ADVANCED PRACTICE NURSING in the Care of Older Adults

SECOND EDITION

LAURIE KENNEDY-MALONE
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I dedicate this book to my husband Chris and my son Brendan for their unwavering support during the writing of this book. To my parents, Nancy and Edward Kennedy, you continue to be models of successful aging that motivate me to continue to be passionate about advanced practice gerontological nursing. To graduates that I have worked with over the years, your continued dedication and expertise in working with older adults is appreciated and admired; thanks to those who also served as contributors to this edition.

—L.K.-M.

To my husband Rick and daughter Erin, thank you both for your patience and encouragement throughout the writing of this book. To my patients, who are also my teachers, thank you for entrusting your health to me; it has been my honor and privilege to serve you and to learn from you.

—L.M.-P.

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—E. G. D.

Preface

With the continued rapid growth of the older adult population, there remains an increased demand for health-care providers to deliver age-specific care and direct disease management. *Advanced Practice Nursing in the Care of Older Adults* will serve as a guide for advanced practice nurses who are privileged to provide care to older adults. Designed as a text for students, as well as a reliable source of evidence-based practice for advanced practice nurses, this book contains information on healthy aging, comprehensive geriatric assessment, and common symptoms and illnesses that present in older adults. Given the complexity of prescribing for older adults taking multiple medications, a new chapter on polypharmacy is included. The book concludes with a chapter on care delivery for patients with chronic illnesses who face end-of-life care.

Throughout the book, case studies are included to provide further practice and review. An important feature of this book is the use of the Strength of Recommendation Taxonomy (SORT) [Ebell, M. H., Siwek, J., Weiss, B. D., Woolf, S. H., Susman, J., Ewigman, B., & Bowman, M. (2004). Strength of recommendation taxonomy (SORT): A patient-centered approach to grading evidence in medical literature *American Family Physician, 69*(3), 548–556], which provides a direct reference to evidence-based practice recommendations for clinicians to consider in the care of older adults.

In Unit I, "The Healthy Older Adult," the first chapter, "Changes with Aging," addresses the normal changes of aging, expected laboratory values in older adults, presentation of illness, atypical disease presentation, bimodal conditions, and the impact of chronic illness on functional capacity. In the second chapter, "Health Promotion," updated information pertaining to health promotion and disease prevention strategies for older adults from *Healthy People 2020* and the U.S. Preventive Services Task Force (USPSTF) is provided, including an immunization schedule and information on the Welcome to Medicare Visit. Also covered is an overview of physical activity, sexual behavior, dental health, and substance use, as well as a section pertaining to the older traveler. Recommendations for exercise and safe physical activity are provided in this unit.

Unit II, "Assessment," opens with a detailed chapter on comprehensive geriatric assessment. Information on physical, functional, and psychological health is delineated, and information on quality of life measures is included. Next is the fifth chapter, "Symptoms and Syndromes," which provides the clinician with a concise description of more than 20 symptoms prevalent in older adults. A rapid reference detailing common contributing factors and associated symptoms and clinical signs that should be worked up for each presenting condition is included. Recommendations for diagnostic tests with accompanying results are used to form a differential diagnosis.

Unit III, "Treating Disorders," provides 11 chapters of concise, updated information pertaining to disease management of illnesses common in older adults, presented by body systems. Each chapter opens with an assessment section that provides the reader with a focused review of systems and the physical examinations needed to obtain pertinent information for diagnosis and treatment of the older adult. Signal symptoms indicating atypical presentation of illness are highlighted at the beginning of each condition. The discussion of each problem and disorder follows a consistent monograph format:

- Signal symptoms
- Description
- Etiology
- Occurrence
- Age
- Ethnicity
- Gender
- Contributing factors
- Signs and symptoms
- Diagnostic tests
- Differential diagnosis
- Treatment
- Follow-up
- Sequelae
- Prevention/prophylaxis
- Referral
- Education

Unit IV, "Complex Illness," addresses complex management of patients requiring chronic illness management, palliative care, and supportive care at end of life, and includes a new chapter on polypharmacy. The text concludes with two appendices—"Physiological Influences of the Aging Process" and "Laboratory Values in the Older Adult"—both of which are ready references for the busy practitioner.

In addition to the content of the book, a **Bonus Chapter, Nutritional Support in the Older Adult,** selected **References,** and other online resources to aid the user in practice and review of the key concepts are available at Davis*Plus*. **Case studies** are provided to support critical thinking and are available for users to complete on their own or for educators to incorporate into their course requirements. To enhance the delivery of competency-based education, the case studies were mapped to the *Adult-Gerontology Primary Care Nurse Practitioner Competencies* (2016).

For the faculty, there are **PowerPoint presentations** and a well-developed **test bank** located on Davis*Plus*. The

Active Classroom Instructors' Guide is an online faculty resource that maps the resources available with the text and includes lecture notes and additional case studies.

This book is written by and for advanced practice nurses involved in the care of older adults across multiple settings of care. While intended as a guide for the management of care for older adults, clinicians are encouraged to deliver individualized, patient-centered care considering the latest clinical practice guidelines on prevention and management of conditions common in older adults.

REFERENCE

National Organization of Nurse Practitioner Faculties. (2016). Adult-Gerontology Acute Care and Primary Care Nurse Practitioner competencies. Retrieved from http://c.ymcdn.com/sites/www.nonpf.org/resource/resmgr/competencies/NP_Adult_Geri_competencies_4.pdf.

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Contents in Brief

unit I

The Healthy Older Adult

CHAPTER

Changes With Aging

Laurie Kennedy-Malone

FUNDAMENTAL CONSIDERATIONS

The aged population continues to be incredibly diverse; it includes some individuals who are nearly twice as old as others and is reflective of growing cultural diversity as well. Knowing what is expected in aging, what diseases are prevalent in aging, and what constitutes successful aging is an immense challenge even for the most skillful advanced clinician. When assessing the aged individual, the advanced practice nurse should be familiar with the range of normal and expected changes associated with aging so that older persons falling outside this range may be identified and interventions taken appropriately and expeditiously.

In the past, wellness was considered the mere absence of disease, but with more information from longitudinal studies of aging, we are learning a great deal about the characteristics of successful physiological and psychosocial agers (O'Brien et al., 2009). A profile of what constitutes successful aging is beginning to emerge, and the illness–health continuum continues to expand to include adults living into old age. This chapter focuses on familiarizing the advanced practice nurse with fundamental underpinnings that serve to guide the approach to assessment and management of the older adult. In addition to appreciating the physiological changes that come with aging, the advanced practice nurse needs to understand how aging changes influence reference laboratory values. Recognizing that presenting features of disease/illness may be different and having a greater awareness of the impact of chronic illness on functional capacity and quality of life provide the advanced practice nurse with a perspective in approaching the older adult that is different from that of younger adults.

PHYSIOLOGICAL CHANGES WITH AGING

The physiological changes associated with the usual aging process have been detailed by system, and the impact of these changes has been described. (These can be found in Appendix A.) Although Appendix A uses a single-system approach, the clinician must be aware that all the systems interact and, in doing so, can increase the older person's vulnerability to illness/disease. For example, the risk of respiratory infection in the geriatric population is considerable, and the physiological influences may include limited chest wall expansion, cilia atrophy, and alterations in the immune system. During the clinical decision-making process, the clinician knowledgeable about physiological changes with aging will be less likely to undertreat a treatable condition. For example, the astute clinician will use the diagnostic process to differentiate the more benign seborrheic keratosis from the more serious melanoma in the aged individual. While educating the older patient, the informed professional will be less likely to attribute a finding to the aging process alone. When clinicians associate findings to aging alone, the older person may conclude that there is no point in changing behavior because the process is inevitable. Additionally, the clinician may take a fatalistic approach and undertreat common conditions such as heart failure and diabetes.

The major impact of these physiological changes can be highlighted with four primary points. First, there is a reduced physiological reserve of most body systems, particularly cardiac, respiratory, and renal. Second, there are reduced homeostatic mechanisms that fail to adjust regulatory systems such as temperature control and fluid and electrolyte balance. Third, there are the changes in the sympathetic response, which contribute to orthostasis and falls, as well as lack of hypoglycemic response. Fourth, there is impaired immunological function: infection risk is greater and autoimmune diseases are more prevalent. The clinician is advised not to be complacent in that some processes previously considered normal, age-related changes are now being refuted. Historically, normal aging studies were conducted using a cross-sectional study method. Today, results are becoming increasingly available from longitudinal studies of aged populations, some of which began in the 1930s (Besdine, 2016; O'Brien et al., 2009).

This more reliable methodology provides some challenges to previously held conclusions. The clinician is encouraged to stay informed regarding the research in expected and successful aging so that this information may be carefully

considered, interpreted, and translated quickly into the clinical setting.

LABORATORY VALUES IN OLDER ADULTS

Healthy individuals of all ages often have asymmetrical distribution of test results. Normality in a statistical sense may be extrapolated incorrectly to normality in terms of health. In addition, the standards previously available to the healthcare worker with which to compare normal laboratory values have been based on randomly collected samples of younger healthy adults. Many factors can influence laboratory value interpretation in the older adult, including the physiological changes with aging, the prevalence of chronic disease, changes in nutritional and fluid intake, lifestyle (including activity), and the medications taken (Dharmarajan & Pitchumoni, 2012).

Clinicians may find that reference ranges, therefore, may be preferable. Reference ranges or intervals, such as age, sex, or race, can be defined demographically. For example, the reference range for older adults might be the intervals within which 95% of persons over age 70 fall. These may be further defined physiologically (e.g., fasting or activity status) or pharmacologically (e.g., medication, tobacco or alcohol use). Even this more precise method does not ensure a healthy sampled population as the standard, and using the reference range method may not differentiate normal aging from disease. The reference values presented for the older adult cohort (see Appendix B) are not necessarily desirable ones. Longitudinal chemical studies support the concept of biochemical individuality; that is, each individual's variation is often much smaller than that of the larger group. Biochemical individuality is of particular importance in detecting asymptomatic abnormalities in older adults. Significant homeostatic disturbances in the same individual may be detected through serial laboratory tests, even though all individual test results may lie within normal limits of the reference interval for the entire group.

The clinician must determine whether a value obtained reflects a normal aging change, a disease, or the potential for disease. Although abnormal laboratory findings are often attributed to old age, rarely are they true aging changes. Misinterpretation of an abnormal laboratory value as an aging change can lead to underdiagnoses and undertreatment in some situations (e.g., anemia or urinary tract infection) and overdiagnosis and overtreatment in others (e.g., hyperglycemia or asymptomatic bacteriuria). At times, the result of a laboratory value may be within the appropriate reference range, yet indicate pathology for the older adult (Dharmarajan & Pitchumoni, 2012). The serum creatinine level may be within the normal range, yet indicate renal impairment in a patient with inadequate protein stores, and different measures might need to be considered. One value of significance to the practitioner with prescriptive privileges is the calculation of creatinine clearance in the estimation of renal function, for instance when dosing enoxaparin (Shaikh & Regal, 2017).

Reduced renal function, particularly the glomerular filtration rate (GFR), affects the clearance of many drugs, and creatinine clearance provides an index of renal function for use in choosing doses of renally eliminated or nephrotoxic drugs (such as digoxin, H_2 blockers, lithium, and water-soluble antibiotics). The Modification of Diet in Renal Disease (MDRD) and Cockcroft-Gault equations both provide useful estimates of the GFR (Boparai & Korc-Grodzicki, 2011). The performance of these two formulas was compared in an older adult population, and the Cockcroft-Gault formula was found to be inferior to the MDRD equation; however, the MDRD equation is not as practical and is more complex to use (Fliser, 2008). The use of serum drug concentration measurements (where these are available) or timed urine specimens is recommended until more acceptable methods of calculating renal function in this population become available.

Finally, when considering which laboratory tests to order, it is worth remembering the doctrine ***primum non nocere,*** to do no harm. Excessive blood sampling may lower the hematocrit; repeated fasting tests may provoke nutritional compromise; and extensive use of tests often requires drugs that may cause adverse reactions. Any risks involved in laboratory testing must be considered with respect to the patient's clinical condition and weighed against the test's expected benefits. The clinician should plan in advance the use for each test result value obtained, especially for less specific or less sensitive tests such as sedimentation rate and serum alkaline phosphatase levels. "Ordering a test requires assessing the likelihood that a patient has specific conditions prior to the order, along with the accuracy of test and as to how it will change management" (Dharmarajan & Pitchumoni, 2012, p. 267). Once laboratory tests are available for review, test results should be discussed with the patients, with abnormal test results interpreted for the aging individual and addressed with the patient and/or caregivers. In addition to understanding the fundamental changes that accompany aging and their influence on interpreting laboratory values and medication management, the advanced practice nurse needs to understand the presenting features of illness/disease in older adults (Dharmarajan & Pitchumoni, 2012).

PRESENTING FEATURES OF ILLNESS/DISEASE IN THE OLDER ADULT

The manifestations of illness and disease in the older adult can be very different, even if the underlying pathological process is the same as in younger individuals. The advanced practice nurse should be aware of what can influence the presentation. Underreporting of symptoms by older adults may occur if they attribute the new sign or symptom to age itself (Amella, 2004). By erroneously associating aging with disease, disuse, and disability, older adults perceive this change as inevitable and either fail to present to the health-care provider or, if they do, fail to challenge the assumption that this represents normal aging. At times an acute symptom such as pain or dyspnea is superimposed on a chronic symptom, and the older adult may not recognize that it represents a new or exacerbated pathology (Bell et al., 2016). The advanced practice nurse is well advised to never attribute something to normal aging without doing a careful and methodical search for a treatable condition.

Certain diseases are more common in the older adult and an understanding of the epidemiology is critical in the interpretation. Certain neoplasms and malignancies such as basal cell carcinoma, chronic lymphocytic leukemia, and prostate cancer have a high prevalence beginning in older adulthood. Neurological conditions such as Parkinson's disease, dementias, stroke, and complex partial seizures are more common to have initial onset in older age. Polymyalgia rheumatica along with giant cell arteritis almost exclusively begins in patients over the age of 50 (Besdine, 2016).

Complicating the care of older adults is when patients develop geriatric syndromes that often involve multiple body systems and have more than one underlying cause (Bell et al., 2016). For patients presenting with one or more of new geriatric giants: frailty, anorexia of aging, sarcopenia, and cognitive impairment, the risk escalates for falls, delirium, injuries, and depression, subsequently placing these patients at dangers for iatrogenic events that could lead to hospitalization, institutionalization, and subsequently, death (Morley, 2017).

Altered Presentation of Illness

Advanced practice nurses managing the care of older adults are challenged to recognize altered, atypical, vague, or even nonspecific signs and symptoms of common conditions in older adults (Auerhahn & Kennedy-Malone, 2010). It is well documented that disease progress may be different for the older adult, especially the frail older adult (Bell et al., 2016). The failure to develop an elevated temperature or fever with an underlying infectious process differs greatly from presentation of illness in a younger patient. The patient with depression may not present with a dysphoric mood but rather agitation and psychotic features. The older adult may present with cardiac manifestations of undiagnosed thyroid disease (Amella, 2004). Additional illustrative examples include jaundice, which is suggestive of viral hepatitis in younger individuals but may represent gallbladder disease or a malignancy in the older adult, and delusions or hallucinations, which are suggestive of bipolar disorder in younger individuals but may represent dementia or medication side effects in the older adult (Williams, 2008).

Because the symptoms or signs of illness or disease may be vague and nonspecific, even a modest change in functional level or behavior should alert the clinician to carefully explore the potential for a treatable condition. Family members or caregivers may report that a patient may no longer be cooperating or participating in individual care. Unusual changes such as these become red flags to the beginning of an atypical presentation of illness. In many cases the progression of the condition is insidious, often presenting as a change in cognition or an alteration in functional status. Other significant changes in patients with altered presentation of illness often include new onset of falls, weakness, fatigue, anorexia, and unexplained tachypnea (Auerhahn & Kennedy-Malone, 2010). Table 1-1 depicts common conditions that often have altered presentation of illness in older adults.

Bimodality of Age of Onset of Clinical Conditions

Understanding of the epidemiology of clinical conditions includes having the knowledge of etiology of the disease,

TABLE 1-1 Presentation of Illness in Older Adults

ILLNESS	ATYPICAL PRESENTATIONS
Acute abdomen	Absence of symptoms or vague symptoms Acute confusion Mild discomfort and constipation Some tachypnea and possibly vague respiratory symptoms Appendicitis pain may begin in right lower quadrant and become diffuse
Depression	Anorexia, vague abdominal complaints, new onset of constipation, insomnia, hyperactivity, lack of sadness
Hyperthyroidism	Hyperthyroidism presenting as "apathetic thyrotoxicosis," i.e., fatigue and weakness; weight loss may result instead of weight gain; patients report palpitations, tachycardia, new onset of atrial fibrillation, and heart failure may occur with undiagnosed hyperthyroidism
Hypothyroidism	Hypothyroidism often presents with confusion and agitation; new onset of anorexia, weight loss, and arthralgias may occur
Malignancy	New or worsening back pain secondary to metastases from slow growing breast masses Silent masses of the bowel
Myocardial infarction (MI)	Absence of chest pain Vague symptoms of fatigue, nausea, and a decrease in functional and cognitive status; classic presentations: dyspnea, epigastric discomfort, weakness, vomiting; history of previous cardiac failure Higher prevalence in females versus males Non-Q-wave MI
Overall infectious diseases process	Absence of fever or low-grade fever Malaise Sepsis without usual leukocytosis and fever Falls, anorexia, new onset of confusion and/or alteration in change in mental status, decrease in usual functional status
Peptic ulcer disease	Absence of abdominal pain, dyspepsia, early satiety Painless, bloodless New onset of confusion, unexplained tachycardia, and/or hypotension
Pneumonia	Absence of fever; mild coughing without copious sputum, especially in dehydrated patients; tachycardia and tachypnea; anorexia and malaise are common; alteration in cognition.
Pulmonary edema	Lack of paroxysmal nocturnal dyspnea or coughing; insidious onset with changes in function, food or fluid intake, or confusion
Tuberculosis (TB)	Atypical signs of TB in older adults include hepatosplenomegaly, abnormalities in liver function tests, and anemia
Urinary tract infection	Absence of fever, worsening mental or functional status, dizziness, anorexia, fatigue, weakness

Source: Amella, E. J. (2004); Bell et al., 2016; Besdine (2016); Chmura & Chan (2006); Peters (2010); Rehman & Qazi (2013); Rowe & Juthani-Mehta, M. (2014); Van Duin (2011); Wester, Dunlop, Melby, Dahle, & Wyller (2013); Williams (2008).

TABLE 1-2 Select Bimodal Presentations of Illness in Younger Adults versus Older Adults

TYPE OF CONDITION	YOUNGER ADULTS	OLDER ADULTS
Dermatological Psoriasis	Late teens to 20s Irregular course which tends to generalize Hereditary factors	50s—males 60s—females Sporadic onset
Gastrointestinal Inflammatory bowel disease Ulcerative colitis (UC) Crohn's disease (CD)	20–40 years old Right lower UC Insidious onset	>60–75 years old a second peak occurs More often older women Proctitis Left-sided UC Higher rates of anemia May present as chronic diarrhea Fistula development Increased cases of associated malnutrition Extraintestinal manifestations including: arthritis spondylitis, uveitis, and erythema nodosum More comorbid conditions May be confused with other forms of colitis
Malignancies Hodgkin's lymphoma	20–30 years old Possible infectious etiology	>50 years old Increased mortality
Neurodegenerative Myasthenia gravis (MG)	Women 20–40 years old More thymus abnormalities	Men—50–70 years old Women—70 years old Dysphonia More frequent ocular form MG Increased rate of AChR seropositivity

Source: Alkhawajah & Oger (2015); del Val (2011); Henseler & Christophers (1985); Louis & Dogu (2007); Montero-Odasso (2006); Shenoy, Maggioncalda, Malik, & Flowers (2011); Smith (2013); Smith, Kassab, Payne, & Beer (1993); Wester, Dunlop, Melby, Dahle, & Wyller (2013); Woon & Lim (2003); Živković, Clemens, & Lacomis (2012).

prevalence and incidence rates, risk factors, age of onset, and gender distribution. There are a number of conditions that are known or suggested to have a bimodal age of onset. In some conditions the difference is not only the decade(s) in life that the disease more likely presents but the dominance of the gender that the condition presents. Myasthenia gravis is one condition that tends to present initially in younger females, with a preponderance in older males (Alkhawajah & Oger, 2015).

Often the presentation of the same illness is different for older adults as compared to their younger counterparts. The onset of the condition may be acute versus progressive, with different symptomatology and clinical signs. For instance, in patients with late onset rheumatoid arthritis the joint involvement is more often in the larger joints such as the shoulder and they experience constitutional symptoms such as fever, malaise, weight loss, and depression (Evcik, 2013). Knowledge of the bimodality of age onset of certain disease conditions will aid the advanced practice nurse in avoiding misdiagnosis or delay in diagnosis due to lack of recognition. Table 1-2 describes medical conditions that present differently in younger versus older adults.

CHRONIC ILLNESS AND FUNCTIONAL CAPACITY

Approximately 80% of those 65 or older have one chronic disease, and 50% have two or more. The most common of these are related to heart disease, arthritis, respiratory problems, cancer, diabetes, and stroke (U.S. Department of Health and Human Services [USDHHS], Centers for Disease Control and Prevention [CDC], 2010). Treating patients with multimorbidities can be very complex and can result in polypharmacy. Patients with multimorbidities are known to have a treatment burden in terms of understanding and self-care management of their conditions. This burden entails not only patients managing the conditions but attending multiple appointments and comprehending and affording complex drug regimens (Wallace et al., 2015).

These conditions often impair functional capacity and limit the person's ability to perform activities of daily living (ADLs) such as bathing and dressing, and instrumental activities of daily living (IADLs) such as managing medications and traveling. More than 25% of community-dwelling Medicare beneficiaries report difficulties performing ADLs, and 14% report difficulties performing IADLs (USDHHS, Administration on Aging [AOA], 2010).

SUMMARY

- Assessment and management of older adults is different from that of younger adults, and it is of critical importance that the advanced practice nurse working with the older adult has the knowledge, skill, and ability to recognize these differences and take them into consideration. This chapter highlighted how the approach of the clinician might be different based on an understanding of the physiological changes of aging and the impact of these changes on medication management and laboratory interpretation; how the presenting features of disease and illness may be different in the older adult; and how the older adult are disproportionately affected with chronic disease and functional impairments.

CHAPTER 2

Health Promotion

Lori Martin-Plank

The concept of health promotion includes activities to which an individual is committed and performs proactively to further his or her health and well-being. This includes not only preventive and health-protective measures but also actualization of one's health potential. The broadest definition, identified by the World Health Organization (WHO), includes healthy lifestyle promotion, creation of supportive environments for health, community action, redirection of health services, and healthy public policy formulation. According to the WHO, by 2050 the world population of those over 60 years old will be at 22%, nearly double what it was in 2015 (WHO, Aging Facts, 2015). In its *Global Strategy and Action Plan for Ageing and Health*, the WHO identifies five priorities for member countries: 1) A commitment to healthy ageing; 2) synchronizing the needs of older persons and health systems; 3) designing age-friendly environments, 4) developing long-term care systems; and 5) research (WHO, 2017). Within the United States, there are several resources for healthy aging, including the Centers for Disease Control and Prevention (CDC) and Health Promotion Web site on Healthy Aging (https://www.cdc.gov/aging/aginginfo/index.htm) and the American Geriatrics Society Health in Aging Web site geared to consumers (http://www.americangeriatrics.org/public).

These resources are available and contain measures that are within the scope of practice for the nurse practitioner (NP) to enhance the visibility of the role while advancing the needs of patients. NPs are in a unique and pivotal position to guide and encourage health-promotion programs and individual efforts. From our nursing background, we bring a holistic orientation to health and wellness, as well as knowledge of developmental tasks and the wellness–illness continuum. Our advanced practice education helps us diagnose and treat patients in a way that supports their return to optimal level of function and/or maximizes their coping abilities within the limits of their existing function. This particular blend of NP competencies is especially valuable in working with older patients. Heterogeneity increases with aging, presenting the NP with the challenge of individualizing health-promotion recommendations for each patient. Most of the literature on older adult health is devoted to treatment of frail older adults, those with geriatric syndromes and dementia (Friedman, Shah, & Hall, 2015). There is a need to develop programs and measure outcomes in promoting health in older adults.

Because older adults have only recently begun to participate in studies on health promotion (Bleijenberg et al., 2017) and because single-focused interventions for health promotion often do not "fit" with the interrelatedness of older adult health-promotion challenges, clear age-specific preventive health guidelines for the older population are scarce. Many disorders in older adults encompass multiple risk factors that involve several systems and interventions to achieve outcomes. This presents a challenge when measuring and synthesizing evidence and reporting outcomes (AGS Guide to Multimorbidity, 2012). Medicare will only pay for A and B level recommendations that meet the U.S. Preventive Services Task Force (USPSTF) stringent evidence guidelines, leaving other beneficial interventions without coverage. Another confounding factor is the way that outcomes for screening are measured in terms of years of life saved. For older adults, quality of life or functional life is a more realistic goal (Friedman, Shah, & Hall, 2015).

The *Healthy People 2020* program has also set specific objectives for prevention in older adults. These include increased use of the Welcome to Medicare visit, an increased percentage of older adults who are up to date on all preventive services, and decreased use of the emergency department for falls by older adults, among others. Because of the focus on chronic disease management and the complexities of multiple comorbidities in older adults, many primary health-care providers are not oriented toward the potential of healthy aging and discount the importance of health promotion in this age group (Friedman et al., 2015).

Current life expectancy is 78.8 years (CDC, National Center for Health Statistics [NCHS], 2017), with many people living to 100 years and beyond. It behooves us to focus on prevention and health promotion in our older patients to maximize the quality of these years. A collaborative plan should include consideration of the patient's health beliefs and goals, present and anticipated levels of function, risks and benefits of proposed interventions, and effectiveness of specific preventive interventions for older adults. The Welcome to Medicare visit provides a good opportunity to focus solely on preventive services and health promotion; this is followed by the Medicare-supported annual prevention visit. Health-promotion activities should be incorporated into every patient encounter, as opposed to being addressed selectively, and should be

individualized to the patient. Recent efforts are being focused on partnering population-based, community-centered programs with personal health initiatives in older adults to make interventions more available and more economical, and to increase socialization opportunities and harness the power of group support.

PRIMARY, SECONDARY, AND TERTIARY PREVENTION

Preventive services are typically divided into the categories of primary, secondary, and tertiary. Primary prevention refers to those activities undertaken to prevent the occurrence of a disease or adverse health condition, including mental health. Health counseling and immunization are examples of primary prevention.

Secondary prevention refers to those tasks directed toward detection of a disease or adverse health condition in an asymptomatic individual who has risk factors but no detectable disease. Screening tests are examples of secondary prevention. The screening test must detect the condition at a stage where it is treatable and a positive outcome is expected after treatment. Mammography for breast cancer screening is an example of secondary prevention.

Tertiary prevention refers to management of existing conditions to prevent disability and minimize complications, striving for optimal level of function and quality of life. Pulmonary rehabilitation for a chronic obstructive pulmonary disease (COPD) patient is an example of tertiary prevention.

HEALTHY LIFESTYLE COUNSELING

The Welcome to Medicare visit (Centers for Medicare and Medicaid, 2011) provides an ideal opportunity for healthy lifestyle counseling. In addition to a thorough history (including some risk assessment, physical activity, diet, and tobacco and alcohol use), home safety and depression assessment are included. The Medicare MedLearn network has a link to guide providers covering all areas (www.cms.gov/Outreach-and-Education/Medicare-Learning-Network-MLN/MLNProducts/downloads//MPS_QRI_IPPE001a.pdf). Healthy lifestyle counseling should be addressed at each visit, using brief motivational interviewing (Lee, Choi, Royce, Yum, & Chair, 2016; Moral et al., 2015; Purath, Keck, & Fitzgerald, 2014).

Physical Activity

Older adults are the least active age group, although recent trends show an increase in physical activity in older adults. The American College of Sports Medicine and the American Heart Association issued updated recommendations for physical activity in all adults, with additional recommendations tailored to adults over age 65 and adults aged 50 to 64 with chronic conditions that are clinically significant or result in functional limitations (Nelson et al., 2007). Counseling on physical activity should include any type of activity that the patient is able and willing to do. The health benefits of regular physical activity are well documented and include flexibility, increased muscle mass, maintenance of desirable weight, decreased insulin resistance, decreased peripheral vascular resistance, lower blood pressure, and a sense of well-being. Whenever possible, the components of aerobic activity (low to moderate), flexibility, balance, and strengthening (weight training) should be included, and the physical activity prescription should be individualized to the patient. Active hobbies, such as gardening, golfing, tennis, dancing, bowling, hiking, and swimming, are beneficial. Tai chi and yoga are helpful for stretching and balance. Frail older adults or older adults with impaired mobility can benefit from armchair exercises and modified ambulation.

A recent study showed a decrease in risk of death in older adults with multiple morbidities who engaged in regular physical activity (Martinez-Gomez, Guallar-Castillon, Garcia-Esquinas, Bandinelli, & Rodriguez-Artalejo, 2017). Patients need to be reassured that expensive equipment or fitness memberships are not necessary to increase physical activity; motivation is the key. There are also many community exercise programs targeted to older adults, as well as Web sites that can be shared if the patient has access to the Internet; these include Exercise is Medicine, the American Association of Retired Persons (AARP), the National Council on Aging (NCOA), and the National Institute on Aging (NIA). Many programs are now targeting exercise and brain health to prevent cognitive decline. Several government and community group programs have handouts for patients.

Before embarking on an exercise program, all patients should have an evaluation of health history, including medications, present physical activity and functional level, potential barriers to exercise, and a physical examination. Older adults with known or suspected cardiac risk factors should have a stress test before engaging in vigorous exercise. All participants should be reminded of the need for adequate hydration and use of caution during extreme weather conditions.

Nutrition

The heterogeneity of older adults is evident in the wide range of nutritional issues affecting them. Before initiating counseling on diet, obtain baseline information on current dietary intake and activity pattern, and combine this with height and weight data and other health status information. For patients in the long-term care setting, this information is obtained easily from chart documentation. For community-dwelling older adults, a brief nutrition screening tool such as the Mini Nutritional Assessment (MNA) can be helpful. The abbreviated MNA consists of six questions, and there is a patient self-questionnaire that can be downloaded or mailed in advance of the visit. The MNA Web site contains a section on tools for clinicians, including a user guide and streaming video (www.mna-elderly.com/tools_for_clinicians.html). It is available in multiple languages as well.

The importance of a healthy, balanced diet to the overall health of older adults cannot be overemphasized. Chronic illness and disability can interfere with the activities of daily living such as shopping or preparing meals. Financial hardship can limit food choices. Prescribed medications can affect absorption of nutrients, sense of taste, or appetite. Depression or social isolation can contribute to poor nutrition. Another problem commonly seen in community-dwelling older adults is obesity. Close to one-half of U.S. older adults are overweight

or obese (Batsis et al., 2017). A recent systematic review of interventions targeting obesity in older adults found that programs combining physical activity and diet had better outcomes, although the findings were of low to moderate quality (Batsis et al., 2017). There is a need for further research to guide clinical interventions to decrease obesity. Overweight and obesity are associated with heart disease, certain types of cancer, type 2 diabetes, breathing difficulties, stroke, arthritis, and psychological problems. Although there is a decline in the prevalence of overweight and obesity after age 60 years, it remains a problem for many older adults. It is a major risk factor for decreased mobility and functional impairment as well as a cardiovascular risk. General guidelines for dietary counseling include:

- Limit fat and cholesterol.
- Maintain a balanced caloric intake.
- Emphasize the inclusion of grains, fruits, and vegetables daily.
- Ensure an adequate calcium intake, especially for women.
- Limit alcohol, if used, to one drink daily for women and two drinks daily for men: one drink = 12 oz beer, 5 oz wine, or 1.5 oz of 80-proof distilled spirits.

Safety

Prevention of injury in the older adult is of paramount importance to continuing functionality and quality of life. Part of this counseling involves reinforcement of extant recommendations, including wearing lap and shoulder seat belts in a motor vehicle, avoiding drinking and driving, having working smoke detectors in the residence, and keeping hot water set below 120°F. For older adults who drive a motor vehicle, periodic assessment of their ongoing ability to drive safely is vital to the older adult and the public at large. Most motor vehicle accidents involve young drivers and older drivers.

Two recommendations are especially important for ensuring the safety of the older adult. The first involves the safe storage and removal of firearms. Possession of a firearm combined with depression, caregiver stress, irreversible illness, or decline in functional abilities can invite self-inflicted injury, suicide pacts, or other acts of violence. Counsel patients to avoid firearms in the home and to use alternative means for self-protection such as alarm systems and pepper mace spray. The second recommendation involves the prevention of falls, the leading cause of nonfatal injuries and unintentional death from injury in older persons. Certain combinations of physiological and environmental factors place some patients at increased risk. About 85% of falls occur at home, in the later part of the day. Office-based providers can assess for falls by asking if there is a history of falling and by performing the Get Up and Go test in the office. If indicated, evaluation of risk factors and a home safety assessment by a home health nurse or a geriatric assessment team can provide direction for preventive intervention and education. Potential recommendations include exercise programs to build strength, modification of environmental hazards, monitoring and adjusting of medications, external protection against falling on hard surfaces, and measures to increase bone density. If urinary incontinence is a contributing factor, a urological work-up may be indicated.

Falls are often alarming to patients and families. In some cases, family members may desire nursing home placement for the patient because of a fall. In other cases, patients may be fearful of ambulation as a result of a fall. Falls also pose a challenge in the long-term care environment. Education and counseling combined with an assessment of the patient's environment are helpful. Keeping water, call bell, telephone, and other necessities available and toileting regularly can minimize the potential for falling in nursing home patients. Several home safety checklists are available on the Internet and can be given to patients for self-assessment.

Aging in Place

In the past few years technology such as SMART HOMES and sensors have been introduced to facilitate aging in place. Most of these technologies are still in their infancy but offer hope in delaying institutionalization and promoting healthy functioning at home. Other programs, primarily in European countries, are targeting at-risk "oldest old" and have designed comprehensive interventions to maintain them at home (Dahlin-Ivanoff et al., 2017). It is anticipated that more technological interventions will be implemented to promote healthy aging in place in the near future.

Sexual Behavior

Assumptions regarding lack of sexual expression in the healthy older adult are unfounded. With the possibility of pregnancy eliminated, many mature adults feel less restraint. As a result of divorce or widowhood, they may seek satisfaction with new partners yet lack the knowledge to protect themselves from sexually transmitted diseases, especially HIV. More than 42% of those living with HIV in the United States in 2013 were people more than 50 years old (CDC, 2017); 39% of deaths from HIV in 2014 were in adults more than 55 years of age (CDC, 2017). Older adults need to be taught methods for safe sex with use of a barrier to avoid sexually transmitted diseases, including HIV and hepatitis B. Using the patient's sexual history, explore patient needs, preferences, and medical or psychological obstacles to sexual expression. This exploration facilitates counseling and interventions to promote healthy sexual behavior.

Dental Health

Counseling regarding dental health in the older adult includes the need for regular visits to the dental-care provider, daily flossing, and brushing with fluoride toothpaste. Many elders have dentures or dental implants and assume that dental checkups are no longer necessary. Oral screening for cancer is still indicated, as is periodic assessment of denture fit and functionality. Another concern is for the condition of the remaining teeth of some older adults. Periodontal disease, erosion of dentin, or other problems may render the teeth nonfunctional for chewing and a potential source for infection. Dependence on others for transportation or lack of available dental resources for patients in long-term care settings further complicates the problem. Caregivers simply may overlook this aspect of preventive health or financial considerations may preclude treatment. Patient and family education regarding dental health is essential.

Substance Use

Counseling about substance use (tobacco, alcohol, and drugs) and injury prevention can be combined naturally

within the issue of safety. Smoking is the leading preventable cause of death in the United States. Smoking cessation yields many benefits to former smokers in terms of reduction of risk for several chronic illnesses and stabilization of pulmonary status. Clear and specific guidelines are available to help health-care providers advise tobacco users to quit and to provide them with follow-up encouragement and relapse prevention management. Quitting smoking may not be a choice for the institutionalized older adult but rather dictated by the policy of the institution. Health-care providers can offer support and encouragement, emphasizing the positive health changes that will result.

Counseling regarding alcohol or other drug use can be preventive or interventional, depending on the initial assessment. Use the Michigan Alcohol Screening Test (MAST), the CAGE questionnaire, or the Alcohol Use Disorders Identification Test (AUDIT) to assess risk. Emphasize the dangers of drinking and driving and the increased risk of falling while under the influence of alcohol or any drug that acts on the central nervous system. Teach patients about the coincidental interactions between alcohol and many prescription drugs, over-the-counter preparations such as acetaminophen, and herbal remedies. The contribution of alcohol abuse to problems such as insomnia, depression, aggressive behaviors, and deteriorating social relationships, should be addressed. Likewise, the problem of dependence on prescription drugs such as analgesics, hypnotics, tranquilizers, and anxiolytics, should be assessed and addressed. Counseling in the form of individual follow-up sessions, group support, or outpatient or inpatient rehabilitation may be indicated. In a group-living situation, the governing body (i.e., resident council) may become involved if the patient's behavior threatens the safety or well-being of the other group members.

SCREENING AND PREVENTION

The following table contains the areas of screening and prevention that are covered by Medicare for older adults and the relevant evidence to support these initiatives.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The USPSTF concludes that the current evidence is insufficient to assess the balance of benefits and harms of screening for hearing loss in asymptomatic adults aged 50 years or older.	I	Moyer for the USPSTF, 2012
The USPSTF recommends that clinicians screen for HIV infection in adolescents and adults aged 15 to 65 years. Younger adolescents and older adults who are at increased risk should also be screened.	A	Moyer for the USPSTF, 2013
The USPSTF recommends that clinicians screen adults aged 18 years or older for alcohol misuse and provide persons engaged in risky or hazardous drinking with brief behavioral counseling interventions to reduce alcohol misuse.	B	Currently under revision, 2017 https://www.uspreventiveservices taskforce.org/Page/Document/ UpdateSummaryDraft/unhealthy -alcohol-use-in-adolescents-and -adults-including-pregnant-women -screening-and-behavioral -counseling-interventions
The USPSTF recommends that clinicians ask all adults about tobacco use, advise them to stop using tobacco, and provide behavioral interventions and U.S. Food and Drug Administration (FDA)–approved pharmacotherapy for cessation to adults who use tobacco.	A	Siu for the USPSTF, 2015
The USPSTF recommends screening for depression in the general adult population, including pregnant and postpartum women. Screening should be implemented with adequate systems in place to ensure accurate diagnosis, effective treatment, and appropriate follow-up.	B	Siu for the USPSTF, 2016

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The USPSTF recommends screening for high blood pressure in adults aged 18 years or older. The USPSTF recommends obtaining measurements outside of the clinical setting for diagnostic confirmation before starting treatment.	A	Siu for the USPSTF, 2015
The USPSTF recommends screening for abnormal blood glucose as part of cardiovascular risk assessment in adults aged 40 to 70 years who are overweight or obese. Clinicians should offer or refer patients with abnormal blood glucose to intensive behavioral counseling interventions to promote a healthful diet and physical activity.	B	USPSTF, 2015
The USPSTF recommends that adults without a history of cardiovascular disease (CVD) (i.e., symptomatic coronary artery disease or ischemic stroke) use a low- to moderate-dose statin for the prevention of CVD events and mortality when all of the following criteria are met: 1) they are aged 40 to 75 years; 2) they have one or more CVD risk factors (i.e., dyslipidemia, diabetes, hypertension, or smoking); and 3) they have a calculated 10-year risk of a cardiovascular event of 10% or greater. Identification of dyslipidemia and calculation of 10-year CVD event risk requires universal lipids screening in adults aged 40 to 75 years. See the "Clinical Considerations" section for more information on lipids screening and the assessment of cardiovascular risk.	B	Bibbins-Domingo for the USPSTF, 2016
The USPSTF recommends one-time screening for abdominal aortic aneurysm (AAA) by ultrasonography in men aged 65 to 75 years who have ever smoked.		Topic under revision, June 2017 by the USPSTF https://www.uspreventiveservicestaskforce.org/Page/Name/topics-in-progress
The USPSTF recommends screening all adults for obesity. Clinicians should offer or refer patients with a BMI of 30 kg/m^2 or higher to intensive, multicomponent behavioral interventions.		Topic under revision, 2017 https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryDraft/obesity-in-adults-interventions1
The USPSTF recommends biennial screening mammography for women aged 50 to 74 years.	B	Siu for the USPSTF, 2016
The USPSTF concludes that the current evidence is insufficient to assess the balance of benefits and harms of screening mammography in women aged 75 years or older.	I	Siu for the USPSTF, 2016
The USPSTF concludes that the current evidence is insufficient to assess the balance of benefits and harms of screening for impaired visual acuity in older adults.	I	Siu for the USPSTF, 2016

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The USPSTF recommends screening for osteoporosis in women aged 65 years and older and in younger women whose fracture risk is equal to or greater than that of a 65-year-old white woman who has no additional risk factors.	B	USPSTE, 2018
Prostate cancer is common in older men.		USPSTF update in progress, 2017 https://screeningforprostatecancer.org/
Screening for cognitive impairment in older adults.		USPSTF update in progress, 2017 https://www.uspreventiveservicestaskforce.org/Page/Document/UpdateSummaryDraft/cognitive-impairment-in-older-adults-screening1
The USPSTF recommends screening for colorectal cancer (CRC) starting at age 50 years and continuing until age 75 years (A recommendation).	A	USPSTF, *JAMA*, 2016; 315(23):2564–2575. doi:10.1001/jama.2016.5989
The decision to screen for colorectal cancer (CRC) in adults aged 76 to 85 years should be an individual one, taking into account the patient's overall health and prior screening history (C recommendation).	C	
The decision to initiate low-dose aspirin use for the primary prevention of CVD and CRC in adults aged 60 to 69 years who have a 10% or greater 10-year CVD risk should be an individual one. Persons who are not at increased risk for bleeding, have a life expectancy of at least 10 years, and are willing to take low-dose aspirin daily for at least 10 years are more likely to benefit. Persons who place a higher value on the potential benefits than the potential harms may choose to initiate low-dose aspirin.	C	Bibbins-Domingo for the USPSTF, 2016
The current evidence is insufficient to assess the balance of benefits and harms of initiating aspirin use for the primary prevention of CVD and CRC in adults aged 70 years or older.	I	Bibbins-Domingo for the USPSTF, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

TABLE 2-1 2017 Adult Immunization Schedule for Older Adults

VACCINE	AGE GROUP	DOSING
PCV13	Over 65	Single dose; for those with chronic health conditions may administer a dose before age 65 and boost with a second dose after age 65
PPSV23	Over 65	Give 1 year after PCV13
Diphtheria-tetanus-pertussis (Tdap)	Any adult—one time substitute for Td	Single dose
Tetanus diphtheria (Td)	Every 10 years after single dose of DTaP	Single dose every 10 years
Influenza	All adults	Annual
Hepatitis B	All with risk factors due to lifestyle, history of diabetes mellitus	Three doses
Herpes zoster (HZV)	Adults aged 50 years or older regardless of whether they had a prior episode of herpes zoster; immunize those who have had Zostavax with Shingrix	Two doses age 50 or older (Shingrix)

See full details and recommendations for special populations and contraindications at: Recommended adult immunization schedule—2017. Retrieved from https://www.cdc.gov/vaccines/schedules/hcp/adult.html

IMMUNIZATIONS

Influenza vaccine is now recommended annually for all adults over 50 years old, unless contraindicated (Table 2-1). Residents of long-term care facilities that house persons with chronic medical conditions are at especially high risk for developing the disease. Health-care workers also should receive the vaccine, preferably before the end of October (Resnick, 2018). Patients with a severe egg allergy or severe reaction to the influenza vaccine in the past and patients with a prior history of Guillain-Barré syndrome should talk with their health-care provider before getting the vaccine.

Tetanus-diphtheria toxoids with acellular pertussis (Tdap) vaccine is administered as a once-in-a-lifetime booster to every adult. Following this, a tetanus-diphtheria (Td) booster is recommended every 10 years.

Pneumococcal vaccine is recommended as follows: Administer a one-time dose to PCV13-naïve adults at age 65 years, followed by a dose of PPSV23 12 months later.

Hepatitis B vaccine is recommended for high-risk persons such as IV drug users, persons who are sexually active with multiple partners, those living with someone with chronic hepatitis B, patients less than 60 years old with diabetes, and all desiring protection from hepatitis B. The initial dose is given, followed 1 month later by the second dose, then the third dose is given 4 to 6 months after the second dose.

Shingrix is a new vaccine for zoster and is recommended over Zostavax. It is administered in two doses. The second dose can be given from 2 to 6 months after the initial one. Persons who have had Zostavax should now be immunized with Shingrix (Resnick, 2018). Those who have had a prior episode of zoster should be vaccinated (CDC, Adult Immunization Schedule, 2017; www.immunize.org).

TRAVEL AND LEISURE

Travel can be one of the most enjoyable experiences one can have. People travel to see new things, understand the world and themselves better, visit friends and family, return to the land of their ancestors, volunteer, challenge themselves, and because it is fun. They travel alone, in groups, and with their families. They go on cruises and they go on safaris. They stay in five-star resorts and in host family homes. They take planes, buses, trains, jeeps, and rickshaws. They scuba dive, hike the Himalayas, and bicycle in Tuscany. They teach and learn. They volunteer in Haiti, Ghana, and Honduras. But travel can pose some unique health risks for the older traveler. The gerontology NP in primary care can provide pre-trip advice to help ensure healthy and safe travel.

Travel Health and Nursing

Travel health is an interdisciplinary specialty that has grown out of the need to protect travelers from illness and injury. It developed in the 1970s as infectious disease and tropical medicine clinicians treating returned travelers recognized that many of the problems they encountered could be prevented by pre-travel evaluations, immunizations, chemoprophylaxis, and counseling about safety, food and water, and insect precautions.

In 1991 the International Society of Travel Medicine (ISTM) (www.istm.org) was formed and established an international body of knowledge to define travel medicine. It is the only body offering an examination to demonstrate competences for physicians, NPs, registered nurses (RNs), physician assistants (PAs), and pharmacists. Those who pass are awarded a Certificate in Travel Health. The American Travel Health Nurses Association (ATHNA) (www.athna.org) was formed in 2004 to promote and support travel health nursing in North America. ATHNA provides many resources for nurses and NPs who specialize in travel health, as well as for those in primary care.

Travel health is rapidly evolving and growing as a specialty but is also growing as a part of primary care. NPs will need to know how to evaluate older travelers and develop a plan of care to keep them healthy while they travel. They need to know how, when, and where to refer to a travel health specialist. The majority of travelers who could benefit from pre-travel consultations do not receive them (Zuckerman,

Brunette, & Leggat, 2015). NPs are in a unique position to educate patients and the public about the benefits of this service.

Medical Tourism

Some people travel abroad to receive medical care. The most common procedures sought outside the United States include joint replacements, cosmetic surgery, cataract lens replacement, cardiovascular surgery, and dental procedures. Some people travel for organ transplants, stem cell treatments, and anti-aging and cancer treatments not available or banned in the United States. While there are some options for high quality, less expensive health care abroad, the patient must do careful research to ensure safe, quality care is rendered by competent providers. Traveling with a patient advocate is advised, as elders recovering from surgery or who are in poor health are more vulnerable to complications and being taken advantage of (Brunette & Kozarsky, 2018).

The Older Traveler

Some of the physiological and psychosocial changes that can occur with aging pose special risks during travel. How a patient functions at home may not be indicative of how well he or she will function in an unfamiliar environment. Diminished musculoskeletal strength, agility, mobility, and endurance can affect a person's ability to navigate safely. Travel often involves more walking and standing than an elder may be accustomed to. Many places abroad are not handicapped accessible. Uneven stairs and walkways, lack of handrails, and lack of elevators can be challenging.

Cardiopulmonary function can decrease with age and contribute to fatigue. Long flights in low humidity and lowered oxygen, in cramped seats, can increase risk of thromboembolic events. The older adults are at increased risk of altitude illness, which affects cardiac and cerebral functioning. Increased air pollution is a significant problem in many countries and affects pulmonary function. The ability to tolerate temperature changes affects the older traveler. Heat and humidity can aggravate underlying conditions, and older travelers will become dehydrated more easily. They are more prone to thermal damage in colder climates. Central nervous system changes affect the older traveler's ability to deal with the stresses of travel. It can be anxiety inducing to be in a place where everything is so different—the language, food, customs, and climate. Jet lag is harder to cope with as one ages. Any traveler can experience unexpected delays and be without food and sleep for hours. This can take an even greater toll on the older traveler.

Sensory changes may result in decreased hearing, which is especially difficult on airplanes or trains with background engine noise. Decreased vision can result in greater risk of injuries. Decreased night vision, longer reaction time, and driving on unfamiliar, poorly lit roads increase the risk of accidents. Bathroom stops may be at longer intervals than needed for an older traveler with diminished bladder capacity or any degree of incontinence. Some facilities may consist only of holes in the floor that the elder may have to balance over to use.

Older travelers have less robust immune systems. Fever is not always a reliable indicator of illness in the older adult. Seroconversion rates decrease with age, rendering some vaccines less effective for older travelers. Although many older travelers are very healthy, many have comorbidities that contribute to the development of health problems abroad. Patients with chronic disease that is well managed at home may decompensate in foreign environments because of heat, humidity, altitude, fatigue, changes in diet, and exposure to infectious diseases.

It is very important that older travelers know what to do if they become ill or injured away from home. Advise the traveler to obtain travel health insurance that includes emergency medical evacuation and repatriation of remains. Medicare does not cover the cost of health care outside the United States. Have the patient bring a hard or electronic copy of his or her medical history, medication list, allergies, and copies of pertinent imaging studies or electrocardiograms (EKGs). The NP with expertise in gerontology can provide pre-travel care that will not only reduce the morbidity and mortality associated with travel but also enhance the elder's travel experience. When destinations or itineraries are complicated or when a patient's condition poses special risks, a visit to or a consultation with a travel health specialist is warranted.

Preparing the Elder in a Primary Care Setting for Travel

To develop an individualized pre-travel plan of care, the NP needs to evaluate the traveler, the destination, and the itinerary. Assessing the traveler consists of reviewing these areas:

- Current health status—stability of preexisting conditions
- Past medical history
- Medications and allergies
- Diet
- Mental status
- Immunization status

CURRENT MEDICAL STATUS

Ideally, the traveler should be seen at least 6 to 8 weeks before the trip to allow for time to optimize preexisting chronic disease and adequate immune response to vaccine-preventable diseases (Gerstenlauer, 2017). Evaluate the patient's current medications. Simplifying medication schedules enhances compliance. Are there any that do not need to be taken on this trip? Are there any factors that will affect your patient's ability to take any medications during travel? Does the patient know how to adjust medication schedules to accommodate air travel and time zone changes? All prescription medications should be brought in original bottles and not in unlabeled pill containers. If your patient gets his or her prescriptions in 90-day supplies, give the patient new prescriptions for smaller amounts for travel, including a few extra in case of delays. Does the patient need to bring a wheelchair, walker, glucometer, hearing aids, C Pap, or nebulizer? Remind them to check all batteries and bring extras. Is adequate electricity reliably available at a current that will work with the equipment? Will adapters be needed and will they work properly?

MEDICATIONS AND ALLERGIES

- Is the patient taking any medication that could prove life threatening if lost or stolen? If so, is it accessible at the patient's destination?

- Does the patient have any life-threatening allergies?
- Does the patient take any medications that require refrigeration? Decompose from heat and humidity? Require syringes? Need a nebulizer?
- Is the patient on oxygen? If so, he or she must notify the airline well in advance of travel.

All medications should be packed in carry-on luggage, not in checked bags. Certain countries restrict bringing in any controlled substances and some other drugs whether legally prescribed in the United States or not. Caution patients about purchasing pharmaceuticals abroad which may be cheaper but also could be counterfeit.

DIET

Does the patient have any special dietary restrictions? Airlines offer diabetic and vegetarian options but may not offer gluten-free or sodium-restricted comestibles. These must be ordered in advance. Cruise ships accommodate many specialty diets but also offer many temptations. Restaurant menus in many countries do not list all the ingredients in the dishes offered, which can be problematic for those with severe food allergies.

MENTAL STATUS

Short-term memory decreases with age. Many elders cope with these changes by adhering to routines that travel may disrupt. Misplacing passports, room keys, or wallets or not remembering hotel names or addresses can be distressing. Family members or travel companions may need to offer additional assistance. Advise that elders carry a hotel's business card that includes the hotel's name, address, and phone number. Taking photographs of hotels, cruise ships, or tour company's names can help the memory impaired who may become confused.

IMMUNIZATION STATUS

All ***routine*** immunizations should be current. This includes influenza, pneumococcal, Td/Tdap (tetanus, diphtheria, and acellular pertussis), zoster, and for some, hepatitis B vaccination. The current schedule of adult vaccination recommendations from the CDC (updated February 2016) is available at http://www.cdc.gov/vaccines/schedules/hcp/adult.html. Certain vaccines may be ***recommended*** based on destination, and some vaccines are ***required*** for entry into some African and South American countries (Table 2-2) (Brunette & Kozarsky, 2018).

Yellow fever vaccinations can only be given by certified yellow fever centers. If the patient is seeing a primary care provider before getting a yellow fever vaccine, be aware of the live virus vaccine rule (Brunette & Kozarsky, 2018). Yellow fever and herpes zoster vaccine are the only live virus vaccines that people over age 50 receive. Immune response can be impaired if live virus vaccines are given within a 28- to 30-day interval of each other. Yellow fever vaccine is not effective until 10 days after administration. If the NP gives a patient a herpes zoster vaccine, that patient cannot receive a yellow fever vaccine for 30 days. If the patient is required to have a yellow fever vaccine for travel, he or she cannot enter a yellow fever country until 10 days after receiving the yellow fever vaccine (or 40 days after receiving a herpes zoster vaccine). If the administration of a herpes zoster vaccine precludes the administration of a time-sensitive yellow fever vaccine, travel plans could be interrupted, with serious financial consequences for the traveler. If the NP has any questions about when to vaccinate a patient whose trip is imminent, discuss this with a travel health specialist. If a patient receives a yellow fever vaccine, he or she cannot receive a herpes zoster vaccine for 28 days. The patient may receive both vaccines on the same day with no decrease in immune response (Brunette & Kozarsky, 2018). Typhoid oral vaccine is a live ***bacterial*** vaccine and will not interfere with live ***viral*** vaccine administration.

TABLE 2-2 Adult Vaccinations for Travel

RECOMMENDED ADULT VACCINATIONS FOR TRAVEL	REQUIRED ADULT VACCINATIONS FOR TRAVEL
Hepatitis A; others are specific to area where traveling including hepatitis B, typhoid, polio, meningococcal, Japanese encephalitis, rabies	Yellow fever for some African and South American countries Meningococcal for Saudi Arabia during the Hajj

After assessing the destination and itinerary (see the following section), decide which vaccines to recommend for this specific patient for this specific trip. The most common vaccines used for protecting travelers are hepatitis A, hepatitis B, typhoid fever, yellow fever, adult booster polio, Japanese encephalitis, meningococcal, and rabies. If the NP does not have access to these vaccines, referral to a travel health specialist will be needed. To help the patient make an informed decision about which recommended vaccines to receive, consider the indications, contraindications, side effects, timing of doses for immune response, and costs. Medicare will cover hepatitis B in very limited patient populations and will not cover the cost of the other recommended vaccines (Official U.S. Government Web site for Medicare, n.d.). Federal regulations require the NP to give patients Vaccine Information Statements (VISs), which are available in many different languages at http://www.cdc.gov/vaccines/hcp/vis/index.html. The most important vaccine a traveler should receive is the influenza vaccine.

All patients should have a copy of their immunization record. If a patient has an incomplete vaccine series, continue the series but do not restart it. For example, if a patient received one dose of hepatitis A several years ago but never received the second, final injection, give the next and final dose now. If the patient received only one dose of hepatitis B years ago, give the second dose now and the third dose 5 months from now, and the series will be complete (Immunization Action Coalition, n.d.).

If a patient cannot complete a series before travel, partial immunization may confer enough protection. Some vaccines can be given on an accelerated schedule; otherwise, do not give a vaccine sooner than the recommended interval between doses. One dose of hepatitis A given just before travel will confer enough protection to make it worth giving to the last-minute traveler. Hepatitis A and B vaccines are also available as a combined vaccine given at 0, 1, and 6 months. If there are at least 21 days before the patient's departure, the vaccine can be given in an accelerated schedule of 0, 7, and 21 to 30 days with a booster at 12 months (Brunette

& Kozarsky, 2018). Typhoid fever vaccine is available in two forms, a single-dose injectable and orally as a series of four capsules given every other day for 1 week. The oral vaccine then takes a week to be effective. The injectable vaccine needs to be boostered every 2 years and the oral vaccine at 5 years, if needed. Before prescribing the oral form, be sure the patient can comply with the proper administration (Brunette & Kozarsky, 2018).

Because of worldwide efforts to eradicate polio, only a few countries require adults to get a polio booster for travel. If your patient has had polio in the past, he or she does not need vaccination. If your patient has been fully vaccinated for polio, a single booster dose as an adult will protect him or her. Japanese encephalitis vaccine, meningococcal vaccine, and rabies pre-exposure vaccine are not usually administered to elders for travel in a primary care setting. Japanese encephalitis vaccine is only advised for long-term stays in high-risk areas. Meningococcal vaccine is only licensed for people ages 2 to 55. Depending on the country visited, length of stay, and potential exposure to rabid animals, decisions regarding rabies pre-exposure vaccine are usually made with a travel health specialist. Cost and vaccine availability play a role in deciding pre-exposure vaccination (Brunette & Kozarsky, 2018). It is important to warn travelers of the risk of rabies and to educate them in animal bite prevention strategies, especially concerning dogs, which are the biggest vector for rabies worldwide.

The *CDC Yellow Book 2018* is an invaluable resource for vaccine administration and is available in paperback and online in its entirety for free and as a free app. Clinicians and travelers can research recommendations for specific countries at http://wwwnc.cdc.gov/travel/. The Advisory Council on Immunization Practice (ACIP) has a section on its Web site (www.immunize.org) called Ask the Expert that can be searched for answers to immunization questions. Listings for travel health specialists and clinics can be found at the ISTM Web site (www.istm.org) and the CDC Web site (wwwnc.cdc.gov/travel/page/find-clinic).

ASSESSING THE DESTINATION AND ITINERARY

The NP needs to know where the traveler is going and what he or she will do there to provide anticipatory guidance for risk reduction. The NP may decide to refer to a travel clinic for the remainder of the pre-trip evaluation. Either way, the following overview will help the NP to understand what comprises a comprehensive pre-travel evaluation.

Mexico, China, India, Peru, Kenya, Australia, Europe, and the Caribbean all pose different risks for the elder traveler. The time of year, duration of the trip, type of accommodations, modes of transportation, and purpose of the trip all influence travel risk. A 70-year-old couple going to the Dominican Republic who plan to stay at an all-inclusive resort will need different advice than a 70-year-old couple traveling to build an orphanage and staying in a host family home. There are Web sites that offer current advice about destinations that the NP can use. Some are free, such as the CDC Web site, and some are subscription based, such as Shoreland (www.travax.com) and Tropimed (www.tropimed.com).

The most common risks for travel to tropical, subtropical, and low-resource countries are trauma and food-, water-, and insect-borne diseases. Because so much information is relayed at the pre-travel visit, it is important to provide written information for review at home. Handouts for insect, food, and water precautions are found on the CDC Web site.

SAFETY

Accidents and injuries are the most common cause of preventable death and disability for travelers. Tourists are 10 times more likely to die from trauma than infectious disease (Brunette & Kozarsky, 2018). The most common risks travelers face from trauma result from motor vehicle, pedestrian, and water accidents; personal safety/crime; natural disasters and environmental hazards; and animal-related injuries.

In many parts of the world, roads and vehicles are poorly maintained. Seat belts and helmets are typically unavailable. Roads are shared by pedestrians, animals, motorbikes, bicycles, trucks, buses, and rickshaws. Traffic accidents are more common because the traveler is unfamiliar with the roads, may be driving on the opposite side of the road, and may need to drive to the left on roundabouts. Road signs and lighting are suboptimal. To help prevent accidents, always take these precautions:

- Wear seat belts when available.
- Avoid driving or riding at night in underdeveloped countries.
- Avoid motorcycles and mopeds altogether.
- Do not drive impaired by alcohol or fatigue or ride with someone who is.
- Do not use cell phones or text or type on GPS systems while driving.
- Avoid overcrowded buses and vans.
- Be alert when crossing the street.

The Association for International Road Travel (ASIRT) has a very helpful Web site with patient handouts for accident prevention (www.asirt.org).

Drowning is the leading cause of accidental death for U.S. travelers visiting countries where water recreation is a major activity (Brunette & Kozarsky, 2018). Warn travelers to avoid diving into shallow water or swimming or boating under the influence of alcohol and remind them to use life vests. Boating in unfamiliar waters, and in unfamiliar boats, increases risk of accidents. Many countries do not have laws and regulations concerning public safety to the same extent as the United States. Outfitters may not be as careful about safety. Divers Alert Network (DAN) (www.DAN.org) provides education, support, and travel and health insurance worldwide for scuba divers. They staff a 24-hour hotline for divers and health-care providers for medical support at 1-919-684-9111.

The U.S. Department of State Web site provides current information about worldwide safety and security at https://travel.state.gov/content/travel/en.html. Personal crime rates vary from country to country. While risk of harm from terrorist activities is low, travelers should be aware of emergency exits and routes and know the location of the U.S. embassy in the countries they are in. Homicide was the second leading cause of death from injuries for U.S. citizens. In Honduras, Colombia, Guatemala, and Haiti, 38% to 52% of all deaths from injuries for U.S. travelers were homicides (Brunette & Kozarsky, 2018). Older adult travelers are seen as wealthy, vulnerable targets and can travel in high-poverty, high-crime areas. Travel during civil unrest or travel at night in unfamiliar places increases the risk of assault.

FOODBORNE AND WATERBORNE ILLNESSES

It is not safe to drink the water in many places in the world. Food and water precautions must be carefully adhered to for preventing disease. The easiest way for travelers to remember this is to tell them to boil it, peel it, cook it, or forget it. Bottled water; carbonated beverages without ice; and coffee, tea, and alcohol are safe to drink. Do not brush teeth or soak dentures in tap water. Some travelers tie a ribbon or small rope around the faucet to remind them not to drink the water. Despite best efforts, traveler's diarrhea is common, and often the health-care provider will prescribe drugs for the traveler to take with him or her in the event that this occurs. Mild diarrhea can be treated with bismuth subsalicylate (BSS). Significant diarrhea can be treated very effectively with azithromycin, 500 mg once daily for 1 to 3 days (Brunette & Kozarsky, 2018), as 90% of the cases are bacterial. Oral rehydration solution (ORS) packets or effervescent tablets help prevent dehydration.

INSECT-BORNE DISEASES

Dengue, chikungunya, and Zika viruses have spread throughout much of the temperate zones of the world, including Central America, the Caribbean, and southern areas of the United States, areas where large numbers of older travelers go for extended stays in the winter. When traveling to areas where insect-borne diseases are a risk, the older traveler should be advised to avoid bites day and night by using insect repellents on exposed skin, treating clothing with permethrin prior to packing, and covering up with clothing, hats, and footwear. Morbidity and mortality for Zika is greater in the older adult. Because there is still much to be learned about this infection, the NP needs to stay informed about new developments at https://www.cdc.gov/zika/. NPs can subscribe to *Morbidity and Mortality Weekly Review* (MMWR) for free at http://www.cdc.gov/mmwr/mmwrsubscribe.html and receive the table of contents and links to articles when published.

Prevention of malaria is essential for any traveler going to a malaria-endemic area, regardless of age. Malaria is more severe in the older adult, and mortality risk from malaria increases with age (Zuckerman et al., 2015). When traveling to malaria-endemic areas, travelers should always practice bite avoidance and consider chemoprophylaxis depending on their specific itinerary and season of travel. Mosquitoes that transmit malaria bite from sunset to sunrise. If a traveler is taking a cruise and spends the day in port in a malaria-endemic area but returns to the ship before sunset, insect repellent and protective clothing may suffice.

Malaria chemoprophylaxis is generally well tolerated in the older adult. Drug choice depends on the destination, side effects, drug interactions, and cost. All medications are started before travel, taken during travel, and taken for a period of time after travel. The CDC provides maps of malaria-endemic areas and guidelines for prescribing (Brunette & Kozarsky, 2018). It is important for patients who are being evaluated for fever or flu-like symptoms for up to a year post travel tell their health-care providers that they have traveled so that insect-borne diseases can be ruled out.

Motion Sickness

Motion sickness can be prevented with pharmacological and nonpharmacological methods. Many large, modern cruise ships are designed to greatly reduce motion sickness. Commonly used medications for motion sickness include dimenhydrinate, promethazine, diphenhydramine, and anticholinergic agents such as scopolamine. Review possible side effects and drug and alcohol interactions carefully. Methods such as closing your eyes, focusing on the horizon line, and acupressure wrist bands can also be helpful.

Jet Lag

Jet lag can be mitigated by getting proper rest before travel and maintaining proper hydration during flight. Zolpidem has been shown to be more effective in the treatment of jet lag than melatonin (Suhner, Schlagenhauf, & Hofer, 2001). The patient should try a dose of zolpidem at bedtime at home before travel to test tolerance. Confusion, ataxia, and falls are potential side effects of zolpidem.

Heat and Humidity

Hyperthermia occurs more frequently in the older adult. Acclimatization to heat and humidity may take several days. Many of the drugs frequently used by the older adult, such as antihistamines, anticholinergics, calcium channel blockers and beta blockers, diuretics, and anti-Parkinson's medications, impair thermoregulation. The older adult traveler should use caution in hot and humid environments, drink adequate fluids, avoid caffeine and alcohol, and avoid overexertion. Self-treatment measures may also include using ORS packets/tablets in clean bottled water.

Altitude Illness

Altitude illness can range from mild shortness of breath to life-threatening acute mountain sickness (AMS). Prior tolerance of altitude does not predict future tolerance. Those with underlying cardiopulmonary disease may experience greater hypoxia. Because the body's normal response to lowered oxygen is to increase the pulse rate, beta blockers can reduce the body's compensatory response. Acetazolamide is used to prevent AMS but needs to be started 24 to 48 hours before ascent, and side effects versus benefits need to be carefully evaluated. If symptoms of AMS develop, descent, if possible, is the best treatment. Many areas, such as Cusco, Peru, are experienced in treating AMS in tourists because it is such a common occurrence.

Respiratory Infections

Low humidity from airline travel and exposure to crowds and air pollution will make the older adult more prone to respiratory infections during travel. If your patient has chronic pulmonary disease, consider giving him or her an antibiotic to self-treat, if infection occurs.

Sexually Transmitted Infections

Older people may be at increased danger from sexually transmitted infections (STIs) because of the decreased perception of risk by both health-care providers and patients, resulting in less screening and treatment. Older adults are less likely to practice safe sex and use condoms. The rate of casual sex increases with travel (Jong & Sanford, 2008). Encounters may be with fellow travelers, locals, or commercial sex workers. Many older women are told they do not need Pap smears based on their age alone. Current sexual history may

determine the need for continued screening for STIs, human papillomavirus (HPV), and cervical cancer.

Fitness for Travel

Travel can be strenuous. Airports and cruise ships are huge. If a trip involves a higher level of activity than what travelers are accustomed to, they need to be sure they are fit for that trip. Tour operators will often give specific suggestions for fitness for walking, hiking, or bicycling trips but not for sightseeing tours. Each traveler should bring a first aid kit and an emergency dental kit and have a plan for getting health care abroad if needed.

National and international travel by the older adult will continue to increase. A knowledgeable NP, either alone or in conjunction with a travel health specialist, can prepare the older patient to safely enjoy travel to many destinations around the world (Table 2-3). It is imperative to not only protect our elder travelers but to help prevent the importation of infectious diseases back to communities in the United States.

TABLE 2-3 Online Resources for Travel Health

Travel Clinic Locator and Certified Yellow Fever Centers	CDC: https://wwwnc.cdc.gov/travel/page/find-clinic ISTM: http://istmsite.membershipsoftware.org/AF_CstmClinicDirectory.asp
Practice Protocols and Standing Orders	ATHNA: www.athna.org ISTM: www.istm.org ACIP: www.immunize.org
Immunizations	CDC: wwwnc.cdc.gov/travel ACIP: www.immunize.org
Safety and Accident Prevention	Association for International Road Travel: www.asirt.org U.S. Department of State: www.state.gov/travel/
Continuing Education in Travel Health	ATHNA, nursing: www.athna.org CDC, nursing: CDC: www2a.cdc.gov/TCEOnline/ ISTM, general: www.istm.org
Vaccine Information Statements	CDC: https://www.cdc.gov/vaccines/index.html See quick link for Vaccine Information Sheets (VIS).
Journals	*Journal of Travel Medicine:* www.istm.org
Destination Information—subscription	Tropimed: www.tropimed.com Shoreland—Travax: www.shoreland.com
Destination Information—free	CDC: https://wwwnc.cdc.gov/travel

SUMMARY

- Evidence-based health promotion for older adults is an evolving science. As the population of older adults increases, lifestyle management for prevention of chronic illness, self-management of chronic conditions, safety, and quality-of-life issues will be more at the forefront. NPs are well positioned to advance health-promotion efforts and keep older adults healthy and functional (Gerstenlauer, 2017).

CASE STUDY

J. S. is a 66-year-old African American woman who presents to your practice for a well-adult physical checkup. She is widowed and works part-time as a mental health technician to support herself. Family history includes father deceased from a stroke at age 50 years, mother living with hypertension and type 2 diabetes mellitus, a half-sister deceased with breast cancer, and a brother with pancreatic cancer and coronary artery disease and end-stage renal disease secondary to type 2 diabetes mellitus.

J. S. has not seen a health-care provider for several years because she had no health insurance. Now she has Medicare, so she is coming in for care. She is a former smoker who quit 5 years ago after smoking 1 pack per day since age 20 years. She has four grown children, all of whom live nearby, and she has eight grandchildren. Vital signs are blood pressure (BP) 150/92, heart rate (HR) 76 (reg), respiratory rate 18 breaths per minute (bpm) (afebrile), and body mass index (BMI) 32.1.

1. What additional subjective data are you seeking?
2. What additional objective data will you be assessing for?
3. What national guidelines are appropriate to consider?
4. What tests will you order?
5. Are there any screening tools that you want to use?
6. What are the priorities for primary, secondary, and tertiary prevention?
7. What is your plan of care?
8. Are there any *Healthy People 2020* objectives that you should consider?
9. What additional patient teaching may be needed?
10. Will you be looking for a consultation?

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CHAPTER 3

Exercise in Older Adults

Lori Martin-Plank

Current population statistics (www.agingstats.gov) indicate that Americans over 65 years of age now represent the most rapidly growing segment of the U.S. population, comprising 15% of the population in 2014. As those born from 1946 to 1964 enter the over-65 age group, these numbers will increase exponentially, expected to reach 21% by 2030, and with that growth is an anticipated skyrocketing of medical costs for chronic health conditions. Lifestyle interventions at any stage can mitigate the effects of chronic illness (Hupin et al., 2015), but they are usually given short shrift during patient encounters (Weiss et al., 2012). Many providers feel inadequately prepared to initiate realistic discussions about lifestyle changes with older adult patients. According to one report, less than 32% of primary care providers and health professionals offer exercise counseling or educate older patients on the benefits of physical activity during an office visit and physical examination (Barnes & Schoenborn, 2012).

Recent statistics related to exercise in older adults (Federal Interagency Forum on Aging Statistics, 2016) show that 12% of adults over age 65 years had an exercise program that met federal physical activity guidelines in 2014, compared to 6% in 1998. This report (Older Americans, 2016) also reveals an increase in obesity in older adults, from 22% in 1988 to 1994 to 35% in 2011 to 2014. Women over 65 years old were more obese than men. These facts underscore the need for a new paradigm to promote increased physical activity in older adults as part of a program of lifestyle intervention for wellness and quality of life. Nurse practitioners are uniquely positioned to assume the primary role in health promotion of both healthy persons and those with chronic illness.

AVAILABLE RESOURCES

Guidelines and position statements for increasing physical activity in adults and older adults have been issued by several authorities, including the American College of Sports Medicine (ACSM) (Chodzo-Zajko et al., 2009; Nelson et al., 2007), American Heart Association (AHA) (Artinian et al., 2010; Marcus et al., 2006), U.S. Preventive Services Task Force (Moyer, 2012), U.S. Department of Health and Human Services (USDHHS) (Physical Activity Guidelines Advisory Committee, 2008). The *Healthy People 2020* initiative has several sections dedicated to health promotion in older adults. Numerous agencies, such as the Centers for Disease Control and Prevention (CDC), American Osteopathic Association (AOA), National Institute on Aging, and Center for Medicare and Medicaid, have programs to promote wellness and quality of life in older adults, including physical activity projects. The ACSM has a Web site, Exercise is Medicine, with extensive resources for providers and the public (http://exerciseismedicine.org), and the Preventive Cardiovascular Nurses Association has a Heart Healthy Toolbox (http://pcna.net/clinical-tools/tools-for-healthcare-providers/heart-healthy-toolbox) with a variety of free tools for use by clinicians. Despite the availability of these tools, they are largely unknown to most health-care providers, who are focused on the day-to-day activities of disease management (Friedman, Shah, & Hall, 2015).

Since the publication of the exercise guidelines for older adults, several studies have demonstrated that adapting these guidelines to individuals and specific populations, such as the oldest old and those with frailty (Bleijenberg et al., 2017; Dahlin-Ivanoff et al., 2015; Lee & Kim, 2017; Tarazona-Santabalbina, 2016), can be successful. One large study at the Veterans Association (VA) also demonstrated the effectiveness of physical activity counseling in primary care in decreasing overall health-care costs (Cowper et al., 2017). Additional studies are now focusing on the relationship of physical activity and cognitive changes, some including diet and supplements (Jonasson et al., 2015; Kobe et al., 2016; Tarazona-Santabalbina et al., 2016; Tse, Wong, & Lee, 2015). While this area (cognitive benefits and exercise) is inconclusive, the future holds promise.

Another area that is being addressed is the relationship of exercise of any kind to a decrease in mortality, both with and without cardiovascular risk factors (Hupin et al., 2015; Kim et al., 2017; Martinez-Gomez et al., 2017; Morey, 2017; Villareal et al., 2017). Specific programs for older adults with osteoarthritis are also effective (Bartels et al., 2016; Fransen et al., 2015). Finally, the American Academy of Family Physicians (AAFP) (Lee, Jackson, & Richardson, 2017; Pescatello, 2014; Zaleski et al., 2015) and other exercise professionals

have given detailed descriptions of how to design an exercise program and write a prescription, as well as how to engage the patient. LeTorneau and Goodman (2014) and Lee and colleagues (2016) have addressed the role of motivational interviewing in facilitating older adult engagement in physical activity and exercise.

BARRIERS AND FACILITATORS TO EXERCISE FOR OLDER ADULTS

In 2006, a subcommittee of the AHA Council on Nutrition, Physical Activity, and Metabolism undertook a review of existing physical activity intervention studies, focusing on subpopulations and settings to gain perspective on the state of the science and to help identify future goals (Marcus et al., 2006). Studies that focused on older adults and exercise found that short-term interventions, both individual and group, face-to-face and by phone, were effective in increasing physical activity when delivered as part of a multifaceted program of educational and cognitive-behavioral participation. Health education alone was ineffective in this population. Health-care personnel–recommended physical activity and an exercise prescription were effective in the short term. None of the studies reviewed had a long enough duration to measure persistence of effort (Marcus et al., 2006).

Patient Barriers

- Lack of time
- Perceived need for equipment
- Perceived barrier to beginning exercise/physical activity
- Disability or functional limitation
- Unsafe neighborhood or weather conditions
- No parks or walking trails
- Depression
- High body mass index (BMI)
- Lack of motivation
- Interpersonal loss or significant life event
- Ignorance of what to do

Patient Facilitators

- Social support
- Positive self-efficacy
- Motivation to engage in physical activity
- Good health, no functional limitations
- Frequent contact with prescriber
- Regular schedule, planned program
- Satisfaction with program
- Insurance incentive
- Improvement in mobility or health condition
- Staff (of exercise facility) support (Franco et al., 2015; Lee et al., 2017; Simmonds et al., 2015)

Medical Contraindications for Exercise Therapy

- Unstable angina
- Uncompensated heart failure
- Severe anemia
- Uncontrolled blood glucose
- Unstable aortic aneurysm
- Uncontrolled hypertension or tachycardia
- Severe dehydration or heat stroke
- Low oxygen saturation

PLAN FOR INCORPORATING EXERCISE INTO PATIENT ENCOUNTER

The ACSM has designed a program for primary care providers called Exercise is Medicine. The goal of this program is to counsel the patient at every visit to increase physical activity. In conjunction with the AHA, the ACSM has also issued guidelines for physical activity in older adults (Nelson et al., 2007). These guidelines can be used to incorporate physical activity and exercise recommendations into patient encounters.

All authorities agree that whenever possible, older adults should engage in physical activities that strengthen muscles, maintain flexibility, promote good balance, and are aerobic in nature; further qualifications include the development of a plan incorporating both preventive and therapeutic goals, and risk management and reducing sedentary lifestyle (Artinian et al., 2010; Chodzko-Zajko et al., 2009; National Institute on Aging, 2011; Nelson et al., 2007). The preferred amount of exercise is 30 minutes per day for 5 days a week of moderate exercise; if weight management is part of this, 60 minutes per day is advised (Villareal et al., 2016). This can be broken up into as little as 10-minute intervals throughout the day. Any increase in physical activity is desirable and has some value over sedentary behavior (Nelson et al., 2007). If older adults with chronic health conditions cannot achieve the 150 minutes per week of aerobic activity recommended, they should be as physically active as possible within the constraints of their conditions and abilities (Chodzko-Zajko et al., 2009).

The initial Welcome to Medicare visit can focus on healthy lifestyle counseling, including assessment of current physical activity level and specific guidance or exercise prescription for the patient. In the case of a patient with disabilities or functional limitations, referral to physical therapy or a community-based program targeting those with physical restrictions is appropriate and more effective. For those who are already very active, an exercise physiologist or ACSM-certified personal fitness trainer can help them to maximize their program benefits. Following up at each visit will emphasize the importance of ongoing exercise in maintaining or promoting healthy aging. Health coaches from the insurance carrier can also be used to keep the patient engaged. Strategies, such as using motivational interviewing (LeTourneau & Goodman, 2014), can be helpful in persuading the patient to adopt a new behavior.

The annual Medicare wellness visit can also be used to reinforce or expand on the importance of lifestyle changes. Exercise prescriptions that are individualized to fit the patient's abilities and preferences are most likely to be implemented (Lee et al., 2017; Zaleski et al., 2015). Actually

handing the patient a program or exercise prescription is more effective than just speaking about it. Knowing resources that are available in the community for group exercise or individual walking programs is a valuable adjunct to counseling. Knowledge of Internet resources for computer-savvy patients is also helpful. Goal setting and self-monitoring by the patient are very effective. A clinician who is aware of some common excuses (e.g., lack of time, no equipment) can counter these with positive suggestions, such as acknowledging the 10-minute benefit, using stairs in the home, or walking around the block.

Clinician judgment should be exercised in assessing the patient and prescribing an exercise routine. Major authorities agree that all adults who do not have symptoms and have no diagnosed chronic health condition, such as osteoarthritis, heart disease, or diabetes mellitus, do not need to consult with a health-care professional about increasing physical activity (Office of Disease Prevention and Health Promotion, 2008). Patients with chronic conditions should consult a health-care provider for physical activity goals that are realistic and safe (Physical Activity Guidelines Advisory Committee, 2008). Healthy adult men over 45 years old and healthy women over 55 years old who are considering a ***vigorous*** exercise program need health-care provider screening and routine stress testing. Sedentary older adults and all adults with cardiac disease or strong risk factors should undergo screening and stress test if they are undertaking a ***vigorous*** exercise program (Lee et al., 2017).

KEY GUIDELINES FOR SAFE PHYSICAL ACTIVITY (PHYSICAL ACTIVITY GUIDELINES ADVISORY COMMITTEE, 2008)

To perform physical activity safely and reduce risk of injuries and other adverse events, people should:

- Understand the risks and yet be confident that physical activity is safe for almost everyone.
- Choose to do types of physical activities that are appropriate for their current fitness level and health goals, because some activities are safer than others.
- Increase physical activity gradually over time whenever more activity is necessary to meet guidelines or health goals. Inactive people should "start low and go slow" by gradually increasing how often and how long activities are done.
- Protect themselves by using appropriate gear and sports equipment, looking for safe environments, following rules and policies, and making sensible choices about when, where, and how to be active.
- Be under the care of a health-care provider if they have chronic conditions or symptoms. People with chronic conditions and symptoms should consult their health-care provider about the types and amounts of activity appropriate for them.

Examples of Common Health Conditions in Older Adults With Exercise Recommendations

HEALTH CONDITION: CONSIDER COMORBIDITIES FOR ALL	RECOMMENDED ACTIVITIES: START LOW INTENSITY, GO SLOW	COMMENTS: CONSIDER COMORBIDITIES FOR ALL
Osteoarthritis	Walking, aquatic activities, tai chi, resistance exercises, cycling	Vary type and intensity to avoid overstressing joints; heated pool
Coronary artery disease	Walking, treadmill walking, cycle ergometry	Supervised program with BP and heart rate monitoring
Congestive heart failure	Walking, treadmill walking, cycle ergometry	Individualize to client; supervised program
Type 2 diabetes mellitus	Resistive, aerobic, aquatic, recreational activities	Proper shoe fit; may need insulin reduction if insulin dependent
Anxiety disorders	Walking, biking, weight lifting	If able to do high-intensity exercise, this benefits anxiety
Depression	Walking, cycling, recreational activities	Group participation helpful to keep patient engaged
Fibromyalgia	Aerobic, aquatic therapy, strengthening, tai chi, Pilates	Heated pool, gentle stretches, counsel about possible increased pain initially
Chronic obstructive pulmonary disease	Cycle ergometer, treadmill walking; individualize	Supervised program—consider pulmonary rehabilitation program
Chronic venous insufficiency	Walking, standing exercises	Supervised program
Osteoporosis	Weight-bearing exercises, weight training	Assess balance and risk for falls before beginning
Parkinson's disease	Walking, treadmill walking, stationary bike, dancing, tai chi, Pilates, boxing	Assess balance and risk for falls before beginning; American Parkinson's Disease Association resources
Peripheral arterial disease	Lower extremity exercises, treadmill walking, walking	Very short intervals initially, progress as tolerated
Age-related sleep disorders	Tai chi, walking, aquatherapy, biking	Assess balance and risk for falls before beginning
Dementia	Walking, recreational activities	Provide safe environment, assess fall risk and ability to participate

Source: Adapted from Goodman, C., & Helgeson, K. (2011). *Exercise prescriptions for medical conditions.* Philadelphia: F.A. Davis.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
To promote and maintain health, older adults need moderate-intensity aerobic activity for a minimum of 30 min on 5 days each week or vigorous-intensity aerobic activity for a minimum of 20 min on 3 days each week.	A	Nelson et al., 2007
To promote and maintain health and physical independence, older adults will benefit from performing activities that maintain or increase muscular strength and endurance for a minimum of 2 days each week. It is recommended that 8–10 exercises be performed on two or more nonconsecutive days per week using the major muscle groups.	A	Nelson et al., 2007
Participation in aerobic and muscle-strengthening activities above minimum recommended amounts provides additional health benefits and results in higher levels of physical fitness.	A	Nelson et al., 2007
To maintain the flexibility necessary for regular physical activity and daily life, older adults should perform activities that maintain or increase flexibility on at least 2 days each week for at least 10 min each day.	A	Nelson et al., 2007
To reduce risk of injury from falls, community-dwelling older adults with substantial risk of falls (e.g., with frequent falls or mobility problems) should perform exercises that maintain or improve balance.	A	Nelson et al., 2007 Lee & Kim, 2017
Older adults with one or more medical conditions for which physical activity is therapeutic should perform physical activity in the manner that effectively and safely treats the condition(s).	A	Nelson et al., 2007 Lee & Kim, 2017
Older adults should have a plan for obtaining sufficient physical activity that addresses each recommended type of activity. Those with chronic conditions for which activity is therapeutic should have a single plan that integrates prevention and treatment. For older adults who are not active at recommended levels, plans should include a gradual (or stepwise) approach to increase physical activity over time. Many months of activity at less than recommended levels is appropriate for some older adults (e.g., those with low fitness), because they increase activity in a stepwise manner. Older adults should also be encouraged to self-monitor their physical activity on a regular basis and to reevaluate plans as their abilities improve or as their health status changes.	C	Nelson et al., 2007

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about SORT evidence rating system, go to www.aafp.org/afpsort.xml.

SUMMARY

- There is strong evidence from a variety of sources that increasing physical activity in older adults will improve health and quality of life in both healthy adults and those with chronic health conditions (Avers, 2016; Bleijenberg et al., 2016; Dahlin-Ivanoff et al., 2016; Fransen et al., 2016; Hupin et al., 2015; Martinez-Gomez et al., 2017; Tarazona-Santabalbina, 2016). Primary care practitioners can play an important role in promoting physical activity for all older adult patients at every visit, using an individualized approach. Resources are available to assist with this effort; further research is needed regarding effective counseling methods and outcomes.

CASE STUDY

A. G., a 72-year-old man, comes in for follow-up of laboratory test results. He has coronary artery disease (CAD), hypertension (controlled), impaired fasting glucose (IFG), benign prostatic hyperplasia, and mild degenerative joint disease (DJD). Since being diagnosed with IFG he has cut back on soda consumption and is also seeing a dietitian for counseling. He is a former smoker (last use 12 years ago) and admits to being a "couch potato." Current medications: Lisinopril 20 mg orally (PO) daily, hydrochlorothiazide 12.5 mg PO daily, simvastatin 20 mg PO daily in the evening, and tamsulosin 0.4 mg PO daily. He has a prescription for nitroglycerin 0.4 mg sublingually as needed for chest pain but has never needed to use it. He occasionally takes Tylenol Arthritis Pain or Aleve if the DJD bothers him. Vital signs are blood pressure (BP) 134/72, heart rate (HR) 70 (reg), respiratory rate 18 breaths per minute (bpm) (afebrile), and BMI 34.2. Pulse oximetry is 99% on room air. Laboratory test results are unremarkable except for high-density lipoprotein (HDL) 32 and fasting blood sugar (FBS) 125. Mr. G expresses frustration at the lack of change in his fasting glucose despite cutting back on soda. He asks you, "What else can I do? I don't want to get diabetes." You encourage him to increase his physical activity level gradually, beginning with walking.

1. What strategies will you use to promote increased physical activity in this patient?
2. What additional objective data will you be assessing for?
3. What national guidelines are appropriate to consider?
4. Will you need to order any tests before he begins this program? If so, what tests?
5. Are there any screening tools that you want to use?
6. What is your plan of care?
7. Are there any *Healthy People 2020* objectives that you should consider?
8. What additional patient teaching may be needed?
9. How will you follow up on the planned increase in physical activity?

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unit II

Assessment

CHAPTER 4

Comprehensive Geriatric Assessment

Evelyn Duffy

Because older individuals represent a richly diverse population, the components of assessment may vary from person to person. People age at different rates and within one person organ systems age at different rates. Psychosocial adaptations, environmental supports, and functional ability can differ dramatically among individuals of the same chronological age. A comprehensive approach to geriatric assessment is recommended because the physical health of the older adult is inextricably related to functional ability, psychosocial health, and a safe and enabling environment. Older individuals who can benefit the most from this approach are the vulnerable older adult (those at risk for decline) and the frail older adult (those already demonstrating decline). This population has less physiological reserve and is at increased risk of iatrogenic complications. Comprehensive geriatric assessment (CGA) helps not only to diagnose treatable conditions and improve patient outcomes, but also to identify potentially preventable conditions. It facilitates the goal of patient-centered care.

CGA has been defined as a multidimensional, interprofessional, diagnostic process to identify care needs, plan care, and improve outcomes for older people (Morley, Little, & Berg-Weger, 2017; Ramani, Furmedge, & Reddy, 2014). A recent Cochrane systematic review of inpatient CGA versus usual care of older adults admitted to acute care or inpatient rehabilitation concluded that older adults were more likely to be alive and in their own homes if they received a CGA (Ellis et al., 2017). These findings were consistent with the work of Partridge, Harari, Martin, and Dhesi (2014) who noted that preoperative CGA is likely to positively impact postoperative outcomes. CGA may be undertaken by an individual provider initially, with the interprofessional team called in for selected patients. Advanced practice nurses may be a part of a team that works together to complete a CGA, or may apply the concepts of CGA to their own patients. Domains of CGA include physical health, psychological health, socio-environmental supports, mobility, functional status, and measure of quality of life. A number of geriatric assessment instruments are focused on these domains. These tools can facilitate the assessment, diagnosis, and evaluation processes, and those with the most clinical utility are highlighted in this chapter. Suggested dimensions within each CGA domain are identified in Table 4-1. Each of these domains is described in greater detail as follows.

PHYSICAL HEALTH

The physical assessment of the older adult includes all the components of a conventional medical history (chief complaint, history of present illness, past history, family and social history, and a review of systems), the conventional physical examination, and appropriate diagnostics based on the findings. The approach to the physical assessment process, however, needs to be tailored to the older adult. Assessment begins with observation of the patient's appearance, language, and behaviors. Does the individual look the stated age? Are they dressed appropriately? Is there evidence of neglect in self-care? What is the manner and content of speech? What behaviors are observed, including facial expressions, eye contact, gestures, or abnormal movements? If a family member or caregiver accompanies the patient, how do they interact?

Interviewing begins before the physical examination and typically continues throughout the examination. Many providers send a geriatric history-taking form to the patient in advance so that they or their family can complete it and the details can be reviewed and expanded on during the visit. Any impairment in vision, hearing, speech, or cognition may affect the patient's ability to communicate effectively. Impairments should be addressed and measures taken to accommodate them during the visit, to prevent the common trap of talking to the family or caregiver rather than the older adult in order to facilitate the visit. The patient's permission needs to be acquired before interviewing the family in most cases. The input of the family or caregiver may provide valuable information about the patient's behavioral or functional changes, which can indicate a change in the patient's overall health status. It is important to address what the patient or

TABLE 4-1 Comprehensive Geriatric Assessment

DOMAINS OF COMPREHENSIVE GERIATRIC ASSESSMENT	DIMENSIONS OF COMPREHENSIVE GERIATRIC ASSESSMENT
Physical health	History taking Physical examination Diagnostics Nutritional assessment Medication review
Functional health	Activities of daily living Instrumental activities of daily living Sensory assessment (hearing, vision) Gait and balance
Psychological health	Cognitive disorders (delirium, dementia, mild cognitive impairment) Affective disorders (depression, anxiety) Spiritual well-being
Socioenvironmental supports	Social network and support Living situation Environmental safety Economic resources
Quality of life measures	Physical conditions Social conditions Environmental conditions Personal resources (mental health, life perspective) Preferences for care

their family considers significant, which often differs from what the provider considers significant.

Many older adults suffer from multiple chronic diseases that require monitoring. There may not be a new chief concern, but if one is reported it is important to put it into the context of the patient's previous health and illness. The history of chronic diseases should be identified and updated with any symptoms of decompensation. If a new concern is identified, a history of present illness is recorded using the seven dimensions of symptomatology as a guide (location and radiation, quantity and quality, aggravating and alleviating factors, associated symptoms and signs, absence of associated symptoms and signs, evolution and course of the symptom, and effect of the symptom on normal daily activities). After taking the history, the provider should summarize for the patient what was heard. This gives the patient the opportunity to clarify or deny any parts of the discussion.

Medication

Medication review should be completed at every encounter. Because older adults are the population most likely to suffer from multiple chronic diseases, they are also most likely to take more medications, both prescribed and over the counter. The more medications a person is taking the more likely they are to have an adverse drug reaction. The most frequently used criteria for evaluating drug use are the Beers Criteria for Potentially Inappropriate Medication Use in Older Adults, first published in 1991 and most recently updated in 2015 (Beers, 1997; American Geriatrics Society [AGS], 2015). A subsequent scale was developed to address some of the limitations

TABLE 4-2 Medication Appropriateness Index Criteria

Is there an indication for the drug?
Is the medication effective for the condition?
Is the dosage correct?
Are the directions correct?
Are the directions practical?
Are there clinically significant drug–drug interactions?
Are there clinically significant drug–disease/condition interactions?
Is there unnecessary duplication with other drugs?
Is the duration of therapy acceptable?
Is this drug the least expensive alternative compared with others of equal usefulness?

of the Beers criteria, the Medication Appropriateness Index (Hanlon & Schmader, 2013; Hanlon et al., 1992; Table 4-2). It is a validated tool that can be used along with the Beers criteria to determine the appropriateness of prescribed medications for older adult patients.

Appropriate prescribing often means that multiple medications are required for adequate management of chronic disease. Appropriate prescribing may actually require adding a medication, such as an angiotensin-converting enzyme (ACE) inhibitor for a patient with diabetes. Older versions of the Beers criteria had identified limitations that led to the development of more explicit criteria, including the Screening Tool of Older Persons' Prescriptions (STOPP) and the Screening Tool to Alert Doctors to Right Treatment (START) (Gallagher et al., 2008). These tools address the issue of appropriate prescribing more specifically and are organized by organ system. The START tool includes medications that may need to be added, while the STOPP tool lists those that should be assessed for possible elimination. An updated version was published in 2015, STOPP/START Criteria for Potentially Inappropriate Prescribing in Older People: Version 2 (O'Mahony et al., 2015).

Examination

The collection of objective data usually begins with the vital signs. These may be completed by ancillary staff, but any abnormalities need to be verified by the provider. A common issue is the necessity to rule out orthostatic hypotension, which is prevalent in older adults due to volume depletion and medication effects. If concerns indicate the possibility of orthostatic drop, the provider needs to accurately measure the blood pressure (BP). The patient should lie down for 5 minutes and then have the BP and pulse rate measured. Have the patient stand and wait for a full minute, then repeat the BP and pulse rate measurements. Note any symptoms of light-headedness or dizziness and any drop in systolic BP greater than or equal to 20 mm Hg or diastolic BP greater than or equal to 10 mm Hg. An increase of heart rate of at least 30 beats per minute (bpm) is also abnormal. BP and pulse rate measurements should be repeated 3 minutes after standing, and if they have returned to baseline the patient has not demonstrated true orthostasis, but you will have confirmed the cause of light-headedness or dizziness.

Pulse oximetry is now readily available in practice settings, and this gives a measure of oxygen saturation. If the respiratory rate is greater than 20 respirations per minute it is classified as tachypnea and may be a sign of infection

or sepsis, lung disease, heart failure, or a metabolic disorder. Weight is another measurement in which accuracy is important, because it is a marker for both nutritional and fluid status. Pain is the fifth vital sign and quantifying pain can be accomplished in various ways, although older adults may prefer verbally descriptive scales.

The physical examination is of critical significance in CGA. In examination of the older adult, functional assessment is as important as careful examination of each body system. In younger adults, neurological and musculoskeletal examination may be less of a focus because health of those systems is obvious, but older adults may have unrecognized muscle weakness, limitations in range of joint motion, or contractures requiring thorough examination. Sensory loss often precludes the ability of the older adult to live independently, and visual and hearing acuity is part of the functional assessment. Measured visual acuity (corrected with lenses) equal to or worse than 20/40 constitutes a visual impairment. Screening for hearing loss can be accomplished using a handheld audiometer, which is more accurate than the whispered voice test. Abnormality in the six cardinal fields of gaze with nystagmus or lack of a downward gaze may reveal neurological disease. Otoscopic examination can identify cerumen impaction, a common cause of conductive hearing loss.

A comprehensive physical examination of the older adult is recommended, and a sample examination is identified in Table 4-3. After completing the examination, discuss the general findings with the patient. Patient teaching should reinforce the positive behaviors taken to improve or maintain health and teach self-assessment and self-care management strategies.

Diagnostics

Chapter 1 discusses the changes associated with aging that are reflected in laboratory values. This section provides some general guidelines on when to order testing. The reader is referred to the disease sections of this text for more specific guidelines. Tests used to screen geriatric patients and tests used to aid in diagnosis have different objectives. Screening can be defined as the presumptive identification of unrecognized disease or defect by the application of tests, examinations, or other procedures. The screening parameters for those over age 65 years, which include laboratory and diagnostic testing, are covered in Chapter 2, Health Promotion.

One of the considerations in ordering tests for diagnostic purposes is whether the test result will alter the diagnosis, prognosis, or management of a condition. Some guidelines for use of diagnostics in decision making include: What is the prevalence of the disease being screened for with the test? Is the test necessary to make the diagnosis? Will the test be accepted and tolerated by the patient? Does the cost or benefit of the test outweigh the risk? Is it the least invasive test available? Can the results be interpreted? Will the results change the treatment of the patient? What is the sensitivity and specificity of the test?

To be of diagnostic value, a test for a given disease must produce patient results that differ substantially from normal results, as well as from results in patients with other diseases that may be mistaken for that disease. Even highly accurate tests produce false results when applied inappropriately and without a firm understanding of the concepts. Most important, consider the test's acceptability to the patient. This is especially significant in the patient who cannot make a choice and give consent. In such individuals, a proxy is needed to gain permission to give the test. Although some patients may be unable to express themselves verbally, their behavior may give nonverbal expression to their feelings toward the test's acceptability.

Nutritional Assessment

Several age-associated changes influence the nutritional status of older adults. These primarily include the loss of lean body mass, a decrease in the basal metabolic rate, and an increase in body fat. Although overall caloric intake should decrease due to decreased metabolism and often decreased physical activity, certain nutrient needs may change as one ages. A nutrient-rich diet is required to meet these changing needs. A nutritional assessment needs to be a part of the CGA, because inadequate intake is common in older adults. An estimated 5% to 30% of older adults living in the community are malnourished; 23% to 60% of hospitalized older adults and 16% to 70% of those in long-term care facilities experience malnutrition (Guyonnet & Rolland, 2015).

Obesity is the most common nutritional disorder in the older adult living in the community, and undernutrition is most common in those in acute and long-term care facilities. The diagnosis of abnormal weight loss may be made if one or more of the following criteria are present: abnormal weight loss of 5% or greater in 1 month, 10% or greater in 6 months; body mass index (BMI) of less than 21 kg/m^2; and serum albumin concentration of less than 35 g/L (Guyonnet & Rolland, 2015). There are four components of nutritional assessment: 1) nutritional history using a validated tool; 2) food intake diary of 1 to 3 days; 3) physical assessment, including anthropometric measurements and signs of nutritional deficiencies; and 4) biochemical markers, if applicable.

Screening for nutritional deficiencies begins with the use of a standardized tool. Several assessment instruments have

TABLE 4-3 Sample Focused Geriatric Physical Examination

SIGNS		PHYSICAL SIGN OR SYMPTOM	DIFFERENTIAL DIAGNOSES
Vital signs	Blood pressure	Hypertension	Adverse effects from medication, autonomic dysfunction
		Orthostatic hypotension	Adverse effects from medication, atherosclerosis, coronary artery disease
	Heart rate	Bradycardia	Adverse effects from medication, heart block
		Irregularly irregular heart rate	Atrial fibrillation
	Respiratory rate	Increased respiratory rate >24 breaths per min	Chronic obstructive pulmonary disease, congestive heart failure, pneumonia
	Temperature	Hyperthermia, hypothermia	Hyperthyroidism and hypothyroidism, infection

TABLE 4-3 Sample Focused Geriatric Physical Examination—cont'd

SIGNS	PHYSICAL SIGN OR SYMPTOM	DIFFERENTIAL DIAGNOSES
General	Unintentional weight loss	Cancer, depression
	Weight gain	Adverse effects from congestive heart failure, medication
Head	Asymmetrical facial or extraocular muscle weakness or paralysis	Bell's palsy, stroke, transient ischemic attack
	Frontal bossing	Paget disease
	Temporal artery tenderness	Temporal arteritis
Eyes	Eye pain	Glaucoma, temporal arteritis
	Impaired visual acuity	Presbyopia
	Loss of central vision	Age-related macular degeneration
	Loss of peripheral vision	Glaucoma, stroke
	Ocular lens opacification	Cataracts
Ears	Hearing loss	Acoustic neuroma, adverse effects from medication, cerumen impaction, faulty or ill-fitting hearing aids, Paget's disease
Mouth, throat	Gum or mouth sores	Dental or periodontal disease, ill-fitting dentures
	Leukoplakia	Cancerous and precancerous lesions
	Xerostomia	Age-related, Sjögren's syndrome
Neck	Carotid bruits	Aortic stenosis, cerebrovascular disease
	Thyroid enlargement and nodularity	Hyperthyroidism and hypothyroidism
Cardiac	Fourth heart sound (S4)	Left ventricular thickening
	Systolic ejection, regurgitant murmurs	Valvular arteriosclerosis
Pulmonary	Barrel chest	Emphysema
	Shortness of breath	Asthma, cardiomyopathy, chronic obstructive pulmonary disease, congestive heart failure
Breasts	Masses	Cancer, fibroadenoma
Abdomen	Pulsatile mass	Aortic aneurysm
Gastro-intestinal	Constipation	Adverse effects from medication, colorectal cancer, dehydration, hypothyroidism, inactivity, inadequate fiber intake
Genital/ rectal	Fecal incontinence	Fecal impaction, rectal cancer, rectal prolapse
	Atrophy of the vaginal mucosa	Estrogen deficiency
	Prostate enlargement	Benign prostatic hypertrophy
	Prostate nodules	Prostate cancer
	Rectal mass, occult blood	Colorectal cancer
Urinary	Urinary incontinence	Bladder or uterine prolapse, detrusor instability, estrogen deficiency
Extremities	Abnormalities of the feet	Bunions, onychomycosis
	Diminished or absent lower extremity pulses	Peripheral vascular disease, venous insufficiency
	Heberden's nodes	Osteoarthritis
	Swan neck deformity	Rheumatoid arthritis
	Pedal edema	Adverse effects from medication, venous insufficiency, congestive heart failure
Musculo-skeletal	Diminished range of motion, pain	Arthritis, fracture
	Dorsal kyphosis, vertebral tenderness, back pain	Cancer, compression fracture, osteoporosis
	Gait disturbances	Adverse effects from medication, arthritis, deconditioning, foot abnormalities, Parkinson's disease, stroke
	Leg pain	Intermittent claudication, neuropathy, osteoarthritis, radiculopathy, venous insufficiency
	Muscle wasting	Atrophy, malnutrition
	Proximal muscle pain and weakness	Polymyalgia rheumatica
Skin	Erythema, ulceration over pressure points, unexplained bruises	Anticoagulant use, elder abuse, idiopathic thrombocytopenic purpura
	Premalignant or malignant lesions	Actinic keratoses, basal cell carcinoma, malignant melanoma, pressure ulcer, squamous cell carcinoma
Neurological	Tremor with rigidity	Parkinson's disease

Source: From Elsay, B., & Higgins, K. E. (2011). The geriatric assessment. *American Family Physician, 83*(1), 48–56.

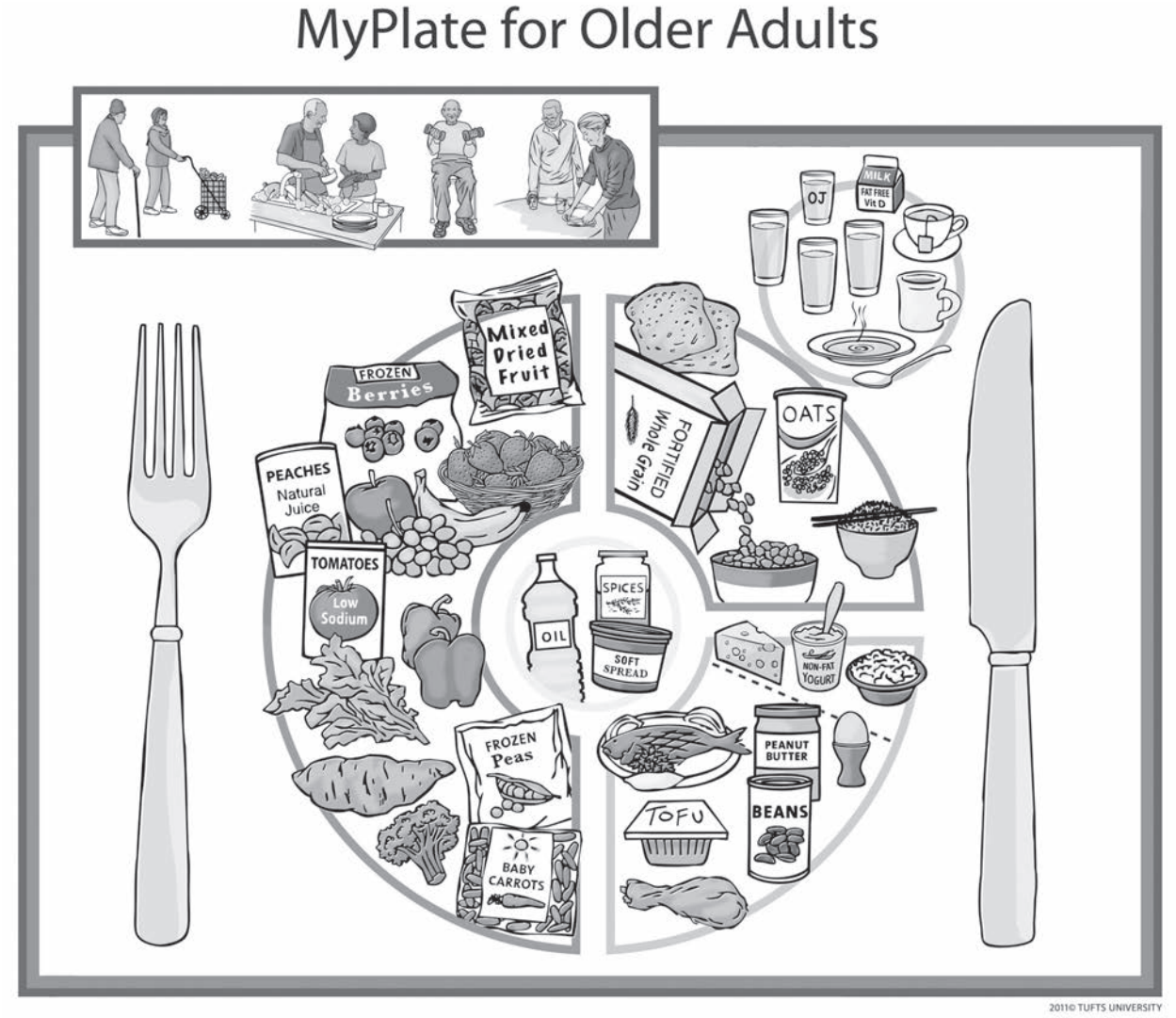

FIGURE 4-1. MyPlate for Older Adults. Copyright 2011, Tufts University. For details about MyPlate for Older Adults, please see http://www.nutrition.tufts.edu/research/myplate-older-adults.

clinical utility in the nutritional evaluation of older adults. The Mini Nutritional Assessment Instrument, a tool that can identify older adults who have or are at risk for malnutrition, and the Nutrition Health Checklist, part of the Nutritional Screening Initiative project by the American Academy of Family Medicine, the American Dietetic Society, and the National Council on Aging for healthy older adults, are the two most often used and available online. A food diary completed by the patient and reviewed by the provider can help to identify both the quantity and the quality of food and fluid intake. MyPlate for Older Adults serves as a guide for evaluation (Figure 4-1).

The physical examination may reveal some indicators of malnutrition, including weight loss, muscle wasting, dry and dull hair, mental impairments, and poor wound healing. In addition to calculating the BMI, there are other noninvasive, quantitative techniques for determining an individual's body fat composition by measuring, recording, and analyzing specific dimensions of the body (height and weight, skinfold thickness, and bodily circumference).

There is no gold standard for biomarkers of nutritional deficiencies in the older adult, but some indicators include prealbumin, transferrin, albumin, chemistries, complete blood count (CBC), vitamin B_{12}, folate, 25(OH) vitamin D, and thyroid panel. As the understanding of the role of pro-inflammatory cytokines on the acute phase protein response, albumin cannot be used as the sole indication of malnutrition. The presence of inflammation needs to be considered when interpreting serum albumin results. A C-reactive protein can help to determine if malnutrition is the result of lack of intake or due to inflammation and hypercatabolism (Guyonnet & Rolland, 2015).

FUNCTIONAL HEALTH

Although functional decline is common with advanced aging, the two are not synonymous. Although functional decline occurs in every system, the distinction between functional usual aging and functional successful aging remains

TABLE 4-4 Selected Elements of Functional Independence/Dependence

ADLs	IADLs
Bathing	Food preparation
Dressing	Household chores
Toileting	Laundry
Feeding oneself	Managing medications
Move from bed to a standing position or chair	Using the telephone
Ability to walk with or without assistive device	Managing finances
Continence of urine	Shopping
Continence of stool	Transportation

unclear. The goal in geriatric care is to improve function, if possible, and, if not, to prevent functional decline or at least slow down the process of functional deterioration. The parameters of function include both activities of daily living (ADLs) and instrumental activities of daily living (IADLs). Basic ADLs include three components: basic self-care, mobility, and continence. IADLs measure community interactions within the home and outside the home (Table 4-4).

Two well-established tools used to evaluate function in older adults are the Katz Activities of Daily Living Scale (Katz et al., 1963) and the Lawton and Brody Scale for Instrumental Activities of Daily Living (Lawton & Brody, 1969). It is important to be cautious about self-report of function (rather than direct observation of function) and to ask, "How do you . . . ?" or "Show me how you. . . ." instead of "Can you . . . ?" in order to determine if patients actually perform the activity. Bathing is often the first ADL that poses problems for older adults.

Falls are a leading cause of death and disability in the older adult, and assessment of both intrinsic and extrinsic factors contributing to falls is important. Ask yourself the question, "Why did this patient fall today?" A valid tool for assessment of falls is the Hendrich II Fall Risk Model, which is useful in acute care, ambulatory, assisted living, long-term care (Hendrich, Bender, & Nyhuis, 2003). In addition to identifying those at risk for falls, it helps to identify appropriate interventions to reduce their fall risk. Mobility can be screened with the Timed Get Up and Go Test. This test involves observing for unsteadiness as the patient gets up from a chair without using the arms, walks 10 feet, turns around, walks back, and resumes a seated position. Timing the process, which should take less than 16 seconds, enhances the sensitivity of this test (Podsiadlo & Richardson, 1991). Patient difficulties performing this test indicate mobility issues, weakness of the extremities, and an increased risk for falling. Tinetti's Performance Oriented Mobility Assessment is a task-oriented test that measures gait and balance abilities (Tinetti, 1986).

PSYCHOLOGICAL HEALTH

Cognitive and affective disorders, specifically dementia, delirium, and depression, are more prevalent in older adults, and clinical definitions of these disorders are addressed in the Diagnostic and Statistical Manual of Mental Health Disorders (DSM), 5th ed. (2013). The DSM-5 describes the characteristics of delirium and mild or moderate neurocognitive disorders. There are a number of validated tools used to screen for cognitive dysfunction. For years the gold standard was the Mini Mental Status Examination (MMSE), but more recent tools have all been validated against that standard. Three tools used in clinical settings have comparable psychometric properties to the MMSE: the Mini-Cog, Montreal Cognitive Assessment (MoCA), and Saint Louis University Mental Status Examination (SLUMS) (Cummings-Vaugh et al., 2014; Tariq et al., 2006; Tsoi et al., 2015). The primary advantage of the Mini-Cog is that it is shorter than the MMSE and measures executive function. It is composed of a three-item recall and the Clock Drawing Test (CDT) and takes about 3 minutes to administer (Borson et al., 2011). Both the MoCA and SLUMS have comparable specificity and sensitivity to the MMSE, but are better at identifying mild neurocognitive disorder and are available online; unlike the MMSE, which is copyrighted.

Older adults are at greater risk of delirium, which is under-recognized and underdiagnosed. There are a number of instruments designed to screen for delirium; however, the best evidence supports the use of the Confusion Assessment Method (CAM), which takes 5 minutes to administer (Inouye et al., 1990). The CAM diagnostic algorithm is based on four cardinal features of delirium: 1) acute onset and fluctuating course, 2) inattention, 3) disorganized thinking, and 4) altered level of consciousness (Shi, Warren, Saposnik, & MacDermid, 2013).

Depression is also under-recognized in older adults. There are several screening tools for depression, though the most widely used in the clinical practice is the Geriatric Depression Scale: Short Form (GDS:SF). It consists of 15 questions requiring a "yes" or "no" response, so it can be completed quickly in any setting (Sheikh & Yesavage, 1986). It has been suggested that a single question sensitive to depression, such as "During the past 4 weeks, how often did you feel downhearted and depressed?" be the first step in screening (Le Strat & Dubertret, 2013). The PHQ-9 is a nine-item depression scale of the Patient Health Questionnaire that assesses depressive symptoms and functional impairment, and screens for symptom severity (Kroenke & Spitzer, 2002).

A recent implementation of a program to improve the quality of spiritual care validated the use of three instruments: HOPE, FICA, and SPIRIT. These tools are effective means of determining the older patient's spiritual beliefs and the comfort associated with these beliefs (Piotrowski, 2013). In that program, they chose to use the HOPE questionnaire with one additional question, "Are you at peace?"

HOPE (Anandarajah & Hight, 2001)

- Sources of **H**ope, meaning, comfort, strength, peace, love, and connection
- **O**rganized religion
- **P**ersonal spirituality and practice
- **E**ffects on medical care and end-of-life issues

FICA (Puchalski & Romer, 2000)

- **F**aith or belief
- **I**mportance or influence
- **C**ommunity
- **A**ddress

SPIRIT (Ambuel, 2005)

- **S**piritual belief system
- **P**ersonal spirituality
- **I**ntegration with a spiritual community
- **R**itualized practice and restrictions
- **I**mplications for medical care
- **T**erminal event planning

SOCIOENVIRONMENTAL SUPPORTS

Social isolation in community-dwelling older adults is all too common. Estimates are as high as 43%, ranging from 10% to 43% (Nicholson, 2012). Social isolation has been shown to contribute to all-cause mortality. While social isolation is not always assessed in the primary care setting, it should be an essential component of a CGA. A valid instrument that is clinically practical in assessment of social support is the Lubben Social Network Scale—6-item version (Lubben et al., 2006). This validated scale can be an important contribution to the assessment of family and friendship relationships, and identifies patients at risk for social isolation who can benefit from intervention. In 1995, Kane recommended including the following statements in a geriatric assessment to help improve understanding of the patient's social support:

- Is there any one special person you could call or contact if you need help? (If yes, identify.)
- In general, other than your children, how many relatives do you feel close to and have contact with at least once a month? (Number.)
- In general, how many friends do you feel close to and have contact with at least once a month? (Number.)

Additional social and economic resources are assessed by exploring the following:

- Living situation
- Housing
- Transportation
- Income
- Assets
- Degree of financial burden resulting from health concerns

QUALITY OF LIFE MEASURES

Quality of life in aging individuals involves identifying the person's general satisfaction with life in a number of the domains identified in the CGA. The Medical Outcomes Study—Short-Form 36 (Stewart & Ware, 1992) is a tool that looks at three of these: the physical, mental, and social domains. Although not specifically designed for the older adult, it remains the gold standard of quality of life instruments because of its longevity, applicability, and ease of administration and analysis. A final assessment involves having a frank discussion with the older adult about preferences for care at present and at end of life, and encouraging the patient to complete an advance directive so these preferences are documented.

SUMMARY

- CGA is a multidimensional process designed to identify care needs, plan care, and improve outcomes for older adults. The domains of CGA include, but are not limited to, physical health, functional status, psychological health, socioenvironmental supports, and measures of quality of life. This chapter covered both the tools and techniques that can be used to conduct a CGA and also highlighted the supportive research where it exists. The evidence of the value of CGA in general has been inconsistent. The evidence is clear that parts of the CGA do improve certain outcomes in older patients, but continued rigorous research is indicated.

CLINICL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The evidence is insufficient to assess the balance of benefits and harms of screening for hearing loss in asymptomatic older adults.	C	Moyer for USPSTF, 2012a
Evidence shows that the benefit of mammography screening increases with age, with women between 50 and 74 years old benefiting the most. The best balance of benefit to harm is when screening every 2 years. For women over 75 years old, evidence is insufficient and decision should be on an individual basis.	A	Siu for USPSTF, 2016
For adults over age 65 who do not present with vision problems, evidence is insufficient to recommend screening in older adults.	C	Chou et al. for USPSTF, 2016
The American Academy of Ophthalmology recommends every 1- to 2-year screening for older adults, while the American Optometric Association recommends screening annually.	C	Teutsch, McCoy, Woodbury, & Welp, 2016

CLINICL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Women age 65 years and over, and younger women whose fracture risk is equal to theirs with no additional risk factors, should be screened for osteoporosis with dual-energy x-ray absorptiometry of the femoral neck.	B	USPSTF, 2011
The USPSTF previously recommended against PSA-based screening in men over age 75, but they now recommend against PSA screening in all age groups.	C	Moyer for USPSTF, 2012b
The American College of Physicians concurred with the recommendation.	C	Qaseem et al., 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

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CHAPTER 5

Symptoms and Syndromes

Evelyn Duffy

ASSESSMENT

Assessment of a symptom based on the presentation of a concern or an evaluation of a geriatric syndrome in the older adult poses challenges to the health-care provider. Using a change in function as a measure of the impact of a symptom on the patient is an effective means of sorting out the differential. Heterogeneity increases with age, so there are a greater number of possibilities to consider in formulating a diagnosis, and the differential diagnosis can be quite broad. Working up a geriatric patient with a single symptom or cluster of symptoms using typical symptom analysis and clinical decision-making tools can be done, but it is often complicated by the fact that patients may ignore symptoms initially because they do not interfere with functional activities or because they attribute the symptoms to the aging process. Conversely, patients may fear loss of independence related to a subtle decline in functional abilities and deliberately choose to conceal this, or they may be unaware of a subtle decline until it becomes apparent to others.

Acuity of symptoms may be blunted because of changes in the efficiency of certain physiological mechanisms. New symptoms often appear in one organ system as an offshoot of pre-existing disease in another organ system. Multiple symptoms may be present because of comorbidities. These factors can result in a delayed or atypical presentation. In many cases, the first hint of illness may be a change in cognitive function or mental status. In the case of the patient with an underlying dementia, the refusal to eat or drink, or a change in the patient's behavior, may signal an acute illness. The term *geriatric syndrome* refers to conditions that have multiple underlying factors that may involve multiple organ systems; some of the most common are delirium, falls, dizziness, and incontinence. The literature reflects that shared risk factors across geriatric syndromes include older age, cognitive impairment, functional impairment, and impaired mobility. For each presenting symptom, the following descriptors should be used to guide the diagnosis:

- Mode of onset of the symptom (events coinciding with onset, similar past episodes, gradual or sudden, total duration)
- Location of the symptom
- Character of the symptom
- Radiation of the symptom
- Precipitating or aggravating factors
- Relieving or ameliorating factors
- Past treatment or evaluation (when, where, who; studies done and results)
- Course of the symptom
- Effect of the symptom on normal daily activities

If the symptom or the patient's demeanor indicates a problem in function or cognition, assess these areas. Perform a thorough medication history, including the use of over-the-counter (OTC) preparations, vitamins, herbal and homeopathic remedies, and any other dietary supplements, and consider their use in formulating diagnostic possibilities. Pharmacotherapeutic or other treatment modalities can complicate or even cause illness in older patients.

BOWEL INCONTINENCE

Description: Bowel incontinence or fecal incontinence (FI) is an involuntary passage of stool or the inability to control stool from expulsion. There are three types of FI: 1) urge incontinence: a person has a desire to defecate but cannot make it to the toilet despite attempts to avoid defecation; 2) passive incontinence: involuntary loss of gas or stool without awareness; and 3) fecal seepage-leakage of stool after a normal bowel evacuation, usually presenting staining of clothing (Shah et al., 2012). This condition can be costly due to use of adult briefs, as well as greater incidence of nursing home placement of individuals with FI (Taminini et al., 2015). It can also be a socially and emotionally devastating

situation that interferes with quality of life and leads to diminished self-esteem and depression.

Etiology: Defecation involves a complex series of events and factors: diet, stool consistency, anorectal sensation, muscle strength and function, as well as neurological integrity, cognition, and motivation (Shah et al., 2012). The ability to defecate has two components: continence and defecation. Continence is the ability to hold fecal material and is under the control of the autonomic and spinal cord nervous system, including the internal and external anal sphincter. Defecation is the evacuation of feces when a person consciously attempts to have a bowel movement (Shah et al., 2012). FI occurs due to a number of reasons, from gastrointestinal (GI) issues to cognitive or neurological diseases. A greater concern exists in a patient who is continent of urine but incontinent of stool. See Table 5-1 for a review of causes of fecal incontinence.

Occurrence: Prevalence in community-dwelling elders is 36% and up to 65% in nursing home residents. In the hospitalized setting, there is reported to be 16% prevalence (Shah et al., 2012).

Age: FI is found more frequently in older adults than in the general population.

Gender: FI occurs more commonly in women than men due to delivery of large babies or from obstetrical trauma related to childbirth (Shah et al., 2012).

Ethnicity: Not significant.

Contributing Factors: There are certain epidemiological risk factors in the older individual associated with FI. In men, kidney disease and urinary incontinence were associated with FI, and in women, depression, urinary incontinence, and chronic diarrhea were associated with FI (Shah et al., 2012).

Diarrhea from GI disease (colitis, irritable bowel syndrome [IBS]), infectious causes such as viral gastroenteritis or *Clostridium difficile (C diff)*, or medication side effects can contribute to acute, short-term bowel incontinence. *C diff* infections occur when a person is exposed to the pathogen while receiving antibiotic treatment for another illness (Keller et al., 2014). Antibiotics suppress the normal bacteria in the colon, allowing malabsorbed carbohydrates or *C diff* to flourish, producing toxins that cause a severe diarrhea. Damage to the colon can cause the individual to become septic and possibly cause death, especially in the older patient (Roos, 2015).

Signs and Symptoms: The first step in the clinical appraisal is to inquire about bowel habits, including incidence of diarrhea and fecal continence. Determine the duration, frequency, severity, and character of the incontinence (gas, liquid, solid). Inquire if there is sensation of rectal fullness, awareness of passing stool or gas, and warning symptoms of cramping or urgency. Associated symptoms of abdominal pain and bloating, anorexia, diarrhea or constipation, nausea and/or vomiting, rectal bleeding, and urinary incontinence should be ascertained. Review prescription and OTC medications carefully. Past medical history may be positive for diabetes, neurological and psychiatric diseases, GI disorders, cancers with radiation, and any condition limiting mobility. Surgical history may include hemorrhoidectomy, anal fissure repair, fistulectomy, anal dilation, or colon resection. Diet review may reveal high fiber intake and excessive fluid intake or foods that may cause irritation and worsen FI such as caffeine, alcohol, chocolate, greasy or spicy foods, dairy products, and various raw fruits and vegetables.

Physical examination includes assessing for significant weight loss. The abdomen should be examined for distention, hyperactive or absent bowel sounds, pain, masses, and bladder distention. The perineum is inspected for gaping of anus, an indication of severe sphincteric dysfunction, hemorrhoids, dermatitis, surgical scars, fissures, and fistulas. The clinician should check the "anal wink" (the reflex contraction of the anal sphincter when the skin around the anus is stroked bilaterally) to test sensation and pudendal nerve function. Asking the patient to bear down will reveal rectal prolapse, if present. The digital rectal examination is performed to detect hemorrhoids or masses, and evaluate baseline and squeeze sphincter tone, as well as the amount and character of stool. Neurological examination includes assessment of gait, general mobility, motor strength, and sensory testing.

Diagnostic Tests: The results of data collected from the history and physical examination will dictate if additional testing is needed. Categorize the diarrhea as inflammatory, fatty, or watery. When an infectious or foodborne cause is suspected, stool tests guided by the clinical presentation are indicated. Exclusion of fecal impaction, particularly in

TABLE 5-1 Causes of Fecal Incontinence

DISEASE	MECHANISM	COMMENTS
Central Nervous System		
Dementia	Impairs awareness of need to defecate Loss of inhibitory control	Manifestations depend on type and severity of dementia
Stroke	Impaired awareness of anal control Slowed motor processing communication with frontal lobe damage	Dependent on size of infarct or hemorrhage within the brain
Brain tumor	Similar to stroke	Dependent on location of tumor
Multiple sclerosis	Decreased external anal sphincter pressures leading to decreased volumes of rectal distention to inhibit the internal anal sphincter	

Continued

TABLE 5-1 Causes of Fecal Incontinence—cont'd

DISEASE	MECHANISM	COMMENTS
Peripheral Nervous System		
Cauda equina syndrome	Compression of spinal nerves	
Polyneuropathies	Various mechanisms leading to destruction of the axon and myelin	Causes include infection, autoimmune disease, hypothyroidism, vitamin B_{12} deficiency, and medications
Diabetes mellitus	Neuropathy of sensory and motor nerves Internal anal sphincter dysfunction	
Shy Drager syndrome	Multisystem atrophy	
Toxic neuropathy	Destruction of nerve cells leading to loss of sensation or motor control	Chemotherapy and radiation therapy
Traumatic neuropathy	Chronic straining during evacuation External anal sphincter weakness or pudendal nerve neuropathy	
Fecal impaction	Altered rectal sensation Liquid stool escaping around solid feces in colon or higher in bowels	
Gastrointestinal (GI)		
GI disease	GI dysfunction/disease with excessive stool volume and rapid gut transit	Causes include viral or bacterial gastroenteritis, colitis, Crohn's disease, celiac disease, colon cancer, irritable bowel syndrome, rectocele, and other GI diseases **Clostridium difficile* can cause severe diarrhea in the elderly and can lead to death
Rectal sphincter dysfunction	Anal sphincter damage and/or pudendal neuropathy affecting sphincters, puborectalis muscle, and rectal sensation	Common causes include delivery of large baby, traumatic childbirth, anal surgery, rectal prolapse, and prolonged straining
Reservoir incontinence	Loss of compliance of the rectal wall leading to urgency, frequency, and leakage of stool	Associated with colectomy, inflammatory bowel disease, chronic rectal ischemia, and prostate/cervical radiation Diminished colonic/rectal capacity is a less common type of FI
Medications		
Laxatives	Medications causing increased gastric motility or increased water in stools	
Fiber products	Increase fluid in the stool to keep it soft and move easier but can lead to diarrhea and FI	
Magnesium	Draws fluid into the bowels through osmosis	Magnesium containing antacids and certain laxatives contain significant amounts of magnesium
Other medications	Antibiotics can cause destruction of the colon's natural flora, leading to diarrhea Other medications that can cause diarrhea include proton pump inhibitors, SSRIs, and NSAIDs	
Other		
Physical deficits	Inability to recognize need to defecate (cognitive problems) or inability to ambulate to toilet without assistance	Common causes of cognitive problems include dementia Common causes of mobility problems include debilitating arthritis, hemiplegia, or any condition affecting gait
Dietary causes	Foods in susceptible individuals include a diet high in fiber, spicy foods, fatty/greasy foods, cured meats, smoked meats, dairy products, caffeine, or sorbitol	Sugar-free gum and artificial sweeteners can cause diarrhea and FI Avoidance of irritating foods can eliminate the FI
Obesity	Inability to ambulate to defecate	This is usually a reversible condition
Excessive fluid intake	Overhydration can lead to diarrhea and possibly cause FI	

Source: Barr & Smith (2014); Keller & Surawicz (2014); Nodrqvist (2014); Shah et al. (2012); Sweetser (2012).

nursing home patients, is essential. Plain abdominal radiograph may show a high impaction when no hardened stool is felt in the rectal vault. If organic bowel disease is suspected (inflammatory disease, ischemic colitis, neoplasm), stools for occult blood, flexible sigmoidoscopy or colonoscopy is indicated. Anorectal manometry tests rectal sensation and compliance through the use of an intrarectal balloon and measures pressure, tone, and reflex contraction of the internal sphincter. It provides comprehensive information about anorectal function and is reasonable in the ambulatory and/or healthy elder who has failed conservative interventions. Other tests that may be considered include colonoscopy, electromyography, anal ultrasound, and magnetic resonance imaging (MRI).

Differential Diagnosis: The differential diagnosis includes fecal impaction, gastroenteritis, foodborne illness, ruptured diverticulum, inflammatory bowel disease, ischemic colitis, irritable bowel syndrome, anal fistula, diabetic neuropathy, rectal prolapse, upper motor neuron lesions secondary to cerebrovascular accident, multiple sclerosis, spinal cord compression, degenerative processes, dementia, depression with self-care deficits, and functional impairment.

Treatment: Elimination of potentially offending agents is an important first step. Fecal impaction requires disimpaction (manually, with enemas, and/or osmotic/stimulant laxatives) and establishment of a preventive bowel regimen (see section on constipation). With loose or liquid stools, a supplement of soluble dietary fiber may be beneficial (adding more bulk to stools); the ideal amount and type have yet to be determined, and response varies greatly. Increasing fiber gradually over a period of several days can help reduce symptoms of abdominal bloating and discomfort that may be associated with increased fiber intake. In the setting of fecal reservoir incontinence, avoidance of dietary fiber may be indicated. Habit training may be effective for elders with dementia and involves frequent and regular toileting, usually after a meal to take advantage of the gastrocolic reflex. Once infection is ruled out, fecal impaction is excluded, and no clear cause is determined for FI with diarrhea, the antidiarrheal medication Imodium (loperamide) may be tried. For rectosphincter dysfunction, biofeedback with sphincter-strengthening exercises is an option for motivated, cognitively intact elders who have some degree of rectal sensation. Sacral nerve stimulation is also a promising, safe, minimally invasive treatment of FI related to rectosphincter dysfunction. Surgical sphincter repair may be considered when incontinence is severe, unresponsive to conservative interventions, and clearly related to anatomical defects, with best outcomes seen with repair of defined defects in the external sphincter.

Follow-Up: Follow-up is indicated to assess resolution of FI and the time frame will be dictated by the identified cause, as well as the severity of the problem. Ongoing monitoring is needed in all cases.

Sequelae: Complications of FI include perineal dermatitis, pressure ulcers in the sacral/perineal area, non-healing wounds, recurrent urinary tract infections (UTIs), anxiety, social isolation, and depression. FI, along with urinary incontinence, is a leading cause of institutionalization of elders.

Prevention/Prophylaxis: Manage constipation. Optimize treatment of underlying medical conditions, such as diabetes and GI disturbances. Adjust diet and laxative use to address loose stools. Consider physical therapy/occupational therapy (PT/OT) intervention to address mobility and toileting ability. Reinforce a regular toileting program for the cognitively impaired and physically frail.

Referral: Urgent spinal imaging/neurosurgical consultation for new-onset fecal/urinary incontinence with lower extremity weakness and paresthesia to rule out spinal cord compression. Additional evaluation with "alarm" symptoms: change in bowel habits, rectal bleeding, weight loss to rule out GI malignancy.

Education: Provide clear information on normal bowel function, FI cause, and rationale for any diagnostic tests, preventive strategies, and treatments. Instruct in appropriate dilution and need for adequate fluid with fiber supplements. Direct to appropriate resources for incontinence pads and briefs as needed.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
With loose or liquid stools, a supplement of soluble dietary fiber may be beneficial.	A	Shah, Chokhavatia, & Rose, 2012
Habit training may be effective for elders with dementia and involves frequent and regular toileting.	B	Nordqvist, 2014
Antidiarrheal medications, such as loperamide (drug of choice) and diphenoxylate, may be considered.	A	Shah, Chokhavatia, & Rose, 2012
Provide biofeedback with sphincter-strengthening exercises.	B	Shah, Chokhavatia, & Rose, 2012
Stimulate the sacral nerve.	A	Shah, Chokhavatia, & Rose, 2012

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CHEST PAIN

Description: Acute, nontraumatic chest discomfort, perceived as pain or as a sensation of tightness, pressure, or squeezing in the chest or thorax area; associated with actual or potential tissue or organ damage.

Etiology: The many different pathophysiologies of chest pain (CP) may be attributed to segmental overlap of neurons of cardiopulmonary and noncardiopulmonary origin. CP may originate from organs within or outside of the thorax; cardiac, pulmonary, esophageal, large vessel, gastric, pancreas, biliary, musculoskeletal, skin, and psychological pathology may be implicated. Life-threatening or high acuity causes include acute coronary syndrome (ACS) (includes acute myocardial infarction [MI] and unstable angina), aortic aneurysm, thoracic aortic dissection, spontaneous tension pneumothorax, pneumonia, pulmonary embolism, esophageal perforation, and esophagitis. Lower acuity causes of CP can be stable angina, pericarditis, myocarditis, gastroesophogeal reflux disease/gastro-peptic, biliary disease, costochondritis, rib fracture/trauma, and herpes zoster (Gupta & Munoz, 2016).

Occurrence: CP is one of the most common causes of emergency department visits, with more than 6 million people evaluated yearly. Of those 6 million visits, approximately 15% are over the age of 65 years (Goldwater, 2014).

Age: The U.S. Census Bureau predicts that by the year 2030, 71 million Americans will be over 65 years of age and by 2050, almost 81 million Americans will be age 65 years or older (Goldwater, 2014). The probability for serious conditions increases with chronological age due to increasing prevalence of coronary artery disease (CAD), hypertension, lung cancer, cardiac arrhythmias, diabetes, and conditions associated with immobilization. Age is a number and the geriatric population can be further defined as elderly (between 75 and 84 years old) and very elderly (85 years of age or older) (Goldwater, 2014). Associated with age is the assessment of frailty. One does not need to be old to be frail, but frailty will occur with aging and frailty must be considered when treatment modalities for CP are considered.

Gender: Atypical presentation of ACS is more common in females, the older adult, and the very older adult.

Ethnicity: African Americans report less typical anginal features than Caucasians. Hispanic women with MI report more prodromal chest pain/discomfort than African Americans and Caucasians. Studies show ethnicity and time to seek medical care for CP increases in many ethnic minorities. African Americans, Asians, Hispanics, and South Asians take longer than those of majority populations to seek care for CP.

Contributing Factors: Esophageal disease not only can mimic, but also coexists with MI in 50% of elders with CAD. A recent heavy, fatty meal can exacerbate reflux esophagitis. Panic disorder with a prevalence of 0.1% to 1.0% may manifest with hyperventilation, which has been linked to coronary artery and esophageal spasm. Medications, illicit drugs (methamphetamines, cocaine, caffeine, stimulants), and prescriptions such as sildenafil (Viagra) which may cause hypotension, and sumatriptan (Imitrex) through coronary vasoconstriction, can trigger CP. Cancer, recent orthopedic surgery, and immobility may result in deep vein thrombosis (DVT), a risk factor for pulmonary emboli.

Signs and Symptoms: Pain of ACS may be retrosternal or poorly localized; may radiate to arms, back, neck, or jaw; may occur at rest; may last longer than 20 minutes; and is unrelieved with nitroglycerin. It may occur with nausea, vomiting, diaphoresis, dyspnea, weakness, fatigue, light-headedness, and/or anxiety. It may be precipitated by stress, exercise, or illness. Stable angina is substernal or precordial pressing, constricting, or heaviness precipitated by exertion and relieved with nitroglycerin. Older adult or diabetic patients with altered pain perception or altered ability to localize the discomfort commonly have atypical presentations. An absence of chest pain with ACS in older adults is common. Dyspnea, not chest pain, is the most common presenting symptom of acute MI in patients over 85 years old.

Thoracic aortic dissection has abrupt onset and severe, retrosternal tearing pain that usually radiates to the back and both arms. Pulmonary embolism may present with acute dyspnea, cough, deep pleuritic chest pain that may not be severe, and hemoptysis. Tension pneumothorax is associated with acute unilateral pleuritic chest pain and dyspnea. Esophageal spasm/gastroesophageal reflux disease (GERD) may mimic myocardial ischemia, may be associated with eating, improves with upright position or antacids, and is accompanied by cough, hoarseness, and dysphagia. Acute pancreatitis may present with knifelike epigastric or lower chest pain radiating to the left shoulder associated with vomiting and relieved by leaning forward. Musculoskeletal pain is usually persistent and is aggravated by movement, cough, or deep respirations. Herpes zoster is described as sharp pain or paresthesia in the mid-thorax unilaterally. Pain may precede rash by several days. History may be positive for CAD with or without angina or risk factors thereof, including hypertension, diabetes, and hypercholesterolemia.

Anemia may predispose to ischemia. Atrial fibrillation is associated with pulmonary emboli, as is recent orthopedic surgery, recent immobilization, DVT, and cancers. GI disorders, degenerative joint disease, and anxiety disorders, as well as smoking and alcohol abuse, can be clues to diagnosis. Signs of serious conditions include acute confusion or anxiousness, pallor/cyanosis, diaphoresis, tachycardia, bradycardia, tachypnea, and hypotension. A new S4 murmur or signs of heart failure (jugular venous distention, wet crackles, S3 murmur, extremity edema) suggest myocardial ischemia. Asymmetrical blood pressures, absent or asymmetrical extremity pulses, and aortic bruit may be present with aortic dissection. Tracheal deviation, unilateral diminished or absent breath sounds, and palpable subcutaneous crepitus may reveal pneumothorax. Rapid, irregular apical pulse suggests atrial fibrillation, whereas unilateral lower extremity swelling and tenderness suggests DVT. Fever suggests an infectious cause. Localized abdominal tenderness with guarding and rebound points to GI origins. Costochondritis pain is worse with chest movement, sneezing, or cough and is not associated with fever, shortness of breath, tachycardia, or other cardiopulmonary symptoms. Diffuse tenderness is

Alternate Differential Diagnosis for Angina for Patients With Chest Pain

NON-ISCHEMIC	PULMONARY	GI	CHEST WALL	PSYCHIATRIC
Cardiovascular	Pulmonary	Esophageal	Costochondritis	Anxiety disorder
Aortic dissection	Embolism	Esophagitis	Fibrositis	Hyperventilation
Pericarditis	Pneumothorax	Reflux	Rib fracture	Panic disorder
	Pneumonia	Biliary	Sternoclavicular arthritis	Primary anxiety
	Pleuritic	Colic	Herpes zoster (before rash)	Affective disorders
		Cholecystitis		Thought disorders
		Choledocholithiasis		
		Cholangitis		
		Peptic ulcer		
		Pancreatitis		

usually seen over one or more costochondral joints. Vesicular rash in a unilateral thoracic dermatome suggests zoster.

Diagnostic Tests: If myocardial ischemia is suspected, immediate 12-lead electrocardiogram (EKG) is essential and may show ST-segment depression or elevation, T-wave inversion, new left bundle branch block (LBBB), or new Q waves. However, the EKG may not change for hours or sometimes not at all. Elevations in cardiac markers (troponins I and T) 3 to 6 hours (COR I LOE A) after the onset of pain indicate myocardial cell damage.

Additional tests include chest x-ray, which may reveal pulmonary edema, pneumothorax, pleural effusion, pneumonia, or lung mass. D-dimer and spiral computed tomography (CT) or ventilation/perfusion (V/Q) scan may confirm pulmonary emboli. Lower extremity Doppler ultrasound diagnoses DVT. Additional testing is guided by the clinical situation, history, and physical findings. Older adult patients with ACS are less likely to have typical EKG findings (i.e., ST-segment elevation). Unstable angina/non-ST elevation MI shows T-wave inversion, absent Q waves, and evolving ST-segment changes.

Differential Diagnosis: The differential diagnosis includes previously discussed emergent conditions, stable angina, pericarditis, pneumonia, pleurisy, lung/chest malignancy, esophageal spasm, esophagitis, GERD, cholecystitis, pancreatitis, peptic ulcer disease, costochondritis, herpes zoster infection, and psychogenic causes.

Treatment: If there is high suspicion of emergent conditions such as ACS or aortic dissection, immediate transport to the emergency department by emergency medical services (EMS) is recommended. For institutionalized elders, conservative management of ACS in the facility may be reasonable. Immediate intervention and early hospital care for suspected ACS is supplemental oxygen, 160 to 325 mg chewed aspirin (if no allergy), and sublingual or IV nitroglycerin when blood pressure is greater than 90 mm Hg systolic and heart rate is greater than 50 beats per minute (bpm). Nitrates should never be administered to patients with a non-ST elevation ACS who received phosphodiesterase inhibitors within 24 to 48 hours of onset of symptoms, or a precipitous fall in the blood pressure could lead to fatal outcomes. Supplemental oxygen to maintain oxygen saturation above 90% is indicated with other suspected emergent conditions.

Analgesic therapy may be recommended for pain control, and morphine IV can be used in a patient when not contraindicated. Treatment of stable angina includes aspirin, beta blockers, angiotensin-converting enzyme (ACE) inhibitors, nitrates, and statins. Long-acting calcium channel blockers may be used for symptom reduction if beta blockers are ineffective or contraindicated. Although only moderately successful, treatment options for esophageal spasm include calcium channel blockers, tricyclic antidepressants (exercise caution with use in older adults), nitrates, botulinum toxin, and dilation. Proton pump inhibitors, H_2 blockers, and/or antacids are the mainstay for GERD. Weight loss is advised for the overweight patient. Costochondritis may respond to local cool or warm packs, rest, or topical or oral analgesics. Careful examination of the skin to detect herpes zoster requires oral antiviral medications and appropriate pain management. Reassurance, cognitive-behavioral therapy, and/or antidepressants may be indicated for psychogenic causes.

Follow-Up: Follow-up is guided by diagnosis and response to treatment.

Sequelae: A 20% to 60% rate of unrecognized MI in older adults with inappropriate hospital discharge and delayed treatment results in poor outcomes and increased mortality.

Prevention/Prophylaxis: Optimize treatment of diabetes, hypertension, CAD, and atrial fibrillation. Encourage daily exercise and smoking cessation. For reflux symptoms, recommend controlling weight, elevating head of bed on blocks, staying upright after meals, and avoiding caffeine, chocolate, spicy foods, peppermint, cigarettes, and alcohol. Encourage the patient to get the Pneumovax and Zostavax vaccinations.

Referral: Suspected life-threatening conditions or unstable status may be referred to the emergency department depending on the patient's and family's wishes. Physician consultation is indicated with uncontrolled pain, uncertain diagnosis, and poor response to treatment.

Education: Teach patients and families to immediately seek medical care with acute new-onset CP or when a change in chronic CP is experienced. Carefully explain causes of pain, appropriate testing and treatment, prevention strategies, and indications for follow-up.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
If there is a high suspicion of an emergent condition, immediate transport to emergency department.	B	Amsterdam et al., 2014
Immediate interventions for suspected ACS: supplemental oxygen, 160–325 mg aspirin, sublingual nitroglycerin.	B	Amsterdam et al., 2014
All patients with CAD should be taking the following medications unless contraindicated:		
■ Aspirin	A	Amsterdam et al., 2014
■ Lipid lowering agent if low-density lipoprotein (LDL) is above target	A	
■ Beta blocker for patients with a history of myocardial infarction (MI)	A	
■ Beta blocker for patients without a history of MI	B	
For patients with CAD and diabetes mellitus (DM)/left ventricular systolic dysfunction, add: ACE inhibitor, unless contraindicated.	A	
Immediate-acting nitroglycerin (sublingual/spray) provides relief of anginal symptoms.	B	
Treatment of esophageal spasm: long-acting calcium channel blockers.	B	Katz, Gerson, & Vela, 2013
Treatment of esophageal spasm: calcium channel blockers, tricyclic antidepressants, nitrates, botulinum toxin, dilation.	C	
Treatment of GERD: proton pump inhibitors, H_2-blocker, antacids.	A	Katz, Gerson, & Vela, 2013
Treatment for GERD: controlling weight, elevating the head of the bed on blocks, staying upright after meals, avoiding specific foods and alcohol, smoking cessation.	C	Katz, Gerson, & Vela, 2013
Treatment for costochondritis: NSAIDs for pain.	C	Ayloo, Cvengros, & Marella, 2013
Treatment for herpes zoster: antivirals.	A	Gan, Tian, & Tay, 2013
Treatment of psychogenic causes: cognitive-behavioral therapy.	A	Campbell et al., 2017

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CONSTIPATION

Description: Constipation is the presence of two or more of the following symptoms: decreased stool frequency, straining, hard stools, sensation of incomplete emptying or anorectal blockage, requirement for manual maneuvers to pass stool, and rarely, loose stools in absence of laxative use (American Gastroenterological Association, 2013). It is the most common digestive complaint and a symptom rather than a disease (Basson, 2017).

Etiology: Colonic motility depends on the integrity of the nervous system impulses and circular smooth muscle tone, and motor complexes stimulated by increasing intraluminal pressure generated by bulk. Any pathophysiological process that interferes with this process can cause constipation. It is thought that changes in colonic motility are not age related but are influenced instead by extrinsic factors such as insufficient fluid, fiber, and exercise (DiGiorgio et al., 2015).

Occurrence: The prevalence of constipation in the general population is 20%, although it can range from 2% to 27% depending on the definition used and the population (Roque & Bouras, 2015).

Age: Constipation is a frequent complaint of older adults, with reports 20% higher than in younger patients. Older adults residing in long-term care facilities have a greater prevalence of constipation; up to 50% to 74% use laxatives daily (Roque & Bouras, 2015).

Gender: At all ages, constipation is reported more frequently in women than in men. Older women are two to three times more likely to report constipation than their male counterparts.

Ethnicity: Not significant.

Contributing Factors: The functional factors described earlier contribute to constipation if they are not the primary etiology. Medications that may contribute to constipation include calcium channel blockers, calcium supplements, narcotics, iron supplements, aluminum-containing antacids, anti-Parkinson's drugs, NSAIDs, and any drug that has anticholinergic side effects. Certain types of laxatives also may be constipating, such as stimulant laxatives, which may cause cathartic colon and laxative dependency, and bulk laxatives taken with insufficient fluids. Neurological disorders, such as Parkinson's disease, stroke, spinal cord disorders, dementia, depression, metabolic factors, and nursing home placement, are also factors that increase the risk for constipation (Wald, 2016; Bardsley, 2016).

Signs and Symptoms: The first part of an assessment should include the use of a tool to determine the person's normal pattern of bowel function. The most common tool is the Bristol Stool Chart, which classifies stools as hard to pass, ideal consistency, or difficult to control. Descriptions and pictures make it easy for patients to identify the type of stool. It is also helpful to include a food diary to determine dietary sources of the problem. Other considerations are mobility, which allows them to easily get on and off the toilet; proper environment for defecation; underlying medical conditions; medications, including OTC and herbal supplements; and remedies already tried for relief (Bardsley, 2015).

A thorough abdominal and rectal examination is necessary to rule out physical abnormalities and then the data is compiled. The ROME III diagnostic criteria are helpful in confirming the diagnosis of constipation, which must include two or more of the following:

- Straining at least at 25% of defecations
- Lumpy or hard stools in at least 25% of defecations
- Sensation of incomplete evacuation for at least 25% of defecations
- Sensation of anorectal obstruction/blockage for at least 25% of defecations
- Manual maneuvers to facilitate at least 25% of defecations (digital evacuation, support of the pelvic floor) (Basson, 2017)

Diagnostic Tests: All individuals should have a fecal occult blood test at the initial visit of a complaint of constipation. Additional diagnostic tests should be done if they can inform contributing causes or alter the treatment. Laboratory tests might include complete blood count (CBC), glucose, thyroid-stimulating hormone, electrolytes, blood urea nitrogen (BUN), and creatinine. An abdominal radiograph may help to determine the extent and distribution of feces. Sigmoidoscopy or barium enema or both may be indicated in patients with a recent onset of constipation when a lesion is suspected. Checking colonic transit times may be indicated in patients who have a normal number of bowel movements but still complain of constipation. Colonic motility testing may be useful in identifying patterns of colonic activity. A colonoscopy should be done if the patient presents with alarm symptoms, anemia, or positive fecal occult blood (Roque & Bouras, 2015)

Differential Diagnosis: The clinician then evaluates the etiology and contributing causes to rule out obstruction, fecal impaction, irritable bowel syndrome, or diverticular disease (Bardsley, 2015).

Treatment: After the specific etiology is established and factors contributing to constipation are identified, the appropriate pharmacological and nonpharmacological interventions are initiated. The primary aim of treatment is for the patient to have regular, predictable, and comfortable bowel movements. Ideally, this should be accomplished with diet and lifestyle changes. An increase in fiber intake should be done gradually to prevent bloating and flatulence. Adults should eat 18 to 20 g of fiber per day. Insoluble fiber found in whole wheat, whole grains, vegetables, and wheat bran is the best choice. It may take up to 4 weeks for the effects to be noted, so the patient may need laxatives in the interim. An adequate fluid intake of 1.5 to 2 L/day is also important. Fruits and fruit juices, especially prune and apricot, which are high in sorbitol, are an alternative for those who cannot increase the fiber intake (Bardsley, 2015).

Laxatives are recommended when diet and lifestyle changes are not sufficient. The recommendation is to start with bulk-forming laxatives. Psyllium or polycarbophil are commonly recommended, but they require increased fluid

intake, which may not be appropriate for some older adults with heart and kidney disease. Alternatives should be based on the presenting symptoms, the nature of the complaint, the side effects, speed of action, and patient acceptability. Stimulant laxatives, such as senna, cascara, or bisacodyl, increase peristalsis and mucus production. Osmotic laxatives such as polyethylene glycol or lactulose act by drawing fluid from the body. Iso-osmotic laxatives increase water content and trigger peristalsis and defecation. They are becoming the first-line treatment for chronic constipation (Bardsley, 2015). Stool softeners are often prescribed for older adults. These are not laxatives and actually may contribute to constipation, "the mush without the push." Discontinuing inappropriately prescribed stool softeners is an option for medication reduction in polypharmacy.

Follow-Up: Chronically constipated individuals often need to have combined nonpharmacological with periodic pharmacological intervention. When laxatives are prescribed, these individuals should be reevaluated every 3 months for the side effects and the responses to treatment (Bardsley, 2015).

Sequelae: Constipation, although uncomfortable, is rarely life threatening. When associated with pain, distention, or vomiting, constipation may be a sign of a life-threatening mesenteric infarction or a partial or complete bowel obstruction. Straining in older adults may have serious effects on the cerebral, coronary, and peripheral arterial circulation. If stool is not eliminated from the colon and the patient becomes dehydrated, stool may harden, resulting in fecal impaction, a large mass of compacted feces at the intestinal level that cannot be evacuated spontaneously. It is estimated that 7% of long-term residents suffer from impactions yearly (Falcon et al., 2016).

Prevention/Prophylaxis: Adequate dietary fluid and fiber and regular exercise habits help to promote colonic mobility. Bowel elimination patterns should be monitored regularly in individuals who are unable to report bowel habits or bowel discomfort.

Referral: Hospitalization may be necessary in some cases of severe constipation and/or impaction, anorexia, dehydration, or nausea or vomiting. Patients with recent-onset constipation associated with bleeding, anemia, or family history of colon cancer; patients with chronic constipation with anemia, abdominal pain, and weight loss; and patients with recent-onset fecal incontinence should be referred to a gastroenterologist. Referral to a specialist also should be considered when there is a failure to alleviate constipation despite escalating attempts (Roque & Bouras, 2015).

Education: In addition to giving instruction in the dietary measures appropriate in preventing constipation, encourage older patients to maintain regular bowel habits and respond to the urge to defecate rather than suppress it. Patient education regarding diet and exercise is challenging when patients have cognitive impairments, mood disorders, uncontrolled pain, or significant muscle weakness. Patients should be taught to take advantage of the gastrocolic reflex, which is strongest about 30 minutes after meal consumption. All individuals should avoid the use of laxatives and OTC medications known to have constipating side effects (Bardsley, 2015).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Ask the patient about a feeling of incomplete evacuation, obstruction, digital manipulation, infrequent bowel movements, and abdominal bloating.	A	Bardsley, 2015
Consider laboratory testing: CBC, electrolytes, BUN, creatinine, calcium, glucose, and thyroid-stimulating hormone if symptoms persist despite conservative treatment.	A	Roque & Bouras, 2015
Patients with red flags for malignancy or complicated disease should have a colonoscopy.	A	AGA, 2013
The initial management of symptomatic constipation is typically dietary modification (high fiber, exercise, and fluid supplementation).	A	Bardsley, 2015
Polyethylene glycol (PEG) and lubiprostone are effective for the management of chronic constipation.	A	Izzy et al., 2016

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COUGH

Description: Cough is the forceful expelling of air from the lungs involving the use of accessory muscles of the chest and constriction of the glottis.

Etiology: Cough is the voluntary or involuntary mechanism used to eject aspirants or noxious substances, including fluids, solids, dust, and gases from the body's upper and lower airways. Cough is also the body's mechanism to remove mucus from the upper or lower respiratory tracts. Involuntary cough is regulated by the vagal afferent nerve, but higher central nervous system (CNS) cortical modulation can inhibit cough or allow for voluntary cough.

Occurrence: Cough is one of the most common complaints for seeking medical attention. It is divided into three categories based on duration: acute, lasting less than 3 weeks; subacute, lasting between 3 and 8 weeks; and chronic, lasting more than 8 weeks.

Age: Cough occurs at any age.

Gender: There are no gender-specific differences, though females tend to seek medical attention more often.

Ethnicity: No direct ties to ethnicity have been documented.

Contributing Factors: Any stimulation, chemical or mechanical, that irritates the receptors of the cough reflex are causes of cough. These receptors are located in the upper and lower respiratory tract, esophagus, pericardium, diaphragm, and stomach. Environmental exposure that causes cough includes multiple different types of irritants such as chemicals, dust, and pollen, along with infectious agents such as the common cold, tuberculosis (TB), or *Bordetella pertussis.* Trauma, including assault, motor vehicle accidents, or falls that lead to contusion of the lungs can cause cough. Medications may cause cough, particularly ACE inhibitors (in 20% of users).

Smoking is a primary cause of chronic cough, but medical services are often not sought by smokers. Cigarette smoking is the most significant preventable cause of disease, and lung cancer is the number one cause of cancer deaths of both men and women in the United States. Cough from chronic obstructive pulmonary disease (COPD) worsens in intensity and duration if the patient continues to smoke. Bronchogenic cancers are often associated with cough, but metastatic cancers to the lungs seldom result in cough until late in the course of the disease.

Signs and Symptoms: A thorough disease history is required, as multiple chronic diseases present with cough as a symptom. Risk factors and work and home environment assessment is essential in narrowing the cause of cough. Using a systematic anatomical approach provides ease in identifying the trigger and illness. Risk factors for cough include recent exposure, smoking, stroke, immigrant status, use of ACE inhibitors, HIV-positive status, and heart disease. The cough may be productive or nonproductive, and the sputum may be a variety of colors or tinged with blood. Identify the onset, including if it is episodic, continuous, or occurs only certain times of the day; the duration and character; and if it is associated with other symptoms, including fever, tachycardia, dyspnea, weight loss, or pain.

The physical examination begins with vital signs that should include temperature, heart rate, respirations, and oxygen saturation. Next is a thorough examination of the nose and oropharyngeal mucosa. Tenderness to percussion of the facial and maxillary sinuses and swollen, mucus-covered, boggy turbinates indicate allergic rhinitis. A "cobblestone" appearance from chronic posterior pharynx and swollen eyelids are other indicators of an allergic origin. Loss of tooth enamel can indicate GERD. The chest must be assessed for adventitious breath sounds. Wheezes indicate bronchitis or asthma caused by mucus in or constriction of the airways. Rhonchi or crackles are suspicions for pneumonia or infiltrates. Diminished or absent lung sounds by auscultation and dullness to percussion are highly suggestive of pneumonia's consolidation. Rhonchi or crackles with cardiomegaly on x-ray or EKG indicate congestive heart failure and may not be accompanied by peripheral edema.

Diagnostic Tests: No specific tests are indicated when the patient has a cough. The selection and extent of testing depends on the findings of the history and physical examination, duration (acute, subacute, or chronic), and previous evaluations.

Acute cough associated with upper respiratory infection does not require further diagnostic evaluation, unless influenza is suspected, then a rapid influenza diagnostic test may be beneficial. Acute cough with physical findings of life-threatening illness should be appropriately evaluated for the diagnosis of concern. A chest radiograph is appropriate for physical findings consistent with pneumonia (fever, heart rate above 100 bpm, respiratory rate greater than 24 breaths/min, chest examination with findings of local consolidation). Viral cultures, serologic assays, and sputum analysis should not be routinely performed in the diagnosis of acute bronchitis.

Subacute cough that has lasted more than 2 weeks without an apparent cause and is associated with post-tussive vomiting, inspiratory whooping, and paroxysms of coughing should have a nasopharyngeal aspirate or polymer swab of the nasopharynx for culture to confirm the presence of *B. pertussis,* whooping cough. Bacterial isolation is the only certain way to confirm the diagnosis. Testing for TB is indicated when there is an increased risk of the population becoming infected, such as in nursing homes or prisons; in a patient with a recent visit to a country with high prevalence of TB; or in an immunocompromised patient.

Chronic cough diagnostic testing in nonsmokers is guided by elimination through treatment of the most common disorders: upper airway cough syndrome (UACS), previously known as postnasal drip syndrome secondary to rhinosinus diseases; GERD; and asthma. Medical history is crucial to determining progression of diagnostic tests for chronic cough. A chest radiograph is appropriate, though most often negative. An abnormal study would guide further diagnostics; a mass suggestive of lung cancer could be followed up with high-resolution computed tomography (CT) scan, bronchoscopy, and biopsy; congestive heart failure would lead

to cardiovascular evaluation; evidence of infection such as TB would direct microbiologic diagnostics. UACS evaluation includes sinus imaging, either plain sinus films or sinus CT scan. Scans or films taken during the episode of a common cold are not diagnostic of bacterial sinus infection. Evaluation for asthma-induced chronic cough is bronchoprovocation challenge. GERD evaluation may include a barium swallow or 24-hour esophageal pH monitoring test.

Differential Diagnosis: The most important first step is determining if the acute cough is potentially related to a life-threatening condition such as pneumonia, congestive heart failure, or pulmonary embolism. Most acute coughs are related to a respiratory tract condition such as the common cold, flu, acute bronchitis, acute exacerbation of COPD, viral syndrome, or exposure to an environmental irritant.

Subacute cough is usually a cough that lingers on from a respiratory tract infection. A post-infectious cough can last 2 to 3 weeks after onset. Consider UACS, transient bronchial hyper-responsiveness, asthma, acute exacerbation of chronic bronchitis, and pertussis. If it appears to be a noninfectious cough, it should be managed and further evaluated as a chronic cough.

Chronic cough most commonly is UACS, asthma, or GERD. Other potential causes may include nonasthmatic eosinophilic bronchitis (NAEB), COPD, drug-induced cough, fungal infection, and mediastinal masses. A nocturnal cough associated with shortness of breath suggests heart failure, and without shortness of breath is more indicative of an allergy.

Diagnosis of unexplained cough or idiopathic cough requires an exhaustive evaluation by a specialist for uncommon causes before the diagnosis is used.

Treatment: Most often the source of the cough is isolated to the respiratory system and the condition is not life threatening. The following treatments are recommended:

Common Cold, Postnasal Drip, and Throat Clearing: Treat with first-generation antihistamine/decongestant preparation to decrease and hasten the resolution of the cough. Naproxen, an NSAID, may also be used to decrease cough. It is thought to work by inhibiting the inflammatory process.

Acute Bronchitis: This diagnosis is applicable in persons without signs or symptoms of acute exacerbation of COPD, acute asthma, common cold, or pneumonia. In the older adult, a high suspicion of pneumonia should be considered until proved otherwise. Most acute bronchitis is viral and self-limiting; antibiotics do not shorten the course of the disease and should not be used. Beta-2 agonists are not recommended for routine treatment unless there is an asthma component. Expectorants such as guaifenesin may be used. Dextromethorphan or codeine may be used for cough suppression for severe coughing, though research has only been done in chronic bronchitis. A cool mist humidifier in the bedroom may be helpful.

Bacterial Pneumonia: Community-acquired pneumonia (CAP) is treated differently than health-care–associated pneumonia. It is of particular concern in older adult patients and those who are immunocompromised because their symptoms may be less intense. Pneumonia most often is caused by *Streptococcus pneumoniae, Staphylococcus aureus, Hemophilus influenza, Moraxella catarrhalis,* or *Pseudomonas aeruginosa*. If pneumonia is suspected, broad-spectrum antibiotic therapy should be initiated empirically. Patients with any respiratory distress and older adult patients with significantly elevated white blood cell (WBC) counts may need to be hospitalized.

Chronic Bronchitis: Stable patients with a sudden change of symptoms, increase in cough, shortness of breath, purulent sputum, and increase in sputum production are considered to be having an acute exacerbation. Treatment includes a combination bronchodilator, beta-agonists, and ipratropium as an inhaler or as a nebulizer, antibiotics, and oral steroids. Dextromethorphan or codeine may be used for cough suppression. Diagnosis and chronic management of COPD is addressed elsewhere in this text.

TB: Antituberculosis drugs should be initiated if the tuberculin skin test is positive and the patient referred to the local health department.

Drug Reaction: ACE inhibitor should be stopped and only restarted if there is compelling need for the category of drug.

Post-Infectious Cough: A treatment trial of inhaled ipratropium for a cough that is adversely affecting quality of life may be used. If ipratropium alone is not successful, use of inhaled corticosteroids can be considered. Antibiotics are not recommended. Severe symptoms may require use of an oral steroid for a short period while other causes of cough are being considered.

Chronic Cough Treatment: Treatment of UACS, asthma, and GERD in a stepwise, systematic approach is most likely to provide a high rate of success. Because UACS is the most common cause of chronic cough, a trial of first-generation antihistamine and decongestant should be tried. Cough should improve in 1 to 2 weeks. If the response is positive, the medication should continue because it may take several weeks for marked improvement. A partial response indicates more than one cause of the cough, and evaluation and treatment for asthma or GERD is reasonable.

Follow-Up: The tenacity of follow-up depends on the severity of the illness and the impact on the patient's life. For pneumonia or bronchitis with minimal dyspnea, a call to check on the patient is appropriate. Rising temperature or increasing difficulty breathing requires immediate attention or emergency department evaluation. Patients with resolving pneumonia should be evaluated in 2 to 3 weeks; a repeat chest x-ray may be taken. Chronic cough may take multiple follow-up visits, evaluation, and treatment trials.

Sequelae: If a cough is caused by lung cancer, the prognosis is ominous. Cough from COPD worsens in intensity and duration if the patient continues to smoke. In older adults, chronic cough can contribute to stress incontinence.

Prevention/Prophylaxis: Preventive measures include smoking avoidance or cessation intervention, evaluation of the work and home environment, and humidification of the sleeping area with a cool mist humidifier. Patients should be provided vaccinations following Centers for Disease Control and Prevention (CDC) guidelines, including annual flu shots, Pneumovax, and Tdap.

Referral: A referral to a specialist can be initiated anytime during the evaluation and treatment of a cough. Some results, such as lesions suspicious for cancer on radiography, require referral to a specialist immediately. A cough specialist, allergist, or pulmonologist referral is appropriate for a chronic cough that is persistent. Other specialists who may be helpful are a GI specialist for evaluation of GERD, an infectious disease specialist for infections and immunocompromised patients, and a cardiologist for heart conditions.

Education: Recommend smoking cessation classes and allergen avoidance training. Cough can last longer than patient expects.

DIAGNOSIS	CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Common cold, postnasal drip, and throat clearing	First-generation antihistamine/decongestant preparation, naproxen	B	Pratter, 2006a, 2006b Smith et al., 2014
Acute bronchitis without signs or symptoms of pneumonia, COPD exacerbation, acute asthma, or common cold	Most acute bronchitis is viral and self-limiting; antibiotics do not shorten the course of the disease and should not be used	B	Braman, 2006a Holzinger et al., 2014
Bacterial pneumonia—community-acquired	Broad-spectrum antibiotic therapy should be initiated empirically; older adult patients with significantly elevated WBC counts or patients with respiratory distress may need to be hospitalized	B	Holzinger et al., 2014 Musher & Thorner, 2014
Chronic bronchitis—sudden change in symptoms	Combination bronchodilator, beta agonists, and ipratropium as an inhaler or as a nebulizer, antibiotics, and oral steroids; dextromethorphan or codeine may be used for cough suppression	A	Irwin et al., 2006
Drug reaction	ACE inhibitor should be stopped	A	Dicpinigaitis, 2006 Kahrilas et al., 2016
Post-infectious cough with other conditions ruled out	Treatment trial of inhaled ipratropium for cough if affecting quality of life; can consider inhaled corticosteroids or use 30–40 mg of prednisone for a limited period of time for severe paroxysms. Cough suppression for failure of other treatments. Antibiotics are not recommended.	B	Braman, 2006b Iyer & Lim, 2013
Upper airway cough syndrome (UACS)	First-generation antihistamine and decongestant	C	Iyer & Lim, 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

DEHYDRATION

Description: Dehydration, or fluid volume depletion caused by too little fluid intake, too much fluid lost, or both, is a common condition in older patients. Fluid balance, intake equals output, is essential to health.

Etiology: Total body water decreases with age. The total body water of an adult age 61 to 74 years is approximately 40% compared with 60% for younger adults (Miller, 2015). Even small decreases in fluid intake can cause dehydration quickly in an older adult, due to a smaller fluid reserve. The ability of the kidneys to concentrate urine declines with age, so that even when deprived of water, urination is not significantly reduced. Also, thirst decreases as a person ages, which is an important self-regulation against dehydration.

Dehydration can be defined as a fluid and electrolyte disturbance arising from either a water depletion or a sodium depletion in which there is accompanying water loss. It can be classified into three different types based on the possible causes (see Table 5-2): isotonic, hypotonic, and hypertonic.

Occurrence: Dehydration has been reported to be the most common fluid and electrolyte imbalance in older adults. The prevalence is increasing, with 23% of patients over 70 years of age and 34% of nursing home patients who were admitted to the hospital diagnosed with dehydration (Theerthakaria & Madlon-Kay, 2016).

Age: Older people are particularly susceptible to dehydration because of age-related changes noted previously, with individuals older than 85 years at the highest risk.

Gender: Not significant.

Ethnicity: African Americans have a greater likelihood of dehydration than Caucasians (Stookey, Pieper, & Cohen, 2003).

Contributing Factors: The causes of dehydration are multifactorial and include decreased mobility and the presence of chronic diseases such as diabetes, cancer, and cardiovascular and renal disease, which make older adults more sensitive to fluid and electrolyte imbalance. Other risk factors for dehydration in older adults include dependency for eating or drinking, mouth pain, poorly fitted dentures, and difficulty swallowing. Vomiting, diarrhea, hemorrhage, and increased metabolic states that result in excess loss of fluids (fever, infection, excess sweating, excess urination) are also contributing factors. The use of certain medications, such as diuretics, anticholinergics, psychotropics, or laxatives, also may increase the risk. Dehydration is the most frequent secondary condition resulting from debilitating issues such as fever, confusion, pain, or pneumonia. Cognitive impairment (delirium, dementia, depression) has been associated with dehydration in older adults.

TABLE 5-2 Types of Dehydration

TYPE OF DEHYDRATION	DESCRIPTION
Isotonic	A balanced loss of both sodium and water causes extracellular fluid loss that increased blood viscosity. Common causes: vomiting, diarrhea, osmotic effects of glucose.
Hypertonic	Excessive water loss results in raised sodium levels and shifts from the intracellular space to extracellular space. Common causes: renal tubular disease, osmotic diuresis, resistance to vasopressin.
Hypotonic	Excessive sodium loss results in extracellular fluid loss. Common causes: overuse of diuretics, chronic salt wasting.

Signs and Symptoms: Assessing for hydration status in the clinical setting can be difficult, as there is no valid tool to assess for risk of dehydration in patients (Burns, 2016; Miller, 2016). A dry, furrowed tongue and mucous membranes; sunken eyes; headaches; dizziness on standing; confusion; concentrated or dark urine; weight loss; and upper body muscle weakness may indicate dehydration. Clinical signs may not appear, however, until dehydration is far advanced. In addition, the usual signs of constipation, disorientation, dry mucous membranes, orthostatic hypotension, and weight loss may be caused by other factors. If one or more of these are present or are a change from baseline, dehydration may be the cause. While poor skin turgor is an expected finding in older adults, it is also a classic sign of dehydration. Clinical assessment should include assessment of skin turgor at the sternum or dorsum of the hand and inspection of oral mucous membranes for dryness and tongue for dryness and longitudinal furrows (Miller, 2016; McCrow, 2016). Signs and symptoms of hypovolemic shock include altered mental status; hypotension; tachycardia; cold, clammy skin; and rapid, deep respirations.

Diagnostic Tests: Serum markers provide the most reliable indicators of dehydration and these include serum sodium, increased serum osmolality, and BUN–creatinine ratio. The serum albumin may be higher than normal when the patient is dehydrated.

Differential Diagnosis: When dehydration is identified, it is important to identify the cause and contributing factors and address these. Weight gain and edema from congestive heart failure can conceal a patient's dehydrated status.

Treatment: Individualize treatment, depending on the type of dehydration. The route of administration (oral or IV or subcutaneous) is determined by what is safe and effective. The recommended daily intake of fluids for older adults should not be less than 1,600 mL/24 hours to ensure adequate hydration, and this is adjusted for climate and physical activity (Theerthakaria & Madlon-Kay, 2016). Regular presentation of fluids to bedridden individuals can maintain adequate hydration status. Provide continuous verbal cues to encourage drinking fluids. Fluids given with medication can be an important source of fluids, so fluids should be encouraged

at this time. Determine the patient's personal preferences of fluid.

Follow-Up: The timing of patient follow-up depends on the seriousness of the identified cause and the response to treatment.

Sequelae: Dehydration has severe consequences for older adults and can have a major impact on quality of life. Dehydration is associated with chronic health problems, including constipation, falls, medication toxicity, increased confusion, and infection. Inadequate fluid intake can have serious medical consequences such as renal failure, hyperthermia, bowel obstructions, delirium, cardiovascular symptoms, and death. A primary or secondary diagnosis of dehydration has been associated with poor outcomes and increased length of stay for older adults who are hospitalized. Older adults admitted with a primary diagnosis of dehydration have a 1-year mortality rate of 42% (El-Sharkawy et al., 2016).

Prevention/Prophylaxis: Educate patients, families, and caregivers that to prevent dehydration the body needs to adequate fluid intake. All liquids are not the same in maintaining fluid balance. Water is the best and should be at least half of the daily intake of fluid. Add milk, fruit and vegetable juices, and non-salty soups for variety. Decrease coffee, tea, alcohol, colas, and liquid diet supplements because they may cause dehydration. The use of a 24-hour fluid balance chart can help determine if the patient is at risk or is dehydrated.

Referral: Refer as indicated by source of symptom. Hospitalization is indicated when the patient has severe signs and symptoms such as dizziness, altered mental status, or lethargy; is hemodynamically unstable; or cannot tolerate oral fluids.

Education: Explain causes of symptoms and measures taken to determine cause and treatment. Aim for 1,600 mL of oral liquids per day unless contraindicated by certain medical conditions such as congestive heart failure. Allow adequate time for eating at mealtimes. Meals can provide two-thirds of daily fluids. Offer fluids regularly during the day. Encourage consumption of fluids with medications. Staff education is critical; staff should report any significant weight loss, change in fluid consumption, or other relevant symptoms, such as dry mucous membranes.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The recommended daily intake of fluids should not be <1,600 mL/24 hours to ensure adequate hydration.	A	Theerthakaria & Madlon-Kay, 2016
Hypodermoclysis is as effective as IV rehydration of older adults with mild-to-moderate dehydration.	A	Theerthakaria & Madlon-Kay, 2016 Thomas et al., 2008
Verbal prompts have been shown to increase fluid intake in nursing home residents.	A	Oates & Price, 2017
Evidence-based practice guidelines for hydration management.	A	Mentes & Kang, 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

DIARRHEA

Description: Diarrhea is the passage of increased stool frequency, liquidity, or volume. It is objectively quantified as passage of three or more loose or watery stools or increased stool weight of greater than 200 g or 200 ml per 24 hours (Shah et al., 2012). Most episodes of diarrhea are caused by viral gastroenteritis, are short lived, and resolve without treatment (Barr et al., 2014). Chronic diarrhea is defined as diarrhea that lasts longer than 4 weeks.

Etiology: In normal bowel motility, water is not actively transported across the intestinal mucosa, it moves passively by osmotic forces generated by solutes such as electrolytes and nutrients. Absorption and secretion take place simultaneously, but when there is either a decrease in absorption or an increase in water within the intestinal lumen, diarrhea occurs. The fundamental cause of diarrhea is the incomplete absorption of water from the intestinal luminal contents. The pathophysiology mechanisms of diarrhea include osmotic, secretory, inflammatory, and altered motility leading to food and liquids being passed too quickly through the body and being excreted through the colon as loose or watery stool. Diarrhea is often accompanied by abdominal cramping, as well as nausea. There are various causes of diarrhea (see Table 5-3). Note that *Clostridium difficile* infection should

TABLE 5-3 Causes of Diarrhea

DISEASE	MECHANISM	COMMENTS
Physical Causes Contributing to the Incidence of Diarrhea		
Dementia	Impairs awareness of need to defecate, loss of inhibitory control	Manifestations depend on type and severity of dementia
Stroke	Impaired awareness of anal control, slowed motor processing communication with frontal lobe damage	Dependent on size of infarct or hemorrhage within the brain
Fecal impaction	Altered rectal sensation, liquid stool escaping around solid feces in colon	
GI disease	GI dysfunction/disease with excessive stool volume and rapid gut transit	Causes include viral or bacterial gastroenteritis, colitis, Crohn's disease, celiac disease, colon cancer, irritable bowel syndrome, rectocele, and other GI diseases *Clostridium difficile* can cause severe diarrhea in the older adult individual and can lead to death
Medication Causes		
Laxatives	Medications causing increased gastric motility or increased water in stools	
Fiber products	Increase fluid in the stool to keep it soft and move easier but can lead to diarrhea and FI	
Magnesium	Draws fluid into the bowels through osmosis	Magnesium containing antacids and certain laxatives contain significant amounts of magnesium
Other medications	Antibiotics can cause destruction of the colon's natural flora, leading to diarrhea Other medications that can cause diarrhea include proton pump inhibitors, SSRIs, and NSAIDs	
Other		
Physical deficits	Inability to recognize need to defecate (cognitive problems) or inability to ambulate to toilet without assistance	Common causes of cognitive problems include dementia Common causes of mobility problems include debilitating arthritis, hemiplegia, or any condition affecting gait
Dietary causes	Other foods in susceptible individuals include diet high in fiber, spicy foods, fatty/greasy foods, cured meats, smoked meats, dairy products, caffeine, and sorbitol	Sugar-free gum and artificial sweeteners can cause diarrhea and FI Avoidance of irritating foods can eliminate the FI
Excessive fluid intake	Overhydration can lead to diarrhea and possibly cause FI	

Source: Barr & Smith (2014); Keller & Surawicz (2014); Nodrqvist (2014); Shah et al. (2012); Sweetser (2012).

be suspected as a cause of diarrhea in patients with watery diarrhea during or shortly after receiving antibiotic therapy or recent hospitalization.

Occurrence: In the United States, an estimated 48 million foodborne diarrheal illnesses occur annually, resulting in more than 128,000 hospitalizations and 3,000 deaths (Barr et al., 2014). The greatest risk factor is contact with another individual with diarrhea.

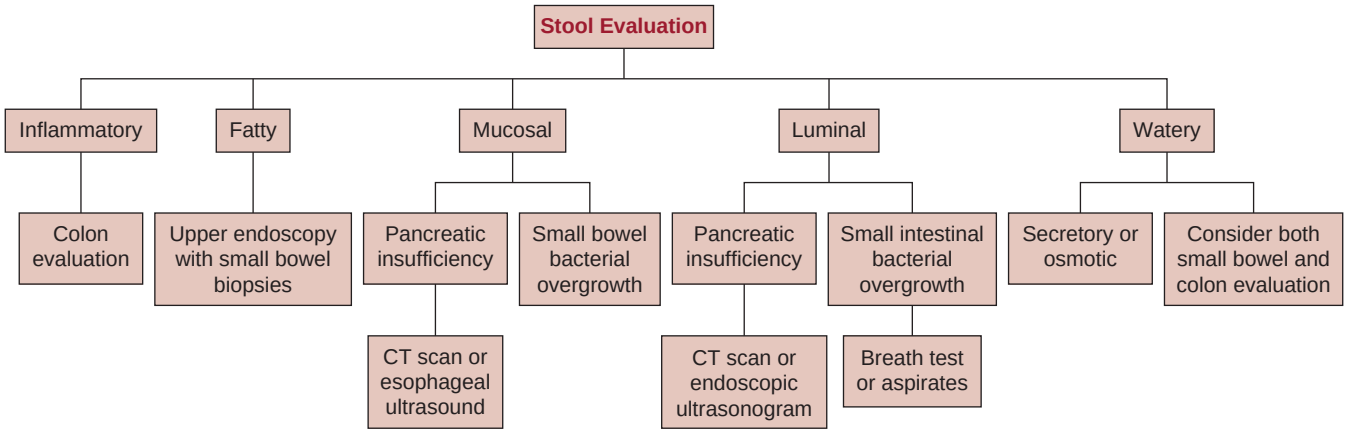
Age: Diarrhea occurs at any age. The severity of illness and the mortality rate is increased in individuals more than 65 years old, especially if the individual has a concurrent medical illness (Wanke, 2016).

Gender: No gender-specific differences.

Ethnicity: No direct ties to ethnicity have been documented.

Contributing Factors: The following can be contributing factors in the development of diarrhea: recent travel, family history of inflammatory/bowel disorders, recent antibiotic usage, exposures to either contacts or foods including tube feedings, whether or not the individual is immunocompromised such as with HIV, recent hospitalization, age of more than 65 years, extended stay in hospital or nursing home, recent chemotherapy, recent GI surgery including post-pyloric tube feeding, and medications. The list of medications that can cause diarrhea include diuretics, beta blockers, NSAIDs, antibiotics, quinidine, chemotherapy, colchicine, laxatives, digitalis, theophylline, prostaglandins, cholinergic agents, and phenolphthalein.

Signs and Symptoms: The initial clinical assessment is to obtain a detailed history to include symptom assessment parameters, including onset, frequency, amount, and character of the stool (the presence of blood, mucus, or atypical color) and any associated symptoms such as abdominal pain, cramping, fever, nausea/vomiting, or bloating. Include the patient's baseline bowel pattern, usual dietary intake, food intolerance history, exposure to others with illness, and any recent dining out experiences. A thorough medication



Type of Diarrhea	Assessment	Common Causes
Inflammatory diarrhea	Frequent, small volume, bloody stools, and may be accompanied by fever and severe abdominal cramping May have leukocytes in stool Laboratory tests may demonstrate elevated C-reactive protein level or elevated sed rate (ESR), and low albumin level	Crohn's disease Ulcerative colitis Ischemic colitis Infectious processes including *C. Difficile*, cytomegalovirus, tuberculosis, and *Entamoeba histolytica*
Fatty diarrhea	History of weight loss Greasy or bulky appearing stools (may be difficult to flush and creates oily toilet requiring a brush to remove)	Malabsorption syndromes Celiac disease Chronic pancreatitis Inadequate bile acid concentration Small intestinal bacterial overgrowth Liver cirrhosis
Watery diarrhea	Classified as osmotic or secretory in origin Differentiate: Fasting decreases stool production in osmotic diarrhea but does not affect secretory diarrhea	Osmotic diarrhea is due to poorly absorbed ions or sugars caused by ingestion of anions such as laxatives and antacids or ions such as magnesium; or carbohydrate malabsorption of poorly absorbed sugars or sugar alcohols such as sorbitol or xylitol Secretory diarrhea is due to disruption of epithelial electrolyte transport caused most commonly by infectious processes—either viral or bacterial, as well as parasitic or peptide-secreting endocrine

FIGURE 5-1. Algorithm to assist in determining the appropriate work-up needed for diarrhea.

review including any OTC herbal remedies or laxatives needs to be conducted. A dietary history 3 to 5 days before onset of symptoms would be helpful, with particular attention to the ingestion of meat, dairy products, and seafood. The associated symptoms of fever, abdominal pain and distention, vomiting, myalgias, and headache with abrupt onset suggest acute diarrhea etiology. In contrast, fatigue, muscle weakness, weight loss, alternating diarrhea and constipation, incomplete evacuation, abdominal pain, and protracted or recurring symptoms suggest chronic diarrhea etiologies.

Physical examination should include vital signs, weight, and assessing for the presence of postural hypotension. Assess the skin for turgor and the condition of the tongue and mucous membranes. Check for evidence of jaundice, rashes, or lesions. Examine the thyroid for enlargement or lesions. Perform a thorough examination of the abdomen for distention, rigidity, tenderness, bowel sounds, masses, hepatomegaly, or splenomegaly. Perform a rectal examination to check for impaction, include hemoccult testing to assess for bleeding, hemorrhoids, polyps, fissures, fistulas, or evidence of malignancy. Include a general inspection for arthritis or lymphadenopathy. Stool should be examined for mucus and the presence of blood. Figure 5-1 describes an algorithm to assist in determining the appropriate work-up needed for diarrhea.

Diagnostic Tests: For most cases of diarrhea of acute onset, no diagnostic testing is warranted unless the individual appears acutely ill. If abnormal findings are observed on physical examination or the individual has additional comorbid conditions, then diagnostic testing may be necessary. CBC and complete metabolic panel (CMP) to include electrolytes and renal function will help identify an infectious process and any electrolyte imbalance, and the presence of dehydration. Abdominal x-rays are helpful to identify the presence of obstruction or ischemia. A barium enema can reveal malignancies, polyps, or abnormalities of the mucosal lining. Colonoscopy or sigmoidoscopy may be useful when

symptoms are persistent and unexplained. If you suspect laxative abuse, you may order a stool laxative screen. Send a stool sample to test for occult blood, *C. difficile,* salmonella, *Escherichia coli,* lactoferrin, campylobacter, leukocytes, ova, and parasites. If the stool tests positive for leukocytes, the sample should be cultured to identify any bacterial etiology.

Differential Diagnosis: Distinguish first between acute diarrhea and chronic diarrhea. In acute diarrhea, consider inflammatory bowel disease, fecal impaction, irritable bowel syndrome, diverticulitis, antibiotic-associated pseudomembranous colitis, bowel ischemia, drug side effects, food poisoning, and virus or bacteria exposure. In chronic diarrhea consider ulcerative colitis, Crohn's disease, microscopic colitis, celiac disease, irritable bowel syndrome, bile acid malabsorption, occult neoplasm, post-radiation entercolitis, and functional problems. A fecal specimen should be obtained for anyone in cases of severe, bloody, inflammatory, or persistent diarrhea or if an outbreak is suspected.

Treatment: In many cases of diarrhea, replacing lost fluid and electrolytes is the only treatment needed, but treatment is dependent on the cause. Frail older adult individuals are at increased risk for dehydration and multisystem failure. If diarrhea is severe, refer the patient for emergent evaluation and treatment. In the hospital, the older adult patient should be treated aggressively with IV fluid therapy with close monitoring of the cardiopulmonary status, renal status, and electrolytes. In cases of acute-onset diarrhea in which a viral cause is likely, provide symptomatic treatment to maintain hydration and electrolyte balance. Use of medications to slow the intestinal motility and the diarrhea may actually extend the length of the illness.

Follow-Up: Scheduling follow-up visits will be determined based on the cause of the diarrhea. In the case of acute diarrhea, reevaluate the patient after 3 to 5 days of following the initial treatment plan. If diarrhea persists, diagnostic studies may be indicated and the patient's hydration status reevaluated.

Sequelae: Depending on the cause and extent of the diarrhea, complications may include dehydration, sepsis, shock, anemia, fluid and electrolyte imbalance, malnutrition, and peritonitis.

Prevention/Prophylaxis: Prevention depends on the cause. Oral replacement of fluids is of highest priority, but in severe cases IV replacement may be warranted. Careful cooking and storage of food is important to avoid spoilage. Check expiration dates on dairy and meat products before using. Clean cutting boards and utensils. Educate about cross-contamination when cooking, such as with raw meats, to prevent exposure to pathogens. For individuals who are traveling to a foreign country, begin appropriate prophylactic treatment (World Health Organization, 2012). Caution travelers about avoiding undercooked foods, raw foods, buffet food, unpasteurized dairy products, untreated water, and ice. Carbonated drinks, bottled water, and boiled water are considered safe for drinking and to use for oral hygiene.

Prevention of transmission of diarrhea to or from others (including young children), in hospitals, and in the long-term care facility is extremely important. Frequent hand washing by patients and health-care providers is the key to preventing transmission of infectious diarrhea. Avoidance of long-term laxative use and prudent use of antimicrobial agents are also important in preventing diarrhea.

Referral: Further work-up may be necessary in individuals who are exhibiting prolonged diarrhea, severe dehydration, recurrent episodes of diarrhea, and in individuals who report blood in stools. Refer to the hospital for consideration of an inpatient stay if IV fluids and/or electrolyte replacements are needed, or if diarrhea persists. The patient may need to be evaluated by a gastroenterologist or an infectious disease specialist depending on the causative agent of the individual's diarrhea. Complications may include dehydration, malnutrition, peritonitis, sepsis, shock, anemia, or electrolyte imbalances, which can be life threatening (such as with hypokalemia).

Education: Explain to the patient, family, and caregivers the cause of symptoms, diagnostic and treatment measures, follow-up, and preventive strategies. Emphasize the need for early intervention to prevent dehydration.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Classify diarrhea into three categories (watery, bloody, or fatty) based on the history, physical examination, and testing.	A	Sweetser, 2012
Specific therapy depends on the underlying cause.	C	Barr & Smith, 2014 Sweetser, 2012
Teach patients the signs and symptoms of dehydration.	C	Barr & Smith, 2014 Arena et al., 2014
Metronidazole 500 mg PO 3 times a day for 10–14 days is the initial and most cost-effective treatment for *C. difficile* diarrhea.	A	McDonald et al., 2018

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

DIZZINESS

Description: Dizziness is an imprecise term commonly used to describe various subjective symptoms by lay persons, especially older adults. The practitioner must understand the patient's personal meaning of dizziness to determine etiology of the dizziness. Patients, however, tend to find it difficult to articulate their symptoms well. Thus, establishing the time course, provoking or aggravating factors, concurrent symptoms, age, pre-existing conditions, as well as interpreting physical examination findings, are crucial in establishing a diagnosis. Despite a thorough history and physical examination, the final cause of dizziness is not identified in some patients. The most common clinical categories of dizziness are vertigo, unsteadiness or gait instability, light-headedness, and disequilibrium.

Etiology: Dizziness may result from mixed pathologies; medications; and neurological, otolaryngological, cardiovascular, or psychiatric causes. Benign paroxysmal positional vertigo (BPPV), a disorder arising from the inner ear, is the most common cause in individuals over age 70 years (Luscher, Theilgaard, & Edholm, 2014). People greater than age 70 years complaining of dizziness with associated neurological deficits but lacking vertigo require a thorough investigation of the cause of their dizziness. Individuals without a neurological deficit and no definitive cause of dizziness identified, can be diagnosed with presbystasis. This is age-related disequilibrium not attributed to any pathology and is characterized by a gradual onset of difficulty walking (Belal & Glorig, 1986).

Occurrence: Dizziness is a common symptom, with 45% of affected nonhospitalized patients seeking care from primary care providers (Sloane, 1989).

Age: Dizziness occurs in 30% of individuals over age 60 years and increases to more than 50% in individuals over age 85 years old (Jönsson, Sixt, Landahl, & Rosenhall, 2004).

Gender: Dizziness occurs more frequently in women.

Ethnicity: Not significant.

Contributing Factors: Contributory medications include antihypertensives, anticonvulsants, psychotropic drugs, and ototoxic drugs. Physiological changes occurring with age can affect equilibrium. Altered sensitivity of the baroreceptors, as well as diminished sensory and motor pathways over time, increase the vulnerability to syncope and gait instability.

Signs and Symptoms: The patient's description of the symptom should help determine the type of dizziness experienced.

Vertigo: Vertigo is illusion of movement of the patient or the environment due to central or peripheral causes. Associated symptoms may include nausea, vomiting, unsteadiness, or visual disturbances. The time course of vertigo assists in determining the appropriate diagnosis. Episodic vertigo following a viral illness and lasting days with nausea and no associated symptoms is usually due to vestibular neuritis. Episodic vertigo lasting for seconds and associated with position changes is probably due to BPPV. Vertigo of sudden onset lasting for minutes can be due to migraine or cerebrovascular disease, especially if neurological deficits are present. Vertigo lasting hours associated with hydropic ear symptoms is probably caused by Ménière's disease. Older adults with head or neck pain post trauma can develop cervicogenic vertigo provoked by movement or sustained positions of the neck.

Presyncope: In presyncope, patients have the sensation of nearly fainting. Associated symptoms include perspiration, fatigue, pallor, palpitations, dimming of auditory or visual stimuli.

Disequilibrium: Patients with disequilibrium describe a sense of imbalance. Symptoms improve dramatically by bracing on a stationary object and are exacerbated by vision deficits or uneven surfaces. Associated symptoms may include chronic hearing loss, chronic lower extremity sensation deficits, poor coordination, and general weakness. Patients with acute gait instability need further evaluation for cerebrovascular disease.

Light-Headedness: Patients often describe presyncope symptoms as light-headedness. This can often be associated with anxiety or depression.

Physical Examination: The physical examination should be comprehensive and include the following components:

General: Observe how the patient moves around. Does he or she hold onto the furniture or walls? Any signs or symptoms of fever?

Head, Eyes, Ears, Nose, and Throat: Test for extraocular movement, watching for nystagmus. Rotatory nystagmus suggests a peripheral cause of dizziness, but this alone is not a conclusive finding. Vertical nystagmus suggests a central lesion and needs further investigation. In many clinical situations, rotatory nystagmus is superimposed on horizontal nystagmus. Check the patient's static monocular or binocular visual acuity and dynamic visual acuity. Evaluate visual fields. Perform a funduscopic examination, looking for papilledema and other abnormalities. Inspect the ears for accumulation of fluid, tympanic membrane abnormalities, or presence of cerumen impaction. Perform Weber's and Rinne's tests if decreased hearing to conversational level auditory stimulus. Examine the neck for range of motion and flexibility.

Cardiovascular: Evaluate central and peripheral function. Auscultate for heart sounds; evaluate for arrhythmias and murmurs. Evaluate the carotid arteries for bruits. Check vital signs, including orthostatic blood pressure and heart rate. A drop in systolic blood pressure of 20 mm Hg or diastolic blood pressure of 10 mm Hg is significant, especially if it reproduces the patient's symptoms.

Neurological: Test for coordination deficits with finger to nose, heel to shin, past pointing, and rapid alternating movements. If coordination tests are abnormal, keep vestibular dysfunction or cerebrovascular insufficiency in the differential. Test sensation including pain, temperature, vibration, proprioception, and Romberg. Patients with cerebellar disease have difficulty standing with their feet together. Patients with decreased position sense or other sensory deficits in their lower extremities are able to compensate with eyes open but

sway with eyes closed. Perform gait assessment, including tandem gait. Complete a motor examination and evaluate deep tendon reflexes. The inclusion of psychological tests (mental status, depression, anxiety) is appropriate if a psychogenic cause is suspected.

Special Diagnostic and Screening Tests: Forced hyperventilation involves asking the patient to breathe 20 to 30 times per minute for 2 to 3 minutes. This normally causes dizziness and finger and perioral numbness. If presenting sensations are reproduced, the dizziness may be related to anxiety.

Have the patient march in place for 30 seconds with the eyes closed and arms extended in front. Be careful not to orient the patient with any sounds (e.g., your voice or a ticking clock). Patients with absent or reduced vestibular function (from prior vestibular damage) rotate more than 30 to 45 degrees while marching.

For the Dix-Hallpike maneuver, assist the patient quickly to lie down on the examination table with the head hanging over the back of the table at an approximate 30-degree angle with the face turned 45 degrees to the right. While holding the patient's head in place for 1 minute, observe the patient for nystagmus and ask whether this reproduces the dizziness (vertigo) symptoms. Bring the patient back to a sitting position and observe for 1 minute. Repeat the test with the head turned 45 degrees to the left. If vertigo is reproduced, the test should be repeated two or three times on the side that caused the most severe symptoms to determine if the nystagmus and symptoms begin to disappear. Patients with benign postural vertigo (BPV) experience severe vertigo 5 to 15 seconds after the head is turned and nystagmus is induced. Position change must be completed in 2 seconds, as the signs typically fade in less than 1 minute, and repeated testing causes the symptoms to disappear. The safety of this test must be considered if the patient is frail or has neck or back problems.

Diagnostic Tests: Diagnostic tests are dependent on the presenting symptoms, the history and physical examination, as well as comorbid factors. The HINTS (head-impulse, nystagmus, test of skew) examination can help distinguish peripheral from central etiologies, as well as delineate which patients may need diagnostic testing (Muncie, Sirmans, & James, 2017). Select testing might include CBC for anemia, serum glucose for hypoglycemia, EKG or Holter monitoring for arrhythmia, audiometry if hearing loss is present, or vestibular function tests (such as videonystagmography or electronystagmography). Neuroimaging (CT, CTA, MRI, or MRA) must be ordered if neurological symptoms are present to evaluate for a central lesion or vascular occlusions causing ischemia.

Differential Diagnosis: Differentiate between vertigo (a sensation of movement), light-headedness (a sensation of being about to faint), and syncope (an actual loss of consciousness). If dizziness has a predictable pattern associated with it (e.g., mid to late morning, mid to late afternoon, 2 to 4 hours after eating), suspect hypoglycemia.

Treatment: Treatment is based on the cause of the dizziness. Physical therapy for balance training or vestibular rehabilitation can be the primary treatment or it may supplement other management approaches. Traditional vestibular rehabilitation, although shown to be effective, requires face-to-face time, resulting in inconsistent completion rates. Recently, however, there are ongoing studies evaluating the efficacy of blended online and face-to-face vestibular rehabilitation protocols in the hope of improving access and promoting consistent completion rates (Geraghty et al., 2014).

Patients with BPV can perform the Epley maneuver, which loosens particles out of the posterior semi-circular canal and back into the utricle. If the cause of light-headedness is medication side effects or volume deficiencies, adjustments are indicated. Support stockings may alleviate symptoms from orthostatic hypotension. Appropriate management of cardiovascular, cerebrovascular, or psychogenic conditions is warranted.

Antihistamine medications like meclizine were initially shown to reduce the severity and frequency of attacks, as well as signs and symptoms associated with positional or continuous vertigo of vestibular origin like vestibular neuritis. However, they have not been shown to be effective in patients with peripheral vestibular diseases like BPV or central vestibular diseases, despite their frequent use for these conditions (Cohen & DeJong, 1972). Benzodiazepines, despite the risk of somnolence and potential for dependency, are another class of drugs frequently used to treat associated symptoms of vertigo, although recent studies have shown no difference between meclizine and diazepam (Shih et al., 2017). These medications require caution, as they can worsen the symptoms of dizziness and have other side effects, which are particularly problematic in older adults.

Follow-Up: Patients should follow up if symptoms worsen, change from one type of dizziness to another, or do not resolve within 2 weeks.

Sequelae: Older adults with dizziness are at high risk for falls and injury, as well as self-induced immobility due to fear of falling. In patients younger than 80 years, the prevalence of falls is 16.5%, whereas in patients older than 80 years, the prevalence is 31.7% (Olsson Moller et al., 2013). Clinicians can view dizziness as a geriatric syndrome that requires specific strategies to reduce significant impairment and complications (Tinetti, Williams, & Gill, 2000).

Prevention/Prophylaxis: Educate older adults to move extremities before rising from positions held for any length of time to compensate for age-related changes in vascularity. Evaluate for medication side effects before adding medications to combat the dizziness.

Referral: Persistent symptoms with unknown etiology require a referral to neurology, cardiology, otolaryngology, or psychiatry as appropriate.

Education: Explain the underlying cause, rationale for treatment, and importance of exercises. Teach the patient the exercises and ask for return demonstration. Explain that this condition may take time to completely resolve, but that it is important to seek care for worsening or persistent symptoms. Encourage the use of assistive devices for safety until the condition resolves.

CLINICAL RECOMMENDATIONS	EVIDENCE RATING	REFERENCES
Balance disorders and presence of dizziness in the elderly is a strong predictor of falls. Falls are the leading cause of accidental death in people older than 65 years.	A	Fernández, Breinbauer, & Delano, 2015
Performing a medication history and review is important for adjusting medications to prevent/resolve medication-related dizziness.	A	Shoair, Nyandge, & Slattum, 2011
No routine neuroimaging tests are indicated in the diagnosis of dizziness unless neurological symptoms are present. However, check glucose levels in diabetics and monitor cardiac rhythm in those >45 years old.	C	Muncie, Sirmans, & James, 2017
Vestibular rehabilitation is effective for management of unilateral peripheral vestibular dysfunction. It is unclear at this time if Internet-based vestibular rehabilitation is superior to traditional vestibular rehabilitation.	B	Geraghty et al., 2014

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DYSPHAGIA

Description: Dysphagia is a swallowing disorder involving the inability to get food from the mouth to the stomach. The problem is divided into oropharyngeal causes related to the inability to initiate the act of swallowing and esophageal causes from disorders of impaired transit through the esophagus.

Etiology: The causes of dysphagia are multiple and diverse. Aging does not cause clinical dysphagia; however, some normal changes of aging, such as changes in nerve and sensory function, delay in pharyngeal swallow, loss of esophageal sphincter tone, decreased lower esophageal sphincter relaxation, delayed emptying of the esophagus, decreased facial muscle and masticatory strength, and oral cavity changes (e.g., impaired masticatory function, reduced salivary flow, xerostoma) can contribute to or aggravate any swallowing problems.

Oropharyngeal dysphagia is usually related to neuromuscular impairments of the tongue, pharynx, and upper esophageal sphincter. Stroke is the most common cause of oropharyngeal dysphagia, but other causes include CNS disorders such as extrapyramidal syndromes, Alzheimer's disease, myogenic disorders (e.g., myasthenia gravis or myopathies), structural disorders (e.g., Zenker's diverticulum, cricopharyngeal stenosis, or tumors). Xerostoma, dry mouth, is a common occurrence in older adults, affecting 16% of men and 25% of women, and adversely affects swallowing by impairing the ability to form an adequate bolus of food to allow for smooth transport of food/fluid.

Esophageal dysphagia is usually a result of a motor disorder (e.g., muscular dystrophy, myasthenia gravis, scleroderma), motility or neurological disorders (e.g., achalasia, multiple sclerosis, amyotrophic lateral sclerosis), or an obstruction from either intrinsic (e.g., carcinoma, stricture, web, diverticula) or extrinsic (e.g., mediastinal tumors, vascular anomalies) sources. Achalasia is characterized by slow, progressive dysphagia for liquids and solids with weight loss, and its etiology is unknown. Esophagitis is another etiology, usually secondary to GERD, herpes virus, or a retained pill (primary offenders include NSAIDs, quinidine, potassium, ferrous sulfate, tetracycline, and alendronate). Drugs with dopamine antagonist action such as phenothiazine and metoclopramide can cause dystonia and dyskinesia, leading to dysphagia.

Age/Occurrence: Swallowing disorders occur in 13.5% of adults and are more common in older adults. Individuals over age 65 years account for up to two-thirds of all people with dysphagia. Forty percent to 60% of nursing home residents develop dysphagia compared to 13% to 33% of noninstitutionalized older adults. Symptomatic dysphagia has been found to occur in 16% of individuals over 87 years old.

Gender: Dysphagia occurs equally in males and females.

Ethnicity: Not significant.

Contributing Factors: Factors that contribute to dysphagia include changes with aging, including decreased body fluid and facial muscle weakness, which decreases masticatory strength. Medications with anticholinergic side effects (e.g., antidepressants, opiates, sedatives, antipsychotics, antispasmodics, antihistamines, and some antihypertensives) may produce slowing or disruptions of the oral phase of swallowing and affect salivation. Other drugs can increase the likelihood of reflux (e.g., calcium channel blockers,

β-adrenergic agents, aspirin, theophylline, nitrates, vitamin C, and NSAIDs), particularly if the patient has inadequate fluid intake.

Signs and Symptoms: Symptoms typically associated with oropharyngeal dysphagia include difficulty initiating swallowing, food sticking in the throat, nasal regurgitation, and coughing during swallowing. The onset of symptoms may assist in the differential diagnosis because sudden onset may suggest a cerebrovascular event, whereas insidious onset is more characteristic of a myopathy, multiple sclerosis, or amyotrophic lateral sclerosis. Voice changes (wet vocal quality) may be associated with swallowing incompetence. Patients with esophageal dysphagia most commonly present with a complaint that food is stuck in the chest (sternum or suprasternal area) and may report choking or a sensation of pressure. Reflux symptoms may be present. If total obstruction occurs, salivation increases, and vomiting may result. Weight loss and aspiration pneumonia may occur. Weight gain may be a sign of dysphagia if the patient consumes significant amounts of processed foods, such as milkshakes, that have a higher calorie count.

Careful history helps to distinguish the type of dysphagia. It is important for the clinician to distinguish between difficulty swallowing solids, liquids, or both. In general, patients with obstructions (e.g., strictures, webs) complain of solid rather than liquid dysphagia, which may progress to both solid and liquid dysphagia. The onset of signs and symptoms is important because rapid progression may indicate infection, irritation, or a food impaction, whereas a more insidious onset suggests dysmotility. Physical examination consists of checking the oral cavity in conjunction with a neuromuscular examination. The mouth should be assessed for signs of irritation, ill-fitting dentures, and any pharyngeal masses. The head and neck should be examined, checking for lymph node or thyroid enlargement. Dentures may block palatal mechanoreceptors, which can lead to poor bolus control, oral transit delays, and longer feeding times. Review all medications and address any that may decrease salivation.

Neurological evaluation must be comprehensive and include a mental status evaluation, cranial nerve examination, and assessment for muscle weakness. A bedside swallow assessment to determine aspiration risk is often inadequate and misleading, so additional diagnostics may be indicated if increased suspicion of dysphagia persists.

Diagnostic Tests: Diagnostic tests are based on the medical history. Videofluoroscopy (modified barium swallow) is important to assess aspiration risk and guide therapy by a speech language pathologist and a radiologist. Other tests that may be indicated based on the history and physical include testing the stool for occult blood and a CBC with indices if associated esophagitis is suspected. Ambulatory 24-hour pH testing of intraesophageal pH and manometry has been found to be useful when evaluating for GERD. If malnutrition is suspected, a serum albumin level may be appropriate with the understanding that inflammatory illnesses common in older adults may result in a low albumin unrelated to actual protein calorie malnutrition. Laboratory screening may be helpful to exclude metabolic, systematic, and myogenic diseases. Upper endoscopy is performed for evaluating structural causes; it also allows for tissue samples to determine etiology and, if needed, therapeutic intervention (e.g., dilatation of esophageal ring). MRI or CT scan is used if a cerebrovascular event is suspected. Fiberoptic endoscopic evaluation of swallowing (FEES) is an option when the patient cannot be transported to the radiology department for the modified barium swallow and is done by a speech language pathologist and an otolaryngologist. FEES is diagnostic and therapeutic, allowing for biofeedback, as patients can be instructed in swallowing techniques and visually observe their swallowing function on a video monitor.

Differential Diagnosis: It is important to differentiate whether the patient has a feeding problem (inability to present food to the mouth) or a swallowing problem (inability to get food from the mouth to the stomach) or both. Cognitive impairments are frequently associated with feeding problems. Evaluation for specific motor, neurological, or obstructive causes should be performed. For the patient complaining of burning associated with swallowing, reflux esophagitis, esophageal infection, and pill esophagitis need to be explored.

Treatment: Treatment targets the specific cause, compensates for mechanical problems, and eliminates or minimizes aspiration risk. Therapeutic strategies include diet modification and swallowing posture or techniques. Management of dysphagia after a stroke is directed toward increasing the sensory awareness of food and improving bolus movement by the chin-tuck and double-swallow technique. A percutaneous endoscopic gastrostomy or nasogastric tube may be used to feed stroke patients at high risk for aspiration, but there is no guarantee it will prevent aspiration. A nutritionist works to ensure appropriate consistency and nutritional content. Stable patients can be treated as outpatients. Hospitalization may be required when dysphagia is associated with total or near-total obstruction of the esophageal lumen. Esophageal dilation (pneumatic or bougie), surgical intervention for an esophageal stent, or laser therapy for late cancer also may be part of the treatment plan, depending on the etiology of the dysphagia. Patients with esophageal spasms can be given calcium channel blockers, and those with associated esophagitis may receive antacids or H_2 blockers, or, in cases of severe esophagitis, a proton pump inhibitor. Referral to a speech language pathologist for specific exercises, compensatory strategies, and recommendations for dysphagia can help to regain control over swallowing. A dental referral should be obtained to assess and treat problems with dentition that may be contributing to the dysphagia.

Follow-Up: Patient monitoring depends on the specific etiology of the dysphagia.

Sequelae: Complications from dysphagia depend on its cause. The most common complications are malnutrition, aspiration, pneumonia, and death. Of stroke patients with dysphagia, 45% to 68% die within 6 months, largely due to nutritional and pulmonary complications related to dysphagia.

Prevention/Prophylaxis: Patients with dysphagia should remain upright with the head midline and slightly flexed when eating or drinking. Extremely hot or cold foods may worsen symptoms of dysphagia. Aspiration precautions include modifying food and fluid consistencies, giving verbal and physical prompts, and allowing additional time for feeding to encourage double swallows and small amounts taken per mouthful.

Referral: Initial consultation with speech pathology for evaluation can be essential in deciding on further assessment. Gastroenterology should be consulted if invasive diagnostic tests are indicated.

Education: Patients and caregivers should be encouraged to provide food and fluids at appropriate consistency. All foods, especially meat products, should be chewed thoroughly. Patients also should be informed that it is important to swallow all medications with plenty of fluid while in an upright position.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Patients with cough and their caregivers should be questioned regarding swallowing problems and if positive further evaluation for dysphagia is indicated.	C	Amin & Belafsky, 2010
Patients with dysphagia should undergo clinical bedside assessment, endoscopy, or fluoroscopy.	C	Amin & Belafsky, 2010
First-line therapy for dysphagia is swallowing therapy, including simply tucking the chin.	C	Amin & Belafsky, 2010 Welch et al., 1993

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FALLS

Description: According to the World Health Organization (WHO), a fall is an event that results in a person coming to rest inadvertently on the ground, floor, or other lower level. Falls are commonly categorized by health-care providers as witnessed or unwitnessed, assisted or unassisted, and by location (community, hospital, or long-term care). In the *International Statistical Classification of Diseases and Related Health Problems (ICD-10)*, falls are categorized as slipping, tripping, and stumbling. Falls are the leading cause of both fatal and nonfatal injuries in older adults. Of serious injuries, fractures and traumatic brain injury are the most common, with falls as the cause for 95% of hip fractures. The total cost for fall injuries in 2013 was 34 billion dollars.

Etiology: The majority of falls are caused by an interaction of several intrinsic and extrinsic risk factors. Intrinsic factors include age, previous falls, weakness, gait/balance problems, poor vision, postural hypotension, chronic medical conditions (e.g., arthritis, dementia, arrhythmia, neuropathies), an occult medical condition (e.g., pneumonia or UTI), and fear of falling. Extrinsic factors include conditions in the environment such as lack of handrails, grab bars, poor lighting, obstacles, slippery or uneven surfaces, psychoactive medications, or being on more than four medications. Additional risk factors include inactivity, improper use of assistive devices, and risky behavior (e.g., use of a ladder).

Occurrence: Approximately 25% of persons 65 years and older and 50% of persons 80 years and older will fall each year, although the true incidence of falling is likely to be higher due to the fact that many falls are not reported. Persons who reside in long-term care are twice as likely to fall as persons who live in the community.

Age: Normal physiological changes with aging can affect vestibular function, vision, and postural reflexes, which increase the risk of falling. Persons who are older than 85 years are four times more likely to fall as persons age 65 to 74 years.

Gender: Males and females are equally likely to fall; however, women are more likely to report falls and fall-related injuries than men. Women have an increased risk of falling due to decreased muscle mass and osteoporosis.

Ethnicity: According to data from the 2014 Behavioral Risk Factor Surveillance System survey, Caucasians and American Indians/Alaskan Natives were more likely to fall compared with African Americans and Asian/Pacific Islanders. American Indians/Alaskan Natives were more likely to report a fall injury compared with all other racial/ethnic groups.

Contributing Factors: The risk of a fall increases as the number of fall risk factors increases, and risk factors increase with age.

Signs and Symptoms: Every older adult patient or their families/caregivers should be questioned if there has been a fall in the past year. Half of patients or families may fail to mention a fall unless there was injury. In addition to asking

about falling, it is important to assess tripping, slipping, or stumbling but not falling, and fear of falling, as both increase risk of falling. For patients who report a fall, it is important to obtain information about the circumstances of the fall, including location, what the patient was doing, how the fall occurred, presence of pain after the fall, and symptoms at the time of the fall (e.g., chest pain, shortness of breath, dizziness/light-headedness, vertigo, diaphoresis, numbness and weakness of extremities, loss of consciousness).

Diagnostic Tests: The details about the fall and symptoms associated with a fall will determine the need for diagnostic testing. Intracerebral injuries need to be considered in any patient on anticoagulants or antiplatelet drugs. Evidence of trauma to the head and loss of consciousness are predictive of intracranial injuries and should guide the decision to obtain diagnostic imaging. Because loss of consciousness is difficult to assess in unwitnessed falls or in persons with cognitive impairment, it is important to determine baseline mental status. It can be challenging to determine if loss of consciousness was the cause or result of the fall.

The decision to obtain radiographic imaging on elders who fall should be based on understanding that low-force mechanisms such as falling from a sitting or standing position has been shown to cause injury, primarily fractures in frail older adults. Hip fracture, one of the most common injuries, should be considered in a person with any of the following: pain in the groin region, inability to bear weight, external rotation and abduction of the affected hip, and the appearance of a shortened leg. A plain radiograph can detect fractures in the majority of persons, however, for patients with negative fracture on plain radiograph, ongoing pain, and inability to function at previous level, an occult fracture should be suspected. An MRI can identify occult fractures, soft tissue damage, and joint disease. For any injury, nondisplaced fractures may not be evident on initial radiographic imaging, so consideration of repeat imaging may be necessary.

For patients who have reported a fall, slipping, tripping, or stumbling in the past year, or who report fear of falling, gait, strength, and balance should be assessed. The timed Get Up and Go Test, 30-Second Chair Stand, or 4-Stage Balance Test can be done in the office in 30 seconds or less. There are no standard laboratory recommendations for patients who are at increased risk of falling or who have fallen, although a suggested work-up would include a basic metabolic panel, CBC, vitamins D and B_{12} levels, thyroid function, and EKG. Diagnostic testing should be individualized based on the history and physical examination findings and comorbidities.

For patients with identified gait and balance issues, it is important to assess multifactorial risks, including assessment for orthostatic hypotension or postural dizziness; cognitive evaluation, such as the Mini-Mental State Examination (MMSE); visual acuity assessment; and assessment of feet, footwear, and mobility aids.

Differential Diagnosis: Determining the cause of a fall can be complex because many falls are multifactorial, resulting from intrinsic and extrinsic factors. A differential diagnosis can be narrowed by assessment of these risk factors, along with subjective and objective findings. Reports of light-headedness, near-fainting, or legs giving out is consistent with cerebral ischemia and would suggest orthostasis, arrhythmias, or other cardiovascular conditions. Difficulty standing from a chair or feeling that legs are not supportive suggests deconditioning or neuromuscular disease. Disequilibrium or feeling the legs are not coordinated could suggest vestibulospinal tract or cerebellar lesions.

The physical examination can help narrow a differential diagnosis or identify additional factors related to a fall. It is important to initially assess gait, strength, and balance; however, the differential diagnosis can be narrowed by including assessment of orthostatic vital signs, muscle tone to determine spasticity or rigidity, sensation, proprioception, vibration to determine neuropathy or a somatosensory problem, cognitive status, and visual problems. The pattern of injury can help sort out the event leading to a fall. For example, a wrist fracture caused by a fall on an outstretched hand suggests that consciousness was preserved while falling, or bilateral wounds on knees could suggest drop attacks.

Treatment: Regardless of having a history of a fall, interventions should address intrinsic and extrinsic risk factors. All older adults with gait, strength, or balance problems should be encouraged to engage in routine exercise or be referred to a community falls prevention program.

Follow-Up: For patients who have fallen and those at risk of falling, ongoing management and monitoring of medical conditions associated with falling is important. Follow-up on exercise or the use of interventions to improve gait, strength, and balance serves as reminders or reinforcement of the importance of fall prevention.

Sequelae: Persons who fall are two to three times more likely to fall again. Approximately 5% to 10% of older adults who fall endure serious injury, including fractures and traumatic brain injury. Injuries result in decreased functional status, subsequent loss of independence, and possible nursing home placement. Falls can also have psychological and social consequences, such as fear of falling or post-fall anxiety syndrome, which can result in social isolation and loss of self-confidence. Approximately 25% to 50% of elders will not ambulate at their previous level after a fall.

Prevention/Prophylaxis: Physiological changes that increase the risk of falling in older adults are inevitable; however, many falls are preventable. Factors associated with the risk of falls in older people need to be addressed and implemented, including optimizing medical conditions, tapering psychoactive medication when appropriate, annual review of all medications, and managing environmental issues (e.g., inappropriate lighting, lack of grab bars, ill-fitting footwear, inappropriate assistive devices). Vitamin D supplementation is widely recommended for all older adults regardless of their history of falls or risk factors, however, the recommended dose varies. Exercise in general has been shown to reduce the prevalence of falls, although evidence on specific exercise at home is lacking. Muscle mass and leg strength can be increased with resistance strength training for older adults. Strength and balance exercise programs are available in many communities. The CDC and National Council on Aging provide resources to help identify and evaluate community falls prevention programs.

Referral: A home safety assessment is essential for patients with identified gait and balance problems. The following

referrals are based on the risk factors for falling, the cause of the fall, and the history and physical examination: physical therapy, occupational therapy, podiatrists or subspecialty provider such as cardiologist or neurologist.

Education: Patients and their families need to be informed that falls are preventable. They need to be encouraged to report all falls, near falls, or fear of falling to their health-care providers.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Group and home-based exercise program, and home safety interventions reduce rate of falls and risk of falling.	A	Gillespie et al., 2012
Exercise may help reduce fear of falling in community-dwelling adults.	A	Kendrick et al. 2014
Exercise as a single intervention can prevent falls in community-dwelling older adults.	A	Sherrington et al. 2016 El-Khoury et al., 2013
Vitamin D supplementation does not reduce falls.	A	Gillespie et al., 2012 Uusi-Rasi et al., 2015
Multifactorial assessment and intervention program reduce rate of falls but not risk of falling.	A	Gillespie et al., 2012

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FATIGUE

Description: Fatigue is a subjective state often described as a feeling of tiredness, weariness, lack of energy, or exhaustion that is unrelieved or only partially relieved by rest. It often results in an inability to initiate normal activity; a reduced capacity to maintain activity; and difficulty with concentration, memory, and emotional stability (Gambert, 2013). Occasional fatigue is common, but pathological fatigue may affect quality of life and impair independence. Chronic fatigue syndrome occurs when fatigue lasts longer than 6 months and is not relieved with rest. Fatigue could also be a symptom of another illness and any older person with this complaint should obtain a medical evaluation and work-up.

Etiology: Physiological fatigue occurs normally with inadequate rest, excess exertion, or insufficient diet. Fatigue that interrupts the individual's activities of daily living (ADLs) may have a physical or psychological cause and be either acute or chronic. Fatigue in older adults may be an early indicator of the aging process, as well as debility or another disorder (Tang & Chen, 2014; Hermans et al., 2014; Manty et al., 2014). See Table 5-4 for common causes of fatigue.

Occurrence: Fatigue occurs in about 25% of the U.S. population (Gambert, 2013; Gluckman, 2014). Under-reporting and under-recognition make the actual prevalence unknown.

Age: Fatigue is common among the elderly, with reports of up to 65% of older patients complaining of fatigue at some point (Tang & Chen, 2014; Hermans et al., 2014; Manty et al., 2014). Among elders living in the community and assisted living facilities, more than 50% report mild fatigue and 7% report severe fatigue on a chronic basis.

Gender: Fatigue is more common in women than in men.

Ethnicity: Not significant.

Contributing Factors: Poor dietary habits, overexertion, alcohol abuse, smoking, stress, chronic illness, drug interactions, misuse of drugs, and sleep apnea may contribute to fatigue symptoms. In the older adult individual it is compounded by a decrease in muscle strength, loss of muscle neurons, muscle atrophy, a decrease in hormone levels, and lack of exercise.

Signs and Symptoms: Obtain a complete medical history and perform a thorough physical examination when an individual complains of fatigue because it may indicate various psychological or physiological illnesses. Conduct a complete symptom assessment, including the onset; duration; severity; and precipitating, aggravating, and relieving factors. Identify other indicators or associated symptoms of fatigue, which may include decreased energy expenditure, decreased endurance, sleep disturbance, attention deficits, somatic complaints (aching body, tired eyes), dyspnea, and weakness. Carefully review the adequacy of the diet, all medications (evaluating for potential medication side effects), activity level (including degree of independence of ADLs), and potential causes or contributing factors. Identify the impact fatigue is having on the person's ADLs and quality of life and current stressors.

TABLE 5-4 Common Causes of Fatigue

Infectious causes	Viral infections • Hepatitis A, B, C • Epstein-Barr virus (mononucleosis) • HIV Bacterial infections • TB • Lyme disease • Occult endocarditis • Osteomyelitis Immunological/rheumatic • RA • Systemic lupus erythematous Celiac disease Fibromyalgia
Medication related	NSAIDs Tetracycline Antipsychotics Antidepressants Sleep medications Pain medications Anxiolytics Statin agents Illicit drugs Alcohol
Chronic fatigue syndrome	No known cause but theories to the cause include viral infections, immune system problems, and hormonal imbalances
Physiological	Inadequate sleep Insufficient rest Overactivity Poor physical conditioning Stress Changes in diet, including decreased protein intake Relocation of one's home or change in job/retirement Frailty Weight loss Sarcopenia Parkinson's disease Vitamin deficiencies including vitamins B_{12} and D
Endocrine related	Hypothyroidism including subclinical hypothyroidism Hyperthyroidism Perimenopausal/postmenopausal Hypogonadism (low testosterone levels)
Neoplastic-related disorders	Any form of cancer Chemotherapy treatments Radiation treatments Anemia related to cancer, as well as its treatment
Toxin-related disorders	Exposure as well as toxicity to: • Carbon monoxide, possibly caused by wood-burning stove, kerosene lamps, automobile exhaust, or coal-burning plants • Lead • Arsenic
Cardiovascular/pulmonary diseases	Congestive heart failure COPD
Psychogenic	Depression Anxiety Somatoform disorders Substance abuse (prescribed and nonprescription drugs, alcohol)

Source: Gambert (2013); Combs et al. (2013); Tang & Chen (2014); Hermans et al. (2014); Manty et al. (2014); Patel, 2015.

The quality and quantity of sleep should always be evaluated. Distinguish between generalized fatigue and actual weakness by testing for muscle strength and presence of localized tenderness. A thorough physical examination will include a mental status examination to screen for dementia and rule out depression.

Diagnostic Tests: The benefit of testing is limited when fatigue is the only symptom. Diagnostic tests on all patients with persistent unresolved fatigue should include CMP, CBC with differential, erythrocyte sedimentation rate (ESR), and/or C-reactive protein, because these are low cost and offer significant screening capacity. Other tests that may be appropriate include thyroid function, urinalysis, and pulmonary function tests. If symptoms and signs indicate cardiac decompensation, a B-type natriuretic peptide (BNP) may indicate degree of heart failure and an EKG may reveal cardiac arrhythmias, enlargement of the heart, myocardial infarction, or abnormalities in the conduction system. Late-life hypothyroidism or masked hyperthyroidism may present with a chief complaint of fatigue.

Differential Diagnosis: Fatigue can be related to many psychological and physiological etiologies. Psychiatric disorders, including depression and generalized anxiety disorder, account for 70% of cases of fatigue. An underlying cause is determined in only 10% of all cases. Fatigue that cannot be relieved by rest or sleep is often a sign of disease.

Treatment: Treatment depends on the etiology identified in the comprehensive work-up. Symptom management includes regular exercise, attention-restoring activities, psychosocial techniques, energy conservation measures, good sleep hygiene, improving diet, and possibly adding nutritional supplements. Pharmacological management is directed at the cause of the fatigue. Psychostimulants may be considered for opioid-related somnolence, cognitive impairment, and depression.

Follow-Up: Follow-up depends on the findings. Monitor the patient periodically as indicated by diagnosis or symptoms, symptom persistence, and disability associated with the symptom. The symptom may be a subclinical one.

Sequelae: The potential for complications relates to the cause of fatigue and the impact the symptom has on the person's function.

Prevention/Prophylaxis: Optimal health maintenance, including maintaining a healthy diet, regular exercise, and good sleep hygiene, may prevent or enable early recognition of signs and symptoms of systemic or psychological illness.

Referral: Referral to a specialist may be indicated based on the results of the work-up.

Education: If the fatigue has a physiological cause, teaching should be related to the findings; psychological counseling, changes in the environment, behavior modification, and stress reduction may be needed. The goal of fatigue management is to provide the patient with self-help tools to eliminate or alleviate fatigue.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
A thorough history and physical examination are crucial in determining the differential diagnosis and identifying the causative agent.	C	Gambert, 2013
Graded exercise has been shown to improve symptoms and physical function in patients with fatigue.	A	Gluckman, 2014 Gambert, 2013
Cognitive-behavioral therapy is an effective treatment for adult outpatients with chronic fatigue syndrome.	A	Gluckman, 2014

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HEADACHE

Description: Headache refers to a symptom that occurs in the head, neck, or face. Headaches decline with aging and any new onset of headache should be considered serious until proved otherwise.

Etiology: *The International Classification of Headache Disorders (ICHD), 3rd edition,* describes two categories of headache: primary and secondary (International Headache Society [IHS], 2013). Primary headaches include those in which there is an intrinsic dysfunction of the nervous system, often genetic in origin, which predisposes the older adult to increased vulnerability to headache attacks. Primary headaches include migraine, tension-type headache (TTH), cluster headache, and other trigeminal autonomic cephalgias. In contrast, secondary headaches are those in which the headache is secondary to an organic or physiological process, intracranially or extracranially. Secondary headaches include headaches attributed to trauma or injury to the head and/or neck; cranial or cervical disorder; nonvascular intracranial disorder; substance abuse or its withdrawal; infection; disorder of homeostasis; disorder of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cervical structures; and psychiatric disorder.

Occurrence: While the prevalence of headache decreases in the older adult, headaches remain a problem with approximately 17% of older adults reporting "frequent headaches" (more than two headaches per month) (Bravo, 2015; Dees, Coleman-Jackson, & Hershey, 2013). The majority of headaches (approximately 66%) are primary, TTH, and migraine. TTH is the most common type of headache in individuals over 65 years old and in the general population, with a lifetime prevalence of 78% (Crystal & Grosberg, 2009). Of greater significance is that secondary headaches are more common in older adults over 65 years old, 15% of which are new-onset headaches, which is significantly increased from an estimated 1.6% for adults under 65 years old (Bravo, 2015).

Age: The prevalence of primary headaches decreases with age; however, the prevalence of migraine in patients over 65 years old is still 10%. New-onset migraine in patients over 60 years old is rare and is a red flag indicating evaluation for a secondary cause (Bravo, 2015).

Gender: TTH is more frequent in women in all age groups, regardless of race or education (Crystal & Grosberg, 2009). Generally, women are affected more often than men; 76% of women and 57% of men report one headache per month. Migraine headaches are more common in women than in men, tending to begin in early adulthood. Cluster headaches are more common in men than in women, with a mean age of onset at age 30 years. Giant cell arteritis is more commonly seen in women.

Ethnicity: Giant cell arteritis is more common in Caucasian populations. Ethnicity does not appear to be related to other headache types.

Contributing Factors: Depending on the type of headache, multiple factors can contribute. Poor posture, cervical osteoarthritis, ill-fitting dentures, tooth pain, and jaw disorders

can contribute to tension headaches. Depression, anxiety, sleep disorders, and general pain syndromes can also contribute to most headache types. Medication side effects, particularly with estrogen, calcium channel blockers, nitrates, anti-Parkinson's drugs, indomethacin, and theophylline, can contribute to headaches. Alcohol can trigger or worsen headaches. Migraines can be triggered by dietary factors such as aged or smoked cheese, chocolate, caffeine, and monosodium glutamate, as well as changes in sleep pattern, weather changes, and smoking. Genetics may play a role in migraine, cluster headache, and giant cell arteritis.

Signs and Symptoms: Headache types, presentation, and treatment options change as patients age (Bravo, 2015). Tension headaches are the most common and are due to mostly extracranial causes, such as muscle tension. Migraine in older adults may appear with sensory or motor phenomenon and may be confused with transient ischemic attack or stroke. The reported symptoms change with age bilaterally or globally with decreasing associated symptoms such as photophobia, phonophobia, nausea, and vomiting. The older adult may complain of increased neck pain, bilateral rhinorrhea, and lacrimation (Bravo, 2015). On a positive note, the older adult frequently has a better response to medication as compared with younger adults. A word of caution for patients who report new-onset auras without a headache; that is, visual, sensory, and/or speech symptoms that may mimic those of migraine headaches. These patients merit immediate referral for evaluation of secondary causes such as transient ischemic attack (TIA), seizure, and intracranial hemorrhage (Bravo, 2015). Two rare primary headaches occurring in older adults are hypnic (developing only during sleep, lasting several hours) and primary cough or Valsalva maneuver headache (precipitated by cough or other Valsalva maneuver, lasting up to 2 hours).

The evaluation of an acute headache in adults is based largely on the history; the physical examination is often normal. Key responses in the history of present illness include the classic, "The worst headache in my life," which may suggest subarachnoid hemorrhage (SAH), a medical emergency. Ask if the headache is "typical;" that is, migraine or TTH. If the typical headache did not improve with usual treatment, other diagnoses should be considered. The onset of the headache is very informative: sudden onset, unprovoked may be SAH or cerebrovascular accident (CVA); sudden onset provoked by agitation, orgasm, cough, or sneeze is usually benign and transient. Lastly, a subacute headache progressing over weeks may indicate an intracranial lesion, subdural hematoma, or hydrocephalus. For the older adult with their first headache, always consider giant cell arteritis (Neblett, Hamilton, Jacobson, & Helseth, 2016). Location, duration, frequency, severity, character, triggering or aggravating factors, and alleviating factors must be considered. Specifically address exacerbating factors such as posture, exertion or Valsalva, and foods (caffeine, monosodium glutamate).

A focused review of systems is important. For example, fever may suggest meningitis, brain abscess, viral syndrome, or dehydration. While vomiting is less common in older adults with migraine, if present, a CVA or SAH should be considered. Visual complaints suggest a possible CVA, acute angle glaucoma, or giant cell arteritis. Other significant findings would be seizures, confusion, or change in mental status, dizziness, weakness, neck pain, recent head trauma, recent travel (Lyme disease, Zika virus, dengue fever), facial pain or tenderness, and eye pain. Ask if other family members or pets are sick. Past medical history should address the patient's immune status, recent change in medication, history of cancer, and hypertension. A family history of migraine or brain tumor may be significant. Alcohol consumption and smoking should be examined, because they frequently intensify headaches. For recurrent/frequent headaches, having the patient keep a headache diary is helpful in identifying patterns and the impact the symptom has on function. Input from family and caregivers on the impact of the headaches on daily life may be helpful.

As noted, the physical examination is often normal, but several components of the examination, to include a focused neurological examination, deserve attention. Observe the patient's general appearance, including facies, and evaluation of mental status. Any sudden change in mental status or mood should be evaluated swiftly. Assess vital signs for extremes in blood pressure, which suggest a neurological event, and temperature elevation, which suggests infection. Test the cranial nerves. Perform a funduscopic examination to assess for papilledema, hemorrhages, exudates, and venous pulsations. Check for pupillary size and look at the cornea for haziness. Check extraocular movements and position of the eyelids. Evaluate gross and fine motor and sensory functions, including gait, balance, and tactile sense. Test deep tendon reflexes for presence and symmetry. Measure muscle strength for grading and equality.

Conduct an examination of the head and neck for lymphadenopathy, thyroid enlargement, carotid bruits, trigger points, meningeal irritation, or limitation in normal range of motion. Palpate the head and neck for tenderness, which may suggest a TTH. Tenderness over the frontal and maxillary sinuses suggests sinusitis. Palpate over the temporal artery; tenderness suggests giant cell arteritis. Palpate the temporomandibular joint for alignment, mobility, and clicking. Examine the ears, nose, throat, and teeth for contributing problems. Evaluate the patient for postural alignment problems, muscle spasms, or trigger points in the back or shoulders.

Diagnostic Tests: Individualize diagnostic tests according to the suspected cause of the symptom. Neuroimaging sometimes is indicated for emergency evaluation of suspected vascular, neoplastic, or infectious disease. If red flags are present, in addition to a full neurological evaluation, neuroimaging should be considered. (Bravo, 2015). Generally, MRI is preferred to CT scan. A CT scan of the brain may be indicated if the patient is immunocompromised, has a fever, or has a history of cancer (Neblett et al., 2016). MRI is considered if the headache is of sudden onset, severe, worsening, unresponsive to treatment, and associated with neurological or constitutional symptoms. Laboratory tests generally are not helpful, unless anemia, infection, or electrolyte imbalance is suspected. ESR or C-reactive protein may be indicated if an inflammatory condition, such as giant cell arteritis, is under consideration. An elevated ESR warrants referral of the patient for temporal artery biopsy to confirm diagnosis. A lumbar puncture may be indicated after a negative CT scan without contrast if the patient has a "thunder-clap headache," suggesting an SAH, fever, or neck stiffness

(Neblett et al., 2016). Sinus or cervical spine x-rays may be indicated.

Differential Diagnosis: There are numerous differential diagnoses to be considered based on the history and examination. Differentiating between urgent headaches in older adults is the first step. Differential diagnoses of primary headaches include TTH, which is the most common, and may be triggered by lack of sleep and emotional or physical stress. The patient may complain of constant bilateral tightening pressure, which does not worsen with ADLs. The neurological examination will be normal. Migraine headaches, as noted previously, with or without aura, are rarely new-onset. Medication-induced headache must be considered, especially with medication overuse or misuse of barbiturate-analgesic-caffeine combinations, codeine, opioids, caffeine, acetaminophen, aspirin, and NSAIDs. These patients may present with daily or near-daily headaches. Other potential diagnoses include sinusitis and cervical spine disease.

The cluster headache is characterized by severe unilateral orbital, periorbital, or temporal pain. The attacks are relatively short (15 to 180 minutes) but can recur up to eight times in one day. The pain is worse in the supine position, which makes the patient very restless, and the patient often wants to pace or rock back and forth during an attack. Autonomic symptoms are also associated with the pain and present as ipsilateral ptosis, miosis, lacrimation, rhinorrhea, nasal congestion, and facial swelling.

Urgent diagnoses include SAH, giant cell arteritis, stroke/CVA, brain tumor, infection, and acute angle glaucoma. The most common acute cerebral bleed is a subarachnoid hemorrhage, which has its highest incidence in women more than 70 years old, with a history including smoking, excessive alcohol ingestion, and hypertension. This individual generally presents with a "thunderclap" or the "worst headache of my life." The pain may be associated with other symptoms, such as confusion, vomiting, and seizures. This individual should be transported to the emergency department for a CT scan and possibly surgery. More commonly, an older adult patient may present with a chronic subdural hematoma, which is defined as one that is more than 20 days old. The peak incidence is in ages 60 to 80 years, and up to 80% of the cases are in men. Most often a chronic subdural hematoma is associated with a fall or other type of minor trauma. Typically, they present with a headache and a change in mental status. The headache is described as mild and generalized all over the head. Although the individual does not appear to be in great distress, he or she should be transferred immediately, preferably to a location where neurosurgical consultation is possible.

Giant cell arteritis occurs predominantly in females over 50 years old who may have a history of polymyalgia rheumatica. She may present with monocular vision loss, flu-like symptoms, and jaw claudication. On examination, unilateral blindness may be present with tenderness in the temporal area. Optic nerve edema may be seen on funduscopic examination. Headache may also warn of an impending stroke, but only in about 25% of all ischemic strokes. The incidence in hemorrhagic strokes is 40% to 60%. Infectious diseases, such as encephalitis and meningitis, may also present with a headache. Generally, both include other symptoms such as fever, altered mental status, and nuchal rigidity. The mortality rate for meningitis in patients older than 65 years is as high as 50% to 70%. When infectious disease is suspected, the patient should be immediately transferred.

Acute angle-closure glaucoma is an emergency. The patient presents with acute unilateral eye and/or forehead pain, blurred vision, halos around lights, injected sclera, and nausea/vomiting. On examination, the pupil is mid-position and the cornea appears hazy. This presentation is considered a serious emergency, and the patient should be seen immediately by an ophthalmologist because the main treatment is decreasing intraocular pressure.

To reiterate, red flags for possible secondary headaches includes systematic symptoms (fever, chills, weight loss), focal or mental status changes, sudden onset or thunderclap presentation, onset over 50 years old, and a change in pattern to recurring headaches (Bravo, 2015).

Treatment: Treatment of headache depends on the presumed etiology. Regardless, acute and preventative medications must be carefully scrutinized, considering the presence of comorbidities and the pathophysiological aspects of aging. The dearth of research studies including older adults limits best evidence. Acetaminophen, NSAIDs, and triptans, as well as combinations, have been established as effective for acute migraine treatment based on available evidence (Marmura, Silberstein, & Schwedt, 2015). Vasoactive substances (triptans or dihydroergotamine) should not be prescribed to patients with cardiovascular disease (CVD), including stroke. If the patient is at low cardiovascular risk, triptans should be considered for acute episodes. Side effects must be recognized to include cognitive and autonomic signs/symptoms (Bravo, 2015).

Management of TTH combines pharmacological and non-pharmacological measures. Analgesics, including NSAIDs and cyclooxygenase-2 inhibitors, are effective. NSAIDs must be used with caution because of the risk of GI ulceration, and renal and hepatic impairment. Concomitant use of aspirin or antiplatelet agents increases the risk of bleeding. Treatment should be limited to 2 days per week to avoid medication overuse headaches. Prophylactic therapy is advised for patients experiencing two to three headache days per week. Tricyclic antidepressants (amitriptyline and nortriptyline) may be effective, but have the potential for anticholinergic side effects. Behavioral intervention such as relaxation, cognitive behavioral therapy, biofeedback, and other lifestyle modifications may be helpful as adjunctive therapy.

For recurrent headaches, most notably migraine, prophylactic therapy may be indicated. In addition to medication, patients should be advised to avoid triggers and to consider behavioral modification. Regular meals, sleep hygiene, hydration, and regular physical activity should be encouraged. Biofeedback and other relaxation techniques, as well as cognitive behavioral therapy, has been helpful for some adults. For older adults without predominant depression or aura, the most effective drugs are the same as those for younger adults. Medication is taken daily for a trial period (usually 1 to 2 months) to evaluate the effect on headache frequency and strength.

All categories of drugs should be started at low doses and incrementally increased to therapeutic dosing. Although beta blockers (propranolol [Inderal LA], 80 mg orally daily; atenolol [Tenormin], 50 to 100 mg orally daily; or nadolol

[Corgard], 40 mg orally daily) have been used prophylactically for migraine and headaches, they are contraindicated in patients with a history of bronchospastic disease, asthma, diabetes, or congestive heart failure. Non-dihydropyridine calcium channel blockers (verapamil, 240 mg orally daily in divided doses, or nifedipine, 30 to 180 mg orally daily) also have been used prophylactically to prevent cluster headaches and are highly effective. Contraindications include congestive heart failure, heart block, hypotension, sick sinus syndrome, and atrial fibrillation. There is moderate evidence that antidepressants are effective for prophylaxis. Low doses of tricyclic antidepressants (amitriptyline [Elavil], 25 to 50 mg orally daily, or desipramine [Norpramin], 50 mg orally daily) have been used for prevention, although these agents may be contraindicated because of adverse effects on the cardiovascular system or anticholinergic effects.

Anticonvulsants have been found to be among the most effective agents for prophylaxis of migraine headaches. Valproic acid (250 to 500 mg in daily divided doses), topiramate (25 to 200 mg in divided doses), and gabapentin (begin with 300 mg increasing to a maximum of 3,600 mg/day) have been used successfully, particularly for migraine management. Each anticonvulsant can increase fatigue, and baseline liver and kidney function studies should be obtained before initiation. Topiramate can cause kidney stones, so it is important that the patient increase fluids while taking the medication.

For abortive treatment of mild to moderate migraine, nonprescription analgesics (NSAIDs or aspirin) with antiemetics and hydration are the first line of treatment. For more severe symptoms, early treatment with a triptan is indicated. Adjunct measures include antiemetics, hydration, and possibly magnesium. Combining NSAIDs with triptans improves the efficacy (Neblett et al., 2016). Triptans are contraindicated in patients with CAD, peripheral vascular disease (PVD), or CVD. Likewise, they should not be used in patients with uncontrolled hypertension or severe hepatic impairment, limiting their use in older adults. Various forms of the triptans are available in oral, injectable, and nasal forms.

For cluster headache, subcutaneous sumatriptan 6 mg or sumatriptan nasal spray 20 mg are effective in improving headache response, as is zolmitriptan nasal spray 5 mg (Robbins, Starling, Pringsheim, Becker, & Schwedt, 2016). While less convenient, high-dose and high-flow oxygen is effective in the acute cluster headache, decreasing the intensity and duration. It is safe and not contraindicated in patients with hypertension or CVD. There is insufficient evidence that prednisone improves the headache response; however, corticosteroids may reduce remission within 24 to 48 hours. If not contraindicated, prednisone, 30 to 60 mg, can be administered orally daily for 1 week, then tapered off for another week. Intranasal lidocaine 4% topical solution, 1 mL in the nostril corresponding to the location of the headache, also has been effective in relieving migraine or cluster headaches. Ergotamine and dihydroergotamine (DHE 45) are effective in migraine treatment, but are contraindicated for older adults due to their effects on peripheral and central circulation. Botulinum toxin has been studied for the treatment of migraine and tension headaches; however, there is currently no evidence of efficacy.

For giant cell arteritis, management requires larger doses of prednisone in order to prevent visual sequelae, 40 to 60 mg and even 80 mg/day. The dose is maintained until remission is reached, which may take 2 to 4 weeks before tapering (Kennedy, 2012). Monitor blood sugar and other side effects of corticosteroids, such as changes in cognition and mood.

Follow-Up: Monitoring depends on etiology and treatment strategies. Once the diagnosis has been made and management strategies identified, patients should be monitored for effectiveness of treatment in person, via secure patient portals, or other preferred methods identified by the patient.

Sequelae: Sequelae depend on cause. Missed diagnosis of acute, life-threatening symptoms can prove fatal. Most common causes have recurrence or chronicity, which affects quality of life. Potential for side effects from medications and medication overuse must be considered.

Prevention/Prophylaxis: Preventive measures include avoidance of triggers, early intervention with medication as soon as symptoms present, and stress reduction techniques as appropriate. Use of alcohol, tobacco, and caffeine in specific types of headache should be monitored.

Referral: Refer patients to an emergency department for urgent conditions when headaches may be a result of hemorrhage, vasculitis, stroke, infectious disease, or acute angle-closure glaucoma. Any abnormal MRI or CT scan results should be referred to a neurologist. Symptoms warranting immediate referral for emergency evaluation include:

- Headache associated with any neurological changes
- Sudden change in mental status
- Headache associated with a fall or other unusual event
- New-onset migraine headache
- Patient complaint of the "worst headache" ever experienced

Education: Patient and family/caregiver education should be ongoing, specifically nonpharmacological management as well as pharmacological management. Evidence-based resources should be provided, as well as Web-based resources from organizations such as the American Headache Society. Provide a headache journal (templates available from the American Headache Society and other venues) for the patient with guidance on how to use. If medications are prescribed, ensure that the patient understands proper use and safety considerations.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Relaxation, biofeedback, and cognitive therapy lead to significant reductions in headache activity.	A	Andrasik & Grazzi, 2014
Acetaminophen, ergots, NSAIDs, triptans are established as effective for acute migraine management. Medications must be individualized based on older adult patient's risk factors.	A	Dees et al., 2013 Marmura et al., 2015
Sumatriptan subcutaneous and via nasal spray is effective in improving headache response in cluster headaches.	A	Robbins et al., 2016
Red flags for secondary headaches include fever, focal or mental status changes, sudden or thunderclap headache.	B	Bravo, 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

HEMATURIA

Description: Hematuria is the presence of red blood cells (RBCs) in the urine. It can be classified as either gross or microscopic. Gross hematuria is suspected when the urine appears either red or brown in color to the naked eye. Microscopic hematuria is identified by laboratory analysis. The American Urological Association (AUA) defines clinically significant microscopic hematuria as three or more RBCs per high-power field on an accurately collected urine specimen. All patients with hematuria require evaluation, as 5% of patients with microscopic hematuria are found to have a malignancy and 30% to 40% of patients with gross hematuria are found to have a malignancy.

Etiology: Hematuria may be renal or contamination from outside the urinary tract. Renal hematuria may result from glomerular or nonglomerular causes. The source of hematuria may be the upper collection system (e.g., renal, ureter) and/or lower collection system (e.g., bladder, prostrate, urethra). Microscopic hematuria is a common finding on routine urinalysis and etiologies range from life threatening to benign incidental causes. The pathophysiology depends on the anatomical site from which blood loss occurred. In older adult patients the most common causes are malignancy or benign prostatic hyperplasia.

Occurrence: The prevalence of asymptomatic microscopic hematuria ranges from 2% to 31% in the adult population. In patients older than 50 years, the prevalence is estimated at 13%.

Age: Incidence of hematuria increases with age. Younger patients are less likely to have an etiology identified.

Gender: There is a slightly higher incidence of hematuria in older women.

Ethnicity: Not significant.

Contributing Factors: Infection, anticoagulation therapy, renal calculi, trauma, anatomical defects such as rectocele, menstruation, atrophic vaginitis, renal disease, or recent urological procedures may all contribute to the presence of hematuria. Factors related to the development of a malignancy include age more than 35 years, male sex, current or past history of smoking, occupational exposures to chemicals or dyes (e.g., painters, printers, chemical plant workers), history of gross hematuria, history of chronic cystitis, history of pelvic irradiation, any exposures to cytotoxic agents such as cyclophosphamide or ifosfamide, plus any history of analgesic abuse.

Signs and Symptoms: A thorough history is important in the assessment of hematuria. Ask all patients about the presence of visible blood in their urine, history of vigorous exercise, recent prostate examination or procedure, recent trauma to the abdomen, recent catheterization, menstruation, renal disease, viral illness, and medications (analgesics, antibiotics, anticoagulants, NSAIDs). Gross hematuria would be recognizable by the red or brownish color of the urine, but there may not be any signs or symptoms related to microscopic hematuria. Symptomatic hematuria with associated flank pain, fever, chills, nausea or vomiting, abdominal pain, or renal colic may represent kidney disease or a kidney stone. A patient may report urinary frequency, lower abdominal pain or dysuria, which may indicate a UTI. Ask about symptoms of prostatic obstruction in males. A physical examination should focus on abdominal or flank pain, urogenital examination, prostate examination, testicular examination, and/or vaginal examination. Because the history and physical examination often fail to diagnose the etiology of hematuria, all patients need to be evaluated with diagnostic tests for glomerular disease.

Diagnostic Tests: A urinalysis with microscopy should be obtained to confirm hematuria. Urine dipstick evaluation may be misleading because it lacks the ability to distinguish RBCs from myoglobin or hemoglobin. Always confirm a

heme-positive dipstick with a microscopic urinalysis. Microscopic hematuria is defined as three or more RBCs per high-power field in a urine collection. Other analysis may include a CMP to identify metabolic abnormalities. An elevated creatinine level may be indicative of acute or chronic renal disease and should be referred to nephrology. If the patient is on anticoagulation therapy, a prothrombin time (PT), international normalized ratio (INR), and CBC should be obtained. Always evaluate hematuria in patients receiving antiplatelet or anticoagulant therapy, as these medications are not a satisfactory explanation as a cause of the hematuria. Note if the patient is taking any other medications that would affect platelet function or decrease clotting abilities. If a glomerular source is ruled out, as well as benign issues, radiological imaging may be indicated to further assess the hematuria. A multiphasic CT urography or IV pyelogram is indicated if there is suspicion for a kidney stone, renal mass, or malignancy. Cystoscopy is indicated in patients older than 35 years of age, patients younger than 35 years if no other reason for hematuria is present, or patients at any age with risk factors for cancer. Cystoscopy is also used to diagnose bladder or prostatic cancers. On initial evaluation for hematuria, urine cytology and urine markers for bladder cancer are not recommended.

Differential Diagnosis: Infection or supratherapeutic anticoagulation should be ruled out as reversible causes of hematuria. More serious causes such as glomerular disease, renal calculi, trauma, anatomical defects, or malignancy must be ruled out.

Treatment: Treatment is directed at the cause. Asymptomatic (isolated) hematuria generally does not require treatment. Appropriate antibiotic therapy for a UTI should resolve the hematuria. If the patient is on anticoagulation therapy, medication adjustment may resolve the problem. If a kidney stone is present, initial treatment may be pain management and oral hydration. The patient with urosepsis, acute kidney injury, anuria, unrelenting nausea, and vomiting should be referred immediately.

Follow-Up: A repeat urinalysis should be obtained 6 weeks after initial treatment for any infection, vigorous exercise, or trauma to make sure that the hematuria has cleared. Referral to a urologist is needed for gross and microscopic hematuria. For persistent hematuria with initial negative urological work-up, a repeat urinalysis should be done yearly and a full repeat evaluation every 3 to 5 years for ongoing hematuria. Referral to a nephrologist may be needed if hematuria persists with renal impairment. Once any benign causes have been treated (e.g., anatomical abnormalities, viral illness, or vigorous exercise) the evaluation should be repeated after the cause has been excluded. Some organizations recommend three repeated analyses due to the potential intermittent nature of hematuria.

Sequelae: Hematuria without an obvious underlying cause is fairly common, particularly in young adults under the age of 35 years. Hematuria may be a sign of a malignancy and should not be ignored, even if it occurs transiently. Hematuria may indicate renal disease, especially when proteinuria is present and, if needed, referral to a nephrologist for a second opinion. A diagnosis of a kidney stone does not rule out the coexistence of a malignancy. Any patient on anticoagulant or antiplatelet therapy needs a full evaluation for hematuria.

Prevention/Prophylaxis: The U.S. Preventive Services Task Force does not endorse routine screening for asymptomatic hematuria due to lack of evidence. "*No major health organization recommends screening healthy, asymptomatic patients with urinalysis in the purpose of cancer detection.*"

Referral: Primary care providers can initiate testing to verify the presence of hematuria and treat if diagnosis is clear. Referral to a nephrologist is indicated for the presence of proteinuria, RBC casts, dysmorphic RBCs, and/or elevated serum creatinine levels. Patients with hematuria persisting after treatment for a UTI or who are taking anticoagulants or in the absence of some benign cause need a referral to a urologist. Of patients referred for evaluation of hematuria or specialty care, 5% to 40% have a urological malignancy. Referral to a urologist is needed for gross and microscopic hematuria.

Education: Counsel patients and family members to seek medical advice about gross hematuria, plus signs and symptoms to report, such as urinary frequency, dysuria, flank pain, and abdominal pain. Educate patients and families about side effects of OTC medications and prescribed medications. Educate the patient about the effects of anticoagulation therapy and to call immediately if the patient notices any hematuria, because this may mean the patient's drug levels are too high.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Further evaluation is need for patients with three or more RBCs per high-power field for a urinalysis.	C	Davis et al., 2012 Sharp, Barnes, & Erickson, 2013
Once benign causes are ruled out, patient should be referred to urologist for further evaluation of hematuria.	C	Davis et al., 2012
Refer to a nephrologist for proteinuria, RBC casts, dysmorphic RBC, hypertension, and/or elevated serum creatinine levels.	C	Davis et al., 2012

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Urinalysis is not recommended in the detection of cancer screening without any signs or symptoms.	C	Sharp et al., 2013
Patients on antiplatelet and anticoagulation therapy need further evaluation by urologist and nephrology.	C	Davis et al., 2012

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HEMOPTYSIS

Description: Hemoptysis is coughing or spitting blood from the respiratory tract. This can range from blood-streaked sputum to frank bleeding. Coughing up more than 100 to 600 mL in 24 hours is indicative of massive hemoptysis.

Etiology: Hemoptysis refers specifically to bleeding from the lower respiratory tract, but sometimes bleeding from the upper respiratory tract or the upper GI tract may mimic hemoptysis. There are multiple potential causes for hemoptysis including, but not limited to, airway disease and trauma, pulmonary infections or inflammation, lung neoplasms, cardiac disease, cocaine use, and medication use (anticoagulants, antithrombotics, NSAIDs). The lungs receive blood from the pulmonary and bronchial arterial systems. The low-pressure pulmonary system produces small-volume hemoptysis, whereas the high-pressure bronchial system produces profuse bleeding. Infection causes mucosal inflammation that can lead to blood vessel rupture, and neoplasms cause mucosal invasion, which erodes the blood vessels. In the primary care setting the most common causes of hemoptysis are acute and chronic bronchitis, pneumonia, TB, and lung cancer.

Occurrence: The most common cause of hemoptysis is airway disease. There is a strong seasonal trend with peak incidence in the spring and decreased incidence in late summer.

Age: Hemoptysis may occur at any age, but the mean age of occurrence is 62 years.

Gender: Male to female ratio is 2:1.

Ethnicity: No direct ties to ethnicity have been documented, but immigrants and persons living in poverty are at higher risk for TB and other pulmonary infections.

Contributing Factors: Smoking history (active or passive exposure); alcohol use; cocaine use; any history of trauma; environmental exposures; hypertension; comorbidities including diabetes or immunocompromised status; or medications such as aspirin, NSAIDs, or anticoagulants can either be causative or contributing factors. Risk factors associated with an increased risk of finding a malignancy with bronchoscopy include male, older than 40 years, greater than 40-pack-per-year smoking history, and duration of hemoptysis longer than 1 week.

Signs and Symptoms: The quantity, characteristics, and duration of the hemoptysis should be established first when assessing the patient. It is also important to know if it has increased in severity, if the cough is worse at night, and if there are any other associated symptoms. The amount of blood loss usually is overestimated by patients and physicians, but an attempt to determine the volume and rate of blood loss should be made. Hemoptysis of more than 100 mL in under 24 hours is potentially life threatening and requires assessment in an emergency department. Any sudden onset of hemoptysis with or without any other associated symptoms can be caused by a pulmonary embolism and needs emergency treatment. A comprehensive history including the patient's known risk factors and the work and home environment are all helpful in narrowing the cause of the hemoptysis. Establish if there are any signs and symptoms of infection (fever, cough, sputum production), malignancy (cachexia, weight loss, smoking history), trauma or foreign body, or any history of immune deficiency. A thorough medication review should also be completed.

Risk factors such as smoking, cocaine use, long-term alcohol abuse, stroke, older age, immigrant status, immunocompromise, and any heart disease are important to assess in trying to find the cause of bleeding. Use of tobacco with long-standing or increasingly severe hoarseness may indicate cancer of the mouth or throat. A history of persistent "heartburn" may be a clue to possible esophageal erosion as the source of bleeding. Chronic cough, dyspnea, and/or sputum production may suggest pulmonary disease.

Vital signs should include oxygen saturation. Tachypnea, tachycardia, use of accessory muscles, cyanosis, fatigue, or altered mentation are signs of respiratory distress and should be treated as a medical emergency regardless of the amount of hemoptysis described or observed. The physical examination should begin with a thorough examination of the nose and oropharyngeal mucosa. The neck, supraclavicular area, and axillae should be palpated for any enlarged lymph nodes,

a possible sign of infection, lymphoma, or other malignancies. Auscultation of the lungs may reveal a focal abnormality consistent with pneumonia (crackles, rhonchi, wheezing); egophony is suggestive of consolidation and is often present in pneumonia. Heart sounds should be auscultated specifically for a murmur, as mitral regurgitation or mitral stenosis can be associated with pulmonary edema or decompensated heart failure, especially in the setting of clinically apparent volume overload. An abdomen distended with ascites may be accompanied by esophageal varices.

Diagnostic Tests: The test of choice is the chest x-ray or chest CT scan. Diagnostic work-up may also include these laboratory tests: CBC, coagulation studies, CMP, arterial blood gas, sputum culture and smear, purified protein derivative, INR, d-dimer, ESR, CT angiogram, or bronchoscopy. If no cause of hemoptysis can be isolated and the radiographic studies are normal, a flexible bronchoscopy for airway examination is still useful to rule out an endobronchial source of bleeding.

Differential Diagnosis: Determine first if the blood is coming from the respiratory tract (hemoptysis) or from the GI tract (hematemesis). Patients with hematemesis typically have nausea and vomiting and a history of gastric or liver disease, and the sputum more likely will be coffee ground in texture and a brown-black color. In hemoptysis, however, the sputum is bright red or pink and a frothy liquid or clotted, and the patient has a history of lung disease. If the source of bleeding is determined to be the lung, consider the possibility of infection, inflammation, malignancies, trauma, autoimmune disease, CVD, and coagulation disorders, among others.

Treatment: The goals of management include bleeding cessation, aspiration prevention, and treatment of underlying cause. If the source of the bleeding has been reasonably isolated to the respiratory system and the condition is not life threatening, and if an infectious or inflammatory etiology is diagnosed, the following diagnoses might be considered.

Acute Bronchitis: Acute bronchitis is predominantly viral and antibiotics do not shorten the course of the disease. If the patient smokes, he or she should be counseled to quit or to cut back significantly. Bronchodilators help open the airways; expectorants such as guaifenesin used to mobilize secretions are about as effective as drinking two additional glasses of water per day. Cough suppressants should be avoided except to help the patient sleep. A cool mist humidifier in the bedroom is useful for loosening secretions.

Chronic Bronchitis: Chronic bronchitis needs a combination bronchodilator as an inhaler or as a nebulizer. Because chronic bronchitis is usually a result of smoking, the patient should be counseled to stop. Antibiotics or oral steroids should be reserved for acute exacerbations heralded by increasing shortness of breath, change in color of the sputum to dark yellow or green, or fever.

Bronchiectasis: Permanent enlargement of some of the airways in the lungs caused by chronic infections, trauma, or immune system problems can result in intermittent hemoptysis. The hemoptysis usually results from dilation of bronchial arteries. Because this is a chronic illness, antibiotic selection is generally based on previous sputum culture/susceptibility results.

Bacterial Pneumonia: Community-acquired pneumonia is most often caused by *Streptococcus pneumoniae. Hemophilus influenza, Chlamydia pneumoniae, Mycoplasma pneumoniae,* and *Legionella pneumophilia* are other major bacteria that cause pneumonia in the otherwise healthy individual. If pneumonia is suspected, broad-spectrum antibiotics should be initiated empirically. Immunocompromised patients, patients with any respiratory distress, and older adult patients with significantly elevated or decreased WBC counts may need to be hospitalized. Patients with pneumonia caused by *Staphylococcus aureus, Enterobacteriaceae,* and *Pseudomonas aeruginosa* may present with a more severe illness and require hospitalization.

Fungal Infection: A fungal infection should be suspected if the pneumonia does not respond to conventional antibiotic therapy. Appropriate antifungal therapy may be initiated, and the patient should be referred to a pulmonologist.

TB: If TB is suspected based on history, signs/symptoms, and chest imaging, the diagnostic work-up should focus both on detecting the bacteria in sputum (acid-fast bacteria [AFB] culture and smear) and on protecting other individuals from contracting the infection. This may include admission to a negative pressure hospital room with airborne isolation for the diagnostic work-up. Treatment with an antituberculosis regimen can be started by the hospital or outpatient clinic; however, these patients need to be referred to the health department who will often manage treatment through directly observed administration of the antituberculosis regimen.

Follow-Up: The tenacity of follow-up depends on the severity of the illness. For pneumonia or bronchitis with minimal dyspnea, a call to check on the patient is appropriate. Worsening temperature, bleeding, or difficulty breathing requires immediate attention or emergency department evaluation. Patients with resolving pneumonia should be evaluated in 2 to 3 weeks, but bear in mind that radiographic resolution may take 6 to 8 weeks, especially in older adult patients or those with underlying lung disease. Mild hemoptysis recurring sporadically over a few years is common in smokers who have chronic bronchitis punctuated with superimposed acute bronchitis.

Sequelae: Death can occur if the etiology is not identified quickly and treated, particularly if it causes cardiovascular collapse or acute respiratory insufficiency. The mortality rate depends on the etiology, and in patients with a malignancy and massive hemoptysis (more than 1,000 mL per 24 hours) the rate is 80%. Chronic lung disease may also result.

Prevention/Prophylaxis: Counseling patients and their families about the benefits of smoking cessation and to decrease alcohol intake are worthwhile. Discussions to promote a healthy lifestyle are important, stressing activity and weight loss.

Referral: Massive hemoptysis requires treatment in the intensive care unit and pulmonary and cardiovascular surgeon consultation. If hemoptysis persists or increases, the patient may need to be referred to a pulmonologist for either bronchoscopy or laryngoscopy. Radiological imaging such as CT scan may be needed to rule out neoplasm followed by a referral to a surgeon or an oncologist.

Education: Discuss risk factors associated with pulmonary embolism, such as immobility or hormone replacement therapies. Provide patient education related to smoking cessation and cessation of street drugs and/or alcohol abuse. Patients with COPD or bronchiectasis should be aware of their own clinical signs/symptoms of worsening or acute exacerbation and have an action plan should one occur.

CLINICAL RECOMMENDATIONS	EVIDENCE RATING	REFERENCES
Moderate hemoptysis has shown similar prognosis of recurrent hemoptysis and mortality to massive hemoptysis; therefore, the more aggressive flexible bronchoscopy and bronchial artery embolization should be considered in moderate hemoptysis.	A	Lee et al., 2014
The initial test of choice is a chest x-ray. Contrast-enhanced CT with angiography adds additional information and is the method of choice.	A	Ittrich, Bockhorn, Klose, & Simon, 2017 Salamone et al., 2017
Patients with a mass on CT and those at risk for cancer should have a flexible bronchoscopy.	A	Bonlokke, Guldbrandt, & Rasmussen, 2015 Cordovilla et al., 2016
Massive hemoptysis is a respiratory emergency. CT angiography (CTA) can play a crucial role in assessing the cause and origin of hemoptysis and directing the interventional radiologist before treatment.	A	Ittrich, Bockhorn, Klose, & Simon, 2017

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xtml.

INVOLUNTARY WEIGHT LOSS

Description: Weight loss is common among older adults and is associated with poor health outcomes and functional decline. Involuntary or unintentional weight loss is a condition in which the patient does not purposefully set out to lose weight for any reason (Wong, 2014). It may be a presenting concern of the patient, reported by the family, or first noted by the provider. There is a well-established association between weight loss and mortality in older adults (Wijnhoven, van Zon, Twisk, & Visser, 2014). The Centers for Medicare and Medicaid Services (CMS) defines involuntary weight loss that must be clinically investigated as 5% in 1 month, 7.5% in 3 months, or 10% in 6 months.

Etiology: The causes of involuntary weight loss are not always clear. In 16% to 18% of patients no identifiable cause is found (Gaddey & Holder, 2014). With aging there is a decline in bone mass and a decrease in muscle mass, which is responsible for a small decline in weight after the seventh decade of life, 0.2 to 0.4 pounds per year. Another age-related phenomenon is a reduction in appetite called anorexia of aging (Wernette, White, & Zizza, 2011). Sarcopenia is a geriatric syndrome that includes decrease in muscle mass and strength, and decline in physical performance related to aging. The resulting increased ratio of fat to muscle may contribute to further weight loss because of the relationship of adipose tissue to inflammatory cytokines, which have been linked to cachexia and weight loss, including tumor necrosis factor-α (THF-α), interleukin-1 β, and interleukin-6. Cachexia is a metabolic disease characterized by the loss of muscle mass with or without the loss of fat. It may be difficult to distinguish cachexia from sarcopenia and the two may coexist. Causes of weight loss may be the result of physiological factors, psychological factors, social factors, or a combination of these (see Table 5-5). While often the primary concern is an undiagnosed malignancy, nonmalignant causes are the most common. Many medications can interfere with appetite and result in weight loss, but often medications are added as treatment rather than removed.

Occurrence: Involuntary or unintentional weight loss occurs in 15% to 20% of older adults (Wong, 2014). Using the CMS criteria, the yearly incidence of weight loss is about 7% in the community. From 30% to 50% of long-term care residents have below-average body weight, but CMS nursing

TABLE 5-5 Causes of Unintentional Weight Loss

PHYSIOLOGICAL FACTORS	
Disease	
Malignancy	Lung, GI, pancreatic, hematological, prostate
Acute illness	Influenza, pneumonia, clostridium difficile
GI disease	Malabsorption, autoimmune, oral health
Endocrine	Diabetes, hyperthyroidism
Cardiovascular	Heart failure
Respiratory	COPD, interstitial lung disease
Infectious disease	HIV, TB
Neurological disease	Dementia, stroke, Parkinson's disease
Rheumatological disease	RA, sarcoidosis, inflammation
Renal disease	Uremic cachexia
Previous surgery	Malabsorption, pain
Chronic pain	
Medications	
See Table 5-6	
Functional Issues	
Decline in ADLs and IADLs	Feeding, purchasing, and preparing food
Dysphagia	Food avoidance, dietary restrictions
Sarcopenia	Muscle weakness, decreased mobility
Falls	Fracture, fear of falling
Psychological Factors	
Depression	
Anxiety	
Grief	
Substance abuse and dependence	Alcoholism, tobacco, cocaine
Social Factors	
Social isolation	
Limited income	
Abuse or neglect	
Environment	Food desert, transportation, safety

TABLE 5-6 Medications That May Cause Weight Loss

MEDICATION	EXAMPLES
Neurological Psychiatric	Antidepressants, lithium, anticonvulsants, stimulants, levodopa, dopamine agonists, selegiline, phenothiazines, haloperidol, benzodiazepines, cholinesterase inhibitors, NMDA receptor antagonists
Cardiovascular	Digoxin, ACE inhibitors, CCB, β blockers, α agonists, hydralazine, statins, diuretics, amiodarone, procainamide, spironolactone
GI	Cimetidine, interferon, diphenoxylate-atropine, proton pump inhibitors, laxatives
Anti-infective	Metronidazole, griseofulvin, most antibiotics, amantadine
Nutritional	Calcium carbonate, iron, potassium, bile acid sequestrants
Musculoskeletal (including pain)	NSAIDs, bisphosphonates, colchicine, penicillamine, aspirin, opiates, allopurinol, colchicine, gold, hydroxychloroquine, methotrexate
Pulmonary	Theophylline, pseudoephedrine, antihistamines
Endocrine	Levothyroxine, Armour thyroid, metformin
Other	Anticholinergics

home data using their criteria reported a 6% incidence when patients with end stage disease or on hospice were excluded (CMS, 2015).

Age: Old age is a risk factor for involuntary weight loss due to the normal physiological changes with aging combined with a higher prevalence of physiological, psychological, and social factors associated with involuntary weight loss.

Gender: No documented significance; however, gender data are affected if the specific underlying disease causing the weight loss is more common in men or in women.

Ethnicity: Not significant.

Contributing Factors: When considering all causes of involuntary weight loss, nonmalignant causes are most common, but in up to 38% the cause is a malignancy. Nonmalignant GI disease is next, followed by psychiatric, endocrine, and cardiopulmonary; alcoholism; dementia; medications; loss of taste and smell; decreased functional status; chronic infection; dentition; and smoking. Social factors such as isolation, abuse or neglect, location of residence (food deserts, safety concerns), lack of transportation, and financial issues have an influence on adequate nutrition with resulting weight loss as well. Many medications contribute to factors related to weight loss and a careful review should be initiated (see Table 5-6). In the Amsterdam Longitudinal Study on Aging, weight loss due to medical reasons, unknown reasons, and a change in eating habits were all associated with increased mortality; however, no association to mortality was found when weight loss was due to intentional loss or social reasons (Wijnhoven, van Zon, Twisk, & Visser, 2014). Their interesting conclusion requires additional investigation. Because weight loss can cause skeletal muscle loss there is an increased risk for falls, functional impairment, protein calorie malnutrition, immune system compromise, anemia, and decreased cognition, all of which increase morbidity and mortality.

Signs and Symptoms: Weight loss may be reported by the patient, their family, or noted by the provider. If clinical documentation is not available, a classic guideline introduced by Marton, Sox, and Krupp (1981) suggests that a change in clothing size, confirmation of history by a family member, or ability of the patient to report exact weight change can

be substituted. A thorough history and complete physical examination are required to identify any treatable underlying cause of the weight loss. History should include a review of cardiac, respiratory, GI, genitourinary, endocrine, hematological, or infectious disease symptoms. An assessment of depression, anxiety, cognitive impairment, substance abuse, and functional decline begin with the history. There should be a focus on oral concerns such as poor dentition, new dentures, mouth lesions, dry mouth, dysphagia, or loss of taste. A review of medications both prescribed and OTC, including supplements, is necessary.

A functional cause of weight loss may be primary or secondary to a physiological or psychological etiology. Determine if the patient has functional limitations that affect the ability to prepare and eat an appropriate diet and if adequate help is consistently available to assist with ADLs if necessary. The Mini Nutritional Assessment (MNA) is supported by evidence as useful in measuring nutritional risk. The tool is accessible at www.mna-elderly.com where you can download the forms and the user's guides at no charge. There is a training video and the possibility of integrating it into the electronic medical record. There are three versions of the MNA: Self MNA, the MNA for clinical use, and the original full MNA. They all include anthropometric measurements and general, dietary, and functional assessments.

Physical examination should include cardiovascular, respiratory, GI, genitourinary, dermatological, and musculoskeletal systems. A comprehensive neurological examination and a mental status examination that includes assessment of mood, cognitive ability, and substance use are essential components. Patients with significant weight loss appear pale and cachectic, with signs of malnutrition such as hair loss, muscle wasting, and loss of subcutaneous fat. Look for evidence of loose skin, petechiae, cyanosis, clubbing of the fingers, and edema. There may be temporal wasting, icterus, dry mucous membranes, and flattened papillae of the tongue. The oral examination should note denture fit; tooth and gum disease; and oral lesions such as ulcers, stomatitis, and candidiasis. Examination of the cardiorespiratory system may reveal loud or palpable murmurs and diminished breath sounds. The liver or spleen may be palpable. Check for masses in the abdomen, breasts, and rectum. Muscle wasting may be apparent on the extremities, and deep tendon reflexes may have a prolonged relaxation phase. Patients may have diminished position and vibratory sense. Check women for ovarian masses, cervical lesions, and any obvious neoplasia. In men, examine the prostate gland for enlargement.

Diagnostic Tests: History and physical examination findings guide the choice of diagnostic tests. The shot gun approach to gathering data that may or may not be significant is no longer an option, as increasing emphasis is placed on evidence-based practice and testing that supports assumptions from the history and physical examination. In our current health-care environment it is more important than ever to remember the advice of Sir William Osler, "Listen to the patients, they will tell you the diagnosis." It has been shown that 70% of the diagnosis can be made with history alone and the physical examination increases to 90% the likelihood of diagnosis.

Additional diagnostic tests serve to confirm the assumptions. Diagnostic screening tests are shown in Table 5-7. A nutritional or diet history and calorie counts add objective data to evaluate the amount and quality of food intake. After the results of the diagnostic work-up are available, consider a neoplastic origin when benign causes of weight loss appear unlikely. Malignant and nonmalignant GI disorders are the most prevalent cause of involuntary weight loss. Evidence of microcytic anemia on the CBC or a positive fecal occult blood result is a reason to refer for colonoscopy. With frail older adults it is important to address the question of treatment options with the patient and family before further invasive and expensive work-up is initiated. If the cause of weight loss is not clear from the diagnostic evaluation, watchful waiting is recommended; watch for further weight loss or specific symptoms and reconsider a functional cause of weight loss. If no abnormalities are detected from the history, physical examination, and diagnostic tests, an organic cause is unlikely (less than 5%). When the findings from the initial work-up are negative, psychological problems, particularly depression and anxiety, are among the most common reasons for weight loss.

Differential Diagnosis: For weight loss that occurs with increased food intake, consider diabetes, thyrotoxicosis, malabsorption, leukemia, lymphoma, and adrenal insufficiency. For weight loss that occurs with normal or decreased food intake, consider alcoholism; malignancy; infection; GI, hepatic, renal, dental, endocrine, respiratory, cardiac, or psychological causes; anorexia nervosa; malnutrition; or a functional origin. After investigation, the cause of weight loss remains unknown in 16% to 28% of patients. Table 5-8 lists the causes of weight loss in the older adult.

Treatment: Treat or manage the identified underlying cause. This will likely involve the inclusion of other disciplines. Regardless of the cause, a referral to a nutritionist should be a priority. Medicare offers specific nutritional benefits to patients with diabetes or chronic kidney disease. Dentistry to assess oral health, speech to evaluate swallowing, occupational or physical therapy, and social work are all key to addressing the problem of involuntary weight loss. Nonpharmacological options should be initiated first. Diet modifications should minimize dietary restrictions, address patient preferences, and accommodate problems of dentition or swallowing. Smaller, more frequent meals can be useful if the patient reports early satiety. Patients with dementia tend to consume most of their daily energy at breakfast, so high-energy foods should be served at this meal, and varying dietary texture can be helpful. Eating with assistance and in the company of others increases calorie intake. For elders in the community, home delivered meal programs are beneficial. Oral nutritional supplements, such as high energy and high protein drinks, if initiated, should be consumed between meals. Evidence supports that their short-term use improves weight and diagnostic parameters, but data in support of their long-term use is lacking. Regular exercise, especially resistance training, stimulates appetite and can prevent increasing sarcopenia. Use of a multivitamin supplement should not be a first-line intervention; nutrient-dense foods and improving intake are preferable. Oral supplements are rich in micronutrients and when added to the treatment plan are superior to a multivitamin. Multivitamins are preferred over individual vitamin supplements.

TABLE 5-7 Diagnostic Screening Tests

TEST	RESULTS INDICATING DISORDER
Albumin Half-life of about 21 days and reflects level about 3 weeks in past	Albumin and pre-albumin are negative acute phase proteins and decrease in response to inflammation. Many chronic diseases in older adults have an inflammatory component that can result in a low albumin and pre-albumin with or without protein calorie malnutrition. Current consensus is that laboratory markers are not reliable by themselves but need to be interpreted in the context of history and physical examination. Historically albumin was interpreted as: 2.8–3.5 g/dl: Mild depletion 2.1–2.7 g/dl: Moderate depletion <2.1 g/dl: Severe depletion
Pre-albumin Short half-life: 2–3 days	Pre-albumin interpretation: 10–15 g/dl: Mild depletion 5–10 g/dl: Moderate depletion <5 g/dl: Severe depletion
Transferrin Half-life 10 days	Low in protein-calorie malnutrition, but also affected by iron status: 100–150 mg/dL: Mild depletion 150–200 mg/dL: Moderate depletion <200 mg/dL: Severe visceral protein depletion
C reactive protein	A positive acute phase reactant can be useful in interpreting the results of the albumin and pre-albumin.
Sedimentation rate	Elevation indicates inflammation and a very high sed rate can occur in temporal arteritis, polymyalgia rheumatic, relapsed Hodgkin's, or malignancy. The test needs to be interpreted in the context of other results and should not be the sole indication of serious illness.
Serum cholesterol	Total cholesterol <130 mg/dl when matched with albumin less than 3.15 g/dl was consistent with protein-calorie malnutrition if signs/symptoms of poor nutrition present.
CBC with differential	Low total lymphocyte count or an unexplained normocytic anemia may indicate malnutrition. A blood loss anemia should raise concern of malignancy.
CMP	Abnormal glucose, sodium, BUN, creatinine, AST, ALT, alkaline phosphatase may be indicative of disease links.
Hemoglobin A1c	Reflects average blood glucose over the last 90 days. Is not accurate in severe anemia.
Thyroid-stimulating hormone	1.9–5.4 μIU/mL, normal reference range. Decreased levels in hyperthyroidism, often associated with weight loss. Increased levels in hypothyroidism, associated with weight change (usually gain, but loss also occurs in older adults).
Urinalysis	Send only if clear indication for its usefulness to screen for blood, protein, or infection if patient exhibits symptoms consistent with the revised McGeer criteria (Rowe & Juthani-Mehta, 2014).
Stool for occult blood	The FIT or Cologuard screening tests are superior to the hemoccult test for screening.

It is common in clinical practice for clinicians to order mirtazapine and other pharmacological agents to stimulate appetite; however, evidence underlying their use is limited, particularly in older adults, and none has gained approval from the U.S. Food and Drug Administration (FDA). Megestrol acetate has evidence for use in cancer or HIV, but studies in older adults have had equivocal results. The risk of DVT and pulmonary embolus is alarming and increasing in patients who are sedentary. Dronabinol is a synthetic form of THC, a naturally occurring component of cannabis sativa (marijuana), which is FDA approved for chemotherapy-induced nausea and vomiting, and anorexia from AIDS or cancer. Only one study has been conducted in older adults, patients with dementia who were refusing food. The CNS side effects are a major risk in a population who has a more vulnerable CNS.

Follow-Up: If results of the standard tests listed earlier are negative, and the history and physical examination are negative, the recommended approach is to watch for further weight loss and to reconsider functional causes. Have the patient or caregiver keep a daily record of food intake, activity levels, and symptoms, and schedule a return visit in 2 to 4 weeks. Supplements may be added with the emphasis that they should be used between meals.

Sequelae: Malnutrition is the first complication to consider in involuntary weight loss. Long-term unexplained weight loss may indicate failure to thrive and the beginning of a downward spiral in health and function. Many patients in acute and long-term care have unrecognized protein-calorie malnutrition. Protein-calorie malnutrition is a common iatrogenic complication of hospitalization.

Prevention/Prophylaxis: Discuss proper nutrition and the need for dental care, if appropriate. Address functional, financial, and social issues that may affect nutrition (e.g., address depression; arrange for socialization at mealtime, if feasible; arrange home delivered meals).

Referral: Involuntary weight loss often has multiple factors and may require the assessment of multiple specialties to derive a diagnosis and plan for treatment. Include the patient and their family in discussions about goals of care. Referral for palliative care may be appropriate. Adult failure to thrive can no longer be used as the primary diagnosis for hospice admission; however consultation with hospice teams can

TABLE 5-8 Meals-on-Wheels Mnemonic for Causes of Weight Loss in the Older Adult

MNEMONIC	CAUSES OF WEIGHT LOSS
M	Medications
E	Emotional problems (depression, anxiety)
A	Alcoholism, anorexia tardive, abuse
L	Late life paranoia
S	Swallowing disorders (dysphagia)
O	Oral problems
N	Nosocomial infections No money (poverty)
W	Wandering and other dementia-related behaviors
H	Hyperthyroidism, hypercalcemia, hypoadrenalism
E	Enteric problems (malabsorption)
E	Eating problems (inability to feed oneself)
L	Low-salt, low-cholesterol diet
S	Social problems Shopping and meal preparation Stones (cholecystitis)

Source: Morley & Silver (1995).

help in determining the primary diagnosis. Always consult a specialist for rapid or acute decline; for positive diagnostic test results indicating malignancy, thyrotoxicosis, or other acute organic illnesses requiring sophisticated diagnostic work-up or management; or for a patient condition beyond your current scope of practice. Refer patients for assistance if social or functional issues may be contributing to weight loss. Refer to an appropriate specialist as noted previously.

Education: Instruct patients about proper nutrition and hydration. Encourage exercise to improve strength or function and to stimulate appetite. Teach patients about medication side effects that may affect nutrition and about signs or symptoms of overmedication or undermedication.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
There is a relationship between depressive symptoms and weight loss. Patients with weight loss should be screened for depression.	A	Kim, Noh, Park, & Kwon, 2014
Oral nutritional supplementation is associated with weight gain and reduced mortality.	A	Morley, 2012
Nutritional supplementation should be given between meals.	A	Morley, 2012
Loss of weight in obese older adults is associated with greater morbidity than in non-obese older adults even when they maintain loss or regain weight.	A	Fabbri et al., 2015
If clinically documented weight loss is not available, a change in clothing size, confirmation of history by a family member, or ability of the patient to report exact weight change can be substituted.	B	Marton, Sox, & Krupp, 1981 classic study
Treatment should address the underlying cause. Involve a multidisciplinary team.	B	Gaddey & Holder, 2014 Wong, 2014
Optimize caloric intake, minimize dietary restrictions, eat in the company of others.	B	Gaddey & Holder, 2014 Morley, 2012
No medications have been shown to reduce mortality. Few studies have been done to determine effectiveness in older adults with involuntary weight loss, and all have considerable side effects.	B	Gaddey & Holder, 2014 Wong, 2014

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

JOINT PAIN

Description: Joint pain can be discomfort or inflammation arising from any part of a joint, including the cartilage, bone, ligaments, tendons, or muscles. It is commonly referred to as arthralgia, which is inflammation within the joint itself (Mayo Clinic, 2016).

Etiology: Any damage to the joints from disease or injury can interfere with movement and cause a lot of pain. Many different conditions can lead to painful joints, including osteoarthritis, rheumatoid arthritis (RA), bursitis, gout, strains, sprains, and other injuries. The description, location, onset, and associated symptoms are important to diagnosing the cause of joint pain. Clarifying the etiology of the pain depends on the clinical presentation, complete history, findings from physical examination, and appropriate diagnostic testing. The pain can be mild (from strained muscles or ligaments that support the joint), chronic (lasting more than 6 months, from degenerative joint disease, arthritis, osteoarthritis), inflammatory (from joint effusion, RA), or severe and acute (from trauma or infection). Drug-induced arthralgia can occur with some medications. Acute joint pain may become a chronic pain if posture, overuse, and weight bearing are not corrected or eased. For example, pain of osteoarthritis of the knee may initially occur intermittently during weight bearing and evolve into persistent chronic pain. Systematic causes of joint pain may include lupus, sarcoidosis, Lyme disease, hemophilia, fibromyalgia, Raynaud's disease, and Paget's disease (Kong, Lauche, & Chang, 2016).

Occurrence: In the United States, an estimated 52.5 million adults have provider-diagnosed arthritis, and 22.7 million report limitations due to their arthritis (CDC, 2015). Neogi (2013), reported that joint pain symptoms requiring medical attention are projected to increase to nearly 67 million by 2030.

Age: Joint pain is found more frequently in the older adult patient as a result of age-related muscle atrophy, demineralization of trabecular bone, atrophy of cortical bone, chronic disease, and loss of bone density. Arthritis (degenerative and inflammatory) is the most common cause of disability in individuals more than 75 years old, and it affects more than 80% of individuals in this age group, with joint pain being the dominant symptom.

Gender: Joint pain occurs more frequently in women than in men.

Ethnicity: Prevalence of certain types of inflammatory disease and systematic disorders are higher in various ethnic groups.

Contributing Factors: Factors that may lead to joint pain include overuse or strain, previous injury or trauma to the joint, alterations in gait or balance, past surgery or joint replacement, known history of joint disease, and postmenopausal women with no hormone replacement therapy. Physical stature, weight, smoking, and family history should be assessed in reviewing the risk factors for osteoporosis. Other contributing factors include obesity, poor nutritional habits, low calcium intake, alcohol consumption, medications, and personal exercise habits. Older adults with obesity may suffer from multi-site pain due to the strain caused by the pressure of the additional weight on the joints. Longitudinal evidence shows that a high body mass index (BMI) precedes and independently predicts foot joint pain and knee pain. Deterioration of the normal gait and mobility may be affected by the combined stressors of lower body joint pain (Zdziarski, Wasser, & Vincent, 2015).

Signs and Symptoms: Using a systematic method to obtain the history, such as the comprehensive OLDCART mnemonic, is essential to identifying the symptom of joint pain. The patient history should identify all joints affected and include the onset of the pain (sudden, insidious), character of the pain (dull, sharp), and duration and location of the pain (including radiation). Inquire about any associated symptoms, alleviating or aggravating factors, and timing (e.g., worse in morning, better late in the day). Morning stiffness points toward an inflammatory arthritis, whereas post-exercise stiffness indicates a degenerative process. Symptoms associated with joint pain may include erythema, warmth, general weakness, stiffness, and decreased range of motion.

Discuss normal activity levels and the possibility of overuse, injury, and strain. Has the patient experienced any past or recent injury to the joint? Has the patient heard any popping, crepitus, or clicking? Discuss the limitation in joint mobility and note the presence of edema, redness, or fever. Review any associated muscle fatigue, joint deformity, or inability to perform certain movements. Red flags include unremitting pain, systematic symptoms (fever, chills, fatigue, weight loss), significant disability, and change in function.

The physical examination includes a complete assessment of the patient. Inspect and palpate each joint and assess the proper range of motion, noting any pain or limitations in movement. Include additional diagnostic maneuvers, if indicated, such as the drawer sign for ligament injury, the McMurray test for meniscal tears, or the Thomas test for hip flexion contracture. Assess the patient's gait, balance, and posture. Inspect each joint, noting the musculature, any atrophy, contractures, nodules, asymmetry, or gross deformity. Note any erythema, inflammation, or soft tissue swelling on or near the joint. Joints affected by RA usually involve the metacarpophalangeal joints and the wrist, while distal involvement of the interphalangeal joints suggests osteoarthritis. Palpate for any tenderness or effusions at the joint and check for crepitus, clicking, or popping. Sensory testing may reveal neurological or vascular involvement (Neogi, 2013). Extra-articular signs (rashes, tophi, conjunctivitis, mouth ulcers) provide additional information toward a differential diagnosis.

Diagnostic Tests: Diagnostic tests are not specifically indicated with a complaint of joint pain; they are useful to identify specific problems consistent with findings from the history and physical examination. A CBC with differential will rule out infection and an ESR may support the concern of an inflammatory process. Marked elevations in alkaline phosphatase level are seen in patients with bone disease (e.g., Paget's disease, metastatic bone disease). Hypercalcemia with marked alkaline phosphatase is seen in patients with hyperparathyroidism or bone carcinoma. Serum creatine kinase (CK) isoenzymes are elevated in persons with muscle trauma and progressive muscular disease. If indicated, obtain

a complete electrolyte panel and urinalysis. Check BUN and creatinine levels to assess renal function. Rheumatoid factor may be of use, but it is negative in approximately 30% of all RA cases and the test has a high false-positive rate. A more specific test is the anti-cyclic citrullinated peptide (anti-CCP), which tests for an antibody present in most patients with RA. X-rays of the joint are not necessary routine, but can be useful to detect fracture, joint space destruction or narrowing, erosion, arthritic changes, and degeneration. Joint aspirations may be done to distinguish inflammatory, noninflammatory, and infectious conditions, and may provide symptomatic relief.

Differential Diagnosis: Other conditions to consider include gout, pseudogout, psoriatic arthritis, connective tissue disease, Reiter's disease, multiple myeloma, scleroderma, lupus, septic arthritis, polymyalgia rheumatic (PMR), cervical radiculopathy, fibromyalgia, and spinal stenosis. PMR is not uncommon in older adults and should be considered when the large joints are involved and the onset of pain is sudden. PMR is diagnosed with symptoms and an elevated ESR.

Treatment: The goal in acute joint pain is to reduce pain and swelling, if present. If the pain is due to acute injury, the mnemonic PRICE is helpful: **P**rotection (brace or wrap), **R**est, **I**ce (15 minutes on and 15 minutes off for several times a day), **C**ompression (elastic wrap), and **E**levation (elevate the joint above the heart level). Emergency intervention is required if you suspect a fracture, avascular necrosis, or sepsis.

Nonpharmacological treatment should be the first line of recommendation. Weight loss of even a small amount can make a difference in pain. Physical therapy can be used to develop a plan for treatment that includes exercise and muscle strengthening. The use of a cane to off load weight and provide stability is an option. Application of moist heat can help with both knee and back joint pain. The use of transcutaneous electrical nerve stimulation and acupuncture is increasing in application.

Pharmacological management should be based on the type and number of joints involved and the presence of comorbidities. Topical NSAID agents have been shown to be as effective as oral agents, especially with knee arthritis. Edwards and colleagues (2016) noted that the NSAID diclofenac sodium topical gel 1% applied up to four times a day resulted in 30% pain reduction of knee osteoarthritis pain. Capsaicin, an enzyme found in hot pepper, depletes substance P when applied to the painful area three times a day for several weeks. Side effects may include burning and erythema, and it should never be applied to open or irritated skin.

Oral NSAIDs are on the Beers' Criteria for Potentially Inappropriate Medication and if used with older adults should be considered for short-term use. Consider the selective Cox-2 inhibitors: moist heat, intra-articular steroid injections, assistive devices, and surgery. Acetaminophen, which was a cornerstone of treatment for osteoarthritis in older adults in the past, is no longer recommended as first-line treatment based on the risk of toxicity and lack of evidence of pain relief. The total dose for an older adult should not exceed 3 g a day from all sources. Newer agents useful in inflammatory arthritis include disease-modifying antirheumatic drugs and biological response modifiers. Intra-articular injections can also provide short-term relief.

Polsunas and colleagues (2016) made the following recommendations for dosing medications: acetaminophen 325 to 1,000 mg every 4 to 6 hours, maximum dosage 3 g per day; tramadol start 25 mg daily, increase 25 to 50 mg every 3 to 7 days to 100 mg every 6 hours. Side effects for tramadol may include seizures and orthostatic hypotension, and tramadol is now a schedule IV medication which requires a U.S. Drug Enforcement Administration (DEA) number to prescribe. Opioid medications include hydrocodone/APAP 5/325 to 10/325 mg every 4 to 6 hours, maximum 3 g per day of APAP; oxycodone 5 to 10 mg every 4 hours. Side effects of this class include an increased risk of falls.

Follow-Up: Monitoring of patients depends on findings. Patients with acute or severe joint pain may need emergency care and intervention. Monitor patients with chronic pain frequently until the clinical work-up is completed. Management of chronic joint pain requires routine visits to monitor the effectiveness of treatments and follow-up laboratory and diagnostic tests, if indicated.

Sequelae: The condition and prognosis relate to the etiology of joint pain.

Prevention/Prophylaxis: Weight reduction to decrease stress on the joint when possible; physical therapy and exercise for strengthening; and moist heat, topical medications, and steroid injections to reduce pain and spasms. Instruct the patient on joint protection and preservation. Alternative methods of pain control such as acupuncture, yoga, medication, or massage therapy may be recommended. Tai chi showed improvement in chronic pain conditions, including low back pain, fibromyalgia, and osteoporosis (Kong, Lauche, & Chang, 2016). These alternatives help with pain, fatigue, and feelings of helplessness of chronic joint symptom conditions such as RA, osteoarthritis, and fibromyalgia. Assistive devices such as canes, crutches, or walkers should be discussed to prevent falls. Information on nutrition, the use of calcium supplements, and smoking cessation should be discussed for further measures.

Referral: Refer patients to a specialist pending the results of the work-up. Refer patients to the appropriate emergency service if you suspect sepsis, fracture, or avascular necrosis. Referral to physical or occupational therapy for exercises to reduce pain and improve strength and endurance is advised (Polsunas et al., 2016).

Education: Teach patients about the appropriate dose of medications and the potential side effects that need to be reported. Instruct patients about appropriate follow-up and referrals. Tell the patient to report any persisting or worsening joint pain and any systemic involvement. Instruct patients on the appropriate methods for pain control. Review the use of any adaptive equipment and the role the patient plays in avoiding disability and slowing progression of the disease. Low-impact exercise is important for obese patients. Hermsen and colleagues (2016) noted education in cognitive appraisals and coping strategies have contributed to positive physical functioning for self-efficacy. Patients with chronic joint pain conditions and higher social support have shown lower levels of depression (Lee, Kahana, & Kahana, 2016). Neogi (2013) noted that joint pain also contributes to the socioeconomic burden, so providers should discourage nonevidence-based costly treatment measures.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Diclofenac sodium topical gel 1% applied up to four times a day resulted in 30% pain reduction of knee OA pain.	A	Edwards et al., 2016
Education in cognitive appraisals and coping strategies have contributed to positive physical functioning for self-efficacy.	A	Hermsen et al., 2016
Tai chi showed improvement in chronic pain conditions including low back pain, fibromyalgia, and osteoporosis.	A	Kong, Lauche, & Chang, 2016
Patients with chronic joint pain conditions and higher social support have shown lower levels of depression.	A	Lee, Kahana, & Kahana, 2016
Aerobic exercise, resistance exercise, or multimodal exercise programs (combination of the two types) can reduce joint pain in older obese adults in the range of 14%–71.4%.	A	Zdziarski, Wasser, & Vincent, 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

PERIPHERAL EDEMA

Description: Peripheral edema is swelling due to an increase in the interstitial fluid volume and is not clinically apparent until the interstitial volume has increased by 2.5 to 3 L. Lymphedema, or lymphatic obstruction, occurs when a compromised lymphatic system causes localized fluid retention and tissue swelling. There are numerous etiologies for peripheral edema ranging from benign to life-threatening clinical conditions.

Etiology: Normally, the distribution of water between the blood and interstitial tissues is maintained by hydrostatic and oncotic pressures in each compartment. Starling's concept—the osmotic pressure of the plasma proteins balances the hydrostatic pressure in the capillaries—helps in the understanding of fluid dynamics. Fluid flows from the vessels to the interstitial area in response to intravascular hydrostatic pressure and the colloid osmotic pressure of the interstitial fluid. In the opposite direction, fluid enters the blood because of the interstitial tissue tension and the oncotic pressure of the plasma proteins. Interstitial fluid also is returned to the blood as lymph. Under steady-state conditions, net fluid flux out of the capillary is balanced by lymph flow back into circulation. Alteration in any of these compartments (intravascular, extravascular, lymphatic) upsets the equilibrium, and edema occurs. Two basic steps occur in edema formation: sodium and water are retained, and capillary hemodynamics are altered.

Edema is common in older adults, has diverse etiologies, and more than one disorder can be causing edema in the same patient. The most common cause in patients older than 50 years of age is venous insufficiency. Other common causes of edema include heart failure, DVT, cirrhosis, renal disease, and drug-induced edema. The swelling that results from lymphedema is nonpitting. It may be primary due to congenital or inherited conditions, or secondary caused by damage to the lymphatic system; cancers (especially breast, ovarian, and prostate); infection; inflammation; or obesity (see Table 5-9).

Occurrence: The frequency of occurrence depends on the underlying etiology.

Age: Peripheral edema occurs frequently in older adults.

Gender: No documented significance is found, but gender statistics vary according to the specific etiology. Lymphedema and venous insufficiency are more common in women.

Ethnicity: While moderate edema does not have a notable ethnic difference, severe venous insufficiency is less common in African American women.

TABLE 5-9 Common Causes of Peripheral Edema

Unilateral, acute	DVT, ruptured Baker's cyst, compartment syndrome
Bilateral, acute	Allergic reaction, trauma, burns, bilateral DVT, acute worsening of renal failure, or congestive heart failure (CHF)
Unilateral, chronic	Venous insufficiency, lymphedema, pelvic tumor, and reflex sympathetic dystrophy
Bilateral, chronic	Venous insufficiency, pulmonary hypertension, CHF, lymphedema, medications, renal disease, idiopathic, dependent edema, anemia

Source: Adapted from O'Brien, J. G., & Miles, T. P. (2013). Edema. *Essential Evidence Plus*. Retrieved from http://www.essentialevidenceplus.com.proxy.its.virginia.edu/content/eee/29

Contributing Factors: Physiological age-related changes increase the vulnerability to fluid retention. The older adult has a smaller amount of total body water overall (80% of body weight for a newborn, 60% for a young adult, 50% for an older adult), and of this, two-thirds is intracellular and one-third is extracellular. As more intracellular fluid is lost over time, extracellular fluid volume starts to comprise more body water. Risk factors for peripheral edema from age-related changes include a decrease in:

- Serum albumin—less than 2 g/dL can cause edema
- Glomerular filtration rate
- Hepatic blood flow
- Sodium-concentrating ability of the kidney
- Myocardial contractility and cardiac output
- Baroreceptor sensitivity

Medications that are used to treat conditions common in older adults can cause peripheral edema:

- NSAIDs cause increased sodium absorption in the kidney due to the blockage of prostaglandin.
- Thiazolidinediones are used as treatment for diabetes (pioglitazone or rosiglitazone) and stimulate sodium reabsorption in the kidney; worsening heart failure is a known side effect.
- Antihypertensives with mechanisms that cause vasodilation or activation of the renin-angiotensin-aldosterone sympathetic nervous system may increase peripheral edema (hydralazine, dihydropyridine calcium channel blockers, and alpha blockers).

Additional contributing factors include dependent positioning of the lower extremities, excessive intake of sodium, and hot weather.

Signs and Symptoms: The patient may complain of fullness, discomfort, aching pain, shoes that are too tight, wounds, gait disturbance, self-image issues, or weight gain. The history should elicit the seven dimensions of the signs and symptoms of edema, emphasizing location (unilateral or bilateral) and chronological evolution (acute or chronic progression). Associated symptoms need to be elicited, particularly shortness of breath or awakening at night with difficulty breathing. The presence or absence of pain is helpful in determining the differential diagnosis. Ask about aggravating and alleviating factors, including the effect of prolonged sitting or standing, and determine the effect of passive leg elevation. A history of malignancy, pulmonary, cardiac, renal, hepatic, endocrine, or PVD is important. Ask about recent weight loss or gain, changes in diet, salt intake, and alcohol use. A thorough review of medications (both prescribed and OTC), particularly new medications or a recent change in dosage is essential. Has the patient been on a trip recently or had periods of prolonged standing or sitting?

The physical examination begins with weight. Assess skin changes, including pigmentation and thickening, lesions, discoloration, texture, temperature, and induration. Evaluate the venous and arterial circulation, checking pulses throughout; capillary refill; and dependent rubor. Varicose veins are usually readily apparent with the inspection of the legs while the patient stands. Assess the extremity for local or diffuse tenderness and for pitting or nonpitting edema. Measure and compare the circumference bilaterally from a fixed reference point above the ankle to the area of maximal edema (e.g., patella, calf, midcalf). Assess any localized enlargement.

Pitting edema has no universal scale for measurement. It is generally defined on a scale of 1+ to 4+ or brawny edema (which is chronic and may not be pitting). Edema is assessed by applying firm pressure with your thumb over the area of swelling. A commonly used scale for grading edema refers to the depth of the impression: 2 mm = 1+, 4 mm = 2+, 6 mm = 3+, and 8 mm = 4+. Another method adds the amount of time it takes for the impression to return to normal: 30 seconds or less = 1+, 30 to 60 seconds = 2+, 60 to 90 seconds = 3+, and 90 to 120 seconds = 4+. To assess whether nonpitting edema represents the presence of lymphedema, a technique that is recommended was defined by Kaposi and later by Stemmer. The Kaposi-Stemmer sign suggests lymphedema when there is an inability to tent the skin over the dorsum of the foot at the base of the second toe.

The examination should also include body systems as indicated by the history, particularly pulmonary, cardiac, hepatic, renal, and endocrine. If arterial insufficiency is suspected, an ankle-brachial index (ABI) measurement should be performed. In unilateral edema, a 1-cm difference in size above the ankle or a 2-cm difference at the calf is significant and should lead to further investigation.

Diagnostic Tests: Diagnostic tests depend on the probable etiology of peripheral edema. Cardiac causes may warrant a chest x-ray, EKG, or echocardiogram. Laboratory studies also depend on the diagnostic considerations: CBC for suspected anemia, with a differential if infection is a possibility; CMP, which includes electrolytes, glucose, BUN, creatinine, albumin, phosphorus, AST, ALT, and bilirubin. More targeted chemistries may be reasonable; for example, a basic metabolic, renal, or hepatic panel. Thyroid function can be assessed with a high sensitivity thyroid-stimulating hormone with a reflexive addition of free thyroxine if abnormal. Additional studies could include ultrasound, venogram, venous Doppler studies, lymphoscintigraphy, or the more invasive lymphangiography. CT scans or ultrasound for pelvic masses and lymphoma may be indicated, as well as a CA-125. Urine studies quantify protein losses and urine electrolytes.

Differential Diagnosis: Peripheral edema is an important clinical sign and its causes are diverse. The differential diagnosis is facilitated in part by noting if edema is unilateral or bilateral and recognizing distinctions between lymphedema, lipedema, and venous stasis. Lipedema is a bilateral, symmetrical deposition of fat in the lower extremities that spares the feet (a distinguishing feature) and occurs exclusively in obese women. A multisystem approach that excludes major organ system dysfunction is useful, especially pulmonary, cardiac, endocrine, hepatic, and renal disease. An accurate diagnosis is important because the mainstay of treatment is reversing the underlying disorder, if possible.

Treatment: Always direct treatment at the cause of peripheral edema: diuretics for heart failure, a high-protein diet for hypoalbuminemia, ACE inhibitors for proteinuria, and thrombolytics for acute DVT. Any drugs known to cause edema should be discontinued or decreased in dosage. Many patients expect that any edema should be treated with a diuretic, but if fluid overload is not the issue, diuresis can

actually cause tissue death. Edema due to venous stasis, for example, should focus on that specific etiology, which may not require diuretics.

Compression Stockings: Compression stockings decrease the capillary filtration rate, thus decreasing edema. They are especially useful in patients with venous insufficiency. There is no evidence to indicate that high compression (30 to 40 mm Hg) is clinically more advantageous than medium compression (20 to 30 mm Hg), and patients are more compliant with medium compression. The stockings can be difficult to pull on, and a stocking donner can be helpful. Give the patient a prescription for two sets to allow for laundering. They need to be replaced every 6 months because the compression properties decrease with wear. Stockings that zip are available. Send the patient to the medical supply store where trained fitters will ensure the proper fit. Compression stockings are not recommended for patients with peripheral artery disease (PAD), and if suspected, an ABI can be ordered to screen for PAD.

Behavioral modifications include intermittent periods of recumbency, the avoidance of environmental heat, and sodium and water restriction as indicated. Exercise improves the muscle pump action and strengthens leg muscles.

Follow-Up: Closely monitor patients for effectiveness of therapy and adverse events. Monitor the weight and limb circumference measurements using the same reference points because a patient's weight may increase 10% before pitting edema is clinically evident.

Sequelae: When administering diuretics, the health-care professional must be alert to potential volume depletion. Older adult patients are very sensitive to diuretics, and their administration may result in falls, weakness, confusion secondary to sodium and other electrolyte abnormalities, urinary incontinence, gout attacks, volume depletion, and acute renal failure. Patients with edema as a result of deep venous insufficiency are prone to recurrent ulceration.

Prevention/Prophylaxis: For patients with recurrent lymphangitis and cellulitis, prescribe intermittent long-term antibiotic prophylaxis. For patients with leg swelling secondary to DVT, thrombolytics may limit the tissue loss, pulmonary embolus, and more extensive thrombosis of the deep venous system.

Referral: Referral to specialists depends on the diagnosis. It would be rare to hospitalize a patient for most causes of peripheral edema, thus treatment is almost always outpatient. Even DVTs are treated in the outpatient setting in consultation with a vascular medicine specialist with low-molecular-weight heparin (enoxaparin), warfarin, or non-vitamin K antagonist oral anticoagulant (NOAC) (rivaroxaban, dabigatran, apixaban, and edoxaban). The NOACs offer benefits above warfarin because they do not require routine monitoring, have predictable pharmacokinetics, fewer drug-drug interactions, and limited drug-food interactions.

Education: Patients need specific information related to the edema and the management of symptoms. Typically, the patient with edema is instructed to avoid highly salted foods (follow a 2 to 3 g sodium diet). If compression stockings are recommended, emphasize the importance of proper sizing and application technique to avoid excessive pressure. Instruct patients to elevate the legs above heart level to decrease peripheral vascular pressure for 10 to 15 minutes three to four times a day. Periodic active muscle contraction exercises are important for individuals who must sit for long periods. Teach patients with peripheral vascular disorders to avoid excessive heat and reduce weight if indicated. Special care of the skin, including proper shoes and prevention of trauma, is important.

CLINICAL RECOMMENDATIONS	EVIDENCE RATING	REFERENCES
When DVT is suspected, use the Wells Clinical Rule, D-dimer, and selective use of imaging to confirm the diagnosis.	A	O'Brien & Miles, 2013
Begin by evaluating whether edema is unilateral or bilateral, painful or nonpainful, and acute or chronic.	B	O'Brien & Miles, 2013 Trayes et al., 2013
Leg edema in immobile patients due to venous insufficiency can be adequately managed with compression and physical therapy alone.	B	Suehiro et al., 2014
Brain natriuretic peptide (BNP) is helpful for distinguishing heart failure from other causes of leg edema.	B	O'Brien & Miles, 2013

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PRURITUS

Description: Pruritus is an unpleasant, irritating, or itching sensation on the surface of the skin that may lead to the desire to scratch. It may be localized or generalized. Pruritus may be acute (less than 6 weeks) or chronic (more than 6 weeks). Characteristic features include scratching and inflammation.

Etiology: The pathophysiology of pruritus remains unknown. Inflammation results from activation of the immune response. Immunoglobulin E (IgE) in the skin surface cells triggers an inflammatory response in which histamines and other neurostimulators are released. Repetitive rubbing and scratching exacerbate the inflammation. As the skin ages, integumentary and vascular changes lead to decreased skin moisture, sweat, and sebum production, all of which contribute to dry skin. The decline in immune function with age produces increased incidence of autoimmune diseases that can cause pruritus. Pruritus can be caused by inflammatory skin disease, exogenous trigger factors (scabies, mites, toxins, allergens, wind), endogenous trigger factors (medications and pH changes), or systemic disease (diabetes, iron deficiency anemia, chronic renal insufficiency, liver disease, tumors, HIV). Psychogenic pruritus (somatoform pruritus) is another common condition. Medications can induce pruritus, including antihypertensives (ACE inhibitors, calcium channel blockers), antibiotics, opioids, and psychotropics. Drug-induced pruritus may be localized or generalized and may start after the first dose of a drug or many weeks or months after being on the drug.

Occurrence: There is no epidemiological data base for pruritus, therefore the overall incidence and prevalence unknown, but it is the most common symptom seen in dermatology, and various skin and systemic diseases are associated with it.

Age: Pruritus is the most common skin complaint in individuals more than 65 years old, and the incidence is about 20% in those more than 85 years old. Analysis of hospital admission data indicates that 11.5% of older adults had a primary diagnosis of pruritus and two-thirds of patients in skilled nursing facilities experience it. Xerosis (dry skin) is the most common cause in older adults.

Gender: Pruritus is more prevalent in women than in men.

Ethnicity: Genetic variation may affect receptors for itch as well as response to anti-pruritic therapies. The prevalence of pruritis is greater in darker skin types (Hajdarbegovic & Thio, 2012).

Contributing Factors: Once a pattern for pruritus has been initiated, continued scratching causes a cascade of inflammatory mediators that provoke the itch sensation and repeat scratching. Additional factors that contribute to dryness include winter weather (low environmental humidity), daily use of cleansers, and bathing without replacement emollients. Sleep deprivation can cause an increase in the release of an inflammatory mediator, and though it is often a result of pruritus, it can also contribute to it.

Signs and Symptoms: A thorough history and physical examination should be done in all patients with pruritus. Focused questions should include:

- How did it start (sudden or gradual)?
- Characterize the sensation—is it prickling, crawling, burning, stinging?
- How severe is it?
- How frequently does it occur and for how long?
- Does anything make it worse or better?
- Where does it itch (local or systemic)?

Asking if and how it might interfere with activities or sleep is important. Asking patients what they think the cause might be might help. Find out about allergies and if there was an exposure to new creams or detergents. Ask about systemic-associated symptoms such as weight loss, fever, heat/cold intolerance (thyroid disease), nausea and vomiting (chronic renal failure), and polyuria/polydipsia (diabetes). Additional history of importance is past medical history, including previous skin conditions, skin care routine, and current medications. Brief psychological screening may provide some insights into the potential cause. A complete physical examination is warranted, examining all surfaces of the skin, including the hair and nails. Assess texture, temperature, moisture, and turgor of the skin. Assess lesions and document using drawings or photos accurately recording the location, shape, and characteristics. Look for secondary lesions such as lichenification (the thickening of the skin from continued scratching). The general physical examination is focused on identifying potential systemic causes and contributors to pruritus and includes palpating lymph nodes and the thyroid gland.

Diagnostic Tests: No specific tests are indicated for a complaint of pruritus, though it might include a CBC, ESR, and/or C-reactive protein to determine infection/inflammation. Patch testing or photo testing may be needed to establish a diagnosis. If a systemic disease is suspected, an appropriate thyroid panel, liver function tests, serum glucose levels, serum iron level, renal values (serum urea and creatinine), stool examination for parasite detection, and HIV testing may be done.

Differential Diagnosis: The differential diagnosis is quite broad and include dermatological causes, hypersensitivities, allergic reactions, scabies, lice, neuropathic and neurogenic causes, and psychiatric causes, as well as pruritus associated with systemic diseases. Pruritus is present in 80% to 100% of persons presenting with cholestasis and jaundice. There may be more than one cause, and frequently a cause is never determined.

Treatment: Treatment goals should be patient-specific and tailored to the patient's physical and mental ability and any concurrent medical issues, severity of the symptoms, and potential for adverse effects of treatments. Goals of treatment include alleviating the itch and maintaining skin integrity. Treatment is aimed at the cause. Xerosis, the most common cause, is treated with emollients that rehydrate the skin and reduce inflammation, and some may additionally include a mild anesthetic that helps decrease the sense of itching.

Topical Therapies: These are most useful with localized forms of pruritus. Topical steroids can reduce inflammation and itching, though these are avoided for generalized itching

or for prolonged periods because they cause skin thinning. An antihistamine topical cream may be used or a menthol cream, which has a cooling effect. Topical immunomodulators act on nerve fibers, and capsaicin topically has been found to be helpful with neuropathic origins such as postherpetic neuralgia. Local anesthetics can decrease transmission in sensory fibers.

Systemic Therapies: These are frequently used for treatment of generalized pruritus. Systemic medications that have antipruritic properties include serotonin and norepinephrine reuptake inhibitors (SNRIs), selective serotonin reuptake inhibitors (SSRIs), and tricyclic antidepressants. Mirtazapine (an SNRI), paroxetine (an SSRI), and doxepin (tricyclic antidepressant) have antihistaminic and serotonergic effects and have been used to treat pruritus. The antidepressants may be helpful in psychogenic causes. Antihistamines, neuroleptics, and opioid agonists and antagonists can be helpful in some circumstances. First-generation antihistamines may help patients experiencing nocturnal pruritus sleep due to soporific effects; however, their anticholinergic properties are problematic in older adults. Gabapentin can be used instead, starting at 100 to 300 mg at bedtime, with a maximum dose of 1,800 mg in divided doses. Systemic steroids should only be used in acute, severe forms of pruritus.

Nonpharmacological Measures: These include removing any environmental allergens if identified, using an occlusive dressing in a localized area to reduce scratching, cool dressings, psychotherapy, and acupuncture. Phototherapy has been used for decades to treat various pruritic dermatoses.

Follow-Up: Monitor the patient frequently to evaluate the effectiveness of treatment, reinforce adherence, and watch for secondary complications.

Sequelae: Secondary infection is a risk when the skin is broken. Neglecting the symptom can have a negative effect on the patient's quality of life, especially through sleep deprivation.

Prevention/Prophylaxis: Avoid triggering or exacerbating factors. Bathing less often in warm (not hot) water, increasing the humidity, and using a soft towel to pat dry gently rather than vigorous rubbing are advised. Apply emollients after bathing (and never in the tub due to fall risk), applying them gently to avoid overstimulating the nerve endings. Eating a healthy diet and drinking plenty of water is advised.

Referral: Refer to a dermatologist or an allergist for severe cases or when treatment is ineffective.

Education: Education should include an explanation of the diagnosis and the triggering and exacerbating factors. In addition to bathing techniques and how and when to use moisturizers, educate the patient to keep fingernails short, wear light and loose clothing, avoid alkaline and alcohol-based cleansers, maintain comfortable temperature and humidification, and alleviate stress. Discussion of breaking the itch-scratch cycle is very important.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Keep cool, use emollients, and take short, tepid showers. Avoid soaking and use moisturizing soaps as opposed to antibacterial soaps.	C	Valdes-Rodriguez, Stull, & Yosipovitch, 2015
First-generation and second-generation antihistamines have an antipruritic effect in management of chronic pruritus.	B	Berger et al., 2013
In patients with chronic atopic dermatitis, exacerbating factors and appropriate treatment options are important and interventions that include educational interventions report significant benefits.	A	Arkwright et al., 2013

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SYNCOPE

Description: Syncope is a sudden and transient loss of consciousness and postural tone resulting from a reduction in oxygen to the brain with immediate spontaneous recovery. While of short duration, it can result in physical injury, accidents, and a decreased quality of life.

Etiology: Etiologies for syncope range from trivial to lethal. In most instances, the loss of consciousness reflects a temporary decrease in cerebral blood flow that is usually secondary to a fall in the systemic arterial blood pressure influenced by metabolic regulation, chemical regulation, and autoregulation.

Reflex-mediated and orthostatic causes are usually more benign, whereas cardiac and neurological causes are more dangerous. In the Framingham Heart Study, cardiac syncope doubled the risk of death compared to those without syncope. The most common cause of syncope is the neurocardiogenic reflex or vasovagal syncope, which represents about one-third of all causes (da Silva, 2014). The second most common reflex syncope that has the highest incidence in older adults is carotid sinus syncope, which is more common in men and reproducible with carotid massage. Other reflex syncope is more situational, triggered by cough, micturition, or defecation. Cardiovascular causes of syncope include orthostatic hypotension, which is particularly common in older adults, and bradycardia or tachyarrhythmias. Valvular heart disease and pulmonary hypertension, while not common causes of syncope, can be treatable. Studies show that the cause of syncope remains elusive in 30% of cases of patients admitted for syncope.

Occurrence: The Framingham Heart Study showed that 3% of men and 3.5% of women had experienced at least one syncopal episode. Approximately one-third of the population will experience a syncopal event in their lifetime. Syncope accounts for about 3% to 5% of all emergency department visits and 1% to 6% of all hospital admissions.

Age: The lifetime incidence of syncope may be 25% in older adults, and 80% of all emergency department admissions for syncope are individuals older than 65 years of age. Some forms of syncope (i.e., vasovagal, carotid sinus hypersensitivity) are more common in younger persons than in older persons; other forms of syncope (i.e., cardiac, orthostatic, micturition related) occur more often in older than in younger individuals.

Gender: Among younger adults, women have nearly twice the rate of syncope as men; among older adults, men have the greater incidence. This likely reflects the most common etiologies in the different age groups.

Ethnicity: Not significant.

Contributing Factors: Cerebral blood flow has been reported to decline 25% with physiological aging, which is thought to be related to vascular stiffening. Hormonal regulation of extracellular volume may become impaired with age. Cardiac reflexes and baroreceptor sensitivity also may become impaired. The kidneys' ability to conserve sodium and water declines. Chronic diseases, such as heart disease, diabetes, renal insufficiency, hypertension, and chronic pulmonary disease, are predisposing conditions. Medications used to treat these chronic diseases, such as diuretics, beta blockers, vasodilators, nitrates, antiarrhythmics, and antihypertensives, also may be offenders. Alcohol is a contributing factor in vasovagal syncope. An emotional response can cause neurally mediated syncope in susceptible individuals in stressful circumstances. Dehydration and orthostatic hypotension may be factors. Situational syncope can occur when the Valsalva maneuver is produced (micturition, defecation, cough, lifting heavy objects, or after meals related to postprandial hypotension). Prolonged recumbency can contribute to micturition syncope, especially in men with prostate enlargement who awaken from a supine sleep and stand to urinate.

Signs and Symptoms: Sudden collapse with loss of motor tone, with or without injury, is the most common presentation of syncope. Self-report and eyewitness accounts of transient loss of consciousness are often unreliable—even when the eyewitness is a health-care professional. The most important first step is determining if it is true syncope. Obtain the patient's description of symptoms preceding the event and of activities that may have precipitated the event. Cardiac-related syncope may be sudden and without warning. A brief prodrome of symptoms, such as nausea, pallor, or diaphoresis, may suggest a vasovagal episode or a seizure. Many older adult patients report presyncopal episodes along with true syncopal events. Determine if any focal neurological symptoms were present (i.e., diplopia, motor and sensory symptoms). Review significant past medical history, prior history of an event, all medications being used, and alcohol intake. Ask about family history for sudden cardiac event. Rapid onset and complete recovery are key diagnostic symptoms along with loss of motor tone. If any one of these symptoms is absent, nonsyncopal causes must be considered. It is possible to have generalized jerking movements with syncope that occur after the loss of consciousness. Rapid reorientation can distinguish this presentation of syncope from a seizure disorder. The recognition of this difference can save a patient from inaccurately being diagnosed with a seizure disorder (Walsh, Hoffmayer, & Hamdan, 2015).

The physical examination should start with checking the patient for evidence of trauma, then focus on the cardiovascular and neurological components. Check blood pressure in both arms and positionally. Determine the compensatory pulse rate. Check the carotids for bruits. Carotid sinus compression sometimes is used to provoke symptoms, but this should be done cautiously and selectively and is contraindicated in patients with a bruit or a history of stroke or myocardial infarction. Examine the heart, assessing for murmurs, signs of arrhythmia, vascular disease, and left ventricular dysfunction. Check oxygen saturation rates. Perform a thorough neurological examination, paying particular attention to focal abnormalities that may suggest neurological syncope. Generalized anxiety disorders can cause hyperventilation and trigger a vasodepressor reaction; this should be evaluated during the physical examination.

Diagnostic Tests: The EKG is the most important diagnostic test with a focus on the rate and rhythm. Diagnosis of cause from the EKG is very low at 2% to 9%, but it is inexpensive and when positive for disease, critical to proper intervention. Positive findings may include sinus bradycardia, prolonged PR interval, a bundle branch block, or ventricular ectopy. Cardiac diagnostic studies may include echocardiogram (valuable in estimating ejection fraction and valvular heart disease), ambulatory EKG monitoring, and electrophysiological studies. A stress test is not indicated unless the event occurred during exercise or was accompanied by chest pain. A chest x-ray, which may reveal cardiomyopathy, is indicated in patients with new abnormal findings, patients with dyspnea, and patients without a recorded baseline. Electroencephalogram and CT scan of the head are reserved for patients with focal neurological abnormalities. Vasovagal syncope may be induced with tilt-table testing, which may be done with or without isoproterenol. It is useful in patients with syncope of unknown etiology or patients with recurrent syncope. Laboratory evaluation is generally of low yield but may include a CBC with differential if infection or anemia is

suspected. Renal function studies and electrolytes may also be considered.

Differential Diagnosis: Distinguish syncope from seizures, dizziness, drop attacks, orthostasis, postprandial hypotension, carotid sinus sensitivity, and arrhythmias. Atypical presentations include myocardial infarction and pulmonary embolism. Dizziness does not involve a loss of consciousness and is often characterized further as vertigo, light-headedness, disequilibrium, or presyncope (sensation of impending loss of consciousness). Drop attacks are sudden drops without warning and may be due to transient basilar artery insufficiency at times precipitated by head movement or neck hyperextension. Metabolic disorders (hypoglycemia, hypoxemia) cause coma/somnolence rather than syncope. Orthostatic hypotension reflects a drop in blood pressure of 20 mm Hg systolic or 10 mm Hg diastolic after changing from a lying to a standing position and is taken 1 minute after standing and again 3 minutes after standing. Consider the relationship to meals with a drop in blood pressure. Carotid sinus sensitivity and arrhythmias (atrioventricular block, sick sinus syndrome, and supraventricular arrhythmias) increase with age. Supraventricular arrhythmias rarely cause syncope, but ventricular tachyarrhythmias, especially those with decreased ejection fraction, are a major concern.

Treatment: When you identify the specific cause of syncope, you can initiate the appropriate treatment. Hospitalization may be required when there is a high risk of death, recurrence, or injury. Twenty-four-hour observation may be an option for patients with confusing presentation. Vasodepressor syncope is best addressed with volume expansion. Six factors predictive of adverse outcomes were noted in almost all research focused on risk stratification: acute coronary syndrome, heart failure, structural heart disease, abnormal EKG, anemia, or hemodynamic instability. Antiarrhythmic therapy is initiated, if indicated, and drug-induced or idiopathic orthostatic hypotension is treated appropriately. Cardiac pacing may be appropriate. Cardiac surgery is the treatment of choice for obstructive heart disease. Always correct underlying anemia and metabolic imbalance, and optimize cardiac and pulmonary status in the older adult patient. Address any age-related, disease-related, or disuse-related changes that impair the patient's compensation for hypotensive stressors, such as dehydration, deconditioning, and orthostatic hypotension. Nonpharmacological approaches to vasodepressor syncope include tilt-table training and lower extremity muscle tensing.

Follow-Up: Unknown causes precipitate a high incidence of syncopal events and in one-third of patients, syncope is a recurring event. Because recurrences may reflect lack of effective therapy or a failure to diagnose correctly, close monitoring is indicated.

Sequelae: Syncope from cardiovascular causes tends to be more dangerous. Sudden death with syncope has been attributed to arrhythmia. Patients with syncope are at risk for fall-related injury (fracture, subdural hematoma) and reduced functional capacity.

Prevention/Prophylaxis: Patients with vasovagal syncope are taught to avoid triggers and, if premonitory symptoms occur, to lie down immediately and elevate the feet higher than the chest. Adequate fluid intake is a precaution, and for selected individuals with vasovagal syncope, a higher salt intake may be advised. Support hose may help prevent reduction in central plasma volume. Patients are encouraged to avoid a sudden change in position. For syncope caused by atrial fibrillation, low-dose warfarin or aspirin therapy may be prescribed. Micturition syncope can be avoided by advising men to sit down to urinate.

Referral: Hospitalization is necessary for patients in whom you suspect an arrhythmia or myocardial infarction as the cause of the syncope and for patients who sustain significant injury during the syncopal event. Consultation may be appropriate with a cardiologist for managing cardiac syncope and with a neurologist for managing neurally mediated syncope. If it is determined that outpatient evaluation is an option, it should occur within 3 to 5 days (Puppala et al., 2015).

Education: In addition to teaching preventive strategies and the avoidance of triggering events, teach individuals with recurrent syncope safety precautions related to driving or the use of dangerous machinery. It is prudent for the health-care professional to understand the driving laws of the state relative to loss of consciousness/syncope.

CLINICAL RECOMMENDATIONS	EVIDENCE RATING	REFERENCES
A 12-lead EKG in all patients coming to the emergency department for syncope should be done.	A	Puppala, Dickinson, & Benditt, 2014 Puppala et al., 2015 Walsh, Hoffmayer, & Hamdan, 2015
Serious outcomes following syncope are unlikely if the following risk factors are absent: systolic blood pressure >90 mm Hg, shortness of breath, EKG with new changes or nonsinus rhythm, history of heart failure, hematocrit less than 30%.	A	Puppala et al., 2015
If patient is not hospitalized after presenting to the emergency department for a syncopal event, outpatient follow-up should be scheduled in 3–5 days.	C	Puppala et al., 2015

CLINICAL RECOMMENDATIONS	EVIDENCE RATING	REFERENCES
Generalized jerking movements may occur after loss of consciousness with syncope. These are unlikely to represent a seizure if patient is rapidly reoriented.	C	Walsh, Hoffmayer, & Hamdan, 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

TREMOR

Description: Tremor is the most common form of involuntary movement and is characterized by rhythmic oscillation of a body part that can be classified according to the circumstances under which it occurs. Only a small fraction of persons with tremor seek medical attention. Tremors may result from physiological or pathological processes.

Etiology: Because of the vast number of causes of tremor, etiological classification is not helpful. Terms used to describe the clinical phenomenology of tremor include rest tremors and action tremors. *Rest tremor* occurs when muscle is not activated voluntarily, and the relevant body part is fully supported against gravity; whereas *action tremor* is present with voluntary contraction of muscle. Action tremors can be subclassified further into postural, kinetic, and isometric tremor. *Postural tremor* is present while voluntarily maintaining a position against gravity. *Kinetic tremor* may occur during any form of voluntary movement of the affected body part. *Isometric tremor* occurs with muscle contraction against a rigid stationary object. The most common tremor is *enhanced physiological tremor* followed by *essential tremor* and then *parkinsonian tremor.*

Occurrence: Tremor is the most common movement disorder encountered in clinical practice. Everyone has a low-amplitude physiological tremor that can be observed when the arms are extended. Present in all muscle groups, it persists throughout the waking state. Enhanced physiological tremor is a physiological tremor that comes and goes with anxiety, caffeine, and fatigue. Estimates suggest 7 million people (or 2.2% of the U.S. population) have essential tremor. Most do not seek help until later age because of slow progression.

In 50% of cases, the disease is familial (autosomal dominant, meaning 50% of an affected individual's children have it). More than 70% of patients with Parkinson's disease have tremor as the presenting symptom. Less common are cerebellar tremor, psychogenic tremor, dystonic tremor, and tremor associated with Wilson's disease.

Age: Most studies report a significant age-associated increase in the prevalence of essential tremor. Essential tremor begins in young to middle-aged people and gradually intensifies with age. Tremors in older adults are more likely to be of the essential or parkinsonian type.

Gender: Tremor afflicts both genders equally, with perhaps slightly more frequency in men than in women.

Ethnicity: Tremor is more prevalent in Caucasians than in African Americans, and is of intermediate prevalence in Hispanics.

Contributing Factors: During times of stress, the amplitude of a physiological tremor increases. Fatigue, anxiety, hyperthyroidism, systemic illness, use of medications, drug withdrawal (especially from alcohol), use of methylxanthines, and excess caffeine intake can exaggerate tremor. Medications that can cause or exacerbate tremor include those that stimulate the sympathetic nervous system and psychoactive medications.

Signs and Symptoms: The first step in the evaluation of tremor is to categorize the tremor based on activation conditions, distribution, and frequency. Determine the duration and age of onset of symptoms, exacerbating or alleviating factors, and any family history of tremor or other neurological disorders. Include any associated symptoms, such as bradykinesia or rigidity (suggesting Parkinson's disease) or ataxia and nystagmus (suggesting cerebellar disease). The patient's medication history, any exposure to toxins, and the presence of illness should be noted. In assessment of the impact of tremor on patients' lives, functional disability in performing ADLs and the patient's subjective assessment of his or her quality of life are useful. History is important, but the diagnosis is based on clinical physical examination findings. Tremor may occur in various body parts, such as the hands, head, facial structures (chin, tongue, lips, and ears), vocal cords, trunk, and legs. Of all tremors, 94% occur in the hands, either unilaterally or bilaterally.

On physical examination it is important to conduct a thorough tremor-focused neurological examination: muscle tone is checked throughout the body, cranial structures (including the mouth and jaw) are examined at rest and in action, and the tongue is observed during rest and protrusion. To distinguish properly between resting and action tremors, patients should be evaluated while supine and when seated with the arms fully supported. (If patients are in a position that does not provide complete support, certain muscles may be active against gravity, producing a tremor that may be classified improperly as a resting tremor.) Once rest tremor is ruled out, the patient performs other maneuvers.

The upper extremities are examined in an outstretched position with the hands supine (palms up), sideways (semi-prone), and then prone (palms down). The semi-prone position enhances essential tremor, whereas the supine position

inhibits it. In the wing position (i.e., with apposition of the index fingers close to each other but not touching), proximal tremor may be identified. Goal-directed activities are performed, such as finger-to-nose, heel-to-shin, and toe-to-finger movements. The patient is asked to recite a standard paragraph and enunciate a sustained vowel. Handwriting samples are obtained (e.g., script, numbers, Archimedes spirals). Gait is evaluated for shuffling and unsteadiness, and Romberg (station) and balance testing are conducted. Careful evaluation is performed for signs associated with tremor syndromes. Bradykinesia and postural abnormalities are evaluated by observing difficulty rising from a seated position, decreased arm swing, and masked facies. Patients with essential tremor typically have handwriting that is shaky and large, whereas the handwriting of patients with Parkinson's disease initially may be of normal size and progressively become smaller (micrographia). Archimedes spirals drawn by essential tremor patients tend to illustrate natural fluctuations in tremor magnitude.

Diagnostic Tests: No specific tests are routinely ordered for tremors. Electromyography is used to subdivide tremors according to their rate and their relationship to posture of limbs and volitional movement. Tremor frequency usually is categorized as low frequency (less than 4 Hz), medium frequency (4 to 6 Hz), and high frequency (greater than 6 Hz).

The diagnosis of tremor is primarily clinical; however, laboratory testing may be necessary to exclude certain conditions that may be associated with tremor, such as metabolic disturbances, including hyperthyroidism (e.g., through thyroid function tests) and Wilson's disease. Brain imaging may be indicated for select patients, particularly patients with tremor that is unilateral, of sudden onset, or associated with atypical clinical features. For difficult cases, single-photon emission computed tomography (SPECT) to visualize the integrity of dopaminergic pathways may be useful in diagnosing Parkinson's disease.

Differential Diagnosis: The differential diagnosis, in general practice, is almost always between Parkinson's disease and essential tremor.

Physiological Tremor: A normal phenomenon, physiological tremor occurs in all contracting muscle groups. Although seldom visible to the naked eye, physiological tremor often may be detected when the fingers are firmly outstretched with a piece of paper placed over the hands.

Enhanced Physiological Tremor (or an Intensification of Physiological Tremor to Detectable Levels): Physiological tremor may be enhanced under conditions of stress, anxiety, fatigue, exercise, cold, hunger, stimulant use, alcohol withdrawal, or metabolic disturbances such as hypoglycemia or hyperthyroidism. Although the tremor is typically low in amplitude and high in frequency (8 to 12 Hz), it may be clinically indistinguishable from essential tremor.

Essential Tremor (4 to 12 Hz): Essential tremor is a persistent postural and kinetic tremor that predominantly affects the hands and forearms. Classically, to show the tremor, the patient is asked to extend the arms in front of the body. The legs are affected less often. Although less frequently involved, the presence of tremor in the head and/or voice is a strong indication of essential tremor and is especially useful in differentiating the syndrome from Parkinson's disease. Head tremor, which is also postural, disappears when the head is supported. Listening to the patient speak or having the patient hold a musical note as long as possible may reveal a quivering intonation. A resting component is present only rarely and typically occurs in the most advanced cases.

Parkinsonian Tremor Syndromes (4 to 6 Hz): Parkinsonian tremor syndromes involve resting tremor that is often asymmetrical. Tremor may be observed when muscles are relaxed, such as when the hands are resting on the lap, and may affect hands, feet, mandible, and lips. Tremor disappears during sleep. Typical is an alternating tremor of the thumb against the index finger—*pill-rolling tremor.* Although rest tremor is a diagnostic criterion for Parkinson's disease, other forms of tremor also may be present.

Treatment: Tremor should be treated if it causes disability. First-line treatment for tremor is oral medication. Beta blockers, anticholinergic medication, and levodopa are useful modalities for resting tremor. Kinetic tremor may respond to beta blockers, primidone, anticholinergics, and alcohol. When there is a lack of response to medical treatment or when tremor results in severe disability, a patient may be considered for neurosurgery. Specific treatments for the most common causes are noted next.

Physiological Tremor: Usually no treatment is required for physiological tremor. When exaggerated, however, it may interfere with activities requiring extreme precision. Identify and remove precipitating causes and contributing causes. If the precipitating cause cannot be removed, propranolol may be effective.

Essential Tremor: Varying degrees of control in essential tremor have been obtained with the beta blocker propranolol and the anticonvulsant agent primidone. Either agent may be considered an appropriate first-line therapy for the symptomatic management of essential tremor. When appropriate, these agents may be administered in combination with benzodiazepines, such as lorazepam or clonazepam. If the medication is of no benefit at a dose that causes adverse effects, dose levels should be tapered down gradually and eventually discontinued. If a medication is documented to be beneficial, it may be continued at the regulated doses, and the next medication may be added to the drug regimen. If the response to a drug is adequate and the dose is well tolerated, you may continue to monitor tolerance and possibly increase the dose.

Physical and psychological measures may be helpful in managing mild tremor. Physical measures may include the application of weights to affected limbs to decrease tremor amplitude. Some patients have experienced benefits with biofeedback, relaxation methods, and other behavioral techniques through alleviation of anxiety or stress that may exacerbate tremor. Alcohol consumption may lead to transient improvement for many with essential tremor. The potential risk of alcohol dependence and abuse among essential tremor patients who drink alcohol to control symptoms is controversial. Alcohol has no impact on the tremor of Parkinson's disease.

Parkinsonian Tremor: The tremor of Parkinson's disease results from a loss of striatal dopamine, and this is the rationale for treatment with either the dopamine precursor levodopa

or dopamine receptor agonists. Dopaminergic and anticholinergic agents are equally effective, but dopaminergic substances additionally improve other parkinsonian signs, and the potential side effects of anticholinergic medications make these drugs undesirable in the older adult. The combination of levodopa and carbidopa reduces levodopa-induced nausea; a typical starting dose is one tablet of Sinemet 25/100 three times daily. Levodopa-carbidopa intestinal gel (brand name Duopa) is a combination of levodopa and carbidopa that is dosed for 16 hours during wakefulness. It is administered via PEG-J tube supplied by an external box. This route of dosing decreases tremor by reducing plasma fluctuations that patients on oral levodopa and carbidopa might experience.

Patients with severe tremors that are resistant to pharmacotherapy may benefit from ablative surgery and/or deep brain stimulation. For those essential tremor patients who fail medication treatment, transcranial magnetic resonance-guided focused ultrasound thalamotomy (product name ExAblate Neuro) has been shown to be successful in the treatment of essential tremor. This procedure is noninvasive.

Follow-Up: Patients should be evaluated for therapeutic effects and side effects within 1 week of starting treatment. Annual monitoring for weight loss, depression, and decline in functional status is necessary.

Sequelae: Functional disabilities may occur in ADLs, including compromised eating, drinking, and preparing food. Decreased caloric intake and weight loss may be observed. Ambulation, especially on stairs, may be hazardous. Withdrawal from social situations may occur, and depression is common.

Prevention/Prophylaxis: Reduce factors that can exacerbate the tremor. Continue medication regimen.

Referral: A neurologist should be consulted for cerebellar tremors, mixed tremors, or Parkinsonian tremor, or when a focal neurological deficit is identified. An ophthalmologist should be consulted when Wilson's disease is suspected. A mental health provider or psychiatrist should be consulted when a hysterical tremor is suspected. Physical therapy or occupational therapy may be helpful in advanced or disabling cases.

Education: Some patients, particularly patients with severe, disabling tremor, may limit their contacts. Patients must be encouraged to learn as much as they can about their disease to help them cope better with the condition's progression. When a diagnosis has been established, the natural history of the condition should be explained to patients. It also may be appropriate to recommend counseling. Use of appropriate coping strategies may reduce stress substantially, preventing possible augmentation of tremor owing to anxiety. Referral to appropriate patient support organizations is helpful for most patients.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The diagnosis of tremor is based on clinical information from the history and physical examination.	C	Deuschl et al., 1998 Sharma & Pandey, 2016
Propranolol and primidone are first-line treatments for essential tremor.	A	Sharma & Pandey, 2016
Tremor amplitude worsens over time if not treated.	C	Sharma & Pandey, 2016
Levodopa-carbidopa intestinal gel improves resting tremors.	B	Fernandez et al., 2016
Transcranial magnetic resonance-guided focused ultrasound thalamotomy successfully treats essential tremor.	B	U.S. FDA, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

URINARY INCONTINENCE

Description: Urinary incontinence (UI) is an involuntary loss of urine (Gibson & Wagg, 2014). It is common in the older individual but often underreported. It can be distressing and affect a person's overall quality of life (Berardelli, 2013). Acute UI is generally a result of illness or the effects of medications and is self-limiting when the cause is determined and addressed. Chronic UI has different forms, including stress incontinence, urge incontinence, overflow incontinence, and functional incontinence. Many older women manifest a combination of urge and stress symptoms resulting in mixed incontinence. Tables 5-10 and 5-11 describe the various types of UI and management strategies.

Etiology: The causes and management of UI are multifactorial, depending on the type of incontinence and also the severity and impact on quality of life for the individual.

TABLE 5-10 Types of Urinary Incontinence

TYPE	SYMPTOMS	TREATMENT OPTIONS
Stress incontinence	Urine leakage associated with increased abdominal pressure from laughing, sneezing, coughing, climbing stairs, or other physical stressors increasing abdominal pressure	Lifestyle interventions Behavioral therapies
Urge incontinence	Urine leakage associated by or immediately preceded by the feeling of an urgent need to void	Lifestyle interventions Behavioral therapies Consider trial of antimuscarinic medications
Mixed incontinence	A combination of stress and urge incontinence, marked by involuntary leakage associated with urgency and also with exertion, effort, sneezing, or coughing	See Management of Stress Incontinence and Urge Incontinence
Overflow incontinence	Urine leakage when the bladder is over-distended and may result in incomplete bladder emptying Symptoms can present as constant dribbling, frequency, hesitation when initiating urination, and nocturia Often associated with bladder outlet obstruction, such as benign prostatic hypertrophy in men and pelvic organ prolapse in women	Neurogenic bladder can also present as overflow UI Assess for the cause such as medication usage and fluid intake Double voiding to empty residual urine from bladder
Functional incontinence	The inability to hold urine due to reasons other than neurological and lower urinary tract dysfunction including delirium, psychiatric disorders, UTI, impaired mobility	Treat underlying cause

Source: Vasavada (2016); Luckacz (2016); Wagg et al. (2015).

TABLE 5-11 Lifestyle Measures and Behavioral Therapies for UI Management

Lifestyle measures	Weight loss Fluid management-restricting fluids Avoiding alcohol, caffeine, and carbonated beverages Avoiding constipation
Behavioral therapies	Pelvic floor exercises including Kegel exercises: • Identify the muscle that starts and stops urination through attempts to start and stop urinating • Contract the identified muscle for approximately 8–10 seconds, and then relax the muscles • Perform 8 to 12 contractions • Repeat 3–4 sets of repetitions daily Bladder training: • Prompted toileting: Ask the individual to void more often • Habit training: Identifying the toileting pattern including UI episodes, schedule toileting routine to prevent UI episodes • Timed toileting: Scheduled toileting at fixed intervals • Combined toileting and exercise therapy: Incorporates pelvic floor strengthening exercises and bladder training interventions • Double voiding: Empty bladder, relax for 1–2 minutes, then lean forward and/or press on bladder and attempt to void again, emptying the remaining urine from the bladder

Source: Luckanz (2016); Wagg et al. (2015).

Anatomical changes, factors related to the individual's medical history, lifestyle, and acute and chronic illnesses, in addition to medications, can result in incontinence that can be either reversible or a permanent condition. Cognitive as well as chronic mental illness, depression, and functional barriers to continence can also affect an individual's ability to maintain urinary continence.

Occurrence: Urinary incontinence is often underreported, with the prevalence of UI being more common in women and increasing with age. Greater than 60% of older individuals experience UI, with the highest occurrence in women (Gibson et al., 2014; da Silva et al., 2012). In the long-term care setting, this rate increases to an average of 85% of the population and is the cause of approximately one in ten nursing home admissions (Vasavada, 2016). UI is not a cause, but it is associated with frailty and functional decline in the older individual (Gibson et al., 2014; da Silva et al., 2012; Omli et al., 2013; Wagg et al., 2015).

Age: Urinary incontinence affects all age groups, with the highest prevalence in older adults who are institutionalized, have cognitive impairment, and are physically frail with functional limitations. Despite popular belief, lifestyle measures and behavioral therapies can be effective interventions when used for individuals, even in individuals with cognitive impairment.

Gender: UI is twice as prevalent in women as in men, and the incidence increases with age, with institutionalized individuals, and those who have at least one deficit in ADLs (Wagg et al., 2015; da Silva et al., 2012). Women are at higher risk for stress incontinence; however, overflow incontinence is more prevalent in men as a result of hyperplasia of the prostate gland.

Ethnicity: Ethnicity is not significant with regard to incontinence in general; however, some data indicate a higher prevalence of stress incontinence among Caucasian women, whereas African American women were found to be at higher risk for urge incontinence.

Contributing Factors: The many contributing factors for UI include pelvic muscle weakness, multiparity, estrogen depletion, pelvic organ prolapse, diabetes, stroke, multiple sclerosis, Parkinson's disease, spinal cord injury, benign prostatic hyperplasia, UTI, fecal impaction, poor fluid intake or excessive fluid intake, smoking, cognitive impairment, depression, immobility or impaired mobility, environmental barriers,

impaired dexterity, visual impairment, obesity, and high-impact physical activities. Incontinence can be a side effect of many medications, including cholinergics, anticholinergics, diuretics, antispasmodics, opiates, hypnotics, calcium channel blockers, ACE inhibitors, alcohol, and caffeine.

Signs and Symptoms: In screening for UI it is important to evaluate whether symptoms are transient or persistent in nature. A thorough history should include:

- The severity and quantity of urine lost, including use of pads or adult briefs
- Onset and duration of the problem and whether the UI is worsening
- Triggering factors or events, including sneezing, coughing, laughing, or during activity
- Constant versus intermittent urine loss
- Associated frequency, urgency, dysuria, pain with a full bladder
- History of UTIs
- Association with fecal incontinence or pelvic organ prolapse
- Additional medical problems, including current or past cancers of the pelvic organs, congestive heart failure, COPD, cognitive impairment, and connective tissue disorders
- Obstetrical history, including difficult deliveries, multiparity, and large babies
- History of pelvic surgeries or other urological procedures, including use of indwelling urinary catheters
- History of spinal or CNS problems and/or surgeries
- Lifestyle issues, including use of tobacco, alcohol, or caffeine; occupational or recreational factors causing severe or repetitive increases in intra-abdominal pressure
- Medications, including use of the following medications that have been associated with UI: cholinergic or anticholinergic agents, alpha blockers, OTC allergy medications, estrogen replacement, beta-mimetics, sedatives, muscle relaxants, diuretics, and ACE inhibitors
- Effect of these symptoms on ADLs, socialization, and relationships, including sexual activity (Vasavada, 2016)

Considering that approximately 10% of nursing home admissions are a result of UI, discussion with patients and caregivers before long-term care placement about symptoms has the potential to prolong the individual's ability to be maintained at home for an extended period of time.

Physical examination should include functional assessment with special attention to mobility, including ability to ambulate to the toilet and dexterity, including the person's ability to remove necessary clothing in time to use the toilet. Vital signs should be completed, looking for the presence of an elevated temperature. Respiratory rate and the presence of dyspnea on exertion may affect continence due to limited stamina to complete necessary tasks for maintaining continence. Mental status, including cognition and evidence of depression, should be assessed. The abdomen should be examined for clues such as bladder distention, pelvic masses, inguinal lymphadenopathy, or tenderness in the suprapubic region. Bladder distention can be found in overflow incontinence secondary to obstruction. In women, malignancy, uterine fibroids, or organ prolapse in the pelvic region creates pressure on the bladder seen in urge, stress, or mixed incontinence. A vaginal examination may also reveal poor perineal hygiene, skin breakdown from urine soaking, or redness and thinning of tissue typical of atrophic changes. Prolapse of genitourinary structures or the rectum may also be seen.

To assess for pelvic floor muscle strength and relaxation, instruct the patient to bear down as though having a bowel movement, then tighten or squeeze by pulling up with the pelvic floor muscles. In patients with pelvic floor relaxation, you can see the inability to contract or weak contractions, and feel a lack of muscle tone when testing during the vaginal examination. Have the patient cough and determine if leakage occurs. Urethral discharge in male patients should also be assessed. Positive neurological findings in the perineal area include hypersensation, hyposensation, or absence of the bulbocavernosus (anal wink) reflex. A rectal examination may reveal fecal impaction, rectal prolapse, hemorrhoids, masses, or, in men, prostatic enlargement. Whenever possible, the examiner should observe the patient voiding, having the patient void into a measurable receptacle. This should be followed by evaluation of a post-void residual (PVR) urine by catheterization or ultrasound of the bladder. Studies are inconclusive with respect to the amount of PVR that is significant, with values ranging from greater than 50 mL to greater than 200 mL.

Diagnostic Tests: Initial evaluation for UI should include a urinalysis to rule out infection and renal abnormalities. This has great significance when UI has an acute onset. The presence of nitrites and leukocyte esterase in the urinalysis is usually indicative of an infectious process. When a UTI is suspected, a culture and sensitivity should be ordered to ensure appropriate antimicrobial therapy. Hematuria may indicate a number of differential diagnoses, including infection, obstruction, kidney stones, or malignancy. Proteinuria is revealing for renal disease and is often associated with poorly controlled diabetes. When renal, metabolic, or obstructive causes of urinary tract dysfunction are suspected, a basic metabolic panel is recommended to evaluate elevated BUN and creatinine levels, and calculate estimated glomerular filtration rate (eGFR) to evaluate for renal disease. Measurement of PVR may reveal incomplete bladder emptying.

When symptoms and basic noninvasive evaluation does not clearly indicate the type of urinary tract dysfunction, and the individual indicates that UI is problematic enough to warrant further treatment, urodynamic testing and referral to a urologist or urogynecologist is indicated. Urodynamic testing evaluates all stages of lower urinary tract function, including filling and storage, as well as bladder outlet abnormalities. Subjective reporting by the patient of sensation such as urgency can also be evaluated with respect to bladder wall compliance, detrusor overactivity, pressure, and flow measurements during testing. Cystoscopy is indicated for evaluation of hematuria to visualize the bladder wall when cancer is suspected, and also when stricture or prostate enlargement is suspected with symptoms of urinary retention.

Differential Diagnosis: UI is a symptom, not a diagnosis. The two mnemonics DRIP and DIAPERS often are used to differentiate transient (acute) from persistent urinary incontinence:

Mnemonic: **DRIP**
- **D**elirium
- **R**estricted mobility
- **I**nfection
- **P**harmaceuticals, polyuria

Mnemonic: **DIAPERS**
- **D**elirium
- **I**nfection, impaction, inflammation
- **A**trophic vaginitis
- **P**sychological, pharmaceuticals, psychotropics
- **E**ndocrine problem
- **R**estricted mobility
- **S**tool impaction

Treatment: Management of UI will depend on the type of incontinence; the patient's preference with regard to his or her perception of how UI affects his or her lifestyle; and the patient's physical condition to tolerate treatments, surgical procedures, and risk for adverse effects, as well as complications due to medications or treatment regimens. For transient UI, treating, eliminating, or modifying the cause usually alleviates the symptom. Delirium or a mild elevation in temperature from the patient's baseline, rather than a fever, may be the first indication of a UTI in the older adult. Appropriate antimicrobial therapy will generally resolve the UI as a result of the infection.

Lifestyle management using behavioral therapies and pelvic muscle exercises or Kegel exercises can be an effective treatment for stress and urge incontinence, performed alone or in combination with biofeedback, to help strengthen periurethral muscles. Toileting programs (see Table 5-11) can be effective interventions for decreasing the impact of or controlling UI, even in patients with cognitive impairment. Nocturia can be a very annoying problem and cause significant issues where quality of life is concerned. Control of comorbidities and symptoms such as edema, in addition to limiting fluids before bedtime, can be helpful to improve sleep hygiene.

Pharmacological treatment for urge incontinence with anticholinergic drugs is effective for symptoms; however, they are not without side effects, especially constipation, blurred vision, and cognitive changes in older adults. These medications, such as tolterodine (Detrol) and oxybutynin (Ditropan), seem to be better tolerated in long-acting and transdermal forms. Drugs that are more selective for muscarinic-3 receptors, such as darifenacin (Enablex), solifenacin (Vesicare), and trospium (Sanctura), which do not appear to cross the blood-brain barrier, appear to have a better side effect profile, and are better tolerated in the older adult population. In the STAR trial, with 70% of subjects more than 65 years old, efficacy and tolerability of solifenacin was found to have better outcomes with regard to incontinence than long-acting tolterodine.

Comorbidities such as narrow-angle glaucoma and potential interactions with other medications are always a concern in the geriatric population and should be considered before prescribing a drug regimen for UI. Topical estrogen therapy (one-quarter applicator nightly) for 2 weeks, then three times a week ongoing, can be an effective remedy for UI as a result of vaginal atrophy. Studies have also indicated that the antidepressant duloxetine can be an effective treatment for stress UI, with approximately a 50% reduction in incontinence episodes and improved quality of life; however, nausea as a significant adverse effect may reduce compliance with this treatment regimen.

For patients with overflow incontinence secondary to prostatic hypertrophy, 5-alpha-reductase inhibitors, finasteride (Proscar), or dutasteride (Avodart) are effective to reduce prostate volume by preventing the conversion of testosterone to dihydrotestosterone. These drugs should not be handled by women of childbearing age, because they can be harmful to the development of a male fetus. Selective alpha-adrenergic antagonists such as tamsulosin (Flomax) relax smooth muscle to reduce urethral resistance and improve urine flow. Nonselective alpha-1 blockers such as doxazosin (Cardura) and terazosin (Hytrin) are effective for UI and can also be used in patients who also require antihypertensive therapy. The provider should consider, however, the cardiovascular side effects due to their nonselectivity, because orthostatic hypotension is an adverse reaction that can have serious consequences, such as increased fall risk and injuries, in the older adult patient. Post-prostatectomy incontinence can be treated long after surgery with behavioral therapy such as pelvic floor muscle training. Studies have found an average of 55% reduction in incontinence using these strategies.

Other treatments for UI include bulking agents such as collagen for stress incontinence, botulinum toxin, sacral nerve stimulation for urge incontinence, and surgical interventions for stress incontinence in women and for prostatic hypertrophy in men. Referral to urology or urogynecology for surgical intervention is appropriate if the patient is inclined to consider surgical intervention and is physically able to tolerate the procedure. Surgical management for pelvic organ prolapse to improve organ support, such as retropubic suspension, sling procedures, and vaginal mesh, are viable options; however, patient education with regard to the high failure rate (5% to 12%) should be discussed when considering these options. In 2008, the FDA warned of potentially serious complications associated with the use of surgical mesh, and in 2016 they changed the classification from moderate risk to high risk when used for pelvic organ prolapse, a frequent cause of stress incontinence. When surgical treatment for UI as a result of pelvic organ prolapse is contraindicated, a pessary is a viable option. In addition to incontinence pads and briefs, pessaries, when properly fitted within the vaginal vault to correct organ prolapse and reduce stress UI, can often be managed by the patient or cared for by a qualified nurse at regular intervals. Nonsurgical management of persistent urinary overflow incontinence includes clean intermittent catheterization. Although the Centers for Medicare and Medicaid Services now reimburses for a single-use sterile catheter, there is no evidence to support a reduction in UTIs with the use of sterile versus clean urinary catheter.

Follow-Up: Follow-up visits will depend on the acuity and severity of symptoms. Treatment of infection and symptoms related to urinary retention requires closer and more frequent follow-up until the problem is resolved due to renal complications that can result. Initially, medication management should be attempted but monitored closely for adverse effects, especially with anticholinergic medications. Side effects of these medications can result in significant complications for the geriatric patient, such as blurred vision, dizziness, and somnolence, as well as contribute to fall risk. Long-acting medications appear to have a lower risk profile, but individual

monitoring is necessary to ensure patient safety. Additionally, dementia exacerbation can occur with the use of anticholinergic medications, resulting in hallucinations, psychosis, or changes in behavior that can lead to injury to the individual or others. Constipation, a common adverse effect of anticholinergics, can exacerbate UI by causing a fecal impaction. Polypharmacy issues also need to be monitored closely with any medication change, requiring close follow-up when starting a new drug regimen.

Sequelae: Possible complications include UTI, hydronephrosis (with overflow or obstruction), renal failure, adverse drug events, or failure of behavioral therapy. Skin breakdown is a significant complication with persistent UI. Urosepsis can occur with unrecognized UTIs. Falls due to UI result from environmental factors or as a side effect of medication management. Social isolation may be a result of uncontrolled UI having a significant impact on quality of life and may lead to depression.

Prevention/Prophylaxis: Ways to help prevent incontinence include:

- Early identification of acute or transient UI
- Teaching patients and family members/caregivers that UI is not a normal part of the aging process and that treatment options are available
- Referral to appropriate specialists for treatment options
- Regular rectal examination in men to detect and treat early prostatic hypertrophy
- Recognition of polypharmacy and adverse reactions when initiating new medications in the geriatric population

Referral: Referral to urogynecology for the female patient to explore treatment options is appropriate if behavioral interventions and medication management are unsuccessful. It is also appropriate to refer to a specialty practice if the diagnosis is uncertain. Refer men with overflow incontinence for urological evaluation urgently if PVR urine volume is significant, to prevent renal complications, and for surgical intervention, if indicated. Certified continence nurse specialists may be an appropriate referral for behavioral therapies, such as biofeedback or for electrical stimulation treatments to improve continence. Further work-up and referral to urology would be indicated if abnormalities in the urinalysis do not resolve after treatment for infection, such as persistent hematuria or proteinuria, and if the patient has a past medical history that includes surgeries for genitourinary diagnoses, or a history of pelvic cancer with or without radiation therapy. Patients with neurogenic bladder as a result of injury or a chronic neurological condition should be followed by a neurologist in collaboration with the primary care provider.

Education: Teach patients, family, caregivers, and healthcare providers, as well as the community, that UI is *not* a normal part of aging and it is a treatable medical problem in many individuals. Behavioral therapy can be provided in the primary care setting, such as Kegel exercises, as well as self-catheterization techniques. Written instructions given to the patient with possible adverse drug effects when starting a new medication can prevent serious consequences of drug effects/complications. Teach patients, especially women and men with bladder outlet obstruction, the signs and symptoms of UTI, including delirium as an indication of acute illness, and that in an older adult a fever may not occur even when there is a UTI. Dietary issues such as adequate fluid intake, limiting bladder irritants such as caffeine and alcohol, and prevention of constipation to avoid fecal impaction can help avoid bladder symptoms and infections. The importance of good perineal hygiene, especially for women and patients who do self-catheterization, should be emphasized. When incontinence cannot be completely avoided, attention to skin care and instruction in the use of skin barriers such as zinc oxide or dimethicone-based products should be taught to the patient or caregivers to prevent skin breakdown.

DIAGNOSIS	CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Urinary incontinence	Combination behavioral therapy and medication management	A	da Silva et al., 2012 Omli et al., 2013 Gibson & Wagg, 2014 Vasavada, 2016 Wagg et al., 2015
Stress urinary incontinence	Alpha-adrenergic medications, serotonin-norepinephrine reuptake inhibitors	A	Gibson & Wagg, 2014 Wagg et al., 2015
Stress urinary incontinence	Surgical procedures	A	Omli et al., 2013
Urge urinary incontinence	Anticholinergic medications (antimuscarinic agents)	A	Gibson & Wagg, 2014 Wagg et al., 2015
Overflow urinary incontinence	Treatment of underlying cause of bladder outlet obstruction, urinary catheterization	C	Lucacz, 2016

Continued

DIAGNOSIS	CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Functional urinary incontinence	Scheduled toileting	A	Gibson & Wagg, 2014 Wagg et al., 2015
Mixed urinary incontinence	Anticholinergic medications (antimuscarinic agents)	A	Gibson & Wagg, 2014 Wagg et al., 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

WANDERING

Description: Wandering in older individuals with disorientation or cognitive impairment is characterized as locomotion (either ambulation or propelling in a wheelchair) in a seemingly aimless pattern. The behavior is often described as having observable patterns such as lapping, pacing, or random movements (Cipriani, Lucetti, & Danti, 2014). The behavior may be frequent, repetitive, temporally disordered, and possibly spatially disordered in nature.

Etiology: Although poorly understood, there are many hypotheses to the causes of wandering behaviors. A medical hypothesis suggested right parietal dysfunction, which leads to spatial perception and memory lapses resulting in these types of behaviors. Another theory is the individual has unmet needs or environmental triggers leading to wandering behaviors (Cipriani et al., 2014; Bright Foundation, 2016) (see Table 5-12).

Wandering behaviors occur in individuals who experience looseness of thoughts, most noticeably in individuals experiencing confusion—either temporary in states of delirium or in individuals with a dementia, most commonly during the middle to later stages of the dementia process. Delirium is an extreme confusion, an acute episode of fluctuating attention, worsening of cognition, and change in level of consciousness, which frequently results in agitated and wandering behaviors (Huang, 2016). Most delirium is reversible but occurs more often in individuals with dementia. Common causes of delirium include illness, medications, and environmental factors (Huang, 2016) (see Table 5-13).

TABLE 5-12 Potential Causes of Wandering Behaviors

Unmet personal needs	• Internal discomfort: need for toileting, hunger, thirst • Loneliness • Perceived threat to the individual's safety • Past history of walking behaviors
Environmental triggers	• Noisy environment • Increased visual distractors • Visualizing other individuals leaving • Shadowing a caregiver

Source: Cipriani et al. (2014); Bright Foundation (2016).

The many causes of dementia include Alzheimer's disease, vascular disease, Lewy Body dementia, Parkinson's disease, alcoholic encephalopathy, normal pressure hydrocephalus, and frontotemporal dementia (also referred to as Pick's disease) (Huang, 2016).

Occurrence: Dementia was estimated to affect 44 million older people worldwide in 2013, with the number expected to explode to 74 million by the year 2030 and 135 million by 2050 (Kales, Gitlin, & Lyketsos, 2015). Wandering behaviors occur in one out of every five individuals with dementia at some point during their illness (Cipriani et al., 2014).

TABLE 5-13 Common Causes of Delirium in the Older Individual

Illness	• UTI • Upper respiratory infections (URIs) including pneumonia • Sepsis • Severe constipation as well as intestinal obstruction • Meningitis • Dehydration • Electrolytes imbalances, especially low sodium and low calcium levels • Severe malnutrition • Hypoxia • Terminal illness
Potential medications	• Medications with anticholinergic properties • Certain antipsychotics • Pain medications, especially when the individual is toxic *See the Beer's list of potentially inappropriate medications frequently prescribed in the older patient
Overstimulation	• Loud noises or multiple conversations • Bright or blinking lights • Large number of people • Change in routine of an individual with cognitive impairment • Overly tired individual with a diagnosis of dementia

Source: AGS (2016); Bright Foundation (2016).

Age: Wanderers do not differ in age (although some studies suggested it occurs more frequently in younger elders) or gender (although some reports suggested it occurs more frequently in males) (Cipriani et al., 2014).

Gender: Women are diagnosed more often with Alzheimer's disease. Women generally live longer than men and therefore are more likely to live to the age when Alzheimer's disease is more common.

Ethnicity: Prevalence related to ethnicity showed frequency of occurrence to be 58% in Caucasians, 67% in African Americans, and 63% in Hispanics.

Contributing Factors: Wandering behaviors occur in over half of individuals with a diagnosis of dementia in the moderate to late stage of this cognitive impairment process. The greatest percentage of wandering occurrence has been reported in seniors residing in a nursing home setting (Cipriani et al., 2014). The most frequent occurrence is between 5:00 pm and 7:00 pm, but it has been reported as early as 2:00 pm and late into the night, which is often referred to as "sundowning" (Cipriani et al., 2014; Bright Foundation, 2016).

Signs and Symptoms: Wandering may be accompanied with agitation and anxiety, causing the individual to be difficult to redirect. Individuals who are anxious and those who are delusional appear to exhibit wandering behaviors more frequently. The consequences of wandering may include falls, exhaustion, malnutrition, anxiety and aggression, elopement, and becoming lost from the individual's home environment (Lin, Zhang, Chen, Ni, & Zhou, 2014; Cipriani et al., 2014; Tilly, 2015; Nagamatsu, Kam, Liu-Ambrose, Chan, & Handy, 2013).

Diagnostic Tests: In an older person who seems to wander aimlessly or become confused or lost, a full medical evaluation should be conducted, including an evaluation for dementia, particularly Alzheimer's disease. A work-up includes a review of the individual's medications; diet, nutrition, and use of alcohol; as well as medications (prescription and nonprescription/street drugs); vital signs and pain level; laboratory tests (blood and urine); and a thorough physical examination including an in-depth geriatric neurological examination. Although no singular test is available for dementia, multiple cognitive assessments are recommended, including the MMSE, the Montreal Cognitive Assessment, the St. Louis University Mental Status Exam, Mini-Cog test, Clock Drawing test, and Memory Impairment Screening. The Mini-Cog test is a short screening test that incorporates a three-item recall within 5 minutes and a clock drawing test. The MMSE, a 30-item test, had been the most widely used test to assess an individual's level of cognitive functioning. Wandering behaviors have been shown to be common in individuals who score a 13 or less on the MMSE (Cipriani et al., 2014).

Treatment: A person-centered approach to assessment, planning, and delivery of care is the key to managing wandering behaviors (Tilly, 2015). When possible, attempt to identify why the person is wandering, and when developing a plan of care, involve the person with dementia in decision making. Identify triggers to the behavior and modify the individual's routine to minimize the triggers; decrease distractions, especially in the afternoon and evening hours or when the person is tired; and do not vary the day-to-day routine. Attempt to engage the individual in nondemanding activities during the time of day the wandering behaviors most frequently occur. Nonpharmacological interventions should be attempted, but when the individual is severely agitated, aggressive, exhausted, or otherwise at risk for harm to self or others, and distraction and redirection are unsuccessful, pharmacological intervention may become necessary.

Sequelae: Consequences of wandering behaviors include accidents, getting lost, fatigue, sleep disturbances, social isolation, injury, and death (Cipriani et al., 2014). Falling with hip fractures and head injuries are common consequences in these individuals. Malnutrition can also occur as a result of an inability to sit for a meal or excessively wandering, which increases caloric requirements and leads to weight loss. Wandering behaviors often lead to early institutionalization in a nursing home when families are unable to manage the individual in the community setting. Disruptive activities are associated with wandering such as agitation, screaming, physical aggression, and disturbed nighttime sleep.

Prevention/Prophylaxis: Care is aimed at balancing a person's need for autonomy with the need to minimize risks. The goal is to provide a pleasant and safe environment where an individual will not wander away, fall, or become exhausted. Wandering is not always problematic if the person is able to ambulate without falling, the behavior does not lead to aggression, or the person does not wander away and become lost. To maximize safety, the living environment needs to be surveyed and modifications made, when necessary, and the area is familiar (minimize changes) and uncluttered. Assure ample lighting, maintain clutter-free walkways, and keep harmful objects out of reach from the confused individual. Other considerations may include putting locks on cabinets and ovens, unplugging dangerous appliances when necessary, removing wall hanging mirrors (the confused may misinterpret the reflection as another person in the room), and providing exits with safety mechanisms. Wandering behavior can be diminished with the installation of simple childproof door knob covers, possibly more complex door locks, or even in-house alarms or bells.

If an individual wanders, identify where the person usually wanders to, what activity the person is usually engaged in when wandering occurs, and what time of day the behavior usually occurs. Identification bracelets or necklaces may help minimize the hazardous consequences if wandering off does occur and the individual becomes lost. Also include identification in clothing and keep a recent photograph of the individual when assistance is needed to locate the person. When wandering occurs, caregivers should approach the individual slowly, speaking softly and in a nonthreatening manner. Act in a nonjudgmental, matter-of-fact manner. Validate the person's feeling with positive, open statements such as "You look concerned" or "What can I do to help?" Distract the individual by refocusing the person's attention on something different, such as looking at old pictures or discussing the weather. Redirect the person in a reassuring manner by saying, "I know you are concerned; let's sit and talk and have a cup of coffee." Possibly suggest going for a walk in the direction you wish the person to go instead of the individual leaving that environment. Avoid asking the person to explain the behavior. In summary, validate, redirect, distract, and redirect the

wandering or confused person to abort the behavior (Jong, 2016).

Referral: Caregivers need to determine the abilities of the wandering individual and create an environment that provides for autonomy as much as possible and ensures safety. Referrals may be made to geriatric care specialists for evaluation and management, home health agencies for assistance in the home setting, and community resources for support groups and available educational resources. When necessary, consideration for a more structured living arrangement may be advised when safety becomes a major concern. Options include sitters in the home setting, adult day cares, assisted living facilities, and nursing homes. In extreme situations, when the elder is at risk for harming self or others, referral to a psychiatrist may be indicated for evaluation.

Education: Educating caregivers, both family and friends, of the wanderer, as well as paid caregivers (nurses, aides, staff), is essential to provide optimal care. The essential information includes the causes of dementia and wandering behaviors and how to identify triggers of this behavior, create a safe environment, and manage wandering individuals while promoting quality of life and patient autonomy.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Falls are associated with an increased tendency to engage in tasks unrelated thoughts that commonly occur in individuals with dementia.	A	Nagamatsu, Kam, Liu-Ambrose, Chan, & Handy: Psychology and Aging, 2013
Elders with dementia frequently engage in wandering behaviors and attempt to exhibit exit-leaving behaviors leading to elopement or becoming lost as a consequence of wandering behaviors.	B	Tilly: Administration on Aging, 2015
The occurrence of wandering in older individuals with dementia, comparison of characteristics, and researchers' theories on causes of wandering behaviors.	A	Cipriani, Lucetti, Nuti, & Danti: Psychogeriatrics, 2014
Definitions of delirium and dementia, discussion of causes and manifestations of these conditions that often lead to wandering behaviors in individuals with cognitive impairment.	C	Huang, 2016
Discussion of consequences of wandering behaviors and strategies to manage.	B	Lin, Zhang, Chen, Ni, & Zhou: International Journal of Gerontology, 2014

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

J. B. is a 77-year-old man who is known to your practice. He is brought in today by his daughter, who reports a new onset of confusion accompanied by UI (first noticed bed was wet a few nights ago). When you see the patient today, he is oriented to place and person (knows you and your office), but not to time, and does not recall much about events of the past few days. He says that he is eating and drinking as usual (but daughter is shaking her head to the contrary). He denies any change in bowel function, but is fearful of sleeping because he might "wet the bed." Daughter states that he has been drinking a lot more water than usual and urinating more frequently. He denies any pain, other than arthritis. He was a regular attendee at the local senior center but has not been there for a week and seems to have forgotten about it.

Past medical history: Known CAD, hypertension, hyperlipidemia, impaired fasting glucose, osteoarthritis of knees.

Medications: Lisinopril 20 mg orally PO once daily in evening; hydrochlorothiazide 12.5 mg PO once daily in morning; metoprolol 50 mg PO once daily in morning; simvastatin 20 mg PO once daily. OTC medications include Aleve, 2 tablets every 12 hours when needed for severe knee pain (infrequent use); topical Icy Hot for knee pain daily; glucosamine-chondroitin preparation daily for joint health; multivitamin for male more than 50 years daily.

Vital signs: Blood pressure (BP) 130/84 mm Hg; heart rate (HR) 60 beats per min, regular; respiratory rate 16 breaths/min; temperature 99.2°F orally; BMI 38.

CASE STUDY—cont'd

Physical findings are unremarkable.

Using the guide to characterizing a symptom located in this chapter, focus on the following questions:

1. What additional subjective data are you seeking?
2. What additional objective data will you be assessing for?
3. What national guidelines are appropriate to consider?
4. What tests will you order? How will you decide on prioritizing in this patient with multiple symptoms?
5. What are the differential diagnoses that you are considering? For each one, map out your clinical reasoning for and against it and make a tentative plan to confirm it or rule it out.
6. Will you be looking for a consultation?

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unit III

Treating Disorders

CHAPTER 6

Skin and Lymphatic Disorders

Laurie Kennedy-Malone

ASSESSMENT

Proper assessment of an older adult's skin, hair, and nails reveals the patient's history of sun exposure, nutrition, socioeconomic status, and education, as well as his or her surgical and medical history. The largest organ of the body, the skin, undergoes certain normal changes with age. The number of cells in the epidermis, such as Langerhans' cells, which are responsible for immune surveillance, begin to decline. Melanocytes also decrease, thereby reducing the skin's natural protection from the sun (Resnick, 2016; Williams, 2009).

Changes With Aging

As people age, the skin tends to be very thin, fragile, and often transparent (Gray-Vickrey, 2010). Skin renewal turnover time increases to approximately 87 days in older adults, compared with 20 days during youth. This fact becomes an important consideration in surgical and dermatological healing. The subcutaneous tissue that houses the eccrine, sebaceous, and apocrine glands thins, thereby providing less protection from heat exposure and less insulation, energy storage, and shock absorption. Secretions aiding temperature control, moisturization, and odor production are also reduced. The skin of older adults therefore tends to be very dry (xerosis). Fat is generally redistributed to the abdomen and thighs, leaving bony surfaces, such as the face, hands, and sacrum, exposed to potential injury, especially skin tears from shearing and friction forces and pressure ulcer development (Doughty & McNichol, 2015). The skin's repair system becomes impaired, with delayed cell regeneration and poor circulation (Resnick, 2016).

Common benign skins lesions in older adults include seborrheic keratoses, cherry angiomas, acrochorda (skin tags), and senile lentigo (Gray-Vickrey, 2010; Williams, 2008). Although the capillaries in older adults tend to be fragile, making the skin of older adults more susceptible to bruising, it is important to discern the cause of the ecchymosis, whether it is trauma-, pressure-, or medication-induced, as in patients taking anticoagulants or corticosteroids (Gray-Vickrey, 2010). It is important to note, however, that "sudden appearance of seborrheic keratoses, telangiectasia or pigmented warty lesions (known as Leser-Trelat sign) may suggest internal malignancy" (Williams, 2008, p. 305).

All of these tissue changes represent a near-catastrophic scenario of aged skin poorly protected from the environment (especially the sun, heat, and cold) and slow to respond to immunological challenges, producing minimal or delayed urticaria and swelling.

Evaluation of Skin and Lymphatics

Assessment of the skin begins with the chief complaint and history, including the dermatological problem's onset, duration, location, size of lesion, color, associated pain or itching, and lost or heightened sensation to touch.

- Investigate associated symptoms such as wheezing, loss of appetite, fever, malaise, headaches, and change in temperature perception.
- Question current and chronic medication history, complete a focused nutritional assessment, and ask about relevant childhood diseases (especially asthma, atopic dermatitis, and chickenpox).
- Ask about contact with chemicals, lifetime occupations, level of lifetime sun exposure, family history of skin cancer, contact with animals, change in environmental habits (e.g., plants, insects, water) or internal habits (e.g., change in soap or laundry detergents, use of any lotions or creams, recent travel, change in diet to include cooking products, exposure to new clothing items or metals such as nickel).
- Ask whether the person has been treated for a similar condition in the past and, if so, ask about the results.
- Note whether the patient appears uncomfortable or pruritic.
- Observe overall hygiene and grooming (Williams, 2008).
- Is the patient incontinent? (Doughty & McNichol, 2015)
- Has the patient had the Zostavax vaccine?
- Is the patient taking any anticoagulation medications? (Elsawy & Higgins, 2011)

Proper equipment for inspection includes good overhead and tangential lighting, a magnifying glass, blades, and collection devices for biopsies and microscopic study. Gloves should

be readily available. Inspect the skin for possible signs of neglect, abuse, or decreased function. Poorly healing wounds or chronic pressure ulcers may signal a problem not only with the patient but with the caregiver's ability to provide adequate care. Welts, lacerations, burns, and distinctive markings may indicate a need for intervention (Gray-Vickrey, 2010; Williams, 2008).

Inspect the hair and nails. The hair may be graying and thinner than in youth. Normally, nails start to form ridges after age 80. A recent (in the past year) change in quality of hair and nails is a significant finding. For example, the hair may have become dryer, duller, and thinner or the nails pitted or brittle, which may be indicative of a systemic condition or nutritional deficiency (Gray-Vickrey, 2010).

Inspection of the skin for even color and overall quality and care is important. Lesions require close scrutiny, description, and classification. Description by type, shape, arrangement, and distribution is a widely accepted and usable format.

- Type: Is the lesion a macule or papule? Is it eroded, excoriated, or hyperpigmented or hypopigmented?
- Shape: Is the periphery of the lesion round, ulcerated, or linear?
- Arrangement: Are multiple lesions grouped (serpiginous, zosteriform, arciform, iris, or bull's-eye pattern) with or without definable borders? (LeBlond, Brown, Suneja, & Szot, 2015).
- Distribution: Is the extent of the lesions isolated, regional, or generalized? Is the pattern symmetrical or over pressure sites, hairline, or sun-exposed areas? Is the distribution characteristic of scabies, seborrheic dermatitis, contact dermatitis, herpes zoster, or lupus?
- Is there any unexplained bruising that could be indicative of elder abuse or neglect? (Elsawy & Higgins, 2011).

The lesion may be classified as primary or secondary. Primary lesions (vesicles, tumors, burrows) arise from normal tissue. Secondary lesions (infections, crusts, and lichenification) arise from changes to the primary lesion (LeBlond et al., 2015). Palpate for moisture, temperature, mobility, and turgor. Testing skin turgor in older patients can be done by pinching the skin on the sternum or forehead. The skin should move easily and return back to place after release; a slower return of the skin may be indicative of dehydration. Lax skin may indicate weight loss, whereas fixed skin may be associated with sclerotic or scarred tissue. Lesions should also be palpated to check whether they are fluctuant, hard, or fixed. At the end of the examination, consider associated pathology, especially thyroid disorders, diabetes, autoimmune conditions, and anemia. Provide wellness instructions, including those concerning proper nutrition, moisturizing, and sun protection. Awareness of the high susceptibility of the elderly to hyperthermia and hypothermia secondary to reduced thermoregulation in the skin should be emphasized (Williams, 2008).

Guidelines for Prescribing Topical Medications

Lesions that concern you or the patient should be examined through biopsy. Lesions that warrant biopsy are those that have changed, bleed, or are painful. Treatment for skin lesions must take impaired absorption, duration, dosage, and delivery method (e.g., cream, powder, ointment, lotion) into consideration. Patients for whom treatment plans fail initially should be referred to a dermatologist.

When prescribing topical treatments it can be a challenge to determine the quantity necessary to dispense. A guideline for determining the quantity was first described by Long and Finlay in 1991 with the introduction of the "fingertip unit." One fingertip unit (FTU) is the amount of ointment or cream dispensed from a tube with a 5-mm opening from the tip of the finger to the first crease. One FTU is equal to approximately 0.5 g. Along with the concept of FTUs, the "Rule of Hand" was developed. This rule says that the body surface of the front and back of an adult hand requires 1 FTU for coverage. Each body part is described by its equivalent surface area in hands. Each arm is equal to three hands, the front torso is seven hands, and so are the back and buttocks. Each leg is equal to six hands, and each foot is two hands. So, if you are treating lesions on one foot, a total of 2 FTUs will be required, or 1 g per treatment. For treatments that are prescribed as twice a day (bid) for 2 weeks, a total of 28 g will be necessary. This guideline results in a more quantitative prescription than writing "dispense 1 week's supply" (Long & Finlay, 1991).

BURNS

Signal Symptoms: Erythema, blistering, serous exudates, eschar, pain, carbonaceous sputum, hypertrophic, pruritus.

Description: Burn injury can be caused by multiple mechanisms. Flame and hot liquids are obvious sources of burn injury. Other causes of burn damage can occur from electricity from high power lines, household current, or lightning strike; radiation or sun exposure; and chemical corrosion. Patients may present varying degrees and depth of tissue damage along with underlying trauma, thus requiring a very thorough physical examination. In addition to what is considered a true burn injury, many exfoliative and necrotizing skin disorders can present in a similar fashion and must be ruled out. Although these skin disorders are not true burns, they are increasingly treated in burn care facilities.

Etiology: Skin forms the largest organ in the body and is constantly in contact with a changing environment. It is composed of three layers: the epidermis, the dermis, and the subcutaneous tissue or hypodermis. The epidermis is the thin outermost layer of the skin. It contains keratinizing cells that migrate from the basal cell membrane and eventually shed over a 2- to 4-week period (Lewis, Heimbach, & Gibran, 2012). At this level of the skin, melanocytes produce melanin, which affects skin color and provides protection from ultraviolet (UV) radiation. The dermis consists of a

dense connective tissue that forms the bulk of the skin. This layer is vascular, with its deeper layer containing hair follicles, associated muscle fibers, cutaneous glands, and nerve fibers. The subcutaneous layer or hypodermis is largely composed of adipose tissue, which provides padding and thermal regulation. Burn injury can affect all of these skin levels in addition to deeper structures, including muscle and bone.

As one ages, there are significant changes in the skin, which becomes thinner, providing a less effective barrier to external stimuli. The dermis atrophies with aging. There are fewer appendages and decreased vascularity. Thus, older adults are at particular risk for burns. Thinner skin combined with decreased vascularity and diminished nerve function often result in a higher incidence of deeper burns. Advanced age results in a weakened immune system. All of these factors, along with the burden of various comorbidities, lead to delayed wound healing and reepithelialization after burn injury in the elderly population (Porro, Deming, Pereira, & Herndon, 2012).

Occurrence: An estimated 2.5 million injuries, 60,000 requiring hospitalization, and 6,000 deaths occur annually in the United States from burns.

Age: Between 2007 and 2011, those aged 65 years and older made up 13% of the U.S. population, but accounted for 30% of fatal home fire victims. This represents an increase from 1980 by 31%. Between ages 65 and 75 years, the mortality rate from burns is twice that of the national average (M. Ahrens, 2014). The Centers for Disease Control and Prevention (CDC, 2016) data indicates that from 2001 to 2014, fire-related burn injury was the twelfth leading cause of nonfatal injury for individuals 65 years and older.

Gender: The American Burn Association's (ABA's) National Burn Registry reported that between 2009 and 2015, across all age categories, males sustained a higher rate of burn injury than women, except for those individuals 80 years and older.

Ethnicity: The ABA's National Burn Registry outlines the following breakdown of reported ethnicity in burn injuries: 58.4% Caucasian, 20.7% African American, 13.0% Hispanic, 4.5% other, 2.5% Asian, and 0.9% Native American. When analyzed by age group, it was found that minorities were overrepresented among those with burn injuries 4 years and younger. As the population ages into adulthood, this characteristic shifts and minorities were no longer found to predominate.

Contributing Factors: Nearly 84% of all burn-related deaths occur in the home (Haynes, 2015). Smoke- and toxic-fume–induced inhalation injury associated with house fires is a larger contributor to death than the burn itself. Inhalation injury is particularly lethal when victims are more than 65 years of age (Peck, 2011). Among the major causes of house fires are cooking equipment, home heating systems, and electrical wiring. Smoking is the leading cause of home fire deaths and has been found to increase the risk of burn injury six-fold (M. Ahrens, 2015; Low, Meyer, Willebrand, & Thomas, 2012). In home fires related to smoking, the risk of death is further compounded by the use of home oxygen.

Human judgment plays a large role in activities that are related to burn injury. Older adults have slow response times and may show impaired judgment due to illness or medications. The ability to sense fire decreases and disabilities can impede escape. Comorbidities such as cardiac disease, renal disease, diabetes mellitus, dementia, or psychological impairment may complicate outpatient care and these individuals may require initial hospital admission for burn assessment and stabilization (Low et al., 2012; Warner, Coffee, & Yowler, 2014). Many older adult individuals live alone or are dependent on an aging spouse for support. This isolation further increases the risk of burn injury.

Signs and Symptoms: A thorough history and physical examination should be undertaken and will provide a majority of the information needed to assess a burn injury and its appropriate management. Ascertain the time and mechanism of the burn injury, as well as duration of contact with the offending agent, and any steps taken to manage the injury. This will assist in determination of wound depth and suitability for outpatient management. Burn injury constitutes trauma, and as such, the initial evaluation should always consist of a primary survey, including the ABCs: airway, breathing, and circulation, followed by a secondary survey including a head-to-toe assessment. After these assessments are completed, it is time to focus on burn depth. Burn injuries involve a graduating zone of damage that occurs along a time continuum, making it difficult to estimate the depth of a burn when first evaluated.

- First-degree burns involving the epidermis are erythematous and painful, but do not blister. Most sunburns fall into this category and are not considered when estimating total burn size.
- Second-degree (partial thickness) burns
  - Superficial dermal burns involve the dermis and are characterised by blisters. The underlying tissue is pink, moist, and hypersensitive to touch. The tissue will blanch with pressure and is associated with dilatation. Superficial dermal burns will often heal within 2 to 3 weeks.
  - Deep dermal burns extend further into the dermis, present with blistering, but the tissue surface appears mottled, pink and white immediately after injury. Capillary refill is slow and usually by the second day from injury the wound bed appears white and dry. Pain is often described more as pressure. Partial thickness burns that are not healed by 3 weeks should be excised and grafted.
- Third-degree (full thickness) burns involve the full dermis extending into the subcutaneous tissue. The burn appears dry, white, charred, and leathery. There is no sensation to pinprick in the wound bed. There may be mottling that does not blanch with pressure. There may be visible coagulated vessels on the surface. Full thickness burns require early excision and autograft.
- Indeterminate burn wounds may hold components of both partial thickness and full thickness, and should be evaluated daily over a period of days to determine the need for excision and autograft (Lewis et al., 2012).

After determining the extent of the burn injury, it is necessary to estimate the total body surface area (TBSA) involved. Small area burns in healthy individuals can be easily managed in the outpatient setting. However, it must be kept in mind that the larger the TBSA involved, the higher the likelihood

TABLE 6-1 Adult Burn Size Estimation

AREA	ADULT (>15 YRS.)	SECOND-DEGREE INJURY	THIRD-DEGREE INJURY	TOTAL
Head	7			
Neck	2			
Anterior trunk	13			
Posterior trunk	13			
Right buttock	2.5			
Left buttock	2.5			
Genitalia	1			
Right upper arm	4			
Right lower arm	3			
Right hand	2.5			
Left upper arm	4			
Left lower arm	3			
Left hand	2.5			
Right thigh	9.5			
Right lower leg	7			
Right foot	3.5			
Left thigh	9.5			
Left lower leg	7			
Left foot	3.5			
	TOTAL			

Note: Lund and Browder technique for those greater than 15 years of age. Adapted from *Care of the outpatient burn* by C. E. Hartford (2012), in D. N. Herndon (Ed.), *Total burn care* (4th ed., pp. 81–92). Edinburgh; New York: Saunders Elsevier.

of a poor prognosis. This is particularly salient in the older adult population due to comorbidities and the psychosocial components of managing a burn injury at home. There are several methods for determining burn size, which can be as complex as laser-assisted mapping to simple visual estimates. The Lund and Browder technique was first published in 1944 and takes into account body surface area in light of age. This is particularly important with infants and small children, as the head occupies a larger surface area. The estimated surface area stabilizes by 15 years of age through adulthood (Table 6-1). A more simple method for estimating the burn size utilizes the patient's palm as representative of 1% of their TBSA. This involves the patient's palm, digits, and thumb in adduction.

After your patient assessment is completed, a decision must be made to transfer to a higher level of care or to continue management in the outpatient setting. The ABA has established criteria for burn center referral to improve patient outcomes (Table 6-2). An important caveat is that any case of suspected elder abuse should be transferred via the emergency medical system to the appropriate emergency department to ensure proper follow-up and reporting. Suspicion of abuse should be triggered by any of the following:

TABLE 6-2 American Burn Association Transfer Criteria

1. Partial-thickness burns greater than 10% of TBSA
2. Burns that involve the face, hands, feet, genitalia, perineum, or major joints
3. Third-degree burns in any age group
4. Electrical burns, including lightning injury
5. Chemical burns
6. Inhalation injury
7. Burn injury in patients with pre-existing medical disorders that could complicate management, prolong recovery, or affect mortality
8. Any patient with burns and concomitant trauma in which the burn injury poses the greatest risk of mortality or morbidity; if trauma poses the greater immediate risk, the patient may be stabilized in a trauma center before transfer to a burn unit
9. Burned children in hospitals without qualified personnel or equipment for the care of children
10. Burn injury in patients who will require special social, emotional, and/or long-term rehabilitative intervention

From Guidelines for the operation of burn centers. (2007). *Journal of Burn Care and Research, 28*(1), 134.

- Burn patterns that do not match the reported event
- Straight or smooth transitions between the area burned and healthy skin, resulting in a stocking- or glove-like appearance
- A long delay between burn injury and request for treatment

Diagnostic Tests/Tools: Laboratory and diagnostic testing should be guided by patient presentation and, if warranted, may be an indication for hospital based evaluation. Of note, smoke inhalation injury may be masked by standard pulse oximetry monitoring. Most systems are designed to detect full saturation of a hemoglobin molecule and cannot differentiate between oxygen and carbon monoxide, resulting in misleading saturation levels (Nitzan, Romem, & Koppel, 2014). A carboxyhemoglobin (COHb) blood level should be obtained whenever an inhalation injury is suspected.

Differential Diagnosis:

- Toxic epidermal necrolysis
- Scalded skin syndrome

Treatment:

Initial Care: First, stop the burn. This should include removing and isolating the causative agent responsible for the injury while protecting the patient and your staff. If a scald has occurred, be certain that soaked clothing has been removed. If chemicals are suspected, irrigate the wounds with copious amounts of water (Lloyd, Rodgers, Michener, & Williams, 2012). Isolate clothing to be sure that others are not contaminated. In evaluating older adult burn patients, it is imperative to consider their physiological differences from younger adults, along with any associated comorbidities. If it is determined that transfer to a higher level of care is appropriate, activate the emergency medical system; do not delay for wound debridement. Cover wounds with dry, clean sheets or towels. Maintain a warm environment to minimize the risk of hypothermia. IV access should be obtained with lactated Ringer's solution as the preferred replacement agent.

TABLE 6-3 Topical Agents and Dressings for Burn Wounds

NAME	USAGE	APPLICATION
Topical antibiotic ointment (bacitracin, mupirocin)	Small area burns: prevention and treatment of infection with gram-positive and some gram-negative bacteria. Mupirocin is active against most strains of MRSA.	Ophthalmic preparations without a dressing are preferred for facial wounds. Apply a thin layer daily to wound, cover with a petroleum impregnated mesh and sufficient gauze as needed to absorb exudate.
Hydrocolloid dressings	Provide a moist protective covering and barrier against exogenous bacteria. Effective for small-area, partial-thickness burns. Not indicated for wounds with large amounts of exudate.	Occlusive dressings may be left in place for 5–7 days for patients with low risk for complication.
Silver sulfadiazine 1%	Prevention and treatment of infection in second- and third-degree burns with eschar burden. Can delay wound healing by impeding epithelialization. Discontinue use once necrotic tissue has sloughed from wound. Contraindicated in those with sulfa allergies or pregnant women.	Do not apply to face, cover burn area with a layer approximately 1/16-inch thick, wrap with sufficient gauze to absorb exudates, change daily.
Petroleum gauze/mesh (Adaptic®, Xeroform®)	Protective covering, 3% bismuth tribro-mophenate in a petrolatum blend (Xeroform®) has bacteriostatic action, can be used with topical antibiotic ointments. May be used in cases of sulfa allergy.	Apply daily, cover with gauze wrapping sufficient to manage exudate.
Aquacel Ag	Hydrofiber silver impregnated antimicrobial dressing. Conforms to wound surface and can absorb high amounts of exudate with autolytic properties.	May be left in place for up to 14 days. Cover with secondary dressing such as hydrogel, foam dressing, or gauze. Not compatible with oil-based products. Care should be taken to monitor function if placed over joints.

Adapted from Warner, P. M., Coffee, T. L., & Yowler, C. J. (2014). Outpatient burn management. *Surgical Clinics of North America, 94*(4), 884–885 and Hartford, C. E. (2012). Care of outpatient burns. In D. N. Herndon (Ed.), *Total burn care* (4th ed., pp. 81–92). Edinburgh; New York: Saunders Elsevier.

Oxygen by nasal cannula would be appropriate for moderate-to-major burns and in any case where pulmonary distress is evident. In cases of suspected inhalation injury, 100% via a nonrebreather mask is indicated.

Wound Care: Burn wounds are frequently colonized with bacteria from the local environment. These wounds are routinely managed with topical antibiotics; systemic antibiotic therapy is not generally indicated (Table 6-3). After administration of appropriate pain medication, wounds should be initially doused with cool tap water to disperse any remaining heat in the tissue. The use of cold fluids is not advised, because this may cause further tissue damage as well as hypothermia. For routine wound care, tepid water is advised. Wounds should be cleaned with a mild soap and rinsed. Antibacterial soaps are not indicated. There are varying approaches to the management of blisters. One theory is that if left in place, the blister provides a protective covering and will allow the wound to heal more quickly. Alternatively, plasmin inhibitors in the fluid of the blister may delay healing while the fluid itself can provide a medium for infection (Hartford, 2012). For small surface area burns, a good guide is to remove what tissue is loose during cleansing and allow intact blisters to remain (Warner et al., 2014). Gently wipe the wound in a circular manner, debriding any loose devitalized tissue and debris during this process. Wounds should be washed in this manner on a daily basis.

The goal of wound dressings is threefold: absorb drainage, provide protection, and decrease pain. Keeping this in mind, the dressings used for small burn wounds should be simple, affordable, and manageable by patients and caregivers. There are virtually no objective comparisons of dressings for small burn wounds, and anecdotal evidence leans heavily toward coverage with simple petroleum-infused mesh with or without antibiotic ointment. When wrapping a burn wound, it is important to consider function. Digits should be wrapped individually to allow for gentle range of motion exercises. Elevate extremities at rest and utilize an ace wrap or compression garments to prevent edema.

Burn wound care utilizes clean rather than sterile technique. Patients and families should be taught proper cleaning techniques, including frequent hand washing and management of supplies within their environment. Wounds should be examined daily during care for redness, swelling, purulent drainage, or odor. This should be reported promptly to the health-care provider, and the addition of systemic antibiotics should be considered.

Patients should be instructed to minimize exposure to UV light. This should include the use of protective clothing and sunscreen over healed burn wounds. They should continue to utilize compression garments in the rehabilitative phase to minimize swelling and scar formation.

Follow-Up: Outpatients should be reevaluated 24 hours after the burn injury. Examine the condition of the wound and surrounding tissues for signs of healing or impending infection. Reexamine respiratory status of patients with an inhalation injury to detect diffuse wheezing and rhonchi. Determine the adequacy of the current pain management regimen. Have the patient or significant other demonstrate proper dressing techniques. Depending on the severity of the burn, the patient may be asked to return once a week until progressive healing is noted; daily visits may be necessary. Have patients return periodically about every 4 to

6 weeks to determine healing, assess progress/compliance with any physical therapy if required, and assess coping mechanisms.

Sequelae:

Pain: There is benefit in utilizing medications with different mechanisms of action to address pain in burn patients. This is especially true for burn patients undergoing any type of procedure that can include wound care or physical therapy (Edkins et al., 2015; Meyer, Wiechman, Woodson, Jaco, & Thomas, 2012). It should be anticipated that for small burns the need for narcotics will be short lived as the wound heals. With deeper or larger burns, there may be a benefit to adding pharmacological agents that act on neuropathic pain, such as gabapentin and pregabalin, while acetaminophen and NSAIDs are beneficial to manage pain and inflammation (McIntyre, Clifford, Maani, & Burmeister, 2016; Retrouvey & Shahrokhi, 2015; Warner et al., 2014). Evaluation of renal function should be assessed with dosages adjusted accordingly.

Pruritis: The process of healing triggers the mobilization of multiple agents, including cytokines and histamine, into the wound bed. These agents, among others, can result in intense pruritus. While multiple additional causes for pruritus have been suggested, the exact causation remains unknown (Hartford, 2012). Significant disruption to wound healing can result through the process of scratching. This can be particularly challenging in the older adult population, because antipruritic agents, such as diphenhydramine or hydroxyzine, can be sedating. Adequate analgesia in conjunction with these agents can be effective, but care must be taken to avoid oversedation. Agents that selectively block H_1 receptors, such as cetirizine or loratadine, can be more effective with less sedating effects and are best administered on a scheduled basis (Warner et al., 2014). The use of moisturizers can aid in management of pruritus in healed burn scars and are essential to prevent drying and cracked skin. As the burn scar begins to mature, it is anticipated that pruritus will begin to diminish over a period of approximately 6 months.

Hypertrophy and Contracture: In healing wounds, the production of collagen is increased. This can be even more exaggerated in infected wounds or in individuals prone to keloid formation. Wounds that remain open for 2 weeks or longer tend to be particularly prone to the development of hypertrophic scarring (Hartford, 2012). Hypertrophy can lead to pain and loss of function through distortion of normal anatomy. The utilization of appropriate topical agents and dressings that act to facilitate a wound's timely closure will help prevent hypertrophy. In addition, the use of pressure garments over nearly closed or healed wounds will aid normal scar progression. Although the action is not completely understood, the use of silicone sheeting over areas of hypertrophy has been a successful adjunct to normal scar progression in some individuals.

Management of scar bands that compromise function should include an evaluation for surgical release. The phenomenon of web creep occurs when hypertrophic scars begin to compromise the web spaces of the fingers and toes. Contractures and underlying tendon damage in the hands may result in impairments, such as Boutonniere deformities. The resulting loss of function can often be restored through surgical intervention. Healed scars at least 6 months out from injury that remain hyperemic and hypertrophic may benefit from resurfacing with a pulsed dye laser, alternated with a fractional carbon dioxide laser, to diminish inflammation and soften scars (Hultman, Edkins, Wu, Calvert, & Cairns, 2013; Hultman, Friedstat, Edkins, Cairns, & Meyer, 2014). Additionally, fat grafting to burn scars that remain chronically painful have shown some benefit (Fredman, Edkins, & Hultman, 2015). Bands and contractures can develop many years after injury, and scars should be monitored for progression.

Prevention/Prophylaxis: In order for a burn injury to occur, multiple factors must come together at one time. These factors include the host, the environment, and the vector. For example, an elderly patient falls asleep while smoking, his sheets ignite, and he is burned in a resulting house fire. It is the relationship between man, his environment, and a potentially hazardous agent that can result in injury. The nurse practitioner can affect each of these components and possibly prevent the initial burn injury from occurring. By using three dimensions of prevention, primary, secondary, and tertiary, the nurse practitioner, by virtue of education, clinical expertise, and anticipatory guidance, plays a vital role in burn prevention (Grant, 2004).

Primary prevention constitutes product design, regulation, and legislation. This also includes educational programs designed to educate the public on safety. In the previous example, the individual smoking in bed would hopefully benefit from smoking cessation programs in his community. Regulation and product design in this scenario would affect the combustibility of clothing, linen, and housing materials exposed to the smoldering cigarette. During an annual physical examination, the nurse practitioner should include education on sound living habits and home safety. Included in this discussion should be a review of home fire safety protocols, including the proper use of smoke alarms, removal of hazards such as frayed electrical cords, visual aids for those with impaired sight, and the appropriate temperature setting for home hot water heaters. This is also an excellent opportunity to discuss the advantages of smoking cessation.

Secondary prevention includes actions that would serve to limit the extent of damage done by a burn, such as cooling wounds initially to mitigate deeper tissue damage. Occupational therapists provide compression garments and ensure proper splitting technique to maintain function. Physical therapists assist with mobilization though stretching and exercise programs. The injury has occurred, but these actions work to limit the scope of damage.

Lastly, tertiary prevention addresses recovery and rehabilitation. Individuals continue to work with therapists to maximize function. Management of healed burn scars with moisturizers and sunscreen can prevent further skin damage. Through tertiary prevention, the individual optimizes his or her level of functioning and minimizes disability resultant from the burn injury.

The nurse practitioners are instrumental in developing and/or referring clients to community-based programs that promote home safety for the elderly (Grant, 2004). Excellent educational programs are available through agencies such as

the National Fire Protection Association, the CDC, the ABA Prevention Campaign, and verified burn centers.

Referral: Burns often require complex care and the involvement of multiple disciplines. In the older adult population, even a small burn may require fluid resuscitation that could complicate care due to pre-existing comorbidities and concomitant medications. The absence of support systems or the suspicion of elder abuse should prompt considerations for hospitalization and possible referral to a specialized burn care facility. The ABA has developed a list of criteria that should signal the need for referral to a burn center (see Table 6-2). A burn center verified by the ABA can provide services to a burn patient through all phases of the patient's care.

For rehabilitation concerns related to nerve compression, development of scar bands, or contractures, it would be advantageous to make a referral to a plastic and reconstructive surgeon. Reconstruction after burn injury should also include evaluation of mature scars that have remained hyperemic and hypertrophic for possible photoablation with laser therapy. Occupational therapy and physical therapy are crucial in preserving and maintaining form and function for burn victims (Lloyd et al., 2012). Referral for these services should be made early in the care of the burn patient. A referral for social services or in-home assistance may be indicated to promote safe return to a living environment. Contact your nearest burn center for referral information. An excellent resource for patient and family support is the Phoenix Society, a national nonprofit organization dedicated to empowering those affected by a burn injury (https://www.phoenix-society.org/).

Education: Education is an ongoing component of patient care. The nurse practitioner should identify early on who will be providing wound care. This individual, or the patient if able, should demonstrate the ability to perform wound care before leaving the clinic or hospital setting. Patients should be provided with appropriate written instructions in clear language for wound care, indicators for infection or tissue compromise, and contact information should questions arise. In addition to wound care, there should be written instructions on medications, including dosing and possible side effects.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Education regarding fire safety and prevention is the most effective intervention to prevent loss of life and injury due to home fires and should be promoted at every visit.	A	American Burn Association Prevention Campaign, 2014 Grant, 2004
Initial pain management for burn injuries with low-dose narcotics sufficient to allow for wound care and therapy. Adjunct therapy with NSAIDs and neuropathic pain modulators as indicated.	A	Edkins et al., 2015 Meyer, Wiechman, Woodson, Jaco, & Thomas, 2012 McIntyre, Clifford, Maani, & Burmeister, 2016 Retrouvey & Shahrokhi, 2015 Warner et al., 2014
Topical antibiotic use for burn wounds with avoidance of prophylactic systemic antibiotics.	C	Warner, Coffee, & Yowler, 2014 Hartford, 2012
Systematic daily care of burn wounds using clean technique, with goal of 1 month for wound closure.	C	Warner, Coffee, & Yowler, 2014 Hartford, 2012
Pressure garments and silicone sheeting to prevent swelling, decrease pruritus, and minimize hypertrophic scar formation.	C	Warner, Coffee, & Yowler, 2014 Hartford, 2012
Evaluation for comorbidities including dementia and other psychiatric disorders associated with burn injury.	C	Low et al., 2012 Warner, Coffee, & Yowler, 2014

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CELLULITIS

Signal Symptoms: Erythema, warmth, blisters, local tenderness, erysipelas.

Description: Cellulitis is an infection of the deep dermis and subcutaneous tissues of the skin; it is most commonly caused by groups A, B, C, and G beta-hemolytic streptococci, *Staphylococcus aureus* (both methicillin-susceptible [MSSA] and methicillin-resistant [MRSA]), and occasionally a gram-negative organism (IDSA, 2014). Cellulitis can also be caused by human or animal bite pathogens.

Etiology: Cellulitis usually occurs when an organism enters the skin through an open area. However, a clear site of entry may not be evident in patients with obesity, edema, or lymphedema (Salmon, 2015).

Occurrence: In the United States, 14.5 million cases of cellulitis are diagnosed annually, with over 650,000 hospital admissions per year attributed to the diagnosis (Raff & Kroshinsky, 2016).

Age: Cellulitis can occur at any age. Facial cellulitis is most common in people 50 years and younger and lower extremity cellulitis is more common in older individuals with lymphedema and venous stasis ulcers (Compton, 2013).

Gender: Cellulitis occurs equally among men and women.

Ethnicity: Cellulitis occurs in all ethnic groups.

Contributing Factors: Predisposing factors include arterial or venous insufficiency, diabetes mellitus, lacerations, obesity, lower extremity edema, lymphedema, human or animal bites (cat bites), tinea infections, recurring cellulitis, burns, trauma, stasis ulceration, ischemia, toe web maceration, IV drug use, and puncture wounds (Compton, 2013; Salmon, 2015). Any break in the skin or mucous membranes is a potential bacterial pathogen port of entry (Dunphy et al., 2015). Acute and chronic sinusitis can lead to periorbital or orbital cellulitis. Consider cellulitis in patients who are on immunosuppressive medications, immunocompetent (HIV infection and AIDS), have drug or alcohol abuse issues, or are prescribed long-term use of corticosteroids or cancer chemotherapy (Dunphy et al., 2015). The fragile skin of the elderly is predisposed to skin tears and edema which increases risk of cellulitis (Compton, 2013).

Signs and Symptoms: Cellulitis in the elderly often presents with atypical symptoms (Compton, 2013). Signs include complaints of trauma or injury to the skin, pre-existing skin conditions, recurrent cellulitis, and worsening of erythema, edema, tenderness, and pain over the past few days. Symptoms are usually sudden and include rapidly spreading warmth, localized tenderness, erythema, edema, and pain (Raff & Kroshinsky, 2016). Systemic symptoms that indicate serious toxicity include fever, hypotension, tachycardia, leukocytosis, lymphadenopathy, and lymphangitis (Dunphy et al., 2015). Cellulitis may be purulent or nonpurulent.

Diagnostic Tests: Unless pus has formed in the wound, a tissue culture is usually not necessary (IDSA, 2014). In cases of extensive infection or suspected systemic toxicity, blood cultures and a complete blood count (CBC) with differential should be obtained to determine the severity of illness and to identify the organism, including methicillin-resistant *Staphylococcus aureus* (MRSA) (IDSA, 2014). C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) may be elevated in patients with severe cellulitis. Patients with diabetes mellitus should have a radiograph of the area and an ESR to rule out the presence of osteomyelitis. Skin biopsies may be necessary to rule out the presence of fungal, viral, parasitic, or mycobacterial etiologies (ISDA, 2014).

Differential Diagnosis:

- Lipodermatosclerosis (tender red plaque on the medial lower legs with venous stasis or varicosities)
- Acute severe contact dermatitis (superficial edema, itching)
- Acute gout (synovial fluid aspiration contains monosodium urate crystals)
- Necrotizing fasciitis (determined by magnetic resonance imaging [MRI] or computed tomography [CT]; diffuse swelling of an arm or leg with bullae (clear or serosanguineous fluid, crepitus, and intense pain)
- Deep vein thromboses (DVTs) (lower extremity Doppler ultrasound)
- Pseudogout (synovial fluid aspiration contains calcium pyrophosphate crystal deposition)
- Osteomyelitis (diagnosed by radiographs, MRI, CT)
- Pyomyositis (diagnosed by radiographs, MRI, CT)
- Early herpes zoster
- Atypical drug eruptions
- Erysipelas (superficial cellulitis represented by red area)
- Venous or varicose eczema (limited or localized erythema, absence of systemic symptoms) (Dunphy et al., 2015)

Treatment: Ask when the patient's last tetanus toxoid booster was given if an open wound is present. If the most recent booster was 10 years ago, 0.5 mL of tetanus toxoid should be administered immediately (CDC, 2016). If the wound is grossly contaminated and the patient's last tetanus booster was 5 to 10 years ago, the practitioner should consider giving another booster at this time (CDC, 2016). An older adult who has not had primary immunization requires tetanus toxoid and tetanus immune globulin (CDC, 2016).

Patients with mild cellulitis may be given oral antibiotics (IDSA, 2014). The drugs of choice (typically for 7 days) include one of the following: dicloxacillin 500 mg four times daily; clindamycin 300 mg four times daily; amoxicillin/clavulanate 875 mg/125 mg twice daily; and a first-generation cephalosporin such as Cephalexin 250 to 500 mg four times daily (ISDA, 2014). For streptococcal cellulitis, penicillin is the drug of choice (IDSA, 2014). Treatment of MRSA (total duration of therapy is 7 to 14 days) should be guided by wound culture results and/or considered when the patient has higher risk factors for MRSA or cellulitis is nonresponsive to primary choice of treatment (IDSA, 2014). Antibiotic choices include one of the following: clindamycin 300 to 600 mg four times daily; doxycycline 200 mg once followed by 100 mg every 12 hours; trimethoprim-sulfamethoxazole 160 mg/800 mg 1 to 2 tablets twice daily; or linezolid

600 mg every 12 hours (IDSA, 2014). Consider the patient's renal function before prescribing any medication. Patients with complications or severe symptoms (especially systemic) should be hospitalized for monitoring and to receive IV antibiotics (IDSA, 2014).

Follow-Up: Outpatients should be requested to contact their health-care providers within 1 week if no improvement with antibiotic use or within 48 hours if fever or inflammation continues.

Sequelae: In patients with cellulitis of the lower extremities there is an increased risk of DVT. Further issues caused by cellulitis include bacteremia, bullae, lymphangitis, and necrosis of the affected area (Herchline, 2015).

Prevention/Prophylaxis: Prevention of cellulitis includes good hand washing, avoidance of injury/trauma, minimizing edema, prevention of peripheral vascular disease, and controlling diabetes.

Referral: The following patients should be hospitalized: those with high fever, diabetes, alcoholism, HIV, anaerobic cellulitis, necrotizing fasciitis, or cellulitis of the orbit or face, or those experiencing extreme pain (IDSA, 2014). Consultation with an infectious disease specialist may be necessary. A surgical consultation may be required for incision and drainage of abscesses and débridement of necrotic tissue.

Education: Patients with cellulitis must adhere to antibiotic dose, frequency, and duration of antibiotic treatment for optimal results. Avoid trauma to the area, keep the area clean, and elevate the affected area if able (especially if edema is present). If there are no contraindications, patients may use NSAIDs and Tylenol for pain relief. If there is no DVT, patient may use compression bandages to decrease edema (especially if edema causes recurrent cellulitis). As with any infection, blood glucose levels may need to be monitored in those with diabetes. There is an increased risk of DVT associated with cellulitis, and education should include signs and symptoms of DVT (report immediately if they occur).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Guidelines were updated to improve diagnosis and treatment of soft tissue and skin infections (cellulitis).	C	IDSA, 2014
Increasing awareness of differential diagnoses associated with red legs improves accuracy of cellulitis diagnosis.	C	Salmon, 2015
Cellulitis is a common skin infection in the elderly. Choosing the right empiric therapy is important.	C	Compton, 2013
Treatment of cellulitis should cover *Streptococcus* and MRSA. Expand coverage for MRSA if risk factors are present. Five days of treatment is recommended with extension if no improvement.	A	Raff & Kroshinsky, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CORNS AND CALLUSES

Signal Symptoms: Thickened skin or hyperkeratotic area on the foot or hand that is often painful. This section will focus on those occurring on the foot.

Description: Corns and calluses are benign skin lesions on the foot. Corns (helomas) can be soft (heloma molle) or hard (heloma duram). They have distinct borders as compared to callosities. Soft corns are spongy and most often found between the fourth and fifth toes. Hard corns form from calluses over time. They have a central core that is painful with weight bearing. They occur on the plantar surface of the foot at the metatarsal heads and on the interphalangeal joints. A callus (tyloma) is a hardened area on the foot's plantar surface. These usually are seen at the metatarsal heads, medially to the first metatarsal head and laterally to the fifth metatarsal head. Melo and colleagues (2015) report lesser-toe deformities including hammer toes, claw toes, and mallet toes, predispose individuals to callus formation on the second through fifth distal surface of the toes.

Etiology: Mechanical forces of compression, shear, and torsion (Hashmi, 2013) put stress on the stratum corneum of the epidermis. This triggers a protective and inflammatory response. The stratum corneum cells form either a plaque (callus) that is superficial or cone shape (corn), which is pointed and deep (Hashmi, 2013).

Occurrence: Corns and calluses are the most common foot ailment seen by podiatrists (26%) in the United Kingdom

(Farndon, 2015). Concannon and Stephens (2015) reported that corns can be found in 14% to 48% of the population.

Age: Corns and calluses are common with increasing age (Feldman, 2017). Older adults have a higher incidence, as the aging process reduces fatty tissue and bones become more prominent (Nordqvist, 2014).

Gender: Females are affected more often than men. This is predominately related to women wearing high heels. Farndon (2015) found that the ratio of females to males seeing a podiatrist for any foot problem was 74% versus 25% in a given day.

Ethnicity: African Americans report 30% greater occurrence than Caucasians (Feldman, 2017).

Contributing Factors:

Intrinsic: Bony prominences, foot deformity, mobility alterations, obesity, diabetes, peripheral neuropathy, peripheral arterial disease, anhidrosis, and hyperhidrosis.

Extrinsic: Poor fitting shoes, not wearing socks or wearing socks that fit poorly, high heels, thin-soled shoes, walking barefoot and standing, walking or running for prolonged periods of time.

Signs and Symptoms: Pain can be the cardinal symptom of both lesions, however, calluses are usually asymptomatic, although they may be of cosmetic concern. Corns are often painful and interfere with daily activities, which is frequently the chief complaint in podiatry practices (Frandon et al., 2015).

Diagnostic Tests: Clinical examination and history taking is usually the method of discovery, however, radiographs may be ordered to assess bone anatomy and foot deformity. Hashmi (2013; Hashmi et al., 2006) reports ultrasound has been used but resolution is poor. Dermatology practices are using optical coherence tomography (OTC) to assess before and after treatment (Hashmi, 2013; Baillie et al., 2011).

Differential Diagnosis:

- Plantar wart (Feldman, 2017)
- Foreign body granuloma (Ramano, 2016)
- Porokeratosis (Mitnick, 2006-2016)
- Underlying ulcer (Feldman, 2017)
- Fungal infection (Feldman, 2017; Mitnick, 2016; Ramano, 2016)

Treatment: Self-maintenance of minor calluses can be done by soaking the feet in warm water and sanding the affected area with a pumice stone. Calluses may be softened with emollients (with and without urea). Barrier creams and occlusive materials, such as silicone sleeves may reduce friction (Hashimi, 2013) and prevent callus and corn formation. Crest pads, off-loading pressure with padding will reduce pressures (Curran, 2015). Salicylic acid plasters and mechanical debridement or paring with a 15-blade scalpel are two successful treatments for removing corns (Fardon, 2013). High-risk patients with some toe deformities may require surgery. Percutaneous digital flexor tendon tenotomy used to straighten a toe when conservative treatment has been unsuccessful (Bus et al., 2016) and electrosurgery are two other methods recognized by Hashmi (2013). Diabetic patients and those with neuropathy should be advised to never use a chemical treatment on their feet. This could damage normal skin without them knowing it (Ramano, 2016).

Follow-Up: Follow-up is determined by the intervention type. Use of salicylic acid plasters usually demand a follow-up appointment within 1 week to assess the progress of the chemical debridement, the need for mechanical debridement of affected skin, and to assess for any complication (Fardon et al., 2013).

Sequelae: Untreated corns and calluses usually result in extended periods of pain, possible alteration in mobility that could result in falls, and a general decline in quality of life. According to the CDC (2015), diabetic patients are especially at risk as these lesions may ulcerate, become infected, and worst-case scenario, require amputation.

Prevention/Prophylaxis: Encourage self-awareness and inspection of the feet as a basic self-care measure. Providers should inspect diabetic feet at every visit. Proper footwear is essential. The shoe must be the proper size and not cause any friction. If a problem area is identified, inspect the inside of the shoe for problems. Lamb's wool and toe pads of felt or silicone may be used to protect the affected area. Prescribed orthotic inserts or therapeutic shoes may be necessary.

Referral: Primary care providers can address the problems associated with corns and calluses; however, podiatric or orthopedic referral for patients with complicated corns and calluses should be made when treatment prescribed has not alleviated or improved the problem. It is recommended that diabetic patients see a podiatrist annually (CDC, 2015) and with more frequency if high risk factors or problems exist.

Education: Advise patients to maintain good foot hygiene: washing feet regularly, keeping toenails clipped, and wearing clean socks and shoes that fit properly. Diabetic patients should be instructed to wash and inspect their feet daily, looking for problematic areas, and to seek medical assistance when lesions are apparent. Counsel patients on the prevention measures mentioned previously.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Emollients with 25% urea are more effective than those with 10% urea in softening and removing calluses.	A	Young & Jones, 2015
Crest pads offload pressure from the distal area of the toes in patients with lesser toe deformities, improving, minimizing, and preventing callus formation.	A	Melo et al., 2015
Seven mm felt padding reduces plantar pressure as compared to lesser depth felt padding.	B	Curran, Ratcliffe, & Campbell, 2015
Corn plasters are an effective treatment to reduce and resolve corns as compared to scalpel debridement on appropriate patients.	A	Farndon et al., 2013
Gold standard treatment to reduce corns and calluses is scalpel debridement.	C	Hashmi, Nester, Wright, & Lam, 2016

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HERPES ZOSTER

Signal Symptoms: Cutaneous eruption of a dermatome distribution; burning, itching, or tingling skin sensation.

Description: Herpes zoster is an acute vesicular eruption caused by a virus histologically identical to the varicella (chickenpox) virus. Herpes zoster is human (alpha) herpes virus 3 (varicella-zoster virus [VZV]), a member of the herpes virus group.

Etiology: Recurrent VZV infection causes herpes zoster. The patient has initial contact with VZV in the form of chickenpox. The DNA virus resides within the neurons. A rash, beginning as maculopapular lesions, develops when the VZV reactivates in the dorsal ganglia. VZV migrates to adjacent sensory dermatomes. The vesicles form scabs within 10 days (Cohen et al., 2013). Older adults have a decrease in cell-mediated immunity, which contributes to the risk of developing herpes zoster and post-herpetic neuralgia (PHN) (Barakzai & Fraser, 2008; Ganty, 2013). If herpes zoster affects adjacent dermatomes, crossing the midline of the body, it is considered to be disseminated herpes zoster. Ten percent to 40% of people who are immunocompromised will contract disseminated VZV (Yoon et al., 2013).

Although chickenpox is one of the most readily communicable diseases, herpes zoster has a much lower rate of transmission. Nonimmune persons are considered contagious 8 to 21 days after exposure to VZV. Mode of transmission is coming into contact with the vesicle fluid. Patients with herpes zoster may be sources of infection for 1 week after the appearance of vesicle lesions. Nonimmune individuals can transmit the infection and should avoid contact with patients with herpes zoster (Fashner & Bell, 2011).

Occurrence: Herpes zoster occurs worldwide, more commonly in older adults. Incidence increases markedly in those 60 years old or older as a result of impaired cell-mediated immunity (Epps et al., 2015). Ten cases of herpes zoster occur for every 1,000 older adults. This increase is, in part, due to older adults living longer (CDC, 2016; Gargliadi et al., 2016). It is estimated that the risk of contracting herpes zoster is greater than 50% for those 85 years of age or older. Approximately 1 million cases occur in the United States each year (CDC, 2016). There was an increase in reported episodes of herpes zoster from 1997/1998 to 2013/2014 by 49.5% (Friesen et al., 2016).

Age: This infection is most common in adults over 55 years old. The risk of herpes zoster increases with age (Dworkin et al., 2009; Sundström, 2015).

Gender: Women are diagnosed with 60% of the cases of herpes zoster (Insinga et al., 2005).

Ethnicity: Herpes zoster is 50% less common in the African American population (CDC, 2016).

Contributing Factors: Individuals are more likely to develop herpes zoster if they are more than 55 years old, have diabetes mellitus, have had an organ transplant, are immunosuppressed (e.g., HIV-infected patients), have certain malignancies, are receiving long-term corticosteroids, are receiving chemotherapy, or are taking radiation treatments. Physiological or psychological stressors can precipitate the development of herpes zoster (Fashner & Bell, 2011; Johnson, Wasner, Saddier, & Baron, 2008; CDC, 2016; Cohen et al., 2013).

Signs and Symptoms: Patients usually experience hyperesthesia with an itching, burning, or tingling pain at the site 4 to 5 days before the eruption appears. Pain in a dermatomal pattern may precede the appearance of the vesicles by weeks, however. Allodynia, the presence of pain without a painful stimulus applied to the area, may also be experienced (Ganty, 2014). Patients often experience a sense of anxiety, malaise, headache, fever, and flu-like symptoms with the onset of herpes zoster. Patients may report sleep disturbances, decreased appetite, and depression. There may be a sense of dysesthesia and paresthesia along the dermatome (Ferri, 2007; Cohen et al., 2013). Some patients complain of intense itching in the involved area (Dworkin et al., 2009). There may be reported pain around the eye if ophthalmic herpes zoster later develops. The eruption is maculopapular for a few hours and then becomes characterized as grouped vesicles on an erythematous base over one dermatome (usually). The vesicles are clear at first and then become cloudy within 3 to 5 days. T5 and T6 are the most common vertebral dermatomes involved, followed by cervical and sacral dermatomes. As vesicles age, they become pustular, then crust over after 7 to 10 days, finally healing in 2 to 4 weeks (CDC, 2016; Ganty, 2014).

Lesions may appear in irregular crops and are typically unilateral. Scarring and change in pigmentation may occur following the healing of the crusted vesicles. The most common distributions are on the trunk or face in elderly. Regional lymph nodes may or may not be swollen and tender (Fashner & Bell, 2011). The pain can last 6 to 12 months after disappearance of the rash (PHN). Ocular complications occur in about one-half of patients with involvement of the ophthalmic division of the trigeminal nerve. These complications may include keratitis, anterior uveitis, and corneal ulceration. Herpes zoster ophthalmicus occurs in 5% to 10% of patients with herpes zoster; permanent loss of vision or cranial nerve palsies can occur (Dworkin et al., 2009). Vesicles on the tip of the nose, known as Hutchinson's sign, have been found to be a precursor to ocular involvement in herpes zoster (Sampathkumar et al., 2009; Cohen et al., 2013). Cutaneous or visceral dissemination, the appearance of numerous varicella-like lesions in extradermatomal sites, may cause pneumonitis, encephalitis, meningitis, myelitis, and hepatitis (Fashner & Bell, 2011; CDC, 2016). The reactivation of VZV in the geniculate ganglion produces vesicles in the mucocutaneous distribution of the peripheral nerves. Known as Ramsey-Hunt syndrome, patients may experience vesicles in the pharynx and external auditory canal, tinnitus, vertigo, one-sided hearing loss, nausea and vomiting, involuntary eye movements and often paralysis on one side of the face (Sampathkumar et al., 2009; Cohen et al., 2013).

Diagnostic Tests: Diagnosis usually is based on clinical appearance and distribution of the eruption and on a careful history of when the rash appeared. The direct immunofluorescence with fluorescein-tagged antibody (DFA) or polymerase chain reaction (PCR) (if available) is preferred over the old standard Tzanck smear. These tests have far greater sensitivity and specificity than the Tzanck smear and allow differentiation between herpes simplex virus (HSV) and VZV infections. The DFA and PCR are useful in complicated cases and epidemiological studies, especially in immunocompromised individuals who may not present with a rash (Sampathkumar et al., 2009; Ganty, 2014). PCR is an expensive test, however, and the results of the test generally are not known for at least a day (Dworkin et al., 2007).

Differential Diagnosis: When the pain of pre-eruptive herpes occurs, the differential diagnosis depends on the dermatome involved:

- Migraine
- Myocardial infarction
- Acute abdomen
- Renal colic
- Pericarditis
- Pleuritis
- Pulmonary embolism

Once the rash or eruption appears:

- Contact allergic dermatitis (linear vesicles)
- Grouped vesicles (viral infection) (Fashner & Bell, 2011; Sampathkumar et al., 2009)

Treatment: Antiviral agents are recommended in the presence of significant pain, serious herpes zoster, or involvement near the eye. PHN is not prevented by antiviral therapy, but these agents may decrease PHN duration. These antivirals block the virus' DNA and block viral cell replication (Beuscher et al., 2017). Give acyclovir 800 mg five times a day for 7 to 10 days; famciclovir 500 mg orally every 8 hours for 7 days, or valacyclovir 1,000 mg orally every 8 hours for 7 days (Fashner & Bell, 2011; Cohen et al., 2013). These drugs must be given within 72 hours after onset of rash to be effective, and their use must be monitored in patients with reduced renal function (Tyring, Beutner, Tucker, Anderson, & Crooks, 2000; Beuscher et al., 2017). IV acyclovir 10 mg/kg every 8 hours is the treatment of choice for herpes zoster in these patients. Patients should be encouraged to stay hydrated and avoid scratching (Cadogan, 2010). Patients with disseminated disease and those who are immunocompromised may need IV antiviral medications (Cadogan, 2010). Topical agents are also effective in treating herpes zoster. The use of cool compresses with 1:20 Burow's solution, calamine lotion, and topical lidocaine (Xylocaine) is recommended for the soothing local effect.

Analgesics may be necessary for the initial prodromal pain associated with herpes zoster. Acetaminophen is recommended initially, but around the clock dosing is needed for optimal pain control. Tramadol, NSAIDs, and opiates are also used as recommended for severe pain. Acute pain may be diminished if antiviral treatment is begun within 72 hours (Cohen et al., 2013).

Gabapentin and pregabalin are recommended for the treatment of PHN. The initial dose of gabapentin is 300 mg on the first day and is titrated up gradually until pain relief is safely reached. The maximum does of gabapentin is 3,600 mg per day. Treatment with gabapentin with 1,800 mg per day was shown to significantly reduce PHN for up to 4 weeks in a study by Fan and colleagues (2014). Pregabalin is also recommended for the treatment of PHN and can be administered 50 mg three times a day or 75 mg twice a day. The dosage can be increased to 300 mg daily after 3 to 7 days as tolerated, followed by 150 mg every 3 to 7 days. The maximum recommended dose is 600 mg per day. Caution is advised when prescribing gabapentin and pregabalin to older

adults, given the side effects of dizziness and ataxia. Dosage adjustment for both of these medications is required for patients at risk for renal impairment (Christo, Hobelmann, & Maine, 2007; Cohen et al., 2013).

The secondary amine tricyclic antidepressants, nortriptyline 10 mg orally or desipramine 10 to 25 mg orally, both given at bedtime, may be helpful, and it may be necessary to gradually increase the dosage until reduction of pain occurs; however, because of the anticholinergic side effects, caution is warranted (Ahmad & Goucke, 2002). It is also recommended to have a baseline EKG done before treatment is begun with tricyclic antidepressants due to possible cardiac side effects (Gundy et al., 2014).

The use of opioids (e.g.., oxycodone) in the treatment of PHN alone or in combination with other therapies has also been studied; greater pain relief was experienced by patients when a combined regimen was prescribed over a single agent (Gilron et al., 2005; Liang et al., 2015). The 5% lidocaine patch has been shown to be the first line in treatment of the pain of PHN; one to three patches are applied in a 24-hour period. Common side effects are skin irritation and rash (Cohen et al., 2013). For PHN pain, capsaicin (Zostrix cream) has been proved beneficial in clinical trials and can be applied topically (Cohen et al., 2013). The capsaicin 8% patch can be applied by a health-care professional to the most painful skin areas (Christo et al., 2007). In cases of severe pain, a transcutaneous electrical nerve stimulator unit may be tried (Christo et al., 2007). Alternative treatments including acupuncture, cupping, and meditation have been shown to be beneficial in reducing pain as well (Cohen et al., 2013).

Follow-Up: Patients should be reexamined in 2 to 4 weeks to monitor progression of rash and as needed for follow-up of PHN.

Sequelae: PHN is the primary complication, occurring almost exclusively in people over 60 years old. Twenty percent of people over the age of 85 years diagnosed with herpes zoster develop PHN (Ganty et al., 2014). This pain persists at least 6 weeks after skin lesions. The pain, characterized as constant, severe, sharp, or burning, may develop into a long-standing, debilitating problem. PHN usually lasts 1 year or less. Some patients experience a post-herpetic itching sensation rather than pain (Dworkin et al., 2009; Oaklander, 2008). Another consequence of herpes zoster is secondary bacterial infection leading to cellulitis, caused by staphylococcus group A or group A β-hemolytic streptococcus. There is also an increased risk of an acute cardiovascular event, such as a cerebrovascular accident or myocardial infarction in the weeks to months after an episode of herpes zoster (Minassian et al., 2015).

Prevention/Prophylaxis: The new vaccine for herpes zoster (Shingrix) is available and is recommended for patients age 50 years and older. Even those people who have had herpes zoster should receive the vaccine to help prevent future occurrences of the disease (Resick, 2018). The immunization may be given without any serological testing, history of varicella, or history of herpes zoster. The vaccine is given in two doses, with the second dose to be given 2 to 6 months after the first dose. Patients should be scheduled for the second visit (Resnick, 2018). With the protection of the Zostavax vaccination waning within the first 5 years and protection uncertain (CDC, 2016), it is recommended that patients are covered with the Shingrix vaccination even if they have had the Zostavax vaccine in the past (Resnick, 2018). Practitioners are advised to review the listed contraindications before administering the herpes zoster vaccine. High-risk individuals, such as immunosuppressed patients and individuals who have not had chickenpox, should be kept from exposure to patients with herpes zoster. Second cases of herpes zoster are rare in immunocompetent patients given that one incidence of herpes zoster boosts immunity and prevents subsequent outbreak (Dworkin et al., 2009).

Referral: Because of probable ocular involvement, patients with lesions on the nose or in the eye area should be referred to an ophthalmologist. Patients with disseminated herpes zoster should be referred to a specialist. Refer patients with severe uncontrollable PHN to a neurologist or pain management clinic (Christo et al., 2007).

Education: Emphasize to the patient the need to stay home and get plenty of rest. Teach patients proper infection control measures and proper disposal of dressings or items of clothing that contain vesicle fluid, especially if they have known contacts that are vulnerable for developing varicella. Patients should avoid contact with immunosuppressed individuals, pregnant women, and individuals who have not had chickenpox.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Antiviral therapy should be initiated within 72 hours of onset of the maculopapular lesions to increase the healing of the herpes zoster and decrease the duration of PHN.	A	Tyring et al., 2000 Beuscher et al., 2017
Regular participation in tai chi boosted the cell-mediating effects of the herpes zoster vaccine.	B	Irwin et al., 2007
In the treatment of PHN, combined use of certain opioids and anticonvulsant drugs provided patients greater pain relief over a prescribed single agent.	A	Gilron et al., 2005 Liang et al., 2015

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The herpes zoster vaccine (Zostavax), unless contraindicated, should be given to persons >60 years old to prevent an outbreak of herpes zoster and subsequent development of PHN.	B	Hurley et al., 2010 Lu et al., 2009 Oxman et al., 2005 Cohen et al., 2013
The use of the high-concentration capsaicin patch provided sustained relief from pain in patients with PHN.	B	Irving et al., 2011 Ganty, 2014
The lidoderm 5% patch provided relief to patients experiencing pain from PHN.	B	Binder et al., 2009 Cohen et al., 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

PRESSURE INJURIES

Pressure injuries are classified through the use of staging systems. These systems describe the degree of tissue loss and the clinical presentation of the injury caused by pressure and/or shear. Pressure injury staging has become the basis for treatment, comparison of outcomes, and, if applicable, reimbursement. It is noteworthy that in April 2016 the National Pressure Ulcer Advisory Panel (NPUAP) updated the Pressure Injury Classification System following a rigorous literature review by a NPUAP-appointed task force, solicited input from stakeholders and the public, and the consensus conference held in Rosemont, Illinois with over 400 stakeholders in attendance. The revisions reflect current scientific and clinical understanding of the etiology of pressure injuries, as well as clarify and make the system more accurate and easy to use (Edsberg et al., 2016).

Only pressure injuries should be staged with the NPUAP Staging System. Non-pressure-related ulcers and wounds are subject to unique staging or classification systems based on wound type: diabetic foot ulcers (Wagner Classification System), venous leg ulcers (Clinical Etiology Anatomy Pathophysiology [CEAP]), skin tears (International Skin Tear Advisory Panel [ISTAP]), medical adhesive or tape injuries (Medical Adhesive Related Skin Injury [MARSI] categories), and burn classification (total body surface area). It is essential to confirm the presence of pressure and/or shear as a causative factor prior to using the NPUAP Staging System for an injury. Because the NPUAP Staging System is based on the extent of tissue damage, an understanding of anatomy is required prior to evaluating tissue types found in wounds. Finally, to ensure an accurate assessment, pressure injury staging should take place only after a wound has been thoroughly cleansed. In 2016, several revisions, which follow, were made to the NPUAP Staging System.

Ulcer Versus Injury: The 2016 NPUAP Pressure Injury Staging System used the term *injury* instead of *ulcer.* After an intensive review, the panel determined that the term *ulcer* did not accurately describe the clinical presentation of a stage 1 pressure injury or a deep tissue pressure injury (DTPI). In short, an ulcer cannot be present without an injury, but an injury can be present without an ulcer.

Roman Versus Arabic Numerals: Roman numerals were changed to Arabic numerals in each of the numbered stages. This change was made to clarify and reduce the potential for confusion between similar terms used in health care, such as Stage IV and intravenous (IV).

Artwork: Artwork was created to illustrate the features of each stage of pressure injury. It is a graphic representation of the tissues present and is to be used for teaching about the extent of injury and the tissues present or absent within each stage of pressure injury. It is available for free color download on the NPUAP website at www.npuap.org.

Suspected Removed, Pressure Inserted in DTPI: The word *suspected* has been removed from the term *deep tissue injury* and the word *pressure* inserted for consistency with the revisions of the other terms.

Teaching Points: Teaching points (key elements of understanding) have been included with each stage. Educators and those responsible for the accuracy, as well as the interrater reliability of serial assessments, can use these teaching points as elements of an orientation or skills competency program for bedside care providers.

Signal Symptoms: Intact skin with a localized area of nonblanchable erythema may be present, which may appear differently in darkly pigmented skin. It is usually over a bony prominence or related to a medical device. Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes. Partial thickness involves loss of skin with exposed dermis, intact serum-filled blister. Full thickness loss of skin with visible adipose (fat) tissue and/or deeper structures such as fascia, muscle, tendon, ligament, cartilage, or bone may be exposed. Wound edges may be open or present with epibole (closed or rolled edges). Depth may be obscured by the presence of necrotic

tissue, resulting in an unstageable pressure injury until such time when the wound bed becomes visible and the degree of tissue damage can be determined. Intact or nonintact skin may be present with a purple or maroon discoloration and/or an epidermal separation revealing a dark wound bed or blood-filled blister (NPUAP, 2016).

Description: A pressure injury is localized damage to the skin and underlying soft tissue usually over a bony prominence or related to a medical device. The injury can present as intact skin or an open ulcer and may be painful. The injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear. The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, comorbid conditions, and condition of the soft tissue. Common terms for pressure injury include *pressure ulcer, decubitus ulcer, bedsore,* and *pressure sore.* There are six pressure injury stages and two categories pertaining to etiology based on anatomical location (Edsberg et al., 2016):

- Stage 1: Intact skin with a localized area of nonblanchable erythema, which may appear differently in darkly pigmented skin. Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes. Color changes do not included purple or maroon discoloration; these may indicate DTPI.
- Stage 2: Partial thickness loss of skin with exposed dermis. The wound bed is visible, pink or red, moist, and may also present as an intact or ruptured serum-filled blister. Adipose (fat) is not visible and deeper tissue is not visible. Granulation tissue, slough, and eschar are not present. These injuries commonly result from adverse microclimate and shear in the skin over the pelvis and shear in the heel.
- Stage 3: Full thickness skin loss in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) is often present. Slough and/or eschar may be visible. The depth of tissue damage varies by anatomical location: areas of significant adiposity can develop deep wounds. Undermining and tunneling may occur. Fascia, muscle, tendon, ligament, cartilage, or bone is not exposed. If slough or eschar obscures the extent of tissue loss, this is an unstageable pressure injury.
- Stage 4: Full thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage, or bone in the ulcer. Slough and/or eschar may be visible. Epibole (rolled wound edges), undermining, and/or tunneling often occur. Depth varies by anatomical location. If slough or eschar obscures the extent of tissue loss, this is an unstageable pressure injury.
- Unstageable: Full thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar. If slough or eschar is removed, a stage 3 or stage 4 pressure injury will be revealed. Stable eschar (i.e., dry, adherent, intact without erythema or fluctuance) on ischemic limbs or heels should not be softened or removed.
- DTPI: Intact or nonintact skin with localized area of persistent nonblanchable deep red, maroon, purple discoloration, or epidermal separation revealing a dark wound bed or blood-filled blister. Pain and temperature change often precede skin color change. Discoloration may appear differently in darkly pigmented skin. This injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface. The wound may evolve rapidly to reveal the actual extent of tissue injury or may resolve without tissue loss. If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle, or other underlying structures are visible, this indicates a full thickness pressure injury (unstageable, stage 3, or stage 4). Do not use DTPI to describe vascular, traumatic, neuropathic, or dermatological conditions.

Etiology: While the science regarding the etiology of pressure injuries has supported the role of tissue deformation, microclimate, nutrition, perfusion, and tissue tolerance, the exact mechanism of pressure ulcer development is poorly understood (Edsberg et al., 2016). Classic references by Kottner, Balzer, Dassen, and Heinze (2009) describe four causation theories: ischemia caused by occlusion of the capillaries leading to vascular insufficiency, tissue anoxia, and cell death; reperfusion injury (i.e., cellular injury resulting from the reperfusion of blood to previously ischemic tissue); impairment of lymphatic function leading to a build-up of metabolic waste products; and mechanical deformation of tissue cells. The type of tissue involved is also important, with epidermis and dermis being more resilient to the effects of pressure than muscle (Kottner et al., 2009). Also, the type of force or combination of forces (i.e., pressure, shear, friction) exerted on the tissue is important. Pressure and shearing forces mainly affect deeper tissue layers, with moisture, pressure, and friction affecting primarily superficial layers (Kottner et al., 2009). Consequently, pressure is believed to be the major causative factor in pressure injury formation. Several factors play a role, however, in determining whether pressure is sufficient to create an injury. The pathological effect of excessive pressure on soft tissue can be attributed to intensity of pressure, duration of pressure, and tissue tolerance (the ability of skin and its supporting structures to endure pressure without adverse sequelae). Injury occurs to the skin and underlying tissues. Ischemia and hypoxia result as the pressure is applied to the area. Waste products accumulate as the ischemia continues, which produces toxins that cause further tissue breakdown.

Medical Device-Related Pressure Injury: Medical device-related pressure injuries result from the use of devices designed and applied for diagnostic or therapeutic purposes. The resultant pressure injury generally conforms to the pattern or shape of the device. The injury *should* be staged using the 2016 NPUAP Pressure Injury Staging System.

Mucosal Membrane Pressure Injury: Mucosal membrane pressure injuries are found on mucous membranes with a history of a medical device in use at the location of the injury. Mucosal tissues are especially vulnerable to pressure from medical devices such as oxygen tubing, endotracheal tubes, bite blocks, orogastric and nasogastric tubes, indwelling urinary catheters, and fecal containment devices. Because the 2016 NPUAP Pressure Injury Staging System for cutaneous injuries is based on the anatomy of the skin, it *cannot* be used to stage mucosal pressure injury.

Occurrence: Definitive information on the occurrence of pressure injuries is limited. Studies to date have been encumbered by methodological issues such as variability in describing the lesions and in differentiating pressure injuries from lesions of other etiologies. Consequently, pressure injuries are common; increase patient morbidity and mortality; and are costly for patients, their families, and the health-care system (Bauer et al., 2016).

The incidence in hospitalized patients ranges from 2.7% to 29% and the prevalence in hospitalized patients ranges from 3.5% to 69%. Patients in critical care units have an increased risk of pressure injuries, as evidenced by a 33% incidence and 41% prevalence. Elderly patients admitted to acute care hospitals for nonelective orthopedic procedures, such as hip replacement and treatment of long bone fractures, are at even greater risk, with a 66% incidence (Engels et al., 2016)

In the nursing home environment, the prevalence of pressure injuries is in the range of 2.6% to 24%. The incidence has been as high as 25% in residents admitted from an acute care hospital. Patients with pre-existing pressure injuries demonstrate a 26% incidence of additional pressure injury formation over a 6-month period. The incidence in chronic care hospitals is reported to be 10.8%, whereas 33% of those admitted to a chronic care hospital have pressure injuries. Long-term follow-up demonstrates that most injuries healed within a year. Persons with spinal cord injury and associated comorbidity are also at increased risk. The incidence of pressure injuries in this population is in the range of 25% to 66%.

Age: It has long been believed that comorbid conditions are associated with pressure injuries, and age is considered a comorbid state. Recent literature reviews suggest, however, that age is a confounding factor and a general indicator of likely deficits in the main areas of risk, including mobility/activity, skin status, perfusion and oxygenation, nutrition, and skin moisture (NPUAP, 2014). Prevalence of pressure injuries in the older adult population is 11.6% to 27.5%, with the increased risk being assigned to those of advancing age. The incidence in skilled care and nursing home facilities is approximately 25%. Patients admitted to a hospital geriatric unit have similarly high prevalence rates: of patients under 70 years of age, only 6% have pressure injuries; of patients over 70 years of age, the prevalence almost doubles to 11.6%.

Gender: Pressure injuries occur equally in men and in women.

Ethnicity: Cited in the literature and mentioned in the NPUAP 2016 Staging System Update is that there may be some difficulty in detecting stage 1 pressure injuries in persons with darker skin pigmentation, as the red-hued changes may appear more subtly in persons with darker skin tones.

Contributing Factors: The main contributing factors to pressure injury development are pressure, shear, adverse microclimate, and malnutrition. Moisture is cited as a related condition because it alters the resiliency of the epidermis to external forces. The primary offending source of moisture is incontinence, and more fecal incontinence than urinary incontinence due to the significantly higher pH of feces (liquid greater than solid) when compared to that of urine. The skin's pH is an acid mantle (i.e., 5.5). Understanding the concept and presentation of moisture associated skin damage (MASD), particularly the two subcategories of incontinence associated dermatitis (IAD) and intertriginous dermatitis (ITD), are essential in the differential diagnosis of pressure injuries from those partial thickness tissue injuries that are not resultant from pressure (Gray et al., 2011).

Signs and Symptoms: A detailed history of the injury should be obtained from the patient or caregiver, including chronic illness, hygiene, nutritional status, immobility, presence and type of prolonged moisture, ability to perform activities of daily living (ADLs), psychological impact of injury, and sources of available support. Ask the patient if he or she has pain over or near bony prominences or beneath medical devices.

Clinical presentation can vary from nonblanching erythema to partial thickness tissue loss and frank tissue necrosis. Nonblanching erythema results from damage to blood vessels and extravasation of blood into the tissue. Its presence suggests that more extensive tissue damage is imminent or has already occurred. The color of the skin can be varying hues of pink to red to darker toned hues of maroon or purple. Pressure-induced nonblanching erythema is occasionally misdiagnosed as a hematoma or ecchymosis. When DTPI is also present, the area is often either indurated or boggy when palpated. Note the injury size; obtain serial measurements of length, width, and depth; note the characteristic of the wound edge (open or epibole [rolled]); note the presence and character of exudate, epithelialization, granulation tissue, deeper structures, and findings such as necrotic tissue, sinus tracts, undermining, tunneling, and other signs of infection, such as induration, warmth, and/or advancing erythema.

Diagnostic Tests: General laboratory tests for the patients with a pressure injury include wound culture and sensitivity. Particularly look for any of the following:

- Signs of local infection (erythema, edema, change in exudate to include purulent or foul-smelling drainage, pain, crepitus)
- Signs of systemic infection (fever, leukocytosis); bone involvement (due to risk of osteomyelitis)
- Presence of chronicity to determine if there is a causative, elusive organism

Evaluation of nutritional status should include total protein, albumin, and prealbumin; in patients with a pressure injury, often these clinical indicators are decreased because of inadequate nutritional stores present for tissue repair. A CBC may reveal increased red blood cell distribution, decreased lymphocyte count (malnutrition), and elevated leukocyte count (osteomyelitis). The erythrocyte sedimentation rate will be increased. X-rays of the appropriate area often reveal bony rarefaction (decreased density), periosteal elevation, and new bone formation.

Differential Diagnosis:

- Skin cancers (neoplasms)
- Fungal or yeast infections
- Herpes simplex (HSV-2)
- Herpes zoster (shingles)
- Lower extremity wounds (venous, arterial, mixed etiology, and neuropathic etiology in origin)
- MASD, particularly IAD and ITD
- Burns (thermal injuries) (Doughty & McNichol, 2015)

Treatment: Effective pressure injury treatment with topical dressings requires consideration of numerous factors. Consider use of the mnemonic I DIP A MOP:

- **I**nfection (eliminate, reduce bioburden)
- **D**ebride necrotic tissue (consider: surgical/conservative, sharp, mechanical, enzymatic, biological, autolytic)
- **I**nsulate the wound
- **P**rotect periwound tissue
- **A**bsorb excess exudate
- **M**aintain a constant moisture level
- **O**bliterate dead space
- **P**revent further injury

Pharmacological Management: Most moisture-retentive dressings are available without a prescription. Some antimicrobials (e.g., silver sulfadiazine, mupirocin), some antifungals (e.g., fluconazole), and some enzymatic debriding agents (e.g., collagenase) are prescriptive.

Follow-Up: Practitioners need to objectively evaluate the patient's progress toward healing at least weekly using standardized methods of assessment (e.g., wound measurements in three dimensions, amount of exudate, patient comfort, etc.). Immediate intervention is indicated if wound deterioration is observed sooner. If the wound deteriorates, is it in accordance with a decline in the patient's overall condition? It is important to communicate this decline as an expected result to patient, caregivers, and staff. The patient's general health and nutritional intake, the stability of the comorbid conditions, the need for psychological support, and the patient's comfort level should be monitored. Signs and symptoms of complications need to be reported and recorded accurately.

Sequelae: Complications include, but are not limited to, cellulitis, bacteremia, osteomyelitis, and sepsis (secondary to pressure injuries).

Prevention/Prophylaxis: Conduct a valid and reliable risk assessment tool (e.g., Braden, Norton) at the first encounter and at regular intervals thereafter for patients at risk. Specific interventions are to be directed at the first encounter and at regular intervals following initial appraisal of skin and should be targeted toward improving subscale scores in any areas in which the patient's score falls below normal. Enlist the support of an interdisciplinary care team (e.g., nurse, physician, physical therapist, caregiver, dietitian, pharmacist) to ensure all goals of care are addressed adequately. Consider support surfaces for bed and/or chair when decreased mobility and moisture are risk factors (McNichol et al., 2015).

Referral: Consider referral to a wound care specialist (wound, ostomy, and continence [WOC] nurse, plastic surgeon, infectious disease practitioner, dermatologist) if wounds fail to progress in spite of evidence-based interventions consistent with the overall plan of care.

Education: Practitioners should provide patients and their caregivers with information that highlights the applicable contributing factors to the development of pressure injury formation and information about techniques being used (or those attempted, but not tolerated by the patient) pertaining to mitigation of those contributing factors.

- Review available and appropriate treatment modalities for each identified stage of wound progression, as well as expected outcomes and anticipated time frames for healing.
- Discuss reportable signs and symptoms of infection and complications.
- Discuss advances in technology and dispel myths that may exist regarding wound care.
- Identify informative Web-based resources and explain that information available via electronic means and/or from well-meaning individuals may not be evidence based.
- Discuss hand hygiene and disposal of soiled dressings.
- Review the importance of positioning, sources of dietary protein, and the amount to be consumed during the wound repair process.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Use a structured approach to risk assessment that includes assessment of activity/mobility and skin status.	B	NPUAP/EPUAP/PPPIA, 2014
Consider the impact of the following factors on an individual's risk of pressure ulcer (injury) development: • Perfusion and oxygenation • Poor nutritional status • Increased skin moisture	C	NPUAP/EPUAP/PPPIA, 2014
Assess localized pain as part of every skin assessment.	C	NPUAP/EPUAP/PPPIA, 2014
Inspect the skin under and around medical devices at least twice daily for the signs of pressure injury on the surrounding tissue.	C	NPUAP/EPUAP/PPPIA, 2014
Consider applying a polyurethane foam dressing to bony prominences (e.g., heels, sacrum) for the prevention of pressure ulcers (injuries) in anatomical areas frequently subjected to friction and shear.	B	NPUAP/EPUAP/PPPIA, 2014

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Offer fortified foods and/or high-calorie, high-protein oral nutritional supplements between meals if nutritional requirements cannot be achieved by dietary intake.	B	NPUAP/EPUAP/PPPIA, 2014
Turn and reposition all individuals at risk for or with existing pressure ulcers (injuries), unless contraindicated.	A	NPUAP/EPUAP/PPPIA, 2014
Use a high-specification, reactive foam mattress for all individuals assessed for being at risk for pressure ulcer (injury) development.	A	NPUAP/EPUAP/PPPIA, 2014
Consider pressure redistribution prior to and after surgery.	C	NPUAP/EPUAP/PPPIA, 2014

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

PSORIASIS

Signal Symptoms: There are several forms of psoriasis. The most common is plaque psoriasis, with erythematous patches, papules, and plaques with silvery scales that may be pruritic. Other forms include inverse psoriasis in intertriginous areas that appears as a smooth plaque; guttate, which is a rare form that presents with pustules; erythrodermic, which can be deadly with generalized rash; and rheumatoid varieties with joint involvement.

Description: Psoriasis is a disease of abnormal keratin synthesis. Inflammation in the lesions results in hyperproliferation with a shortening of the cell cycle that results in 28 times the normal production of epidermal cells (Habif, 2016; Wolff, Johnson, Saavedra & Roh, 2017). This results in the development of papules with a fine scale that coalesce and form distinctive, well-demarcated, round to oval plaques with a silvery scale. If the scale is removed traumatically, the presence of bleeding points, Auspitz sign, is noted. When the lesion is present in intertriginous areas, the moist environment macerates the scale, so the lesion appears as a smooth plaque. This is referred to as inverse psoriasis. Other less common forms of psoriasis include the guttate or acute eruptive psoriasis, which presents with small papules that are generalized over the trunk and extremities. Pustular psoriasis, in contrast to papular types, include pustular psoriasis of the palms and soles or generalized pustular psoriasis (Zumbusch's psoriasis), which is an acute systemic illness that may be fatal. Frequently, those with psoriasis have involvement of either the fingernails or toenails. Fingernail involvement is more common (Habif, 2016; Wolff et al., 2013).

Psoriasis can also affect the joints, causing a seronegative spondyloarthropathy, psoriatic arthritis. Other autoimmune diseases, such as inflammatory bowel disease, may also be present (Ferri, 2018). Generally, symptoms occur in those age 30 to 55 years. Joint symptoms occur before skin psoriasis in about 15% of cases. Symptoms include early morning stiffness; warm, red joints; and effusions.

John Updike is credited with first describing the "heartbreak of psoriasis." The phrase was used in advertisement in the 1960s, but it is a genuine concern because psoriasis may have a more profound effect on the emotional health of patients than on the physical health. The skin changes and stigma of psoriasis can result in depression, and depression can exacerbate psoriasis (Ferreira, Abreu, Reis, & Figueiredo, 2016).

Etiology: Recent understanding of the pathogenesis includes the probability of psoriasis being an immune-mediated disorder. This is supported by the responsiveness of psoriasis to treatment with immunosuppressive drugs. Although there appears to be a genetic link to psoriasis, it is a polygenic trait with an 8% incidence when one parent has psoriasis, and it increases to a 41% rate if both parents have been affected. About one in three patients with psoriasis has a family history of the disease. In addition to the genetics, environmental factors are often the trigger, including skin trauma (Koebner phenomenon), streptococcal or viral infection, stress, medications, smoking, and alcohol consumption (Ferreira et al., 2016; Patterson, 2016).

Occurrence: Psoriasis affects about 2% of the population of the United States, or about 7.5 million Americans. Most have less than 5% of their body surface affected. About 30% of those also have psoriatic arthritis (National Psoriasis Foundation, 2017). Chronic stable plaque psoriasis is the most common type, affecting 80% to 90% of those with psoriasis. Guttate psoriasis most often occurs in younger adults and represents about 10% of patients with psoriasis.

Age: The peak incidence of psoriasis occurs at 22.5 years of age, although it can occur even in childhood and may have a later onset at age 55 years or over. Women tend to have earlier onset, and earlier onset is associated with more severity of the disease. Psoriatic arthritis occurs most often in

those between the ages of 30 and 50 years (National Psoriasis Foundation, 2017).

Gender: There is an equal incidence of the disease in males and in females.

Ethnicity: Prevalence is highest among Scandinavians, with low incidence in West Africans, Japanese, and Inuits. There is a very low incidence among North and South American Indians. African Americans also have a low incidence of 1.3% compared with 2.5% of Caucasians.

Contributing Factors: Classic links to the development of psoriasis include drugs, physical trauma (Koebner's phenomenon), infection, and stress. A streptococcal or viral infection often precedes the onset of guttate psoriasis by 2 weeks. Drugs that may precipitate or exacerbate psoriasis include lithium, beta blockers, antimalarial agents, and systemic steroids. Several lifestyle factors or behaviors have been associated with a higher rate of psoriasis. Smoking was reported by 37% of those with psoriasis, and 78% of those reported smoking before they were diagnosed. This compares to a rate of smoking of only 13% in the general population. Psoriasis is more prevalent in those who abuse alcohol and may increase the severity of the disease. Multiple studies have noted a relationship between obesity and the development of psoriasis (Menter et al., 2011).

Signs and Symptoms: Patients may present with a papular rash that is getting worse, with shiny scales on the lesions. They may complain of pruritus. In guttate psoriasis, the lesions are distinct small papules with thin scales over the trunk and extremities. Chronic plaque psoriasis may involve a few plaques or may have many plaques scattered over the entire body. If the patient presents with a sudden onset of lesions, it is important to elicit a history of possible upper respiratory infection 2 to 3 weeks before the onset of the lesions.

Physical examination will reveal well-demarcated erythematous lesions with silvery scales. The scales may be thin or very thick. They are not easily removed, and if forcefully removed there may be points of bleeding, known as the Auspitz sign. The lesions of plaque psoriasis are generally symmetrical in distribution and may be located on the scalp, trunk, and buttocks, often affecting the extensor surface of the extremities. When the lesions form in the skinfolds, the moisture affects the development of scales, and the surface may be shiny. This inverse pattern psoriasis may be difficult to distinguish from *Candida* or tinea. The distinct demarcated border is helpful in differentiation. Nails also may be affected with the presence of pitting, onycholysis, subungual hyperkeratosis, red-brown coloring (oil drop sign), and nail plate dystrophy.

Diagnostic Tests: The distinct presentation of the lesions of psoriasis makes a clinical diagnosis possible. When fungal infections are a possibility, KOH tests are the gold standard for that diagnosis. Biopsies to confirm the diagnosis of psoriasis are rare, but when they are necessary, it is essential to take the sample from an intact lesion that has not been scratched and is not bleeding. If biopsy is required, a referral to a dermatologist will ensure accurate testing.

Laboratory testing, including blood chemistry profile, liver function tests, serum uric acid, a CBC with differential, antinuclear antibody titer, or rheumatoid factor, is directed at the inflammatory process and to rule out the possibility of infection. If the patient is presenting with guttate-type psoriasis, a throat culture to screen for streptococcus should be considered.

Differential Diagnosis:

Plaque psoriasis:
- Seborrheic dermatitis
- Nummular eczema
- Tinea corporis
- Lichen planus
- Pityriasis rubra pilaris
- Mycosis fungoides
- Atopic dermatitis

Guttate Psoriasis:
- Secondary syphilis
- Pityriasis rosea
- Maculopapular drug eruption

Inverse Psoriasis:
- Candidiasis
- Tinea

Psoriatic Nails:
- Tinea unguium
- Onychomycosis caused by yeast or molds

Treatment: While treatment decisions will generally be made by the dermatologist or, in the case of psoriatic arthritis, by the rheumatologist, the primary care provider is always responsible for the oversite of the patient's care. Knowledge of contraindications, side effects, adverse drug reactions, and drug-drug interactions are all a critical responsibility of the advanced practice nurse provider. Establishing good communication with the specialist is a necessity. It is also crucial that comorbidities which are common with psoriasis are optimally managed. The staging of psoriasis helps to determine the choice of treatment:

- Mild, affecting less than 5% of the body
- Moderate, affecting more than 5% but less than 10% of the body
- Severe, affecting more than 10% of the body

The hand is representative of about 1% of the body. When hands, feet, face, or genital areas are involved, because of the possible effect on ADLs, even with less percent involvement it may be considered more severe (Menter et al., 2011). The emotional impact of psoriasis is also considered in determining the severity and may increase the ranking even with less severe involvement. Eighty percent of patients have mild-to-moderate disease.

In addition to severity, the age of the patient and the type of psoriasis needs to be considered. To ensure the appropriate choice of treatment, referral to a dermatologist should be initiated. Dermatologists are the experts who treat and manage the multiple nuances of this disease. However, primary care providers need to be knowledgeable regarding the treatment options, because the treatments have implications for ongoing primary follow-up.

Topical Treatments: Topical treatments are the first line of treatment and recommended for mild-to-moderate psoriasis. For most patients in that category, the topical treatments are safe and effective. The treatments are also used as adjunctive

TABLE 6-4 Topical Treatment for Psoriasis

TOPICAL TREATMENT	MECHANISM OF ACTION	SIDE EFFECTS/PRECAUTIONS
Corticosteroids	Anti-inflammatory, antiproliferative, immunosuppressive, vasoconstrictive	Skin atrophy, telangiectasia, striae distensai, acne, folliculitis, and purpura
		May exacerbate some superficial skin conditions: rosacea, tinea
		Rebound of psoriasis
		With high potency, possible systemic side effects
Vitamin D analogues	Binds to vitamin D receptors	Burning, pruritus, edema, peeling, dryness, erythema may improve with continued treatment
Calcipotriene topical	Inhibition of keratinocyte proliferation and enhancement of differentiation	Rarely systemic effects of hypercalcemia and parathyroid suppression; apply after UVA therapy
Retinoids Tazarotene topical	Normalizes abnormal keratinocyte differentiation, diminishes hyperproliferation	Local irritation, may be reduced with lower concentrations; combine with moisturizers or topical steroids
	Decreases inflammation	
Calcineurin inhibitors Pimecrolimus topical	Block the synthesis of inflammatory cytokines	Burning and itching reduced with continued therapy
		Cautious use with light therapy
		Not FDA approved for psoriasis
Salicylic acid	Keratolytic; reduce keratinocyte-to-keratinocyte binding	Risk of systemic toxicity; do not use with oral salicylates
	Reduce pH stratum corneum	Use after UVA therapy
Anthralin	Prevent T lymph activation Normalize keratinocyte differentiation	Skin irritation and staining

therapy for patients with more extensive disease who are receiving phototherapy or systemic therapy. When making a choice of topical treatments, the patient's goals are important to consider. The vehicle for the medication should be chosen to match those goals and the severity of the disease. Problems with topical treatments include the time needed for application, the need for prolonged treatment, and incomplete clearance of lesions. All of these factors influence the patient's likelihood of adherence to therapy. Patients may be on concurrent topical therapy with the use of more than one topical agent. Agents include corticosteroids, vitamin D analogues, retinoids, calcineurin inhibitors, salicylic acid, anthralin, coal tar, and nonmedicated topical moisturizers (Table 6-4) (Menter et al., 2011).

Phototherapy: Phototherapy requires treatment by a dermatologist. Narrow-band ultraviolet B (UVB) is often used in combination with topical therapies and offers the possibility of home therapy. A more aggressive treatment is chemophototherapy with psolarens (an oral medication) and long-wavelength UV light (PUVA). The treatment inhibits mitosis by stopping DNA replication. It increases the risk of squamous cell carcinoma and possibly melanoma. It also induces photoaging. Although it has more risk than UVB, it is more effective (Menter et al., 2011).

Systemic Therapy: Systemic therapy is reserved for those patients with severe disease and requires management by a dermatologist with expertise in treatment of psoriasis. Methotrexate is very effective but has the potential for hepatotoxicity and should be avoided in renal impairment (this will eliminate many older adults). Drug interactions are common. Biologicals include T-cell and tumor necrosis factor (TNF) inhibitors. Baseline laboratory data are essential and need to be repeated frequently to monitor for toxicities. Live vaccines must be avoided in patients being treated with biologicals. All of the TNF inhibitors carry the increased risk of infection, especially upper respiratory infection. Coordinating the care of older adults on systemic therapy with the dermatologist is essential to adequate management (Menter et al., 2011).

Follow-Up: Follow-up should be on a case-by-case basis. A key to follow-up is coordination with the dermatologist, who, in other than the mildest cases, should be managing the disease process. It is important to consider the "heartbreak of psoriasis." The incidence of depression and suicidal ideation is higher in psoriasis and may not be directly related to the severity of the disease. All patients should be screened for depression. Patients with psoriasis have a higher incidence of arthritis, heart disease, diabetes, cancer, and hypertension, and all of these are increased in older adults as well (Menter et al., 2011).

Sequelae: Psoriasis is a chronic disease with exacerbations and remissions. In a small percentage of those with psoriasis, more serious types, such as psoriatic erythroderma or generalized acute pustular psoriasis, may occur. These diseases have much higher morbidity and may even result in death. Five percent of those with psoriasis will develop psoriatic arthritis.

Prevention/Prophylaxis: Triggers should be avoided: smoking, excess alcohol use, and skin trauma. Keeping the skin dry but well hydrated may decrease pruritus and the urge to scratch. Drugs known to exacerbate psoriasis should be avoided. Stress management, adequate rest, and a well-balanced diet to achieve ideal body weight are all important.

Referral: Except in the mildest cases of psoriasis, the patient should be co-managed with a dermatologist.

Education: In addition to ways to avoid triggers that can cause exacerbations, patients need to understand the treatment options and partner with providers to make choices that increase adherence. Teaching needs to include information regarding medication side effects and special considerations, such as the importance of not getting live vaccines if currently being treated with biologicals. In the current health-care environment, retail clinics and pharmacies are offering vaccines, so patients need to be especially conscious of the recommendations related to their treatments and the guidelines regarding vaccines. The emotional toll of this disease needs to be acknowledged, offering patients connection to support groups. Patients also need to be informed of reputable Internet sources on psoriasis.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Psoriasis is associated with depression and suicidal ideation. In one study of 217 patients with psoriasis, almost 10% indicated a wish to be dead, with 7.2% of patients with severe psoriasis indicating active suicidal ideation. This compares to a rate of 2.4%–3.3% in the general medical patient population. The recommendation to screen patients with psoriasis for depression and suicidal ideation is supported.	A	Gupta & Gupta, 1998 Ferreira, B. I. R. C. et al., 2016
Early studies of topical calcineurin inhibitors, tacrolimus, and pimecrolimus did not show efficacy, but when used under occlusion, they were efficacious. The conclusion that they lacked the ability to penetrate thick plaque was supported by their successful use in the treatment of thin lesions on the face and in intertriginous areas. Although not FDA-approved for treatment of psoriasis, two double-blind studies strongly support their effectiveness in treating psoriasis on the face or intertriginous areas, without the skin atrophy common with chronic steroid treatment.	A	Gribetz et al., 2004 Menter et al., 2011
Vitamin D analogue calcitriol, used in topical treatment of plaque psoriasis, was found to have slower onset of action but a longer disease-free period than betamethasone dipropionate. The risks of toxicity are minimal compared to topical steroids.	A	Menter et al., 2011
Tumor necrosis factor (TNF) inhibitors are an effective treatment for severe psoriasis and psoriatic arthritis. Side effects may discourage their use. However, for treatment of psoriasis, TNF inhibitors are only approved as monotherapy while they are often combined with methotrexate in the treatment of rheumatoid arthritis and inflammatory bowel disease. The risks associated with the combination therapy may overestimate the risk when using TNF inhibitors alone.	C	Menter et al., 2011

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SKIN CANCER

Signal Symptoms: Poikiloderma refers to telangiectasia, reticulate hyperpigmentation and atrophy. These are signs of long-term sun exposure and indicate significant risk for development of skin cancer. Squamous cell carcinoma (SCC) skin cancer may evolve from actinic keratosis, which present as discrete lesions or a cluster of crusted, scaly papules on sun-exposed skin. These are called actinic keratosis and may be tender or itchy. Any lesion with changing pigment, abnormal symmetry, crusting, bleeding, or tenderness is a possible skin cancer.

Description: Neoplasms of the skin are the most common type of cancers in humans. Non-melanoma skin cancers (NMSCs) are basal cell carcinoma (BCC), which arises from the basal keratinocyte layer of the epidermis and SCC, which originates in the squamous cells of the epithelium. Malignant melanoma (MM) is a tumor arising from pigmented cells. Melanoma causes abnormal proliferation of specialized cells that produce melanin in the skin, eyes, and hair.

Etiology: The major risk factor for the development of skin cancers is exposure to UV radiation (Welsh et al., 2011). Patients who have or had an occupation requiring them to spend extensive time outdoors are susceptible to the development of skin cancer. There is a high recurrence rate for BCC.

Occurrence: Skin cancer is the most common form of cancer in the United States. The incidence of NMSCs has increased by 100% in the Medicare population and by 35% in the fee-for-service population from 2006 to 2012 (Rogers, Weinstock, Feldman & Coldiron, 2015). BCCs have long been thought to be more prevalent than SCCs. Current research indicates changing trends. Rogers and colleagues (2015) found a 1:1 ratio in BCCs to SCCs in the United States, and global trends are similar. Each year, more than 1 million new cases of skin cancer are diagnosed in the United States. Melanoma occurs less frequently but is responsible for more deaths. It is estimated that over 9,000 persons will die of MM each year (Guy et al., 2015; U.S. Cancer Statistics Working Group, 2016). The incidence of MM has doubled from 1982 to 2011 (Guy et al., 2015). Melanoma, though deadly in the later stages of development, has an excellent prognosis if treated early (Guy et al., 2015).

Age: The incidence of all types of skin cancers increases with age and the degree and intensity of sun exposure.

Gender: SCC presents three times more frequently in men than in women (Nolen, Beebe, King, Bryn, & Limaye, 2011). MM is equally prevalent in men and in women. It poses significant mortality risks for both (Guy et al., 2015).

Ethnicity: Skin cancers are more prevalent in fair-skinned persons, blue-eyed persons (especially people with blonde, red, or light brown hair), and people of Celtic ancestry. In the United States, residents of states where year-round sunshine is abundant are at a high risk, especially those people who spend an extended amount of time outside. The highest incidence of BCC in the world occurs in Australia (Nolen et al., 2011). SCC is more common than BCC in darker skinned persons. Acral lentiginous melanomas (ALMs) are the most common melanoma subtype in dark skinned persons (Marchetti, Chung, & Halpern, 2015). They are characterized by location and are found on the palms, plantar surface of feet, and subungually. A pigmented streak of the cuticle, longitudinal melanonychia, is diagnostic (Marchetti et al., 2015).

Contributing Factors: Certain patient phenotype characteristics are associated with risk for developing skin cancer: albinism, hair and eye color, tendency to burn, and skin freckling (Lin et al., 2011). UV sun exposure is the most significant contributing factor to the development of skin cancer (Welsh et al., 2011). Other factors include tanning bed use, late sequelae to burns, scars, chronic ulcers, genetic disorders, and exposure to radiation (Welsh et al., 2011). Patients who report severe sunburns in childhood are at risk for developing skin cancer. Patients who have had ionizing radiation, whether as a treatment for a condition such as acne or accidental exposure, are at risk for developing BCC and SCC. Inorganic arsenic, pitch, tar, and radium exposure also have been linked to the development of skin cancer (Welsh et al., 2011). Patients who have had a renal transplant and immunosuppression are at risk for developing skin cancer and cancer of the lips (Nolen et al., 2011). Presence of multiple and atypical nevi are contributing factors (Welsh et al., 2011), but a recent study of over 500 persons with melanoma showed younger patients had few nevi and no atypical nevi preceding the diagnosis of melanoma (Geller et al., 2016). Human papillomavirus and xeroderma pigmentosum may also play a role in the development of SCC. Certain genetic predispositions can contribute to the development of skin cancer, and there is a familial tendency to develop melanoma. For patients diagnosed with familial atypical mole and melanoma syndrome or dysplastic nevus syndrome, there is a very high lifetime risk of developing melanoma (Haenssle et al., 2016).

Signs and Symptoms: Question patients about any new or changing lesions. Is the lesion painful? Does it itch or bleed? Has it healed and then reappeared? Ask about past history of any previous actinic keratosis, excised or treated lesions, and prior diagnosed skin of cancer. Question patients about any family history of skin cancers. Ask about their history of sun exposure, severe sunburn, burns, scars, ulcers, radiation dermatitis, use of tanning beds, current and prior occupations, and family history of skin cancer.

Presentation of the skin cancer will depend on the type of tumor. There are several types of BCCs. The most common are nodular, morpheaform, and superficial. Nodular BCC initially presents first as a dome-shaped papule that is white-to-pink with a raised pearly border and prominent telangiectasia (James, Elston, Berger, & Andrews, 2011). Over time, these lesions may develop a central erosion and bleed easily (James et al., 2011). BCCs can also be pigmented, with a brown glossy appearance, which comprise 6% of all BCCs (James et al., 2011). Patients may describe this lesion as a pimple that did not heal. Superficial BCC often appears on the trunk and extremities. Characteristics of superficial BCCs are the presence of a well-circumscribed translucent or bright pink to red patch of skin surrounded often by telangiectasia. This type of BCC resembles other chronic skin conditions

such as psoriasis, eczema, discoid lupus erythematous, and Bowen's disease. The least common type of BCC is the sclerosing or morpheaform tumor. Found on the head and neck and occasionally the trunk of the body, the lesion appears to be a hypopigmented tumor that eventually is surrounded by irregular telangiectasia with atrophic scar-like appearance (Nolen et al., 2011).

SCC often originates at sites of chronic inflammation or old scars. Actinic keratoses, precursors to SCC, appear as round or irregular-shaped erythematosus or tan plaques with a scaly or rough surface. Other precursor lesions of SCC include cutaneous horn, leukoplakia, and keratoacanthoma. These are often rapidly growing lesions that may arise from another benign, pre-malignant, or malignant lesion (James et al., 2011). Signs of malignancy of SCC include elevation, ulceration, or inflammation of the lesion; the original lesion also may have enlarged in size. In later stages of SCC, the surface may appear crusted, and a horn of keratin forms. SCC appears on sun-exposed as well as on non–sun-exposed areas of the body. They may be tender to touch, owing to their rapid growth and inflammatory process. Common locations are the scalp, ears, lower lip, and dorsa of the hands.

For patients with suspected melanoma, the mnemonic ABCDE guides the clinician in determining if the clinical characteristics of a suspicious lesion warrant close surveillance and/or biopsy for histological evaluation.

- Asymmetry
- Border irregularity
- Color variation
- Diameter greater than 6 mm
- Elevation of a previously flat lesion, evolving, and enlarging (Bibbins-Domingo et al., 2016)

It is important to note that the classic ABCDE pattern of MM development is not always present. The tool is used to alert patients and nondermatologists about suspicious lesions (Rastrelli et al., 2013). Melanoma can mimic benign lesions.

Major Signs: Change in size, change in shape, change in color. If one or more major signs exist, refer for expeditious biopsy.

Minor Signs: Inflammation, crusting or bleeding, sensory change, diameter of greater than 6 mm. If three or four minor signs exist with a major sign, consider referral.

Patients may be concerned about a new, pigmented lesion or a change in an already existing one. Patients may report associated itching, burning, or pain in a mole. Superficial spreading melanoma is a flat to slightly raised pigmented lesion with irregular borders, commonly found on the backs of men and the lower legs of women. Lentigo maligna melanoma, an irregularly pigmented macula with notched borders, occurs on sun-exposed areas, especially on the faces of older adults. Nodular melanoma, brown or black papules usually located on the trunk, head, and neck, is characterized by rapid growth. Clinical evaluation for skin cancer includes a total body skin examination and palpation of regional lymph nodes, liver, and spleen (Rastrelli et al., 2013).

Diagnostic Tests: Skin cancer is diagnosed through biopsy. Biopsy of the suspected lesion is necessary to confirm the diagnosis via histological examination of the tissue. An adequate tissue sample should be excised, and an elliptical excision generally is necessary for larger lesions. Excisional biopsy is recommended for any pigmented lesion (Rastrelli et al., 2013; Nolen et al., 2011).

Differential Diagnosis:

- Actinic keratoses
- Seborrheic keratoses
- Keratoacanthoma
- Atypical nevi
- Blue nevus
- Dermatofibroma
- Venous lakes
- Pyogenic granulomas
- Intradermal nevus
- Sebaceous hyperplasia
- Molluscum contagiosum
- Psoriasis
- Eczema
- Discoid lupus erythematous
- Bowen's disease (Nolen et al., 2011)

Treatment: Several factors need to be considered before skin cancer therapy begins: the patient's age and general health, whether or not the patient is immunocompromised, size and location of the tumor, the pathology of the tumor, and the cosmetic concerns of the patient. BCC may be treated by excisional surgery, electrodessication and curettage, cryotherapy, ionizing radiation, and Mohs' micrographic surgery (MMS). A chemotherapeutic agent such as 5-fluorouracil, interferon, and retinoids are topical agents for superficial or small BCCs and early-diagnosed SCC. SCC may be treated in the same manner as BCC; however, because of its more truculent growth pattern, wider excision and MMS are the preferred methods of treatment. Recurrent BCC and SCC should be treated with surgical excision (Nolen et al., 2011). Treatment of MM is surgical. An excisional margin surrounding the tumor is made, depending on the thickness of the tumor. Chemotherapy and radiation are used for palliative measures in the treatment of metastatic disease.

A number of different treatment options are available for the management of actinic keratosis (AK), including cryotherapy, topical chemotherapy using 5-fluorouracil, immunomodulators, and photodynamic therapy, all of which have been shown to reduce lesion count in patients with AK (Patel, Armstrong, & Eisen, 2014; Englet & Hughes, 2012). Cryotherapy, topical 5-fluorouracil, and photodynamic therapy are the most commonly used treatments for AK. A meta-analysis by Patel and colleagues (2014) found photodynamic therapy had a larger treatment effect on thin AKs of the scalp and face compared to cryotherapy and topical chemotherapy. It is relatively easy to use and is an effective treatment. Other treatments can include electrodessication and curettage or photodynamic therapy. Combination therapies can include both field-directed and lesion-directed therapy (Huang & Clark, 2011).

Follow-Up: Follow-up for a patient with diagnosed skin carcinoma is essential, because the recurrence rate of skin cancer is high; 50% of persons with BCC and SCC have a reappearance of a cancerous lesion within 5 years. A person who is susceptible to skin cancer may develop another cancerous lesion at any time. Precancerous lesions should be examined regularly every 6 to 12 months using a head-to-toe

skin examination, including a careful inspection of the previous site of a lesion. In patients with SCC, palpation of regional lymph nodes is suggested. During subsequent visits of patients with malignant melanoma, a thorough review of systems is imperative to elicit clinical signs and symptoms of metastasis.

Sequelae: BCC rarely metastasizes; however, if it is not treated early, the carcinoma may invade the surrounding tissue and bone. Advanced SCC lesions of the lips, pinna, and genitalia often metastasize. Recurrent NMSC has a higher rate of recurrence and eventually may lead to the development of metastatic disease (Nolen et al., 2011). The larger the NMSC in size, the higher the rate of recurrence. The anatomical location of the NMSC also influences the recurrence rate; lesions on the face, ears, vertex, and scalp are known as high-risk cancer areas. Five-year prognosis for MM is determined by the thickness of the tumor. Tumors less than 0.76 mm are associated with a 98% 5-year survival rate; tumors 0.76 to 1.49 mm, with an 87% to 94% 5-year survival rate; and tumors 1.50 to 3.99 mm, with a 66% to 83% 5-year survival rate. Patients with tumors greater than 4.0 mm have a less than 50% 5-year survival (American Cancer Society, 2016).

Prevention/Prophylaxis: Because the incidences of all skin cancers increase with age, it is important to discuss sun care protective behaviors with all older adults (Lin et al., 2011). The CDC recommends easy options for sun protection. Because UVR exposure increases the risk for skin cancer with all skin types, individuals need to seek shade when UVR is strongest, during the hours of 10:00 a.m. to 4:00 p.m., and avoid burning. According to the Skin Cancer Foundation, sunscreen should have a sun protective factor (SPF) of at least 30 or higher, broad spectrum, and water resistant for extended outdoor activity. Tanning beds or booths should be avoided, as they pose a significant risk for the development of melanoma (Biniol, Boyle & Gandini, 2012; CDC, 2016).

Areas of the body for sunscreen application include head, neck, and ears; exposed areas of the front of torso; back of torso; each arm; dorsum of hand and shoulder; each upper and lower leg; and each foot. One teaspoon of sunscreen should be applied separately to the upper leg and one teaspoon applied to the lower leg. It is necessary to reapply sunscreen after toweling off from swimming or exercise and after heavy sweating. Wide-brim hats that shade the face, head, ears, and neck; long-sleeved shirts; and long pants are recommended. Sunglasses that are wrap-around type should be worn and can be purchased to block up to 100% of UVR (U.S. Cancer Statistics Working Group Department, 2014).

Patients need to be routinely asked about their ability to conduct skin self-examinations. Recommending partner participation can enhance one's thoroughness in examining hard-to-reach areas of the body. Providing individuals with printed directions on how to position mirrors to examine the body, the use of good lighting, and suggesting magnifying lenses for those with impaired eyesight can help ensure a thorough examination has been carried out by the patient. Taking the time to give patients adequate instructions in skin self-examination has been found to be a successful strategy in ensuring older adults routinely conduct or seek assistance in regular skin examinations and the use of sunscreen (Janda et al., 2011). High-risk patients can be provided body maps to record areas of suspicious lesions to bring with them at the time of clinical examinations. These patients should be questioned if they are regularly being followed by a dermatology practice. If not, primary care providers are well positioned to begin triaging (to include referring patients to a dermatologist) patients who present with suspicious skin lesions (Goulart et al., 2011).

Yearly physical examinations should include assessment of the head, scalp, and skin, and an accurate recording of descriptions of any suspicious lesions. Patients should be fully undressed, to complete an accurate physical. New, unusual, or changing lesions or moles should be evaluated by a dermatologist. Lesions that have variegated colors, irregular elevations, or irregular borders should be examined by biopsy. Immunosuppressed organ recipients are at high risk for developing skin cancer at rates faster than the general patient population and should be scheduled for regular skin examinations.

Referral: When a suspicious lesion is found, referral to a dermatologist for evaluation and possibly biopsy is necessary. Whole-body photographs and dermoscopy may be used to follow patients with suspicious lesions. A tutorial on dermoscopy is available on the Internet at http://www.dermoscopy.org. Oncology referral is needed for metastatic SCC and MM.

Education: Advise older patients that skin cancers are a common occurrence as one ages, especially for patients at risk. Older adults should perform a monthly self-evaluation of the skin. Any suspicious lesion that does not heal in a reasonable time needs to be examined by a primary care provider. Patients also need to report any slow-growing, flesh-colored, or pigmented lesion, noting if the lesion has irregular borders, changes in color, ulceration, bleeding, or horn formation. Sun exposure, especially during the hours of 10:00 a.m. to 4:00 p.m., should be avoided. Year-round broad-spectrum sunscreen that blocks UVA and UVB light is recommended. Vulnerable areas, such as the head and neck, should be covered with protective clothing.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
In outdoor occupational settings the provision of information regarding sun protection to workers through instruction, small media, or both increase sun protective behaviors.	C	Guide to Community Preventive Services, August 2013
Multicomponent community wide interventions including a combination of: • Individual directed strategies • Mass media campaigns • Policy changes Is effective in increasing sunscreen use.	C	Guide to Community Preventive Services, August 2013
In high school and college education through class instruction, use of brochures/flyers, and participation in an educational program did improve self-care practices, including increased use of sunscreen and sunglasses.	B	Guide to Community Preventive Services, August 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.
Source: U.S. Department of Health and Human Services. *The Surgeon General's Call to Action to Prevent Skin Cancer.*

SUPERFICIAL FUNGAL INFECTIONS

Signal Symptoms

Dermatophyte Infection (Tinea): Erythema, scale, raised border, clearing center, may present with vesicles if inflammatory.

Candidiasis (Moniliasis): Pustules, red, denuded, glistening surface with a scaling border and satellite pustules. In the oral cavity and vagina, the scale and inflammatory cells form the classic "cottage cheese curds."

Tinea Versicolor (Pityriasis Versicolor): Hypopigmented macules and patches or scaly red or shades of brown macules or patches may be slightly pruritic.

Description:

Superficial fungal infections include the dermatophytes, candidiasis, and tinea (pityriasis) versicolor. The warm, moist surface of the skin allows fungal infections to survive. Fungal infections are not self-limiting and without treatment will spread (Salmon & Fuller, 2013). Dermatophyte infections are referred to clinically as *tinea* followed by the region of the body that is infected: tinea capitis (the head), tinea barbae (the beard area), tinea facea (the face except for the beard area), tinea manuum (the hand), tinea corporis (the body), tinea cruris (the groin), tinea pedis (the foot), and when there is nail involvement, tinea unguium or onychomycosis. Lay terms for the dermatophytes include ringworm, jock itch, and athlete's foot.

Candidiasis may be termed monilia, yeast, or thrush and generally presents in intertriginous areas or on mucous membranes. Tinea versicolor, which in almost all other countries is referred to as pityriasis versicolor, is not caused by a dermatophyte, and is therefore not a true tinea. It is a chronic scaling change in color that affects the areas of the body with increased sebaceous activity.

Etiology:

Dermatophytes: The dermatophytes are a group of fungi that infect the dead keratin of the stratum corneum, hair, and nails. They are limited to these areas because they require keratin for growth. They are responsible for most of the fungal infections of the skin, hair, and nails. Patients who are chronically infected may have a genetic predisposition to dermatophyte infection. One way to classify dermatophytes is by the origin of the infection: anthropophilic, transmission from person to person by fomites or direct contact; zoophilic, transmission from animal to person by fomites or direct contact; or geophilic, from the soil. The latter two sources result in a more rapid and more severe inflammatory response. Another classification is based on the tissue primarily involved: epidermomycosis when the epidermis is infected, trichomycosis when it involves the hair or hair shaft, and onychomycosis when the nail is affected. The dermatophytes within the stratum corneum disrupt the horny layer, lead to scaling, and create an inflammatory response. Three fungal species are primarily responsible for the infection: *Trichophyton, Epidermophyton,* and *Microsporum. T. rubrum* are the most common cause of skin and nail infection, with *Trichophyton* responsible for 80% of infections (Clinard & Smith, 2015; Wolff, Johnson, Saavedra, & Roh, 2017).

Cutaneous Candidiasis: Candida albicans is an oval, yeast-like fungus that lives in the normal flora of the mouth and gastrointestinal tract. It reproduces through budding, the development of pseudohyphae, and true hyphae. Conditions that compromise the immune system or alter the normal body flora contribute to the development of *Candida* infections. The yeast infects the outer layer of the skin with the primary

lesion, a pustule that dissects under the outer skin layer and peels it away. The result is a glistening surface with a scaling, advancing border and often the presence of satellite lesions. *Candida* grows best in warm, moist environments, and often the lesions end as they meet the area of dry skin.

Tinea Versicolor: Tinea versicolor is caused by organisms in the *Malassezias* species. (Wolff et al., 2017). The organisms are lipophilic yeasts that reside in the keratin and hair follicles, areas with increased sebaceous activity (Habif, 2016; Wolff et al., 2017). It is not known to be contagious, but is an overgrowth of a normal flora. As sebum production declines later in life, the incidence of tinea versicolor also declines.

Occurrence: Immunosuppressed patients are at higher risk for fungal infections of all kinds and are more likely to have infections that do not resolve. Older adults are at higher risk for many fungal infections because of the decline in the immune system with aging, as well as the increased incidence in comorbidities and the use of multiple medications. Functional compromise can lead to a decline in personal hygiene, which also increases the risk factors for fungal infection. Dermatophyte infections are increasing as there is an increase in travel, migration, and international sports (Salmon & Fuller, 2013). Tinea versicolor is more common in young adults due to its link to sebum production (Wolff et al., 2017).

The feet and nails are the most common areas of the body affected by dermatophytes in the older adult. Onychomycosis due to tinea unguium increases in incidence with advancing age, while toenail onychomycosis due to *Candida albicans* declines with aging. When the dermatophytes are inaccurately diagnosed and treated with topical steroids, a phenomenon called tinea incognito may occur. The rash initially disappears, but when treatment with steroids is discontinued, the rash returns with much more extensive involvement and may appear without scale. It is not responsive to subsequent topical steroid treatment, and a thorough history is necessary to make the proper diagnosis of tinea.

Candida most often causes intertriginous infections in those over 65 years old, but it can also be the cause of onychomycosis. Although it seldom causes infection in toenails, it may be the identified cause in half of all fingernail infections, especially in women and those who often immerse their hands in water (Gelotar, Vachhani, Patel, & Makwana, 2013). Tinea versicolor thrives in heat and humidity. It has an estimated prevalence in the U.S. population between 2% and 8%, however, the actual prevalence may be much higher because many of those with the disease do not seek medical evaluation.

Age: Tinea of the scalp and face (tinea capitis and tinea facialis) are primarily diseases of children. Tinea of the body, trunk, and extremities (tinea corporis) can occur at any age. The initial presentation of tinea pedis (athlete's foot) is most common in adults aged 20 to 50 years, and the initial presentation of tinea unguium (onychomycosis) commonly occurs in those age 45 to 54 years. Tinea unguium can affect any age, but the incidence in toenails increases with aging and is most common in the oldest old. Approximately 50% of those over 70 years old are affected (Wolff et al., 2017).

Candida of the skin occurs more often in older adults because of the decline in immune response with aging, as well as a decline in hygiene among many older adults. While not a common cause of toenail infection, when it occurs it is more likely in an older adult, while fingernail fungus is rarely seen in older adults.

Tinea versicolor is more common when sebaceous glands are most active during puberty and in young adults ages 15 to 24 years. Tinea versicolor is less common in those over 65 years old because sebum production declines with age (Wolff et al., 2017).

Gender: Tinea barbae is an infection that only occurs in males and is more common in rural areas. Males also more often experience tinea cruris and tinea pedis, and are more likely to have toenails infected with dermatophytes than females, while females are more likely to have *Candida*-infected fingernails. Gender differences have not been found in cases of *Candida* in other parts of the body or in cases of tinea versicolor.

Ethnicity: Although African Americans are less likely than other races to be diagnosed with dermatophytosis, African American children are at highest risk for tinea capitis. This is attributed to genetic and cultural factors, as well as socioeconomic status. Ethnic differences have not been noted for either *Candida* or tinea (pityriasis) versicolor.

Contributing Factors: Fungal infections are more common in warmer climates. Excessive sweating increases risk. Any of the diseases that compromise the immune system, atopy, and topical and systemic steroids increase risk. Diabetics are of noted higher risk and those with abnormalities of the keratin, as in psoriasis and ichthyosis (Nenoff et al., 2013). Age alone increases the risk, particularly of onychomycosis. Tinea manuum is rare in individuals who do not also have tinea pedis. A common presentation is two feet–one hand syndrome, which is believed to be a result of the patient touching or scratching the infected feet. Tinea pedis occurs more frequently in those who wear occlusive footwear that promotes warmth and sweating, the perfect environment for fungal growth. Socioeconomic status also appears to contribute to the incidence of dermatophyte infections, with tinea pedis more prevalent in developed countries and tinea capitis more prevalent in poorer countries.

Cutaneous candidiasis is more common in adults with diseases that compromise the immune system, those taking medications that cause immune decline, adults treated with broad-spectrum antibiotics, and those with poor hygiene that results in moisture in intertriginous areas (Habif, 2016; Wolff, et al., 2017). Other contributing factors include hot, humid weather; tight underwear; poor hygiene; and inflammatory diseases occurring in skinfolds.

Like other fungal infections, tinea (pityriasis) versicolor is more common in an environment of heat and humidity. It grows best where sebaceous activity is highest and may be more common in those with oily skin. Immune-compromised individuals and those on steroids are also more susceptible. While the color change may be more noticeable on black skin, there is no noted racial difference (Habif, 2016; Wolff et al., 2017).

Signs and Symptoms: Well-defined borders with scale are a hallmark of each of the superficial fungal infections. Tinea is often dry and scaly, while *Candida* presents with initial pustules that become eroded, confluent, and evolve into patches with sharply demarcated borders and fine scale, surrounded by satellite pustules. Either can also be noted in thickened,

discolored nails, onychomycosis. As the infection in the nail progresses, the nail becomes brittle, splinters easily, and the nail plate may pull away from the nail bed (onycholysis).

Tinea capitis presents as hair loss (alopecia). Gray patch tinea capitis often is circular in shape with the presence of many broken-off hairs and the scalp assumes a dull gray color due to the arthrospores that are formed by the fungi. Black dot tinea capitis occurs when hairs break off near the surface and give the appearance of dots. The dots may be scattered over the scalp and not form a classic round patch. A kerion is an inflammatory mass on the scalp that is painful and may include pustules and result in cervical or occipital adenopathy.

Tinea of the face and body presents as the classic ringworm pattern with a clearing center and a raised, scaling leading edge. Tinea cruris presents as an erythematous pruritic rash that begins in the groin and spreads onto the thighs. It is usually unilateral and rarely involves the scrotum. The lack of scrotum involvement can help to differentiate tinea from *Candida* of the groin, where scrotum involvement is common (Habif, 2016).

Tinea pedis, athlete's foot, is a common dermatophyte infection and presents with many different signs and symptoms. Habif (2016) notes that although it may present in the classic ringworm pattern, involvement between the toes or on the soles of the feet is more common. Three primary types are interdigital, moccasin type, and vesicular.

- Interdigital tinea pedis results in erythema and scaling present between the fourth and fifth toes, although any interdigital space can be affected. Secondary infection with bacteria is more common in the macerated toe space.
- Moccasin-type tinea pedis affects the entire sole of the foot, and the chronic silvery white scale may also occur on the palms of the hands, where it is referred to as painter's palm. The creases of the palms and the soles of the feet are thickened and the creases are white in color, hence the appearance of paint that was not fully washed away. Because of the mild nature of this infection and its subtle presentation, it may be present for years before the patient seeks treatment.
- Vesicular-type tinea pedis may appear on the sole or the top of the foot and may represent a secondary bacterial infection in someone with chronic interdigital tinea pedis. It can also occur as an id reaction to the fungal infection, in which case the blisters may even involve remote sites.

Toenails are most often infected with dermatophytes, and there are three classic presentations:

- Distal and lateral subungual onychomycosis (DLSO), which presents with onycholysis (the pulling away of the nail plate from the nail bed), hyperkeratosis, and yellow-brown discoloration.
- Superficial white onychomycosis (SWO), in which the dermatophyte invades the surface of the nail and the dorsal nail plate is chalky white.
- Proximal subungual onychomycosis (PSO) appears as a chalky, white color at the base of the nail.

When a patient presents with a nail problem it is important to think broadly about the differential, as only 50% of nail problems are onychomycosis (Westerberg & Voyak, 2013). Not every toenail with abnormal appearance is infected with fungus. Other common considerations should be trauma, frequent immersion in water, psoriasis, atopic dermatitis, or HIV infection.

Cutaneous candidiasis occurs intertrigo, in large skinfolds and interdigital areas, as well as in nails. Habif (2010) describes two presentations of *Candida*: 1) Pustules form, become macerated in the skinfold, and develop papules with a fringe of moist scale at the border and intact pustules outside the intertriginous area; 2) Red, moist, and shiny lesions form that lack the pustules, because they macerate as soon as they form. Small intact pustules appear outside the border, and these satellite lesions are helpful in making the diagnosis.

Tinea (pityriasis) versicolor appears as macular, well-demarcated lesions that are light brown on untanned skin and white on tanned skin. The lesions vary in size and may become confluent and cover large areas of the body. They are finely scaled and sometimes pruritic. The trunk is the most common area of the body affected, but the lesions can spread to the upper arms, neck, and face.

Diagnostic Tests: History of the concern is critical in diagnosis, because in most superficial fungal infections diagnosis is primarily presumptive, based on clinical presentation. The fungal lesions evolve and for the most part are asymptomatic, so they may be found coincidentally by the provider on physical examination or presented by the patient when self-treatments have failed. Taking the history along with inspection is recommended. Asking the patient to identify what part of the lesion looks like the initial presentation allows for identification of the primary lesion and can sort out confusing signs of lesions that are secondary and may mask the primary disease.

For fungal skin involvement, the gold standard of diagnosis is direct microscopic examination of a potassium hydroxide wet mount preparation (Habif, 2016). Scale is obtained using a surgical blade and placed on a slide. Although fungal infection in the hair or nails may need to be cultured, most tinea does not require culture because treatments are effective against any of the specific species. Culture may also be needed for definitive diagnosis of lesions on the foot. Because nail involvement will require prolonged oral treatment to eradicate the infective agent, clinical diagnoses should be confirmed by laboratory testing. Approximately 90% of nail infections are due to dermatophytes; 8% are caused by a mold, *Scopulariopsis;* and 2% are due to a yeast, *Candida,* that may not respond to treatments useful in treating dermatophytes (Habif, 2016; Westerberg & Voyak, 2013). Garcia-Doval and colleagues (2010) present a clinical diagnostic rule for tinea unguium. In their study, they found that if the patient with suspected onychomycosis had greater than 25% involvement of the sole of the foot with plantar desquamation and the presence of interdigital tinea pedis, the positive predictive value of the rule was 81%, which is higher than that of laboratory tests.

Differential Diagnosis:

Scalp Lesions:

- Seborrheic dermatitis
- Psoriasis
- Atopic dermatitis
- Alopecia areata
- Bacterial folliculitis
- Neoplasia needs to be considered with presence of a kerion

Lesions on the Trunk:
- Allergic contact dermatitis
- Atopic dermatitis
- Nummular eczema
- Psoriasis
- Pityriasis rosea
- Tinea (pityriasis) versicolor
- Early presentation of herpes zoster

Lesions in the Groin:
- Intertrigo
- Psoriasis
- Seborrheic dermatitis
- Erythrasma

Lesions on the Feet: Between the toes:
- Bacterial infections: erythrasma, impetigo, *Pseudomonas*
- *Candida* intertrigo

Moccasin type:

- Allergic contact dermatitis
- Atopic dermatitis
- Dyshidrotic eczema
- Psoriasis

Vesicular:

- Allergic contact dermatitis
- Dyshidrotic eczema
- Bacterial infection
- Bullous disease

Nail Involvement: Distal and lateral subungual onychomycosis (DLSO):

- Psoriatic nails
- Onychogryphosis (thickening and hardening)
- Onychauxis (overgrowth or thickening)

Superficial white onychomycosis (SWO):

- Trauma
- Contact or irritant dermatitis

Tinea Versicolor:
- Vitiligo
- Pityriasis alba
- Seborrheic dermatitis
- Secondary syphilis
- Pityriasis rosea

Treatment: In older adults, response to treatment may be decreased due to slower cell turnover and comorbid conditions, particularly diabetes and peripheral vascular disease. Tables 6-5 and 6-6 offer topical and systemic treatment options, respectively. For topical agents, the treatment is twice a day for 2 to 4 weeks and should be continued for 1 week after the lesions clear. Application should extend beyond the leading edge of the lesion. Within a class of medication, the options are comparable and should be selected based on cost and the preferred base (lotion, cream, ointment). Topical treatments are not useful for tinea capitis, and failure is common in tinea manuum and tinea unguium.

Newer agents are more likely to cure tinea pedis than the older generation of antifungals, including clotrimazole, which is fungistatic, whereas terbinafine is fungicidal. Terbinafine 1% cream applied twice daily for 1 week has a high cure rate for interdigital tinea pedis and butenafine twice a day for a week is also very effective (Habif, 2016). Econazole and ciclopirox also have antibacterial properties and may be the drugs of choice if bacterial involvement is suspected.

When tinea pedis is vesicular, the use of aluminum acetate solution (Burow's solution) compresses applied for 30 minutes several times a day is a helpful drying agent and facilitates the penetration of the topical antifungal treatment. Oral antifungal drugs may be used to treat the acute infection (Habif, 2016). In hyperkeratotic, moccasin-type tinea, keratolytic agents, such as ammonium lactate lotion or topical urea, can reduce the hyperkeratosis and allow for improved penetration. Applying a plastic occlusive dressing over the keratolytic agents enhances their effectiveness. Oral terbinafine daily for 4 weeks may result in cure rates of 95% (Habif,

TABLE 6-5 Topical Treatment for Superficial Fungal Infections

DRUG CLASS	PREPARATION	USE
Azoles	Clotrimazole	Dermatophytes of the trunk or extremities
	Miconazole	Cutaneous candidiasis
	Ketoconazole	Tinea (pityriasis) versicolor
	Econazole	
	Oxiconizole	
	Sulconizole	
Allylamines	Naftifine	Dermatophytes of the trunk or extremities
	Terbinafine	Tinea (pityriasis) versicolor
	Butenafine	
Naphthionates	Tolnaftate	Dermatophytes of the trunk or extremities
Pyridone	Ciclopirox	Dermatophytes of the trunk or extremities
		Candidiasis
		Tinea (pityriasis) versicolor
		Onychomycosis SWO
Polyene	Nystatin	Candidiasis

TABLE 6-6 Systemic Treatment for Superficial Fungal Infections

DRUG CLASS	PREPARATION	USE
Allylamines	Terbinafine	Tinea corporis, cruris, capitis, pedis
		Onychomychosis
Azoles	Itraconazole	Dermatophyte infections
	Fluconazole	Tinea (pityriasis) versicolor
		Onychomychosis
Mitotic inhibitor	Griseofulvin	Tinea corporis, cruris, capitis, pedis
		Tinea (pityriasis) versicolor

2016). For tinea pedis it is important to assess the condition of the nails and treat any reservoir that may be present.

Topical treatments are rarely useful in the treatment of tinea unguium, except for SWO. In the United States, three topical treatments are approved by the FDA. Ciclopirox topical (Penlac Nail Lacquer) is the oldest; new agents include tavaborole (Kerydin) and efinaconazole (Jublia). Ciclopirox alone has a clinical cure rate of only 6% to 9%, however, it is also used in combination with oral agents and in that combination had a complete cure rate of 68% (Westerberg & Voyak, 2013). An intriguing alternative was presented by Derby, Rohal, Jackson, Beutler, and Olsen (2011). In a small study (including only 18 subjects) of the effectiveness of the lay treatment of onychomycosis with Vicks VapoRub applied topically once a day for 48 weeks, 83% of the participants reported satisfactory results, with 27.8% experiencing a complete clinical and mycological cure; 56% had partial clearance, and 17% had no improvement.

An alternative treatment of tinea (pityriasis) versicolor is selenium sulfide 2.5% lotion or shampoo applied for 10 to 15 minutes followed by showering off for 2 weeks. Weekly application for several additional months may prevent recurrence. The skin color alterations may take 1 to 2 months to resolve completely.

Research supports a variety of dosing options for all of the oral treatments, from daily to once-a-month pulse therapy. The duration of therapy depends on the site of involvement as well as the response. Although oral terbinafine is least toxic of the antifungals, all of the agents have the potential of serious side effects. Before recommending any oral treatment, consultation is recommended.

Mechanical treatments of nails infected with fungus offer another option for resolution of the infection. Total nail removal is an option, but can have unintended consequences.

Follow-Up: Follow-up of the patient with superficial fungal infections needs to be addressed on a case-by-case basis. A follow-up in 4 weeks is a reasonable option, especially for those patients on oral therapy, where hepatitis may appear after 4 weeks of treatment (Baran et al., 2008). For oral therapy, a baseline CBC and liver function tests are recommended with retesting every 4 to 6 weeks (Habif, 2016). Infection of the toenails requires 6 weeks or longer to judge results, and in tinea versicolor 1 to 2 months are required before the lesions resolve. Patient education regarding expected outcomes and possible side effects can help in the assessment of when or even whether follow-up is necessary.

Sequelae: The superficial fungal infections for the most part do not present a serious health threat to older adults. Their lack of symptoms is one reason they are often chronic before they are addressed. In older adults, onychomycosis can result in cellulitis and foot ulcers. The risk of superinfection with bacteria and resulting severe consequences is greatest in those with diabetes, peripheral vascular disease, or immune compromise. The *Candida* species may have systemic consequences and are often painful; treatment and prevention are key. The side effects of the treatments may be more concerning than the disease, and these risks are important to weigh in decision making. Serious consequences include hepatic toxicity, CyP-450 interactions, neutropenia, and agranulocytosis.

Prevention/Prophylaxis: All fungus grows best in warm, humid environments. The key to prevention of all types is to keep the skin cool and dry. Avoiding occlusive footwear, wearing absorbent materials, and practicing good hygiene offer the best primary prevention. Tinea pedis, tinea manuum, and onychomycosis are difficult to completely eradicate; recurrence is common, and the diseases may become chronic in nature. The interdependence of these infections requires the complete resolution of all three in order to prevent recurrence. Tinea versicolor is the result of the conversion of the normal *Malassezia* skin flora to a mycelial form that is pathological and causes the scaly lesions. Older age actually decreases the risk of this disease, because sebaceous activity declines with age. Prophylactic therapy with topical or oral antifungals has been effective in preventing recurrence. The *Candida* species are part of the normal flora in the mouth, gut, and vaginal tract. The key to prevention of recurrence is keeping the skinfolds dry. Use of a hairdryer to thoroughly dry the area after bathing, the use of aluminum acetate solution (Burow's solution), and the application of antifungal or absorbent powder have all been shown to prevent recurrence (Habif, 2016; Wolff et al., 2017).

Referral: When definitive diagnosis is needed for suspected fungal infections of the skin, hair, or nails, referral is often necessary so that direct visualization of a wet mount preparation can be performed. When the hair or nails are affected, systemic treatment is usually required. A culture is necessary to rule out the other diseases that can mimic fungal infection and determine the causative organism.

Education: Patient education should center on the cause of the infection and the risk of reinfection/recurrence. Prevention requires avoidance of situations that cause occlusive, warm, moist environments. This can be accomplished by wearing absorbent materials, loose clothing, and open-toe shoes in the summer, and wearing protective footwear in showers and around pools. Patients can be reassured that tinea versicolor is the result of a change in the normal body flora and not contagious, and that coloration changes will resolve with treatment. Prophylactic antifungal therapy may be required, especially for tinea pedis, tinea manuum, or onychomycosis.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
A rule for the clinical diagnosis of onychomycosis was developed. Data supported that the rule was at least as accurate as laboratory tests. When dermatologists considered onychomycosis was the most likely diagnosis and plantar desquamation affected >25% of the sole of the foot, treatment could be started without further confirmation. The rule needs to be validated with primary care practitioners.	B	Garcia-Doval et al., 2010

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Oral ketoconazole is not a recommended treatment for dermatophyte infections. Newer antifungals are more effective, and it is the most toxic antifungal with the risk of hepatitis and drug interactions.	A	Habif, 2016
A small study of topical Vicks VapoRub applied to nails affected with fungus confirmed by microscopy and culture was found to be as effective as topical ciclopirox 8%, the only FDA-approved treatment for onychomycosis. Participants were followed for 48 weeks, and the outcome measure was clinical and mycological cure. Patient satisfaction was also measured. The cost of treatment is less than one-fourth that of the prescription treatment.	B	Derby et al., 2011
White socks are not useful in the prevention of tinea pedis. The color of the socks does not make a difference; the fabric does. Cotton socks are preferable to synthetics.	C	Habif, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

Mrs. Jones is a 70-year-old woman you are admitting to the skilled nursing facility after a total hip replacement. When you obtain her past medical history, she reports a problem with a chronic rash under her breasts and in her supragluteal fold. She has tried a number of topical antifungal treatments without success. She has never seen a dermatologist. While she finds the rash a nuisance and experiences an occasional increase in itchiness, it is something she can live with. Her primary care provider diagnosed the rash as *Candida* and encouraged her to keep the area clean and dry and to use antifungals. Recently, her 42-year-old daughter was diagnosed with psoriasis and asked about a family history of the disease. Mrs. Jones was unaware of anyone in her or her husband's family with that diagnosis.

On physical examination, you note a shiny, erythematous lesion with symmetrical distribution under both breasts. It is well demarcated without drainage and without the presence of any satellite lesions. Examination of the gluteal fold reveals a similar well-demarcated, shiny, erythematous lesion without drainage. On close inspection of the rest of the body around the umbilicus, you note an erythematous patch with silvery scale. You ask Mrs. Jones about the lesion, and she states that it has been there for years. The lesion is worse in the winter, but she attributes this to the closures on her skirts and pants causing irritation. She reports some improvement when applying OTC hydrocortisone cream, although when she stops using the cream, the lesion returns. For this case, the answers and rationale for your decision making need to be provided.

1. What clues in Mrs. Jones' history can help you determine her diagnosis?
2. What signs on physical examination are most helpful to defend or refute the differential?
3. How would you classify the severity of Mrs. Jones' disease? Be specific in your explanation.
4. What would be the treatment(s) of choice in her case?
5. What are some important points to include in patient education?
6. What would require a referral to a dermatologist?

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CHAPTER 7

Head, Neck, and Face Disorders

Lori Martin-Plank

ASSESSMENT

A systematic and thorough examination of the head and neck begins with the inspection of the face, head, and scalp. Assess the shape, size, and symmetry of the patient's features. Examine the shape of the skull, palpating the bones of the head for any anatomical irregularities, masses, or areas of tenderness. Inspect the hair for distribution, quantity, and any balding patterns, noting any uniform alopecia, nits, or seborrhea. The scalp should be carefully inspected for any skin lesions on sun-exposed areas, noting pigment changes, tenderness, scales, or lumps. Assess for scars or bruising patterns. Note the symmetry of the patient's features, facial expressions, the presence of involuntary tics or tremors, periorbital edema, or facial drooping; palpate over the temples for any abnormality or tenderness in the temporal arteries. The presence of pain and nodules on palpation is not a normal finding even in very old individuals. Cranial nerve VII (facial nerve) should also be assessed at this time, noting any facial asymmetry, weakness, drooping of the lower eyelid, and unilateral paralysis (Bickley & Szilagyi, 2012; LeBlond, Brown, Suneja & Szot, 2015).

Eyes

Begin the inspection of the eyes by noting the position and symmetry of the surrounding skin and tissue, as well as the presence and position of the eyebrows and eyelashes. Screen visual acuity before proceeding with any other examination of the eye. Using a Snellen chart from 20 feet, or a handheld chart approximately 12 inches from the patient, assess visual acuity in each eye separately, then together; if the patient wears corrective lenses, he or she should have them on for this test. Note the condition of the skin around the eyes, check the sclera, and note the color of the conjunctiva. Assess the presence of exophthalmos, xanthelasma, pinguecula, ptosis, edema, and skin lesions. The lids should be carefully examined for the presence of hordeolum, chalazion, ectropion, and entropion, which is most common among elderly persons. Examine the cornea for any scarring, presence of pterygium, corneal arcus, and opacities. Note that basal and squamous cell carcinomas are common around the eye. Palpate for any tenderness over the lacrimal gland and assess the patency of the lacrimal duct. Examine the extraocular motions of the eye (cranial nerves III, IV, and VI). Check the visual fields (cranial nerve II), corneal reflexes (cranial nerve V), and pupillary reactions (cranial nerves II and III) with direct and consensual reactions. Carefully perform a funduscopic examination; most elderly patients need dilation for accurate assessment of the fundus. If you see the fundus, note the narrow, pale appearance of the arterioles common in elderly persons. Be careful to note any abnormalities in blinking (Parkinson's disease), dull or blank staring (hypothyroidism), residual facial paralysis (Bell's palsy or cerebrovascular accident), and skin changes. Decreased elasticity and turgor is a normal aging pattern; the skin around the eyes becomes thin and wrinkles appear. This is a normal change in older adults and makes skin turgor a poor determinant of hydration status (Bickley & Szilagyi, 2012; LeBlond et al., 2015).

Sinuses

Inspect and palpate over the frontal and maxillary sinus areas. Note any gross tenderness or inflammation in the sinus area or around the eyes.

Nose

Inspect the external nose for any asymmetry, inflammation, gross septal deviation, or deformities. If applicable, assess the function of the olfactory nerve (cranial nerve I) by having the patient identify a familiar-smelling item with the eyes closed. The olfactory sense greatly diminishes as a normal part of aging. Assess for patency of each nostril. Examine the internal nose, noting any discharge, bleeding, or edema. Check the status of the turbinates and position of the septum using a large otoscope speculum. The color and consistency of the inferior and middle turbinates as well as the presence of any polyps should be noted (Bickley & Szilagyi, 2012).

Ears

Carefully examine the external ears, noting their position and symmetry on the head, as well as any abnormal lesions, deformities, and the presence of tophi, keloids, or cysts. Estimate auditory acuity (cranial nerve VIII) by using the whisper test, testing each ear separately. Use the Weber and Rinne tests

to assess for any conductive or sensorineural hearing loss. Palpate the tragus for any tenderness, as well as for any preauricular or postauricular adenopathy. When examining the middle ear, note any inflammation, discharge, erythema, or cerumen in the canal. If visible, inspect the tympanic membranes and surrounding landmarks for abnormalities. Note any foreign bodies, dull membranes, alterations in the cone of light reflex, and presence of fluid or scarring. Be careful to inspect the posterior ear and helix for any skin lesions or carcinomas (LeBlond et al., 2015).

Oral Cavity

Perform a complete assessment of the lips, mouth, oral mucosa, and pharynx, noting the color, moisture, and presence of any abnormal lesions on and around the lips. Assess for any herpes simplex I, chancres, angular stomatitis, mucous retention cysts, angioedema, and fissures. Check oral mucosa for color, nodules, ulcers, or white patches (Bickley & Szilagyi, 2012). If the patient has dentures, these should be removed to do a complete oral examination. Examine the fit of the dentures and assess the consistency of the gums under the dentures. If natural teeth are present, note any loose or broken teeth as well as caries. Because periodontal disease is the primary cause of tooth loss in the adult, do a careful oral and gum examination. Examine the gums for bleeding, discoloration, swelling, and retractions. Note the attrition of the teeth (exposed dentin) and enamel loss from years of chewing.

Note the condition of the hard palate and the presence of torus palatinus, thrush, or other lesions. Assess the gag reflex and rise of the palate (cranial nerves IX and X). Carefully examine the tongue for symmetry, enlargement (hypothyroidism), growths, protrusions, or abnormal movements (cranial nerve XII) and the dorsum for any papillary atrophy. A swollen, red, painful tongue may indicate vitamin B or riboflavin deficiency. Note any inflammation or obstruction in the parotid (Stensen) or submaxillary (Warton) ducts. Thoroughly examine the area underneath the tongue, the floor of the mouth, and the tonsils, soft palate, uvula, and posterior pharynx, noting any lesions, inflammation, or exudate and the color. Examine the strength and movement of the temporal and masseter muscles (cranial nerve V), as well as any crepitus in the mandible junction. Complete assessment should also include evaluation of the voice (cranial nerve X) and speech (cranial nerves V, VII, X, and XII).

Neck

Inspect the neck for symmetry, masses, scars, tracheal position, and deviation. Look for the presence of thyroid inflammation or goiters. Carefully palpate for any lymphadenopathy, noting that loss of lean muscle makes it easier to feel nodes in the cervical region of elderly persons. Assess supraclavicular, tonsilar, superficial, deep, and posterior cervical chains and submaxillary, submental, occipital, preauricular, and postauricular nodes, noting any inflammation, tenderness, or change in size, position, or shape. Hard, fixed nodes imply malignancy; tender nodes are typical of inflammation (Bickley & Szilagyi, 2012). Gently palpate and auscultate the carotid arteries one side at a time for any nodularity or bruits. Note any jugular vein distention that occurs when the patient is seated. Check to see if the patient uses any neck muscles to breathe. Carefully inspect and palpate the thyroid gland, noting any inflammation or nodules (unilateral). Examination of the range of motion of the neck should include flexion, extension, rotation, and lateral bending. Check the strength of the trapezius and sternomastoid muscles (cranial nerve XI).

A complete and thorough detailed examination of the head and neck has the potential to allow the examiner to discover multiple variants from the normal. Develop a clear and concise pattern of examination to ensure appropriate evaluation of these areas. Keep in mind the normal variants of the older person and how these differ from pathological or abnormal findings.

CATARACT

Signal Symptoms: Diminished vision in one or both eyes, poor night vision, sensitivity to glare. Because progression is gradual, changes may go unnoticed until visual loss is advanced.

Description: A cataract is an opacification of the lens that interferes with the passage of light through the lens, decreasing visual acuity. The location, size, and density of this opacity influence the degree of visual impairment (Harper, 2018; Liu et al., 2017). Nuclear sclerotic cataracts affect contrast sensitivity, progress slowly, and tend to preserve functional reading vision while frequently causing nearsightedness. In contrast, posterior subcapsular cataracts progress more rapidly and interfere with reading vision.

Etiology: Most cataracts are related to the aging process. The gradual thickening or hardening in the lens is believed to be caused by oxidative damage to the lens protein.

Occurrence: The rate of visually significant cataracts in persons 50 to 59 years old is 6.8%. Of persons over 80 years old, 68.3% have cataracts (Harper, 2018). Cataracts tend to be bilateral, although the rate of progression varies between the eyes.

Age: Although subtle changes in visual acuity related to cataract formation occur by age 50, people over 75 years old experience most visually significant cataracts.

Gender: Cataracts occur equally in men and women.

Ethnicity: Not significant.

Contributing Factors: Diabetes mellitus, hypertension, poor nutrition, cigarette smoking, high alcohol intake, trauma to the eye, long-term exposure to ultraviolet (UV) B radiation (sunlight), and a strong family history of cataracts are risk factors. Use of certain substances, including oral

glucocorticoids, also contributes to cataracts. An association between cataracts and glaucoma and intraocular inflammation exists (Jacobs, 2016).

Signs and Symptoms: The patient with a cataract initially may present with an improvement in near vision, requiring a new prescription for corrective lenses; distance vision will worsen (Harper, 2018). Patients also may experience blurred vision or sensitivity to glare from bright light or from automobile headlights during night driving. Complaints of having difficulty reading and distinguishing contrast sensitivities and of seeing a yellow tint or washed-out colors are common. Some patients may not seek evaluation of their symptoms by a health-care provider owing to denial or fear of loss of independence (driver's license being revoked), whereas other patients seek prompt assessment in the hope of early intervention. A recent history of falls, accidents, or injury is suspicious. On physical examination, visual acuity test results are abnormal for one or both eyes (evaluate near and distance vision). Examine the eye using an opaque light for opacification of the lens. The cataract may be visible to the naked eye. The red reflex may be absent, or the cataract may appear as a black area.

Diagnostic Tests: No diagnostic testing is required other than that for visual acuity and examination of the eye, unless other visual problems are suspected. Refer the patient to an ophthalmologist for complete evaluation after initial screening. Pupillary dilation and slit lamp examination will reveal white, gray, or brownish opacities if the cataract is developed. Dark areas on the red reflex in a dilated eye indicate small cataracts; the red reflex may not be visible if the cataract is large (Jacobs, 2016).

Differential Diagnosis:

- Corneal scarring
- Retinal detachment
- Macular degeneration
- Chronic glaucoma
- Diabetic retinopathy

Treatment: Treatment is determined after full evaluation by the ophthalmologist. Ultimately, surgical intervention is required with extraction of the cataract and immediate implantation of a plastic intraocular lens, unless contraindicated by other disease conditions (Thompson & Lakhani, 2015). Standard treatment is phacoemulsification. Recently, laser has been added to assist with this, but outcomes are no better with laser (Day, Gore, Bunce, & Evans, 2016). A variety of types of implantable lenses are used depending on patient vision assessment and lifestyle. These include lenses that adjust for astigmatism and refractive errors. Patients with posterior capsular opacification may require neodymium:yttrium-aluminum-garnet (Nd:YAG) laser capsulotomy. This should not be performed at the same time as other cataract surgery. Collaboration with the ophthalmologist is indicated for patients with severe cardiac, respiratory, or neuromuscular conditions that may prevent the patient from lying still or supine as required during the surgery. Patients with diabetes and hypertension should be instructed in medication use preoperatively. Patients receiving anticoagulant therapy also should be managed collaboratively and individually. Patients on clopidogrel after cardiac stent are encouraged to delay surgery until the clopidogrel is no longer needed (Jacobs, 2017). There is no hard and fast rule for patients on aspirin or warfarin maintenance (Jacobs, 2017). Patients receiving anticoagulant therapy also should be managed collaboratively and individually. Patients who are or have been on alpha receptor therapy, such as tamsulosin for prostatic enlargement, may experience intraoperative floppy iris syndrome (IFIS). Treatment of these patients may require special techniques during surgery to prevent this (Olson et al., 2016; Thompson & Lakhani, 2015). Although still considered an alternate practice pattern, intracameral antibiotic injection during surgery is becoming more common and has been shown to reduce rates of endopthalmitis (Hashemian et al., 2016; Herrinton et al., 2016; Javitt, 2016).

The degree of visual impairment and its influence on the patient's usual daily functioning determine the timing of the surgery (Preferred Practice Panel-Cataract, 2016; Thompson & Lakhani, 2015). Initially, a conservative treatment plan may include a prescription for glasses and periodic reevaluation by the ophthalmologist, coupled with a plan for environmental safety and optimization of the patient's functional abilities within his or her visual limitations. Surgery is performed in an outpatient or short procedure unit, with the patient returning home immediately after discharge. With the advent of multifocal implantable lenses and toric lenses for astigmatism, glasses may not be required after surgery, however, insurers currently only support monofocal lenses. More research needs to be done before multifocal lenses are perfected (Alio et al., 2017; deSilva et al., 2016; Harper, 2018; Lawless et al., 2016; Thompson & Lakhani, 2015).

Follow-Up: Immediate postsurgical care includes eye protection and application of topical agents prescribed by the ophthalmologist. The patient may be cautioned to avoid straining, lifting, or bending. Postoperative follow-up is usually within 24 to 48 hours. The family or health-care team should heighten precautions for environmental safety to avoid injury. Intensive supervision of mentally compromised patients is required to prevent damage to the operative site. Future follow-up includes a reevaluation of visual acuity, prescription of corrective lenses if indicated, and monitoring of the unaffected eye for cataract development.

Sequelae: Possible sequelae to cataract surgery include faulty wound closure with aqueous humor leakage, blindness secondary to choroidal hemorrhage, endophthalmitis, toxic anterior segment syndrome (TASS), inflammation, retinal detachment, prolapse of the iris into the corneal wound, and secondary glaucoma (Preferred Practice Panel-Cataract, 2016; Thompson & Lakhani, 2015).

Prevention/Prophylaxis: The patient should be protected from exposure to UVB radiation by wearing sunglasses designed for this and by wearing a hat with a wide brim.

Referral: After the initial visual screening, refer the patient to an ophthalmologist for complete examination and treatment, including postsurgery follow-up.

Education: Provide the patient with information on age-related visual changes and the importance of protecting the eyes from sunlight with UV-blocking sunglass lenses; maintaining a nutritionally balanced diet including green, leafy vegetables; avoiding tobacco and high alcohol intake; and having periodic eye examinations every 2 years. Instruct the patient to seek prompt evaluation of any vision changes, and reassure the patient that with proper treatment cataracts do not result in permanent loss of visual acuity (Jacobs, 2017).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
There is no evidence from randomized, controlled trials (RCTs) that supplementation with antioxidant vitamins (beta carotene, vitamin A, or vitamin E) prevents or slows the progression of age-related cataract.	A	Olson et al., 2016
The decision to recommend cataract surgery should be based on consideration of the following factors: visual acuity, visual impairment, and the potential for functional benefits.	B	Thompson & Lakhani, 2015 Olson et al., 2016
Ophthalmologists and other physicians managing patients taking alpha antagonists should be aware of the risks of intraoperative floppy iris syndrome (IFIS).	B	Jacobs, 2017 Thompson & Lakhani, 2015
Extracapsular cataract surgery by phacoemulsification with intraocular lens (IOL) implantation provides safe and effective treatment for patients with cataract.	A	Olson et al., 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

EPISTAXIS

Signal Symptoms: Bleeding from the nose or into the posterior nasopharynx.

Etiology: Ninety percent of the time the bleeding is the result of a spontaneous rupture of a blood vessel in the anterior septum in Kiesselbach's plexus (Morgan & Kellerman, 2014). Local causes of epistaxis include trauma, irritation or inflammation of the nasal mucosa, a septal defect, or paranasal tumors. Systemic causes include arteriolosclerosis, hematological disorders, or hereditary hemorrhagic telangiectasias (Nguyen, 2016; Scott, 2014). Approximately 10% of nosebleeds are idiopathic.

Occurrence: Most people have at least one episode of minor, nonrecurring epistaxis. Because self-treatment is often effective and the episode is not reported, statistical data are scarce. Several sources indicate a lifetime incidence of 60% with fewer than 10% requiring medical intervention (Morgan & Kellerman, 2014; Nguyen, 2016). Typically, people older than age 50 years have more serious bleeding that requires medical attention (Morgan & Kellerman, 2014).

Age: Epistaxis occurs most frequently in young children and in elderly persons (Nguyen, 2016).

Gender: Epistaxis occurs more frequently in males than in females (Nguyen, 2016).

Ethnicity: Not significant.

Contributing Factors: Local trauma to the anterior portions of the nose (digital, as in nose picking, and blunt, as in nasal fractures) is usually the cause of epistaxis. Low humidity in desert climates or from central heating, foreign bodies, intranasal polyps or neoplasms, irritants (smoking), intranasal steroids, inflammation from rhinosinusitis, and systemic medications such as NSAIDs, anticoagulants, and antiplatelets may contribute secondarily to the occurrence of epistaxis. Septal abnormalities also are contributing factors. Systemic diseases including hypertension; bleeding disorders such as aplastic anemia, leukemia, and thrombocytopenia; liver disease; and hereditary coagulopathies may contribute to the occurrence of epistaxis (Blomgren, 2014; Morgan & Kellerman, 2014; Nguyen, 2016).

Signs and Symptoms: Usually no signs or symptoms are associated with epistaxis other than external bleeding or an awareness of blood dripping down the posterior nasopharynx.

Diagnostic Tests: No laboratory studies are indicated for minor and nonrecurring epistaxis. For more substantial or recurrent epistaxis, a complete blood count (CBC) including platelet count, bleeding time, prothrombin time (PT), and partial thromboplastin time may be useful. In a patient who is on warfarin therapy, an international normalized ratio (INR) should be drawn if epistaxis occurs (Ruhl & Swindell, 2015). If bleeding is severe and the need for blood replacement is anticipated, type and crossmatch also should be done.

Differential Diagnosis:

- Trauma
- Inflammation due to rhinitis or infection
- Vascular disorders
- Clotting disorders and blood dyscrasias
- Intranasal foreign bodies
- Hereditary telangiectasia
- Tumors

Treatment: Most episodes of epistaxis are mild and self-limited, resolving spontaneously. Epistaxis may be controlled by pinching the nostrils together for 5 to 10 minutes, keeping the patient in an erect sitting position with head tilted forward to prevent blood from going down the posterior nasopharynx. Anterior bleeding that does not stop with manual pressure alone may be controlled with two sprays of oxymetazolone 0.05% applied directly to the bleeding site followed by 10 to 15 minutes of manual pressure (Morgan & Kellerman, 2014). The mucous membrane can be topically anesthetized with 4% lidocaine, and a silver nitrate stick then applied to the bleeding site once it is located. Other devices include nasal tampons such as Merocel or balloons for packing (Scott, 2014). The user must be skilled in handling these devices properly. If packing is needed, adequate pain management must be prescribed and it should be removed within 2 to 3 days (Ruhl & Swindell, 2015).

Follow-Up: For recurrent episodes, follow-up visits are recommended as needed. If medications have been implicated as likely causes, follow-up by a home health nurse for medication management education may be helpful. Posterior epistaxis, comorbidities, or recurrent bleeding may necessitate admission or referral to an ear, nose, and throat (ENT) specialist for management (Ruhl & Swindell, 2015).

Sequelae: Anemia can be a complication of excessive or frequent bouts of epistaxis. Infection can result from nasal packing. Severe blood loss can exacerbate existing diseases. Older patients must be assessed for dyspnea, chest pain/pressure, and syncope (Morgan & Kellerman, 2014).

Prevention/Prophylaxis: Discourage patients from nose picking and rubbing, and excessive forceful blowing of the nose; advise them to increase the humidity in the home, especially in winter months. Some patients may be prescribed bacitracin, saline gel, or petrolatum to apply twice weekly in the area of Kiesselbach's plexus (Nguyen, 2016; Scott, 2014).

Referral: Consultation with an ENT specialist is indicated for the following conditions:

- Bleeding that is not controlled after 15 minutes of compression
- Evidence of massive bleeding
- Recurrent bleeding within the first hour
- Second episode of epistaxis within 1 week
- Uncontrolled bleeding from the posterior nasopharynx

The consulting physician may initiate interventions, such as the insertion of Merocel nasal tampons or an intranasal balloon to control bleeding. Intractable epistaxis may require transantral arterial ligation or embolization (Nguyen, 2016).

Education: Discourage picking of the nose and advise patients to increase the humidity in the home, especially in winter months, by means of a humidifier. Petrolatum or saline gel may be rubbed over the nasal septum twice daily to decrease dryness of mucosa. Teach the patient how to manage simple epistaxis at home, with instructions to seek medical attention for excessive (bleeding for more than 1 hour) or recurrent epistaxis. Use of nasal saline spray for humidification is encouraged. Prolonged hot showers and hot, spicy foods should be avoided. Instruct the patient to avoid nose blowing and to open the mouth when sneezing (Nguyen, 2016).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Simple measures are helpful in stopping most nosebleeds: nose blowing to clear clots, pinching the nose for 15 minutes, and topical decongestants.	C	Ruhl & Swindell, 2018
Cauterizing visible bleeding vessels with silver nitrate ($AgNO_3$) or electrocautery are equally effective.	B	Ruhl & Swindell, 2018
Patients with resistant or posterior bleeds are likely to need posterior packing, surgery, or embolization.	C	Ruhl & Swindell, 2018
Routine coagulation tests for epistaxis are not useful unless the patient is taking an anticoagulant.	B	Ruhl & Swindell, 2018

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GLAUCOMA, ACUTE AND CHRONIC

Glaucoma encompasses several different eye diseases characterized by progressive neuropathy of the optic disc, leading to loss of vision. The optic disc is composed of vascular, neural, and connective tissue. The axons of the retinal ganglion cells form a rim around a shallow cup in the optic disc. Apoptosis of the ganglion cells of the retina leads to a thinning of the nerve fibers in the inner layer and axons, with atrophy of the disc and the appearance of cupping or expansion of the cup/disc ratio (Salmon, 2017; Weinreb, Aung, & Medeiros, 2014). The two most common glaucomas are primary angle closure with acute or chronic closure and primary open-angle (POAG). Current research has identified specific genetic associations, but research in this area is ongoing.

GLAUCOMA, ACUTE (PRIMARY ANGLE-CLOSURE)

Signal Symptoms: Unilateral eye pain, visual blurring with halos around lights, conjunctival injection, and photophobia; nausea and vomiting may also occur. Approximately two-thirds of patients with angle-closure glaucoma will have vision loss without experiencing an acute episode.

Description: Acute glaucoma, also known as *angle-closure* or *narrow-angle glaucoma*, is an obstruction to the outflow of aqueous humor from the posterior to the anterior chamber through the trabecular meshwork, canal of Schlemm, and associated structures. It typically results in an elevation of intraocular pressure, damaging the optic nerve and causing loss of peripheral vision, eye pain, and redness. This type of glaucoma is uncommon but may occur as a primary disease or secondary to other conditions, and constitutes an ophthalmic emergency (Hoffman, 2018; Salmon, 2017; Weinreb et al., 2014). Associated presenting symptoms may complicate diagnosis or result in misdiagnosis.

Etiology: The precise pathophysiology of angle-closure glaucoma is unknown. It is a disorder of the lens, iris, and retrolenticular system. In the acute form, pupillary blockage limits the progress of the aqueous humor through the trabecular network. The peripheral iris, which blocks the trabecular meshwork, is displaced forward (Mantravadi & Vadhar, 2016; Weinreb et al., 2014).

Occurrence: Uncommon; accounts for 10% of all glaucoma. This is more common in families with prior history.

Age: The predominant age range for acute glaucoma is 60 to 70 years old.

Gender: Acute glaucoma occurs more often in women.

Ethnicity: Acute and primary angle-closure glaucoma are more common in Inuit and Asian populations.

Contributing Factors: Contributing factors include an anatomically narrow anterior chamber angle, requiring identification with the use of a special examination technique called *gonioscopy*. This examination technique is beyond the scope of the primary care practitioner, but the condition can be evaluated during the comprehensive eye examination. Other risk factors include hyperopia; small cornea; shallow anterior chamber; Asian ancestry; female gender; family history of glaucoma (first-degree relatives have a 2% to 5% risk over a lifetime); and use of antihistamines, a/b-Adrenergic agonists, tricyclic antidepressants, SSRIs and SNRIs, anticholinergics and cholinergic agents—these drugs may precipitate an acute episode (Mantravadi & Vadhar, 2016; Prum et al., 2015).

Signs and Symptoms: The presentation is often dramatic, but the diagnosis can be missed because of the associated symptoms. The history reveals severe, unilateral eye pain; blurred vision; lacrimation; reports of seeing colored halos around lights; and a red eye. Headache, nausea, and vomiting frequently accompany eye pain, causing eye pain to be overlooked. Emotional stress also is common. Examination reveals circumcorneal, conjunctival injection, tearing, and a fixed mid-dilated pupil that is nonreactive to light; the cornea is steamy or cloudy. Visual acuity, if evaluated, shows a loss in the affected eye.

Diagnostic Tests: Immediately refer patients for a complete ophthalmic examination (Mantravadi & Vadhar, 2016; Weinreb et al., 2014).

Differential Diagnosis:

- Conjunctivitis
- Hyphema
- Uveitis
- Corneal trauma or infection

Treatment: The patient with acute glaucoma needs an immediate consultation with and referral to an ophthalmologist. Permanent visual loss occurs within 2 to 5 days if this condition is untreated. Surgical treatment includes peripheral iridectomy or laser iridotomy. Intraocular pressure must be lowered preoperatively, which may require the use of an osmotic diuretic intravenously or orally and miotic eye drops. As the primary health-care provider, you must communicate to the specialist any medical conditions that need monitoring with the use of these agents. Bilateral treatment is indicated, because patients are at risk for developing the same problem in the other eye (Mantravadi & Vadhar, 2016; Prum et al., 2015)

Follow-Up: The ophthalmologist treating the patient determines follow-up treatment. Periodic eye examinations are recommended by preventive guidelines.

Sequelae: If treated promptly, acute glaucoma is not associated with sequelae. If untreated, permanent visual loss occurs.

Prevention/Prophylaxis: Knowing the risk factors for acute angle-closure glaucoma, the health-care provider should educate patients about those risk factors (see under Education). Periodic eye examinations are recommended for prevention.

Referral: Refer the patient immediately on presentation and evaluation of symptoms.

Education: Teach patients with known risk factors (see under Contributing Factors) the importance of regular eye examinations and reporting of symptoms.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Acute angle-closure glaucoma is an ocular emergency that requires immediate lowering of IOP with topical/oral/parenteral medications, usually followed by iridectomy or laser iridotomy.	A	Prum et al., 2016
For acute angle-closure glaucoma, topical and systemic agents are usually necessary to adequately decrease the IOP, and laser iridotomy is the definitive surgical treatment.	A	Prum et al., 2016

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GLAUCOMA, CHRONIC (PRIMARY OPEN-ANGLE)

Signal Symptoms: None initially: silent; tunnel vision, night blindness, halos around lights.

Description: Chronic open-angle or primary open-angle glaucoma is an insidious optic neuropathy characterized by increased intraocular pressure (IOP) severe enough to damage the optic nerve. Progressive over time with a gradual visual field loss, chronic glaucoma is called the *silent blinder*, because it often goes unnoticed until the later stages. Variants occur in which IOP does not increase, but the optic nerve still becomes damaged; this is termed *normal tension glaucoma* and is common in Asian populations (Jacobs, 2016; Mantravadi & Vadhar, 2016; Salmon, 2017). Conversely, some patients have increased IOP but show no optic nerve changes or visual defects; these are referred to as glaucoma suspects (Prum et al., 2015) or those with ocular hypertension (Mantravadi & Vadhar, 2016)). Secondary glaucomas are the result of prior or concurrent ocular disease or trauma; they may have open angles caused by steroid-induced pressure increases.

Etiology: The etiology of chronic glaucoma is unknown, but the disease is associated with increased IOP, optic nerve degeneration, and visual field loss. Because IOP is the one factor that can be measured, most reevaluation and treatment decisions are influenced by this measurement (Mantravadi & Vadhar, 2016).

Occurrence: Prevalence of chronic glaucoma increases in persons over 40 years old, approaching 15% by age 75; 80% of cases are the open-angle type. Primary open-angle glaucoma causes 15% to 20% of all blindness in the United States and is the primary cause of blindness in African Americans (Salmon, 2017). In 2010, there were 2.8 million in the United States with POAG and this is predicted to rise to 3.4 million by 2020 (Jacobs, 2016).

Age: The incidence increases in persons over 40 years old, with another increase in incidence in persons between ages 60 and 75 years. Onset is earlier in African Americans.

Gender: Chronic glaucoma occurs equally in both men and women.

Ethnicity: African Americans, Afro-Caribbeans, and Hispanics are at a higher risk than the general population for developing chronic glaucoma (Salmon, 2017).

Contributing Factors: Risk factors include older age, a positive family history, African American ancestry or Latino/Hispanic ethnicity, type 2 diabetes mellitus, hypertension, myopia, low ocular perfusion, thinner central cornea, and genetic mutations (Jacobs, 2016). There is some association with migraine headache, but further research is needed to confirm this. Risk factors for secondary open-angle glaucoma include long-term use of topical or oral corticosteroids. It is important to note if the patient had prior LASIK surgery, because this can alter corneal thickness. Cataract surgery can also lower IOP, so this history is essential (Mantravadi & Vadhar, 2015).

Signs and Symptoms: The disease is asymptomatic in the early stages. By the time symptoms develop, significant neural damage has occurred. Eye health specialists frequently discover chronic open-angle glaucoma during routine eye examinations. Patients may present with a complaint of blurred vision, a request for new corrective lenses, or occasionally a report that they see a halo around lights (Jacobs, 2016; Weinreb et al., 2014).

On routine vision screening, visual acuity may be normal or unchanged from previous screening; visual fields may be decreased, however. The skilled examiner may detect changes in the cup-to-disc ratio on funduscopic examination—a cup with a diameter of more than 50% of the vertical disc diameter is highly suspect for glaucoma (Jacobs, 2016). Because it is difficult to perform a funduscopic examination on older patients without the benefit of a mydriatic agent, referral for specialized ophthalmological examination is essential. The ophthalmological examination may reveal increased IOP or signs of optic nerve damage without increased pressure.

Diagnostic Tests: Visual acuity should be measured and compared with prior measurements; pupillary assessment should be included. A dilated funduscopic examination and slit lamp examination of the anterior segment is conducted, assessing cup-to-disc ratio, asymmetry between discs, or other abnormalities. Applanation tonometry with the Goldmann applanation criterion standard should be measured to determine IOP. IOP has a circadian rhythm and is highest in the morning. Time of day, method, and measurements in both eyes are noted and compared with any prior measurements. If glaucoma is suspected, pachymetry should be used to measure central corneal thickness. If corneal thickness is outside of an established normal range, an adjustment in the IOP number is made based on this, raising or lowering the number. Because a thin central cornea is a risk factor, those with thicker central corneas but increased IOP have a correction factor calculated into the adjusted IOP (Prum et al., 2015).

Automated visual field testing is done to rule out any visual field deficits (visual field assessment by confrontation is of no value) (Jacobs, 2016). Gonioscopy is done to rule out other causes of IOP increase, including angle-closure glaucoma. Various imaging modalities, including fundus photography, optical coherence tomography, and scanning laser polarimetry, are used to preserve a permanent visual picture of the optic disc and are to be repeated later for comparison or evidence of disease progression (Jacobs, 2016).

Differential Diagnosis:

- Cerebral neoplasia
- Other optic neuropathies
- Vascular occlusive disease
- Trauma

Treatment: Chronic open-angle glaucoma cannot be cured. The goal of treatment is to halt progression of the disease and preserve existing vision as follows:

- IOP control within an identified range
- Stable visual fields
- No further progression of optic nerve damage

Mainstay treatment modalities include:

- Topical eye drops that reduce aqueous production or encourage outflow
- Laser trabeculoplasty
- Trabeculectomy or filtering surgery

Medical treatment with eye drops is usually first-line therapy, unless there are contraindications. Consultation between primary care provider and ophthalmologist may be indicated for patient safety and accurate identification of comorbidities and current medications (Table 7-1).

If topical ophthalmic agents do not effectively lower IOP, systemic carbonic anhydrase inhibitors are added. These also may be used before surgery to lower the IOP. If medical therapy is unsuccessful, selective laser trabeculoplasty or filtration surgery (trabeculectomy) to improve aqueous outflow is indicated. The laser treatment is considered for elderly patients who cannot tolerate surgery, although the outcome is not as desirable as with surgery. In either case, medication still may be required after surgery (Jacobs, 2017; Prum et al., 2015).

The U.S. Food and Drug Administration (FDA) has approved the use of a miniscule device called the Glaukos iStent® Trabecular Micro-Bypass Stent for patients with glaucoma and cataract. At the conclusion of cataract surgery, this stent is inserted into the canal of Schlemm, allowing the aqueous humor to bypass trabecular obstruction and drain directly from the anterior chamber into Schlemm's canal, lowering IOP (Jacobs, 2017; Mantravadi & Vadhar, 2015).

Follow-Up: Monitor patients for compliance with the treatment regimen. If eye drops are used, ensure that the patient can instill these, or teach a family member or neighbor to do it. Monitor medications for adverse effects; the range and frequency of side effects to glaucoma medications are significant. Ensure that patients have regular ophthalmological follow-up visits. If visual loss is severe, evaluate the environment for safety and risk of falls or injury. If the patient is still a licensed motor vehicle operator, driver's license retesting may be indicated in the interest of public safety.

Sequelae: If initial presentation is monocular, the other eye may become affected. Changes in the patient's overall health may have implications for the treatment plan.

Prevention/Prophylaxis: The U.S. Preventive Services Task Force (USPSTF) found insufficient evidence to recommend screening for glaucoma (USPSTF, 2013). Primary care providers need to be aware of risk factors and discuss these with patients who have not been diagnosed but are at risk. Patients who are glaucoma suspects or have actual primary open-angle glaucoma must be reminded of the need for eye examinations, continued monitoring, and adherence to the prescribed regimen. African Americans should have periodic eye examinations every 3 to 5 years, beginning at age 40 years. Anyone with a family history of glaucoma should have an annual eye examination, beginning at age 40 years. Patients with hypertension or diabetes should have regular eye examinations, as recommended by their health-care provider. All patients over 65 years old should have annual eye examinations (Prum et al., 2015).

Referral: Refer patients initially for ophthalmological examination and diagnosis. Collaborate on the management plan if the patient has significant medical conditions that affect treatment options, particularly pharmacological choices. Refer patients for periodic follow-up eye examinations or for treatment of complications if they arise.

Education: Educate the patient about the chronic and progressive nature of the disease, the need to follow the treatment regimen as prescribed, and the need for regular follow-up eye examinations and reporting of symptoms, if any. Reassure the patient that, although the disease is not curable, it can be managed. Educate patients with known risk factors about the need for eye examinations despite the absence of

TABLE 7-1 Glaucoma Drugs

DRUG CLASS	ACTION	EXAMPLES	SIDE EFFECTS	CONTRAINDICATIONS
Prostaglandin analog	Lower IOP 25%–30% by increasing trabecular or uveoscleral outflow Once-daily dosing	Lantanoprost Travoprost Bimatoprost Tafluprost	Iris color change, uveitis, eyelash growth, conjunctival injection	History of herpes keratitis, macular edema
Beta blockers	Decrease aqueous production; lower IOP 20%–25%	Betaxalol Carteolol Levobunolol Metipranolol (OptiPranolol) Timolol gel	Fatigue, bronchospasm, hypotension, bradycardia, corneal toxicity, depression, impotence	Congestive heart failure (cardiology consult) Nonselective are contraindicated in asthma, chronic obstructive pulmonary disease Hypotension, >first-degree heart block
Adrenergic agonists	Decrease aqueous production, enhance aqueous outflow; lower IOP 20%–25%	Apraclonidine Brimonidine Dipivefrin Epinephrine	Conjunctival injection, fatigue, headache	Use of monoamine oxidase inhibitors
Parasympathomimetic agents	Increase trabecular outflow; lower IOP 20%–25%	Pilocarpine Carbachol	Corneal toxicity Increased myopia Cataract Decreased vision	Other types of malignant glaucoma
Carbonic anhydrase inhibitors (primarily oral route)	Decrease aqueous production; lower IOP 15%–20%	*Topical:* Dorzolamide Brinzolamide *Oral:* Acetazolamide Acetazolamide sustained release Dichlorphenamide Methazolamide	*Topical:* Corneal edema, metallic taste, conjunctivitis *Oral:* Stevens-Johnson syndrome Metallic taste Renal calculi Aplastic anemia Thrombocytopenia	Sulfa allergy Sickle cell disease Renal calculi Thrombocytopenia

symptoms. Asymptomatic patients may question the need for regular treatment. Explain that treatment is preventing disease progression, which is why there are no symptoms. The frequency or complexity of the medication regimen may influence compliance. Reinforce the role of medication in controlling IOP and preventing visual loss.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Prostaglandin analogs are the most effective drugs at lowering IOP and can be considered as initial medical therapy unless other considerations, such as cost, side effects, intolerance, or patient refusal, preclude this.	A	Li et al., 2016 Mantravadi & Vadhar, 2015
Measurement of central corneal thickness (CCT) aids the interpretation of IOP readings and helps to stratify patient risk for ocular damage.	B	Prum et al., 2016
The IOP can be lowered by medical treatment, laser therapy, or incisional glaucoma surgery (alone or in combination). The choice of initial therapy depends on numerous considerations, and discussion of treatment with the patient should include the relative risks and benefits of the three options.	B	Prum et al., 2016

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HEARING LOSS

Signal Symptoms: Cannot hear or hears but does not understand (especially in a group); turns up radio or television louder to hear (noted by family, friends, or neighbors); inability to hear in one ear; tinnitus; feeling like people are "mumbling." Because presbycusis is gradual and insidious, hearing loss may go unnoticed until it has progressed significantly.

Description: Hearing loss is the decreased ability or complete inability to hear. The loss may involve the external, middle, or inner ear and can be unilateral or bilateral.

Etiology:

Sensorineural: A lesion in the organ of Corti or in the central pathways, including the eighth nerve and auditory cortex, causes sensorineural hearing loss. Presbycusis, noise-induced hearing loss, and ototoxic drug-related hearing loss all are sensorineural. Because there is no cure for this, amplification is usually required (Contrera et al., 2016).

Conductive: Conductive hearing loss is caused by a lesion involving the outer and middle ear to the level of the oval window. Various structural abnormalities, cerumen impaction, perforation of the tympanic membrane, middle ear fluid, damage to the ossicles from trauma or infection, otosclerosis, tympanosclerosis, cholesteatoma, middle ear tumors, temporal bone fractures, injuries related to trauma, and congenital problems are some of the causes.

Mixed: Mixed hearing loss includes sensorineural and conductive components.

Retrocochlear: Retrocochlear hearing loss is caused by a lesion between the cochlea and the brain, such as an acoustic neuroma or a meningioma.

Occurrence: Occurs in about 25% of adults more than 50 years old and in over 80% of those more than 80 years old. It is the most common sensory deficit in older adults (Walker et al., 2013).

Age: Sensorineural hearing loss increases with age.

Gender: Presbycusis is more severe in men than in women. This is attributed to environmental causes.

Ethnicity: Not significant.

Contributing Factors: Exposure to loud noises; heredity; ototoxic drugs, such as aminoglycoside antibiotics, salicylates, loop diuretics, cisplatin, and quinine; eustachian tube obstruction; chronic middle ear infections; and chronic cerumen impaction are contributing factors. Most cases of sudden unilateral hearing loss in older adults are due to thrombotic or embolic obstruction of the internal auditory artery.

Signs and Symptoms: The patient may complain that people mumble. There may be difficulty hearing associated with pain, pressure, discomfort, vertigo, or loss of balance. Associated symptoms may include tinnitus, dizziness, blockage, popping, pressure, crackling, trouble hearing distant sounds, or stiffness. A history of noise exposure, prior ear problems, or familial hearing problems is significant.

Otoscopic examination and inspection of the external auditory canal and middle ear may reveal redness, foreign objects, discharge, scaling, lesions, fluid, or cerumen impaction. Expect to see minimal cerumen, a pink color, and hairs in the outer one-third of the ear. The tympanic membrane should have no perforations and should be a translucent pearly gray. Changes in the tympanic membrane may be consistent with conductive hearing loss.

Diagnostic Tests: Primary care provider awareness requires asking about or screening for hearing loss in older adults (Walker et al., 2013). The self-administered Hearing Handicap in the Elderly (HHIE-S) is a 10-question instrument that can be given to patients in the waiting room or administered in the long-term care setting (Gross, 2016). Alternatively, asking a single question—"Do you have a hearing problem now?"—can be effective in uncovering a problem. Use of a portable pure-tone audiometer in the primary care office is also a quick screening test, and a medical assistant or office nurse can be instructed in how to administer the test and record results. Anyone with a hearing threshold of 40 dB or greater should be referred to audiology for evaluation. If there is a history of trauma or sudden hearing loss, urgent consultation with an otolaryngologist is warranted, and a computed tomography (CT) scan may be ordered. The Weber and Rinne are not good screening tests for hearing loss, but they can help to identify if the loss is sensorineural or conductive (Weber, 2015).

Differential Diagnosis:

- Ménière's disease
- Acoustic neuroma (unilateral, with tinnitus)
- Cholesteatoma (unilateral)
- Embolic or thrombotic phenomenon, associated with hypertension (sudden onset, unilateral)
- Meningioma (unilateral)
- Trauma (history of ear trauma, unilateral)
- Otitis media with effusion (retracted tympanic membrane, fluid behind tympanic membrane)
- Acute otitis media (unilateral, ear pain; bulging, immobile tympanic membrane)
- Central auditory processing disorder (seen in dementia)

Treatment: Treat the cause or refer the patient as appropriate. Encouragement, follow-up, and support are important for the patient with hearing loss.

Cerumen Impaction: Treat the patient with cerumenolytic agents, manual removal, or irrigation followed by manual removal as needed (AAFP, 2013). If cerumen impaction is a chronic problem, instruct the patient on instillation of an over-the-counter agent to soften cerumen.

Acute Otitis Media: Antibiotic treatment; an analgesic, such as acetaminophen or an NSAID, may be indicated for pain initially. In the presence of tympanic perforation, ossicle damage, tumor, tympanosclerosis, otosclerosis, sudden hearing loss, or temporal bone injury, refer the patient to a specialist (Weber, 2016a).

Presbycusis: Educate and support the patient so that no further damage occurs. Have the patient reduce noise exposure and avoid ototoxic medications. Refer to an audiologist for hearing aid evaluation. Refer the patient for lip-reading instruction when appropriate, and instruct the family to speak clearly. Consult a hearing rehabilitation company about the availability of special audio equipment. Patients with profound hearing loss should also be evaluated for suitability for cochlear implant (Mosnier et al., 2015).

Follow-Up: See patients as indicated by the cause of the symptom.

Sequelae: Possible complications depend on the cause of the symptom but may include permanent hearing loss. The patient may need a hearing aid or assistive listening device. Middle ear problems may progress to chronic ear problems, such as perforations and cholesteatoma. Social isolation and depression may result from inability to communicate.

Prevention/Prophylaxis: Advise the patient to use protective devices to guard against occupational or recreational hearing loss, to equalize ear pressure when diving, to chew gum or use decongestants in airplanes, to avoid flying or diving if upper respiratory infection is present, and to avoid ototoxic medications. Teach the patient proper techniques for cerumen removal and avoidance of Q-tip–type ear swabs or other foreign bodies in the ear. Hearing screening tests are recommended for persons over 65 years old and persons who report hearing difficulty. The USPSTF recently revised its recommendation and does not recommend periodic screening for hearing loss in asymptomatic adults (USPSTF, 2013). The American Speech-Language-Hearing Association recommends screening every 3 years for adults more than 50 years of age (American Speech-Language-Hearing Association, 1997). Residents of long-term care facilities should be screened for hearing loss on admission and as needed thereafter (Echalier, 2013).

Referral: Refer to or consult with appropriate specialist as indicated by cause of symptom. All patients with unexplained hearing loss should have an otoscopic and audiometric evaluation.

Education: Explain cause of symptoms, measures taken to determine the cause, and symptomatic treatment, if any. Advise the patient when to seek medical care. Teach the importance of using hearing aids, if indicated, because this can make a significant difference in the patient's quality of life. Older patients with multiple comorbidities may require the services of a multidisciplinary team to learn how to use a hearing aid effectively (Weinstein & Taylor, 2015). Advise the patient to contact rehabilitation centers to learn lip-reading skills or sign language if a hearing aid is not indicated. Hearing rehabilitation services can also assess the need for assistive devices such as flashing smoke detector alarms, an amplified telephone, or an amplified television device. Provide support and help the patient resist the temptation to withdraw socially. Caution the patient to avoid solicitation by hearing aid salespeople, particularly if they are offering a device without proper testing of the patient or a device that is very inexpensive. For patients in a long-term care setting, education of staff is essential to enhance communication and improve quality of life (Echalier, 2013).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
In 2012, the U.S. Preventive Service Task Force concluded that the current evidence is insufficient to assess the balance of benefits and harms of screening for hearing loss in asymptomatic adults ≥50 years. This recommendation applies to asymptomatic adults ≥50 years. It does not apply to persons seeking evaluation for perceived hearing problems or for cognitive or affective symptoms that may be related to hearing loss. These persons should be assessed for objective hearing impairment and treated when indicated.	B	USPSTF, 2013
Sudden hearing loss warrants urgent evaluation and treatment by a specialist.	C	Gross, 2018a
Patients who report hearing loss should be referred for full audiometry.	C	Gross, 2018a
Order imaging and/or ears, nose, and throat referral if vertigo, sudden onset, neurological symptoms, unilateral loss, or primarily low-frequency loss.	C	Gross, 2018a, 2018b
Patients with presbycusis may benefit from amplification or assistive listening devices.	C	Gross, 2018b

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HORDEOLUM AND CHALAZION

Signal Symptoms: Increased lacrimation with lump on eyelid.

Description: A hordeolum (stye) is an acute, purulent area of inflammation in the meibomian gland or gland of Zeis, commonly referred to as a "stye." The hordeolum typically contains *Staphylococcus aureus* bacteria; it can occur internally (underside of the eyelid) or externally at the lid margin (Ghosh & Ghosh, 2017); and some styes are sterile. A chalazion is a sterile mass on the eyelid caused by an inflammation and obstruction of a meibomian (oil-secreting) gland of the upper or lower eyelid. Meibomian glands lubricate the lid margins. The chalazion is more chronic in nature, painless, and lipogranulomatous without erythema. It can interfere with vision at times and develops a rubbery consistency (Vagefi, 2018).

Etiology: Blockage in a duct leading to the eyelid surface from the meibomian gland or obstruction of the gland of Zeis results in inflammation or infection (usually from *Staphylococcus*) called hordeolum. If the blockage becomes chronic, progressing to the formation of a hard, granulomatous mass, it is called a chalazion.

Occurrence: Common.

Age: Can occur at any age.

Gender: Occur equally in both men and women.

Ethnicity: Not significant.

Contributing Factors: Previously unresolved blepharitis, poor hygiene, immunosuppression, and skin conditions, such as acne rosacea or seborrheic dermatitis, all are contributing factors to development of hordeolum or chalazion (Fort, 2018; Vagefi, 2018).

Signs and Symptoms: A hordeolum is painful and erythematous; it may present initially as a grossly swollen and tender eyelid, then localize (Walker & Adhikari, 2016). A chalazion is a slow-developing, painless hard mass, with inflammation of the meibomian gland and possible involvement of the surrounding tissue. Physical examination with inversion of the eyelid reveals a red, elevated mass that may become large and press against the cornea (Vagefi, 2018).

Diagnostic Tests: None unless recurrent, then biopsy of lesion to rule out malignancy.

Differential Diagnosis:

- Foreign body
- Contact dermatitis
- Rosacea
- Atopic dermatitis
- Blepharitis
- Orbital cellulitis
- Acute dacriocystitis
- Sebaceous cell, meibomian gland, or basal cell carcinoma (Ghosh & Ghosh, 2017; Vagefi, 2018)

Treatment: Warm compresses are applied to the affected eyelid for 10 minutes, four times daily, to facilitate opening and drainage. Allow purulent lesions to drain without squeezing them. Massage of the eyelid after warm compresses may help to soften secretions. Many lesions resolve spontaneously without treatment. In cases of secondary infection, antibiotic eye drops or ointment may be used; combination antibiotic and steroid should be used only by an ophthalmologist. If the hordeolum progresses to preseptal cellulitis, prescribe a systemic antibiotic, such as dicloxacillin (Fort, 2018). Treatment of chalazion includes warm compresses for 15 minutes four times a day. Incision and drainage by an ophthalmologist may be needed if there is no resolution after 2 months. Antibiotics are not indicated because it is granulomatous in nature (Ghosh & Ghosh, 2017). A large nondraining hordeolum or chalazion may require emergency treatment if cellulitis occurs (Vagefi, 2018; Walker & Adhikari, 2016). Cochrane reviews on use of acupuncture in acute external hordeolum report low level certainty of effectiveness in China (Cheng et al., 2017). Another Cochrane review on the use of nonsurgical interventions for acute internal hordeolum found no evidence for or against nonsurgical treatment (Lindsley, Nichols, & Dickersin, 2017).

Follow-Up: See patient in 2 to 4 weeks to evaluate treatment.

Sequelae: Complete resolution may take several weeks to months; frequent recurrence may indicate underlying malignancy (Ghosh & Ghosh, 2017).

Prevention/Prophylaxis: Prevention strategies include advising the patient to perform proper lid hygiene (helps prevent recurrence) by gently scrubbing lids with diluted baby shampoo daily or directly applying baby shampoo with cotton-tipped applicator, then rinsing. Frequent hand washing is advised. Warm compresses should be initiated at the first sign of eyelid irritation or if chalazion starts to return; a clean cloth should be used for each warm compress to eye (Ghosh & Ghosh, 2017).

Referral: Refer to or consult with an ophthalmologist if the patient has a visual change, eye pain, or impairment to the eye, or if the chalazion does not heal spontaneously in 6 weeks, because surgical removal may be necessary.

Education: Explain the problem and treatment. Discuss prevention strategies.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Initial treatment includes warm compresses 4 times daily and topical erythromycin ointment for hordeolum.	C	Wolter, 2017
Lesions refractory to treatment should be referred for incision and drainage (both hordeolum and chalazion).	C	Walker & Adhikari, 2016 Wolter, 2017
Sebaceous carcinoma should be suspected in patients who are middle-aged and older with persistent chalazia.	C	Wolter, 2017

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

AGE-RELATED MACULAR DEGENERATION

Signal Symptoms: Age-related macular degeneration (AMD) is the leading cause of blindness in adults. It is a disease that results in a loss of central vision, which is most crucial for activities of daily living (ADLs) and instrumental activities of daily living (IADLs). The severity of central vision loss varies depending on the type of macular degeneration. Most recently, efforts have been made to standardize the terminology and classification of AMD by defining it as one disease with three stages: early, intermediate, and late. Late AMD is characterized by significant vision loss, which may occur with gradual loss due to "dry" macular geographic atrophy, or with rapid onset as a result of the neovascularization of "wet" macular degeneration (Wenick, Bressler, & Bressler, 2018).

Dry, Nonexudative or Atrophic Macular Degeneration: Gradual loss of central vision in one or both eyes, with complaints of difficulty reading or driving, blurred or fuzzy vision (late), and the concern that straight lines appear wavy. In early dry AMD there is the presence of drusen, yellow deposits under the retina. In the intermediate stage, pigmentary disturbances in the retinal pigment epithelium (RPE) occur, and in the late stage there is geographic atrophy (GA) with severe central vision loss.

Wet, Exudative or Neovascular Macular Degeneration (NVAMD): This characterizes one form of late AMD with accelerated central vision loss in a period of weeks to a couple of months. The fragility of the neovasculature results in subretinal hemorrhage or fluid accumulation that destroys the visual center. The disease usually presents in one eye initially, but there is a greater than 40% risk of development in the other eye within 5 years.

Description: The macula in the center of the retina comprises millions of photoreceptor cells containing visual pigments for central vision. These are nourished by the retinal pigment epithelium (RPE), part of a complex basement membrane that becomes sclerotic with age. AMD is defined as early, intermediate, and late, depending on the severity of vision loss and pathological findings. Dry AMD is most common, accounting for 90% of cases. Onset of visual loss is gradual and may not be noticed if only one eye is affected. The cause is unknown, but there is the presence of drusen, disintegration of the RPE and light-sensing cells. Late AMD with neovasculature occurs in 10% of cases of AMD but accounts for 90% of all AMD blindness. Patients with early and intermediate forms of AMD may develop neovascularization of the choroid area with leakage of new blood vessels and fluid into the retinal pigment epithelium, which causes scarring and additional damage to the macula. The risk ranges from 2% to 20% depending on the size of drusen present. The presence of GA does not protect against the development of NVAMD, with an incidence of 16%, which increases to 36% if NVAMD is present in the other eye (Wenick et al., 2018).

Etiology: A challenge in identifying the cause of dry AMD is the difficulty in distinguishing the early form from normal changes with aging and the lack of symptoms at this stage. As the macula ages, the RPE, Bruch's membrane, and choroidal vessels are all affected. Hard drusen may by noted in almost all older eyes. It is the increase in size and number of drusen that is one indication of early AMD. Genetic predisposition and risk factors certainly contribute to the development of AMD. The role of inflammation in the development of AMD is the focus of research on the role of the complement pathway (Gemenetzi & Lotery, 2016). NVAMD has been more aggressively studied in attempts to understand the origin of the angiogenesis. As a result of these studies, several pro-angiogenic factors have been identified. The vascular endothelial growth factor (VEGF) is the most important in intraocular angiogenesis and its role has resulted in new treatment options for NVAMD (Velez-Montoya et al., 2014).

Occurrence: Common; macular degeneration is the leading cause of legal blindness in adults more than 55 years old. Risk rises with age; occurrence is 30% in adults over 75 years old. In North America, 15 million people have nonexudative AMD and 1.7 million have NVAMD. These numbers may be an underestimate, as those with early AMD may have no visual changes and may not have been diagnosed. Those with early AMD and NVAMD are at low risk of increasing visual

loss, with only one-third progressing to intermediate AMD in the next 5 years (Wenick et al., 2018).

Gender: Occurs with equal frequency in males and females.

Ethnicity: Caucasians are more likely to experience visual loss from AMD than are African Americans.

Contributing Factors: The primary risk factor for AMD is advancing age. Other risk factors include family history, smoking, exposure to sunlight, nutritional deficits, and cardiovascular disease. History of AMD in one eye is a risk factor for developing it in the other eye (Brady, Bressler & Bressler, 2018).

Signs and Symptoms: In early AMD there may be no symptoms, particularly if it is monocular. Later, patients may note a decrease in visual acuity or blurred vision that improves in bright light. In late NVAMD, straight lines appear wavy, and there may be a hole in central vision. White-yellow drusen spots are seen on funduscopic examination. Until recently, the primary screening device for NVAMD was the Amsler grid test, which, when administered, may reveal wavy lines or missing lines in central vision; however, it has low sensitivity (a high number of false negatives). New perimetry devices are now available for both clinical and home monitoring with better sensitivity and specificity for identifying NVAMD (Wenick et al., 2018). On funduscopic examination of those with NVAMD, choroidal neovascular membranes (CNVMs), subretinal fluid, hemorrhage, and subretinal lipid deposits may be present.

Diagnostic Tests: All patients should have a comprehensive eye examination by an ophthalmologist, including visual acuity with best correction, funduscopic examination, Amsler grid or perimetry assessment, fluorescein angiography, and fundus photos, including optical coherence tomography (Brady et al., 2018; Wenick et al., 2018).

Differential Diagnosis:

- Unexplained loss of visual acuity
- Diabetic retinopathy
- Ocular histoplasmosis
- Trauma with scar
- Hypertension

Treatment: The Age-Related Eye Disease Study (AREDS) found that patients at high risk of late AMD lowered their risk by 25% when treated with high doses of vitamin C, vitamin E, beta carotene, zinc oxide, and cupric oxide. This effect was confirmed through 10 years of follow-up. The efficacy of this was demonstrated for those with intermediate AMD or late monocular AMD to prevent further loss or involvement of the other eye. It is not recommended for those without AMD or those with early AMD who have a low 5-year risk of worsening vision. The risk versus benefits of this high-dose vitamin treatment needs to be evaluated for each patient. The AREDS2 evaluated the effect of substituting the carotinoids, lutein and zeaxanthin, as well as omega-3 (DHA) and eicosapentaenoic acid (EPA) for beta carotene and a lower zinc dose on progression of AMD (AREDS2 Research Group, 2013). The conclusion after 5 years was that eliminating beta carotene and lowering zinc did not make a difference in progression to advanced AMD, and the addition of lutein and zeaxanthin, DHA, and EPA did not further reduce progression. However, they concluded that there may be value in substituting lutein and zeaxanthin for beta carotene, as beta carotene increased the risk of developing lung cancer, especially in smokers. Trials are currently underway for the use of intravitreal injections of lampalizumab for the treatment of GA. The initial data seems promising and this would be a breakthrough for treatment of advanced nonexudative or dry macular degeneration (Bohadorani & Singer, 2017). Low-vision enhancement aids are available to help patients with dry AMD. For patients with bilateral advanced and end stage untreatable AMD, a visual prosthetic implant may be considered.

First-line therapy for NVAMD includes monoclonal antibodies that have anti-VEGF abilities. These drugs are injected directly into the vitreous area of the eye and bind with the angiogenic VEGF or receptor and interrupt the neovascularization. They are not able to repair the basic defect or attack the source of VEGF, so they must be used repeatedly to suppress the angiogenic process (Brady et al., 2018). Ranibizumab and bevacizumab are two humanized monoclonal antibodies that are used to treat NVAMD successfully (Brady et al., 2018). They are injected intravitreally at monthly intervals; current focus is on determining optimal spacing and length of treatment for best results. Local side effects include eye pain, uveitis, and endophthalmitis. There are also potential systemic cardiovascular and thrombotic effects from these drugs, although they have proved minimal. Aflibercept is a newer agent that improves visual outcomes with less frequent injections; preliminary studies show it to be as effective as ranibizumab, with better outcomes in visual acuity and less frequent dosing or maintenance; further studies are in progress (Arroyo, 2017b).

Laser photocoagulation therapy is no longer first-line treatment since the advent of anti-VEGF therapies. Photodynamic therapy is now being studied as a possible combination therapy with anti-VEGF treatment (Arroyo, 2017b). Research into surgical options has yielded disappointing results; likewise, use of statins for AMD has not shown positive results, but recent studies with a small number of participants showed some promise (Arroyo, 2017b; Leung et al., 2018). Research is currently underway to find safe, effective treatments for both dry and wet AMD. Research into treatment using stem cells is actively being pursued with some recent success (Janigian, 2018).

Follow-Up: All patients with AMD should be seen periodically by an ophthalmologist for reevaluation of disease progression and, where applicable, further treatment. Patients may be given an Amsler grid or the newest option of an at-home perimetry monitor to use weekly or monthly to check for visual changes.

Sequelae: Loss of central vision limits functional capacity and ability to read. Most AMD patients cannot drive. Environmental safety also is compromised; falls and hip fractures can occur in patients with AMD. Patients using high-dose vitamin and mineral therapy may have yellowing of skin. Smokers are at increased risk of lung cancer from beta carotene included in the first AREDS vitamin supplement formula.

Prevention/Prophylaxis: There is no specific prevention. The USPSTF has determined that there is not enough evidence to support visual screening of older adults for AMD (USPSTF,

2014). Annual eye examinations in persons over 65 years old can facilitate early detection and, in the case of wet AMD, early intervention to limit visual loss. Smokers should be encouraged to quit.

Referral: All patients more than 65 years old should be referred to an ophthalmologist for an annual eye examination. Patients less than 65 years old with visual symptoms also should be referred. Patients with AMD should be referred to the local chapter of the Blind Association for services, vision enhancement devices, and support groups.

Education: Teach patients the importance of seeking evaluation for visual symptoms as soon as they occur. Explain disease process and community support services. Educate the family about the need for environmental safety. Caution patients to avoid unproven remedies. Enforce the role of smoking as an independent risk factor in AMD.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Anti-VEGF injections are first-line agents to treat wet AMD.	A	Brady, Bressler, & Bressler, 2018 Leung et al., 2018
Antioxidant formulations as used in the AREDS and AREDS2 trials should be recommended to those with intermediate or advanced AMD.	A	Wenick, Bressler, & Bressler, 2018 Leung et al., 2018
There is inadequate to support the use of antioxidant formulations in those without AMD or with early AMD.	A	Wenick, Bressler, & Bressler, 2018

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

ORAL CANCER

Signal Symptoms: Nonhealing sore in the mouth or lip, unexplained lymph node swelling in head/neck area, difficulty chewing or swallowing.

Description: Oral cancer is a malignant tumor of the oral squamous epithelium, which includes the lip, buccal mucosa, upper and lower alveolar ridge, floor of the mouth, retromolar trigone, hard palate, and anterior two-thirds of the tongue. The oropharynx includes the soft palate at the back of the mouth, part of the throat behind the mouth, tonsils, and base of the tongue (Figure 7-1). The oral cavity has a rich lymphatic supply with initial spread to lymph nodes in levels I to III of the neck. The risk for lymphatic spread in cancers of the lip is lower than other oral cancers (NCCN, 2016).

Etiology: Oral cancer is associated with chronic irritation of the squamous epithelial lining of the oral cavity. Tobacco and heavy alcohol use have previously been the primary risk factors in the development of oral cancers. In recent years, there has been a global increase in the incidence of human papilloma virus (HPV)-related oral cancers, particularly in North America and Northern Europe (ACS, 2016; Gillison, Chaturvedi, Anderson, & Fakhry, 2015). In certain subsets of oropharyngeal cancers, HPV, especially HPV-16, is now considered to be the leading cause of the increased incidence (Gillison et al., 2015). HPV-associated head and neck cancers are commonly confined to the oropharynx and are associated with improved prognosis and response to treatment (van Monsjou et al., 2013).

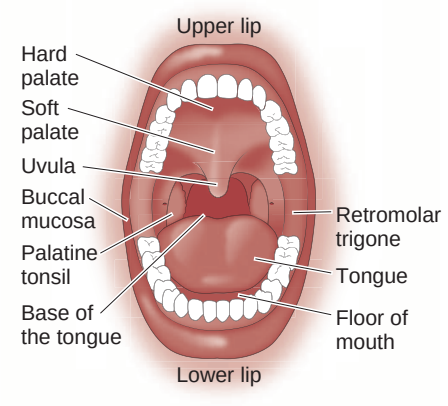

FIGURE 7-1. Oral anatomy.

Occurrence: The National Cancer Institute Surveillance Epidemiology and End Results (SEER) estimates that in 2016, 48,330 people in the United States will be diagnosed with oral cancer, with estimated deaths from this disease at 9,570 (SEER Stat Fact Sheet, 2016). The incidence of new cases has been rising on average 0.6% each year over the last 10 years and from 2009 through 2013. The Caucasian and non-Hispanic population had the highest incidence, followed by the African American population (SEER, 2016). The lowest incidence was seen in Hispanics. Death rates for oral cancers

have not changed significantly from 2004 through 2013; the overall 5-year survival rate in the United States is about 64% (SEER, 2016). If cancer has spread to a distant part of the body, the 5-year survival rate is 38% (Cancer.net, 2016).

Age: The median age at diagnosis for oral cancers from 2009 through 2013 in the United States (including all races and both sexes) was 62 years of age; the median age for death from this cancer was 67 years old, with death rates higher in men of African American descent (SEER, 2016).

Gender: Men are almost three times more likely to develop oral cancer than women, with the number of new cases of oral cavity and pharynx cancer 11.1 per 100,000 in men and women combined per year based on 2009 through 2013 cases (SEER, 2016).

Contributing Factors: Tobacco use and heavy alcohol consumption, alone or synergistically, are strongly related to the development of oral cancer. Pipe smoking and sun exposure have been implicated in lip cancer. Leukoplakia and erythroplasia are often precursors to oral cancer. In addition to HPV, relationships between oral cancer and Epstein-Barr virus, herpes simplex virus, and immunodeficiency states also have been found (ACS, 2016). Ill-fitting dentures are a risk factor for development of oral cancer, however, duration of denture use in general is not (Manoharan, Nagaraja & Eslick, 2014). In Asian populations, several genetic polymorphisms, combined with environmental carcinogens such as betel quid chewing, have been implicated in the development of oral cancer (Guha1, Warnakulasuriya, Vlaandere, & Straif, 2014).

Signs and Symptoms: Nonhealing, ulcerative lesions on the lip, tongue, or oral mucosa usually are noted first. Leukoplakia, a white patch on the mucosa that cannot be rubbed off, is the most common precancerous lesion. There is often no pain, so detection is contingent on a careful soft tissue examination by a dentist. Erythroplasia is a nonpainful, flat, or even depressed erythematous change of the mucosa without a patch lesion with high risk for malignant transformation (Yardimci, Kutlubay, Engin, & Tuzun, 2014). Presenting symptoms of oral cancers may include a lump or thickening in the mouth, soreness or a feeling that something is caught in the throat, difficulty chewing or swallowing, ear pain, difficulty moving the jaw or tongue, hoarseness, numbness of the tongue or other areas of the mouth, or swelling of the jaw causing dentures to fit poorly or become uncomfortable (NIDCR, 2013). Oral pain, bleeding lesions, and unexplained weight loss are usually late signs of malignancy. Patients may complain of numbness in the skin around the chin when the lesion involves a nerve. Enlarged submandibular or submental lymph nodes may be found in advanced disease.

Diagnostic Tests: More than 90% of all oral cancers are squamous cell carcinomas and involve lesions on the mucosal surfaces (Schiff, 2016). The challenge is to differentiate cancerous lesions from a number of benign lesions that also occur in the oral cavity. Definitive diagnosis depends on tissue biopsy and is recommended after 2 weeks of nonhealing. Endoscopy may also be performed to evaluate extent of the lesion. Depending on location and potential extent of oral cancer, and the possibility of second primary site or distant metastases, various diagnostic strategies will be employed by the specialist seeing the patient. Magnetic resonance imaging (MRI), CT scan, positron emission tomography (PET), or PET/CT scan will show extent of infiltration, regional lymph node spread, second primary tumors, or distant metastases. CT scan is superior in detecting early bone invasion and lymph node metastasis, but MRI is preferred for evaluating the extent of soft tissue involvement and for providing a three-dimensional image of the tumor. (Poon & Stenson, 2016). Other adjunctive studies include vital cytology staining, biological markers, routine dental radiographs, and thorough dental examination.

Differential Diagnosis:

- Oral candidiasis (white patches are scraped off and bleed underneath)
- Melanocytic lesions
- Oral lichen planus (lacy white lesions on buccal mucosa)
- Oral hairy leukoplakia
- Viral or idiopathic lesions (herpes, aphthous ulcers, mucositis)
- Gingivitis
- Oral cysts
- Intravascular/extravascular blood lesions (Finkelstein, 2016)

Staging: The tumor, node, metastases (TNM) staging system of the American Joint Committee on Cancer (AJCC) staging system and the Union for International Cancer Control (UICC) system is used by oncology specialists in the staging of oral cancers (AJCC, 2016; Sobin, Brierley, & Gospodarowicz, 2015). The TNM system describes the anatomical extent of disease by the size of the primary tumor, the presence or absence of regional nodal involvement, and presence or absence of metastatic disease (Sobin et al., 2016) (see Figure 7-2). Additionally, oral cancers are given a histological grading, GX to G4 (cell differentiation of the tumor) and stage grouping (stage 0 to stage IVC) (AJCC, 2016; Cancer.net, 2016).

Treatment: Treatment for oral cancers ideally requires a multidisciplinary approach with three main treatment options: surgery, radiation therapy, and chemotherapy, or a combination of one or all of these. Treatment depends on the extent of disease, comorbid illness, potential side effects, and functional outcomes. Surgery and radiation therapy are the standard of care for early-stage and resectable locally advanced cancers (NCCN, 2016). Radiation therapy may be the primary treatment modality, particularly in early stage disease, unresectable tumors, and as adjuvant therapy after surgery to prevent recurrence or if there is possible microscopic residual disease. Radiation treatment is generally given 5 days per week, for 6 to 7 weeks (Gross et al., 2016). Chemotherapy is not considered a first-line therapy in early-stage oral cancers, but may be used in conjunction with radiation therapy in more advanced cases (Gross et al., 2016).

Follow-Up: Post-treatment surveillance is recommended for lifetime with more frequent follow-up the first 2 to 4 years, as 80% to 90% of all recurrences in patients treated for curative intent will occur in this timeframe (NCCN, 2016). The 5-year overall survival rate is approximately 70% for stage I and II oral cancer (Gross et al., 2016) and 83.3% for localized oral cavity and pharynx cancer (SEER, 2016). During the 1 to 2 years, the patient should be seen frequently for focused oral

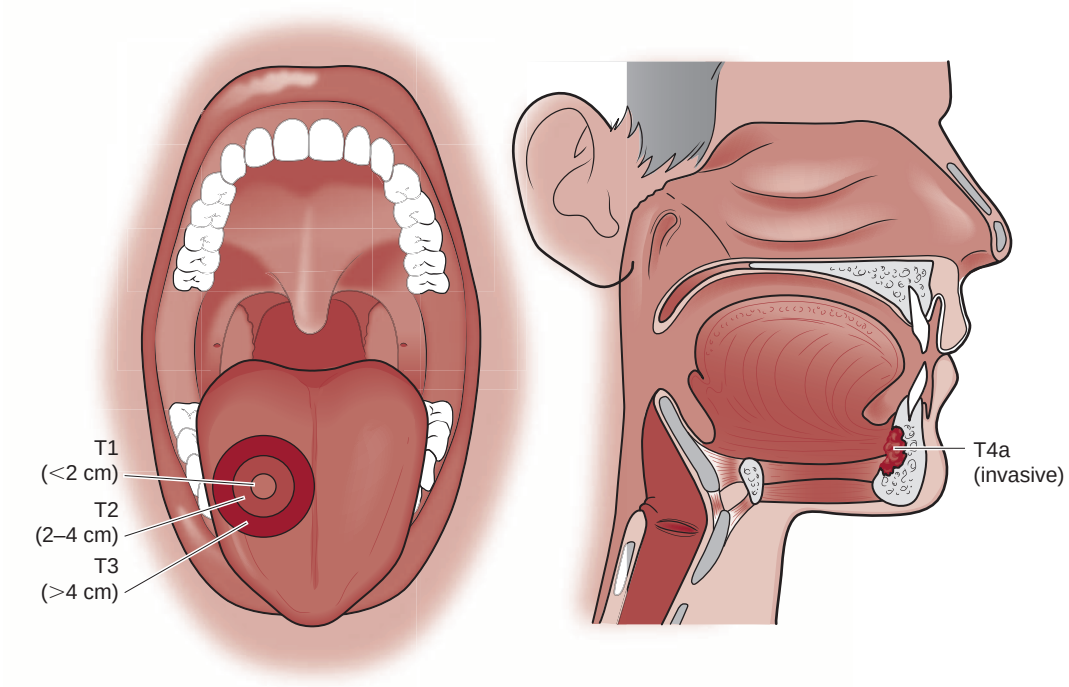

FIGURE 7-2. Oral cancer staging.

examinations and to assess for recurrence of symptoms and late effects of treatment, in addition to yearly complete physical examinations. Thereafter, follow-up visits should occur per recommendation of the surgeon or oncologist.

Sequelae: Complications and late effects from oral cancer treatment include disfigurement and oral problems such as chronic xerostomia, dental problems, difficulty chewing or swallowing, chronic pain, hypothyroidism, and malnutrition. Cosmetic and functional outcomes are often very good with advanced surgical reconstruction techniques and microsurgery (NCCN, 2016). Regardless of the treatment modality, patients undergoing treatment for oral cancers may have substantial side effects, loss of function, and changes in appearance with significant impact on quality of life (Sandstrom et al., 2016). Other complications may include secondary infections, second primary cancers, or metastases to the lungs, regional lymph nodes, or adjacent organs.

Prevention/Prophylaxis: The most effective way to control oral cancer is to combine early diagnosis with timely and appropriate treatment. Smoking cessation, treatment for alcoholism, adequate nutrition, and yearly oral examinations (see Figure 7-3) can help prevent oral cancer. Edentulous patients should remove dentures for oral examination. Wearing a hat with a brim or using lip gloss with sunscreen can prevent sun exposure of lips. Routine dental examination of asymptomatic and symptomatic patients can lead to detection of earlier stage cancers and premalignant lesions, however, there is no definitive evidence that shows screening can reduce oral cancer mortality (NIH, 2016). The USPSTF (Moyer, 2014), Cochrane Database of Systematic Reviews (Brocklehurst et al., 2010), and American Dental Association Council on Scientific Affairs Expert Panel (Rethman et al., 2010) all agree that there is insufficient evidence to justify screening for oral cancer in the general population. The American Dental Association does recommend that providers screen for signs of potentially malignant lesions early-stage cancer during routine oral examinations, particularly for patients who use tobacco or have heavy alcohol consumption (Moyer, 2014). A review by Omaña-Cepeda and colleagues (2016) found substantial evidence that with proper training, dentists and the dental team can be very effective in counseling patients to quit smoking and are essential to implement guidelines that help patients quit.

Referral: Refer the patient to an otolaryngology surgeon and/or oncologist. Initial consultation with a dentist who specializes in oral cancer detection may also be indicated. Also refer the patient and family to the National Cancer Institute Helpline (1-800-4-CANCER) for information about resources and support, and to a local support group if available. If nutrition is a problem, refer to a nutritionist who works with cancer patients. Many cancer programs, particularly academic and comprehensive cancer centers, have multidisciplinary teams that include surgeons, medical and radiation oncologists, nurses, advance practice clinicians, dieticians, and social workers to manage the complex care that may be required for these patients.

Education: Teach patients about the risk factors for developing oral cancer, including smoking, chewing tobacco, alcohol abuse, HPV transmission, and poor nutrition. Discuss strategies to help the patient deal with these issues. Advise patients to report any nonhealing oral lesion within 2 weeks of onset.

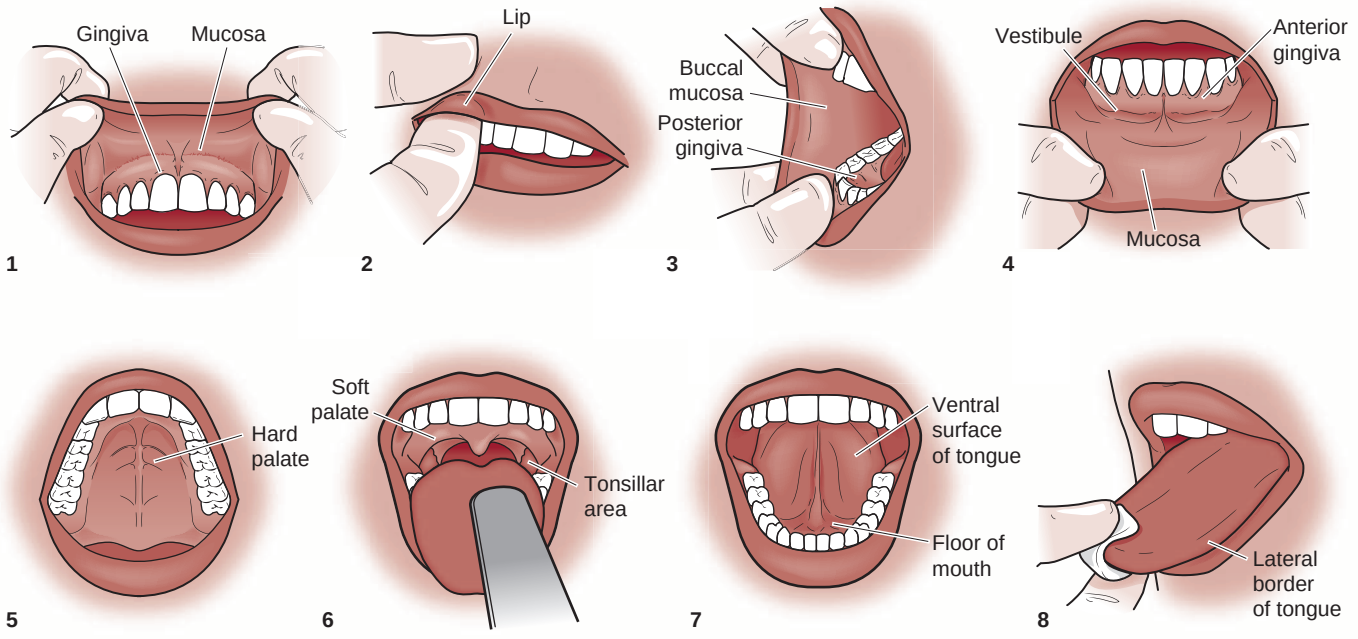

FIGURE 7-3. Oral cancer screening.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Curative treatment for oropharyngeal cancer typically consists of surgery, radiation therapy, or radiation therapy in combination with chemotherapy.	A	National Comprehensive Cancer Network Guidelines, 2016
Current evidence is insufficient to assess the balance of benefits and harms of screening for oral cancer in asymptomatic adults.	I	Moyer, 2014
Patients presenting with metastatic disease or very advanced disease may benefit from systemic chemotherapy, local radiation to palliate symptoms, or both.	A	National Comprehensive Cancer Network Guidelines, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

RETINOPATHY

Signal Symptoms: Blurred vision, painless visual loss, poor night vision, poor color vision, floaters.

Description: Retinopathy, a disease of the retina, includes several types: hypertensive, diabetic, and, less commonly in older adults, sickle cell; polycythemia vera; and infectious causes such as toxoplasmosis or cytomegalovirus in HIV.

Etiology: The pathology of the retinopathy relates to the underlying cause. Systemic causes common to older adults include hypertension (arteriosclerotic retinopathy), diabetes (diabetic retinopathy), and AMD (see previous discussion). Hypertensive or arteriosclerotic retinopathy is related to the severity of the hypertension. Diabetic retinopathy is a highly specific microvascular complication of type 1 and 2 diabetes. It is related directly to the duration of diabetes, the patient's age at onset, and glycemic control. Diabetic retinopathy is either proliferative or nonproliferative. Nonproliferative retinopathy is most common in type 2 diabetics and is characterized by dilation of veins, microaneurysms, retinal edema and hemorrhages, and hard exudates. Proliferative

retinopathy is common in type 1 diabetics and includes neovascularization, vitreous hemorrhage, and retinal detachment; secondary glaucoma also can occur because of blockage of outflow channels by new vessels. Diabetic retinopathy often coexists with hypertensive retinopathy.

Occurrence: Diabetic retinopathy is the leading cause of new blindness in people 20 to 74 years old. Up to 27% of people with type 1 diabetes have some retinopathy present in 5 to 10 years of diagnosis, and after 10 years the prevalence is 70% to 90% (Yanoff & Cameron, 2016). Nearly 80% people with type 2 diabetes develop retinopathy after 15 to 20 years and it is present in one-third at the time of diagnosis (Hammes, Lemmen, & Bertram, 2014). Diabetic retinopathy develops earlier in older patients, but proliferative retinopathy is less common. Proliferative retinopathy is more common with insulin use. According to the National Eye Institute, diabetic retinopathy affects 7.7 million people age 40 years and older. Hypertensive retinopathy is seen with poorly controlled hypertension.

Age: Older adult patients are more likely to have hypertensive or diabetic retinopathy because of the increase in chronic disease with age. Approximately one-half of all diabetes cases occur in persons over 55 years old.

Gender: Men develop diabetic retinopathy sooner than women.

Ethnicity: Diabetes is more common in Native Americans, Asian Americans, African Americans, Hispanic Americans, and Pacific Islanders than in Caucasians. Hypertension is also more prevalent among African Americans.

Contributing Factors: Contributing factors in diabetic retinopathy include age at onset, type of diabetes, duration of disease, poor glycemic control (persistent elevated hemoglobin A1c), arterial hypertension, dyslipidemia, nephropathy, smoking, and stage at which retinopathy is detected (early detection of nonproliferative is preferred) (Hammes et al., 2014). Duration of disease is the best predictive factor for retinopathy (Yanoff & Cameron, 2016). Causes of other types of retinopathy include poorly controlled hypertension and AMD. Multiple sclerosis, sarcoidosis, and coagulopathies are also associated with proliferative retinopathy (Bearelly, Mathura, & Jampol, 2008).

Signs and Symptoms: Retinopathy usually develops without symptoms until advanced stages of the disease. If presenting with symptoms, they may be the insidious painless onset of decreased visual acuity. Patients may report blurred vision, poor night vision, poor color vision, or "floaters and flashers." The ophthalmoscopic examination may be the first point of detection in an asymptomatic patient. All patients with known diabetes or hypertension should have regular comprehensive eye examinations, including a funduscopic examination. The funduscopic changes seen in patients with hypertension include arteriolar narrowing, arteriovenous nicking, changes in arteriolar light reflex, and tortuosity of vessels. Classic nonproliferative changes in patients with diabetes are microaneurysms and hard and soft exudates. Clinical findings in patients with proliferative retinopathy are cotton-wool spots, deep hemorrhages, and neovascularization.

Diagnostic Tests: Dilated funduscopic examination and comprehensive eye examination, fundus photos, ocular coherence tomography (OCT), and fluorescein angiography are used to diagnose diabetic retinopathy (Crandall & Shamoon, 2016; Janigian, 2018). Hypertensive retinopathy is a clinical diagnosis based on physical examination findings during slit-lamp examination. Hypertensive retinopathy presents as disc edema and can be classified by ophthalmology using the Keith-Wagener-Barker or the recent simplified Mitchell-Wong grading system (Aissopou et al., 2015; Downie et al., 2013). The intent of these grading systems was to correlate hypertensive retinopathy with a systemic disease such as left ventricular heart failure, pulmonary edema, stroke, or myocardial infarction. Although not specific for the diagnosis of retinopathy, regular monitoring of hypertension and blood glucose is essential in disease management and prevention of complications.

Differential Diagnosis:

- Retinal detachment
- Cytomegalovirus retinitis (usually associated with HIV)
- Toxoplasmosis (associated with latent infection)
- Retinal vasculitis
- Retinal vein occlusion
- Hyperviscosity syndromes

Treatment: Prevention and early detection are fundamental to preserving vision. Annual ophthalmological examinations are required. For patients with diabetes, the goal is to optimize glucose control. Patients with hypertension or hypertension and diabetes should be treated to control blood pressure (goal is 130/80 mm Hg for those with diabetes, 140/90 mm Hg without diabetes). Blood pressure reduction should be slow and deliberate to minimize end-organ damage; treatment of the underlying pathology is essential. The parameters and goal of treatment in patients with hypertension and diabetes should be clear and concise.

For patients who have diabetes with nonproliferative retinopathy, macular edema is responsible for most vision loss. If macular edema is clinically significant, treatment with anti-VEGF is superior to laser therapy, but intravitreal steroid injection may also be used (Yanoff & Cameron, 2016). For those with diabetes with severe nonproliferative disease, panretinal laser photocoagulation can lower the risk of progression to proliferative diabetic retinopathy (Crandall & Shamoon, 2016). For patients who have diabetes with vitreous hemorrhage and advanced neovascularization, vitrectomy can be of significant benefit (Crandall & Shamoon, 2016).

Follow-Up: Follow-up depends on the findings. A retinal specialist should follow up with patients with retinopathy. The primary care provider is responsible for regular disease management. The staging of retinopathy and the treatment plan require specialty follow-up.

Sequelae: Not all retinopathy is progressive. The condition and its outcome are related to the cause of the retinopathy and the degree of successful disease management. For hypertensive retinopathy, arteriovenous nicking and arteriolar narrowing are irreversible.

Prevention/Prophylaxis: Older adult patients should have an annual ophthalmological examination. During routine office

visits, perform a funduscopic examination on the eyes of patients with diabetes and hypertension. Promptly refer the patient to a retinal specialist at the earliest report of vision change or abnormal examination findings. Patients with diabetes and hypertension should be aware of the goals of treatment and the need to control blood pressure and blood glucose. Home monitoring of blood glucose and blood pressure may be advised.

Referral: A retinal specialist should see all patients with retinopathy. All patients with diabetes and hypertension need to see an ophthalmologist annually. Communication between the specialist and primary care provider is essential for optimal patient treatment.

Education: Patients should report any alteration in vision. Patients should know how the goals of blood pressure and blood glucose control relate to prevention of complications. Finally, patients should understand the importance of follow-up care. The goals of treatment should be a partnership between patient and health-care provider.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCE
Refer patients with macular edema, severe nonproliferative retinopathy, or any degree of proliferative retinopathy to an ophthalmologist immediately for treatment.	A	Yanoff & Cameron, 2016 AAO, 2016
Patients with type 1 diabetes should have annual screenings beginning 5 years after diagnosis. Those with type 2 diabetes should have annual screenings beginning at the time of diagnosis.	A	AAO, 2016
Optimal glycemic control reduces the risk and progression of retinopathy.	A	Diabetes Control and Complications Trial (DCCT) Research Group, 1993
Optimal glycemic control and blood pressure control reduces the risk and progression of retinopathy.	A	Yanoff & Cameron, 2016
Laser photocoagulation reduces the risk of vision loss in patients with proliferative retinopathy, clinically significant macular edema, and some cases of severe nonproliferative retinopathy.	A	ACCORD Study Group & ACCORD Eye Study Group, 2010
In macular edema that is clinically significant, treatment with anti-VEGF is superior to laser therapy, but intravitreal steroid injection may also be used.	A	Yanoff & Cameron, 2016 AAO, 2016
Aspirin therapy does not prevent the progression of retinopathy, nor is it contraindicated for prevention of cardiovascular disease in the presence of retinopathy, because it does not increase the risk of retinal hemorrhage.	A	AAO, 2016 ACCORD Study Group & ACCORD Eye Study Group, 2010

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

RHINITIS

Signal Symptoms: Clear rhinorrhea (anterior or posterior drainage), nasal congestion and itching, and sneezing. May also present with itching of the palate, ear canals, eyes, and reduced olfaction.

Description: Rhinitis is an inflammation of the nasal mucosa and includes allergic rhinitis (AR), nonallergic rhinitis (NAR), and nonallergic rhinitis with eosinophilia syndrome (NARES) (McCullan, 2014).

Etiology: AR is classified by pattern (e.g., seasonal or perennial), symptom frequency, severity, and evidence of immunoglobulin E (IgE) sensitization to an allergen. Symptoms occur due to the response of the nasal mucosa to airborne allergens

in genetically predisposed individuals. The mechanism of the three-phase type 1 hypersensitivity response experienced in AR is due to IgE-mediated mast cell degranulation and activation (Welch & Kau, 2014). In the sensitization phase, the genetically predisposed person inhales an allergen, which is presented by dendritic cells to T cells in lymph nodes (Rote & McCance, 2014). This causes T-helper 2 cells to increase and release cytokines, causing B cells to class-switch into plasma cells that produce antigen-specific IgE antibodies (Settipane & Schwindt, 2013). During the challenge phase, cross-linking of mast cells initiates degranulation of the mast cell and release of symptom-eliciting mediators. The final phase, elicitation, has an early and late-stage response. The early phase response (sneezing, itching, rhinorrhea), occurring within 5 to 30 minutes of exposure, is primarily due to histamines. The late phase response (primarily nasal congestion) occurs 2 to 8 hours after exposure and is due to the influx of basophils, eosinophils, and T-lymphocytes into the nasal mucosa. Priming is a process in which a susceptible person becomes more sensitive to smaller amounts of the allergen and more reactive to nonallergic substances such as irritants (Settipane & Schwindt, 2013).

AR may be seasonal, caused by pollens from trees, grass, or weeds pollinating in the spring or fall. Pollen from flowers is rarely allergenic, because the pollen is heavier and is carried by bees from plant to plant (American Academy of Allergy, Asthma, & Immunology, n.d.). Perennial AR is often related to environmental exposure to pollutants, animal dander, dust, and indoor and outdoor molds. Response to irritants such as cigarette smoke, pollution, and odors such as perfumes and diesel may cause rhinitis but is not IgE mediated.

NAR is not mediated by the production and interaction of IgE, yet symptoms are similar to AR (rhinorrhea and nasal congestion). However, the patient usually does not experience itching, sneezing, or conjunctival symptoms (McMullan, 2014; Settipane & Kaliner, 2013). The subtypes of NAR include vasomotor, atrophic nonallergic, rhinitis medicamentosa, drug-induced, hormonal, gustatory, infectious, or NARES. The cause of NAR is not well understood, but is thought to be due to atopy, nociceptive nerve sensor and ion channel protein dysfunction, and autonomic dysfunction (Settipane & Kaliner, 2013).

Vasomotor rhinitis symptoms have been linked to temperature changes, inhaled irritants, bright lights, and strong odors. Atrophic NAR is probably due to degeneration and atrophy of nasal membranes and bony structures. Rhinitis medicamentosa is frequently due to the overuse of topical over-the-counter (OTC) nasal decongestants. Drug-induced rhinitis may occur with use of alpha-receptor agonists, angiotensin-converting enzyme (ACE) inhibitors, aspirin, beta blockers, NSAIDs, oral contraceptives, and phosphodiesterase—5 selective inhibitors. Hormonal rhinitis may occur in patients with hypothyroidism, who are menstruating, or are pregnant. Gustatory rhinitis can occur after food or alcohol ingestion and is thought to be mediated vagally. Infectious rhinitis is most commonly caused by acute viral syndrome. Chronic infectious rhinitis results from a secondary bacterial infection. Marked by sneezing fits, profuse rhinorrhea, nasal itching, and reduced olfaction, NARES is thought to be a precursor to asthma, aspirin-aggravated respiratory disease, and nasal polyps (McMullan, 2014; Settipane & Kaliner, 2013).

Occurrence: AR occurs in 10% to 30% of adults (approximately 60 million people) in the United States. NAR occurs in 7% of the population, affecting 22 million people (Settipane & Kaliner, 2013; Settipane & Schwindt, 2013).

Age: AR is most common between ages 10 and 39 years, declining after age 40 years. NAR is a condition of adulthood and is more common in older adults.

Gender: Adult men and women have an equal incidence of rhinitis (McMullan, 2014). Women who are 40 years old and older have a higher incidence of NAR (Settipane & Kaliner, 2013).

Ethnicity: Not significant.

Contributing Factors: Most important, AR usually is associated with a strong family history of atopic disorders, including asthma, atopic eczema, urticaria, food allergy, stinging-insect-venom allergy, and anaphylaxis. Exposure to airborne allergens in the environment, such as animal dander, dust mites, molds, or seasonal pollens, triggers AR. A variety of factors are thought to contribute to NAR including anatomical abnormalities, infections, medications (especially in the elderly), immunodeficiency, tumors of the nasopharynx and paranasal sinuses, pregnancy, hypothyroid disease, granulomatous disease (Wegener's disease and sarcoidosis), and leakage of cerebrospinal fluid. Vasomotor rhinitis can be triggered by strong odors or fumes, temperature or barometric pressure changes, and psychological factors. Age-related changes in the geriatric population should be considered, because physiological changes in this age group, as well as medication side effects and drug–drug interactions, may contribute to the development of symptoms (Hellings et al., 2013; Settipane & Kaliner, 2013).

Signs and Symptoms: Patients with seasonal AR report rhinorrhea, sneezing, obstructed nasal passages, and pruritic eyes, nose, and oropharynx during the spring and fall. Patients with perennial AR have similar symptoms associated with exposure to environmental allergens, typically in their homes.

Review of Symptoms: Questions to ask patients with symptoms of rhinitis include: Does the patient associate the symptoms with a season, place, time of day, or activity? Is there a family history of allergic diseases? Do symptoms seem to occur during times of stress or during weather or temperature changes? What is the patient's quality of life (sleep disturbance, fatigue, missed work)? Does the patient use intranasal drugs such as cocaine?

Physical Examination: Although the patient history is key in establishing the diagnosis, the physical examination will assist in ruling out other differential diagnoses. A full head, eyes, ears, nose, and throat (HEENT), skin, and respiratory examination should be performed. Objective findings consistent with AR includes atopic dermatitis, scleral erythema and injection, allergic shiners and Dennie's lines (infraorbital hyperpigmentation and creasing), salute sign (creasing of the nasal dorsum), swollen and pale turbinates, and tonsillar enlargement. As asthma often accompanies AR, the patient also must be assessed for wheezing (McMullan, 2014).

Postnasal discharge and congestion are common complaints in vasomotor rhinitis; symptoms associated with AR

may coexist. Individuals with atrophic rhinitis report a bad taste along with congestion and thick postnasal discharge. In AR, pink-to-red, dry nasal mucosa is found on examination. Symptoms of infection such as fever and purulent nasal discharge should be ruled out on history and physical examination.

Diagnostic Tests: Skin testing involves scratching the surface of the skin with a single stylus for each allergen. Depending on the practice, providers may begin with skin prick testing and proceed to intradermal testing if the results are negative (Hollander, 2014). It is important to note that some medications (antihistamines, tricyclic antidepressants, oral and topical corticosteroids, and leukotriene antagonists) will interfere with skin allergy testing. Total serum IgE testing may be used when skin testing is not possible due to medications or skin conditions; however, mild elevation of IgE is not useful in the diagnosis of allergies and serum IgE testing should not be used as a screening test (Hollander, 2014). Radiographic sinus films or CT scan should not be used in the absence of recurrent infection (Seidman et al., 2015). Patients with chronic rhinitis may be referred for evaluation of obstructive sleep apnea in the presence of sleep-disordered breathing (Seidman et al., 2015).

Differential Diagnosis:

- Viral or bacterial upper respiratory infection
- Acute or chronic sinusitis
- Otitis media
- Nasal polyps
- Deviated nasal septum
- Hypothyroid disease
- Tumor
- Foreign body in the nose

Treatment:

Allergic Rhinitis: Pharmacological therapy is directed at control of the specific patient symptoms. Avoid first-generation antihistamines, such as diphenhydramine, because of safety issues related to sedating effects. The lowest possible dose of these medications should be used in older adults to avoid excessive sedation. Because of their anticholinergic effects, these drugs may worsen certain conditions that are common in the older adult patient, such as benign prostatic hypertrophy, bladder neck obstruction, and narrow-angle glaucoma.

Intranasal corticosteroid nasal sprays are the most effective treatment for AR due to their ability to prevent early and late phase responses (McMullan, 2014). It is best to begin treatment with intranasal sprays or in combination with oral antihistamines. Oral leukotriene receptor antagonists such as monelukast are not considered first-line treatment but may be effective for patients with asthma and seasonal AR (McMullan, 2014; Seidman et al., 2015). Topical intranasal antihistamine preparations are helpful in avoiding systemic side effects but may cause local irritation and dryness of the nasal mucosa.

Intranasal Antihistamines:

- Azelastine (Astelin) nasal spray, two sprays each nostril twice daily
- Olopatadine (Patanase), two sprays each nostril twice daily

Intranasal Corticosteroids:

- Beclomethasone dipropionate (Qnasl), two sprays each nostril once daily
- Budesonide (Rhinocort), two sprays each nostril twice daily
- Ciclesonide (Omnaris, Zetonna), two sprays each nostril once daily
- Flunisolide (Nasarel, Nasalide), two sprays each nostril twice daily
- Fluticasone furoate (Veramyst), two sprays each nostril once daily
- Fluticasone propionate (Flonase), two sprays each nostril once daily or one spray each nostril twice daily
- Mometasone furoate (Nasonex), two sprays each nostril once daily
- Triamcinolone acetonide (Nasacort), two sprays each nostril once daily

While second-generation antihistamines are not as effective as intranasal steroids, some patients cannot tolerate nasal sprays and find the once-daily dosing and rapid symptom relief to be advantageous. Most have few side effects, however, cetirizine and levocetirizine may be sedating (Seidman et al., 2015).

Second-Generation Antihistamines:

- Loratadine (Claritin), 10 mg once daily (10 mg every other day in hepatic or renal insufficiency with glomerular filtration rate less than 30 mL/min)
- Desloratadine (Clarinex), 5 mg once daily (give every 48 hours to start with hepatic or renal insufficiency)
- Cetirizine (Zyrtec), 10 mg once daily (5 mg once daily with hepatic or renal insufficiency)
- Levocetirizine (Xyzal) 5 mg once daily
- Fexofenadine (Allegra), 180 mg once daily, 60 mg twice daily (60 mg once daily with decreased renal function)

These antihistamines should not be prescribed for patients taking antifungal medications, macrolide antibiotics, tricyclic antidepressants, or class Ia antiarrhythmics, because their interaction with these drugs may cause life-threatening cardiac dysrhythmias. They also are contraindicated for patients with a history of congestive heart failure, coronary artery disease, or liver disease.

Nonallergic Rhinitis:

- Avoid known irritants.
- Nasal corticosteroids (see earlier) for NARES.
- Nasal corticosteroids and elimination of topical decongestant sprays in rhinitis medicamentosa.
- Ipratropium bromide (Atrovent) nasal spray 0.03%, 0.06%, two sprays each nostril two to four times daily, is an anticholinergic for local control of rhinorrhea.
- Pseudoephedrine, 30 to 60 mg orally every 4 hours, a decongestant, decreases nasal mucosa swelling but has little effect on other rhinitis symptoms; it is best used briefly and intermittently because of central nervous system (CNS) side effects. It is contraindicated in older adult patients with poorly controlled hypertension, coronary artery disease, and a history of cerebrovascular accident.
- Oral corticosteroids are recommended only for severe nasal obstruction, such as that caused by rebound rhinitis or nasal polyps, and require individualized dosing.

Follow-Up: The patient should return in 2 to 4 weeks after beginning therapy to review the response to medications and the effectiveness of environmental control.

Sequelae: Epistaxis, acute and chronic sinusitis, and the development of nasal polyps are complications of rhinitis.

Prevention/Prophylaxis: Teach patients with AR to avoid irritants and to try to control environmental risk factors. Discuss the need to take intranasal steroid sprays and/or antihistamines regularly to control symptoms.

Referral: When symptoms persist or worsen, or are accompanied by comorbidities such as nasal polyps or asthma, refer the patient to an otolaryngologist or allergist (McMullan, 2014). In patients with chronic rhinitis, the otolaryngologist may prescribe a short-term course of antibiotics along with oral prednisone. Sinus surgery may be indicated in order to correct obstruction, a major component of recurrent sinusitis. An allergist may skin prick test patients in order to determine their sensitivities and place the patient on allergy immunotherapy.

Education: Avoid the use of OTC nasal sprays for more than 3 consecutive days, and warn patients about the sedating side effects of first generation antihistamines. Teach the patient with perennial allergies to use their medications (intranasal steroid sprays and second-generation antihistamines) regularly. Waiting until symptoms have progressed may result in the lack of efficacy of either the oral antihistamine and/or the intranasal steroid spray.

Discuss specific environmental control with the patient. For patients with house dust mite sensitivity, advise them to use mite-impermeable mattress covers, wash floors each day, wash bedding in hot water twice weekly, and remove upholstered furniture from the bedroom. For control of seasonal pollen from trees and grass in the spring, and weeds in the summer and fall, it is essential that patients keep their windows closed and air conditioning on during periods of high pollen counts, especially from 5:00 am to 10:00 am. Pollen easily enters the screens of windows and deposits on furniture and bedding. High-efficiency particulate air (HEPA) filters can be used in bedrooms and living areas. Patients should also be encouraged to shower and change their clothes when coming in from the outside. Clothes should be dried in an automatic dryer, not outdoors. Outdoor mold exposure can be decreased by avoiding areas of decaying plants, rotting leaves, and other debris. Wearing a mask when working outside also may be beneficial (National Institute of Environmental Health Sciences, n.d.; Seidman et al., 2015).

Patients with animal dander sensitivity can be more difficult to treat if the family chooses to keep the pet. Pets must be kept out of the patient's room at all times, and a HEPA filter should be run in the bedroom. Because animal dander is sticky, bed clothes should be clean before entering the bedroom. Washing the hands and face after pet exposure also is advised (National Institute of Environmental Health Sciences, n.d.). There may be some benefit gained from washing dogs twice weekly for 5 minutes and then blowing them dry; no such benefit has been found with bathing cats (Seidman et al., 2015).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Topical corticosteroid intranasal sprays are the most effective treatment of AR when used regularly.	A	Seidman et al., 2015
Oral second-generation antihistamines are effective, either alone or in conjunction with intranasal steroid sprays, for treatment of AR with sneezing and itching.	A	Seidman et al., 2015
Patients with comorbidities such as asthma, atopic dermatitis, sleep-disordered breathing, conjunctivitis, and otitis media should receive further testing.	A	McMullan, 2014
Patients should implement environmental controls such as frequent washing of bedding and floors, using mite-impermeable mattress covers and HEPA filters, keeping pets out of the bedroom, and keeping the windows closed.	B	Seidman et al., 2015
Mold exposure can be decreased by eliminating sources of moisture, avoiding areas of decaying plants, rotting leaves, and other debris. Wearing a mask when working outside can also be of benefit.	A	National Institute of Environmental Health Sciences, 2018
Specialized allergy testing (skin or blood testing) may be performed on patients who do not respond to empiric treatment.	B	Seidman et al., 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

A 76-year-old woman presents today with complaints of nasal drainage, clearing of throat, and occasional nasal congestion, especially on waking in the morning. She has recently moved into an independent living center after living in her home for 40 years. She states that, although she has had these symptoms before, generally the symptoms appeared in the spring, and she associated the nasal drainage with pollination. Because it is winter, she could not identify the trigger of her symptoms.

Chief complaint: Persistent "runny nose" for 3-week duration, associated clearing of throat, and nasal congestion on awakening in the morning.

Objective data: Blood pressure (BP) 130/84, temperature 98.6, pulse 78, respiratory rate 20.

1. How will you use this information to prepare for today's visit?
2. What additional subjective data are you seeking, to include history of any allergies?
3. What additional objective data will you be assessing for?
4. What are the differential diagnoses that you are considering?
5. What laboratory tests will help you rule out some of the differential diagnoses?
6. What is your treatment, and what specific information about the prescription will you give to this patient?
7. What are the potential complications from the treatment ordered?
8. What additional specific laboratory tests will you consider ordering?
9. What additional patient teaching may be needed?
10. What type of specialist will you be consulting for this patient?

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CHAPTER 8

Chest Disorders

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ASSESSMENT OF THE CARDIOVASCULAR SYSTEM

Key symptoms for cardiovascular assessment include dizziness, syncope, orthopnea, angina, edema, and claudication (Bickley, 2013; Kane, Ouslander, Resnick, & Malone, 2018). Differentiating normal from abnormal cardiac function in an older adult can be a challenge because one in two older persons has cardiac disease. More specifically, almost one-half of all individuals older than age 60 years have severe coronary artery narrowing with a respective increase in myocardial demand. Of these, less than 50% have clinical signs and symptoms of this process. Cardiovascular disease is the most prominent cause of disability in this age group and the leading cause of death, accounting for about one-half of all deaths among older adults.

ASSESSMENT OF RISK FACTORS FOR CORONARY ARTERY DISEASE

Although some risk factors for coronary artery disease (CAD) can be remedied, others cannot. The two most important risk factors for atherosclerosis that cannot be remedied are advanced age and male gender. The major remediable risk factors are high blood pressure (BP), cholesterol levels, and smoking.

Nearly 13 million Americans have CAD, and the incidence increases with age (Keller, Sabatino, Winland-Brown, Porter, & Keller, 2015). Since the 1970s, the mortality rate from CAD has shown a pronounced decline in the United States. Most of this decline can be attributed to changes in lifestyle and improved technologies such as thrombolytic drugs. The decline in cardiovascular disease has occurred in older as well as younger age groups, suggesting that the effects of risk factor modification persist well into later life.

Blood Pressure

The systolic blood pressure (SBP) rises with age; the diastolic blood pressure (DBP) remains the same or drops a bit. Established hypertension is a risk factor for cardiovascular disease in the geriatric age group, with systolic elevations posing a greater risk than diastolic elevations. Overall, the risk for both genders of experiencing a cardiovascular event or death is two to three times higher in those with significant hypertension than in those who are normotensive. Isolated systolic hypertension (ISH) is defined as an SBP greater than 160 mm Hg and a DBP of less than 90 mm Hg. Aggressive treatment of ISH has demonstrated a significant reduction in stroke, myocardial infarction (MI), and sudden cardiac death.

Cholesterol

Serum cholesterol levels rise with age, up to age 60 years; thereafter, these levels begin to drop. The risk of the effects of an elevated cholesterol level persists from middle age to extreme old age. Aggressive efforts are often indicated to lower low-density lipoprotein (LDL) levels and raise high-density lipoprotein (HDL) levels for cardiac protection. Controversy exists about the role of statins in primary prevention of low-risk older adults (Keller et al., 2015; Patterson, Gazziano, & Greenland, 2014).

Smoking

Smoking is an independent significant risk factor for atherosclerosis and coronary heart disease (CHD) (Carter et al., 2015). Smoking cessation is strongly advised as both primary prevention (Patterson et al., 2014) and secondary prevention for those with known CAD or peripheral vascular disease (American Heart Association [AHA]/American College of Cardiology [ACC], 2013).

Additional proven or postulated risk factors for cardiovascular disease in older adults include obesity, lack of exercise, left ventricular hypertrophy, and impaired glucose tolerance. Older adult individuals are most likely to have a combination of risk factors, which has a cumulative effect of increasing the risk of CAD. Control of hypertension is clearly the most potentially remediable risk factor. Evidence is compelling for discontinuation of cigarette smoking at any age. More information is needed regarding the effectiveness and feasibility of lowering cholesterol levels, weight reduction, improved exercise plans, and strict control of blood glucose levels, with respect to the incidence of CHD, particularly in those of most advanced age.

Cardiac risk assessment tools, including the Global Framingham Risk Assessment, are available at https://www.nhlbi.nih.gov/health-pro/guidelines/in-develop/cardiovascular-risk-reduction/risk-assessment/. Assessment for metabolic syndrome and obesity, including body mass index (BMI) and waist circumference, also enhances risk assessment.

Physiological Changes

The size of the heart remains essentially unchanged, although some increase in left ventricular wall thickness has been demonstrated, even in older individuals who do not have cardiovascular disease. Left ventricular hypertrophy is usually due to increased cardiac demand, most likely caused by an increase in peripheral resistance. Peripherally, the vessels become atherosclerotic and arteriosclerotic, and the SBP increases with age. Structural and physiological changes in the aging cardiovascular system cause decreased capacity to endure stresses, decline in physical activity performance, and limited functional reserve capacity (Keller et al., 2015). Fat deposits accumulate around the sinoatrial (SA) node. The number of pacemaker cells usually has decreased by age 75 years. Baroreceptors become less sensitive with age, and the response to changes in BP often is blunted.

These physiological changes have little functional impact on the aging heart at rest, but with exercise or stress, they render the aging heart less capable of increasing and sustaining an increase in cardiac output. The maximal heart rate declines approximately 30% between ages 20 and 80 years. Cardiac dilation and increased stroke volume compensate somewhat for the diminished heart rate during exercise (Keller et al., 2015).

Symptom Assessment

Using the Onset, Location, Duration, Characteristics, Aggravating factors, Relieving factors, Timing (OLDCART) mnemonic or Precipitating/Palliating factors, Quality, Radiation, Severity, Timing (PQRST) mnemonic, ask the patient about chest pain, shortness of breath, dizziness, palpitations, or edema. Establish the patient's usual baseline activities of daily living (ADLs) and activity level and ask how symptoms are affecting this. Some older adults experience palpitations as a nervous feeling, so include that in your questioning. Chest pain may be presented as heartburn or indigestion, particularly if the patient also has gastroesophageal reflux disease (GERD). Shortness of breath may be felt as anxiety but is a more common presentation of an acute coronary event than chest pain. Atypical presentation for MI includes vague symptoms such as nausea, decreased ADL status, and fatigue without chest pain (Cash & Gunter, 2016; Keller et al., 2015).

CLINICAL EXAMINATION FEATURES

In the actual physical assessment of the older adult, the apical impulse is difficult to locate, as the chest wall undergoes changes due to the intercostal muscles weakening, resulting in an increased anteroposterior diameter (Bickley, 2017).

Auscultation in the geriatric patient frequently reveals changes in normal heart sounds, extra heart sounds, bruits, and murmurs. The S_1 is more easily heard, and splits of the first heart sound are more easily detected because of an ejection sound that occurs when the aortic valve cusp tissue fails to fold into the vessel wall during ejection. A change in the loudness of S_1, accompanied by a slow heart rate, may indicate heart block. The S_2 split on inspiration is narrower or absent because of decreased compliance of the pulmonary vasculature. An opening snap occurs when the mitral or tricuspid valve produces minor vibrations of increased intensity caused by more rapid cusp movement or a resistance to inflow caused by cusp fusion (Bickley, 2017). An audible opening snap is the best physical sign of mitral stenosis, but it becomes less obvious in the older adult whose valves are rigid and calcified.

Ventricular filling sounds, best heard with the stethoscope bell, are caused by the halting of the ventricle after ventricular filling. The physiological S_3, heard most clearly at the apex, disappears by the fourth decade as the ventricle stiffens and filling becomes less rapid. An S_3 in an individual more than 50 years old is usually pathological and reflects an increased filling rate, indicating heart failure or mitral regurgitation (Bickley, 2017). Early diastolic filling is reduced in healthy older adults resulting in an end-diastolic volume maintained by an increase in atrial contribution to left ventricular filling. This condition may cause an S_4, which in the absence of other findings, is considered normal in older adults.

The carotid arteries should be assessed routinely for the presence of bruit. Asymptomatic carotid bruit is a risk factor for stroke (Bickley, 2017). Studies demonstrate that a vessel is occluded more than 50% by the time a bruit can be heard. In older adults assess the top level of the internal jugular pulsation when evaluating jugular venous pressure. Do not use the external jugular veins, because they may be sclerosed and appear to be falsely distended or may yield a falsely low pressure reading.

Prolonged extra heart sounds (murmurs), particularly systolic ones, are quite common in elderly individuals. Systolic murmurs, which often indicate aortic valve disease, occur in more than 50% of individuals over age 70 years. The soft systolic ejection murmur is due to the dilation and the decrease in compliance of the aorta caused by stiffening of the aortic cusps without obstruction (aortic sclerosis). Loud murmurs, which usually indicate aortic stenosis, are often accompanied by a slow rising carotid pulse and left ventricular hypertrophy. An apical pansystolic or late systolic murmur also occurs frequently in older persons, resulting from floppy valves that become regurgitant over time. The two most common causes of mitral regurgitation are papillary muscle dysfunction (usually due to MI) and mitral annular calcification. Diastolic murmurs, which are almost always pathological in elderly persons, may be caused by heart block, aortic regurgitation, or mitral stenosis (LeBlond, Brown, Suneja, & Szot, 2014).

In the older adult, additional features of cardiac disease may be seen on physical examination of systems other than the heart. The eye examination may reveal the thick corneal arcus seen with lipid abnormalities or the funduscopic findings of atherosclerosis. The skin may demonstrate xanthomas or cholesterol nodules and changes in skin temperature,

indicating a metabolic or peripheral vascular disease causing or contributing to cardiac disease. The presence of edema must be determined. The clinical cardiac examination features in the geriatric patient are:

- Structural wall changes are common.
- S_1 is more easily heard.
- S_2 split is narrower.
- S_4 is common.
- Systolic murmurs are common.
- Bruits (carotid, abdominal) are common.

ASSESSMENT OF THE RESPIRATORY SYSTEM

Key symptoms warranting evaluation include increasing dyspnea and persistent cough (Kane et al., 2018). In order to assess the respiratory system accurately in the older adult, the nurse practitioner must be familiar with changes in tissue and in the musculoskeletal and respiratory systems that are caused by aging.

The aging process is characterized by a loss of elasticity and flexibility in collagen and elastin tissue components; this impedes the normal expiratory recoil of the lung. The concurrent decrease in body water composition dries mucous membranes and interferes with the protective functions of the upper airway in expelling foreign material. Loss of elastin also affects the alveoli and the basement membrane of the capillary wall, where gas exchange occurs. A thickening occurs in both areas, limiting the amount of diffusion (Bickley, 2017; Shanker, Rojas & Caufield, 2017). Musculoskeletal changes, including calcification of costal cartilage and weakening of inspiratory muscles, result in a relaxation of the skeletal contours and an increased anteroposterior diameter, causing a barrel chest, although not as pronounced as that found in patients with chronic obstructive pulmonary disease (COPD). An exaggeration of the thoracic curvature may also occur, with some tracheal displacement. Expiratory muscles must work harder because expiration is no longer a passive act, resulting in use of accessory muscles. The residual capacity of the lungs is increased and inspiratory reserve capacity lessened. Air tends to fill the apices and not the bases. Because of decreased muscular strength, the cough reflex is not as forceful or as effective. All of these changes occur gradually and are hardly noticeable unless a physiological challenge or stress arises. Even in such an event, exertional dyspnea may be the only observed change in the healthy older adult. Healthy lifestyle behaviors also serve to mitigate these age-related changes.

Healthy older adults or those with stable, chronic problems are unlikely to seek care unless there is a change in functional capabilities related to breathing, such as decreased exercise tolerance, shortness of breath with exertion, or easy fatigability. In frail older adults an increase in respirations, sweating, or anorexia may be the only indication of a respiratory problem. History questions should address the symptom(s) using the OLDCART or PQRST symptom analysis formats. Is there a prior history of the problem(s) or of any respiratory problem(s)? Does the patient use tobacco, or has he or she used it in the past? Is there a history of occupational exposures or lung problems? Has the possibility of tuberculosis (TB) been eliminated? Is the patient a silent aspirator? Any changes in medications, including the use of over-the-counter (OTC), herbal, and homeopathic remedies, should be addressed. Is there a cough, sputum production, or hemoptysis? Is the patient wheezing? If there is pain, have cardiac or musculoskeletal causes been ruled out? If there is dyspnea, is it related to activity, or does it occur at rest or when lying down? How many pillows are used? Specific questions about changes in endurance, stair-climbing, or ADLs are necessary to quantify the extent of the problem. Does the problem interfere with eating? Has there been a 10-pound weight loss or gain in the past 6 months? Has the patient received the influenza vaccine and pneumococcal vaccine? Is HIV a consideration?

Physical assessment of the respiratory system begins with general observation of the patient: rate and ease of respirations at rest, conversational ability (limited by breathing problems), use of accessory muscles, posture, color, pursed lips, circumoral or nail bed cyanosis, and capillary refill. The chest and upper back should be exposed for full inspection and examination. Are both sides symmetrical, with equal respiratory effort and diaphragmatic movement? Is there any evidence of kyphosis, lordosis, or scoliosis? Rapid respiratory rates are common with pneumonia, fever, or COPD.

Palpation of the posterior chest to confirm equal chest expansion is done by placing the thumbs on the chest wall near the vertebrae at T9 and T10 and spreading the hands. A small fold of skin should be between the thumbs. When the patient takes a deep breath per your instruction, the thumbs should move apart symmetrically. If pneumonia or consolidation is suspected, palpating for tactile fremitus may be helpful. Using the ball of the hand (metacarpophalangeal area of the palm) or the ulnar side of the hand, feel the vibrations in a side-to-side pattern while the patient says "ninety-nine." The vibrations should feel the same on either side and should be stronger in the upper areas. Increased fremitus occurs with marked consolidation, such as lobar pneumonia. Percussion of the posterior chest for resonance and symmetry is the next step, again using a side-to-side approach. Dullness is indicative of increased density, as found with pneumonia, tumor growth, or pleural effusion. Hyperresonance is found with air trapping in emphysema or with pneumothorax. Auscultation of posterior breath sounds for symmetry, presence or absence of adventitious sounds, and air movement is done with the patient seated and the arms resting on the knees to retract the scapulae. The patient is instructed to take a deep breath through the mouth, with the mouth open, inhaling and exhaling. Decreased breath sounds are common with emphysema, pleurisy, or pleural effusion. Wheezing is commonly heard with asthma or acute bronchitis, although it may also be part of the background of chronic bronchitis or COPD. Crackles are heard with congestive heart failure (CHF), pulmonary edema, or pneumonia; basilar crackles that clear with coughing or deep breathing are not pathological. Older patients become light-headed with deep breathing and may need this part of the examination to be spaced out over time. If pneumonia is suspected, vocal fremitus techniques such as bronchophony, egophony, or whispered pectoriloquy may be indicated. Examination of the anterior chest follows the same sequence. The right middle lobe is auscultated anterolaterally.

ASTHMA

Signal Symptoms: Wheezing, shortness of breath, cough (especially at night), chest tightness.

Description: The Global Initiative for Asthma (GINA) provides an operational definition of asthma as a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role. The chronic inflammation is associated with airway hyperresponsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or early in the morning. These episodes are usually associated with widespread, but variable, airflow obstruction within the lung that is often reversible either spontaneously or with treatment (Global Strategy for Asthma Management and Prevention, 2016 update).

Etiology: Both GINA and the National Asthma Education and Prevention Program Expert Panel Report–3 (NAEPP-EPR-3) (2007) cite a lack of agreement on definition and etiology due to the complexity and variation in manifestation of asthma as well as the lack of identification of the phenotype qualities of asthmatics. Characteristics that are agreed on are inflammation, airway hyperresponsiveness, airway obstruction, and clinical symptoms (NAEPP-EPR-3, 2007).

Occurrence: In the United States, asthma is more prevalent in African Americans—the age-adjusted rate for those over 18 years of age is 8.7%; the rate for Caucasians is 7.6%; and the rate for Hispanics/Latinos is 5.8% (U.S. Department of Health and Human Services, Centers for Disease Control and Prevention [CDC], 2016, March). Worldwide, it is estimated that asthma affects up to 334 million people and is responsible for 346,000 deaths each year (Global Asthma Network, 2014; Global Strategy for Asthma Management and Prevention, 2016 update).

Age: The age-adjusted rate for adults 65 to 74 years old is 6.9%; for age 75 and older it is 6.2% (U.S. Department of Health and Human Services, CDC, National Center for Health Statistics, 2016).

Gender: In adult asthma there are more females with the disease than males (Wheaton et al., 2016). The age-adjusted rate for females over 18 years old is 9.7%; for males it is 5.4% (U.S. Department of Health and Human Services, CDC, National Center for Health Statistics, 2016).

Ethnicity: African Americans have a higher rate of asthma in the United States.

Contributing Factors: Exposure to allergens, respiratory infections, family history/genetic predisposition, atopy, occupational irritants, tobacco smoke (smoking and passive smoke exposure)), high levels of indoor and outdoor pollution, and obesity; rhinitis is a specific factor in older adults (Global Strategy for Asthma Management and Prevention, 2016 update; Miller & Sawlani, 2013; Tarasidis & Wilson, 2015). Multiple genes contribute to the development of asthma in different ethnic groups; research into genetic phenotypes is ongoing (Global Strategy for Asthma Management and Prevention, 2016 update; Toskala & Kennedy, 2015).

Signs and Symptoms: Recurrent wheezing, cough (especially occurring at night), recurrent chest tightness, and recurrent breathing difficulty (Miller & Sawlani, 2013). Symptoms may occur in conjunction with a respiratory infection, weather changes, contact with environmental allergens, strong emotional reactions, animal fur, mold, exercise, or other triggers. Medications including aspirin, NSAIDs, and beta blockers can exacerbate symptoms. Older adults are less likely to sense dyspnea related to airway obstruction, even in the presence of cough, chest tightness, or wheezing (Vaz Fragoso, 2016). Other symptoms may include rhinorrhea with postnasal drip.

Diagnostic Tests: Spirometry or pulmonary function testing, particularly forced expiratory volume in 1 second (FEV_1), FEV_6, forced vital capacity (FVC), and FEV_1/FVC ratio before and after bronchodilator challenge, showing an improvement of 12% and 200 mL, indicates reversible airway obstruction (Brigham & West, 2015). This is helpful in differentiating asthma from COPD, particularly in an older adult with a prior smoking history. A history of atopy or allergen testing may establish an allergic component. In a patient with dyspnea, measurement of brain natriuretic peptide, also known as B-type natriuretic peptide (BNP), and a chest x-ray may be needed to eliminate other conditions. If spirometry is near normal, bronchoprovocation such as a methacholine challenge test may help to differentiate other conditions with a similar presentation (NAEPP-EPR-3, 2007; Vaz Fragoso, 2016). Older adults tend to have increased hyperresponsiveness to bronchoprovocation even when adjusting for the baseline amount of airway obstruction, atopy, or smoking status (Vaz Fragoso, 2016). Vaz Fragoso (2016) suggests that in response to a low provocative challenge (PC-20), a PC-20 threshold for hyperresponsiveness should be decreased to less than 4 mg/mL in lieu of the standard less than 8 mg/mL. The American Thoracic Society (ATS) has endorsed the use of FeNO, the fractional nitrous oxide measurement in exhaled breath, as a biomarker for eosinophilic inflammation and a complimentary tool to assess airway disease, including asthma (Bjermer et al., 2014). At the present time, use and utility of this test are still evolving.

Differential Diagnosis:

- COPD
- CHF
- Pneumonia
- Upper respiratory infection (URI)
- Pulmonary embolism (PE)
- Anxiety disorder
- Vocal cord dysfunction (VCD)
- Cough secondary to drugs
- GERD
- Allergic bronchopulmonary aspergillosis (ABPA)

Treatment: The NAEPP-EPR-3 (2007) has identified four components of asthma management:

- Assessment and monitoring measures, including patient history, physical examination, and objective testing, such as spirometry, to diagnose asthma and determine severity and initial and ongoing level of control

Assessing severity and initiating treatment for patients who are not currently taking long-term control medications

Components of Severity		Classification of Asthma Severity ≥12 years of age			
			Persistent		
		Intermittent	Mild	Moderate	Severe
Impairment Normal FEV_1/FVC: 8–19 yr 85% 20–39 yr 80% 40–59 yr 75% 60–80 yr 70%	Symptoms	≤2 days/week	>2 days/week but not daily	Daily	Throughout the day
	Nighttime awakenings	≤2x/month	3–4x/month	1x/week but not nightly	Often 7x/week
	Short-acting $beta_2$-agonist use for symptom control (not prevention of EIB)	≤2 days/week	>2 days/week but not daily, and not more than 1x on any day	Daily	Several times per day
	Interference with normal activity	None	Minor limitation	Some limitation	Extremely limited
	Lung function	• Normal FEV_1 between exacerbations • FEV_1 >80% predicted • FEV_1/FVC normal	• FEV_1 >80% predicted • FEV_1/FVC normal	• FEV_1 >60% but <80% predicted • FEV_1/FVC reduced 5%	• FEV_1 <60% predicted • FEV_1/FVC reduced 5%
Risk	Exacerbations requiring oral systemic corticosteroids	0–1/year	≥2/year →		
		← Consider severity and interval since last exacerbation. → Frequency and severity may fluctuate over time for patients in any severity category. Relative annual risk of exacerbations may be related to FEV_1.			
Recommended Step for Initiating Treatment		Step 1	Step 2	Step 3	Step 4 or 5
				and consider short course of oral systemic corticosteroids	
		In 2–6 weeks, evaluate level of asthma control that is achieved and adjust therapy accordingly.			

Key: FEV_1 forced expiratory volume in 1 second; FVC, forced vital capacity; ICU, intensive care unit

FIGURE 8-1. Classifying asthma severity and initiating treatment in youths ≥12 years of age and adults. FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity. *(National Asthma Education and Prevention Program [2007, August]. Third Expert Panel on the Diagnosis and Management of Asthma. Expert Panel Report 3: Guidelines for the diagnosis and management of asthma.* Section 4: *Stepwise approach for managing asthma in youths ≥12 years of age and adults. Bethesda, MD: National Heart, Lung, and Blood Institute. Available from* www.ncbi.nlm.nih.gov/books/NBK7222.*)*

- Patient, family, and professional education for partnership in asthma management
- Control of comorbidities and environmental factors affecting asthma
- Pharmacotherapy

Treatment in older adults may need to be individualized based on comorbidities.

Recommendations from the NAEPP-EPR-2 (1997) relevant to older adults that are incorporated into current asthma guidelines include:

- Review of patient technique in using inhaler medications/devices is essential; functional, visual, or cognitive impairments may require more intense and prolonged instruction for safe and efficacious use.
- A comprehensive medical history is essential to safely integrate asthma medications without aggravating pre-existing medical conditions such as osteoporosis, glaucoma, or cardiac problems. Conversely, medications such as beta blockers or aspirin prescribed for a pre-existing condition may need to be reevaluated if they are exacerbating the asthma.
- COPD may coexist with asthma; a trial of systemic corticosteroids will establish reversibility and benefit.

Initial assessment of severity is optimized when the patient has not been started on long-term controller therapy. Asthma is classified as intermittent or persistent; persistent is further classified as mild, moderate, or severe based on certain parameters (NAEPP-EPR-3, 2007) (see Figure 8-1).

Once the severity is established, treatment goals at all levels aim to control asthma and evaluate responsiveness to the plan of care (Figure 8-2) using a stepwise approach to management (Figure 8-3).

Components of Control		Classification of Asthma Control ≥12 years of age		
		Well Controlled	**Not Well Controlled**	**Very Poorly Controlled**
Impairment	Symptoms	≤2 days/week	>2 days/week	Throughout the day
	Nighttime awakenings	≤2x/month	1–3x/week	≥4x/week
	Interference with normal activity	None	Some limitation	Extremely limited
	Short-acting beta$_2$-agonist use for symptom control (not prevention of EIB)	≤2 days/week	>2 days/week	Several times per day
	FEV_1 or peak flow	>80% predicted/ personal best	60%–80% predicted/ personal best	<60% predicted/ personal best
	Validated questionnaires ATAQ ACQ ACT	 0 ≤0.75* ≥20	 1–2 ≥1.5 16–19	 3–4 N/A ≤15
Risk	**Exacerbations requiring oral systemic corticosteroids**	**0–1/year**	**≥2/year**	
		Consider severity and interval since last exacerbation.		
	Progressive loss of lung function	Evaluation requires long-term follow-up care		
	Treatment-related adverse effects	Medication side effects can vary in intensity from none to very troublesome and worrisome. The level of intensity does not correlate to specific levels of control but should be considered in the overall assesment of risk.		
Recommended Action for Treatment		• Maintain current step. • Regular follow-ups every 1–6 months to maintain control. • Consider step down if well controlled for at least 3 months.	• Step up 1 step. • Reevaluate in 2–6 weeks. • For side effects, consider alternative treatment options.	• Consider short course of oral systemic corticosteroids. • Step up 1–2 steps, and • Reevaluate in 2 weeks. • For side effects, consider alternative treatment options.

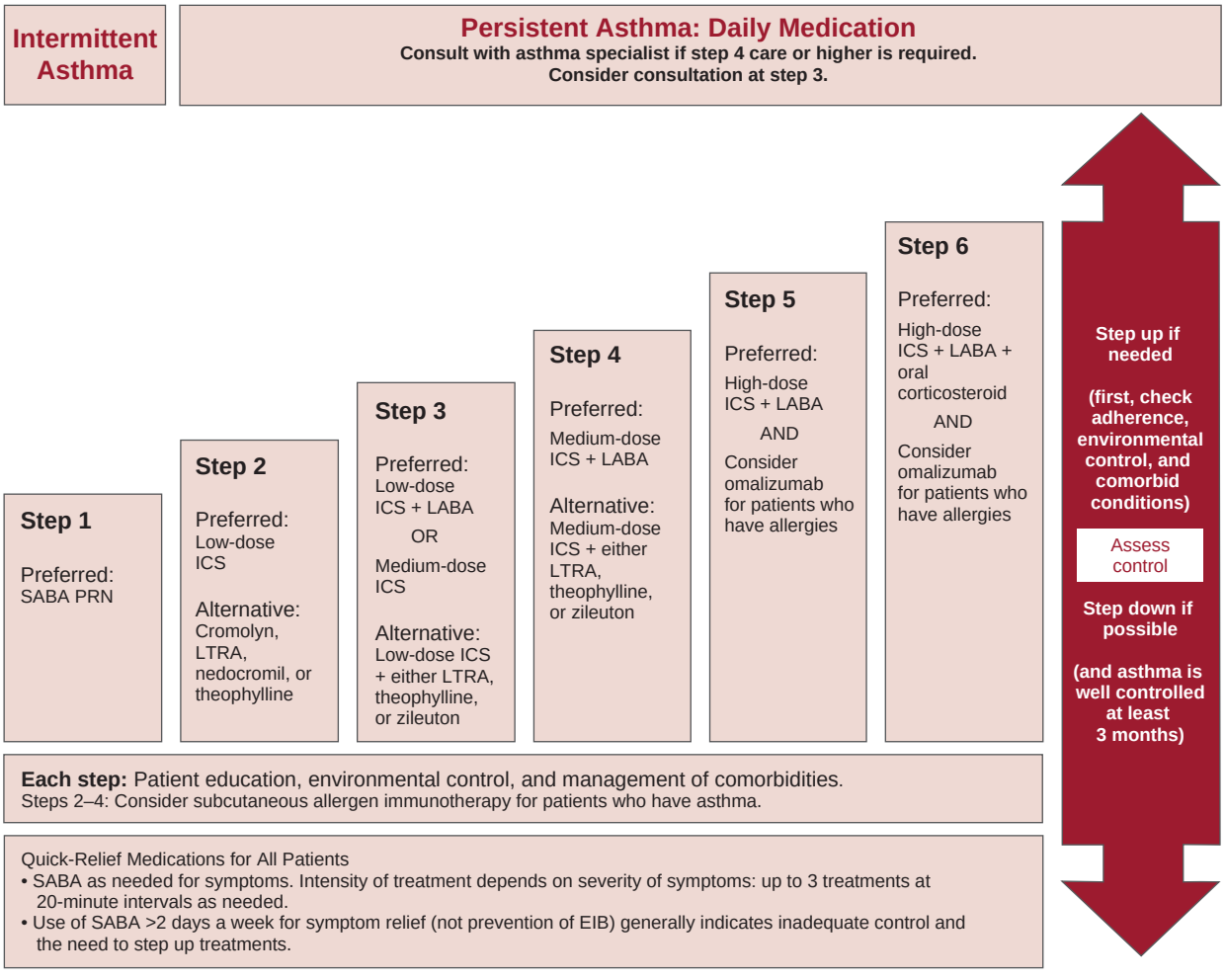
*ACQ values of 0.76–1.4 are indeterminate regarding well-controlled asthma.
Key: EIB, exercise-induced bronchospasm; ICU, intensive care unit

FIGURE 8-2. Assessing asthma control and adjusting therapy in youths ≥12 years of age and adults. EIB, exercise-induced bronchospasm. *(National Asthma Education and Prevention Program [2007, August]. Third Expert Panel on the Diagnosis and Management of Asthma. Expert Panel Report 3: Guidelines for the diagnosis and management of asthma.* Section 4*: Stepwise approach for managing asthma in youths ≥12 years of age and adults. Bethesda, MD: National Heart, Lung, and Blood Institute. Available from* www.ncbi.nlm.nih.gov/books/NBK7222.*)*

Asthma is a chronic health problem, just as hypertension and diabetes are. Regular chronic maintenance visits are required for optimal management. Depending on level of control, patients should be seen every 1 to 6 months for reevaluation. An interval symptom history is highly important in ongoing monitoring of control and adjustment of stepped treatment. An increase in symptoms indicating that asthma is not well controlled necessitates stepped up management and more frequent monitoring.

All patients who are able to read and comprehend should have an asthma action plan that is updated whenever changes in treatment are made (Apter, 2015). A printable asthma action plan including information on controlling environmental triggers is available at www.nhlbi.nih.gov/health/public/lung/asthma/asthma_actplan.pdf. The plan may need to be adapted to older adults who cannot or will not perform peak expiratory flow rate (PEFR) monitoring. Although one study in 2006 found no additional benefit of PEFR monitoring in terms of health-care utilization, lung function, or quality of life (Buist, Vollmer, Wilson, Frazier, & Hayward, 2006), it is recommended that patients with moderate to severe asthma use a diary every day to record the PEFR and their symptoms (Bailey & Gerald, 2016). Pharmacotherapy for asthma follows the guidelines in the NAEPP-EPR-3 (2007) outlined in Figure 8.3.

Patients who experience symptoms less than twice per month may be managed with a short-acting beta agonist (SABA) such as albuterol used as required for symptoms. Levalbuterol is an alternative with fewer side effects than albuterol. Patients are instructed to contact their primary care provider if symptoms increase, necessitating more frequent use of the SABA, or if their use of the medication does not alleviate symptoms. Prior to a step-up in treatment, inhaler adherence and technique must be assessed.



Alphabetical order is used when more than one treatment option is listed within either preferred or alternative therapy.
EIB, exercise-induced bronchospasm; ICS, inhaled corticosteroid; LABA, long-acting inhaled beta$_2$-agonist; LTRA, leukotriene receptor antagonist; SABA, inhaled short-acting beta$_2$-agonist

FIGURE 8-3. Stepwise approach for managing asthma in youths ≥12 years of age and adults. EIB, exercise-induced bronchospasm; ICS, inhaled corticosteroid; LABA, long-acting inhaled beta-2 agonist; LTRA, leukotriene receptor antagonist; SABA, inhaled short-acting beta-2 agonist. *(National Asthma Education and Prevention Program [2007, August]. Third Expert Panel on the Diagnosis and Management of Asthma. Expert Panel Report 3: Guidelines for the diagnosis and management of asthma.* Section 4: *Stepwise approach for managing asthma in youths ≥12 years of age and adults. Bethesda, MD: National Heart, Lung, and Blood Institute. Available from* www.ncbi.nlm.nih.gov/books/NBK7222.*)*

For persistent asthma, initiate stepwise therapy with the goal of well-controlled asthma. For patients with mild persistent asthma by severity classification, Step 2 therapy with a low-dose inhaled corticosteroid (ICS) such as fluticasone should be initiated. The patient should be followed up in 2 to 6 weeks to evaluate level of control and modify treatment as indicated.

Patients with moderate persistent asthma should be started on Step 3 therapy with either a medium-dose ICS or a combined low-dose ICS and long-acting beta agonist (LABA) such as fluticasone/salmeterol, budesonide/formoterol, or momentasone/formoterol. Follow up at 2 to 6 weeks for evaluation of response and adjustments to therapy. Referral should be considered for patients requiring Step 3 or higher therapy.

Patients with severe persistent asthma should be started on Step 4 treatment with a medium-dose ICS and LABA combination and reassessed at 2 to 6 weeks for response and level of control.

For Steps 2 through 4 alternative treatments include a leukotriene modifier antagonist (LTRA) such as montelukast, either alone (Step 2) or in combination with an ICS (Steps 3 through 4). A recent Cochrane review (Chauhan & Ducharme, 2014) found that LTRAs were safe but not as effective as monotherapy ICS for asthma. Research continues into the role of leukotrienes in asthma (Singh, Tandon, Dastidar, & Ray, 2013). Theophylline is also listed as an alternative treatment in Steps 2 through 4. Vaz Fragoso (2016) discourages the use of this medication in older adults due to toxicity and adverse effects. Other considerations in older adults with comorbidities include individualizing therapy; for example, for a patient with heart disease, increasing the dose of ICS and avoiding a LABA is prudent; for a patient with glaucoma or osteopenia, adding a LABA or LTRA instead of increasing

the dose of ICS is preferable (Saag, Furst, & Barnes, 2016; Vaz Fragoso, 2016). In short, guidelines are not meant to be absolute and do not preclude the use of clinical judgment.

Patients with severe persistent asthma should be referred for add-on treatment. Treatment at Step 5 is usually with a high-dose ICS/LABA and possible addition of omalizumab for patients with an allergic component. Studies have demonstrated that omalizumab, a humanized monoclonal antibody, is safe and effective for difficult-to-control asthma in older adults who meet allergic criteria. Treatment with omalizumab should be discontinued if the patient has not responded to the therapy within 4 months of initiation (Chang et al., 2014). Practitioners must be prepared to treat anaphylaxis in the unlikely event that it occurs.

Step 6 is reserved for patients who are not well controlled at Step 5. Therapy includes high-dose ICS and LABA and oral corticosteroid. Omalizumab also should be considered for those with an allergic component. Close follow-up of these patients is essential to avoid further morbidity and even mortality due to asthma. Oral corticosteroid therapy should be limited to short "bursts" to avoid drug-related events.

Asthma exacerbations in older adults frequently result in emergency department visits or hospital admissions. Aggressive treatment with SABAs and systemic (IV or oral) glucocorticoids is the standard of care, along with frequent spirometric assessment (Fanta, 2016.). Patients who do not respond well within 4 to 6 hours should be hospitalized (Fanta, 2015). It is critical to encourage patients to adhere to prescribed medication regimens. Evidence indicates that only 30% to 40% of patients currently take their asthma medications as prescribed (Sumino & Cabana, 2013).

Stepping down therapy is a gradual process compared to stepping up treatment. If a patient has been stable and well controlled for 3 months or more, it is generally safe to decrease the dose of ICS by 25% to 50% (GINA, 2016; Hagen, 2014).

Follow-Up: Regular monitoring should be done 1 to 3 months after initiation of treatment, and then every 3 to 12 months, depending on the level of control and severity. Patients should be seen within 1 week after an asthma exacerbation, and within 2 to 6 weeks when changes are made in stepwise management (Fanta, 2016; GINA, 2016; Khalid, 2015). Treatment step-down can be considered if the patient has had good control for 3 months; follow-up should be scheduled every 1 to 3 months after treatment step-down has been initiated (GINA, 2016).

Sequelae: Asthma in older adults is associated with increased bronchial hyperresponsiveness and a more rapid decline in lung function than in older adults without asthma (Hanania & Busse, 2016; Vaz Fragoso, 2016). Older adults also have increased treatment-related adverse events. Comorbidities such as COPD, cardiovascular disease, osteoporosis, glaucoma, diabetes mellitus, and other health problems complicate management and may limit treatment options. Older adults have the highest rate of asthma-associated mortality.

Prevention/Prophylaxis: There is no known strategy to prevent asthma. Patients with asthma should be given an influenza vaccine annually. Immunization with the pneumococcal pneumonia vaccine is also advised if the patient has not already been immunized. Partnership with the patient/family in education to recognize triggers, identify changes in status, and seek prompt medical assistance to avoid complications is key to success. Avoidance of environmental factors is also essential.

Referral: According to the NAEPP-EPR-3 (2007) report, consultation with an asthma specialist is advised at Step 4, with a suggestion to consider a consultation at Step 3. Older adults who have multiple comorbidities, have been treated in the emergency department, or have been hospitalized due to an asthma exacerbation within the past year should also be managed in collaboration with an asthma specialist. Patients who require ongoing oral corticosteroids or more than two courses of oral corticosteroids in a year should be referred to an asthma specialist. Patients having any complications that affect asthma or those who require further testing should also be referred (Fanta, 2016; GINA, 2016; NAEPP-EPR-2, 1997); patients with characteristics of both asthma and COPD fall into this category (Fanta, 2016; GINA, 2016). Patients/families may also be referred to a community-based asthma education program, although many of these initiatives are more focused on childhood asthma. Referral to case management through local Area Agency on Aging services can be helpful if the patient has unmet care needs or financial problems with obtaining medications.

Education: Patient/family education is an important component of asthma management in older adults. In addition to educating patients on the disease and the need for frequent monitoring and immediate reporting of increased symptoms, a written asthma action plan should be initiated, detailing how to identify and deal with environmental triggers, medications (including dosage and frequency), and when to seek emergent care. Instruction in use and care of inhalers and spacers, if used, is also important. If the patient is on an ICS, instruct on the need for daily use and oral hygiene to prevent a fungal infection. Clarification of the action and role of quick relief inhaler versus daily controller therapy is essential for medication adherence. The role of patient as partner in asthma management should be emphasized (Peláez, Bacon, Lacoste, & Lavoie, 2016).

Risk factors predictive of uncontrolled asthma in adults are African American race, less education, living in poverty, a BMI of more than 30 kg/m^2, insurance status, history of gastroesophageal symptoms, prior exacerbations, and current tobacco use (Miller & Sawlani, 2013; Schatz et al., 2013). Gearing education to the health literacy level of the patient is critical to gaining patient understanding and partnership. Patients who are still smoking should receive brief motivational counseling at each visit and referral to a Quitline or community-based smoking cessation program when ready to quit. Referral to the local Area Agency on Aging for assistance with insurance issues may also be beneficial.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The hallmark of asthma is partial reversibility of airflow obstruction on spirometry following beta-2 agonist administration.	C	Kraft & Oppong, 2015
Short-acting beta-2 agonists (SABAs) are indicated only as needed rather than on a scheduled basis for all levels of asthma severity.	A	Kraft & Oppong, 2015
ICSs are the preferred controller medication for all but mild intermittent asthma.	A	Kraft & Oppong, 2015
Long-acting beta agonists (LABAs) are the preferred add-on agent due to lack of control, but they should never be used as monotherapy without ICSs.	A	Kraft & Oppong, 2015
It is important to educate patients on the proper technique for inhaled medications and trigger avoidance.	B	Kraft & Oppong, 2015
The best strategy for management and improved outcomes is early treatment of asthma exacerbations.	A	Humphrey, 2016
Initial treatment (of asthma exacerbation) should include oxygen for most patients, SABA for all patients, ipratropium bromide for severe exacerbations, and systemic corticosteroids for most patients.	A	Humphrey, 2016
Initial treatment response in the emergency department, rather than severity of attack presentation, is a better predictor of need for hospitalization.	C	Humphrey, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CARDIAC ARRHYTHMIAS

Signal Symptoms: No symptoms at times; feeling of heart "beating out of the chest."

Description: An arrhythmia is a disturbance of cardiac rhythm. Cardiac arrhythmias, which occur in either the presence or absence of underlying heart disease, may be life threatening or may be an incidental finding. Arrhythmias may be differentiated by type or mechanism.

Atrial fibrillation (AF) is the most common sustained rhythm disorder. AF consists of numerous fibrillatory P waves that vary in shape, size, and timing. The ventricular response is often chaotic and rapid if the atrioventricular (AV) conduction is intact. AF can be clinically classified as follows:

- Paroxysmal: less than 7 days self-terminating or with intervention; episodes may occur with varied frequency
- Persistent: greater than 7 days and requires intervention to terminate
- Long-standing persistent: continuous AF more than 12 months
- Permanent: used when the patient and clinician make a joint decision to stop further attempts to restore or maintain sinus rhythm
- Nonvalvular: in the absence of rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve or mitral valve repair (January et al., 2014)

Other prevalent forms of arrhythmias in a geriatric population are:

Sick Sinus Syndrome: 1) Pathological bradyarrhythmia with an alternating supraventricular tachyarrhythmia (bradycardia-tachycardia syndrome); 2) supraventricular tachyarrhythmia—three primary categories.

Arrhythmias Primarily of Atrial Origin: Atrial premature beats, ectopic atrial rhythms, multifocal atrial tachycardia, atrial flutter, and AF.

Arrhythmias Arising Primarily Within the AV Node: AV nodal reentrant tachycardia, junctional premature beats, and nonparoxysmal junctional tachycardia.

Arrhythmias Partially Supraventricular in Origin: Pre-excitation syndromes.

Ventricular Arrhythmias: Ventricular ectopic beats are the most common variety.

Etiology: Most arrhythmias are thought to be caused by abnormalities either in impulse formation (disordered automaticity) or in impulse conduction (allowing reentry), or by a combination of the two. Increased automaticity is an accentuation of the inherent ability of many cardiac tissues to generate an independent rhythm. Reentry consists of a wave of excitation repeatedly circulating around a fixed anatomical obstacle. Triggered arrhythmias are caused by altered cellular depolarization.

Occurrence: Studies show that asymptomatic older patients with no known structural heart disease have an incidence of up to 40% of sinus arrhythmia, supraventricular or ventricular premature beats, or supraventricular tachycardia.

Age: The prevalence of ventricular arrhythmia increases with age, occurring in 80% of healthy older adults ages 60 to 85 years. AF is diagnosed in 4.8% of women and 6.2% of men over age 65 years at baseline examination, and its prevalence is strongly associated with advanced age, particularly in women. Although no specific data are available, the incidence of junctional arrhythmias and tachycardias related to the accessory pathway may be lower in older adult individuals because of an age-related reduction in accessory pathway conduction.

Gender: Arrhythmias occur slightly more often in men than in women (see also section on Age).

Ethnicity: Not significant.

Contributing Factors: Pre-existing heart disease is a contributing factor to arrhythmias, with structural disease becoming more prevalent with age. The muscular response and contractility decreases as the patient ages. The patient experiences a reduction in cardiac function and contractility. This reduction is responsible for a decreased stroke volume and ejection fraction. The average older adult does not experience adverse symptoms with normal everyday activities. However, stressors (whether they are physical, psychological, environmental, or social) can increase the risk of illness in the patient. These illnesses are taxing to the patient's system, and the increased energy demands place an increased demand on the cardiac system. The aging process can also have an adverse effect on the cardiac valves. The valves tend to become thick and stiff secondary to arteriosclerosis and atherosclerotic plaques. Fibrosis or calcification in the vicinity of the AV node causes conduction disturbances. Atrial arrhythmias may be caused by a mechanical obstruction to atrial emptying with subsequent left atrial dilation, myocardial ischemia, and increased sympathetic activity.

An age-related factor associated with tachyarrhythmias is increased left atrial size. This enlargement may contribute to the increase in supraventricular ectopy. Increases in ventricular ectopic beats may be related to left ventricular enlargement. The overload of ionized calcium in the older myocardium may contribute to ectopy. Age-related factors associated with bradyarrhythmias include an age-related decline in the number of pacemaker cells and the presence of fat deposits around the SA node. His bundle cells are replaced with fibrous tissue, and adipose tissue and amyloid are deposited; this is also associated with conduction disturbance.

Systemic diseases (i.e., thyrotoxicosis, infection, hypoxemia, hypercapnea) can cause circulatory disturbances that may provoke an arrhythmia. Drugs that can cause an arrhythmia include digitalis and other antiarrhythmics, aminophylline, and alcohol. Electrolyte disturbances, particularly hyperkalemia, hypokalemia, hypercalcemia, and hypocalcemia, can precipitate ectopic beats. Family history of sudden cardiac death may indicate a predisposing factor for rhythm disorder, such as hypertrophic obstructive cardiomyopathy, congenital prolonged QT syndrome, or the presence of an aberrant conduction pathway.

Signs and Symptoms: The history, physical examination, and electrocardiogram (EKG) studies represent the cornerstone to evaluation of arrhythmias. Arrhythmias may cause symptoms due to a reduced blood flow or inadequate cardiac pump function. In the history, the patient may describe sensations that accompany abnormal cardiac rhythm such as pounding, racing, or skipped beats. Older adults are less likely to complain of palpitations and more likely to present with manifestations of heart failure or hypoperfusion (i.e., impaired mental function, dizziness, syncope). There appears to be a peak in syncopal episodes after age 65 years in both men and women. The Framingham study showed an increased incidence of syncope after age 70 years, from 5.7 events per 1,000 person-years in men ages 60 to 69 years, to 11.1 events in men ages 70 to 79 years (Moya et al., 2009). Along with the history of present illness, previous diagnosis and treatment for arrhythmia and cardiac disease should be elicited.

In the physical examination, concentrate on the cardiac and peripheral vascular systems. Check the pulses for 1 full minute to determine rate and regularity (Bickley & Szilagyi, 2013; Goolsby & Grubbs, 2015). Assess normal and extra heart sounds. S_1 intensity may provide information about the relation of atrial to ventricular contraction. The longer the PR interval, the softer the S_1. Note intermittent extra heart sounds (S_3 and S_4). The jugular vein must be assessed. In AV dissociation (when the atria and ventricles contract independently), giant A waves (cannon waves) may be observed. Provoking maneuvers should not be attempted by the advanced practice nurse but may be attempted by a cardiologist. These maneuvers include carotid massage (for atrial arrhythmias only), mild exercise, psychological stress, pharmacological stress, and electrical programmed stimulation (EPS).

Other indicators of hemodynamic response to an arrhythmia include level of consciousness and skin temperature and color. The history should guide the remainder of the episodic examination because it is also helpful in identifying the contributing factors that may precipitate or aggravate an arrhythmia.

Diagnostic Tests:

TEST	RESULTS INDICATING DISORDER
Rhythm strip	Rhythm disturbance may be present, or if transient, it may be absent
12-lead EKG	Acute MI or ischemia, prolonged QT intervals, and pre-excitation may be identified as precipitators of an arrhythmia

Continued

TEST	RESULTS INDICATING DISORDER
Ambulatory EKG monitoring	Quantifies arrhythmias with reference to symptoms Identifies patients at risk for sudden death by documenting ventricular arrhythmias and heart rate variability
Implantable loop recorder	In infrequent syncope this long-term device (14 months) may identify the precipitant
Echocardiogram	Identifies structural, functional, and hemodynamic abnormalities in the cardiovascular system
Cardiac electrophysiological study	Evaluates specific arrhythmias to distinguish these, determine location and characteristics, and guide therapy
Implantable cardioverter-defibrillator (ICD)	ICD firings may be due to recurrent arrhythmia
Electrolyte panel	Identify electrolyte disturbance (i.e., hypokalemia, hyperkalemia, hypomagnesemia, hypocalcemia) that may be causative or contributory
Coronary and LV angiography	Identifies CAD and LV function
Cardiac MRI	Especially useful in recognizing arrythmogenic RV dysplasia, or infiltrative diseases

Diagnostic testing includes a 12-lead EKG done with a rhythm strip, lasting at least 2 to 3 minutes. Include exercise testing for those with a clinical history suggesting exercise-induced arrhythmia. Ventricular arrhythmias can occur in individuals with or without cardiac disorders. The standard resting 12-lead EKG is indicated in all patients who are evaluated for ventricular arrhythmias (Domino, 2017; Pedersen et al., 2014). An ambulatory 24-hour EKG (Holter monitoring) helps to quantify arrhythmias with reference to symptoms. Ambulatory EKG is indicated when there is a need to clarify the diagnosis by detecting arrhythmias, QT interval changes, T-wave alternans, or ST changes; to evaluate risk; or to judge therapy (Pedersen et al., 2014). Event recorders are best suited for documenting less-frequent but more-prolonged bouts of arrhythmias, although not used with as much frequency. Patient-activated loop memory devices, which record the EKG before the symptomatic event, are most useful. His bundle electrograms involve the insertion of a transvenous electrode catheter into the right ventricle, to record depolarizations and the intervals. This method is most useful for distinguishing AV block from an ectopic focus.

Echocardiography is recommended in patients with ventricular arrhythmias who are suspected of having structural heart disease (Domino, 2017; Pedersen et al., 2014). An echocardiogram is used more commonly than magnetic resonance imaging (MRI) or cardiac computed tomography (CT) scan because it is inexpensive in comparison. The echocardiogram is a useful tool in assessing for valvular disorders, left ventricular function and wall motion, and the ejection fraction. The electrophysiological (EP) testing is performed using intracardiac recordings, electrical stimulation, and drugs to assess and document ventricular tachycardia, guide ablations, and evaluate loss of consciousness in patients with arrhythmias as the suspected cause. EP testing is recommended for diagnostic evaluation of patients with remote MI with symptoms suggestive of ventricular tachyarrhythmias, including palpitations, presyncope, and syncope (Domino, 2017; Pedersen et al., 2014). EP testing is recommended in patients with syncope of unknown cause with impaired left ventricular function or structural heart disease (Domino, 2017; Pedersen et al., 2014).

In EPS, multipolar catheter electrodes are introduced into the venous or atrial circulation and advanced to various intracardiac positions to monitor the electrical activity or to induce an arrhythmia. EPS is most useful for determining sinus node dysfunction, AV block, intraventricular conduction disturbances, pre-excitation syndromes, supraventricular tachycardia, ventricular tachycardia, and unexplained syncope or palpitations. It is used also to monitor cardiac activity in survivors of sudden cardiac death.

Differential Diagnosis: Clarify the diagnosis with precision. Note reversible and precipitating causes of an arrhythmia and coexisting diseases.

Treatment: The incidence of asymptomatic arrhythmias of questionable clinical significance is high. Arrhythmias are never treated in isolation; some are benign and some are lethal. Patients with hemodynamic compromise resulting from arrhythmia need to be hospitalized as soon as possible. Prehospital care for threatening hemodynamic instability secondary to tachyarrhythmia may include direct current cardioversion, and the nurse practitioner should be skilled in this intervention. After thorough investigation, the goals of arrhythmia treatment include the alleviation of bothersome symptoms, the prevention of complications from sustained arrhythmias, and the avoidance of sudden death associated with certain arrhythmias.

The risk of treatment is considerable. Aggressive therapeutic treatment is indicated when patients are symptomatic and the urgency of therapy depends on the associated hemodynamic disturbance. A comprehensive treatment plan must address the three cornerstones of AF management: rate control, rhythm control, and prevention of thromboembolism. Goals of the therapy should be to obtain symptom control, stroke prevention, and a reduction in hospital recidivism (January et al., 2014). Three major factors determine how well a patient tolerates an arrhythmia: heart rate, duration of the arrhythmia, and presence and severity of associated underlying heart disease. Arrhythmias often cannot be controlled unless underlying cardiac problems are discovered and treated. No antiarrhythmic "wonder drug" exists, and the bothersome and potentially dangerous side effects necessitate determination of clear indications and use of the utmost caution. Pharmaceutical agent selection is based on the electrophysiology of the rhythm disturbance, the mechanism of action, and the side effects of the drug. Several new drugs are useful as an alternative to warfarin for the prevention of stroke and systemic thromboembolism in patients with paroxysmal to permanent AF and risk factors for stroke or systemic embolization who do not have a prosthetic heart valve or hemodynamically significant valve disease, severe renal failure, or advanced liver disease (January et al., 2014). Conditions common in older adults that can affect the choice, dosing, efficacy, and safety of antiarrhythmic therapy include decreased hepatic or renal function, decreased serum albumin levels, and electrolyte abnormalities.

The broad categories of arrhythmia treatment include medications, pacemakers, antitachycardia devices, implantable automatic cardioverter-defibrillators, catheter ablative procedure, and specific maneuvers. Defibrillation is the required intervention in ventricular fibrillation or pulseless ventricular tachycardia and is the most important determinant of survival in cardiac arrest. Patients defibrillated within 2 minutes have a 60% hospital discharge survival rate (Hansen et al., 2014). Vagal maneuvers (carotid massage or the Valsalva maneuver) may terminate or slow AV nodal reentry or AV reentry types. These maneuvers are associated with a high risk of emboli dislodgment. The carotid arteries should be assessed for bruit before administering massage. Because of the high mortality rate associated with antiarrhythmic surgery, it is not recommended in older adult individuals. The specifics of treatment are not detailed in this text, because all assessment and management must be done in close collaboration with a physician specialist; much of the treatment is initiated by the specialist in the hospital setting.

Follow-Up: Because of their low therapeutic ratio, drug dosing and plasma concentrations of the antiarrhythmics are based on therapeutic monitoring. Side effects are significant and include the negative inotropic effect, which can precipitate heart failure or proarrhythmia in the presence of structural heart disease. Extracardiac side effects can also be significant, including anticholinergic effects, gastrointestinal effects, and neurological toxicity with some antiarrhythmic agents. Monitor patients after pacemaker insertion with regular follow-up appointments and EKG testing, specifically looking for pacemaker failure, infection, thromboembolism, perforation or dislodgment, and complicating arrhythmias.

Sequelae: Regardless of age, the nature and severity of underlying heart disease are of much greater prognostic significance than the arrhythmia alone. The following rhythm disturbances have been reported to carry a poor prognosis in patients with CAD: frequent ventricular premature contractions (VPCs) (greater than 10 per min), multiform VPCs, ventricular couplets, R-on-T phenomenon, and ventricular tachycardia.

Syncope can occur secondary to asystole. Syncope is an intermittent symptom that may be caused by an underlying arrhythmia with or without cardiac disease. Treatment goals are prevention of symptom recurrence, improvement of quality of life, and prolongation of survival. Permanent cardiac pacing may relieve the symptoms but may not affect the survival rate. Syncope recurs in approximately 20% of the patients (Moya et al., 2009; Saklani, Krahn, & Klein, 2013). Bradycardia can contribute to complete heart block and to the development of heart failure in patients with associated ventricular dysfunction. Tachycardia can precipitate angina and circulatory arrest in patients with CAD. Bradyarrhythmias or tachyarrhythmias can cause systemic embolism and stroke. AF can result in heart failure and a low cardiac output state. The risk of stroke related to AF increases with age. The Cardiac Arrhythmia Suppression Trial (CAST) demonstrated that patients treated for prognostically significant arrhythmias may have a higher mortality rate from sudden cardiac death if Class IC (flecainide and encainide) agents are used (Greenberg et al., 1995).

Prevention/Prophylaxis: All patients with AF should be considered for long-term low-intensity warfarin or the newer anticoagulant therapy. Aspirin, though less effective in preventing stroke, is an alternative for some patients in whom warfarin is contraindicated. Electrolyte imbalances should be monitored and metabolic disturbances treated. Patients receiving digitalis should be monitored for toxicity.

Referral: All patients with treatable arrhythmias require collaborative management. Clinically significant ventricular arrhythmias or any symptomatic arrhythmia is managed by the specialist in the coronary care unit. A primary care provider may handle many atrial arrhythmias and low-grade ventricular arrhythmias; however, a specialist should be consulted for all clinically significant ventricular arrhythmias, arrhythmias resistant to routine therapy, or arrhythmias whose clinical significance is in doubt.

Education: Carefully instruct all patients receiving antiarrhythmics about the therapeutic effects, side effects, and potentially adverse effects of these medications. Because patients with pacemakers are vulnerable to external electrical fields, they should be instructed to recognize this potential and avoid exposure. Patients with pacemakers must also be aware of signs and symptoms of pacemaker failure.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Oral anticoagulants are useful as an alternative to warfarin for the prevention of stroke and systemic thromboembolism in patients with paroxysmal to permanent AF and risk factors for stroke or systemic embolization who do not have a prosthetic heart valve or hemodynamically significant valve disease, severe renal failure, or advanced liver disease. Based on shared decision making.	B	January et al., 2014
A 1-minute delay in defibrillation is associated with a reduction in odds of survival of up to 21%.	C	Scottish Intercollegiate Guidelines Network, 2007

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Permanent cardiac pacing may relieve the symptoms but may not affect the survival rate. Syncope recurs in approximately 20% of the patients.	C	Moya et al., 2009
EP testing is recommended in patients with syncope of unknown cause with impaired left ventricular function or structural heart disease.	C	Pedersen et al., 2014
EP testing is recommended for diagnostic evaluation of patients with remote MI with symptoms suggestive of ventricular tachyarrhythmias, including palpitations, presyncope, and syncope.	C	Pedersen et al., 2014
Echocardiography is recommended in patients with ventricular arrhythmias who are suspected of having structural heart disease.	B	Pedersen et al., 2014
Ambulatory EKG is indicated when there is a need to clarify the diagnosis by detecting arrhythmias, QT-interval changes, T-wave alternans, or ST changes, to evaluate risk.	C	Pedersen et al., 2014
The standard resting 12-lead EKG is indicated in all patients who are evaluated for ventricular arrhythmias.	B	Pedersen et al., 2014

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Signal Symptoms: Dyspnea, chronic cough with or without sputum production, decreased activity tolerance, wheezing.

Description: COPD is defined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) as "a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases" (GOLD, 2017, p. 2). Cardinal symptoms of COPD include dyspnea, chronic cough, and/or sputum production (GOLD 2017; Han, Dransfield, & Martinez, 2016).

A characteristic of COPD is chronic airflow limitation resulting from a parenchymal destruction (emphysema) and small airways disease (e.g., obstructive bronchiolitis) that vary among individuals and evolve at different intervals over time (GOLD, 2017). Chronic inflammation is responsible for structural changes, narrowed passages of the small airways, and lung parenchyma destruction, leading to loss of alveolar attachments to the small airways and a reduction in lung elastic recoil (GOLD, 2017). The ability of the airways to remain open is diminished during expiration as a result. Characteristics of COPD are airflow limitation and mucociliary dysfunction, both of which are likely attributed to loss of small airways (GOLD, 2017).

The most significant risk factor for COPD is cigarette smoking (CDC, 2016, Sept. 16; GOLD, 2017). Long-term cumulative exposure to noxious particles and gases, in combination with various other host factors such as airway hyperresponsiveness, inadequate lung growth and development during childhood, genetics, occupational/industrial gases or fumes, and indoor/outdoor air pollution are all contributing risk factors for COPD (GOLD, 2017; GOLD, 2017a; Liu, Li, Ding, Wang, & Wen, 2016; World Health Organization [WHO], 2017). Long-term asthma has been attributed to some cases of COPD and advancing age is a factor with symptoms presenting often after the ages of 40 to 50 years old (WHO, 2017). Patients with symptoms and/or exposure to risk factors for COPD should be considered (GOLD, 2017). The presence of persistent airflow limitation with lung function measurements (spirometry) is required to confirm the diagnosis of COPD (GOLD, 2017).

Etiology: The most significant etiological factor in the development of COPD and the progression of the disease is cigarette smoking (CDC, 2016, Sept. 16; GOLD, 2017). However,

individuals who are non-tobacco users may also develop airflow limitations and COPD (GOLD, 2017). Long-term asthma has been attributed to some cases of COPD and advancing age is a factor with symptoms presenting often after the ages of 40 to 50 years old (WHO, 2017).

Occurrence: COPD is a chronic disease with substantial morbidity and mortality worldwide, lending to social and economic burdens, long-term disability, and premature death (American Lung Association [ALA], 2017; Criner et al., 2015; GOLD, 2017). COPD is the third leading cause of death in the United States, with over 3 million or 5% of all deaths globally attributed to COPD (CDC, 2016, Sept. 16; GOLD, 2017). An approximate 15.7 million Americans or 6.4% have been diagnosed with COPD (CDC, 2016, Sept. 16). Over one-half of adults with abnormally low pulmonary function tests were unaware they had COPD, thus the prevalence of COPD may be underrepresented (CDC, 2016, Sept. 16; Criner et al., 2015).

Age: Individuals aged 65 to 74 years and age 75 years and older have the highest incidence of COPD (CDC, 2016, Sept. 16). COPD development occurs gradually over time and most commonly becomes apparent after 40 to 50 years of age (WHO, 2017). Advancing age is a risk factor for COPD, although the evidence is unclear if healthy aging leads to COPD or if it is a result of the cumulative sum of exposures throughout life (GOLD, 2017).

Gender: Anatomical and physiological differences between the male and female gender influence the course of respiratory disease and treatment response (Pinkerton et al., 2015).

Women have an increased prevalence of COPD (CDC, 2016, Sept. 16; GOLD, 2017), although some studies have suggested women are more susceptible to the detrimental effects of smoking than men, leading to greater severity of disease and burden of small airway disease despite a similar history of tobacco exposure (GOLD, 2017; Jenkins, 2017; Pinkerton et al., 2015). Evidence reveals that women who have COPD and have fewer pack-years of smoking than men have demonstrated an early-onset of COPD, experienced greater dyspnea, and progress to severe COPD more rapidly than men (GOLD, 2017; Jenkins, 2017). When compared to men, women with comparable pack-year history smoking have shown more loss of lung function, airflow obstruction, and exercise intolerance than men (GOLD, 2017; Jenkins, 2017). The differences found in women are presumably a result of the manner in which tobacco smoke is metabolized in women differently than men, rendering more higher and more prolonged toxic exposure to tobacco smoke (Pinkerton et al., 2015).

Ethnicity: Native Americans/Alaska Natives and multiracial non-Hispanic individuals have been found to have the greatest prevalence of COPD (CDC, 2016, Sept. 16).

Contributing Factors: Cigarette smoking is the most important risk factor for COPD (CDC, 2017; GOLD, 2017). Inhaling other pollutants and exposure to particles (e.g., second-hand smoke, pipe, water pipe, or cigar use), environmental and occupational exposures (e.g., biomass fuel exposure, chemicals, dust, fumes), and genetic determinants (e.g., alpha-1 antitrypsin deficiency) are other causative factors in COPD (GOLD, 2017; Liu et al., 2016; WHO, 2017). Chronic bronchitis and frequent respiratory infections have been associated with a greater risk of COPD (GOLD, 2017).

Signs and Symptoms: COPD is a complex, chronic disease and therefore requires a comprehensive assessment of symptoms. Symptoms may include dyspnea, chronic cough with or without sputum production, recurrent lower respiratory infections, wheezing, chest tightness, fatigue, weight loss, and/or anorexia (GOLD, 2017; GOLD, 2017a). Signs may include increased anteroposterior diameter of the thorax, use of accessory muscles for respiration, prolonged expiration, hyperresonance on percussion, decreased heart and breath sounds, tachypnea, neck vein distention during expiration in absence of heart failure, ruddy or cyanotic skin color, and clubbing of nail beds (Bickley, 2017). Nocturnal and seasonal exacerbation of symptoms are common. Weight loss, fatigue, and anorexia pulmonary cachexia are commonly associated with severe to very severe COPD (GOLD, 2017). Symptoms of anxiety or depression may be present when assessed and are attributed to an increased risk of COPD exacerbations and less than optimal health (GOLD, 2017).

"Chronic and progressive dyspnea is the most characteristic symptom of COPD" (GOLD, 2017, p. 25). Dyspnea is considered a cardinal symptom described by COPD patients as breathlessness, air hunger, chest heaviness, or gasping for breath (GOLD, 2017). Chronic cough is commonly an initial symptom of COPD, and in about 30% of patients cough with tenacious sputum production is present (GOLD, 2017). Because the COPD patient usually has several manifestations of COPD, symptoms often vary.

A thorough history to include pattern of symptom development, past medical history, family history, exposure to risk factors, history of exacerbations, and/or hospitalizations for respiratory conditions aid in establishing the diagnosis of COPD (GOLD, 2017). The impact of symptoms such as activity limitations, socioeconomic impact (e.g., missed work days) and comorbid conditions (e.g., cardiovascular heart disease, heart failure, osteoporosis) are supportive in developing the diagnosis of COPD.

The severity of symptoms and airflow limitation associated with COPD are contributing factors impacting quality of life and ADLs. Limitations of functional activities vary daily, such as physical exertion, daily function, lifestyle management, social activities, household chores, sleep, and work performance (GOLD, 2017).

Physical examination is not typically diagnostic in early COPD and may be normal (GOLD, 2017). This does not preclude a diagnosis of COPD and can aid in establishing a baseline and identifying comorbidities (GOLD, 2017). In the early to middle stages, chest auscultation may reveal longer expirations, with wheezing on forced expiration. Wheezes can sometimes be heard (most frequently with chronic bronchitis); the presence of crackles suggests possible pulmonary edema or pneumonia.

As the severity of the disease progresses, signs of airway limitations are more apparent on physical examination (GOLD, 2017). Physical examination with advancing disease may reveal decreased breath sounds, hyperinflation (e.g., resonance to percussion), use of accessory muscles of respiration, wheezes, crackles in the lung bases, pursed lip breathing, and/or distant heart sounds (Bickley, 2017; Han et al., 2017). Hyperinflation restricts diaphragmatic movement and

TABLE 8-1 Global Initiative for Chronic Obstructive Lung Disease: Post-Bronchodilator Fev_1 Classification of Severity of Airflow Limitation

Patient Criteria: FEV_1/FVC <0.70

FEV_1 ≥80% predicted	GOLD 1: Mild
50% ≤FEV_1 <80% predicted	GOLD 2: Moderate
30% ≤FEV_1 <50% predicted	GOLD 3: Severe
FEV_1 <30% predicted	GOLD 4: Very Severe

Adapted from *GOLD Pocket Guide to COPD Diagnosis, Management, and Prevention, A Guide for Health Care Professionals*, 2017.

the chest takes on a barrel-like appearance (Bickley, 2017). As the cardiovascular system attempts to compensate, tachycardia or a gallop rhythm commonly occurs. Right-sided heart failure and cor pulmonale seen in the later stages may present as jugular venous distension, hepatomegaly, or peripheral edema (Bickley, 2017). Patients frequently present for treatment due to health-related issues, such as upper or lower respiratory infections, thus presenting an opportunity for the clinician to assess symptomatology of COPD.

Diagnostic Tests: Spirometry is the gold standard for measuring airflow limitation (GOLD, 2017; Guirguis-Blake, Senger, Webber, Mularski, & Whitlock, 2016). Airflow limitation that is not fully reversible is evident if post-bronchodilator FEV_1/FVC is less than 0.70 and FEV_1 is less than 80% predicted (refer to Table 8-1) (GOLD, 2017a). The U.S. Preventive Services Task Force (USPSTF) recommends spirometry only in symptomatic patients, as there was no direct evidence to support screening asymptomatic individuals (Guirguis-Blake et al., 2016). Symptoms for considering a diagnosis and performing spirometry include dyspnea, cough, chronic sputum production, recurrent lower respiratory tract infections, history of risk factors, family history of COPD, and/or childhood factors (GOLD, 2017a). Although the GOLD guidelines (2017) are focused on spirometry results, FEV_1 decreases with age and, therefore, the age-related change needs to be factored into interpretation of spirometry results (GOLD, 2017). Spirometry has good sensitivity, although a peak expiratory flow measurement alone is unreliable when used as the only diagnostic tool because of weak specificity (GOLD, 2017). Because there is a weak correlation between FEV_1, symptoms, and impairment impacting an individual's health status, a more formal assessment of symptoms is indicated.

The updated 2017 GOLD guidelines added important multidimensional recommendations regarding COPD management and treatment. In addition to results of spirometry, assessing the impact of COPD, the burden of disease, the risk of disease progression, and COPD exacerbation risk are integral components (GOLD, 2017; Karloh et al., 2016). Thirteen disease-specific instruments have been identified as COPD assessment tools (Karloh et al., 2016). The most comprehensive disease-specific questionnaires with reliability and validity focused on health status include the Chronic Respiratory Questionnaire (CRQ) and the St. George's Respiratory Questionnaire (SGRQ) (GOLD, 2017; Karloh et al., 2016). Both tools are comprehensive, but are lengthy, and the scoring systems are too complex to use for clinical settings (GOLD, 2017; Karloh et al., 2016). Brief and simple, yet valid and reliable assessment tools recommended for clinical practice include the COPD Assessment Test (CAT) and the COPD Control Questionnaire (The CCQ) (GOLD, 2017; Karloh et al., 2016). Of the two, the CAT is widely utilized, applicable worldwide, and useful to predict clinical correlation of disease-related impact (GOLD, 2017; Karloh et al., 2016).

Radiographic imaging with a chest x-ray alone is not diagnostic of COPD but can provide value in excluding differential diagnoses, detecting the presence of concomitant pulmonary disease (bronchiectasis, pleural diseases, pulmonary fibrosis), pneumonia, or cardiac diseases (e.g., cardiomegaly) (GOLD, 2017). Chest radiography in early COPD appears normal (Dunphy, Winland-Brown, Porter, & Thomas, 2015). Abnormalities associated with COPD may be present on a chest x-ray, such as lung hyperinflation, hyperlucency of the lungs, or tapering of vascular markings (GOLD, 2017). Structural lung disease may be seen on chest imaging (e.g., emphysema, gas trapping, or airway wall thickening) consistent with COPD findings. A chest x-ray in those having advanced COPD with emphysema may reveal hyperinflation, bullae or blebs, and a flat hemidiaphragm. A chest x-ray is not recommended for screening of lung cancer. The USPSTF (2017) recommendations for lung cancer screening include an annual screening with low-dose computed tomography (LDCT) in adults age 55 to 80 years with a 30 pack-year smoking history and who currently smoke or have smoked in the past 15 years.

Pulse oximetry is a noninvasive intervention to assess a patient's arterial oxygen saturation and need for supplementation oxygen therapy (GOLD, 2017). If pulse oximetry is less than 92%, arterial or capillary blood gases are recommended (GOLD, 2017).

Laboratory tests should include a complete blood count (CBC) with differential to rule out anemia, polycythemia (occurs in advanced COPD with hypoxemia), detecting the presence of infection consistent with an increased white blood cell (WBC) count, or if eosinophilia is present, considering an allergic or asthmatic component (Dunphy et al., 2015). Serum $alpha_1$-antitrypsin levels are recommended for individuals presenting with COPD at early ages (less than 45 years of age), in non-tobacco users with clinical emphysema, and individuals with a family history of COPD at a young onset (Dunphy et al., 2015).

An EKG is recommended as a baseline in patients with signs of advanced COPD or with concurrent cardiac disease. In COPD patients and older adults of any age, atrial arrthymias are common (Dunphy et al., 2015). Peaked P waves in leads II, III, and aVF are often apparent in patients with pulmonary disease (Dunphy et al., 2015). If concomitant cardiac involvement is severe, an EKG in advanced COPD may demonstrate evidence of right-axis deviation, sinus tachycardia, and pulmonary hypertension with the presence of ongoing S waves in the lateral precordial leads.

Differential Diagnosis: Carcinoma of the lung, asthma, interstitial lung disease, heart failure, pneumonia, bronchiectasis, TB, obliterative bronchiolitis, diffuse panbronchiolitis, chronic rhinitis, chronic sinusitis, obstructive sleep apnea, and/or PE.

Treatment: Individualize treatment goals according to the stage of disease, assessment of symptomatology, patient goals, comorbidities, and risk COPD exacerbation. Treatment is targeted toward improvement of health status and functional status, quality of life, prevention of disease progression, avoidance of exacerbations or complications, prevention of treatment side effects, and management of exacerbations (GOLD, 2017). The GOLD syllabus identifies four segments of a COPD management program: 1) diagnosis and assessment, 2) prevention and maintenance therapy, 3) management of stable COPD, and 4) management of COPD exacerbations. Because diagnosis and assessment has been addressed in the Assessment/Diagnostic Tools section, the remaining segments will be focused on prevention and management.

Prevention and Prophylaxis:

- COPD is almost 100% preventable by avoidance of smoking. Early detection of COPD may alter the course and progression (CDC, 2016).
- All COPD patients should have annual influenza vaccines unless allergic (CDC, 2017a). The pneumococcal polysaccharide vaccine (PPSV23) is recommended for all patients at age 65 years (CDC, 2017a). Prior to age 65, adults 19 to 64 years of age at high risk (e.g., cigarette smoking, chronic conditions, COPD) are recommended to have the PPSV23 (CDC, 2017a). For individuals who receive a PPSV23 prior to age 65 years, revaccination is recommended with PPSV23 at age 65 years (CDC, 2017a).

Risk Factor Reduction:

- Identification and reduction of exposure to risk factors is key in the prevention and treatment of COPD (GOLD, 2017).
- Smoking cessation is the most effective and cost-effective intervention and should be promoted at every visit (CDC, 2017; GOLD, 2017). Counseling (brief or intensive), educational, and community resources are helpful. Pharmacotherapy (e.g., nicotine replacement therapy, buproprion, varenicline, or combination therapy) consistently should be offered as a component of an intervention program (CDC, 2017; GOLD, 2017).
- Identify and reduce personal exposure to occupational or environmental risk factors (fumes, gases, dust, indoor/outdoor air pollutants).
- Vaccination for influenza and pneumococcal (PCV13 and PPSV23) are recommended unless contraindicated.
- Adequate hydration and nutrition are important.

Management of Stable COPD:

- Individualize assessment to reduce current symptoms and risk of exacerbations. Tailor plan for escalating/de-escalating treatment-based interventions dependent on symptomatology and COPD exacerbation risk.
- Establish a partnership facilitating patients to become active partners in their care. Educate patients on the nature of the disease process, chronicity, and risk factors for progression. The role of self-management and the health-care team is recognition of instability and when to seek medical assistance. Adherence to treatment regimen should be included in education provided.
- Discussion of healthy living, nutrition, and physical activity is essential.
- Pulmonary rehabilitation is recommended in patients with high symptom burden to reduce symptomatology and risk of exacerbations (GOLD, 2017; McCarthy et al., 2015). Pulmonary rehabilitation has been shown to be a highly effective therapeutic intervention to improve dyspnea, health status, and exercise intolerance (Criner et al., 2015; GOLD, 2017a).
- Long-term supplemental oxygen therapy in patients with severe resting hypoxemic COPD has been shown to improve survival (GOLD, 2017a). Additional benefits include reductions in nocturnal hypoxemia and arrhythmias, reduction in polycythemia, dyspnea, increased exercise tolerance, and improved cognitive status. Criteria for oxygen therapy include PaO_2 = 55 mm Hg or SaO_2 = 88% with or without hypercapnia confirmed twice over a 3-week period; or PaO_2 = 55 to 60 mm Hg or SaO_2 = 88% if there is evidence of pulmonary hypertension, polycythemia, or peripheral edema suggesting heart failure (GOLD, 2017).

Anxiety and depression are comorbidities associated with COPD and attributed to poor health outcomes (Panagioti, Scott, Blakemore, & Coventry, 2015). Assess for symptoms and provide psychological interventions as indicated. Holistic care can facilitate self-management and self-care. Pharmacological management is the cornerstone of COPD treatment and is directed at symptom reduction, reducing the risk and severity of COPD exacerbations, and improving exercise tolerance and health status (GOLD, 2017). The GOLD (2017) guidelines recommend initiation therapy along with subsequent escalation and/or de-escalation pharmacological management.

When dyspnea is unrelieved by medications and interferes with functioning and quality of life, consider evaluation for surgical options. Lung volume reduction therapy, lung resections, transplants, and laser bullectomy are available in selected circumstances (GOLD, 2017).

Clinician–patient communication should include a discussion of disease progression and patient choices for care, including high technology (intensive care unit [ICU], ventilator), subacute, long-term ventilator dependent, long-term care, home care, and surgical options. End-of-life care to include advance directives, resuscitation, palliative care and hospice considerations, and place of death preferences are important components.

Management of COPD Exacerbations: COPD exacerbations are defined as "an acute worsening of respiratory symptoms that result in additional therapy" (GOLD, 2017a, p. 31). They are attributed to progression of disease, worsening health status, and mortality (Wedzicha, 2015). Most commonly, COPD exacerbations are precipitated by respiratory tract infections (GOLD, 2017).

COPD exacerbations are classified as mild, moderate, or severe (GOLD, 2017a). Interventions aligned with severity include: mild (short-acting bronchodilator [SABDs]; treatment only; moderate (SABDs and/or oral corticosteroids); or severe (emergency department visit or hospitalization required) (GOLD, 2017a). Severe exacerbations may be associated with acute respiratory failure, which can be non-life threatening or life-threatening (GOLD, 2017).

Treatment modalities include short-acting beta$_2$-agonists with or without anticholinergics, long-acting bronchodilators,

systemic corticosteroids, antibiotics, noninvasive mechanical ventilation, and/or invasive mechanical ventilation (GOLD, 2017a).

Individualized COPD action plans for management of COPD exacerbations with a brief patient educational session has been found to significantly reduce the risk of hospitalization (Howcroft, Walters, Wood-Baker, & Walters, 2016). Goals for treatment include minimizing the negative impact relative to the current exacerbation and preventing future events (GOLD, 2017).

Pharmacological Management

Pharmacological therapy for COPD has a significant role in reducing symptoms, reducing exacerbation frequency and severity, along with improving exercise tolerance, health status, and quality of life (Dunphy et al., 2015; GOLD, 2017). Individualized treatment regimens are required based on assessment of symptoms severity, airflow limitation, and/or severity of COPD exacerbation (GOLD, 2017). A step-wise approach to therapy is utilized in the treatment of COPD. No current drug therapy has been proved to influence the long-term, progressive decline in COPD lung function (GOLD, 2017). Classes of medications to in the treatment of COPD are:

Inhaled Beta$_2$-Agonist Bronchodilators: Bronchodilators are widely used in the treatment of COPD to reverse bronchoconstriction by relaxing smooth muscles in the airways (GOLD, 2017). Acute bronchospasms lead to permanent airway remodeling and, therefore, prevention is paramount (Dunphy et al., 2015). There are two types of beta$_2$-agonists: SABAs and LABAs. SABAs, known as "rescue" inhalers, are prescribed for intermittent symptoms on an as-needed basis. SABAs include medications such as albuterol or levalbuterol. Onset is rapid, about 15 to 30 minutes, and duration of action is 4 to 6 hours.

Beta-agonists are sympathomimetic agents with adverse effects that may include transient tachycardia, nervousness, palpitations, and and/or arrhythmia (GOLD, 2017). SABAs are not recommended for regular use and in patients who have recurrent symptoms. Modification of the treatment regimen with a step-wise approach would be indicated (GOLD, 2017).

LABAs in COPD are used for maintenance therapy to prevent acute bronchospasms and not for relief of acute symptoms (Dunphy et al., 2015). The duration of LABAs is about 12 hours (GOLD, 2017). Formoterol and salmeterol are two examples of LABAs that have demonstrated improvement in COPD patients (Ferguson & Make, 2017).

Inhaled Anticholinergic/Antimuscarinic Bronchodilators: Inhaled antimuscarinic bronchodilators block the bronchoconstriction effects of acetylcholine on muscarinic cholinergic receptors in the smooth muscle of the airways (GOLD, 2017). The short-acting antimuscarinic (SAMA) agent, ipratroprium, has a duration of 6 to 8 hours and is used in long-term and acute COPD management (Criner et al., 2015; Ferguson & Make, 2017). Tiotropium, a long-acting anticholinergic/antimuscarinic, is used for maintenance treatment of COPD. Duration of action is about 24 hours. Tiotropium has been shown to decrease symptoms of dyspnea along with the frequency of COPD exacerbations and hospitalizations (Criner et al., 2015; Ferguson & Make, 2017). Tiotropium demonstrated more benefits, such as fewer COPD exacerbations, reduced hospital admissions, and improved quality of life, as compared to ipratropium (Cheyne, Irvin-Sellers, & White, 2015). Antimuscarinic agents have anticholinergic properties and should be used cautiously in patients with narrow-angle glaucoma, prostatic hypertrophy, or bladder neck obstruction, and are contraindicated in patients who are allergic to atropine (Dunphy et al., 2015).

Combination Bronchodilator Therapy: When used in combination, bronchodilators with different mechanisms of action may improve the degree of bronchodilation with a lower risk of increasing a monotherapy agent (GOLD, 2017). Combination inhaled therapy with LABAs, ICSs, or long-acting inhaled anticholinergics are used for stable COPD patients with symptoms and may be more effective than the agents as individual components (Farne & Cates, 2015). In patients with stable, moderate to very severe COPD, a maintenance combination of an ICS/LABA or ICS and inhaled long-acting anticholinergic therapy are recommendations (Criner et al, 2015). For patients with severe COPD and persistent symptoms, triple therapy is recommended with a combination ICS/LABA and inhaled long-acting anticholinergic (Ferguson & Make, 2017).

Glucocorticoids: Glucocorticoids are utilized in the management of COPD for the anti-inflammatory effects on COPD-associated inflammation (Criner et al., 2015). Two types of glucocorticoids are available: ICSs and systemic corticosteroids. ICSs are used in combination with LABAs for optimal symptom management in persistent symptoms. Fluticasone/salmeterol and budesonide/formoterol are examples of combination pharmacological agents available. Instruct the patient to rinse the mouth after using. ICSs are not recommended for use as monotherapy due to greater efficacy when combined with a LABA (GOLD, 2017). In a large meta-analysis reviewing 71 randomized controlled studies with 73,062 individuals, lung function and quality of life was improved on combination inhalers (LABA and ICS) as compared to monotherapy with ICS (Kew, Dias, & Cates, 2014).

Systemic corticosteroids (e.g., prednisolone) have nonspecific anti-inflammatory properties and are used for short-term treatment in the management of an acute COPD exacerbation. Systemic corticosteroids are associated with numerous side effects and, therefore, have no role in long-term management of COPD (GOLD, 2017).

Methylxanthines: Theophylline, a nonspecific phosphodiesterase inhibitor, is the most common of methylxanthines, although its use has decreased significantly. Theophylline is an oral bronchodilator that has been found to be modestly effective with a small therapeutic range, potential for toxicity, many drug-to-drug interactions, and wide range of toxic adverse effects (GOLD, 2017).

Inhaled Delivery Technique: Delivery of inhaled pharmacological agents in COPD management is user dependent and has been identified as a long-term issue in COPD management (Sanchis, Gich, & Pederson, 2016).

Follow-Up: A newly diagnosed patient should be seen frequently until an optimal treatment plan is in place and disease education has been completed. For stable patients, routine reassessment every 3 to 6 months is advisable, with

instructions to the patient or caregiver to schedule an interim visit if there is a change in status, increased symptoms, or an infectious process. For stable patients with other significant comorbidities, visits every 3 months are advisable. A team model for chronic care is optimal for COPD management. Periodic reassessment of pulmonary function and assessment of patient report of breathing status can guide therapy. Patients who are unstable or have had an acute exacerbation will need more frequent follow-up visits initially. Collaborative management with these patients is strongly advised. During the immediate post-acute period, a pulmonary specialist should manage the patient. Pulmonary rehabilitation is helpful in moderate to severe disease (GOLD, 2017).

Sequelae: COPD is a multisystem disease and can lead to infections, particularly respiratory (pneumonia, recurrent bronchitis, viral infections), cor pulmonale, pulmonary hypertension, malnutrition, acute or chronic respiratory failure, steroid-induced myopathy or Cushing's syndrome, bullous lung disease, polycythemia, and sleep-related hypoxemia. Depression is frequently associated with COPD (Remels et al., 2007; Shapiro et al., 2010).

Prevention/Prophylaxis: Avoidance of tobacco, smoking cessation, and avoidance of second hand smoke are helpful.

Referral: Collaborative management is advised in all patients. Refer patients to a pulmonary specialist during instability, for hospital and post-acute care, and for oxygen therapy evaluation. Refer patients to a surgeon if the option of surgery is deemed appropriate. Refer patients to physical therapy for reconditioning; occupational therapy may also be helpful in teaching breathing techniques and energy-conserving measures. Consult with a dietary health professional when applicable. Post-acute COPD patients in the community should be referred for home care. Refer all patients to community resources and support groups. Palliative care and hospice options are considerations.

Education: Teach the patient, family, and/or caregivers about the disease process and its management; the association of risk factors such as smoking and importance of cessation; pharmacological therapy; oxygen therapy and precautions; and seeking early care with symptoms of exacerbation or infectious processes (GOLD, 2017). Self-management education and coaching are integral components of a chronic care model approach (GOLD, 2017). Self-management skills patients need to learn include monitoring and managing signs and symptoms of COPD; treatment adherence; and maintaining regular follow-up with health-care providers.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Smoking cessation is the most effective and cost-effective intervention and should be promoted at every visit.	A	GOLD, 2017 CDC, 2016
In patients with respiratory symptoms, particularly dyspnea, spirometry should be performed to diagnose airflow obstruction. Spirometry should not be used to screen for airflow obstruction in asymptomatic individuals.	B	GOLD, 2017 Guirguis-Blake et al., 2016
Pulmonary rehabilitation consisting of exercise training, muscle strengthening, nutritional counseling, energy conservation, ADLs, breathing modifications, medication education (including use of inhaler devices), and oxygen use is beneficial, particularly in moderate-to-severe COPD.	A	GOLD, 2017
Supplemental oxygen therapy in patients with hypoxemic COPD improves survival.	A	GOLD, 2017
Bronchodilators are the foundation of symptomatic management; they are given on an as-needed basis for symptom management.	A	GOLD, 2017
ACP, ACCP, ATS, and ERS recommend that clinicians prescribe monotherapy using either long-acting inhaled anticholinergics or long-acting inhaled beta agonists for symptomatic patients with COPD and FEV_1 <60% predicted. Clinicians should base the choice of specific monotherapy on patient preference, cost, and adverse effect profile.	A	GOLD, 2017 Cheyne et al., 2015 Farne et al., 2015 Kew et al., 2014
Long-term monotherapy with oral corticosteroids is not recommended in COPD.	A	GOLD, 2017

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Regular treatment with ICSs improves symptoms, lung function, and quality of life and reduces exacerbations in COPD patients with an FEV_1 <60% predicted.	A	GOLD, 2017 Cheyne et al., 2015 Criner et al., 2015 Farne et al., 2015 Kew et al., 2014
Education improves patient response to exacerbations.	B	GOLD, 2017
Prospective end-of-life discussions can lead to effective end-of-life decisions as a result of understanding advance directives.	B	GOLD, 2017

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

HEART FAILURE

Signal Symptoms: Dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, progressive activity intolerance, weakness, progressive edema.

Description: As a cardiac syndrome, heart failure is a secondary process associated with multisystem pathophysiology involving neurochemical regulation, abnormal cardiac muscle structure, and/or wall function. The older term *congestive heart failure* does not represent symptom presentation in all heart failure patients; therefore, the preferred term is *heart failure.*

Etiology: CAD and hypertension are the most common etiologies of heart failure. Other causes of heart failure include valvular heart disease, dysrhythmias (e.g., AF), COPD, obstructive sleep apnea, fluid volume overload, and hyper- or hypometabolic disorders (e.g., diabetes, anemia, thyroid disease, infection). Heart failure with reduced left ventricular ejection fraction (HF*r*EF), previously referred to as *systolic heart failure*, is due to a large, thin left ventricular wall that has lost the elasticity required to effectively eject enough blood to maintain normal cardiac output (Yancy et al., 2013). In contrast, heart failure with preserved LVEF (HF*p*EF), previously referred to as *diastolic heart failure*, is caused by thick, stiff-walled ventricles and may occur with normal or near-normal LVEF (Yancy et al., 2013).

Occurrence: Prevalence of heart failure with preserved and reduced LVEF are evenly distributed, with 650,000 new cases diagnosed each year (Mozzafarian et al., 2016). The lifetime risk of developing heart failure is 20% for those over 40 years of age or older. The 5-year mortality rate is approximately 50% (Mozzafarian et al., 2016). Heart failure is the fourth most common hospital discharge diagnosis in those over age 65 (CMS, 2015). Hospital readmissions within the first 30 day are correlated with increased mortality risk (Krumholz et al., 2013).

Age: More common in older adults.

Gender: More common in men in the general population, but higher in women within the long-term care setting.

Ethnicity: African Americans have higher rates of heart failure.

Contributing Factors: Nonmodifiable risk factors include gender and race. Advanced age is an independent risk factor for heart failure. Modifiable risk factors for heart failure are tobacco use, SBP of more than 140 mm Hg, fasting glucose of more than 125 mg/dL, elevated cholesterol, obesity, diet, and physical inactivity. Use of NSAIDs, COX-2 inhibitors, glucocorticoids, and thiazolidinediones are common precipitating factors for heart failure in the geriatric population (Maxwell & Jenkins, 2011; Trelle et al., 2011; Yancy et al., 2013).

Heart failure is caused by myocardial structural or functional damage. To maintain cardiac output, stroke volume, and vital organ perfusion, the sympathetic nervous system and renin-angiotensin-aldosterone system (RAAS) are activated to release neurohormones. Angiotensin II, the end product of the RAAS and a potent vasoconstrictor, leads to increased systemic vascular resistance, enhances the release of catecholamines from noradrenergic nerve endings, and directly stimulates the adrenal cortex to increase secretion of aldosterone. Vasopressin (antidiuretic hormone) and aldosterone secretion cause sodium and fluid retention with subsequent increase in blood volume. Increased systemic vascular resistance, decreased arterial pressure, increased venous pressure, increased blood volume, and decreased venous compliance further increase ventricular wall stress and myocardial oxygen demand. Ultimately, these normal physiological responses become maladaptive and result in damage to the heart, cardiopulmonary, renal, and systemic vasculature, and cause progression of heart failure.

Signs and Symptoms: Geriatric patients with severe heart failure may be asymptomatic, whereas others with mild disease can suffer incapacitating symptoms (Ahmed, 2011).

Left ventricular heart failure leads to pulmonary venous congestion, subsequent right ventricular dysfunction, and portal and systemic venous congestion. Clinical symptoms of dyspnea on exertion or at rest, orthopnea, dry cough (worse in recumbent position), and/or paroxysmal nocturnal dyspnea indicate increased pulmonary venous congestion. Progressive lower extremity edema (not due to venous insufficiency or hypoalbuminemia) and abdominal symptoms (pain, distention, nausea) related to hepatic enlargement are symptoms suggestive of right ventricular failure or systemic venous congestion.

Signs that suggest heart failure include weight gain, tachycardia, S_3 or S_4 heart sound, laterally displaced point of maximum impulse (PMI), neck vein distention, rales in bilateral lower lobes that are not due to atelectasis, positive hepatojugular reflux, and ascites. Cognitive impairment is common in older adults with heart failure.

Heart failure classification is based on risk, cardiac structural changes, symptom presentation, and functional impairment. The New York Heart Association (NYHA) classifies heart failure based on functional limitation (Yancy et al., 2013). This system is difficult to use with the elderly population, who may have functional decline related to comorbid conditions. The heart failure rating system is based on risk factors, structural changes, and history of heart failure symptoms. The ACC/AHA system classifies heart failure in four stages as the syndrome progresses along the natural trajectory and is used in conjunction with, not as a replacement for, the NYHA functional classification system. Stages A and B refer to risk factors and present opportunities for primary and secondary prevention measures.

- Stage A: Risk factors are present for heart failure without structural changes, history of signs or symptoms, or functional limitations.
- Stage B: Risk factors and structural changes in the heart are present without signs or symptoms of heart failure.
- Stage C: Risk factors, structural changes in the heart, and past or current signs or symptoms of heart failure are present. At this stage, the patient is diagnosed with heart failure that does not regress back to former stages.
- Stage D: Heart failure is refractory despite maximum medical management. Patients are symptomatic at rest or with minimal exertion.

Diagnostic Tests:

Two-Dimensional Echocardiography Combined With Doppler Flow Studies: Two-dimensional echocardiogram is considered the most useful diagnostic tool for evaluation of ventricular ejection fraction and structural abnormalities for prognostic and interventional purposes (Ahmed, 2011; Yancy et al., 2013). Two-dimensional echocardiograms are essential to accurate diagnosis and treatment plan development. Echocardiogram is essential for those with unknown history of heart failure and clinical presentation to evaluate ventricular function, size, wall thickness, wall motion, and valve function criteria. Diagnosis of HF*p*EF is supported by 1) evidence of clinical heart failure symptoms, 2) normal or mildly abnormal LV systolic function, and 3) evidence of abnormal LV relaxation, filling, diastolic distensibility, or diastolic wall function (Nugeuh et al., 2016).

Electrocardiography: EKGs may indicate left atrial enlargement, left ventricular hypertrophy, arrhythmias, or ST-T wave changes associated with MI or ischemia.

Cardiac Angiography: Cardiac catheterization may benefit geriatric patients who are candidates for revascularization and may reveal hemodynamic abnormalities, the presence and severity of valvular heart disease, or CAD.

Chest X-Ray: Chest x-ray may reveal cardiomegaly, increased pulmonary vascularity, interstitial or pulmonary edema, and assists in evaluating alternative etiologies that may contribute or cause heart failure-like symptoms.

Brain Natriuretic Peptide (BNP) or N-Terminal proBNP (NT-proBNP): BNP and NT-proBNP are helpful in narrowing differential diagnoses or establishing prognosis. BNP elevation occurs slowly; therefore, levels are not helpful in acute heart failure episodes. BNP levels of less than 100 pg/mL indicate that heart failure is unlikely. Levels between 100 and 500 pg/mL are indeterminate. Levels of more than 500 pg/mL may suggest heart failure, but are not appropriate as "stand-alone" tests.

Blood Chemistries and CBC: CBC, comprehensive metabolic profile (CMP), and thyroid-stimulating hormone (TSH) levels should be obtained initially and periodically to evaluate renal, liver, metabolic, and hemodynamic status.

Serial Cardiac Enzymes: Cardiac enzymes (CK-MB, troponin) may reveal myocardial ischemia or infarction. Cardiac enzymes require time-sensitive processing; therefore, have limited value in the long-term care setting. If the presentation is questionable, transport via emergency medical services to an acute care facility is appropriate.

Differential Diagnosis: Atypical symptom presentation in geriatric patients necessitates a thorough health history that includes evaluation of risk factors with correlation to physical examination findings and diagnostic test results. Dyspnea on exertion, the most common heart failure symptom, may be caused by numerous disease processes, including asthma, COPD, anemia, renal failure, dysrhythmias, cardiomyopathies, valvular heart disease, primary pulmonary hypertension, PE, abdominal masses, deconditioning, and anxiety neurosis. Differentiation between heart failure with preserved LVEF or reduced LVEF is crucial and best determined by two-dimensional echocardiography (Ahmed, 2011; Yancy et al., 2013).

Treatment: Treatment options for heart failure are targeted at symptom management, treatment of the causative disease process(es), and the resulting multisystem pathophysiology. Fragility, multiple comorbid disease processes, polypharmacy, age-related changes in pharmacokinetics and pharmacodynamics, and prevalence of geriatric syndromes require individualized treatment plans.

Angiotensin-Converting Enzyme Inhibitors: Unless contraindicated, angiotensin-converting enzyme (ACE) inhibitors (ACEIs) are first-line agents in the treatment of asymptomatic HF*r*EF or in patients with recent or remote history of MI regardless of LVEF. ACEI contraindications include unilateral or bilateral renal artery stenosis, hypersensitivity to ACEIs, angioedema, pregnancy, serum potassium more than 5.5

mEq/L that cannot be reduced, and hypotension in patients at risk for cardiogenic shock. ACEI therapy should be discontinued as a last resort in heart failure patients due to cardioprotective properties. All ACEIs should be started at low doses and gradually titrated over several weeks to achieve target doses daily based on renal function, potassium levels, BP, and tolerance. Obtain baseline serum potassium, serum creatinine, and blood urea nitrogen (BUN) levels before initiation and with dosage adjustments. Dosage adjustments should be made accordingly for symptoms of dizziness, light-headedness, or unsteadiness. ACEI cough is a common side effect and is characterized as a dry nonproductive cough that starts as a tickling cough, may be worse at night, and can occur at any point in treatment. ACEI cough is a classwide side effect and is not dose dependent.

Angiotensin II Receptor Blockers: Angiotensin II receptor blockers (ARBs) are an appropriate substitute for ACEIs, as they do not inhibit kininase and are associated with lower incidence of cough and angioedema (Yancy et al., 2013). Isosorbide dinitrate and hydralazine combination therapy is an appropriate alternative in patients who experience hypotension, hyperkalemia, angioedema, or renal insufficiency with ACEI or ARB therapy, and those who do not respond to treatment with ACEIs and beta blockers.

Angiotensin Receptor/Neprilysin Inhibitors: Angiotensin receptor/neprilysin inhibitors (ARNIs) are ARBs combined with an inhibitor of neprilysin, an enzyme that degrades natriuretic peptides, bradykinin, adrenomedullin, and other vasoactive peptides (McMurray et al., 2014). ARNIs are approved as a substitute for ACEIs or ARBs in those with symptomatic HF*r*EF and should be not used concomitantly with these agents and with a 3-day washout period (McMurray et al., 2014). Clinical trials have shown 20% reduction in cardiovascular death or heart failure hospitalization (McMurray et al., 2014; Yancy et al., 2016).

Beta-Adrenergic Blockers: Carvedilol, metroprolol succinate, and bisoprolol are the only beta blockers indicated for treatment of heart failure and with evidence to support morbidity and mortality reductions in heart failure patients. Unless otherwise contraindicated (heart block, bradycardia, hypotension, severe respiratory disease), beta blockers are recommended for patients with recent or remote history of MI regardless of LVEF. Use of carvedilol or metoprolol succinate has been shown to decrease morbidity and mortality in heart failure patients and is recommended in patients with Stage C heart failure and/or a history of MI regardless of LVEF.

Beta blockers are strongly associated with reduced risk of mortality and reduced rehospitalization during 30-, 60-, and 90-day follow-up periods. Carvedilol is the best option in those with advanced heart failure. Beta blockers should be used in combination with ACEIs, if possible, for synergistic effect. Monitor geriatric patients closely for volume status, emotional side effects, and functional decline. Contraindications include bronchospastic lung disease (e.g., asthma), symptomatic bradycardia, or advanced heart block without a pacemaker. Beta blocker initiation should be started only in a euvolemic patient. Initial dosages should be low and titrated slowly based on response to treatment and tolerance. Carvedilol may have a greater hypotensive effect in the geriatric patient due to the vasodilatory effect of alpha-1 receptor blockade properties. Metoprolol succinate may be better tolerated in those with lower baseline BP levels. Consider carvedilol or bisoprolol in patients who require crushed medications.

Diuretic Therapy: Loop diuretics are used to reduce symptoms of peripheral or pulmonary congestion. Loop diuretics inhibit the Na+/K+/Cl- symporter in the ascending loop of Henle, whereas thiazide-type diuretics affect the Na+/Cl- in the distal convoluted tubules. Use of diuretics in the treatment of heart failure without ACEIs, ARBs, or beta blockers activates the RAAS and may hasten progression of heart failure. Most heart failure patients are resistant to the effects of hydrochlorothiazide; therefore, loop diuretics are preferred diuretics in heart failure treatment. When used in equipotent dosages, loop diuretics appear to have comparable diuretic effects (1 mg bumetanide = 40 mg furosemide = 10 to 20 mg torsemide). Furosemide should be initiated at 20 mg daily oral dosage in the older adult patient with mild symptoms, with dosage titration based on effect and tolerance. Bumetanide is 40 times more potent than furosemide and is less ototoxic, but it is associated with myalgias in some patients.

Fluid overload refractory to loop diuretics results from downregulation in renal response and sodium retention (Trullás et al., 2016). The addition of a thiazide diuretic to a loop diuretic may be an indicated and efficacious option to reduce acute heart failure symptoms if a sub-therapeutic response to loop diuretic dosage increase is noted (Trullás et al., 2016). Metolazone (2.5 to 5 mg daily), a thiazide-like agent, is a long-acting diuretic that is effective in increasing the effects of loop diuretics if given 30 minutes before the loop diuretic dosage. Combination therapy with metolazone requires close monitoring of potassium and magnesium levels. Torsemide may be advantageous, because absorption is unimpaired and the response is less variable than the other loop diuretics. Amiloride, triamterene, and ethacrynic acid should only be considered if there is a life-threatening allergy to sulfa due to side effects and inability to protect against damaging neurohormonal activity. Spironolactone, an ARB, is an effective therapy in conjunction with ACEIs or ARBs to more completely block the deleterious effects of the RAAS in patients with Stage C and D Class III–IV HF*r*EF and in patients with LVEF of 40% or less who have adequate renal function without hyperkalemia. Additionally, providers should consider ability to monitor for renal dysfunction and hyperkalemia before initiation of therapy.

Aldosterone Antagonists: Aldosterone antagonists are recommended at low dosages once daily and should not be initiated in men with serum creatinine levels of 2.5 or more; in women with serum creatinine levels of 2.0 or more; or potassium levels of more than 5.0.

Digoxin: Digoxin is recommended in the treatment of heart failure patients without bradycardia as a fourth-line agent in those who have persistent symptoms despite optimal therapy with diuretics, ACEIs, and beta blockers. In addition, digoxin may be helpful to control ventricular response due to AF in men with heart failure with LVEF of less than 50% (Yancy et al., 2013, 2016). To reduce morbidity and mortality, lower therapeutic range (0.5 to 0.9 ng/mL) is advised (Yancy et al., 2013). Evidence from several trials supports the use of digoxin to improve functional status and decrease heart failure

BOX 8-1
R U Watching?

R - Reconcile medications post hospital

U - Up-titrate medication to target dosages as tolerated (document failure attempts)

Watching

- Daily weight until stable x 4 weeks, then weekly
- Notification parameters (Call >3 # daily or >5 # weekly)

BOX 8-2
ABCs of Transitions

A - ACEI/ARB or ARNI

B - Beta antagonist
Restart when stable on follow-up

C - Closely monitor labs

D - Diet (no added salt)
Teach families to read nutrition labels

E - Exacerbation plan

F - Follow-up visit within 7 days post hospitalization; telephone follow-up within 3 days

hospitalization without improvement in mortality. Geriatric dosages should not exceed 0.125 mg daily (American Geriatrics Society, 2015; Beers Criteria Update Expert Panel, 2015). In geriatric patients with renal dysfunction, every-other-day dosage may be appropriate. ACEIs, beta blockers, diuretics, nitrates, calcium channel blockers (CCBs), and aldosterone antagonists are helpful in treating HF*r*EF. Concurrent treatment with beta blockers and non-dihydropyridine calcium channel antagonist agents is contraindicated. There is no evidence supporting the use of CCBs in the treatment of heart failure (Yancy et al., 2013).

Follow-Up: Fatigue and weakness without vital sign changes may occur with titration of any medication, but are transient and should resolve within several days following dosage titration. Ask the patient about the presence of symptoms of heart failure as noted previously. Asking specific questions specific to triad symptoms (nocturnal dyspnea, orthopnea requiring two or more pillows, and dyspnea with exertion) is considered an optimal approach to symptom recognition. Ask general questions related to the patient's quality of life such as sleep patterns, sexual difficulties, symptoms of depression, and coping behaviors. Assess for caregiver overburden. Immediate follow-up post-hospitalization and frequent follow-up may reduce readmission rates (Boxes 8-1 and 8-2).

Sequelae: Complications of heart failure involve hepatic and renal end-organ disease. Fifty percent of heart failure sufferers die from sudden cardiac death. All patients with heart failure should have an advance care plan and documented goals of care, including the Medical Orders for Scope of Treatment.

Prevention/Prophylaxis: Reducing cardiac risk factors in older adult affects coronary disease as strongly as it does in younger age groups. Encourage risk factor reduction using age-specific guidelines to facilitate changes in lifestyle. Patients with heart failure are at risk for a vascular event, although there is conflicting evidence regarding efficacy of prophylactic antithrombotic treatment (aspirin or warfarin) without other indications.

Referral: Referral for interprofessional team and chronic care case management should be considered for all patients with heart failure. Warning signs for the need for hospitalization include weight gain that is resistant to diuresis, palpitations, persistent or recurrent dizziness, agitation or cognitive changes, inability to sleep because of paroxysmal nocturnal dyspnea, abdominal pain, and inability to walk.

Practice of best evidence involves shared decision making between an experienced, well-coordinated, proactive primary care team in a patient-centered palliative care model. Referral to cardiology is indicated when the diagnosis is unclear, if the patient remains symptomatic despite appropriate therapy, and in those with significant cardiac disease. Hospice should be considered when a patient has Stage D Class IV heart failure, has an ejection fraction of 20% or less, is unable to tolerate optimal therapy due to hypotension or renal failure, or has symptoms that are not controlled with maximized medical management.

Education: Education and use of support groups are important for patients with heart failure, because noncompliance is a major cause of morbidity and unnecessary hospital admissions. Instruct patients and their families about the natural progressive trajectory of heart failure, necessary medications, dietary restrictions, heart failure symptoms that necessitate contact with provider, and prognosis. Discussions regarding advance directives should begin early in the trajectory. Explain typical symptoms of worsening heart failure (orthopnea, paroxysmal dyspnea, leg edema, or exercise intolerance) and instruct patients to contact their health-care provider if these occur. Mild aerobic exercise increases functional capacity and improves the quality of life for heart failure patients, but should be undertaken with provider supervision. Dietary sodium should be restricted to 2 or 3 g per day. Discourage alcohol use and tobacco use. Fluid restriction is not necessary unless there is hyponatremia, but patients with heart failure should avoid excessive fluid intake. Advise patients to contact the health-care provider if weight increases more than 2 pounds in a day or 4 pounds in a week. Nurse practitioners should recommend that patients with heart failure receive vaccination against influenza and pneumococcal disease.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
ACEIs are recommended for all patients with HF*r*EF unless contraindicated.	A	Yancy et al., 2013, 2016 Muth, Gensichen, Beyer, Hutchinson, & Gerlach, 2009
ARBs are an appropriate option for those who have HF*r*EF are intolerant to ACEIs.	A	Pitt et al., 1999 Yancy et al., 2013, 2016 Zannad et al., 2010
ARNIs are an appropriate substitute in those who have HF*r*EF and cannot tolerate ACEIs or ARBs	A	Yancy et al., 2013, 2016 McMurray et al., 2014
Beta blockers should be used in combination with ACEIs if possible for synergistic effect.	A	Poole-Wilson et al., 2003 Muth et al., 2009 Yancy et al., 2013
Beta blocker initiation should be started only in a euvolemic patient. Initial dosages should be low and titrated slowly based on response to treatment and tolerance. Transient fatigue is common with initiation and dosage titration.	A	Rienstra et al., 2013 Yancy et al., 2013
Beta blockers (1 of the 3 specified) are recommended for patients with or without recent or remote history of MI, regardless of LVEF.	A	Rienstra et al., 2013 Yancy et al., 2013
Avoid use of diuretics in the treatment of heart failure without ACEIs, ARBs, or beta blockers (activates the renin-angiotensin-aldosterone system and may hasten progression of heart failure).	C	Harrington, 2016 Triposkiadis et al., 2009
Aldosterone antagonist is recommended in HF*r*EF/NYHA Class II–IV given appropriate renal function (>30 ml / min/1.73m^2) and serum potassium is less than 5 mEq/L. Class II patients should have a history of prior hospitalization and elevated natriuretic peptides for consideration of therapy. Providers should consider ability to monitor for renal dysfunction and hyperkalemia before initiation of therapy. Renal function and electrolytes should be checked in 2 to 3 days and again in 7 days following initiation or dosage adjustment.	A	Pitt et al., 1999 Yancy et al., 2013 Zannad et al., 2010
Isosorbide dinitrate and hydralazine combination therapy is an appropriate alternative in patients who experience hypotension, hyperkalemia, angioedema, or renal insufficiency with ACEI or ARB therapy, in African Americans with NYHA class III–IV HF*r*EF receiving optimal therapy with ACEIs and beta blockers, unless contraindicated.	B A	Yancy et al., 2013
Combined use of ACEI, ARB, and aldosterone antagonist is potentially harmful in those with HF*r*EF.	B	Cohn & Tognoni, 2001 Yancy et al., 2013 Zannad et al., 2010

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Digoxin may be helpful in those with HF*r*EF to reduce hospitalization to control ventricular response due to AF as an add-on agent for control of heart failure symptoms resistant to ACEIs, beta blockers, and diuretics without contraindication.	A	Yancy et al., 2013
Dietary sodium restriction of 3 g/day is reasonable. Fluid restriction is not necessary unless there is hyponatremia or Stage D Class IV symptom management.	C	Yancy et al., 2013
Discourage alcohol use and tobacco use.	C	AHA, 2016 Mozzafarian et al., 2016 Yancy et al., 2013
Use of NSAIDs, COX-2 inhibitors, glucocorticoids, and thiazolidinediones are common precipitating factors for heart failure in the geriatric population.	A	Trelle et al., 2011 Yancy et al., 2013
Vaccination against influenza and pneumococcal disease is recommended unless contraindicated for heart failure patients.	A	ACIP, 2016 Moberley, Holden, Tatham, & Andrews, 2008

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

HYPERTENSION

Hypertension is one of the most important preventable contributors to cardiovascular disease and death. With the growing number of the world population over the age of 60 years and an increase in life expectancy, clinicians will be treating more and more patients with hypertension (Williamson et al., 2016).

Signal Symptoms: Hypertension is usually asymptomatic, except in the case of hypertensive crisis, and is often referred to as the "silent killer" (Basile & Bloch, 2016).

Description: The ACC/AHA 2017 guidelines for the detection, evaluation, and management of hypertension in adults incorporate new information regarding BP-related risk factors for cardiovascular disease and new BP thresholds for initiating antihypertensive drug therapy (Whelton et al., 2017). According to the new guidelines, BP should be defined as normal, elevated, or stages one or two (Whelton et al., 2017). Normal BP is defined as less than 120/less than 80 mm/Hg. Elevated BP is defined as 120 to 129/less than 80 mm/Hg. Stage one is defined as SBP 130 to 139 mm/Hg or DBP 80 to 89 mm/Hg. Stage two is defined as SBP greater than or equal to 140 mm/Hg or DBP greater than or equal to 90 mm/Hg (see Table 8-2) (Whelton et al., 2017).

Although hypertension is generally asymptomatic, it is a key risk factor for cardiovascular disease and contributes to approximately 45% of deaths due to heart disease and 51% of deaths due to strokes (Kjeldsen et al., 2014). Hypertension is a prevalent disorder in older adults and is one of the most common diagnoses seen in primary care (James et al., 2014).

TABLE 8-2 Classification of Hypertension

SYSTOLIC BLOOD PRESSURE	DIASTOLIC BLOOD PRESSURE	CLASSIFICATION
130–139 mm Hg	or 80–89 mm Hg	Stage 1 hypertension
≥140 mm Hg	or ≥ 90 mm Hg	Stage 2 hypertension
<120 mm Hg	and <80 mm Hg	Normal
120–129 mm Hg	and <80 mm Hg	Elevated

Adapted from Whelton et al., 2017.

Both SBP and DBP rise with age (Logan, 2011). However, the diastolic component of BP usually begins to level off around the age of 50 years and slowly declines over time (Logan, 2011). Isolated systolic hypertension is very common in older adults. It is defined as an SBP of 160 mm Hg or greater and is associated with a higher risk of MI and stroke (Logan, 2011). Hypertension is a major risk factor for

cardiovascular and renal disease, but is a modifiable cause of mortality (Logan, 2011).

Numerous clinical trials, including studies in older adults, document effective treatment and management of hypertension to improve survival rates with cardiovascular benefits (Alhawassi, Krass, & Pont, 2015). There are multiple, robust, randomized controlled trials that provide solid evidence-based treatment guidelines for hypertension in older adults (Alhawassi et al., 2015). When making a diagnosis of hypertension, it is also important to screen and manage other cardiovascular disease risk factors such as smoking, diabetes, hyperlipidemia, overweight, lifestyle factors, and sleep apnea (Whelton et al., 2017).

Etiology: Hypertension develops in response to an increase in cardiac output or an increase in peripheral resistance. The exact etiology of primary hypertension remains unclear but is thought to be the result of a number of genetic and environmental factors that affect cardiovascular and renal structure and function. Secondary hypertension results from some known causes such as renal disease, obstructive sleep apnea, prescription and OTC medications, alcohol, and primary aldosteronism (Whelton et al., 2017). Uncommon causes of secondary hypertension include Cushing's syndrome, hypothyroidism, hyperthyroidism, primary hyperparathyroidism, and acromegaly (Whelton et al., 2017).

Occurrence: It is estimated that 29% to 31% of adults in the United States have hypertension. With an aging population, the number is likely to grow, as hypertension occurs in the majority of persons over the age of 65 years (www.cdc.gov).

Age and Gender: Gender differences are noted in both younger and older populations. Men under the age of 45 years are more likely to develop hypertension than women. However, after the age of 60 years women are more likely than men to develop hypertension (Alhawassi et al., 2015).

Ethnicity: Hypertension is more common in African American adults than in Caucasians and Hispanics, and develops at a younger age (www.cdc.gov).

Contributing Factors: Contributing factors in the development of hypertension include genetics, obesity, stress, lifestyle factors, loss of elasticity of blood vessels, and age (Basile & Bloch, 2016).

Aging is a major contributing factor to hypertension. As one ages, there is associated BP elevation caused by changes in arterial vessel structure and function (Logan, 2011). Collagen in blood vessels becomes more rigid, leading to sclerosis and fibrosis of the vessels (Logan, 2011). This stiffening of the blood vessels causes a widening of the pulse pressure, commonly found in older adults (Logan, 2011). Furthermore, structural changes in blood vessels amplify systolic pressure with each heartbeat, causing an increase in systolic pressure and a fall in diastolic pressure (Logan, 2011).

Older adults are also more sensitive to salt due to reduced ability to excrete sodium. This increased salt sensitivity is caused by a decline in renal function and reduced generation of natriuretic substances, such as prostaglandin E2 and dopamine, resulting in elevated blood pressure (Logan, 2011). The prevalence of diabetes and glucose intolerance also increases with age and is a contributing factor to vascular injury and subsequent elevation in BP (Logan, 2011). Progressive renal dysfunction as part of the aging process may contribute to the pathophysiology of hypertension in the geriatric population (Logan, 2011).

Secondary causes of hypertension such as renal artery stenosis, sleep apnea, primary hyperaldosteronism, and thyroid disorders should be considered and ruled out in older adults before assuming a diagnosis of hypertension (Logan, 2011).

Diagnostic Tests: All adults 18 years and older should be screened for elevated BP. Before labeling an individual with a diagnosis of hypertension, the clinician should use an average of two or more readings obtained on two or more occasions (Whelton et al., 2017). Out of office and self-monitoring of BP measurements are recommended to confirm the diagnosis of hypertension (Whelton et al., 2017). As part of the evaluation and work-up for primary hypertension, the following studies should be considered:

- CMP
- CBC
- Lipid panel
- Thyroid panel
- Urinalysis
- EKG
- Echocardiogram, uric acid level, urinary albumin to creatinine ratio (optional) (Whelton et al., 2017)

Differential Diagnosis: Differential diagnoses to consider are drug-induced hypertension, chronic kidney disease, obstructive sleep apnea, hyperaldosteronism, hypothyroidism, hyperthyroidism, hyperparathyroidism, and Cushing syndrome when confirming a diagnosis of hypertension (Basile & Bloch, 2016). Accurate diagnosis of hypertension and identification of other cardiovascular risk factors is important to identify patients who would benefit from BP management (Kjeldsen, 2014). Health-care provider measurement of BP tends to be poor and often fails to look for the white-coat effect, masked hypertension, orthostatic hypotension, or postprandial hypotension, which leads to overestimation of BP and inappropriate treatment (Morley, 2014).

White coat hypertension in older adults is described as having an elevated office BP with a normal ambulatory or home BP with the patient receiving antihypertensive treatment. Research finds that medications do not have a significant effect on lowering BP in individuals with true white-coat hypertension (Franklin et al., 2012).

Masked hypertension is described as having a normal office BP with an elevated out-of-office BP, determined by ambulatory or home BP monitoring. Studies indicate 15% to 30% of older adults have masked hypertension with associated increased cardiovascular risks (Peacock, 2014).

Orthostatic hypotension is defined as a significant fall in BP when moving from a supine or sitting to a standing position. Because orthostatic hypotension is a significant risk factor for falls and fractures in older adults, screening for orthostatic hypotension should be an important part of the clinician's work-up for hypertension (Shaw & Clayton, 2014). Orthostatic testing involves a measurement of BP from two different body positions, with the most common sitting then standing. Orthostatic hypotension is present with a decrease in systolic pressure of 20 mm Hg or a decrease in diastolic pressure of 10 mm Hg within 3 minutes of standing when compared with BP from a sitting or supine position (Shaw & Clayton, 2014).

Postprandial hypotension is another important consideration in the work-up for hypertension in older adults. Postprandial hypotension is a drop in SBP within 2 hours of a meal, which can also increase the risk for falls in older adults (Nair, Visvanathan, & Piscitelli, 2016).

Although a single, carefully taken BP may be a predictor of future cardiovascular risk, the AHA recommends using ambulatory or home BP readings in conjunction with office monitoring as a better method for diagnosing hypertension and for subsequent guidance in treatment decisions (Logan, 2011). The USPSTF also recommends obtaining BP measurements outside of the clinical setting for diagnostic confirmation before starting treatment (National Guideline Clearinghouse, 2015).

Ambulatory or home BP monitoring is considered a better predictor of cardiovascular risk and provides better data for clinicians to make treatment decisions than a few office readings (Crabtree & Stuart-Shor, 2014). Evidence-based guidelines recommend home BP monitoring along with office monitoring, as it helps with patient engagement with treatment and improvement in shared decision making (Crabtree & Stuart-Shor, 2014). The clinician should assess the patient's capability of doing home BP monitoring and provide adequate instructions.

Sleep apnea, pain, nocturnal hypoglycemia, excessive aldosterone production, and pheochromocytoma should all be considered as causes of hypertension in older patients and should be ruled out before initiating treatment for hypertension (Morley, 2014). Lifestyle risk factors such as smoking, excessive alcohol consumption, and lack of exercise should also be discussed with the patient. Before initiating treatment for hypertension, a diagnostic work-up is recommended, including EKG, urinalysis, CMP, lipid panel, CBC, TSH, and free T4 (Morley, 2014).

Treatment: Treatment of hypertension is recommended for noninstitutionalized, ambulatory, community-dwelling adults (65 years of age or older) with an average SBP greater than or equal to 130 mm Hg (Whelton et al., 2017). Treatment goal should be SBP less than 130 mm Hg (Whelton et al., 2017). For older adults (65 years of age or older), both community dwelling and those residing in long-term care facilities with complex comorbid conditions and/or limited life expectancy, treatment should be based on clinical judgement, goals of care, and a team approach to determine risk/benefit for intensity of BP targets and choice of antihypertensive medications (Table 8-3) (Whelton et al., 2017).

There is scant research for treatment of hypertension for those ages 80 years or older, although some evidence exists for a BP goal of less than 150/90 mm Hg in this age group (James et al., 2014). Management of hypertension in geriatric patients can be challenging. These patients often have organ damage or cardiovascular disease, leading to a different response to treatment than in younger patients (James et al., 2014). Clinical judgment must play an important role in using evidence-based guidelines regarding treatment decisions and must be individualized for each patient in the geriatric population (Logan, 2011).

Poor adherence to a medication regimen due to pill burden, the cost of medications, or cognitive impairment is common in older adults (Jones, 2016). This is often the cause of poorly controlled BP and a major predictor for nursing home placement in frail older adults (Jones, 2016). Hypertensive therapy often involves a high number of medications, usually with an assortment of classes (Jones, 2016). Studies provide strong evidence that as the number of medications increase, patient adherence falls (Jones, 2016). It is important to complete a medication reconciliation during each visit and specifically ask about how prescriptions are being filled and medications are being taken (Jones, 2016). To increase compliance, clinicians should prescribe once-a-day medications whenever possible, as frequency of dosing contributes to poor medication adherence (Jones, 2016). Lack of medication adherence should not be mistaken for treatment-resistant hypertension (Jones, 2016).

TABLE 8-3 2017 ACC/AHA Guidelines for the Treatment of Hypertension in Adults

Normal blood pressure	Evaluate yearly.
Elevated blood pressure or Stage 1 hypertension with estimated cardiovascular risk less than 10%	Start treatment with nonpharmacological interventions and reevaluate in 3 to 6 months. Nonpharmacological interventions should include: weight loss, heart healthy diet, sodium reduction, potassium supplement unless contraindicated due to chronic kidney disease or use of drugs that inhibit potassium excretion, increased activity, moderation in alcohol consumption, and smoking cessation. Goal of treatment <130/80 mm Hg.
Stage 1 hypertension with estimated cardiovascular risk 10% or greater	Initial treatment should include nonpharmacological interventions and antihypertensive drug therapy and reevaluate in 1 month. Goal of treatment <130/80 mm Hg.
Stage 2 hypertension	Initial treatment should include nonpharmacological interventions and antihypertensive drug therapy with two agents of different classes and reevaluate in 1 month. Goal of treatment <130/80 mm Hg.

Adapted from Whelton et al., 2017.

Hypertension in older adults is treated with the same medications as with younger patients, but the clinician must take into account age-related changes in metabolism and pharmacodynamics (Sherman, 2013). Before any consideration of antihypertensive medication therapy the clinician should first consider nonpharmacological interventions for BP reduction. Weight loss and reduction of sodium intake can be effective therapy in many older adults (DeSimone & Crowe, 2009). Other lifestyle modifications such as smoking cessation, exercise, and moderation in alcohol consumption should be discussed with the patient (Sherman, 2013).

When initiating antihypertensive drug therapy, first-line agents include thiazide diuretics, CCBs, and ACEIs or ARBs (Whelton et al., 2017). Simultaneous use of an ACEI and ARB is contraindicated and potentially harmful to the patient (Whelton et al., 2017). The goal of the 2017 ACC/AHA guidelines is to facilitate evidence-based practice in the management of hypertension in adults (Table 8-4).

Medications should be started at the lowest dose and gradually increased to meet the targeted BP goal. Most older

TABLE 8-4 Evidence-Based Dosing for Antihypertensive Agents

MEDICATION	STARTING DAILY DOSE	TARGETED DOSE IN RANDOMIZED CONTROLLED TRIALS
Thiazide Diuretics		
Hydrochlorothiazide	12.5–25 mg	25–100 mg
Chlorothiazide	12.5 mg	12.5–25 mg
Indapamide	1.25 mg	1.25–2.5 mg
ARBs		
Lorsartan	50 mg	100 mg
Valsartan	40–80 mg	160–320 mg
ACEIs		
Enalapril	5 mg	20 mg
Lisinopril	10 mg	40 mg
CCBs		
Amlodipine	2.5 mg	10 mg
Diltiazem ER	120–180 mg	360 mg
Beta Blockers		
Metoprolol	50 mg	100–200 mg
Atenolol	25–50 mg	100 mg

Source: James et al., 2014.

adults will require more than one medication along with lifestyle changes to reach the BP goal (Sherman, 2013). Review of the literature indicates 70% of older adults require two or more drugs to reach BP goals (Sherman, 2013). Specific comorbid illnesses may guide the initial and subsequent choice of drug therapy (Sherman, 2013). ACEIs are recommended for patients with renal disease and diabetes. Beta blockers are recommended for those with CAD or CHF (National Guideline Clearing House, 2015).

Follow-Up: Follow-up is important, as the majority of patients with hypertension are not adequately controlled. Per ACC/AHA guidelines, once treatment has been started for hypertension, low-risk adults with elevated BP or stage one hypertension with low cardiovascular risks should be reevaluated in 3 to 6 months (Whelton et al., 2017). Adults with stage one and stage two hypertension with high cardiovascular risks should be reevaluated in 1 month (Whelton et al., 2017). Comorbid conditions such as diabetes, heart failure, and chronic kidney disease will influence the frequency of follow-up visits.

Sequelae: Hypertension is associated with serious adverse effects, such as left ventricular hypertrophy, heart failure, stroke, and kidney disease (Basile & Bloch, 2016).

Prevention/Prophylaxis: One of the primary preventative measures for hypertension is lifestyle modification. Excess weight, excess sodium intake, inadequate physical activity, smoking, inadequate intake of fruits and vegetables, and excessive alcohol consumption are all modifiable factors that can contribute to the onset of hypertension (Basile & Bloch, 2016).

Referral: The clinician should consider referring to a hypertension specialist when the BP goal cannot be attained or for the management of complicated patients who may benefit from consultation (James et al., 2014).

Education: Patient education is important in preventing hypertension and/or adhering to treatment once diagnosed. There are many patient education resources available for health-care professionals. The AHA has disease-specific pocket guides available for health-care professionals to give to their patients, or patients can be referred to their website. UpToDate also has patient education materials available on hypertension. Lifestyle modifications and medication adherence must be reinforced with every patient.

Clinicians should refer to evidence-based guidelines, such as those provided by ACC/AHA, when assessing and treating older adults with hypertension, but recommendations are not a substitute for clinical judgment for individual patients. Older adults often have comorbid conditions or lifestyle practices that may impact treatment outcomes. Balancing competing risk factors for hypertension-related risk of stroke, MI, and heart failure with treatment-related risks, such as fall-related fractures and adverse reactions to medications, is challenging and requires a patient-centered approach to treatment in older adults (Williamson et al., 2016).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
In patients older than 50 years, SBP more than 140 mm Hg is a much more important risk factor for cardiovascular disease than DBP.	A	Campbell-Scherer, Green, & Barry, 2017
Self-monitoring of BP at home or work is a practical way to assess differences between out of office and office readings.	B	Campbell-Scherer, Green, & Barry, 2017
Most patients with hypertension have essential hypertension, and more extensive testing for secondary hypertension is not necessary unless BP control is not achieved or if the patient has symptoms of catecholamine excess, bruits, or unprovoked hypokalemia.	B	Campbell-Scherer, Green, & Barry, 2017

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Lifestyle modification (smoking cessation, sodium restriction, exercise, weight control, etc.) lowers BP and should be encouraged in all patients with all stages of hypertension.	A	Campbell-Scherer, Green, & Barry, 2017
Thiazide diuretics (e.g., chlorthalidone and hydrochlorothiazide) are the drugs of first choice for most patients given their enormous cost-effectiveness advantage and lack of evidence of superiority of any other medications.	A	Campbell-Scherer, Green, & Barry, 2017
While diuretics are the cornerstone for treating hypertension, patients with diabetes, chronic kidney disease, heart failure, and CAD should have tailored treatment.	B	Campbell-Scherer, Green, & Barry, 2017
Reducing the number of daily doses increases adherence to BP lowering medication.	A	Campbell-Scherer, Green, & Barry, 2017

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

ISCHEMIC HEART DISEASE

Signal Symptoms: Angina pectoris is the initial manifestation of ischemic heart disease (IHD). Typical angina is substernal chest discomfort associated with physical exertion or emotional stress and relieved with rest or nitroglycerin (Fihn, 2012). Women report epigastric discomfort; nausea; radiation of discomfort to the arms, neck, and interscapular areas; dyspnea;and fatigue (Mieres, 2014).

Description: IHD is also referred to as coronary heart disease and occurs when there are one or more signs or symptoms of inadequate supply of blood to the myocardium. The most common problems resulting in IHD are related to obstruction of the epicardial coronary arteries due to atherosclerosis. Angina pectoris occurs when myocardial oxygen demand exceeds oxygen supply. Patients are considered stable when symptoms are manageable with either medical or revascularization therapy (Kannam, 2016). IHD is a chronic disorder that typically cycles through defined phases: asymptomatic, stable angina, accelerating angina, and acute coronary syndrome (ACS)/unstable angina/acute MI. The progression of the disease is not necessarily linear (Fihn, 2012).

Etiology: Atherosclerosis involving the coronary arteries is the common etiology of IHD. Evidence supports a multifactorial pathophysiology of coronary atherosclerosis that includes obstruction of the coronary arteries, reducing the quantity of oxygen-rich blood to the myocardium, and dysfunction of the coronary microvasculature and endothelium.

Occurrence: Cardiovascular disease is the number one cause of death in both men and women. Among persons 60 to 79 years of age, approximately 25% of men and 16% of woman have IHD. It is projected that in 2016 there will be approximately 660,000 Americans who have a new coronary event, either a first hospitalization for an MI or CHD death, and about 305,000 will have a recurrent event. Silent MIs are estimated to involve 160,000 additional persons (Mozzafarian, 2016). There are trends indicating that MI has declined significantly over time, including over the past decade. Mean predicted 10-year risk for CHD among adults aged 30 to 74 years has decreased from 7.2% during 1999 to 2000, to 6.5% during 2009 to 2010.

Age: The prevalence of CHD increases with age. The average age at first MI is 65.1 years for men and 72 years for women.

Gender: Men have CHD at a younger age. Women lag behind men by 10 years. In those 40 to 59 years of age, men 6.3%, women 5.6%; in those 60 to 79 years of age, men 19.9%, women 9.7%; and in those 80 years of age and older, men 32.2%, women 18.8%.

Ethnicity: In a meta-analysis involving 17 population-based cohorts worldwide, 60,211 participants without cardiovascular disease at baseline were studied. The 10-year event rate for formation of carotid intima-media thickness was 6.7% in Asians, 7.8% in Hispanics, 8.1% in Caucasians, and 9.2% in African Americans. Ethnicity appears to significantly modify the association between risk factors and atherosclerotic disease. The association between increase in age and atherosclerotic disease was weaker in African American and Hispanic populations. SBP elevations in Asians were strongly related to onset of disease. HDL cholesterol and smoking were associated less with disease onset in African Americans (Gijsberts, 2015).

Contributing Factors: In the absence of an established model to predict outcome, consider the following risks:

1. Sociodemographic: Age is the single, strongest determinant of survival. Sex and ethnicity have fewer effects on risk. Lower socioeconomic status is associated with worse outcome.
2. Cardiovascular: Smoking, hypertensions, dyslipidemia, family history of premature CAD, obesity, and sedentary lifestyle have the greatest risk of complications.
3. Coexisting medical conditions: Diabetes, chronic kidney disease, chronic pulmonary disease, and malignancy influence the prognosis.
4. Cardiovascular comorbidities: Heart failure, peripheral artery disease, and cerebrovascular disease are strong risk factors for mortality.
5. Psychosocial characteristics: Depression is strongly associated with worse survival rates. Poor social support, poverty, and stress are associated with poor outcome.
6. Health status: Patient's symptoms, functional capacity, and quality of life are associated with poor survival and incidence of ACS.
7. Anginal frequency: The more frequent the angina, the stronger prediction of ACS and hospitalization.
8. Cardiac disease severity: The degree and distribution of stenosis are meaningful predictors of outcome. (Fihn, 2012)

Signs and Symptoms: A detailed assessment of chest pain includes the quality, location, severity, and duration of pain; radiation; associated symptoms; and provoking and alleviating factors. The quality of chest pain associated with IHD is most often described as squeezing, gripping, suffocating, or heavy. Women and older adults can present with atypical symptoms such as nausea, vomiting, mid-epigastric discomfort, or sharp pain. Anginal pain typically lasts minutes; is located in the substernal area with pain that may radiate to the neck, jaw, epigastrium or arms; often is aggravated by exertion or emotional stress; and relieved with rest or nitroglycerin. Patients with angina must be categorized as stable or unstable. Unstable angina is defined as new onset increasing in frequency, intensity, duration, or at rest.

Physical examination may be normal or nonspecific. The patient may present with heart failure, valvular disease, or cardiomyopathy. Bruits over vascular areas and/or diminished pulses might suggest vascular disease. Elevated BP or xanthomas may indicate risk factors for IHD (Fihn, 2012).

Diagnostic Tests: A resting EKG is the initial test, followed by stress testing.

TEST	RESULTS INDICATING DISORDER
EKG	ST-segment depression or elevation or T-wave inversion in the absence of left ventricular hypertrophy supports the diagnosis of ischemia
	Q waves may be evidence of an old MI
Exercise stress testing	Reproduces ischemic symptoms
	Defects in myocardial perfusion with exercise
Exercise stress testing	Cardiac risk identified in patients with a decrease in the BP, S_3, rales, or prolonged downsloping ST-segment depression after exercise
Myocardial perfusion imaging	Perfusion defects Left ventricular dysfunction
Echocardiogram	Assesses the severity of left ventricular dysfunction
Stress echocardiogram	Multiple reversible wall motion abnormalities
Cardiac catheterization	For patients who fail pharmacological therapy, have had an MI, or have unstable angina, to determine the severity of disease

Differential Diagnosis: Aortic dissection, pericarditis, PE, pneumothorax, pneumonia, pleuritic, esophagitis, spasm, reflux, biliary colic, cholecystitis, choleolithiasis, peptic ulcer, pancreatitis, costochondritis, fibrositis, rib fracture, herpes zoster, anxiety, panic disorder, affective disorders (depression) (Fihn, 2012).

A resting EKG is recommended in patients when there is suspected cardiac cause of chest pain (Evidence Rating B). Those with evidence of prior MI, especially Q waves in multiple leads or an R wave in V1; persistent ST-T-wave inversions particularly in leads V1 to V3; left bundle-branch block or bifascicular block, second or third-degree atrioventricular block, or ventricular tachycardias or hypertrophy have a worse prognosis than those with a normal EKG (Fihn, 2012).

When the evaluation is complete, the determination of the probability of IHD is sufficient to recommend further testing. The typical angina pain and the older age of the patient have a high likelihood of IHD. Diabetes has the greatest influence on increasing the probability of IHD (Fihn, 2012).

For those who have suspected IHD and a prior MI, Q waves or symptoms of heart failure, complex ventricular arrhythmias, or an undiagnosed heart murmur, an echocardiography to evaluate the LV systolic and diastolic function and heart structures is recommended (Fihn, 2012).

If there is a probability that IHD may be present, stress testing is the most common noninvasive test used to diagnose IHD. Stress testing is used to induce ischemia either by exercise or pharmacological stress agents. However, the production of ischemia is dependent on the stress imposed and submaximal exercise testing can fail to produce ischemia. Coronary artery stenosis under 70% may be undetectable. For those able to exercise, standard exercise EKG testing is recommended. The patient must have an interpretable EKG and a moderate physical ability to exercise. For those unable to exercise, pharmacological stress with nuclear myocardial perfusion imaging (MPI) or echocardiography is recommended.

Those patients who have survived sudden cardiac death, life-threatening ventricular arrhythmias, or heart failure should undergo coronary angiography (Fihn, 2012). Angiography is useful in patients with unacceptable ischemic symptoms despite guideline-directed medical therapy (Fihn, 2014). However, it should not be offered to those who are unwilling or are not candidates for revascularization.

Treatment:

MEDICAL THERAPY TO PREVENT MI AND DEATH		
CLINICAL RECOMMENDATION	**EVIDENCE RATING**	**REFERENCES**
CHOLESTEROL MANAGEMENT		
Lifestyle modification including physical activity and weight management.	B	Fihn, 2012
Dietary therapy inclusive of reduction of saturate fats of <7% of total calories, trans fatty acids to <1% of total calories, and cholesterol to <200 mg/d.	B	Fihn, 2012
Moderate or high dose of a statin should be prescribed if not contraindicated.	A	Fihn, 2012
For those who do not tolerate statins, bile acid sequestrants, niacin or both are reasonable.	B	Fihn, 2012
BLOOD PRESSURE MANAGEMENT		
Lifestyle modification including weight control, physical activity, alcohol moderation, sodium reduction; increased consumption of fresh fruits, vegetables, and low-fat dairy products.	B	Fihn, 2012
Antihypertensive drug therapy should be started for BP 140/90 mmHg or higher.	A	Fihn, 2012
DIABETES MANAGEMENT		
For those with short duration of diabetes, a goal HbA1c of 7% or less.	B	Fihn, 2012
A HbA1c between 7% and 9% is reasonable for patients according to age, history of hypoglycemia, presence of microvascular or macrovascular complications, or presence of coexisting medical conditions.	C	Fihn, 2012
Therapy with rosiglitazone should not be initiated.	C	Fihn, 2012
PHYSICAL ACTIVITY		
30–60 minutes of moderate-intensity aerobic activity at least 5 days a week.	B	Fihn, 2012
Physical activity history and an exercise test to guide prognosis and prescription.	B	Fihn, 2012
Cardiac rehabilitation and physician-directed, home-based programs.	A	Fihn, 2012
Complementary resistance training at least 2 days per week.	C	Fihn, 2012
WEIGHT MANAGEMENT		
BMI and/or waist circumference should be assessed at every visit, and encouragement for weight maintenance or reduction to maintain or achieve BMI 18.5 to 24.9 and a waist circumference less than 40 inches in men and less than 35 inches in women.	B	Fihn, 2012
Initial goal of weight loss should be to reduce body weight by approximately 5% to 10% from baseline.	C	Fihn, 2012

Continued

SMOKING CESSATION		
Smoking cessation and avoidance of exposure to second hand smoke.	B	Fihn, 2012
MANAGEMENT OF PSYCHOLOGICAL FACTORS		
Screen for depression	B	Fihn, 2012
ALCOHOL CONSUMPTION		
Limit alcohol to 1 drink (4 ounces of wine, 12 ounces of beer, or 1 ounce of spirits) a day.	C	Fihn, 2012
ANTIPLATELET THERAPY		
ASA 75 to 162 mg daily in the absence of contraindication.	A	Fihn, 2012
Treatment with clopidogrel when ASA is contraindicated.	B	Fihn, 2012
Dipyridamole is not recommended.	B	Fihn, 2012
BETA BLOCKER		
Beta-blocker therapy should be started and continued for 3 years in all patients with normal LV function after MI or ACS.	B	Fihn, 2012
Beta-blocker therapy should be used in all patients with LV systolic dysfunction (EF ≤40%) with heart failure or prior MI (carvedilol, metoprolol succinate, or bisoprolol have been shown to reduce risk of death).	A	Fihn, 2012
Beta blockers considered for chronic therapy for all other patients with coronary or other vascular disease.	C	Fihn, 2012
RENIN-ANGIOTENSIN-ALDOSTERONE BLOCKER THERAPY		
ACEIs for patients who have hypertension, diabetes, LVEF ≤40%, or chronic kidney disease.	A	Fihn, 2012
ARBS for patients with hypertension, diabetes, LV systolic dysfunction, or chronic kidney disease who are intolerant of ACEIs.	A	Fihn, 2012
Influenza vaccine is recommended.	B	Fihn, 2012
Estrogen therapy; vitamin C, E, and beta-carotene, folate or vitamin B_6 and B_{12}, chelation therapy, garlic, coenzyme Q10, selenium or chromium.	No benefit	Fihn, 2012

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MEDICAL THERAPY FOR SYMPTOM RELIEF

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
ANTI-ISCHEMIC MEDICATIONS		
Beta blockers for initial therapy for symptom relief.	B	Fihn, 2012
CCBs or long-acting nitrates for symptom relief in those intolerant to beta blockers or contraindicated.	B	Fihn, 2012
Sublingual nitroglycerin spray for immediate relief of angina.	B	Fihn, 2012
Long-acting nondihydropyridine CCB instead of beta blockers as initial therapy.	B	Fihn, 2012
Ranolazine as a substitute for beta blockers if initial treatment with beta blockers leads to unacceptable side effects or is ineffective.	B	Fihn, 2012
Ranolazine in combination with beta blockers if beta blockers alone is not successful.	A	Fihn, 2012

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MEDICAL THERAPY FOR SYMPTOM RELIEF IN PATIENTS WITH REFRACTORY ANGINA

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Enhanced external counterpulsation (EECP)	B	Fihn, 2012
Spinal cord stimulation	C	Fihn, 2012
Transmyocardial revascularization	B	Fihn, 2012

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REVASCULARIZATION DECISION TO IMPROVE SURVIVAL

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Recommended in patients with unprotected left main or complex CAD.	C	Fihn, 2012
CABG to improve survival in patients with significant (≥50% diameter stenosis) left main.	B	Fihn, 2012
PCI is an alternative to CABG in stable patients with significant unprotected left main who may have low risk of PCI complications or have a significantly increased risk of adverse surgical outcomes.	B	Fihn, 2012
CABG is beneficial in patients with significant (≥70) stenosis in two major coronary arteries with severe or extensive myocardial ischemia.	B	Fihn, 2012

Continued

REVASCULARIZATION DECISION TO IMPROVE SURVIVAL		
CLINICAL RECOMMENDATION	**EVIDENCE RATING**	**REFERENCES**
CABG is reasonable in patients with mild to moderate LV systolic dysfunction (EF 35%–50%) and significant stenosis when viable myocardium is present.	B	Fihn, 2012
CABG is recommended over PCI to improve survival in patients with diabetes, multivessel CAD, particularly if a left internal mammary artery (LIMA) graft can be used on the left anterior descending artery (LAD)	B	Fihn, 2012

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Follow-Up: See patient at regular intervals for chronic health management; instruct in emergency measures and lifestyle management.

Sequelae: IHD remains a major public health problem with more than 10 million people with angina. Survival rates of patient have been improving; however, it is still the number one cause of death in men and women. Patients with symptoms of angina have MIs at a rate of 3% to 3.5% per year. Many people with IHD do not return to work after coronary revascularization and 15% to 20% rate their health as fair or poor. IHD continues to be associated with considerable morbidity despite the decrease in mortality rate. The costs of caring for patients with IHD are estimated at $156 billion in the United States for both direct and indirect costs (Fihn, 2012).

Prevention/Prophylaxis: Healthy, active lifestyle; control of hypertension, diabetes, cholesterol.

Referral: There are certain medical conditions, such as HIV and connective tissue disease, which may result in increased IHD. These patients need to be referred to Infectious Disease and Rheumatology to provide optimum management of these disorders. Ongoing reassessment of the patient's adherence to the therapy, adverse events, treatment goals, effectiveness of interventions, and risk factor reduction should be done. For those who have not met their goals, especially in the management of diabetes, depression, smoking cessation, and chronic kidney disease, referral to an appropriate specialist may be considered. If angina becomes unstable, the patient should know to seek emergency medical care (Fihn, 2012).

Education:

CLINICAL RECOMMENDATION	**EVIDENCE RATING**	**REFERENCES**
INDIVIDUALIZED EDUCATION PLAN INCLUDING:		
Importance of medication adherence for managing symptoms and slowing disease progression.	C	Fihn, 2012
Explanation of medication management and cardiovascular risk reduction strategies.	C	Fihn, 2012
Comprehensive review of therapeutic options.	B	Fihn, 2012
Description of appropriate levels of exercise.	C	Fihn, 2012
Information on recognition of worsening cardiovascular symptoms and appropriate actions.	C	Fihn, 2012
Adherence to diet that is low in saturated fat, cholesterol and transfat; high in fresh fruits, whole grains and vegetables; reduction in sodium intake.	B	Fihn, 2012
Common symptoms of stress and depression and techniques to minimize.	C	Fihn, 2012

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LUNG CANCER

Signal Symptoms: Cough, dyspnea, weight loss, anorexia, hemoptysis.

Description: Lung cancer is a malignant neoplasm originating in the parenchyma of the lung or airways (Midthun, 2015). Approximately 95% of all lung cancer is classified as non–small cell lung cancer (NSCLC) or small cell lung cancer (SCLC) (Latimer & Mott, 2015; Midthun, 2015). NSCLC comprises 80% of cancer diagnoses and includes adenocarcinoma, squamous cell cancer, and large cell cancer (Latimer & Mott, 2015; Midthun, 2015).

Etiology: Research has shown conclusively that approximately 90% of lung cancer cases are associated with tobacco smoking (CDC, 2016b; Mannino, 2016). Additional risk factors have also been identified. Radiation therapy can increase the risk of a second primary lung cancer in patients who have received this for other malignancies (Mannino, 2016). Environmental toxins such as radon, asbestos, and second-hand smoke are also risk factors (CDC, 2016b; Mannino, 2016; Samet, 2016). Patients with pre-existing diseases that involve the lungs, such as COPD, prior lung cancers, sarcoidosis, and scleroderma, are at increased risk for developing lung cancer. Studies have shown that patients with pulmonary fibrosis have a sevenfold increased risk for developing lung cancer (Tomassetti et al., 2015). Genetic predisposition to carcinoma of the lung continues to be studied, but there is growing evidence that genetics can affect the risk of developing lung cancer and the prognosis (Mannino, 2016; Midthun, 2015).

Occurrence: Lung cancer is the most common cause of cancer-associated deaths in the United States. It is the second most common cancer among men and women. Annually, more than 200,000 new cases are diagnosed (CDC, 2016a). Lung cancer is responsible for 26.5% of all cancer deaths (National Cancer Institute, 2016).

Age: Lung cancer can occur at any age, but there is a dramatic increase in new diagnosis after age 40 years. Most recent statistics show 32.4% diagnosed between 65 and 74 years, 27.4% between 75 and 84 years, and 9.3% among those more than 84 years old (National Cancer Institute, 2016).

Gender: More men have lung cancer than do women, but the incidence rate for women is rising.

Ethnicity: African American men have a higher incidence of lung cancer than Caucasian men. No significant difference exists between African American and Caucasian women.

Contributing Factors: The cumulative dose or pack-years for cigarette smoking is related directly to the risk of developing lung cancer. The more cigarettes smoked per day and the longer the individual has smoked, the higher the risk of developing lung cancer. The risk of lung cancer is 30 times higher in smokers than in nonsmokers. Cigar and pipe smokers and passive smokers have double the risk. The risks of developing NSCLC steadily decline for people who quit smoking and approach that of a nonsmoker after 15 years of abstinence. Individuals who have smoked for more than 20 years probably always will have a slightly increased risk of developing the disease, even after 15 years as a nonsmoker. There is no change in risk of SCLC with smoking cessation; early age at beginning to smoke is a significant risk factor. Asbestos exposure alone increases the risk of developing pulmonary carcinoma, but when combined with smoking, the risk increases to 100 times that of a nonsmoker. Other inhaled agents implicated in the development of the disease are radon, arsenic, nickel, chromates, halo ethers, alkylating compounds, and polycyclic aromatic hydrocarbons. Radon is a risk factor; air pollution also is thought to contribute to the development of lung cancer. Sarcoidosis carries a threefold increased risk; scleroderma increases risk of bronchoalveolar cancer.

Signs and Symptoms: Signs and symptoms of lung cancer are usually present only after the disease has progressed beyond the early stages. Routine screening for the disease with chest x-ray and/or sputum cytology is not recommended because it is neither specific nor sensitive (Deffebach & Humphrey, 2016). Low-dose chest CT scan can identify early-stage asymptomatic lung cancer. The USPSTF (2013) and National Comprehensive Cancer Network (NCCN) (2016a) recommend that adults age 55 to 80 years who have a 30 pack-year history and currently smoke or have quit within the past 15 years, undergo a low-dose CT scan to screen for lung cancer. Screening should be discontinued once the person has not smoked for 15 years or develops a health problem that can substantially limit life expectancy or undergoes curative surgery. Symptoms suggestive of lung cancer include a new or changing cough, hoarseness, hemoptysis, anorexia, cachexia, unexplained weight loss, dyspnea, hypoxia, wheezing, unresolving pneumonia, and chest wall pain (Latimer & Mott, 2015; Midthun, 2015). Patients initially may present with symptoms related to extrathoracic disease, including tracheal obstruction, esophageal obstruction with dysphagia, laryngeal nerve paralysis, phrenic nerve paralysis with elevated hemidiaphragm, sympathetic nerve paralysis or Horner's syndrome, pleural effusion owing to lymphatic obstruction, Pancoast's syndrome involving the eighth cervical and first and second thoracic nerves, or superior vena cava syndrome from vascular obstruction (Midthun, 2015).

Patients with pericardial and cardiac extension of tumor may have symptoms of arrhythmia, tamponade, or failure. The presenting illness in patients with lung cancer may be paraneoplastic syndromes, including hypercalcemia, hypophosphatemia, hyponatremia with syndrome of inappropriate antidiuretic hormone, and hypercoagulable states (Midthun, 2015). Occasionally, skeletal-connective syndromes, clubbing, and osteoarthropathy are initial symptoms of lung cancer.

Diagnostic Tests: After a history and physical examination suggestive of disease, the following tests are recommended (note that tissue biopsy results are required for diagnosis):

Noninvasive Testing:

- Chest x-ray: This may show nonspecific abnormalities, hilar masses, atelectasis, pleural effusions or masses, and infiltrates.

- CT scan, preferably with contrast: CT scan can assist in defining the characteristics, size, and location of the primary tumor, lymphadenopathy if present, and any abnormalities of the liver and adrenal glands (Latimer & Mott, 2015).
- Fluorodeoxyglucose positron emission tomography (FDG-PET): This is a test that measures the metabolic uptake of glucose by cells; lung cancer cells have a higher cellular glucose uptake than normal cells. It is helpful in diagnosing metastatic disease (Latimer & Mott, 2015).
- MRI of the brain, with contrast: An MRI can assist in identification of brain metastases (NCCN, 2016b; NCCN, 2017).

Invasive Testing:

- Sputum for cytology: This is positive in most centrally located tumors.
- Transthoracic needle aspiration is usually reserved for specific circumstances.
- Fiberoptic bronchoscopy with biopsies has a very high sensitivity rating for diagnosis.
- Bronchoscopy with CT-guided transbronchial needle aspiration has an important role in diagnosis and staging of lung cancer.
- Endoscopic ultrasound, endobronchial ultrasound, and mediastinoscopy also contribute to staging; mediastinoscopy is considered the gold standard for identifying mediastinal involvement (Latimer & Mott, 2015).
- If abnormalities suggest more extensive cancer, further testing will be conducted looking for extrathoracic, metastatic disease.

Staging: NSCLC staging was revised in 2009 and follows the tumor, node, metastasis (TNM) classification (Edge et al., 2009). Correct staging is essential at diagnosis to identify treatment options, prognosis, and potential for recurrence. An updated version of TNM staging is scheduled to be released in late 2017.

SCLC staging follows the two-stage Veterans Administration Lung Study Group staging system of limited disease (LD) and extensive disease (ED). Limited disease is confined to one hemithorax, the mediastinum, and the supraclavicular lymph nodes on the same side as the tumor. Extensive disease is any disease extending beyond this, including disease in the contralateral nodes or pleural effusion (Glisson & Byers, 2014).

Differential Diagnosis: TB, infectious granuloma, pneumonia, empyema, bronchiectasis, abscess, sarcoidosis, pneumonitis, and asbestosis all can mimic lung cancer.

Treatment: Surgical resection, radiation therapy, and chemotherapy are indicated for NSCLC; the stage of the disease, patient's functional status, and histology of the tumor determine the treatments. Recent advances in genotyping and targeting of altered signal mechanisms in lung cancer have allowed targeted treatment with immunotherapy, monoclonal antibodies, anti–vascular endothelial growth factor (anti-VEGF) agents, and other modalities (NCCN, 2016b, 2017). The EGFR gene is mutated in 15% to 30% of non-Asian patients and 30% to 60% of Asian patients with adenocarcinoma, which is associated with improved response to tyrosine kinase inhibitors (Midthun, 2015). Multimodal chemotherapy and radiation is the treatment of choice for limited stage SCLC; cisplatin and etoposide are the preferred agents (NCCN, 2016b). Prophylactic cranial irradiation is done in select patients. In some patients with very limited disease, surgical resection may be done (NCCN, 2016b). Treatment of extensive stage SCLC is chemotherapy alone with cisplatin or carboplatin, up to six cycles, then watchful waiting. Use of prophylactic cranial irradiation is recommended in patients who have a complete or partial response to initial therapy (NCCN, 2016b). Treatment after relapse is dependent on response to initial chemotherapy. If there is a positive response, repeating the treatment is done. If nonresponsive to initial chemotherapy, "salvage chemotherapy" with a nonplatinum, single agent such as topotecan, gemcitabine, or a taxane is attempted (NCCN, 2016b). Clinical trials are now underway with other chemotherapy agents. Treatment for older adults should be individualized. A comprehensive geriatric assessment is often conducted and used in determining treatment options. The Karnofsky Performance Scale is also used by some specialists (www.hospicepatients.org/karnofsky.html).

Follow-Up: Follow-up is individualized; generally, patients should be seen for follow-up every 3 to 4 months after treatment for lung cancer. During years 3 to 5, follow-up should be every 6 months. Yearly examinations are advised thereafter.

Sequelae: Disability and death are the chief sequelae of lung cancer. The 5-year survival rate is 17.7% (National Cancer Institute, 2016).

Prevention/Prophylaxis: Prevention of lung cancer should focus on smoking cessation. Ask about and record the tobacco use status of every patient, advise patients who smoke to quit, and offer smoking cessation treatment at every office visit. Screening for lung cancer in asymptomatic patients is not advised. Screening in high-risk patients has not demonstrated any decrease in mortality; newer methodologies studying biomarkers have not been proved to date. The National Comprehensive Cancer Network (NCCN, 2016a) and the USPSTF (2013) have issued guidelines endorsing screening with low-dose helical CT scan in high-risk patients. There is currently no recommended chemoprevention for lung cancer (Keith & Miller, 2014).

Referral: On suspecting lung cancer, refer the patient to a pulmonary specialist. At a later stage, referral to palliative care or hospice may be indicated. Give patient information on the American Lung Association Information Line (1-800-LUNGUSA). Referral to a mental health specialist is indicated in severe depression unresponsive to treatment.

Education: Many smoking cessation programs are available. Examples include the National Cancer Institute Tobacco Quitline, the American Academy of Family Physicians Stop Smoking Kit, various pamphlets from the American Cancer Society, and A Healthy Beginning Counseling Kit from the American Lung Association; the Web site www.smokefree.gov is another resource. The primary health-care provider can assess the smoker's motivation to quit and offer brief motivational interventions at each visit. Provide information about support groups and community programs available to help with smoking cessation.

Once the patient is diagnosed with lung cancer, education about type of cancer, treatment options, and prognosis is very important. This is typically done by the oncology team, but primary care providers can monitor for patient questions and communicate this to the specialty team.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Low-dose CT scan should be used to screen high risk individuals for lung cancer.	B	USPSTF, 2013 NCCN, 2016a
Surgery is the treatment of choice for patients with local or only locally advanced non-small cell carcinoma (Stage I through Stage IIIA).	C	Latimer & Mott, 2015 NCCN, 2017
Treatment for unresectable non-small cell carcinoma may involve radiotherapy and chemotherapy.	C	NCCN, 2017
Chemotherapy, combined with radiotherapy in limited disease, is the mainstay of treatment for small cell carcinoma.	B	NCCN, 2016b
Both radiotherapy and chemotherapy are used in treatment of superior vena cava (SVC) syndrome because of non-small cell or small cell lung cancer.	B	NCCN, 2016b NCCN, 2017
Early palliative care results in improved quality of life and a decreased incidence of depression in patients with newly diagnosed non-small cell lung cancer.	B	Latimer & Mott, 2015

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MYOCARDIAL INFARCTION

Signal Symptoms: Typical symptom is prolonged chest pain (more than 20 minutes' duration); atypical symptoms include shortness of breath, neurological symptoms (confusion, weakness), and worsening of heart failure.

Description: Reeder and Kennedy (2016) define MI as a pathological clinical episode, triggered by myocardial ischemia, manifesting in myocardial injury or necrosis. MIs can be classified as acute ST elevation MI (STEMI) or non–ST elevation MI (NSTEMI).

Etiology: The hallmark of ACS is the sudden imbalance between myocardial oxygen consumption (MVO2) and demand, which is usually the result of coronary artery obstruction. This imbalance may also be caused by other conditions, including excessive myocardial oxygen demand in the setting of a stable flow-limiting lesion; acute coronary insufficiency due to other causes such as vasospastic angina, coronary embolism, coronary arteritis; noncoronary causes of myocardial oxygen supply-demand mismatch include hypotension, severe anemia, hypertension, tachycardia, hypertrophic cardiomyopathy, severe aortic stenosis; nonischemic myocardial injury and multifactorial causes that are not mutually exclusive (Amsterdam, 2014, p. 16).

Atherosclerotic plaque formation has been noted in persons younger than 20 years of age. Gradually, this atherogenic plaque builds up inside the intimal linings of the coronary artery(ies), resulting in both hardening of the arterial walls and narrowing of the coronary vessels. This atherosclerotic plaque contains lipid (cholesterol)–rich content, covered by a fibrous cap, which can ulcerate or fissure. Rupture of this vulnerable plaque can result in thrombosis, which partially or totally occludes the lumen of the affected artery(ies), either at the location where the thrombus formed or further downstream where a smaller artery could be blocked. The results are essentially the same—an abrupt decrease in coronary blood flow to the myocardium, termed *myocardial ischemia.* Prolonged myocardial ischemia can lead to tissue death and MI. Although there are other causes of MI due to reduced myocardial blood flow, including hypotension and prolonged coronary vasospasm, or severe hyperthyroidism (which can increase the heart's demand for oxygen), the most frequent cause of MI is due to atherosclerosis. Another factor that plays an important role in ACS is platelet activation and aggregation (Amsterdam, 2014; Riffo, 2015). Damaged blood vessels result in exposed collagen fiber in the basement membrane. Platelets subsequently adhere to this collagen

fiber and become activated. On activation, platelets then release chemicals, including adenosine diphosphate (ADP) and thromboxane (a vasoconstrictor), which causes additional platelet aggregation. A platelet plug (or clot) is formed, which decreases blood flow to the blood vessel (Amsterdam, 2014; Riffo, 2015).

Occurrence: When looking at all-cause mortality, coronary disease is the most frequent cause of death in the United States. In persons age 55 to 74 years in the United States, coronary disease occurs more frequently in men, with a median age of 68 years (Amsterdam, 2014). Women of all ages have a greater risk of longer hospitalizations and complications (Amsterdam, 2014). In persons older than 75 years, coronary disease contributes to significant morbidities and mortality (Amsterdam, 2014). In persons age 75 years or older who present with MI, more than 60% have NSTEMI with associated in-hospital mortality, accounting for approximately 80% of all in-patient hospital deaths (Kyriakides, Kourouklis, & Kontaras, 2007; Tullman, Haugh, Dracup, & Bourguignon, 2007). In addition, persons older than 65 years with MI are associated with higher post-discharge mortality, theoretically because of the higher incidence of diffuse coronary disease and existing poor myocardial perfusion. Data published by the CDC (2011) on coronary disease and MI found that males overall had a significantly higher myocardial rate than females; however, with successive age groupings, the prevalence of MIs significantly increased. When identifying level of education with MI and coronary disease, persons who had less than a high school diploma experienced almost twice the occurrences of these conditions when compared with those who had college education.

Age: In 2010, the prevalence of CHD was greatest among persons aged 65 years and older (19.8%). Postmortem studies show that coronary atherosclerosis is widespread, even among asymptomatic adults. Aging itself may be a risk factor for MI in both men and women (CDC, 2011).

Gender: Before menopause, due to estrogenic cardioprotective effect, women have a lower occurrence of MI than men. Coronary disease is much more prevalent in men than in women up to age 74 years, after which the occurrence increases steeply with age in both genders. On average, women develop heart disease 10 years later than men and suffer MIs and sudden death 20 years later than men. Thus, by age 75 years, postmenopausal women have an equal risk to men of coronary disease and MI.

Ethnicity: The incidences and occurrences of patients having MIs and CHD by race and ethnicity have been reported by the CDC (2011). Specific groups were identified at increased risk, including those persons who are multiracial, Native Americans, and Alaskan natives, who have substantially higher prevalence of MI than non-Hispanic Caucasians or African Amerians (CDC, 2011). Overall, in patients with NSTEMI, African American patients (when compared to Caucasians) were younger, with concomitant comorbidities including hypertension, renal insufficiency, diabetes, and heart failure (CDC, 2011).

Contributing Factors: Risk factors for heart disease and MI in elderly individuals are essentially the same as in the younger population. Risk factors include hypertension, hyperlipidemia, diabetes, insulin resistance, physical inactivity and sedentary lifestyle, overweight or obesity, family history of heart disease, poor diet, sleep apnea, alcohol, and stress (www.nhlbi.nih.gov/health/health-topics/topics/hd). Cigarette smoking is associated with new coronary events for elderly men and women. The risks of MI drop rapidly after smoking cessation and, at 5 years, the risk for new coronary events is similar to that of a nonsmoker (Mendis, Puska, & Norrving, 2011).

Aging alters the cardiovascular system in ways that reduce cardiac reserve and efficiency, thus compromising the ability to respond to stress or illness. The cardiovascular system becomes less compliant as diastolic filling of the ventricles declines and afterload increases secondary to increased stiffness in the ascending aorta. The result is moderate left ventricular hypertrophy, which creates a more precarious balance between myocardial oxygen supply and demand. Other changes in the elderly cardiovascular system include decreased responsiveness to beta-adrenergic stimulation, decreased baroreceptor sensitivity, and an increased dependence on a higher end-diastolic volume to maintain cardiac output (NIH, 2015).

Signs and Symptoms: With advancing age, the presentation of acute MI will be less likely to include the classic symptoms of crushing substernal chest pain, nausea, vomiting, and diaphoresis. Rather, atypical symptoms such as a vague ache or discomfort may be present. Elderly persons may not recognize that throat, shoulder, arm, jaw, or abdominal pain may be referred cardiac pain or angina equivalent. Dyspnea is the second most common symptom of MI in both younger and older populations. For patients 85 years and older, syncope, acute confusion, or stroke may be the only presenting symptom. Some elderly patients may present only with faintness, weakness, giddiness, or restlessness (Domino, 2016).

On physical examination, the patient may be anxious and weak and may appear cyanotic. Arrhythmias may be noted on the EKG. The skin may be diaphoretic, cold, and clammy. Thrills, heaves, and an abnormal PMI may be palpated. Peripheral pulses may be irregular, slow, fast, or thready. Auscultation may reveal an S_3 or S_4, pericardial friction rub, murmurs, or crackles. The physical examination must focus on ruling out diagnoses other than MI.

Diagnostic Tests: Along with a chest x-ray examination, CBC, clotting profile, electrolytes, and cardiac biomarkers including troponin, evaluation for acute MI includes serial 12-lead EKGs (Amsterdam, 2014; Domino, 2016). Changes on the EKG indicative of STEMI include ST-segment elevation in leads facing the infarction. ST-segment depression is sometimes present. With the increased incidence of NSTEMI, the presence of a single normal EKG does not rule out MI, and therefore serial EKGs are necessary. However, EKG findings may be nondiagnostic or difficult to interpret in older adults because of possible abnormalities, such as left bundle branch block, left ventricular hypertrophy, and previously unrecognized MIs.

In older adults, the cardiac enzyme profile may be atypical. Creatine kinase (CK), an enzyme found in myocardial cells, is released into the bloodstream when cells are damaged, approximately 6 to 8 hours after the onset of pain. Because the baseline CK in elderly patients may be significantly

lower than normal, even those with MI may not develop CK levels high enough to be interpreted as abnormal, owing to decreased muscle mass. CK-MB, an isoenzyme of CK, is released on the death of myocardial cells and not during ischemia. The presence of CK-MB is also found in skeletal muscle, which can result in false-positive results from noncardiac conditions such as renal failure, head or chest trauma, PE, acute and chronic muscular disorders, hypothyroidism, or hyperventilation. Therefore, CK-MB measurements do not provide additional information to the algorithms of STEMI and are not preferred over cardiac troponins (Rains, 2014). Cardiac troponin T (cTnT) is a cardiac-specific marker for MI, with rises in concentration levels soon after chest symptoms begin (Rains, 2014). Although elevations may not occur for 6 to 12 hours after onset of symptoms, in approximately 80% of patients use of cTnT alone can result in a definitive diagnosis of STEMI. Lactate dehydrogenase levels are occasionally useful in patients with delayed presentation because this enzyme remains elevated for several days. Other tests for MI may include echocardiography or stress thallium scans (Rains, 2014).

Differential Diagnosis: The pain from MI can be similar to that of acute pericarditis, PE, acute aortic dissection, or costochondritis. Many conditions can present as cardiac disease in the elderly, including cor pulmonale, pneumonia, esophageal spasm, GERD, hiatal hernia, gallbladder disease, osteoarthritis of the spine, inflammation at the chondrocostal junction, muscle injury, and panic disorder (McPhee & Papadakis, 2016).

Treatment: The ACC and AHA have examined pharmacological and interventional treatments for acute MI and established guidelines for care. Nitrates, the cornerstone of therapy for ischemic pain, reduce preload and improve coronary perfusion. Chest pain should be treated with sublingual nitroglycerin, repeated three times, 5 minutes apart, unless the patient is hypotensive, has right ventricular infarction, or has aortic stenosis (Amsterdam et al., 2014). IV nitrates have been shown to limit infarct size and reduce pain, complications, and mortality. IV nitroglycerin is started at 5 mcg/min on an infusion pump and titrated to desired effect. Frequent monitoring is required because harmful effects can be seen if the SBP drops below 90 mm Hg. Morphine sulfate, 2 to 4 mg IV, repeated at 5- to 15-minute intervals, is also effective for pain relief and can be given with anxiolytics (Amsterdam et al., 2014). Supplemental oxygen (2 to 6 L/min nasal cannula) should be given if oxygen saturation is less than 94% or arterial oxygen saturations are low.

Thrombolytics: Reperfusion in patients who present to hospitals with percutaneous coronary angioplasty and intervention (PCI) capability has become the standard of care (Amsterdam et al., 2014). In hospitals without PCI capability and where patients cannot be transferred to a hospital with PCI capability within 90 minutes, treatment with fibrinolytic (thrombolytic) therapy initiated within 30 minutes of presentation should be performed unless contraindicated (HAA, 2014). The use of thrombolytics in STEMI patients has been shown to decrease mortality by up to 30% (Amsterdam, 2014; Morse et al., 2009). Thrombolytic agents include tissue-type plasminogen (tPA) and streptokinase (SK). Indications for thrombolysis are based on the existence of chest pain and specific EKG changes. Studies show that this therapy is frequently omitted for elderly patients. Reasons for this include frequent delay in seeking medical care, atypical presentation of MI, and a higher prevalence of NSTEMIs. Fear of hemorrhage following use of thrombolytics must be weighed against the proven gains in survival for all age groups, especially for those presenting early with MI. A variety of studies suggest that SK may be as beneficial as tPA in treating the elderly MI patient, which is considerably less expensive (Morse et al., 2009).

Aspirin: Aspirin therapy in post-PCI STEMI patients without demonstrated aspirin resistance should be initiated at 162 to 325 mg daily and, depending on the type of stent implanted, the recommended maintenance dose to be used with Ticagrelor is 81 mg (Amsterdam, 2014). In patients at risk for increased bleeding after stent implantation, lower-dose aspirin is reasonable. Aspirin (non-enteric coated) should be given as soon as possible if there is no obvious contraindication. Patients taking nonselective NSAIDs (excluding aspirin) and cyclooxygenase-2 inhibitors should have these agents discontinued at the time of presentation with STEMI and not readministered during the course of hospitalization due to increased risk of mortality, reinfarction, myocardial rupture, heart failure, shock, or hypertension (Amsterdam et al., 2014).

Thienopyridines: Concurrent use of dual antiplatelet therapy (aspirin and thienopyridines) in MI patients (STEMI or NSTEMI) following PCI with stent implantation is the gold standard for secondary prevention of subsequent cardiovascular events. Use of thienopyridines, including clopidogrel or prasugrel, is important to decrease the risk of death and stroke, and to prevent reinfarction or in-stent thrombosis (Amsterdam et al., 2014). Thienopyridines should be initiated as a loading dose early, either before or at the time of primary or nonprimary PCI, and continued for a minimum of 12 months after bare metal stent placement in patients who are not at high risk for bleeding (Amsterdam et al., 2014). In patients for whom nonemergent coronary artery bypass is planned, thienopyridine therapy should be discontinued for a minimum of 5 days for those patients receiving clopidogrel and 7 days for those patients receiving prasugrel (Amsterdam et al., 2014).

Parenteral anticoagulants are recommended in all patients with acute coronary syndrome or myocardial patients regardless of whether or not reperfusion therapy was received. Low-molecular-weight heparin (LMWH), unfractionated heparin, bivalrudin, enoxaparin, or fondaparinux is recommended in patients undergoing PCI (Amsterdam et al., 2014). If coronary artery bypass grafting is planned, anticoagulants should be withheld for 5 to 7 days before the procedure due to risks of excessive bleeding.

For patients not receiving thrombolytics, low-dose subcutaneous heparin or LMWH is indicated for prophylaxis of deep venous thrombosis. Older adult patients with large anterior MIs and heart failure, as well as those with documented left ventricular thrombus, should receive full heparin anticoagulation.

Angiotensin-Converting Enzyme Inhibitors: ACEIs reduce mortality in post-MI patients. ACEIs are recommended and should be continued indefinitely for every patient recovering from an

STEMI and for those with MI with an LVEF less than 40% and no obvious contraindication(s) to their use. For patients with preserved LVEF with hypertension, chronic kidney diseases, or diabetes, ACEIs are recommended unless contraindicated (Amsterdam et al., 2014).

Angiotensin Receptor Blockers: ARBs are recommended in those patients who are intolerant of ACEIs, who have heart failure with LVEF of 40% or less, or who have hypertension.

Aldosterone Blockade: Aldosterone blockade is recommended for post-MI patients without severe renal dysfunction or hyperkalemia, with heart failure, with LVEF of 40% or less, or with diabetes who are already on a therapeutic dose of ACEI and beta blockade (Amsterdam et al., 2014).

Beta Blockers: Beta blockers have been shown to limit infarct size, decrease chest pain, improve prognosis, and reverse cardiovascular remodeling. They are generally well tolerated in patients ages 65 to 75 years. "The use of B-Blockers in this population should be tailored based on other concomitant cardiovascular conditions and completeness of revascularization" (Motivala, 2016). Conditions that contraindicate the use of beta blockers in the elderly include bronchospastic lung disease, marked bradycardia, hypotension, depression, acute heart failure, and diabetes. "B-Blockers have consistently been shown to improve outcomes in a variety of cardiovascular settings and therefore remain widely used" (Motivala, 2016).

In acute MI, metoprolol or atenolol is given 2.5 to 5 mg IV every 5 minutes, for a total of 15 mg and 10 mg, respectively. Beta blockers should be initiated in the emergency department before PCI and within 24 hours in patients presenting with STEMI, MI, acute coronary syndrome, or left ventricular dysfunction with or without heart failure, unless contraindicated. Beta blocker use in this population has increased over time (Motivala, 2016). Contraindications to beta blocker therapy include heart failure, evidence for a low cardiac output state, or risk for cardiogenic shock (Amsterdam et al., 2014).

Statins and Lipid-Lowering Therapy: A lipid profile that includes total cholesterol, LDL, HDL, and triglycerides should be performed and assessed on all acute coronary syndrome or MI patients within 24 hours of admission. Lipid-lowering therapy is recommended before discharge, with the LDL goal to be less than 100 mg/dL, preferably with further LDL reduction to less than 70 mg/dL (Amsterdam et al., 2014). Dietary therapy is recommended for all patients, including consumption of omega-3 fatty acids, weight management, and physical activity.

CCBs have limited use in STEMI and NSTEMI patients. Hypotension-induced tachycardia has been associated with the use of short-acting CCBs. Long-acting CCBs should be used sparingly, if needed, with additional precautions in those patients already taking digoxin or beta blockers (Kyriakides et al., 2007).

In terms of interventional management of elderly MI patients, guidelines by both the AHA and ACC have established "door to needle" time of less than 30 minutes and "door to balloon" time within 90 minutes of initial presentation to the hospital for the best possible outcomes (Amsterdam et al., 2014). Percutaneous transluminal coronary angioplasty (PTCA) with PCI may be a valuable alternative in those for whom thrombolytics are contraindicated. Coronary artery bypass graft (CABG) is preferred for patients with left main stenosis and those with moderately severe left ventricular depression. Emergency PTCA or CABG in older adult patients presenting with cardiogenic shock or heart failure is associated with high morbidity and mortality.

Glucose Control: Hyperglycemia (defined as blood sugar greater than 180 mg/dL) in patients with STEMI has been associated with adverse outcomes, including increased morbidity and mortality, as well as prolonged hospitalizations (Amsterdam et al., 2014). Maintaining glucose control may decrease the inflammatory response and improve LVEF (Amsterdam et al., 2014).

Follow-Up: Risk stratification for future cardiac events following stabilization from MI is necessary. Tests to evaluate for ischemia or myocardium at risk include low-level exercise test, nuclear perfusion scanning, and dobutamine echocardiography. Left ventricular function can be determined by echocardiography or radionuclide ventriculogram. Routine coronary angiography is not recommended for all MI patients, but should be performed on patients with demonstrable ischemia. Patients at high risk for sudden death from arrhythmias should undergo electrophysiology (EP) evaluation.

Sequelae: Increasing age is associated with more complications after MI, including heart failure, arrhythmias, pulmonary edema, cardiogenic shock, cardiac rupture, and death.

Prevention/Prophylaxis: Secondary prevention in the older adult MI patient includes use of aspirin, beta blockers, and ACEIs, unless there are absolute contraindications to these agents. Lipid-lowering therapy is also beneficial. Reduction of risk factors is necessary. Cardiac rehabilitation can be as beneficial in older MI patients as in younger ones. Emotional support remains important.

Referral: Suspicion of acute MI should prompt transfer of the patient to an environment equipped with cardiac monitoring and the ability to administer advanced cardiac life support. As hospitalization continues, consultation with a variety of disciplines, such as surgery, social work, and physical therapy, may be necessary.

Education: Individualized teaching following MI for each patient and family should be provided, using age-specific teaching methods. Teach the basic definitions of CAD, angina, and MI. Patients need information on the healing process following MI, when to return to work or resume normal daily activities, and when to resume sexual relations. Give an exercise prescription. Involve patients and families in a discussion of the psychological adjustment following MI. Encourage risk factor reduction. Health-care practitioners should work with patients to set goals and design plans. Give patients information on community resources, such as the AHA or support groups in their area. Smoking cessation should be strongly encouraged and pharmacological and nonpharmacological therapies discussed. Medication teaching and review are very important. Health-care providers should be aware of all medications prescribed for the patient and any OTC medications, including vitamins, minerals, and supplements. Patients should bring in all of their medications at each visit. Teach patients about the desired effects and common

side effects of their medications. Emphasize the importance of taking aspirin and anticoagulants as directed to prevent in-stent thrombosis. Review what to do if medication cannot be taken or obtained. Discuss the use of nitrates for any chest pain, while avoiding the usage of nitrates in patients taking medications such as sildenafil, tadalifil, or vardenafil, due to sudden drops in BP, resulting in hypotension.

Teach older adult patients about altered pain perception that can occur with age and with diseases such as diabetes. Teach patients and their families about warning signs such as chest pain or pressure, shortness of breath, indigestion, choking, sweating, dizziness, palpitations, severe weakness, and loss of consciousness. Establish a clear plan for obtaining prompt medical attention.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The presence of elevated cardiac enzymes associated with clinical or EKG evidence of ischemia is diagnostic for MI.	A	Amsterdam et al., 2014
Treat all patients with oxygen, pain control medications, and antiplatelet therapy including aspirin and heparin (or enoxaparin).	A	Amsterdam et al., 2014
Nitroglycerin may be given to relieve ischemic chest pain, except in patients with hypotension and right ventricular infarction.	A	Amsterdam et al., 2014
Give thrombolysis within 30 minutes when PCI is not available or when >90 minutes of delay is expected in door to balloon time.	A	Amsterdam et al., 2014
Treat most patients with aspirin or clopidogrel, beta blockers, and statins (LDL target of 70–100 mg/dL) if no contraindications are noted.	A	Amsterdam et al., 2014

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

PNEUMONIA

Signal Symptoms: Chills or hyperthermia, fever, sweating, chest discomfort or dyspnea, a new cough with or without sputum production or with chronic cough, a change in sputum color. Nonspecific symptoms include fatigue, abdominal pain, headache, myalgias, loss of appetite, and worsening of other chronic illnesses. In older adults, initial symptoms may not include a cough or chest pain but fever and confusion. Atypical pneumonia symptoms generally have a more gradual onset and may include a dry, hacking cough with more constitutional symptoms such as headache, coryza, shaking chills, myalgias, and sore throat (Wunderink & Waterer, 2014; Musher & Thorner, 2014).

Description: Community-acquired pneumonia (CAP) is an acute lower respiratory tract infection of the lung parenchyma in which consolidation occurs as exudate fills the alveoli. CAP is usually infectious, can be bacterial or viral, and refers to pneumonia that begins outside of the hospital or is diagnosed within 48 hours of admission without recent (14 day) residence in a long-term care facility. Health-care–acquired pneumonia (HAP) is acquired from a hospital or nursing home (Donovan, 2015).

Ventilator-associated pneumonia (VAP) is the most frequently acquired infection in the ICU in mechanically ventilated patients, with the highest risk occurring in the first 5 days post intubation. Multiple factors contribute to VAP, including micro aspiration, bacterial laden tube, secretions around the cuff, impairment of clearance of secretions within the airway, and pathogenic material from surrounding structures (mouth, sinuses, stomach) (Kalanuria, Zai, & Mirski, 2014).

Etiology: The most common cause of bacterial CAP in patients who were admitted to the hospital was *streptococcus pneumoniae*, followed by *mycoplasma pneumoniae, Staphylococcus aureus, legionella pneumophilia*, and *enterobacteriacaea*. Pneumococcal pneumonia was found in only 5% of CAP inpatient cases (CDC, 2016; Jain et al., 2015).

HAP in older patients is caused by a variety of organisms (*Pseudomonas aeruginosa, Klebsiella pneumoniae, S. pneumoniae, Escherichia coli, Sarratia proteus,* and *Enterobacter)* and it is common to have a mixture of multiple organisms. In nursing home residents *S. Aureus*, *MRSA, S. pneumoniae*, and *H. influenza* are reported more often as the causative organism (Marrie & File, 2016). The most common pathogens found in VAP differ depending on the length or ventilation (Kalanuria et al., 2014).

Occurrence: Pneumonia and influenza together are the eighth leading cause of death in the United States, and CAP is the leading cause of death due to infectious disease. The

annual occurrence rate of CAP in the United States is about 5.6 to 6.11 cases per 1,000 persons, with the highest rates among adults 65 years or older (Marrie & File, 2016). Estimated annual deaths from CAP is over 55,000 (Kochanek, Murphy, Xu & Tejada-Vera, 2016). The incidence of CAP for each pathogen increased with age. In the EPIC study (CDC, 2016), 93% had radiographic evidence of pneumonia and yet a pathogen was identified in only 38%. About 23% of pathogens were viral and 11% were bacterial. Rhinovirus, influenza A or B, and streptococcus pneumonia were the most common pathogens identified in the study. Persons 65 to 79 years old had a rate of hospitalization approximately nine times that of those under 50 years, and persons 80 years and older were 25 times more likely to be hospitalized (Jain et al., 2015).

The mortality rate for CAP is dependent on the treating site but generally is less than 3% in the outpatient setting, 5% to 10% in hospitalized patients, and as high as 25% in intubated ICU patients. In ICU patients on vasopressors, the mortality rate may be as high as 50% (Restrepoa, Faverioa, & Anzueto, 2013).

Age: Pneumonia is the most common cause of death from infectious disease in older patients. Elderly persons experience a more significant increase in risk than that of younger adults from dying of pneumonia (Jain et al., 2015; Restrepoa et al., 2013).

Gender: More men than women are diagnosed with pneumonia.

Ethnicity: Not significant.

Contributing Factors: Comorbidities, influenza, chronic pulmonary conditions, smoking, alcoholism, malnutrition, colonization of the oropharynx by gram-negative bacteria, institutional setting, decline in immune function, hospitalization, use of sedating medications, recent surgery or trauma, and diminished cough reflex may contribute to development of pneumonia (Marrie & File, 2016; Restrepoa et al., 2013; Torres, Peetermans, Viegi, & Blasi, 2013).

The main risk factor for HAP is mechanical ventilation. Other significant risk factors according to File (2016b, p. 1) include:

- Age 70 years or older
- Chronic lung disease
- Depressed consciousness
- Aspiration
- Chest surgery
- The presence of an intracranial pressure monitor or nasogastric tube
- Agents that increase gastric pH (H_2 blockers, antacids, proton pump inhibitors)
- Transport from the ICU for diagnostic or therapeutic procedures
- Previous antibiotic exposure, particularly to third-generation cephalosporins
- Reintubation or prolonged intubation
- Hospitalization during the fall or winter season
- Mechanical ventilation for acute respiratory distress syndrome
- Frequent ventilator circuit changes
- Paralytic agents
- Underlying illness

TABLE 8-5 CURB-65 Criteria for Evaluation of Pneumonia Severity and Treatment Site

CLINICAL FACTOR	POINTS	SCORING
Confusion (new disorientation)	1	Scoring 0-1: Low risk; consider outpatient treatment 2: Brief hospitalization or closely monitored outpatient treatment ≥3: Severe, hospitalize and possible ICU
B**U**N >19 ng/dL	1	
Respiratory rate ≥30 breaths/min	1	
BP: Systolic <90 mm Hg OR Diastolic <60 mm Hg	1	

Adapted from Dunphy, Winland-Brown, Porter, & Thomas. (2015). *Primary care: The art and science of advanced practice nursing* (4th ed.). Philadelphia: F.A. Davis.

The most common risk factors in pneumonia-related deaths are respiratory disease, cardiovascular disease, and cancer. Other common risk factors for increased mortality include older age, severity of pneumonia, and either existing or new onset of comorbidities such as cardiovascular, respiratory, neurological, diabetes, COPD, and acute kidney injury (Holter et al., 2016).

The CURB-65 score (Table 8-5) and the CAP index are used to stratify risk in patients with CAP. The five indicators that comprise the CURB score include elevated BUN levels, low DBP, a rapid respiratory rate, confusion, and age greater than 65 years. There is a 21-fold increase in mortality if patients have two or more of these symptoms. The pneumonia severity index (PSI) was designed to predict the 30-day mortality rate of patients with CAP and assigns points to contributing factors such as comorbidities, age, gender, nursing home residence, physical examination, and laboratory and radiographic findings to determine which patients may need to be hospitalized (File, 2016d).

Signs and symptoms associated with pneumonia-related mortality include dyspnea, chills, altered mental status, tachypnea, hypotension, hypothermia, and hyperthermia. Laboratory abnormalities associated with increased mortality are hyponatremia, hyperglycemia, abnormal liver function studies, hypoalbuminemia, azotemia, hypoxemia, and azotemia. Risk factors for drug-resistant *S. pneumoniae* (DRSP) include age of more than 65 years, alcoholism, antimicrobial therapy within the past 3 months, exposure to children in day care, multiple comorbidities, or immunocompromise (Musher & Thorner, 2014; Wonderink & Warterer, 2014).

Risk factors for VAP include the insertion of an endotracheal tube and device-related issues, immunocompromise, adult respiratory distress, COPD, patient positioning, medications, and personnel related (breach in technique) (Kalanuria et al., 2014).

Signs and Symptoms: Typical symptoms include fever, chills, cough, and rusty or thick sputum, with associated gastrointestinal upset or anorexia, malaise, and diaphoresis; pleuritic chest pain may also be present. These symptoms are frequently absent in the frail and elderly, leading to a lag time before diagnosis. In the older patient, mental status changes (i.e., confusion), new onset or increased frequency of falls, increased respiratory rate, hypotension, anorexia, a marked functional decline, and new onset of urinary incontinence

are typical symptoms (High, 2016). Physical examination of the chest reveals crackles that do not clear with cough or deep breathing. Increased respiratory rate (more than 24 breaths/min) is typical. A fever of 100°F with tachycardia may be present and signs of dehydration may occur. The patient may appear anxious, restless, or withdrawn (Marrie & Toumanen, 2016; Musher & Thorner, 2014; Wonderink & Warterer, 2014).

Symptoms of VAP include new or increasing pulmonary infiltrate, decreased oxygenation with an increased respiratory rate, and a decreased tidal volume. Fever and increased purulent secretions are common, and patients usually have diffuse rhonchi with an inability to mobilize secretions and may have either leukocytosis or leukopenia. Crackles, diminished breath sounds, and tachypnea, as well as wheezing and hemoptysis, are common findings (Kalanuria et al., 2014; Kollef, 2016).

Diagnostic Tests: The diagnosis of CAP may be difficult in patients with underlying chronic comorbidities, especially those with pulmonary fibrosis, lung cancer or other chronic lung diseases, and heart failure. Recommendations for diagnostic testing continue to be controversial, however, the chest x-ray is considered the gold standard for the diagnosis of pneumonia (File, 2006d; Mandell et al., 2007). Other routine testing for patients treated on an outpatient basis is not recommended. Newer recommendations include testing for changes in C-reactive protein (CRP) and/or urine specific antigen when there is a question about when, or if, to start antibiotic therapy (Bel et al., 2015; CDC, 2016; File, 2016a; National Institute for Health and Care Excellence [NICE], 2014). CT scan of the chest is often ordered and is more accurate than a chest x-ray, especially in the emergency department setting. Studies have shown that ultrasound of the chest has better sensitivity and specificity than a chest x-ray and is equal to that of a CT scan (Bourcier et al., 2014). Pulmonary infiltrate, lobular consolidation, or opacities found on chest x-ray, CT scan, or ultrasound confirm the diagnosis of pneumonia; however, newer studies demonstrate that chest x-rays have a low sensitivity and low positive predictive rate, but a high specificity rate and high negative predictive value (Bartlett, 2016; Self, Courtney, McNaughton, Wunderink, & Kline, 2013). Both the CPIS and CURB-65 are useful tools that may aid in predicting the severity of CAP and the need for hospital admission. The use of the PSI tool for use in risk stratification and decision making has been validated in multiple studies (Yealy & Fine, 2015).

Other diagnostic tests are usually reserved for candidates admitted to the hospital following the Infectious Disease Society of America/American Thoracic Society (IDSA/ATS) guidelines (2007). Sputum for gram stain is recommended for all patients but is difficult to obtain in practice, particularly in an outpatient setting. For hospital patients, a urinary antigen test for pneumococcal disease is indicated (Mandell et al., 2007).

Diagnostic testing for VAP includes chest x-ray or CT scan, and endotrachial aspirate for culture. There is little evidence to support invasive sampling over noninvasive sampling of sputum and secretions. In addition, the guidelines suggest that positive cultures from noninvasive samplings and clinical criteria should be the basis for treatment in VAP (Kalil et al., 2016). The updated ISDA guidelines do not recommend the use of biomarkers or CRP values when deciding to start antibiotic treatment. While only about 15% of VAP patients have positive blood cultures, they are still recommended as they may reveal other infectious processes not related to VAP (Kalanuria et al., 2014; Kalil et al., 2016).

Differential Diagnosis: The differential diagnoses of CAP are dependent on the results of the chest x-ray. Differentials for those with a positive chest x-ray include:

- Aspiration pneumonitis
- Pulmonary infarction
- Heart failure
- Exacerbation of pulmonary fibrosis or bronchiectasis
- Acute eosinophilic pneumonia
- Pulmonary vasculitis
- Hypersensitivity pneumonitis
- Injury from cocaine use

For those without an abnormal chest x-ray the differentials are:

- COPD exacerbation
- Influenza
- Pertussis
- Acute bronchitis
- Viral syndrome in asthmatic patients (Wunderink & Waterer, 2014)

Early stage VAP occurs within 48 hours of intubation, while later stage VAP emerges after the fourth day of intubation and is more likely to be associated with multi-drug resistant (MDR) bacteria. Differential diagnosis for VAP includes ventilator-associated respiratory infection (VARI) and ventilator-associated tracheobronchitis (VAT) (Kalanuria et al., 2014; Kalil et al., 2016).

Treatment: A crucial element in treatment is determining the severity of the illness and the setting, but other factors including oral intake, adherence, substance abuse, mental illness, cognitive impairment, patient function, and living situation (File, 2016d). For community-dwelling older patients, the presence of a responsible caregiver and a supportive home environment is necessary. Temporary measures to increase support and monitor status of response may be introduced by ordering home health services. If the patient fails to recover or deteriorates despite these interventions, hospitalization should be contemplated. IDSA/ATS guidelines advocate the use of the pneumonia patient outcome research team (PORT) PSI prediction rule as a guideline for determining outpatient treatment versus hospitalization (which can be accessed at http://pda.ahrq.gov/clinic/psi/psicalc.asp).

Use of any prediction rule or tool should be tempered by clinical judgment. Objective criteria for hospital admission include (File, 2016c; Kalanuria et al., 2014; Kalil et al., 2016):

- Inability to take oral medications or fluids
- Acute mental status changes
- Severe acute metabolic, hematological, or electrolyte abnormalities
- Acute concomitant medical condition (e.g., malignancy, hepatic disease, renal insufficiency, cardiac disease)
- Hypoxemia on room air (PaO_2 less than 60 mm Hg)
- Multilobular involvement on chest x-ray
- Secondary suppurative infection

- Severe vital sign abnormality (SBP less than 90 mm Hg, pulse greater than 125/min, respirations greater than 30/min)

The latest consensus guidelines from the IDSA/ATS for CAP, as well as recent studies, support the following recommendations:

- Outpatient treatment for previously healthy adults with no risk factors for DRSP should consist of a macrolide or doxycycline.
- Outpatient treatment for healthy adults who have been on an antibiotic in the previous 3 months should consist of a macrolide or respiratory flouroquinolone.
- For those with comorbidities, including diabetes mellitus, malignancies, alcoholism, chronic lung disease, cardiovascular disease, chronic liver or renal disease, immunosuppressive conditions or immunosuppressive drugs, or risk for DRSP, treatment should consist of a respiratory fluoroquinolone or a combination of a beta-lactam and a macrolide.
- For those in an area with more than 25% macrolide-resistant *S. pneumoniae,* consider treating with a beta-lactam plus either a macrolide or doxycycline.
- For inpatient treatment outside the ICU, a respiratory fluoroquinolone or a beta-lactam and macrolide concurrently; cefotaxime, ceftriaxone, and ampicillin are preferred.
- For severe CAP requiring ICU admission, treat with a beta-lactam antibiotic, plus azithromycin or a respiratory fluoroquinolone.
- Patients with risk factors for *Pseudomonas* should be treated with a beta-lactam antibiotic (piperacillin/tazobactam, imipenem/cilastatin, meropenem, doripenem, or cefepime), plus an aminoglycoside and azithromycin or an antipseudomonal fluoroquinolone (levofloxacin or ciprofloxacin).
- For risk factors for MRSA, treat with vancomycin or linezolid (Watkins & Lemonovich, 2011).
- Treatment should be instituted as soon as possible and transition from IV to oral therapy should occur as soon as the patient is hemodynamically stable, shows clinical improvement, can ingest oral medications, and has no gastrointestinal tract problems. Duration of therapy is 5 to 7 days, provided the patient is afebrile for 48 to 72 hours (Donovan, 2015; File, 2016d; Kalil et al., 2016; Lee, Giesler, Gellad, & Fine, 2016; Mandell et al., 2007; Wunderink & Waterer, 2014).

Treatment for HAP and VAP is guided by the IDSA/ATS guidelines (Kalil et al., 2016). Once the potential for HAP/VAP is identified and specimens have been obtained, rapid empirical treatment with local antibiogram-driven choice of antimicrobial should be started. Knowledge of local resistance patterns, extent of MRSA, and other regional factors is essential. Treatment should be tailored to optimize antibiotic therapy and prevent resistance using pharmacokinetic and pharmacodynamic data (blood levels). In addition, patients on IV antimicrobial therapy should be switched to oral therapy as soon as possible (Kalil et al., 2016; Mandel, 2007). The use of glucocorticoids in the treatment of HAP/VAP has had varying results and should be reserved for patients who may benefit from their anti-inflammatory effects, ICU patients who are severely ill and have an elevated CRP, and patients whose risk for adverse effects from their use is limited (File, 2016c).

Follow-Up: Follow-up depends on comorbidities and response to prescribed treatment regimen. All patients diagnosed with CAP should schedule a follow-up office visit within a few days after the initial diagnosis, as well as after completion of treatment, with future follow-up as needed. Patients should be instructed to call if symptoms worsen, if no response occurs after 3 to 5 days of treatment, or if symptoms reoccur. In most cases, follow-up chest x-ray is not necessary. In patients with comorbidities, smokers, and those over 40 years old, a repeat chest x-ray after 7 to 12 weeks may be indicated (File, 2016c).

Sequelae: The older patient with comorbidities is especially prone to complications and has a higher long-term mortality rate than those without comorbidities (Restrepoa et al., 2013). Serious complications can occur with CAP, including respiratory failure, pleural effusions, septic shock, renal failure, and empyema. Increased mortality is largely attributed to comorbidities and underlying disease, especially vascular and cardiac disease, COPD, diabetes, and renal disease. HAP and VAP patients are at risk for pneumonitis, CHF, acute respiratory syndrome, atelectasis, and pleural effusion (Holter et al., 2016; Kalanuria et al., 2014; Restrepoa et al., 2013).

Prevention/Prophylaxis: Older patients and all health-care workers should have an annual influenza vaccine unless highly allergic. Latest guidelines recommend that the pneumococcal polysaccharide vaccine 23 and the pneumococcal conjugate vaccine be administered in patients 65 years and older. Patients who are hospitalized for pneumonia may be immunized before discharge (Musher, 2016).

Prevention of VAP is facilitated by maintaining endotracheal tube cuff pressure, elevating the head of the bed, using chlorhexidine for oral care, performing a daily sedation assessment, and providing deep vein and ulcer prophylaxis (Kalanuria et al., 2014).

Referral: Physical therapy measures for strengthening and ambulation may be initiated during the acute recovery period and continued during convalescence or replaced by restorative nursing measures. For those with chronic comorbidities, follow-up or referral to the appropriate specialist is indicated.

Education: Provider education regarding appropriate prescribing of antibiotics is helpful in decreasing drug-resistant organisms. Teach groups of older adults (especially adults with comorbidities) about preventive measures including care after discharge, follow-up, and possible vaccination. Educate patient and caregivers about the importance of nutrition, hydration, deep breathing exercises, and smoking cessation (UpToDate, 2016).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Consider pneumonia in patients with cough, dyspnea, or sputum production, especially if accompanied by fever, altered breath sounds, or rales.	C	Bartlett, 2016
Perform a chest x-ray to confirm the diagnosis of pneumonia. Newer studies suggest US and CT are more accurate.	C C	Bartlett, 2016 Bel, Hausfater, Chenevier-Gobeaux, Blanc, Benjoar, Ficko, ...& Le Bel, 2015 CDC, 2016 File, 2016d NICE, 2014
Routine diagnostic tests are optional for outpatients, because most do well with empirical antibiotic treatment.	C C	Bartlett, 2016 Wunderink & Waterer, 2014
Sputum gram stain and culture, as well as legionella and pneumococcal antigen testing, are recommended for inpatients, particularly those with severe pneumonia, in whom identification of an organism is more likely to prompt a change in the antibiotic agent.	C	Kalil et al., 2016
The Pneumonia Severity Index and CURB-65 are validated prognostic models that may be used to help determine site of treatment in patients with CAP.	A C	Mandell et al., 2007 File, 2016c
Administer antibiotic therapy as soon as possible after the diagnosis is made and before leaving the emergency department if the person is being admitted.	C C	Mandell et al., 2007 Moran, Rothman, & Volturo, 2013
Treat previously healthy outpatients with a macrolide or doxycycline.	B C	Mandell et al., 2007 Musher & Thorner, 2014 Wunderink & Waterer, 2014
Treat outpatients with comorbidities or recent antibiotic use with levofloxacin or moxifloxacin alone or a beta lactam plus a macrolide.	B C	Mandell et al., 2007 Musher & Thorner, 2014
Treat inpatients empirically until pathogen is identified with a 2nd or 3rd generation bata lactam plus a macrolide, or a respiratory fluoroquinolone.	A C	Mandell et al., 2007 Wunderink & Waterer, 2014
VAP EVIDENCE		
In the absence of medical contraindications, elevate the head of the bed at an angle of 30 to 45 degrees for a patient at high risk for aspiration (e.g., a person receiving mechanically assisted ventilation and/or who has an enteral tube in place).	A C	Institute for Clinical Systems Improvement (ICSI), 2011 Kalanuria, Zai, & Mirski, 2014
Cuff pressure should be maintained at 20 to 25 cm H_2O. Minimal leak technique is discouraged.	B	American Thoracic Society (ATS) Documents, 2004 ICSI, 2011
Effective infection control measures: staff education, compliance with alcohol-based hand disinfection, and isolation to reduce cross-infection with multidrug-resistant pathogens should be used routinely.	A C	ATS Documents, 2004 File, 2016c

Continued

VAP EVIDENCE

Intensive insulin therapy is recommended to maintain serum glucose levels between 80 and 110 mg/dL in ICU patients to reduce nosocomial bloodstream infections, duration of mechanical ventilation, ICU stay, morbidity, and mortality.	A	ATS Documents, 2004

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

PULMONARY EMBOLISM

Signal Symptoms: Acute onset dyspnea, tachypnea, tachycardia, pleuritic type chest pain, anxiety, symptoms of deep vein thrombosis (DVT). Difficult to diagnose due to variability in presentation (Belohlavek, Dytrych, & Linhart, 2013). Symptoms are nonspecific; some pulmonary emboli are asymptomatic. Clinical symptoms alone should not be used to confirm or exclude PE (Ho & Smouse, 2015).

Description: PE is the occlusion of the pulmonary artery or one of its branches by a traveling thrombus originating from elsewhere in the body (Thompson, 2016). A blood clot is the most common embolism, although fat, air, bone marrow, tumor cells, and foreign material also can occlude the pulmonary vasculature (Konstantinides et al., 2014). PE is one component of venous thromboembolism (VTE), which also encompasses DVT.

Etiology: PE usually is caused by a dislodged thrombus from one of the veins of the legs or pelvis (DVT). Most emboli are believed to arise from areas of decreased blood flow in the lower extremity proximal veins (iliac, femoral, popliteal). More than 50% of patients with proximal vein DVT also present with PE (Thompson, 2016).

Occurrence: Estimated incidence of 60 to 70 per 100,000 in general population; PE can develop in as many as 40% to 50% of patients with DVT (Belohlavek et al., 2013). Seventy percent of patients presenting with PE are also found to have DVT in the legs (Wilbur & Shian, 2012). PE is the third leading cause of vascular death after MI and stroke (Goldhaber & Bounameaux, 2012). The estimated mortality rate in patients with PE is 12% (Wilbur & Shian, 2012).

Age: The incidence of PE increases steadily with age; however, in older age the incidence of PE is higher in men than in women (Lavorini et al., 2013).

Gender: Overall prevalence of PE is similar in males and females, but there is a higher recurrence rate in men (Robert-Ebadi et al., 2010). Women of reproductive age have slightly increased incidence of PE because of the association of PE with pregnancy and increased risk with oral contraceptive use (Lavorini et al., 2013).

Ethnicity: Incidence varies by race; highest incidence is in those of African American descent, with Europeans in North America and Europe almost as prevalent. There is lower incidence in Hispanic, Asian, and Native American populations (Zakai & McClure, 2011).

Contributing Factors: Risk factors for PE are indicated by Virchow's triad of stasis or prolonged immobility, hypercoagulation, and venous trauma (endothelial injury with inflammation of the vessel lining). Stasis may be occupational or medical (e.g., CHF, postsurgical paralysis, chronic illness or debilitation, tumor compression). Venous trauma damaging the deep veins of the legs; prolonged immobility for less than 48 hours; orthopedic procedures from arthroscopy to hip, knees, or pelvic repair; or trauma from falls, burns, or crush injuries may contribute to thrombus formation and lead to PE (Ho & Smouse, 2015). Medical disorders that increase PE risk include heart failure, ischemic stroke, acute respiratory failure or intubation, sepsis, acute rheumatic disease, and inflammatory bowel disease (Lavorini et al., 2013). Presence of a central venous catheter and insertion of a pacemaker, use of estrogen, advanced age, obesity, and malignancy are also risk factors. A past history of DVT or PE is associated with recurrence. Cardiovascular risk factors for PE include hypertension, diabetes, cigarette smoking, and increased cholesterol (Ozaki & Bartholomew, 2012). A rare but strong risk factor for PE includes deficiencies of natural coagulation inhibitors like antithrombin, Protein C and S. These usually account for about 1% of PE cases (Lavorini et al., 2013). A small proportion of cases have been associated with Factor V Leiden and prothrombin (Factor II) G20210A. Pregnancy can lead to a five-fold increased risk of PE. Hormone replacement therapy (HRT) is believed to increase PE risk by two- to four-fold (Lavorini et al., 2013). Although patient risk factor identification is crucial in the initial diagnostic work-up, up to 30% of PE cases develop idiopathically (Belohlavek et al., 2013).

Signs and Symptoms: Signs and symptoms of PE tend to be nonspecific. Less than 20% of patients present with classic symptoms of chest pain, dyspnea, or hemoptysis. The Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED II) study showed that 97% of patients had more than one of the symptoms of dyspnea, pleuritic chest pain, and tachypnea. The variation in presentation may occur when these symptoms are attributed to aging or existing

comorbidities (Ho & Smouse, 2015). Dyspnea can range from acute to severe. Severe dyspnea, as seen with central PE, is usually described as having typical angina-type character. Dyspnea seen with a small peripheral PE is usually mild and transient, making PE difficult to diagnose (Konstantinides et al., 2014). Other findings may include anxiety or apprehension, cough, tachypnea, and accentuation of the pulmonic component of S_2 and may be accompanied by diaphoresis, syncope, tachycardia, S_3 or S_4 gallop, hypoxemia, or hemoptysis (Ozaki & Bartholomew, 2012). Auscultation of the lungs may be normal or may show localized wheezing, consolidation, or friction rub. Jugular venous distention and atrial arrhythmia may be present. Examination of the legs, especially of the iliofemoral and popliteal areas, may reveal signs of DVT. The arms, abdomen, and pelvic areas also should be examined for pain, erythema, or palpable vein cords. In the case of a massive PE, hypotension, circulatory instability, and cyanosis may be present.

Diagnostic Tests: Clinical judgment in conjunction with clinical prediction scoring tools and diagnostic studies is recommended. The Wells Clinical Prediction Scoring Tool for Pulmonary Embolism (www.mdcalc.com/wells-criteria-for-pulmonary-embolism-pe) considers clinical symptoms, tachycardia, recent immobility or surgery, hemoptysis, malignancy, and prior DVT/PE to help assign a pretest probability of PE (low, intermediate, or high) (Wilbur & Shian, 2012). The Revised Geneva Score is sometimes used to stratify patients by probability of PE. The Simplified Pulmonary Embolism Severity Index (PESI) (www.mdcalc.com/simplified-pesi-pulmonary-embolism-severity-index) is a prediction rule for 30-day mortality from symptomatic PE; this tool is used more selectively as appropriate. No matter what scoring method is used, it is helpful to determine pretest probability in order to categorize patients into subgroups to determine likelihood of presence of PE. Future research is needed to develop standardized models of predictability (Lavorini et al., 2013).

The chest x-ray is often abnormal, but usually nonspecific. It is used primarily to rule out other causes of symptoms such as pneumonia or CHF. EKG changes that show signs of right ventricular strain, T-wave inversion in leads V1 to V4, QR pattern in V1, new-onset right bundle branch block, or S1Q3T3 complex can sometimes be seen in severe cases of PE. Mild PE cases may only reveal sinus tachycardia or atrial arrhythmias such as AF (Konstantinides et al., 2014). In a patient deemed to be at low or intermediate risk of PE, a negative enzyme-linked immunosorbent assay (ELISA) D-dimer test can rule out the possibility of PE. The ELISA or ELISA-derived D-dimer assays have a sensitivity of 95% and can exclude PE in patients with a low to intermediate pretest probability (Konstantinides et al., 2014). The sensitivity of D-dimer testing also increases with the extent of PE; highest concentrations are seen in patients with perfusion scan defects involving 50% of the pulmonary circulation (Lavorini et al., 2013). The specificity of D-dimer tests decreases with increased age, to only 10% in patients older than 80 years. D-dimer can also be elevated in the patient with cancer, during hospitalization, and in pregnant patients. Some studies suggest age-adjusted cut-offs to improve D-dimer testing in the elderly (Konstantinides et al., 2014). Patients with intermediate to high pretest probability would require further testing to rule out PE (Ozaki & Bartholomew, 2012).

Pulmonary catheter angiography has long been considered the gold standard to diagnose PE, but the Multidetector CTA (MDCTA) is often used as the test of choice because it is noninvasive and has a lower rate of complications compared to pulmonary catheter angiography (Ho & Smouse, 2015). The advancements in the quality of MDCTA has led to increased usage (Belohlavek et al., 2013). MDCTA is widely available, and when used with IV contrast, the MDCTA can outline thrombi in the pulmonary arteries. MDCTA has almost replaced lung scanning and angiography as the standard for PE diagnosis (Lavorini et al., 2013). Limitations for using a MDCTA include patients with renal dysfunction, allergy to contrast media, possible pregnancy, critical illness, requiring ventilator support, or recent MI. In this case, a ventilation/perfusion (V/Q) scan may be done. If there is a high suspicion of PE but a negative MDCTA, a V/Q scan may be ordered also. However, most clinicians feel more comfortable with MDCTA as a diagnostic tool due to its ability to provide anatomical demonstration of presence of a clot versus relying on the V/Q scans to assess the probability of embolism based on V/Q mismatch (Lavorini et al., 2013). V/Q scanning is done in the nuclear medicine department and may not be as readily available as MDCTA in many cases (Belohlavek et al., 2013).

Echocardiography is not recommended to diagnose PE, but can help determine risk of mortality or adverse outcome. An echocardiogram can evaluate right ventricular function and detect a free-floating thrombus in the right heart, which can be associated with PE and increased mortality (Ho & Smouse, 2015). In patients with a massive PE, transesophageal echocardiography (TEE) should be done to assess right ventricular function.

The myocardial markers, including cardiac troponin levels and BNP or NT-proBNP have no role in the diagnosis of PE. These markers indicate a biochemical response to PE. They have been shown to forecast right ventricular dysfunction and mortality. Elevated troponin levels have been shown to reflect severity of PE and have been associated with increased mortality (Belohlavek et al., 2013).

Differential Diagnosis:

- Pneumonia
- MI
- Pericarditis
- CHF
- Pleural effusion
- Panic attacks
- Hyperventilation syndrome
- Pneumothorax
- Esophageal rupture
- Gastritis
- Gastric or duodenal ulcer
- GERD
- Asthma
- Musculoskeletal pain
- Mediastinitis

Treatment: Anticoagulation is the cornerstone of treatment. Goal of anticoagulation is to prevent early death, recurrence, or fatal VTE/PE. If no contraindications, anticoagulation should be initiated immediately in patients with high or moderate suspicion of PE while awaiting diagnostic test

results (Lavorini et al., 2013). Acute phase therapy includes unfractionated heparin (UFH), low-molecular weight heparin (LMWH), or fondaparinux. Parenteral heparin should overlap with initiation of vitamin K antagonist (VKA) (Konstantinides et al., 2014).

In patients with a massive PE, acute RV failure and low systemic output is the leading cause of death. Treatment should be individualized and supportive, and may include the use of vasopressors. Oxygen administration and/or mechanical ventilation may be needed to reverse hypoxia (Konstantinides et al., 2014). Pharmacological treatments, including thrombolytic drugs or surgical or interventional reperfusion treatments such as surgical or catheter embolectomy procedures may also be considered in hemodynamically unstable patients (Lavorini et al., 2013).

In patients at low risk for bleeding, LMWH, subcutaneous (SQ) fondaparinux, IV or SQ unfractionated heparin (UFH) can be used. LMWH or fondaparinux is preferred due to lower risk of inducing major bleeding or heparin-induced thrombocytopenia (HIT) (Konstantinides et al., 2014). In patients with severe renal impairment (creatinine clearance less than 30 mL/min), UFH is preferred because of ease in monitoring via activated partial thromboplastin time (aPPT) testing (Lavorini et al., 2013). UFH is recommended in patients in whom thrombolysis is being contemplated, morbidly obese patients (questionable subcutaneous absorption), or those at high risk of bleeding because of the short half-life of UFH and the ability to rapidly reverse UFH with protamine (Konstantinides et al., 2014). Oral anticoagulation should be initiated simultaneously with or shortly after parenteral anticoagulation. Warfarin (Coumadin) has been the gold standard VKA for over 50 years and is monitored by measuring the international normalized ratio (INR). UFH, LMWH, or fondaparinux should be continued for at least 5 days and until INR is within the 2 to 3 range for 2 consecutive days to ensure adequate anticoagulation (Konstantinides et al., 2014). Warfarin can be used for long-term maintenance of 6 months or more. INR monitoring should begin with baseline measurement, then again 2 to 3 days after initiation. Monitoring then tapers to twice weekly until the INR is within the therapeutic range, then once weekly or every other week, and finally, monthly. ACCP guidelines allow clinicians to consider INR monitoring up to every 12 weeks in patients who are stable. Dosage is adjusted based on INR results (Wigle et al., 2013).

Dabigitran, rivaroxaban, apixaban, and edoxaban are the new oral anticoagulants (NOACs). They are not vitamin K dependent and are approved for treatment of PE. NOACs overcome some limitations of standard therapy, such as the need for regular monitoring and dose adjustments (Lavorini et al., 2013). Studies have shown NOACs have similar efficacy and safety profiles compared to traditional therapies in terms of initial and long-term treatment of PE (Ho & Smouse, 2015). CHEST 2016 guidelines recommend the use of NOACs over VKA therapy in patients with PE and no cancer (Kearon et al., 2016). VKA therapy is suggested over LMWH in patients with PE and no cancer who are not treated with NOACs. The decision to use NOACs can be influenced by whether cost is covered by insurance plans and patient and physician satisfaction with current therapy (Lavorini et al., 2013). Currently, NOAC therapy can be very expensive (Goolam-Mahomed, 2013).

LMWH may be used long term in patients with cancer associated with PE due to greater compatibility with chemotherapy (Lavorini et al., 2013). LMWH is used for PE treatment during pregnancy because VKAs are teratogenic.

Routine use of venous filters to prevent PE recurrence is not recommended. Filters may be indicated in cases if anticoagulation is contraindicated or if PE has recurred despite adequate anticoagulation. It is recommended that the filter be removed as soon as possible to avoid secondary vena cava thrombosis or thromboembolism (Lavorini et al., 2013).

Follow-Up: Follow-up visits are on an individualized basis. Anticoagulation usually is continued for a minimum of 3 to 6 months after PE. Experts differ on optimal length of therapy and dosing adjustments in specific situations. CHEST 2016 guidelines recommend at least 3 months of anticoagulant therapy for provoked or unprovoked PE. The risk-benefit ratio should be evaluated after 3 months of therapy for the unprovoked PE. For the unprovoked first-time VTE with low to moderate bleeding risk, CHEST guidelines recommend extended anticoagulant therapy (no scheduled stop date). If bleeding risk is high, 3 months of anticoagulant therapy is recommended. In any case of extended anticoagulant therapy, risk–benefit for continued treatment should be reassessed at periodic intervals (i.e., annually). Extended therapy is recommended for the patient with active cancer and PE regardless of bleeding risk, keeping in mind the need to reassess risk–benefit periodically (Kearon et al., 2016).

Sequelae: Unrecognized PE can be fatal and is associated with overall mortality of up to 30% (Thompson, 2016); many are diagnosed at autopsy. Most deaths occur within the first week of diagnosis and are usually due to recurrent VTE and shock (Thompson, 2016). After acute PE, symptoms may resolve or can lead to chronic thromboembolic pulmonary hypertension, which can progress to right heart failure if untreated (Goolam-Mahomed, 2013). Other complications include alveolar collapse, atelectasis, and pulmonary infarction. Complications from anticoagulation include uncontrolled bleeding and hematoma.

Prevention/Prophylaxis: PE is one of the most common preventable causes of hospital deaths in the United States, but only one-third of hospitalized patients receive adequate prophylaxis (Ozaki & Bartholomew, 2012). Patients should avoid controllable risk factors for PE such as sedentary lifestyle, obesity, and smoking. Encourage frequent ambulation, use of antiembolic stockings or graded compression hose, safety precautions to avoid injury to lower extremities, evaluation of possible hypercoagulation pathology, and prophylactic anticoagulation after orthopedic surgery of hip or knee. Prolonged sitting should be avoided. Consideration of prophylactic anticoagulation in any immobilized hospitalized patient and use of sequential compression devices (SCDs) may be helpful. In select patients with very increased DVT/PE risk and bleeding, a temporary IVC filter is an option (Tapson, 2015).

Referral: Refer patients to internal medicine or emergency medicine for emergent evaluation and initial treatment plan. When stable, patients can be managed on warfarin or NOAC therapy. Health-care professionals skilled in initiation and assessment of warfarin therapy and adjustments can greatly influence outcomes (Wigle et al., 2013). Although no regular

monitoring of NOAC therapy is required for dosing, compliance is of utmost importance in order to achieve adequate therapeutic anticoagulant benefits (ten Cate, 2013).

Education: Provider education about awareness of PE in differential for chest pain or dyspnea is needed. Teach the patient, family, or caregiver about risk factors and preventive measures, disease pathology, monitoring for signs of recurrence, and need for compliance with anticoagulation therapy. Instruct the patient to use a soft toothbrush and an electric razor and to report bleeding gums, hemoptysis, and any blood in stool or urine. Advise patients to avoid aspirin, to consult with their health-care provider before taking any OTC medications, and to take precautions to avoid bumping or bruising.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Prevention of VTE should be considered according to the patient's baseline risk.	A	Ozaki & Bartholomew, 2012
Diagnosis begins with estimating probability based on a validated clinical decision rule, then using a validated diagnostic algorithm integrating findings from D-dimer, ventilation/perfusion (V/Q) scan, lower extremity ultrasound, and spiral (helical) CT scanning with IV contrast (i.e., CT pulmonary angiography [CT-PA]).	A	Belohlavek et al., 2013 Ho & Smouse, 2015 Konstantinides et al., 2014 Lavorini et al., 2013
In patients with acute PE, we recommend early initiation of vitamin K antagonist (VKA) (e.g., same day as parenteral therapy is started) over delayed initiation, and continuation of parenteral anticoagulation for a minimum of 5 days and until the INR is 2.0 or above for at least 24 hours. Studies have shown NOACs have similar efficacy and safety profiles compared to traditional therapies in terms of initial and long term treatment of PE in some patient populations.	B	Ho & Smouse, 2015 Konstantinides et al., 2014 Lavorini et al., 2013
In patients with acute PE, we recommend initial treatment with parenteral anticoagulation (LMWH, fondaparinux, IV UFH, or subcutaneous UFH) over no such initial treatment.	A	Konstantinides et al., 2014 Lavorini et al., 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

PULMONARY TUBERCULOSIS

Signal Symptoms: Productive, prolonged cough; fatigue; low-grade fever; night sweats; poor appetite; weight loss.

Description: Pulmonary TB is a chronic necrotizing infection caused by a slow-growing acid-fast bacillus (AFB), *Mycobacterium tuberculosis*. TB is the most important, fatal infection among humans. The primary site for most cases of TB is the lungs. Extrapulmonary TB can affect any organ or tissue.

Etiology: The infection spreads by inhalation of airborne particles or droplets produced by persons with active pulmonary or laryngeal TB during coughing, sneezing, singing, and other expiratory efforts. The development of infection after exposure depends on the exposed person's ability to mount an effective immune response on the cellular level. In the initial 2 to 4 weeks before cellular immunity response occurs, direct pulmonary infection may develop, or lymphohematogenous circulation may lead to miliary, meningeal, or TB adenitis. When T cells recognize the specific antigen, they become sensitized, engaging the macrophages in destroying or containing the tubercle bacilli. This leads to healing, with no residual or calcified lymph nodes in the pulmonary or tracheobronchial areas. This latent stage is typical of 90% to 95% of infected persons, leaving them at lifelong risk for reactivation. Tuberculin skin testing documents exposure at 2 to 10 weeks after exposure.

Occurrence: TB is one of the world's deadliest diseases.

- One-third of the world's population is infected with TB.
- In 2015, 10.4 million people around the world became sick with TB. There were 1.8 million TB-related deaths worldwide.
- TB is a leading killer of people who are HIV-infected.

A total of 9,557 TB cases (a rate of 3.0 cases per 100,000 persons) were reported in the United States in 2015. The overall number of TB cases in the United States increased over the previous year in 2015, after having declined yearly during 1993 through 2014. Despite a slight increase in case count, the TB incidence rate per 100,000 persons has remained relatively stable at approximately 3.0 since 2013.

Age: Children and adolescents are more likely to have primary disease; adults and older adults are more likely to have recrudescent disease. TB disease in children under 15 years of age (also called pediatric TB) is a public health problem of special significance because it is a marker for recent transmission of TB. Also of special significance, infants and young children are more likely than older children and adults to develop life-threatening forms of TB disease (e.g., disseminated TB, TB meningitis). Among children, the greatest numbers of TB cases are seen in children less than 5 years of age and in adolescents older than 10 years of age.

Gender: TB occurs in men more frequently than in women.

Ethnicity:

- Native Americans or Alaska Natives: 6.1 TB cases per 100,000 persons
- Asians: 18.2 TB cases per 100,000 persons
- African Americans: 5.0 TB cases per 100,000 persons
- Native Hawaiians and other Pacific Islanders: 18.2 TB cases per 100,000 persons
- Hispanics or Latinos: 4.8 TB cases per 100,000 persons
- Caucasians: 0.6 TB cases per 100,000 persons

Risk Factors: Overall, about 5% to 10% of infected persons who do not receive treatment for latent TB infection will develop TB disease at some time in their lives. For persons whose immune systems are weak, especially those with HIV infection, the risk of developing TB disease is much higher than for persons with normal immune systems. Persons who are at increased risk include those with:

- HIV infection
- Substance abuse issues (especially IV drug use)
- Recent infection with *M. tuberculosis* (less than 2 years ago)
- Chest x-ray findings suspicious of previous TB with no treatment or ineffective treatment
- Diabetes mellitus
- Silicosis
- Prolonged corticosteroid therapy
- Other immunosuppressive therapy
- Cancer of the head and neck
- Hematological and reticuloendothelial diseases
- End-stage renal disease
- Intestinal bypass or gastrectomy
- Chronic malabsorption syndromes
- Low body weight (less than 10% ideal body weight)

Other factors associated with increased risk include homelessness, residence in a congregate setting (e.g., nursing home, boarding home, prison, mental health facility), low socioeconomic status, and health-care work in a high-risk area.

TABLE 8-6 Differentiating Between Latent TB Infection and TB Disease

LTBI	TB DISEASE
No symptoms or physical findings suggestive of TB disease.	Symptoms may include one or more of the following: fever, cough, chest pain, weight loss, night sweats, hemoptysis, fatigue, and decreased appetite.
TST or IGRA result usually positive.	TST or IGRA result usually positive.
Chest radiograph is typically normal.	Chest radiograph is usually abnormal. However, may be normal in persons with advanced immunosuppression or extrapulmonary disease.
If done, respiratory specimens are smear and culture negative.	Respiratory specimens are usually smear or culture positive. However, may be negative in persons with extrapulmonary disease or minimal or early pulmonary disease.
Cannot spread TB bacteria to others.	May spread TB bacteria to others.
Should consider treatment for LTBI to prevent TB disease.	Needs treatment for TB disease.

Note: Physical examination may be unrevealing: Nonspecific signs such as fever or weight loss may be the only findings. In some persons, a positive tuberculin test reaction is the only manifestation. Chest examination may show post-tussive apical rales. If pleural effusion is present, percussion in the area may be dull.
Source: https://www.cdc.gov/tb/topic/testing/default.htm

Signs and Symptoms: Typical presentation includes cough, hemoptysis, weight loss, anorexia, adenopathy, fever, night sweats, decreased activity level, and pleuritic pain (Table 8-6). In the average population, the onset is gradual and may go undetected for some time. In the older patient, these findings are not usually present, or they are so subtle and so intermingled with other chronic illness symptoms as to be undistinguishable. Weight loss, dyspnea, or anorexia may be the only symptoms. Typical simulations include pneumonia, bronchitis, or CHF with pleural effusion. Extrapulmonary TB may manifest with symptoms typical to the site involved (e.g., urinary incontinence or frequency and urgency for bladder TB).

Diagnostic Tests:

Tuberculin Skin Test (TST): The TST is used to determine if a person is infected with *M. tuberculosis.* If a person is infected, a delayed-type hypersensitivity reaction is detectable 2 to 8 weeks after infection. The skin test is administered intradermally using the Mantoux technique by injecting 0.1 ml of 5 TU purified protein derivative (PPD) solution. The reading and interpretation of TST reactions should be conducted within 48 to 72 hours of administration. For more information about tuberculin skin testing, visit the CDC website for additional resources (see Resources) and refer to Appendix C.

Key Points:

- Training is essential for health-care providers to gain proficiency in the administration and interpretation of the TST.
- The TST should not be performed on a person who has written documentation of either a previous positive TST result or treatment for TB disease.
- Patients or family members should never measure TST results; this should only be done by a trained health-care professional.
- Interpretation of the TST result is the same for persons who have had BCG vaccination because a majority of BCG cross-reactivity wanes with time.
- A TST that was not measured and recorded in millimeters (mm) of induration must be repeated.

Interferon–Gamma Release Assays (IGRAs): IGRAs are used to determine if a person is infected with *M. tuberculosis* by measuring the immune response to TB proteins in whole blood. Specimens are mixed with peptides that simulate antigens derived from *M. tuberculosis* and controls. In a person infected with *M. tuberculosis,* the white blood cells recognize the simulated antigens and release interferon-gamma (IFN-γ); results are based on the amount of IFN-γ released. As noted earlier, there are two U.S. Food and Drug Administration (FDA) approved IGRAs commercially available in the United States:

- QuantiFERON®-TB Gold-in-Tube test (QFT-GIT)
- T-SPOT® TB test

Key Points:

- Advantages of IGRAs include:
  - Requires a single patient visit to conduct the test.
  - Does not cause booster phenomenon.
  - Laboratory test not affected by health-care worker perception or bias.
  - Results can be available within 24 hours.
  - Unaffected by BCG and most environmental mycobacteria.
- Limitations of IGRAs include:
  - Blood sample must be processed within 8 to 30 hours after collection.
  - Limited data exist on use in groups such as children younger than 5 years of age, persons recently exposed to TB, immunocompromised persons, and those who will be tested repeatedly (serial testing).

For more information, see Latent Tuberculosis Infection: A Guide for Primary Health Care Providers at https://www.cdc.gov/tb/publications/ltbi/.

Differential Diagnosis: Pneumonia, lymphoma, fungal infections, CHF, pleural effusion, and lung cancer can mimic TB.

Treatment: Before treatment, obtain baseline values for liver function, bilirubin, CBC, BUN, creatinine, and serum uric acid. If ethambutol (EMB) is used, baseline visual acuity should be measured. The goal of treatment is safety and efficacy in the shortest time period. For newly diagnosed, active TB, initial treatment consists of combined therapy using four first-line drugs: isoniazid (INH), rifampin (RIF), pyrazinamide (PZA), and EMB, until culture results are complete. Follow-up cultures should be done, usually monthly, until negative, to determine response to treatment. If the culture is not negative after 3 months, suspect drug resistance or noncompliance and reevaluate. After culture is negative, obtain one further culture at treatment completion. For drug-resistant TB, different culture guidelines apply. Several treatment options are available. The most commonly used options are presented in Table 8-7 (for further information, consult with the CDC).

Note: HIV-positive individuals require specific modifications in therapy and CDC guidelines should be consulted. Multidrug-resistant TB also requires different regimens (see CDC guidelines).

TABLE 8-7 Latent TB Infection Treatment Regimens

DRUGS	DURATION	INTERVAL	COMMENTS
Isoniazid	9 months	Daily	Preferred treatment for: • Persons living with HIV • Children aged 2 to 11 years • Pregnant women (with pyridoxine/vitamin B_6 supplements)
		Twice weekly *	Preferred treatment for: • Pregnant women (with pyridoxine/vitamin B_6 supplements)
Isoniazid	6 months	Daily	
		Twice weekly *	
Isoniazid and Rifapentine	3 months	Once weekly*	Treatment for: • Persons 12 years or older Not recommended for persons who are: • Younger than 2 years old • Living with HIV/AIDS taking antiretroviral treatment • Presumed infected with INH or RIF-resistant M. tuberculosis • Women who are pregnant or expect to become pregnant within the 12-week regimen
Rifampin	4 months	Daily	

*Use directly observed therapy (DOT).
Note: Due to the reports of severe liver injury and deaths, CDC recommends that the combination of rifampin (RIF) and pyrazinamide (PZA) should generally not be offered for the treatment of latent TB infection.

Daily for 2 months:
INH 5 mg/kg (300 mg maximum)
RIF 10 mg/kg (600 mg maximum)
PZA 15 to 20 mg/kg (2 g maximum)
EMB 15 to 25 mg/kg (1 g maximum)

Then daily for 4 months:
INH and RIF (see preceding dosage schedule)

In lieu of daily therapy for 4 months, the same agents (INH, RIF) can be used as follows:
INH 15 mg/kg (900 mg maximum) two or three times weekly by directly observed therapy (DOT)
RIF 10 mg/kg (600 mg maximum) two or three times weekly by DOT

The basis for treatment is availability of two drugs to which the bacterium is susceptible. Prolonged treatment is needed. Compliance is key to successful control of disease.

Follow-Up: See Culture Guidelines. Follow-up chest x-ray examination may be done at therapy termination to evaluate response. Periodic liver enzymes are necessary, especially if the patient is taking INH, to monitor for effects on hepatotoxicity. For the frail, older adult in a long-term care facility, more frequent monitoring for adverse effects of treatment, including anorexia, polyneuropathy, or development of medication-induced hepatitis, is warranted. DOT is normally used in these settings, so compliance is less of a concern. Refer community-dwelling older adults to the local or state health department for follow-up, monitoring of medication compliance and side effects, patient and family education, and testing of close contacts. TB is a reportable disease.

Many agencies charged with monitoring and control have outreach services, such as home visits. Emphasize to patients that compliance is crucial to successful control. If no follow-up visitation is available through the monitoring agency, see the patient for monthly follow-up visits in the office.

Sequelae: Possible complications include development of drug-resistant organisms, particularly if a patient is noncompliant with the prescribed treatment. Secondary infection of cavitary lesions and development of treatment-associated hepatitis or polyneuropathy are possible. If treatment is ineffective, spread of disease to other close contacts can occur.

Prevention/Prophylaxis: For older patients residing in long-term care facilities, PPD testing before admission to the facility is required unless there is documented evidence of a positive test result in the past. Two-step testing is recommended initially. Annual retesting is recommended. Patients with a positive PPD reaction need a chest x-ray to evaluate for active or latent disease. Staff members are required to have tuberculin skin testing at initial employment and annually.

When targeted testing reveals a positive tuberculin skin reaction but no evidence of active TB, it is often referred to as latent tuberculosis infection. The person has been exposed to and infected with *M. tuberculosis* but does not have active disease and cannot infect others.

The decision to institute chemoprophylaxis is a clinical judgment, based on a comparison of individual factors with the risk of developing TB (see Contributing Factors) versus the risk of INH toxicity. Chemoprophylaxis is with INH, 300 mg orally daily for 6 months in an otherwise healthy person; 9 months is considered optimal if compliance is not an issue. Alternatively, INH, 15 mg/kg orally twice weekly by DOT, may be substituted. For HIV-positive persons or close contacts of patients with drug-resistant tuberculosis, see CDC recommendations. A shorter course of RIF and PZA previously recommended has been associated with fatal and severe liver injuries and so is no longer recommended.

Referral: Patients may be referred to a government-associated community agency, such as the health department, or to an infectious disease or pulmonary specialist for initial evaluation and management recommendations. Refer patients with concurrent positive HIV status or confirmed AIDS to specialized treatment services, or collaborate in management with specialists in this area. Refer patients with severe anorexia or malnutrition to a dietitian.

Education: Teach patient, caregivers, close contacts, and paraprofessional providers about the nature of the disease, its mode of transmission, screening and control measures, and follow-up required. Teach the patient or caregiver about medications, drug actions and possible side effects, length of treatment, and need for compliance.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
We recommend performing an interferon-γ release assay (IGRA) rather than a tuberculin skin test (TST) in individuals 5 years or older who meet the following criteria: 1) are likely to be infected with MTB, 2) have a low or intermediate risk of disease progression, 3) it has been decided that testing for LTBI is warranted, and 4) either have a history of BCG vaccination or are unlikely to return to have their TST read. Remarks: A TST is an acceptable alternative, especially in situations where an IGRA is not available, too costly, or too burdensome.	A	Lewinsohn et al., 2017 ISDA Guideline
We recommend that acid-fast bacilli (AFB) smear microscopy be performed, rather than no AFB smear microscopy, in all patients suspected of having pulmonary TB.	A	Lewinsohn et al., ISDA Guideline

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
While both IGRA and TST testing provide evidence for infection with MTB, they cannot distinguish active from latent TB. Therefore, the diagnosis of active TB must be excluded prior to embarking on treatment for LTBI. This is typically done by determining whether symptoms suggestive of TB disease are present, performing a chest radiograph and, if radiographic signs of active TB (e.g., airspace opacities, pleural effusions, cavities, or changes on serial radiographs) are seen, then sampling is performed and the patient managed accordingly.	A	Lewinsohn et al., 2017 ISDA Guideline
Guidelines recommend that persons at low risk for MTB infection and disease progression NOT be tested for MTB infection. We concur with this recommendation. However,we also recognize that such testing may be obliged by law or credentialing bodies. If diagnostic testing for LTBI is performed in individuals who are unlikely to be infected with MTB despite guidelines to the contrary: ■ We suggest performing an IGRA instead of a TST in individuals 5 years or older Remarks: A TST is an acceptable alternative in settings where an IGRA is unavailable, too costly, or too burdensome. ■ We suggest a second diagnostic test if the initial test is positive in individuals 5 years or older. Remarks: The confirmatory test may be either an IGRA or a TST. When such testing is performed, the person is considered infected only if both tests are positive.	C	Lewinsohn et al., 2017) ISDA Guideline

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RESTRICTIVE LUNG DISEASE

Signal Symptoms: Rapid, shallow respirations; dyspnea; decreased activity tolerance; easy fatigability; nonproductive, irritating cough provoked by deep breathing or exertion.

Description: Restrictive lung disease refers to a heterogeneous group of disorders that share a common abnormal ventilatory function. Restricted breathing is characterized by small tidal volume and rapid rate. The hallmark restrictive pattern is a decrease in lung volumes, principally total lung capacity and vital capacity (Lutfi, 2017; Kanaparthi, 2012).

Etiology: Restrictive lung diseases, which have a variety of etiologies, are divided into subgroups based on the location of the pathology.

Restrictive/Parenchymal/Interstitial/Intrinsic: In addition to a decrease in total lung capacity and vital capacity, residual volume is decreased. Forced expiratory flow rates are maintained.

- Sarcoidosis
- Idiopathic pulmonary fibrosis
- Pneumoconiosis
- Occupational lung disease
- Drug/radiation-induced interstitial lung disease

Restrictive/Extraparenchymal/Extrinsic: Abnormalities can be predominantly in inspiration or in inspiration and expiration.

Neuromuscular:

- Diaphragmatic weakness/paralysis
- Myasthenia gravis (limitations may be inspiratory and expiratory)
- Muscular dystrophies (limitations may be inspiratory and expiratory)
- Cervical spine injuries (limitations may be inspiratory and expiratory)
- Guillain-Barré syndrome (limitations may be inspiratory and expiratory)

Chest Wall:

- Kyphoscoliosis
- Obesity
- Ankylosing spondylitis (limitations may be inspiratory and expiratory)

The mnemonics **P**leural, **A**lveolar, **I**ntrinsic, **N**euromuscular, **T**horacic (PAINT) and **S**pace, **P**leural, **I**nterstitial, **C**hest Wall, **E**xtrathoracic (SPICE) are helpful reminders of the possible causes of restrictive lung disease.

Occurrence: The incidence of restrictive lung disease is undeterminable because several distinct entities are involved. Statistics are available for select causes of restrictive lung disease. Studies reference an overall prevalence of three to six cases per 100,000 persons for intrinsic lung diseases. Prevalence of idiopathic pulmonary fibrosis (IPF) is 27 to 29 cases per 100,000 persons; in adults over age 75 years, the prevalence increases to over 175 cases per 100,000 persons (Kanaparthi, 2012). Occupational lung diseases are common in farmers and in people who work with silica, asbestos, beryllium, organic solvents, or cotton. The prevalence of sarcoidosis in the United States is 10 to 40 cases per 100,000 persons.

Age: Occupationally induced disease and IPF are seen predominantly in the older population; other restrictive lung diseases may occur at any age.

Gender: The incidence is higher in men than women for occupational types of restrictive lung disease.

Ethnicity: African Americans in the United States have a prevalence of sarcoidosis that is 10 to 17 times greater than Caucasians.

Contributing Factors: Risk factors vary with etiology, including exposure to occupational dust, abnormalities in skeletal structure, genetics, and autoimmune disorders (King, 2012).

Signs and Symptoms: Patients have a gradual onset of dyspnea, initially occurring only with exertion and progressing to dyspnea at rest. The breathing pattern is rapid and shallow. A nonproductive cough may be present (Behr, 2012). A careful, detailed history is essential, including prior systemic diseases, occupational or environmental exposures, family history, social history, and history of drug use (Alhamed & Cosgrove, 2011). Amiodarone, nitrofurantoin, hydralazine, gold, chemotherapeutic agents, and procainamide can cause drug-induced disease (Kanaparthi, 2012). Prior radiation can result in fibrosis. Use of tobacco should also be ascertained; it is common for patients to have a mixed pattern of obstructive and restrictive disease. Physical findings may reveal skeletal abnormalities, such as kyphoscoliosis, limiting lung expansion. The initial presentation of breathing problems often occurs after an acute respiratory viral infection.

Physical assessment of the lung initially may be unremarkable. In intrinsic disease, with progression of the disease, inspiratory crackles ("Velcro") typically are heard at the bases. Cyanosis and clubbing of fingers and toes may occur in IPF. In the end-stages, signs of right-sided heart failure, including cor pulmonale, appear (Behr, 2012; Kanaparthi, 2012; King, 2012).

Diagnostic Tests: Because of the diverse nature of the conditions leading to restrictive lung disease, it is challenging to address diagnostic testing and results. Many results are specific to the causative condition. Routine testing including CBC, chemistry profile, and liver function tests is standard.

TEST	RESULTS INDICATING DISORDER
PFT	Normal FEV_1/FVC ratio but decreased FVC and FEV_1; decreased total lung capacity, residual volume, and functional residual capacity. Residual volume–to–total lung capacity ratio is normal to low. Most have a gas exchange problem with marked decrease in single breath diffusing lung capacity for carbon monoxide. Diagnosis of restriction and extent of restriction is based on total lung capacity (Kanaparthi, 2012).
Chest x-ray	Increased interstitial markings, especially in lower fields. Hilar and mediastinal lymphadenopathy in sarcoidosis, some lymphomas, and silicosis. Pleural effusion and thickening with collagen-vascular disease, lymphoma, and asbestosis. A scattered reticulonodular pattern and ground glass opacities are common (Behr, 2012).
High-resolution CT scan	In idiopathic pulmonary fibrosis, patchy, peripheral bibasilar reticular abnormalities in the subpleural area; with advanced disease, subpleural fibrosis and honeycomb pattern are present.

In the late stages, arterial blood gases help to identify the degree of hypoxemia and carbon dioxide retention. In select cases, bronchoscopy and biopsy may be indicated (Gulati, 2011).

Differential Diagnosis:

- Infectious or neoplastic diseases
- COPD
- CHF
- Wegener granulomatosis
- Goodpasture syndrome
- Bechet disease
- Sjögren syndrome
- Systemic sclerosis
- Pneumoconiosis
- Tuberous sclerosis
- Eosinophilic pneumonia

Treatment: Therapy depends on the cause of disease; specific diagnosis obtained from clinical evaluation, imaging, and lung biopsy; and the disease progression. Occupational exposures should be avoided. Therapy with corticosteroids, cytotoxic agents, and immunosuppressive agents has been the primary treatment for most interstitial diseases. Duration of treatment is still unknown, and objective data to support use of cytotoxics and immunosuppressants is lacking or low quality (Kanaparthi, 2012). Cytotoxic agents, including azathioprine (Imuran) and cyclophosphamide (Cytoxan), are given concurrently with prednisone or in place of it if the patient cannot tolerate high-dose prednisone therapy (Kanaparthi, 2012).

The ATS and other global organizations' official statement on the evidence-based treatment of IPF was issued in 2011 and updated in 2015 (Raghu et al., 2011; 2015), citing the weakness of the evidence for current treatments including corticosteroids, cytotoxics, immunosuppressants, and other miscellaneous drugs, and encouraging individual patient and specialist discussion before implementing any drug therapy. The ATS statement recommends against treatment of IPF with corticosteroids alone or in combination

with cytotoxics or immunosuppressants (Raghu et al., 2015). Nintedanib, a tyrosine kinase inhibitor, and pirfenidone received conditional support for use in the 2015 Update (Raghu et al., 2015). Research indicates that IPF is related more to fibroblastic proliferation than inflammation (Daccord & Maher, 2016; Cerri, Spagnolo, Luppi, & Richeldi, 2012). Lung transplantation is the only treatment to prolong survival in IPF (Puglisi, 2016; King, Pardo, & Selman, 2011); post-transplant the patient will be on immunosuppression for life (Whelan, 2012). Studies using stem cells are ongoing. Prior studies with tumor necrosis factor inhibitors and other atypical drugs have proven unsuccessful (Puglisi, 2016; King et al., 2011). In the end stage of restrictive lung diseases, administer supplemental oxygen for supportive care; consider palliative care and hospice.

Follow-Up: Follow-up visits are scheduled as indicated by symptoms and comorbidities. Periodic chest x-rays or pulmonary function tests (PFTs) may help to chart diseases course and evaluate response to treatment.

Sequelae: Use of corticosteroids or immunosuppressives may result in increased risk of infection. Pulmonary hypertension and right-sided heart failure may occur. Restrictive lung diseases are chronic and there is no known cure.

Prevention/Prophylaxis: Give patients the pneumococcal pneumonia and influenza vaccines. Advise patients to avoid known exposures, tobacco use, and persons with acute, infectious upper respiratory illness.

Referral: Initially refer patients to a pulmonary specialist for bronchoscopy and possible biopsy; thereafter, collaborative management is appropriate. If immunosuppressives are used, refer the patient for initial recommendations and periodic reevaluation.

Education: Teach the patient and family about chronic disease management, regular self-care habits, and early intervention in acute illness. Discuss prognosis and preferred choices for end-stage disease.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Interstitial lung disease diagnosis can be made with a chest x-ray, pulmonary function tests (PFTs) consistent with restrictive lung disease, and typical findings on high-resolution CT. Lung biopsy is necessary in atypical cases.	C	ATS, 2002
No treatment has been shown to have consistent benefit.	C	Reust, 2011
Corticosteroids, supplementary oxygen, and pulmonary rehabilitation may provide symptomatic relief.	C	Reust, 2011
Standard treatment regimens have included corticosteroids plus azathioprine or cyclophosphamide. However, a recent Cochrane review concluded that there is little good-quality information on the efficacy of noncorticosteroid agents for IPF.	C	Davies, Richeldi, Walters, & Davies, 2007
At present, there is no evidence for an effect of corticosteroid treatment in patients with IPF. On the other hand, other fibrotic lung diseases, such as nonspecific interstitial pneumonia (NSIP), are reported to show a better response to corticosteroids. Making a clear distinction between IPF and other entities grouped under the umbrella term interstitial lung disease is, therefore, essential, because this may have therapeutic and prognostic implications.	C	Richeldi, Spagnolo, Davies, et al., 2010

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UPPER RESPIRATORY TRACT INFECTION

Signal Symptoms: Nasal congestion, rhinorrhea/mucopurulent discharge, sneezing, sore throat, cough, headache, malaise, low-grade fever.

Description: Upper respiratory tract infection (URI), most frequently the common cold, is a self-limited infection of the upper respiratory track, usually caused by a virus which

results in inflammation of the nasal passages. Most URIs are self-limiting and accompanied only by minor somatic complaints. In addition to the common cold, acute laryngitis, acute rhinosinusitis, and acute pharyngitis are included as URIs.

Etiology: The majority of URIs are caused by a virus, most commonly rhinovirus, influenza, cornonavirus, and respiratory syncytial virus. However, there are over 200 viruses associated with the common cold and 100 subtypes of the rhinovirus alone. New viruses, including the metapneumovirus and bocaviruses, have recently been identified. There are three modes of transmission: hand to hand (most common), small particle droplet, and large particle droplet. The incubation period is 1 to 3 days, but could last as long as 2 weeks.

Occurrence: URIs are the third most common reason for office visits in the United States, with approximately 500 million noninfluenza viral respiratory infections annually (Sexton & McClain, 2016).

Age: Occur much more frequently in children than in adults and decrease with age.

Gender: Occur equally in men and women.

Ethnicity: Not significant.

Contributing Factors: Risk factors for developing URIs include exposure to infected individuals, psychological stress, lack of sleep, smoking, and contact between nose or conjunctiva and contaminated fingers. Older persons with diabetes contract more frequent URIs than the general population.

Signs and Symptoms: The most common signs and symptoms include nasal obstruction and stuffiness, sneezing, and scratchy throat. Other signs and symptoms include cough, hoarseness, malaise, headache, and fever higher than 100°F (less than1%). Physical examination may reveal mucopurulent nasal drainage, nasopharyngeal mucosal swelling, and lymphadenopathy (Sexton & McLain, 2016). Symptoms lasting more then 7 to 10 days, reports of facial pain with purulent nasal discharge, sudden onset of symptoms, or high fever may be indicative of a bacterial infection (Aring & Chan, 2016).

Diagnostic Tests: No diagnostic tests are indicated for the nonspecific URIs. Diagnosis is clinical based on symptoms. If symptoms persist for more than 7 to 10 days or are indicative of a bacterial infection, an erythrocyte sedimentation rate and CRP may aid in the diagnosis (Aring & Chan, 2016). A rapid strep or culture should be obtained if strep pharyngitis is suspected.

Differential Diagnosis:

- Influenza
- Allergic rhinitis
- Chronic or bacterial sinusitis
- Foreign body
- Streptococcosis
- Catarrhal phase of pertussis (Turner, 2015)

Treatment: URIs usually are managed on an outpatient basis. Patients with significant COPD or cardiac disease should be evaluated on an individual basis. URIs are treated with rest, increased fluid intake, and symptom relief measures, such as humidified air (not recommended for asthma patients). OTC medications may be taken for pain, fever, congestion, or cough relief. A recent Cochrane review found some benefit to various OTC combination agents, including analgesics, antihistamines, and decongestants, in terms of limiting duration and symptoms when compared to no treatment (DeSutter, van Driel, Kumar, Lesslar, & Skrt, 2012). Antihistamine-decongestant combinations were most effective, although more side effects occurred in those who used combination therapy. Controversy still remains about the use of antihistamines for viral illness unless an allergic component has been identified (DeSutter, Saraswat, & van Driel, 2015).

Topical nasal steroids may be used; however, in a recent Cochrane review (Hayward et al., 2015) studies demonstrated no benefit from using them. Topical and oral decongestants are available but topical preparations are preferred, owing to fewer systemic side effects, and should be discontinued after 3 days. Nasal and oral decongestants are associated with elevated BP and should be used cautiously in older adults. A Cochrane review on the use of nasal saline irrigation for acute URIs found limited benefit for symptom alleviation (King, Mitchell, Williams, & Spurling, 2015). Likewise, a Cochrane review of echinacea products for prevention or treatment (limiting duration of symptoms) of URIs was confounded by the variability of echinacea products on the market. There was some evidence that *Echinacea purpurea* administration limited duration of symptoms (Karsch-Völk et al., 2014).

Antibiotics are not indicated for viral URIs (Chow et al., 2012; Institute for Clinical Systems Improvement [ICSI], 2011; Khandelwal, Lathren, & Sloane, 2012; Sexton & McLain, 2016; Spellberg et al., 2011). Guidelines are in agreement that consideration of bacterial illness should be deferred until the patient has had symptoms for 1 week or more that are worsening despite self-care measures. If a reevaluation at that time determines that a bacterial infection is likely, treatment with amoxicillin or amoxicillin clavulanate is instituted. The overprescribing of antibiotics for viral infections is of global concern and should be avoided in URIs. Watchful waiting is often advised before starting antibiotic therapy (Aring & Chan, 2016; Essak & Pignatari, 2013).

Follow-Up: The patient should return if symptoms last more than 7 to 10 days or if he or she develops a high fever associated with systemic symptoms, difficulty breathing, or facial pain with purulent nasal drainage (Aring & Chan, 2016).

Sequelae: Possible complications include lower respiratory tract infection, sinusitis, and aggravation of asthma symptoms. In older individuals with comorbidities, URI may contribute to the exacerbation of other symptoms (e.g., COPD, hyperglycemia, CHF) or may lead to pneumonia.

Prevention/Prophylaxis: Advise the patient to perform frequent proper hand washing, avoid touching the face, and avoid contact with infected people. Pneumococcal and influenza vaccinations are recommended for all older adults.

Referral: Usually neither referral nor consultation is necessary if the patient has an uncomplicated URI.

Education:

Provider Education: Diagnosis of nonspecific URI or acute rhinopharyngitis denotes an infection that is typically viral

and in which sinus, pharyngeal, and lower airway symptoms may be present but not prominent (Sexton & McClain, 2016; University of Michigan Health System, 2011).

Antibiotic treatment of adults with nonspecific URI does not improve illness resolution and is not recommended. There are no studies specifically testing the impact of antibiotic treatment on complications of acute URIs in adults. Life-threatening complications of URIs are rare.

Purulent nasal or pharyngeal secretions (commonly seen in patients with uncomplicated URIs) do not predict bacterial infection and do not benefit from antibiotic treatment (Aring & Chan, 2016).

Patient Education: Explain the disease process, signs and symptoms, and treatment (including side effects of medications). Discuss prevention strategies, including hand washing and when to contact a health-care provider. Educate patients and families about the dangers of antibiotic resistance owing to inappropriate prescribing (Sexton & McClain, 2016; University of Michigan Health System, 2011).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The diagnosis of upper respiratory tract infection is based on clinical signs and symptoms. This is an acute infection that is typically viral in origin and in which sinus, pharyngeal, and lower airway symptoms are present but not prominent.	B C	Wong, 2009 Sexton & McClain, 2016
Purulent nasal discharge or sputum does not predict bacterial infection or benefit from antibiotics.	A C	Wong, 2009 Aring & Chan, 2016
Antibiotics are ineffective for the treatment of the common cold in children and adults.	B C C	Wong, 2009 Sexton & McClain, 2016 Aring & Chan, 2016
Antihistamine and decongestant combinations may help alleviate nasal symptoms in older children and adults. Newer-generation nonsedating antihistamines are ineffective.	B C	Wong, 2009 Sexton & McClain, 2016

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VALVULAR HEART DISEASE

Signal Symptoms: Asymptomatic in early stage; fatigue and dyspnea are common later.

Description: Valvular heart disease (VHD) is damage to a valve or valves of the heart, causing cardiac dysfunction. The most prevalent types of VHD in elderly persons are the result of calcific and degenerative valve disease. The most prevalent VHD disorders in the elderly are aortic stenosis and mitral regurgitation. VHD may be defined in terms of stages of disease progression from A to D. Patients with stage A VHD are defined as at risk for development of the disease; stage B VHD is defined as progressive mild to moderate disease but asymptomatic; stage C is defined as severe asymptomatic and may be further stratified into C1 and C2, with C1 representing compensation by the ventricle and C2 representing decompensation; and stage D is defined as severely symptomatic. As defined in the stages, patients may be asymptomatic in early stages, but fatigue and exertional dyspnea are common (Nishimura et al., 2014).

Aortic Stenosis

In aortic stenosis (AS), there is obstruction of the left ventricular outflow tract, which may be supra- or sub-valvular. AS is graded as mild, moderate, severe, or critical as defined by aortic gradient and area. Mild AS is defined as aortic valve area of 1.5 cm^2 and mean pressure gradient of less than 20 mm Hg; moderate AS is defined as aortic valve area of 1.0 to 1.5 cm^2 and mean pressure gradient of 20 to 40 mm Hg; severe AS is defined as aortic valve area less than 1 cm^2 and mean pressure gradient greater than 40 mm Hg; and critical AS is defined as aortic valve area less than 0.5 cm^2/m^2 BSI and mean pressure gradient of greater than 80 mm Hg. The hemodynamic hallmark of AS is pressure overload, left ventricular hypertrophy with a resultant gradient.

Etiology: AS diagnosed before age 60 years is usually caused by a congenital bicuspid valve, whereas AS after age 60 years is usually caused by calcific degeneration of the valve. Occasionally, a patient after 60 years of age may present with a

calcific bicuspid or unicuspid aortic valve. Other less common causes of AS include rheumatic fever, endocarditis, systemic lupus erythematous, and Fabry's disease.

Occurrence: In the absence of a congenitally malformed valve, occurrence increases with age due to focal thickening or calcification of the valve. At the time of surgical intervention, 40% of patients older than 70 years will be noted to have a bicuspid valve, while it will be much higher in the age group younger than 70 years (Roberts & Ko, 2005).

Age: Congenital valvular heart disease is present at birth; acquired valvular heart disease is found in older age.

Gender: More prevalent in males.

Ethnicity: Not significant.

Contributing Factors: Factors associated with degenerative aortic valve stenosis include age, male gender, current cigarette smoker, high serum concentrations of lipoprotein (a), and history of hypertension (Mohty & Pislaru, 2016).

Signs and Symptoms: Many of these patients are asymptomatic. Prognosis is poor for patients who exhibit symptoms. The triad of symptoms that may be associated are syncope, angina, and dyspnea (Otto, 2016). The survival rate decreases to 2 to 3 years for the patients who exhibit symptoms (Yeo & Low, 2007) and do not have appropriate intervention. Angina is an early and more common symptom and can occur in the presence or absence of CAD. Presyncope followed by effort syncope occurs in about one-third of the patients with symptoms and is related to a fixed cardiac output due to the obstruction of the left ventricle created by AS. Exertional dyspnea indicates left ventricular dysfunction and heart failure.

Physical Examination Findings: The classic crescendo-decrescendo systolic murmur is heard at the second right intercostal space and radiates to the carotids. The murmur in the elderly may radiate to the apex (Soriano, Fernandez, Cassel, & Leipzig, 2007). This murmur, which peaks in intensity in mid to late systole, may also be associated with a thrill if greater than grade IV. The murmur intensity does not correlate with the severity of AS; often in the setting of heart failure the AS murmur may become softer as the cardiac output falls. S_1 is often soft, and the aortic component of S_2 is soft or absent. The S_2 may also be paradoxically split due to the late closure of the aortic valve. An S_4 is common, representing forceful atrial contraction in the setting of left ventricular hypertrophy. Other findings may include delayed carotid upstroke, a carotid and apical pulse lag time, systemic hypertension, prominent "A" waves in the jugular venous pulse, and a sustained and forceful apical impulse (O'Gara & Loscalzo, 2015).

Diagnostic Tests: In AS, the EKG is abnormal in most cases, demonstrating QRS or T-wave changes reflecting left ventricular hypertrophy. Chest x-ray examination is not recommended in routine screening of asymptomatic patients, but may provide information on valve calcification, cardiac chamber sizes, and pulmonary vasculature. Chest films may show cardiac enlargement when heart failure is advanced.

Echocardiography is the standard test for assessing aortic valve stenosis and may demonstrate thickening and calcification of the aortic valve with decreased mobility of the leaflets. Doppler measurement of intracardiac blood velocity can help determine hemodynamic severity. The echocardiogram is useful for providing detail on the valve morphology. Useful questions that can be answered include, but are not limited to: Is there congenital absence of valve cusp, degree of valve calcification, valve cusp mobility, aortic valve area, aortic valve pressure gradient, size of aortic root, left ventricular size, ejection fraction, posterior and septal left ventricular wall thickness, and concomitant valvular dysfunction and estimation of pulmonary artery pressures?

The frequency of echocardiogram assessment of AS is dependent on the degree of severity and symptoms. Exercise testing has limited utility in the evaluation of patients with AS. Pharmacological echocardiogram may be done in patients with low gradient AS and can help to risk stratify patients in this category. Cardiac catheterization measurement of the systolic pressure gradient across the aortic valve is the definitive method for assessing AS in patients being considered for surgery or when there is a discordance between the clinical findings and the diagnostic tests. In patients with congenital AS there may also be abnormalities noted in the coronary arteries, which can be evaluated with the left heart catherization. Right heart catherization will provide information on hemodynamics. Cardiac MRI may be used to stratify patients with AS and assess the effect of chronic left venticle pressure overload, volume, and overall function.

Treatment: No medical therapies are available to delay the progression of AS. The patient with asymptomatic AS will need to be frequently monitored for the development of symptoms and progression of disease (Bonow et al., 2008; European Task Force, 2007). Symptoms of aortic stenosis (angina, heart failure, syncope) are associated with substantial valvular obstruction and a risk of sudden death; therefore, surgical management is necessary. Aortic valve replacement is associated with a higher mortality rate (9% to 12%) in older adults than in younger individuals. Factors associated with greater operative risk include emergency surgery, left ventricular dysfunction, right-sided heart failure, female gender, significant coronary disease, cachexia, additional valve replacement, renal insufficiency, or concomitant CABG. The proper selection of the type of valve is important in older adult patients. The bioprosthetic valves have fewer structural failures and are advantageous in that their use obviates the need for long-term anticoagulation (which is associated with substantial morbidity and mortality in older adults). The disadvantage of this valve type is that the tissue degrades and many patients may require reoperation in 10 years. Mechanical valves are more durable and have better hemodynamic profiles, but these require lifelong anticoagulation therapy.

Aortic balloon valvuloplasty should be considered as an alternative method of treatment; however, because it is associated with rapid restenosis and significant residual outflow obstruction, it is reserved as a palliative procedure for the symptomatic patient who is not a surgical candidate or as a bridge to surgery.

Secondary to advanced age, 30% of the patients with symptomatic, severe AS are not able to undergo surgery to replace the aortic valve. There has been a rapid growth in the use of a new procedure called transcatheter aortic valve implantation. This procedure involves implanting a bioprosthetic

valve within the diseased aortic valve through a catheter. The option of this procedure is becoming more common in cardiac centers across the nation. Early results from the Transcatheter Valve Therapy Registry indicate decrease in 30-day operative mortality and 1 year mortality (Grover et. al, 2017).

The AHA and ACC 2014 guideline recommendation for AS intervention include:

CLINICAL RECOMMENDATION	RECOMMENDATION	REFERENCES
Surgical AVR in patient with low or intermediate surgical risk.	A	Nishimura et al., 2014
Transcatheter aortic valve replacement (TAVR) for patients who have a prohibitive surgical risk and a predicted post TAVR survival greater than 12 months.	A	Nishimura et al., 2014
TAVR for patient who have high surgical risk.	B	Nishimura et al., 2014
TAVR is not recommended in patients in whom comorbidities would preclude the expected benefit from treatment of AS.	B	Nishimura et al., 2014
Balloon aortic valvuloplasty (BAV) as a bridge to surgical AVR or TAVR in severely symptomatic patients.	C	Nishimura et al., 2014

Adapted from Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP, Guyton RA, et al. 2014 AHA/ACC Guideline for the Management of Patients with Valvular Heart Disease: Executive Summary: A report of the American College of Cardiology/American Heart Association Task Force of Practice Guidelines. *Circulation.* 2014 June 10. 129 (23): 2440-92.
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Other Class I recommendations for AVR in patients with AS include: patients with high gradient AS who have symptoms by history or on testing (stage D1), asymptomatic patients with severe AS (stage C2) and left ventricular fraction less than 50%, and patients with severe AS (stage C or D) when undergoing other cardiac surgery (Nishimura et. al, 2014).

Mitral Regurgitation

Mitral regurgitation (MR) is retrograde blood flow during systole from the left ventricle into the left atrium through an incompetent mitral valve. The mitral valve apparatus consists of mitral valve annulus, chordae tendineae, and papillary. Distortion of either of these structures may lead to an incompetent mitral valve and thus mitral regurgitation. MR is categorized by grades A to D. Grade A is patients at risk for MR, grade B is progressive MR, grade C is asymptomatic severe MR, and grade D is symptomatic severe MR. The progression of untreated disease may lead to pulmonary hypertension and a failing left ventricle.

Etiology: Some of the common causes of MR are rheumatic heart disease, mitral valve prolapse, and ischemic heart disease (Maganti, Rigolin, Sarano, & Bonow, 2010). In the older adult population, the most common causes of significant MR are myxomatous degeneration and ischemic heart disease. Congenital MR is rare, but may be seen with cleft mitral valve in persons with Down's syndrome. MR may be further delineated as acute or chronic. Common causes of acute MR include chordal rupture, papillary muscle rupture, leaflet perforation, and trauma. Common causes of chronic MR include rheumatic fever, mitral leaflet prolapse, CAD, papillary muscle dysfunction, congenital heart disease, left ventricular dilatation, myxomatous degeneration, and hypertension.

Signs and Symptoms: The symptoms associated with MR can be quite variable from a state of asymptomatic to symptoms of left ventricular dysfunction, shortness of breath, paroxysmal nocturnal dyspnea, fatigue, as well as concomitant right-sided heart failure.

Physical Examination Findings: The murmur of MR is a holosystolic murmur heard at the apex and may radiate to the axillae and may be associated with an S_3 gallop. Due to the dilatation of the left atrium, may of the patients will have AF. Other examination findings may include edema, displaced apical impulse, and crackles on lung examination (Maganti et al., 2010).

Diagnostic Tests: In MR, the EKG shows left atrial enlargement or AF and the chest x-ray examination shows left ventricular dilation; in nonrheumatic forms of MG these are less distinctive. Echocardiography delineates overall ventricular function and Doppler studies show the jet and severity of the regurgitation, as well as the mechanism. Transesophageal echocardiography is a more precise way of visualizing the regurgitant jet.

Treatment: Medical management of MR includes use of ACEIs, digitalis, diuretics, and vasodilators to reduce the symptoms of heart failure and reduce the regurgitant volume. Anticoagulation may be needed if the patient has AF. Mitral valve surgery is considered in asymptomatic and symptomatic patients with progressive disease before signs of irreversible left ventricular dysfunction are present. The mortality rate

associated with mitral valve replacement could be as high as 14% in the elderly patient. Mitral valve repair is associated with a lower operative mortality than mitral valve replacement and is preferred, as preservation of the existing valve architecture allows synchrony of left ventricular contraction. When mitral annular calcification is the cause, medical therapy is prudent because the operative risk is substantially higher in patients with this disease process. Acute MR from papillary muscle rupture or chordal rupture requires patient stabilization followed by surgery, which still carries a high mortality rate. Other therapeutic options for repair or the mitral valve may include MitraClip (O'Riordan, 2013).

The AHA and ACC 2014 guideline recommendations for MG intervention include:

CLINICAL RECOMMENDATION	RECOMMENDATION	REFERENCES
MV surgery is recommended for symptomatic patients with severe primary MR (Stage D) and ejection fraction greater than 30%.	B	Nishimura et al., 2014
MV surgery is recommended for asymptomatic patients with severe primary MR and left ventricular dysfunction (ejection fraction 30% to 60% and/or systolic diastolic dimension greater than 40 mm.	B	Nishimura et al., 2014
MV repair is recommended when procedure is limited to posterior leaflet in the setting of chronic severe primary MR.	B	Nishimura et al., 2014
MV repair is recommended over MV replacement in patients with chronic MR involving anterior or posterior leaflets.	B	Nishimura et al., 2014
If patient undergoing other cardiac surgery and has chronic primary MR, they should also have MV repair or replacement surgery at the same setting.	B	Nishimura et al., 2014

Adapted from Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP, Guyton RA, et al. 2014 AHA/ACC Guideline for the Management of Patients with Valvular Heart Disease: Executive Summary: A report of the American College of Cardiology/American Heart Association Task Force of Practice Guidelines. *Circulation.* 2014 June 10. 129 (23): 2440-92.
A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

General Principles

Diagnosis: Often the first indication of VHD may be the auscultation of a cardiac murmur. It is important for advanced practice nurses to have good cardiovascular assessment skills. Auscultatory and other associated findings are listed in the table that follows and can be a useful guide in approaching the cardiovascular examination of all patients.

VALVULAR HEART DISORDER	MURMUR	OTHER CARDIAC FINDINGS
Aortic stenosis	Systolic murmur heard at second right intercostal space and may radiate to carotids	Decreased cardiac upstroke. Best heard with patient sitting up and exhaling.
Aortic regurgitation	Diastolic blowing murmur at left sternal border	Rapidly collapsing pulse (Corrigan's pulse), preservation of A2 component of second heart sound, widened pulse pressure.
Mitral stenosis	Diastolic rumble	May be associated with signs of right heart failure including edema, ascites, elevated jugular venous pressure.
Mitral regurgitation	Holosystolic murmur heard at apex and may radiate to axilla	AF, ventricular gallop (S_3), displaced apical impulse laterally, crackles on lung examination.
Mitral valve prolapse	Mid-systolic click heard at apex	May be associated with mitral regurgitation.
Tricuspid stenosis	Mid-diastolic rumble heard at left sternal border; may increase with inspiration	Very rare finding but may be associated with right atrial myxomas or carcinoid syndrome.
Tricuspid regurgitation	Holosystolic murmur heard at right sternal border and increase with inspiration	May be associated with signs of right heart failure including edema, ascites, elevated jugular venous pressure.

VALVULAR HEART DISORDER	MURMUR	OTHER CARDIAC FINDINGS
Pulmonic stenosis	Harsh mid-systolic murmur heard at the 2nd left interspace and may radiate to the left carotid	May be associated with a palpable thrill. The second heart sound is widely split.
Pulmonic regurgitation	High-pitched diastolic murmur	Accentuated P2 of the second heart sound. May be louder in setting of pulmonary hypertension.

Follow-Up: For patients treated medically for valvular disease, close follow-up to monitor the effectiveness of treatment, adverse effects of medication, and progressiveness of the disease process is indicated. Medication therapy, particularly the use of anticoagulation therapy, requires meticulous attention. Surgically treated patients are monitored for valve function, fluid balance, and anticoagulation. Periodic echocardiographic, electrocardiographic, and chest x-ray monitoring may be indicated.

Sequelae: Untreated VHD may lead to progressive heart failure, dysrhythmias, and death. Valve replacement risks include thrombus formation, infection, or rupture at the attachment points to the valve ring. Infective endocarditis, which may occur with artificial valves, has a high risk of mortality and requires reoperation. There is a high prevalence of gallstones in patients with prosthetic valves, thought to be due to low-grade intravascular hemolysis. In patients who are not surgical candidates, the symptoms of heart failure are progressive and disabling. Even in surgical patients, the symptoms of heart failure may recur or persist.

Prevention/Prophylaxis: In patients with prosthetic valves, the risk of thromboembolism decreases with an individualized antithrombotic regimen. The specific therapy is determined based on the comorbid state and the patient's overall status. Consider prophylactic antibiotic therapy (endocarditis prophylaxis) before any surgical or dental procedures in all patients with valvular disease, especially patients with valve replacement, rheumatic heart disease, aortic regurgitation, or mitral valve prolapse with significant MR murmurs.

Referral: All patients with symptoms of progressive valvular disease must be managed collaboratively with the physician. Many require further collaboration with a cardiologist and or cardiac surgeon. The Practice Guidelines for Cardiothoracic Surgery Concerning Valvular Heart Disease include the indications for surgery. In general, these include symptoms that cannot be controlled with medical therapy or indications of a threat to survival (i.e., angina, dyspnea, effort syncope or progressive impairment of ventricular contractility, and infective endocarditis).

Education: Older adults constitute 40% to 60% of all cases of endocarditis. Instruct all at-risk patients in the importance of good oral hygiene and antibiotic prophylaxis. Patients should be taught to monitor and report febrile illness.

Teach all patients with valve disease requiring medication therapy to report lack of therapeutic effect or any adverse effects of the drugs. Teach patients to be aware of drug-food interactions (e.g., green leafy vegetables and anticoagulants). Teach patients to have prothrombin time/INR checked on a regular basis if they are taking anticoagulant medications. Teach the patient with disabling heart failure about energy-conservation measures. Patients with hemodynamically significant valvular heart disease may need to limit vigorous physical activity. Deterioration may be rapid and symptoms insidious, so patients are taught to report any changes in condition.

CASE STUDY

You are assigned to see R. G., a 66-year-old man known to the practice where you have your clinical experience. When you go to review his chart it is in the inactive file because he has not been seen for more than 7 years. His last visit was for bronchitis, and he was prescribed albuterol metered-dose inhaler (MDI) as needed. He was given laboratory slips for a complete metabolic profile, EKG, and PFTs, but there are no results on the chart. A follow-up postcard was sent to him 2 months after the visit.

On review of his chart you note that he has never come for a comprehensive visit, only episodic problems. He was diagnosed with hypertension 10 years ago and prescribed hydrochlorothiazide, 12.5 mg orally once daily; his last refill was 2 years ago. History is sketchy since he was seen mainly for sick visits. Here is what you can obtain from the record:

Divorced; worked as a carpenter, also did handyman jobs

Last insurance through carpenters' union

Significant family history: Father died from heart problem at age 42; mother died from lung cancer at age 60; younger brother has type 2 diabetes mellitus, heart disease

Smoker, 1-1/2 packs/day since age 15 years

Occasional alcohol; during military service drank four six-packs of beer every weekend

Denies any drug use, uses OTC Advil for "aches and pains on the job"

Reported that he had a tetanus booster in the emergency department 8 years ago for a work-related injury

Last visit: BP 130/84, heart rate (HR) 76, respiratory rate (RR) 18, BMI 28

Continued

CASE STUDY—cont'd

1. How will you use this information to prepare for today's visit?
   Today's Visit:
   Chief Complaint: "I'm really feeling my age these days. There are times when I feel like I can't catch my breath. [Pause to breathe] I know I've put on a few pounds but I didn't think I'd feel so winded. This retirement is for the birds; I can't seem to get into the groove." [Pause to breathe]
   Objective: BP 160/92, HR 110, RR 28, BMI 27
   66-year-old Caucasian male, ruddy complexion, fingertips yellowed, prominent sternocleidomastoids, looks older than stated age
   Diminished breath sounds, crackles at bases of lungs bilaterally
   Heart sounds distant, possible S4
   Feet cyanotic when dependent, +2 edema bilaterally
2. What additional subjective data are you seeking?
3. What additional objective data will you be assessing for?
4. What national guidelines are appropriate to consider?
5. What tests will you order?
6. What are the differential diagnoses that you are considering?
7. What is your plan of care?
8. Are there any *Healthy People 2020* objectives that you should consider?
9. What additional patient education may be needed?
10. Will you be looking for a consultation?

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CHAPTER 9

Peripheral Vascular Disorders

Catherine Ratliff and David Strider

ASSESSMENT

A comprehensive vascular assessment may be integrated into the general history and physical examination for the patient with vascular disease. A systematic head-to-toe examination, done consistently for each vascular patient, will minimize the chance of missing subtle signs related to vascular deficits.

The clinician should begin with auscultation of the carotid arteries bilaterally. Cardiac murmurs will usually radiate into the carotid arteries, and an underlying bruit may not be discerned. The subclavian arteries should then be auscultated over the upper anteromedial chest area. During this phase of the examination, the jugular veins should be inspected for jugular vein distention. This can best be appreciated when the patient is lying down with the head of the bed elevated 15 degrees.

The clinician should proceed to the palpation of the brachial, radial, and ulnar arteries. Such palpation should be scored on a scale of 0 to 4 (4 = pulsatile, bounding; 3 = easily palpable with full triphasic pulse; 2 = biphasic pulse that may be a bit harder to locate initially; 1 = monophasic pulse that may come and go depending on position of fingertips; and 0 = absent pulse by palpation, which may be detected by Doppler ultrasound). All palpable pulses should be graded in the assessment and documented.

The clinician should evaluate any pain, coolness, paresthesia, motor weakness, discoloration, or tissue loss in the hands. Careful inspection of the distal fingers and the nail beds may reveal signs of end tissue malperfusion. The finger joints should be inspected for cyanosis and/or petechiae. The fingernails should be checked for clubbing, which portends chronic hypoxemia. Routinely, in any patient confirmed or suspected to have vascular disease, blood pressure should be checked in both arms at this point.

The heart should then be auscultated to permit delineation of rhythm regularity, S_1, S_2, and any extrasystolic sounds such as S_3, S_4, or pericardial rub. Murmurs should be noted and classified as per the cardiac assessment guidelines. The abdomen should be auscultated for celiac artery, superior mesenteric artery, and renal artery bruits, which are best heard 4 to 8 cm above the umbilicus and 1 to 2 cm to the right and left of midline abdomen. Then the clinician should listen to the femoral artery in each groin for the presence of bruits. Following this, the abdomen should be lightly palpated just to the left of midline, approximately 4 cm above the umbilicus, to evaluate for a pulsatile abdominal aorta. In slender individuals, the abdominal aorta is often fairly easy to palpate. A bounding, pulsatile abdominal aorta in an individual may suggest aneurysmal formation. The clinician should note and document any tenderness or guarding associated with the abdominal palpation. There are many other causes for abdominal tenderness that the clinician should be aware of, in addition to arterial and venous disease, to include pancreatitis, liver contusions, cholecystitis, appendicitis, splenic infarction, pyelonephritis, cystitis, gastroenteritis, peptic ulcer, and small bowel obstruction.

Once the abdominal examination is complete, the clinician should progress to the vascular assessment of the lower extremities. The femoral arteries should be palpated over the groin area and documented on the scale of 0 to 4. Any pulse deficits should be followed up with a Doppler examination. The popliteal fossa should be inspected and palpated bilaterally in order to locate the popliteal artery and assess for the possibility of popliteal artery aneurysms. The clinician should inspect the calves and feet for edema, skin turgor, and skin pigmentation. Thick, hyperpigmented skin in the lower calves and feet may be an indication of chronic venous insufficiency, whereas thin, shiny, pale skin is more consistent with peripheral arterial insufficiency. The dorsalis pedis and posterior tibial pulses should be palpated. If those pulses are not palpable, Doppler ultrasound should be used. Any Doppler signals should be located and identified with an indelible marker pen. At this time, the heel, the medial and lateral malleoli, and the toes of each foot should be inspected for breakdown. As in the hands, any pedal pain, coolness, paresthesia, motor weakness, discoloration, or tissue loss should be documented. Dorsal and plantar flexion of the feet should be assessed and recorded with muscular strength graded on a scale of 1 to 5.

The vascular examination then turns to a careful evaluation of the cranial nerves (CNs), usually focusing on CN II to CN XII. Evaluation of pupillary light and accommodation response (CN II and III); following the eyes in a lateral, medial, superior, and inferior gaze (CNs III, IV, and VI); and evaluation of facial smile, scowl, and opening eyes against

pressure (CNs V and VII) should be completed. Gross hearing assessment, with follow-up by Weber and Rinne testing, may ascertain any CN VIII deficits. Evaluating the tongue protrusion, uvula rise, cough reflex, gag reflex, and shoulder shrug will provide information on the integrity of CNs IX, X, XI, and XII. Document the conformation of the uvula, which may be bifid or singular (some connective tissue disorders have associated bifid uvula).

Completion of the CN examination provides the healthcare team with an excellent neurological baseline before interventions for carotid artery disease. Assessment findings related to vertebrobasilar insufficiency may include balance problems, dizziness, and coordination deficits. Focal neurological deficits such as arm, leg, and/or facial weakness on one side of the body; loss of speech; difficulty speaking or swallowing; and unilateral or partial vision loss signify transient cerebral ischemia that may be related to severe carotid artery stenosis (Dua, Romanelli, & Upchurch, 2016).

Following the neurological examination, the clinician should ascertain any specific end organ perfusion deficits, as shown by the following signs and symptoms:

- Pain in the feet, calves, thighs, and or buttocks, either at rest or with ambulation (peripheral arterial disease [PAD])
- Any open wounds or blisters on the distal aspects of feet and/or hands (PAD or chronic venous stasis ulcers)
- Abdominal pain, nausea, vomiting, and/or diarrhea after a meal (mesenteric ischemia)
- Persistent abdominal/back pain (abdominal aortic aneurysms or dissection)
- Acute unilateral CN deficits (carotid artery disease)
- Acute balance/coordination deficits (possible vertebrobasilar malperfusion)
- Acute chest and/or back pain (after ruling out myocardial ischemia and pulmonary embolus, one needs to quickly and thoroughly evaluate the patient for thoracic aortic dissection and/or expanding aneurysm)

Once the focused vascular assessment and abbreviated history are completed, the clinician will have achieved a very sound baseline with which to guide future interventions to delineate and treat vascular lesions.

ABDOMINAL AORTIC ANEURYSM

Signal Symptoms: Persistent or intermittent pain in the middle or lower abdomen, often radiating to the lower back, which is characteristic of a rapidly expanding, leaking, or ruptured abdominal aortic aneurysm (AAA). Most AAAs are asymptomatic.

Description: The abdominal aorta is the large artery that provides blood to the digestive organs, liver, kidneys, spleen, and lower extremities. It extends inferiorly from the descending aorta to the iliac arteries. An AAA is a dilation of the abdominal aorta that is one and one-half to two times greater than the size of the nondilated proximal or distal aorta. The AAA involves all three layers of the arterial wall.

Etiology: Most AAAs are atherosclerotic in nature; other causes include trauma, infection, and inflammation. Connective tissue disorders, such as Ehlers-Danlos syndrome and Marfan syndrome, predispose the patient to AAA formation (Strider et al., 2013; Yip & Sawatxky, 2014). Most AAAs are infrarenal (65%), occurring below the renal arteries. Patients with AAAs are more likely to have arterial aneurysms in other locations such as the thoracic aorta, the common iliac arteries, the common femoral arteries, and the popliteal arteries.

Occurrence: AAAs are the thirteenth leading cause of death in the United States. Mortality rates for ruptured aneurysms are 70% to 90%, compared with 5% operative mortality for elective open surgical repair and 2% to 3% for endovascular stent AAA exclusion.

Age: More frequent in adults over 50 years old; prevalence rate is 2% to 4%.

Gender: Onset occurs around age 50 years for men and 60 years for women. Incidence steadily increases with age and peaks at age 80 years. AAA is five times more likely in men than in women.

Ethnicity: There is no dominant ethnic group that develops AAA, but there is a familial history associated with AAA development.

Contributing Factors: Risk factors for developing AAA include arteriosclerotic heart disease and arteriosclerotic changes of other vessels, smoking history, hypertension, chronic obstructive pulmonary disease, obesity, family history of AAA, diabetes mellitus, cystic medial necrosis, Marfan syndrome, and previous spinal cord injury (Schermerhorn et al., 2015; Sodem, Zetterwal, & Ultee, 2016). Aortic dissection involves a splitting apart of the aortic wall layers, such that two or more blood flow channels are created within the aorta. Acute aortic dissection may produce intimal flaps that occlude major take-off vessels, leading to end-organ ischemia. Progressive dissection may lead to weakening of the entire aortic wall, resulting in aneurysm formation and possible rupture. Isolated celiac and/or superior mesenteric artery dissection may occur, although this is much less likely than the initial aortic dissection (Dimusto, Oberdoerster & Criado, 2015).

Signs and Symptoms: Most patients with AAAs are asymptomatic (66% to 75%), except in the presence of dissection, rupture, or impending rupture. A pulsatile abdominal mass at or slightly above the umbilicus in the epigastrium, as noted by palpation of the abdomen, is consistent with but not conclusive for an AAA. These are easier to palpate in thin individuals; however, if noted in an obese individual, they are usually quite large. AAA should be suspected in individuals with a femoral or popliteal aneurysm, because 30% of individuals with a peripheral aneurysm also have an AAA. In patients with symptomatic AAAs, the complaints include mild-to-severe abdominal, flank, or lower back pain. Other symptoms may include nausea and vomiting, gastrointestinal bleeding, and lower extremity ischemia. Rupture is the most lethal clinical presentation, with symptoms including sudden onset of

severe abdominal and back pain, hypotension, and the presence of a pulsatile, expanding mass. There is a high mortality rate associated with ruptured AAAs; therefore, the possibility of ruptured AAA in the differential diagnosis should be considered in any adult patient with acute abdominal or flank pain (Schermerhorn et al., 2015).

Diagnostic Tests: The best initial screening test is an ultrasound of the abdominal aorta. In cases where surgery is planned, angiography and computed tomography (CT) scan with IV contrast are indicated to size the aneurysm and diagnose any tears or perforations.

Differential Diagnosis: It is important to evaluate for and treat hypertension and heart disease, because basic and early steps in AAA management include blood pressure control and recognition or enhancement of cardiac function. An ectatic abdominal aorta without aneurysm may be palpated and confused with AAA. Other acute causes of abdominal and back pain may mimic a ruptured AAA and must be ruled out. Major imaging modalities for a patient with suspected or known AAA include abdominal ultrasound, CT scan, magnetic resonance angiogram (MRA), and angiogram.

Treatment: The rate of AAA growth for an individual is unpredictable. Some aneurysms may remain stable for long periods, whereas others may enlarge quickly. Medical management of an asymptomatic, small AAA should include blood pressure control, regulation of heart rate, and smoking cessation. Beta blockers have been shown to slow the long-term growth of aortic aneurysms. Angiotensin receptor blockers (ARBs), such as losartan, may also have a major role in critical blood pressure and heart rate control for patients with aortic aneurysms and/or dissections.

Initial treatment for symptomatic AAA, whether ruptured or not, involves aggressive blood pressure control. Mean arterial pressure should be maintained between 60 and 70 mm Hg, and systolic blood pressure should be kept between 100 and 120 mm Hg. Arterial pressure monitoring is recommended, and IV antihypertensive agents, such as esmolol, nicardipine, nitroprusside, labetalol, and/or nitroglycerin, should be used to rapidly and consistently maintain blood pressure in these ranges. Cardiology should be consulted, and a transthoracic echocardiogram should be done to assess heart function. If the patient is to be medically managed, transition to oral antihypertensive agents should begin, using beta blockers, calcium channel blockers, clonidine, ARBs, and labetalol as needed to maintain very tight blood pressure control.

The two invasive options for excluding an AAA when it becomes symptomatic or becomes more than 5 cm in diameter include open surgical repair and endovascular repair. Elective operative repair has a mortality rate of 2% to 5%. Urgent repair (where cardiac and other known risk factors have not been optimized) of an intact symptomatic AAA has a mortality rate of 15% to 20% (Cooper, 2014; Schermerhorn et al., 2015). In emergent repair in which the AAA has ruptured, there is a 50% mortality rate in patients who reach the hospital. Average postoperative length of stay for an elective AAA surgical repair is 5 days. The major cause of death after AAA repair is myocardial infarction. Other complications include renal failure, limb ischemia, ischemic colitis, hemorrhage, pneumonia with prolonged ventilator dependence, and paraplegia (Buck, Ultee & Zetterwall, 2016; Yiu & Cheesy, 2016).

Endovascular grafts are a more recent addition to the treatment modalities for elective AAA repair. The graft is deployed through the femoral or the iliac artery. Not all AAAs are anatomically appropriate for stenting. Experimental AAA stent trials began in 1993 in the United States. Current success rates are higher than 90%, with a mortality rate of less than 2%. Most patients are discharged from the hospital in 1 to 2 days (Gordon & Toursarkissian, 2014). Complications after endovascular treatment may include arterial injury at site of access, arterial embolization, endoleak (blood flow outside the lumen of the graft but within the original aneurysmal sac), post-implant syndrome (back pain and fever without elevated white blood cell count or other signs of infection), and graft limb distal thrombosis (Schermerhorn et al., 2015).

Follow-Up: Initial management of patients with small aneurysms (fewer than 4 cm) should include serial evaluation with ultrasound every 3 to 6 months. Any symptomatic AAA or any asymptomatic AAA larger than 5 cm in diameter should be repaired using either open surgical or endovascular technique (Sodem, Zetterwall, & Ultee, 2016; Cooper, 2014).

Sequelae: The most common complication of AAA is rupture. Infrequent complications are thrombi to the lower extremities and preexisting infection of the aneurysm (referred to as a mycotic aneurysm), with *Salmonella* and *Staphylococcus aureus* being the most commonly identified organisms. Fungal infections of the aorta pose significant challenges and require long-term IV antimycotics, such as fluconazole after 6 weeks of an IV course of antifungal agents, until a definitive surgical repair is done.

Prevention/Prophylaxis: Because most AAAs are atherosclerotic in nature, the same long-term preventive measures for reducing coronary artery disease should be applied. These include lifestyle modification, control of hypertension, beta blockade and/or ARB therapy, statin therapy, daily aspirin, and maintenance of euglycemia if diabetic (Sodem, Zetterwall & Ultee, 2016). Early abdominal ultrasonographic screening is recommended for first-degree relatives of individuals who already have been diagnosed with an AAA. The prevalence of AAA in the first-degree relatives of affected families is reported to be 15% to 33%. Large studies in Western Europe and North America support the selective ultrasonographic screening of men over age 65 years for AAA and all women and men over the age of 50 years if a first-degree relative has been diagnosed with an AAA (Schermerhorn et al., 2015).

Referral: Referral to a vascular surgeon is essential for all patients with symptomatic AAAs. Furthermore, any patient with a known AAA, even if not symptomatic, should be screened with serial ultrasound imaging at least once a year.

Education: Patients and their families should be taught the importance of follow-up; ways to manage hypertension, hypercholesterolemia, smoking, and other risk factors; and the signs and symptoms that should be reported to the physician immediately (sudden onset of abdominal or back pain, dizziness). The risks of operative versus nonoperative treatment should be explained thoroughly to the patient and his or her family.

CHRONIC LYMPHEDEMA

Signal Symptoms: Swelling of the affected body part, usually the limb, because of impaired flow of lymph fluid.

Description: Lymphedema is a progressive condition of abnormal lymph accumulation in the interstitial tissues due to a disruption of lymph flow. The lymph system is a low-pressure system that includes capillaries and depends on muscle movement to advance the fluid to the vascular system. Most of the lymph drains into the thoracic duct on the left side of the body and into the brachiocephalic vein.

Etiology: Lymphedema is swelling caused by impaired lymph transport, which results in an accumulation or pooling of protein-rich fluid in the interstitial space. Lymphedema is most commonly caused by surgery, radiation, or infection that damages the lymphatic system.

Occurrence: Lymphedema may be classified as primary (idiopathic) or secondary (acquired). Primary lymphedema is congenital absence or abnormalities of the lymphatic system. It affects 1 to 2 million individuals in the United States. Secondary lymphedema affects 2 to 3 million individuals in the United States (Ratliff, 2016). One of the most common causes of secondary lymphedema in the United States is related to treatment of cancer.

Age: Not confirmed as a risk factor for lymphedema.

Gender: With primary or congenital lymphedema, females are affected twice as often as males. With secondary lymphedema in the United States, females may be affected more often, but that is because a common cause of secondary lymphedema is breast cancer, which is primarily a woman's disease.

Ethnicity: Not significant.

Contributing Factors: The types of cancer associated with secondary lymphedema include breast cancer, gynecological cancer, lymphoma, melanoma, and urological cancers. Surgery, radiation, trauma, infection, malignancy, or scar tissue can cause secondary lymphedema by obstructing the lymphatic system. For example, patients with a history of breast cancer with axillary node dissection or radiation, groin node dissection or radiation, and a postoperative infection have an increased risk for developing lymphedema. Morbid obesity can also increase the rate of secondary lymphedema (Ratliff, 2016).

Signs and Symptoms: Swelling or edema is the characteristic symptom of lymphedema. Additional symptoms may include heaviness or tightness, aching and fatigue in the affected limb, restricted range of motion, recurring cellulitis in the affected extremity, and hardening and thickening of the skin. The Stemmer sign (or Kaposi-Stemmer's sign) is another clinical indication of lymphedema in which one is unable to pinch a fold of skin at the base of the second toe on the dorsal aspect of the foot or between the second and third finger. Skin that does not fold up into a pinch is considered a positive sign of lymphedema (Ratliff, 2016; Lewis & Walsh, 2014).

Diagnostic Tests: Diagnosis of lymphedema is typically made through clinical presentation and medical history, such as a cancer diagnosis. Lymphoscintigraphy is the imaging gold standard for lymphedema (Lewis & Walsh, 2014); however, it is expensive and invasive, and so only indicated if the patient is a surgical candidate. Bioimpedance spectroscopy (BIS) measures the composition of tissues (e.g., fluid such as lymph) and may be used for early diagnosis as well as determining the effectiveness of therapy (Ratliff, 2016).

Differential Diagnosis: Knowing if the patient had a prior history of comorbid conditions that can cause swelling of the extremities, such as cardiac disease, venous ulcer disease, renal disease, hepatic disease, trauma, and infection, is also important. Patients with rheumatoid arthritis, obesity, lipedema (painful fat syndrome), and venous ulcer disease are at greater risk for lymphedema because these conditions add additional stress to the impaired lymphatics. Lymph nodes are located around most joints, so patients undergoing such surgical procedures as total knee replacements may be at greater risk for developing lymphedema because lymph nodes can become injured during the surgery. Also, procedures such as vein stripping can exacerbate mild lymphedema (Lewis & Walsh, 2014).

Treatment: Complete decongestive physiotherapy (CDP) is the gold standard for lymphedema management. CDP is a specialized massage technique designed to stimulate the lymph vessels, break up subcutaneous fibrous tissue, and redirect the lymph fluid to areas where lymph flow is normal. It involves four steps: manual lymph drainage (MLD), compression bandaging, exercises, and skin care.

Conservative Treatment: The use of multilayer compression bandages and/or compression stockings is an important component for all patients with lymphedema to prevent the reaccumulation of lymph fluid in the limb.

Pharmacological Treatment: Diuretics are not beneficial with lymphedema because they draw off excess water in the interstitial spaces but not the protein. Lymphedema is a high-protein edema, and the high osmotic pressure from the increased protein in the interstitial space causes rapid reaccumulation of edema. In addition, the higher concentration of protein in the edema fluid causes increased fibrosis and induration of the skin. Diuretics are not contraindicated for the treatment of other conditions in lymphedema patients, but they should not be used as primary treatment for lymphedema.

Surgical Treatment: Surgery is reserved for individuals with a positive lymphoscintigram who do not respond to more conservative methods such as CDP. Reconstructive or restorative procedures create lymph-venous shunts or autologous vessel transplantations. Debulking procedures remove excess tissue to reduce the size and weight of the limb (Lewis & Walsh, 2014).

Follow-Up: Patients with lymphedema need regular follow-up by health-care providers to make sure that the CDP is effective and adequate and, if not, appropriate modifications can then be made to the care plan.

Sequelae: Lymphangitis and cellulitis are complications seen with lymphedema. Once patients develop these infections, they are at greater risk for developing them again.

Prevention/Prophylaxis: Lifelong CDP is essential to prevent worsening of the lymphedema and to avoid complications.

Referral: Patients with lymphedema should be referred to a lymphedema specialist.

Education: It is important to teach patients with lymphedema about the disease process, its lifelong treatment with compression bandaging, the importance of avoiding complications such as lymphangitis, and about resources, such as the National Lymphedema Network, that are available to them.

PERIPHERAL VASCULAR DISEASE

Signal Symptoms: Pain, intermittent claudication of the feet, tissue loss in affected leg/arm.

Description: Peripheral vascular disease (PVD) refers to a disease or process that alters blood flow to or from the extremities and vital organs other than the heart. These processes may involve the arterial, venous, or lymphatic systems, but most often are due to enlarging atherosclerotic plaques in the distal aorta or in major bifurcations or areas of angulation in the iliac, femoral, and popliteal arteries. Most activity limitations and limb loss are associated with peripheral arterial disease (PAD).

Etiology: Atherosclerotic plaques may be fatty streaks, fibrous plaques, or complicated calcified lesions. Fatty streaks are early lesions that occur in the intima of arteries. Fibrous plaques and areas of intimal thickening are the most frequently occurring type of lesion. Complicated (heterogeneous) plaques, as well as calcified fibrous plaques with potential for necrosis and thrombosis, are associated most often with symptoms in PAD patients (Dua & Lee, 2016).

Occurrence: PAD is referred to as an age-related disease. More diabetics than nondiabetics are diagnosed with PAD. The prevalence of PAD for the U.S. adult population is 12%, with an annual incidence of 2.4%. The prevalence of critical limb ischemia (CLI) for the same population is 1.3%, with an annual incidence of 0.4%. This percentage is expected to increase as the number of older adults in the population increases (Dua & Lee, 2016).

Age: Nearly 20% of individuals who are more than 70 years old have PAD, compared with less than 8% of individuals who are younger than 70 years old.

Gender: Symptomatic PAD is two to five times more prevalent in men than in women.

Ethnicity: There are few available data to support an ethnic predisposition for the development of PAD.

Contributing Factors: Smoking remains the most important risk factor, with up to 80% of cases of intermittent claudication associated with tobacco use. Diabetes mellitus is another important risk factor. More than 80% of diabetics surviving 20 years from the time of diagnosis have some type of arterial disease. Of patients with a gangrenous lesion of the feet requiring amputation, more than 50% are diabetic. Other associated risk factors include hypertension, high serum cholesterol, obesity, sedentary lifestyle, strong family history, vasculitis, and hypercoagulopathy (Pomposelli et al., 2015; Shirasu, Hoshima, & Nishiyama, 2016; Moain, Dabrn, & Steffen, 2016).

Signs and Symptoms: Intermittent claudication is the early symptom of PAD. It is described as a painful cramping of the muscles of the leg during walking. It goes away when the patient stops walking and resumes after the patient starts walking again. It also may be described as a sensation of tiredness or fatigue. One-third of patients with proven arterial stenosis report symptoms of claudication. Ischemic rest pain occurs constantly and is differentiated easily from claudication. It is described as a burning sensation and localizes to the metatarsal heads or to an ischemic ulcer. Pain is often worse at night when the leg is elevated. Relief may occur with dependency of the foot. Ischemic rest pain requires immediate attention. Ulceration or gangrene develops at an area of external pressure or at the site of a minor injury, with gangrene representing the end-stage of PAD. Acute arterial ischemia results from arterial thrombosis, embolism, or trauma, with symptoms that are sudden in onset. It often causes the five Ps: pain, pallor, pulselessness, paresthesia, and paralysis. Other symptoms may include decreased sensation and mottling of the extremity. Acute arterial ischemia may be due to thrombus from the left atrium, sudden onset of atrial fibrillation, dislodging of calcium plaque in the aorta, and/or intrinsic hypercoagulopathy. A thorough patient history and physical examination are essential to determine the stage and type of vascular disease (Cucato, Ritti-Dias, & Franco, 2016; Johnston, Robinson, & Tracci, 2016).

Diagnostic Tests: Ankle-brachial index and systolic arterial pressure measurement with continuous-wave Doppler are first-line tests. In cases where surgical intervention is being considered, angiography is indicated. Assessment of distal arterial system patency can be done by Doppler ultrasound, which can also detect and pinpoint occluded areas (Alahdab, Wang, & Elraihyah, 2015).

Differential Diagnosis: Buerger's disease and Raynaud's phenomenon also should be considered when diagnosing PAD. Buerger's disease may occur in individuals less than 35 years old who are smokers, and it may affect the upper as well as the lower extremities. This rare disease causes inflammation of the blood vessels, which can result in clot formation. Raynaud's phenomenon affects the fingertips, the tips of the ears and nose, and the feet. Patients with Raynaud's phenomenon have symptom exacerbation with any cold temperatures. Other diagnoses that mimic the symptoms of PAD include gout, arthritis, diabetic neuropathy, sciatica, and severe venous insufficiency (Dua & Lee, 2016).

The hallmark signs and symptoms of venous ulcers include distal lower extremity edema; warmth of the foot; large, irregular, copiously draining ulcers in the distal calf

or foot dorsum; and hyperpigmentation of the surrounding skin. Arterial insufficiency ulcers usually are found over the medial or lateral malleolus, heel, or distal aspect of the toes. Wounds associated with arterial ischemia are small and drain minimally, but are extremely painful.

Treatment: The main goal of PAD treatment is to slow the progression of the disease. Treatment may be conservative, pharmacological, operative, or endovascular. Risk factor recognition and lifestyle modification are the central tenets for managing patients with long-term PAD.

Conservative Treatment: Conservative treatment involves modification of risk factors, including smoking, blood pressure control, and diet (Hirsch et al., 2005). Smoking is the number one modifiable risk factor, and there are numerous methods available to help individuals stop smoking (Zkhari, 2013; VanDevanter, Zhou, & Katigbak, 2016; Arens, White, & Masengill, 2014). In addition, managing diabetes and hyperlipidemia are key measures. Exercise is an essential element of PAD management because collateral vessels are strengthened and stimulated to grow with the onset of an exercise program such as walking. Formalized vascular rehabilitation programs exist; however, they are not currently recognized by insurers and rarely are reimbursed. Home exercise programs should focus on exercising three or more times per week for at least 30 minutes per day. Patients should be instructed to walk until pain develops, walk a few steps more through the pain, rest until pain goes away, then resume walking. Over time, the patient should be able to walk farther with fewer rest breaks. Meticulous foot care is important to prevent complications of ulceration and gangrene. Patients should be instructed about wearing properly fitted footwear, daily inspection of feet and legs, avoidance of walking barefoot, application of lotion on feet and legs, and meticulous toenail trimming. Podiatrist consultation is recommended for patients with fungal overgrowth and/or ingrown toenails. (Han, Sadarangani, & Wyatt, 2016; Moyer-Harris, 2015).

Pharmacological Treatment: Pharmacological treatment is provided with conservative treatment. Currently, aspirin (81 to 325 mg daily) is one of the mainstays for such treatment. Aspirin has been shown to prevent progression of disease in patients with claudication. Cilostazol (Pletal) may be effective in relieving symptoms of claudication. The use of pentoxifylline (Trental) is also available for treatment of claudication; however, studies have not shown significant improvement in claudication symptoms and cilostazol has superiority (Stevens et al., 2012). Vasodilators and anticoagulants have not proved effective in relieving pain symptoms. Clopidogrel (Plavix) 75 mg daily is an antiplatelet medication that helps reduce the risk of stroke, heart attack, and other atherosclerotic problems. Clopidogrel is frequently used for at least 3 months after peripheral arterial stents have been placed. Medications for the management of hypertension, diabetes, and hyperlipidemia should be used with the goals of euglycemia, normotension, and attainment of a healthy lipid profile (Iannito, Dickman, & Lakhani, 2014; Mackey & Whitaker, 2015; Moyer-Harris, 2015). Most patients with cardiovascular disease benefit significantly from a statin, a beta blocker, an aspirin, daily multivitamins with B-complex vitamins, vitamin E, and calcium supplements (Pomposelli et al., 2015; Sherrod, Sherrod, & Cheek, 2015).

Surgical Treatment: Surgical treatment of PAD involves revascularization of the affected extremity. Surgical options should be considered when pain limits the patient's lifestyle or when ulceration or gangrene is present. Surgical options involve using vein graft or synthetic graft material. Long-term patency rate is dependent on smoking cessation, exercise, antiplatelet therapy, and vigilant diabetic control, as well as the location, length, and diameter of the graft.

Endovascular Treatment: Endovascular options for the treatment of PVD include balloon angioplasty and endoluminal stents, which are minimally invasive for the patient. Angioplasty involves inflating a balloon across a stenotic lesion. Stents are devices that are deployed in a stenotic lesion to keep the vessel open. Other interventions include thrombolysis with fibrin-degrading infusions, mechanical thrombectomy, atherectomy, and subintimal recanalization.

Follow-Up: Follow-up should include evaluating the patient's response to pharmacological intervention and the progression of risk factor modification. Pulse volume recording should be done at least once a year to assess lower extremity perfusion.

Sequelae: A history of intermittent claudication approximately doubles the risk of mortality resulting from ischemic heart disease. In patients with claudication, 25% develop worsening symptoms and 5% require an amputation over their lifetime. There appears to be differences in the presentation of PAD in men versus women, however many of the risk factors and treatments are similar for both genders (Tomczyk & Treat-Jacobson, 2009).

Prevention/Prophylaxis: Prevention of PAD focuses on slowing the progression of disease in patients with symptoms. This can be accomplished through risk factor modification, as discussed earlier. Good foot care, in conjunction with exercise, smoking cessation, glucose management if diabetic, and lipid management can prevent the development of tissue loss or gangrene.

Referral: Referral for surgical or endovascular treatment should occur when claudication becomes disabling to the patient, when ischemic rest pain or gangrene is present, or when nonhealing ulceration is present.

Education: Education needs to focus on proper follow-up and the modification of risk factors as discussed earlier; patients also need to be taught to report any new onset of symptoms, including nonhealing ulcers. There is a need for more research on the differences between the treatment for PAD in men and in women (Cucato, Ritti-Dias, & Franco, 2016; Han, Sadarangani, & Wyatt, 2016; Dua & Lee, 2016).

VENOUS DISEASE (CHRONIC VENOUS INSUFFICIENCY)

Signal Symptoms: May be asymptomatic but could also present with swelling that subsides with elevation of lower extremities, eczematous skin changes, dull ache in lower extremities, and presence of varicosities.

Description: Venous leg ulcers (also known as chronic venous insufficiency ulcers, venous stasis ulcers, lower extremity venous ulcers, or varicose ulcers) are the most common leg ulceration. They typically occur over the medial malleolus, but can also occur over the lateral malleolus. The ulcers are irregular and shallow with granulation tissue and fibrin present in the wound bed. Exudate is moderate to heavy, but if there is an arterial component, the amount of exudate may be decreased. These wounds often have a foul odor as well. The area around the wound often presents with dermatitis from the increased permeability of the venous capillaries causing protein to leak into the interstitial space, becoming irritating to the epidermis and dermis. The loss of protein and red blood cells into the subcutaneous tissue results in a brownish discoloration of the skin referred to as hemosiderin staining. Over time, the induration and hyperpigmentation is associated with fibrosis of the adipose tissue of the leg, which is called lipodermatosclerosis.

Etiology: Venous disease is caused by ambulatory venous hypertension. Normally, blood flows from the superficial veins to the deep veins. Unidirectional valves and contraction of the calf muscles assist in blood flowing from the superficial to the deep veins. If there are any abnormalities (i.e., valvular incompetence, deep vein thrombosis, and/or failure of calf muscle pump), the deep and superficial veins become distended, increasing venous pressures.

Occurrence: Venous ulcers are the most common leg ulcers, accounting for up to 90% of all leg ulcers (Ratliff, 2016). In the United States, it is estimated that approximately 2.5 million people have clinical symptoms of chronic venous insufficiency (Lal, 2015; O'Donnell et al., 2014). Venous leg ulcers are estimated to affect 4% of adults 65 years of age or older, or about 600,000 people in the United States (Lal, 2015).

Age: As one ages, the incidence of venous ulcers increases (Alavi et al., 2016).

Gender: Women are affected more than men, with multiple pregnancies or pregnancies close together increasing the risk.

Ethnicity: There are no data to support an ethnic predisposition to venous disease.

Contributing Factors: Risk factors for venous leg ulcers include advanced age, obesity, pregnancy, thrombophilia, systemic inflammation, anticardiolipin antibody, venous thromboembolism (VTE)/phlebitis, varicose veins, pulmonary embolus, sedentary lifestyle or occupation, reduced mobility, simultaneous insufficiency of two out of three venous systems, trauma/surgeries/leg fractures, impaired calf muscle pump, restricted range of motion of the ankle, family history of venous disease, injection drug user, and previous leg wound (WOCN, 2013). The presence of comorbid conditions such as heart failure, lymphedema, and orthopedic procedures can also increase the risk of venous leg ulcers (WOCN, 2013).

Signs and Symptoms: Edema that worsens with standing and decreases with leg elevation is the classic sign of venous leg ulcers. The edema may be pitting or nonpitting. Other signs include hemosiderosis (hemosiderin staining), venous dermatitis (manifested as erythematous, scaly, pruritic skin), ankle flaring (cluster of spider veins), varicose veins, scarring from previous ulcers, and lipodermatosclerosis.

Diagnostic Tests: In many cases of venous ulcers, diagnosis may be made by clinical assessment alone, which includes skin changes in the leg, extent and location of edema, capillary refill less than 2 seconds, and palpation of pedal pulses. Duplex ultrasonography imaging with or without color has become the standard diagnostic tool for assessing venous disease. Duplex imaging produces images of blood flow and its direction through vessels, pinpointing the anatomical site of reflux or obstruction; thickened, abnormal vein walls; and the presence and age of a thrombus. Duplex scanning can be used to calculate the superficial venous pressures and allows for quantification of venous reflux and valve closure times. An ankle-brachial pressure index is also recommended to rule out significant arterial disease, especially before application of compression therapy.

Differential Diagnosis: Lower leg edema, which is one of the classic signs of venous disease, is also seen with other diseases as well. Deep vein thrombosis usually presents with pain and the presence of a thrombus on ultrasound. Patients with heart failure usually present with dyspnea, orthopnea, and a decreased ejection fraction. Patients with cirrhosis will have abnormal liver functions, and those in renal failure will have proteinuria. The diagnosis of venous disease can be narrowed by categorizing the edema according to its duration (acute or chronic), distribution (unilateral or bilateral), and accompanying symptoms (e.g., pain, itching, skin changes).

Treatment: Treatment for venous ulcers includes management with compression therapy, leg elevation, medications, and surgery.

Conservative Treatment: Compression therapy is the gold standard for venous ulcers and is used by those with chronic venous disease to manage the edema and increase venous return. Compression has been shown to improve healing of venous ulcers when compared to no compression (O'Donnell et al., 2014; O'Meara et al., 2012), as well as reducing venous ulcer recurrence (Ratliff, 2016; Mauck et al., 2014; O'Donnell et al., 2014). Compression is also effective in the improvement of symptoms of chronic venous disease, reduction of edema and pain, and improvement of quality of life (Sippel, Seifert, & Hafner, 2015).

Compression can be achieved with the use of a single layer wrap (bandage) or the use of multiple layer wraps (bandages) (Mauck et al., 2014). There are also compression garments such as the reusable Velcro wraps like CircAid®, sleeves like tube-shaped Tubigrip™, compression stockings, and intermittent pneumatic compression pumps (Ratliff, 2016).

Another feature of compression products is based on the type of material in the wrap (bandage). Elastic (adapts to changes in limb size) should be prescribed for those who are sedentary, versus inelastic (will not change with limb size), which should be prescribed for those who frequently ambulate. For patients with mixed venous and arterial disease (ankle-brachial index of greater than 0.5 to 0.8), a trial of supervised modified light compression up to 30 mm Hg may help decrease edema and promote ulcer healing (Ratliff, 2016). Leg elevation above the heart for 30 minutes three or four times per day when used with compression can also help reduce edema (Robinson & Melo, 2014).

Pharmacological Treatment: Horse chestnut seed extract (HCSE) in a dose of 300 mg twice daily may reduce edema and pain in venous leg ulcers (Pittler & Ernst, 2012). People with bleeding disorders, kidney disease, or liver disease should not take HSCE. There may also be interactions with certain medications, such as lithium, diabetic drugs, and anticoagulants, that might result in low blood sugars and slower blood clotting (WebMD, 2016). Aspirin may be effective when used with compression therapy with the recommended dosage of 300 mg per day, as long as there are no contraindications to its use (Kimmel & Robin, 2013). Pentoxifylline (Trental) 400 mg three times per day has been used to treat venous ulcers. It has been shown effective when used with compression and may be beneficial as monotherapy in those patients who cannot tolerate compression (Jull et al., 2012). Topical steroids may provide short-term improvement of venous dermatitis. Oral antibiotics are only warranted in cases of suspected cellulitis (Carmel & Bryant, 2016).

Surgical Treatment: Surgery should be considered when topical therapy and compression are not able to heal the venous ulcer. Surgical options to treat venous disease include interruption of the perforating veins with subfascial endoscopic perforator surgery (SEPS), laser ablation, radiofrequency ablation (RFA), and sclerotherapy (Carmel & Bryant, 2016; Robinson & Melo, 2014). Surgical procedures available for valvular incompetence include artificial valve insertion, direct valvuloplasty, and autogenous vein valve transplantation (Carmel & Bryant, 2016). Skin grafts and bioengineered human skin equivalents may be used for those with large or refractory venous ulcers, but they must be used in conjunction with compression therapy to improve healing (Carmel & Bryant, 2016).

Follow-Up: The goals of care are to reduce edema, promote ulcer healing, and prevent reoccurrence of the ulcer (Robinson & Melo, 2014).

Sequelae: Compliance with compression, ulcer size, and duration of the ulcer affect ulcer healing. Ulcers larger than 5 cm^2 and present for more than 6 months are predictive for nonhealing, so early assessment and treatment with compression therapy is important to reduce complications such as infection from nonhealing ulcers (Carmel & Bryant, 2016).

Prevention/Prophylaxis: The daily use of compression therapy for the rest of one's life is recommended to promote healing and reduce the chance of reoccurrence.

Referral: Patients with venous leg ulcer disease with impaired calf muscle pump function may benefit from physical therapy that includes flexion and extension exercises, as well as walking programs to improve calf muscle pump function (Robinson & Melo, 2014).

Education: Patients with venous ulcer disease should be instructed to use compression stockings every day for the rest of their lives and replace these compression stockings at least every 6 months. They should elevate the affected leg or legs above the heart several times a day. Physical activity is recommended to increase venous return and should be performed based on the patient's medical condition, but may include ankle flexion exercises, brisk walking, and sitting in a rocking chair and using the feet to push down to plantar flex the ankles.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Smoking cessation, dietary management, exercise, and diabetic management are key treatments to slow the progression of peripheral arterial disease.	A	Zakhari, 2013 Arens, White, & Masengill, 2014 Cucato, Ritti-Dias, & Franco, 2016 Iannito, Dickman, & Lakhani, 2014 Mackey & Whitaker, 2015 Conte et al., 2015 Dua & Lee (2016)
Hypertension treatment is an essential treatment for patients with known abdominal aortic aneurysms.	A	Scher, Drew, & Cottrell, 2015 Gordon & Toursarkissian, 2014
Patients with known abdominal aortic aneurysms should receive periodic ultrasound, magnetic resonance imaging, or CT-guided images at least once a year.	A	Sodem, Zetterwal, & Ultee, 2016 Strider, Keeling, & Tullmann, 2013 Cooper, 2014

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Endovascular grafts for complex aortic aneurysms permit a lower perioperative risk profile than does the open surgical procedure for such patients. After the procedure, nursing proficiency with aortic endograft is key to the patient's safe postoperative progression.	A	Buck, Ultee, & Zetterwall, 2016 Sodem, Zetterwall, & Ultee, 2016 Cooper, 2014 Schermerhorn, Buck, & O'Malley, 2015
Compression improves healing of venous leg ulcers compared to no compression.	A	O'Meara et al., 2012 Mauck et al., 2014
Compression stockings reduce the chance of venous ulcer recurrence when compared with no compression.	A	Mauck et al., 2014
Manual lymph drainage may offer an additional benefit to compression bandaging for swelling reduction in breast cancer-related lymphedema.	A	Ezzo et al., 2015
Clinicians should prescribe one or more of the following for patients diagnosed with peripheral arterial disease: aspirin, beta blocker, statin agent.	B	Iannito, Dickman, & Lakhani, 2014 Mackey & Whitaker, 2015 Conte et al., 2015 Sherrod, Sherrod, & Cheek, 2015 Dua & Lee, 2016 Lewis, Dindyal, & Raynor, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

M. J. is a 76-year-old woman who lives on the side of a very steep mountain. The home health nurse has visited her once a week for the last year. She has running water, electricity, and a coal stove with back-up oil heat for very cold winter nights. She uses the telephone for communication. She has diabetes mellitus, hypertension, hypothyroidism, and is in atrial fibrillation. She has never been in the hospital before.

Her current medications include metformin (250 mg twice a day), losartan (50 mg/day), levothyroxine sodium (Synthroid) (50 mcg/day), digoxin (0.125 mg/day), furosemide (Lasix) (10 mg/day), aspirin (81 mg/day), simvastatin (20 mg/day), and warfarin (Coumadin) (4 mg/day, with 6 mg on Sundays). Allergies are to penicillin (hives) and to metoprolol (hypotension and dizziness). M. J. stopped smoking 5 years ago, but until then she smoked one-half pack a day.

Last laboratory test results (1 week ago) were: hemoglobin A1C (Hgb A1C) 8.3, international normalized ratio (INR) 1.7, sodium 129, potassium 5.8, chloride 102, CO_2 20, blood urea nitrogen (BUN) 45, creatinine 1.5, glucose 289, white blood cell (WBC) count 6.8, Hgb 10.1, hematocrit 30.2, platelets 215,000.

The home health nurse calls you from the patient's house, where she notes that the patient had a 5-minute spell of visual loss in her right eye. It has not recurred. During this period, M. J. noted that her left hand and left leg felt "funny," and her left foot and leg were so weak that she could not walk.

M. J. has a heart murmur, described by the home health nurse as loudest over the right sternal border and more prominent in the systolic phase. Furthermore, the home health nurse tells you that she hears bruits on both sides of the neck, with the right being louder than the left. The blood pressure in the right arm is 135/72 mm Hg and 110/66 mm Hg in the left arm. Heart rate is irregular at 75 to 90 beats per minute. The left hand and fingers have always been cooler than the right. Lungs are remarkable for wheezes. The patient has no fever. Oxygen saturations at room temperature are 90% to 92%, which has been her baseline.

Continued

CASE STUDY—cont'd

1. What concerns do you have regarding M. J. at this point?
2. Do you need to see M. J. fairly soon, or can she come to see you at her regularly scheduled appointment in 3 weeks?
3. If you were to see M. J. in the next 24 hours, what additional tests would you perform?
4. Based on the laboratory results given, what changes would you make in her medication profile?
5. Which arm will you use for blood pressure recordings in the future?
6. What systolic blood pressure range would you order for M. J., as a general target?
7. If M. J. has a repeat episode of right eye blindness, lasting 30 minutes this time, what would you do?

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CHAPTER 10

Abdominal Disorders

Laurie Kennedy-Malone

ASSESSMENT

Accurate history taking of abdominal complaints is essential for completing an assessment of the older adult. The physical examination is often unremarkable, and laboratory findings may not provide diagnostic information because the presentation of illness in an older adult is usually subdued.

Gathering the Abdominal History

Generally, the chief complaints of abdominal conditions can be categorized into symptom cluster areas for further exploration: abdominal pain/discomfort, gastroesophageal reflux disease (GERD)/regurgitation symptoms, nausea/vomiting, diarrhea/incontinence, and difficult defecation and constipation (Crowell et al., 2015; Koloski et al., 2017). Explore with the patient any episodes of anorexia, dyspepsia, dysphagia, heartburn, nausea, regurgitation, vomiting, painful or difficult defecation, diarrhea, tenesmus, or constipation. Determine the sequence of events that triggered each symptom. Inquire about precipitating factors such as a meal, position of the body, use of caffeine, alcohol use, or smoking. Were any other symptoms present that can be clustered to form a differential diagnosis? It is important to discern from patients what therapeutic measures they have initiated to alleviate the symptoms (Saccomano & Ferrara, 2013). Determine if the treatment is a previously prescribed regimen, an over-the-counter (OTC) medication, an herbal product, or a home remedy. Ask the patient when the last time a fecal occult blood test was performed and if he or she has had a recent colonoscopy or sigmoidoscopy examination.

Abdominal Symptoms

Although abdominal pain is a common acute problem with which patients present to their health-care providers, older adults may not experience the typical symptoms of abdominal disorders that younger patients report. If patients present with abdominal pain, it is important to discern the characteristic of the pain. How quickly did the pain begin? Was the pain abrupt? Is the pain progressive or intermittent? Has the patient ever experienced this type of pain before and, if so, what was the diagnosis and treatment? Has the patient noted any improvements with self-treatment, positioning, vomiting, or defecation? What is the referral pattern? Is the pain worse at night or during the day? Is it relieved or aggravated by food? If the patient reports triggers to abdominal pain by food, ask more specific questions pertaining to food to discern if the patient is having a specific food intolerance(s) or possible food poisoning. Does the patient have any associated symptoms of regurgitation, reflux, cough, nausea, vomiting, constipation, or diarrhea? You can ask about the severity of pain by asking the patient to rate the pain on a scale from 1–10, however, realizing that in older adults pain may be blunted despite the underlying pathology (Saccomano & Ferrara, 2013). Gather a history of previous abdominal surgeries. Inquire about alcohol use or drug abuse. Inquire about any history of jaundice, heart disease, or peripheral vascular disease. Review all current medications (Spirit, 2010).

Inspection

The physical examination of the abdomen begins with inspection, checking for overall symmetry while observing the patient lying down. The practitioner can consider the abdomen to be divided into four quadrants, visualizing an imaginary vertical line from the sternum to the pubis passing though the umbilicus and a horizontal line drawn through the umbilicus. In addition to being familiar with the underlying organ systems and referred pain in the four quadrants, the nurse practitioner should be familiar with other anatomical landmarks, such as epigastric, periumbilical, suprapubic, and McBurney's point, which is one-third of the distance from the right anterior superior iliac spine to the umbilicus (Rabinowitz & Roberts, 2015).

Carefully examine for scars, lesions, dilated veins, or other marks. If surgical scars are detected, review with patient surgical history if not recorded. Determine if the abdomen is concave or protuberant. Is an umbilical, abdominal wall, incisional, or inguinal hernia present? If a hernia is suspected, have the patient raise the head and note if there is presence of a bulge. (O'Laughlen, 2009). Is a lift noticeable? Is there abdominal distention? If so, is the distention local or generalized? Causes of generalized distention include abdominal fat, peritoneal fluid, or gaseous distention, whereas local distention often is indicative of obstruction, or organ or structural enlargement. Examine the umbilicus. Signs of ascites include

a flat or everted umbilicus (Williams, 2008). Check for any bulging in the flanks as a sign of ascites.

Examine the skin of the abdomen for color, noting if there is any jaundice found in liver disease or biliary tract obstruction, bruising, or telangiectasias (O'Laughlen, 2009). If a bluish discoloration is detected around the umbilicus this is known as Cullens' sign, which is often found in patients with bleeding in the peritoneum. If the bluish discoloration is on the flanks, this is known as Grey Turner's sign and is often indicative of retroperitoneal bleeding, as in pancreatitis (Rabinowitz & Roberts, 2015). Jaundice in the area of the umbilicus is known as Ransohoff's sign and is a result of a ruptured common bile duct (Williams, 2008). Note if there are any pulsatile movements across the abdomen. If present, this sign may be indicative of a vascular aneurysm or a wide arterial pulse pressure (Williams, 2008). Visible intestinal peristalsis may be indicative of bowel obstruction (Rabinowitz & Roberts, 2015).

Auscultation

Auscultations follow inspection of the abdomen. Using the diaphragm, place the stethoscope lightly against the skin. Listen for bowel sounds, vascular bruits, and rubs. Bowel sounds are produced by peristalsis. A murmur heard in the abdomen may be an aortic aneurysm. A constant systolic-diastolic bruit may occur when the patient has an arteriovenous fistula in the renal vessels. Other sites to auscultate for bruits during abdominal examination include the iliac arteries and the femoral arteries.

When auscultating bowel sounds, take the time to listen for presence of bowel sounds. Generally, people who are hungry have active bowel sounds. Bowel sounds described as high pitched, rumbling, or tinkling are known as borborygmi. A rushing sound is associated with bowel obstruction. If bowel sounds are absent, listen for 5 minutes. Suspect peritonitis, mesenteric thrombosis, or advanced intestinal obstructions when bowel sounds are absent (O'Laughlen, 2009). Listen for a loud succussion splash over the abdomen of a patient in whom you suspect a gastric obstruction or dilations (Williams, 2008). Nurse practitioners should not, however, rely on auscultation alone for diagnostic purposes, as studies have refuted the usefulness of auscultation when differentiating patients who have normal versus pathological bowel sounds (Felder, Margel, Murrell, & Fleshner, 2014).

Percussion

Percussion of the abdomen is performed to determine the density of tissue of the abdominal organs by the sounds emitted when tapped (LeBlond, Brown, Suneja, & Szot, 2015). It is normal to hear tympanic sounds over the stomach, whereas dullness is expected over a distended bladder, the liver, and the spleen (O'Laughlen, 2009). Dullness noted over other areas of the abdomen is a deviation from the norm. Definitive percussion is used to ascertain the size and shape of the liver and spleen. Because the liver decreases in size starting at about age 50 years, the normal range of the liver size in an older person is 6 to 12 cm (Williams, 2008).

Palpation

Perform light palpation to discern abdominal masses; enlarged organs such as liver, spleen, and kidneys; and areas of tenderness. If a mass is detected, the nurse practitioner needs to determine if the mass is intra-abdominal or within the abdominal wall. Using the palm and extended fingers of the right hand, press about 21 cm deep. If a patient has complained of abdominal pain, always palpate in a quadrant away from the identified location. (Rabinowitz & Roberts, 2015).

Once you discover the tender area, maintain pressure over the area to determine the consistency of the pain. If the pain diminishes despite the applied pressure to the area, inflammation is unlikely. If you suspect intra-abdominal tenderness, proceed to deep palpation. Check for referred rebound tenderness by applying pressure with the tips of the fingers to a site distant from the areas of questionable tenderness, and then quickly remove them from the abdomen (Saccomano & Ferrara, 2013). Assessment of the patient's facial expression is warranted, especially in older adults with difficulty with communication (O'Laughlen, 2009). If tenderness is elicited remote from the areas palpated after release of pressure, consider peritoneal irritation (LeBlond et al., 2015). Specific maneuvers to detect tenderness can lead the examiner to making a differential diagnosis; for instance, tenderness medial to McBurney's point (Rabinowitz & Roberts, 2015) suggesting a Meckel diverticulum should be distinguished from typical appendicitis tenderness. A mass detected in the lower abdomen on palpation warrants further rectal and/or pelvic examination.

Palpation of the kidneys should include checking for costovertebral angle tenderness, which is often indicative of renal disease, but also may be musculoskeletal in origin (O'Laughlen, 2009). While the bladder is not normally palpable, it may be detected on palpation if bladder distention is suspected. Rectal examination includes determining presence or absence of stool, checking for presence of hemorrhoids, and testing sphincter tone and guaiac. Ask the patient to strain gently as if having a bowel movement to relax the anal sphincter.

ACUTE KIDNEY INJURY

Signal Symptoms: Fatigue, altered mental status, anorexia, shortness of breath.

Description: Acute kidney injury (AKI), previously known as acute renal failure (ARF), is defined as the abrupt loss of kidney function over a period of hours to days (Palevsky, 2012). This can be either a minimal loss without any residual effects, with the patient's renal function returning to previous baseline before the injury, or can result in chronic kidney disease (CKD). In addition, any episode of AKI will increase the risk for CKD later in life. AKI has now been classified as one of a number of acute kidney diseases and disorders (AKDs), and can occur with or without other acute/chronic kidney diseases (KDIGO, 2012).

Etiology: Renal function with urine formation is based on four steps that occur in specific areas of the kidney. First, blood from the renal arteries is delivered to the glomeruli and filtered, resulting in an ultrafiltrate. The ultrafiltrate, free of protein and blood elements, then flows into the renal tubules, which reabsorb and secrete solute and/or water from the ultrafiltrate. The final tubular fluid, urine, leaves the kidney, draining through the renal pelvis and ureter and into the bladder, leaving the body through the urethra. Any disruption in the process of urine formation can result in the deterioration of renal function and AKI.

AKI is defined as an increase in serum creatinine by 50% within 7 days or an increase in serum creatinine by 0.3 mg/dl in 2 days or urine volume of less than 0.5 ml/kg/hr for 6 hours (KDIGO, 2012). Serum creatinine is a by-product of muscle metabolism that is excreted unchanged by the kidneys. Assessment of renal function is made by estimation of the glomerular filtration rate (eGFR) (KDIGO, 2012). Serum creatinine alone should not be used to determine renal function (Levey, 2015). eGFR is the most widely accepted and most useful overall index of kidney function (KDIGO, 2012). Changes in serum creatinine and urine output are indicators of a change in eGFR. Normal eGFR varies according to age, sex, and body size. In young adults, it is 120 to 130 mL/min and declines with age starting after 40 years. AKI can occur before a change in the serum creatinine or urine output is noted. eGFR is best calculated using the 2009 Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) (KDIGO, 2012).

The formula is as follows:

$$\text{eGFR} = 141 \times \min(S_{Cr}/\kappa, 1)^{\alpha} \times \max(S_{Cr}/\kappa, 1)^{-1.209} \times 0.993^{\text{Age}} \times 1.018\ [\text{if female}] \times 1.159\ [\text{if African American}]$$

eGFR is less accurate in patients who have AKI due to the rapid changes in kidney function. Patients who have extremes of muscle mass, such as the frail older adult, the critically ill, cancer patients, body builders, or those with unusual diets, will also have inaccurate eGFR. Creatinine clearance, where urine is collected over 24 hours and the amount of creatinine is measured, is a more accurate indicator of the eGFR in these patients.

The causes of AKI can be grouped into three categories:

- Prerenal, caused by reduced renal perfusion
- Intrinsic, in which there is damage to the renal parenchyma
- Postrenal, where there is obstruction to the flow of urine

Furthermore, AKI can be characterized as:

- Oliguric, which is less than 400 mL urine output in 24 hours
- Nonoliguric, which is greater than 400 mL urine output in 24 hours
- Anuric, which is less than 100 mL urine output in 24 hours (Safirstein, 2005)

Anuria is uncommon and may be caused by complete obstruction or a major vascular event such as aortic dissection or ruptured aortic aneurysm in AKI. Prerenal, intrinsic, and postrenal all present clinically with either oliguria or nonoliguria. A nonoliguric state is associated with much better renal prognosis and recovery of renal function.

Occurrence: Although risk for AKI increases with age, it can occur at any age or ethnicity.

Age: Although AKI can occur at any age, its incidence is highly associated with older adults, and risk increases across the age span, most notably in those age 85 years and older (Silver et al., 2015; USRDS, 2015).

Gender: Across age groups, a higher percentage of men are affected by AKI than women (USRDS, 2015).

Ethnicity: The rates of diagnosing AKI across all ethnic groups has doubled since 2013 (USRDS, 2015).

Contributing Factors: A prior history of CKD and/or diabetes mellitus increases the risk for an AKI event (USRDS, 2015). AKI can also be caused by volume depletion, certain medications, cardiac issues, major surgery, trauma, sepsis, poisonous plants, and animals. After sustaining a severe burn, it is very common to develop AKI (Wu et al., 2016).

Use of radioopague dye can induce AKI, which is also known as contrast-inducted nephropathy (Zuo et al., 2016). Factors that predispose the patient to vascular disease and a detailed history of hypertension, diabetes mellitus, smoking, hyperlipidemia, claudication, stroke, myocardial infarction, and atrial fibrillation should be noted. Patients with atrial fibrillation may develop acute renal infarction as the precursor to AKI (Yousuf et al., 2016).

In prerenal AKI, any condition in which there is volume depletion can lead to renal hypoperfusion, including vomiting, diarrhea, hemorrhage (either surgical or gastrointestinal [GI]), excessive diuresis, and burns. Volume depletion and decreased cardiac output are the main causes of prerenal AKI. Sepsis, hypoxemia, anaphylactic shock, pancreatic disease, and liver disease may also cause AKI. In addition, any condition that affects the renal vascular system, such as renal artery stenosis, renal vein thrombosis, hepatorenal syndrome, NSAIDs, and angiotensin-converting enzyme (ACE) inhibitors/angiotensin blockers, also can induce AKI.

Conditions that cause intrinsic AKI are focused on the area of the kidney that is damaged. These include the glomeruli, tubules, and interstitium. The most common cause of damage to the structures of the kidney is prolonged hypoperfusion and nephrotoxins such as radiopaque contrast agents or certain medications. Vascular damage to the kidney from ischemia or vasculitis leads to glomerular AKI. Systemic lupus erythematosus, antineutrophil cytoplasmic antibody (ANCA)-associated granulomatous polyangiitis, Goodpasture's syndrome, cryoglobulinemia, postinfectious glomerulonephritis, polyarterisnodosa, hemolytic uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP) hepatitis B virus, and HIV are all conditions that can lead to the development of glomerular AKI. Tubules of the kidney may be damaged by ischemia, cellular edema, or interstitial edema, or become obstructed by cellular debris. AKI in this area of the kidney is called acute tubular necrosis (ATN). Any severe and prolonged prerenal cause of AKI will cause ATN. In addition, cyclosporine, tacrolimus, amphotericin B, aminoglycosides, ethylene glycol, radiopaque contrast agents, high-dose vitamin C, and Fleet's Phosphosoda (a commonly used bowel prep) can lead to ATN. Rhabdomyolysis causes tubular damage by accumulation of a greater quantity of myoglobulin than can be filtered by the kidney. Interstitial

inflammation or nephritis is usually caused by an immunological or allergic reaction and can be caused by certain medications.

Postrenal causes of AKI include any condition in which there is an impediment of the excretion of urine anywhere from the collecting tubules to the urethra. In the tubules, crystal or protein particles can precipitate, causing obstruction. This is associated with tumor lysis, myeloma, and calcium oxalate from ethylene glycol (antifreeze) ingestion and rhabdomyolysis. Ureteral obstruction can be caused by calculi, clots, fungus balls, tumors, or trauma. Bladder obstruction, either mechanical or chemical, will also cause postrenal AKI. Prostatic disease, either hypertrophy or cancer, is the most common type of postrenal AKI.

Signs and Symptoms: Predominant symptoms are usually those of the underlying illness or injury, which then leads to AKI. The patient may have symptoms directly resulting from alterations in kidney function, such as decreased to no urine output, flank pain, edema, or hypertension. The patient may have noticed a change in his or her urine consistency or color, such as foamy urine, which indicates proteinuria, or dark brown/red urine that could be indicative of red blood cells (RBCs). Patients may have nausea, vomiting, and diarrhea, leading to volume depletion. Specific questions regarding use of medications that can cause renal injury, including NSAIDs and antihypertensive medications such as ACE inhibitors (ACEIs) and angiotensin receptor blockers (ARBs), as well as recent antibiotic use, need to be asked of the patient. Factors that predispose the patient to vascular disease and a detailed history of hypertension, diabetes mellitus, smoking, hyperlipidemia, claudication, stroke, myocardial infarction, and atrial fibrillation should be noted. In addition, ask patients whether they have experienced any fatigue, arthritis, weight loss, cough, hemoptysis, arthralgias, or sinus symptoms, which may indicate a systemic illness, such as vasculitis or systemic lupus erythematosus. Use of illicit drugs, such as cocaine, ecstasy, and heroin, can also cause AKI. In addition, ask about any past history of drug use, tattoos, and piercings, which increase the risk for hepatitis B, hepatitis C, and HIV and can cause glomerular disease.

Use of radiopaque dye can also induce AKI, which is known as contrast-inducted nephropathy (Zuo et al., 2016). Past history of cancer, specifically prostate, cervical, or bladder cancer, may be indicative of an obstruction. Any family history of kidney disease increases the risk of a patient developing kidney disease in his or her lifetime as well. Patients with signs and symptoms of renal failure such as weakness, easy fatigability, anorexia, altered mental status, weight loss, dysgeusia, nausea, cold intolerance, neuropathy, tremor, asterixis, seizures, and uremic fetor, where the patient has an ammonia-like or urine-like odor to the breath, may be suffering from end-stage renal disease (ESRD) and need hospital admittance for further evaluation.

Physical examination includes assessment of weight, orthostatic blood pressures, mucous membranes, and skin turgor and neck veins for the presence of volume depletion or overload. A funduscopic examination positive for atrioventricular nicking, exudates, or arterial narrowing is indicative of hypertension. Cardiac examination may reveal an S_3, indicative of volume overload or congestive heart failure, and the presence of a pericardial rub may indicate pericarditis, a late finding in ESRD. Lung assessment may reveal tachypnea and rales. Skin assessment should include inspection for any rash, skin lesions, livedo reticularis, or purpura that may indicate vasculitis or autoimmune disease. Periorbital, lower extremity or pretibial, and presacral edema can indicate advancing renal disease from any cause. Abdominal assessment should include auscultation for renal or abdominal bruits, as well as evaluation for abdominal masses, costovertebral angle tenderness, and bladder distention. Assess for asterixis, a tremor of the wrist when it is extended, often seen in patients with ESRD.

Diagnostic Tests: Urinalysis is a noninvasive tool that can provide information regarding AKI. Urine is analyzed for sediment, specific gravity, urine sodium, urine protein, and urine creatinine levels. The appearance of certain urinary casts are cylindrical particles associated with certain diseases, assisting in determination of the cause of the AKI. Specific gravity reflects the ability of the kidney to concentrate urine and can reflect a patient's volume status. Patients who have impaired urine concentrating ability, such as those with ATN, will have low specific gravity. Urine sodium can assist in differentiating between ATN and volume depletion but can be falsely elevated in patients on diuretics. Further laboratory tests may show hyperkalemia, hypocalcemia, hyponatremia, and acidosis. A complete blood count (CBC) should also be drawn to evaluate for leukocytosis and anemia.

Radiographic studies are performed to assist in the diagnosis and cause of AKI. Renal ultrasound is most common and can be used to assess kidney size, obstruction, and the presence of masses. In AKI, kidneys will be of normal size, as compared to CKD, where they are small. If masses are noted on the kidneys with ultrasound, an abdominal computed tomography (CT) scan will help to identify the lesion. Renal biopsy is only performed when laboratory data have been unrevealing and no reversible causes could be identified as to the cause of AKI. It is performed percutaneously, and the most common complication is bleeding. Assessment of the patient's activated partial thromboplastin time (aPTT), prothrombin time (PT)/international normalized ratio (INR), and platelet count must be performed before the kidney biopsy. Renal biopsy is avoided in patients who have a "solitary" kidney, either from congenital absence or acquired absence, such as nephrectomy for renal cell carcinoma.

Differential Diagnosis:

- Hypovolemia
- Low cardiac output
- Sepsis
- Glomerulonephritis
- Ischemic nephritis
- Hyperkalemia
- Urethral obstruction
- CKD (Rahman, Shad & Smith, 2012).

Treatment: Treatment is indicative of the cause of AKI. In volume depletion, fluid resuscitation with isotonic saline and/or packed RBCs to reestablish circulating volume, as well as use of vasopressors to maintain blood pressure, may be needed. Withdrawal of the offending medication and avoidance of other nephrotoxic agents are of the utmost importance. Dose adjustment of renally metabolized medications is needed as well. Antihypertensive medications should be

judiciously titrated to avoid any further renal hypoperfusion. In obstructive AKI, treatment of the obstruction often results in renal recovery. Usually, rapid correction of prerenal and postrenal causes leads to full recovery of kidney function. Avoidance of exogenous sources of potassium, such as nutritional supplementation and IV fluids, should be discontinued. Treatment of hyperkalemia includes administration of IV calcium gluconate, insulin, beta-2 agonists (albuterol), dextrose, and occasionally kayexalate. Hyperphosphatemia may also occur in AKI, and calcium acetate or calcium carbonate can be used to reduce absorption of phosphorus from the GI tract. Platelets are often dysfunctional in AKI and may require use of DDAVP to activate platelets. Due to lower levels of circulating erythropoietin and decreased bone marrow responsiveness, anemia is often found in AKI, requiring transfusion of RBCs to increase oxygen-carrying capacity to the kidney.

In the event that there is no recovery in kidney function after correction of the cause of the AKI, hemodialysis may need to be initiated. This is especially important in the face of continued acid-base disturbances, electrolyte abnormalities, volume overload, and uremia. Studies have found that early initiation of renal replacement therapy for patients with severe AKI reduced overall mortality during the first 90 days after diagnosis (Zarbock et al., 2016). The risk of developing ESRD is higher in patients who already have CKD and sustain dialysis-requiring AKI.

Follow-Up: Patients who are unable to fully recover to their previous level of kidney function will need to be closely monitored and followed by a nephrologist. For Medicare patients age 66 years and older with an AKI hospitalization in 2011, the cumulative probability of a recurrent AKI hospitalization within 2 years was 48% (USRDS, 2015). Unfortunately, only 20% of patients had a nephrology visit within 1 year of a hospital admit for AKI. Among patients without pre-existing CKD or diabetes, less than 5% had nephrology follow-up within 1 year (USRDS, 2015). Follow up with a nephrologist 3 to 4 weeks after discharge to evaluate serum creatinine is important. Patients who have a new diagnosis of CKD stages 1 through 5 will require follow-up with a nephrologist for the remainder of their lives. With patients with concomitant conditions of hypertension, diabetes, and strict disorders it is highly advised that current practice guidelines are followed for these patients (Silver et al., 2015).

Sequelae: CKD is a direct result of AKI if there is no return to previous level of function. Patients with CKD are at an increased risk for stroke and peripheral vascular disease. In addition, they are at an increased risk for cardiovascular death, left ventricular hypertrophy, coronary artery disease, and congestive heart failure. Management of CKD includes management of anemia of chronic disease, secondary hyperparathyroidism, cholesterol, and hypertension.

Prevention/Prophylaxis: Prevention of AKI begins first with identifying those at risk for CKD. Close monitoring of GFR and urine for albuminuria, especially in older adults, is paramount. When there is a decline in GFR, prompt referral to a nephrologist is of the utmost importance. Avoidance of nephrotoxic medications, such as NSAIDs, as well as polypharmacy, will decrease risk of AKI. Recognition of medications that need adjustment for levels of kidney dysfunction is also very important in the prevention of episodes of AKI.

Referral: All patients who have AKI need to be hospitalized. Evaluation and management by a nephrologist is warranted. After resolution of the AKI and discharge from the hospital, follow-up with a nephrologist is needed in 2 to 4 weeks to evaluate serum creatinine and eGFR until recovery or plateauing occurs.

Education: Patients should be educated on signs of congestive heart failure as well as avoidance of certain OTC medications such as NSAIDs, cimetidine, and phosphate enemas. Patients should be instructed to avoid diets high in salt and to strive for less than 2 grams of sodium per day (Silver et al., 2015). In addition, tight glycemic control, with hemoglobin A1c (HbA1c) of less than 7%, as well as optimal blood pressure control of less than 130/80 mm Hg, is paramount. Smoking cessation and maintenance of healthy body weight are also important.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
GFR is the best indicator of kidney function.	C	Palevsky, 2012
Assessment of renal function is made by EGFR.	C	KDIGO, 2012
A prior history of CKD and/or diabetes mellitus increases the risk for an AKI event.	C	USRDS, 2015
Serum creatinine alone should not be used to determine renal function.	C	Levey, 2015
Serum creatinine alone should not be used to monitor kidney function. eGFR should also be used.	C	

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BLADDER CANCER

Signal Symptoms: Painless hematuria (most common presenting symptom), urinary frequency or urgency, urinary tract infection, flank pain, mild suprapubic pain; often asymptomatic.

Description: The majority of bladder cancers are transitional urothelial cell cancer; 3% are squamous cell cancer; and the rest are adenocarcinoma or small cell tumors (National Comprehensive Cancer Network [NCCN], 2016). In bladder cancer, the posterior and lateral walls of the bladder are involved more frequently than the superior wall. Bladder cancer can be categorized as *superficial*, *invasive*, and *metastatic*. Superficial, or early, bladder cancer occurs when the lesion is located on the surface of the mucosa or when the tumor penetrates the mucosa and submucosa only. Invasive bladder cancer develops when lesions pervade the bladder muscularis or the perivesical fat. Metastatic bladder cancer is characterized by lymph node, visceral, or bone tissue involvement.

Etiology: Bladder cancer has been linked to smoking, chemical exposure, and exposure to certain medications such as cyclophosphamide. There is a relationship between HPV infection and urothelial cancer. Squamous cell bladder cancer has been linked to chronic infection with *Schistosoma haematobium* (Hussein et al., 2016).

Occurrence: In the United States, each year more than 75,000 new cases of bladder cancer are diagnosed, and more than 16,000 deaths can be attributed to bladder cancer. Bladder cancer is the sixth most common type of cancer (National Cancer Institute, 2016).

Age: Median age of diagnosis is 69 years in men and 71 years in women. Bladder cancer is rarely diagnosed in patients under the age of 40 years. The age of onset is younger in current smokers than in never-smokers (American Cancer Society [ACS], 2016).

Gender: Bladder cancer is three times more common in men than in women (ACS, 2016).

Ethnicity: In the United States, Caucasian males have the highest risk, with roughly twice the incidence seen in African American and Hispanic men (ACS, 2016).

Contributing Factors: Smokers are three times more likely to develop bladder cancer as compared with non-smokers. Women exposed to second-hand smoke are at increased risk for bladder cancer. Some industrial chemicals have been linked with bladder cancer, including benzidine and beta-naphthylamine. Additionally, painters, machinists, printers, hairdressers, and truck drivers are at increased risk due to chemical exposure. Pelvic irradiation, certain drugs (e.g., cyclophosphamide), and abnormal tryptophan metabolism contribute to the development of bladder cancer. Excessive coffee consumption, use of some artificial sweeteners (e.g., saccharin sodium and cyclamate sodium), and overuse of phenacetin-containing analgesics have been suggested risk factors. *S. haematobium* infection is prevalent in Africa and the Middle East and has been associated with the development of bladder cancer, particularly in patients who smoke (Antoni et al., 2016). Exfoliation of cancer cells by cystoscopy, brushing, or transurethral biopsy or resection may spread bladder cancer cells to other sites within the bladder or may cause irritation from instrumentation. There is an association with developing bladder cancer and a long history of indwelling catheters and urinary calculi.

Signs and Symptoms: Gross, painless hematuria, pyuria, burning, and urinary frequency are common in the presentation of bladder cancer. The presence of unexplained hematuria in a patient over the age of 40 years denotes urothelial cancer until proved otherwise. Symptoms of advanced cancer may include pelvic or flank pain and lower extremity edema, resulting from lymphatic or venous blockage. Patients also may complain of abdominal pain, anorexia, and bone pain.

The physical examination may be normal in most patients; however, the clinician should palpate the pelvic area to identify a solid pelvic mass, palpate and percuss for evidence of any kidney enlargement, and perform a prostate examination on men and a pelvic examination on women. Additionally, the examination should be directed toward searching for possible sites of metastasis in the lungs, liver, bone, and lymph nodes.

Diagnostic Tests: Cystoscopy is the gold standard for the initial diagnosis and staging of bladder cancer (NCCN, 2016). Cystoscopy can miss tumors during the initial transurethral resection of bladder tumors (TURBT); therefore, a repeat cystoscopy is performed. The procedure begins with a bimanual examination under anesthesia (EUA) to determine whether or not a palpable mass is present and, if present, whether or not it is mobile. An EUA during cystoscopy is effective at identifying locally advanced disease, which may present as gross extravesical extension, invasion of adjacent organs, or pelvic sidewall involvement. If a mass is felt, the bimanual examination is repeated after the resection to see if it is still present. Fluorescence cystoscopy uses an intravesical photoactive protoporphyrin, which accumulates in neoplastic rather than normal tissue. Studies have shown that this test can detect more tumors than regular cystoscopy, but the cost is higher and there is a slightly higher false-positive rate (Kotodziej, Krajewski, Matuszewski, & Tupikowski, 2015). Urine-based tumor markers that look at tumor-related proteins, DNA, or RNA have been developed and are used to supplement cystoscopy but are not sufficiently sensitive to replace cystoscopy.

Imaging studies may be used to define the location and the extent of the tumor, as well as to detect sites of multifocal disease. CT scan is replacing the IV pyelogram (IVP) as the procedure of choice, but IVP remains an appropriate alternative where CT scan is not readily available. CT scans should include both the abdomen and pelvis; scans need to be done with and without contrast, and they should include delayed images to identify defects in the collecting system. Although CT scan provides better visualization of tumors than ultrasound, it may miss tumors less than 1 cm in size, particularly those in the bladder trigone or dome, and it cannot differentiate depth of bladder wall invasion (i.e., mucosal versus lamina propria or muscularis propria). Magnetic resonance imaging (MRI) is as reliable as CT scan for staging of invasive or locally advanced disease.

Urine dipstick, cytology, and screening for tumor-specific molecular markers in the urine have been used for screening but are not recommended for screening asymptomatic patients due to low specificity, low sensitivity, and cost, respectively (Cho & Dana, 2010).

Laboratory studies should include an alkaline phosphatase, CBC, and chemistry profile. If the alkaline phosphatase is elevated, a bone scan should be considered (NCCN, 2016).

Differential Diagnosis:

- Neurogenic bladder
- Nephrolithiasis
- Urinary tract infection
- Benign prostatic hypertrophy
- Other genitourinary cancers (Xiong, Jia, & Wang, 2016)

Treatment: Management of bladder cancer depends on the stage of the disease. Initially, surgical intervention to remove the bladder tumors is warranted. Some patients may require a cystectomy with a urostomy, continent urostomy, or a replacement bladder. For patients with multiple recurrent superficial bladder tumors, the urologist may request collaboration with an oncologist for chemotherapy after surgery. Recent clinical trials of checkpoint inhibitors have indicated efficacy in the treatment of advanced and metastatic bladder cancer (Apolo, Vogelzang, & Theodorescu, 2015). Advanced disease generally requires surgery, radiation, and chemotherapy with combination agents. The patient's age and health status at the time of diagnosis must be considered in the management of bladder cancer (Hall et al., 2014; NCCN, 2016).

Follow-Up: Patients with superficial low-grade bladder cancer require a cystoscopy at designated intervals, although the value of repeated testing has been questioned. The need for supplemental nutritional support, pain management, prevention of complications such as skin breakdown, and an advance directive should be discussed during future follow-up care. Patients with a urostomy may need assistance from an ostomy nurse.

Sequelae: Metastasis to other parts of the body can occur. Survival of the untreated patient may be less than 2 years.

Prevention/Prophylaxis: Encourage patients who smoke to quit, and exhort all patients to decrease exposure to harmful chemicals.

Referral: Refer patients with clinically significant hematuria to a urologist. An oncologist also may be involved in the patient's management of the disease. Patient and family support is important at this time; information pertaining to hospice services should be provided. Patients with a urostomy may seek support from a local chapter of the United Ostomy Associations of America (UOAA) at 1-800-826-0826 or http://www.ostomy.org/Home.html.

Education: Older adults with bladder cancer may need to be educated about palliative support measures when the disease becomes terminal.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Urine dipstick is not recommended for screening of bladder cancer in the general population due to lack of specificity.	A	ACS, 2016; NCCN, 2016
Routine screening for bladder cancer is not recommended for the general population.	B	NCCN, 2016
Patients should be counseled to eliminate active and passive smoking for prevention of bladder cancer.	B	ACS, 2016; NCCN, 2016
Cystoscopy is recommended in all patients with symptoms suggestive of bladder cancer. It cannot be replaced by cytology or by any other noninvasive test.	A	NCCN, 2016

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BOWEL OBSTRUCTION

Signal Symptoms: Cramping abdominal pain, nausea, vomiting, obstipation.

Description: Bowel obstructions are classified as mechanical or nonmechanical and partial or complete, and are located in either the small or large bowel (Ansari, n.d.; Bordeianou & Ye, 2017). The acute development of a large bowel obstruction (LBO) is considered an abdominal emergency (Jaffe & Thompson, 2015).

Etiology: Normal flow of gastric and intestinal contents is interrupted (Bordeianou & Ye, 2017). A mechanical bowel obstruction results when there is a complete or partial blockage of the lumen of the bowel by an adhesion, tumor,

or hernia (Di Saverio et al., 2013). Postoperative adhesions cause approximately 60% to 70% of small bowel obstruction (SBO) (Di Saverio et al., 2013). Signs of an LBO are subtle compared to acute onset of SBO symptoms (Jaffe & Thompson, 2015). Bowel distention in LBO leads to bowel ischemia and perforation if not treated.

Occurrence: An estimated 300,000 admissions annually are due to SBO in the United States (Frasure, Hildreth, Takhar, & Stone, 2016; Taylor & Lalani, 2013).

Age: Bowel obstruction from all causes, except intussusception, is more prevalent in older adults.

Gender: Occurs equally in men and women.

Ethnicity: Not significant.

Contributing Factors: Previous abdominal or pelvic surgery with formation of adhesions is common in SBO (Bordeianou & Ye, 2017). Additional causes for mechanical obstruction include "diverticulitis, foreign bodies (including gallstones), volvulus (twisting bowel on its mesentery), and intussusception (telescoping of one segment of bowel into another), and fecal impaction" (Ansari, n.d., para. 3).

Signs and Symptoms: Older adults often present for evaluation late in their illness and often have atypical or nonspecific complaints (Springer, Bailey, Davis, & Johnson, 2014). The most common symptoms reported are cramping abdominal pain, nausea, vomiting, and obstipation (Ansari, n.d; Bordeianou & Ye, 2017).

Inspect the abdomen for evidence of surgical scars and external hernias; the level of the obstruction determines the amount of abdominal distention (Bordeianou & Ye, 2017). Auscultate bowel sounds. In acute mechanical bowel obstruction the bowel sounds are hyperactive and are described as "high-pitched tinkling" (Bordeianou & Ye, 2017). Palpation of the abdomen may reveal abdominal wall or groin hernias. Patients with a strangulating bowel may have a tender abdomen, continuous pain, and absent bowel sounds (Ansari, n.d.). In mechanical obstruction of the large bowel, symptoms are similar to an SBO, but appear more gradually. The patient complains of persistent constipation leading to abdominal distention; vomiting is uncommon. Physical examination reveals loud borborygmi, no abdominal tenderness, and an empty rectal vault. Perform a digital rectal examination to determine if fecal impaction or rectal mass is a source of the obstruction (Bordeianou & Ye, 2017).

Diagnostic Tests: Standard laboratory evaluation include CBC with differential and a metabolic panel to assess the patient for presence and severity of hypovolemia, leukocytosis, and metabolic abnormalities (Bordeianou & Ye, 2017).

The initial imaging of a patient with a suspected bowel obstruction is the plain abdominal x-ray with flat and upright views (Khan, n.d.). Plain films can identify "multiple air-fluid levels, distention of small bowel loops and the absence of gas in the colonic section" (Di Saverio et al., 2013, p. 3). If x-rays do not show an obstruction and there is a high index of suspicion, CT scan should be used to further evaluate. CT scan is found to have as much as a 90% sensitivity in identifying the presence of bowel strangulation and can identify the etiology and the site of the obstruction (Khan, n.d.). Ultrasonography is often used in the evaluation of acute abdominal pain and can be used to identify obstruction along with its cause.

Differential Diagnosis:

- Cholecystitis
- Constipation
- Diverticulitis
- Gallstones
- Abdominal hernia
- Appendicitis (Nobie, n.d.).

Treatment: SBO is a common reason for older adults to be admitted under general surgery and may be treated by nonoperative management (NMO) or surgery (Springer et al., 2014). Initial treatment of a bowel obstruction consists of hospitalization for nasogastric suctioning and IV fluid administration (Di Saverio et al., 2013). In the absence of clinical sign of strangulation or peritonitis, the patient can be treated with NMO up to 72 hours. Systematic signs of "tachycardia, fever, focal tenderness, and elevated lactate levels can indicate intestinal ischemia" (Di Saverio et al., 2013, p. 6). Immediate surgical intervention is indicated for suspected vascular insufficiency, perforation, or strangulation of the bowel.

Follow-Up: Di Saverio and colleagues (2013) recommend NMO should be attempted for 3 to 5 days, then surgical management used for unresolved obstructions.

Sequelae: Delay in surgery may increase morbidity and mortality outcomes in older adults (Di Saverio et al., 2013).

Prevention/Prophylaxis: Caregivers must understand the importance of avoiding fecal impactions in patients at risk for this condition.

Referral: Immediate surgical referral with suspected bowel strangulation or after 3 days if no resolution after NMO (Di Saverio et al., 2013).

Education: Inform older adults with diagnosed, untreated internal and external hernias of the possible complication of bowel obstruction.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
SBO is routinely diagnosed with plain film radiographs that show distended small bowel loops, air-fluid levels, and decreased large bowel air.	C	Khan, n.d.
CT scan should be done when radiography is inconclusive or strangulation is suspected.	C	Khan, n.d.
Discretion should be used when deciding to use nonoperative management beyond 24–48 hours in older adults.	C	Springer et al., 2014

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CHOLECYSTITIS

Signal Symptoms: Acute onset of right upper quadrant and/or epigastric pain that is persistent, with radiation to back or shoulders, and usually related to history of fatty meal intake prior to onset (Abraham, Rivero, Erlikh, Griffith, & Kondamudi, 2014).

Description: May fall into one of three categories: acute gallbladder inflammation related to cystic duct obstruction by gallstones, acalculous cholecystitis without presence of stones or sludge, or chronic gallbladder inflammation (Katabathina, Zafar, & Suri, 2015).

Etiology: Gallstones are mainly comprised of cholesterol, and stone formation is associated with high caloric intake, diagnoses of diabetes mellitus, dyslipidemia, obesity, and the metabolic syndrome (Abraham et al., 2014). Cholecystitis usually occurs when a gallstone obstructs the cystic duct; however, acalculous cholecystitis may be found in approximately 10% of cases, specifically in critically ill states (Katabathina et al., 2015). Trapped bile caused by the blockage of the cystic duct leads to inflammation, impaired circulatory flow, and ischemia. The risk of bacterial infection climbs, reaching 70% by 7 days with lack of appropriate treatment. Common culprit organisms include *Escherichia coli*, *Enterobacter*, *Klebsiella*, and *Enterococcus*. Acute acalculous cholecystitis (AAC) is precipitated by stagnant bile and ischemic states. The critically ill patient who has suffered trauma, recent surgery, shock, sepsis, and total parenteral nutrition is at risk for AAC. Additional comorbid diagnoses of heart failure or dehydrated states may contribute to AAC. The possibility of undetected gallbladder cancer needs to be considered in all older adults presenting with symptoms of cholelithiasis (Makino, Yamaguchi, Nariatsu, Toshiaki, & Ichiro, 2009).

Occurrence: Gallstones may be an incidental finding via imaging, and 10% to 20% of these individuals may exhibit symptoms 5 to 20 years after imaging (Abraham et al., 2015). Ninety percent of cholecystitis cases are associated with gallstones. In the older adult, cholecystitis can present with atypical features and likely a complicated disease course (Bergman et al., 2015).

Age: During reproductive years, women are more prone to gallstones than men (Dua et al., 2013). As age increases, the risk for gallstones increases regardless of gender (Kuy, Sosa, Roman, Desai, & Rosenthal, 2011). Thirty percent of those older than 70 years are at risk for gallstones (Demehri & Alam, 2016).

Gender: Twice as many women as men have cholecystitis (Dua et al., 2013).

Ethnicity: A high prevalence for cholecystitis exists in older Native Americans and Caucasians; the disease is less prevalent in African Americans. The prevalence of gallbladder disease is highest in the Pima, Hopi, and Navajo groups (Abraham et al., 2014).

Contributing Factors: Nonmodifiable risk factors for cholecystitis include advancing age, female gender, race, and family history of gallbladder disease (Abraham et al., 2014). Obesity is a known predisposing factor to cholecystitis (Abraham et al., 2014; Lee, Han, & Min, 2009). Diabetes, alcoholic cirrhosis, inflammatory bowel disease, chronic hemolysis, pancreatic insufficiency, and hyperlipidemia predispose patients to gallstone development. Gallstone formation may be attributed to use of exogenous estrogens prescribed for postmenopausal hormone replacement, and clofibrate, used to treat hyperlipidemia. Either past or current use of thiazide diuretics is associated with a modest increase in the formation of gallstones (Gorroll, 2014). Gallbladder stasis, a precursor to gallstone formation, occurs with extended total parenteral nutrition. Patients who experience rapid weight loss are at high risk for the development of gallstones. Cholelithiasis is common in patients who have undergone bariatric surgery (Decker, Swain, Crowell, & Scolapio, 2007).

Signs and Symptoms: The most common type of presentation includes the acute onset of right upper quadrant and/or epigastric pain that is moderate to severe in nature, persistent, and without any relieving factors (Abraham et al., 2014). Associated symptoms may include nausea, vomiting, malaise, fever, and poor appetite. A change in mental status in the older adult may be the only outward sign. Physical

examination may reveal right upper quadrant subcostal tenderness, guarding, and pain on inspiration (Murphy's sign) (Adedeji & McAdam, 1996; Katabathina et al., 2015). The absence of Murphy's sign does not exclude the acute illness. Biliary colic pain can localize to the midepigastric region; thus, it is important to question the patient about generalized upper abdominal pain (Trowbridge, Rutkowski, & Shojania, 2003). In a patient who has reported symptoms for days, rebound tenderness may suggest perforation. Jaundice on examination would raise concern for complicated clinical course secondary to biliary obstruction.

Diagnostic Tests: Mild leukocytosis with increased band formation is the most common abnormality seen on laboratory studies; however, white blood cell (WBC) counts may not be elevated in the older adult (Katabathina et al., 2015). Significant leukocytosis would raise concern for complicated cholecystitis secondary to perforation or gangrene (Abraham et al., 2014). Elevation in lipase, amylase, and elevated liver function tests would prompt further evaluation for gallstone pancreatitis. In chronic cholecystitis, however, laboratory values may be normal.

Real-time ultrasonography of the gallbladder and biliary tree is the diagnostic procedure of choice for both acute and chronic cholecystitis, showing gallstones, thickening of the gallbladder wall, and if the common bile duct is obstructed, dilation of the biliary tract (Abraham et al., 2014). An additional imaging modality called the hepatobiliary iminodiacetic acid scan (HIDA) is useful if there is concern for cystic duct obstruction or biliary dyskinesia. CT scan is often performed in evaluation of acute abdominal pain when diagnosis is unclear; yet, it is less sensitive at identifying gallstones than an ultrasound. In situations where choledocholithiasis is questioned, a magnetic resonance cholangiopancreatography (MRCP) is highly sensitive and specific (97% and 98%, respectively) for biliary tree obstruction. Elevated bilirubin greater than 4 mg/dL and a common bile duct dilated greater than 6 millimeters should prompt evaluation with MRCP to exclude the presence of choledocholithiasis. In cases where this is confirmed, an endoscopic retrograde cholangiopancreatography (ERCP) is warranted for stone extraction; however, an ERCP may also be used as a diagnostic tool if the clinical picture remains unclear but high suspicion exists for biliary obstruction.

Differential Diagnosis:

- Perforated peptic ulcer in older adults
- Appendicitis
- Liver abscess
- Diverticulitis
- Hepatitis
- Acute pyelonephritis
- GI carcinoma
- Acute pancreatitis
- Myocardial ischemia
- Herpes zoster
- Left lower lobe pneumonia
- Gastritis
- Cholangitis
- Irritable bowel syndrome
- GERD (Gorroll, 2014)

Treatment: Acute episodes require hospital admission in preparation for cholecystectomy; at this time antibiotics are given intravenously and selected based on targeting enteric pathogens. For uncomplicated cases in which there is no penicillin allergy, evidence of multi-organ involvement, or other hospital-acquired infection, agents such as amoxicillin/clavulanic acid or ceftriaxone and metronidazole are given (Demehri & Alam, 2016). Laparoscopic cholecystectomy should be undertaken in 24 to 48 hours, and delay of surgery for resolving inflammation is no longer indicated. Supportive measures to include analgesics and antiemetics may be given as needed. The risk of conversion to open cholecystectomy is linked with male gender, older age (greater than 60 years), and prior abdominal surgeries (Abraham et al., 2014).

A cholecystectomy for acalculous cholecystitis in the critically ill patient may be on hold and delayed until health improves, with a percutaneous cholecystostomy tube as an initial approach to treatment (Demehri & Alam, 2016; Macrì et al., 2006; Winbladh, Gullstrand, Svanvik, & Sandström, 2009). The general surgeon may weigh risks and benefits of a permanent cholecystostomy versus interval cholecystectomy in a patient with multiple comorbidities (Fuks et al., 2015). The recurrence risk of events following conservative management of cholecystitis is high; therefore, surgical intervention should be undertaken if there are no contraindications. In the event of choledocholithiasis, an ERCP may be indicated for stone extraction prior to cholecystectomy (Demehri & Alam, 2016). If the clinical status is unknown regarding choledocholithiasis, a cholecystectomy with intraoperative cholangiogram is indicated to ensure no occult stones remain. Cholangitis requires prompt recognition in order to provide IV antibiotics, biliary decompression via ERCP, and close monitoring for sepsis. Subsequent cholecystectomy is indicated after biliary decompression.

Although used less often now than in the past, oral ursodeoxycholic acid may be considered for dissolution of gallstones if surgery is contraindicated; however, the gallbladder must be functioning and the cystic duct patent (Abraham et al., 2014). Recurrence rate of stones is more than 50%, even with ursodeoxycholic acid. Extracorporeal shock wave lithotripsy is a possible alternative in nonsurgical candidates, yet potential complications to include biliary pancreatitis and hematomas may raise concern for the procedure.

Follow-Up: Postoperative management of routine cholecystectomy includes monitoring for impending infection, adverse drug reactions and interactions, and changes in functional and mental status. For the frail older adult who has undergone percutaneous cholecystostomy, observation for complications is crucial. The catheter of a cholecystostomy tube should be replaced every 3 months if used for long-term purposes, and the catheter should be flushed with normal saline every 24 hours (Katabathina et al., 2015). For older women, the dosages of estrogen preparations may need to be reduced, if applicable, due to the idea that estrogen may contribute to gallstone development (Demehri & Alam, 2016).

Sequelae: Complications of acute cholecystitis include ischemia and inflammation within the gallbladder lumen (Fuks et al., 2015), gallbladder necrosis with perforation and/or abscess, sepsis, choledocholithiasis, cholangitis, and pancreatitis (Katabathina et al., 2015). Frail older adults, especially those who are diabetic, are at high risk for complications from cholecystitis (Gorroll, 2014).

Prevention/Prophylaxis: The importance of resting and avoiding risk factors after acute cholecystitis or an exacerbation of chronic cholecystitis needs to be emphasized.

Referral: A surgical consultation is necessary if acute cholecystitis is suspected. A gastroenterologist should be consulted for the frail older adult who is not a surgical candidate, or in the clinical scenario of choledocholithiasis. Biliary pain in patients with a prior history of a cholecystectomy should be evaluated for a retained common bile duct stone. Referral to a gastroenterologist for evaluation by ERCP, MRI, or endoscopic ultrasonography is highly recommended (Agrawal, Morrissey, & Thakkar, 2012).

Education: To prevent complications, encourage patients with chronic cholecystitis and patients for whom surgery is contraindicated to report early signs and symptoms of an acute attack. Patients also should be educated that gallstones could recur in the common bile duct, despite prior cholecystectomy.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
For patients with cumulative risk factors for cholecystitis, early cholecystectomy is recommended before there is a progression of the disease leading to complications of acute cholecystitis.	B	Cho, Han, Yoon, & Ahn, 2010 Riall, Zhang, Townsend, Kuo, & Goodwin, 2010
Laparoscopic cholecystectomy is the preferred treatment for cholecystitis, barring any clinical contraindications.	A	Abraham et al., 2014
A positive Murphy's sign is a useful diagnostic tool when examining older adults presenting with symptoms of acute cholecystitis; a negative sign, however, should be cautiously interpreted.	C	Adedeji & McAdam, 1996 Abraham et al., 2014
Older adults who underwent ultrasonograpahic percutaneous cholecystostomy for the treatment of acute cholecystitis had complete resolution of symptoms within 48 hours.	B	Macrì et al., 2006
There was a negative correlation between BMI and the severity of cholecystitis; more complicated disease was found in males who were not obese.	B	Hyeon Kook, Ho-Seong, & Seog Ki, 2009
Older adult patients who underwent an early laparoscopic cholecystectomy did not experience any more complications or infections than younger adults who had the same procedure.	A	Fuks, Duhaut, Mauvais, Pocard, Haccart, Paquet, . . . Regimbeau, 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CHRONIC KIDNEY DISEASE

Signal Symptoms: Fatigue, anorexia, altered mental status, weakness, dysgeusia, pruritis, nausea, vomiting, edema.

Description: CKD continues to be a growing worldwide problem. According to CDC data, the prevalence of CKD was 25% higher from 2007 through 2014 than in 1988 through 1994 (CDC, 2015). *Healthy People 2020* has developed 14 objectives to attempt to reduce new cases, complications, and economic costs of CKD. CKD, when left untreated, results in ESRD, which requires dialysis in order for the patient to survive. It is estimated that 25% of the Medicare budget is spent on CKD (CDC, 2015). With the increased prevalence of diabetes and an aging population, incidence of CKD will continue to rise. Unfortunately, many people are unaware that they have CKD.

Etiology: CKD is defined as abnormalities of kidney structure or function, present for more than 3 months (Inker, 2014). Markers of kidney damage include albuminuria, urine sediment abnormalities, electrolyte abnormalities, abnormalities detected by histology or imaging, or a history of kidney transplantation. CKD is then further classified based on cause, GFR category, and albuminuria category (KDIGO, 2013). Following are the stages of CKD:

- Stage 1: GFR is greater than or equal to 90 mL/min with other evidence of CKD damage
- Stage 2: GFR 60 to 89 mL/min
- Stage 3a: GFR 45 to 59 mL/min
- Stage 3b: GFR 30 to 44 mL/min
- Stage 4: GFR 15 to 29 mL/min
- Stage 5: GFR less than 15 mL/min or on dialysis

Diabetes is the leading cause of kidney failure, followed by hypertension. Other causes of kidney disease include glomerulonephritis, inherited polycystic disease, autoimmune diseases, infections, and urological diseases.

Occurrence: The CDC estimates that more than 20 million people in the United States have CKD (CDC, 2015).

Age: Although CKD can affect any age group, incidence increases with age. For U.S. adults age 30 to 49, 50 to 64, and 65 years or older with no CKD at baseline, the residual lifetime incidences of CKD are 54%, 52%, and 42%, respectively (CDC, 2015). The prevalence of CKD in adults 30 years or older is projected to increase from 13.2% currently to 14.4% in 2020 and 16.7% in 2030 (CDC, 2015).

Gender: CKD affects females more than males (CDC, 2015).

Ethnicity: African Americans have a higher prevalence of CKD than Caucasians, according to USRDS data. This is closely followed by Hispanics/Mexican Americans (CDC, 2015).

Contributing Factors: Diabetes mellitus is the leading cause of CKD and is the leading risk factor in the development of CKD. This is followed closely by hypertension, both a cause and a consequence of renal disease. Inflammatory diseases, such as systemic lupus erythematosus, and malignancy, such as renal tumors or multiple myeloma, also increase the risk of kidney failure. Vascular disease, chronic pyelonephritis, urinary stones, systemic infections, gout (Roughley, Belcher, Mallen, & Roddy, 2015; Zhe & Hang, 2016), certain medications, and radiopaque dye also heighten the risk of developing CKD. AKI often predisposes a patient to the development of CKD. A family history of renal disease is an important risk factor in the development of CKD. Racial and ethnic minorities have an increased risk of CKD and are more likely to progress from CKD to ESRD. In addition, the presence of inherited disease, such as autosomal dominant polycystic kidney disease, will lead to development of CKD.

Exposure to toxins can also play a role in the development of CKD. Exposure can be occupational, environmental, or recreational. There is a strong association between smoking and development of CKD. Chronic smoking causes nephrosclerosis, which is caused by microvascular atherosclerosis, resulting in hypertension. Illicit substances associated with CKD include heroin and cocaine. Recently, methamphetamines and ecstasy use have been associated with CKD. Cocaine use is associated with acute rises in blood pressure, leading to AKI from hypertension. Although heroin use was initially thought to cause focal glomerulosclerosis, it is now thought that substances mixed with the heroin or even transmittable diseases, such as hepatitis and HIV associated with IV injection of street drugs, are the cause of the CKD (Connor, 2011). Methamphetamines cause accelerated hypertension, causing renal damage. Ecstasy use can lead to rhabdomyolysis, drug-induced vasculitis, and severe hyponatremia. Recurrent episodes of rhabdomyolysis injury could lead to CKD for the patient (Connor, 2011). Exposure to heavy metals such as lead, cadmium, mercury, and organic solvents has been strongly linked to CKD; thus, screening for occupational hazards is a must.

Signs and Symptoms: Unfortunately, CKD does not produce symptoms until renal injury is quite advanced. It is often known as a silent disease. Many individuals are unaware that they have CKD until it progresses to stages 3 or 4. In stages 1 and 2, BUN and creatinine levels are normal or nearly normal, and acid-base, fluid, and electrolyte balances are maintained through the adaption of remaining nephrons. In stage 3, there is moderate impairment of GFR, and BUN and creatinine levels begin to increase. The patient usually remains asymptomatic, but levels of erythropoietin and parathyroid hormone are abnormal. Early symptoms of kidney disease, if any, may be similar to other illnesses. Only when the patient develops stage 4 or 5 of kidney disease do more profound symptoms of CKD occur. These include fatigue, loss of appetite and weight loss, dyspnea, edema in upper and lower extremities, confusion and difficulty with concentration, pruritus, dysgeusia, and foul-smelling breath (CDC, 2015).

Although diagnosis of kidney disease is based heavily on laboratory data and diagnostic imaging, a careful history may lead to clues associated with the diagnosis of CKD. Careful review of any kidney disease in the family is imperative, including questions relating to any hereditary diseases, such as autosomal dominant polycystic kidney disease. If the patient has a past history of pregnancy, ask whether there were any complications, including proteinuria or preeclampsia. Any past urological conditions could indicate postrenal causes of CKD.

Any history of diabetes mellitus or hypertension should immediately raise a strong suspicion for the presence of CKD. In addition, the patient should be questioned regarding any history of congestive heart failure, cirrhosis, or GI symptoms that may cause prerenal CKD. Disorders of the urinary tract, recent systemic infections, unusual rashes, or skin lesions should also raise suspicion of CKD. A through medication history should also be obtained to identify medications that may be contributing to CKD.

Diagnostic Tests/Tools: As stated previously, estimation of eGFR is the best indicator to level of kidney function and functioning renal mass. GFR declines after damage to the kidney structurally. GFR and albuminuria should be assessed yearly in patients with CKD and with increased frequency in those patients who have high risk of rapid progression of CKD. Serum creatinine alone as a decline in eGFR may occur before any rise in creatinine is noted. eGFR is best calculated using the 2009 Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) (KDIGO, 2013).

The formula is as follows:

$$\text{eGFR} = 141 \times \min(S_{Cr}/\kappa, 1)^{\alpha} \times \max(S_{Cr}/\kappa, 1)^{-1.209} \times 0.993^{\text{Age}} \times 1.018\,[\text{if female}] \times 1.159\,[\text{if African American}]$$

eGFR in CKD is more accurate than those in patients with AKI because of their steady state of kidney function. A creatinine clearance, in which a volume of urine is collected over 24 hours and the amount of creatinine secreted is measured, is a more accurate indicator of eGFR because it is a direct measurement.

Albuminuria, defined as the abnormal loss of albumin in the urine, is also indicative of CKD. Albumin is a plasma protein that is the principal component in most kidney diseases. It is a very common finding in CKD, indicative of glomerular diseases. Albuminuria of more than 30 mg/24 hours for 3 months is indicative of CKD (KDIGO, 2013).

In addition to albuminuria, there are other markers that indicate damage to the kidneys. These include abnormalities in urinary sediment and abnormal findings on imaging studies. Examination of the urinary sediment for WBCs, RBCs, or casts may indicate the presence of acute or chronic kidney disease and requires further investigation. Imaging studies should also be performed to suggest the cause of CKD. Ultrasound examination may indicate the presence of kidney stones, hydronephrosis, cysts, or masses. Small kidneys on ultrasound suggest CKD, whereas "large" kidneys may indicate tumors, cysts, or infiltrating diseases. CT scan may reveal tumors, obstructions, cysts, or calculi.

Further laboratory data that should be assessed in patients with possible CKD include CBC and electrolytes. Fasting lipids and HbA1c should be evaluated in the diabetic patient. Serum calcium, phosphorus, parathyroid hormone, and vitamin D levels should be drawn to evaluate for the changes in mineral metabolism and bone structure that begin early in CKD. Iron studies, vitamin B_{12}, and folate should also be completed and replacement begun, if warranted.

Differential Diagnosis:

- Acute kidney injury
- Cirrhosis
- Uropathic obstruction
- Nephrotic syndrome
- Tubulointerstitial disease (Bazari, 2014)

Treatment: Referral to a nephrologist should occur if 1) GFR is less than 30 mL/min, 2) there is a decline in GFR greater than 3 mL/min, 3) albuminuria is present, 4) it is difficult to control hypertension, and 5) there is unexplained anemia with GFR less than 60 mL/min (Johnson, 2011). Further investigation using the previous diagnostic tests will assist the nephrologist in the determination of the cause of the CKD. Once the stage of CKD is identified, management of CKD is aimed primarily at preventing progression of the disease through medications, risk factor reduction, lifestyle changes, and education of the patient and family regarding kidney disease. Eventually there is decline in kidney function to ESRD. Patients then must decide between renal replacement therapy—hemodialysis or peritoneal dialysis—in order for the patient to survive, or opt for no renal replacement therapy and end-of-life care.

Patients with CKD have a greater risk for cardiovascular disease than those without. As stated previously, hypertension is both a cause and a sequela of kidney disease. It is associated with a faster decline in kidney function by increasing albuminuria and furthering the development of cardiovascular disease. Thus, reducing the risk of progression of CKD and advancement of cardiovascular disease is paramount. Reduction of blood pressure to target levels is the most important treatment step in managing CKD. The targeted blood pressure for patients with hypertension as recommended by KDIGO is systolic blood pressure less than 140 mm Hg and diastolic blood pressure less than 90 mm Hg (2012). Patients who are at greatest risk for complications or already have evidence of cardiovascular disease may need more aggressive hypertension.

Multiple medications are frequently required in CKD patients to achieve goal blood pressure. ACEIs and ARBs are first-line treatment as antihypertensive medications. These agents reduce albuminuria and slow the rate of decline in kidney function in both diabetic and nondiabetic kidney disease. When beginning a patient on an ACEI or ARB, serum creatinine and GFR should be measured at 1 week and 4 weeks after initiation of therapy. If the initial rise in creatinine is more than 30% above the baseline value, the ACEI or ARB should be discontinued. ACEIs or ARBs should also be withdrawn if serum potassium concentration exceeds 6 mmol/L despite dose reduction, potassium diet restriction, and diuretic therapy. In addition, treatment response to ACEIs or ARBs can be monitored by albuminuria. Reduction in urinary albumin excretion will decrease risk of development of end-stage kidney disease and cardiovascular disease. Furthermore, sodium restriction of less than 2 g per day is recommended for blood pressure control. This is especially important in "salt sensitive" groups; for example, African Americans, older individuals, and those with diabetes, hypertension, and CKD. These groups are more salt sensitive and have a greater response in their blood pressure with increased intake of salt.

Because of the increased salt and water retention with CKD, use of a diuretic should be included in the antihypertensive regimen of the CKD patient. Thiazide diuretics often become less effective as GFR falls below 40 mL/min due to decreased renal blood flow. Usually a loop diuretic, such as furosemide, torsemide, or bumetanide, is used. Side effects of loop diuretics include hypokalemia and hypomagnesia, which can lead to increased cardiovascular risk of arrhythmias. Thus, serum electrolytes should be monitored, and the patient should be instructed to report any paresthesias, rash, or tinnitus. Beta blockers such as carvedilol, metoprolol, or bisoprolol should be used in patients with CKD when a proper dose of ACEI, ARB, or diuretic is not enough to lower blood pressure. Although these agents do not provide any renoprotective effects, they do assist in reducing cardiovascular mortality in high-risk patients. Beta blockers should be avoided in patients with bradycardia or heart block.

Calcium channel blockers (CCBs) are also effective agents that can be used to lower blood pressure in CKD patients; however, there are differences between these medications' effects on reducing proteinuria. Nondihydropyridine CCBs (verapamil, diltiazem) reduce proteinuria among patients with advanced nephropathy, whereas dihydropyridine CCBs (nifedipine, felodipine, amlodipine) will not have this effect unless coupled with an ACEI or ARB (Kalitzidis, 2011). They should not be used as monotherapy in CKD patients but in combination with an ACEI or ARB to achieve optimal blood pressure control. Central alpha-adrenergic agonists decrease the effects of sympathetic activity to lower blood pressure. Vasodilators such as hydralazine and minoxidil are often used as fourth-line agents when other antihypertensive treatments have failed. Vasodilator therapy does not reduce proteinuria and has not been shown to improve renal outcomes. Side effects of minoxidil include increased hair growth and risk of pericardial effusion. Hydralazine is the leading cause of drug-induced lupus syndrome and should be avoided in any patient with an autoimmune disease.

Tight glycemic control in CKD patients reduces cardiovascular disease and CKD progression risk in both type 1 and 2 diabetes mellitus. An HbA1c of less than 7.0% is the goal of patients with diabetes and CKD (KDIGO, 2012). Although achieving this can be difficult for some patients and is associated with increased risk of hypoglycemia, tight control reduces not only albuminuria but also retinopathy and neuropathy. Insulin can be used to treat diabetes in CKD, and doses should be tailored to attain the goal HbA1c while avoiding hypoglycemia. Insulin types and doses should be individualized to each patient and to the patient's level of CKD. First-generation sulfonylureas are metabolized in the kidney and may lead to longer half-lives with greater risk of hypoglycemia and, thus, should be avoided in CKD. Metformin, a biguanide, is also metabolized in the kidney and increases risk for lactic acidosis and so should also be avoided. Exenatide (Byetta) is not recommended for use in patients with a GFR less than 30 mL/min and has actually been found to cause renal failure in a number of patients (Hahr, 2011).

Anemia is a common complication of CKD. It worsens as kidney function declines, becomes prevalent in CKD stage 3, and continues through stage 5 into ESRD. The primary cause of anemia is decreased secretion of erythropoietin from decreased functioning renal cells, resulting in fatigue, decreased exercise capacity, decreased cognition, and impaired immunity, leading to decreased quality of life. Workload of the heart is increased with anemia, leads to left ventricular hypertrophy and cardiomyopathy, and increases the patient's risk of death from ischemic heart disease and heart failure. Target hemoglobin goals are 11 to 12 g/dL in patients with CKD (KDIGO, 2012). In addition, iron studies should be completed to evaluate for a true iron deficiency or functional iron deficiency. Iron replacement is given orally as ferrous sulfate. IV iron is usually reserved for those patients severely iron depleted or those patients on hemodialysis. Erythropoietin-stimulating agents can be given once iron deficiency is corrected, other causes of anemia have been excluded or treated, and hemoglobin is consistently below 10 g/dL. Epoetin alfa and darbepoetin alfa are two genetically engineered agents that can be administered to CKD patients to increase bone marrow production of RBCs.

Changes in mineral metabolism and bone structure begin early in CKD. This not only encompasses bone disease but also vascular and soft tissue calcification. As kidney function worsens and GFR continues to decline, the kidney must work harder to excrete phosphorus. This then reduces the formation of the active form of vitamin D (1,25D). Low levels of 1,25D, coupled with low serum calcium levels from decreased gut absorption, lead to a rise in parathyroid hormone. This leads to secondary hyperparathyroidism due to hypocalcemia, hyperphosphatemia, and low 1,25D levels. The increased parathyroid hormone affects bone metabolism and increased bone turnover, causing decreased cortical bone and bone strength. This increases risk of fracture and causes increased "bone pain" in CKD patients. Development of secondary hyperparathyroidism can be decreased by dietary restriction of high-phosphate foods such as fast food, dairy products, colas, and processed food products. A phosphate binder, which binds to the phosphate in food and is eliminated in the bowel movement, is also prescribed. Calcium acetate, lanthanum carbonate, and sevelemer carbonate are the common phosphate binders used today. In addition, administration of vitamin D (Rocaltrol, Hectoral, and Zemplar) also helps to reduce parathyroid hormone secretion.

Patients with CKD are also at increased risk for malnutrition and hypoalbuminemia. These are associated with poor outcomes when the time comes for the patient to initiate dialysis. The restriction of protein in the CKD population has been a topic of ongoing debate. Current recommendations from KDIGO (2012) include a protein intake of 0.8 gm/kg/day in patients with CKD. Patients need to be monitored for malnutrition with serum albumin levels and body weight assessments.

Smoking cessation is strongly encouraged in patients with CKD. Smoking compounds the risk for cardiovascular mortality in patients with CKD and hastens progression of kidney disease. It should be addressed with each office visit and risks reviewed with the patient.

Unfortunately, despite optimal treatment and practices by the patient, kidney disease may continue to progress until kidney failure occurs. Patients then develop uremia, which is the accumulation of toxins within the blood. This leads to anorexia, nausea, vomiting, asterixis, muscle weakness, platelet dysfunction, pericarditis, mental status changes, and possibly coma. At this point, dialysis is initiated to "clean" the blood of these toxins and will need to continue throughout the patient's lifetime.

Follow-Up: Care of the patient with CKD is in the hands of the nephrologist for the rest of the patient's life span. Unfortunately, timely referral to help keep the patient from progression to ESRD is often not done. In addition, some patients are not aware that they have CKD. It has been found that the worse the patient's kidney function is, the more aware they are of their disease (CDC, 2012). According to KDIGO, any patient with the following criteria should be referred to nephrology for further evaluation:

- AKI or abrupt sustained fall in GFR
- eGFR less than 30 ml/min/1.73 m2
- A consistent finding of significant albuminuria (ACR greater than 300 mg/g [greater than 30 mg/mmol]) or AER greater than 300 mg/24 hours
- Progression of CKD
- Urinary RBC casts, RBC 420 per high power field sustained and not readily explained
- CKD and hypertension refractory to treatment with four or more antihypertensive agents
- Persistent abnormalities of serum potassium
- Recurrent or extensive nephrolithiasis
- Hereditary kidney disease

The nephrologist continues to manage the patient in an attempt to avoid any further progression of CKD. Tight glycemic control, optimal blood pressure control, dietary phosphate restriction, and management of anemia are paramount. Patient education of CKD continues with every office visit as well. In addition, once the patient reaches stage 4, "options classes" are held with the family and the patient to discuss dialysis modalities (i.e., peritoneal dialysis or hemodialysis). Referral for renal transplant should be made before the patient begins dialysis. If the patient chooses not to proceed with dialysis, referral should be made for palliative care to discuss end-of-life decisions.

Sequelae: The major sequela of CKD is ESRD. In addition, CKD is associated with an increased risk for cardiovascular disease. Thus, lifestyle changes, such as maintaining healthy body weight, restriction of salt and high-fat diet, active participation in an exercise program, smoking cessation, limited alcohol intake, and tight glycemic and blood pressure control are important, not only to reduce cardiac risk, but also to slow progression of kidney disease.

Prevention/Prophylaxis: Early screening for albuminuria in patients at risk for CKD is paramount. In addition, other risk factors associated with CKD, including level of GFR, level of albuminuria, age, sex, race/ethnicity, elevated blood pressure, hyperglycemia, dyslipidemia, smoking, obesity, history of cardiovascular disease, ongoing exposure to nephrotoxic agents, and others should not be ignored (KDIGO, 2012). Tight glycemic control, optimal blood pressure control, tobacco cessation, low-salt diet, avoidance of nephrotoxic medications, and optimal weight with healthy lifestyle all assist in reduction of risk of CKD.

Referral: All patients with CKD should be referred to a nephrologist for ongoing monitoring.

Education: Patients with CKD are educated regarding their disease and the manifestations of this disease. Continued education of low sodium and low protein diet, compliance with medications, tight glycemic control, and avoidance of NSAIDs and contrast dye is important. Patients are encouraged to continue to follow up with their primary care physician for other aspects of their health care.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Assessment of renal function is made by eGFR.	C	KDIGO, 2012
Albuminuria of >30 mg/24 hours for 3 months is indicative of CKD.	C	KDIGO, 2012
Patients with or without diabetes mellitus should be treated primarily with an ACEI or ARB to decrease albumineria.	C	KDIGO, 2012
Target hemoglobin A1c for people with diabetes should be <7%.	C	KDIGO, 2012

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CIRRHOSIS OF THE LIVER

Signal Symptoms: Jaundice, fatigue, spider angiomas, palmar erythema, nodular liver.

Description: Cirrhosis occurs when there is chronic insult to the liver, resulting in fibrous and nodular regeneration of the existing hepatocytes. Forty percent of patients are asymptomatic initially when cirrhosis is diagnosed as part of a routine examination (Heidelbaugh & Bruderly, 2006). Cirrhosis is the eighth leading cause of death in the United States and a significant financial burden, with annual direct costs of approximately $2 billion and indirect costs of $10 billion (Ge & Runyon, 2016).

Etiology: Chronic alcoholism, viral hepatitis, and nonalcoholic fatty liver disease (NAFLD) are the most common causes of cirrhosis, together accounting for 80% of patients on the wait list for liver transplant from 2004 through 2013 (Wong et al., 2015). However, nonalcoholic steatohepatitis is expected to become the number one indication for liver transplant due to the increasing prevalence of fatty liver disease (Ge & Runyon, 2016). The cause of primary biliary cirrhosis, a chronic inflammatory disease of the liver in which the intrahepatic bile duct is destroyed, is unknown. Other less common causes include autoimmune hepatitis, primary sclerosing cholangitis, medications, Wilson's disease, Alpha-1 antitrypsin deficiency, right-sided heart failure, and others (Heidelbaugh & Bruderly, 2006).

Occurrence: In the United States, data from 1999 through 2010 estimated the prevalence of cirrhosis at 0.27%, or approximately 633,323 adults (Scaglione et al., 2015). True estimates are difficult to derive, as many patients with cirrhosis are asymptomatic, and an estimated 69% of patients are unaware that they have liver disease.

Age: Cirrhosis, which has an increase in onset in older adults, is the twelfth leading cause of death, predominantly in patients with alcoholic liver disease (Heidelbaugh & Bruderly, 2006; Starr & Raines, 2011). Onset of primary biliary cirrhosis usually occurs before age 65 years.

Gender: Overall, being male is independently associated with cirrhosis (Scaglione et al., 2015). However, cirrhosis of the liver is equally prevalent in men and women with chronic alcoholism.

Ethnicity: The overall prevalence of cirrhosis is higher in non-Hispanic African Americans and Mexican Americans (Scaglione et al., 2015).

Contributing Factors: Chronic alcohol consumption, combined with a poor nutritional intake, contributes to cirrhosis. Drug-induced cirrhosis can occur in patients taking large doses of vitamin A, aldomet, isoniazide, and methotrexate. Certain infectious diseases (tertiary syphilis, brucellosis, schistosomiasis) have predisposed patients to a risk of developing

cirrhosis. Patients with Wilson's disease, sarcoidosis, heart failure, and hemochromatosis may go on to develop cirrhosis (Heidelbaugh & Bruderly, 2006). A history of the following chronic conditions can contribute to the development of biliary cirrhosis: biliary obstruction, cystic fibrosis, primary sclerosing cholangitis, and congenital biliary cysts (Heidelbaugh & Bruderly, 2006). Chronic hepatitis B and C viruses also place patients at risk for developing cirrhosis (Chen, Chu, Yeh, & Liaw, 2007; Starr & Raines, 2011). Risk factors for developing cryptogenic cirrhosis, which occurs in patients with NAFLD, include obesity, diabetes mellitus, hypertriglyceridemia, and significant weight loss in patients who have had jejunoileal bypass surgery (Gill & Wu, 2006; Riley, Taheri, & Schreibman, 2009).

Signs and Symptoms: Many patients can be asymptomatic until advanced disease sets in. Symptoms may include anorexia, fatigue, generalized pruritus, jaundice, and easy bruising of the skin. When questioned, reports of weight loss, weakness, and GI bleeding may be common. An assessment tool, such as the AUDIT (Alcohol Use Disorders Identification Test) or the CAGE Questionnaire (Ewing, 1984), is recommended to discern alcohol-related problems. It allows the practitioner to explore the patient's history of chronic alcohol use and the status of functional impairment. Physical examination may reveal evidence of scleral icterus, Kayser-Fleischer ring around the cornea of the eye, xanthoma, palmar erythema, spider angiomas, dilated abdominal veins (caput medusae), clubbing of fingers, changes in the nail bed (Terry's nails, with two-thirds of the nail bed appearing white), Dupuytren's contracture, asterixis, and in men, decreased body hair, gynecomastia, and testicular atrophy. Palpation of the liver may reveal a firm, nodular liver; splenomegaly; and ascites (Heidelbaugh & Bruderly, 2006; Starr & Raines, 2011).

Diagnostic Tests: CBC with indices may reveal anemia, which is typically multifactorial in nature. Potential etiologies, depending on etiology and manifestation of the underlying liver disease, may include chronic GI bleeding, alcohol toxicity, folate deficiency, anemia of chronic disease, splenomegaly, suppression of bone marrow, and hemolysis. A decreased serum albumin level and prolonged PT/INR occur in cirrhosis. The serum aspartate aminotransferase (AST) level is elevated; the alanine aminotransferase (ALT) level is usually above normal but not to the degree of the AST. The ALT is a more specific test for recognizing liver disease than the AST (Duke, 2012). The ammonia level is elevated in patients with hepatic encephalopathy. The alpha-fetoprotein (AFP) level is elevated in patients with hepatitis C and is also a tumor marker for hepatocellular carcinoma (Duke, 2012; Starr & Raines, 2011).

Abdominal ultrasound can be ordered initially when cirrhosis is suspected, given clinical symptom presentation and other diagnostic studies as surface nodularity, ascites, increased parenchymal echogenicity of the liver, and the direction of portal blood flow can be detected using Doppler scanning (Heidelbaugh & Bruderly, 2006). Liver biopsy, either percutaneous or transjugular, is the gold standard for diagnosing liver cirrhosis, with a sensitivity between 80% and 100% (Bravo, Sheth, & Chopra, 2001). However, biopsy may be unnecessary if clinical assessment, laboratory findings, and imaging results significantly suggest cirrhosis and biopsy would not alter patient management. Additionally, noninvasive measures such as liver ultrasound elastography and laboratory studies like AST to platelet ratio and Fibrotest/Fibrosure have emerged, although a standard has not been identified (Chou & Wasson, 2013; Lin et al., 2016).

Differential Diagnosis:

- Liver cancer
- Alcoholic hepatitis
- Congenital hepatic fibrosis
- Portal hypertension
- Schistosomiasis (O'Shea, Dasarathy, & McCullough, 2010)

Treatment: Treatment of cirrhosis is primarily dependent on the cause of the disease. Cessation of all alcohol intake is imperative in all cases (O'Shea, Dasarathy, & McCullough, 2010). Patients should also avoid or have dose-reduction with hepatotoxic medications such as NSAIDs, acetaminophen, systemic antifungals, and statins. Herbal medications have also been shown to be hepatotoxic, including germander, mistletoe, green tea extracts, kava kava, and others (Pittler & Ernst, 2003). Review the patient's immunizations and determine the need for influenza, hepatitis A and B, and pneumococcal pneumonia vaccines. Nutritional therapy is of the essence, as 20% to 60% of cirrhosis patients suffers from malnutrition (Ge & Runyon, 2016). Nutritional support includes at least 1.0 to 1.5 g protein/kg dry body weight; cirrhosis patients with ascites should have a 2,000 mg daily sodium restriction. It is highly recommended that patients with cirrhosis have small, frequent nutritious feedings, including a small snack at bedtime and a small morning meal, to improve overall nitrogen balance without exacerbating hepatic encephalopathy. Multiple-vitamin supplements should be prescribed, including adequate intake of vitamins A, D, and K. It is recommended, however, that a multiple vitamin without iron be recommended, unless iron-deficiency anemia is present (O'Shea et al., 2010).

Treatment for complications of cirrhosis may include antibiotics for infections such as spontaneous bacterial peritonitis, diuretics if the patient has ascites, beta blockers for portal hypertension, and symptomatic relief of pruritus (Heidelbaugh & Bruderly, 2006). For hepatic encephalopathy treatment or prevention, lactulose and/or Rifaximin are commonly used (NeSmith, Ahn, & Flamm, 2016). Therapeutic paracentesis may be needed for recurrent, especially large volume, ascites and eventually a transcutaneous intrahepatic portosystemic shunt (TIPS) may be necessary. Esophagogastroduodenoscopy (EGD) can be used to screen for esophageal varices, especially in patients who cannot undergo nonselective beta blocker prophylaxis. Variceal banding can be utilized to prevent or treat variceal hemorrhage, which has been associated with a mortality rate near 15% (Cabrera, Tandon, & Abraldes, 2016).

Follow-Up: Surveillance of the older adult with cirrhosis depends on the stability of the patient and the presence of complications. Stable older adults should have repeat CBCs, complete metabolic profile, and PT/INR 6 months to 1 year after initial diagnosis and every 6 months to identify cirrhosis complications such as anemia, thrombocytopenia, coagulopathy, metabolic derangements, and others. Additionally, model for end-stage liver disease (MELD) score should be

calculated every 6 months (Harrison, Hogan, Floros, & Davies, 2016). Unstable patients may need to be monitored more frequently.

All adults with cirrhosis should undergo liver ultrasonography of CT imaging every 6 months to screen for hepatocellular carcinoma (HCC) (Bruix & Sherman, 2011; Ge & Runyon, 2016). The use of alpha fetoprotein laboratory test in conjunction with ultrasonography for HCC detection remains controversial, as it results in increased detection rates, but also increased costs and false positives. All cirrhosis patients should undergo EGD for esophageal variceal screening and guideline-based repeat EGD for surveillance (Ge & Runyon, 2016). On return visits, observation and testing for changes in mental status and depression may be indicated. Other complications of cirrhosis, listed under Sequelae, will require additional medical therapy.

Sequelae: Complications from cirrhosis include ascites, spontaneous bacterial peritonitis resulting from uncontrolled ascites, portal hypertension, hepatopulmonary syndrome, portopulmonary hypertension (Kochar, Nevah, Moises, & Fallon, 2011), esophageal variceal bleeding, renal failure, and hepatic encephalopathy and hepatorenal syndrome. Patients with ascites and cirrhosis are at risk for developing an umbilical hernia (Eker et al., 2011). The incidence of developing cholelithiasis increases in patients with cirrhosis (Pessaux & Lermite, 2008). There is a very high risk of developing hepatocellular carcinoma, at approximately 5% per year, which is the leading cause of death for patients with cirrhosis, and therefore surveillance (as stated previously) is necessary (Ge & Runyon, 2016).

Alcoholic hepatitis may occur with cirrhosis and is generally defined as alcohol-related acute onset of symptomatic hepatitis. Severity is measured with calculation of discriminant function (DF), which is a score based on PT and serum bilirubin (Gholam, 2016). General management of these patients includes alcohol abstinence, fluid management, nutritional support, and acid suppression (such as proton pump inhibitors [PPIs] or H_2 receptor blockers) to prevent mucosal bleeding (Saberi, Dadabhai, Jang, Gurakar, & Mezey, 2016). Patients with moderate to severe alcoholic hepatitis have 1 month mortality rates as high as 23% (Yu, Xu, Ye, Li, & Li, 2010). In severe alcoholic hepatitis (DF greater than or equal to 32), the use of nonselective beta blockers has been associated with increased risk of acute kidney injury and should therefore be discontinued (Sersté et al., 2015). Additionally, patient with severe alcoholic hepatitis should be treated with a 4-week course of prednisolone (40 mg/day for 28 days, typically followed by discontinuation or a 2-week taper), unless steroid therapy is contraindicated, as this has been shown to reduce mortality rates in these patients (Thursz et al., 2015).

Prevention/Prophylaxis: Cessation of alcohol consumption is crucial to the prognosis of cirrhosis. Avoidance of hepatotoxic medications, especially NSAIDs, is recommended for patients with cirrhosis. All patients with cirrhosis should be given the hepatitis A and B vaccines, unless they are shown to be immune to the diseases. The polyvalent pneumococcal vaccine and annual influenza vaccines also are recommended for these patients. Preventive strategies for patients with cirrhosis need to focus on the prevention of complications by instituting measures such as routine follow-up with laboratory testing and abdominal imaging, as well as endoscopic screenings for varices and variceal prophylaxis, unless contraindicated.

Referral: A gastroenterologist should be consulted when varices are suspected and variceal bleeding occurs. An EGD is performed to manage complicated patients. Patients presenting with a new onset of ascites need to be referred for consideration of diagnostic paracentesis (Starr & Raines, 2011). Referral to a hepatologist for patients with cirrhosis should be considered early on to plan management for complications and formulate plans for end-stage liver disease. Additionally, all patients with a MELD score greater than or equal to 15 or complications of cirrhosis should be referred to a transplant center for evaluation (Kanwal et al., 2010; Runyon & AASLD Practice Guidelines Committee, 2009).

Education: Instruct patients to eliminate all alcohol consumption. Recommend an alcohol treatment program and provide the telephone number for the nearest chapter of Alcoholics Anonymous. Patients should be requested not to self-medicate with OTC medications, including herbal products. Patients with cirrhosis should eat small, frequent meals of a balanced diet containing 1.0 to 1.5 mg protein/kg body weight per day, unless contraindicated by advanced disease. Patients with encephalopathy should not be driving. For patients with end-stage liver disease, palliative care measures should be initiated.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Patients who have cirrhosis associated with a MELD score of 15 or greater or with complications of cirrhosis should be referred to a transplant center.	A	Kanwal et al., 2010 Runyon & AASLD Practice Guidelines Committee, 2009
Patients with severe alcoholic hepatitis with or without hepatic encephalopathy and lacking contraindications to steroid use should be considered for a 4-week course of prednisolone (40 mg/day for 28 days, typically followed by discontinuation or a 2-week taper).	A	O'Shea et al., 2010 Uribe et al., 1978

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Patients should be highly encouraged to abstain from drinking any alcohol.	B	Borowsky, Strome, & Lott, 1981
It is highly recommended that patients with cirrhosis have small, frequent, nutritious feedings, including a small snack at bedtime and a small morning meal, to improve overall nitrogen balance.	A	
Ascites should be treated with salt restriction and diuretics.	A	Kanwal et al., 2010 Licata et al., 2009
Persistent hepatic encephalopathy should be treated with disaccharides or rifaximin (Xifaxan).	B	Kanwal et al., 2010 Bass et al., 2010

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CLOSTRIDIUM DIFFICILE

Signal Symptoms: Profuse, watery, malodorous diarrhea; abdominal discomfort; fever.

Description: *Clostridium difficile*-associated diarrhea (CDAD) and colitis are a consequence of the colonization and subsequent infection of the colon by this organism following a disruption of the normal flora of the gut by antibiotic exposure (Keller & Surawicz, 2014). Once established in the colon, *C. difficile* produces two potent toxins, *Toxin A* and *Toxin B*, that cause fluid secretion, inflammation, and injury to the colonic mucosa, resulting in diarrhea and/or colitis. Due to increasing antibiotic usage, new and dangerous strains continue to emerge (Trier, 2009). Intestinal colonization by toxigenic *C. difficile* strains does not always result in infection or clinical symptoms, meaning some individuals may be asymptomatic carriers. Clinical presentation of *C. difficile* diarrhea varies from mild diarrhea as the only symptom to fulminant, life-threatening colitis.

Etiology: CDAD is no longer a hospital-acquired infection only, as outpatient incidences are rising (Lessa et al., 2015). Risk factors include antibiotic exposure, older adults, and healthcare facility exposure; however, the presence of inflammatory bowel disease, compromised immunity, liver and kidney disease, and prior GI procedures also play a role in increasing the risk for CDAD (Keller & Surawicz, 2014; Surawicz et al., 2013). Less-known risk factors include intensive care admission, prior hospitalization within several months, ventilatory support, enteral feedings, use of histamine blockers and PPIs, malnourished states, and hypoalbuminemia (Kim, Lee, & Jeong, 2010; Morrison et al., 2011; Tleyjeh et al., 2013).

Bacterial spores are spread via the fecal-oral route and thrive on surfaces such as toilets, hospital equipment, and door handles (Keller & Surawicz, 2014). These spores have the potential to survive for months, even in harsh circumstances (Jump, Pultz, & Donskey, 2007; Keller & Surawicz, 2014). Asymptomatic carriage of the organism by patients is not uncommon and may hinder adequate infection control measures. The transfer of patients harboring *C. difficile* infection (CDI) from one hospital or long-term care facility to another enables spread of the infection within geographical regions (Cohen et al., 2010; Kee, 2012).

Occurrence: Recent estimations report 29,000 individuals with CDAD succumbed to the illness in 2011, with around 453,000 cases per year (CDC, 2015). Two-thirds of the cases per year are associated with hospital or nursing home admission, while the remaining population may be without any recent health-care exposure. The incidence rate of outpatient infections is increasing, and the overall incidence of CDAD has doubled in prevalence from 2000 to 2015 (Lessa et al., 2015). The incidence in long-term care patients varies from 4% to 50% (Kee, 2012).

Recurrence and risk of death are more common with hospital-acquired infections (Lessa et al., 2015). Recurrence of CDAD is defined by the presence of diarrhea and a positive stool test, usually occurring 1 to 3 weeks after treatment completion for CDAD (Chopra & Krishna, 2014). Different strains of the bacteria may be responsible for recurrent infection, as 50% of reinfections are a different bacterial strain of *C. difficile*.

Age: Although the infection is difficult to manage in all ages, older adult patients can be particularly challenging to treat. Individuals 65 years and older have an increased risk for CDAD along with severe disease manifestations (Keller & Surawicz, 2014; Louie et al., 2013; Surawicz et al., 2013). Advancing age correlates with difficulty in successful treatment, while recurrence risk also increases with advancing age. The concern for decreased immunity in the older adult population is felt to contribute to complicated disease course (Louie et al., 2013).

Gender: Females have been shown to have higher incidence rate of CDAD than males (Lessa et al., 2015).

Ethnicity: CDAD occurs in all ethnic groups, but Caucasians have been found to have higher incidence rates than other ethnicities (Lessa et al., 2015).

Contributing Factors: The main contributing factor to the development of CDAD is antibiotic exposure. All antibiotics, including antibiotics used in treatment for CDAD, have the potential to stimulate the development of CDAD. Common offending antibiotics include fluoroquinolones, broad-spectrum penicillin agents, clindamycin, and second- and third-generation cephalopsorins (Clark & Wiselka, 2008; Hensgens, Goorhuis, Dekkers, & Kujper, 2012; Hessen, 2010; Keller & Surawicz, 2014). Further characterization of antibiotic offenders (Clark & Wiselka, 2008) may be classified by tier, with clindamycin, second- and third-generation cephalosporins, pencillins, and quinolones as high risk; macrolides, tetracyclines, and aminoglycosides noted to be medium risk; and metronidazole and vancomycin rare risk for CDAD. Fluoroquionolones are associated with a strain identified in the older population known to have difficulty with treatment response and increased recurrence (Keller & Surawicz, 2014; Surawicz et al., 2013).

Advancing age is a known risk factor. Patients with prolonged length of stay in a health-care facility can be exposed to the infection, especially if they share common toilets, have severe underlying disease (malignancies), use PPIs and H_2 blockers, need tube feeding, require ventilatory support, are malnourished, and show poor host immune response (Hessen, 2010; Kim, Lee, & Jeong, 2010; Morrison et al., 2011; Tleyjeh et al., 2013). Patients with prior GI surgeries are at risk. The use of perioperative antibiotic prophylaxis has been known to contribute to CDAD (Carignan et al., 2008; Cohen et al., 2010). Advanced age, limited mobility and difficulty with activities of daily living, history of depression, heart failure, immunocompromised status, and prior history of CDAD are associated with severe disease in the older adult (Rao et al., 2013).

Signs and Symptoms: The incubation period has not been established. Symptoms can appear immediately after beginning antibiotic therapy, or they may not develop until several weeks to months after completion of antibiotic therapy (Trier, 2009). Older adults can present atypically, with lack of fever; alteration in mental status or confusion can be an early sign (Bartlett & Gerding, 2008; Crogan & Evans, 2007).

Mild cases may present with only crampy lower abdominal pain and watery stool; moderate to severe cases present with profuse, watery diarrhea; abdominal pain and tenderness; tenesmus; nausea; anorexia; leukocytosis; hypoalbuminemia; and malaise (Surawicz et al., 2013). Complicated cases are characterized by at least one of the following criteria: intensive care, hypotension, ileus, mental status changes, fever, WBC count greater than 35,000 or leukopenia, serum lactate greater than 2.2 mmol/l, and organ failure (Surawicz et al., 2013). High suspicion for megacolon or perforation should be raised if marked tenderness to palpation; toxin injury to the colon results in lost functionality and decreased/absent peristalsis (Carter, Rood, & Lyras, 2012). Sepsis secondary to bacteremia may occur, with 20% mortality rate (Johnson et al., 2012). Other complications include chronic diarrhea, electrolyte imbalance, hypoalbuminemia with anasarca, and reactive arthritis (Kee, 2012). Endoscopic evaluation via colonoscopy or flexible sigmoidoscopy may be undertaken when diagnosis is uncertain. The presence of adherent white and yellowish plaques that may coalesce, termed pseudomembranous colitis, are highly specific for *C. difficile* infection (Kee, 2012). However, the absence of pseudomembranous colitis does not exclude the presence of *C. difficile.*

Diagnostic Tests: The triad of leukocytosis, abdominal distension, and hypoalbuminemia raises concern for severe CDAD in a patient with profuse, watery diarrhea (Keller & Surawicz, 2014; Surawicz et al., 2013). Suspicion for CDAD should prompt obtaining stool samples as soon as possible, with the nucleic acid amplification test (NAAT) by polymerase chain reaction (PCR) as the best option for confirming CDAD (Surwaricz et al., 2013). This is approved by the U.S. Food and Drug Administration (FDA) and notable for providing quick results. False-negative results of stool specimens have been documented if empirical treatment was commenced prior to obtaining the stool sample; therefore, delay in diagnostic testing could present unclear results (Sunkesula, Kundrapu, Muganda, Sethi, & Donskey, 2013).

The European Society of Clinical Microbiology and Infectious Diseases recently completed a large review of existing literature regarding diagnostic methods, with updated guidelines encouraging the use of a two-step approach to diagnosing the disease (Crobach et al., 2016). Commercial tests evaluated included two enzyme immunoassays (EIAs) and NAATs. The authors concluded that stand-alone tests may not be sufficient for correct diagnosis of *C. difficile* infection. This newer approach recommends starting with NAAT by PCR or glutamate dehydrogenase EIA (GDH EIA), with negative results likely true negatives. Positive results of the NAAT or GDH EIA would prompt an EIA with toxin A/B, with positive indicating likelihood of infection and negative indicating possibility of carrier status (Crobach et al., 2016). Peri-rectal swabs for diagnosis of infection may also be used in the clinical scenario of paralytic ileus. If suspicion remains high for CDAD despite negative testing, providers must use their best clinical judgment and treat appropriately (Crobach et al., 2016).

Initial baseline laboratory studies should include a CBC with differential; complete metabolic panel; thyroid panel; stool samples to include cultures, ova, and parasites; lactic acid; and a CT scan if concerning abdominal examination or subjective symptoms to exclude megacolon or perforation. Endoscopic evaluation via colonoscopy or flexible sigmoidoscopy may be indicated if persistent diarrhea and/or worsening clinical status occurs despite appropriate medical treatment. Diagnostic endoscopic evaluation is also helpful when the diagnosis is unclear, such as negative stool studies but persistent symptoms despite medical therapy.

Differential Diagnosis:

- Acute diarrhea caused by salmonella
- *Shigella*
- *Campylobacter jejuni*
- *E. coli* 0157:H7
- *Cryptosporidium*
- *Giardia*
- *Microsporida*

- *Isospora*
- *Cyclospora*
- Diarrhea associated with hyperthyroidism or diabetic neuropathy
- Irritable bowel syndrome
- Dietary causes, including nonabsorbable carbohydrates, caffeine, lactose intolerance, and gluten sensitivity
- Microscopic and collagenous colitis
- Pancreatic insufficiency
- Antibiotic-induced chemical effects
- Known antibiotic effects of bacterial and fungal overgrowth
- Food poisoning
- Bacterial or viral gastroenteritis
- Inflammatory bowel disease (Yassin, Young-Fadok, Zein, & Pardi, 2001)

Treatment: Contact isolation must be employed for hospitalized individuals who have suspected or known CDAD. Enteric isolation must continue until resolution of diarrhea or discharge (Surawicz et al., 2013). Supportive measures to include oral and IV fluid replacement, low residue diet, and pharmacological therapy such as antiemetics are indicated. The offending antibiotic agent must be discontinued if clinically possible. A coexisting infection (e.g., urinary tract infection, pneumonia) would preclude complete discontinuation of all antibiotic therapy, as these conditions would still need treatment. In this scenario, less-known offending antibiotics such as ampicillin, sulfonamides, erythromycin, tetracycline, and first-generation cephalosporins may be cautiously used (Hessen, 2010). Antidiarrheals should be avoided due to risk of megacolon (Surawicz et al., 2013).

Treatment of choice for non-severe episodes is vancomycin 125 mg orally four times a day for 10 days (McDonald et al., 2018). Fidaxomicin is an alternative antibiotic that may be used for initial non-severe or even severe episodes; however, if vancomycin or fidaxomicin is unavailable, metronidazole may be given 500 mg orally three times a day for 10 days. A fulminant presentation, which would be characterized by shock, ileus, hypotension, or megacolon, would require vancomycin 500 mg orally four times a day and intravenous metronidazole 500 mg three times a day. If an ileus is present or the individual is unable to receive oral medications, vancomycin enemas would be indicated for direct topical therapy (McDonald et al., 2018; Surawicz et al., 2013). Subsequent recurrences should be treated similar to original episode if uncomplicated. Repeated recurrences may require vancomycin in a pulse dosed fashion or tapering. Fecal microbiota transplants (FMTs) are indicated after the third recurrence and show promising results for long-standing resolution.

Metronidazole and vancomycin are best absorbed via the oral route, but complicated clinical courses may require administration via other routes. Vancomycin is not highly effective intravenously, which leads to the need for colonic enemas. Fidaxomicin is also an FDA-approved antibiotic therapy for CDAD, with similar effectiveness to vancomycin; however, cost remains a barrier to obtaining it in some facilities and as an outpatient (Cornely et al., 2012; Crawford, Husgen, & Danziger, 2012; Louie et al, 2013).

Emerging therapies for CDAD include FMT, which utilizes donor stool that is transplanted to the recipient's colon, most commonly via colonoscopy (Yoon & Brandt, 2010; Musgrave, Bookstaver, Sutton, & Miller, 2011). Other routes include nasogastric tube, enemas, or upper endoscopy. Oral formation of *C. difficile* strains in capsules holds promise for the future, with further studies needed (Gerding et al., 2015).

Follow-Up: The first month following treatment has been shown as crucial due to colonic flora disruption, raising concern for recurrence (Kelly, 2012). Recurrence is identified as prior resolution with new onset diarrhea and a positive stool sample. Repeated stool testing during the same episode is not necessary, and a "test of cure" via negative stool studies is not recommended (Crobach et al., 2016, p. S79). Risk factors for relapse following treatment for CDAD include renal insufficiency, antibiotic exposure during active CDAD, severe strains of *C. difficile,* and advanced age (Louie et al., 2013). Individuals with diabetes and/or sepsis are at risk for metronidazole treatment failure (Jung et al., 2010). Therapeutic response should be based on clinical signs and symptoms, not repeat diagnostic testing.

Sequelae: Chronic colonization, paralytic ileus, toxic megacolon and fulminating colitis, chronic diarrhea, electrolyte imbalances, hypoalbuminemia, and reactive arthritis are complications of CDI.

Prevention/Prophylaxis: Infection control in health-care settings and reduction of risk factors are paramount. Contact isolation should be implemented without delay. The use of electronic rectal thermometers should be avoided (Cohen et al., 2010). Dedicated hospital equipment for the individual is necessary to prevent spread of infection. Proper hand washing is imperative. Equipment and rooms must be sanitized with bleach cleaning agents to kill spores, per facility protocol.

Referral: Signs and symptoms of ileus or fulminating colitis, failure to respond to conventional therapy, and persistent diarrhea that is unexplained require referral. Inpatient older adults with signs of acute abdomen need a surgical referral. Gastroenterology consultation is recommended for bloody diarrhea, concern for secretory diarrhea, fecal incontinence, and failure to improve symptomatically with pharmacological therapy.

Education: Avoid unnecessary antibiotics. Universal precautions include utilizing hand washing with soap and water, along with bleach cleaning agents to sanitize all surfaces at home and in hospital settings. Do not use antidiarrheal agents. Contact provider if recurrent diarrhea persists after treatment for CDAD.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Reducing the frequency and duration of antimicrobial therapy can reduce the risk of recurrence of CDI.	A	Cohen et al., 2010
Vancomycin is the drug of choice for initial episodes of uncomplicated cases of CDI.	A	McDonald et al., 2018
Removing the provoking antimicrobial agent may reduce the rate of CDI recurrence.	A	Cohen et al., 2010
Low serum albumin was found to be associated with relapse of *C. difficile* colitis following initial treatment.	B	Nair, Yaday, Corpuz, & Pitchumni, 1998
Diabetes mellitus and sepsis were identified as independent risk factors for metronidazole treatment failure in CDI.	B	Jung et al., 2010
Removal of environmental sources (electronic thermometers with disposal covers) can reduce the spread of CDI.	B	Cohen et al., 2010

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

COLORECTAL CANCER

Signal Symptoms: A change in bowel habits, persistent diarrhea, bleeding from the rectum, blood in the stool, unexplained anemia, bloating, unexplained abdominal pain, increased gas, abdominal distention, vomiting, weight loss, fatigue, persistent constipation or feeling that the bowels do not completely empty, rectal pressure, rectal pain, and/or reduction in diameter of stool (National Cancer Institute, 2016).

Description: Approximately 50% of neoplasms in the colon occur in the sigmoid and descending colon (54%) (Wasif, Etzioni, Maggard, Tomlinson, & Ko, 2011). Approximately 95% of colorectal cancers are adenocarcinomas (World Cancer Research Fund International, 2012). Leiomyosarcomas account for less than 2% (Sarcoma Alliance, 2006), lymphomas around 0.5% (ACS, 2007), and melanomas are rare, with incidence of less than 2% (Kayhan & Turan, 2003). Neuroendocrine tumors with subsets, including aggressive and indolent, are less than 2% (Bernick et al., 2004).

Etiology: Colorectal cancer results from complex interactions between inherited susceptibility and environmental factors, but the exact nature and contribution of these factors continues to be researched (National Cancer Institute, 2016). Nutritional factors play an important role in its development. High-fat, high-carbohydrate diets with highly processed sugars and carbohydrates and high intakes of red meat, processed meats, and alcohol (which may block the absorption of folic acid) and low-fiber diets appear to be factors. Smoking, inactivity, obesity, high insulin levels, and excessive adipose tissue centrally located all appear to influence the risk of colon cancer (Giovannucci, 2003).

Occurrence: Colorectal cancer is the third most common malignant neoplasm worldwide and the second leading cause of cancer deaths in men and women combined in the United States (National Cancer Institute, 2016). Worldwide, 917,000 deaths occur yearly from colon cancer (Jonker et al., 2007). Colon and rectum cancer represent 8.0% of all new cancer cases in the United States. Approximately 90% of all colorectal cancers occur after age 50 (American Cancer Institute, 2012). Colorectal cancer incidence and mortality rates have been declining, largely attributable to the contribution of screening for prevention and early detection (Smith et al., 2015). Cancer rates vary in levels of education (Albano et al., 2007).

Age: The incidence of colorectal cancer increases with age. Ninety percent of the cases of colorectal cancer occur in people over 50 years old, with a peak incidence in those in their seventies (ACS, 2012).

Gender: Men are more likely to be diagnosed with colon cancer than women of the same age (Jemal et al., 2009).

Ethnicity: African Americans have a higher incidence of new colorectal cancer diagnosis and higher mortality rates from colorectal cancer than non-Hispanic Caucasians (SEER 18 2009-2013, Age-Adjusted) with Native Americans and Latino Americans below 2% (Agrawal et al., 2005).

Contributing Factors: Lifestyle, education, and specific genetic disorders (familial adenomatous polyposis and hereditary nonpolyposis colorectal cancer) are associated with high risk of developing colorectal cancer (Lynch et al., 1993) and inflammatory bowel diseases.

Signs and Symptoms: Clinical symptoms include rectal pressure, passing bright red blood, constipation, bloating, diarrhea, weight loss, melenonic stools, nausea, and loss of appetite. Patients presenting in the clinic with microcytic anemia should be evaluated for rectal bleeding and possible colorectal cancer.

Diagnostic Tests: Initial screening tests that primarily detect cancer include fecal occult blood tests (FOBTs) such as Hemoccult SENSA (Beckman Coulter, Inc.), immunochemical-based fecal occult blood tests (FITs), and stool tests for exfoliated DNA (sDNA) (Smith et al, 2015). Flexible sigmoidoscopy and colonoscopies can detect cancer and advanced lesions and are used for a biopsy and diagnosis. Additional work-up can include digital rectal examination to palpate for any identifying masses, CBC with differential, chemistry panel, carcinoembryonic antigen (CEA), C-reactive protein (CRP), CT scan to determine metastases, and a positron emission tomography (PET) scan for staging purposes. A new screening method, CT colonography or virtual colonoscopy, is a procedure that allows imaging the colorectal region by CT radiographs.

Differential Diagnosis:

- Diverticulitis
- Weight loss
- Blood in the stool
- Mass in the colon
- Prostatic carcinoma
- Sarcoma
- Inflammatory bowel disease
- Hemorrhoids
- Benign colonic polyps
- Peptic ulcer disease (PUD)
- Functional bowel disorders (Tamparo, 2016)

Treatment: The extent of treatment for colorectal cancer depends on how invasive the cancer is in the colon and surrounding sites, and the condition of the patient at the time of diagnosis. The most widely used staging system among clinicians is the TNM system maintained by the American Joint Committee on Cancer (AJCC) and the International Union for Cancer Control (UICC). This system can be used for many types of cancer (Edge & Compton, 2010). The staging of colorectal cancer is predicted using the Dukes classification. Patients should be referred to a surgeon for consideration of a radical resection. Presence of metastatic disease does not rule out surgery. The chemotherapeutic agent 5-fluorouracil combined with Avastin is an effective adjuvant therapy after a surgical resection of stage III colorectal cancer, even in older adults.

Follow-Up: After curative surgery, oncology follow-up is essential, which may include CT scan and CEA markers regularly, depending on stage of cancer. Primary care providers will need to closely monitor for symptoms of fatigue, weight loss, and change in bowel habits with annual FOBTs, and colonoscopy within 1 year. Annual physical examinations are encouraged.

Sequelae: Complications of colorectal cancer include obstruction and perforation, and possible colostomy. Post-resection recurrence of colorectal cancer is common. Radiation proctitis with burning and diarrhea may occur. Bowel adhesions and chronic pain may also be a complication. The liver and bone are two sites known for metastasis after colorectal cancer.

Prevention/Prophylaxis: The ACS recommends that beginning at age 50 years all asymptomatic individuals with no risk factors should have an annual FOBT. FOBT at home is recommended over FOBT with a single stool sample collected by the clinician during a digital rectal examination (DRE) or a guaiac-based toilet bowel FOBT test. FITs may be more patient friendly (Smith et al., 2015). Clinicians should educate patients that stool tests require annual commitment. The gold standard for screening is delineated in Table 10-1 (ACS, 2012). Prolonged prophylactic use of aspirin, calcium,

TABLE 10-1 American Cancer Society Guidelines on Screening and Surveillance for the Early Detection of Colorectal Adenomas and Cancer—Women and Men at Increased Risk or at High Risk

RISK CATEGORY	AGE TO BEGIN	RECOMMENDATION	COMMENT
Increased Risk			
People with a single small (<1 cm) tubular adenoma	5–10 years after the baseline colonoscopy, assuming that all visible polyps were completely removed	Colonoscopy	If the examination is normal, the patient can thereafter be screened as per average risk guidelines.
People with a large (1 cm+) adenoma, multiple adenomas (greater than 3), or adenomas with high-grade dysplasia or villous change	Within 3 years after the baseline colonoscopy assuming that all visible polyps were completely removed	Colonoscopy	If normal, repeat examination in 3 years; if normal then, the patient can thereafter be screened as per average risk guidelines.
Personal history of curative-intent resection of colorectal cancer	Within 1 year after cancer resection	Colonoscopy	After the 1-year colonoscopy the interval to the next colonoscopy should be 3 years (i.e., 4 years after surgery) and then 5 years (i.e., 9 years after surgery). Subsequent colonoscopies should occur at 5-year intervals until the benefit of continued surveillance is outweighed by diminishing life expectancy (Kahi et al., 2016).

TABLE 10-1 American Cancer Society Guidelines on Screening and Surveillance for the Early Detection of Colorectal Adenomas and Cancer—Women and Men at Increased Risk or at High Risk—cont'd

RISK CATEGORY	AGE TO BEGIN	RECOMMENDATION	COMMENT
Increased Risk			
Either colorectal cancer or adenomatous polyps, in any first-degree relative before age 60, or in two or more first-degree relatives at any age (if not a hereditary syndrome)	Age 40 or 10 years younger than age at diagnosis of the youngest affected relative (Rex et al., 2009)	Colonoscopy	Every 5 years (Rex et al., 2009). Colorectal cancer in relatives more distant than first-degree does not increase risk substantially above the average risk group.
High Risk			
Family history of familial adenomatous polyposis (FAP)	Puberty	Early surveillance with endoscopy, counseling to consider genetic testing	If the genetic test is positive, colectomy is indicated. These patients are best referred to a center with experience in the management of FAP.
Family history of Lynch syndrome (hereditary nonpolyposis colon cancer [HNPCC])	Age 2 (?)	Colonoscopy and counseling to consider genetic testing	If the patient has not had genetic testing, colonoscopy every 1–2 years starting by age 25 years or 5–10 years before the age of earliest colorectal cancer diagnosed in the family, whichever is younger. At age 40 begin annual colonoscopy. If the patient has a positive gene test, yearly colonoscopy is recommended (Johns Hopkins Medicine, 2016). These patients are best referred to a center with experience in the management of HNPCC.
Inflammatory bowel disease, chronic ulcerative colitis, Crohn's disease	Cancer risk begins to be significant 8 years after the onset of pancolitis or 12–15 years after the onset of left-sided colitis	Colonoscopy with biopsies for dysplasia	Every 1–2 years. These patients are best referred to a center with experience in the surveillance and management of inflammatory bowel disease.

and vitamin D supplementation may reduce the risk of colon cancer, as might weight loss and a diet high in fruits and vegetables.

Referral: A gastroenterologist should be consulted for colonoscopy and a biopsy of the lesion should be obtained to determine the kind of cancer. A surgical consultation with a cancer surgeon, usually found at a major medical center, is advised. A team including a radiologist, oncologist, cancer surgeon, nutritionist, pharmacist, and nurse will develop a plan of care individualized to the patient and type of cancer. The team, along with valuable input from the patient and family, will then decide the treatment. Hospice may be a necessary choice and is available in all communities.

Education: Teach older adults about the importance of routine surveillance for rectal bleeding and to report any change in bowel habits.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Prevention of colorectal cancer by screening is cost effective, and if caught early, colorectal cancer is curable. Recommend annual FOBT; screening colonoscopy at age 50 based on meta-analysis of research studies involving colonoscopy and annual screenings; 75% of colorectal tumors do not appear to be due to inherited genetic mutations. Some experts suggest screening African Americans beginning at age 45 (Rex et al., 2009).	A	ACS, 2012

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
An adenoma is a polyp that resembles the normal lining of the colon but differs microscopically. They can occur without symptoms and may be precursors to adenocarcinoma of the colon. Fewer than 10% of all adenomas become cancerous, however, more than 95% of colorectal cancers develop from adenomas. Many can be detected radiographically. Patients have many choices in deciding kinds and costs of screening tools; shared decisions should be made after appropriate counseling.	B	Buetow & Buck, 2006 Association of Directors of Anatomic and Surgical Pathology, 2014
Rare cancer (<2%). Routine screening recommended unless diagnosed, then annually.	A	Sarcoma Alliance, 2006
Colorectal cancer (advanced neoplasia) is significantly higher in men than in women. This could warrant refinement of the screening recommendations for colorectal cancer.	A	Regula et al., 2006
Diet and body weight with hyperinsulinemia are important risk factors for colorectal cancer. The majority of colon cancers may be prevented by consuming foods that are higher in dietary fiber, such as fruits and vegetables; keeping BMI within normal ranges and avoiding smoking have been proven to reduce the incidence of colorectal cancer.	A	Giovannucci, 2003
The most modifiable determinants of cancer risks are weight control, dietary choices, and levels of activity. Recommendations are to maintain a healthy weight, get regular physical activity, limit processed meat and red meat, and avoid alcohol and tobacco. Collaborative work in communities to promote healthy worksites, schools, and marketing of foods and beverages that have good nutritional value, particularly to youth, is advised.	A	American Cancer Institute, 2012
Appropriate staging of colorectal cancer improves outcomes and allows for appropriate follow-up. Established guidelines are correlated to the staging of colorectal cancer and provide practitioners a better prognostic picture.	A	
Colorectal cancer mortality rate is declining, but incidence rates are increasing. Obesity, diabetes, and diets high in processed foods all seem to correlate to higher rates. Providing patients with facts on colorectal cancer risks and preventive measures may reduce the incidence.	A	Jonker et al., 2007
The USPSTF recommends screening for colorectal cancer using fecal occult blood testing, sigmoidoscopy, or colonoscopy beginning at age 50. Diagnosing colorectal cancer early has improved mortality rates.		USPSTF, 2015

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DIVERTICULITIS

Signal Symptoms: Impaired cognitive status, lower quadrant pain.

Description: Diverticulitis is an inflammatory condition that involves perforation of one or more colonic diverticula, which are herniations of the mucosa through the muscularis of the colon. It usually occurs in the sigmoid or descending colon. Inflammation of the diverticulum begins at the apex when the narrow opening of the lumen is exposed to fecal residue. Mucosal erosion within the diverticulum also can occur, leading to diverticulitis.

Etiology: Diverticula are common in older adults. Age-related changes in the elastic matrix of the colon and the resulting sluggish fecal mass are thought to cause increased intraluminal pressure of the colon. Diverticular disease is rare in societies that consume high-fiber diets. It is thought that a low-fiber diet produces less bulky stool and increased intracolonic pressure. Diverticulitis is thought to develop when one or more diverticula are perforated (macroperforation or microperforation). A pseudodiverticulum occurs when only the mucosa and submucosa of the colon are affected (Sheth, Longo, & Floch, 2008). Approximately 85% of all cases of diverticulitis involve the left colon and sigmoid. Because this perforation is usually a localized process, free intraperitoneal air or diffuse peritoneal signs are usually not evident; their presence would indicate a more severe case and the possible need for surgical consultation.

Occurrence: An estimated 70% of older adults develop diverticulosis by age 85 years, with the prevalence rising to 80% for adults in their nineties. Approximately 10% to 25% of the population with diverticulosis goes on to develop symptoms of diverticulitis, which is usually most severe in older adults (Hall, 2014).

Age: Diverticulitis is found most commonly in older adults, most commonly in the sixties to eighties.

Gender: Diverticulitis occurs more commonly in women than men (Commane, Arasardnam, Mills, Mathers, & Bradburn, 2009).

Ethnicity: Diverticulitis is found almost exclusively in Westernized countries or populations that have begun to consume a refined Western diet. People of Asian descent are more likely to have right-sided diverticulitis (Kvansnovsky, Papagrigoridia, & Bjarnason, 2014).

Contributing Factors: While there are patients who may not have been diagnosed with diverticulosis prior to an acute case of diverticulitis, it is important for the nurse practitioner to recognize known risk for developing diverticulosis. Obesity, as well as sedentary lifestyle, has been found to contribute to diverticular disease. One study, however, found a relationship between the development of diverticulosis in patients who had seven bowel movements a week as compared to patients who had less than seven; suggesting, too, that a diet high in fiber was not protective of the formation of diverticulosis (Perry & Sandler, 2013). Patients who regularly consumed processed and red meat were found to have a high prevalence of diverticular disease; whereas regular inclusion of fruits and cereal in the diet has been shown to reduce the risk for developing diverticulosis. In one Danish study, there were high indications of developing diverticular disease among siblings, however, there may be more of a connection because of shared environmental risks (Strate, Erichsen & Barron, 2013).

Advancing age is the primary nonmodifiable risk factor for developing diverticulitis (Commane et al., 2009). Chronic constipation and the need to strain to defecate contribute to diverticulitis. These two conditions lead to colonic wall weakness and raise the intraluminal pressure (Jacobs, 2007). Long-term use of NSAIDs, aspirin, antiplatelets, and immunosuppressant drugs increases the incidence of diverticulitis (Nagate, Niikurs, & Aoki, 2014). The development of diverticulitis also has been associated with chronic cigarette smoking and adult polycystic kidney disease. There is a relationship between physical inactivity and the development of diverticulitis (Mulligan, 2015). A recent study also indicated a connection between increased body mass index (BMI) in men (over 30 years) and diverticulitis (Strate, Liu, Syngal, Aldoori, & Giovannucci, 2008). Similarly, patients with increased waist circumference have a tendency to develop diverticular disease (Strate et al., 2008).

Signs and Symptoms: Clinical presentation of diverticulitis in an older adult may be suppressed despite the presence of severe disease. Mental confusion may be the first overt indication of diverticulitis in older adults with known diverticular disease. Patients also may report recent anorexia. They may report a low-grade fever. History of left lower quadrant pain aggravated by movement and fever may be present. They may report that the pain is precipitated by eating and describe a colicky pain (Salzman & Lillie, 2005). Pain also may be reported in the flank, back, or right side of the abdomen. Patients may describe the pain as dull, aching, and intermittent. A sensation of "bloating" may be the initial complaint offered. Associated complaints of nausea and vomiting suggest obstruction (Wilkins, Embry, & George, 2013). Obstipation, constipation, or diarrhea; abdominal cramping without the abdominal pain; and fever also may occur (McQuaid, 2010). Patients may experience abdominal or perirectal fullness. If the bowel is inflamed adjacent to the bladder, the presentation may mimic a urinary tract infection, especially if the patient reports dysuria. Joint pain may be reported in the hip, knee, and/or thigh (Frattini & Longo, 2006). Acutely ill patients with a moderate-to-severe episode of diverticulitis may present with lethargy, reflecting signs of fluid depletion and sepsis.

Bowel sounds may be normal in mild disease; however, bowel sounds become hypoactive until there is an obstruction, when a tinkling sound may be heard. Hyperresonance may reflect intestinal obstruction. Localized tenderness is usually present in the left lower quadrant. Rebound tenderness, involuntary rigidity, and guarding are signs of peritonitis (Wilkins, Embry, & George, 2013). A mass palpated in the left lower quadrant may indicate an abscess. Occult rectal bleeding occurs in about 25% of patients with diverticulitis. Tenderness may be elicited during the rectal examination.

Patients in acute distress may have pyrexia, tachycardia, and impending signs of hypovolemia (Jacobs, 2007).

Diagnostic Tests: A CBC, amylase, lipase, urinalysis, C-reactive protein sedimentation rate, and plain abdominal radiographs can be initially ordered (Mulligan, 2015, Wilkins, Embry, & George, 2013). Leukocytosis may be present. Normal amylase and lipase will help distinguish diverticulitis from other causes of acute abdominal pain. Urinalysis may reveal sterile pyuria due to adjacent colonic irritation; however, if mixed colonic flora are present, consider the presence of a colovesical fistula (McQuaid, 2010). Abdominal x-rays may reveal perforation (free air) and bowel obstruction. Patients should be scheduled for a CT scan of the abdomen and pelvis. CT scans performed with oral, IV, and rectal contrast can enhance the accuracy of the diagnostic image (Rafferty, Shellito, Hyman, & Buie, 2006). Sigmoidoscopy, colonoscopy, and barium enema are usually avoided during acute diverticulitis because these tests may cause further perforation or leakage of bowel contents (Jacobs, 2007). These tests may be performed several weeks after the resolution of the acute episode. Gram stain and testing for *C. difficile* should be considered in patients with diarrhea (Strate et al., 2008).

Differential Diagnosis:

- Acute appendicitis—suspect if right lower quadrant symptoms or nonresolution with medical therapy
- Inflammatory bowel disease (Crohn's disease)
- Complicated PUD—suspect if pneumoperitoneum or peritonitis
- Ischemic colitis—suspect if high-risk patient, bloody diarrhea, or thumb printing
- Pseudomembranous colitis—suspect if antibiotic use or diarrhea
- Vascular ectasia—consider leaking abdominal aortic aneurysm
- Carcinoma of the colon—suspect if weight loss or bleeding
- Urological disorders, such as infection or ureteric colic
- Gynecological carcinomas or abscesses
- Pelvic inflammatory disease
- Infectious colitis
- Small bowel obstruction
- Endometriosis
- Ovarian cyst
- Testicular torsion
- Gastroenteritis
- Bowel obstruction
- Irritable bowel syndrome
- Kidney stone (Wilkins, Embry & George, 2013)

Treatment: Diverticulitis is found to be polymicrobial and results from bacteria normally found in the GI tract (Spirit, 2010). The selection of antibiotics for the treatment of diverticulitis needs to consider the most common bacteria found in the colon, which are gram-negative aerobic rods and anaerobic microorganisms (Rafferty et al., 2006). For mild cases, use broad-spectrum antibiotics such as ciprofloxacin 250 to 500 mg every 12 hours or levofloxacin 500 mg or moxifloxacin 400 mg once a day, plus metronidazole 250 to 500 mg three times a day for 7 to 10 days. An alternative choice is amoxicillin and clavulanate potassium 875 mg twice a day for 7 to 10 days. It is important to follow up with the patient within 48 to 72 hours and inquire about pain, ability to tolerate clear liquid fluids, and if the patient has become febrile (McQuaid, 2010), because improvement should occur if a medication regimen such as this is followed (World Gastroenterology Organization, 2007). Before recommending outpatient treatment, ensure that there is a reliable support system available. Patients treated at home should be able to tolerate oral fluids and lack peritoneal signs (Lutwak & Dill, 2013). The diet can be advanced if there has been improvement in symptoms during this time (Alonso et al., 2010).

Hospitalization should be considered for older adults with diverticulitis, owing to the uncertainty of the severity of the disease because of possibly subdued presentation and comorbid diseases (Lutwak & Dill, 2013). Acute treatment for hospitalized patients consists of bedrest; restriction of any fluids by mouth; nasogastric suction if nausea, vomiting, or other indication of obstruction is present; and IV fluids and electrolytes. It is recommended to try a single-agent parenteral antibiotic for adequate coverage of bowel flora for the treatment of severe diverticulitis. Selection of antibiotics used in the treatment of acute diverticulitis include:

- Ciprofloxacin 250 to 500 mg IV every 12 hours plus metronidazole 500 mg IV every 6 hours
- Ceftizoxime 2 g IV every 12 hours
- Cefoxitin 2 g IV every 8 hours plus metronidazole 500 mg IV every 6 hours
- Moxifloxacin 400 mg IV every 24 hours
- Ampicillin-sulbactam 3 g every 6 hours
- Piperacillin-tazobactam 3.375 g every 6 hours or 4.5 g every 8 hours
- Ticarcillin-clavulanate 3.1 g every 4 hours

Pay special attention to the patient's renal function and creatinine clearance level. Aminoglycosides should only be considered in patients with normal renal function (Rafferty et al., 2006). Antibiotics may need to be adjusted following culture from any drainage or aspiration.

Follow-Up: Older adults with mild cases treated at home with prescribed therapy should expect improvement by the third day. If, however, patients are not able to tolerate fluids and/or fail to respond to narcotic analgesics, refer for hospitalization. Hospitalized patients require daily monitoring for persistent signs and symptoms of diverticulitis, laboratory values, and response to treatment. Patients need to be aware of the high rate of recurrence; approximately 30% of patients with diverticulitis have a recurrence (Mulligan, 2015). Surgical consultation may be necessary if the patient does not respond to treatment and continues to have an elevated WBC count, fever, rebound tenderness, pain, and tachycardia. Approximately 25% of patients with diverticulitis require surgical intervention. Once the acute diverticulitis has resolved, consider referring the patient for further diagnostic imaging studies such as colonoscopy or contrast enema x-ray (Lau et al., 2011; McQuaid, 2010; Rafferty et al., 2006).

Sequelae: Complications include anemia, bowel perforation, peritonitis, pericolonic or intramesenteric abscess, colovesical fistula (most common fistula in patients with diverticulitis), hemorrhage, and bowel obstruction. Of patients with diverticulitis, more than 50% eventually have bowel obstruction (Wilkins, Embry, & George). Complications from

diverticulitis tend to be most severe in patients who are immunocompromised, including those with diabetes, CKD, cirrhosis, or those on immunosuppressant therapies (Hall et al., 2011). Risk of bleeding in diverticular disease increases in patients taking anticoagulants and antiplatelets, aspirin, CCBs, steroids, and NSAIDs (Mulligan, 2015).

Prevention/Prophylaxis: Recognition of early signs and symptoms of diverticulitis helps prevent severe cases (Maconi, Barbara, Bosetti, Cuomo & Annibale, 2011). The use of a symbiotic mixture was found to be beneficial in preventing recurrence of constipation-related abdominal pain in patients with diverticulum disease (Lamiki et al., 2009). Evidence does not support that the inclusion of nuts, seeds, corn, and popcorn in the diet contributes to the development of diverticulitis (Strate et al., 2008).

Referral: Severe episodes of diverticulitis require consultation with a gastroenterologist for hospitalization. Patients with localized abscesses need to be referred for consideration of a CT scan-guided percutaneous drainage (Mulligan, 2015). Repeated episodes may require surgical consultation for an elective sigmoid resection.

Education: Provide information on a high-fiber diet or fiber supplementation or both (Strate et al., 2008; Tarleton & DiBaise, 2011). Teach patients to increase their fluid intake, unless otherwise cautioned, especially when taking fiber supplements. Diverticulitis recurs in approximately one-third of all patients who receive medical management only. Encourage obese older adults to consider weight reduction, as obesity has been found to be a contributing factor to complicated cases of diverticulitis (Wilkins, Embry, & George, 2013).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Despite long-time popular belief, current evidence is lacking to show that including nuts, seeds, corn, and popcorn in one's diet contributes to the development of diverticulitis.	B	Strate, Liu, Syngal, Aldoori, & Giovannucci, 2008
Regular intake of fiber may be beneficial in the prevention and recurrence of diverticular disease.	B	Tarleton & DiBaise, 2011
Patients with known diverticular disease benefited from taking a prescribed symbiotic mixture; recurrence of diverticular disease was prevented in patients with constipation-predominant symptoms.	B	Lamiki et al., 2009
CT scan of the abdomen and pelvis is highly recommended as the imaging study of choice in patients with suspected acute diverticulitis.	A	Ambrosetti, Jenny, Becker, Terrier, & Morel, 2000
Regular use of NSAIDs or aspirin has been associated with an increased risk of developing diverticulitis and diverticular bleeding.	B	Strate, Liu, Huang, Giovannucci, & Chan, 2011

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ESOPHAGITIS

Signal Symptoms: Dysphagia, regurgitation, pyrosis.

Description: Esophagitis is an inflammation of the lining of the esophagus.

Etiology: There are a number of different causes for esophagitis. Patients may develop esophagitis if they ingest medication improperly; have chronic medical conditions; ingest caustic chemicals; are exposed to radiation treatments; have a bacterial, viral, or fungal infection (especially those patients who are immunocompromised); or have a history of GERD (Aparanji, Annavarappu, Russell, & Dharmarajan, 2012). *Candida albicans* is the most common fungal infection (Kliemann, Pasqualotto, Falavigna, Giaretta, & Severo, 2008). The herpes simplex virus type 1 also can cause esophagitis, as can cytomegalovirus. Bacterial esophagitis is rare but can coincide with a fungal or viral infection, making it difficult to diagnose. In GERD, esophagitis is a common complication. Hiatal hernia, an intrinsic factor to the development of reflux esophagitis, increases in incidence with increasing age (Masaoka, Kondo, Yano, Araki, & Nobutomo, 2012).

Eosinophilic esophagitis results from inflammation and is clinically evident by dense esophageal eosinophilia with severe squamous epithelial hyperplasia.

Occurrence: It is estimated that as much as 59% of the general population experiences some symptoms of esophagitis (Cohen et al., 2014). One study found as many as 19% of the population suffers from asymptomatic esophagitis (Lee et al., 2016).

Age: Esophagitis can occur at all ages.

Gender: Esophagitis occurs equally in men and women.

Ethnicity: Not significant.

Contributing Factors: In older adults, normal aging changes such as decreased salivation, gastric motility, and delayed gastric emptying can contribute to the development of esophagitis. Obesity and conditions that contribute to limited upper body mobility, such as spinal cord injuries, may increase the risk of esophagitis. Infectious agents are known to cause esophagitis in patients with certain viral, bacterial, parasitic, and fungal infections. Predisposing factors for patients who develop *Candida* esophagitis include radiation therapy, chemotherapy, certain hematological malignancies, AIDS, alcoholism, and malnutrition. Swallowing certain medications such as aspirin, antibiotics, ferrous sulfate, certain chemotherapeutic agents, NSAIDs, quinidine, steroids, alendronate, alprenolol, vitamin C, phenytoin, calcium preparations, theophylline, and potassium chloride contributes to pill esophagitis due to chemical irritation of the esophageal mucosa. Patients often report that insufficient fluid intake and not remaining upright after the medication is taken contributed to the symptoms of esophagitis (Aparanji et al., 2012).

Substances that can weaken the lower esophageal sphincter (coffee, peppermint, alcohol, spicy foods, citric fruits, chocolate, nifedipine, verapamil, and progesterone) can contribute to esophagitis. Several systemic disorders place patients at risk for esophagitis, including pemphigus vulgaris, bullous pemphigoid, Stevens-Johnson syndrome, lichen planus, inflammatory bowel disease, sarcoidosis, scleroderma, chronic granulomatous disease, and motility disorders of the esophagus. Patients with left atrial enlargement may experience esophagitis due to the pressure of the left atrium exerted on the distal esophagus (Aparanji et al., 2012).

Signs and Symptoms: A history of dysphagia and pain on swallowing is common. Associated pyrosis, regurgitation, coughing, wheezing, and progressive hoarseness may occur. A fever may be present in patients with an infectious process. Patients should be questioned about medication usage, history of radiation treatments, smoking, and intake of substances that weaken the lower esophageal sphincter; sleeping habits; and use of any tight or restrictive clothing. Review patient medical history for chronic conditions that can contribute to esophagitis.

Physical examination usually produces no positive findings for simple esophagitis, but can be used to rule out other etiologies of abdominal pain. Oral thrush may be found in patients with *C. albicans*. Palpate for any upper abdominal masses or tenderness. Perform a rectal examination to detect any frank bleeding and stool guaiac to detect occult blood. Examine the oral cavity for sores and the skin for signs of immunosuppression or systemic diseases like scleroderma (Devuni & Birk, 2017a).

Diagnostic Tests: If suspicion was aroused on the physical examination, stool should be checked using the guaiac test to determine if there has been any intestinal bleeding. Laboratory studies are not required when antacids, position change, or both relieve pyrosis. For older adults who complain of persistent dysphagia or odynophagia with or without fever, a barium swallow or an endoscopy (with brush and biopsy if structural mucosal damage is suspected), or both are ordered. pH monitoring studies may be considered; however, in patients with eosinophilic esophagitis, the results are usually normal and, as such, pH monitoring can be utilized to rule out GERD over eosinophilic esophagitis in differential diagnosis (Akhondi, 2017).

Differential Diagnosis:

- Esophageal stricture (upper GI sinus)
- Esophageal carcinoma
- Cholecystitis and biliary colic
- GI foreign body
- Acute coronary syndrome
- Angina pectoris
- Myocardial infarction
- PUD (Devuni & Birk, 2017b)

Treatment: For infectious esophagitis, temporary symptomatic relief can be obtained with sucralfate slurry, 1 g/10 mL orally four times daily. Viscous lidocaine (2%), 15 mL orally every 4 hours as needed to swish and swallow, can be used for short-term temporary relief, unless contraindicated by potential drug interactions or history of cardiac or hepatic disease. For mild cases of *C. albicans* infection, nystatin oral suspension, 400,000 to 600,000 units four times daily spaced evenly over 24 hours, is prescribed. Ketoconazole should not be given at the same time as antacids or H_2 blockers. For severe cases of herpes simplex virus–induced esophagitis, IV acyclovir may be given, adjusting the dosage for the patient's weight and creatinine clearance. Esophagitis from radiation can be treated with viscous lidocaine. For erosive esophagitis, use of PPIs is recommended for 4 weeks, followed by reevaluation. In moderate to severe cases, PPIs have been found to be consistently superior to other options, including H_2 receptor blockers, in esophageal healing; however, agent selection and dose should be tailored to each patient (Kroch & Madanick, 2017).

Follow-Up: Patients should report progress at least 1 week after treatment. An endoscopy may be repeated if the patient is still symptomatic but compliant after the initial treatment. Yearly endoscopy is recommended thereafter for patients with severe cases of esophagitis. Patients with eosinophilic esophagitis generally do not respond to treatment with PPIs (Akhondi, 2017).

Sequelae: Ulceration and bleeding, if reflux esophagitis is present, can occur after esophagitis. Barrett's esophagus with possible adenocarcinoma may be a long-term complication (Erichsen et al., 2012).

Prevention/Prophylaxis: Because of the high recurrence rate of esophagitis, patients should be instructed to follow all nonpharmacological measures unless otherwise instructed. Maintenance therapy for esophagitis may be prescribed for an extended time, although the dose and need should be periodically evaluated (Freedberg, Kim, & Yang, 2017).

Referral: A gastroenterologist should be consulted for the endoscopy and for patients with severe or nonresponsive esophagitis.

Education: Patients with reflux esophagitis should be instructed to raise the head of the bed 4 to 6 in. with shock blocks. Factors that increase abdominal pressure, such as wearing tight restrictive clothing, should be avoided. The patient should avoid smoking and ingestion of fatty foods, coffee, chocolate, mints, citric juices, alcohol, and large quantities of fluids with meals. Remind patients not to break or crush extended-release or delayed-release tablets. Teach the patient the importance of swallowing medications with an adequate amount of fluids.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Patients with erosive reflux disease are at increased risk for developing adenocarcinoma.	B	Erichsen et al., 2012
The incidence of hiatal hernia was found to increase with age.	B	Masaoka et al., 2012
Hiatal hernia is an independent risk factor for asymptomatic erosive esophagitis	B	Lee et al., 2016
PPIs have been found to be superior to H_2 receptor blockers in esophageal healing, preventing relapse of symptoms at 6 months, and maintaining symptom remission for more than 6 months.	A	Kroch & Madanick, 2017

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GASTRIC CANCER

Signal Symptoms: Weight loss and incessant abdominal pain are the most frequent symptoms at the time of diagnosis, reported by greater than 52% of patients (Mansfield, 2017). Weight loss is usually a consequence of decreased intake, and may be derived from dyspepsia, anorexia, nausea, and/or dysphagia. A palpable abdominal mass is the most common physical finding in gastric cancer and is usually indicative of advanced disease (Mansfield, 2017).

Description: Gastric cancer develops when cells in the stomach begin to grow rapidly and uncontrollably. Eventually, this rapid cell growth will begin to interfere with the normal functions of the stomach. Adenocarcinoma accounts for approximately 90% to 95% of gastric cancers. Adenocarcinoma arises from the stomach mucosa. About 4% of gastric cancers are classified as lymphoma and are usually found in the wall of the stomach. Carcinoid tumors are found in the hormone-generating cells of the stomach and make up 3% of stomach cancers. Carcinoid tumors are unlikely to metastasize to other organs. GI stromal tumors (GISTs) form in the interstitial cells of Cajal in the stomach wall. GIST tumors can be cancerous or noncancerous. Additional types of gastric cancer include small cell carcinoma, leiomyosarcoma, and squamous cell carcinoma, however, these types of cancer very rarely present in the stomach (ACS, 2016). Gastric cancers are then further subdivided by histopathological and anatomical criteria. Recent work has identified that further subgroupings by gene expression exist and suggest a new classification of gastric tumors (Shah et al., 2011).

Early gastric cancers generally are confined to the mucosa or submucosa. Advanced gastric carcinomas penetrate the muscularis propria with lymph node involvement. Gastric cancer usually begins in the distal portion of the stomach and spreads via the lymph, circulatory system, peritoneal seeding, and direct extension (Rowser, 2013).

Etiology: The etiology of gastric cancer is multifactorial. The difference in type and/or location of gastric cancer may also yield different etiologies. Intestinal-type gastric cancer accounts for 70% to 80% of gastric cancers (Cornett & Dea, 2015). It is strongly linked to environmental factors and is thought to progress from chronic gastritis to atrophic gastritis, intestinal metaplasia, dysplasia, and finally, adenocarcinoma (Chan & Wong, 2016). An infection by the bacteria *Helicobacter pylori* allows the microbes to tunnel into the mucosal lining of the stomach and cause inflammation and cellular damage (Jenks, 2015). It is estimated that gastritis caused by *H. pylori* infection is attributable to 60% to 90% of gastric cancers (Cornett & Dea, 2015; Li et al., 2014).

Diffuse-type gastric cancers are most derivable from an acquired or hereditary mutation in the cell adhesion protein E-cadherin. "Familial diffuse gastric cancers account for 1% to 3% of gastric cancers" (Cornett & Dea, 2015, p. 1603). Proximal gastric tumors differ from more distal tumors in

that they are not associated with severe gastritis, and carcinomas in the cardia of the stomach are more strongly associated with environmental and chemical carcinogens than distal carcinomas (Chan & Wong, 2016).

Gastric cancer is additionally associated with the interaction of bacterial, dietary, environmental, lifestyle, and host risk factors. Patients who regularly consume diets containing highly salted and preserved foods (salted, pickled, and/or smoked) and foods high in refined carbohydrates have an increased risk for gastric cancer (Compare et al., 2001). Smoking is also associated with gastric cancers. It has been suggested that diet and lifestyle factors account for one-third to one-half of all gastric cancers (Chan & Wong, 2016).

Occurrence: Each year in the United States, more than 22,220 new cases of gastric carcinoma are diagnosed. Of those, approximately 10,990 will die from the disease (Chang & Wong, 2015). The incidence worldwide is much greater. In Japan and the developing regions of Eastern Asia, Eastern Europe, Chile, Colombia, and Central America the incidence is estimated at 70 per 100,000 people, compared to 6.7 per 100,000 people in the United States (ACS, 2017; Cornett & Dea, 2015). Causing 754,000 deaths a year, gastric cancer is the fourth highest cause of cancer death worldwide (World Health Organization [WHO], 2017).

The incidence of gastric cancer worldwide has diminished greatly over the past few decades (Li et al., 2014). The decline began even before the recognition of certain risk factors, such as *H. pylori* infections. It has been proposed that the worldwide decrease in gastric cancers may be in part be due to the popularization of refrigerator use, which decreased the use of salt-based food preservation techniques and hindered food contamination by bacterial or fungal sources (Chan & Wong, 2015). Refrigeration additionally allowed people to preserve fresh fruits and vegetables, making them more available. Studies have shown that consuming fruits and vegetables is protective against gastric cancer (Chan & Wong, 2016).

Age: The average age at onset of gastric cancer is 69 years. Of patients diagnosed with gastric cancer, approximately 60% are over 65 years of age (ACS, 2017a).

Gender: Gastric cancer is found predominantly in men with a ratio of 3:2 in both developed and developing countries (Chan & Wong, 2015; Jemal et al., 2011Ford). The ACS (2017b) estimates that in the United States in 2017 there will be 17,750 new cases of gastric cancer in men and 10,250 new cases in women. In 2017, it is also estimated that 6,720 men and 4,240 women will die of gastric cancer in the United States (ACS, 2017b).

Ethnicity: Ample differences have been found in the incidence of gastric cancer among people of different ethnic groups, even when living in the same region (Chan & Wong, 2015). Within the United States, the annual incidence of gastric cancers is highest in Asian and Pacific Islanders (11/100,000), followed closely by non-Hispanic African Americans (10.7/100,000), Hispanic persons (10.1/100,000), Native American and Alaska Natives (8.6/100,000), and non-Hispanic Caucasians (5.5/100,000) (ACS, 2017b).

Contributing Factors: As previously mentioned, gastric cancer is associated with bacterial, dietary, environmental, lifestyle, and host risk factors. It is estimated that one-half of the world's population is infected with *H. pylori;* however, individual risk for developing gastric cancer is 2%. The longer the duration of *H. pylori* infection, the higher the risk of developing gastric cancer, as the bacteria can cause significant DNA damage over time. This may contribute to the increased incidence of gastric cancers in developing countries where individuals are more likely to become infected with *H. pylori* at a young age and carry the disease to old age untreated (Jenks, 2015). Chronic *H. pylori* gastritis increases the "relative risk of gastric carcinoma 3.5 to 20 fold" (Cornett & Dea, 2015, p. 1603).

It is unknown why some individuals infected with *H. pylori* develop gastric cancers while some develop ulcers, while still most are symptomatically unaffected. Inherited traits may have some influence over if someone infected with *H. pylori* will go on to develop gastric cancer (ACS, 2016). Numerous studies strongly suggest that a diet high in salt and salt-preserved foods, such as salted fish and cured meat, increases the risk of developing gastric cancer. High dietary salt intake damages the stomach mucosa, making it easier for carcinogens, such as *H. pylori,* to invade. Some studies suggest a synergistic effect between *H. pylori* and salt intake in causing gastric cancer (Chan & Wong, 2016). In 2015, the WHO declared a positive association between eating processed meat and stomach cancer. This is thought to be due to the dietary nitrates contained in "sausages, bacon, ham, beef jerky, corned beef, and other smoked, salted, fermented, or cured meats" (Chan & Wong, 2016, para. 26). Additionally, diets high in fried foods and low in fruits and vegetables are also associated with an increased gastric cancer risk.

There has been some linkage proposed between occupations in coal and tin mining, steel and iron processing, and manufacturing rubber with an increased risk of gastric cancer; however, further research is needed to determine the overall effect of these environmental effects and developing gastric cancer (Raj, Mayberry, & Podas, 2003). Regular ingestion of alcohol has not been consistently associated with an increased gastric cancer risk; a European study advised that daily consumption of wine may even be protective. Cigarette smoking has, however, been linked to 18% of gastric cancers (La Torre et al., 2009) and recent studies have linked water pipe smoking with increased incidence of gastric cancer (Lai, Koriyama, Tokudome, Tran, Tran, & Nandakumar, 2016). Individuals with a low socioeconomic status have a twofold increase in risk of distal gastric cancers, while those with a higher socioeconomic status are at a higher risk of proximal gastric tumors.

Obesity, defined as having a BMI greater than or equal to 25 kg/m^2, was correlated with an increased risk of gastric cancer, and there was a positive correlation with occurrence of gastric cancer and the individual's BMI increasing (Kyrgiou et al., 2017). Other medical conditions associated with an increased risk for gastric cancer include gastric polyps, gastric ulcers, pernicious anemia, and Epstein-Barr virus. History of previous abdominal radiation and having undergone gastric surgery, especially a partial gastrectomy (Billroth I or II), also increases an individual's risk for developing gastric cancer. Aggregation of gastric cancers within families account for 10% of incidences, while gastric cancers which are truly hereditary are culpable for 1% to 3% worldwide. Lastly, individuals with blood type A have a 20% increase in developing gastric cancer when compared to those with blood types O, B, or AB (Chan & Wong, 2016).

Signs and Symptoms: Early detection of gastric cancer is often difficult because of the absence of clinical presentation. Patients often describe a vague sensation of fullness after a meal that is relieved by belching, nonspecific complaints of abdominal pain of varying intensities, nausea, anorexia, dyspepsia, vomiting, and evidence of GI blood (hematemesis and melena), and changes in bowel habits. Epigastric discomfort is present in more than 75% of patients, and the presentation may be similar to that of a gastric ulcer. Weight loss and pallor, if the patient is anemic, may be the only signs noted during physical examination. Examination of the skin may reveal diffuse seborrheic keratosis. A palpable mass in the abdomen is felt in less than 20% of patients.

Many patients with gastric cancer do not become symptomatic until their disease becomes metastatic. Metastatic distribution most commonly involves the liver, peritoneal surface, and distant lymph nodes. Liver involvement is usually diffuse and not singularly palpable, however, it is often associated with an elevated serum phosphatase level. Ascites is often the first sign of peritoneal spread. Because gastric cancers can be spread through the lymphatic system, patients with metastatic disease often have enlargement of the left supraclavicular lymph nodes (Virchow's node), a periumbilical nodule (Sister Mary Joseph's node), and left axillary node (Irish node) (Mansfield, 2017). Additionally, in advanced disease a rigid rectal mass may be palpated (Blumer's shelf) and/or an enlarged ovary may be detected in females (Krukenberg's tumor). The patient's stool may also be found to be guaiac-positive (Cornett, 2015).

Diagnostic Tests: For patients presenting with symptoms of gastric cancer, the following laboratory studies should be considered: CBC with indices to determine presence of anemia, comprehensive chemistry profile, and FOBT to detect bleeding in the intestinal tract. Patients will need to be referred for an endoscopy with a biopsy and cytological examination (Mocellin & Pasquali, 2015).

A chest abdominopelvic CT scan with oral and IV contrast should be used to evaluate for metastasis, especially if the provider is concerned about disease in the liver, adnexa, distal lymph nodes, or ascites. A paracentesis should be performed on patients with ascites and the fluid should be examined for cytology and chemical evaluation. If metastatic disease is found on CT scan, the suspected lesion must be confirmed by biopsy due to the risk of false-positive findings. CT scans can miss peritoneal and hematogenous metastases smaller than 5 mm and do not precisely evaluate the depth of primary tumor invasion and existence of lymph node involvement. In the absence of distant metastatic disease, endoscopic ultrasonography (EUS) is the preferred tool to determine the depth of invasion of primary gastric tumors, as well as to accurately evaluate for nodal staging and occult metastases (Mansfield, 2017; NCCN, 2016). Additionally, epidermal growth factor receptor 2 (HER2) testing should be performed on the tumor biopsy if metastatic adenocarcinoma is documented or strongly suspected (NCCN, 2016). HER2 determination can be used to guide treatment choices in advanced disease.

Differential Diagnosis:

- Chronic/acute gastritis
- Functional dyspepsia
- PUD
- Esophageal stricture
- Esophageal carcinoma
- Reflux esophagitis
- Lymphoma
- Malignant neoplasms of the small intestine
- Crohn's disease
- Sarcoidosis
- Mesenteric ischemia
- Gastroenteritis
- Irritable bowel syndrome
- Gastric ulcers (Cabebe, 2017)

Treatment: A partial or complete gastric resection with adjacent lymph nodes is the treatment of choice for gastric cancer. The NCCN recommends resecting 15 or more regional lymph nodes at the time of tumor resection (Cornett & Dea, 2015). One systematic review found that extended surgery carries increased mortality risks associated with spleen and pancreas resection (McCulloch, Nita, Kazi, & Gama-Rodrigues, 2007). Extensive cancer or metastases negate the need for "curative" surgery, however, surgery is sometimes used as a palliative treatment to relieve pain, bleeding, or a blockage caused by the cancer. Chemotherapeutic agents (most commonly, 5-fluorouracil) have been used alone or in combination with other treatments, such as radiation. Targeted therapies such as monoclonal antibodies (trastuzumab [Herceptin] and ramucirumab [Cyramza]) attack cancer cells without affecting healthy cells, unlike standard chemotherapy in patients whose tumors carry the amplification of the epidermal growth factor receptor 2 (HER2) (Cornett & Dea, 2015). Targeted therapy is used in gastric cancer for patients with stage IV disease or with a recurrence of disease.

Follow-Up: Patients who have undergone a gastric resection should be seen by their provider for continued surveillance to monitor for complications and cancer recurrence or metastatic disease every 3 to 6 months for the first 1 to 2 years after surgery, followed by every 6 to 12 months for the next 3 to 5 years, then annually. Evaluation with a CBC and chemistry profile, along with a CT scan of the chest and abdomen with contrast, should be assessed at each visit. Additionally, patients should be monitored for nutritional deficiencies, including B_{12} and iron insufficiency (NCCN, 2016). Serological cancer markers can be elevated in patients with gastric cancers; however, these tests have low rates of sensitivity and specificity, hindering their use as diagnostic tests, and are rarely used for monitoring unless the patient is undergoing neoadjuvant therapy (Mansfield, 2017).

Sequelae: Malnutrition, hemorrhage, obstruction, possibly evasive cancer, metastases, and recurrence are complications from gastric cancer. Additionally, patients may experience reflux, dumping syndrome, hypoglycemia, and diarrhea following a gastric resection (Jeon, Choi, Lee & Noh, 2016).

Prevention/Prophylaxis: Patients should eliminate the use of food products that contain nitrates and are highly salted. Screening for *H. pylori* in high-risk populations has been advocated (Li et al., 2014). Consumption of fruits, especially citrus fruits, and vegetables has been found to protect against gastric cancer, possibly because of their vitamin C content. Vitamin C is thought to decrease the accumulation of carcinogenic agents in the stomach (Chan & Wong, 2016). Dietary fiber and folate have also been shown to reduce the

risk of gastric cancers in some studies, but further investigation is needed. The use of NSAIDs has been shown to inversely associate with distal gastric cancer risk. This is most evident in patients with a history of *H. pylori* infection (Chan & Wong, 2016).

Referral: A gastroenterologist should be consulted for the endoscopy and complicated management problems, including annual follow-up endoscopy. Patients with stages I to III disease should be seen by a surgeon for possible resection. After receiving a surgical resection, patients may need to be followed by a nutritionist. Additionally, patients with gastric cancer should see a medical oncologist to evaluate for the role of neoadjuvant, adjuvant, or palliative chemotherapy. Given the advanced age and the cognition of patients with gastric cancer, careful attention to emotional support and their quality of life measures need to be considered when caring for older adults (Baldacci, 2015). For patients with advanced disease, referral for palliative care or local hospice should be made (Cornett & Dea, 2015).

Education: After partial and complete gastrectomy, teach patients about the importance of adequate nutrition. Consuming six small meals a day may be necessary instead of the usual three. Supplementation with vitamins, especially vitamin B_{12}, and minerals, such as calcium and iron, should be prescribed. Patients may prefer taking nutritional supplements in place of one or more meals each day.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Epidermal growth factor receptor 2 (HER-2) testing should be performed on the tumor biopsy if metastatic adenocarcinoma is documented or strongly suspected.	A	NCCN, 2016
Treatment of *H. pylori* reduced the incidence of gastric cancer, suggesting the benefits of treatment in preventing gastric cancer.	A	Li et al., 2014
Upper GI endoscopy with biopsy is the recommended initial diagnostic test.	C	Mocellin & Pasquali, 2015
Persons with diets high in salted and preserved foods (salted, pickled, and/or smoked) and in refined carbohydrates have an increased risk for gastric cancer.	C	Compare et al., 2011

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

GASTRITIS

Signal Symptoms: Anorexia, nausea and vomiting, epigastric pain, or discomfort with tenderness.

Description: Gastritis is an inflammation of the mucosal lining of the stomach. A number of conditions can cause gastritis, including infection, autoimmune disorders, exogenous substances, and stress. Patients may be asymptomatic; however, dyspepsia and GI bleeding are common findings in older adults (Zayouna & Piper, 2016).

Etiology: Gastritis represents a group of disorders characterized by inflammation of the stomach lining. Each disorder has distinct clinical attributes, pathogeneses, and histological features. Gastritis is divided first into erosive and nonerosive types; a possible inflammatory process within each group may be categorized as acute or chronic. It is also classified according to the area of the stomach involved (i.e., cardia, body, antrum). Gastritis may be caused by infectious agents such as *H. pylori, E. coli, Staphylococcus aureus, Clostridium perfringens,* and *Streptococcus* and viruses such as cytomegalovirus (CMV), herpes virus, and Epstein-Barr virus. *Candida* may be found in immunocompromised patients, but also in alcoholic patients or those who have ingested corrosive chemicals (Lauwers, Fujita, Nagata, & Shimizu, 2010). Acute erosive gastritis occurs when there is damage to the surface epithelium of the stomach. Erosion results from contact from exogenous agents such as NSAIDs, acetylsalicylic acid, chemotherapeutic agents, and alcohol; infections; or bile secretions (Pilotto, Sancarlo, Addante, Scarcelli, & Franceschi, 2010). In the case of burns, trauma, sepsis, or prolonged hypotension, mucosal hypoxia occurs, leading to acute erosive gastritis (Pilotto et al., 2010).

Chronic gastritis can be classified further as nonatrophic, atrophic, and specific types related to precipitating factors, such as chemical or radiation gastritis. Chronic nonspecific gastritis results from ongoing injury to the gastric mucosa causing chronic inflammation and gastric atrophy (Chen, Ying, Kong, Zhang & Li, 2013). It is estimated that 90% of patients with chronic gastritis have been exposed to *H.*

pylori (Chey & Wong, 2007). There are a number of types of uncommon chronic gastritis, including autoimmune, reactive chemical, noninfectious granulomatous, lymphocytic, eosinophilic, radiation, and ischemic (Franceschi, Di Mario, Leandro, Maggi, & Pilotto, 2009).

Occurrence: Exact incidence of gastritis is unknown.

Age: Although gastritis occurs in all ages, it is a common occurrence in older adults. Although patients of all ages can be infected with *H. pylori*, extensive atrophic damage generally occurs after age 50 years (Chen et al., 2013; Mapel, Roberts, Overhiser, & Mason, 2012). Pernicious anemia can develop as a result of long-standing autoimmune atrophic gastritis, which often occurs around the sixth decade (Orton, 2012).

Gender: Atrophic gastritis occurs equally in men and women; autoimmune gastritis is more prevalent in females (3:1 ratio). In a study by Mapel and colleagues (2012), women were found to have increased incidence of gastritis over men, especially in the fifth decade of life.

Ethnicity: Worldwide, the risk for developing *H. pylori* infection is greatest among Asians and Hispanics. Chronic gastritis resulting from *H. pylori* infection is higher in the United States among African Americans than Caucasians. Autoimmune atrophic gastritis occurs more frequently in individuals of northern European descent (Zayouna & Piper, 2016).

Contributing Factors: Many factors can precipitate the development of gastritis. Acute gastritis can be caused by physiological stressors, hypovolemic shock, portal hypertension, aspirin, NSAIDs, alcohol, radiation, chemotherapy, gastric lymphoma, Crohn's disease, and *H. pylori* and other bacterial, viral, and fungal infections. Stress, trauma, and burns can trigger this acute condition (Wehbi & Dache, 2016). Patients on mechanical ventilation are at risk for developing acute gastritis. Patients with atrophic gastritis from long-term *H. pylori* infection and chronic damage to the intestinal mucosa generally also are found to have pernicious anemia (Zayouna & Piper, 2016). Autoimmune gastritis is related to the loss of parietal and chief cells and includes achlorhydria, hypergastrinemia, loss of pepsin and pepsinogen, and anemia. Chronic gastritis can develop because of gastric atrophy, *H. pylori* infection, bile and pancreatic secretions, and pernicious anemia. Exposure to long-term smoking makes the stomach lining more vulnerable to the development of chronic gastritis. Most patients who have had a previous gastrectomy develop gastritis (Chen et al., 2013).

Signs and Symptoms: Anorexia, nausea (with or without vomiting), and epigastric distress (dyspepsia) often aggravated by eating are common with gastritis (Mapel et al., 2012).

Halitosis may be noted. Patients may report a bloating sensation and early satiety (Huang, Sun, Wang, Tao, Wang, & Tan, 2015). Physical examination may be unremarkable in cases of chronic gastritis. Palpate for abdominal masses and liver tenderness. Perform a rectal examination to test for occult blood. Any unexplained weight loss should be noted. GI bleeding may be exhibited by coffee-ground vomitus, melena, hematochezia, or the passing of bright red blood in a nasogastric tube. Any patient suspected of GI bleeding should be examined for changes in mental status; coolness of the extremities; and pallor of the nail beds, mucous membranes, and conjunctiva. Assessment should include watching for any changes in cardiac output, such as decreasing blood pressure and increased heart rate.

Diagnostic Tests: Diagnostic studies include CBC with indices to detect blood loss and anemia, stool guaiac test, and endoscopic gastroduodenoscopy with a biopsy, which is the definitive diagnostic test for gastritis. If *H. pylori* infection is suspected, noninvasive testing includes antibody testing, urea breath tests (UBTs) ^{13}C and ^{14}C, and fecal antigen tests (Gurney, Carvalho, Gonzalez, Galaviz, & Sonstein, 2014). Though more costly, UBTs and fecal antigen tests have excellent sensitivity and specificity (95%) and can be used for diagnosis and post-therapy evaluation. PPI use will need to be restricted for 14 days and antibiotics for 28 days before UBT and fecal antigen testing (not serology) to ensure more accurate diagnosis and avoid false-negative results (Chey & Wong, 2007; Fashner & Gitu, 2015; Gurney et al., 2014). The serological testing for *H. pylori* is not able to distinguish between prior exposure or active infection; therefore, the utility of this test is limited in clinical scenarios. During endoscopy, a biopsy-based *H. pylori* test can be ordered and remains the gold standard for diagnosis.

Differential Diagnosis:

- GERD
- Biliary tract disease
- Food poisoning
- Functional dyspepsia
- PUD
- Perforated or penetrated ulcer
- Gastric carcinomas
- Pancreatic disease
- Esophageal rupture
- Myocardial colic (McQuaid, 2017)

Treatment: Patients with acute gastritis should avoid offensive agents such as alcohol, NSAIDs, aspirin, and cigarettes (Wehbi & Dache, 2016). Acute hemorrhagic gastritis requires hospitalization for IV fluids, IV PPIs, nasogastric aspiration, transfusion of blood products as necessary, and monitoring of vital signs. H_2 blockers, such as famotidine, ranitidine, or nizatidine, can be given for 6 to 8 weeks. An oral dose of ranitidine 300 mg or nizatidine 300 mg daily at bedtime can be ordered. The dosage of the H_2 blockers may have to be reduced, however, depending on renal status of the patient. If treatment for *H. pylori* is indicated, treatment options are delineated in Table 10-2 (Chey & Wong, 2007; Gurney et al., 2014).

Follow-Up: A repeat endoscopy is advised after 6 weeks for patients who had severe gastritis or who continue to have symptoms despite treatment. Obtain a CBC and check stool for occult blood at subsequent office visits every 3 to 6 months after the diagnosis of gastritis. At each follow-up appointment, review all drugs, including OTC medications and home remedies. (Wehbi & Dache, 2016).

Sequelae: Acute hemorrhagic gastritis has a high mortality rate in older adults. For the patients with *H. pylori*, there is a risk for developing gastric carcinoma; after eradication, the risk decreases (Adamu, Abdullahi, Weck, Gao, & Brenner, 2010; Carrasco & Corvalan, 2013). Patients with autoimmune gastritis may develop pernicious anemia, given the

TABLE 10-2 *Helicobacter Pylori* Regimens

PATIENT CHARACTERISTICS FOR TREATMENT	MEDICATION AND DOSAGE
Patients who are not allergic to penicillin and have not previously received a macrolide	Standard dose PPI twice daily plus clarithromycin 500 mg twice daily, and amoxicillin 1,000 mg twice daily for 10–14 days
Patients who are allergic to penicillin and who have not previously received a macrolide or metronidazole or are unable to tolerate bismuth quadruple therapy	Standard dose PPI twice daily, clarithromycin 500 mg twice daily, metronidazole 500 mg twice daily for 10–14 days
Patients who are allergic to penicillin, failed a course of prior therapy, or have previously received a macrolide	Bismuth subsalicylate 525 mg or subcitrate 420 mg four times daily, metronidazole 250 mg four times daily, tetracycline 500 mg four times daily, standard dose PPI twice daily for 10–14 days

loss of parietal cell mass and subsequently anti-intrinsic factor antibodies (Miceli et al., 2012). It is important to note that unexplained or refractory iron deficiency anemia can develop in patients with either *H. pylori*–associated atrophic gastritis or autoimmune atrophic gastritis.

Prevention/Prophylaxis: Prophylaxis with acid-suppressive drugs can reduce the incidence of acute stress gastritis. Older adults who are high-risk intensive care unit patients, including those with severe burns, central nervous system trauma, coagulopathy, sepsis, shock, multiple trauma, mechanical ventilation for longer than 48 hours, hepatic or renal failure, and a history of peptic ulcer or GI bleeding, benefit from prophylaxis medication to prevent acute gastritis. Critically ill older adults need to be monitored for signs and symptoms of hypovolemic shock secondary to acute gastric bleeding (Wehbi & Dache, 2016). Alternative anti-inflammatory or nonnarcotic analgesics should be considered in the treatment of pain or inflammation in older adults with a history of gastritis, dyspepsia, or other upper GI clinical events (Laine, Curtis, Cryer, Kaur, & Cannon, 2010).

Referral: Older patients with acute hemorrhagic gastritis require hospitalization, probably with intensive care unit admission. Consultation with a gastroenterologist for endoscopy and management of complicated cases of gastritis is recommended.

Education: Advise patients to report any black, tarlike stools or frank bleeding to the health-care practitioner immediately. All alcohol, caffeine, salicylates, tobacco, and NSAID product use should be discontinued.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
A strong association between *H. pylori* and developing chronic atrophic gastritis exists.	B	Weck, Gao, & Brenner, 2009
Vitamin B_{12} deficiency was found in patients diagnosed with autoimmune atrophic gastritis.	B	Miceli et al., 2012
Patients with asymptomatic chronic gastritis predisposed patients taking metformin to experience GI side effects from the medication.	A	Huang et al., 2015

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GASTROENTERITIS

Signal Symptoms: Sudden onset of diarrhea, abdominal pain with distention, flatulence, vomiting.

Description: An infectious response of the GI tract to various microorganisms that can be viral, bacterial, or parasitic in origin.

Etiology: Caused by exposure to toxins and drugs or viruses, bacteria, and parasites:

- Viruses such as norovirus, formerly called the Norwalk-like virus and astrovirus
- Bacteria such as *Salmonella, Campylobacter, Shigella, E. coli,* and *Vibrio cholerae*
- Parasites such as *Giardia lamblia*

Occurrence: Approximately 30% to 40% of gastroenteritis diarrhea in the United States is thought to be viral in origin. Exact incidence of infectious gastroenteritis is unknown because of underreporting of symptoms. Noroviruses cause approximately 23 million cases of acute gastroenteritis each year and are the leading cause of outbreaks of gastroenteritis.

Norovirus outbreaks peak in the winter months. Norovirus accounts for around 19 to 21 million illnesses, 56,000 to 71,000 hospitalizations, and 570 to 800 deaths annually (Wikswo et al., 2015).

Age: Occurs at all ages, but its incidence and the mortality rate from infectious diarrhea are higher in older adults. Epidemic cases of gastroenteritis occur in nursing home populations and long-term care facilities are the most frequently reported settings of outbreaks transmitted through person-to-person contact or environmental contamination (Wikswo et al., 2015).

Gender: Equally prevalent in both sexes, although diarrhea is more common in women.

Ethnicity: Not significant.

Contributing Factors: Specific to the older adult population, age-related factors such as decreased motility, mucosal atrophy, and decreased gastric acidity inhibit natural defense mechanisms against infectious agents. The use of H_2 blockers, PPIs, antacids, anticholinergic drugs, and narcotics increases the potential for developing gastroenteritis (Reuben et al., 2016). Gastroenteritis also can be caused by emotional stress, viral or bacterial infection, food intolerance, and organic (shellfish, certain mushrooms) or inorganic (sodium nitrate) poisons. Travel to another country with a change in surroundings or to an area of poor sanitation standards and facilities can contribute to the development of gastroenteritis. Nursing home populations are susceptible to epidemics because of contact with health-care workers who may not use proper hand washing techniques. Some viruses, such as norovirus, can be transmitted by an airborne route. Fecal-to-oral contact has been identified as a mode of transmission of organisms (Wikso et al., 2015).

Signs and Symptoms: History of possible fecal-to-oral contact; exposure to other patients with gastroenteritis; and ingestion of certain food products such as mayonnaise, custards, fried rice, vegetables, beef, poultry, bean sprouts, or raw seafood should be explored. Try to estimate the length of time that has elapsed since the patient ingested the food product suspected of contamination. Travel to a foreign country or region that may have contaminated water should be recorded, as should any previous history of diarrhea and related symptoms, and the duration of the current episode. A sudden onset of diarrhea, abdominal pain with distention, flatulence, and vomiting may be reported. Associated anorexia, headache, fatigue, dizziness, and myalgias are also symptoms of gastroenteritis. Fever may or may not occur in the older adult. Mental confusion may result from dehydration. The stool's color, odor, amount, and frequency should be described, and the presence of any blood or mucus in the stool should be discerned. Patient use of prescription drugs, OTC medications, and home remedies need to be included in the history. In the physical examination, the skin is checked for signs of rashes or dehydration, and the lymph nodes are assessed for lymphadenopathy. Abdominal examination may reveal distention, hyperactive bowel sounds, and tenderness. Perform a rectal examination to note any bleeding, the color of the stool, and to check for an impaction (Reuben et al., 2016).

Diagnostic Tests: CBC with differential should be ordered. WBC shift to the left may suggest an infection, and a decreased hemoglobin value indicates anemia from probable blood loss. Serum electrolyte evaluation shows an increased sodium level in dehydrated patients and decreased potassium resulting from the diarrhea. Elevated serum creatinine and BUN levels also occur in dehydration. Stool samples for blood, ova and parasite, leukocytes, and bacteria are ordered to try to identify the microorganism in patients with bloody diarrhea, dehydration, or signs of inflammatory disease, and symptoms lasting more than 3 to 7 days. Cultures in immunosuppressed patients are also warranted. Cultures may be obtained in traveler's diarrhea (Barr & Smith, 2014).

Differential Diagnosis:

- Fecal impaction
- Fecal incontinence
- Colorectal cancers
- Adverse reaction to medications
- Diverticulitis
- Malabsorption
- Pseudomembranous colitis if the patient has been prescribed an antibiotic within 2 months of onset of symptoms
- Clostridium difficile (Reuben et al., 2016; Swita, Winter, & Christensen, 2015)

Treatment: Lost fluids and electrolytes must be replaced. Clear liquids and specially formulated rehydration liquids, such as Gatorade, should be given as tolerated. Clear broths and crackers may be added to the diet as tolerated. Early refeeding decreases symptom duration and improves nutritional outcomes (Barr & Smith, 2014). Patients should avoid caffeine, dairy products, alcohol, fruits, bran, vegetables, and red meats. Solid foods should be added gradually, starting with rice or potatoes (Swita, Winter, & Christensen, 2015). Physical activity should be limited, to avoid unnecessary exertion. Care should be taken to prevent any skin excoriation or pressure sores. Antibiotic therapy is specific to the bacterial or parasitic organisms identified from the stool culture:

- *Campylobacter* strains have been shown to have fluoroquinolone resistance and are best treated by azithromycin or other macrolides. For all antibiotics, either single-dose therapy or 3 days of therapy are sufficient for resolution of symptoms (Riddle et al., 2016).
- *G. lamblia* infection is treated with metronidazole, 250 mg three times daily for 5 to 7 days.
- Severe cases of traveler's diarrhea (enterotoxigenic *E. coli*) can be treated with azithromycin 500 mg daily for 3 days (Riddle et al., 2016).

Follow-Up: Contact outpatients 4 days after the onset of symptoms to determine progress. Request nursing home staff to provide a verbal report of the patient's condition on the third or fourth day. Note any further outbreak of gastroenteritis. When contacting the patient at home, question if there have been any additional symptoms, including fever and neurological developments such as paresthesia, motor weakness, and cranial nerve palsies. For older adults with chronic diarrhea or other persistent GI symptoms, refer to a gastroenterologist (Reuben et al., 2016).

Sequelae: Dehydration, anemia, metabolic acidosis, and hypovolemic shock could occur in untreated cases of severe infectious diarrhea.

Prevention/Prophylaxis: Older adults with travel plans to foreign destinations should be counseled to avoid contaminated water and other high-risk beverages and foods to prevent traveler's diarrhea (Riddle et al., 2016).

Referral: Refer to a specialist when symptoms persist beyond 4 days, when severe dehydration develops, or when the patient has bloody stools.

Education: Gastroenteritis, although generally self-limiting, can be debilitating. Infections with some microorganisms, such as *G. lamblia,* can become chronic and result in lactose intolerance, which is common in older adults. Any new case of diarrhea not resolved in 3 to 4 days requires health-care provider intervention. Use of OTC antiperistaltic agents, such as loperamide, is contraindicated in infectious diarrhea, but may be used for adjunctive therapy in patients receiving antibiotics for traveler's diarrhea (Riddle et al., 2016).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Balanced electrolyte rehydration is favored over other oral options in the older adult with severe diarrhea. Other individuals can keep up with electrolyte replacement by using water, juices, soups, sports drinks, and saltine crackers.	A	Riddle et al., 2016
Oral rehydration is the first, preferred step in treating acute diarrhea.	C	Barr & Smith, 2014
Antibiotics reduce the duration and severity of traveler's diarrhea,	A	Barr & Smith, 2014
Testing for *Clostridium difficile* toxins Aand B should be performed in patients who develop unexplained diarrhea after a hospitalization of 3 days or more.	C	Barr & Smith, 2014
Routine testing for ova and parasites in acute diarrhea is not needed in developed countries unless the patient is in a high-risk group (diarrhea >7 days, immunocompromised, travel, infants in daycare, bloody diarrhea with few fecal leukocytes).	C	Barr & Smith, 2014
Probiotics and prebiotics are not recommended for prevention of traveler's diarrhea.	C	Riddle et al., 2016

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GASTROESOPHAGEAL REFLUX DISEASE

Signal Symptoms: Regurgitation, pyrosis, hoarseness, chronic cough, atypical chest pain.

Description: GERD is a common disorder characterized by various symptoms ranging from mild heartburn to more severe physical complaints caused by the reflux of gastric contents into the esophagus. These symptoms may or may not occur in patients who present with laryngopharyngeal and/or respiratory symptoms. Influencing factors in the development of GERD include a weakened lower esophageal sphincter (LES) pressure, hiatal hernia, abnormal esophageal clearance, slowed esophageal peristalsis, esophageal mucosal resistance, and delayed gastric emptying (Hart, 2013).

Etiology: The presentation of GERD is a complex problem than involves reduction in the tone of the lower esophageal sphincter, episodic relaxation of the lower esophageal sphincter at inappropriate times, decreased secondary peristalsis, and faulty mucosal resistance to caustic fluids.

Occurrence: GERD affects more than 20% of the U.S. population (El-Serag, Sweet, Winchester & Dent, 2014). Additionally, it is estimated that at least 50 million Americans experience heartburn at least twice a week (Kroch & Madanick, 2017).

Age: Approximately 10% to 30% of older adults experience weekly symptoms of heartburn or regurgitation.

Gender: The ratio of men to women with GERD is equal. Severe esophagitis is more common in men than in women (2:1). Barrett's esophagus is more common in men than in women (10:1) (Kubo et al., 2013).

Ethnicity: Severe esophagitis is more common in Caucasians than in non-Caucasians.

Contributing Factors: Intrinsic factors that contribute to the development of GERD include LES incompetence, hiatal hernia, esophageal motility disorders, delayed gastric emptying, and gastric distention. Certain medications can trigger GERD. Nitrates, CCBs, benzodiazepines, tricyclic antidepressants, beta agonists, and anticholinergics lower the LES tone. NSAIDs, aspirin, steroids, iron sulfates, gelatin capsules, antibiotics, potassium chloride tablets, vitamin C, quinidine, and bisphosphonates contribute to esophagitis, local erosions, or ulcers. Additional medications that contribute to GERD due to decreased esophageal motility include theophylline, sedatives, and narcotics. Substances that weaken the LES pressure include caffeine, mints, chocolate, alcohol, spicy foods (including yellow onions and garlic), and citrus fruits. Smoking, alcohol, obesity, and certain dietary products (food substances that contain lactose in some patients lead to gastric distention) may contribute to the development of the symptoms of GERD (Hart, 2013). There is an increased risk in developing nonerosive esophagitis in patients with metabolic syndrome. Patients with nasogastric tubes are known to be at risk for having symptoms of GERD. Patients with certain medical conditions that affect gastric motility such as stroke, diabetes, scleroderma, myasthenia gravis, and Parkinson's disease are at risk for developing GERD (Basheshati, Hejazi, Andrews & Storr, 2014).

Signs and Symptoms: Determine from patients precipitating factors related to the development of GERD, length of time they have been experiencing symptoms, and what regimens and/or lifestyle changes they have tried. Patients should be questioned about medication usage, smoking, and intake of substances that can weaken the LES; sleeping habits; and use of any tight or restrictive clothing. Determine if any activity triggers GERD, such as bending over, exercise, or being recumbent. Review the patient's dietary habits and elicit if there are food products that the patient regularly ingests that aggravate symptoms of GERD.

Question the patient about history of heartburn, pyrosis, belching, halitosis, regurgitation, nausea, atypical chest pain (radiation of pain to back, neck, and/or jaw), hoarseness, recurrent laryngitis, cough, choking, pharyngeal tightness, globus sensation, sense of fullness, persistent clearing of the throat, sore throat, recent dental caries, dysphagia, odynophagia, anorexia, weight loss, vomiting, otitis media, bronchospasms, chronic bronchitis, and asthma (Becker et al., 2014; Richter, 1996). Determine if the patient's sleep is disturbed because of GERD symptoms. Review the medications the patient is taking to determine if any medications reduce the LES pressure, decrease the LES pressure, and/or irritate the esophageal mucosa. Question the patient about any previous history and/or treatment for *H. pylori*. Ask about any family history of esophageal cancer or hiatal hernia (Basheshati et al., 2014).

Physical examination usually produces no positive findings. Conduct a thorough examination of the oral cavity and a respiratory examination. Palpate for any upper abdominal masses or tenderness. Perform a rectal examination to detect any frank bleeding.

Diagnostic Tests: If suspicion was aroused on the physical examination, stool should be checked using the guaiac test to determine if there has been any intestinal bleeding. Laboratory studies are not required when antacids, position change, or both relieve pyrosis. For older adults who complain of persistent dysphagia or odynophagia with or without fever, barium swallow or an endoscopy (with brush and biopsy if structural mucosal damage is suspected) or both are ordered. Any patient presenting with laryngopharyngeal symptoms of GERD should be considered for a laryngoscopy (Hart, 2013). A majority of GERD patients have a negative endoscopy.

Differential Diagnosis:

- Nonulcer dyspepsia
- Esophageal spasms
- Esophagitis
- Esophageal stricture (upper GI sinus)
- Esophageal carcinoma
- Cholecystitis
- PUD (Hart, 2013)

Treatment: The goals for the treatment of GERD include reducing symptoms, healing esophagitis, managing esophageal healing, improving quality of life, and preventing complications (Kroch & Madanick, 2017). Important to the management of GERD are the lifestyle changes or nonpharmacological measures that patients need to incorporate along with the medication regimen. These include avoidance of smoking, alcohol, and food products such as chocolate, mints, spicy or acidic foods, and caffeine. For older adults, PPIs remain the mainstay of treatment, given the rapid relief of GERD symptoms and esophageal healing that result from PPI treatment. Once-a-day dosing is beneficial to aid in medication compliance. It is important to note that older adults will require long-term maintenance therapy for GERD. A step-down approach can be initiated in patients with GERD by reducing the dosage of the PPI or by adding an H_2 blocker in the evening. While PPIs can be used long-term in patients who are unable to sustain remission without them, agent and dosage should be tailored to the individual patient with the dosage and need for the drug periodically reviewed in order to use the lowest effective dose (Freedberg et al., 2017; Kroch & Madanick, 2017). The use of prokinetic agents is not recommended in older adults given the serious side effects. Pharmacological recommendations are listed in Table 10-3.

Follow-Up: Review lifestyle changes and the impact modifications have had on the patient's symptoms of GERD (Daniele, Oh, O'Donnell & Clark, 2015). Evaluate compliance with the medication regimen. Determine if the patient has experienced any complications or alarming changes in status since the last evaluation.

Sequelae: Ulceration and bleeding, if reflux esophagitis is present, can occur after esophagitis. In esophagitis with esophageal stricture, Barrett's esophagus with possible adenocarcinoma may be a long-term complication (Bansal & Fitzgerald, 2013). GERD itself is associated with a 10% to 15% risk of Barrett's esophagus (Shaheen, Falk, Iyer, Gerson, & American College of Gastroenterology, 2016). Abdominal obesity, not specifically increased BMI, was found to be associated with the development of Barrett's esophagus in both men and women (Kubo et al., 2013). Older adults are also at risk for developing pulmonary complications of GERD, such as chronic cough, recurrent pneumonitis, and aspiration pneumonia. Patients who present with otolaryngological symptoms of GERD may develop chronic laryngitis, laryngeal polyps, laryngotracheal stenosis, and laryngeal carcinoma

TABLE 10-3 Pharmacological Recommendations for Gastroesophageal Reflux Disease

Treatment of Erosive or Nonerosive Gastroesophageal Reflux Disease

Proton Pump Inhibitors

DRUG	DOSAGE
Dexlansoprazole	30 mg daily
Esomeprazole	40 mg daily
Lansoprazole	15 to 30 mg daily
Omeprazole	20 to 40 mg daily
Pantoprazole	40 mg daily
Rabeprazole	20 mg daily
H_2-Receptor Antagonists	
Nizatidine	150 mg q12h
Famotidine	20 mg q12h
Ranitidine	150 mg q12h
Cimetidine*	400 mg four times daily or 800 mg q12h

*Cimetidine is not recommended for use in older adults.

(Patrick, 2011). Given the reflux of gastric content to the pharynx, dental erosions are also a common consequence of laryngopharyngeal reflux. The risk of chronic sinusitis has also been found in patients with laryngopharngeal reflux (Lin, Chang, Yao & Li, 2015).

Prevention/Prophylaxis: Prevention of GERD should be aimed at eliminating or at least modifying the extrinsic factors that contribute to the development of the condition. Patients should be encouraged to make lifestyle adjustments and avoid potentially harmful medications to prevent further complications.

Referral: A gastroenterologist should be consulted for the endoscopy and for patients with anemia, severe or non-responsive reflux disease, dysphagia, odynophagia, unexplained weight loss, or evidence of hemorrhage. When a patient presents with laryngopharyngeal reflux, he or she should be referred for an indirect or direct laryngoscopy. If a patient presents with dysphagia, odynophagia, unexplained weight loss, or GI bleeding, or if a patient did not respond to initial therapy, refer to a gastroenterologist (Katz, Gerson, & Velva, 2013; Müller, Gockel, Konig, Kuhr, & Eckardt, 2011; Bashashati et al., 2014; Galindo, Vassalle, Marcus & Triadafilopoulos, 2013).

Education: Patients with reflux esophagitis should be instructed to raise the head of the bed 4 to 6 in. with shock blocks or a foam wedge that can be placed at the head of the bed. While there has been limited evidence to suggest that making lifestyle changes to decrease abdominal pressure, such as avoiding tight, restrictive clothing, counteracts the symptoms of GERD, medical practitioners should encourage patients to adopt this strategy, especially given the small number of patients in studies that made this recommendation (Katz et al., 2012). The patient should avoid smoking and ingestion of fatty foods, coffee, chocolate, mints, citric juices, alcohol, and large quantities of fluids with meals. Remind patients not to break or crush extended-release or delayed-release tablets. Patients should avoid eating a meal for at least 3 hours before becoming recumbent. Teach the patient the importance of swallowing medications with an adequate amount of fluids. An oral PPI needs to be ingested 60 minutes before a meal. It is suggested to then wait 30 minutes after the PPI has been taken before eating.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Older adults reported typical symptoms of GERD at postprandial times.	B	Furuta et al., 2012
Despite negative results of pH monitoring indicating no evidence of GERD, patients were found to continue PPI use.	B	Gawron et al., 2012
Screening for Barrett's esophagus can be considered in men with more than 5 years of GERD symptoms and/or frequent GERD symptoms who also have two or more risk factors: age >50, Caucasian, central obesity, smoking history, and history of Barrett's esophagus and/or esophageal adenocarcinoma in a first-degree relative.	B	Shaheen et al., 2016
Patient's with Barrett's esophagus should receive once-daily PPI, unless twice daily therapy is needed to adequately control symptoms.	B	Shaheen et al., 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

HERNIA

Signal Symptoms: A dual dragging sensation with an inguinal hernia; male patients may report pain and swelling in the scrotum; patients with strangulated abdominal hernia usually have a tender mass with associated fever, nausea, and vomiting.

Description: A hernia is the protrusion of tissue through a weakened section in the abdominal wall. Abdominal wall hernias usually occur at the groin (inguinal) and umbilicus. Hernias are classified by their severity as reducible, incarcerated, or strangulated. A reducible hernia moves easily through the anatomical defect. An incarcerated hernia does not return to a normal position automatically or when manipulated externally. A strangulated hernia results when an incarcerated hernia develops edema with ischemia to the entrapped bowel.

In addition to these classifications, inguinal hernias may be either direct or indirect. A direct inguinal hernia passes through the posterior inguinal wall, whereas an indirect or congenital inguinal hernia enters in through the internal abdominal inguinal ring along the spermatic cord through the inguinal passage, to exit out the external inguinal ring.

Etiology: Hernia development has been linked to recurrent Valsalva maneuvers and dysfunctional connective tissue resulting from malnutrition or long-term steroid use. In older women, a history of multiple pregnancies and relaxation of the pelvic musculature, combined with loss of extraperitoneal fat, are considered etiological factors in the development of an obturator hernia (Haraguchi et al., 2007).

Occurrence: In the United States, more than 700,000 herniorrhaphies are performed annually. The most common type of hernia is the inguinal hernia, which accounts for 80% of all cases, followed by the femoral hernia, which is 5% of all hernias. The remaining 15% include umbilical, epigastric, obturator, and those resulting from a surgical incision (Greenfield, 2001).

Age: The incidence of femoral and obturator hernias in women increases with age (Haraguchi et al., 2007). The incidence of male indirect (congenital) hernias decreases with age, but that of direct inguinal hernias increases.

Gender: Indirect inguinal hernias are eight to ten times more common in men than in women, yet the indirect inguinal hernia is the most common type of hernia in women. Older women are three to five times more likely than men to develop a femoral hernia. The obturator hernia is a rare condition that occurs predominantly in women (Haraguchi et al., 2007; Kielar, Duigenan, & McInnes, 2014).

Ethnicity: Not significant.

Contributing Factors: A chronic cough, ascites, abdominal surgery, obesity, and symptomatic prostatism can contribute to development of a hernia because of the associated risk factor of increased intra-abdominal or intrathoracic pressure (Kielar et al., 2014; Lau, Kim, Haigh & Tejirian, 2012). Incisional abdominal hernias can be found in patients with abdominal organ transplants (Smith et al., 2015). Chronic straining for bowel movements, straining to urinate, and lifting heavy objects may be precursors to hernia formation. For women, a weakened pelvic floor after childbirth can contribute to the development of femoral herniation. Older women who are underweight and have a history of chronic illness can develop obturator hernias (Haraguchi et al., 2007). Hernias also may form at the site of a surgical incision or a large scar. Patients with known liver cirrhosis with ascites have a 20% chance of developing an umbilical hernia (Eker et al., 2011). Family history of organ herniation is also recognized as an intrinsic factor contributing to the development of hernias.

Signs and Symptoms: When patients present with signs and symptoms suggestive of a hernia, question them about how long the discomfort (swelling, pain, mass) has been present. Ask if the pain is aggravated by any activity, such as heavy lifting or positioning. Inquire about abdominal surgical history (LeBlanc, LeBlanc, & LeBlanc, 2013). Question if there is a family history of hernias. Patients may report a dual dragging sensation with an inguinal hernia (Kielar et al., 2014). Male patients may report pain and swelling in the scrotum. Hernias may be asymptomatic, only to be discovered as part of a routine physical examination. A reducible hernia presents as a nontender mass that becomes more pronounced after a Valsalva maneuver. An incarcerated hernia, also a nontender soft mass, is found in the abdominal, femoral, or inguinal area and remains even after gentle manipulation. Patients with strangulated abdominal hernia usually have a tender mass with associated fever, nausea, and vomiting. No attempt should be made to reduce a strangulated hernia.

Physical examination reveals decreased flatus, high-pitched or tinkling bowel sounds, abdominal distention, and tenderness of the mass. When examining a patient with a suspected hernia, assess the patient in both the supine and standing position, first observing the suspected mass before change in position. Have the patient also perform the Valsalva maneuver. If possible, attempt to identify the location of the hernia sac and the fascial opening of the hernia when palpating the abdomen (Kielar et al., 2014). Identify the border of the suspected hernia (LeBlanc et al., 2013). Although rare, a patient with a chest wall bulge on the lower chest wall may have an abdominal intercostal hernia (Erdas, Licheri, Calò, & Pomata, 2014). For a suspected inguinal hernia, ask the patient to flex the leg on the side you will examine and then gently palpate the groin area. Then, with fingertips placed over the femoral area, external ring and internal ring should be inserted up into the inguinal canal (LeBlanc et al., 2013).

Gross screening for an inguinal hernia in a male patient is made easier with the patient coughing because "hernia bulges can be felt either against the examining finger (direct hernia) or at the tip of the finger, as it approaches the internal ring (indirect hernia)" (Amerson, 1990, p. 484). A femoral hernia can often be elicited by having a patient cough or strain; the bulge will be felt below the inguinal ligament (LeBlond et al., 2015). A positive Howship-Romberg, which is pain experienced with internal rotation of the hip along with intestinal obstruction, has been found in patients diagnosed with an obturator hernia (Ramser, Messmer, Zbinden, Von

Holzen, & Nebiker, 2014). A mass is detected in the femoral triangle; patients experience pain down the medial aspect of the thigh all the way to the knee as a result of compression of the obturator nerve (LeBlond et al., 2015).

Diagnostic Tests: In an uncomplicated asymptomatic hernia, laboratory and diagnostic tests are unwarranted. If there is questionable strangulation from a prolonged incarcerated hernia, laboratory studies for complications may reveal leukocytosis, elevated serum amylase, and guaiac-positive stool. The use of higher-resolution multidetector CT scan allows for a clearer image of the anatomy studied. Axial CT scan should be requested first if a hernia is suspected (Kielar et al., 2014). Abdominal series are done postoperatively to look for signs of perforation (free air) or obstruction of the bowel (multiple air-fluid levels). Ultrasonography may be ordered for a patient with a suspected recurring hernia or hydrocele (LeBlanc et al., 2013).

Differential Diagnosis:

- Femoral lymphadenopathy
- Femoral artery aneurysm
- Psoas abscess
- Undescended testicle
- Muscle strain (LeBlanc et al., 2013)

Treatment: Surgical repair of strangulated inguinal, umbilical, and femoral hernias is recommended immediately unless the patient is a poor surgical risk. These patients need to be hospitalized, receive IV solutions, and remain on NPO status. Small, direct inguinal hernias and painless, indirect inguinal hernias do not need immediate attention; however, surgery generally is recommended within 1 week of diagnosis. Patients who are not surgical candidates can be fitted for a truss and monitored for signs of prolonged incarceration. Patients with a small, direct, nonpainful hernia should be observed for reduction of the hernia when supine.

Follow-Up: For patients with reducible hernias, observation of the hernia during subsequent physical examination is recommended (Fitzgibbons et al., 2006). Patients suspected of having an incarcerated hernia should be followed up within 1 week to determine if they are experiencing any tenderness of the mass and if a general surgeon has seen them. For the postoperative patient who has had a herniorrhaphy, assess for wound healing and recurrence of the hernia. The mortality rate is increased for patients who have undergone an emergent hernia repair (Altom et al., 2011).

Sequelae: An untreated inguinal, umbilical, or femoral hernia may become incarcerated and strangulated, with subsequent intestinal gangrene. The likelihood of incarceration is greatest for femoral hernias (Kielar et al., 2014). Postsurgical complications of a herniorrhaphy include wound hematomas and superficial wound infections. In one study, 17% of patients who had laparoscopic ventral hernia repair needed subsequent abdominal surgery for reasons such as unplanned bowel obstruction (Patel, Love, Ewing, Warren, Cobb, & Carbonell, 2017). Nerve entrapment is a serious complication of an inguinal herniorraphy. Approximately 50% of hernia recurrence occurs within 5 years, and an additional 20% occurs 15 to 20 years after surgical repair (Sevonius, Gunnarsson, Nordin, Nilsson, & Sandblom, 2011; Slater et al., 2014).

Prevention/Prophylaxis: Cessation of cigarette smoking is recommended to reduce intrathoracic pressure from chronic coughing. Encourage the use of fiber, fluids, and stool softeners for patients who strain with defecation. Patients should avoid lifting heavy objects without proper support. As a preventive measure, encourage weight reduction for obese patients, especially those who have had prior abdominal surgery (Lau et al., 2012).

Referral: Refer the patient to a general surgeon when a hernia is detected. A strangulated hernia requires immediate attention.

Education: Inform the patient with a reducible hernia of the potential complications of an untreated hernia and the importance of reporting immediately any new clinical signs and symptoms, such as increase in pain, swelling, and/or fever. Teach patients to avoid straining to defecate or urinate. Encourage smoking cessation to reduce the probability of a chronic cough. Instruct the patients on proper techniques for lifting heavy objects. Weight reduction should be encouraged for obese patients. Patients also should be advised to discuss with their surgeon postoperative restrictions and plan for rehabilitation and home support given the probable restrictions to activity (Ritcher, 2014).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
In a retrospective review of patients with laparoscopic ventral hernia repair, 17% of the 733 patients required a subsequent abdominal operation.	A	Patel, Love, Ewing, Cobb, Carbonell, A., . . . Carbonell, A. M., 2017
Both increasing BMI and older age were found to directly related to incarceration of noninguinal abdominal wall hernias.	A	Lau, Kim, Haigh, & Tejirian, 2012
A positive Howship-Romberg sign, which is pain elicited with internal rotation of the hip, is common in older patients with an obturator hernia.	C	Ramser, Messmer, Zbinden, Von Holzen, & Nebiker, 2014

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Patients with cirrhosis and ascites benefit from elective umbilical hernia repair.	B	Eker et al., 2011
No significant difference was found in patients with inguinal hernias with mild symptoms who delayed surgical repair and those who did not.	B	Fitzgibbons et al., 2006

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

IRRITABLE BOWEL SYNDROME

Signal Symptoms: Abdominal pain (may be described as colicky, pain relieved by defecation), abdominal distention, mucus in the stool, and the sensation of incomplete evacuation; patients often complain of associated symptoms such as fatigue, flatulence, headache, backache, and dyspepsia.

Description: Irritable bowel syndrome (IBS) is a chronic functional GI disorder characterized by chronic abdominal pain and altered bowel function, in the absence of an attributable organic cause (Longstreth et al., 2006). The subtypes of IBS are diarrhea prominent (IBS-D), constipation prominent (IBS-C), or mixed diarrhea and constipation (IBS-M), and unspecified (IBS-U) (Capili, Anastasi, & Chang, 2016). IBS is generally felt to be caused by alterations in GI motility and visceral hypersensitivity (increased gut sensitivity to stimuli) (Capili et al., 2016; Kanazawa et al., 2016).

Etiology: Although IBS is felt to be due to multiple interacting factors, the exact etiology remains uncertain (Camilleri, 2012). While the focus remains on distortions, intestinal motility, and altered visceral sensation, research has begun to investigate other contributing factors such as inflammation, changes in intestinal flora, food sensitivities, and genetic predispositions (Capili et al., 2016; Jeffery et al., 2012; Lembo, Zaman, Jones, & Talley, 2007; Liebregts et al., 2007). There has also been an association made between increased stress and IBS (Chang, 2011; Kanazawa et al., 2016).

Occurrence: A common presentation for patient referral, IBS accounts for approximately 30% of all referrals to a gastroenterologist (Drossman, Camilleri, Mayer, & Whitehead, 2002). It is estimated that 5% to 15% of the general population suffer from IBS (Capili et al., 2016).

Age: Of cases of IBS, 50% are diagnosed before age 35 years. Because most cases of IBS present by age 50 years, this syndrome is often a chronic condition in older adults. A new diagnosis of IBS in the sixth decade or later is extremely rare (Kurniawan & Kolopaking, 2014).

Gender: Prevalence of IBS is twice as great in women as in men in the United States (Kurniawan & Kolopaking, 2016).

Ethnicity: IBS has a higher prevalence among Caucasians than among other ethnic groups (Minocha, 2008).

Contributing Factors: Female gender and family history of IBS are associated with the development of IBS. Traumatic life events, such as physical or sexual abuse, may trigger the onset of IBS (Koloski, Talley, & Boyce, 2005; Perona et al., 2005). Stressful psychosocial situations may hasten an exacerbation of IBS. IBS is common in patients who also have diagnosed anxiety or depression (Kurniawan & Kolopaking, 2014). Small bowel overgrowth is a common finding in older adults with IBS (Chatterjee, Park, Low, Kong, & Pimentel, 2007; Lupascu et al., 2005). Recent findings have indicated that the 5-HT (serotonin) concentration was higher in patients who had diarrhea predominant IBS than those with constipation dominant IBS (Kurniawan, & Kolopaking, 2014).

Certain food products such as fructose, sorbitol, and lactose, have been known to alter bowel motor function. Review any new prescribed and OTC medications to determine if side effects include constipation or diarrhea. Given the increased prevalence of diverticulosis and intra-abdominal surgeries such as hysterectomy and cholecystectomy, it has been suggested that these conditions may cause or contribute to symptoms that occur in IBS in older adults. It is important to note that common medical conditions such as diabetes, stroke, and Parkinson's disease are known to alter defecation patterns (Minocha, 2008). Symptoms of IBS have been found in about one-fourth of patients who have had a prior GI infection (Kurniawan & Kolopaking, 2014).

Signs and Symptoms: A thorough dietary history is essential to establish a differential diagnosis of IBS, ruling out the possibility of food intolerance. Review recent travel history with the patient. Explore the patient's psychosocial history to determine the relationships between stressful events and exacerbation of IBS. Determine the onset of symptoms, including the time of day when the pain and GI disturbances usually occur. A thorough GI review of systems is important in patients with IBS because GI conditions such as GERD, nonobstructive dysphagia, and functional dyspepsia are often found in patients presenting with IBS symptoms (Kurniawan & Kolopaking, 2014). A report of postprandial abdominal pain suggests biliary tract disease, pancreatitis, or PUD. Patients often report loose and frequent stools or alternating

constipation and diarrhea. Abdominal pain may be described as colicky. Pain often is relieved by defecation. Abdominal distention, mucus in the stool, and the sensation of incomplete evacuation are other presenting symptoms.

Patients often complain of associated symptoms such as fatigue, bloating, flatulence, headache, backache, and dyspepsia. It is common for older adults to present with non-GI-related symptoms such as lethargy, back pain, headache, or urinary frequency (Agrawal, Khan, & Whorwell, 2009). Older adults tend to have greater incidence of rectal urgency with fecal incontinence than younger patients (Minocha, 2008). If patients report fever, unexplained weight loss, dysphagia, or rectal bleeding, these are not symptoms of irritable bowel and should be evaluated closely, especially with a past personal history of any malignancy and a family history of colon cancer.

The physical examination is usually unremarkable (Kurniawan & Kolopaking, 2016). Abdominal tenderness may be elicited, especially in the left lower quadrant, but is not pronounced. Presence of an abdominal mass, lymphadenopathy, hepatosplenomegaly, or ascites in a patient with IBS should prompt further investigation. A routine digital rectal examination generally reveals a tender rectum that is either empty or full of hard, firm feces. It is important to discern if there are any disorders that may contribute to difficult or painful defecation, such as fissures, external hemorrhoids, or rectocele. Detection of a rectal mass on digital rectal examination is concerning and must be further evaluated to rule out malignancy.

Diagnostic Tests: The Rome III criteria for the diagnosis of irritable bowel has been simplified. It is important to discern from the patient the time frame of symptom presentation and duration to help distinguish IBS from other conditions that can mimic this chronic functional disorder of the bowel (see Table 10-4). A new presentation of IBS in older adults is one of the alarm symptoms, so when ordering diagnostic tests in older adults, determine the length of time since the last evaluation and the nature of the current symptoms. There is no biological indicator for IBS. In general, a CBC and erythrocyte sedimentation rate can rule out anemia and inflammation (Furman & Cash, 2011). If diarrhea is the presenting symptom, stool culture and examination for occult blood, ova and parasites, bacteria, and celiac sprue in patients with diarrhea are recommended. A test for *Clostridium difficile* should also be ordered in acute onset of diarrhea, especially in the setting of recent confinement to a health-care facility, recent antibiotic use, or other causes of altered GI flora, such as intestinal infection. For patients with constipation, plain abdomen x-ray series and colonic transit markers may be useful in determining functional causes, as well as stool burden (Minocha, 2008).

Patients with documented involuntary weight loss equal to or more than 5% within a 6-month period, or with signs of obstruction, should have an abdominal CT scan and a small bowel series. Colonoscopy with biopsies may be considered in patients with diarrhea-predominant symptoms to rule out microscopic colitis (Furman & Cash, 2011). Patients with persistent constipation should be evaluated for hypothyroidism, which can contribute to constipation. If the presenting symptoms are bloating and abdominal distention with cramping and diarrhea, a 3-week trial of a lactose-free diet is recommended to rule out lactose intolerance.

TABLE 10-4 Rome III Criteria for the Diagnosis of Irritable Bowel Syndrome

Symptoms present for at least 3 days per month in the past 3 months (with symptom onset at least 6 months previously) with at least two of the following features:

- Improved with defecation
- Onset associated with a change in stool frequency
- Onset associated with a change in stool form (appearance)

Source: Ford et al., 2014.

Differential Diagnosis:

- Food intolerance
- Diverticular disease
- Parasitic diseases
- Biliary tract disease
- Colonic polyps
- Neoplasms
- Pancreatic disease
- Abuse of cathartics
- Latent celiac disease
- Crohn's disease
- Ulcerative colitis
- Ischemic colitis
- Chronic prostatitis
- Thyroid dysfunction
- GI infection (viral or bacterial)
- Lactose malabsorption (Furman & Cash, 2011; Minocha, 2008)

Treatment: The management of IBS depends on the presentation of the patient's condition. In all patients with mild symptoms that do not significantly impair quality of life, lifestyle and dietary changes should be initiated. If symptoms do not respond, or are moderate to severe, pharmacological interventions can supplement diet and lifestyle modification. Treatment should begin with education and reassurance that, while this is a chronic disease, there is no increase in malignancy. Dietary modifications should include avoiding foods that trigger symptoms.

Additionally, improvement of symptoms has been demonstrated with a traditional IBS diet (avoiding high fat, caffeine, gas-producing foods, insoluble fiber, and large meals) (Böhn et al., 2015). Also, a trial of gluten avoidance and lactose avoidance can be instituted to assess improvement. If the patient presents predominantly with constipation, psyllium has been shown to improve symptoms (Ford & Talley, 2012). It should be initially prescribed at one-half to one tablespoon daily, to prevent gas and bloating side effects, and then titrated up until effective. For diabetic patients who choose bulk-forming products, caution should be given regarding using any products that have high sugar content. Patients should be warned that increased bloating might occur initially with fiber products, but usually resolves in 2 to 3 weeks.

Polyethylene glycol (PEG) 3350 is an FDA-approved osmotic laxative used for short-term constipation; however, while it has been shown to improve constipation, it has little effect on abdominal discomfort (Chapman, Stanghellini, Geraint, & Halphen, 2013). PEG should be started at

17 g in 8 ounces of water per day, and titrated up or down (maximum 34 g a day) for effect. Lubiprostone is a chloride channel activator used in the treatment of IBS-C; a dose of 8 to 24 mcg twice a day with food, to prevent nausea, is recommended. Results of clinical trials show efficacy in reducing chronic constipation and abdominal pain with bloating in patients with IBS-C (Lembo et al., 2011). Another agent is Linaclotide, a guanylate cyclase agonist that stimulates intestinal fluid secretion and transit that has been shown to be effective in treatment of IBS-C (Chey et al., 2012; Rao et al., 2012). It is dosed in 290 mcg and 145 mcg once daily on an empty stomach, at least 30 minutes prior to the first meal of the day to avoid diarrhea. Because it is a newer medication and long-term effects are still unknown, it is usually used in IBS-C patients who have failed PEG treatment. The most common side effect is diarrhea, although this tends to resolve after a few weeks of treatment.

Alosetron, a selective 5-HT(3) antagonist, is FDA approved for IBS-D treatment in females only, but significant adverse side effects such as ischemic bowel and toxic megacolon limits this use in routine practice (Bleser, 2011). Historically, this drug resulted in adverse effects, but updated prescribing guidelines by the FDA has improved the safety profile. Providers who choose to prescribe this must be aware of all risks, benefits, and have a dedicated prescribing outline in order to meet safety requirements. Close follow-up is recommended, and prescribing should be done by an experienced provider who is familiar with the full ramifications of this drug.

Foods that may exacerbate IBS-D should be avoided (e.g., caffeinated beverages, histamine-releasing foods like wine and beer, sorbitol-containing candies or gums, citrus fruits for persons with fructose intolerance, and milk products for persons who have known lactose intolerance) (Bohn, Storsrud, Törnblom, Bengtsson, & Simren, 2013).

Antispasmodics are not recommended for treatment of IBS-D in older adults because of the anticholinergic side effects of these medications. If diarrhea is severe, loperamide 2 mg every 4 to 8 hours can be taken, but antidiarrheal medications are not beneficial in treating global IBS-D symptoms. Another treatment often used for IBS-D is bile acid sequestrants, as up to 50% of IBS-D patients have been found to have bile salt malabsorption (Wedlake et al., 2009). Early studies point to the use of rifaximin, an antibiotic, for consideration of the treatment of IBS without constipation and can be considered in patients who have failed other treatments (Menees, Maneerattannaporn, Kim, & Chey, 2012).

A newer agent recently approved for the treatment of IBS-D is a combination mu-opioid receptor agonist and delta-opioid receptor antagonist named Eluxadoline, in doses of 100 mcg and 75 mcg to be given twice a day with food. This has been shown effective in improving both the diarrhea and abdominal pain symptoms associated with IBS-D (Lembo et al., 2016). It should be noted that pancreatitis developed in 0.3% of study patients and any patient on Eluxadoline who experiences pancreatitis-like symptoms should stop the medication and notify their provider. Eluxadoline is also contraindicated in individuals with chronic alcohol abuse, chronic history of pancreatitis, known or concern for biliary duct obstruction, sphincter of Oddi disease, pancreatic structural diseases, known mechanical obstruction, or severe liver impairment (Child-Pugh Class C).

Follow-Up: With chronic IBS patients, a positive relationship between the health-care provider and the patient is mutually beneficial. Dietary intervention and stress-reduction techniques should be reviewed and evaluated. In patients with newly diagnosed IBS, evaluation of persistent symptoms is necessary because IBS is a diagnosis of exclusion. Patients with IBS have been found to have multiple comorbidities that need to be considered. Functional dyspepsia and depression were the most common comorbidities in patients with chronic constipation (Nellesen, Chawla, Oh, Weissman, Lavins & Murray, 2013).

Sequelae: Older adults with a chronic history of IBS may have a concomitant illness, such as diverticulosis (Agrawal et al., 2009; Minocha, 2008). Complications may develop because of other pathological processes mistaken for IBS symptoms. Patients with uncontrollable diarrhea are at risk for dehydration because of fluid and electrolyte loss; this is especially true in older adults. Fecal impaction may result from chronic constipation, especially in an immobile, cognitively impaired older patient.

Prevention/Prophylaxis: The patient should use stress reduction techniques during emotionally stressful situations. Patients should avoid all food products that are known to irritate their bowels (Bohn et al., 2013). Patients with known food intolerance must read nutrition labels and be informed of the inactive contents of medications, which may contain irritating substances.

Referral: Refer patients to a gastroenterologist for colonoscopic imaging. Refer again if patients exhibit persistent abdominal pain or uncontrollable diarrhea despite compliance with treatment and if signs of GI bleeding are present. Patients may benefit from psychotherapy, biofeedback, or hypnosis. Cognitive behavioral treatment is a very common nonpharmacological treatment in patients with IBS (Kurniawan & Kolopaking, 2014).

Education: Inform patients that because the increase of dietary fiber could aggravate their IBS symptoms, they should increase gradually to the recommended dose. It may take 3 to 4 weeks to reach a therapeutic level sufficient to produce results. Eating smaller, frequent meals may ease the IBS symptoms in older adults and enhance overall quality of life (Kurniawan & Kolopaking, 2014). Inform patients with chronic IBS that although the disease itself does not lead to a more serious illness, any change in symptoms should be reported to the health-care practitioner.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Patients with IBS without constipation treated for 2 weeks with rifaximin versus placebo experienced significantly less bloating and other global symptoms of IBS than before the treatment started.	A	Pimentel et al., 2011
A systematic review and meta-analysis of randomized controlled trials (RCTs) that tested the use of rifaximin against placebo in patients with IBS without constipation showed the efficacy of rifaximin in reducing global symptoms of IBS, including bloating.	A	Menees et al., 2012
Older adults diagnosed with IBS presented with noncolonic symptoms such as lethargy, headache, joint pain, and back pain.	B	Agrawal, Khan, & Whorwell, 2009
Women with IBS reported greater psychological stress with the increased severity of abdominal pain/discomfort regardless of the bowel pattern.	B	Heitkemper et al., 2011
Lubiprostone has been well tolerated in patients with chronic constipation.	A	Drossman et al., 2009 Lembo et al., 2011
Eluxadoline reduced symptoms of IBS with diarrhea in men and women, with sustained efficacy over 6 months with 100 mg dosed twice daily.	A	Lembo et al., 2016
Implementation of the traditional IBS diet (avoidance of large meals, high-fat foods, caffeine, insoluble fibers, and gas-producing foods) was associated with improvement in IBS symptoms.	A	Böhn et al., 2015

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LIVER CANCER

Signal Symptoms: Cachexia, jaundice.

Signal Description: The liver is the most common organ in the body for metastasis from other cancers. Common sites of primary tumors that metastasize to the liver are the lungs, colon, pancreas, stomach, breast, and gallbladder. Hepatocellular cancer is associated with cirrhosis of the liver.

Etiology: The hepatic filtration of arterial and portal venous blood is a major reason for the high prevalence of metastases from primary cancerous sites in the body to the liver. Metastases also may result from an extension from an abdominal tumor or through the lymphatic system. Malignant tumors of the liver are primarily adenocarcinomas.

Occurrence: Primary liver cancer, rare in the United States, accounts for about 2% of all cancers or approximately 39,000 cases per year (National Cancer Institute, 2016b). A malignant lesion is 20 times more likely to be from a metastatic source than from a primary lesion.

Age: Mean age of diagnosis is between 50 and 60 years.

Gender: More prevalent in males than in females (ACS, 2016).

Ethnicity: Primary liver cancer is prevalent in people from Africa and Asia because of the widespread occurrence of hepatitis B virus and hepatitis C virus (ACS, 2016).

Contributing Factors: The incidence of liver cancer increases for patients who have a history of cirrhosis of the liver, hepatitis B virus, and hepatitis C virus. Hemochromatosis, porphyria cutanea tarda, alpha-1 antitrypsin deficiency, and Wilson's disease place patients at higher risk of developing liver cancer (NCCN, 2016).

Signs and Symptoms: Complaints of weakness, malaise, weight loss, sweating, and anorexia may be reported. Pain in the upper abdomen can be associated with the cancer and also may be reported. Physical examination reveals cachexia. Auscultation may reveal a bruit over the tumor

and tenderness of the liver. Hepatomegaly and ascites may be present. A mass may be palpable. In more advanced cases, jaundice may appear (NCCN, 2016).

Diagnostic Tests: Laboratory findings may be nonspecific and are related to the underlying liver disease. Findings may include thrombocytopenia, hypoalbuminemia, hyperbilirubinemia, and hypoprothrombinemia. Additionally, patients may have mild anemia and electrolyte disturbances (ACS, 2016). Serum alkaline phosphatase, AST, ALT, and gamma-glutamyl transpeptidase (GGT) are often abnormal in a nonspecific pattern. PT/INR may be elevated.

Patients at high risk for hepatocellular carcinoma (HCC) should be screened using ultrasound every 6 months (Bruix & Sherman, 2011; NCCN, 2016). Identification of a liver nodule that is smaller than 1 cm should be reimaged with an ultrasound in 3 months. Nodules that are larger than 1 cm should be reimaged with contrast-enhanced MRI. Nodules that demonstrate arterial hypervascularity and venous or delayed phase washout are consistent with HCC. Those nodules that do not demonstrate arterial hypervascularity and venous or delayed phase washout should be reimaged with another contrast-enhanced study such as a CT scan or MRI. Lesions that demonstrate arterial hypervascularity and venous or delayed phase washout are diagnostic for HCC. Lesions that do not demonstrate arterial hypervascularity and venous or delayed phase washout should undergo percutaneous biopsy.

An elevated serum α-fetoprotein is not specific to liver cancer (National Cancer Institute, 2016a). A rise in the α-fetoprotein is concerning for the development of HCC in patients with cirrhosis. Elevated serum α-fetoprotein found at levels of more than 500 g/L in patients with cirrhosis is diagnostic for HCC. However, HCC is often found with lower levels of α-fetoprotein when high-risk patients are undergoing routine screening.

Differential Diagnosis:

- Cirrhosis
- Chronic hepatitis B or C infection
- Metastatic malignancy of the liver

Treatment: Advancements in the surgical treatment of hepatic metastatic cancers include tumor ablation and microscopic glass beads containing a radioactive element. Surgical resection of the liver is beneficial only if the patient has a resectable tumor; even so, the survival rate remains low. Liver transplant may be considered in patients with early stage liver cancer, as well as advanced disease. Intrahepatic chemotherapeutic agents, such as 5-fluorouracil and floxuridine, may alter the growth of the tumor, but the prognosis remains the same. Sorafenib can be used for patients with unresectable or metastatic disease. Older patients with known renal function impairment may require a reduced dosage of these agents compared with younger patients, however, often the treatment is palliative at best (NCCN, 2016).

Follow-Up: Patients and family members should be given the opportunity to explore adjunctive methods of pain relief and relaxation. After diagnosis of liver cancer, the focus should be on holistic palliative care, including the intervention of hospice and services provided by the ACS.

Sequelae: The prognosis is poor because the tumor grows rapidly and often metastasizes to the lungs or bones. The survival rate is only 4 to 6 months.

Prevention/Prophylaxis: Hepatitis vaccine is recommended for high-risk patients. Avoidance of chemical exposure, as part of occupational safety, is suggested. Annual ultrasound screening for patients with chronic hepatitis B or cirrhosis should be considered as a preventive measure for liver cancer.

Referral: Refer the patient to an oncologist when the diagnosis of liver cancer is suspected. Local hospice care services should be contacted for care for the patient and family.

Education: Although no further medical treatment is aimed at reversal of the disease, the patient needs to know that you will collaborate with the hospice nurses to provide for the patient's comfort throughout the disease process. The patient can be referred to http://www.liverfoundation.org/support/ for basic information about liver cancer.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Prevention of HCC should focus on preventing infection with hepatitis B virus (HBV) and hepatitis C virus (HCV), treating patients with viral hepatitis who are candidates for treatment, avoiding environmental toxins, and attempting to prevent the development of cirrhosis in patients with liver disease.	A	ACS, 2016 NCCN, 2016
Patients with chronic HBV infection who are at increased risk for HCC should undergo routine surveillance.	A	Bruix & Sherman, 2011 NCCN, 2016
All patients with cirrhosis, regardless of etiology, should undergo surveillance for HCC.	B	Bruix & Sherman, 2011 NCCN, 2016
Surveillance in high-risk patients should be performed with ultrasonography every 6 months rather than annually.	B	Bruix & Sherman, 2011 NCCN, 2016

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NEPHROLITHIASIS

Signal Symptoms: Vague flank pain, hematuria, renal colic, abdominal pain.

Description: In the Western hemisphere, most cases of nephrolithiasis, or kidney stones, are calcium salts, uric acid, cysteine, and struvite. These substances begin to form crystals and become attached to the kidney, eventually developing into stones. Calculi range in size from microscopic to several centimeters in diameter. Nephrolithiasis often attempt to travel through the urinary system and out through the urine. Pain occurs when the calculi become lodged (Curhan, Aronson, & Preminger, 2015).

Etiology: Kidney stones develop from the supersaturation of urine with stone-forming salts that occurs either by overexcretion of salt or underexcretion of urine. Some preformed nuclei form to create a calculus. Abnormal crystal growth inhibitors are formed as well because of hypocitraturia or magnesium deficiency. Approximately 80% of diagnosed kidney stones contain calcium; 10% to 20% are struvite stones, resulting from urinary tract infections; 10% to 15% are uric acid calculi; and 1% contain cystine.

Occurrence: One out of 11 Americans will be diagnosed with a kidney stone in their lifetime. Approximately 50% of patients who have had previous urinary calculi have a recurrence in 5 to 10 years.

Age: Episode onset often occurs between 20 and 60 years of age. The incidence of kidney stones for women peaks around age 55 years.

Gender: Men are more likely than women to form uric acid and calcium stones; struvite stones are more common in women due to the higher incidence of urinary tract infections. Cystine stone formation occurs equally in men and women (Han, Segal, Seifter, & Dwyer, 2015).

Ethnicity: The incidence of kidney stones in Caucasians is three to four times greater than in African Americans; however, the prevalence in African Americans has recently increased 150%. Asian women have the lowest occurrence of kidney stones.

Contributing Factors: A major contributing factor to the development of calculi is decreased fluid intake leading to high concentration of urine. Certain food substances that augment the formation of kidney stones include dairy products, chocolate, green leafy vegetables (calcium oxalate stones) and eggs, fish, poultry, peanuts, and wheat (cystine stones). Certain medications such as triamterene, indinavir, acetazolamide, acyclovir, and sulfa can contribute to the development of nephrolithiasis (Curhan et al., 2015; Tan & Lerma, 2015). A history of hyperparathyroidism, sarcoidosis, Cushing's syndrome, Paget's disease, and immobilization may contribute to the development of calcium phosphate stones. Chronic urinary tract infections may be precursors to struvite stone formation. Family history has also been found to contribute to stone formation, as well as increased oxalate absorption. Bowel disorders that cause chronic acidic urine states, such as patients that have undergone bariatric surgeries, can increase nephrolithiasis frequency. Individuals with metabolic syndrome or those that encompass four to five traits of the disease are more likely to experience kidney stones (Richman, O'Bell, & Pareek, 2014; Tan & Lerma, 2015).

Signs and Symptoms: Patients may have abrupt, severe, colicky pain in either flank or pain that originates in the flank and radiates to the groin. The pain sensation can begin as vague flank pain. Pain spreading downward suggests movement of the stone along the ureter. Associated nausea and vomiting may occur. Hematuria is also common (Curhan et al., 2015; Januchowski, Dabecco, & Verdone, 2014).

Physical examination may reveal local abdominal and costovertebral angle tenderness. Men complaining of groin pain should have a testicular examination to rule out testicular torsion, prostatitis, or epididymitis. Women with pain radiating to the labia should have a pelvic examination to rule out possible ovarian torsion, cysts, pelvic inflammatory disease, or tumors.

Diagnostic Tests: For patients who have not yet passed a stone, urine collection for recovery of stones or gravel should be ordered. A urinalysis (UA) reveals the presence of leukocytes, bacteria, and blood, which can be indicative of a urinary tract infection. The presence of crystals in a UA can aid in the determination of the type of stone present. Although abdominal plain films are inexpensive and quick, they can fail to detect radiolucent stones, small stones, or obstructions (Curhan et al., 2015). It has been found that patients that undergo an ultrasound initially do not need further imaging studies; similar results are found with ultrasound when compared to CT scans with decreased radiation exposure (Smith-Bindman et al., 2014; Tan & Lerma, 2015). The ability to view the entire urinary system is provided by IVP; however, there is a large amount of radiation exposure with IVP, lower sensitivity than CT scans, and multiple films may be required over several hours (Curhan et al. 2015; Tan & Lerma, 2015). A noncontrast helical CT scan is still the gold standard for nephrolithiasis detection. The helical scan is quick and often detects stones and obstructions not seen on other imaging studies (Curhan et al., 2015; Han et al., 2015; Januchowski et al., 2014; Tan & Lerma, 2015).

Differential Diagnosis:

- Papillary necrosis
- Hydronephrosis
- Ileus
- Diverticulitis
- Appendicitis
- Bowel obstruction
- Mesenteric ischemia
- Ovarian cyst
- Testicular torsion
- Constipation
- Arterial aneurysm
- Cholecystitis
- Acute cystitis
- Pyelonephritis
- Ectopic pregnancy

- Prostatitis
- Renal cell carcinoma
- Narcotic seekers

Treatment: Size and location of the stone, coupled with the length of time since the onset of symptoms, directs treatment. Stones less than or equal to 4 mm usually pass on their own; stones 4 to 5 mm have a 50% chance of passing without intervention. A stone of more than 10 mm requires referral to a urologist for possible intervention that may include extracorporeal shock-wave lithotripsy, ureteroscopy, or percutaneous nephrolithiotomy. IV fluids have not been found to aid in stone passage, but are recommended if patients are dehydrated (Curhan et al., 2015; Januchowski et al., 2014). Patients not in distress and without obstruction should drink at least 2 L of fluid daily and strain the urine until a stone has passed (Tan & Lerma, 2015). NSAIDs have been found to be as effective in pain management as narcotics (Han et al., 2015). CCBs and alpha adrenergic blockers, such as Tamsulosin, relax smooth muscle and are often prescribed to aid in stone passage (Curhan et al., 2015; Januchowski et al., 2014).

Follow-Up: A 24-hour urine collection 3 to 6 months after treatment for nephrolithiasis is recommended. At this time, also review with the patient the need for compliance with dietary restrictions and fluid requirements.

Sequelae: Immediate complications of unresolved kidney stones include obstruction, urinary tract infection, and sepsis. A high incidence of recurrence of kidney stone formation exists. Metabolic causes of kidney stones need to be ruled out when dietary measures are unsuccessful. Additional complications of nephrolithiasis include renal failure, ureteral perforation, perinephric abscess, pyelonephritis, and sepsis (Januchowski et al., 2014).

Prevention/Prophylaxis: An adequate daily fluid intake is essential. Patients should be encouraged to increase their fluid intake during heavy exercise or when traveling long distances. To prevent recurrent stones, review the patient's dietary habits to discern if there are any excessive food products that may be factors in kidney stone formation (calcium, purine, protein).

Referral: Refer the patient to a urologist when:

- Obstruction is detected.
- The stone is more than 5 mm.
- The stone has not passed within 24 to 48 hours of the onset of pain.
- The patient has complicated diagnostic reports.
- The patient has severe nausea and vomiting, intractable pain, urosepsis, anuria, or renal failure.

Education: Inform all patients with nephrolithiasis that there is a high probability of a second occurrence of a kidney stone. Advise the patient, if possible, to strain urine with the provided strainer when experiencing symptoms of kidney obstruction. A daily fluid intake of 2 to 3 L is recommended. Explain to patients that fluid intake needs to be increased when urine appears dark yellow. Provide information about any food restrictions that may be deemed necessary to prevent specific stone formation (Curhan et al., 2015; Elmahdy & Persad, 2014; Han et al., 2015; Richman et al., 2014; Shah & Camillo Calle, 2016; Tan & Lerma, 2015).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
A noncontrast helical CT scan is the gold standard for nephrolithiasis detection; however, initial ultrasonography reduces radiation exposure with no significant difference in outcomes.	B	Smith-Bindman et al., 2014
Alpha adrenergic blockers and CCBs aid in stone passage by relaxation of smooth muscle.	B	Curhan et al., 2015
There is an increased risk of stone development in patients with 4 to 5 traits of metabolic syndrome.	B	Richman et al., 2014
NSAIDs and narcotics have been found to be beneficial for pain relief in kidney stone sufferers.	B	Tan & Lerma, 2015

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NONALCOHOLIC FATTY LIVER DISEASE

Signal Symptoms: Fatigue, right upper quadrant pain or fullness, possible hepatomegaly.

Description: Nonalcoholic fatty disease (NAFLD) is the most common liver disease in Western countries (Byrne & Targher, 2015). NAFLD refers to a spectrum of liver disorders that include simple steatosis (fatty liver), and nonalcoholic steatohepatitis (NASH). NASH is characterized by hepatocyte injury, inflammation, and fibrosis, which can ultimately lead to cirrhosis and end-stage liver disease (Byrne & Targher, 2015). Risk factors for the development of NASH include elevated fasting blood glucose, low high-density lipoprotein (HDL), and elevated fasting cholesterol (Wilkins, Tadkoh, Hepburn, & Schade, 2013). Alcohol use must be minimal to meet the criteria for NAFLD (Bayard, Holt, & Buroughs, 2006). The maximum intake of alcohol is two standards drinks a day for men and one for women.

Etiology: Risk factors for the development of NAFLD include obesity, insulin resistance, hypertension, and dyslipidemia (Dyson, Anstee, & McPherson, 2015). Insulin resistance is the most important risk factor for the development of NAFLD. It has been suggested that there may be a genetic predisposition to NAFLD, especially in patients who also develop metabolic syndrome (Croke & Sampson, 2012).

Occurrence: NAFLD is the most common liver disease in Western countries (Byrne & Targher, 2015). It has been predicted that NAFLD will become the most frequent indication for liver transplantation by 2030. In the United States, it is estimated that 95 million adults are affected by NAFLD and the prevalence will continue to rise (Malhotra & Beaton, 2015). It is estimated that up to one-third of the population has evidence of steatosis on imaging (Dyson et al., 2015).

Age: The majority of patients initially diagnosed with NAFLD are in their forties or fifties (Sheth & Chopra, 2016a). The risk of nonalcoholic steatohepatitis, cirrhosis, and hepatocellular carcinoma is increased in the elderly with NAFLD (Bertolotti et al., 2014).

Gender: NAFLD is more common in men than women (Byrne & Targher, 2014). The incidence of NAFLD is approximately 30% to 40% in men and 15% to 20% in women (Bryne & Targher, 2015).

Ethnicity: Hispanics have the highest rate of NAFLD and non-Hispanic African Americans have the lowest incidence of NAFLD (Pan & Fallon, 2014). African Americans have been found to have less steatosis than Caucasians.

Contributing Factors: The most common risk factors associated with the development of NAFLD are obesity, hypertension, dyslipidemia, and insulin resistance (Sheth & Chopra, 2016a). Aging was found to be a risk factor for the development of NAFLD in premenopausal women, but the same could not be said for men (Hamaguchi et al., 2012). NAFLD may be present in patients who have jejunoileal bypass or gastric bypass, or who have experienced starvation and/or protein energy malnutrition. Also at risk are patients on total parental nutrition (Sheth & Chopra, 2016a). Drug-induced NAFLD can be caused by aspirin, cocaine, valproate, highly active antiretroviral, tetracycline, NSAIDs, amiodarone, tamoxifen, methotrexate, and corticosteroids (Patel & Sanyal, 2013). In patients with coexisting conditions, such as ulcerative colitis, Graves' disease, or type 1 diabetes, consider an autoimmune cause for NAFLD (Croke & Sampson, 2012). There is evidence that NAFLD is linked to other chronic disease such as sleep apnea, colorectal cancer, osteoporosis, psoriasis, and endocrine diseases such as polycystic ovary syndrome (Bryne & Targher, 2015).

Signs and Symptoms: Most patients with NAFLD are asymptomatic (Wilkins et al., 2013). Patients may present with fatigue, jaundice, pruritus, right upper quadrant discomfort or fullness, and muscle weakness (Wilkins et al., 2013). Inquire about weight loss. On examination, look for evidence of jaundice of the skin, scleral icterus, and spider angiomas. Patients with more advanced cases may also have anorexia and nausea. Patients may have unexplained abnormal liver blood tests as part of a routine physical or drug monitoring, such as statins. On physical examination, patients with NAFLD may have hepatomegaly (Sheth & Chopra, 2016a). Patients with more advanced illness may present with asterixis and caput medusa radiating from around the umbilicus (Sheth & Chopra, 2016a).

Diagnostic Tests: NAFLD is a diagnosis of exclusion. The most common abnormal laboratory tests are elevated ALT and AST (Sheth & Chopra, 2016a). ALT and AST may be elevated two to five times the upper limits. It is important to exclude other causes of elevated transaminases levels. Hepatitis A, B, and C need to be ruled out, as does autoimmune hepatitis. NAFLD is the most common reason for elevated transaminases (Vernon, Baranova, & Younossi, 2011). Hemochromatosis needs to be ruled out. It may be useful to rule out Wilson's disease, thyroid disease, celiac disease, and alpha-1 antitrypsin deficiency. A fasting glucose, lipid panel, PT, albumin, and total bilirubin should be obtained (Wilkins et al., 2013). An ultrasonography of the liver will assist in the diagnosis of NAFD by identifying fatty infiltration in the liver (Wilkins et al., 2013). An ultrasonography of the liver has a sensitivity of 82% to 89% and a specificity of 93% for identifying a fatty liver. For patients suspected of liver fibrosis, commercially available serologic markers are available to order (Wilkins et al., 2013). A liver biopsy should be considered for patients who are likely to have a more advanced liver disease, including morbid obesity, diabetes, advanced age, and an AST/ALT ratio greater than 1. Patients with persistent elevations in liver enzymes despite lifestyle changes may be considered for liver biopsy. Patients should be screened for alcohol and drug use.

Differential Diagnosis:

- Alcoholic hepatitis
- Auto immune hepatitis
- Hepatitis B
- Hepatitis C
- Thyroid disease
- Alpha 1 antitrypsin disease
- Medication induced hepatitis

- Wilson's disease
- Pregnancy related hepatitis
- Wilson's disease (Wilkins et al., 2013)

Treatment: Management of NAFLD should focus primarily on risk factors such as insulin resistance and hyperlipidemia (Sheth & Chopra, 2016b). Overweight or obese patient with NAFLD should consider a weight loss program. It has been shown that weight loss and exercise reduce liver enzyme levels and steatosis (Greenberger et al., 2009). Weight loss should not exceed 1 to 2 pounds a week. Patients should avoid rapid weight loss. Treatment for hyperlipidemia (atorvastatin, gemfibrozil) has been shown to improve liver enzymes and liver steatosis (Ghamar-Chelreh et al., 2012). Statin drugs should not be withheld, due to mild elevations of liver enzymes. The initial dose of statin drugs should be low and liver enzymes checked in 2 weeks, and then monthly for the first 3 months (Baynard et al., 2006). If there is an increase in transaminases of two times the baseline value, statin therapy should be discontinued.

Medications for the treatment of insulin resistance (pioglitazone, rosiglitazone) have been shown to improve transaminases levels and decrease hepatic steatosis (Sheth & Chopra, 2016b). Morbidly obese patients should be considered for bariatric surgery. Obese patients who are unable to lose weight with lifestyle changes should be considered for bariatric surgery; individual risk for complications from surgery need to first be evaluated (Greenberger et al., 2009). Orlistat reduces the absorption of dietary triglycerides and has been shown to improve liver enzymes and steatosis in obese patients (Sheth & Chopra, 2016b). Hepatitis A and B vaccines should be given to patients without serologic evidence of immunity. Patients with NAFLD should avoid alcohol consumption.

Follow-Up: Patients on statin drugs should be monitored every 6 months for liver function and adherence to overall treatment pan. Patients should be monitored for weight loss. Patients with decompensated liver disease should be referred to a specialist.

Sequelae: NAFLD in the elderly is often more severe and can carry a worse prognosis than in the younger population (Bertolotti et al., 2014). The elderly have increased risk for complication (cardiovascular disease, extrahepatic neoplasms) than in the younger population. Risk factors for the development of hepatocellular carcinoma in NAFLD include the elderly, type 2 diabetes, and cirrhosis (Bertolotti et al., 2014).

Prevention/Prophylaxis: Weight loss should be encouraged in obese patients. Patients should be encouraged to exercise (Bertolotti et al., 2014). Patients with diabetes, hypertension, and hyperlipidemia should be closely monitored and managed to help prevent the progression of NAFLD.

Referral: Consider referral to a specialist when the clinical parameters are indicating a severe disease state. Patients whose liver enzymes remain elevated despite weight loss, those who are morbidly obese, those with AST/ALT twice the upper limits of normal, and those with NASH whose clinical picture suggests the likelihood of cirrhosis should be referred (Sheth & Chopra, 2016b).

Education: Patients should be advised that NAFLD is usually caused by the same conditions that cause an increased risk for cardiovascular disease (Sheth & Chopra, 2016b).

The management of NAFLD is directed at risk factors, specifically diabetes, elevated lipids, and obesity (Sheth & Chopra, 2016b).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Orlistat reduces the absorption of dietary triglycerides and has been shown to improve liver enzymes and steatosis in obese patient.	C	
Treatment for hyperlipidemia has been shown to improve liver enzymes and liver steatosis.	C	Ghamar-Chehreh et al., 2012
Obese patient should lose weight; it is not recommended to lose >1–2 pounds per week. Rapid weight loss can worsen NAFLD, particularly after bariatric surgery.	C	Sheth & Chopra, 2016b
Medications for the treatment resistance (rosiglitazone, pioglitazone) have been shown to improve transaminase levels and decrease hepatic steatosis.	C	Sheth & Chopra, 2016b
Long-term use of statins in patients with NAFLD had a significant reduction in hepatic steatosis compared to patients not taking statins.	B	
Patient should refrain from any alcohol consumption.	C	Sheth & Chopra, 2016b

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Patients with NAFLD should receive hepatitis A and B vaccine if they do not have immunity.	C	Sheth & Chopra, 2016b

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PEPTIC ULCER DISEASE

Signal Symptoms: Gastric ulcers: history of dyspepsia, epigastric pain, or right upper quadrant pain that radiates to the back after the ingestion of a meal. Duodenal ulcers: pain 1 to 3 hours after a meal (patients with gastric ulcers have pain immediately on eating). Nocturnal pain may occur in duodenal ulcers in the older adult; definitive symptoms may be absent, and vomiting and anorexia may be reported instead of epigastric pain.

Description: PUD involves ulcerations of the mucous membrane of the stomach or duodenum (Fashner & Gitu, 2015). The disease is classified according to the nature and anatomical location of the lesion. Gastric ulcers generally are found along the lesser curvature of the stomach between antral and acid-screening mucosa, occurring anywhere in the stomach, from the cardia to the pylorus. Duodenal ulcers develop in the duodenal bulb or in the immediate postbulbar area (Barkun & Leontiadis, 2010).

Etiology: The etiology of PUD is characterized best as an imbalance between the noxious agents to which the GI mucosa is exposed (primarily hydrochloric acid and pepsin) and the protective factors (mucus production, bicarbonate secretion) that the mucosa uses to resist destruction from such noxious agents. An increase in destructive influences (e.g., with NSAID use) can destroy the mucosa, leading to the development of an ulcer (Lockrey & St. Lim, 2011).

Occurrence: Approximately 25 million Americans have experienced PUD. Annually, more than 500,000 people are diagnosed with PUD (Barkun & Leontiadis, 2010).

Age: Seventy percent of those affected by PUD will be age 25 to 64 years (Ramakrishnan & Salinas, 2007).

Gender: Gastric ulcers may be more prevalent in older women, with duodenal ulcer occurrence more common in men.

Ethnicity: Prevalence of PUD secondary to *H. pylori* is higher in African Americans and Hispanics than in Caucasians (Feinstein et al., 2010).

Contributing Factors: *H. pylori* infection is a factor in more than 90% of duodenal ulcers and 80% of gastric ulcers (Chey & Wong, 2007). Use of aspirin and other NSAIDs because of their ability to inhibit prostaglandin synthesis has been shown to increase the risk of PUD (Laine et al., 2010). Other medications that may contribute to PUD include steroids, potassium chloride, bisphosphonates, chemotherapeutic medications, selective serotonin reuptake inhibitors, antidepressants, and antihypertensives (Gonzalez-Perez, Saez, Johansson, Nagy, & Rodrigues, 2014; Ramakrishnan & Salinas, 2007). Spironolactone was found to increase the risk for GI bleeding in patients (Gulmez et al., 2008).

There has been some association with smoking, drinking alcohol, and the use of caffeine in contributing to PUD, although more evidence is needed to support these risk factors. Smoking can hinder healing of ulcerations. Certain infectious conditions and other comorbidities such as tuberculosis, Crohn's disease, hepatic cirrhosis, chronic renal failure, and sarcoidosis are associated with developing PUD (Crosby & Dexter, 2013). Patients who have critical illness, have undergone major surgery, or experienced hypovolemic shock are at high risk for PUD (Ramakrishnan & Salinas, 2007). Question patients about a family history of PUD, as there is an increased likelihood of developing a duodenal ulcer if a first-degree relative has had one in the past (Crosby & Dexter, 2013).

Signs and Symptoms: Older adults presenting with symptoms of PUD must be evaluated for alarm symptoms of upper GI bleeding, anemia, fatigue, and melena (Chey & Wong, 2007). Persistent severe epigastric pain can be indicative of perforation. Patients with gastric ulcers have a history of dyspepsia, epigastric pain, or right upper quadrant pain that radiates to the back after the ingestion of a meal. Pain may be described as gnawing or burning. Patients with duodenal ulcers have pain 1 to 3 hours after a meal, and pain may be relieved with eating food and/or taking antacids. The pain associated with gastric ulcers generally occurs immediately with eating. Nocturnal pain may occur. In older adults, definitive symptoms may be absent. Vomiting and anorexia may be reported instead of epigastric pain (Ramakrishnan & Salinas, 2007).

Physical examination of a patient with PUD may be normal. Palpation may reveal upper abdominal tenderness and guarding. Rigidity of the abdomen and absence of bowel sounds may suggest perforation. Patients with chronic duodenal ulcer disease may exhibit signs of dehydration if nausea and vomiting accompany the other symptoms. In patients with suspected GI bleeding, signs of shock may be detected. Guaiac stool testing should be done.

Diagnostic Tests: Endoscopic gastroduodenoscopy with a biopsy for the detection of peptic ulcer, malignancy, and *H. pylori* remains the gold standard in diagnostic testing for this disease, especially in patients 55 years or older and/or with alarm symptomatology (Fashner & Gitu, 2015). In the diagnostic breath test to detect the presence of *H. pylori,* patients

are given C-13–labeled or C-14–labeled urea to drink. The marked carbon is absorbed and is measured as carbon dioxide in the patient's expired breath (Collazo, 2012). PPI use will need to be restricted for 14 days and antibiotics for 28 days before UBT and fecal antigen testing to avoid false-negative results (Fashner & Gitu, 2015). Serologic antibody testing for *H. pylori* shows results as positive or negative, and this is not able to identify active versus past infection; therefore, it is not helpful in diagnosing the presence of active *H. pylori* infection (Fashner & Gitu, 2015).

Differential Diagnosis:

- Angina
- Esophagitis
- Gastritis
- Gastroenteritis
- Celiac disease
- Acute coronary syndrome
- Gastric carcinoma
- GERD
- Gallbladder disease
- Zollinger-Ellison disease
- Nonulcer dyspepsia
- IBS (Fashner & Gitu, 2015; Lockrey & St. Lim, 2011)

Treatment: First-line therapy, triple drug regimen:

- Select PPI twice daily, plus amoxicillin 1 g twice daily, plus clarithromycin 500 mg twice daily for 10 to 14 days; or
- PPI twice daily, plus clarithromycin 500 mg twice daily, plus metronidazole 500 mg twice daily for 10 to 14 days (if penicillin allergic); or
- Sequential therapy of PPI twice daily and amoxicillin 1 g twice daily for 5 days, followed by PPI twice daily, clarithromycin 500 mg twice daily, and metronidazole 500 mg (or tinidazole 500 mg) twice daily for 5 days (complex and needs more validation in the United States).

Second-line therapy:

- PPI twice daily, amoxicillin 1 g twice daily, clarithromycin 500 mg twice daily, and tinidazole or metronidazole 500 mg twice daily for 10 days (may be an option for patients instead of sequential therapy).
- PPI twice daily, bismuth subsalicylate 525 mg or subcitrate 400 mg four times daily, metronidazole 250 mg four times daily, and tetracycline 500 mg four times daily for 10 to 14 days (used if first-line treatment failure).
- PPI twice daily, amoxicillin 1 g twice daily, and levofloxacin 500 mg once daily (needs further validation in the United States).

Encourage patients to complete the therapeutic regimen despite the cessation of pain. Eradication of *H. pylori* reduces the risk of recurrence of PUD (Collazo, 2012; Ford, Delaney, Forman, Moayyedi, & Ford, 2011). For PUD not related to *H. pylori* infection, treatment with PPIs should be scheduled for 4 to 8 weeks for complicated duodenal ulcer and 8 to 12 weeks for gastric ulcer (Malfertheiner et al., 2012). If PUD is secondary to NSAIDs, a minimum of 8 weeks of PPI therapy should be completed.

Follow-Up: If a gastric ulcer was detected on endoscopy and the patient continues to have symptoms after 8 weeks of treatment, referral for endoscopic examination with a biopsy is indicated. Repeat endoscopy should be at the discretion of the gastroenterologist, as the literature is unclear regarding definitive need for surveillance endoscopy. However, if concern for malignancy is present, repeat endoscopy would be prudent. Periodic stool guaiac testing and blood counts can detect bleeding. Confirm eradication of *H. pylori* 4 weeks after treatment therapy via stool antigen test or urea breath test (Malfertheiner et al., 2007). It is important that PPI therapy has been held for at least 14 days prior to documenting eradication.

Sequelae: Complications originating from PUD in older adults include gastric bleeding with accompanying anemia (Huang & Lee, 2014), perforation, gastric outlet obstruction, and choledochduodenal fistula (Lockrey & St. Lim, 2011; Xi, Jia, Lin, Geng & Zheng, 2016).

Prevention/Prophylaxis: Any patients with previous history of GI bleeding, multiple and/or high NSAID use (including aspirin), over 70 years of age, on corticosteroids, and *H. pylori* infection are at risk for developing PUD (Laine et al., 2010; Lockrey & St. Lim, 2011). Signs and symptoms of impending recurrence of PUD, including epigastric pain, anorexia, and weight loss, should be identified early. Guaiac stool testing should be performed for all patients taking NSAIDs.

Referral: Refer the patient to a gastroenterologist for initial endoscopy and for treatment of upper GI bleeding. PPI therapy, compared with histamine blockers and placebo, has been shown to reduce mortality after upper GI bleeding secondary to PUD; however, studies are unclear if the use of IV PPI therapy prior to an endoscopy is associated with decreased mortality, recurrent bleeding, or surgery (Leontiadis et al., 2007). Refer again if epigastric pain and dyspepsia persist despite treatment and if signs of GI bleeding are present (Ramakrishnan & Salinas, 2007).

Education: Avoidance of aspirin and other NSAIDs and tobacco is essential. Reduction of stressful events, coffee (including decaffeinated forms) and other caffeine products, and alcohol is recommended. Caution patients about taking OTC medication preparations without professional advice. Patients with history of GI bleeding should notify the healthcare provider of any recurrent symptoms and overt bleeding, such as melena or hematochezia.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Assess patients for NSAID use if they are ≥65 years, for prior GI clinical conditions including dyspepsia, and for low-dose aspirin use to determine if medication should be avoided or used with protective measures to reduce GI bleeding.	B	Laine, Curtis, Cryer, Kaur, & Cannon, 2010
Spironolactone was found to increase the risk for GI bleeding in patients.	A	Gulmez et al., 2008
There is a decreased mortality rate from bleeding peptic ulcers when PPI therapy is utilized for treatment after diagnosis.	A	Leontiadis et al., 2007
Treat patients with PUD symptoms and *H. pylori* infection with standard protocol to reduce the risk the risk of ulcer recurrence and promote healing.	A	Ford, Delaney, Forman, Moayyedi, & Ford, 2011
Upper endoscopy is needed for patients with PUD who are older than 55 years and/or with alarm symptoms, or in those with ulcers who do not improve with therapy.	A	Talley, Vakil, & Moayyedi, 2005

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

A 71-year-old woman presents with a 4-week history of dyspepsia and abdominal pain that she describes as gnawing. She states that for years she has had occasional heartburn, but she cannot recall having abdominal pain. She states that she also has arthritis and has taken Motrin every day for the past 2 years. For her stomach pain she has been taking Tums extra strength a couple of times a day. She states that the pain began soon after visiting her sister, who is a nurse at a mission in Nicaragua.

Chief Complaint: "My stomach hurts and wakes me up from my sleep. I find myself belching after meals and I feel bloated."

Objective: Blood pressure (BP) 118/70 mm Hg, heart rate (HR) 90 beats/min, respiratory rate 18 breaths/min, BMI 29.

1. How will you use this information to prepare for today's visit?
2. What additional subjective data are you seeking?
3. What additional objective data will you be assessing for?
4. What are the differential diagnoses that you are considering?
5. What laboratory tests will help you rule out some of the differential diagnoses?
6. What is your treatment and specific information on the prescription you will give to this patient?
7. What are the potential complications from the treatment ordered?
8. What additional specific laboratory tests will you consider ordering?
9. What additional patient teaching may be needed?
10. What type of specialist will you be consulting for this patient?

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CHAPTER 11

Urological and Gynecological Disorders

Lori Martin-Plank

ASSESSMENT

Changes in the female reproductive system occur well before the life stage of older adulthood, considering that the average age of menopause is 51 years. A plethora of choices are now available to women in this age group for prevention and management of postmenopausal problems such as atrophic vaginitis, incontinence, osteoporosis, dyslipidemia, and cardiac problems. The nurse practitioner will likely encounter a variety of physical findings during assessment of the older female patient, based on these emerging choices.

A comprehensive medication history is very important to identify medications or dietary supplements that affect the reproductive system, including hormone replacement therapy and vaginal applications. Equally important is a thorough history, including family history of breast or reproductive organ cancer in a first-degree family member or in the patient herself. The patient should be questioned about any prior breast surgery, including surgery for breast augmentation or reduction. History of mammograms and gynecological examination with a Papanicolaou (Pap) smear (unless the patient has had a total hysterectomy, including removal of the cervical cuff) should be included in the assessment process. Does the patient do breast self-examination? Has she ever discovered a lump or had a biopsy or ultrasound? The patient should also be questioned about breast pain, nipple discharge, rash, trauma, or swelling.

Normal aging changes in the female breast include the atrophy of glandular breast tissue and replacement with connective tissue. There may be a decrease in breast size or breasts may become pendulous and flabby, owing to loss of elasticity. Areolar nipple ducts may be palpable, feeling stringy and firm. Nipple retraction may result from breast atrophy; however, the possibility of malignant changes causing nipple retraction must be ruled out. The inframammary ridge assumes more prominence. Axillary hair decreases.

Physical examination of the female breasts includes inspection for symmetry, lumps, dimpling, or nipple retraction. Optimally, the patient should be sitting, with her hands placed on her hips, raising them over her head during the inspection. Patients with large, pendulous breasts should lean forward, so the examiner can observe for attachment to chest wall or retraction; a bimanual hand technique can be used to examine the breasts at this time also. While the patient's arms are at her sides, the examiner palpates the axillary area for lymphadenopathy, probing deeply while manipulating the patient's arm for maximal access. The patient is then positioned supine with a small towel or pillow under the side to be examined and the corresponding arm placed over the head. Using the finger pads, the examiner palpates in one of three patterns:

- Concentrically from the nipple to the periphery, including the breast tail
- Extending out from the nipple in a pattern similar to spokes on a wheel
- In a linear pattern from top to bottom, starting medially, moving laterally with each line

Breast self-examination technique may also be taught at this time, if the patient desires.

Age-related changes in the male breast include gynecomastia in some patients. Examination of the male breast and axillary areas follows the same sequence as for the female breast, but in an abbreviated format, because less breast tissue exists.

Assessment of the urogenital system of older men and women includes a complete sexual history. Sexual orientation, patterns of sexual expression, current or desired activity, protective practices to avoid transmission of sexually transmitted infections (STIs) including HIV, past or recent history of STIs, number of current partners, and problems with expression of sexuality should be explored (Heidelbaugh & Tortorello, 2012). Review of the medication regimen for potential effects on libido and impotence is important.

If the patient is in a community living situation, are opportunities provided for privacy and intimacy? If medications or medical problems interfere with sexual expression, what interventions are possible? Age-related changes in the reproductive system of both sexes do not interfere with libido

and sexual satisfaction, although physiological response time is slower and more prolonged, and orgasmic response is more generalized.

Assessment of the genitoreproductive system of the older female patient includes historical elements such as menarche, obstetrical history, menopause, history of STIs including herpes, condyloma, surgeries, or malignancies. Medication history and sexual history were discussed previously. The patient should be questioned about urinary symptoms and about any vulvovaginal itching, discharge, or bleeding.

Normal aging changes in the female genitoreproductive system include atrophic changes in most structures. Pubic hair becomes gray and sparse, and the labia flatten. Vaginal epithelium is thinner, drier, and itchy, with less rugae; decreased secretions and alkalinity predispose patients to painful intercourse, friability, and vaginitis (Williams, 2008). The vagina becomes shorter, narrower, and less elastic; tissue is pale pink and shiny. Sexual abstinence or infrequent intercourse intensifies atrophic changes. Undergarments often irritate sensitive external structures. The uterus shrinks, and the ovaries, which atrophy, are not palpable. The pelvic floor muscles, sacral ligaments, and other supporting structures relax, frequently leading to organ (bladder, rectum, and uterus) prolapse.

Examination of the female genitoreproductive system is the same in older patients as in younger patients. With an older patient, before positioning her in the lithotomy position with stirrups, external inspection may be performed by having the patient flex one knee and abduct it. Patients with arthritis may be helped by taking pain medication before the examination. Equipment should be assembled and ready, to limit time spent in the stirrups. Lubrication of the speculum with water before insertion is important for comfort. The choice of speculum is an individual one; the Pederson speculum, with narrow blades, is frequently chosen for virginal or postmenopausal women with a narrow introitus. Examination may reveal relaxation of internal organs, including a cystocele or rectocele. The cervix, which appears pale and shiny, may be flush with the vaginal mucosa or may protrude into the vagina with uterine prolapse (LeBlond, Brown, Suneja & Szot, 2015).

On bimanual examination, the ovaries should not be palpable and the uterus should be small and firm. Some patients may require local hormone application before examination. Pap smears are no longer recommended for women over 65 years old who have had adequate prior screening and are not at high risk for cervical cancer (Guirguis-Blake, Henderson, & Perdue 2017; USPSTF, 2012).

The genitourinary and reproductive systems of the older male patient undergo more gradual changes than those of the older female patient. Pubic hair turns gray and becomes sparse. Penile size decreases slightly. Scrotal contents hang lower; the testes decrease in size and firmness, and less sperm are produced owing to the increase in connective tissue in the tubules. Testosterone production declines slowly, resulting in slower arousal and more prolonged erection before ejaculation. Ejaculation is less intense and shorter, with less seminal fluid and rapid detumescence. Prostatic tissue enlargement is common, frequently resulting in nocturia, urinary hesitancy, decreased urine flow, retention (sometimes with overflow incontinence), and less forceful ejaculation.

When taking the history related to the genitourinary and reproductive systems of the older male patient, ask about difficulty urinating, hesitancy, decreased force of stream, frequency with decreased amount, dribbling, or nocturia. Ask about rashes or lesions that have developed or reoccurred. If nocturia is present, establish its onset and frequency, and explore possible contributing factors other than prostatic enlargement. These include diuretics, caffeine or alcohol ingestion, increased fluid consumption in the evening, habit of frequent urination, mild heart failure, or dependent edema. Other pertinent questions concern urinary symptoms such as frequency, urgency, dysuria, hematuria, or documented urinary tract infection (UTI).

Examination of the urogenital system of the older male patient is essentially the same as for a younger male patient. The normal changes of aging have been described earlier. Examination of the prostate is conducted with the rectal examination, which begins with inspection and palpation of the genitalia for lesions. Examine the penis for any lesions to include herpes simplex, chancre, or chancroid (Williams, 2008). If red, raised hyperkeratotic lesions on the scrotum are detected, consider Fordyce disease, a benign condition. Look for evidence of intertrigo, which could be caused by candida (Williams, 2008). On palpation note if there is any swelling or masses of the scrotum, proceeding to palpation of the inguinal canal for hernia and then to palpation of the inguinofemoral areas for lymphadenopathy. If the older male has a swollen left testicle, consider that the patient may have a left renal vein obstruction. A unilateral scrotal mass could be an inguinal hernia or a hydrocele (Williams, 2008).

For examination of the prostate through the rectum, the patient is placed in the left lateral decubitus position, or standing and leaning forward over the examination table with toes pointed inward, for easier access to the anorectal area. The examiner uses a well-lubricated, gloved finger, advanced above the anal canal, to palpate the anterior wall of the rectum for the prostate gland. The gland is normally heart shaped with a central groove that can be felt. The normal surface is smooth, elastic, and nontender on palpation. Absence of the central groove in a smooth gland or protrusion of the gland more than 1 cm into the rectum is typical of benign prostatic hypertrophy (LeBlond et al., 2015). A hard, irregular nodule may indicate carcinoma. Either finding requires further work-up. If ecchymosis is detected in the perineal area or scrotum, consider a pelvic fracture (Williams, 2008).

ATROPHIC VAGINITIS

Signal Symptoms: Vaginal dryness, dysuria, vulvar and vaginal itching, urinary frequency, blood-tinged vaginal discharge, dyspareunia.

Description: Atrophic vaginitis, also called vulvovaginal atrophy, urogenital atrophy, or adhesive vaginitis, is a noninfectious, sometimes inflammatory, postmenopausal process in which the female genital and urological tissue thins and becomes fragile. Changes in vaginal pH due to a hypoestrogenic state present a more favorable environment for bacterial invasion by trichomonas, candida, and bacterial vaginosis, as well as by resident skin and rectal flora. Dysuria, increase in UTIs, and urinary frequency, as well as dyspareunia, are potential consequences (Bachmann & Santen, 2011a). This can have a significant negative effect on a woman's sexual health and quality of life (North American Menopause Society [NAMS], 2013). Up to 70% of women do not address this issue with health-care providers due to its personal nature (Nappi & Kokot-Kierepa, 2010; Pearson, 2011; Reimer & Johnson, 2011). Unlike vasomotor symptoms associated with menopause, atrophic vaginitis will not resolve with time and without treatment (Wysocki, Kingsberg, & Krychman, 2014). Left untreated, these symptoms can cause not only discomfort and have a negative impact on sexuality, they can negatively impact quality of life, including sexual relationships and emotional well-being (NAMS, 2013).

Etiology: Estrogen deprivation leads to atrophy of the vaginal and vulvar epithelium. Atrophic vaginitis, a common disorder in postmenopausal women, can be surgically induced, created by the natural aging process, or brought on through primary ovarian failure. Postmenopausal estrogen depletion can cause deterioration of tissue, decrease in blood flow, loss of elasticity, decreased rugae, thinning of tissues and epithelium, and increased pH, and these lead to the symptoms (Lester et al., 2015).

Occurrence: This disorder affects all postmenopausal women to some degree, unless estrogen therapy is provided. Women who experience earlier menopause, have diabetes, or have lower body mass may experience more pronounced symptoms (Pearson, 2011). Moreover, symptoms are prevalent in menopausal women but more so in postmenopausal breast cancer survivors (Lester, Bernhart, & Ryan-Wenger, 2012). This may be due to premature menopause with associated symptoms in young breast cancer survivors or due to chemotherapy or enodocrine therapy, such as aromatase inhibitors (Lester et al., 2015).

Age: Atrophic vaginitis is predominantly a problem of postmenopausal women. The average age of natural menopause in the United States is 52.5 years.

Gender: Occurs in women only.

Ethnicity: Not significant.

Contributing Factors: Estrogen-deficient states accompanying metabolic disorders and changes of normal aging create the risk of atrophic vaginitis. Changes in vaginal epithelium and pH caused by estrogen deficiency provide an environment in which pathogenic bacteria and fungi can flourish. Drugs also may alter vaginal secretions and clinical findings (Bachmann & Santen, 2011b).

Signs and Symptoms: Itching, discomfort, burning, dyspareunia, and, at times, a thin blood-tinged vaginal discharge or bleeding after intercourse as the epithelium thins characterize atrophic vaginitis. As vaginal secretions decrease, vaginal dryness can be another bothersome symptom. Complaints of urinary frequency, urgency, and stress incontinence are common. On physical examination, signs include pale, dry, nonrugated vaginal walls with patches of erythema or petechiae or both. The vaginal canal is short and narrow. A watery, white vaginal discharge without foul odor may be found. Estrogen deficiency can lead to loss of uterine support and subsequent uterine descensus (Tan, Bradshaw, & Carr, 2012). The examination may also reveal sparse vulvar hair, decreased subcutaneous fat within the mons pubis and labia majora, volume reduction of labia minora, retracted clitoris, and inadequate vaginal lubrication with pale, dry, and shiny introitus (Lester et al., 2015).

Diagnostic Tests:

Atrophic Vaginitis Diagnostic Tests

TEST	RESULTS INDICATING DISORDER
Pelvic examination with speculum examination and Pap smear (may do wet mount and KOH preparation if infection is suspected)	Pale, dry, nonrugated vaginal mucosa; Pap smear results should be normal; vaginal pH by litmus paper will be ≥5.
Urinalysis to rule out UTI if symptoms	Variable; if dipstick is positive for WBCs and nitrites, a culture and sensitivity should be done. If negative, UTI is not cause of symptoms.

KOH, potassium hydroxide; *Pap,* Papanicolaou; *UTI,* urinary tract infection.

Differential Diagnosis:

- Malignancy: Both atrophic vaginitis and malignancy such as cervical cancer can present with spotting. An endometrial biopsy and a Pap smear would be done if malignancy is suspected, and this would differentiate malignancy from atrophic vaginitis.
- Lichen sclerosus: Whitish lesions on the vulva, squamous cells involved, may be immune mediated. Biopsy is needed to diagnose.
- Lichen simplex chronicus: Pruritus results in scratching and thickened skin plaques or patches in the area (as opposed to thin skin in atrophic vaginitis). A potassium hydroxide (KOH) wet mount or fungal culture may be done to rule out candidiasis.
- Squamous cell hyperplasia: Pruritus results in scratching and inflammatory changes in squamous cells of the vulvar area seen on biopsy.
- Lichen planus: Papular, purple lesions that are pruritic; thought to be immunological in origin. Biopsy and direct immunofluorescence study show clumps of immunoglobulin M (IgM) and complement blended with dead keratinocytes.

- UTI
- STIs and other infections, such as *Candida albicans* and bacterial vaginosis (Samra-Latif, 2012): *Candida* is a fungal organism that presents as pruritis, burning, and with thick, white discharge. Bacterial vaginosis is also pruritic with a thin, white, adherent discharge and may have a fishy odor when mixed with KOH, referred to as a whiff test. Wet mount with KOH will show clue cells in bacterial vaginosis and hyphae in candidiasis.

Treatment: Treatments for atrophic vaginitis include nonprescription therapies (lubricants and moisturizers, herbal products), prescription therapies (vaginal estrogen, ospemifene), and investigational and off-label therapies (raloxifene, lasofoxifene, bazedoxifene, conjugated estrogens, intravaginal DHEA, testosterone) (NAMS, 2013). Use of vaginal dilators and/or sexual activity also promotes a healthy vaginal epithelium (Bachmann & Santen, 2011b). Endometrial biopsy may be required periodically to detect hyperplasia (Samra-Latif, 2012). Hormonal modalities should only be used after careful evaluation of the patient for risks and benefits and discussion with the patient.

Nonprescription Therapies: First-line therapies to alleviate atrophic vaginitis symptoms include vaginal lubricants, moisturizers plus regular sexual activity with a partner, device, or masturbation (NAMS, 2013). Vaginal lubricants and moisturizers are over-the-counter (OTC) products. There are no published reports on the potential irritation from these products; women should be encouraged to test them on a small patch of skin for 24 hours before using intravaginally. Herbal alternatives were tested in a clinical trial that concluded dietary supplements such as black cohosh and soy have no beneficial effect on atrophic vaginitis (Reed et al., 2008).

Prescription Therapies: Many clinical trials have demonstrated the effectiveness of vaginal estrogens. A 2006 Cochrane review of 19 efficacy studies reported all products alleviated symptoms with similar efficacy (Suckling, Lethaby, & Kennedy, 2006). The safety of low-dose vaginal estrogen has been extensively examined, especially related to endometrial thickening and venous thromoembolism, and concluded to have a lower risk profile compared with common doses of systemic estrogen. This is because vaginal estrogen produces very low serum levels (NAMS, 2013). Potential contraindications to vaginal estrogen are postmenopausal women with undiagnosed vaginal/uterine bleeding and controversial in women with estrogen-dependent breast or endometrial neoplasia. The safety of hormone therapy in women with breast cancer is an ongoing concern and it is recommended that breast cancer survivors using adjuvant therapy, such as aromatase inhibitors or tamoxifen, discuss hormone therapy options with their oncologist (NAMS, 2013).

Ospemifene is a selective estrogen receptor modulator (SERM) that is U.S. Food and Drug Administration (FDA) approved for treatment of moderate to severe dyspareunia. It should not be used for breast cancer, as the drug has not been adequately studied in this population.

Investigational and Off-Label Therapies: Raloxifene is a SERM with no apparent estrogen-agonist effect on the vagina, while lasofoxifene has produced significant improvements in vaginal pH and vaginal maturation index (VMI) but currently it is not approved for that purpose. Bazedoxifene in conjunction with conjugated estrogens has demonstrated improvement in atrophic vaginitis with no apparent increase in endometrial hyperplasia; however, bazedoxifene alone has no apparent positive effects on the vagina (Kagan et al., 2010). Intravaginal DHEA is an androgen derivative available in the United States as a dietary supplement. Clinical trials have demonstrated improvements in VMI and reduction in vaginal symptoms (Labrie et al., 2010). Although testosterone cream has been used in the past as a treatment for vulvar lichen sclerosis, Cochrane review established it to be no better than placebo for that purpose (Chi et al., 2011). Current trial data are insufficient to support use of vaginal testosterone for atrophic vaginitis (NAMS, 2013).

Follow-Up: Expected response is quick, with resolution of symptoms within 2 to 3 months. If this does not occur, the patient should be reevaluated and reexamined for other causes of symptoms. Patients treated with continuous menopause hormone therapy need regular return visits every 3 months to check side effects, blood pressure, and response to therapy. When efficacy is achieved, treatment can be discontinued. If symptoms reoccur, reinstitute short-term treatment.

Sequelae: With changes in the vaginal pH of postmenopausal women (pH >5.0) and the loss of normal acidity, bacterial species grow in the vagina that are not found there commonly. Infections can become frequent and chronic.

Prevention/Prophylaxis: Recognition of early signs and symptoms of atrophic vaginitis can lead to the individual seeking treatment to prevent atrophy, dryness, infections, urinary and urethral problems, and sexual dysfunction. Intermittent use of topical vaginal estrogen can prevent recurrence of atrophic vaginitis, provide adequate levels of hormone, and give soothing relief. Use of a vaginal lubricant in conjunction with a regular vaginal moisturizer also may be helpful, especially before coitus.

Referral: Gynecology referral is appropriate for patients who do not respond to treatment or have vaginal bleeding. Patients who present with severe estrogen depletion, evidenced by marked perineal and vaginal changes, along with pelvic floor relaxation, need gynecological referral before initiating topical treatment.

Education: Use water-soluble lubricants for patients with atrophic vaginitis. Counsel the patient regarding the benefits of regular sexual activity. Identify age-related difficulty associated with intravaginal application of creams and address these needs with sensitivity.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
First-line therapies for atrophic vaginitis include nonhormonal lubricants with intercourse and possible regular use of long-acting vaginal moisturizers.	A	NAMS, 2013
For symptomatic women with moderate to severe symptoms or those who don't respond to lubricants/moisturizers, vaginal or low-dose systemic estrogen is the therapeutic standard.	A	Wysocki, Kingsberg, & Krychman, 2014
For women with a history of breast or endometrial cancer, management depends on a woman's preference, need, understanding or potential risks, and consultation with her oncologist.	C	Lester et al., 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

BREAST CANCER

Signal Symptoms: None; breast mass.

Description: Breast cancer is the most common noncutaneous cancer in U. S. women and was estimated to affect 61,000 with *in situ* disease, 246,660 with invasive disease, and expected to result in 40,450 deaths in 2015 (American Cancer Society [ACS], 2015). Men represent approximately 1% of breast cancer incidence and deaths (ACS, 2015). Incidence and death rates for breast cancer increase with age; however, there is a marked decrease in incidence rates for women age 80 years or older, possibly due to reduced screening. The median age at breast cancer diagnosis was 61 years in 2008 through 2012, which means half of women diagnosed with breast cancer were 61 years old or younger at time of diagnosis. Currently, women living in the United States have a 12.3% or one in eight lifetime risk of developing breast cancer (Howlander et al., 2015).

Breast cancers are classified as malignant, neoplastic tumors that start in the tissue of the breast. The two most common types are ductal and lobular carcinomas, with ductal carcinomas having a higher prevalence (ACS, 2015). Breast cancers may be invasive or noninvasive (also called *in situ*). Ductal carcinoma *in situ* (DCIS) is a condition in which abnormal cells replace the normal epithelial cells of the breast ducts and is considered a noninvasive form of breast cancer. Lobular carcinoma *in situ* (LCIS) has cells that look like cancer cells growing with breast lobules and is thought to be a marker for developing invasive lobular cancer (ACS, 2015).

Most breast cancers are invasive, which means cancer cells have broken through the walls of the glands or ducts where they originated and subsequently grow into surrounding breast tissue. The prognosis of invasive breast cancer is influenced by disease stage at first diagnosis. This is determined by a classification of tumor stage by either the TNM Classification of Malignant Tumors (TNM) or the Surveillance, Epidemiology, and End Results (SEER) summary stage system.

Clinical settings utilize the TNM staging system, including information about tumor size, how far it has spread within the breast and to adjacent tissues (T), the extent of spread to nearby lymph nodes (N), and presence or absence of distant metastases (spread to distant organs) (M). Once the T, N, and M are determined, a stage of 0, I, II, III, or IV is assigned, with stage 0 being *in situ*, stage I being early stage invasive cancer, and stage IV being the most advanced disease with distant organ metastases (Edge et al., 2010). The SEER staging system is more simplified and is commonly used in reporting cancer registry data, and in public health research and planning (Young et al., 2001).

Occasionally, breast cancers form in other areas of the breast. Included in this are inflammatory breast cancer, triple-negative breast cancer, Paget's disease of the nipple, Phyllodes tumor, and angiosarcoma (ACS, 2011). The exact cause of breast cancer is unknown, with environmental and genetic contributions.

Occurrence: An estimated 231,840 cases of invasive breast cancer, plus 60,290 additional cases of *in situ* disease was predicted among women in 2015. At the same time, 40,290 women were expected to die from breast cancer in 2015, which is second only to lung cancer deaths. In men, 2,350 new cases of breast cancer and 440 deaths were predicted in 2015. In 2014, there were more than 3.1 million living U.S. women with a history of breast cancer; some of these women are cancer free, while others were undergoing treatment (ACS, 2015).

Age: The median age of diagnosis of breast cancer is 61 years; median age of death is 68 years. Risk is positively related to increased age, placing older women at high risk. Incidence of *in situ* breast cancer increased in the 1980s and 1990s and is linked to increases in mammography screening. The incidence increases were greater in women age 50 years or older than in women under age 50 years. Incidence rates

of *in situ* breast cancer have stabilized since 2000 among women age 50 years or older, and in women under age 50 years since 2007.

Such trends are thought to reflect trends in mammography screening. The historic increases in breast cancer incidence is thought to reflect delayed childbearing and having fewer children, which are known breast cancer risk factors. Invasive breast cancer rate increases in the 1980s reflect greater use of mammography for screening, which diagnosed cancers 1 to 3 years earlier then would have been without the screening. Increases in the prevalence of mammography screening, rising rates of obesity, and use of menopausal hormones may also contribute to increases in breast cancer risk. Similarly, the reduced rates of invasive breast cancer noted between 2002 and 2003 has been linked to publication of the Women's Health Initiative clinical trial data in 2002, which triggered widespread reduction in hormone therapy use (ACS, 2015).

Gender: Female gender is one of the two most significant risk factors for developing breast cancer. Excluding cancers of the skin, breast cancer is the most common cancer among U.S. women and accounts for 29% of newly diagnosed cancers (ACS, 2015).

Ethnicity: Breast cancer is slightly more prevalent in Caucasian women age 60 to 84 years, followed by African American, Hispanic, Asian/Pacific Islander, and Native American/Alaska Native women (CDC, 2015). African American women have a higher mortality rate at all ages (ACS, 2015).

Factors that contribute to health-care disparities that exist in cancer are socioeconomic status (education, income, and employment), age, access to and use of health-care services, behaviors, social environment, exposure to carcinogens, and treatment (U.S. Cancer Statistics Working Group, 2010). Overall breast cancer death rates decreased from 1989 to 2012 by 36%, and this decrease occurred in younger and older women. The breast cancer death rate has been level since 2007 among women younger than 50 years (Howlander, 2015). Decline in breast cancer deaths is attributed to improvements in breast cancer treatment and early detection (Berry, 2005); however, screenings are lower among minority, low income, inner-city women (Smith-Bindman et al., 2006). Survival is lower among women with more advanced stage at diagnosis, as well as in impoverished women, those with less education, and those with a lack of health insurance (Shi et al., 2015).

Contributing Factors: The major factors that affect cancer risks are age and gender. As age increases, so does cancer risk. Ethnicity has already been discussed and is a nonmodifiable risk factor along with age, family history, early menarche, and late menopause. Modifiable risk factors include postmenopausal obesity, use of combined estrogen and progestin menopause hormone therapy, alcohol consumption, and not breastfeeding. Several strategies are suggested to help reduce the risk of breast cancer, and these include avoiding weight gain and obesity, engaging in regular physical activity, minimizing alcohol intake, and selecting alternatives to menopause hormone therapy for menopause symptom management (ACS, 2015).

Family and Personal History: A women's risk of breast cancer increases by 50% if she has a first-degree relative diagnosed with breast cancer. Compared to women without a family history, risk of breast cancer is about two times higher for women with one first-degree female relative, nearly three times higher for those with two relatives, and almost four times higher for women with three or more relatives diagnosed with breast cancer. If one of those affected relatives was diagnosed at a young age, risk is further increased. Approximately 5% to 10% of breast cancers are linked to an inherited genetic mutation. The most commonly occurring mutation is of the BRCA1 and BRCA2 genes, which are inherited from a parent. Women in the general population have a 7% risk of developing breast cancer by age 70 years, however, the average risk for BRCA1 and BRCA2 mutation carriers is approximately 57% to 65% and 45% to 55%, respectively (ACS, 2015). Partner and Localizer to BRCA2 (PALB2) mutations are thought to confer risk similar to BRCA2 mutations (Antoniou et al., 2014).

Compared to women who were never diagnosed with breast cancer, women with a personal history are about one and one-half times more likely to develop a new breast cancer (ACS, 2009). DCIS or LCIS is considered a precursor to invasive cancer, and it may increase a woman's risk for developing a new invasive breast cancer. Women with a history of DCIS are eight to ten times more likely to be diagnosed with an invasive breast cancer compared to women without a history of DCIS (Lopez-Garcia et al., 2010). Previous chest radiation and treatment of other cancers increases the risk of breast cancer (Henderson et al., 2010; Patterson, 2010). Benign breast conditions have been linked to breast cancer risk and are categorized into three general groups: nonproliferative lesions (overgrowth of breast tissue; little or no effect on breast cancer risk), proliferative lesions without atypia (abnormal cells; small increase in risk of breast cancer, one and one-half to two times the risk of those without one of these lesions); and proliferative lesions with atypia (about four to five times higher risk than average risk) (Santen, 2014).

Dense breast tissue (an indicator on mammogram of the amount of glandular and connective tissue in the breast relative to fatty tissue) is a contributing factor to breast cancer. Dense breast tissue decreases the ability to detect masses early (Henderson et al., 2010; Katapodi et al., 2009). Compared to women with 11% to 25% breast density, those with 26% to 50% or more than 50% have about a 1.5 or 2.3 times, respectively, higher risk of breast cancer (Bertrand et al., 2015). Breast density is usually lower among women with higher body weight, and drugs such as tamoxifen are known to decrease density, while menopause hormone therapy is known to increase density (Boyd et al., 2007). A more recent finding is that alcohol may increase breast density (Trinh et al., 2015).

Endogenous hormone levels are linked to breast cancer. Early menarche and/or late menopause increases the risk of breast cancer. Nulliparity, childbirth delayed until after age 30, and lack of breastfeeding increase breast cancer risk. These findings are thought to be related to the number of menstrual cycles in the woman's lifetime, thus, amount of exposure to endogenous hormones (ACS, 2015). Factors such as recent oral contraceptive use and estrogen plus progesterone menopause hormone therapy for menopause symptom management, particularly post menopause, increase risk. Hormonal (estrogen plus progesterone products) birth control

may be associated with a small increase in breast cancer risk, especially among women who began use before 20 years of age or before their first pregnancy. Risk appears to diminish when women stop taking it and after 10 years the risk is similar to that of women who never took oral contraceptives (Bassuk & Manson, 2015).

High bone mineral density in postmenopausal women has been associated with increased risk of breast cancer, although study findings are equivocal (Grenier et al., 2011). It is suggested this risk is associated with cumulative estrogen exposure (ACS, 2015). There is solid evidence that suggests breastfeeding for a year or more slightly reduces a woman's overall risk of breast cancer (Faupel-Badger et al., 2013). A global review of 47 studies found a reduced risk of 4% for every 12 months of breastfeeding (Collaborative Group on Hormonal Factors in Breast Cancer, 2002). The primary theory is that breastfeeding inhibits menstruation, thus reducing the lifetime number of menstrual cycles. Yet, despite the theory about reducing lifetime number of menstrual cycles, there continue to be claims that abortion is linked to increased risk for developing breast cancer. However, a large body of scientific evidence confirms there is no link between either spontaneous or induced abortion and breast cancer (ACS, 2015).

Signs and Symptoms: Signs and symptoms of breast cancer range from nonexistent to masses detected by palpation. Symptoms include a breast lump or mass, bloody nipple discharge, change in the size or shape of the breast, changes of the skin, inverting of the nipple, and changes in the skin texture (e.g., peeling, flaking, redness, or pitting over the breast) (Watkins, 2009). Early symptoms of breast cancer are painless; symptoms are usually evident when the cancer grows. Irregularly shaped, painless, hard masses have an increased probability of being cancerous. Due to the spread of cancer to the lymphatic system, a lump or swelling in the axilla should be further evaluated (ACS, 2015).

Diagnostic Tests: For high-risk women or women who have noted a breast mass, history taking and clinical breast examination is the first stage of breast cancer diagnosis. A diagnostic mammogram is used to determine breast disease in women who have breast symptoms or abnormal screening mammography. This diagnostic procedure is distinguished from screening mammography. Routine mammography screening is recommended by the ACS beginning at 45 years of age for average-risk women. At age 55 years women may go to biennial screening or continue with annual screening. However, the ACS states that women 40 to 44 years old should have the choice to begin annual screening. Women should continue screening mammography if their overall health is good and they have a life expectancy of 10 years or more. However, the ACS does not recommend clinical breast examination for breast cancer screening among average-risk women at any age.

The U.S. Preventive Services Task Force (USPSTF) made its final recommendations on screening for breast cancer (2016), which apply to women age 40 years and older who have no signs or symptoms of breast cancer and don't already have breast cancer or a high-risk lesion. Individualized recommendations about breast cancer screening in high-risk individuals should come from the woman's own clinicians. The USPSTF recommendations are based on research studies on screening mammography. These final recommendations are summarized as:

- The value of mammograms increases with age, such that women ages 50 to 74 years benefit the most. The best balance between benefit and harm seems to be when screening occurs every 2 years.
- Starting breast cancer screening before age 50 years is an individual decision and women should consult their clinician to determine whether screening is right for them.
- There is insufficient evidence to determine effectiveness of screening for women over age 75 years.
- There is insufficient evidence to determine effectiveness of screening with three-dimensional mammography.
- There is insufficient evidence to determine the effectiveness of additional screening with other methods for women with dense breasts who have had a negative mammogram.

Here is the overall recommendations from the USPSTF on screening for breast cancer:

- Biennial screening mammography for women age 50 to 74 years. Grade B
- Starting screening mammography in women 40 to 49 years should be an individual decision and may be more appropriate for those who place a higher value on the potential benefit than the potential harms. Grade C
- Current evidence is insufficient to assess the balance of benefits and harms of screening mammography in women 75 years and older. I Statement
- There is insufficient evidence to assess the benefits and harms of three-dimensional mammography as a primary screening method for breast cancer. I statement
- Current evidence is insufficient to assess the balance of benefits and harms of adjunctive screening for breast cancer using breast ultrasonography, magnetic resonance imaging (MRI), three-dimensional mammography, or other methods in women identified to have dense breasts on an otherwise negative screening mammogram. I Statement

Mammography is a low-dose x-ray that allows visualization of the breast's internal structure (ACA, 2015). Women with breast implants need to have an increased number of x-rays to provide a thorough evaluation of the breast tissue. Mammography provides information on calcifications, micro or macro, or masses in the breast. Mammography cannot diagnose breast cancer. This type of screening is ineffective on younger women because the breast tissue is very dense and may obscure masses or tumors. Suspicious findings on mammography must be confirmed by biopsy.

Currently, there are three types of mammography: film, digital, and digital breast tomosynthesis (three-dimensional) (ACS, 2015). Mammography may cause follow-up examinations due to false-positive findings that are most often found on screening mammogram (Hubbard et al., 2011). It is thought that the introduction of tomosynthesis will reduce false positives and detect more invasive cancers compared to digital mammography alone (Friedewald et al., 2014).

MRI is also used for women at high risk for cancer in conjunction with mammography. MRIs have an increased sensitivity to detecting breast cancer, but also lead to an

increased rate of false positives. Breast mass biopsy is used for confirmation.

Breast ultrasound is often used to further evaluate abnormal findings from a screening or diagnostic mammogram or from a clinical breast examination. It has been demonstrated that ultrasound detects more cancer than mammography alone in women with dense breast tissue, although it can increase the likelihood of false-positive results (Berg et al., 2012).

Excisional biopsy or large core biopsy is recommended for all palpable, solid lesions. Image-guided large core breast biopsy is the choice for nonpalpable lesions and abnormal breast calcification (Agency for Healthcare Research and Quality [AHRQ], 2012).

Differential Diagnosis: Not all breast masses are cancerous. Certain benign breast conditions exist. These conditions must be evaluated and malignancy ruled out. These benign conditions are classified as nonproliferating lesions, proliferating lesions without atypia, and proliferating lesions with atypia.

- Benign nonproliferating breast mass: Nonproliferating lesions do not seem to affect future cancer risk.
  - Adenosis
  - Fibroadenoma
  - Fibrosis
  - Fat necrosis
  - Duct ectasia
  - Lipomas
  - Neurofibromas
  - Mastitis
- Proliferating lesions without atypia: Increase cancer risk by approximately one to two times the normal.
  - Radial scar
  - Sclerosing adenosis
  - Ductal hyperplasia
  - Papillomas
- Proliferating lesions with atypia: Increase cancer risk by four to five times the normal (Ferrara, 2011).
  - Atypical ductal hyperplasia
  - Atypical lobular hyperplasia

Treatment: The treatment for breast cancer confirmed by biopsy includes neoadjuvant therapy, adjuvant therapy, chemotherapy, surgical therapy, and endocrine therapy. Treatment is based on stage of disease, molecular profiling (hormone-receptor positive; HER2/neu positive; triple negative [ER, P, and HER2/neu negative]) (National Cancer Institute [NCI], 2016).

Neoadjuvant therapies include the therapies given to reduce the size of the tumor, allowing for improvement of outcomes and breast conservation (Waljee & Newman, 2007). Following is a summary of treatment of invasive breast cancer by stage (ACA, 2016).

Stage I: Relatively small, these breast cancers have either not spread to lymph nodes or a tiny area of cancer is found in the sentinel lymph node (first node to which cancer is likely to spread). Surgery is the main treatment and is generally breast-conserving surgery (BCS), although mastectomy is sometimes performed. Nearby lymph nodes are checked with sentinel lymph node biopsy (SLNB) or axillary lymph node dissection (ALND). Radiation therapy is likely to be performed if BCS is done. Adjuvant systemic therapy (chemotherapy or hormone therapy) may be recommended. Tumors less than 1 cm across usually do not need chemotherapy, but if the tumor was hormone receptor positive, hormone therapy (tamoxifen or an aromatase inhibitor) may be added as adjuvant therapy.

Stage II: These cancers are treated either with BCS or mastectomy, and nearby lymph nodes must be checked with SLNB or ALND. Women who have BCS are treated with radiation therapy following surgery. If chemotherapy occurs after surgery, then radiation therapy follows. It should be noted that in some cases, breast reconstruction can be done during the surgery to remove cancer. But, if radiation is to occur after surgery, it is considered better to wait for reconstruction until after all radiation treatments are complete.

Systemic therapy is recommended for women with stage II breast cancer. Some are given before surgery (neoadjuvant therapy) while others follow surgery (adjuvant therapy). Neoadjuvant treatments are a good option for large tumors, because they shrink the tumor before surgery and often enough make BCS a possibility. Chemotherapy can be given before or after surgery; HER2-targeted drugs are started along with chemotherapy and continue after surgery for a total of 1 year. Herceptin and Perjeta are the most commonly used drugs for this purpose. Hormone therapy is given if the cancer is hormone receptor-positive. Tamoxifen is commonly used for premenopausal women and an aromatase inhibitor is commonly used for postmenopausal women.

Stage III: Stage III breast cancers are large (more than 5 cm) or the cancer has spread to many nearby lymph nodes. Included in this stage are some inflammatory breast cancers that have not spread beyond nearby lymph nodes. Treatment for inflammatory breast cancer can vary from the usual treatments for stage III.

Neoadjuvant therapy is a common start for treatment of stage III breast cancer. The purpose is to shrink the tumor before surgery. Another option is to treat with surgery first. Because these tumors are relatively large, mastectomy is a common occurrence, although in women with large breasts, BCS is a possibility. Surgery is usually followed with adjuvant systemic chemotherapy and/or hormone therapy and/or HER2 hormone treatment (trastuzumab). Radiation is recommended following surgery and chemotherapy. For hormone receptor-positive tumors, hormone therapy is prescribed for 5 years. HER2/neu positive tumors are treated with HER2-targeted drugs starting at the time of chemotherapy and continuing for 1 year.

Stage IV: Stage IV breast cancers have spread beyond the breast and nearby lymph nodes to other parts of the body. The most common sites are bones, liver, and lungs. It may also spread to the brain or other organs, including ovaries, uterus, and cervix. Drug therapies, hormone therapy, chemotherapy, targeted drugs such as trastuzumab (Herceptin) and pertuzumab (Perjeta), or a combination of these are the primary treatments. Surgery and/or radiation therapy may be useful but are less often used. Treatment can shrink tumors, slow their growth, improve symptoms, and help women live longer; however, these cancers are not generally considered curable (ACA, 2016).

Follow-Up: Follow-up of breast masses and breast cancer is based on the specific diagnosis, cancer staging, and treatment rendered.

Sequelae: Survival rates are calculated as relative survival. From 2003 through 2012, breast cancer death rates declined annually by 1.8% in Caucasians, 1.5% in Hispanics, 1.4% in African Americans, and 1.0% in Asians/Pacific Islanders, but remained unchanged among Native Americans/Alaska Natives (Howlander et al., 2015). Decline in breast cancer mortality is attributed to improved breast cancer treatment coupled with early detection (ACA, 2015).

However, as trends in breast cancer death rates decline, there is a striking divergence in long-term breast cancer survival between African American and Caucasian women, with African American women demonstrating higher mortality rates. Congruent with improved breast cancer treatment and mortality decline, the divergence between African American and Caucasian women widened so that by 2012 breast cancer death rates were 42% higher in African American women (ACA, 2015). This difference in mortality may reflect many factors, including differences in stage at diagnosis, obesity and comorbidities, and tumor characteristics. Access, adherence, and response to treatment may also be factors. The racial disparity may also reflect differences in mammography screening rates, although recent data suggest screening rates are similar between African American and Caucasian women (Newman, 2015).

Prevention/Prophylaxis: Screening mammography recommendations were discussed previously. Screening mammography remains the most effective means of breast cancer screening.

It is no longer recommended to teach breast self-examination during health examinations for women between the ages of 40 and 69 years due to potential overdiagnosis and overtreatment. Approximately one in three women will have a false-positive screening result (Hubbard et al., 2011). The age to discontinue screening mammography remains unclear.

Genetic testing is recommended for individuals with a mutation detection probability of at least 10%. Mammography, ultrasound, and MRIs are recommended for women with a lifetime risk greater than 29% or a heterozygous risk greater than 19% starting at age 25 years. Mutation carriers may obtain prophylactic surgery of a bilateral salpingo-oophorectomy or bilateral mastectomy (Metcalfe et al., 2008). These screenings are currently not supported in the general population. Chemoprevention has been shown to decrease breast cancer risk in the context of risk factors (Kelly et al., 2010). Chemoprevention should be discussed with high-risk women; this has been associated with decreased breast cancer risk and death (Armstrong et al., 2007).

Lifestyle modification has been shown to have a positive impact on cancer diagnosis. These modifications include maintaining a healthy body mass index (BMI), increased physical activity, and improved general nutrition (Wishart, 2010). High soy intake and breastfeeding have been associated with a reduction in breast cancer risk (Schmitz et al., 2010; Trock, Hilakivi-Clarke, & Clarke, 2006).

Referral: All breast masses should be further evaluated for malignancy. Referrals to surgeons, oncologists, geneticists, genetic counselors, mental health services, and appropriate support groups are made as necessary. Negative mammography with a positive breast mass may need further evaluation, especially in young women with dense breast tissue.

Education: It is important to instruct patients on current breast screening recommendations. It should be noted that breast screening recommendations vary and may change regularly. Therefore, all clinicians must keep abreast of current screening recommendations made by the NCI, the ACS, and professional organization evidence-based clinical practice guidelines.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Women who are age 45 to 54 years should be screened annually.	A	ACS, 2015
Starting breast cancer screening with mammography prior to age 50 years is an individual decision women should make in consultation with their clinicians.	A	USPSTF, 2016
Effectiveness of mammography for women over age 75 years cannot be determined as there is insufficient evidence.	C	USPSTF, 2016
Clinical breast examination for breast cancer screening among average-risk women at any age is not recommended.	A	ACS, 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CYSTITIS

Signal Symptoms: Dysuria, frequency, urgency, suprapubic tenderness, change in urine character (hematuria, color change, change in odor), mental status changes (Hooton, 2012; Juthani-Mehta et al., 2009).

Description: Cystitis is a pathogenic invasion of the wall of the bladder, usually resulting from an ascending infection via the urethra, of bowel flora organisms from the perineum (Beveridge, Davey, Phillips, & McMurdo, 2011; Dielubanza & Schaffer, 2011). Cases of cystitis are classified as complicated or uncomplicated. Uncomplicated cases (focus of this chapter) are defined as acute genitourinary symptoms with evidence of pyuria and bacteriuria in a nonpregnant person with no history of abnormal urinary tract (Hooton, 2012; Rowe & Juthani-Mehta, 2014). All other cases are considered complicated, but this does not account for diversity of complicated syndromes and mislabels complicated syndromes that can be managed with short-term therapy; that is, acute uncomplicated pyelonephritis that is managed in the outpatient setting (Hooton, 2012).

Asymptomatic bacteriuria (ASB) (a urinary culture with more than 10^5 colony-forming units) with no corresponding urinary tract symptoms is prevalent in the older adult (especially those residing in nursing homes) and is not diagnostic (Juthani-Mehta et al., 2009; Schmiemann, Kniehl, Gebhardt, Matejczyk, & Hummers-Pradier, 2010). The USPSTF has found that evidence does not support the routine screening of nonpregnant adults for asymptomatic bacteriuria and that no benefit is derived from it (Lin & Fajardo, 2008). The American Urological Association (AUA) and Infectious Disease Society of America (Badalato & Kauffman, 2016; Gupta et al., 2011; Nicolle, 2005) do not recommend treatment for such.

Etiology: The most common organism identified in the development of cystitis in adults of all ages is *E. coli,* which transcends across community dwelling and long-term care residing older adults (Rowe & Juthani-Mehta, 2014; Grabe et al., 2011). Other common organisms include klebsiella, proteus, and enterococcus, with responsible organisms being the same across community and long-term care facility (LTCF) populations (Rowe & Juthani-Mehta, 2014).

Occurrence: UTI is the second most commonly diagnosed hospital infection and represents close to 5% of all emergency department visits for all U.S.-based adults older than 65 years of age (Rowe & Juthani-Mehta, 2014). Foxman and Brown (2003) noted that the self-reported rate of annual UTI incident is 12% in young women, with half of all women reporting one UTI by age 32 years. UTI also accounts for approximately 30% to 40% of all LTCF health-care acquired infections (Tsan et al., 2010; Cotter et al., 2012).

Age: Incidence of cystitis/UTI increases with age; difficult to calculate due to ASB.

Gender: Men have a 20% incidence of UTI, with lifetime prevalence of 1% (Griebling, 2005). After age 65 years, the rate of cystitis in men significantly increases, but is still approximately one-half that of women (Dielubanza & Schaffer, 2011). Incidence in postmenopausal women can range from 0.07/women/year to 0.13/women/year in women greater than 85 years old with great degrees in prevalence (Rowe & Juthani-Mehta, 2014). Community-dwelling men 70 years of age and older have a prevalence rate of ASB from 3.6% to 19%, whereas their institutionalized counterparts have a prevalence rate from 15% to 40% (Nicolle et al., 2005). Community-dwelling women 70 years of age and older have a prevalence rate of ASB from 10.8% to 16%, whereas their institutionalized counterparts have a prevalence rate from 25% to 50% (Nicolle et al., 2005).

Ethnicity: Not significant.

Contributing Factors: Predisposing factors to the development of cystitis in older adults include indwelling catheters, urethral or condom catheters, incontinence (urinary and fecal), cognitive impairment, neurological conditions that impair bladder emptying, and diabetes, which can lead to neurogenic bladder (Beveridge et al., 2011). Other predisposing factors to cystitis include sexual intercourse, functional disability, sickle cell disease, prior antibiotic therapy, genetic predisposition, and functional or structural genitourinary tract abnormalities (including urethral strictures, uterine or bladder prolapse, ureteral weakness, and vesicoureteral reflux or renal calculi) (Dielubanza & Schaffer, 2011; Nicolle, 2005; Schmiemann et al., 2010). Frail older adults in institutionalized settings are at additional risk due to lack of adequate fluids and immobility.

The elder adult's compromised anatomical defenses related to decline of aging allow unheeded bacterial ascent of the genitourinary tract and subsequent colonization of pathogens (Dielubanza & Schaffer, 2011; Badalato & Kauffman, 2016). Benign prostatic hyperplasia, incontinence, urinary retention, and institutionalization are all contributing factors to the increase in the prevalence of cystitis in older adult men (Dielubanza & Schaffer, 2011). The woman's shorter urethra is an anatomical variance as to why women are more susceptible to UTIs over their lifetime, along with the urethra's proximity to rectal and vaginal flora, increasing the risk of cross-contamination. The length of the male urethra provides a protective barrier against ascending bacterial infection, especially before age 50 years (Dielubanza & Schaffer, 2011). The loss of estrogen in older adult women leads to increased infection through increased vaginal pH and the loss of protective lactobacillus.

Signs and Symptoms: The older community-dwelling patient presents with similar symptoms of UTI as a younger person, but also may endorse or present with an atypical presentation of mental confusion, anorexia, malaise, and incontinence possibly being the first symptoms of cystitis in an older patient, especially for patients with indwelling catheters or the frail older adults (Woodford & George, 2009). Caution must be exercised though, and the differential must be explored before cystitis can be diagnosed in the absence of concurrent urinary-specific signs and symptoms (dysuria, change in urine character, etc.). Symptoms such as nonspecific functional decline, increased confusion, and other nonspecific signs and symptoms may be erroneously attributed to cystitis in the absence of key urinary tract symptoms and,

therefore, should be thoroughly examined in the differential (Beveridge et al., 2011). Patients should be questioned about their sexual history, including use of spermicide (which can alter flora, leading to increased uropathogen colonization) (Dielubanza & Schaffer, 2011). Women may report pelvic pain or vaginal or cervical discharge that may indicate an ascending infection. If fever, flank pain, and other systemic symptoms are reported, consider obstruction when ordering diagnostic tests.

Physical examination may reveal fever, tachypnea, and tachycardia. Suprapubic tenderness may be elicited on palpation. Percussion for costovertebral angle tenderness may be positive with reported flank pain and is usually suggestive of pyelonephritis (Hooton, 2012; Beveridge et al., 2011).

Vaginal examination in women should rule out discharge, irritation, and erythema (Dason, Dason, & Kapoor, 2011). In men, the prostate gland should be examined gently to assess for enlargement, bogginess, and tenderness to rule out prostatitis, which is considered a complicated UTI.

Diagnostic Tests: Bacteriuria and pyuria are the main laboratory clinical manifestations of cystitis (Hooton, 2012; Rowe & Juthani-Mehta, 2014). Pyuria does not diagnose bacteriuria. In the older adult, the presence of localized genitourinary symptoms (see Signal Symptoms) and pyuria on urinalysis are required for diagnosis (Rowe & Juthani-Mehta, 2014). Certain gram-negative organisms will be dipstick positive for the presence of nitrates, but atypical organisms are not detected in this manner (Woodford & George, 2009).

Urine culture is preferred but not required in the presence of uncomplicated cystitis (Hooton, 2012). The presence of greater than 10^5 colony-forming units/mL of a single bacterium in a culture of freshly voided urine is generally considered to be a significant bacteriuria (Beveridge et al., 2011). A true diagnosis involves examination of urine and clinical picture of illness (Badalato & Kauffman, 2016).

Due to the prevalence of asymptomatic bacteriuria in institutionalized elders (15% to 50%) with concomitant pyuria (90%), bacteriuria plus pyuria in this population is not sufficient for diagnosis without additional genitourinary clinical symptoms (Juthani-Mehta et al., 2009). Additional radiographic testing may be necessary if obstruction is suspected or if an abnormal and persistent pattern of infection needs to be further explored (Dielubanza & Schaffer, 2011; Grabe et al., 2011).

Although considered a complicated UTI, catheter-associated UTI (CAUTI) is widely recognized as the most common health-care associated infection (AUA, 2014). Careful consideration needs to be taken to differentiate between ASB and CAUTI. The AUA emphasizes the need for careful surveillance because of the potential risk of multidrug-resistant organisms. Experts agree that ASB in the face of an indwelling catheter does not require antibiotic treatment, even though administration rates remain as high as 32% (Badaloto & Kauffman, 2016).

McGeer criteria were developed in 1991 to provide standardized guidance for infection surveillance activities in long-term care facilities. Updates to the guidelines were made in 2012, making significant changes to the diagnosis of UTI with and without the presence of an indwelling catheter (Stone et al., 2012). Key points with regard to infection surveillance: All symptoms must be new or acutely worse, alternative noninfectious causes of symptoms should be evaluated/considered before event is deemed an infection, and identification of infections are not to be based on a single piece of evidence that should always consider the clinical presentation (Stone et al., 2012).

Differential Diagnosis:

- Urethritis
- Prostatitis
- Epididymitis
- Vaginitis
- Obstructive uropathy
- Complicated pyelonephritis (uncomplicated pyelonephritis can be treated successfully in the outpatient setting) (Michels & Sands, 2015).

Treatment: Gupta and colleagues (2011) and Hooton (2012) outlined initial and empirical therapy options for the treatment of uncomplicated UTI in women that highlighted nitrofurantoin 100 mg twice daily for 3 days over trimethoprim-sulfamethoxazole related rising resistance rates and nitrofurantoin's lesser side effect profile. Another option is Fosfomycin given in a 3 g one-time dose (Hooton, 2012; Gupta et al., 2011). Short-course therapy optimally balances pathogen eradication with minimizing adverse drug effects (including vaginal and bowel flora alterations) (Dielubanza & Schaffer, 2011). Fluoroquinolones and narrow spectrum cephalosporin antibiotics should be reserved for special circumstances related to known microbial sensitivity and/or the patient's reported allergy list (Hooton, 2012).

Uncomplicated treatment of UTI in the adult male can be treated empirically for 7 days with a fluoroquinolone or sulfamethoxazole-trimethoprim DS, taking into consideration local antimicrobial resistance patterns (Grabe et al., 2013). Older men may require longer therapy for 10 to 14 days (Gilbert, Chambers, Eliopoulos, & Saag, 2015).

Utilizing the updated McGeer criteria and supported by the American Geriatrics Society, Zarowitz and colleagues (2016) presented several therapeutic algorithms that provide guidance in the assessment of the older adult with signs and symptoms, along with objective physical examination findings that are consistent with UTI, and then directs the selection of appropriate empirical antibiotic therapy prior to that time a culture and sensitivity results. Highlights note the exclusion of odorous or cloudy urine as an indicator for urine culture or empiric therapy, discontinuing antibiotic therapy if cultures are negative, and no longer using fluoroquinolones as first-line empirical therapy for the treatment of noncatheter-related UTI.

Treatment recommendations have been changing to reflect trends in pathogen resistance, antibiotic adverse effects, and best evidence-based practice. Current recommendations include multiple treatment regimens based on the needs of the patient. All providers should be aware of the patient's renal function or preexisting renal disease before prescribing an empirical antibiotic or any medicine for that matter. Always encourage good hydration and the use of analgesics such as Tylenol and short-term NSAID therapy for fever and pain. Phenazopyridine 200 mg by mouth every 8 hours for 3 days can be added to these regimens for the relief of dysuria (Gilbert et al., 2015).

Follow-Up: Follow-up test-of-cure urinalysis and urine cultures are not indicted for uncomplicated cases of cystitis (Hooton, 2012; Gupta et al., 2011; Nicolle et al., 2005). Relapse occurs when symptoms persist and the same organism is found in the culture specimen shortly after cessation of treatment. Cystitis treated with the correct antimicrobial therapy resolves 90% of the cases. Recurrence is not uncommon, though, and over 25% of the time another infection occurs within a year (Dielubanza & Schaffer, 2011). Change in treatment is warranted if the organisms are resistant to the original treatment.

Sequelae: Untreated symptomatic cystitis can lead to pyelonephritis, sepsis, shock, and death.

Prevention/Prophylaxis: Hooton (2012) outlined strategies for prevention of recurrent uncomplicated cystitis that included reduction of frequency of intercourse, abstaining from spermicidal use, and practicing good voiding habits. Prophylactic use of antibiotics by patients with indwelling catheters is not recommended (Woodford & George, 2009). For older adults who require frequent instrumentation of the lower genitourinary tract or who have frequent cystitis, suppressive antimicrobial therapy may be considered on an individual basis (AUA, 2014; Nicolle, 2005). A Cochrane review (2012) noted no significant evidence suggesting the ingestion of cranberry products would prevent UTI significantly; however, there was a noted slight stranding in a decrease of UTI in persons who were taking cranberry products. Rowe and Juthani-Mehta (2014) noted newer cranberry product formulations being introduced that showed a reduction of pyuria and bacteriuria in LTCF taking them.

Referral: In complicated cystitis that has progressed to pyelonephritis or urosepsis, consultation with a specialist is recommended for hospitalization. This recommendation can also be applied to recurrent UTI.

Education: Adequate hydration remains a cornerstone in the prevention and treatment of any infectious process. Have the patient perform timed and double voiding in an effort to lower the post-void residual of those suspected of urinary retention serving as a nidus for infection. Patients should also be instructed to complete the full course of antibiotic therapy prescribed unless notified by their provider.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Evidence does not support the routine screening of nonpregnant adults for asymptomatic bacteriuria. No benefit has been found to be derived from it.	A	Lin & Fajardo, 2008 Nicolle et al., 2005
The presence of more than 10^5 colony-forming units/mL of a single bacteria in a culture of freshly voided urine is consistent with significant bacteriuria in symptomatic subjects.	A	Beveridge et al., 2011 Juthani-Mehta et al., 2009 Nicolle, 2005
Antimicrobial selection for empirical therapy should be based on the patient's individualized history and requirements (including prior antibiotic use, symptoms, urine culture results, and institutional susceptibilities). Treatment should be reevaluated and modified as appropriate.	A	Gupta et al., 2011 Nicolle, 2005
Fluoroquinolones should not be used for first line empiric treatment of uncomplicated UTIs.	A	FDA, 2016 Hooton, 2012 Gupta et al., 2011
Follow-up test-of-cure urinalysis and urine cultures are not indicted for uncomplicated cases of cystitis.	A	Grabe et al., 2011 Gupta et al., 2011 Nicolle et al., 2005
Careful infection surveillance and special attention to special population groups (geriatrics, neurogenic bladder, etc.) in those at risk for CAUTI.	B	AUA, 2014
Consider prophylactic antimicrobial prophylaxis for recurrent cystitis and those who undergo frequent lower urinary track instrumentation.	B	AUA, 2014 Nicolle, 2005

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
In patients with complicated, unresolved, recurrent, or worsening cystitis, specialist referral is recommended. In complicated cystitis that has progressed to pyelonephritis or urosepsis, urgent evaluation and consultation with a specialist is recommended for further testing and hospitalization as appropriate.	B	Hooton, 2012 Nicolle, 2005
Urine culture is preferred but not required in the treatment of acute, uncomplicated cystitis.	B	Hooton, 2012
Cranberry products can be helpful to reduce recurrent cystitis.	B	Rowe & Juthani-Mehta, 2014 Grabe et al., 2011

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ENDOMETRIAL CANCER

Signal Symptoms: Postmenopausal bleeding.

Description: Endometrial cancer, which is the abnormal proliferation and neoplastic transformation of endometrial tissues, is the most common gynecological malignancy in the United States.

Etiology: Type I tumors, or adenocarcinoma of the endometrium, have a histological precursor of endometrial intraepithelial hyperplasia (also known as atypical endometrial hyperplasia), usually occur in the uterus, and account for 75% of endometrial cancers (Creasman et al., 2006). Type II consists of clear cell and papillary serous tumor etiologies, is considered high grade, and has a poorer prognosis. Uterine sarcomas, a third type, are unrelated to either of the other types and occur very rarely, but also have a poor prognosis.

Occurrence: Endometrial cancer represents 6% of all cancers in women; it is the most common gynecological cancer in developed countries. Although most women with endometrial cancer present with an early-stage disease and have an excellent chance of cure, in 2016 there will be approximately 10,470 deaths and 60,050 cases of uterine cancer (ACS, 2016). The lifetime probability of developing endometrial cancer for all women in the United States is 3%.

Age: Advancing age is the most important risk factor for endometrial cancer; 5% of tumors occur in women younger than 40 years old. Most tumors occur after menopause, with 63 years being the average age of diagnosis.

Gender: Endometrial cancer is limited to women.

Ethnicity: Incidence rates are higher in Caucasian women, but the mortality rate is almost twice as high in African Americans as it is in Caucasians. Asian women have the lowest rates of endometrial cancer (Allard & Maxwell, 2009; Yap & Matthews, 2006).

Contributing Factors: Obesity and diabetes (glucose intolerance) have been correlated with endometrial carcinoma. Strong evidence exists that endogenous or exogenous estrogen has a role in the development of endometrial cancer. There is a high incidence of this cancer in women with polycystic ovarian syndrome. An association exists between menstrual abnormalities and infertility and endometrial cancer. Of women with endometrial cancer, 20% to 30% are nulliparous. The use of estrogen after menopause substantially increases the risk of endometrial cancer. A program of estrogen plus progesterone for postmenopausal therapy decreases the risk. Low parity, early menarche, and late menopause have been associated with endometrial carcinoma. Use of tamoxifen in postmenopausal women is also a risk factor. Use of hormonal contraception is a negative risk factor. Women who have Lynch syndrome, which is an autosomal dominant genetic condition causing hereditary nonpolyposis colorectal cancer, are at high risk for endometrial, ovarian, colon, and other malignancies (Heald et al., 2010). They should be screened for endometrial cancer every 1 to 2 years beginning at age 35 years. Smoking is a negative risk factor for type 1 endometrial cancers, but it increases the risk for type II cancers (ACOG, 2015).

Signs and Symptoms: Postmenopausal bleeding is the most common symptom associated with endometrial carcinoma. Occasionally, an incidental finding of hyperplasia may be found on Pap testing or hysterectomy performed for other reasons.

Diagnostic Tests: A thorough history and physical examination, including a pelvic examination, is essential to rule out pelvic masses and other urogenital causes of bleeding. A rectal examination should also be done to eliminate this as the bleeding source. The diagnosis of endometrial cancer is a histological diagnosis; endometrial sampling, cytology after

hysterectomy, or curettage sample are required to be examined for an accurate diagnosis. Transvaginal ultrasound to evaluate endometrial thickness is frequently used in postmenopausal women as an initial study. If the endometrial thickness or stripe is less than 4 mm, endometrial biopsy, the gold standard for diagnosis, may be deferred (ACOG, 2015). However, if the uterine bleeding continues, a histological evaluation of the uterus should be obtained. Premenopausal women will need an endometrial biopsy; a pelvic ultrasound may be done to rule out other causes of abnormal bleeding, such as uterine leiomyomas.

Differential Diagnosis:

- Infection
- Atrophy
- Polyps
- Other causes of genital tract bleeding

Treatment: Endometrial cancer is staged from stage I (cancer is in the lining of the uterus) to stage IV (cancer has spread to other organs). The primary treatment for stage 1 cancer is surgery (total hysterectomy and bilateral salpingo-oophorectomy). This surgery also allows accurate staging of the cancer for appropriate treatment. Radiation therapy is used for women with intermediate or high risk. Hormonal therapy and chemotherapy may be used for advanced disease.

Follow-Up: Operative and histological findings assign the risk for recurrence. Most episodes of recurrence are within 3 years of treatment; this can include vaginal/pelvic areas, abdomen, or chest. Post-treatment surveillance is performed by oncology or gynecology (Sartori et al., 2010).

Sequelae: Various factors influence the prognosis, including histological differentiation, depth of invasion, and lymph node metastases. Histological type also influences the outcome.

Prevention/Prophylaxis: Do not use estrogen therapy without progesterone in the presence of an intact uterus.

Referral: All cases of suspected endometrial cancer must be referred to a gynecologist for evaluation and treatment. Endometrial sampling should be discussed with a gynecologist for women at risk for endometrial cancer.

Education: Explain the significance of postmenopausal bleeding to every woman at the time of menopause. Estrogen should not be taken without progesterone in the postmenopausal period in the presence of an intact uterus.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
All major organizations do NOT recommend routine screening of asymptomatic women by ultrasound or endometrial biopsy, unless they have Lynch syndrome.	C	Carter, 2015
All women with postmenopausal bleeding or older than 35 years with abnormal uterine bleeding should be evaluated for endometrial cancer.	C	Bode & Seehusen, 2011
First-line treatment for most women is surgical total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH-BSO), which is both therapeutic and prognostic (allows staging).	B	American College of Obstetricians and Gynecologists (ACOG), 2015
Prognosis is good for most patients, with an overall 5-year survival rate of more than 75%; prognosis for uterine sarcoma is worse.	B	ACOG, 2015

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OVARIAN CANCER

Signal Symptoms: None; most women are asymptomatic until the disease has metastasized.

Description: Cancer of the ovary is the most lethal of pelvic malignancies in women. It is actually several different types of cancer, with epithelial ovarian cancer predominating (Murphy, 2012). Within epithelial ovarian cancer are several subtypes: serous, mucinous, endometroid, and clear cell adenocarcinomas; the rest are undifferentiated and mixed epithelial carcinomas. As histological and genetic mutations are studied further, it is anticipated that valuable clinical information will be uncovered to influence outcomes (McLemore, Miaskowski, Aouizerat, Chen, & Dodd, 2009; Vaughan et al., 2011). Serous malignancies are thought to arise in the fallopian tube fimbria; clear cell and endometroid tumors may develop from endometriosis and retrograde menstruation (Murphy, 2012).

Etiology: Unknown.

Occurrence: Each year, approximately 23,000 new cases of ovarian cancer are diagnosed, making ovarian cancer the thirteenth cause of cancer deaths in the United States. Late diagnosis is the primary reason for the poor prognosis. For women in the United States, the lifetime risk for developing ovarian cancer is 1.3%. In 2016, it was estimated that 14,240 women would die of the disease (NCI, 2016). The lifetime probability of developing the disease is 1 in 55 for women in the United States (Murphy, 2012).

Age: For ovarian cancer, almost 70% of all new cases occur in women older than 55 years. Fewer than 1.3% of ovarian cancers occur in women less than 20 years old. The peak incidence of invasive ovarian cancer is age 63 years (NCI, 2016). Hereditary ovarian cancers occur in younger women.

Gender: Ovarian cancer only occurs in women.

Ethnicity: The incidence of ovarian cancer is highest in the United States, Europe, and Israel; it is lowest in Japan and in developing countries. Ovarian cancer incidence is higher in Caucasian women than in African American, Hispanic, or Asian women (NCI, 2016).

Contributing Factors: Increasing age and family history are the most important risk factors for ovarian cancer. The most significant risk factor is family history of the disease, as it is estimated that 20% of the cases are familial (PDQ Board, 2016). The risk depends on the number of affected first-degree and second-degree relatives, and their age at diagnosis with ovarian and breast cancer. This holds true for relatives on the maternal and paternal sides. Families with BRCA1 and BRCA2 mutations are at increased risk for breast and ovarian cancer. Overall lifetime risk for women with this genetic mutation for ovarian cancer is 20%. Some individuals are at risk for ovarian cancer as part of their colorectal cancer genetic risk, as with Lynch syndrome. Late menopause and early menarche may be associated with a slightly higher trend in ovarian cancer. Use of the oral contraceptive pill reduces the risk of ovarian cancer, as does pregnancy; multiple pregnancies further reduce the risk. Likewise, an increase in ovarian cancer risk among nulliparous women is reported. Obesity, and the use of talcum powder, may also increase the risk (Lederman et al., 2013).

Signs and Symptoms: Ovarian cancer may be totally asymptomatic. The woman may experience pelvic or abdominal pain or pressure, vaginal discharge or bleeding, gas/bloating, early satiety, constipation, or urinary symptoms (frequency/urgency). Because many of these symptoms are nonspecific and occur with other health conditions, they are frequently overlooked by patient and health-care professional alike (Vaughan et al., 2011). A group of four symptoms, commonly found in women with ovarian cancer, has been identified. It has been named the Ovarian Cancer Symptom Index (OCSI). These symptoms include: 1) bloating, 2) pelvic or abdominal pain, 3) difficulty eating or feeling full quickly occurring more than 12 times a month, and 4) urinary symptoms (McLemore et al., 2009).

Diagnostic Tests: A complete history and physical examination, including a pelvic examination, is recommended for all sexually active women. Because ovarian enlargement cannot always be palpated, it makes pelvic examination a limited diagnostic test. Rectovaginal examination may be necessary to detect ovarian enlargement.

Transvaginal ultrasonography is the best initial study for suspected ovarian cancer. A mass of more than 4 cm is highly suspect; necrosis or hemorrhage, nodularity, or walls of more than 3 mm in thickness also raise the index of suspicion. Laparoscopy, with subsequent confirmatory histopathological tissue report, is the gold standard diagnostic tool for ovarian cancer (Fowler, 2011). A complete blood count (CBC), cancer antigen 125 (CA-125), chest x-ray, and liver function tests (LFTs) are also part of the initial evaluation (National Comprehensive Cancer Network [NCCN], 2015).

The USPSTF recommends against screening for ovarian cancer in all women due to its low incidence. For women who have a family history of hereditary ovarian or breast cancer, the USPSTF recommends genetic counseling and BRCA testing (USPSTF, 2015).

Differential Diagnosis:

- Stool-filled sigmoid colon
- Distended bladder
- Irritable bowel syndrome
- Benign ovarian mass
- Pelvic inflammatory disease
- Peritoneal tuberculosis
- Pelvic kidney, diverticular abscess
- Cysts (dermoid, functional, cystadenoma)
- Tubo-ovarian abscess (Fowler, 2011)

Treatment: Typical treatment is surgical excision, usually a staging laparotomy, with total abdominal hysterectomy and bilateral salpingo-oopherectomy. However, extent of surgery is specific to stage and dependent on whether the cancer is primary or metastatic. For advanced cases, debulking of the tumor is done. Chemotherapy with carboplatin and taxane usually follows surgery, and there is a period of remission. Most cancers return within 1 to 4 years and eventually become resistant to the platinum-based chemotherapy. Palliative chemotherapy with gemcitabine, liposomal doxorubicin, and bevacizumab has yielded a more than 15% response (Seiden, 2012).

Follow-Up: Surveillance includes regular office follow-up, usually every 3 months for the first 2 years, with pelvic examination. Imaging and CA-125 measurement are done only if recurrence is suspected (NCCN, 2015).

Sequelae: On average diagnosis, only 15% of new cases of ovarian carcinoma have not spread beyond the ovary.

Prevention/Prophylaxis: In women who are high risk due to genetic mutations (BRCA 1 or 2), a risk-reducing bilateral salpingo-oophorectomy (RRSO) may be recommended, ideally at the age of 35 to 40 years, on completion of child-bearing (NCCN, 2015). However, once this surgery is performed, these women are at higher risk of cardiovascular events and osteoporosis due to premature menopause.

Referral: Women with a significant family history of ovarian or breast cancer should be referred for genetic counseling. Women with a significant family history should be referred to a gynecological oncologist for discussion of RRSO surgery.

Education: Routine screening for women without family history of ovarian cancer is not currently recommended. Women should also be aware of the OCSI.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Women with a family history of BRCA gene mutations should be referred for genetic counseling. They should be counseled on the risks, benefits, and limitations of genetic screening.	B	USPSTF, 2015
The best initial study for women with suspected ovarian cancer is transvaginal ultrasound.	C	PDQ Board, 2016
Surgery is required with histopathological evaluation for definitive diagnosis and staging.	A	NCCN, 2015
Chemotherapy for ovarian cancer should include a platinum-based drug and a taxane-based drug.	A	NCCN, 2015
Women with early disease at diagnosis and absence of residual tumor after treatment have better prognosis and improved survival.	A	Fowler, 2011

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BENIGN PROSTATIC HYPERPLASIA (BENIGN PROSTATIC HYPERTROPHY)

Signal Symptoms: Urinary frequency, nocturia, urgency hesitancy, weak or intermittent urine stream, straining to void, sensation of incomplete voiding, dysuria (with infection).

Description: Benign prostatic hyperplasia (BPH), also called benign prostatic hypertrophy, is the benign growth of the prostate that may lead to obstruction of the bladder outlet. Cellular elements of the prostate are involved, and dysfunction in normal cell apoptosis with excessive growth of epithelial and stromal cells within the prostate occurs (Deters, Costabile, Leveillee, Moore, & Patel, 2012).

Etiology: Originates in the transitional and periurethral zones of the prostate and is hormonally influenced by testosterone and dihydrotestosterone (DHT) production. In younger males who have BPH, there may be a genetic component (Scher & Eastham, 2015).

Occurrence: Occurs universally in older men; genetics is thought to play a part in males who develop it at a younger age.

Age: Seen in 50% of men over 50 years old and in 90% of men by age 85 years.

Gender: Occurs in men only.

Ethnicity: Not significant. Asian males are less likely to require surgery than African Americans and Caucasians.

Contributing Factors: Risk factors include male gender, age of more than 40 years, and intact testes. There is an association between obesity and diabetes and BPH.

Signs and Symptoms: Symptoms are lower urinary tract symptoms (LUTS), occur on a continuum, and do not necessarily reflect the degree of prostatic enlargement. The onset of symptoms is gradual and includes increased frequency of urination, nocturia, hesitancy, urgency, and weak urinary stream. These symptoms are not specific for BPH and progress gradually over a period of years.

The AUA has developed a self-administered symptom questionnaire that addresses the occurrence over the past month of the symptoms mentioned here. History elements should include:

- History of type 2 diabetes, which can cause nocturia
- Symptoms of neurological disease, suggesting a neurogenic bladder
- Sexual dysfunction, which is correlated with lower urinary tract symptoms
- Gross hematuria or pain in the bladder region suggestive of a bladder tumor or calculi
- History of urethral trauma, urethritis, or urethral instrumentation that could lead to urethral stricture
- Family history of BPH and prostate cancer
- Treatment with drugs that can impair bladder function (anticholinergic drugs) or increase outflow resistance (sympathomimetic drugs)

On physical examination, the prostate may be enlarged or normal sized. It should feel smooth, with a rubbery consistency. Nodularity or extreme hardness raises suspicions of malignancy. Prostate size does not correlate with degree of obstruction or severity of symptoms. The suggested rationale for this is that rectal examination is limited to palpation of the peripheral zone of the prostate and does not reach the periurethral zone where symptoms originate. In cases of advanced obstruction, the bladder may be palpated on examination. Focal neurological examination assessing the sacral nerve roots is also helpful.

Diagnostic Tests: Urinalysis is recommended to rule out infection (pyuria or bacteriuria) or malignancy (suggested by hematuria). If the urinalysis is positive for bacteria, culture and sensitivity testing is indicated. Blood urea nitrogen and creatinine should be measured if renal insufficiency or obstructive uropathy is suspected. Optional testing includes serum prostate-specific antigen (PSA), postvoid residual measurement, and maximal urinary flow rate. PSA may be elevated in BPH, prostatitis, acute urinary retention, prostatic infarction, increased physical activity, ejaculation, and prostatic cancer; the test is used primarily to screen for prostate cancer (Cunningham & Kadmon, 2016a). Specialized tests, such as IV pyelogram, transrectal ultrasound, and cystourethroscopy, are not indicated routinely. In specific instances, they may be performed as a guide to therapy choices or to rule out other conditions.

Differential Diagnosis:

- Prostate cancer (may coexist with BPH)
- Urethral stricture
- Neurogenic bladder
- UTI
- Prostatitis
- Detrusor muscle failure
- Infection
- Bladder neck contracture
- Bladder calculi
- Carcinoma of the bladder

Treatment: For patients with mild symptoms and a UAU-SI score less than 8, or men with moderate to severe symptoms who have not developed the complications of lower urinary tract symptoms or bladder outlet obstruction (renal insufficiency, urinary retention, or recurrent infection), a program of watchful waiting, with instruction to avoid medications known to worsen symptoms, is prescribed (AUA Education and Research, Inc., 2010). Medication groups to be avoided include decongestants and other sympathomimetics and anticholinergics (Cunningham & Kadmon, 2016b). Patients should also be instructed in lifestyle self-management such as limiting fluids in the evening and avoiding alcohol and caffeinated beverages, which act as mild diuretics, in addition to the drug categories mentioned previously (McVary et al., 2011). Double voiding before leaving the house may reduce symptoms.

For mild-to-moderate symptoms, alpha-adrenergic blockers such as alfuzosin (Uroxatral), doxazosin (Cardura), terazosin (Hytrin), and tamsulosin (Flomax) relax smooth muscle of the bladder neck and prostate and can increase peak urinary flow rate; all alpha-adrenergic blockers are equally effective (AUA Education and Research, Inc., 2010). Taking the medication at bedtime minimizes hypotension, the primary side effect. Dutasteride (Avodart) and finasteride (Proscar), 5-alpha reductase inhibitors, block the conversion of testosterone to dihydrotestosterone, the major intraprostatic androgen in men, and are used when prostate enlargement is identified. Side effects include decreased libido, ejaculatory dysfunction, and impotence. Treatment for 6 months or longer is needed for maximal benefit. Long-term treatment has been associated with increased risk of high-grade prostate cancer (McNicholas & Kirby, 2012). Finasteride and an alpha blocker used concurrently slow the progression of BPH; the greatest effect is seen in patients with significantly enlarged prostates (McNicholas & Kirby, 2012).

For moderate to severe BPH, surgical treatment may be the primary option. Patients with an AUA score more than 20, renal insufficiency secondary to obstruction, markedly large prostates, or acute urinary retention that must be treated with a catheter are prime candidates (Flannery, 2010). In recent years, many treatments have proliferated in this category. Transurethral resection of the prostate (TURP) has been the standard for years, with open prostatectomy for glands of more than 40 g. Newer treatments include transurethral incision of the prostate (TUIP) for gland enlargement of less than 30 g, transurethral needle-aspiration ablation of the prostate (TUNA), transurethral electrovaporization of the prostate (TVAP), transurethral or visual laser-assisted prostatectomy (TULIP/VLAP), insertion of a urethral stent, and transurethral microwave therapy (TUMT). Stents are usually reserved for complex cases. Some of the newer laser/microwave procedures may need to be repeated after several years.

Follow-Up: Scheduling of follow-up depends on the course of treatment. Advise watchful waiting and evaluate with the AUA symptoms index every 6 to 12 months. Patients receiving drug therapy should be evaluated for symptoms and side effects every 6 months. Annual digital rectal examination for prostate cancer is indicated, and usually PSA testing and urinalysis are done.

Sequelae: Complications of BPH include urinary retention and bladder outlet obstruction (BOO), requiring catheterization and/or surgery; renal insufficiency; UTIs; bladder stones; gross hematuria; prostatitis; treatment complications such as urethral stricture; treatment side effects; and possibly treatment-induced impotence.

Prevention/Prophylaxis: No preventive measures are known; BPH occurs with aging.

Referral: Patients should be referred to a urologist if comorbid conditions exist, if there is repeated hematuria or UTIs, if surgical options are indicated, and to rule out acute prostatitis or prostate cancer. Patients with BPH symptoms that do not respond to medical management should be referred for evaluation. Consider collaborative management for drug therapy.

Education: Educate the patient about BPH and its treatment options and side effects. Instruct the patient to avoid any drugs that can increase retention or cause symptoms to flare up, including caffeine, alcohol, sedatives, OTC sleeping pills, and OTC cold and allergy remedies. Instruct the patient to report hematuria, UTI symptoms, or increased retention.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Watchful waiting is an option; the need for medical treatment is based on symptoms (AUA symptom score 3) and the degree of patient bother.	C	AUA (reaffirmed 2014)
Alpha-adrenergic antagonists are equally effective and work rapidly; patients can expect moderate improvement in symptoms.	A	McNicholas & Kirby, 2012
5-Alpha reductase inhibitors modestly improve AUA symptom score and reduce the risk of acute urinary retention or need for invasive surgery.	A	McNicholas & Kirby, 2012
Combination therapy with an alpha blocker and a 5-alpha reductase inhibitor is superior to monotherapy, particularly in patients with higher prostate volumes and prostate-specific antigen (PSA) levels.	B	McNicholas & Kirby, 2012

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DRUG-INDUCED ERECTILE DYSFUNCTION

Signal Symptoms: None; noncompliance with medication regimen, responses on sexual history query or questionnaire.

Description: Erectile dysfunction (ED), previously known as impotence, is the consistent inability to achieve and maintain erection sufficient for sexual intercourse for at least 3 months (Bella, Lee, Carrier, Benard, & Brock, 2015) In a broader sense, ED encompasses problems with arousal, libido, orgasm, sensation, and relationships. Drug-induced ED refers to that which is caused by a drug or drugs (Cunningham & Khera, 2016).

Etiology: Drug-induced ED may be caused by many medications or medication interactions (see Contributing Factors).

Occurrence: An estimated 20 to 30 million U.S. men experience ED on a chronic basis.

Age: Incidence of ED increases with age. The prevalence is approximately 52% for men between ages 40 and 70 years; in men over 70 years old, prevalence is increased further. Men over 70 years old with chronic medical problems, such as diabetes, have a prevalence of more than 90%. Because the condition frequently is underreported, the percentage is probably higher. Approximately 25% of erectile problems are related to drugs (McVary, 2015).

Gender: Occurs in men only.

Ethnicity: Not significant.

Contributing Factors: The following factors contribute to ED (Cunningham & Khera, 2015a; Heidelbaugh & Barry, 2016; Tacklind, Fink, MacDonald, Rutks, & Wilt, 2012):

- Stress
- Long-term alcohol use
- Tobacco use
- Recreational drug use (cocaine, marijuana, opiates)
- Antiandrogens
- Anticholinergics
- Anticonvulsants
- Antidepressants (including selective serotonin reuptake inhibitors, monoamine oxidase inhibitors, tricyclic antidepressants)
- Antipsychotics
- Centrally acting depressants (sedatives/hypnotics, tranquilizers, opiates)
- H_2 blockers
- Interpersonal conflict
- Levodopa
- Lithium
- Stimulants such as amphetamines
- Beta blockers
- Spironolactone
- Methadone
- Cytotoxic agents
- Antihypertensives (clonidine, methyldopa, thiazides, calcium channel blockers)
- Urologic drugs (alpha blockers)
- Anti-inflammatories (baclofen, naproxen)

Various processes are involved, including effect on libido, neurochemical mediation, and drug side effects.

Signs and Symptoms: There may be no reported symptoms unless the provider includes a sexual history, is aware of the potential for ED when prescribing certain medications, and asks about baseline sexual activity at that time, then periodically reassesses this during follow-up visits. The patient should be asked about sexual interest, sexual ability, and

sexual activity. Also, if a provider detects noncompliance to a prescribed medication regimen in an otherwise cooperative patient, the provider should inquire whether ED occurs when the medication is taken. Drug-induced ED can be associated with starting a new medication.

Medication history, including OTC remedies, alcohol use, tobacco use, and use of recreational drugs is also important. The CAGE test for alcoholism, a screening questionnaire for depression, or other psychological screens as needed may be included as part of the history. A history of surgical procedures, especially of procedures involving the prostate, bladder, or colorectal area, and including the lymphatic channels, may reveal potential sources of impotence (Cunningham & Khera, 2016a).

Physical examination is performed to rule out other causes of ED. The cause may be multifactorial, especially in an older patient. Evaluate the patient's overall appearance and mobility, then assess the vital signs, specifically checking for orthostatic hypotension. Palpation of the thyroid may reveal a goiter. Gynecomastia may be related to certain drugs, such as cimetidine. Abdominal or femoral bruits may highlight an abdominal aortic aneurysm or vascular obstruction at the bifurcation of the abdominal aorta.

Diminished peripheral pulses suggest a circulatory problem. Lack of sensation or inability to discriminate between sharp and dull may indicate peripheral neuropathy, especially if the history is positive for diabetes. Abnormal reflexes point to a neurological problem. Decreased mobility may indicate a neurological or musculoskeletal problem contributing to ED. Examination of the genital area may reveal testicular atrophy or penile plaques, as seen in patients with Peyronie's disease, or an enlarged prostate. Assess for the bulbocavernosus reflex; absence of this reflex indicates penile neuropathy. Some men using 5-alpha-reductase inhibitors for BPH or male pattern baldness experience a decreased libido, ED, and/or ejaculatory problems (Cunningham & Khera, 2016b).

Diagnostic Tests: Diagnostic testing is not indicated specifically for drug-induced ED, but is performed to eliminate other possible causes of impotence. ED is not always a sexual health issue and other causes may require exploration. Diagnostics may include (Cunningham & Khera, 2016a):

- Fasting glucose and hemoglobin A1c
- CBC
- Comprehensive metabolic profile
- Thyroid-stimulating hormone
- Lipid profile
- Serum total testosterone
- Serum prolactin

Differential Diagnosis:

- Vascular
- Endocrine
- Neurological
- Neurovascular
- Substance abuse
- End-organ disease
- Psychogenic
- Social causes

Treatment: Whenever possible, decrease the dose, eliminate the medication, or substitute another medication from available drugs that do not cause ED. If this is not possible, or if the patient continues to experience the problem after these changes have been made, consider other treatment measures. Adding a second drug while continuing the original drug may be effective. Counsel the patient regarding alcohol, recreational drug, and tobacco use (Cunningham & Khera, 2016b).

Phosphodiesterase-5 inhibitors may be considered once cardiac risk factors, chronic illness, medication assessment, and underlying etiology are examined. Medications in this classification include sildenafil citrate (Viagra), vardenafil (Levitra), tadalafil (Cialis), and avanafil (Stendra), and are selective inhibitors of cyclic guanosine monophosphate (cGMP)–specific phosphodiesterase type 5. Tadalafil has a longer duration and avanafil has a rapid onset. During sexual stimulation, nitric acid is released into the corpus cavernosum, resulting in increased cGMP levels, which prevents smooth muscle relaxation and increases blood flow to the penis. Phosphodiesterase-5 inhibitors enhance this effect by inhibiting an enzyme that degrades cGMP in the corpus cavernosum. These medications are contraindicated with nitrates and in advanced heart disease; cytochrome influences include increased plasma levels with inhibitors of CYP3A4 or CYP2D9 and decreased plasma levels with inducers of CYP3A4. Side effects include headache, flushing, UTI, abnormal vision (blue-green, blurring, photosensitivity), nasal congestion, diarrhea, dizziness, and rash. PDE-5 inhibitors are effective in treating drug-induced ED in patients taking antidepressants/antipsychotics and antihypertensives/diuretics (Bella et al., 2015; Cunningham & Khera, 2016b). Phosphodiesterase-5 inhibitors were also found to be effective in treating ED related to prior cancer therapy (Miles et al., 2011). For other types of treatment, such as penile implants or injection, refer the patient to a urologist specializing in this area.

Follow-Up: Individualize follow-up according to cause and treatment. Periodic reassessment of treatment for recurrence of the problem is helpful. For medication treatment, monitor for side effects and response.

Sequelae: Disruption of sexual function may result in depression or relationship problems. Complications may arise from treatment.

Prevention/Prophylaxis: Whenever possible, avoid prescribing medications with a high risk for causing ED, and include a sexual history in routine history and physical examination whenever prescribing a new medication with ED as a potential side effect.

Referral: Refer patients to a urologist to evaluate for other causes or multifactorial causes of ED. Refer patients who are experiencing the problem to a support group. Refer for psychological services when indicated.

Education: Teach the patient about medication side effects and that they cannot be used in conjunction with nitrates. Have the patient report sexual dysfunction. Educate the patient about normal age-related changes in sexual functioning and how to adapt.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCE
There is no recommended diagnostic testing to serve as a gold standard for ED; history and physical examination are sufficient in making an accurate diagnosis in the majority of cases.	C	Heidelbaugh & Barry, 2016 McVary, 2015
The PDE5 inhibitors are the most effective drugs in the treatment of ED, including ED associated with diabetes and spinal cord injury, and in men with sexual dysfunction secondary to antidepressants.	A	Bella, Lee, Carrier, Benard, & Brock, 2015 Cunningham & Khera, 2016b

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PROSTATE CANCER

Signal Symptoms: Early stage often asymptomatic. Obstructive voiding symptoms (hesitancy, intermittent urinary stream, decreased force of stream) are generally indicative of advanced disease.

Description: Prostate cancer is a malignant neoplasm of the prostate, which is a male sex accessory gland. The prostate has been equated to the size of a walnut, encircling the urethra like a doughnut. The role of the prostate is to secrete fluid into the ejaculate that accompanies sperm and seminal fluid to make up semen. Ninety-five percent of prostate cancers are adenocarcinomas.

Etiology: Approximately one-half of prostate cancers demonstrate a genetic component. Testosterone has been identified as a prerequisite for the development of prostate cancer. Prostate cancers exhibit high levels of androgen receptors (Small, 2012).

Occurrence: Prostate cancer is the leading cause of cancer death in men (Mulhelm, 2015), second only to lung cancer (Siegel, 2016). Prostate cancer is the most common cancer in men, with approximately a 17% lifetime risk (Ballentine Carter, 2013; Mulhelm, 2015). The older adults are more frequently affected, with 60% of cases diagnosed after age 65 years; 70% of deaths are in men older than 75 years (Mulhelm, 2015).

Age: Mean age at diagnosis is 71 years.

Gender: Prostate cancer is a disease confined to men.

Ethnicity: African Americans have the highest incidence of prostate cancer in the world, with Asian and Hispanic men at lower risk than Caucasian men (ACS Facts & Figures, 2016). Diagnosis in African Americans tends to be at a more advanced stage, and disease-specific survival is lower in this group (ACS Cancer Facts & Figures for African Americans, 2016–2018).

Contributing Factors: Age greater than 50 years, African American race, diet high in fat. Family history appears significant with a 2.5-fold increased risk with a first-degree relative affected before age 50 years, and even greater if the affected relative is a brother rather than a father, if the affected relative is younger than 55 years, or if two or more first-degree relatives are affected. No link has been determined with prior vasectomy or BPH (ACS Prostate Cancer, 2016).

Signs and Symptoms: There are usually no symptoms with early disease. Symptoms generally occur in advanced stages, making early detection desirable. Obstructive voiding symptoms (hesitancy, intermittent urinary stream, decreased force of stream) generally reflect locally advanced disease with growth into the urethra or bladder neck. Locally advanced tumors may result in hematuria and hematospermia (Small, 2012). If rectal obstruction occurs, a large bowel obstruction or difficulty in defecation may be present (McCance, Huether, Brashers, & Rote, 2014). Prostate cancer that has spread to the regional pelvic lymph nodes occasionally causes edema of the lower extremities or discomfort in pelvic or perineal areas. Metastasis occurs most often to the bone, resulting in pain and pathological fractures (Small, 2012).

Diagnostic Tests: Screening discussion should be held with men around the age of 50 years who do not possess comorbidities that restrict life expectancy to less than 10 years, unless risk factors dictate earlier. Controversy continues in terms of screening recommendations. The following table reflects current screening statements by expert groups and the date declared.

GROUP / SOCIETY	RECOMMENDATION
American Academy of Family Physicians (2012)	Do not perform PSA-based screening for prostate cancer.

GROUP / SOCIETY	RECOMMENDATION
ACS (2010)	• Beginning at 50 years of age, asymptomatic men who have at least a 10-year life expectancy should have an opportunity to make an informed decision with their physician about screening. • Men at higher risk, including African American men and men who have a first-degree family history (father or brother) of prostate cancer diagnosed before 65 years of age, should receive this information beginning at 45 years of age. • When screening is performed, PSA testing should be used with or without digital rectal examination. Frequency of testing depends on PSA level.
American College of Physicians (2013)	• Inform men between 50 and 69 years of age about the limited potential benefits and substantial harms of screening. • Do not screen men who do not express a clear preference for it. • Do not screen men older than 69 years or men with a life expectancy of less than 10 to 15 years.
AUA (2013)	• Use shared decision making for men 55 to 69 years of age. • Individualize screening decisions for higher-risk men 40 to 54 years of age. • Do not screen men younger than 40 years, older than 70 years, or who have a life expectancy of less than 10 to 15 years.
European Urological Association (2013)	Baseline PSA is recommended for men 40 to 45 years to initiate a risk-adapted follow-up approach with the purpose of reducing prostate cancer-mortality and the incidence of advanced and metastatic prostate cancer.
U.S. Preventive Services Task Force (2012)	Do not perform PSA-based screening for prostate cancer.

Digital rectal examination (DRE) is the only method for physically examining the prostate, with awareness that only part of the gland can be palpated, allowing for irregularities to be missed. DRE is considered abnormal if the prostate is enlarged, asymmetrical, nodular, or tender (Swartz, 2014). The effectiveness of DRE for prostate cancer screening is not well established and is considered to have low sensitivity (Ballentine Carter, 2013). Screening with PSA and DRE may detect cancer at an earlier stage than if no screening is performed (Ballentine Carter, 2013).

PSA is a glycoprotein expressed by normal and neoplastic prostate tissue and is excreted in the ejaculate to liquefy semen (McCance & Heuther, 2014). Escape of PSA in large amounts into the bloodstream occurs when the basement membrane of the prostate epithelial cells is disrupted. Both the PSA and DRE may produce false-positive or false-negative results. A PSA level of 4.0 ng/mL or greater historically has been used to recommend referral for further evaluation or biopsy, which remains a reasonable approach for men at average risk for prostate cancer. Five-alpha reductase inhibitors finasteride and dutasteride are known to lower PSA levels (Hoffman, 2016). Transurethral ultrasonography with biopsies is indicated when the PSA level is elevated, when the percentage of free PSA is less than 25%, or when an abnormality is noted on DRE. When biopsy is undertaken, up to six biopsies on each side are preferred for evaluation. Seminal vesicles may be sampled in high-risk patients (Small, 2012).

Differential Diagnosis:

- Benign nodule prostate growth
- Subacute prostatitis
- Prostatic intraepithelial neoplasia
- Prostate stones

Treatment: Treatment depends on stage of the tumor, anticipated effects of treatment, and the age, general health, and life expectancy of the patient. The Gleason score determines the grade of the prostate cancer. The Gleason score grades tumors on a 1 to 5 range based on the degree of glandular differentiation and structural architecture. Grade 1 represents the most well-differentiated appearance, and grade 5 represents the most poorly differentiated. The International Society of Urologic Pathologists (2014) adopted a new grading system that includes a modified Gleason score. This new grading system is often reported in conjunction with the Gleason score on a pathology report and offers a more accurate assessment of risk (Yang, 2016). Treatment options include active surveillance for a tumor of incidental histological findings, to radical prostatectomy, radiation, or androgen deprivation therapy.

Follow-Up: Follow-up depends on current treatment and patient-specific risk. It may be best shared by the cancer specialist and the primary provider. Routine history and physical examinations, cardiovascular health screenings, surveillance for diabetes, depression screening, annual quality of life assessment, and evaluation and management of any sexual dysfunction should be considered.

- PSA: Every 6 months for next 5 years (Skolarus, 2014). If PSA is elevated after treatment, refer back to cancer specialist.
- DRE: Annually after radiation. Discontinue after radical prostatectomy if PSA undetectable (Skolarus, 2014).

For patients with radical prostatectomy, any PSA greater than 0.2 ng/ml is abnormal and raises concern for recurrence or progressive cancer. For patients with radiotherapy who previously had a low PSA, a rising PSA, particularly above 2, indicates recurrence. A "PSA bounce" is described in men who were treated with brachytherapy or external beam radiation who experience an increase in PSA up to 30 months following the end of treatment (Skolarus, 2016).

Sequelae: Complications of treatment may include incontinence, ED, mild colitis, and radiation cystitis. After nerve-sparing prostatectomy, urinary continence returns in under 6 months in about 50% of men. The rate of ED after external beam radiation ranges from 10% to 80%, and with brachytherapy approximately 15% to 60%.

Prevention/Prophylaxis: PSA screening remains controversial because many of the prostate cancers detected are low grade and slow growing. The USPSTF's (2012) latest

recommendation is against screening for prostate cancer, with the AUA (2013) opting for PSA testing after a discussion with shared decision making. The ACS (2010) recommends that asymptomatic men who have at least a 10-year life expectancy have an opportunity to make an informed decision with their health-care provider about whether to be screened for prostate cancer. Men at average risk should receive this first information beginning at age 50 years. Men at higher risk, including African Americans and men with a first-degree relative (father or brother) diagnosed with prostate cancer before age 65 years, should receive this information at age 45 years. Men with an appreciably higher risk (multiple family members diagnosed with prostate cancer before age 65 years) should receive this information at age 40 years. Asymptomatic men with less than a 10-year life expectancy based on age and health status should not be offered prostate cancer screening. Screening, when completed, is recommended with PSA with or without DRE.

Referral: Appropriate referral sources may include urology, oncology, and radiology.

Education: Review risk factors and significance of reporting family history. Encourage screening per health-care providers' recommendations and current guidelines.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Informed decision making after shared discussion of risks and benefits of screening.	C	ACS, 2016
Health-care providers should periodically discuss prostate cancer screening with men who are expected to live at least 10 years and are old enough to be at significant risk for prostate cancer.	B	AUA, 2013
When screening, perform with PSA testing every 2 to 4 years.	B	Hoffman, 2016
Stop screening after age 69 or earlier if comorbidities limit life expectancy to less than 10 years.	C	Hoffman, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

PROSTATITIS

Signal Symptoms: Patients may appear acutely ill with fever, chills, and malaise. Myalgia, cloudy urine, and urinary symptoms such as dysuria, frequency, and urgency may also occur (Meyrier & Fekete, 2016). Sexual dysfunction, psychological issues, and urogenital pain may occur in chronic cases (Rees, Abrahams, Doble, and Cooper, 2015).

Description: Prostatitis includes both acute and chronic bacterial prostatitis and prostatitis syndrome, also termed chronic pelvic pain syndrome (CPPS). The National Institutes of Health Consensus Classification of Prostatitis identifies four categories of prostatitis based on clinical presentation: type I, acute bacterial prostatitis; type II, chronic bacterial prostatitis; type III, CPPS with either type IIIA inflammatory components or type IIIB noninflammatory components; and type IV, asymptomatic prostatitis. Acute bacterial prostatitis is an acute bacterial infection of the prostate and, if left untreated, it can progress to sepsis or development of prostatic abscess. By definition, an organism must be identified on culture. Chronic prostatitis is defined by symptoms being present for at least 3 months and urine cultures obtained over the course of the illness repeatedly growing the same bacterial strain. CPPS is pelvic pain in the absence of bacteria localized to the prostate (Gill & Shoskes, 2016).

Etiology: Most common pathogens in prostatitis include *Escherichia coli, Klebsiella, Proteus mirabilis, Enterococcus faecalis,* and *Pseudomonas aeruginosa* (Gill & Shoskes, 2015; Meyrier & Fekete, 2016). *E. coli* is the most commonly isolated organism in chronic prostatitis, and these strains have been found to have a higher virulence factor and greater degree of biofilm formation, making this form more difficult to treat (Meyrier & Fekete, 2016).

Occurrence: Prevalence of prostatitis is approximately 8% (Rees et al., 2015). Some degree of prostatic inflammation is present in 4% to 36% of the male population (McCance et al., 2014).

Age: Bimodal incidence peaks between 20 and 40 years, and older than 60 years (Gill & Shoskes, 2016).

Gender: Occurs in men only.

Ethnicity: Not significant.

Contributing Factors: History of UTI, cystitis, urethritis, functional or anatomical anomalies.

Signs and Symptoms: The predominant symptom is pain in the prostate, perineum, scrotum, testes, penis, urinary bladder, or lower back. Acute bacterial prostatitis frequently

includes symptoms similar to acute cystitis or pyelonephritis and includes malaise, low back and perineal pain, high fever, chills, dysuria, inability to empty the bladder, nocturia, and urinary retention. Men are acutely ill and may look toxic (McCance et al., 2014). Chronic bacterial prostatitis is the most frequent cause of recurrent UTIs in men. Symptoms are variable and are similar to those of an acute bladder infection, such as frequency, urgency, dysuria, perineal discomfort, low back pain, and urinary dysfunction (McCance et al., 2014). These men often do not appear ill looking but present with recurrent or relapsing UTIs, urethritis, or epididymitis with the same bacterial strain (Rees et al., 2015).

Physical examination may reveal a tender, boggy prostate on rectal examination. The prostate gland may be warm, swollen, firm, or irregular. Prostatic massage is contraindicated, as it increases the risk for bacteremia, is very painful, and has no benefit (Meyrier & Fekete, 2016). Patients may also complete the National Institutes of Health Chronic Prostatitis Symptom Index assessing pain, urination, impact of symptoms, and quality of life at www.prostatitis.org/symptomindex.html.

Diagnostic Tests: Diagnosis of acute bacterial prostatitis is often done on report of symptoms. Suspected acute prostatitis diagnostics include urinalysis, urine and blood cultures, urine Gram stain, and CBC with differential. Urine should be a midstream collection, and the presence of more than 10 white blood cells (WBCs) per high-power field suggests a positive diagnosis. Suspected chronic prostatitis/CPPS tests include urinalysis, urine culture, and postvoid residual, if sensation of incomplete emptying of bladder. Suspected chronic bacterial prostatitis testing includes two-glass preprostatic and postprostatic massage test and a positive urine culture. In most cases, imaging is not required. Order computed tomography (CT) scan or MRI if malignancy or abscess is suspected. Transrectal ultrasound is indicated if prostatic calculi or abscess is suspected. Sexually active men younger than age 35 years and older men who engage in high-risk sexual behavior should be tested for *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. Measurement of free and total PSA would contribute to diagnosis of exclusion of prostate cancer (Rees et al., 2015).

Differential Diagnosis:

- Acute cystitis: rapid onset and rapid response to antimicrobial therapy
- Urethritis: clinical diagnosis, mucopurulent or purulent urethral discharge, Gram stain for WBC count
- Pyelonephritis: usually results from an inadequately treated ascending infection; positive costovertebral angle tenderness, may have hematuria, may have abdominal pain; more common in younger men and pregnant women
- Acute urinary retention: physical assessment of distended bladder, immediate catheterization to alleviate
- Benign prostatic hyperplasia (see BPH in this chapter)
- Urinary tract stones: acute, crampy pain on urination; sludge or gravel may be seen in urine specimen, especially if specimen is strained; may be accompanied by nausea, vomiting
- Bladder cancer
- Prostatic abscess
- Enterovesical fistula
- Foreign body within the urinary tract (see Urinary Stones) (Bope & Kellerman, 2017)

Treatment: Antibiotics are indicated in acute bacterial prostatitis and recommended in chronic bacterial prostatitis with empiric therapy started while awaiting any culture (Meyrier & Fekete, 2016). If the patient is acutely ill with acute bacterial prostatitis, the health-care provider may need to consider hospitalization and parenteral antibiotics (Prostatitis, 2012). Fluoroquinolones such as ciprofloxacin (Cipro) 500 mg twice a day or levofloxacin (Levaquin) 500 mg daily are recommended. They offer good activity against typical and atypical bacterial strains in addition to *P. aeruginosa,* and both penetrate the prostate tissue well (Meyrier & Fekete, 2016).

Oral medications are recommended for 10 days for acute cases. Trimethoprim/sulfamethoxazole (Bactrim, Septra) may be considered, but drawbacks include no activity against *P. aeruginosa*, poorer tissue penetration, and increasing resistance in some geographical areas. In chronic cases, antibiotics are given for 2 weeks, then the patient is reassessed and antibiotics continued only if cultures are positive or the patient reports positive effects from the treatment. A total treatment period of 4 to 6 weeks is recommended, and a 6- to 12-week course may be needed to eradicate the causative organism and to prevent recurrence (Rees et al., 2015). Patient may need to be evaluated for need for stool softeners if painful or difficult defecation. Evaluate the need for analgesics, antipyretics, or anti-inflammatory medications, and warm water baths.

Follow-Up: For patients with acute bacterial prostatitis, a repeat urine culture after the treatment regimen is recommended. Patients with chronic prostatitis or relapse should receive extended antimicrobial therapy. Refer patients with symptoms of BPH, intractable infection, or prostatodynia to urology.

Sequelae: Unresolved acute prostatitis can lead to bacteremia or pyelonephritis. Prostatic abscess rarely occurs as a complication of acute bacterial prostatitis except in immunocompromised patients.

Prevention/Prophylaxis: There are no prevention strategies for prostatitis.

Referral: Refer patients with symptoms of BPH, intractable infection, or prostatodynia to a urologist.

Education: Educate the patient and family regarding the cause of symptoms and treatment. Explain the need for extended antibiotics and importance of adherence with treatment. Encourage follow-up appointments as recommended. Condoms should be worn to prevent reintroduction of bacteria into the urethra during sex, and anal intercourse should be avoided with acute bacterial prostatitis. Patients should be advised to avoid bladder irritants such as alcohol and caffeine. Emphasize that prostatitis can recur and to report symptoms at onset.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Antimicrobial therapy should be guided by bacterial cultures and sensitivities for chronic bacterial prostatitis.	B	Rees, Abrahams, Doble, & Cooper, 2015
Optimal duration of antibiotic treatment for acute bacterial prostatitis is 4 to 6 weeks.	C	Rees, Abrahams, Doble, & Cooper, 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

Mrs. C., a 66-year-old woman, presents to your practice with a complaint of recent onset of leaking urine. She has no prior history of this. On further questioning you discover that she is newly married, after being widowed for 15 years, during which time she had no sexual partners. She and her new husband have just returned from a cruise, and she reports that he is a "passionate" lover and she is having difficulty meeting his needs for daily intercourse. Other symptoms that Mrs. C. reports are vaginal burning and general discomfort "down there."

Medications: Takes fish oil at bedtime, vitamin D_3 1,000 IU daily. No known drug allergies.

Vital signs: Blood pressure (BP) 128/76 mm Hg, pulse 82 (regular), respiratory rate 16 breaths/min, temperature 98.2°F (oral), BMI 23.4.

A staff member has obtained a urine specimen for dipstick. Findings are as follows:

Nitrite—negative

Leukocyte esterase—negative

Blood—trace

Protein—trace

Glucose—negative

Ketones—negative

Specific gravity—1.015

1. What additional subjective data are you seeking?
2. What additional objective data will you be assessing for?
3. What national guidelines are appropriate to consider?
4. What tests will you order?
5. Are there any screening tools that you want to use?
6. What are the differential diagnoses that you are considering?
7. What is your plan of care?
8. Are there any *Healthy People 2020* objectives that you should consider?
9. What additional patient teaching may be needed?
10. Will you be looking for a consultation?

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CHAPTER 12

Musculoskeletal Disorders

Laurie Kennedy-Malone

ASSESSMENT

To make an accurate assessment of the musculoskeletal system in the older adult, the nurse practitioner needs to be familiar with changes in the musculoskeletal system caused by aging. For example, aging often brings about a decrease in height, resulting from a decrease in the length of the trunk with respect to the length of the extremities. An older person may tilt the head backward to compensate for the bend in the thoracic spine, producing the typical posture of those in this age group. Because of the loss of subcutaneous fat caused by aging, bony prominences became more noticeable. Some absolute loss of muscle mass occurs, with some muscles diminishing and others atrophying. Without continued use, muscles stiffen and range of motion (ROM) becomes impaired as an older person ages (Manini, Gundermann, & Clark, 2016).

Often when an older person experiences a limitation in functional ability resulting from pain, weakness, or physical impairment, he or she seeks medical attention. A goal of the nurse practitioner in gathering the subjective information from the older adult is to try to determine if there is a pattern of symptoms (Browne & Merrill, 2015).

To obtain an accurate history of musculoskeletal problems, ask the patient these questions:

- Is there any pain, swelling, stiffness, change in temperature perception (hot or cold), limitation of movement, weakness, body deformity, or paralysis?
- Are the symptoms constant or intermittent?
- What was the sequence of events that triggered the onset of each symptom and what are the precipitating factors? (Browne & Merrill, 2015)
- Is the muscle, joint, and/or periarticular signs and symptoms related to an accident, illness, or prior trauma?
- Is there a family history of chronic musculoskeletal conditions?
- Review occupational and hobby history to determine an underlying contributing factor to pain and gross deformities.
- Is there a history of obesity?
- Is there a trigger to the complaint such as lack of mobility, temperature, or position (as in sleeping)? Often the patient complains of one or more of these symptoms; however, the condition may actually be a neurological condition (e.g., Parkinson's disease) or a systemic disorder (e.g., thyroid disease). Additionally, patients may be experiencing a new musculoskeletal condition concomitant with an existing condition, such as a new onset of gout with pre-existing osteoarthritis (OA) of the interphalangeal joints.

In addition, the nurse practitioner should:

- Determine if the condition has been triggered by medications such as statins, with the known side effect of myalgias (Gillet & Norrell, 2011), or steroids, which can contribute to bone loss.
- Briefly review dietary history to determine pattern on calcium intake.
- Be alert for other symptoms the patient may describe; for example, constitutional symptoms of fever and malaise may be clustered to form a differential diagnosis (Resnick, 2016).
- Review prior therapy and results, determining what the patient has used in the past to alleviate symptoms to include herbal products such as chondroitin, use of complementary or alternative treatments such as heat or cold, massage, ultrasound, and/or acupuncture or pressure.

Pain or Stiffness

Initially, the nurse practitioner will need to determine if the presenting symptoms arise from the joint, tendons, muscles, or periarticular structures, such as bursae. Further questioning needs to help distinguish whether the condition is acute or chronic, to include an exacerbation of a former condition that had been deemed in remission or resolved. Is the patient experiencing an inflammatory or noninflammatory condition? It is important to remember that both can coexist in patients. Most importantly, determine how the pain and stiffness is affecting function and quality of life (Browne & Merrill, 2015).

When a patient complains of pain or stiffness with movement, the history of the complaint should be discerned. Ask if the patient has experienced any severe trauma in the past that may be now manifesting itself as an articular

degeneration. Because patients who have had a structural deformity or amputation typically place excessive strain on the joints for years, as older individuals they may now experience degeneration of the bone and surrounding musculature. An overextension or a recent increase in activity may lead to muscle soreness, followed by disuse, atrophy, and chronic pain. Adhesive capsulitis or frozen shoulder is a common condition in patients experiencing pain in the shoulder and, in turn, limiting their ROM. Knowing the history of the presenting symptom may help you distinguish between a local inflammation and a systemic problem. Questioning the patient about the time of day when the symptoms of pain and stiffness occur can be helpful too. Rheumatic disease, for example, is associated with pain on waking, whereas osteoarthritic pain and stiffness worsen as the day progresses. It is important to remember that within the current cohort of older adults a stoic attitude toward pain may be displayed despite the presence of an acute or chronic musculoskeletal condition (Abdulla et al., 2013).

Weakness and Paralysis

Patients with musculoskeletal problems often complain of weakness and paralysis. Determine whether the disability is local or generalized, constant or intermittent. Local weakness or paralysis may be due to disuse because of pain, trauma, or a neurological problem. Generalized weakness may indicate a systemic disorder or recent deconditioning related to extensive immobility or frailty, as in the case of patients with sarcopenia (Brown & Clark, 2016). Patients with an underlying thyroid disorder may have constant weakness, whereas those with rheumatoid arthritis (RA) may experience weakness intermittently (Resnick, 2016).

Muscular weakness should always be differentiated from subjective fatigue. Generally, proximal weakness results from a myopathy and distal weakness from a neuropathy. The patient should be asked about his or her ability to carry out certain activities of daily living (ADLs), such as:

- Does the patient have difficulty combing his or her hair?
- Can he or she lift objects?
- Does the patient have any trouble standing up after sitting in a chair?

Patients with a proximal weakness of the upper extremities may have difficulty grooming the hair or lifting objects. Those with a proximal weakness of a lower extremity may have trouble crossing the legs at the knees or walking. If a patient reports difficulty fastening buttons or turning doorknobs, a neuropathy involving the upper extremity is likely.

Deformity

Another common complaint in patients with a musculoskeletal disorder is deformity. In obtaining the history from a patient with a deformity, the examiner needs to know how long the patient has noticed the deformity:

- Did it occur suddenly?
- Was it the result of trauma?
- Has there been any change in the deformity since its onset, such as a new onset of pain, swelling, or erythema?
- Is there a family history of similar nodularity?

Physical Examination

Focus of the physical examination of the musculoskeletal system includes inspection, palpation, ROM, and joint special maneuvers. Observe for any noted asymmetry, atrophy, wasting, masses, and erythema of the muscles. Examine the skin specifically for any lesions or change in color that may be related to an acute or chronic musculoskeletal condition. Ecchymosis in the area of a joint may be indicative of tendon rupture. Joints and surrounding structures should be examined to determine presence of any deformities, nodularities, subluxation, abnormal angulation, limitation of movement, stability, and malalignment. Palpation may reveal swelling, change in temperature, tenderness, and crepitus (Williams, 2008). Engage the patient in active ROM of joints necessitated by the specific complaint. Specific provocative maneuvers related to pathology occurring in that affected joint(s) is warranted during a focused examination.

GAIT

The initial physical examination of the musculoskeletal system can begin with observing the patient's gait. Overall assessment of gait includes an extensive examination of the lower extremities to include podiatric issues (examining patient's feet). Any gait abnormalities or problems in maintaining balance should be noted. Assessment of gait needs to consider the patient's stance and arm swing (Salzman, 2010). Observe for any limping or asymmetrical leg movement or noted deformities. An abnormal stance is referred to as an antalgic gait, often resulting from chronic hip pain and muscle atrophy. Note if the patient is having difficulty in shifting weight from one leg to the other and instead relies on shoulder movement from side to side. This abnormality in arm swing is referred to as a lurch (Salzman, 2010).

Another important aspect of the gait examination is to note the patient's ability to sit in and rise from a chair. If a patient relies on a rocking motion and/or arm strength to stand, consider quadriceps weakness. More sophisticated testing is warranted if the patient demonstrates difficulty in performing this task, because he or she could be at a high risk for falling (Salzman, 2010).

POSTURE

Note the patient's posture for any abnormalities of the trunk and spine. Examine the patient while the patient is standing erect to note any changes in the spinal curvature such as scoliosis. In older adults, kyphosis of the thoracic spine is accentuated. While looking at the older person from the side, the examiner may note that the spine appears to form the number 3. If the curve appears more to be a sharp angle, this is called a gibbus. A gibbus resulting from a vertebral compression fracture may be the first evidence of osteoporosis (Williams, 2008).

RANGE OF MOTION

Patients presenting with a functional limitation should be asked to demonstrate active ROM, which should be performed smoothly and effortlessly. Remember that joint movements include flexion, extension, abduction, adduction, and external rotation. If the patient has limited active ROM, passive ROM should be performed. If the passive range exceeds the active range, the limitation is due to a muscle weakness.

Patients may resist passive ROM because of fear, a neurological disorder, or a joint abnormality.

JOINT PAIN

When patients complain of joint-related pain, the practitioner needs to determine if the underlying condition is articular, periarticular, or both. When examining the patient's joints, the joints should be inspected in a relaxed position and then in flexion and in extension. Abnormalities of the position or carrying angle, joint deformity, erythema, swellings, nodulation, and muscle changes should be assessed. In an older patient with muscle changes, the limb or portion of the body will appear thinner, which indicates atrophy. To determine whether bone structure changes have occurred, appearance and symmetry should be observed. Enlargement, excessive curvature, and irregularity may indicate sequelae of childhood rickets, osteoporotic fractures, Paget's disease, OA, tophaceous gout, or bone tumors. Crepitation or a crackling noise may be heard when the joints are put through ROM. Crepitation is produced by the rubbing together of bone or irregular cartilage surfaces (LeBlond, Brown, Suneja & Szot, 2015).

JOINT SWELLING AND NODULARITY

Specific questions about the duration of any swelling, presence of pain, limitation of movement, evidence of erythema, and locking or buckling of the joint are important to assess. Erythema is usually associated with active inflammation and accompanied by swelling. Examples of joint nodulation are the nodules found in the interphalangeal joints in patients with RA and OA. Heberden's nodes are nodules of the distal interphalangeal joints; Bouchard's nodes are nodules of the proximal joints. Both are found in patients with OA. Rheumatoid nodules are described as firm, nontender nodules often found on the dorsum of the wrist, elbow, or metacarpophalangeal joint. Haygarth's nodes are spindle-shaped enlargements of the middle interphalangeal joints that occur in RA (LeBlond et al., 2015). Patients presenting with "sausage-like" digits known as dactylitis may have reactive or psoriatic arthritis (LeBlond et al., 2015).

Any joint deformity should be palpated to determine whether it fluctuates because of fluid or is firm because of a thickening or enlargement. If there is fluid, the cause may be recent trauma, inflammation, or joint infection. An articular enlargement of RA is usually soft, whereas a joint deformity found in OA is usually firm. Erythema may also be indicative of septic arthritis. If the joint swelling persists for more than 3 days, this may indicate an arthritic condition. Also examine the bursal areas around the joints, palpating for swelling, tenderness, ganglions, and presence of nodules. Tophaceous gout appears as salmon-colored nodules that do not transilluminate, as is possible when examining a superficial inflamed bursa (Williams, 2008). A ganglion is usually a soft cyst-like swelling, often found near joints. Swelling of the bursa is usually tender on palpation. All masses should be measured across their greatest diameter (LeBlond et al., 2015).

MUSCLE WEAKNESS

For the patient complaining of muscle weakness, inspect for evidence of muscle atrophy and palpate the muscles during contraction and rest. Any fluttering or fasciculations of the muscles should be noted. To determine if a patient is having true muscle weakness, the patient should perform against the examiner's resistance. One side should be compared with the other and a numerical value for tested muscle strength recorded. Flexor and extensor muscles should be tested for strength (LeBlond et al., 2015).

BURSITIS, TENDINITIS, SOFT TISSUE SYNDROMES

Signal Symptoms: Pain, tenderness in soft tissue areas, erythema, and swelling.

Description: Bursitis is the inflammation of a bursa, which is a sac lined with synovial membranes that secrets synovial fluids. Tendinitis is the inflammation of a tendon, which is the noncontractile portion of a muscle (Brown & Clark, 2016).

Etiology: Underlying causes of acute bursitis and tendinitis include micro-trauma and mechanical stressors. Chronic conditions such as gout, RA, and calcium pyrophosphate dihydrate can contribute to the development of bursitis (Berkoff, Sandbulte, Stafford, & Berkowitz, 2016). Superficial bursitis can be further divided as acute traumatic/hemorrhagic and chronic. Acute traumatic/hemorrhagic bursitis results from forceful contact, often resulting with bleeding into the bursa. Bleeding is more common and can occur spontaneously in patients with coagulopathies and prescribed anticoagulants. Patients with chronic superficial bursitis frequently sustain micro-trauma from repeated friction (Khodale, 2017). Common origins of bursitis include prepatellar, olecranon, trochanteric, and retrocalcaneal (Aaron, Patel, Kayiaros, & Calfee, 2011). Supraspinatus tendinitis, bicipital tendinitis, and Achilles tendinitis are common sites of inflamed tendons.

Occurrence: May occur anytime, but increases after middle age.

Age: Range of 53 to 71 years old with a median age of 62 years old (Brown & Clark, 2016).

Gender: In studies, females outnumber males. These conditions are often related to occupation, activities, and hobbies that may be gender prevalent.

Ethnicity: Not significant.

Contributing Factors: Certain diseases and conditions increase the risk of developing bursitis, such as RA, OA, gout, thyroid disease, chronic kidney disease, and diabetes (Khodale, 2017). Olecranon bursitis is known to occur in patients on hemodialysis in the arm with the arteriovenous fistula (Senecal & Leblanc, 2001). Septic bursitis often results from a nearby tissue infection such as cellulitis or an aspiration of a large bursa. Chronic septic superficial bursitis occurs in patients with the chronic diseases mentioned previously, patients who are alcoholics, and those who are

immunocompromised (Khodale, 2017). In patients with intrabursal rheumatoid nodules or gouty tophi, the risk for septic bursitis increases (McAfee & Smith, 1988). Idiopathic bursitis may result from minor, often unrecognized traumas, especially in older adults.

Certain occupations and activities that involve leaning on elbows or pressure on knees can contribute to olecranon and prepatellar bursitis (Khodale, 2017; McAfee & Smith, 1988). Risk factors for developing Achilles tendinitis include sports or activities that involve running or jumping, prior history, family history, hypertension, hyperlipidemia, diabetes, elevated body mass index (BMI), and fluroquinolone antibiotics (Asplund & Best, 2013; Klein, Weil, Weil, & Fleischer, 2013). Plantar fasciitis also is the result of load-bearing micro-traumas over time (Owens, 2017).

Signs and Symptoms: Patients with presentation of soft tissue syndromes should be asked about the origin of the presentation. If there is associated pain, have the patient describe the pain (throbbing, dull, burning, constant, intermittent). Inquire about all activity that has occurred to include occupation, hobbies, ergonomic positioning, postural disorders and trauma, and even minor injuries that may have predisposed the patient for the development of a soft tissue condition. Question if the symptoms are alleviated when not performing an activity or in a work setting. Identify all presenting and associated symptoms such as swelling, erythema, pain, reported decrease in ROM and limitation on ADLs, and alterations in sensations of hot or cold (Browne & Merrill, 2015). Question patient about prior history or family history of soft tissue conditions, aspiration of bursa, recent fever, fluroquinolone use, and chronic medical conditions. Inquire as to when the condition began and if the condition progressed. Ask how long there has been noted limitation in movement or restriction in ADLs. Inquire if there is a particular time of the day the clinical presentation occurs. For instance, when the patient steps out of bed, is there pain after sustained pressure on the area, prolonged sitting, or standing? Does the patient experience any morning stiffness? Have the patient identify where the pain and/or swelling is located and if prior areas have been involved (Owens, 2017). Question if there is any referred pain. Try to discern if the condition was traumatic or nontraumatic (Browne & Merrill, 2015; Khodale, 2017).

In patients with suspected Achilles tendon disorders, look for evidence of an effusion into the adjacent bursa. Palpate for any heel pain. A tendon rupture can be assessed by squeezing the calf muscle. If the tendon has a tear, the ankle will not flex (Asplund & Best, 2013).

Physical examination needs to begin with assessment for fever. Look for evidence of ecchymosis, erythema, swelling, and tenderness. Note if there is any point tenderness. Examine for any associated nodularity, such as tophi or rheumatoid nodules in patients with suspected bursitis (Khodale, 2017). Perform passive ROM; determine ROM first on the nonacutely affected side. In patients presenting with symptoms of plantar fasciitis, passive dorsiflexion of the toes and ankle may elicit pain in the medial plantar area (Owen, 2017).

For patients complaining of shoulder pain and tenderness, supraspinatus tendinitis or tenosynovitis and subacromial bursitis are the most common shoulder problems to be presented in practice. Patients with these conditions most often complain of pain located at the shoulder tip. On clinical examination, palpable tenderness is normally noted over the superior aspect of the shoulder, and, on testing shoulder movements, painful active abduction is typically found between 70 and 140 degrees for tendinitis (Barratt, 2009). In subacromial bursitis, on clinical examination localized tenderness at the shoulder tip and a soft feeling may also be noted at the humeral head. When the patient's shoulder movements are tested, there is a complaint of superior shoulder pain on both active and passive abduction, but minimal pain is experienced on resisted abduction because the supraspinatus muscle and tendon are not inflamed. Pain happens with passive abduction of the glenohumeral joint due to the subacromial space becoming restricted, compressing the subdeltoid bursa, which is exhibited as shoulder tip pain (Barratt, 2009).

The inflammatory condition of a nontraumatic tear of one or more of the rotator cuff muscles usually occurs in patients over 50 years old, because people in this age group tend to develop muscle fibrosis or long-term tendinitis or bone spurs. These everyday conditions can lead to the rotator cuff muscles tearing. On testing patients' shoulder movements by either active abduction or lateral or medial rotation, pain and weakness are often noted. Patients may also be unable to abduct their shoulders or hold abduction at 90 degrees (Barratt, 2009).

Diagnostic Tests: Any patient in which septic arthritis is being considered should have a blood cell count with differential, C-reactive protein, and erythrocyte sedimentation rate (ESR). Consider blood cultures on immunocompromised patients. If the cause of the bursitis remains unknown, aspiration is recommended: order crystal analysis, Gram culture, blood cell count, and glucose measurement (Khodale, 2017). Ultrasound can be useful in visualizing a large bursa. Color Doppler can be ordered to help determine the presence of inflammatory bursitis (Khodale, 2017). In the case of patients with periarticular pain in cases where fractures have been ruled out, MRI may be beneficial to diagnose tendon ruptures (Asplund & Best, 2013; Emam, Farmakidis, Lee, & Wainapel, 2016). When determining if a patient has plantar fasciitis, generally imaging is not needed for acute pain unless the patient is having atypical presentation or history of foot trauma. Radiographs can show calcifications in the soft tissue. Thickened heel aponeurosis is identified by ultrasound. Radiology tests can also rule out fractures and determine the presence of heel spurs. Ultrasound and MRI can be useful diagnosing complicated plantar fasciitis cases that would include tears or rupture (Owens, 2017).

Differential Diagnosis:

- Fracture
- Degenerative joint disease
- Cellulitis
- Septic and inflammatory arthritis
- Joint effusions
- Morel-Lavallee lesions (Khodale, 2017).
- Heel contusion
- Tarsal tunnel syndrome
- Tumor
- Plantar facia rupture (Owens, 2017).

Treatment: The treatment of acute soft tissue syndromes starts conservatively. The PRICEMM mnemonic is used for pain control in bursitis and tendinitis conditions as indicated:

- P— Protect with padding, braces, and changes in techniques.
- R— Rest, avoid activities that exacerbate pain.
- I— Ice; cryotherapy can relieve pain and decrease inflammation.
- C— Compression; elastic dressings can ease pain in olecranon bursitis.
- E— Elevation; raise the affected limb above the level of the heart.
- M—Modalities; electrical stimulation or ultrasound.
- M—Medications; NSAIDs, acetaminophen, and/or corticosteroid injection. (Simpson & Howard, 2009; Khodale, 2017)

Corticosteroid injection can be effective when other treatment fails. A mix of corticosteroids and local anesthetic is injected into the tender site. At times of suspected infection/sepsis, the bursa is aspirated for content and no cortisone is injected. A 4-week course of sensitivity-susceptible antibiotics is advised. Outpatient treatment is effective in 40% to 50% of cases, with hospitalization advised for severe cases.

Indications for therapeutic injection for soft tissue conditions include bursitis, tendinitis, trigger points, ganglion cyst, neuromas, entrapment syndromes, and fasciitis (Foster, Voss, Hatch, & Frimodig, 2015). Absolute contraindications for therapeutic injection include cellulitis, acute fracture, history of allergy, and joint prosthesis. The effects of injection are inversely related to solubility of the agent; therefore, the less soluble, the longer acting the agent (Cardone & Tallia, 2002). When using anesthesia, obtain informed consent and assess for toxicity, which can include hives, flushing, nausea, and pain. Fat atrophy, changes in pigmentation, and tendon rupture can occur. It is important to remember that with the introduction of steroids comes hyperglycemia, occurring in patients with diabetes mellitus (Foster et al., 2015).

Short-term treatment for plantar fasciitis includes rests with site specific taping, stretching, encouraging the use of proper footwear, icing and anti-inflammatory medications as tolerated. If symptoms are still occurring, patients may require physical therapy, the use of night splints, and corticosteroid injections. For patients with chronic plantar fasciitis, extracorporeal shock waves, which are focused, single-pressure pulses of microsecond duration may be included in treatment (Owens, 2017). Side effects of high-energy pulses are petechiae and hematomas. Limited evidence showed that casting or surgery may be effective when conservative measures fail.

Alternative treatment for epicondyle pain includes the use of acupuncture. Randomized and quasi-randomized controlled trials examining the effects of acupuncture on lateral epicondyle pain were selected from six studies. The majority of these studies, five out of six, expressed strong evidence suggesting that acupuncture is effective in the short-term relief of lateral epicondyle or tennis elbow pain (Bisset, Coombes, & Vicenzino, 2011).

The review for surgery of the rotator cuff has shown that it may not lead to any difference in pain compared with different exercise programs. Arthroscopic surgery showed no difference except in recovery time. Side effects that have occurred in the studies included pain, infection, difficulty moving the shoulder after the operation, wasting of the shoulder muscle, and the need to have another surgical procedure (Green, Johnston, & Bell, 2008). There were no differences in side effects between types of surgery.

Conservative treatment is also recommended for Achilles tendon disorder. Studies have found that eccentric calf exercises and low energy shock wave treatment was beneficial for recovery (Asplund & Best, 2013). For patients with probable Achilles tendon rupture, referral to an orthopedist is recommended.

Follow-up: Generally, an acute concern; however, aspiration or surgery as listed may be needed. Patients may benefit from physical therapy if soft tissue condition is not resolved.

Sequelae: Left untreated, these conditions may lead to limitations of movement and, thus, the inability of an older adult to perform ADLs. Untreated septic bursitis can lead to sepsis. Achilles tendon disorders can progress to Achilles tendon rupture (Asplund & Best, 2013).

Prevention/Prophylaxis: Secondary preventive measures include avoidance of mechanical stressors, stretching exercises, the use of compressive bandages, and control of chronic conditions that contribute to soft tissue syndromes.

Referral: Refer to orthopedist for injections, surgery, and for cases that are not responding to treatment regimen or with complications (Asplund & Best, 2013; Khodale, 2017; Owens, 2017). In some cases in the literature, an acupuncturist has been shown to be effective for pain (Bisset et al., 2011).

Education: Inform patients about ways to avoid micro-trauma and mechanical stressors. Encourage participation in recommended exercising to include stretching for tendon injuries. Inform patients with history of Achilles tendinitis not to take fluroquinolone antibiotics. Patients will need consent before injections. Give presurgical education if needed.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Advanced age was found to be related to the need for advanced therapy for patients with Achilles tendinitis.	B	Klein, Weil, Weil, & Fleischer, 2013
In patients with suspected septic bursitis, aspiration should be performed.	C	Baumbach, Lobo, Badyine, Mutschler, & Kanz, 2014

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Patients with suspected septic bursitis should be initially treated empirically to cover the most common organisms that cause septic bursitis: *Staphylococcus aureus* and *Streptococcus.* Treatment regimen can be modified once culture results become available.	B	Baumbach, Lobo, Badyine, Mutschler, & Kanz, 2014
Fibrin sealant was found to prevent recurrence of non-septic olecranon bursitis.	C	Berkoff, Sandbulte, Stafford, & Berkowitz, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

FRACTURES

Signal Symptoms: Soft tissue swelling, dislocation, altered ROM, ecchymosis, local tenderness, pain with any motion.

Description: Fractures are a common cause of disability in older adults. A compound fracture or open fracture occurs when fragments of the bone pierce the skin or mucosa; an impacted fracture occurs when fragments wedge together. Common sites for fractures include proximal humerus, distal radius, pelvic ramus, proximal femur, proximal tibia, and thoracic and lumbar vertebral bodies (Southerland & Barrie, 2014).

Etiology: Many fractures are due to an indirect or direct injury. Vertebral fractures may result from an activity such as heavy lifting or bending over that puts sudden stress on the spine. Fractures most attributed to osteoporosis include femoral neck fractures, pathological fractures of the vertebrae and lumbar, and thoracic spine vertebral fractures; whereas skull fractures, fractures of the facial bone, and open proximal humerus fractures were found to be the ones least likely to be a result of a patient having osteoporosis (Warriner et al., 2016). When a fracture is caused by multiple myeloma, bone marrow is replaced by malignant plasma cells and, thus, bone is destroyed.

Occurrence: More than 250,000 hip fractures occur in the United States each year. Estimates show that 30% of people age 65 years and older fall each year; in that group, 5% of those falls result in fractures.

Age: Fractures are more prevalent in women who are older than menopausal age due to decreased protective benefits of estrogen to bone, causing some degree of osteoporosis. Increasing age is a risk factor for all types of fractures (Southerland & Barrie, 2014).

Gender: Older females tend to outnumber males because the majority of fractures in this age group occur due to menopausal osteoporosis (Southerland & Barrie, 2014).

Ethnicity: Those at high risk for osteoporosis include Caucasian and Asian American women. These ethnic groups have a higher prevalence of fractures than African American women because of their increased risk of osteoporosis. A systematic review of fractures related to osteoporosis found that African American males had a low probability of sustaining fractures of the tibia, fibula, patella, ribs, and sacrum, whereas Caucasian women had the highest probability of attributing one of these fractures to osteoporosis (Warriner et al., 2016).

Contributing Factors: Multiple factors exist that predispose older adults to sustaining fractures. These include low vitamin D levels, osteoporosis, low BMI, malnutrition, and sedentary lifestyle; medications such as long-term high steroid doses; and impaired vision, neurological disease, on dialysis, prior history of fractures, poor balance and difficulty with gait, orthostatic hypotension, and muscle atrophy. Consider also household hazards like throw rugs and poor lighting that can contribute to falls (Southerland & Barrie, 2014). Review patient medications. In addition to long-term high steroid use, the following are known to increase fracture risk in patients: anticoagulants, antipsychotics, selective serotonin reuptake inhibitors, and levothyroxine in male patients (Southerland & Barrie, 2014). Another cause of fractures is injuries sustained in motor vehicle accidents (MVAs), pedestrian involved MVAs, and falls from heights (Smeltzer, 2010). Patients with diagnosed diabetes have been found to be at increased risk for fractures (Schneider et al., 2013).

Signs and Symptoms: Determine from the patient how the injury occurred and what specific activity the patient was doing prior to the suspected fracture. Inquire about when the injury occurred, other related injuries, prior history of fractures, and what the patient has done to alleviate any pain and discomfort related to the fracture (Eiff & Hatch, 2017; Smeltzer, 2010). Symptoms include soft tissue swelling, ecchymosis, abrasion, edema, skin discoloration, local tenderness, and pain with any motion. Fractures often are a result of injury to the affected area. It is important to palpate the area of maximum tenderness to determine if more than one bone may be involved (Eiff & Hatch, 2017). For vertebral compression fractures, however, trauma may not be the precipitating factor. An older adult may report pain, pressure, spasms,

and swelling in the injured area. A patient with a femoral neck fracture may report groin pain. Physical examination will reveal soft tissue swelling, ecchymosis, local tenderness, and pain with motion (particularly in weight-bearing limbs) (Southerland & Barrie, 2014).

Diagnostic Tests: Recommend ordering radiographs of the affected area/injury to rule out and identify area and type of fracture. Radiographs of the affected area are needed in two and three views (Eiff & Hatch, 2017). Also important is a complete blood count (CBC) to determine if internal blood loss has occurred during an injury. Further laboratory studies include a Westergren ESR. An elevated ESR may indicate whether an infectious process was a cause of the fracture.

Differential Diagnosis:

- An infectious process/osteomyelitis
- Neoplastic process/multiple myeloma
- Osteoporosis

Treatment: For the pain related to the fracture, narcotic analgesics and/or short-term NSAIDs, if not prohibited, are helpful. Depending on the site of the fracture, casting, walking boot, elastic wrapping, splints, immobilization, traction, or surgical intervention may be used (Eiff & Hatch, 2017). The most common surgical intervention for a fractured hip is the open reduction with internal rotation, especially for fractures of the intertrochanteric or subtrochanteric region.

Follow-up: The long-term management of a patient with a fracture depends not only on the location of the fracture, but also on the etiology of the fracture, whether osteoporosis, an infectious process, or neoplasia (Buckley & Page, 2016). Adequate nutrition and wound care are important for proper healing. It is common in nursing homes to start patients on zinc and vitamin C supplements; however, no study is conclusive in this treatment modality. Patients should be questioned about any muscle weakness or paresthesia after the incident. Patients may have return appointments with an orthopedist, physical therapist, or both. The function of the affected area should be evaluated. Determine what impact the injury had on the patient's ADLs and instrumental ADLs (Eiff & Hatch, 2017).

Sequelae: The complications resulting from a fracture depend on many factors, including comorbidities, health status of the patient before the injury, and the location of the fracture (Buckley & Page, 2016). Patients are susceptible to postoperative anemia, hypovolemic shock, infection, incontinence, decubiti, subdural hematoma, dehydration, electrolyte imbalance, hypothermia (especially with an open fracture), and phlebitis after sustaining the injury and throughout the recovery period (Smeltzer, 2010). The risk for developing venous thrombosis and subsequent pulmonary embolism increases for patents with fractures that restrict mobility and ADLs. Evaluate the prothrombin time (PT)/international normalized ration (INR) level for patients who are taking anticoagulants or any condition that affects coagulation, as anticoagulation therapy may need to be initiated and/or adjusted (Mears & Kates, 2015).

Soft tissue injuries sustained with the fracture also increase the risk for clot formation, as well as compartment syndrome. Another complication of fractures that penetrate the muscle is gas gangrene (Eiff & Hatch, 2017). Patients who develop edema, ecchymosis, and/or abrasions are at risk for delay of surgical repair when there is accompanying soft tissue damage (Smeltzer, 2010). Older adults who sustain rib fractures are at risk for developing pneumonia or acute respiratory distress syndrome (Buckley & Page, 2016). For hip fractures, approximately 15% of patients are readmitted to the hospital during the first 6 months. There is also a high mortality rate associated with hip fractures (Mears & Kates, 2015). Loss of physical and social function also may follow an injury. Monitor for signs and symptoms of depression and lack of sleep.

Prevention/Prophylaxis: Weight-bearing exercise programs and balance training should be encouraged of older adults at risk for fractures. Cataract surgery in patients whose vision is impaired reduces the number of fractures. The modification of the patient's environment, including removal of hazards such as throw rugs and small pieces of furniture; improvements in lighting; and installation of grab bars, raised toilet seats, and ramps helps prevent injuries (Southerland & Barrie, 2014). Patients at risk for fracture should be evaluated to determine their Vitamin D and calcium intake; recommendations for women over age 50 years and men over age 70 years is 120 mg a day. Encourage patients to regularly include foods containing calcium in their diets so they do not depend completely on calcium supplementation. For patients with osteoporotic fractures, treatment with first line bisphosphonates should be initiated.

Referral: Rehabilitation needs will include an orthopedic specialist for complex casting and surgical needs. Physical therapist/occupational therapist referrals to further evaluate and treat functional physical impairments and to improve ADLs are in order to provide as much independent living as possible. Keep in mind that older patients may be hesitant to resume previous normal activities due to falls. This may be resolved by the involvement of a rehabilitation team.

Education: After an injury, older adults are often hesitant or afraid to repeat the activity that led to the incident. Patients with osteoporosis need to be instructed on safe practices for lifting, bending, and reaching for objects. If the injury occurred after a fall, assessment of the home environment and consideration of adaptive equipment are suggested.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
For patients with fractures of the proximal humerus, treatment needs to be individualized; patients would benefit from care delivered by a multidisciplinary team.	C	Vachtsevanos, Hayden, Desai, & Dramis, 2014

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Femoral neck, pathological fractures of the vertebrae, and lumbar and thoracic vertebral fractures were found to be the most common type of fractures related to a diagnosis of osteoporosis.	B	Warriner, Patkar, Curtis, Delzell, Gary, Kilgore, & Saag, 2011
Patients diagnosed with diabetes found to be at an increased risk of fracture; for those patients with poor glycemic control the risk was even greater.	B	Schneider, Williams, Brancati, Blecker, Coresh, & Selvin, 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

GOUT

Signal Symptoms: Polyarticular in older adults, soft tissue tenderness, tophi, podagra (metatarsophalangeal [MTP]).

Description: Gout, an inflammatory disease associated with malfunctioning metabolism of purine, leading to overproduction or underexcretion of uric acid, results in deposits of sodium urate crystals in the joints, periarticular tissues, subcutaneous tissues, and kidneys. Primary gout is the clinical disease caused by hyperuricemia; secondary gout usually occurs as a result of extended use of agents that decrease uric acid excretion. Gout is the most prevalent inflammatory condition occurring in older adults. Although it is a common presentation in older adults, the diagnosis of gout is less than routine because gout flares in older adults mimic other arthritic conditions, such as septic arthritis, and can coexist on the osteoarthritic growths of the distal and proximal interphalangeal joints (Ning & Keenan, 2011).

Initial presentation of gout in older adults tends to be polyarticular and directly associated with chronic conditions, such as hypertension and renal impairment, and the specific treatments for these conditions, namely diuretic use (Roberts, 2010). Physiological stressors, common in older adulthood, can contribute to the rapid development of an acute gout attack; for example, gout beginning in a dehydrated patient or postsurgical procedure (Doherty, 2009). Additionally, adults with hypertension, diabetes, cardiovascular disease, and metabolic syndrome are at high risk for developing gout (Chang-Fu, Grainge, Zhang, & Doherty, 2015). The deposition of monosodium urate crystals in synovial fluid and other tissues occurs or the formation of tophi or uric acid stones in the kidney occurs in patients with chronic gout.

Etiology: Clinically, hyperuricemia is defined when the serum urate level is more than 6.8 mg/dL (Khanna et al., 2012). With levels more than10 mg/dL, the chance of an acute attack of gout is more than 90%. In 70% to 90% of patients with diagnosed gout, underexcretion of urate rather than a metabolic overproduction causes elevation of the plasma urate level. Although hyperuricemia is a risk factor for gout, some patients with a normal serum acid level develop acute gouty arthritis. The mere presence of intrasynovial urate crystals is not sufficient to cause flares of gouty arthritis. The majority of patients who develop gout have been hyperuricemic for about two decades.

Occurrence: For every 100,000 people in the United States, 100 cases of gout occur. It is estimated that 3.9% of U.S. adults (age 18 years or over) are affected by gout annually (approximately 8.3 million people) (Lawrence et al., 2008; Zhu, Pandya, & Choi, 2011). The frequency of gout increases in older adults; the highest lifetime prevalence is between the ages of 70 to 79 years (7.3%), with peaks in men ages 75 to 84 years (Black et al., 2015).

Age: Primary gout usually begins in the forties to sixties, whereas a new presentation in older adults occurs in the sixties to seventies as a result of factors contributing to a cause of secondary gout. Prevalence of gout increases with advanced age in men, peaking at 75 to 84 years.

Gender: The prevalence of gout in women is lower at all ages than in men, whose risk increases with age. The risk of gout increases in women after menopause due to decreased estrogen levels, use of diuretics, prevalence of renal insufficiency, and longevity over males (Chang et al., 2015).

Ethnicity: There is a high prevalence in Pacific Islanders, and people from Samoa and the Philippines. Limited data suggest an increased incidence of gout in African Americans as compared to Caucasians; however, clinically recognized gout is extremely rare in Africans living in Africa. In England, gout affects 16.4 of every 1,000 men and 2.9 of every 1,000 women.

Contributing Factors: Factors associated with primary gout in men include positive family history, obesity, trauma, hypertension, hyperlipidemia, hypertriglyceridemia, diets high in purine (especially organ meats, anchovies, sardines, scallops, oatmeal), alcohol consumption (especially beer and moonshine whiskey), dietary intake of high-fructose corn syrup products, lead intoxication, dehydration, fasting (which causes ketosis), binge eating, analgesic nephropathy, nephrolithiasis, urolithiasis, and polycystic kidney disease (Choi &

Curhan, 2008; Neogi, 2011). Research data have indicated elevated risk for gout in connection with the following conditions: insulin resistance/diabetes mellitus, the metabolic syndrome, renal insufficiency, chronic kidney disease, hypertension, cardiovascular disease, heart failure, and organ transplantation (Neogi, 2011). Common causes of secondary gout include chronic kidney disease, hypothyroidism, and hyperparathyroidism. Elevated uric acid levels may result in myeloproliferative diseases, common in older adults, when cell lysis occurs as a result of chemotherapy (El-Zawawy, et al, 2017).

Drugs leading to increased risk for gout include thiazide and loop diuretics, low-dose aspirin (less than 1 g/day), ethambutol, pyrazinamide, levodopa-carbidopa, nicotinic acid, tacrolimus, and cyclosporine (Choy, 2005; Neogi, 2011). Diuretics are the most common cause of secondary gout because they cause intravascular depletion, lower the glomerular filtration rate, and increase the reabsorption of urate. Patients with combined use of loop diuretics and thiazide diuretics may have a five-fold greater risk of gout flares (Chang et al., 2015).

Signs and Symptoms: Review the patient's history for evidence of excessive alcohol consumption, dietary habits (including foods that are high in purine), medical diagnosis of gout, family history of gout, diabetes mellitus, increased BMI, exposure to lead, consumption of illicit whiskey, consumption of high-fructose beverages and additional food products, trauma, and all medication use. The following questions are helpful to ask the patient in order to understand the presentation of symptoms:

- Did the pain occur suddenly or become noticeable gradually?
- Is the pain dull, throbbing, or unbearable to touch?
- Has the pain ever occurred before?
- If so, how long did it last, and was the swelling in the same joint?
- How was the pain and swelling treated?

In middle-aged men, the classic presentation of an acute gout attack is a hot, swollen, erythematous MTP joint of the great toe known as podagra. Usually, in the first presentation of gout in middle-aged men, joint involvement is monarticular. In older adult women, joint involvement with gout is usually polyarticular and often occurs in joints above the waist. The proximal interphalangeal (PIP) joints and the distal interphalangeal (DIP) joints should be examined and the instep, heel, ankle, knee, wrist, and olecranon bursa palpated for signs of swelling and tenderness. An acute attack can escalate over an 8- to 12-hour time frame with pain and erythema in the affected joints (Burns, Workman, Imboden, Hellman, & Stone, 2013). Tophi, subcutaneous deposits of sodium urate, are common in chronic gout, developing approximately 10 years after the initial gout attack as a result of persistent hyperuricemia (West & Janson, 2015). Examine the helix of the ear, olecranon bursa, prepatellar bursa, Achilles tendon, over Heberden's nodes, and finger pads for signs of tophi. Fever may be present. Although rare, gout may be found in the spine (Jegaprogason, 2014).

Diagnostic Tests: The gold standard for confirming the diagnosis of gout is the presence of monosodium urate (MSU) crystals in the synovial fluid. Arthrocentesis is indicated for every patient in whom a diagnosis has never been proved by joint aspiration and for those in whom a possibility of septic arthritis exists (Burns et al., 2013). Musculoskeletal ultrasonography and dual-energy computed tomography (DECT) is increasingly being used to diagnose gout, as urate crystals may be visualized on the articular cartilage. Radiographic findings in chronic gout will show tophi and bony erosions (Igel et al., 2017).

A prior history of gout or pseudogout does not rule out the possibility of acute septic arthritis. Joint aspiration will also rule out septic arthritis and pseudogout. Even in the presence of crystals in the joint fluid, blood cultures are indicated if any sign of systemic toxicity is present. Septic arthritis can occur in patients with active crystalline arthropathy.

Elevated serum uric acid level is indicative of hyperuricemia; however, patients with high uric acid levels may be asymptomatic. Check the CBC to determine if leukocytosis is present and to rule out sepsis. Serum creatinine and blood urea nitrogen (BUN) should be ordered initially on patients with suspected gout to determine the presence of renal insufficiency. Elevated ESR or C-reactive protein will indicate an inflammatory process but will not distinguish one arthritic condition from another.

Differential Diagnosis:

- Pseudogout
- Traumatic joint injury
- Septic arthritis
- Cellulitis
- Hemarthrosis
- RA
- OA
- Seronegative spondyloarthropathies (Goroll & Mulley, 2014)

Pseudogout (calcium pyrophosphate deposition disease [CPDD]) is caused by the deposition of calcium pyrophosphate rather than the deposition of uric acid derivatives that cause gout. Also, in pseudogout, synovial fluid samples obtained with aspiration have positive birefringence. This finding is in direct contrast to the negative birefringence in gout.

Treatment: NSAIDs, corticosteroids, and oral colchicine are acceptable first-line options for treatment of acute gout, with treatment started within 24 hours of attack onset. Management of the older adult with gout requires careful monitoring. Older adults are susceptible to renal insufficiency, may have other concomitant diseases, and experience hypersensitivity to some of the medications used to treat younger patients with gout. NSAIDs should be used cautiously in the treatment of gout in older adults who have a history of heart failure, renal failure, and gastrointestinal conditions (Fravel & Ernst, 2011). As with any NSAID, renal function must be monitored. NSAIDs can endanger existing renal function, especially when the creatinine clearance is less than or equal to 30 mL/min. Additional concern with NSAID use in older adults is the potential for gastrointestinal bleeding due to NSAID-induced peptic ulcers. An exacerbation of hypertension can occur with excessive use of NSAIDs in older adults. Extensive use of NSAIDs can lead to fluid retention and antagonism of diuretic therapy, which may precipitate heart failure (Fravel & Ernst, 2011).

Indomethacin is effective in the treatment of acute gout. The usual dose is 25 to 50 mg orally two to three times daily until the symptoms cease, then begin to taper the dose for 5 to 7 days. Liquid indomethacin is available for patient use. Although indomethacin has been traditionally favored in the treatment of gout, there has been no research documenting the advantage to its use over other NSAIDs such as naproxen. Recommendations from the American College of Rheumatology (ACR) 2012 guidelines on the management of gout did not reach a consensus on any specific NSAIDs as first-line treatment (Khanna et al., 2012). As with any NSAID, renal function must be monitored. NSAIDs can endanger existing renal function, especially when the creatinine clearance is less than or equal to 30 mL/min. Cyclooxygenase-2 inhibitors may be better tolerated in older adults with history of peptic ulcer.

Colchicine can be given for acute gout attacks orally, as it functions as an anti-inflammatory and does not lower the uric acid level. It is most effective if given within 24 hours of an attack. The oral colchicine dose is 1.2 mg followed by a single 0.6 mg dose 1 hour later. Dose should not exceed 1.8 mg a day for an acute flare. Colchicine may be used daily or every other day, dosed per renal function, and as prophylaxis against flares while lowering uric acid levels, as the risk of flare during this time is increased (Janson, 2015).

Gastrointestinal toxicity commonly precedes a therapeutic response. Because oral colchicine is associated with side effects, it should be administered cautiously to patients with impaired renal or hepatic function or gastrointestinal disease or to postoperative patients because of the potential for vomiting. Patients for whom colchicine and NSAIDs are contraindicated may be given intra-articular steroid injections for monarticular joint involvement. Short-term corticosteroids can be initiated in patients unable to tolerate NSAIDS or colchicine, and used cautiously in patients who are diabetic or immunocompromised. Xanthine oxidase inhibitors and uricosuric agents should not be prescribed for an acute gout attack (Fravel & Ernst, 2011). However, ongoing pharmacological urate-lowering therapy should not be stopped during a gout flare.

Follow-up: Patients having a first acute attack of gout should be followed up with 72 hours after initiating treatment to determine effectiveness and presence of any side effects. Patients who have more than two attacks of gout in a year are considered to have chronic disease. Before initiating a long-term medication regimen, uric acid level, BUN, creatinine, serum lipid level, CBC, and urinalysis should be ordered.

For patients with chronic gout, urate-lowering therapy with xanthine oxidase inhibitors, such as allopurinol or febuxostat, is the first-line treatment option. The starting dose for allopurinol is recommended at no higher than 100 mg/day, to be increased every 2 to 3 weeks until the desired uric acid level is less than 6 mg/dL at a minimum, if not lower, until less than 5 mg/day is achieved (Khanna et al., 2012). In patients with moderate to severe kidney disease, a lower starting dose should be considered (Khanna et al., 2012). Recommendations from the ACR found that monotherapy with allopurinol at doses of 300 mg could be raised to 300 mg daily even with renal impairment. Patients, however, will need to be educated on side effects of drug toxicity (Khanna et al., 2012).

In patient populations known for high risk for allopurinol hypersensitivity (Koreans with stage 3 or worse chronic kidney disease and Han Chinese and Thai), screening for HLA-B 5801 should be considered before prescribing this medication (Khanna et al., 2012). Febuxostat is available in 40 to 80 mg daily doses. Dose adjustment is not necessary in the older adult with stage 2 or 3 chronic kidney disease (30 to 89 mg/dL) or mild hepatic insufficiency. Allopurinol or febuxostat should not be discontinued during a gout flare, as this may lead to further fluctuation of uric acid levels, exacerbating the current flare. Alternative treatment to chronic gout is low-dose indomethacin 25 mg twice a day or another NSAID (Fravel & Ernst, 2011). Once medications are initiated for gout, target serum urate levels should be 6 mg/dL or less according to 2012 ACR guidelines (Khanna, 2012). Uric acid levels of less than 5 are recommended for patients with tophi.

For patients with chronic tophaceous gouty arthropathy, the ACR guidelines recommend combination therapy with one xanthine oxidase inhibitor (allopurinol or febuxostat) and one uricosuric agent when the target urate levels are not achieved (Khanna et al., 2012). The usefulness of probenecid in the older adult is limited because it is effective only when the creatinine clearance is less than 30 to 40 mL/min and is contraindicated in patients with renal insufficiency and those on diuretics.

Recent advances in understanding the inflammatory process of gout have led to the discovery of new anti-inflammatory medications. Current research is directed to the use of monoclonal antibodies and medications targeting specific cytokines contributing to gout inflammation. Several new urate-lowering drugs are being used for individuals with chronic tophaceous gout that are refractory to allopurinol and febuxostat. Pegloticase is a new IV medication administered every two weeks that degrades uric acid and resolves tophi; the greatest concern administering this medication is infusion reactions (Igel et al., 2017).

Sequelae: Older adults who have pre-existing hypertension or primary renal disease, or who use NSAIDs and diuretics, are at risk for developing renal insufficiency, chronic urate nephropathy, and acute hyperuricemia nephropathy. Uric acid nephrolithiasis is also a complication of gout. Untreated chronic gout may lead to multilobular, tender subcutaneous tophaceous gout, which can be deforming and impair ADLs. Progressive renal failure is common in patients with gout and has been known to contribute to up to 20% of deaths (Rosenberg, 2005). Left untreated, elevated uric acid levels have been shown to contribute to the development of cerebrovascular disease and vascular dementia (Feig, Kang, & Johnson, 2008). Patients with draining tophi are at risk for infection, and untreated chronic tophaceous gout can lead to severe joint destruction (Khanna et al., 2012). Given the complexity of comorbid conditions common in patients with gout, risk for polypharmacy and drug–drug interactions should be of great concern to primary care providers (Dalbeth, 2013).

Prevention/Prophylaxis: Thiazide diuretics and salicylates should be avoided in patients with gout. Patients should be cautioned about the use of alcohol and told to avoid foods high in purine, especially during an acute gout flare. For patients with an elevated BMI, weight reduction is recommended. Maintaining control of diabetes and hyperlipidemia can aid in the reduction of developing gout flares.

Referral: Patients may need a referral to a rheumatologist if they require intra-articular injections of steroids or joint aspiration, have complications from treatment, or have unusual presentation of the disease. A patient also may benefit from a referral to a dietitian for information on how to follow a low-purine diet.

Education: Patients need to be informed that during an acute attack they should rest the affected area and limit weight bearing if the first MTP joint is involved. Patients should be encouraged to be well hydrated with an intake of 3 L/day of fluids unless contraindicated. The use of alcohol should be discouraged. Smoking cessation, weight loss for obese patients, physical exercise, and overall healthy diet are recommended lifestyle changes for all patients with gout (Khanna et al., 2012). Patients should consult their primary healthcare provider regarding selection of any over-the-counter (OTC) medications.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
A presumptive diagnosis of gout can be determined in many cases based on history, clinical presentation, and use of the ACR's decision rule; however, if possible, joint aspiration and polarized light microscopy should be performed to verify diagnosis and rule out septic arthritis or pseudogout.	B	Khanna et al, 2012 2012 ACR Guidelines for Gout
A diagnosis of gout should lead to an assessment of potentially modifiable risk factors (e.g., dietary patterns, alcohol intake, and obesity) and associated comorbidities (e.g., hypertension and dyslipidemia) that may need intervention to reduce gout risk and decrease hyperuricemia.	B	Khanna et al, 2012
Weight reduction with daily aerobic exercise and limiting the intake of red meat, alcohol, certain seafood, and sugar-sweetened beverages help to reduce serum uric acid levels and the risk of gout. Also, low-fat dairy products, vegetables, legumes, and whole grains may decrease the risk of gout by reducing insulin resistance.	C	Choi, 2010
Medications that increase serum urate levels (e.g., thiazide diuretics, low-dose aspirin, niacin, and cyclosporine) should be stopped, if feasible, and alternative medicines used.	B	Khanna et al, 2012

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

OSTEOARTHRITIS

Signal Symptoms: Morning joint stiffness lasting less than 30 minutes; joint stiffness that improves with mild activity. Clinical findings include Bouchard's nodes (PIP joints), Heberden's nodes (DIP joints), and joint crepitus. Weight-bearing joints such as the knees and hips are most affected by OA.

Description: OA encompasses both symptoms and the structural remodeling of articular cartilage with inflammation of synovitis and ligament (Loeser et al., 2012). The resulting joint pain and loss of function is the leading cause of disability in older adults in the United States (Ashford & Williard, 2014). According to the Centers for Disease Control and Prevention (2015), as the U.S. population ages, the number of adults with arthritis is expected to increase to 67 million by 2030.

Etiology: The etiology of OA is unknown; however, it is recognized that OA is not age dependent. However, aging-related changes in cartilage contributes to the remodeling response of the matrix (Loeser et al., 2012). The underlying pain experienced in OA is related to increased pressure in the subchondral bone, the stretching of the joint capsule, pressure on the ligaments, bone spur formation, and inflammatory irritation of innervated tissue surrounding the affected joints (bursitis) or within the joints (synovitis) (Ashford & Williard, 2014; Ayhan, Kesmezacar & Akgun, 2014).

Occurrence: Approximately 26.9 million people in the United States have OA (Arthritis Foundation, 2016). An estimated 49.7% of adults 65 years or older reported provider-diagnosed arthritis from 2010 to 2012 (Barbour et al., 2013).

However, by 2040 an estimated 78 million Americans ages 18 years or older are projected to have doctor-diagnosed arthritis (Hootman et al., 2016).

Age: OA is common in older adults. Of persons ages 65 or older, 49.7% reported doctor-diagnosed arthritis (Barbour et al., 2013). An estimated 33% to 90% of the population over 65 years old is thought to have OA. According to the Arthritis Foundation (2016), one in two adults will develop symptoms of knee OA during their lives.

Gender: Among people 55 years and older, women are affected more often than men. From 2010 to 2012, 26% of women and 19.1% men reported doctor-diagnosed arthritis (Barbour et al., 2013).

Ethnicity: OA of the knee is more common in African American women than in Caucasian women. Also, the prevalence of OA in male and female Alaska Natives is considerably lower than Caucasians. Four million Hispanic adults never reported doctor-diagnosed arthritis as a medical condition when questioned in a patient history as compared to 5.9 million non-Hispanic African Americans (Barbour et al., 2013). The incidence for arthritis for non-Hispanic Asians is reportedly 1.2 million (Barbour et al., 2013).

Contributing Factors: Increasing age, female sex, obesity, previous joint injury (torn meniscus, intra-articular mechanical damage), prior surgeries, occupation, hobby, and prolonged sports activity involving the weight-bearing joints contribute to the development of OA. Patients with concomitant low bone density and related nutritional deficits are found to have OA (Ashford & Williard, 2014). Family history is a known contributing factor for OA of the hand (Chao & Kalunian, 2010; Ringdahl & Pandit, 2011). According to Loeser and colleagues (2012), abnormal remodeling of tissues may be resultant of inflammatory mediators. Other factors include calcium pyrophosphate or uric acid acromegaly, hyperparathyroidism, and diabetes mellitus. Conditions that change joint mechanics, such as untreated hip dislocation, may also predispose an individual to OA.

Signs and Symptoms: Complaints of morning joint stiffness lasting less than 30 minutes or stiffness that improves with activity and accompanying muscle spasms may indicate OA. Persistent pain and limitation of motion in the affected joint may be reported. Bouchard's nodes (nontender nodules of the PIP joints), Heberden's nodes (nontender nodules of DIP joints of the hands and feet), or both may be found (LeBlond et al., 2015). The carpometacarpal joint located at the thumb base is often involved, and crepitus may be elicited; however, it is rare in OA that the metacarpophalangeal joints are involved (Onat et al., 2015). Consider secondary causes of OA if these joints are involved, such as gout, pseudogout, trauma, or occupational risk (Chao & Kalunian, 2010).

In women, erosive OA often occurs in the PIP joints and DIP joints, manifesting red, tender joints that eventually result in joint erosion, joint deformity, and subsequent ankylosis. An autoimmune disorder to consider is also psoriatic arthritis, as these symptoms can mimic inflammatory OA. The MTP joints may be involved in OA. Clinical criteria for patients with OA of the knee include having crepitus (a grinding sensation of the joint) of the affected joint. In addition to pain and morning stiffness, patients with OA of the knee often report limited ROM and bony enlargement of the knee (Ashford & Williard, 2014; Zhang et al., 2010). Some patients may complain of knee locking and unsteadiness. On examination, patients with OA of the hip and knee may present with a counteractive gait; patients are limping to avoid pain of the affected hip and/or knee. Examine bilateral quadriceps muscles for signs of weakness and an internal and/or external hip rotation that may be reduced (Zhang et al., 2010).

Patients with OA of the cervical spine often complain of paresthesia in the arms waking them from their sleep. This sensation generally improves when the limb is lightly shaken. Examination of the cervical spine may show some restricted joint movement and muscle tenderness. When OA affects the lumbosacral spine, patients may report pain across the lower back with radiation to the buttocks and the posterior thigh. If nerve root compression has occurred, patients may complain of pain in the lower leg known as pseudoclaudication. It is important to assess the patient's current functional status and pain level initially and at every subsequent visit and determine if any adaptive equipment is necessary (Allen et al., 2016; Chao & Kalunian, 2010).

Diagnostic Tests: Results of bilateral standing radiographs of the affected areas with OA may reveal joint space narrowing, subchondral cyst formation, subchondral bony sclerosis, and osteophytosis, resulting in proliferative bone spurs. The narrowing of the joint space occurs due to the loss of cartilage. Bony spurs known as osteophytes develop at the margin of the joint as a protective measure for the damaged joint structure (Ashford & Williard, 2014). The cyst formation can be seen on x-ray beneath the surface of the joints. Two views of the affected joint are recommended, with the exception of the sacroiliac joint and pelvis (Chan et al., 2014). Other types of imaging tests, such as ultrasound and MRI, may be used to detect damage to cartilage, ligaments, and tendons, which cannot be seen on x-ray. Arthrocentesis should be considered for joint effusions to rule out crystalline disease or infection. A needle is gently inserted into the joint to withdraw a small amount of synovial fluid from the joint. The specimen should be tested for chemistry, viscosity (thickness), blood cell counts, overall appearances, blood cultures for suspected microorganisms, and presence of crystals to exclude diagnosis of gout and calcium pyrophosphate crystals. The synovial fluid in OA is usually clear, viscous, and has less than or equal to 2,000 white blood cells per μL. Baseline laboratory studies (CBC, liver function tests [LFTs], BUN, and creatinine) should be obtained before initiating long-term drug therapy for monitoring purposes (Chao & Kalunian, 2010).

Differential Diagnosis:

- Osteoporosis (radiographs)
- Metastatic disease (radiographs)
- Multiple myeloma (bone marrow is infiltrated; lytic lesions are common in the axial skeleton); anserine bursitis (knee involvement)
- Polymyalgia rheumatic
- RA
- Crystalline disease
- Septic arthritis
- Reiter syndrome
- Bursitis (Shelton, 2013)

The presence of any systematic symptoms in patients with OA should alert practitioners that the patient may have a developing concomitant inflammatory condition (e.g., polymyalgia rheumatica or RA), crystalline disease, or septic arthritis (Ayhan et al., 2014). When ruling out conditions that mimic OA in specific joints such as hips, knees, and spine, additional conditions must be considered when the presence of pain is described in the specific area. Radiculopathies and neuropathy must be considered in patients with referred pain. Conditions that involve soft tissue and joint support mechanism (e.g., bursitis, tendinitis, and ligament instability and meniscal pathology) should be included in differential diagnosis. Bone tumors and avascular necrosis need to be considered in patients complaining of pain in the large joints of the body (Ringdahl & Pandit, 2011).

Treatment: A multifaceted approach to the treatment of OA remains the mainstay of therapy. The recommendation for nonpharmacological therapies, such as walking, can be beneficial and should be advocated (Allen et al., 2016). Water therapy has been shown to improve the function of patients with OA with no evidence of inflammation (Ringdahl & Pandit, 2011). In noninflammatory OA, acetaminophen is the medication of choice in doses of 2 to 3 g per day (Abdulla et al, 2013). Acetaminophen toxicity has been a growing concern, so caution needs to be taken when recommending acetaminophen. Advise the patient to avoid using multiple acetaminophen products and to restrict alcohol intake (Ali, 2011). For patients who are not getting relief from acetaminophen and exercise, the cyclooxygenase type 2 (COX-2) selective agents should be tried, especially in patients with a history of gastrointestinal bleeding and who are anticoagulated. Starting doses are celecoxib 50 to 100 mg PO twice daily. The lowest starting dose should be used in the older adult, especially in older adult patients weighing less than 50 kg.

Selection of a nonselective NSAID should be based on dosing frequency, toxicity potential, and cost to the patient (Ali, 2011). Older adults should be started on a small dose, increasing the dose gradually. Combination use with a proton-pump inhibitor is recommended (Ali, 2011; Shelton, 2013). The use of NSAIDS should be avoided in older adults with a calculated creatinine clearance less than 35 ml/min. Nonacetylated salicylates such as magnesium trisalicylate can be used, 500 to 750 mg two to three times daily in patients who cannot afford COX-2 medications, though caution is recommended in patients who need cardioprotection (Ali, 2011). Tramadol can be given at 50 mg every 4 to 6 hours; maximum dose in patients age 75 years and older should not exceed 300 mg/day. Other opiates such as codeine and oxycodone can be used for patients with severe OA pain or those who cannot tolerate NSAIDS; however, serious addictions remain a possibility with all opiates, including tramadol. A slow tapering schedule is recommended to avoid withdrawal symptoms (Stoehr et al., 2009; Barsotti, Mycyk, & Reyes, 2003).

Reports have shown that glucosamine and chondroitin sulfate (1,500 mg/1,200 mg per day) may relieve the pain of OA (Sawitzke, Shi, & Finco, 2010). A study by Zeng and colleagues (2015) showed a correlation with safe effectiveness of pain reduction of glucosamine plus chondroitin in knee OA over the COX-2 celecoxib due to side effects of gastrointestinal tract type in the latter. Capsaicin cream 25% applied twice daily to the affected joint has also been shown to reduce pain (Reid, Shengelia, & Parker, 2013). When only one or a few joints are inflamed, intra-articular corticosteroid injections may be beneficial; however, use of these injections should be limited to only a couple times a year (Ali, 2011). The use of topical diclofenac sodium gel (DSG) 1% for OA of the hand was found to relieve the local arthritic pain (Altman et al., 2009). Applying 4 g of DSG four times a day to the knee was found to be effective when rescue acetaminophen was permitted as adjunct to the topical corticosteroid (Baraf, Gloth, Barthel, Gold & Altman, 2011). A patch form of DSG is available with similar efficacy as the gel formulation (Ali, 2011). Viscosupplementation is another nonpharmacological option for patients with OA; an intra-articular injection of the highly viscous joint lubrication has been shown to be effective for 6 months (Abdulla et al., 2013). Patients with severe pain and restricted mobility with OA may benefit from surgical intervention or reconstructive joint surgery (Allen et al., 2016).

Follow-up: Patients should be reevaluated in about 2 to 3 weeks initially, to determine the effectiveness of the treatment. At this time, the patient should be weighed if obesity is a contributing factor; diet and exercise should be reviewed. Response to pharmacological measures can be reevaluated in patients who have been prescribed NSAIDs. A CBC, creatinine clearance, and liver function profile should be ordered at this time, then every 3 months to monitor for elevated liver enzymes due to analgesics. Question the patient about any new onset of dyspepsia, abdominal pain, or bleeding related to medication for OA. Determine if patients are interested in referrals for orthotics and physical and occupational therapy (Shelton, 2013).

Sequelae: Because OA is a slowly progressive disease, joint deformity and functional disability may occur in individuals who have difficulty responding to the therapeutic regimen.

Prevention/Prophylaxis: Physical activity conditions joints and leads to weight maintenance. Weight reduction and avoidance of joint trauma may prevent further joint deformity in the patient with OA. A recent study found that patients who regularly consumed foods that are part of the Mediterranean diet were found to have a lower prevalence of OA of the knee (Veronese et al., 2016).

Referral: Patients may need a referral to a rheumatologist if they have complications from treatment or an unusual presentation of the disease. Patients with involved joint deformities should be referred to an orthopedic surgeon for possible joint replacement (Allen et al., 2016).

Education: Patients need education on specific strategies for ROM and joint protection (Allen et al., 2016). Information in the treatment plan should include the importance of exercise, such as water exercise and aerobic and resistance exercises as tolerated. Other nonpharmacological therapies shown to be beneficial to patients with OA are scheduled rest periods, weight reduction, and the safe use of heat, cold, and medications to control or alleviate pain. According to Allen and colleagues (2016), information should be provided about acquiring walkers, canes, elevated toilet seats, and any orthotics as needed. Lastly, it is essential to review all medications, especially OTC drugs, to avoid duplication of NSAIDs.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Viscosupplementation is another nonpharmacological option for patients with OA; an intra-articular injection of the highly viscous joint lubrication has been shown to be effective for 6 months.	A	Abdulla et al., 2013
Patients with severe pain and restricted mobility with OA may benefit from surgical intervention or reconstructive joint surgery.	C	Allen et al., 2016
The use of topical diclofenac sodium gel (DSG) 1% for OA of the hand was found to relieve the local arthritic pain.	A	Altman, Dreiser, Fisher, Chase, Dreher, & Zacher, 2009
Although the diagnosis of OA is often made without x-rays, results of bilateral standing radiographs of the affected areas with OA reveal joint space narrowing, subchondral cyst formation, subchondral bony sclerosis, and osteophytosis resulting in proliferative bone spurs. The narrowing of the joint space occurs due to the loss of cartilage. Bony spurs known as osteophytes develop at the margin of the joint as a protective measure for the damaged joint structure.	C	Ashford & Williard, 2014
The presence of any systematic symptoms in patients with OA should alert practitioners that the patients may have a developing concomitant inflammatory condition (e.g., polymyalgia rheumatica or RA), crystalline disease, or septic arthritis.	A	Ayhan, Kesmezacar, & Akgun, 2014
Applying 4 g of DSG four times a day to the knee was found to be effective when rescue acetaminophen was permitted as adjunct to the topical corticosteroid.	A	Baraf, Gloth, Barthel, Gold, & Altman, 2011
Patients with involved joint deformities should be referred to an orthopedic surgeon for possible joint replacement.	C	Chao & Kalunian, 2010.
Abnormal remodeling of tissues may be resultant of inflammatory mediators. Other factors include calcium pyrophosphate or uric acid acromegaly, hyperparathyroidism, and diabetes mellitus.	C	Loeser, Goldring, Scanzello, & Goldring, 2012
MCPs are rarely a symptom in OA.	C	Onat et al., 2016
Selection of capsaicin cream 25% applied twice daily to the affected joint also has been shown to reduce pain.	C	Reid, Shengelia, & Parker, 2013
Reports that have shown glucosamine and chondroitin sulfate (1,500 mg/1,200 mg per day) may relieve the pain of OA.	A	Sawitze, Shi, & Finco, 2010
Serious addictions remain a possibility with all opiates, including tramadol. A slow tapering schedule is recommended to avoid withdrawal symptoms.	C	Stoehr et al., 2009

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
A recent study found that patients who regularly consumed foods as part of the Mediterranean diet were found to have a lower prevalence of OA of the knee.	A	Veronese et al., 2016
Safe effectiveness of pain reduction of glucosamine plus chondroitin in knee OA over the COX-2 celecoxib due to side effects of gastrointestinal tract in latter.	A	Zeng et al., 2015
On examination, patients with OA of the hip and knee may present with an antalgic gait; patients are limping to avoid pain on the affected hip and/or knee. Examine bilateral quadriceps muscles for signs of weakness; internal and external hip rotation may be reduced.	A	Zhang et al., 2010
The carpometacarpal joint located at the thumb base is often involved, and crepitus may be elicited; however, it is rare in OA that the metacarpophalangeal joints are involved.	C	Zhang et al., 2008

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

POLYMYALGIA RHEUMATICA

Signal Symptoms: New onset of stiffness and aching in neck, shoulders, pelvic girdle; unable to get out of bed in the morning without extreme difficulty; difficulty in lifting arms over one's head.

Description: Polymyalgia rheumatica (PMR) is a clinical inflammatory syndrome characterized by fatigue, aching, pain, and stiffness, primarily in the neck, shoulders, hips, and pelvic girdle, occurring primarily in older adults. Older adults may relate the presentation of aches and pains to old age and delay seeking treatment (Patil & Dasgupta, 2013). Initially, patients may report unilateral stiffness, but eventually muscle aching with guarded ROM will affect both sides of the body (Muller et al., 2016). Musculoskeletal symptoms have been linked to nonerosive articular and extra-articular synovitis (Paget & Spiera, 2006). Systemic manifestations include low-grade fever, anorexia, depression, weight loss, and malaise (Saad, 2015).

Round cell infiltration and synovial proliferation are found in patients with PMR. Patients are often found to have an elevated ESR and C-reactive protein level (Mackie & Pease, 2013). If both of these inflammatory markers are not elevated, the diagnosis of PMR cannot be discarded (Dasgupta et al., 2010; Mackie & Pease, 2013). There is a close association with patients presenting with PMR developing giant cell arteritis (GCA), a common vascular condition in older adults. It has been suggested that, given the similar clinical pathology of these two conditions, they may actually be manifestations of the same disease (Unwin, Williams, & Gilliland, 2006).

Approximately 50% of patients with PMR go on to develop GCA. Assessing for signs and symptoms of vasculitis early is important to avoid the complications of GCA, namely, irreversible blindness (Salvarani et al., 2008). Patients need to be alerted to the symptoms of GSA, as they are additive to PMR.

Etiology: The etiology of PMR is unknown. A relationship between the presence of the HLA-DR4 haplotype and presentation of PMR has been suggested (Saad, 2015).

Occurrence: PMR affects 100,000 individuals 52 years old and older in the United States. The incidence of PMR increases in northern states that have populations with similar ethnic backgrounds to northern European countries (Saad, 2015). Environmental exposures have been suggested as a trigger for the development of this condition (Caylor & Perkins 2013).

Age: This disease occurs predominantly in adults over 50 years old, with an average age of presentation of 72 years old (Caylor & Perkins, 2013). PMR and GCA occur 10 times more frequently in adults over 80 years old than in adults under 60 years old.

Gender: PMR occurs two to three times as often in women as in men (Hancock et al., 2014).

Ethnicity: PMR is six times more common in Caucasians than in African Americans, Alaska Natives, Asians, and Hispanics (Gonzalez-Gay & Pina, 2015). Patients who are descendants from northern European countries have the highest rate of PMR and GCA, whereas the lowest prevalence has been noted in the Japanese (Matteson & Dejaco, 2017).

Contributing Factors: Advanced age and a possible genetic predisposition are thought to contribute to the development of PMR. Environmental exposures have been suggested as a trigger for the development of this condition (Caylor & Perkins 2013). Patients with a history of myocardial infarction, peripheral vascular disease, and stroke have been shown to have a tendency to develop PMR (Patil & Dasgupta, 2013).

Signs and Symptoms: Often, the onset of PMR occurs in patients who report good health prior to symptom presentation. Patients complain of fatigue and generalized malaise and may attribute the symptoms to influenza or the exacerbation of existing arthritic conditions, such as OA (Buttgereit, Dejaco, Matteson, & Dasgupta, 2016). Additionally, patients usually have bilateral, proximal aching and stiffness in the neck, shoulders, upper arms (Gonzalez-Gay & Pina, 2015), hips, thighs, and lower back, with approximately 95% of patients reporting aching and pain in the shoulder girdle (Caylor & Perkins, 2013). Stiffness occurs in the morning and may last more than 30 minutes to 1 hour; however, on exertion, fatigue and aching may return (Matteson & Dejaco, 2017). Limiting mobility and/or positional changes may also exacerbate the musculoskeletal pain.

When the patient is questioned, there may be no report of actual peripheral joint pain. Musculoskeletal pain and related symptoms may be present about 1 month before the diagnosis is made; however, sudden onset of PMR can occur as well in older adults (Mackie & Pease, 2013). Fever, anorexia, weight loss, apathy, night sweating, fear, and depression are also constitutional symptoms of PMR (Buttgereit et al., 2016). Muscle weakness usually is not elicited on physical examination. Tenderness may be detected on palpation to the muscle groups mentioned earlier. Examine for contracture of the shoulder or adhesive capsulitis due to extended limitation of movement resulting from persistent pain over time (Saad, 2015). Patients may report a decreased ability to grasp small objects. Check for signs of carpal tunnel syndrome, such as paresthesia of the thumb and index and middle fingers due to flexor tenosynovitis (Caylor & Perkins, 2013). Distal extremity swelling with pitting edema may be present over the dorsum of hands, wrists, ankles, and tops of feet (Salvarani et al., 2008). Examine for evidence of claudication in the lower extremities and/or pitting edema (Caylor & Perkins, 2013; Kermani & Warrington, 2011). Diagnostic studies may reveal anemia; checking for signs of pallor is important.

Because GCA is associated with PMR, the work-up should include questions to evaluate for this vasculitis, such as complaints of occipital or temporal headaches, visual disturbances (amaurosis fugax and transient diplopia), high fever, jaw and tongue pain, sore throat, hoarseness, choking, cough, ear pain, painful scalp, and limb claudication as a result of vascular occlusion (Ezeonyeji, Borg, & Dasgupta, 2011; Salvarani et al., 2008). Assess for tender temporal arteries and visual acuity. Funduscopic examination may reveal retinal hemorrhages, cotton-wool patches, and edema of the optic disc (Paget & Spiera, 2006). Neurological findings are also common in patients with GCA due to occlusion of internal carotid or vertebral arteries; neuropathies, transient ischemic attacks, and stroke have been known to occur in patients with extensive GCA (Salvarani et al., 2008). Consider, too, that patients with GCA may develop thoracic aortic aneurysm or even dissection, thus, attention to potential for these presenting conditions is warranted, especially for those who have history of hypertension (Caylor & Perkins, 2013).

Diagnostic Tests: While there are no diagnostic criteria for PMR in patients age 50 years old and older, the European League Against Rheumatism (EULAR) and the ACR proposed the following algorithm for a patient that presents with bilateral shoulder pain and elevated inflammatory markers to differentiate patients with possible PMR from patients with mimicking conditions (Dasgupta et al., 2012a; Dasgupta et al., 2012b)

SCORING	WITHOUT ULTRASOUND	WITH ULTRASOUND
Morning stiffness >45 minutes	2	2
Hip pain/limited ROM	1	1
Absence of rheumatic factor and/or anti-citrullinated protein antibody (anti-CCP)	2	2
Absence of peripheral joint pain	1	1
Ultrasound presence of subdeltoid bursitis or biceps tenosynovitis or glenohumeral synovitis in at least one shoulder, and at least one hip with synovitis or trochanteric bursitis	N/A	1
Subdeltoid bursitis, biceps tenosynovitis or glenohumeral synovitis on both shoulders on ultrasound	N/A	1

Required criteria: age ≥50 years, bilateral shoulder pain and abnormal ESR or C-reactive protein. Using only clinical criteria (without ultrasound), a score of ≥4 out of a total possible score of 6 had a 68% sensitivity and 78% specificity for discriminating PMR from comparison subjects. When ultrasound criteria were included, a score of ≥5 out of a total possible score of 8 had a sensitivity of 66% and specificity of 81% for discriminating PMR from comparison subjects. N/A, not applicable.
Reproduced with permission from Dasgupta et al. (2012a). *Arthritis and Rheumatism, 64,* 943–954.2.

An elevated ESR (more than 40 mm/hr) and C-reactive protein are common findings in PMR. With the trend now to use ultrasound findings in patients whom you are trying to establish a diagnosis of PMR, look for evidence of subdeltoid bursitis, tenosynovitis, hip synovitis, glenohumeral joint bursitis, and trochanteric bursitis (Buttgereit et al., 2016; Mackie & Pease, 2013). For patients with GCA, the ESR generally is found to be 100 mm/hr or greater. There have been cases of patients who had a positive temporal artery biopsy for GCA with normal C-reactive protein (Parikh et al., 2006). Ultrasound of the temporal artery is also being used to diagnosis GCA (Gonzalez-Gay & Pina, 2015). Obtain CBC with indices to determine presence of normochromic, normocytic anemia, and thrombocytosis, which are common in patients with PMR (Caylor & Perkins, 2013).

For patients with symptoms of claudication, imaging studies can be beneficial in determining presence of lower

extremity vasculitis (Kermani & Warrington, 2011). Liver enzyme tests often show elevated alkaline phosphate in patients with GCA (Salvarani et al., 2008). Patients with symptoms of GCA need to be referred for temporal artery biopsy (Caylor & Perkins, 2013; Patil & Dasgupta, 2013).

Considering the need for long-term corticosteroids, diagnostic studies should also include urea and electrolytes, thyroid-stimulating hormone and dipstick urinalysis, and bone density testing if no recent results are available for the patient (Dasgupta et al., 2010).

Differential Diagnosis: The following are differential diagnoses for polymyalgia rheumatica (Aydeniz, Altinda, Öüt, & Gürsoy, 2012; Caylor & Perkins, 2013; Mackie & Pease, 2013; Patil & Dasgupta, 2013):

- Late-onset RA (specifically, the absence of rheumatoid factor and normal anti-citrullinated protein antibody [ACPA] can rule out initial diagnosis of late-onset RA)
- Polymyositis (patients who have demonstrated proximal weakness characteristic of polymyositis would have a positive muscle biopsy)
- Fibromyalgia (generally fibromyalgia begins in younger adulthood and the ESR is normal)
- Hypothyroidism (thyroid hormone is not elevated as a result of PMR)
- Hyperparathyroidism (parathyroid hormone level is not elevated as a result of PMR)
- OA (radiographic changes would demonstrate evidence of OA and not PMR)
- Bursitis and tendinitis (constitutional symptoms generally are not evident in patients with bursitis and tendinitis)
- Rotator cuff injuries
- Carcinomatosis
- Chondrocalcinosis
- Depression
- Systemic lupus erythematosus (antinuclear antibody [ANA] would not be significantly elevated in patients with PMR; slight ANA elevation can be expected in older adults absent of disease)
- Multiple myeloma (patients with multiple myeloma do not respond to corticosteroids and noted absence of Bence Jones protein in urine or serum or urine electrophoresis)
- Infective endocarditis
- Vasculitis
- Parkinson's disease (ESR is not elevated in patients with Parkinson's disease [PD] and corticosteroid therapy would not produce a clinical response to the stiffness experienced
- Polyarticular calcium pyrophosphate deposition disease (arthrocentesis of affected joints would be negative for this condition)
- Late-onset ankylosing spondylitis (radiographic findings of syndesmophytes)
- Inflammatory amyloidosis
- Hypothyroidism (TSH levels)
- Paraneoplastic syndrome from lung cancer (corticosteroid would not produce a clinical response and radiographic imaging would be negative) (Matteson & Dejaco, 2017).

In patients with symptoms typical of PMR and a normal ESR, consider also drug-induced myalgias from lipid-lowering agents, beta blockers, and dipyridamole (Goëb, Guillemant, Vittecoq, & Le Loët, 2004; Mackie & Pease, 2013; Snyder, 1991).

Treatment: If the patient presents with signs and symptoms only of PMR, start low-dose prednisone of 10 to 12.5 mg/day with dosing increasing to 25 mg daily (Matteson & Dejaco, 2017). The patient's weight should be considered when dosing prednisone (Cimmino, Parodi, Montecucco, & Caporali, 2011). Symptoms should begin to resolve after 24 hours, and the ESR, C-reactive protein, anemia, and thrombocytosis should begin to normalize in 7 to 10 days, taking often 1 month to return to normal when higher levels were present in patients with GCA. However, immediate relief from corticosteroids may not be indicative of PMR, as multiple conditions that mimic PMR would also show sign and symptom improvement after prednisone is initiated (Matteson & Dejaco, 2017). Prednisone is tapered off slowly, continuing over months, when the symptoms have resolved and the ESR returns to normal. A slow tapering plan of 1 mg every 4 weeks until discontinuation is recommended and patient is in remission; however, consensus has not been reached as to a tapering schedule (Buttgereit et al., 2016). Patients presenting with visual disturbances or other symptoms of GCA need a higher dose of oral corticosteroids. Prednisone 40 to 80 mg in divided daily doses is the suggested starting dose, again with a gradual tapering depending on symptoms and laboratory values. Treatment may last 1 to 3 years (Dejaco et al., 2015).

Follow-up: Assess the patient for proximal pain, morning stiffness, resolution of constitutional symptoms, and adverse reaction to corticosteroids (Dasgupta et al., 2010). For patients with PMR, the ESR and/or C-reactive protein need monitoring until the levels decrease and previously reported symptoms are alleviated. The CBC can be repeated to determine if the anemia has resolved. Generally, a return to normalization of ESR and C-reactive protein resolves within 2 to 4 weeks of treatment of corticosteroids in patients with PMR and GCA (Gonzalez-Gay & Pina, 2015). Initially, the patients will need to return every couple of weeks to evaluate the clinical response to therapy. This is followed by approximately an every-3-month surveillance to determine response to treatment and any adverse reactions to the long-term corticosteroids. Additional monitoring of urea and electrolytes and glucose should continue every 3 months while on corticosteroids (Dasgupta et al., 2010).

Follow up on the results of the dual-energy x-ray absorptiometry scan. Consider prophylactic therapy to prevent osteoporosis with bisphosphonates with calcium and vitamin D supplementation of 800 to 1,000 IU daily (Patil & Dasgupta, 2013). Patients also presenting with GCA need to be monitored in the same way, with repeated eye examinations as warranted, including examination for cataracts resulting from corticosteroid therapy (Paget & Spiera, 2006). Because patients with GCA are at risk for developing aortic aneurysm, follow-up abdominal examination for aortic aneurysm is needed. This complication is of great concern, especially with patients who are at high risk for developing aortic aneurysms, such as patients who smoke, are hypertensive, and have arteriosclerotic heart disease (Caylor & Perkins, 2013; Gonzalez-Gay & Pina, 2015).

Sequelae: Generally, the patient begins to exhibit a reduction in symptoms within 24 hours after starting treatment, however, one study did indicate that approximately 1 month after treatment with corticosteroids only 55% of patients showed complete response to treatment (Patil & Dasgupta, 2013). Patients should be alerted to the signs of GCA. Patients with untreated GCA are at risk for blindness (Freeman, 2016). Patients should be informed that they must complete all of the prescribed medication to avoid a relapse. Recent systematic review studies on PMR found that female patients, those with elevated ESR rates, and those with peripheral arthritis were at high risk for relapse (Dejaco et al., 2015).

Despite the initial symptom relief, extended treatment with corticosteroids is the mainstay of treatment. Patients often relapse following an initial case of PMR; up to 25% to 50% of patients have a recurrence of PMR (Patil & Dasgupta, 2013). There has also been indication that a percentage of patients with PMR eventually develop late-onset RA (Macke & Pease, 2013).

Patients with PMR may be at risk for developing vascular disorders, especially cardiovascular disorders (Hancock et al., 2014); thus, these patients should be screened carefully. Practitioners will need to continue to monitor patients for any new symptoms following treatment for PMR. When examining a national database of patients with PMR, Muller and colleagues (2013) found a trend of new cancer diagnoses in patients with PMR without pre-existing cancer 6 months after initial diagnosis.

Prevention/Prophylaxis: No preventive measures exist for PMR, but because the disease can recur, patients should be advised to contact their health-care providers when any of the prevailing signs and symptoms reappear. Patients on long-term corticosteroids should be up to date on immunizations for influenza and pneumonia (Caylor & Perkins, 2013; Patil & Dasgupta, 2013).

Referral: For patients with signs of visual disturbance, funduscopic changes, or both, referral to an ophthalmologist is warranted if the patient can be evaluated immediately. If not, these patients should be referred to an emergency department for immediate evaluation for probable temporal artery biopsy. Consultation with a rheumatologist is warranted for patients who have GCA, have spinal involvement, who do not respond initially to corticosteroid treatment, are unable to take corticosteroids, and are candidates for a disease-modifying agent, or have prolonged duration of PMR (greater than 2 years) (Caylor & Perkins 2013; Dasgupta et al., 2010; Patil & Dasgupta, 2013). Consider also referring younger patients (under 60 years) with symptoms of PMR to a rheumatologist (Helliwell, Hider, & Mullen, 2013).

Education: Patients should be taught the precautions needed when steroids are being taken. In addition to being at risk for osteoporosis, the likelihood for developing infections, fractures, diabetes, peptic ulcers, cataracts, depression, and weight gain increases with steroids. Patients should be instructed to include an adequate amount of dietary calcium and vitamin D in their diets (Helliwell et al., 2013). Given the risk of vascular disorders, patients who smoke should be highly encouraged to begin smoking cessation (Hancock et al., 2012). Patients who do not have GCA should be alerted to potential symptoms, such as visual disturbance, headache, temporal and scalp tenderness, jaw pain (especially while chewing), and limb pain (Caylor & Perkins, 2013).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Careful consideration of the patient's weight must be taken into consideration when prescribing corticosteroids to patients with PMR.	A	Cimmino et al., 2011
For patients taking 20 mg of prednisone a day for treatment of PMR, there was a lower percentage of relapse than patients who were prescribed 10 mg of prednisone a day.	A	Buttgereit, Dejaco, Matteson, & Dasgupta, 2016
The use of ultrasound improves diagnostic accuracy of PMR by detecting subdeltoid bursitis, a prominent sign of PMR.	A	Buttgereit, Dejaco, Matteson, & Dasgupta, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

RHEUMATOID ARTHRITIS

Signal Symptoms: Morning stiffness greater than 1 hour; joint swelling and pain, especially at the small joints of the hands, wrists, and feet; symmetrical inflammatory polyarthritis.

Description: RA is a chronic systematic inflammatory process evidenced by symmetrical polyarthritis. It is the most common inflammatory arthropathy. Patients generally

experience episodes of remission and acute exacerbations of this disease. It is an incurable autoimmune condition that affects synovial joints in the body. This ongoing synovial membrane attack results in synovial proliferation; pannus formation; and destruction of bone, cartilage, and ligaments leading to joint damage and deformity (West & O'Dell, 2015).

Pain and stiffness result from the damage and inflammation of the joints and soft tissues. RA is associated with decreased physical function and overall diminished quality of life. Loss of functionality is the after effect of joint destruction. Extra-articular features can result in damage to the heart, lungs, and other vital organs. There are multiple presentations of RA, which can include vasculitis, rheumatoid nodules, scleritis, pericarditis, neuropathy, interstitial fibrosis, Sjögren's syndrome, and Felty's syndrome. RA has been shown to increase cardiovascular risk and reduce overall life expectancy by 30%. Risk for myocardial infarction and stroke is doubled in patients with RA (Ishchenko & Lories, 2016).

Older patients with RA have already sustained significant joint damage, as well as multiple comorbidities. These comorbidities may be directly related to the RA or may be a result of long-term treatments. Osteoporosis may occur after long-term use of corticosteroids. Older people may be hesitant about asserting themselves when exacerbations of their condition occur. Some may find the current approach of self-empowerment difficult. As with all conditions affecting older adults, comorbid factors such as impaired mental, visual, and auditory faculties and muscle weakness must be factored into the patient's individual treatment plan (Ishchenko & Lories, 2016).

Etiology: The exact cause of RA is unknown. There is a demonstrated relationship between the presence of the class II human leukocyte antigen (HLA) gene and RA. This relationship has a familial component. Antibodies for RA (rheumatoid factor) have been identified in the serum many years prior to clinical symptoms (West & O'Dell, 2015).

Occurrence: Approximately 1.3 million U.S. adults over the age of 18 years have RA, representing 0.6% of the population. This is based on the National Arthritis Data Workgroup (Ma, Chan, & Carruthers, 2014).

Age: Although the condition can affect all ages, there is a greater prevalence of RA in the elderly; 2% prevalence over the age of 60 years and 2.8% over the age of 70 years. The mean age of onset is 40 to 60 years, with cases peaking between ages 60 and 80 years for patients developing late-onset RA (Ishchenko & Lories, 2016).

Gender: Prevalence is two to three times higher in women than men. This occurrence can range up to 5% in women over age 65 years; however, late-onset or elderly-onset RA tends to have a more equal gender distribution (Yung, 2017).

Ethnicity: Prevalence is approximately 1% in Caucasians. There is a variance among races from 0.1% in rural Africans to 5% in Native Americans.

Contributing Factors: Risk factors for the development of RA include positive family history, female gender, and the presence of the HLA haplotype DR4 and DRB1. For patients who smoke and have the identified haplotype DRB1, the risk increases (Bang et al., 2010). Numerous studies have demonstrated that smoking is a significant risk factor for RA; the risk is 2.5 times greater for women smokers (Yung, 2017).

Signs and Symptoms: The clinical presentation for patients with longstanding RA before age 60 years reflects the duration of the individual's disease and concomitant conditions. For patients with older adult–onset RA, the presentation may be gradual onset of morning stiffness, swelling, and pain in multiple joints. In others, the attack may be more acute. In older adults, constitutional symptoms with RA may include low-grade fever, weight loss, malaise, and depression. Joint involvement is usually accompanied by pain and early morning stiffness that gradually improves during the day. This differs from the OA type of stiffness that presents after prolonged inactivity. Pain is usually present regardless of weight bearing or movement (Manno & Bingham, 2011).

During examinations of the patient with known RA, the metacarpophalangeal, wrist, PIP, MTP, shoulder, ankle, and elbow joints should be examined for ROM, tenderness, erythema, warmth, and swelling. Clinical joint findings prevalent in patients with a chronic history of RA include hyperflexion of the PIP joints and flexion of the DIP joints (called swan neck deformities), flexion of the PIP joints and extension of the DIP joints (called boutonniere deformity), ulnar deviation of the metacarpophalangeal joint, and knee and ankle effusions (Shah & St. Clair, 2015). A gentle squeeze test for tenderness of joints of hands and feet will vary on exacerbation of condition. The skin should be checked for subcutaneous nodules, which are generally less than 1 to 3 cm in diameter and feel firm and fixed on palpation; these often are found proximal to the elbow. Rheumatoid nodules may also develop in the lungs and pericardium. Extra-articular inflammation from RA may present in the eyes, lungs, and heart. Systemic evaluation also includes an eye examination to check for keratoconjunctivitis, scleritis, and corneal ulcers. Conditions of the lung may include pleuritis and pneumonitis. Pericarditis, a possible manifestation of RA, warrants a cardiac examination. The clinician also should examine the patient for evidence of nerve entrapment and sensory neuropathy. All patients should be questioned about the impact of RA on their lifestyle and current ability to carry out ADLs.

There are several standardized tools used to measure the functional status of patients with RA. Recommended assessment is at least once yearly, more often during active disease. Several commonly used functional status measures are listed in the ACR 2015 treatment guidelines (Sing et al., 2015). One of the more common validated instruments used is the Disease Activity Score (DAS28), measuring disease activity in 28 joints. The initial presentation is usually one of an inflammatory polyarthritis (many painful joints).

Some simple rules will help you to identify patients who have an inflammatory polyarthritis, such as RA, who should be referred promptly to a specialist rheumatology team for diagnosis as soon as possible. Joint destruction may be detectable on radiographic imaging several months after the onset of symptoms. Erosions at the joints may take several years to occur. The ACR, jointly with the EULAR, have established new classification criteria for RA (Aletaha et al., 2010).

Diagnostic Tests: Blood tests, such as rheumatoid factor (RF), ESR, and the C-reactive protein, are useful in the presence of diagnostic indicators on physical examination. The

laboratory test anti-citrullinated peptide antibodies have been shown to be an important indicator for destructive disease when accompanied by a high RF titer. Testing for antibodies to cyclic citrullinated peptide (anti-CCP antibodies) is newer than the RF testing and is associated with higher sensitivity and specificity for RA. The RF is positive in 70% to 80% of patients with RA, with an 86% specificity. Anti-CCP antibody testing for RA is becoming standard and more severe disease manifestations are associated with its positivity (West & O'Dell, 2015). Anti-CCP antibodies may be detected before the RF develops, and are found in up to 40% of RF-negative patients. Radiographs of the hands and feet are needed to look for early signs of erosions, which are an important factor indicating the need to start an aggressive treatment approach aimed at halting further joint damage progression. Additional radiographic findings in RA include soft tissue swelling, symmetrical joint space narrowing, and joint subluxations. A CBC may show normochromic, normocytic anemia, mild leukocytosis, and thrombocytosis (Lee, Beck, & Hall, 2008; Majithia, Peel, & Geraci, 2009; Manno & Bingham, 2011).

Differential Diagnosis: Differential diagnoses for RA include the following (Matjithia et al., 2009; Manno & Bingham, 2011; Olivieri et al., 2009; Villa-Blanco & Calvo-Alen, 2009):

- Polymyalgia rheumatica (rapid response to corticosteroid therapy)
- Crystal arthropathies, including gout and pseudogout (joint aspiration would reveal monosodium urate crystal or calcium pyrophosphate rhomboidal crystals)
- Septic arthritis (Gram stain and culture from arthrocentesis)
- OA (can be ruled out with radiographs)
- Spondyloarthropathy
- Remitting seronegative symmetrical synovitis with pitting edema syndrome
- Arthritis related to connective tissue disease or systemic vasculitis
- Polymyositis (normal muscled enzymes and muscle biopsy would be expected in RA)
- Malignancy-related arthritis
- Hypertrophic osteoarthropathy
- Sarcoidosis
- Infectious arthritis (hepatitis B and C, HIV, and others)

Treatment: The drug management of RA is one of symptom and disease control. Symptom control includes corticosteroid treatment, analgesia, and NSAIDs. Disease control includes traditional disease-modifying therapies, which may be used in combination with corticosteroids. The early or long-term use of corticosteroids remains a controversial topic, chiefly because of their long-term side-effect profile. Traditional nonbiological disease-modifying antirheumatic drugs (DMARDs), which suppress the immune response, include medications such as methotrexate hydroxychloroquine, leflunomide, and sulfasalazine. Biological DMARDs are newer therapies that target specific cytokines of the inflammatory response. They are administered subcutaneously, intravenously, and by mouth (Kalden, 2016).

When using NSAIDs to treat older adults with RA, the NSAID selection should be based on the shortest half-life and the lowest effective dose. Low-dose oral corticosteroids may provide relief and are recommended as a short-term therapy only for less than 3 months, until DMARD treatment is established (Sing et al., 2015).

Disease control treatment approaches start with the prompt introduction of a "traditional" DMARD as a single therapy or a combination of two or more DMARDs. DMARDs suppress the immune response and prevent joint damage, although they may take up to 3 months to have an effect. Traditional DMARDs include:

- Methotrexate (oral, intramuscular, or rarely, subcutaneous given as a once-weekly dose; co-prescribed with folic acid, usually 5 mg once a week when the weekly dose of methotrexate is administrated to decrease nausea, diarrhea, and prevent MTX-associated macrocytic anemia)
- Sulfasalazine
- Leflunomide
- Hydroxychloroquine

There are added risks and long-term consequences of immune suppression associated with drugs such as methotrexate. Tuberculosis and hepatitis testing is done before initiating the immune-suppressing medications. Often more than one DMARD is prescribed at a time for efficiency. Patients require close monitoring for potential toxic effects such as renal and hepatic toxicity, NSAID-induced gastritis, and central nervous system toxicity. RA patients with comorbidities on combinations are at a risk for drug interaction. The new biological agents are used with close observation of benefit versus risk of cancer and fungal infections.

The list of biological DMARD agents in recent years has significantly grown, targeting numerous proinflammatory cytokines such as tumor necrosis factor (TNF) alpha, interleukin (IL)-1, and IL-6. Some of the TNF inhibitor biological agents approved for use are etanercept, adalimumab, infliximab, certolizumab, and golimumab. Other biological therapies commonly used are rituximab and abatacept. The latest consensus statement on biological agents for the treatment of rheumatic disease reported that there is evidence indicating that TNF receptor antagonists are effective for the treatment of RA with methotrexate. There is, however, no evidence that any of the current TNF receptor antagonists are better than any others in its class, so if the first agent fails, another medication of this class can be tried. Severe events include infections, potential worsening of heart failure, and demyelinating disease. TNF receptor antagonists should not be prescribed to patients with current, active infection and should be used with caution in older adult patients with other immune-suppressing conditions.

Recently, studies have concluded that anti-TNF agents could be administrated to elderly patients with RA with similar effectiveness and tolerability as in younger patients (Ishchenko & Lories, 2016). Another cytokine is IL-6, which, importantly, may be involved in the pathogenesis of RA. The blocking of this receptor with the biological agent tocilizumab has led to reduction of the acute-phase response of RA, with elevated cholesterol being the main side effect known at this time (Singh, Beg, & Lopez-Olivio, 2011).

There are many unscientific claims to improving and curing RA in the general marketplace. Support groups and gentle exercise are recommended regardless of age. Cycles of

exacerbations are common. RA is not a diagnosed condition that exists without need of support.

Follow-up: Routine evaluations for patients with RA should include the patient's response to the pharmacological and nonpharmacological therapy. Medications should be adjusted if the patient does not obtain symptomatic relief from the current therapy. Progression of the articular and extra-articular disease should be monitored through physical examination. Any change in the patient's ability to carry out ADLs, as well as his or her psychosocial status, should be evaluated at each visit using any one of the recommended functional assessment tools for RA.

Sequelae: Older adults who have had RA for years eventually experience more severe disease with increased joint deformities. Comorbidities such as septic arthritis, Sjögren's syndrome, Felty's syndrome, and pericarditis may exist in patients with a history of RA. Patients with older adult-onset RA may tend to have a milder course of the disease with periods of remission; however, patients experiencing a more rapid decline have also been reported. Complications from the medication regimen need to be considered and monitored.

Prevention/Prophylaxis: Although RA cannot be prevented, a program of gentle ROM exercises can help maintain function and muscle strength. Water exercise programs have been shown to be an effective therapy for patients with RA (Eversden, Maggs, Nightingale, & Jobanputra, 2007; Hall, Skevington, Maddison, & Chapman, 1996).

Referral: Patients experiencing a period of prolonged inflammation despite therapy or one of the comorbidities mentioned earlier should be referred to a rheumatologist (Wasserman, 2011). Patients can also benefit from referral to a physical therapist and an occupational therapist, to assist them in their exercise programs, splinting needs, additional orthotics, and adaptive equipment requirements, all aimed at maintaining function and independence for as long as possible.

Education: Patients should be taught the importance of incorporating periods of rest and exercise into their daily lives. Medication education is important in RA because of the potential for side effects associated with a complicated drug regimen. Because there is no cure for RA, patients must be skeptical about antidotes promised for RA and check with their health-care providers if they have any concerns about treatments. Patients can contact the Arthritis Foundation (800-282-7800) for information; a booklet, *Overcoming Rheumatoid Arthritis,* is available (Oliver, 2009).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
An increased fear of falling was found in women with RA who had associated foot deformities.	B	Morpeth, Brenton-Rule, Carroll, Frecklington, & Rome, 2016
Late-onset RA may progress slowly over several months or may occur suddenly, mimicking an acute crystalline arthritis.	C	Manno & Bingham, 2011
Mild anemia of chronic disease is a common clinical find in older adults with RA.	C	Wasserman, 2011
Patients in active RA were found to have decreased HDL cholesterol levels and increased incidence of insulin resistance.	B	Ostojic & Bartolovic, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

Ruby, an 86-year-old, thin, osteoporotic woman, gets up onto a stepstool to reach something on the top shelf of her cabinet. She loses her balance and falls onto her left side. She finds that she cannot sit up without pain. Her daughter later finds her and calls an ambulance to take her to the emergency department. Per radiograph, left femoral fracture is present. Surgery is scheduled for open reduction with internal fixation (ORIF). Blood clot prevention injections and pain medications are prescribed. Next, the patient is monitored for postoperative anemia and infection. After discharge, Ruby has rehabilitation for inpatient and then nursing home temporary care for 30 days. She is discharged to her daughter's home with home health and assistive equipment.

Continued

CASE STUDY—cont'd

1. How will you use the information to prepare for today's visit?
   Today's visit:
   Chief complaint: "I am still recovering from my fall last month and now am staying at my daughter's but want to go home."
   Objective: Blood pressure (BP) 100/60 mm Hg, pulse 87, respiratory rate 20 breaths/min
2. What additional subjective data are you seeking?
3. What additional subjective data will you be assessing for?
4. Are there any national guidelines you want to consult for the postoperative and rehabilitation care of this patient with an ORIF related to osteopenia of the left femur?
5. What tests will you want to order as part of this follow-up visit?
6. Are there any new differential diagnoses you need to consider?
7. What is your plan of care?
8. What additional patient teaching may be needed?
9. Are there additional community resources to consider?
10. Will you be looking to add any referrals for this patient?

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CHAPTER 13

Central and Peripheral Nervous System Disorders

Laurie Kennedy-Malone

ASSESSMENT

The neurological assessment of the older adult begins with the initial encounter with the patient, taking into consideration the overall appearance, hygiene, facial expression, gait, and posture (Gladstone & Black, 2002a). Whether it is the masked facial expression of Parkinson's disease (PD) or the facial dystonia of progressive supranuclear palsy (PSP), certain neurodegenerative conditions are often visually initially recognizable and should be considered in the differential diagnosis when patients present with progressive neurological signs and symptoms.

Early in the neurological examination is an assessment of mental status to ensure accuracy of the patient's comprehensive history or chief complaint. The interview should begin with an assessment of level of consciousness, speech pattern, mood and affect, concentration ability, short-term memory, and orientation. This information can usually be obtained during the course of a standard interview. "The key to appreciating a memory impairment or subtle dementing illness is to perceive incongruities among the patient's appearance, dress, language or behavior" (Williams, 2008, p. 236). There are several short screening tools that can be administered to the patient to assess cognition:

- The Mini-Cog is a short dementia assessment that combines three-word recall with clock-drawing capability. Patients are given a total score reflecting accuracy in clock drawing and recollection of the given three words. A score of 0 to 2 is a positive screen for dementia (American Association of Neuroscience Nurses, 2009).
- The St. Louis University Mental Status Examination (SLUMS), a 12-item assessment, has been shown to detect mild neurocognitive disorders. A patient's level of high school is considered when interpreting the score. A score of 19 or below in any patient is indicative of dementia (Tariq et al., 2006). If any questions regarding the mental status arise, family members may be asked for additional information to investigate the patient's cognitive abilities further.
- The Montreal Cognitive Assessment (MoCA Version 7.1) is a tool designed to measure the status of a patient's attention, concentration, executive function, memory, and orientation. It has been tested on patients with a variety of neurodegenerative conditions, as well as those with a history of substance abuse (Berstein, Lacritz, Barlow, Weiner, & DeFina, 2011). The score can be adjusted for patients with a lower level of education (Johns et al., 2010). A score of 26 or higher out of a possible score of 30 is considered normal cognition.

The focused interview for a neurological complaint should include asking about the following common signs and symptoms in older adults:

- Loss of consciousness or presyncope, including a full description of any episodes and their precipitating factors.
- Any episodes of seizures, "spells," shaking palsy, dizziness, weakness or paralysis, tremors, involuntary movements, pain, numbness, paresthesias, gait problems, or restlessness of the legs.
- Any speech or language disturbances or change in vision or hearing, including diplopia, ptosis, or tinnitus reported by the patient or family.
- Any mood swings, new onset of headaches, and change in level of awareness or reversal or alteration in judgment or memory.
- Pertinent family history of any acute neurological or neurodegenerative disease or unknown conditions with specific symptomatology.
- Information about previous occupations and hobbies that may have involved trauma or chemical exposure for each patient.
- All medications (including over-the-counter [OTC] medications), diet, and sleep patterns.
- A careful assessment of social history, including any current or previous substance abuse.

The positive findings determined in the history of present illness and review of systems will guide the episodic neurological examination. A complete neurological examination, however, begins with testing of the cranial nerve function.

Cranial Nerve Function

CRANIAL NERVE I (OLFACTORY)

Make sure to test one nostril at a time using familiar scents. This sense may be lessened or absent because of nasal disease, head trauma, smoking, use of cocaine, or normal aging. It may also be congenitally absent. Patients with hyposmia or anosmia are at risk for safety issues (rancid foods; inability to detect smoke, gas, or other noxious chemicals) and malnutrition (American Association of Neuroscience Nurses, 2009). A distorted sense of smell in both nostrils of familiar odors has been found in patients early in the progression of neurodegenerative conditions such as Alzheimer's disease (AD), whereas a unilateral, nonoccluded loss of smell can be indicative of frontal brain tumor (Williams, 2008). In patients with multi-infarct dementia, studies have shown that the olfactory sense is not as impaired as for patients with AD. For patients with PD, anosmia is one of the earliest signs, but in patients with parkinsonism plus syndromes, such as progressive supranuclear, palsy generally remains intact (Gladstone & Black, 2002a).

CRANIAL NERVE II (OPTIC)

Funduscopic examination will reveal disc or small vessel abnormalities. Assess pupillary reactions to light and visual fields. Presbyopia, the loss of accommodation, is a normal aging change. Older adults experience difficulty with depth perception, and contrast sensitivity is reduced as one ages (American Association of Neuroscience Nurses, 2009). Older patients should be encouraged to continue ophthalmic examinations; the goal should remain for vision to be corrected as needed to 20/20 (Gladstone & Black, 2002a).

CRANIAL NERVES III, IV, AND VI (OCULOMOTOR, TROCHLEAR, AND ABDUCENS)

These nerves may be examined together by testing extraocular movements and convergence. Inspect for ptosis. While weakening of the levator muscle's ability to attach to the lid can be contributed to aging, there are several conditions that cause acquired ptosis, such as myasthenia gravis, diabetes, stroke, brain tumor, and Bell's palsy. Generally, pupils are smaller in an older adult, a condition known as senile mitosis (Larner, 2006). One study found that patients with AD had even smaller pupils than patients of similar age without dementia (Gladstone & Black, 2002a). A hallmark of PSP is supranuclear vertical gaze palsy; however, limitation of vertical gaze and convergence are common age-related changes noted on testing of cranial nerves (Gladstone & Black, 2002a).

CRANIAL NERVE V (TRIGEMINAL)

The motor function of this nerve can be assessed by palpating the temporal and masseter muscles. If the patient has no teeth, this test may be difficult to interpret. The sensory function of this nerve should be tested with sharp and dull stimuli in all three branches: ophthalmic, maxillary, and mandibular. If any abnormalities are noted, light touch and temperature sensation should also be assessed. The corneal reflex should be assessed using a wisp of cotton. Patients who wear contact lenses may have diminished corneal reflexes. Older adults may naturally have decreased lacrimal secretions; certain medications, such as anticholinergics, may exacerbate this condition (American Association of Neuroscience Nurses, 2009; Gladstone & Black, 2002a).

CRANIAL NERVE VII (FACIAL)

Observe the patient's face at rest and continue to observe throughout the examination for any asymmetry. Motor function may be tested by asking the patient to smile, frown, raise both eyebrows, close eyes tightly and resist the examiner's attempts to open them, and puff out both cheeks. A peripheral injury to this nerve, such as Bell's palsy, may affect both the upper and the lower face, whereas a central lesion mainly affects the lower face (Gladstone & Black, 2002a).

CRANIAL NERVE VIII (ACOUSTIC)

Assess auditory acuity using the whisper test. If hearing loss is present, test for lateralization using the Weber test and for air versus bone conduction using the Rinne test. The ear canals should be checked for cerumen impaction or ruptured tympanic membrane, which are leading causes of conductive hearing loss and could contribute to overall hearing loss. Presbycusis, a normal aging change, results in older adults losing the ability to hear at higher tones (Larner, 2006).

CRANIAL NERVES IX AND X (GLOSSOPHARYNGEAL AND VAGUS)

Observe the quality of the patient's voice. Hoarseness may indicate vocal cord paralysis; a nasal quality may indicate paralysis of the palate. Difficulty in swallowing could indicate palatal or pharyngeal weakness. Inspect movement of the palate and pharynx when the patient swallows. With a bilateral lesion of the nerve, the palate will fail to rise. With unilateral lesion, one side of the palate will fail to rise and will be pulled, along with the uvula, to the normal side (Gladstone & Black, 2002a). Test the gag reflex by stimulating the back of the throat on each side. Older adults experience a decrease in taste perception of saltiness, sweetness, sourness, and bitterness (American Association of Neuroscience Nurses, 2009).

CRANIAL NERVE XI (SPINAL ACCESSORY)

Observe for atrophy or fasciculations in the trapezius muscles. Test motor strength bilaterally with a shoulder shrug (Gladstone & Black, 2002a).

CRANIAL NERVE XII (HYPOGLOSSAL)

Inspect for fasciculations of the tongue at rest. Look for asymmetry of movement, atrophy, or deviation from midline. Atrophy or fasciculations suggest peripheral lesions (Gladstone & Black, 2002a). With unilateral lesions, the protruded tongue will deviate toward the affected side (LeBlond, Brown, Suneja & Szot, 2015).

Motor Function

Assessment of motor function should focus on inspection of body position and involuntary movements (tremors, tics, or fasciculations). Note that the incidence of benign essential

tremor increases with aging. Inspect the patient's muscle bulk, comparing sizes and contours of muscles. Pay particular attention to the hands, shoulders, and thighs. Atrophy of the hand muscles may occur with normal aging. Muscular atrophy may also result from diseases of the peripheral nervous system, motor neuron disease, rheumatoid arthritis, muscle disuse, and malnutrition. Fasciculations paired with muscle atrophy often implies front-temporal dementia (Gladstone & Black, 2002b).

Motor assessment also includes inspection of muscle tone, best assessed by the patient's resistance to passive stretch. Inspect tone in all extremities during flexion and extension. Increased tone that is worst at extremes of the range is known as spasticity; resistance that persists throughout and in both directions is known as lead-pipe rigidity. Decreased resistance suggests peripheral nervous system disease, cerebellar disease, or the acute stage of spinal cord injury. If a passive resistant movement in any direction is noted, consider dementia reflecting bilateral frontal lobe dysfunction; this irregular movement is referred to as partonia or greenalite (Gladstone & Black, 2002b). In patients with PD, cogwheel rigidity, ratchet-like jerking movements, can be detected by the examiner on passive flexion and extension of the muscle (Gladstone & Black, 2002b).

Assess muscle strength against resistance in all major muscle groups and grade on a scale from 0 to 5, with 5 being active movement against full resistance. Test the biceps and triceps; grip strength; finger abductions; opposition of the thumb; trunk strength; flexion, extension, abduction, and adduction at the hip; flexion and extension at the knee; and dorsiflexion and plantarflexion of the foot during passive range-of-motion exercises (Gladstone & Black, 2002a). If you note weakness, assess strength against gravity alone or with gravity eliminated. Many clinicians make further distinctions by using plus or minus signs toward the stronger end of the scale (Williams, 2009). Determine if there are patterns of objective weakness that are asymmetrical, distal, and/or proximal only (Gladstone & Black, 2002b).

It is critical to accurately assess the presentation of weakness in older adults because it is a common complaint. It is first important to determine if it is subjective weakness, which is actually found to be fatigue rather than the loss or alteration of normal strength. Patients, however, may experience both fatigue and true weakness. Differentiate between constant versus intermittent weakness, and establish the patterns of weakness:

- Weak arm and leg (same side): hemiparesis or hemiplegia
- Weak legs, normal arms: paraparesis
- All four limbs weak: tetraparesis
- One limb weak: monoparesis
- Proximal muscle weakness (often from a myopathy)
- Distal muscle weakness (often from a neuropathy) (Gladstone & Black, 2002a)

Determining subjectively the patient's limitations in activities of daily living (ADLs) can further help the practitioner establish the severity of the weakness. Ask questions pertaining to function of the upper extremities, such as the ability to lift one's arms over the head for grooming or dressing (proximal upper extremity weakness) versus the ability to use utensils or turn a door handle (distal upper extremity weakness). For lower extremity, proximal weakness questions can be asked about the ability to climb stairs or cross one's legs.

An overall test of weakness and mobility is the Get Up and Go Test. It includes a number of tasks such as standing from a sitting position, walking, turning, stopping, and sitting down, all of which are important tasks needed for a person to be independently mobile, as well as to determine if the patient is having proximal weakness of the lower extremities (Williams, 2009).

Test coordination using rapid alternating movements of the hands, fingers, and feet and point-to-point movements. In some neurodegenerative conditions such as PD, movements may be slow and irregular with imprecise timing. The inability to perform repetitive movements in a rapid rhythmic method is called dysdiadochokinesia (Gladstone & Black, 2002c).

Patients with appendicular ataxia may have difficulty with finger-to-nose testing. The examiner may note undershooting (hypometria) and overshooting (hypermetria) of a target (dysmetria), as well as the decomposition of a movement or the inability to complete a movement (LeBlond et al., 2015).

Gait function should be tested. Assess tandem gait, walking on toes and heels, hopping in place, and shallow knee bends on each side. Romberg's test is to evaluate position sense and is performed with the patient's feet together and eyes closed for 20 to 30 seconds without support. Only minimal swaying should occur in patients with a normal finding (Gladstone & Black, 2002d). This test may be combined with the pronator drift test, in which the patient stands with eyes closed and both arms straight forward, palms up. Normally, this position may be held for 20 to 30 seconds. After instructing the patient to maintain this position, tap the patient's arms briskly downward. The arms should return smoothly to the horizontal position; a downward drift indicates muscle weakness. Allow extra time for the elderly patient to perform gait and coordination maneuvers (Williams, 2008).

Sensory Function

Assessment of sensory function helps to establish a lesion of the sensory cortex, the level of a spinal cord lesion, or the location of a peripheral lesion. Sensory function is tested using pain and temperature (spinothalamic tract), position and vibration (posterior column), and touch (posterior horn). When you locate an area of hypersensitivity or sensory loss, map out its boundaries in detail. Unilateral sensory loss suggests a lesion in the spinal cord or higher pathways; a symmetrical sensory loss suggests a neuropathy such as that experienced by persons with diabetes. Touch and vibratory sensations may diminish because of normal aging (Gladstone & Black, 2002d). Discriminative sensations test the ability of the sensory cortex to analyze and interpret stimuli. These techniques include stereognosis, number identification, two-point discrimination, point localization, and extinction.

Reflexes

During assessment of the reflexes, the patient should be relaxed, with limbs positioned symmetrically. Palpate the tendon to locate position and make sure the tendon is slightly stretched. Strike the tendon briskly and note the speed, force, and amplitude of response. Grade on a scale from 0 to 4 pluses, with the normal response at 2 pluses. The older

adult may have diminished or absent reflexes, usually affecting lower extremities before upper extremities (Gladstone & Black, 2002d).

If the response is asymmetrical, check the force and location of the strike. Nurse practitioners who elicit reflexes on patients with multi-infarct dementia may, however, note brisk asymmetrical reflexes as a sign of focal corticospinal tract dysfunction (Gladstone & Black, 2002d). If the response is symmetrically diminished, the examiner may use reinforcement, in which isometric contraction of other muscles may increase reflex activity. Ask the patient to clench the teeth when testing upper extremities and to lock hands together and pull hands against each other when testing lower extremities (Gladstone & Black, 2002d).

Assess all reflexes: biceps (C5, C6), triceps (C6, C7), brachioradialis (C5, C6), patellar (L2, L3, L4), and ankle (S1). Assess the abdominal reflex by stroking each side of the abdomen both above (T8, T9, T10) and below (T10, T11, T12) the umbilicus (Gladstone & Black, 2002d). Evaluate the plantar response by stimulating the lateral aspect of the sole of the foot from the heel to the ball of the foot, curving medially across the ball. Look for flexion of the toes as a normal response. If dorsiflexion of the big toe and fanning of the other toes is noted, this constitutes a positive Babinski response, indicating a central nervous system (CNS) lesion. This response may also be noted in certain unconscious states (e.g., drug or alcohol intoxication, postictal state) (LeBlond et al., 2015).

The clinical significance of positive findings when testing primitive reflexes such as palmomental, glabellar, and grasp remains controversial. Numerous studies have shown that primitive reflexes are common findings in the general aging population without any CNS disorder. If a patient when tested has several primitive reflexes versus just one, the likelihood of underlying neurological condition increases (Gladstone & Black, 2002d).

Abnormal or Involuntary Movements

With the incidence of neurodegenerative conditions increasing with older adulthood, the practitioner needs to assess for any involuntary movements such as fasciculations, spasms, chorea, tremors, tics, and athetosis. Abnormal movements could also be a side effect of psychotropic medications that can cause extrapyramidal symptoms (Gladstone & Black, 2002c). Make sure to document completely all findings of the neurological examination, especially if additional diagnostic testing is indicated. The identification of subtle nervous system abnormalities has been shown to be associated with the risk of stroke and even death in healthy older adults (Inzitari et al., 2008). Nurse practitioners should distinguish pathology from age-related neurological changes. One study showed that over 50% of community-dwelling older adults were found to have at least one extrapyramidal sign (Gladstone & Black, 2002b).

BRAIN TUMOR

Signal Symptoms: Headaches, with or without nausea and vomiting; seizures; changes in mental function or personality; motor or language deficits; visual problems.

Description: A brain tumor is a malignant or benign neoplasm of the brain or its supportive structures, which may arise from glial cells, blood vessels, connective tissue, meninges, pituitary, or pineal glands. Gliomas and meningiomas are the most common type of brain tumor in adults (National Brain Tumor Society, 2016). Other types of primary brain tumors include CNS lymphoma arising from lymphocytes, medulloblastoma, oligodendroglioma, and ganglioma from the neurons (National Brain Tumor Society, 2016; Perkins & Liu, 2016).

Etiology: The etiology of brain tumors is largely unknown. CNS tumors have been associated with rare genetic conditions, including Li-Fraumeni syndrome, neurofibromatosis 1 and 2, and Von Hippel-Lindau disease (Michaud & Batchelor, 2016; Perkins & Liu).

Occurrence: Malignant brain tumors are a rare cancer, but the incidence has been increasing over the past 50 years. In 2014, 1.4% of all new cancer cases were primary brain tumors (Perkins & Liu, 2016). There were an estimated 23,380 new cases and 14,320 deaths. The incidence of a new brain tumor is 6.4 per 100,000 people each year. The 5-year survival rate is 33.4% (Perkins & Liu, 2016).

Age: Peak prevalence is between 55 to 64 years (Perkins & Liu, 2016).

Gender: Occur more commonly in males than females (American Cancer Society, 2017; Michaud & Batchelor, 2016; Perkins & Liu, 2016).

Ethnicity: Caucasians have a higher incidence compared to African Americans across most histologies. Exceptions include meningioma, tumors of the pituitary, and craniopharyngioma, where rates for African Americans significantly exceed those of Caucasians (Michaud, Schiff, & Batchelor, 2016). Malignant brain tumors are less common among Asians and Native Americans (Michaud, Schiff, & Batchelor, 2016).

Contributing Factors: High-dose ionizing radiation is the only proven risk factor. The increase in CNS lymphoma is associated with the increase in immunocompromised persons. Prior radiation and chemotherapy are associated with brain tumors (Michaud & Batchelor, 2016; Perkins & Liu, 2016).

Signs and Symptoms: Signs and symptoms are produced by tumor infiltration of nervous tissue, displacement of brain structures by tumor mass, or increased intracranial pressure (ICP) (Wong & Wu, 2016). Headaches occur more than half of the time and are often associated with seizures, visual disturbances, and persistent nausea with vomiting. Generalized headache can be associated with increased ICP.

Memory loss, cognitive changes, and motor or language deficits are present in about 30% of brain tumor patients. Psychomotor function may slow in patients with primary brain tumors. Personality changes may be marked or subtle,

including changes in mood, concentration, and intellectual functions (Wong & Wu, 2016).

Partial motor, sensory, or grand mal seizures occur in slightly more than 30% of patients with brain tumors. New-onset focal seizures in individuals over 40 years old are suspicious for brain tumor until proved otherwise. Syncope due to an increase in ICP can mimic a seizure with a few tonic-clonic jerks. Papilledema may be present in increased ICP, though less commonly in the elderly (Wong & Wu, 2016).

Focal neurological changes are related to the area of the brain invaded by tumor. Frontal and parietal lobe tumors may cause changes in memory, behavior, and cognitive function. Memory, hearing, vision, and emotions are affected most often by temporal lobe tumors. Symptoms of temporal lobe tumors may mimic symptoms of affective or psychotic thought disorders. Visual changes can occur with occipital lobe tumors, in addition to speech, motor, and sensory changes for left-sided occipital masses and an inability to grasp abstract concepts for right-sided occipital masses. Lesions in the cerebellum affect balance and coordination. Pituitary tumors may present with the symptoms of hypothyroidism, hypercortisolism, diabetes insipidus, or visual changes (Wong & Wu, 2016).

Diagnostic Tests: The following tests are recommended for a person with suspected brain tumor:

- Complete blood count (CBC) to assess for anemia or infection.
- Thyroid-stimulating hormone (TSH) because hypothyroidism or hyperthyroidism can be associated with mental status changes or hypothyroidism with pituitary tumors.
- Venereal disease research laboratory (VDRL) if neurosyphilis is a possibility.
- Chemistries to assess for electrolyte and chemical imbalances.
- Gadolinium-enhanced magnetic resonance imaging (MRI) of the brain is the preferred radiological examination for either brain tumor or suspected bleeding in the brain. Computed tomography (CT) scan with contrast enhancement is acceptable in special circumstances, such as when a patient is uncooperative or when MRI is unavailable. Magnetic resonance spectroscopy (MRS) is used in specialized circumstances.
- Positron emission tomography (PET) scan is not used to diagnose tumors of the CNS, but may have an ancillary role to examine for metastasis or after treatment.
- Electroencephalogram (EEG) is useful for evaluating possible seizure activity.
- Lumbar puncture should not be performed before MRI or CT scan because of the potential for fatal brain herniation to result. Lumbar puncture can yield pressure readings and cerebrospinal fluid for cytology, protein, glucose, and tumor markers.
- Testing blood for tumor markers, α-fetoprotein, or β–human chorionic gonadotropin is useful for the diagnosis of some brain tumors.
- Histological examination of a biopsy specimen of brain tissue may be preferred when feasible (Wong & Wu, 2016).

A careful history of symptoms is invaluable. Complete physical examination should include skin survey for stigmata of neurocutaneous syndromes or melanoma, lymph node examination, abdominal examination for hepatomegaly or splenomegaly, and rectal examination with guaiac stool testing. Breast and pelvic examinations in women and cardiopulmonary examination are recommended. The neurological evaluation should include a mental status evaluation, testing for cognitive deficits or memory loss, and assessment for personality changes. Family members may be able to provide clues about subtle personality changes. Ophthalmic examination is essential to assess for papilledema, although this may not be present in patients 55 years old and older. Test also for asymmetry of strength, sensation, visual fields, reflex activity, cranial nerve function, and radicular signs.

Differential Diagnosis:

- Stroke: There is acute onset of headache with persistent focal neurological deficits. CT scan or MRI is diagnostic.
- Aneurysm: In acute rupture, there is a sudden intense headache, often with signs of meningeal irritation. MRI is preferred for diagnosis.
- Arteriovenous malformation: There is a chronic unilateral throbbing headache with no prodromal or associated symptoms. CT scan or MRI may be diagnostic.
- Meningitis: Headache usually is associated with fever and nuchal rigidity. Cerebrospinal fluid is obtained through a lumbar puncture for diagnosis.
- Abscess: Abscess is often associated with fever or other signs of infection.
- Syphilis: Neurosyphilis may occur at any time during infection and up to 35 years later. Symptoms vary according to the area infected. Diagnosis is based on cerebrospinal fluid abnormalities and a reactive serological test for syphilis.
- HIV: Various neurological manifestations can be caused by the virus or opportunistic infections; the incidence of CNS lymphoma is increasing in these patients.
- Subdural hematoma: Persistent headache occurs after trauma, with poorly defined intellectual impairment. This may be ruled out by noncontrast CT scan or MRI.
- Postconcussion syndrome: A dull, constant headache occurs within 24 hours of trauma. The patient may complain of loss of concentration, giddiness, irritability, and anxiety.
- Trauma: History of injury to the head or neck is present.
- Temporal arteritis: This is usually characterized by a slow-onset headache that is worse at night, temporal in more than one-half of patients, often with jaw claudication and temporal artery tenderness as well as an elevated erythrocyte sedimentation rate (ESR).
- Normal-pressure hydrocephalus: This condition often presents with dementia, gait disturbances, and incontinence of bladder or bowel.
- Multi-infarct dementia: This may present with impaired cognition and symptoms of upper motor neuron disease in patients with a history of hypertension, atrial fibrillation, diabetes, carotid artery disease, or smoking.
- Alzheimer's disease: This presents with progressive dementia, memory disturbances, and behavioral changes, which are usually gradual over years.
- Chemical poisoning: History of exposure to chemicals, such as carbon monoxide, pesticides, or industrial materials, is present.

- Migraine: Migraine is characterized by an often-unilateral headache with a pulsating quality, associated with nausea and vomiting, photophobia, or phonophobia; prodromal or aura symptoms may be present (Perkins & Liu, 2016).

Treatment: Treatment of brain tumor includes surgery, radiation, chemotherapy, glucocorticoids, anticonvulsants, immunotherapy, and gene therapy (NCCN, 2016).

Follow-Up: Prognosis depends on the tumor type, patient age, functional neurological status, extent of resection, tumor location, and extent of metastasis at diagnosis. Tumor grade, patient age, functional status, and complete surgical resection are the important prognostic factors for survival in people with malignant gliomas (Michaud, Schiff, & Batchelor, 2016). During the first year after treatment, the patient should have a focused history and physical examination, including a neurological and funduscopic examination every 3 months. MRI of the brain is recommended 2 to 6 weeks after radiation, then every 2 to 4 months for 2 to 3 years, and then less frequently (NCCN, 2016). Patients receiving palliative care should be seen as necessary for pain and symptom control; hospice care is recommended for these patients.

Sequelae: Various neurological deficits, personality changes, seizure disorders, and chronic head pain can result from brain tumor. Paraplegia, hemiplegia, and quadriplegia are also consequences of brain tumor. Bradycardia, hypertension, and respiratory arrest can occur with increased ICP. Brain herniation is a life-threatening emergent complication.

Prevention/Prophylaxis: Instruct patients to avoid radiation exposure.

Referral: Refer patients to a neurosurgeon, oncologist, radiation oncologist, and hospice, as appropriate.

Education: Genetic counseling is warranted in hereditary syndromes of brain tumor.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Gadolinium-enhanced MRI of the brain is the recommended diagnostic examination for suspected brain tumor.	A	NCCN, 2016 Wong & Wu, 2016
Patients with persistent headache that is different from their usual pattern with protracted nausea, vomiting, seizures, neurological symptoms, or positional worsening should be evaluated for brain tumor.	A	Wong & Wu, 2016
Hospice should be offered if life expectancy is less than 6 months, and in patients who are not candidates for surgery or chemotherapy, have deteriorating neurological status, and have poor or worsening functional status.	C	Perkins & Liu, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

PARKINSON'S DISEASE

Signal Symptoms: Tremor, rigidity, akinesia, impaired postural reflexes.

Description: PD is a chronic progressive neurodegenerative disorder resulting in the loss of dopamine-producing cells in the substantia nigra, located in the basal ganglia. The loss of the inhibitory neurotransmitter dopamine results in an imbalance with the neurotransmitter acetylcholine, which is primarily responsible for worsening symptoms leading to immobility. Symptoms of PD include resting tremor, rigidity, akinesia, bradykinesia, and impaired postural reflexes (resulting in postural instability) (Schoneburg, 2014). Subtle motor impairments may precede the development of overt clinical signs and symptoms by many years. The Braak hypothesis (Chen, 2013) indicates six pathological stages of PD, but clinical symptoms do not appear until stage 4 to 6. This stage correlates with 60% to 80% loss of substantia nigra neurons and striatal dopamine. It is the second most common neurodegenerative disease. It affects motor and autonomic function, mood, and cognition.

Etiology: The cause of idiopathic primary PD is unknown. Genetic causes, viral causes, mitochondrial dysfunction, oxidative stress, and possibly chronic inflammation may cause PD, and these causes are being researched. Secondary PD (Parkinson syndrome) may be caused by head injury, toxins, heavy metals, herbicides, pesticides, dopamine-depleting drugs, encephalitis, hypoparathyroidism, and Wilson's disease. Parkinsonism (idiopathic PD is the most common form) includes known variants of PD, including multisystem atrophy (MSA), olivopontocerebellar atrophy (OPCA), Shy-Drager syndrome (SDS), progressive supranuclear palsy

(PSP), corticobasal ganglionic degeneration (CBGD), Lewy body dementia (LBD), vascular parkinsonism, and neuroleptic-induced parkinsonism.

Occurrence: Approximately 1.6% of the population (100 to 150 cases in 100,000) is diagnosed with PD. In the United States, 60,000 people are diagnosed each year. A recent study suggests that this is an underestimate. The study identified a sharp rise in PD prevalence from 170 per 100,000 in 2000 to 256 per 100,000 in 2007 (Chillag-Talmor et al., 2011).

Age: PD is predominantly discovered in adults ages 55 to 69 years; however, 15% of newly diagnosed cases occur in persons younger than 49 years old.

Gender: The age-standardized prevalence and incidence of PD is greater in men than in women for all races, with a mean sex ratio of 155 men per 100 women (Wright-Willis, Evanoff, Lian, Criswell, & Racette, 2010).

Ethnicity: PD can be found in all cultures. The cumulative incidence of PD is 45 per 100,000 for people (40 to 65 years old). The age- and gender-adjusted rates per 100,000 was 54 for Caucasians, 40 for Latinos, and 23 among African Americans (Dahodwala, 2010).

Contributing Factors: Stress is not a direct causative factor in PD, but it may contribute to worsening symptoms. A major life stressor is often reported with diagnosis of PD (a move, retirement, loss of a job, a fire, or loss of a child). Carbon monoxide exposure, MPTP (an illicit drug), and a combination of genetic and environmental factors contribute to the diagnosis of PD. It is known that in certain family's genetic factors do result in risk of developing PD.

Signs and Symptoms: An initial clinical symptom of PD is a resting tremor, initially unilateral, that disappears with movement. Tremor also is noted in the lips, chin, and tongue. Patients may make a motion of the thumb and forefinger known as pill rolling. Although common, tremor is not present in every patient with PD. Occasionally, a symptom of internal tremor is reported but may not be observed. Rigidity, as shown by increased resistance to passive range of motion, is a classic sign and is known as cogwheel rigidity, which can be noted in the wrists and elbows. Patients experience a slowing of movements termed *bradykinesia*. Patients also report having difficulty initiating movement. Autonomic movements, such as the normal pattern of arm swinging during ambulation, are decreased. Symptoms have an asymmetrical onset.

Other associated manifestations include masked facial expression, decreased blinking, and delayed ability to show facial expressions. Patients also experience an associated softening of the voice, hypophonia, and drooling (due to less frequent swallowing). The size of the patient's handwriting often changes (micrographia). Flexed posture is common, with a bowed head, kyphotic back, and a trunk that leans forward. The gait may become faster with the body propelled forward (festination). Ask the patient about associated symptoms, such as shuffling gait, constipation, seborrhea, myalgia, impotence, and urinary incontinence.

Depression is common and affects 30% to 40% of Parkinson's patients (Blonder & Slevin, 2011). Dementia may occur with or without associated Alzheimer's involvement in 20% to 30% of people with PD. Patients and family members may report mood swings, hallucinations, and insomnia. Executive function may be impaired (i.e., the ability to plan, organize, pay attention, remember details, strategize, and manage time and space). Non-motor symptoms are common, including orthostasis, bladder and sexual dysfunction, sleep disorders, fatigue, and anorexia. Early in the diagnostic phase, the patient's cognitive status needs to be assessed, given the prevalence of dementia developing in patients with PD. Assess the patient's mood as well.

The neurological examination will reveal most PD symptoms, and noting the patient's movement, coordination, and gait is crucial. Note any differences in speed in alternating simultaneous pronation and supination of the hands, as well as in finger tapping. The pull test (Di Giulio, 2016) is a physical test of postural stability (balance). The examiner stands behind the patient, informs the patient that he or she is going to be pulled with two hands on the shoulders backward, then the patient is pulled, and the recovery is documented. Note whether the postural reflexes are impaired or absent. Ask the patient to ambulate and observe gait, including arm swing, as well as tandem (heel-to-toe in a straight line) walking, turning abruptly, and walking on toes. Observe stature for kyphosis and forward bending of the head. Smell is commonly impaired (hyposmia) early in PD; therefore, evaluating cranial nerve I (olfactory nerve) could be useful (Doty, 2012). Check the patient's ocular movements; impairment of upward or downward gaze is common in progressive supranuclear palsy but not in PD.

Examine the patient's upper and lower extremities. Test for range of motion and strength, noting any cogwheel rigidity. Strength generally does not deteriorate despite the rigidity. Tendon reflexes are almost always normal. To test for tremor, have the patient rest the arms on the legs while seated. Note the frequency and amplitude of the tremor. Hypotension is common in patients with PD. An orthostatic blood pressure check with the patient lying, sitting, and standing is recommended at each visit (Sanchez-Ferro, Benito-Leon, & Gomez-Esteban, 2013). The presence of two cardinal signs—bradykinesia and resting tremor, rigidity, or postural instability—is essential to establishing the diagnosis of PD.

Diagnostic Tests: PD is established by history and physical examination, including the neurological examination (Seppi, 2011). There is a growing interest in neuroprotection to slow disease progression, and there are developing technologies that may detect PD in first-degree relatives earlier. These patients may benefit from discoveries of effective neuroprotective therapies, including antioxidants and exercise. Histological markers are not widely used to establish the diagnosis of PD. A simple blood test detecting phosphorylated alpha-synuclein, which is common is people with PD, has been discovered but it is not yet widely available. A DaTSCAN (ioflupane [^{123}I]) injection, also known as phenyltropane, can indicate the presence of PD and is reimbursed by Medicare. An MRI may be ordered to rule out suspected brain lesions or abnormalities, such as normal-pressure hydrocephalus.

Differential Diagnosis: Olfactory dysfunction, rapid eye movement (REM) sleep behavior disorder, autonomic dysfunction (constipation), and depression are strongly linked to PD.

- Drug-induced parkinsonism: Confirmed by withdrawal of suspected medication, such as neuroleptics and metoclopramide.

- Cortical basal ganglionic degeneration: Distinguished by unilateral coarse tremor, ideomotor ataxia, limb dystonia, and lack of response to levodopa.
- Essential (benign familial) tremor: Tremor is an action tremor 6 to 8 Hz compared with PD tremor, which is 3 to 6 Hz.
- Huntington's disease: This disorder is genetic in origin and characterized by chorea, clumsiness, and cognitive decline.
- Shy-Drager syndrome: This syndrome is distinguished by early and prominent autonomic nervous system dysfunction and poor response to dopamine.
- Progressive supranuclear palsy: Supranuclear gaze abnormality, facial spasticity, and axial rigidity are present; tremor is usually absent.
- Creutzfeldt-Jakob disease: A rapidly progressive syndrome of mental deterioration, lead-pipe rigidity, myoclonus, aphasia, apraxia, and hallucinations.
- Normal-pressure hydrocephalus: Gait apraxia, urinary incontinence, and dementia are present.
- Striatal nigra degeneration: Autonomic dysfunction and dystonia are present.
- Olivopontocerebellar atrophy: Distinguished by inherited parkinsonism, progressive ataxia, dementia, difficulty with balance, and dysarthria.
- Multisystem atrophy: Includes olivopontocerebellar atrophy, striatal nigral degeneration, and Shy-Drager syndrome (Pruitt, 2014).

Treatment: Drug therapy focuses on correcting the imbalance of dopamine and acetylcholine.

- Patients with mild disease and no interference with ADLs may not require treatment.
- With tremors and rigidity causing impairment of the patient's ability to perform ADLs and a disability level that is mild-to-moderate, treatment may include amantadine, which is thought to augment dopamine release from presynaptic nerve terminals or to inhibit dopamine reuptake. Initial dose is usually 100 mg with breakfast. In 5 to 7 days, add amantadine 100 mg with lunch, then increase daily dose to 300 mg.
- Supplements are not prescribed but often used as self-treatment for PD symptoms, including co-enzyme Q10 (ubiquinone), vitamins D and E, and S-adenosylmethionine (SAMe), resveratrol, and creatine, as well as low-dose melatonin (.3 mg) for sleep.
- Always start low and slow with all medications. Asses for and treat depression early because it is common in PD and can contribute to worsening of symptoms.
- Patients with disability require carbidopa and levodopa to replenish the depleted dopamine in the brain by increasing the dopamine precursor levodopa to stimulate dopamine receptors. Carbidopa and levodopa can be started using 25/100 (carbidopa 25 mg, levodopa 100 mg) Sinemet two tablets once a day. The dosage can be increased by two tablets every 3 to 5 days until the total daily dose is two tablets of Sinemet 25/100 three times a day.
- Dopamine agonists include ropinirole (Requip) 0.25 mg three times a day and pramipexole (Permax) 0.125 mg three times a day.
- Give the catechol-O-methyltransferase inhibitor (COMT) entacopone (Comtan) 200 mg with each dose of levodopa or 1,600 mg daily. Blocking the 3-O-methylation of levodopa prolongs the action of the levodopa dose; thus, this inhibitor allows a larger amount of levodopa to reach the brain. Tolcapone (Tasmar) 100 mg three times daily can cause fulminant liver failure (check liver function); unless it has been most effective (given with levodopa) within 3 weeks, it may be best to try another COMT inhibitor or Stalevo. Stalevo is a combination of carbidopa, levodopa, and entacapone. Stalevo dosages reflect the amount of levodopa (50, 75, 100, 125, 150, and 200) in each pill combined with carbidopa and Comtan 200 mg.
- When the dosage of levodopa reaches 600 to 1,000 mg (1 g) per day, an adjunct medication is recommended. Large doses of levodopa over time can contribute to the on-off phenomenon and abnormal involuntary movements (AIMs). The agonists may be used alone or to extend the usefulness of levodopa. Taking medication on a consistent schedule lowers the likelihood of AIMs.
- Selegiline (Deprenyl, Eldepryl), a monoamine oxidase–B inhibitor, inhibits the enzyme responsible for inactivating dopamine. Adding selegiline 5 mg/day (increasing to 10 mg/day within 1 week) can improve the wearing-off effect of levodopa (doses to be given before lunch). Rasagiline (Azilect) is a monoamine oxidase–B inhibitor that can be used alone or with Sinemet. It is recommended once a day and is available in 0.5 mg or 1 mg doses. "The advantage of agonist therapy is the reduction in the prevalence of motor fluctuations" (Smith, Wichmann, Factor, & DeLong, 2011).
- Apomorphine (Apokyn) and carbidopa/levodopa (Parcopa) are "rescue" medications, which means they are used when a person "freezes," becoming unable to move. Apokyn is a subcutaneous injectable (2 to 4 mg), and Parcopa is an immediate-release formulation that dissolves on the tongue. Doses start with 25/100 and are also available in carbidopa/levodopa ratios 10/100 and 25/250.
- Duodopa is administered by using a pump and PEG-J or naso-jejunal tube. It is a gel and most often indicated for persons with late PD, as it offers the possibility of more on time when the patient can function.
- Rytary is an extended release carbidopa/levodopa and is used for people who experience symptoms of wearing off.
- Neupro (Rotigotine) is a dopamine agonist patch.
- Other medications that may be used for symptoms (including memory, hallucinations hypotension, and constipation) include memantine, rivastigmine, quetiapine fumarate, midrodrine, linaclotide, and lubiprostone, as well as MiraLAX and Senokot. Orthostasis and constipation may also be treated with adequate water intake as well as medication.

Deep brain stimulation (DBS) has been approved for treatment of Parkinson's symptoms in the United States. Stimulation of the thalamus is used for tremor-predominant PD, and stimulation of the globus pallidum and subthalamic nucleus is used for bradykinesia, akinesia, dyskinesia, and rigidity.

(DBS is not appropriate for those with profound depression and dementia.)

Treatment must include exercise from day of diagnosis. Exercise activity promotes neuroplasticity. Neuroplasticity is the brain's ability to reorganize and repair throughout a lifetime. All sustained enriched activity exerts an acute and sustained effect on neuroplasticity. Consistent exercise of 20 to 30 minutes, 5 or more days a week; or two to three times a week for 45 to 60 minutes, is therapeutic (Bray, 2016). Exercise can be divided throughout the day into 10-minute sessions, if necessary. Moderate intensity exercise is adequate to obtain benefit (Loftus, 2014).

Follow-Up: Ask the patient if any medication seems to be wearing off or if the patient has had any falls, because medication and surgery do not improve balance, and falls are associated with disease progression. Ask about sleep disturbances and constipation, because they are linked to PD, increase stress, and compromise quality of life. Depression is common in PD patients, adversely affecting quality of life, and must be treated aggressively.

Sequelae: PD results in chronic and progressive immobility. As the disease advances, the patient is at risk of injury from falls due to postural instability. The hazards of immobility (Olson, Johnson, & Thompson, 1990) can prove deadly, including megacolon from constipation, aspiration pneumonia, pulmonary embolism, decreased appetite from inactivity (anemia), urinary tract infections, and sepsis from disruption in skin integrity. With medical therapies, surgery, and consistent exercise, a patient with PD can have a normal life expectancy.

Prevention/Prophylaxis: No preventive measures exist for idiopathic PD, but exercise can promote neuroplasticity and is safe (Rosenthal, 2013).

Referral: Refer to a neurologist patients with unusual presentation or bothersome complications and patients who do not respond to the initial medication regimen. Patients benefit from a physiatrist who can recommend therapy to address axial rigidity and postural instability, as well as adaptive equipment (rolling walker). Home health-care nurses have techniques to enhance independence while maintaining a safe environment. Refer patients with voice changes or difficulty swallowing to a speech pathologist.

Education: Provide patients and family members with written information about PD, including information about local support groups. Contact the National Institute of Neurological Disorders and Stroke for educational resources at www.ninds.nih.gov/disorders/Parkinsons_disease/parkinsons_disease.htm. For care of patients with PD, contact the Mount Sinai Beth Israel Medical Center Parkinson and Movement Disorders Center, New York City at 212-844-8482.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Olfaction dysfunction is a "pre-clinical" sign of Parkinson's disease.	A	Doty, 2012
Depression affects 30%–40% of patients with PD.	A	Blonder & Slevin, 2011
Beyond the benefits on physical health, exercise gives patients a more active role in the management of their PD.	A	Rosenthal & Dorsey, 2013
New method to investigate postural instability in PD using computer-controlled motors to deliver precise pulls to the shoulders of subjects while standing.	A	DiGiullo et al., 2016

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PERIPHERAL NEUROPATHY

Signal Symptoms: Paraesthesia, allodynia, sensory ataxia, autonomic dysfunction, distal symmetrical sensorimotor dysfunction.

Description: Peripheral neuropathy is a disorder of the peripheral nervous system resulting in sensory, motor, and autonomic dysfunction. Peripheral neuropathy is often used interchangeably with the term *polyneuropathy* to describe sensorimotor abnormalities affecting many nerves in a symmetric pattern (ClinicalKey, 2014). Neuropathy is defined as nerve damage with symptoms ranging from numbness or tingling, to pricking sensation (paresthesia) or muscle weakness, allodynia (abnormally sensitivity leading to an exaggerated intense or distorted experience of touch (National Institute of Health [NIH], 2016). Peripheral neuropathy occurs in various forms, depending on the type of nerve damage involved, sensory, motor, and autonomic, or combination. Symptoms of peripheral neuropathy may

be experienced over a period of days, weeks, or years (NIH, 2016; Mayans & Mayans 2015; Levine & Saperstein, 2013).

Peripheral nerves are composed of bundles of long neuronal axons as they exit the nervous system. Some nerves are contained within a myelin sheath generated by Schwann cells, whereas others are unmyelinated (Azhary, Farooq, Bhanushali, Jajid, & Kassab, 2010). Peripheral nerves exhibit distinct characteristic responses to stressors: Wallerian degeneration, axonal degeneration, and segmental demyelination. The specific mechanisms inducing the pathological changes are unknown. Wallerian degeneration is characterized by degeneration of the axon distally to a focal lesion, which interrupts the continuity of the axon. This is often seen in focal mononeuropathies due to trauma or nerve infarction (Shields, 2010).

Axonal degeneration, often referred to as a dying-back syndrome, is due to axonal degeneration at the distal area of the axon. This is usually symmetrical and with progression; the axons usually degenerates in a distal to proximal gradient. Axonal degeneration is the most common type of pathological reaction in generalized polyneuropathies and is often metabolic related (ClinicalKey, 2014).

Segmental demyelination is focal degeneration of the myelin sheath while the axon remains intact. This is found in focal mononeuropathies and in generalized sensorimotor or motor neuropathies. Segmental demyelination occurs in some hereditary polyneuropathies and acquired segmental polyneuropathies, and is often immune-mediated or inflammatory (Shields, 2010).

Peripheral neuropathies related to Wallerian degeneration or axonal degeneration have a less favorable prognosis because axons must regenerate and reinnervate muscle, sensory organs, blood vessels, and other structures before recovery is seen. However, recovery of segmental demyelination is rapid due to remyelination occurring quickly, thus restoring normal nerve conductivity of the axon and function (Shields, 2010). In general, the underlying pathophysiology of peripheral neuropathy is presented as an axonal loss (the most common), demyelinating, or both (ClinicalKey, 2014).

Etiology: Peripheral neuropathy has a variety of systemic, metabolic, toxic, and hereditary causes, as well as infections. The most common treatable causes are listed here (Jones III, Lawson, & Backonja, 2016; ClinicalKey, 2014):

- Diabetes mellitus (type 1 and 2): Diabetes mellitus frequently affects the peripheral nervous system and is the most common cause of neuropathy (Isenberg et al., 2015). Prolonged hyperglycemia can damage nerves and contribute to a number of health issues, including the development of diabetic neuropathy, in which nerves are permanently damaged (Otis, Sanderson, Hardway, Pincus, & Tun, 2013). Diabetes is a leading cause of peripheral neuropathy in the United States (NIH, 2016). Approximately 60% to 70% of people with diabetes have some mild to severe forms of nervous system damage that can affect sensory, motor, and autonomic nerves, and present with various symptoms.

The most common peripheral neuropathy subtype, distal symmetric polyneuropathy (DSP), had a prevalence of 3.4% to 3.7%, increasing to 4.2% to 5.3% in those older than 75 years (Callaghan, Price, & Feldman, 2015).

- Moderate-heavy use of alcohol: Alcohol frequently causes distal, painful, sensory peripheral neuropathy due to the adverse neurotoxic effect and associated nutritional deficiencies (ClinicalKey, 2014).
- Guillain-Barre syndrome: This is an immuno-mediated demyelinating peripheral neuropathy, the most common cause of acute generalized motor weakness. It typically presents in an ascending pattern.
- Cryptogenic peripheral neuropathy is responsible for up to 40% of patients referred to an acute care setting (ClinicalKey, 2014).

Other causes (not inclusive) to consider include hypothyroidism/hyperthyroidism, HIV, hepatitis B and C, Lyme disease, syphilis, cytomegalovirus, Epstein-Barr virus, varicella zoster virus, heavy metals (arsenic, lead, thallium, mercury, gold), chemotherapy, medications (colchicine, amiodarone, metronidazole, isoniazid, thalidomide, disulfiram, lithium), multiple myeloma, environmental exposure (Agent Orange, ethylene oxide, acrylamide, hexacarbons, carbon disulfide),vitamin $B_{12,}$vitamin B_6 deficiency and toxicity, thiamine deficiency and copper deficiency, cirrhosis, celiac disease, Crohn's disease, ulcerative colitis, rheumatoid arthritis, sarcoidosis, systemic lupus, Charcot-Marie-Tooth disease, amyloidosis, and hypertriglyceridemia (which may exacerbate diabetic neuropathy) (ClinicalKey, 2014).

Trauma, the most common acquired injury, from motor vehicle accidents, falls, sports, related surgical procedures (nerves can be partially or completely severed, crushed, compressed, or stretched), repetitive tasks (leads to entrapment neuropathies, carpal tunnel, ulnar neuropathy) may result in irritation to tendons, muscles becoming inflamed and swollen, constricting the narrow passageways through which nerves pass, contributing to a nerve dissection from accidental injury or surgery (NIH, 2016). Some causes of peripheral neuropathy will be idiopathic (Trivedi et al., 2013).

Occurrence: The overall prevalence of peripheral neuropathy is not well established due to the heterogeneity of the various peripheral nervous system diseases. An estimated 20 million people in the United States have some form of peripheral neuropathy (NIH, 2016) with 8% being older than 55 years and 24% being older than 65 years (Levine & Saperstein, 2013). Peripheral neuropathy affects at least 20% of adults with diabetes mellitus (Farhat & Yezback, 2016).

Age: The incidence of peripheral neuropathy increases with age and affects up to 10% of persons over age 65 years (ClinicalKey, 2014; Callaghan et al., 2015).

Gender: A diagnosis of peripheral neuropathy is not gender specific (Mold et al., 2004).

Ethnicity: Peripheral neuropathy occurs more frequently in non-Hispanic African Americans and Mexican Americans than in Caucasians (Gregg et al., 2007).

Contributing Factors: Hyperlipidemia, especially hypertriglyceridemia, may exacerbate diabetic neuropathy (ClinicalKey, 2014). Military herbicides such as Agent Orange, used between 1961 and1971 during the Vietnam War, can be a contributing factor (Yi, Hong, Orr, & Yi, 2014). Yi and colleagues (2014) conducted a large-scale study on 111,726 Korean Vietnam War veterans, exploring the prevalence of various disorders of endocrine, nervous, circulatory,

respiratory, and digestive systems, and the adverse effects of Agent Orange on human health. They examined associations between Agent Orange exposure and certain morbidities after adjusting for major health-related covariates using data from national health insurance claims dated January 2000 through September 2005.

This study supported the long-term neurotoxic effect of Agent Orange exposure and demonstrated that neurological disorders, such as spinal muscle atrophy, Alzheimer's type disease, and peripheral neuropathy, occurred with long-term exposure to Agent Orange in Korean and Vietnam War veterans. However, Yi and colleagues (2014) advise that the results should be interpreted with discretion for various reasons, such as possible false-positive associations due to analyzing several diseases simultaneously (Yi et al., 2014). The Veterans Affairs Administration (VA) presumes veterans' early onset of peripheral neuropathy to be related to their exposure to Agent Orange or other herbicides during service when the disease appears within 1 year of exposure to a degree of at least 10% disabling by VA regulations (www.publichealth.va.gov, 2016). The prevalence of various neurological disorders, except demyelinating diseases including multiple sclerosis, were elevated in the high-exposure group compared to the low-exposure group. Future research is needed to better understand the long-term effect of Agent Orange exposure on human health (Yi et al., 2014).

Signs and Symptoms: Sensory nerve damage symptoms may vary because the sensory nervous system has a broad range of functions. Large sensory fibers enclosed in myelin are responsible for vibration, light touch, and position sense. Damage to these nerves impairs touch, resulting in general decrease in sensation, which is felt mostly in the hands and feet. With progression of damage, sensory loss ascends the lower extremities in a symmetrical pattern. When sensory loss is at or above the level of the knee, the axons to the distal fingertips become involved, creating a length-dependent process beginning in the upper extremities (Shields, 2010). It is common for patients to feel that they are wearing gloves and stockings when they are actually not.

Paresthesia and dysesthesias (sense of numbness, tingling, prickling, pins and needles sensations, and band-like sensations on feeling and pressure) are not uncommon complaints (Shields, 2010; Otis et al., 2014). During the neurological sensory examination, there will be a distal to proximal loss of various sensation, along with a predominant complaint of pain and thermal sensation, suggesting a small fiber neuropathy (Shields, 2010; NIH, 2016). Loss of position sense contributes to gait disturbances and difficulties with coordination of complex tasks, such as buttoning clothing or maintaining balance with eyes closed (NIH, 2016). Patients will be at high risk for pressure sores, trauma, and burns not perceived by them. Pain may be described as allodynia, a dull, aching sensation, and intermittent lancinating pulses of pain (Trivedi et al., 2013; NIH, 2016; Otis et al., 2014).

Small fiber neuropathy presents with distal painful paresthesias with intact deep tendon reflexes and normal vibration and position sense. The symptoms can be multifocal or proximal, such that isolated small fiber neuropathy can present as chest or trunk, face, and scalp pain, or vary asymmetrically involving only a single limb (Levine & Saperstein, 2013). Progression to the upper extremities may occur within weeks or months from the onset. These symptoms are usually found in diabetes, glucose intolerance, metabolic syndrome, hypothyroidism/hyperthyroidism, alcohol abuse, and vitamin B_{12} deficiency. In distal symmetrical sensorimotor polyneuropathy there is hyperalgesia or allodynia, especially in the feet, with pain triggered by the least amount of activity, such as the touching of a bed sheet (Farhat & Yezback, 2016). The pain may migrate to the hands years after onset and is chronic. Presenting symptoms include loss of function (loss of sensation to pinprick, temperature, vibration, and proprioception), tingling or burning (early sign of nerve damage), and pain starting symmetrically in the lower extremities. Small cell fiber neuropathy is also associated with early autonomic dysfunction (Mayans & Mayans, 2015; Levine & Saperstein, 2013).

Autonomic nerve damage may include the inability to control bowel and bladder function (constipation, diarrhea), orthostasis (occurs with advanced autonomic involvement), arrhythmias, dry eyes, and dysphagia (NIH, 2016; Mayans & Mayans, 2015). Early in the course of autonomic neuropathy, patients may notice anhidrosis (absence of sweating or reduced sweating) of the head and neck areas due to a distal to proximal gradient pattern occurring in the limbs and thorax, reflecting a compensatory hyperhidrosis (Levine & Saperstein, 2013).

Motor nerve damage is commonly associated with muscle weakness, painful cramps, fasciculation (uncontrolled muscle twitching visible under the skin), muscle atrophy, and decreased reflexes (NIH, 2016). It produces weakness in a distal to proximal gradient with a length-dependent axonal degeneration. As with sensory neuropathy, it begins with the toes and, as polyneuropathy spreads, it ascends up to the distal lower extremities to the level of the knees, at which time motor involvement in the hands is observed. There is weakness in dorsiflexion of the feet and ankles, which can result in partial or complete foot drop that usually causes the feet to slap while walking and predisposes the person to stumbles and falls, especially on uneven floor surfaces (Shields, 2010; Levine & Saperstein, 2013; Mayans & Mayans, 2015).

Identification of peripheral neuropathy begins with a detailed history and physical examination (Jones III et al., 2016), as the lack of symptoms does not rule out neuropathy. About 50% of patients with diabetes are asymptomatic (Farhat & Yezback, 2016). Begin with an extensive medical history. Ask about the following:

- Positive symptoms that occur first due to either overactive or inappropriate nerve conduction activity, such as cramping, twitching, burning, and painful paresthesia described as burning, stinging, pins and needles, stabbing and electric shocks, freezing, aching, and broken glass, which mainly affect small fiber nerves (Mayans & Mayans, 2015; Trivedi, Siverstri, & Wolfe, 2013; ClinicalKey, 2016). Pain is often worse at the end of the day during rest or when lying in bed. A variety of positive symptoms often coexist (Trivedi et al., 2013; ClinicalKey, 2016).
- Negative symptoms due to reduced nerve conduction activity, such as numbness, weakness, balance difficulty, difficulty walking, and poor sensation (Mayans & Mayans, 2015; Trivedi et al., 2013) typically begin in the distal feet or toes and, with time, progress upward

to the knees; hands may be affected at this time (ClinicalKey, 2016). Sensory neuropathy is the most common cause for painful or burning feet (Trivedi et al., 2013). Sensations of a sock rolled up at the end of the shoe, walking on glass or golf balls, or sunburn may be used to describe the symptom and often reflect large fiber nerve involvement (ClinicalKey, 2016).

- Autonomic symptoms include early satiety, constipation or diarrhea, impotence, sweating abnormalities, sexual dysfunction, and orthostasis (Mayans & Mayans, 2015; ClinicalKey, 2016).

A thorough social history provides guidance to the identification of potentially reversible causes of peripheral neuropathy. Ask about the following:

- Sexual history to determine risk of HIV
- Current occupation (exposure to chemical toxins such as heavy metals, lead, or arsenic)
- Military background (exposure to chemicals such as Agent Orange)
- Lifestyle habits (drug, alcohol, nicotine abuse)
- Dietary habits (vitamin deficiencies, anemias, OTC supplements, herbs)
- Infectious diseases (viral or other infectious diseases, vaccinations (NIH, 2016; Mayans & Mayans, 2015)

Obtaining the personal or family history during childhood may reveal clumsiness, suggesting a hereditary neuropathy such as Charcot-Marie Tooth disease, which is a common hereditary neuropathy and a demyelinating peripheral neuropathy seen in up to 150,000 persons in the United States. Ask about signs and symptoms of type 1 and 2 diabetes, thyroid disease, renal disease, and hepatic or autoimmune diseases (Mayans & Mayans, 2015; ClinicalKey, 2016). Ask about a history of chemotherapy with respect to type, dose, and timing (ClinicalKey, 2016; Mayans & Mayans, 2015).

When performing the physical examination, include a skin examination of the hair and mucus membranes, because many infections, toxins, autoimmune disorders, and genetic causes of peripheral neuropathy cause changes in these areas. The skin may present with decreased hair over the involved areas, especially the distal lower extremities, and discoloration of the toes and feet, ranging from pale to an erythematous or dusky color. The skin may be unusually dry or diaphoretic or cool to touch. Mees lines (pale bands across the fingernails) or palmar desquamation may be indicative of arsenic, thallium toxicity, or chemotherapy for cancer (ClinicalKey, 2016; Mayans & Mayans, 2015). Look for muscle atrophy of the intrinsic hands and feet muscles. Look for high arches, hammer toes, and inverted champagne bottle-like legs, which may suggest a hereditary neuropathy. Pes caves with hammer toe deformities are commonly seen in Charcot-Marie Tooth disease. Charcot joints are also seen in diabetic peripheral neuropathy (Mayans & Mayans, 2015; ClinicalKey, 2016).

Sensory examination includes response to light touch, temperature, painful stimuli, vibration, and proprioception, which may indicate small or large sensory nerve fiber impairment (NIH, 2016). Compare both sides for any differences and determine if they are normal, increased, or decreased (Voltrubec & Thong, 2013; ClinicalKey, 2016). Look for reduced sensation, hyperesthesia, or dysesthesias to pinprick and light touch in a stocking-glove distribution (ClinicalKey, 2016). A monofilament testing of the toes is helpful for DSP and can identify asymptomatic patients at risk for developing DSP (Izenberg et al., 2015). While performing the foot examination, inspect the feet for cracks, ulcers, and callosities; evaluate the quality of pedal pulses; assess the skin for temperature changes, as well as for any bony or muscular changes. Sensory perception of the various locations of the soles of the feet is checked with a 10-gauge monofilament. Monofilament testing only assesses large fiber neuropathy rather than mild neuropathy from small fiber deficits. Weak or absent pulses suggest circulatory issues and will require further evaluation (Farhat & Yezback, 2016). Sensory ataxia with finger–nose dysmetria (the inability to control the range of movement when trying to touch an object with an index finger) may also exist when the eyes are closed; this usually resolves when the eyes are opened. A wide-based unsteady gait due to sensory loss may exist in some patients (ClinicalKey, 2016).

Also during the examination, there may be vibratory loss and reduced proprioception in distal-to-proximal gradient, absent or reduced deep tendon reflexes (usually with the ankles first as it progresses upward), and positive Romberg sign, suggesting a sensory neuropathy (ClinicalKey, 2016). Vibratory sensory impairment and ankle reflex testing test large fiber function (Farhat & Yezback, 2016).

Remember that a loss to sensation in the feet often occurs with healthy aging; therefore, such loss of sensation in the older adult, especially with diabetes mellitus, is not always indicative of peripheral neuropathy. Age-related sensory loss is usually generalized and involves more proximal areas of the lower extremities, as in the knees. In peripheral neuropathy, sensory loss changes occur more in the feet. This is a key distinction in the diagnosis of diabetic peripheral neuropathy in elderly patients (Liu, Brooks, McCosker, Molyneaux, & Yue, 2013).

The motor examination involves evaluating muscle tone, strength testing, reflexes, and coordination. Motor weakness is manifested by the reduced ability to move toes and, with severe disease, patients have proximal weakness, including bilateral foot drop and reduced grip strength (ClinicalKey, 2016; Votrubec & Thong, 2013).

A comprehensive history and physical examination provides guidance to the type of neuropathy and diagnostics testing. Peripheral neuropathy can be classified as polyneuropathy, mononeuropathy, and multiple mononeuropathy.

Mononeuropathy is focal involvement of one nerve resulting from a localized process, as in compression or entrapment, such as carpal tunnel syndrome (Mayans & Mayans, 2015). Carpal tunnel syndrome/medial neuropathy, ulnar nerve compression at the elbow, and compression at the peroneal nerve of the fibular head and lateral femoral cutaneous nerve at the inguinal ligament are often seen in persons with diabetes, compared to 10% of the general population (Izenberg & Saperstein, 2013). Multiple mononeuropathy (mononeuritis) may be due to several nerves coming in contact with each other, which can occur simultaneously or sequentially, as in vasculitis (Mayans & Mayans, 2015)

Polyneuropathy involves two or more contiguous nerves, usually symmetric and length dependent, creating a stocking-glove pattern paresthesia. Many times, long nerves are affected initially, which leads to complaints of symptoms in

the legs and feet, followed by the hands at a later time. This is commonly seen in those with diabetes (Mayans & Mayans, 2015; Trivedi et al., 2013).

Diagnostic Tests: Although there are no standardized tests for peripheral neuropathy, the consensus of expert opinion is that the following laboratory testing should be routinely ordered: CBC, sedimentation rate, fasting glucose, and possibly hemoglobin A1c, thyroid studies, renal function, and vitamin B_{12} level. If the vitamin B_{12} level is under 400, methylmalonic acid and homocysteine level should be ordered due to their great diagnostic yield (Mayans & Mayans, 2015; Jones III et al., 2016; Votrubec & Thong, 2013). Additional tests are done if the diagnosis remains unclear after initial testing and a careful history, such as HIV testing in high-risk populations.

The differential diagnosis of peripheral neuropathies depends on the results of nerve conduction studies and electromyography (NCS/EMG), which are complementary and should be performed together (Levine & Saperstein, 2013; Mayans & Mayans, 2015). These are considered an extension of the neurological examination and yield valuable information in differentiating between a single or multiple mononeuropathy or polyneuropathy, enabling the classification of a polyneuropathy as axonal, demyelinating, or mixed, because certain neuropathies affect nerves in a different pattern. Electrodiagnostic testing is crucial to diagnosing DSP (Izenberg et al., 2015).

Nerve conduction studies measure the degree of damage in large nerve fibers by revealing symptoms caused by degeneration of the myelin sheath or axon. EMG detects abnormal electrical activity in the motor neuropathy, which helps to differentiate between muscle and nerve disorders (NIH, 2016; Jones III et al., 2016). Diagnostic findings may show an absent or reduced sural nerve sensory response, which will be confirmatory in most cases; an absent or low-amplitude H response; absent medial plantar mixed sensory response (if the patient is under 40 years old); fibrillation in intrinsic foot muscles; and demyelinating peripheral neuropathy, which show prolonged latencies, delayed conduction velocities, and absent late responses while axon-loss peripheral neuropathy is demonstrated with absent or reduced amplitudes with normal conduction velocities and latencies. Once the clinical pattern of the neuropathy is identified, further diagnostic testing may be done in a strategic manner (Levine & Saperstein, 2013) by asking:

- Is it acute, subacute, or chronic?
- Is it affecting motor, sensory, or autonomic nerves, or a combination?
- If the process is purely sensory, does it affect small fibers, large fibers, or both?
- Is the disease proximal or distal?
- Is the disease symmetric or asymmetric?
- Is the disease lower motor or upper motor involved? (Levine & Saperstein, 2013)

Additional testing includes MRI to evaluate for tumors, herniated disc, or causes of neuropathy; nerve biopsy (to evaluate for degree of nerve damage, but is difficult to perform and may result in neuropathy itself); and skin biopsy (reveals damage present in small fibers in contrast to anatomical nerve biopsy) (NIH, 2016). Serum protein electrophoresis or serum immunofixation electrophoresis are recommended in patients over age 60 years because monoclonal gammopathy is a common cause of peripheral neuropathy in this age group. If the history and physical examination warrants, laboratory testing for paraneoplastic, autoimmune, infections, or toxic etiologies can be performed (Mayans & Mayans, 2015; Votrubec & Thong, 2013). Other tests may include alanine transaminase (Votrubec & Thong, 2013).

Once the evaluation is complete and it is believed that the neuropathy is idiopathic, it is important to continue to follow-up with the patient intermittently. Most idiopathic neuropathies are very slow, progressive, and do not involve weakness. If the patient is thought to have a progressive disabling condition in terms of weakness, then further evaluation should include a lumbar puncture, as well as nerve biopsy (Levine & Saperstein, 2013).

Differential Diagnosis:

- CNS lesions such as syrinx (cyst in the spinal cord or brain) (Jongen, Hans, Benzon, Huygen, & Hatrick, 2014)
- Spinal cord injury. Spinal cord disease such as transverse myelitis or cervical cord compression can also lead to bilateral sensory abnormalities in a symmetrical manner, but is distinguished from peripheral neuropathy by the presence of upper motor neuron abnormalities such as hyperreflexia (ClinicalKey, 2014; Jongen, Hans, Benzon, & Hatrick, 2014)
- Tumor (Jongen et al., 2014)
- Cerebrovascular accident (Jongen et al., 2014)
- PD (Jongen et al., 2014)
- Restless leg syndrome (Jongen et al., 2014)
- Venous insufficiency (Jongen et al., 2014)
- Radiculopathy (bilateral L5-S1) may cause compression of the nerve root due to degenerative disease, resulting in pain and paresthesias of the distal feet (ClinicalKey, 2014)
- Peripheral vascular disease (may cause numbness and tingling in the distal extremities, but reflexes and motor strength are normal on examination as well as EMG testing) (ClinicalKey, 2014)
- Multiple sclerosis with lesions affecting the sensory nervous system either intracranial or within the spinal cord may present as peripheral neuropathy, although central cause of sensory changes may typically present in a non-length-dependent and asymmetric pattern (ClinicalKey, 2014).

Treatment: Management of peripheral neuropathy involves treatment of the underlying cause and supportive therapy. Treating peripheral neuropathy or neuropathic pain begins by identifying and treating the underlying causes or cause as early as possible to prevent the development of chronic treatment-resistant neuropathic syndrome, as well as to help alleviate the symptoms (Jongen et al., 2014; Trivedi et al., 2013). Peripheral nerves have the ability to regenerate axons (as long as the nerve cells had not died), which can lead to functional recovery over time. Correcting the underlying cause can result in the neuropathy resolving on its own as the nerves regenerate (NIH, 2016). Neuropathic pain (Serpell et al., 2013) that is due to a direct result of a lesion or somatosensory system disease (Jongen et al., 2014; Jones III

et al., 2016; Trivedi et al., 2013) is widespread, with an estimated prevalence of over 1% of the general population.

Neuropathic pain can be triggered by a variety of diseases and conditions; however, the mechanisms causing the discomfort have specific characteristics related to the damage and/or dysfunction of the nervous systems (Serpell et al., 2013). Neuropathic pain is characterized by a change in sensitivity to sensory stimuli that may lead to an attack of evoked pain which comes in various forms, as hyperalgesia (increased pain from a stimulus that normally causes pain); hyperalgesia (diminished pain in response to a normal painful stimuli); hypoesthesia (decreased sensitivity to stimulation); and allodynia (pain due to a stimulus that does not normally cause pain, as in diabetic neuropathy) (Jones III et al., 2014; Trivedi et al., 2013). Pain in peripheral neuropathy may not be limited to one region, but can occur in surrounding areas called secondary hyperalgesia (as in herpes zoster). This is not referred pain, as there is no nerve lesion involved in referred pain (Jones III et al., 2014).

The frequency, intensity, and quality of neuropathic pain differs with the etiology of the pain (Trivedi et al., 2013). There is no "gold standard" in the treatment of neuropathic pain. (Jongen et al., 2014). Therefore, management of pain will involve a combination of treatment strategies to alleviate symptoms (Jones III et al., 2014). The provider should substantiate their treatment of choice based on available scientific evidence and mode of action, and the patient's situation (Jongen et al., 2014). Medications proved most effective for managing neuropathic pain provide 30% to 50% improvement (Jones III et al., 2013). Neuropathic pain is present in 65% to 80% of idiopathic polyneuropathies and up to one-third of diabetics and patients with AIDS, but is uncommon in para protein-associated neuropathies (Trivedi et al., 2013). Because individual medications have limited benefits, medications are often combined in therapy to better improve the outcome, followed by other treatment modalities (Jones III et al., 2016). Medications used to treat chronic neuropathies fall under the following categories: antidepressants, anticonvulsants, antiarrhythmics, and narcotics (NIH, 2016).

PHARMACOLOGICAL AGENTS FOR NEUROPATHIC PAIN

Gabapentin/Pregabalin: These drugs work by acting on the alpha2 delta sub unit of calcium channel on central sensory neurons. Clinical trials have shown a significant effect in the treatment of post-herpetic neuralgia and diabetic peripheral neuropathy (Jones III et al., 2016). Gabapentin has been approved by the U.S. Food and Drug Administration (FDA) for the treatment of diabetic peripheral neuropathy (Farhat & Yezback, 2016). Gabapentin used in combination with morphine to treat neuropathic pain has an opioid-sparing effect (Jongen et al., 2014). Gabapentin has been shown to be the most favorable agent based on safety and efficacy in a recent meta-analysis conducted by Rudroju and colleagues (2013). The significant dose-limiting side effect with gabapentin is sedation and dizziness. Other side effects include weight gain, blurred vision, peripheral edema, and balance instability (Farhat &Yezback, 2016; Trivedi, 2013; Jones III et al., 2016). The general starting dose for gabapentin is 100 to 300 mg bid. The recommended dose is 300 to 1,200 mg three times a day. This medication is increased by 300 to 400 mg increments every 5 to 7 days to 3,600 mg daily divided into three to four doses (Trivedi et al., 2013). The pharmacokinetics are variable and, therefore, the dose is individualized according to the patient. Reduce dose in those with renal impairment (Votrubec & Thong, 2013; Trivedi et al., 2013). For diabetic peripheral neuropathy, the starting dose may be 100 to 300 mg daily, with the target dose ranging between 900 to 3,600 mg daily in divided doses (Farhat & Yezback, 2016).

Pregabalin, approved by the FDA for treatment of diabetic neuropathy in 2012 (Farhat & Yezback, 2016), has also been approved for pain from spinal cord injury (Jones III et al., 2016). The starting dose is 50 mg three times a day, with the recommended dose being 50 to 300 mg bid (Trivedi et al., 2013). Neuropathic pain effective dose has been around 75 to 600 mg per day in divided doses (Jones III et al., 2016.). Higher doses of pregabalin at 900 mg daily have been studied, with the outcome showing that doses greater than 300 mg resulted in adverse effects, such as somnolence, dizziness, confusion, ataxia, and weight gain. Pregabalin is habit forming and classified as a schedule V drug. Avoid stopping this medication abruptly because symptoms of insomnia, nausea, headache, diarrhea, and seizures may occur. Gradually taper down the dose over a week (Farhat & Yezback, 2016).

Tricyclic Antidepressants (TCAs) (Nortriptyline, Desipramine, Imipramine): These are the most extensively studied agents for neuropathic pain and generally the first-line option for neuropathic pain related to post-herpetic neuralgia and especially for diabetic peripheral neuropathy (Farhat & Yezback, 2016; Trivedi et al., 2013). TCAs have been listed on the Beers criteria recently as potentially inappropriate agents due to the potential for adverse side effects. Less common adverse side effects include weight gain, heart block, and risk of cardiac arrhythmias. Desipramine, nortriptyline, and imipramine have lower incidence of anticholinergic effects and have been shown to be efficacious for the treatment of diabetic peripheral neuropathy (Farhat & Yezback, 2016). TCAs primarily inhibit the reuptake of serotonin and noradrenaline, but have additional effects on antagonism of N-methyl-D-aspartate, serotoninergic histaminergic, muscarinic, and alpha-adrenergic receptors in controlling pain.

Amitriptyline has been first-line medication for diabetic peripheral neuropathy for many years, however, compared with other TCAs, this agent has a higher likelihood of causing anticholinergic effects. There are specific recommended doses of this agent for patients with renal or hepatic impairment, although the drug is renally excreted (Farhat &Yezback, 2016).

Desipramine has the lowest incidence of adverse side effects, making this agent the most favorable of the TCA agents for patients unable to tolerate other TCAs, and is preferred in the older adult. The initial dose is 25 mg at bedtime, titrating up every 3 to 7 days for a single dose of 100 to 125 mg at bedtime. Educate patients on the avoidance of abrupt discontinuation of the medication to avoid withdrawal symptoms (Farhat & Yezback, 2016). Tricyclic antidepressants used to treat neuropathy are dosed lower than when used to treat depression. They are usually dosed once daily (Farhat & Yezback, 2016).

Norepinephrine and Serotonin Reuptake Inhibitors (Venlafaxine, Duloxetine): Serotonergic and noradrenergic pathways

are involved in pain, making serotonin reuptake inhibitors (SNRIs) another treatment for peripheral neuropathy (Farhat & Yezback, 2016). Duloxetine is usually used for neuropathic pain associated with diabetes and musculoskeletal pain (Jones III et al., 2016). According to Farhat & Yezback (2016), a Cochrane review (2014) showed significant improvement in pain with the use of duloxetine dosed at 60 to 120 mg, but no studies revealed any difference in efficacy between 60 mg daily and 120 mg daily, with less tolerability at higher doses. For this reason, duloxetine should be dosed at 60 mg daily. The initial dose can be started at 20 to 30 mg per day based on tolerance and safety. Caution should be taken in patients with renal and hepatic impairment (Farhat &Yezback, 2016). Monitor for side effects, such as nausea, vomiting, dizziness, and agitation (Votrubec &Tong, 2013).

Venlafaxine's starting dose is 37.5 mg daily and is increased by 75 mg daily until the target dose is achieved at 225 mg extended release daily. Venlafaxine has been shown to be effective at doses greater than 150 mg daily because lower doses under 150 mg daily have been associated with serotonergic, not serotonin, norepinephrine effects. Medication will require hepatic and renal dosage adjustments (Farhat & Yezback, 2016). For both agents, side effects to observe include nausea, which may occur the first week at the start of the treatment and resolve around the sixth day of treatment, sedation, gastrointestinal upset (constipation, diarrhea), dry mouth, and elevated blood sugars. Monitor liver function as well as blood pressure. Contraindications for these medications include patients with hepatic dysfunction, hypertension, uncontrolled glaucoma, and those taking MAOIs. Adjust medication dosage based on side effects (Votrubec & Thong, 2013). Venlafaxine has a high potential for QT prolongation even at a therapeutic dose; however, routine EKG monitoring is unnecessary unless persistent symptoms of toxicity develop or other risk factors are present (Farhat & Yezback, 2016).

Opioids/Tramadol: The epidemic of opioid overdose deaths due to misuse and abuse, as well as significant CNS side effects on multiple organs, make this class of drugs a second- or third-line therapy for treatment in severe neuropathic pain. The key principle when considering prescribing for neuropathic pain is to establish treatment goals and change administration to a longer-acting preparation as soon as a stable dose is achieved (Jones III et al., 2016; Jongen et al., 2014).

Tramadol, an opioid receptor agonist, has been shown to be effective for the treatment of neuropathic pain. Side effects of nausea, headache, and constipation may limit its use. If first-line options fail and a second line is required, tramadol may be started 50 mg once daily or 50 mg twice daily (Farhat & Yezback, 2016; Trivedi et al., 2013). Tramadol is titrated up at increments of 50 mg every 3 to 7 days on a three or four times a day schedule. For diabetic peripheral neuropathy, the maximum dose is 200 mg daily (Farhat & Yezback, 2016). Abuse and misuse of this class IV controlled substance should be considered when prescribing this medication (Farhat & Yezback, 2016).

Other agents approved by the FDA in 2012 include tapentadol ER for diabetic pain management. It is a combination of an opioid receptor agonist and norepinephrine reuptake inhibitor. The recommended dose is 100 to 250 mg twice daily with maximum dose 500 mg daily, but opioid-naïve patients should be started at a lower dose of 50 mg bid. This is a class II scheduled controlled substance. Common side effects include somnolence, dizziness, constipation, and nausea/vomiting. Morphine and oxycodone have also been used to treat diabetic peripheral neuropathic pain (Farhat & Yezback, 2016).

Topical Agents: Topical agents are usually most appropriate for chronic localized pain, as in herpes zoster, but their use for treating diffuse chronic diabetic neuropathy is limited (NIH, 2016). A high dose capsaicin patch (8%) has been found effective to relieve neuropathic pain in the treatment of post-herpetic neuralgia and HIV neuropathy (Jongen et al., 2014). The patch, applied once every 3 months, is applied over the area of the pain and associated allodynia. This agent should be considered early in therapy because delayed pain relief allows patients to tolerate other forms of pain management, such as physical treatment modalities (Jones III et al., 2016). Further studies are needed to confirm its efficacy (Farhat & Yezback, 2016).

Lidocaine patch (5%) is another agent that can be considered early in the treatment of neuropathic pain. The patch is applied every 12 hours up to 3 months (Jones et al., 2016). Although this has been approved for post-herpetic neuralgia, lidocaine patch may be used for diabetic peripheral neuropathy, where in the presence of sensory loss, patients report pain relief. The most common side effect may include skin irritation and is resolved after removal of the patch (Jones III et al., 2016). Further studies are needed to evaluate its efficacy (Farhat & Yezback, 2016).

As an adjunct therapy to pharmacological management of neuropathic pain, cognitive behavioral training is an effective psychological therapy approach for reducing the severity of chronic pain for a variety of painful conditions (Otis et al., 2014; Jones III et al., 2016). Cognitive behavior training helps patients focus on well behavior functioning and normal participation in activities rather than the pain itself. Often, patients may exaggerate the severity and effect of pain experienced, which is considered a predictor of poor outcome to pharmacotherapy and the likelihood of discontinuation of treatment (Jones III et al., 2016).

Combination Therapy: Several agents may be ineffective or have dose-limiting side effects when used as monotherapy. While more studies are needed, some combinations of medications have been safe and efficacious. The combination of gabapentin and nortriptyline has been effective in providing some pain relief, as well as duloxetine and pregabalin (Farhat &Yezback, 2016). Gabapentin used in combination with morphine to treats neuropathic pain has an opioid-sparing effect (Jongen et al., 2014).

Alternative Agents: In a systematic review of treatments for diabetic peripheral neuropathy conducted by Cakici, Fakkel, Neck, Verhagen, and Coert (2016), alpha lipoic acid (ALA) dosed at 600 mg oral once daily revealed no significant benefit for the improvement of pain if given the same dose intravenously. The study did show that AlA, botulinum toxin A, mexidol, reflexology, and Thai foot massage did have some beneficial effect on diabetic peripheral neuropathy symptoms.

Follow-Up: Referral should be considered in patients of unknown etiology or when pain remains refractory to

multiple pain medications (ClinicalKey, 2014). If the patient is thought to have a progressive disabling condition in terms of weakness, then further evaluation with referral to a neurologist should be made, as a lumbar puncture and nerve biopsy may be required (Levine & Saperstein, 2013). Patients should be referred to a neurologist when onset of the disease is rapid, symmetrical, and multifocal, along with EMG reports revealing either a demyelinating or axonal disease processes (Cleveland Clinic, 2010).

Sequelae: Peripheral neuropathy has a devastating effect on the older population because of the sum of negative effects on this group already affected by other neurological or systemic diseases, worsening the quality of life for this population (Marmiroli & Cavaletti, 2016). The negative impact includes gait impairment due to sensory and motor deficits, thereby increasing the risk of falls and injuries; amputations; ulcerations; and chronic pain (Callaghan et al., 2015). Mood and sleep disturbances, as well as impaired ability to work, are also complications related to uncontrolled neuropathic pain (Trivedi et al., 2013).

Prevention/Prophylaxis: Several health-promoting activities patients can engage in prophylactically include:

- Avoid repetitive motions, cramped positions, constant exposure to offending toxins, and excessive alcohol consumption.
- Practice good foot care, washing feet in lukewarm water and mild soap with moisturizing emollients and observing feet daily for any cuts, bruises, or lesions.
- Wear ankle braces, foot orthosis, and proper shoes to help reduce the risk of falls and injury, the development of ulcers, or the worsening of slow-healing wounds due to sensory impairment.
- Utilize assistive walking devices to help maintain stability due to muscle leg weakness, facilitate safety, and reduce falls and injury.
- Wear hand or wrist splints if the hands or wrists are involved (Cleveland Clinic, 2010).
- Make lifestyle modifications, including exercising, especially yoga and tai chi (NIH, 2016). Tai chi has proved beneficial for those with chronic painful conditions because it empowers individuals, allowing them to take an active role in their outcome. Tai chi improved Hgb A1c and plantar sensation in patients with diabetic peripheral neuropathy. Yoga has been shown to improve glycemic control, as well and nerve function (Jones III et al., 2016). Smoking cessation is also helpful. Smoking constricts blood vessels that nourish the peripheral nerves and can worsen neuropathic symptoms (NIH, 2016).
- Support groups provide an avenue for patients and facilitate engagement in the treatment strategy (Jones III et al., 2014).

Referral: Referral to a neurologist should be considered when the etiology is idiopathic or when pain remains refractory to multiple pain medications (ClinicalKey, 2014). If the patient is thought to have a progressive disabling condition in terms of weakness, then further evaluation with referral to a neurologist should be made, as a lumbar puncture and nerve biopsy may be required (Levine & Saperstein, 2013). Patients should be referred to a neurologist when onset of the disease is rapid, symmetrical, and multifocal, along with EMG reports revealing either a demyelinating or axonal disease processes (Cleveland Clinic, 2010).

Patients who cannot complete the tandem stand should be referred to physical therapy for balance and gait training. Reconditioning and appropriate exercises can help with pain control and reduce the risk of falls and injuries (Cleveland Clinic, 2010). Refer patients to podiatry to ensure appropriate shoes for extra depth and special orthotics, especially in patients with loss of proprioception or gait disturbances (Cacchione, 2010; Jeffrey, 2000). An ankle-foot orthosis may be effective in improving ambulation in persons with foot drop (Botek, Anderson, & Taylor, 2010). Refer to occupational therapy for appropriate assistive ambulation devices, splints, and braces (Shy, 2007). Refer patients to hematology/oncology when M-protein is present on SPE for possible lymphoproliferative disorder (Pourmand, 2002).

Education: Teach the patient, family, and/or caregivers about the disease process and its management, the role of common causes of the disease, the interventions for palliation, fall precautions, the need for good supportive shoes, foot care, and about healthy eating to ensure all vitamins and minerals are included in the daily diet. Advise patients of the need for regular follow-up visits, even if no symptoms are present. Instruct patients to avoid crossing the legs at the knee to avoid prolonged pressure or constriction and further nerve damage.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Higher doses of pregabalin at 900 mg daily have been studied with the outcome showing that doses greater than 300 mg resulted in adverse effects consisting of somnolence, dizziness, confusion, ataxia, and weight gain.	A	Ziegler & Fonseca, 2015
Venlafaxine has been shown to be effective at doses greater than 150 mg daily because lower doses under 150 mg daily have been associated with serotonergic, not serotonin, norepinephrine effects (Farhat & Yezback, 2016).	C	Trivedi et al., 2013

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The combination of gabapentin and nortriptyline have been effective in providing some pain relief as well as duloxetine and pregabalin.	A	Farhat &Yezback, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

RESTLESS LEGS SYNDROME

Signal Symptoms: Urge to move legs when lying down.

Description: Restless legs syndrome (RLS) is a common neurological disorder characterized by an intense urge to move the legs (or sometimes the arms) associated with discomfort. Patients may not be able to resist the urge to move their legs. Symptoms are reported as a need to move due to sensations variously described as aching, burning, creeping, crawling, cramping, gnawing, pulling, painful, tense, throbbing, tingling, restless, itchy, tense, tearing, and tugging. Movement of affected limbs provides relief, but discomfort recurs unless the movement continues. RLS symptoms have circadian rhythmicity, peaking between midnight and 3:00 a.m. Symptoms are elicited by sitting or lying down. It is a common sleep disorder.

Etiology: The cause of RLS is unknown; however, RLS is found in families and may have a genetic component. There is an indication that the brains of patients with RLS have reduced relative availability of iron and that this contributes to the symptoms (Conner, 2011).

Occurrence: It is estimated that 9% to 20% of elders are affected by RLS, with an estimated prevalence of 10% to 35% in individuals over 65 years of age.

Age: Average age of symptom onset is 42 years (range: 20 to 80 years and older).

Gender: Because women are more likely to be iron deficient than men, RLS is more common in women, with a female/male ratio of 5:1.

Ethnicity: RLS has a prevalence of 5% to 15% in the Caucasian population. It is less common in Asian and African American populations. It is more common in Caucasian women than in African American women.

Contributing Factors: Peripheral neuropathy (diabetes mellitus) is an assumed cause of secondary RLS. A positive family history of RLS is often noted in patients with this condition. Antidepressants, antipsychotics, antiemetics, and neuroleptics can aggravate RLS symptoms. Anemia, especially iron deficiency (there is a relationship between the dopaminergic system and iron), vitamin B_{12} deficiency, or folate deficiency can contribute to symptoms. Renal patients receiving dialysis are at risk for secondary RLS. Herbal and OTC medications can worsen RLS symptoms. Medications for hypertension, nausea, colds, allergies (antihistamines), and depression can make RLS worse. Patients diagnosed with attention-deficit disorder are known to have RLS.

Signs and Symptoms: Symptoms are worse at night and there is a strong need to move a limb or limbs, which may be associated with paresthesias or dysesthesias. An urge to move the legs is usually accompanied or caused by uncomfortable and unpleasant sensations in the legs (though sometimes the urge to move is present without the uncomfortable sensations, and sometimes the arms or other body parts are involved in addition to the legs). The urge to move or unpleasant sensations begin or worsen during periods of rest or inactivity such as lying or sitting. Akathisia is a major symptom of RLS. The focused interview for RLS should include the following questions:

- Ask about the urge to move or unpleasant sensations and if they are partially or totally relieved by movement such as walking or stretching.
- Ask if the urge to move or unpleasant sensations are worse in the evening or night than during the day, or if the urge to move only occurs in the evening or night (when symptoms are very severe, the worsening at night may not be noticeable, but must have been previously present).
- Ask about sleep patterns. Does the patient keep his or her partner awake?
- Does the patient have trouble sitting still and need to stand up and walk around?
- Is the patient tired during the day from an interrupted night of sleeping?

Diagnostic Tests: A CBC to begin the work-up of anemia should be followed by specific studies indicated by the results. A sleep study may be helpful to those who experience the symptoms mainly during the night.

Differential Diagnosis: Differential diagnosis may be challenging unless the suspicion of RLS is considered. Patients may not associate restless legs with disturbed sleep. Common disorders to rule out are listed, but many disorders may masquerade as RLS, such as:

- Hypotensive akathisia
- Radiculopathy
- Vascular claudication
- Neurogenic claudication
- Neuroleptic-induced akathisia

- Neuropathy
- Chronic pain syndrome associated with (lumbar, cervical) positional discomfort
- Nocturnal leg cramps
- Hypnic jerks
- Depression with somatic syndrome
- Volitional movements
- Foot tapping
- Leg rocking
- Lower limb arthritis
- Fibromyalgia
- Varicose veins (Garcia-Borreguero et al., 2011)

Diabetic neuropathy is damage to the peripheral nerves (microvascular injury) due to diabetes (high blood sugar damages nerves) and is most common in legs and feet. Symptoms include numbness, tingling, hypersensitivity, muscle weakness, and sharp pain.

Leg cramps (at night) are sudden, painful, and involuntary contractions of the muscles of the legs; these may have no real cause and are just bothersome. If leg cramping is disturbing sleep, assess and consider these causes as possibilities: diuretics, Addison's disease, PD, muscle overextension and over exercise, diabetes, hypoglycemia, hemodialysis, calcium channel blockers, peripheral artery disease, nerve compression, diabetes, and dehydration.

Periodic limb movements are repetitive cramping or jerking of the legs during sleep. It only occurs during sleep. It may be associated with RLS but is not the same thing. *Periodic* refers to the fact that the movements are repetitive and rhythmic, occurring about every 20 to 40 seconds. It is a sleep disorder and can disturb sleep and result in daytime sleepiness.

Radiculopathy refers to neuropathy, which occurs from compression on nerves in the spine, resulting in pain, tingling, numbness, and weakness. "Sciatica" pain radiating to the legs is an example. Pain can occur from a herniated disk, bone spur, osteoarthritis, tumor, infection, scoliosis, and stenosis. Box 13-1 lists the essential diagnostic criteria for RLS.

BOX 13-1

Essential Diagnostic Criteria for Restless Legs Syndrome

An urge to move the legs, usually accompanied or caused by uncomfortable and unpleasant sensations in the legs (sometimes the urge to move is present without the uncomfortable sensations, and sometimes the arms or other body parts are involved in addition to the legs)

The urge to move or unpleasant sensations begin or worsen during periods of rest or inactivity, such as lying or sitting

The urge to move or unpleasant sensations are partially or totally relieved by movement, such as walking or stretching, at least as long as the activity continues

The urge to move or unpleasant sensations are worse in the evening or night than during the day, or only occur in the evening or night (when symptoms are very severe, the worsening at night may not be noticeable, but must have been previously present)

SUPPORTIVE CRITERIA

Positive family history

Positive response to dopaminergic drugs

Periodic leg movement during wakefulness or sleep as assessed with polysomnography or leg activity

ASSOCIATED FEATURES

Natural clinical course of the disorder: RLS can begin at any age, but most patients seen in clinical practice are middle-aged or older. Most patients have a progressive clinical course, but a static clinical course is sometimes seen. Remissions of a month or more are sometimes reported.

Sleep disorders are a frequent but unspecific symptom of RLS.

Medical evaluation/physical examination: The neurological examination is usually normal. Probable causes for secondary RLS should be excluded. A low serum ferritin (<45–50 mcg/L) may be found in RLS patients (Richards, 2015).

Treatment: RLS is a lifelong condition for which there is no cure. Conservation therapy or complementary care may be enough to manage symptoms, including avoiding alcohol and caffeine, a trial of relaxation techniques, massage, acupuncture, daily exercise, stretching, application of heat or cold, good sleep hygiene, and small doses of melatonin (0.3 mg) (Zhdanova, 2001) and vitamins needed to correct the specific anemia. Recommend that patients find distractions while sitting if RLS occurs in this position. Recommend participation in support groups to learn of new supportive measures (Mitchell, 2011). For patients who experience RLS at night, promote good sleep hygiene (sleep at a consistent time; sleep in a cool, dark environment; relax before sleep).

For medical treatment of RLS, extensive data are available for levodopa and dopamine agonists, especially pramipexole and ropinirole, and to a small extent, cabergoline, pergolide, and rotigotine (Comella, 2014). Medical treatment for RLS often starts at age 50 to 60 years, though RLS may develop by age 40 years, and treatment may begin when symptoms are persistent and disturbing (Garcia-Borreguero et al., 2011). Sedatives (ramelteon [Rozerem]), anticonvulsants (neurontin [Gabapentin]), and pain relievers (opioids) may be beneficial and are drug options for treatment of RLS. Dopaminergic agents are used to treat RLS; start and use low doses and change doses slowly. Patients report the use of magnesium oxide (400 mg po q hs) to be helpful. There is not enough research yet to confirm this supplement is effective.

Follow-Up: Ask about sleep, exercise, and relationships. Sleep disturbances can cause hypertension and tension in relationships. Check blood pressure at each visit.

Sequelae: RLS can cause difficulty in falling asleep or staying asleep. Sleep deprivation can cause relationship problems, hypertension, and daytime sleepiness that can contribute to motor vehicle accidents.

Prevention/Prophylaxis: There is no prevention for RLS, but recommend daily exercise and review the tenets of sleep hygiene to improve sleep (sleep in a cool, quiet, dark environment; go to bed and get up at the same time; get enough sleep to feel rested). Cognitive behavioral therapy may be beneficial. Sequential compression devices have been used as an effective therapy for RLS (Lettieri & Eliasson, 2009).

Referral: Patients may benefit from participating in yoga, Qi gong, or treatment by an acupuncturist. Refer to a neurologist or a sleep or movement disorders specialist if unusual case or nonresponse to treatment.

Education: Provide patient and families with written information about RLS, including support groups in the community. Contact the Restless Legs Syndrome Foundation at info@rls.org.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The European Restless Legs Syndrome Study Group (EURLSSG) Task Force reached a consensus and agreed on diagnostic and treatment algorithms.	C	Garcia-Borreguero et al., 2011
Pneumatic compression devices are effective therapy for RLS: a prospective, randomized, double-blinded, sham- controlled trial.	A	Lettieri & Eliasson, 2009
There is a growing body of evidence demonstrating the effectiveness of nonpharmacological treatments, including lifestyle changes, physical activity programs, pneumatic compression, massage, near-infrared light therapy, and complementary therapy.	B	Mitchell, 2011
The long-term treatment of restless legs syndrome/ Willis-Ekbom disease: evidence-based guidelines and clinical consensus best practice guidance: report from the International Restless Legs Syndrome Study Group.	A	Garcia-Borreguero, et al., 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

SEIZURE DISORDERS

Signal Symptoms: Brief periods of confusion, loss of consciousness, staring with little or no motor activity, bizarre behavior, memory changes, falls, cognitive changes, sleep disorders, twitching, and involuntary movements.

Description: *Seizure* is a transient occurrence of signs and symptoms attributable to abnormal, excessive, or synchronous neuronal activity in the brain and can result from many causes (AANN, 2016; Fisher et al., 2014). *Epilepsy* is a disease of the brain defined by at least two unprovoked (or reflex) seizures separated by at least 24 hours; one unprovoked (or reflex) seizure and the probability of further seizures similar to the general recurrence risk of at least 60% after two unprovoked seizures occurring over the next 10 years; or the diagnosis of an epilepsy syndrome. Epileptic seizures may be classified as focal (partial/localized), generalized (involving both hemispheres), or unclassified. Generalized seizures are divided into absence (typical or atypical), myoclonic, clonic, tonic, tonic-clonic, and atonic (Berg et al., 2010). Further, epilepsy is considered to be resolved for individuals who had an age-dependent epilepsy syndrome and are now past the applicable age or have been seizure free for the last 10 years with no seizure medication for the last 5 years (Fisher et al., 2014). In contrast, a *provoked seizure (acute symptomatic)* is a single seizure caused by an acute, reversible condition caused by a known precipitating factor (AANN, 2016). In the older adult, precipitating factors could be a nervous system infection, acute traumatic brain injury, and fluctuations in blood sugar or electrolyte levels. Lastly, a *nonepileptic event* is a common and paroxysmal clinical event of diverse etiology that mirrors but is not an epileptic seizure (Fisher et al., 2014). The focus of this section is on new-onset seizures and epilepsy in older adults.

Etiology: In older adults, the most common causes of new-onset epilepsy and seizures are secondary factors to include cerebrovascular diseases, primary neuron degenerative disorders associated with cognitive impairment, intracerebral tumors, and traumatic brain injury. However, underlying etiologies can be found in only 50% (NINDS, 2015; Liu, Yu, & Lu, 2016). Understanding the epidemiology of epilepsy in older adults is important because of the association with poorer quality of life. There are unique challenges in terms of diagnosis and treatment within this group because of differences in clinical presentations and etiology compared with younger patients, presence of comorbidities and cognitive difficulties, and physiological changes that affect pharmacological management (Acharya & Acharya, 2014). Recognizing that persons over age 65 years are a heterogeneous group, recommendations have been made to divide patients into three

groups based on age: 65 to 74 years (young-old); 75 to 84 years (middle-old); and over 85 years (old-old). These groups are further subdivided based on health status: patients with only epilepsy, patients with epilepsy and multiple medical comorbidities, and the frail older adults. In addition, consideration should be given to persons living independently and those living in nursing homes.

Cerebrovascular disease accounts for 20% to 50% of seizures in older adults. Likewise, 10% of patients with stroke are at risk of developing seizures within 5 years. Hemorrhagic strokes are more likely to be associated with seizures than infarcts. In contrast, patients with transient ischemic attacks have a relatively low incidence of seizures (Acharya & Acharya, 2014; Gibson & Whiteley, 2013; Phabphal, Geater, Limapichat, Sathirapanya, & Setthawatcharawanich, 2013).

Neurodegenerative disease accounts for 10% to 20% of epilepsy that is due to dementia and neurodegenerative disease associated with cognitive impairment. Patients with AD have a 10 times higher risk of epilepsy, which increases as the disease progresses. Recognition of seizures in this population may be difficult, however, older adults do respond well to antiepileptic drugs (AEDs) (Acharya & Acharya, 2014).

Trauma, particularly head trauma, accounts for up to 20% of epilepsy in the older adult. Being age 65 years or older has been identified as a risk factor for developing epilepsy after a head injury.

Brain tumors are responsible for approximately 10% to 30% of seizures in the older adult and are more likely with low-grade, slow-growing tumors (Acharya & Acharya, 2014).

Occurrence: The incidence and prevalence of seizure disorders is highest in the older adult, therefore, older adult patients are the fastest growing demographic within patients with epilepsy. Approximately 2.3 million adults currently live with epilepsy in the United States (NINDS, 2015). Among older U.S. Medicare beneficiaries, prevalence rates are 10.8/1,000, with incidence rates of 2.4 per 1,000 (Faught et al., 2012). The prevalence rate of epilepsy in persons 65 years and older is 1.5%, with the prevalence being higher among nursing home residents than community-dwelling older adults. Prevalence and incidence rates may be underestimated due to the difficulty in identifying seizures and diagnosing epilepsy. In addition, status epilepticus is more frequent in older adults, with an annual incidence of 86/100,000 in patients over 60 years, twice that in the general population (Acharya & Acharya, 2014). These rates indicate an under-recognized public health issue. Overall rates are higher for African American beneficiaries and lower for Asians and Native Americans than for Caucasian beneficiaries. The higher rate of hypertension and stroke may account for the higher rates among African American men. Incidence rates are slightly higher for women than men and increase with age for all gender and races groups. Status epilepticus is more frequent in the older adult, with the annual incidence 86/1000,000 for patients over 60 years, twice that of the general population. The incidence is higher as patients age, with the mortality almost 50% in patients over 80 years (Acharya & Acharya, 2014).

Age: Some older adults may continue to experience seizures after age 60 years due to the natural course of the disease. There is an increased incidence in new-onset seizures after age 60 years (see Occurrence).

Gender: Not significant (see Occurrence).

Ethnicity: Although there is no well-understood difference in the incidence of seizures among various ethnic groups, there is a belief that the incidence of seizures among ethnic minorities may be under-reported. In some ethnic groups (e.g., Hispanic) neurocysticercosis may be a possible cause for seizures.

Contributing Factors: Under-diagnosis of seizures is common in the older adult. The most common initial diagnoses are syncope, altered mental state, and confusion. As noted, stroke and other cerebrovascular diseases are the most important risk factor for new-onset seizures. Epilepsy is mainly due to mechanical stimulation of a stroke lesion, nerve cell degeneration around the lesion, and glial scar formation (Liu et al., 2016). Cerebrovascular disease is the most common cause of epilepsy in the older adult. Less-frequent causes are neurodegenerative diseases such as AD, trauma, and brain tumors. As in younger people, acute symptomatic seizures may arise from drugs, metabolic disturbances, infections, acute strokes, and head injury.

Cerebrovascular disease accounts for 20% to 50% of epilepsy cases in the older adult. About 10% of patients who have had a stroke are at risk of developing seizures within 4 years. Hemorrhagic strokes are more likely associated. Transient ischemic attacks have a low incidence of seizures.

Neurodegenerative Diseases. Approximately 10% to 20% of epilepsy in older adult is due to dementia and neurodegenerative diseases associated with cognitive impairment. Patients with AD have a 10 times higher risk of epilepsy. Both focal and generalized seizures may occur. Recognition may be difficult, however, once diagnosed, patients respond well to AEDs.

Trauma and Brain Tumors. Age of 65 years or older is a risk factor for developing epilepsy after a head injury, which accounts for approximately 20% of epilepsy cases. Brain tumors account for 10% to 30%, especially low-grade, slow-growing tumors (Acharya & Acharya, 2014).

Multiple drugs have been shown to either cause seizures or potentially lower seizure thresholds. These include psychotropic drugs, theophylline, narcotics, tramadol (Ultram), antimicrobials, chemotherapeutic agents (methotrexate, chlorambucil), general anesthetics (ketamine, enflurane), local anesthetics, stimulants, antiarrhythmics (verapamil, mexiletine, procainamide, propranolol), diphenhydramine, baclofen, and chlorpromazine.

Signs and Symptoms: Types of seizures and epilepsy syndromes were reclassified by the International League Against Epilepsy (ILAE) in 2010 (Berg et al., 2010). In the 1989 system, seizures were classified as partial (limited to part of the brain) and generalized (involving both hemispheres of the brain). Under the new system, seizures are classified as focal (originate within networks in a hemisphere) and generalized (originate in a point and quickly spread to involve bilateral networks). Rather than using labels (e.g., simple partial, myoclonic), the labels have been removed and more descriptive terminology is used. The term *semiology* is used to describe the sensory/motor/behavioral presentations. A summary of the types and potential symptoms of generalized seizures is presented in Table 13-1. A summary of the types of focal seizures and their symptoms is presented in Table 13-2. An important point is that new onset (or first-ever)

TABLE 13-1 Generalized Seizures—Consciousness Is Impaired and There Are Symptoms of Motor Dysfunction Involving Both Sides of the Brain

SEMIOLOGY OF GENERALIZED SEIZURES	DESCRIPTION	POSSIBLE SYMPTOMS
Absence (typical)	Lapses of awareness lasting from a few seconds to up to 30 seconds; no warning and no residual effects	Blank staring or blinking, may have clonic component; perseverative speech; eyes may roll upward, eyelid fluttering, objects are dropped
Myoclonic	Sudden brief shock-like muscle contractions, usually unilateral, often occur while falling asleep or waking up	Sudden, unexpected jerking of the limbs, tremor-like; jerking is repetitive; may be dramatic resulting in falls
Clonic	Generalized seizure without the initial tonic phase	Rhythmic jerking, rhythmic muscle spasm, extremity involvement
Tonic-clonic	Increased muscle tone followed by rhythmic jerking, which gradually subsides; lost or impaired consciousness; the entire seizure, composed of tonic and clonic action, lasts for several minutes	Rigidity, person falls to the ground, a loud cry, loss of bladder or bowel function, breathing may stop, followed by labored breathing
Atonic seizures	Abrupt loss of muscle tone, usually with a brief loss of consciousness; alertness regained after striking floor	Head nods, eyelids droop, person may fall to the ground; injury from falls

TABLE 13-2 Focal Seizures*

PREVIOUS TYPES WITH CURRENT DESCRIPTIONS	DESCRIPTION	POSSIBLE SYMPTOMS
Simple partial seizures; focal seizure with or without impairment of consciousness or awareness	Consciousness is not lost or impaired	Rhythmic twitching of a limb or part of a limb, unusual taste, smell, buzzing noise, feeling of falling, paresthesias
Complex partial seizures; focal seizures with impairment of consciousness or awareness	Consciousness is lost or impaired	Loss of consciousness; change in awareness; automatisms such as fiddling with clothes or objects, wandering, making gestures, nonsensical talking
Secondarily generalized seizures; a seizure evolving to a bilateral convulsive seizure	A simple or complex partial seizure that spreads to involve both cerebral hemispheres	Generalized tonic-clonic seizure

*An electrical disturbance in one area of a cerebral hemisphere.

seizure age is not an independent predictor of seizure recurrence, and older patients are less likely than younger ones to sustain a seizure-related injury. Treatment for older patients should be based on risk factors for recurrence rather than age (Lawn et al., 2013).

Diagnostic Tests: A detailed history is imperative from the patient and, if possible, an eyewitness. The diagnostic approach is similar to that of a younger person. To begin, ascertain if the symptoms were epileptic seizures or nonepileptic in nature (see Differential Diagnosis). Older adult patients may not have prominent automatisms, as in focal seizures. Postictal confusion can last from hours to days. In the initial work-up of a new-onset seizure in an older adult, multiple diagnostic tests are needed. Blood tests include blood glucose because extreme hypoglycemia or hyperglycemia can cause provoked generalized tonic-clonic seizures (GTCS); CBC (an elevated white blood count can indicate a systemic or CNS infection, which can lead to a provoked GTCS); and electrolyte panel (hyponatremia, hypernatremia, or uremia can lead to a provoked GTCS). ESR, renal function tests, liver function tests, and calcium tests should be done. Baseline tests allow decisions to be made about potential medications. A thyroid screen (i.e., TSH) is done because hypothyroid neuropathy may be confused with focal seizure activity.

A toxicology screen may be indicated because illicit substances may cause an unprovoked GTCS. A rapid plasma reagin (RPR) may be done to rule out the possibility of syphilis, which, in tertiary form, may present as a seizure disorder. A lumbar puncture may be performed if there is concern for a primary CNS infection. A basic electrocardiogram (EKG) can confirm syncope in association with a cardiac arrhythmia. In the event that there is an abnormal EKG, an echocardiogram may be required. Tilt-table testing can be useful to detect baroreceptor and vasopressor dysfunction or sympathetic failure due to autonomic neuropathy in older people. Electroencephalography (EEG) and brain MRI are the most important tests for unprovoked seizures, although a routine EEG may be of limited use in older adults due to low sensitivity and specificity. An ambulatory prolonged EEG may be indicated. The MRI is the preferred test, as it is more accurate

than a CT scan. A brain MRI with and without contrast should be done to rule out old scar tissue, mesial temporal sclerosis, or other structural reasons for the seizures. Neuroimaging with contrast increases the ability to identify tumors, inflammatory disease, and abscesses. An EEG is indicated after any unprovoked seizure event to support the diagnosis of idiopathic generalized or focal-onset epilepsy. A normal result does not prove the patient does not have a potential for seizures; multiple EEG studies may be indicated.

Differential Diagnosis: The most common differential diagnoses for seizures include (Acharya & Acharya, 2014):

- Neurological: transient ischemic attack, transient global amnesia, confusional migraine, RLS
- Cardiovascular: syncope, orthostatic hypotension, cardiac arrhythmias, carotid sinus syndrome
- Drug intoxication
- Infections
- Endocrine/metabolic disturbances (e.g., hypoglycemia, hyperglycemia, hyponatremia, hypokalemia)
- Sleep disturbances: obstructive sleep apnea, hypnic jerks
- Dementia
- Psychological: nonepileptic, psychogenic seizures
- Drug induced: alcohol withdrawal (peak incidence of first seizures occurs late in life); use of one or more drugs in high doses with co-existing illness (antihistamines, antidepressants, antipsychotics, hypoglycemic drugs)

Diagnosing new-onset seizures in older adults is challenging due to the presentation and comorbidities. As noted, the most common seizures in the older adult are focal in onset. Confusion and memory problems are common presenting symptoms and the postictal phase may be prolonged. A reliable history and a witnessed event are the most important diagnostic factors, especially in new-onset seizures. Obtain information concerning family history of seizures, any head injuries or other history of trauma (observe bruises, cuts, burns), and any past history of seizures. Information concerning whether the individual has lived outside of the United States (particularly in unincorporated areas with poor sanitation) can raise suspicion of neurocysticercosis. This parasitic infection results from ingestion of eggs from the adult tapeworm and is the most common parasitic infection of the brain with epilepsy as the most common presentation. With the immigration of people from Latin America to the United States, this disease is a growing problem (Sharma, Singh, & Mathew, 2015).

Obtain a complete description of the seizure, including any aura (a warning of the seizure), along with a complete description of the patient's behavior during the seizure and in the postictal period. If the seizure was observed, note if there was evidence of pallor, cyanosis, abnormal movements, tongue biting, urinary incontinence, impaired continence, or postictal symptoms such as confusion, headache, and drowsiness. As noted previously, seizures are associated with dementia and cerebrovascular disease in older adults with new-onset seizures; therefore, it is imperative to include cognitive function screening. An estimate of the time for each phase of the seizure is helpful. How the patient felt before and after the seizure can offer insight into the potential nature of the cause of the event. A thorough neurological examination is performed, with particular attention paid to any focal or localizing symptoms. If the individual is currently taking AEDs, screen for any symptoms of toxicity, including nystagmus, ataxia, and slurred or slowed speech.

Treatment: Patients with provoked seizures need to be treated for the underlying cause and usually do not need AEDs. With unprovoked seizures, the decision to treat depends on whether the patient has a single seizure or recurrent seizures (epilepsy). Unlike management in younger adults, AEDs may be appropriate after a single seizure in the older adult because of the high rate of recurrence. The goal of management should be the maintenance of a normal lifestyle, with complete control of seizures with minimal side effects. Seizures in the older adult tend to respond better to AEDs than those in young individuals and can often be appropriately controlled with monotherapy. Pharmacotherapy continues to be the mainstay of treatment for seizures, however, AED management can be complicated because of the coexistence of other diseases (e.g., dementia, polypharmacy), and the likelihood of dose-related and adverse side effects.

AEDs are commonly prescribed to the older adult and to about 10% of nursing home residents for a variety of reasons, including migraine prophylaxis, psychiatric disorders, pain management, and seizures. AEDs are the fifth most common cause of drug side effects in the older adult and, therefore, need to be carefully prescribed and monitored. The pharmacokinetics and pharmacodynamics of AEDs are distinct in older adult patients. Physiological changes relevant to pharmacotherapy in the older adult include decreased gastrointestinal motility, decreased serum albumin, increased body fat to lean mass ratio, decreased total body water, decreased liver mass and blood flow, decreased cytochrome P450 enzyme activity, decreased renal flow and weight, and decreased glomerular filtration rate, among others. For example, reduction in serum albumin levels leads to reduction in protein binding and increase in free fraction of the drug. This is an issue for high protein bound drugs such as phenytoin, carbamazepine, and valproic acid because of the increased risk of toxicity.

Older adults have a narrower therapeutic window than younger adults. Many AEDs are metabolized by the liver, especially the P450 enzyme, which is decreased. The potential for drug interactions is a major consideration in selecting an AED. Older AEDs (e.g., phenytoin, phenobarbital, carbamazepine) are enzyme-inducers, which increases the likelihood of interactions with other medications. Newer medications such as lamotrigine and levetiracetam are better choices for older adults on multiple medications. Calcium and vitamin D supplements should be considered because of the risk of osteoporosis with AED use. Pharmacodynamic changes include alterations in receptor distribution and density, receptor affinity and function, neurotransmitter release, and autonomic and homeostatic function. Toxicity from medications is poorly recognized in the older adult and can be attributed to multiple causes. Older adults with multiple chronic illnesses are more likely to be taking medications that interfere with AEDs. Table 13-3 includes a summary of important information concerning AEDs (those in bold are recommended).

Benzodiazepines (diazepam, lorazepam) are most commonly used for rescue or only in the acute setting. Although these are generally considered CNS depressants, the older adult may experience idiosyncratic effects such

TABLE 13-3 Major Anticonvulsants

GENERIC NAME	BRAND NAME	DOSAGE	INDICATIONS	SELECTED SIDE EFFECTS	COMMENTS
Carbamazepine	Tegretol	200–400 mg daily in divided doses; up to 1,600 mg daily	Partial, PGTC	Hyponatremia Osteoporosis Leukopenia	Other forms: Tegretol XR Carbatrol
Gabapentin	Neurontin	900–1,200 mg daily in divided doses; up to 3,600 mg daily	Partial	Gastrointestinal Dizziness	Decreased drug interactions
Lacosamide	Vimpat	100–200 mg daily in divided doses; up to 400 mg daily	Partial	Fatigue, nausea, dizziness	No generic available
Lamotrigine	Lamictal	Monotherapy 25 mg for 2 weeks, 50 mg for the next 2 weeks, then increases of 50–100 mg/week. 100–400 qd	New onset focal; new onset unprovoked generalized	Sedation, dizziness, dose-related rash	Fewer interactions with other medications
Levetiracetam	Keppra	500–1,000 mg daily; up to 3,000 mg daily	Partial, PGTC, Myoclonic	Fatigue, anxiety, depression Gastrointestinal	Decreased drug interactions
Oxcarbazepine	Trileptal	300–600 mg daily in divided doses; up to 2,400 mg daily	Partial	Dizziness Hyponatremia, nausea Leukopenia	
Phenobarbital	Phenobarbital	Usual starting dose in adults is 60 mg/day, with additional 30-mg increases every 2–4 weeks to a target dose of 90–120 mg/day, or higher if clinically tolerated	Partial, PGTC, Myoclonic	Sedation Behavior problems Psychotic episodes cognitive impairment	Considered high-risk medication in older adult
Phenytoin	Dilantin	300 mg daily in divided doses; up to 600 mg daily; dose to saturation kinetics	Partial, PGTC	Osteoporosis Dizziness	Once a day dosage if tolerated Other forms: Phenytek
Pregabalin	Lyrica	150 mg daily in divided doses; up to 600 mg daily	Partial	Weight gain Dizziness Psychotic episodes Sedation	
Primidone	Mysoline	100–125 mg daily in divided doses; up to 2,000 mg daily	Partial, PGTC, Myoclonic	Same as Phenobarbital	Metabolizes to phenobarbital
Tiagabine	Gabitril	4–8 mg daily in divided doses; up to 56 mg daily	Partial	Fatigue, tremor, problems concentrating Psychotic episodes	
Topiramate	Topamax	100–200 mg daily; initiate with titration schedule; up to 400 mg daily	Partial, PGTC	Somnolence Dizziness Behavior problems Weight changes, renal stones	
Valproic acid	Depakote	500–1,000 mg daily in divided doses; up to 60 mg/kg daily	Partial, PGTC, Absence	Weight gain, hair loss, thrombocytopenia Higher risk of cognitive problems and parkinsonism, retinal dysfunction	
Zonisamide	Zonegeran	100–200 mg daily; up to 600 mg daily	Partial	Sedation Weight loss Impaired cognition	Once daily dosage

as restlessness, hyperactivity, and psychosis. Phenobarbital has been used in the treatment of seizures since the 1950s. Some older adult patients may have been on the medication for many years and will continue to require consistent monitoring. Phenobarbital is not recommended for a patient with new-onset seizures.

The newer AEDs offer some advantages in working with the older adult, including a better side-effect profile. However, they are more expensive, and the name brands may not be covered by various insurance programs. Oxcarbazepine is a second-generation carbamazepine but with fewer side effects. This drug is only 38% protein bound and can be introduced more quickly in the older adult than carbamazepine. Lamotrigine is well absorbed orally by the older adult. Although the medication is eliminated by hepatic metabolism, then excreted renally, the drug is safely used in the older adult with renal failure. Side effects are minimal, and several studies have shown that it is well tolerated by the older adult. Lamotrigine has some mood-stabilizing effects, which may also help in treatment of the older adult. Levetiracetam is not metabolized by the P450 system and is entirely renally excreted. Dosage in renal failure may need adjustment. Gabapentin is also completely excreted unchanged in the urine. This drug is generally well tolerated in the older adult and is less likely to be involved in drug interactions, however, high dosages are often required for effective seizure control.

Topiramate has demonstrated good efficacy in older adult patients, but may interact with digoxin and warfarin. Pregabalin has oral bioavailability exceeding 90%, is not bound to plasma proteins, and readily crosses the blood-brain barrier. Dosage must be titrated slowly to avoid adverse cognitive effects. Felbamate is primarily metabolized by the liver and has a number of drug interactions. Used carefully, this can be a good choice in pharmaco-resistant seizures. Tiagabine is also metabolized by the liver and has a drug interaction profile similar to carbamazepine. Zonisamide is 40% bound to protein and its major elimination pathway is hepatic, but can be safely used in the older adult.

Caution should be exercised in the use of generic medications, because these have been shown to not have the same efficacy as name-brand medications. A patient who is stable on generic medications can become toxic or have breakthrough seizures if switched to name-brand medications. Similarly, the patient who is stable on name-brand medications and is switched to generic may experience a significant decrease in serum levels, leading to breakthrough seizures.

Seizure surgery (e.g., temporal lobectomy) can be considered in the older adult if the seizures are medically intractable and the seizures are related to a focal structural lesion. Limited data are available on the outcomes from surgery in those over age 65 years. Patients would be referred to a comprehensive epilepsy center for evaluation.

Vagus nerve stimulation (VNS) has also been used in patients with refractory epilepsy. VNS is provided by a surgically implanted device programmed to deliver a brief electric stimulation on a schedule. The efficacy of this treatment has been established in general populations, but the data on the older adult are sparse.

Follow-Up: Seizure management is a significant challenge, and the patient and any caretakers should be seen frequently. A seizure calendar/journal is maintained by the patient and should be reviewed on each visit. Precipitating factors for an increase in seizures, such as stress, sleep deprivation, or viral illness, should be noted. Questions concerning potential medication side effects are asked on each visit. For some medications, periodic levels are monitored and adjusted to ensure therapeutic levels. The incidence of depression in adults with epilepsy has been reported as high as 50%. Causes of the depression include lifestyle changes, loss of confidence and independence, as well as biochemical alterations in the brain due to the seizures. The potential loss of freedom following a seizure is heightened by driving restrictions, which are based on the laws of the individual state and range from 6 months to at least 1 year seizure free.

Sequelae: Sudden unexpected death in epilepsy (SUDEP) is defined specifically as the sudden, unexpected, witnessed or unwitnessed, nontraumatic, and nondrowning death in patients with epilepsy with or without evidence for a seizure, and excluding documented status epilepticus, in which postmortem examination does not reveal a structural or toxicological cause for death. Individuals with epilepsy have a mortality rate two to three times greater than that of the population at large. SUDEP accounts for 8% to 17% of these deaths and is more likely to occur in those with frequent seizures. Patients and their families need to be warned of the potential for SUDEP and the importance of controlling the variables that can lead to SUDEP, such as abrupt cessation of AEDs.

Convulsive status epilepticus incidence is almost twice that of the general population. Associated mortality rate is also higher in older patients, 38% in those over 60 years and over 50% in those over 80 years. Nonconvulsive status epilepticus (NCSE) is equally common in the older adult, but much more difficult to diagnose because it can present with general symptoms such as confusion, psychosis, lethargy, or coma, or a more focal cognitive disturbance with aphasia. These symptoms are very general and may not be associated with any observable seizure activity. Due to the nonspecific nature of the symptoms, the presence of seizures on the EEG is the most important diagnostic step. Incidence rate of all types of status epilepticus in the older adult is 54.2 per 100,000 people.

Prevention/Prophylaxis: Prevention of potential causative factors such as stroke, meningitis, and active management of disease processes such as AD and dementia is an important part of prevention of seizures. Care in preventing falls may also decrease the incidence of head injuries leading to seizures. Prophylactic use of AEDs following head injury and stroke remains controversial but may be indicated.

Referral: For new-onset seizures, referral to a neurologist/epileptologist or to a comprehensive epilepsy center would be in the best interest of the patient. If traditional medications become difficult to manage, the patient should be referred to a neurologist.

Education: Comprehensive education should be provided to patients and their families/caregivers. This education includes seizure recognition and first aid, treatment plans and side effects, safety practices to prevent injury during seizures, and lifestyle considerations appropriate for age (e.g., driving, work, sleep, coping skills, stress management). Community resources, as well as evidence-based Web-based resources, should be discussed (e.g., Epilepsy Foundation,

National Institute of Neurological Disorders and Stroke [NINDS]). Educating patients and their families about SUDEP, ascertaining seizure precipitants, and promoting compliance with treatment are important strategies. Very specific information should be provided to patients who are nonadherent with medications or appear to be in denial of the diagnosis. Patients should maintain a seizure calendar that chronicles seizures and all precipitants. Strategies to ensure compliance with daily medication include the use of medication boxes and telephone alarms. Patients should inform the provider of any changes in other medications to avoid potential interactions. Inform patients and family members that abrupt cessation of medications is very dangerous and that all AEDs should be carefully weaned.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Low dose AED monotherapy decreases adverse effects and drug interactions.	A	Schmidt & Schachter, 2014 Hanlon, Semla, & Schmader, 2015
It is essential to understand the etiology in the decision to treat the older adult with seizures.	B	Acharya & Acharya, 2014
Comprehensive epilepsy education should be provided to older adults with epilepsy and their caregivers.	B	England, Liverman, Schultz, & Strawbridge, 2012
Patients with epilepsy should be screened for depression.	A	Fiest et al., 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

STROKE

Signal Symptoms: Sudden weakness or numbness on one side of the body, language impairment, trouble seeing in one or both eyes, alteration in balance and/or coordination, severe headache without known cause.

Description: Stroke is a rapidly evolving cerebrovascular syndrome of sudden onset, with symptoms suggestive of a nonepileptic focal neurological deficit. There are two major types of strokes: ischemic, indicating blockage of an artery, and hemorrhagic, usually resulting from a leak or rupture in an artery wall.

Etiology: Approximately 87% of all strokes are ischemic (Mozaffarian et al., 2016) and develop when an artery in the brain is obstructed by a clot that eithers forms in the brain or travels from somewhere else. Ischemic strokes are classified into casual subtypes: cardioembolic, small vessel occlusion (lacunar stroke), large vessel atherothrombotic, other unusual determined etiology, and undetermined etiology. Cardioembolic strokes occur when a blood clot forms elsewhere in the body, travels to the brain through the circulatory system, and becomes lodged in a cerebral artery. Cardioembolic strokes are generally associated with atrial fibrillation, valvular disorders, heart failure, and other states associated with thrombus formation. Large vessel strokes occur when a major artery in the brain is blocked, presumably by a blood clot that forms as a result of an inflammatory response to an unstable atherosclerotic plaque. Lacunar infarcts occur in the small arteries that branch from main cerebral arteries. Unusual etiologies of stroke include nonatherosclerotic vasculopathies, hypercoagulable states, or hematological disorders. An exhaustive work-up, an incomplete work-up, and more than one possible source can lead to a label of undetermined etiology (Adams et al., 1993).

Hemorrhagic stroke usually results from either a nonstructural or structural cause, where a weakened area of an artery in or around the brain ruptures. The most common nonstructural hemorrhage is caused by pathophysiological changes that occur in small arteries from long-standing hypertension, often triggered by an accelerated hypertensive episode. Structural etiologies include ruptured aneurysms and arteriovenous malformations. Incidence is varied by race and mechanism. Approximately 10% of all events are intracerebral hemorrhage and 3% are subarachnoid hemorrhage (Mozaffarian et al., 2016). *Hemorrhagic transformation* is a term that refers to bleeding into infarcted tissue and is thought to be associated with reperfusion. It occurs within days or weeks from stroke onset.

A transient ischemic attack (TIA) is an episode of focal neurological impairment of presumed ischemic origin, typically lasting only a few minutes with no permanent deficits. The episode of neurological dysfunction is caused by focal brain, spinal cord, or retinal ischemia without acute infarction (Kernan et al., 2014). Absence of tissue findings makes TIA a tissue-based definition rather than a time-defined definition. Transient hypotension, in conjunction with significant carotid stenosis, may also cause a TIA. The significance

of TIA lies in the heightened short-term risk of stroke, with recent studies finding that with early treatment in TIA clinics, stroke within 90 days of a TIA dropped to between 5.9% and 11.7%, down 50% from previous studies (Amarenco et al., 2016).

Occurrence: Stroke is the leading neurological cause of death and disability in the United States, with an estimated 795,000 people each year suffering a new or recurrent stroke (Mozaffarian et al., 2016).

Age: Approximately 6.6 million Americans 20 years old and older have had a stroke. The prevalence of silent cerebral infarction rises from 6% to 28% with increasing age. With an aging population, by 2030 prevalence is expected to increase 20.5% in those 18 years old and older, adding 3.4 million more people with stroke (Mozaffarian et al., 2016).

Gender: The stroke incidence rate is higher for men compared with women at younger ages, but not at older ages. Lifetime risk for women is higher than men, with rates one in five for women and approximately one in six for men ages 55 to 75 years. Prevalence of TIA increases with age. True prevalence is difficult to ascertain, as many fail to report neurological symptoms to their health-care provider (Mozaffarian et al., 2016).

Ethnicity: Native Americans, Hispanics, and African Americans have more TIAs and are at higher risk for stroke and stroke mortality than Caucasians. African Americans carry the highest risk and Hispanics carry a moderate risk, while Caucasians carry the lowest risk (Mozaffarian et al., 2016).

Contributing Factors: Risk factors for stroke include advanced age, family history of stroke, hypertension, diabetes, smoking, prior history of TIA or stroke, hyperlipidemia, cardiovascular disease, carotid stenosis, atrial fibrillation, obesity, sedentary lifestyle, alcohol abuse, and some infections (endocarditis, bacterial meningitis). Less common risk factors include sleep apnea, drug abuse, migraine, hypercoagulable disorders, hormone replacement therapy or oral contraceptive use, sickle cell anemia, and other inflammatory processes (Meschia et al., 2014).

Signs and Symptoms: TIA is a precursor to stroke in 15% of all cases. Patients having a TIA or ischemic stroke may report sudden onset of the following symptoms: weakness, numbness, or paralysis in the face, hand, arm, or leg (usually unilateral); difficulty speaking or understanding verbal communication; blurred, doubled, or decreased monocular or binocular vision; loss of balance or coordination; and severe headache (Jauch et al., 2013). Patients with a hemorrhagic stroke may report any of these symptoms, along with nausea and vomiting, altered mental status, sensitivity to light, and neck stiffness. Physical signs may include one or more of the following: decreased visual acuity or field cut, diplopia, slurred speech, aphasia, and hemiparesis or sensory changes of the face, arm, or leg. A decrease in coordination or unsteady gait can occur. Elevated blood pressure is common. Acute confusion can occur.

Any other focal signs on neurological examination should be assessed carefully. Physical examination should include repeated measures of vital signs and examination of the head and neck for signs of trauma and infection, checks of the peripheral and carotid pulses, auscultation of the neck for bruits, and a complete neurological examination for focal abnormalities. The consequent impairment depends on the location and extent of the infarction (Hemphill et al., 2015). Recommended is the National Institutes of Health Stroke Scale (NIHSS), a validated tool that standardizes approach to the neurological examination (Jauch et al., 2013; Hemphill et al., 2015).

Diagnostic Tests: All patients with symptoms consistent with stroke should be transported via emergency medical services (EMS) to the nearest certified stroke facility for rapid assessment and early treatment. Even for patients who symptoms have resolved, immediate diagnostic tests should include a noncontrast CT scan or MRI of the head to evaluate for presence of hemorrhage, EKG, and laboratory assessment (oxygen saturation, CBC, prothrombin time/activated partial thromboplastin time [PT/aPTT], troponin, lipids, and complete blood chemistry) (Jauch et al., 2013). A head CT scan is almost 100% sensitive for intracerebral hemorrhage (ICH). Ischemic events may not be visible on CT scan for 24 to 48 hours and may be noted anywhere in the cerebral cortex, cerebellum, or brainstem. MRI is helpful in early diagnosis of ischemic or hemorrhagic strokes but may not be available in all areas. If a hemorrhagic stroke is confirmed, consider referral to a neurosurgeon. Perfusion CT and MRI may be considered for patients eligible for reperfusion outside the typical time window. Further diagnostic testing includes carotid duplex studies, CT angiography and/or magnetic resonance angiography, noninvasive methods to look for intra- and extracranial vascular occlusion in the head or neck (Macellari et al., 2014), as well as cardiac monitoring and an echocardiogram. Treatment choices will depend on results of initial assessments and time elapsed between onset of symptoms and confirmed diagnosis (Jauch et al., 2013; Hemphill et al., 2015). The purpose of acute hospitalization is to establish stroke etiology in order to prevent subsequent events.

Differential Diagnosis:

- Seizure with Todd's paralysis: loss of consciousness, seizure activity, incontinence, postictal state
- Migraine aura: more common in women, history of similar event, preceding aura, with or without headache, photophobia
- Syncope/dizziness/vertigo: common types include neurocardiogenic (vasovagal) situational, orthostatic, and syncope related to cardiac ischemia or arrhythmia. Associated with sweating, pallor, and nausea. May have dim vision, decreased alertness, and transient loss of consciousness. Isolated peripheral vertigo with subsequent stroke is 0.18% (Atzema et al., 2016)
- Transient global amnesia: poor recall of recent events with impaired ability to retain new memories without other cognitive or focal neurological impairment
- Toxic-metabolic disorders: hypoglycemia, hyponatremia, drug or alcohol intoxication, ruled out based on history and laboratory findings
- Multiple sclerosis: paroxysmal transient attacks of ataxia and dysarthria
- Brain tumor: gradual progression of symptoms, other primary malignancy, seizure at onset, ruled out based on brain imaging studies
- Systemic infections: acute illness with recrudescence of previous stroke symptoms

- Conversion disorder: inconsistent clinical examination, nonfocal symptoms, neurological findings in a nonvascular distribution, increased risk in younger age with psychiatric history

Treatment: Emergency transport to the nearest designated stroke center should be initiated for patients with suspected stroke, regardless of the severity of neurological deficits, and be evaluated as high priority triage. Patients should be given nothing by mouth (NPO) with IV fluids (isotonic sodium chloride) started to maintain euvolemic status in most patients. Patients with fever (greater than 38°C [100.4°]) should be treated with antipyretics and an aggressive search for the source of fever initiated. Hypoglycemia and hyperglycemia should be identified and treated promptly. Both can produce symptoms that mimic ischemic stroke, and both may worsen neuronal injury. The goal for hypoglycemia treatment is to achieve normoglycemia. Hyperglycemia during the first 24 hours is associated with poor outcomes, thus glucose level goals of 140 to 180 mg/dL with avoidance of hypoglycemia is advised. Supplemental oxygen is recommended to attain oxygen saturation greater than 94% (Jauch et al., 2013).

For eligible patients who present within 4.5 hours of the onset of symptoms and a hemorrhagic event is ruled out, recombinant tissue-type plasminogen activator (alteplase), an IV thrombolytic agent, has been established to decrease long-term disability. The most important factor in successful treatment is early administration (Demaerschalk et al., 2016). Additionally, for patients who have a large vessel occlusion, intra-arterial treatment with mechanical thrombectomy within 24 hours of symptom onset has been found to be safe and effective, demonstrating improved outcomes and reduced mortality. Other investigational methods of reperfusion, including intra-arterial thrombolysis remain unproven. Endovascular and research availability depends on location; treatment should only be performed at an experienced stroke center (Powers et al., 2018). The American Stroke Association guidelines support that cities, counties, regions, and states develop a stroke system of care that will enable patient access to optimal care (Powers et al., 2018; Higashida et al., 2013).

Early administration of aspirin within 24 to 48 hours following stroke has shown benefit and is recommended in ischemic events, but cannot be given within 24 hours of alteplase administration. Urgent anticoagulation to prevent recurrent stroke, avoid neurological worsening, or improve outcomes is not recommended (Jausch et al., 2013).

Hypertension is common in acute stroke and may spontaneously decline within 90 minutes of stroke onset. Aggressive use of antihypertensives may reduce perfusion pressure and prolong or worsen ischemia. Rapid reduction of blood pressure regardless of the degree of hypertension may be harmful. The consensus recommendation is to lower the blood pressure only in the event the systolic pressure is greater than 220 mm Hg or diastolic blood pressure is greater than 120 mm Hg. However, a systolic pressure greater than 185 mm Hg or a diastolic pressure greater than 110 mm Hg is a contraindication for thrombolytic therapy. Therefore, the management of blood pressure in acute ischemic stroke will depend on whether the patient is a candidate for thrombolytic therapy. There is no evidence to determine which antihypertensive should be used; an individualized approach is best (Jauch et al., 2013).

Hemorrhagic events should be initially managed in an intensive care or stroke unit. For patients taking anticoagulation medications, reversal may be indicated. If patients present with systolic pressures 150 to 220 mm Hg, aggressive reduction is considered safe down to 140 mm Hg. Prophylactic antiepileptics are not recommended. Ideally, neurosurgical care should be available, though the usefulness of surgery for most patients is not well established. Patients should be monitored at frequent intervals for signs of cerebral edema, and control of other comorbid diseases are also indicated (Hemphill et al., 2015).

Follow-Up: Indications for follow-up depend on the type and extent of the stroke. Rehabilitation should begin during the acute phase as soon as the patient can tolerate, with all patients undergoing a formal rehabilitation needs assessment prior to discharge. Post-acute goals support the pursuit of the highest level of functioning possible. A stroke-specific rehabilitation environment is preferred, if available. Rehabilitation targets should be designed around 1) functional outcomes, 2) lifestyle interventions, 3) enhancement of cardiorespiratory fitness, 4) prevention of complications and deconditioning, and 4) social reintegration. Education regarding stroke, rehabilitation, recovery, and what to expect are critical for the survivor, family, and caregivers. Whenever possible, the stroke survivor, family, and caregivers should be involved in decision making regarding treatment and care planning. Use of standardized assessment instruments will be helpful in establishing baseline, setting goals, and tracking progress. Standardized stroke assessment instruments are found in Box 13-2 (Winstein et al., 2016; VA DoD, 2010).

Whenever possible after a stroke, patients should have regular contact with a care team with expertise in stroke rehabilitation to assess for changes in level of function, control medical comorbidities, manage pain, assess for depression/anxiety, address complex rehabilitation issues, and implement prevention strategies for secondary stroke prevention (Winstein et al., 2016). Community- or home-based rehabilitation services are recommended following discharge from either acute hospitalization or inpatient rehabilitation, when feasible, and should include a team approach with nursing, medical interventions, and specialized equipment as needed. Given today's electronic climate, it is reasonable to consider other methods for follow-up at home, especially for those in rural environments, including the use of discharge phone calls, telehealth, and Web-based or smart-phone enabled support (Higashida et al., 2013).

Sequelae: Stroke survivors are at high risk for complications related to immobility, such as skin breakdown and contractures, loss of muscle strength, and pulmonary compromise. Risk assessments should routinely be performed for other common complications including malnutrition, bowel and bladder incontinence, deep vein thrombosis, pain, falls, seizures, and osteoporosis. Assessment of cognition and memory, communication abilities, sensorimotor impairment, and psychosocial status should be undertaken regularly in the subacute and chronic phases of stroke recovery. Speech and swallowing difficulties also may persist after a stroke. Depression and anxiety are also common. Stroke survivors should be screened for depression both during the acute phase and

BOX 13-2

Standardized, Validated Instruments for Assessment of Stroke Survivors

Global disability (Rankin scale)
Measures of disability/ADLs (Barthel Index; Functional Independence Measure [FIM™])
Instrumental activities of daily living (PGC Instrumental Activities of Daily Living; Frenchay Activities Index)
Mental status (Folstein Mini-Mental State Examination; Neurobehavioral Cognition Status Exam [NCSE])
Speech and language (Boston Diagnostic Aphasia Examination; Porch Index of Communicative Ability [PICA]; Western Aphasia Battery)
Paresis/strength (Motricity Index, muscle strength, grip pinch dynamometry)
Tone (Modified Ashworth scale)
Sensorimotor impairment (Fugl-Meyer, Chedoke McMaster Stroke Assessment, impairment inventory)
Upper extremity function (Action Research Arm Test, Box and Block Test, Chedoke Arm and Hand Activity index, Wolf Motor Function Test)
Balance (Berg Balance Scale, Functional Reach Test)
Mobility (walking speed, Timed Up and Go, 6-minute walk test, functional ambulation category, observational gait analysis)
Self-reported impairments, limitations, and restrictions (Stroke Impact scale, motor activity log, Activities-Specific Balance Confidence Scale)
Technology for monitoring activity and participation (accelerometers, step activity monitors, pedometers)
Depression (Beck Depression Inventory [BDI]; Center for Epidemiologic Studies Depression [CES-D]; Geriatric Depression Scale [GDS])

Sources: Winstein et al. (2016). Department of Veterans Affairs, Department of Defense, and the American Heart Association/American Stroke Association, 2010.

regularly thereafter, with treatment as indicated (Winstein et al., 2016). Monitoring of the caregiver burden and access to available community resources is also imperative (Camak, 2015). Finally, all stroke survivors are at risk for recurrent stroke and other cardiovascular events. Appropriate strategies should be implemented for prevention/prophylaxis.

Prevention/Prophylaxis: Strategies to prevent stroke are similar to strategies used to prevent other cardiovascular diseases: control of hypertension, diabetes, dyslipidemia, sleep apnea, and cardiac disease through lifestyle and pharmacotherapeutic agents. Encourage patients to eat healthy, increase physical activity levels, lose weight (if indicated), avoid heavy alcohol intake, and stop smoking. Secondary stroke prevention with antiplatelet agents is recommended for patients with prior noncardioembolic ischemic TIA or stroke and identified carotid disease, unless otherwise contraindicated.

Aspirin 50 to 325 mg, clopidogrel, and the combination of aspirin/extended-release dipyridamole are all acceptable treatment options. Anticoagulation therapy is recommended in stroke patients with known arrhythmias or hypercoagulable states. If on warfarin, the INR must be monitored regularly and should be kept within the range of 2.0 to 3.0. Newer agents are available that may be preferable because they are as effective and may be safer (have lower risk profiles), do not require routine blood tests, nor dietary adjustment in patients with embolic stroke of unknown etiology; ongoing screening for atrial fibrillation will alter treatment course if found. Carotid endarterectomy (CEA) or carotid angioplasty and stenting (CAS) may be considered for stenosis greater than 50% (Kernan et al., 2014).

Referral: Referrals depend on the type and severity of impairment from the stroke and the influence of any comorbid illnesses. Referrals to physiatry (rehabilitation medicine), neurology, vascular surgery, psychiatry, and cardiology for consultation are common.

Education: Stroke education should address stroke prevention, treatment, and recovery. For stroke survivors, emphasize the cause of the stroke, control of individual risk factors to prevent future events, and importance of early identification with EMS activation and treatment of recurrent stroke. A home exercise program to maintain or improve functional levels should be introduced and strongly encouraged. Programs with balance training components have been shown to reduce falls (Winstein et al., 2016). The individually tailored exercise program will also enhance cardiorespiratory fitness and reduce risk of recurrent stroke, as well as improve cognition and memory. Information to access stroke support groups for patients and caregivers should be provided. Emphasis should be placed on the importance of periodic follow-up with the primary care provider for recovery monitoring, surveillance for complications, ongoing management of risk factors, and secondary prevention. The National Stroke Association can be contacted at info@stroke.org and 1-800-STROKES (1-800-787-6537).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Transport rapidly to the closest certified stroke center. May involve air transport and hospital bypass.	B	Jauch et al., 2013 Powers et al., 2018
A stroke rating scale, preferably the NIHSS, is recommended for initial assessment.	B	Powers et al., 2018

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
IV alteplase is recommended within 3 hours of ischemic stroke for those who meet eligibility. IV alteplase in the 3- to 4.5-hour window is also recommended for patients who are eligible.	A B	Powers et al., 2018
For patients eligible for alteplase, benefit is time dependent, and should be initiated as quickly as possible. Door-to-needle should be within 60 minutes in ≥50% of patients.	A B	Powers et al., 2018
Endovascular therapy recommended if the patient meets all of the following: a) Prestroke modified Rankin Scale score 0 to 1 b) Received alteplase within 4.5 hours on onset c) Caused by occlusion of internal carotid or middle cerebral artery d) Age ≥18 years e) NIHSS score ≥6 f) ASPECTS score of ≥6, and g) Treatment initiated within 6 hours of symptom onset	A	Powers et al., 2018
Endovascular therapy recommended within 6 to 16 hours in select patients.	A	Powers et al., 2018
Endovascular therapy recommended within 16 to 24 hours in select patients.	B	Powers et al., 2018
Emergency imaging of the brain recommended prior to initiating acute stroke therapy.	A	Jauch et al., 2013
Noninvasive imaging of the cervical vessels should be performed routinely as part of the evaluation for patients with suspected TIAs.	A	Jauch et al., 2013
Noninvasive imaging of the intracranial vasculature with CT angiography or magnetic resonance angiography to exclude intracranial stenosis/occlusion. Reliable degree of intracranial stenosis requires catheter angiography to confirm abnormalities detected.	A	Jauch et al., 2013
Patients with transient neurological symptoms should undergo neuroimaging evaluation within 24 hours of symptom onset or as soon as possible in patients with delayed presentation. MRI is preferred modality, but if not available, head CT scan should be performed.	B	Jauch et al., 2013
Anticoagulation with goal to prevent early recurrent stroke, halt neurological worsening, or improve outcomes is not recommended in acute ischemic stroke.	A	Jauch et al., 2013
Anticoagulation to manage noncerebrovascular conditions is not recommended due to risk of intracranial hemorrhagic complications.	A	Jauch et al., 2013
Initial dose of aspirin 325 mg is recommended within 24 to 48 hours of stroke onset.	A	Jauch et al., 2013
Stroke unit care that incorporates rehabilitation is recommended.	A	Jauch et al., 2013

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Swallow assessment prior to eating, drinking, or taking medications is recommended in all stroke patients.	B	Jauch et al., 2013 Hemphill et al., 2015
Patients with large infarctions are at high risk for edema and increased intracranial pressure. Measures to decrease risk and close monitoring during the first few days are recommended.	A	Jauch et al., 2013
Routine blood tests (CBC, chemistry, coagulation studies, and fasting lipid panel) are reasonable in the evaluation of suspected stroke/TIA.	B	Jauch et al., 2013
Baseline EKG is recommended to screen for atrial fibrillation or other cardiac arrhythmias.	B	Jauch et al., 2013
For patients with a TIA or ischemic stroke within the past 6 months and ipsilateral severe (70%–99%) carotid artery stenosis, CEA is recommended if the perioperative morbidity and mortality risk is <6%. For patients with ipsilateral moderate (50%–69%) carotid stenosis, CEA is recommended depending on patient-specific factors, such as age, sex, and comorbidities. When degree of stenosis is <50%, there is no indication for CEA or CAS.	A B A	Kernan et al., 2014
For patients with ischemic stroke or TIA with persistent or paroxysmal nonvalvular atrial fibrillation, anticoagulation with a vitamin K agonist (VKA) (target INR, 2.5; range, 2.0 to 3.0), apixaban, dabigatran, and rivaroxaban are all recommended. For patients unable to take anticoagulation, aspirin 325 mg/day is recommended.	A (VKA, apixaban) B (dabigatran, rivaroxaban) A (aspirin if no anticoagulation)	Kernan et al., 2014
In patients with ischemic stroke or TIA in sinus rhythm that have a left atrial or ventricular thrombus identified, anticoagulation with a VKA is recommended for ≥3 months.	C	Kernan et al., 2014
For patients with ischemic stroke or TIA who have dilated cardiomyopathy (LVEF ≤35%) or restrictive cardiomyopathy without clot, the effectiveness of anticoagulation is uncertain and decision should be individualized.	B	Kernan et al., 2014
For patients with ischemic stroke or TIA who have rheumatic mitral valve disease and atrial fibrillation, long-term VKA therapy with a target INR of 2.5 (range 2.0–3.0) is recommended.	A	Kernan et al., 2014
For patients with ischemic stroke or TIA who have mechanical prosthetic heart valves, warfarin is recommended with an INR target of 2.5 (aortic) and 3.0 (mitral).	B (aortic) C (mitral)	Kernan et al., 2014
For patients with noncardioembolic ischemic stroke or TIA, antiplatelet agents rather than oral anticoagulation is recommended to reduce the risk of recurrent stroke and other cardiovascular events. Aspirin (50 to 325 mg/day), the combination of aspirin and extended-release dipyridamole, or clopidogrel are all acceptable options for initial therapy.	A A (aspirin alone) B (aspirin and dipyridamole, clopidogrel)	Kernan et al., 2014
For patients with large vessel stenosis, the addition of clopidogrel 75 mg/d to aspirin for 90 days might be reasonable.	B	Kernan et al., 2014

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Combination of aspirin and clopidogrel when continued for 2 to 3 years, increases the risk of hemorrhage and is not recommended long-term.	A	Kernan et al., 2014
In ICH, 1 to 4 days after bleeding has stopped, it is okay to use low-molecular weight heparin or low dose unfractionated heparin for venous thromboembolism prevention.	A	Hemphill et al., 2015
Initial monitoring and management of ICH patients should take place in an ICU or stroke unit.	B	Hemphill et al., 2015
In ICH, clinical seizures should be treated with antiseizure medications.	A	Hemphill et al., 2015
For most with supratentorial ICH, usefulness of surgery is not well established.	A	Hemphill et al., 2015
Cerebellar ICH patients who are deteriorating or have brainstem compression and/or hydrocephalus should undergo surgical decompression as soon as possible.	B	Hemphill et al., 2015
BP should be immediately controlled in all ICH patients. It is safe to reduce systolic BP to <140 mm Hg.	A	Hemphill et al., 2015
All ICH patients should have access to multidisciplinary rehabilitation.	A	Hemphill et al., 2015
Appropriate management of medical comorbidities (hypoglycemia and hyperglycemia, hyperthermia, hypoxia, arrhythmias, etc.) during the acute, subacute and chronic phases of stroke recovery can significantly improve outcomes.	A	Kernan et al., 2014 Hemphill et al., 2015 Winstein et al., 2016
Prolonged cardiac monitoring (~30 days) for atrial fibrillation is useful in TIA/stroke patients with an unclear origin within 6 months of the index event.	C	Kernan et al., 2014
Stroke survivors receive rehabilitation based on benefit and tolerance.	B	Winstein et al., 2016
For stroke survivors who qualify and have access, care in an inpatient rehabilitation facility is preferred to a skilled nursing facility.	B	Winstein et al., 2016
Targeted injection of botulinum toxin into upper limbs is used to improve range of motion and lower limbs to reduce spasticity.	A	Winstein et al., 2016
Balance training program for those with poor balance and fall risk.	A	Winstein et al., 2016
Intensive, repetitive, mobility training for all stroke survivors with gait limitations.	A	Winstein et al., 2016
ADL training tailored to individual needs and discharge setting.	A	Winstein et al., 2016 VA/DoD, 2010
Family, social support, and community resources should be identified early and included in the plan of care.	C	Winstein et al., 2016 VA/DoD, 2010

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Stroke survivors should be regularly screened for depression and treated accordingly throughout the subacute and chronic phases of stroke recovery.	B	Winstein et al., 2016 VA/DoD, 2010
Individual exercise program indicated to enhance cardiorespiratory fitness and reduce the risk of stroke recurrence.	A (improved fitness) B (reduction stroke risk)	Winstein et al., 2016
Stroke patients with functional limitations in ADLs and mobility should be considered for community- or home-based rehabilitation post discharge.	A	Winstein et al., 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

Sue is a 68-year-old healthy woman with no significant medical history. She is in the office today with complaint of intractable nausea and vomiting for the past 5 weeks with an 11-pound weight loss. On review of systems she also has noted a dull, persistent headache, difficulty with concentration, and some blurred vision. She states these other problems are likely due to this gastrointestinal "bug" that will not go away. She denies similar symptoms in the past. Her mother died from complications of diabetes at 42 years old, her father died from a heart attack in his late fifties, one brother died in a car accident in his late teens, and the other died from complications of diabetes in his late teens. She is a nurse, married, with two adult children who are in good health. She drinks a beer or glass of wine on most days, sometimes two. She has never used tobacco products or illegal drugs.

1. What additional subjective data are you seeking?
2. What additional objective data will you be assessing for?
3. What are the differential diagnoses that you are considering?
4. What laboratory tests will help you rule out some of the differential diagnoses?
5. What radiological examinations would you order?
6. What is your treatment and specific information on the prescription you will give to this patient?
7. What are the potential complications from the treatment ordered?
8. What are the additional specific laboratory tests you may consider ordering?
9. What additional patient teaching may be needed?
10. Will you be looking for a consultation?

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CHAPTER 14

Endocrine, Metabolic, and Nutritional Disorders

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ASSESSMENT

Accurate history taking of complaints associated with endocrine, metabolic, and nutritional disorders is essential for completing the examination of the older adult. Recognizing that normal aging changes of the endocrine system primarily are related to a decrease in pancreatic function (inability to sufficiently secrete insulin) and alterations in thyroid function, the nurse practitioner needs to screen older adults periodically for diabetes mellitus and thyroid disease because the incidence of these conditions increases with age (Cai, McNeilly, Luttrell, & Martin, 2012)

Overall, the nurse practitioner managing the care of older adults needs to differentiate among four clinical states:

1. Endocrine function that is altered relative to that of younger patients, but is an expected consequence of normal aging
2. Altered endocrine function secondary to coincident nonendocrine disease, but is not of known pathological significance
3. Iatrogenic changes in endocrine function that largely reflect the polypharmacy seen in the older adult population
4. Authentic endocrinopathy (Davis, Davis, & Leinung, 2007)

Recognition of Endocrine, Metabolic, and Nutritional Disorders

Because changes in the endocrine system may appear subtle to the older person or atypical as compared to younger patients, it may be difficult to pinpoint the onset of the presentation (Silverman, 1990). Reevaluate the patient's family history for endocrine and metabolic disease (Resnick, 2016). Explore with the patient any difficulty with temperature regulation, changes in skin texture, or distribution of body hair. Review with the patient any episodes of unexplained weight loss or gain, new or increased fatigue, weakness, malaise, and recent infections (Ahmed & Haboubi, 2010; Leslie & Hankey, 2015).

Focusing the Endocrine, Metabolic, and Nutritional Disorder Review of Systems

Given the vagueness or atypical presentation of endocrine, metabolic, and nutritional disorders in older adults, specific questions directed at these conditions are imperative during the review of systems:

- Is there any alteration in the ability to carry out activities of daily living (ADLs) or instrumental ADLs due to fatigue or subjective weakness?
- Has there been any acute changes in memory or mood?
- Have there been any decrease in appetite or reported change in weight?
- Has the patient noticed any excessive thirst, appetite, or urination?
- Is the patient experiencing incontinence for the first time?
- Has the patient experienced any repeated yeast infections?
- Has the patient had a skin ulcer that has failed to heal?
- Are there any mouth ulcerations?
- Are there any new episodes of chest pain, palpitations, tachycardia, dyspnea?
- Has the patient noticed a new onset of constipation or diarrhea?
- Has the patient noticed any complaints of paresthesias or carpal tunnel syndrome?
- Has the patient reported any hoarseness of the voice?
- Has the patient experienced vertigo or gait disturbances?
- Are there any other symptoms that could be clustered to form a differential diagnosis?
- Is there any changes in the color or texture of the skin?
- Is there any noticeable dryness to hair, loss of hair?
- Has the patient noticed any intolerance to heat, sweating or flushing?
- Are there any changes to tolerance of cold?

- Is there any new presentation of abdominal pain and associated symptoms such as nausea or vomiting?
- Is the male patient reporting impotence?

It is important to determine from the patient what therapeutic measures have been initiated or prescribed in the past to alleviate any of the symptoms and what response occurred following treatment (LeBlond, Brown, Suneja, & Szot, 2015).

Approach to the Physical Examination

The physical examination should focus on the clinical signs of endocrine, metabolic, and nutritional disorders prevalent in older adults, recognizing, however, that they may present atypically. Evaluation of older vital signs and previous weight and height should be noted when recording current measures; note differences that could be indicative of unintentional weight loss (malnutrition and/or failure to thrive), vertebral fractures (reduction in inches), and alteration in temperature, pulse, respiration, and blood pressure (underlying thyroid disease). Examine the skin for changes in texture, temperature, evidence of infection and/or rashes, or open, nonhealing wounds. Inspect the hair distribution and note any dryness, coarseness, or brittleness of hair. Determine if the hair distribution of the eyebrows has changed (known as madarosis or Queen Anne's sign), which is a presentation found in hypothyroidism (Kumar & Karthikeyan, 2012).

Note any changes to the nail beds. Are they brittle, cracked, peeling, or discolored? Examination of the tongue and mouth could reveal signs of nutritional deficiencies (depapillated tongue and cheilitis caused by vitamin B_{12} deficiency). Perform a thyroid examination, looking for enlargement, tenderness, and evidence of goiter or nodularity (Williams, 2008).

A thorough cardiac and respiratory examination is essential given the new manifestations of conditions such as atrial fibrillation, hypertension, or heart failure in patients with undiagnosed hyperthyroid disease. Examine the lower extremities for edema; nonpitting edema is a clinical sign of pretibial myxedema. Neurological examination in older patients with undiagnosed diabetes mellitus may reveal alterations in temperature, vibratory sense, and sensation. A fine tremor in the fingers and hands may be noted. Reflexes in an older adult may be altered by abnormal thyroid function. Testing the patient's proximal and distal strength is also important, because weakness can manifest in thyroid disease. As patients and/or their caregivers may report changes in cognition, an objective assessment of mental status is warranted in patients with untreated thyroid disease (Resnick, 2016).

Medication Review

A thorough review of all medications, including over-the-counter (OTC) medications and home remedies, is important to discern if drug–drug interactions are responsible for alterations in the absorption of medications. Certain medications may be responsible for a new onset of anorexia, diarrhea, or constipation (Elsawy & Higgins, 2011). Essential, too, is a thorough review of how the patient and/or medical facility are administering the medications (timing the doses to not conflict with food). Finally, review patient adherence and understanding of the medication regimen.

ACUTE PANCREATITIS

Signal Symptoms: Mild-to-severe sharp epigastric pain with possible radiation to the back, chest, or flanks. Nausea and vomiting accompanies the pain in up to 90% of patients.

Description: Acute pancreatitis is the result of acute inflammation of the pancreas, an organ that produces enzymes such as amylase, lipase, trypsin, and chemotrypsin to aid in digestion, as well as the hormone insulin, which controls the level of glucose in the blood. It is located in the upper abdomen, behind the stomach. The enzymes are secreted into the duodenum through the pancreatic duct and join with bile produced by the liver to digest food. Generally, these enzymes do not become active until they reach the small intestine; however, the enzymes can become activated within the pancreas where they attack the pancreatic cells, causing damage and inflammation (Quinlan, 2014). Acute pancreatitis typically has a sudden onset and in a majority of patients resolves in 3 to 5 days with treatment and without complications. However, some patients progress to developing complications, which can include chronic pancreatitis, pancreatic pseudocyst, necrosis, organ failure, and death with a mortality rate of up to 30% in patients with severe acute pancreatitis (Quinlan, 2014).

Etiology: The two most common causes of acute pancreatitis are obstruction by gallstones, accounting for 40% to 70% of cases, and alcohol, accounting for 25% to 35% of cases (Tenner, Baillie, DeWitt, Vege, & American College of Gastroenterology, 2013; Yang, Vadhavkar, Singh, & Omary, 2008). If not due to gallstones or alcohol use, two less common but useful causes to investigate in the older adult are drug-induced pancreatitis and hypertriglyceridemia-induced pancreatitis, typically with serum triglycerides over 1,000 mg/dL (Goyal, Smith, Bayer, Rutherford, & Shelnut, 2016). Other possible causes of acute pancreatitis are many and include viral infections (mumps, coxsackie B, mycoplasma pneumonia, and campylobacter), abnormalities of the pancreas itself, obstruction by pancreatic neoplasm, hereditary diseases such as cystic fibrosis, autoimmune disorders such as systemic lupus erythematosus, complication of endoscopic retrograde cholangiopancreatography (ERCP) (occurs in approximately 3% of post-ERCP patients), and trauma to the abdomen (Forsmark, Baillie, AGA Institute Clinical Practice and Economics Committee, & AGA Institute Governing Board, 2007). The list of drugs causing acute pancreatitis is long and includes sulfonamides, corticosteroids, thiazide diuretics, NSAIDs, estrogens, and antibiotics such as tetracycline, among others (Carroll, Herrick, Gipson, & Lee, 2007).

Several studies, including a meta-analysis by Majumder, Gierisch, and Bastian (2015) suggest cigarette smoking is in independent risk factor for both acute and chronic

pancreatitis. Gardner and Berk (2010) recorded median ages of onset for various etiologies, with biliary tract related (median age 69 years) and trauma related (median age 66 years) having a median age within the geriatric age group. Despite numerous etiologies, it should be noted that up to 15% to 25% of patients with acute pancreatitis are idiopathic with no identifiable cause.

Occurrence: Acute pancreatitis is the leading gastroenterology cause of hospitalization with nearly 275,000 hospitalizations annually (Peery et al., 2012). Eighty percent of pancreatitis is considered mild, and those persons generally recover without complication. The other 20% have severe disease with local and systemic complications. The overall mortality rate for acute pancreatitis is approximately 1%, although this rate increases depending on certain factors like age, comorbidity, and disease severity (Yadav & Lowenfels, 2013). For severe acute pancreatitis with organ failure, the death rate can be as high as 30% (Working Group IAP/APA Acute Pancreatitis Guidelines, 2013).

Age: Acute pancreatitis is more common in the elderly, with the incidence rate (per 100,000 population) for those age 75 years and older double that of those age 35 to 44 years in males and nearly triple that in females for the same age comparison groups (Yadav & Lowenfels, 2013). Mortality, likewise, increases with age. A study by Fan, Choi, Lal, and Wong (1988) found mortality for acute pancreatitis at 5.9% for those age 50 years and younger and 21.3% for those age 75 years and older. The reasons are attributed to diminished organ function, comorbid illnesses, increased susceptibility to infection and ischemia, and age-associated changes in the pancreas and biliary system (Skolnick, Feller, & Nanda, 2008).

Gender: Acute pancreatitis affects males and females nearly equally (Yadav & Lowenfels, 2013); however, alcohol-related etiology is more common in men than in women, although the rates equalize when level of alcohol consumption is equal. Gallstone etiology is more common in women compared to men.

Ethnicity: African Americans have a two to three times higher risk of acute pancreatitis than do Caucasians (Yadav & Lowenfels, 2013). The hospitalization rates of African Americans are three times higher than for Caucasians and are more pronounced for males than females. For those ages 35 to 64 years, African American hospitalization rates are 10 times higher than for any other group (Gardner & Berk, 2010).

Contributing Factors: Biliary tract disease, binge alcohol use, recent surgery, family history of high triglycerides (typically over 1,000 mg/dL), and increasing age are some of the contributing factors. There is some disagreement on other potentially contributing factors. Some studies cite medications as causing up to 2% of acute pancreatitis cases (Quinlan, 2014); others assert that factors such as medications, hypercalcemia, hyperparathyroidism, and infection are falsely attributed as causative factors to acute pancreatitis because the evidence supporting these is lacking and therefore causative links are only theoretical (Tenner et al., 2013).

Signs and Symptoms: Examination findings will vary depending on severity of the attack. With a mild attack, the epigastric area may be minimally tender; however, with severe episodes, there may be abdominal distention, tenderness, and guarding. Respirations may be shallow due to diaphragmatic irritation. There may be ecchymotic discoloration of the flank (Grey-Turner's sign) or in the periumbilical area (Cullen's sign), which reflect intra-abdominal hemorrhage that occurs in about 1% of cases and is associated with a poor prognosis (Steven & Conwell, 2010).

Diagnostic Tests: Amylase and lipase amounts are generally three times the normal amount during acute pancreatitis and are the most common laboratory markers used to establish a diagnosis of acute pancreatitis. Amylase is the most frequently ordered test, which rises within 6 to 12 hours of onset of pain and peaks around 24 hours, returns to normal within 3 to 5 days, and has a sensitivity of 81% to 95% (Basnayake & Ratnam, 2015). Lipase has a higher sensitivity for acute pancreatitis (85% to 100%), rises within 8 hours of onset of symptoms, peaks at 24 hours, and returns to baseline within 8 to 14 days; it is now the preferred diagnostic blood test. However, they can both be nonspecific depending on the time the abdominal pain began, other possible intra-abdominal processes, and chronic comorbid diseases such as renal insufficiency. Amylase levels may be normal in an alcoholic, and plasma lipase becomes the more sensitive and specific test, but bear in mind that the level of the pancreatic enzyme does not correlate with the severity of the disease. Another potential marker of acute pancreatitis is trypsinogen activation peptide (TAP) that becomes markedly increased and may be useful in detection of early acute pancreatitis. Other useful laboratory tests are a metabolic panel to look at blood urea nitrogen (BUN), creatinine, glucose, and calcium levels, as well as liver function tests, triglycerides, a complete blood count (CBC), arterial blood gases (ABGs), and a urinalysis (Carroll et al., 2007).

Radiological evidence is used to confirm or exclude the clinical diagnosis, establish a cause, assess severity, and provide guidance for therapy. An abdominal x-ray is generally done first and helps to exclude other causes of abdominal pain, such as an obstruction or bowel perforation. Findings in more severe disease is an ileus or "colon cut-off sign" reflecting paucity of air in the colon due to spasm of the descending colon. This may be followed by an abdominal ultrasound, which is 87% to 98% sensitive for detection of gallstones, as well as for showing a diffusely enlarged hypoechoic pancreas. Abdominal computed tomography (CT) scan with contrast is the standard imaging technique for detecting acute pancreatitis and for assessing severity (78% sensitive and 86% specific for severe acute pancreatitis) (Steven & Conwell, 2010). However, it is not recommended at initial presentation because there is no evidence that it improves outcomes and complete extent of necrosis may not be clear until 72 hours after onset (Vengadakrishnan & Koushik, 2015).

Endoscopic ultrasonography (EUS) is especially helpful in documenting stones and tumors and is useful in obese patients. It can also be useful in determining which patients with acute pancreatitis would benefit from a therapeutic ERCP (100% sensitive and 91% specific for gallstones). Magnetic resonance cholangiopancreatography (MRCP) may be ordered to assess for inflammation or calcium deposits or for changes in the ducts of the pancreas. MRCP has been found to be as accurate as CT scan in predicting severity of pancreatitis

and identification of pancreatitis complications (Stimac et al., 2007). It is a newer, noninvasive technique and is comparable to ERCP for cholelithiasis detection but does not have the interventional capacity for stone extraction, stent insertion, or biopsy, as the ERCP does. However, advantages include no radiation exposure compared to CT scan, gadolinium contrast in MRI has a lower risk of renal complications than iodine contrast in CT scan, and for patient with contraindication to contrast, nonenhanced MRI can still detect pancreatic necrosis. ERCP may be used for the less common causes of pancreatitis such as sphincter of Oddi dysfunction or pancreatic duct strictures. It is indicated for those persons who have not passed a stone during the acute attack and those with evidence of sepsis, biliary obstruction, cholangitis, elevated bilirubin worsening, or persistent jaundice or worsening pain in the presence of an abnormal ultrasound (Carroll et al., 2007; Cherian, Slvaraj, Natrayan, & Venkataraman, 2007).

Most patients will have mild disease, but it is important to know as early as possible whether the attack is mild or severe for purposes of management and use of resources. There are several scoring systems used to identify early prognostic signs to predict the severity of pancreatitis, including the Atlanta Classification of Severe Acute Pancreatitis, Ranson's criteria, the APACHE II scale (Acute Physiology and Chronic Health Evaluation), the Systemic Inflammatory Response Syndrome (SIRS) Score, and the Computed Tomography Severity Index (CTSI). Each has its pros and cons. With all scoring systems, the higher the prognostic score, the poorer the clinical outcome.

The Atlanta classification is both used to diagnose acute pancreatitis and rate the severity of pancreatitis. Diagnosis is made based on the Atlanta classification when two of three criteria are met: abdominal pain consistent with acute pancreatitis, laboratory abnormalities (serum amylase and/or lipase three times the normal limit or higher), and imaging findings consistent with acute pancreatitis. To rate severity, it uses prognostic signs (a score of 3 or higher on the Ranson criteria and an 8 or higher on the APACHE II) and presence of local pancreatic necrosis (peripancreatic fluid collection, etc.) and organ failure (Alexakis & Neoptolemos, 2005; Quinlan, 2014).

The Ranson criteria was the first system and remains the best known and most widely used, but has drawbacks in that it requires 2 days' worth of objective measures for the total score, five to be obtained on the first day and six evaluated within 48 hours of the onset of pain, sensitivity being only 73% and 77% to predict mortality, and the threshold for abnormal values depends on whether the pancreatitis is caused by alcohol or gallstones (Balthazar, 2002). The criteria on admission or diagnosis include:

- Age of more than 55 years
- White blood cell (WBC) count greater than 16,000/mm^3 (16.0×10^9/L)
- Blood glucose greater than 200 mg/dL (11.1 mmol/L)
- Serum lactate dehydrogenase greater than 350 U/L
- Aspartate aminotransferase (AST) greater than 250 U/L

During the initial 48 hours:

- Hematocrit decrease greater than 10%
- BUN increases greater than 5 mg/dL (1.8 mmol/L)
- Serum calcium less than 8 mg/dL (2 mmol/L)
- Base deficit greater than 4 mmol/L (4 mEq/L)
- Fluid sequestration greater than 6,000/mL
- PaO_2 less than 60 mm Hg

Scoring: One point for each criterion met. The prognostic implication is as follows: 0 to 2 means 2% mortality; 3 to 4 means 15% mortality; 5 to 6 means 40% mortality; and 7 to 8 means 100% mortality.

The APACHE score can be used to assess the patient at any point during the illness, but is cumbersome to use; sensitivity is 77% and specificity is 84%. The equation includes the following factors: age, rectal temperature, mean arterial pressure, heart rate, PaO_2, arterial pH, serum potassium, serum sodium, serum creatinine, hematocrit, WBC count, Glasgow Coma Scale score, chronic health status. *Scoring:* Can be calculated at www.sfar.org/scores2/apache22.html#calcul.

The CT Severity Index developed by Balthazar (2002) is the only scoring system to use evidence of necrotic pancreas as criteria. A key criterion for identification of patients at higher risk for fatal pancreatitis is pancreatic necrosis.

CT grade:

- A is normal pancreas (0 points)
- B is edematous pancreas (1 point)
- C is B plus mild extrapancreatic changes (2 points)
- D is severe extrapancreatic changes plus one fluid collection (3 points)
- E is multiple or extensive fluid collections (4 points)

Necrosis score:

- None (0 points)
- Less than one-third (2 points)
- Greater than one-third but less than one-half (4 points)
- Greater than one-half (6 points)

Scoring: CT grade plus necrosis score. The maximum score is 10, and a score of 6 or higher is considered severe disease.

The presence of systemic inflammatory response syndrome (SIRS) has been associated with an increase in mortality from acute pancreatitis. A score based on the presence of SIRS can be used on a daily basis to reliably predict acute pancreatitis severity. One study found that patients with persistent SIRS from admission, presence of SIRS on admission that was not persistent, and no SIRS had mortality rates of 25%, 8%, and 0%, respectively (Mofidi et al., 2006). Another study found that the presence of three or four SIRS criteria is associated mortality (Singh et al., 2009). SIRS is present if the patient meets two or more of the following criteria:

- Temperature greater than 38.3°C (100.4°F) or less than 36.0°C (96.8°F)
- Heart rate greater than 90 beats/minute
- Respiratory rate greater than 20 breaths/minute or $PaCO_2$ less than 32 mmHg
- WBC count greater than 12,000 cells/mL, less than 4,000 cells/mL, or greater than 10% immature (band) cells

At initial presentation, pancreatitis severity should be estimated using an established classification method, such as the Atlanta classification system, rather than clinical judgement alone. Additionally, hemodynamic status should be assessed immediately on presentation to allow timely treatment and fluid resuscitation (Quinlan, 2014; Tenner et al., 2013).

Differential Diagnosis:

- Cholangitis
- Cholecystitis
- Choledocholithiasis
- Cholelithiasis
- Colon cancer
- Colonic obstruction
- Diabetic ketoacidosis
- Dissecting aneurysm
- Diverticulitis with/without perforation
- Duodenal or gastric ulcers
- Gastric or pancreatic cancer
- Gastric outlet obstruction
- Gastric volvulus
- Hepatitis
- Intestinal infarction
- Mesenteric ischemia
- Myocardial infarction
- Perforated peptic ulcer
- Pneumonia
- Tubo-ovarian abscess (Quinlan, 2014)

Treatment: For acute pancreatitis, a short stay in the hospital is generally required for IV fluid replacement and pain management. Aggressive fluid resuscitation of 5 to 10 mL kg/hr is recommended in the management of acute pancreatitis, unless contraindicated due to cardiac, renal, or other condition (Gardner, Vege, Pearson, & Chari, 2008; Haydock et al., 2013). Fluid status should be frequently reassessed based on blood urea nitrogen (BUN) and hematocrit. Clinical signs and adequate fluid resuscitation have been linked to reduced mortality (Working Group IAP/APA Acute Pancreatitis Guidelines, 2013). Antibiotics are generally not indicated for a mild case (Steven, Parsi, & Walsh, 2009). For patients with gallstone pancreatitis, a cholecystectomy should be performed during the hospital stay, generally after at least 24 hours of treatment. Patients can begin on a low-fat, low-residue, soft diet.

Persons admitted with severe acute pancreatitis or certain high-risk patients (older adults, gross obesity, diabetes) may require intensive care because multiple complications can develop within hours or days. These patients are kept on NPO status with aggressive fluid hydration; enteral nutrition is safer and more effective regarding the suppression of the immune-inflammatory response (Tenner et al., 2013; Working Group IAP/APA Acute Pancreatitis Guidelines, 2013). In rare cases, acute pancreatitis can cause hypoxia, and this is more common in the older adult. Antibiotics should be used if there is evidence of pancreatic necrosis. If there is vomiting, a nasogastric tube may be inserted to remove fluid and air (Neoptolemos, 2009). Clear liquids followed by a diet high in carbohydrates and low in fat will be instituted, and the patient will be advised to have more frequent but smaller meals.

Follow-Up: Treatment of the patient must address underlying causes of pancreatitis. If it is alcohol consumption, treatment should go beyond just telling the patient to quit drinking. An endocrinology consultation may be assistive for those with hypertriglycerides or hypercalcemia-induced pancreatitis. Those with medication-induced pancreatitis may benefit from a pharmacology consultation. The acronym PANCREAS can be used to remember management particulars: Perfusion, Analgesia, Nutrition, Clinical management, Radiology, ERCP, Antibiotics, and Surgery (Khaliq, Dutta, Kochhar, & Singh, 2010).

Sequelae: Gallstones may be the cause of acute pancreatitis and will need to be removed. Removal of the gallbladder may be done by cholecystectomy if the pancreatitis is mild. If the pancreatitis is more severe, the gallstones may be removed using ERCP. A cholecystectomy will be performed after a full recovery from acute pancreatitis. Other possible complications can be an abscess or pseudocysts. Newly diagnosed patients with acute pancreatitis are at risk for developing prediabetes or diabetes after their initial attack (Das et al., 2013). Acute pancreatitis can also cause kidney failure through acute tubular necrosis. Repeated episodes of acute pancreatitis can lead to chronic pancreatitis.

Prevention/Prophylaxis: Depending on the cause of the acute pancreatitis episode, the person needs to be counseled on the cause, such as medication-induced pancreatitis, alcoholism, or gallstone pancreatitis.

Referral: A dietary consultation may be helpful for the patient in order for changes to be made to lessen reoccurrence, especially if the acute pancreatitis is due to high triglycerides or calcium. A referral for behavior modification or a treatment program can be useful for the person who continues to drink alcohol.

Education: It is necessary for persons who have had an episode of acute pancreatitis to understand the mechanism behind the episode so if there are changes to be made, they understand the importance of them.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Total enteral nutrition is equal to or more effective than total parenteral nutrition for nutritional management of patients with severe pancreatitis.	A	Besselink, Santvoort, Buskens, & Gooszen, 2006 Al-Omran, Al-Balawi, Tashkandi, & Al-Ansary, 2010
Evaluate for less common causes of pancreatitis (e.g., sphincter of Oddi dysfunction, pancreas divisum, and pancreatic duct strictures) with endoscopic retrograde cholangiopancreatography.	C	Carroll et al., 2007 Cherian et al., 2007

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Contrast-enhanced CT scan is the standard imaging technique for detection of acute pancreatitis. CT scan is not generally indicated for patients with mild, uncomplicated pancreatitis but should be reserved for cases of clinical or biological worsening.	C	Steven & Conwell, 2010 Bollen et al., 2011 Arvanitakis et al., 2004 Zaheer, Singh, Qureshi, & Fishman, 2013
It is controversial whether antibiotics reduce mortality in patients with necrotic pancreatitis.	B	Steven et al., 2009 Jiang, Huang, Yang, & Xia, 2012
Urgent endoscopic retrograde cholangiopancreatography is indicated in patients with or at risk for biliary sepsis.	A	Kapetanos, 2010
Aggressive hydration, 250 to 500 mL of normal saline or lactated Ringer's should be given to all acute pancreatitis patients, unless contraindicated by other comorbidities (renal, cardiovascular, or other). This is most critical in the first 12 to 24 hours.	A	Tenner et al., 2013
Fluid needs should be reassessed within 6 hours of admission and over the following 24 to 48 hours. The goal of aggressive hydration is a reduction in BUN.	B	Tenner et al., 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CHRONIC PANCREATITIS

Signal Symptoms: Fifty percent to 80% of people experience upper abdominal pain, often in the mid or left upper region and radiating in a band-like fashion to the midback, and it is usually dull or boring in quality. The pain may feel worse after eating or drinking, and also become chronic and disabling. The pain is not fleeting or transient; it tends to last several hours and for some, several weeks. Most persons experience intermittent attacks of pain at unpredictable intervals over years.

Description: Chronic pancreatitis is inflammation of the pancreas that does not heal or improve, gets worse over time, and leads to permanent damage. It occurs due to the same underlying causes of acute pancreatitis; the digestive enzymes attack the pancreas but it now leads to chronic and irreversible inflammation, leading to fibrosis and calcification. This, in turn, results in exocrine and endocrine insufficiency, causing diabetes mellitus and steatorrhea, and often, chronic, disabling pain. The TIGAR-O classification system is based on risk factors for chronic pancreatitis. TIGAR-O stands for Toxic-metabolic, Idiopathic, Genetic, Autoimmune, Recurrent, Obstructive (Conwell et al., 2014).

Etiology: The majority of chronic pancreatitis cases have been attributed to excessive alcohol use, typically 80 g per day for 6 to 12 years, and can account for up to 70% of cases (Duggan, Ní Chonchubhair, Lawal, O'Connor, & Conlon, 2016; Hobbs, Johnson, & Graham, 2016; Spicak et al., 2007). Chronic pancreatitis can also be triggered by an acute attack that damages the pancreatic duct, which causes the pancreas to become inflamed and slowly destroyed. Other causes are hereditary disorders of the pancreas, cystic fibrosis, hypercalcemia, hyperlipidemia or hypertriglyceridemia, certain autoimmune disorders, obstruction by tumors, systemic diseases such as systemic lupus erythematosus (SLE) or hyperparathyroidism, some medications, as well as some causes that are still unknown (Goulden, 2013; Hobbs et al., 2016). Hereditary chronic pancreatitis has an estimated prevalence of 1 in 300,000 (Ellis, Lerch, Whitcomb, & Consensus Committees of the European Registry of Hereditary Pancreatic Diseases, Midwest Multi-Center Pancreatic Study Group, International Association of Pancreatology, 2001).

Autoimmune pancreatitis has been found in association with Sjögren's syndrome, primary biliary cirrhosis, and renal tubular acidosis (Epstein et al., 1982; Nishimori et al., 1994). With autoimmune chronic pancreatitis, other characteristics include diffuse enlargement of the pancreas, diffuse narrowing of the main pancreatic duct, increased circulating levels of gamma globulin, and the presence of autoantibodies (Ghazale et al., 2008; Raina et al., 2009). Cigarette smoking has also been identified as a risk factor for chronic pancreatitis and accelerates disease progression (Coté et al., 2011; Maisonneuve et al., 2005; Yadav et al., 2009).

Occurrence: The prevalence of chronic pancreatitis is estimated at 50 per 100,000 people (Yadav & Lowenfels, 2013). Based on U.S. Census Bureau estimates of the 2015 U.S. population at 321,418,820, this would translate to approximately 160,709 Americans with chronic pancreatitis in 2015 (U. S. Census Bureau, 2015).

Age: The onset of chronic pancreatitis related to alcoholism occurs in patients in their late thirties to forties. Older adults may develop idiopathic or obstructive pancreatitis (Shah, Farah, Goldwasser, & Agrawal, 2008).

Gender: Chronic pancreatitis is more common in men then in women (Yadav & Lowenfels, 2013). Alcohol plays a larger role in chronic pancreatitis in men; females tend to have chronic pancreatitis of idiopathic, autoimmune, or gallstone origins.

Ethnicity: There is a two- to three-fold greater increased risk of pancreatitis in African Americans than Caucasians (Yadav & Lowenfels, 2013). Additionally, African Americans with chronic pancreatitis have been shown to be more likely to be male, current or former smokers, and have etiology related to alcohol consumption (Wilcox et al., 2016). Also, African Americans were more likely to have advanced pancreatic morphological than Caucasians.

Contributing Factors: Alcohol use remains the greatest contributing factor, accounting for 70% of the disease (Hobbs et al., 2016). Idiopathic "late-onset" pancreatitis is more common in older adults. Obstructive chronic pancreatitis can be caused by an ampullary tumor or adenocarcinoma of the pancreas. Pancreatic stones also have been known to develop in older adults (Shah et al., 2008). Prolonged use of medications known to contribute to acute pancreatitis can lead to the development of drug-induced chronic pancreatitis (Shah et al., 2008).

Signs and Symptoms: The most important clue to a proper diagnosis of chronic pancreatitis is an accurate medical history. The most consistent presentation for chronic pancreatitis includes abdominal pain, typically dull, constant, epigastric pain with radiation to the back, neck, or flank, that can be worsened by food intake (Braganza, Lee, McCloy, & McMahon, 2011; Duggan et al., 2016; Pitchumoni, 1998). Pain is sometimes accompanied by nausea, vomiting, and weight loss (Braganza et al., 2011). Persistent pain is responsible for most hospitalizations related to chronic pancreatitis; however, some patients, as many as 20%, may have silent pancreatitis or only present with signs and symptoms of endocrine and/or exocrine pancreatic insufficiency (Duggan et al., 2016; Layer et al., 1994; Pitchumoni, 1998).

The pancreatic enzymes that are grossly elevated in acute pancreatitis may be normal or only mildly elevated. At times the abdominal pain goes away as the condition worsens, because the pancreas is no longer making enzymes. Other symptoms are related to the exocrine pancreatic insufficiency that impedes the body's ability to break down complex foods and properly absorb products of digestions. These symptoms include nausea and vomiting, weight loss, diarrhea, and oily stools (steatorrhea). Even if one's appetite is good, many with chronic pancreatitis lose weight due to these malabsorption deficiencies, even in the absence of steatorrhea, due to the pancreatic exocrine malfunctions (Dumasy, Delhaye, Cotton, & Deviere, 2004). However, it has been estimated that protein and fat deficiencies do not occur until over 90% of pancreatic function has been lost (DiMagno, Go, & Summerskill, 1973).

If hyperlipidemia is present, the funduscopic examination may reveal a milky white hue in the retinal blood vessels. Occasionally, a tender fullness or mass may be palpated in the epigastric area, suggesting a pseudocyst or an inflammatory mass in the abdomen. Patients who have protein and fat deficiencies associated with advanced disease may exhibit decreased subcutaneous fat, temporal wasting, sunken supraclavicular fossa, and other signs of malnutrition. Severe or rapid weight loss should be a red flag for the health-care provider, as it generally is pointing toward pancreatic cancer.

Diagnostic Tests: Symptoms between acute and chronic pancreatitis can be similar. Serum amylase and lipase levels may be slightly elevated in chronic pancreatitis but are often normal or even decreased with the atrophy of the gland itself. If there is obstruction of the biliary tract, one may see elevated bilirubin, serum alkaline phosphatase, and hepatic transaminase levels. In the more advanced stages of pancreatitis, when malabsorption is present, the health-care provider may order blood, urine, and stool tests (testing fecal fat content) to help diagnose and also monitor progression. However, maldigestion and malabsorption do not occur until more than 90% of the pancreas is destroyed.

Pancreatic function tests at present are direct tests, such as determining duodenal aspirates, but although they are sensitive, they are invasive and expensive. Generally, x-rays of the abdomen are first, which may show pancreatic duct calcifications in 30% of chronic pancreatitis patients. Transabdominal ultrasound, CT scan, MRI, and endoscopic ultrasound (EUS) may be further used to diagnose chronic pancreatitis and may show calcifications, pancreatic enlargement, pancreatic pseudocysts, or pancreatic ductal dilation. The sensitivity and specificity for ultrasound is 60% to 81% and 70% to 97%, respectively; for MRI/MRCP 88% and 98%, respectively; for CT 75% to 90% and 85%, respectively; and for EUS 80% to 100% for both sensitivity and specificity (Bolondi, Li Bassi, Gaiani, & Barbara, 1989; Luetmer, Stephens, & Ward, 1989; Mayerle et al., 2013).

While EUS has the highest possible sensitivity and specificity, it is invasive, has associated risks, and its accuracy is limited by the skill of the ultrasonographer (Bolondi et al., 1989; Nair et al., 2007). The invasiveness and associated risks are also concerns for ERCP and, actually, ERCP is also one of the possible etiologies for chronic pancreatitis, when used to extract gallstones during an acute attack. Magnetic resonance cholangiopancreatography (MRCP) is becoming the choice test for diagnosing chronic pancreatitis due to its ability to identify calcifications and duct obstructions in the disease but without the radiation exposure associated with CT scan imaging and invasive procedural risks of EUS and ERCP. Subsequently, ERCP is becoming limited to cases with potential need of therapeutic intervention. Although late-stage chronic pancreatitis is often readily apparent on imaging tests, the diagnosis of early ("minimal change") chronic pancreatitis is more challenging (Shah et al., 2008).

Differential Diagnosis:

- Ampullary carcinoma
- Cholangitis

- Cholecystitis
- Chronic gastritis
- Crohn's disease
- Intestinal perforation
- Mesenteric artery ischemia
- Myocardial infarct
- Pancreatic cancer
- Peptic ulcer disease
- Pneumonia (Ritcher, 2014)

Treatment: Optimal management of chronic pancreatitis includes pain management, improvement of exocrine pancreatic insufficiency leading to malabsorption, and management of any complications (Hobbs et al., 2016; Steven & Conwell, 2010). General measures to help improve pain include smoking cessation; cessation of alcohol use; consumption of small, low-fat meals; and adequate hydration. Alcohol and smoking cessation are essential (Goulden, 2013; Hobbs et al., 2016). Patients with chronic pancreatitis due to an alcohol etiology who avoid alcohol have been shown to have improved disease course (Pitchumoni, 1998; Strum, 1995); those who continue to drink have been shown to have increased physical impairment and mortality (Ammann, Akovbiantz, Largiader, & Schueler, 1984; Steer, Waxman, & Freedman, 1995).

Related to meal supplementation, patients who consumed supplements with medium-chain triglycerides can have improvement in pain, steatorrhea, and weight loss (Shea, Bishop, Parker, Gelrud, & Freedman, 2003). In general, patients who follow these general recommendations early in disease progression tend to require minimal, nonopioid pain medications for pain management. Pancreatic enzyme supplementation has also been shown to have a benefit in pain management in chronic pancreatitis (Singh & Toskes, 2003). Limiting caffeinated beverages and not smoking or consuming alcohol is strongly advised. The pancreas is the key player in the digestion of fat, so if the pancreas cannot properly digest fat, one can have fatty or oily stools (steatorrhea) (Keller & Layer, 2005). Patients should be educated to consume low-fat diets with supplementation of vitamins and antioxidants, specifically vitamin E, riboflavin, choline, magnesium, copper, manganese, and sulfur (Bhardwaj, Thareja, Prakash, & Saraya, 2004; Goulden, 2013).

Pain relief treatment options consist of analgesics coupled with antidepressants; NSAIDs and acetaminophen are first-line agents with long- and short-acting narcotics such as Ultram. Narcotic addiction is a common consequence of treatment and should be closely monitored (Huffman, 2011). In the case of exocrine pancreatic insufficiency, supplementation with enzymes is the recommended therapy and has long been used to treat pain associated with chronic pancreatitis (Hobbs et al., 2016; Shah et al., 2008). However, the effectiveness of pancreatic enzyme supplementation is widely debated, although given its benefit to malabsorption, lack of adverse reactions, and noninvasive nature, it is reasonable to try them because of the potential benefits with minimal risks (Hobbs et al., 2016).

Follow-Up: If alcohol use and/or smoking are among the habits of the patient, these are best treated by a team approach using a chemical dependency counselor, psychologist, or mental health nurse practitioner. An alcohol treatment program may be appropriate. The patient will be seen back for follow-up on symptoms and clinical progression of the disease, with any treatment modifications needed, based on symptoms and presentation.

Sequelae: Acute onset of abdominal pain can also occur with large volume alcohol ingestion. Chronic pancreatitis can also lead to calcification of the pancreas, where the tissue hardens and surgery may be necessary to remove part of the pancreas. Long-standing inflammation in the pancreas caused by chronic pancreatitis is a risk factor for developing pancreatic cancer. Benign cysts, called pseudocysts, are formed of pancreatic fluid and surrounded by a fibrous wall. However, these cysts can also fill with fluid and debris, become infected, and rupture. Biliary obstruction and gastric outlet obstruction may occur because of compression of the bile duct. If the beta cells of the pancreas are destroyed, diabetes may develop, and insulin and diet will be recommended along with frequent blood glucose monitoring (Steven & Conwell, 2010).

Other complications of chronic pancreatitis include development of duodenal obstruction, pancreatic ascites, and pleural effusion from fluid accumulation, pseudo-aneurysms, and splenic vein thrombosis. When medical management no longer relieves pain, when patients are significantly malnourished, or when quality of life is decreased greatly, surgical intervention should be considered. Some studies have suggested that early surgical intervention may prevent disease progression and reduce the need for opioid pain medication, but this remains controversial (Alexakis et al., 2004; Nealon & Thompson, 1993). To date, the most recent Cochrane review of surgical intervention in obstructive chronic pancreatitis found that early surgical intervention seemed beneficial but could not be definitively demonstrated (Ahmed Ali et al., 2015). Finally, patients with chronic pancreatitis are at risk for developing diabetes. Caution is advised when regulating glycemic control in older adults to prevent episodes of hypoglycemia regardless of diabetic etiology (Shah et al., 2008).

Prevention/Prophylaxis: Once a normal diet is resumed (low in fat and high in protein and carbohydrates), the person will be placed on pancreatic enzymes (a minimum of 30,000 units of lipase) for treatment of the steatorrhea and malabsorption. The enzymes are taken with every meal and help digest food and help the person gain weight. Response to enzyme therapy is measured by a 72-hour stool fat quantification. A daily proton pump inhibitor may be added for those with refractory therapy because gastric acid may denature exogenous enzymes. Medium-chain triglycerides (MCTs), a form of dietary fat, are more easily digested and absorbed than the long-chain triglycerides found in most foods. MCTs are available as oil that can be mixed with fruit juice and are a good source of calories for people with chronic pancreatitis who have lost weight and do not respond to dietary changes or pancreatic enzyme supplements (Freedman, 2010). Those persons with a known reason for chronic pancreatitis, other than smoking or alcohol, should have those reasons corrected, if possible.

Referral: Surgical intervention is indicated when there is an anatomical complication correctable by mechanical intervention and includes an abscess, fistula, and fixed obstruction such as a stone, pseudocyst, stenosis, or a varietal

hemorrhage due to splenic vein thrombosis. Pancreatic resection is reserved for those with small duct disease or pain unresponsive to medical management (Steven & Conwell, 2010).

Education: All persons with chronic pancreatitis should have a consultation with a dietitian concerning a diet low in fat. They should also have instruction on how to cook low-fat meals and how to portion out small, frequent meals.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Pancreatic enzyme supplementation is indicated for steatorrhea and malabsorption and may help relieve pain in patients with chronic pancreatitis.	B	Freedman, 2010 Nair et al., 2007 Hobbs et al., 2016
Contrast-enhanced CT scan is the recommended initial imaging study in patients with suspected chronic pancreatitis.	B	Nair et al., 2007 Conwell et al., 2014
MRCP and endoscopic ultrasonography provide similar diagnostic performance as endoscopic retrograde cholangiopancreatography for evaluation of pancreatic parenchyma and the duct system.	C	Nair et al., 2007 Mayerle et al., 2013
ERCP drainage of pseudocysts results in a similar rate of pain relief as surgery, with equivalent or lower mortality.	B	Adler et al., 2005 Makary et al., 2005
Pancreatin (pancreatic enzyme supplementation) should be given to chronic pancreatitis patients with weight loss or abdominal signs/symptoms attributable to malabsorption or maldigestion.	A	Mayerle et al., 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

DIABETES MELLITUS, TYPES 1 AND 2

Signal Symptoms: Anorexia, paresthesias, proteinuria, chronic skin infections, blurred vision, nausea, gastroparesis, yeast vaginitis, impotence, fatigue, weight loss or gain, polydipsia, polyuria, polyphagia.

Description: Diabetes mellitus is a chronic state of hyperglycemia that is divided primarily into two types: type 1 and type 2. Formerly, type 1 diabetes was called *juvenile diabetes* or *insulin-dependent diabetes* and type 2 diabetes was called *adult onset diabetes* or *non–insulin-dependent diabetes,* primarily to signify the age of onset of disease and whether or not insulin therapy was the required treatment. Diabetes in the older adult is a different disease than in the younger adult. Aging is a major risk factor for diabetes because a decline in beta-cell function and blood insulin levels, along with a decrease in physical activity and muscle mass, are common changes that increase the likelihood of impaired glucose tolerance or diabetes. Kalyani and Egan (2013) identify five reasons that older adults have reduced insulin effectiveness: 1) increased abdominal fat, 2) decreased physical activity, 3) sarcopenia, 4) mitochondrial dysfunction, and 5) hormonal changes with lower IGF-1 and DHEA. Furthermore, older adults often do not have the increase in fasting hepatic glucose production that is seen in middle age, resulting in normal or near-normal fasting glucose with elevated postprandial glucose (Hornick & Aron, 2008; Kalyani & Egan, 2013). This combination of normal changes with aging and lack of typical presentation results in the under-diagnosis of diabetes in the older adult.

Etiology: Type 1 diabetes most often occurs in childhood and is characterized by low or absent levels of insulin, often resulting from an autoimmune response leading to pancreatic beta-cell antibodies. Individuals with type 1 diabetes require insulin therapy for survival. Type 2 diabetes usually occurs in adulthood and is thought to be the result of insulin resistance and deficiency. This early understanding of diabetes has been expanded by the discovery of other hormones involved in the regulation of glucose, including insulin, glucagon, amylin, and incretin hormones. These discoveries have resulted in new classes of medications to treat diabetes. These hormones include pancreatic hormones, insulin and amylin secreted by the β cells and glucagon from the α cells, as well as the gut incretin hormones, gastric inhibitory polypeptide (GIP) and glucagon-like peptide-1 (GLP-1). Genetic predisposition for developing diabetes is more evident in type 2 diabetes. Hyperglycemic states that have not reached levels that warrant a type 2 diabetes diagnosis are identified as impaired glucose tolerance. Evidence indicates that individuals with impaired glucose tolerance have an increased risk of developing type 2 diabetes within 5 to 10 years of onset.

Occurrence: According to the Centers for Disease Control and Prevention (CDC, 2015), it is estimated that 30.3 million children and adults in the United States, or approximately 9.4% of the population, have diagnosed diabetes. Type 1 diabetes is only about 5% of that total. Adults older than 18 years have a prevalence of 30.2 million, with 7.2 million of those undiagnosed, a total of 12.2% of all adults. This percent increases with aging so that the prevalence in adults more than 65 years old reaches 25.2%. A 2015 estimate of those with prediabetes included 33.9% of adults more than 18 years old, which is approximately 84.1 million people. The incidence in adults more than 18 years old in 2015 was 1.5 million new cases of diagnosed diabetes.

Diabetes is costly and has high morbidity and mortality rates. The total direct and indirect costs of treating diagnosed diabetics in 2017 was estimated to be $327 billion up from 2012 when it was $245 billion dollars (American Diabetes Association, 2018). This translates to a cost 2.3 times higher than care for those without diabetes. Morbidity includes blindness, end-stage renal disease, and lower extremity amputations. Macrovascular complications from diabetes substantially increase the risk of morbidity and death from coronary artery disease, stroke, and peripheral vascular disease. Complications may result from either type 1 or type 2 diabetes. Evidence indicates that the degree of severity with respect to microvascular, neuropathic, and possibly macrovascular complications seems to be related to the number of years an individual has had hyperglycemia and the magnitude of the glucose elevation. In 2014, 7.2 million hospital discharges listed diabetes as the primary diagnosis in adults more than 18 years old. That same year, a total of 14.2 million emergency department visits listed diabetes as the diagnosis. Diabetes was the seventh leading cause of death in the United States in 2015.

Age: The onset of type 1 diabetes usually occurs in childhood, with the major incidence of disease occurring in individuals less than 30 years old. The onset of type 2 diabetes usually occurs after age 40 years, with a mean age of 51 years. Age cannot be the only factor used to determine the classification of diabetes, however, because type 1 or type 2 diabetes can occur at any age.

Gender: Type 1 diabetes affects men and women equally. Type 2 diabetes affects men more frequently than women.

Ethnicity: Type 1 diabetes primarily affects Caucasians. Native Americans and Alaskan Natives have the highest prevalence of type 2 diabetes for both men and women, with non-Hispanic African Americans and Hispanics next, followed by Caucasians and Asians.

Contributing Factors: Although the exact cause of type 1 diabetes is unknown, it has been linked to environmental, autoimmune, and viral toxins. A predisposition for acquiring type 1 diabetes has not been identified. Contributing factors for the development of type 2 diabetes have strongly been associated with obesity, sedentary lifestyle, and familial history. The following risk factors have been identified as significantly increasing an individual's risk of developing type 2 diabetes: age 45 years and older, a positive family history (parents or siblings with diabetes), hyperlipidemia, a history of gestational diabetes, or women delivering infants weighing 9 pounds or more, obesity (body mass index [BMI] greater than or equal to 25 kg/m^2), smoking, physical inactivity (exercising less than three times a week), high blood pressure (greater than 140/90 mm Hg,), and a history of impaired fasting glucose or impaired glucose tolerance.

An association has been noted between treatment with the higher potency statins, rosuvastatin, atorvastatin, and simvastatin, and new onset of diabetes (Carter, et al., 2013; Laakso & Kuusisto, 2017). High potency statins are particularly diabetogenic, causing insulin insensitivity and decreased insulin secretion. Pravastatin is the least diabetogenic statin. The value of statins' ability to decrease the risk of cardiovascular disease outweighs the risk of developing diabetes, but it does provide another indication for vigilant monitoring for the development of diabetes. The greater number of risk factors present increases the risk of development of the disease.

Signs and Symptoms: Among adults 40 years and older, diabetes often is discovered as an incidental finding during the work-up for cardiovascular, renal, neurological, or infectious diseases. The primary presentation may be due to complications of underlying and undiagnosed hyperglycemia in conditions such as stroke, myocardial infarction and ischemia, intermittent claudication, impotence, peripheral neuropathy, proteinuria, retinopathy, slow wound healing, or fatigue. Often the classic symptoms of polyuria, polydipsia, and polyphagia with weight loss are attributed to other disease entities and overlooked among older adults. The classic triad may be absent entirely due to an impaired thirst mechanism and an increased renal threshold for glucose. Screening is recommended for adults of any age who are overweight and present with any of the additional risk factors (ADA, 2016). Screening should begin for the general population at 45 years old and be repeated at 3-year intervals. More frequent screening is recommended for individuals with several risk factors.

The determination of whether or not the diabetes is type 1 or type 2 is based on ketonuria, age of onset, and BMI. However, more definitive diagnostic tools are available to distinguish between them, including the use of antibody testing and testing C-peptide levels. Antibodies to islet cells and beta cells are present in 70% to 80% of type 1 diabetics and would not be present with type 2 diabetes. C-peptide is a by-product of insulin production, and extremely low levels are consistent with type 1 diabetes. Type 2 diabetes primarily occurs in individuals who are 40 years and older, have no or minimal ketonuria, and have a BMI of more than 27.

Diagnostic Tests: The criterion for the diagnosis of diabetes is either two fasting blood glucose readings with results greater than or equal to 126 mg/dL or a random blood glucose reading greater than or equal to 200 mg/dL if symptoms of diabetes are present. In older adults, the glucose reading after a 2-hour oral glucose tolerance test (OGTT) rises more rapidly than the fasting glucose (Kalyani & Egan, 2013). Therefore, diabetes will be diagnosed sooner with the OGTT than with fasting glucose alone. Hemoglobin A1c (Hb A1c) measurement greater than or equal to 6.5 is indicative of type 2 diabetes (ADA, 2017; Florkowski, 2013). In the absence of unequivocal disease, the same test should be repeated at least once; so, for example, if the Hb A1c is greater than or equal to 6.5, a second Hb A1c result should be compared rather than comparing it to a fasting glucose level (ADA, 2016). An advantage of using the Hb A1c is that fasting is not required and it is less effected by day-to-day issues in the patient, such as illness or stress. An individual with a casual

TABLE 14-1 Comparison of Hb A1c to Average Glucose Level

HB A1C (%)	GLUCOSE (MG/DL)
6.0	126
6.5	140
7.0	154
7.5	169
8.0	183
8.5	197
9.0	212
9.5	226
10.0	240

Source: Adapted from American Diabetes Association. (2010). Insulin basics. Retrieved from http://forecast.diabetes.org/files/images/InsulinChart_4.pdf

plasma glucose level greater than or equal to 200 mg/dL, but without symptoms, should have fasting plasma glucose, 2-hour OGTT, or Hb A1c measured. Fasting is defined as no caloric intake for at least 8 hours. Parameters for the estimated average glucose levels based on Hb A1c levels can be found in Table 14-1.

When hyperglycemia has been established, fasting urine for ketones should be performed to help differentiate between type 1 and type 2 diabetes and the need for insulin therapy. Ordering C-peptide levels and antibodies to islet cells and beta cells, as indicated previously, will help to clarify any uncertainty. It is also important to screen for nephropathy using the urine albumin to creatinine ratio (ADA, 2016; Farmer et al., 2014).

Differential Diagnosis:

- Drug toxicity and endocrine disorders that may affect glucose tolerance, interfere with insulin secretion, and induce insulin resistance.
- Drugs associated with hyperglycemia include alcohol, beta-adrenergic agents, calcium channel blockers, corticosteroids, lithium salts, rifampin, asparaginase, diazoxide, diuretics, glycerol, niacin, phenytoin, and sympathomimetics.
- Drugs associated with hypoglycemia include anabolic steroids, beta-adrenergic blockers, chloroquine, disopyramide, pentamidine, salicylates, warfarin, ethanol, and sulfonamides.
- Endocrine disorders that may induce hyperglycemia include Cushing's syndrome, glucagonoma, acromegaly, and pheochromocytoma.

Treatment: The goal of treatment for patients with type 2 diabetes is glycemic control, good nutritional status with weight management, and exercise. Evidence shows that patients with type 2 diabetes benefit from maintaining near-normal glucose levels. The 2016 ADA Diabetes Standards of Care modified previous recommendations for glucose control, blood pressure goal, screening for nephrology, and treatment with a statin. They recommend individualized treatment plans based on the health and functional ability of patients in three categories based on the presence of chronic illnesses and functional status: healthy, complex/intermediate, and very complex/poor health. For example, fasting values vary from 90 to 130 and an Hb A1c target of less than 7.5% for the healthy older adult to a fasting value of 100 to 180 and Hb A1c target of less than 8.5% for the very complex. Table 14-2 has the details for each group. These new guidelines are based in part on the data from the Analyses from the Action to Control Cardiovascular Risk in Diabetes (ACCORD) study, which concluded that the risk of hypoglycemia and the morbidity resulting from those episodes may outweigh the microvasculature benefits of tighter control.

Aggressive glycemic control that compromises functional independence and quality of life, as well as increasing the risk of hypoglycemic episodes, would be counterproductive. As the options for treatment of diabetes continues to increase, it is more possible to develop patient-centered care plans. Inzucchi and colleagues (2015) prepared an update of their work from 2012 on patient-centered care plans that included more head-to-head trials of antihyperglycemic agents to allow for improved evidence-based practice. Treatment goals and the choice of agent to reach those goals should be made after a careful assessment of the patient's health beliefs, comorbidities, life expectancy, functional status, economic situation, and availability of support services.

Nonpharmacological Treatment: Nonpharmacological therapy is the recommended first-line therapy for newly diagnosed patients with mild-to-moderate hyperglycemia. Lifestyle modifications of weight loss and exercise are particularly important in lowering Hb A1c. Although weight loss in frail elders is not recommended due to the risk of sarcopenia, exercise of even a modest nature can be beneficial in decreasing insulin resistance. If after 3 to 6 months nonpharmacological treatment fails or the hyperglycemia is severe (fasting plasma glucose 200 to 300 mg/dL or casual plasma glucose 250 to 350 mg/dL), oral agents may be added to the treatment regimen. Metformin is also recommended for prevention or delay of type 2 diabetes in patients with a BMI greater than or equal to 35 kg/m^2 or anyone with a history of gestational diabetes or patients with a rising Hb A1c in spite of lifestyle modifications (ADA, 2016).

Pharmacological Treatment: Pharmacological treatment options for diabetes include oral agents and injectables. There are now seven classes of oral glucose-lowering agents: biguanides, sulfonylureas, meglitinides, thiazolidinediones, dipeptidyl peptidase-4 (DPP-4) inhibitors, alpha-glucosidase inhibitors, and the newest agent, sodium-glucose cotransporter 2 (SGLT2) inhibitors. Other oral agents that may be used in the management of type 2 diabetes include the bile acid sequestrant and the dopamine-2 agonist. Injectables include insulin and the glucagon-like peptide–1 (GLP-1) receptor agonists.

Oral Agents: Metformin (Glucophage, Glucophage XR) is a biguanide that acts by decreasing hepatic glucose production, decreasing glucose intestinal absorption, and increasing insulin sensitivity. It is now considered first-line therapy in treating type 2 diabetes and may be initiated with lifestyle changes in many individuals (ADA, 2016). This agent generally does not increase endogenous insulin production and has few hypoglycemic effects. In addition to glucose control with improved Hb A1c of 1% to 2%, patients may lose weight and

TABLE 14-2 Setting Treatment Goals for Older Adults

PATIENT CHARACTERISTICS/ HEALTH STATUS	RATIONALE	REASONABLE A1C GOAL‡	FASTING OR PREPRANDIAL GLUCOSE	BEDTIME GLUCOSE	BLOOD PRESSURE	LIPIDS
Healthy (few coexisting chronic illnesses, intact cognitive and functional status)	Longer remaining life expectancy	<7.5% (58 mmol/mol)	90–130 mg/dL (5.0–7.2 mmol/L)	90–150 mg/dL (5.0–8.3 mmol/L)	<140/90 mmHg	Statin unless contraindicated or not tolerated
Complex/intermediate (multiple coexisting chronic illnesses* or 2+ instrumental ADL impairments or mild-to-moderate cognitive impairment)	Intermediate remaining life expectancy, high treatment burden, hypoglycemia vulnerability, fall risk	<8.0% (64 mmol/mol)	90–150 mg/dL (5.0–8.3 mmol/L)	100–180 mg/dL (5.6–10.0 mmol/L)	<140/90 mmHg	Statin unless contraindicated or not tolerated
Very complex/poor health (LTC or end-stage chronic illnesses** or moderate-to-severe cognitive impairment or 2+ ADL dependencies)	Limited remaining life expectancy makes benefit uncertain	<8.5%† (69 mmol/mol)	100–180 mg/dL (5.6–10.0 mmol/L)	110–200 mg/dL (6.1–11.1 mmol/L)	<150/90 mmHg	Consider likelihood of benefit with statin (secondary prevention more so than primary)

This represents a consensus framework for considering treatment goals for glycemia, blood pressure, and dyslipidemia in older adults with diabetes. The patient characteristic categories are general concepts. Not every patient will clearly fall into a particular category. Consideration of patient and caregiver preferences is an important aspect of treatment individualization. Additionally, a patient's health status and preferences may change over time.
ADL, activities of daily living.
‡ A lower A_{1c} goal may be set for an individual if achievable without recurrent or severe hypoglycemia or undue treatment burden.
* Coexisting chronic illnesses are conditions serious enough to require medications or lifestyle management and may include arthritis, cancer, congestive heart failure, depression, emphysema, falls, hypertension, incontinence, stage 3 or worse chronic kidney disease, myocardial infarction, and stroke. By "multiple," we mean at least three, but many patients may have five or more (27).
** The presence of a single end-stage chronic illness, such as stage 3–4 congestive heart failure or oxygen-dependent lung disease, chronic kidney disease requiring dialysis, or uncontrolled metastatic cancer, may cause significant symptoms or impairment of functional status and significantly reduce life expectancy.
† A_{1C} of 8.5% (69 mmol/mol) equates to an estimated average glucose of ~200 mg/dL (11.1 mmol/L). Looser A_{1c} targets above 8.5% (69 mmol/mol) are not recommended as they may expose patients to more frequent higher glucose values and the acute risks from glycosuria, dehydration, hyperglycemic hyperosmolar syndrome, and poor wound healing.

improve lipid profiles. Side effects include nausea, vomiting, diarrhea, abdominal pain, anorexia, and taste disturbances.

A rare but potentially life-threatening reaction is the development of lactic acidosis. Metformin is contraindicated in patients with renal insufficiency because the risk of lactic acidosis is increased in these patients and, while uncommon, has a very high mortality rate. The estimated glomerular filtration rate (eGFR) at which metformin was contraindicated had been less than 60, but recent recommendations have lowered that to an eGFR of less than 45, or in some cases, less than 30 with dose adjustment. In hospitalized patients at risk for acute or chronic renal insufficiency, consider holding metformin. Other contraindications include hepatic insufficiency, congestive heart failure, and alcohol abuse. Data presented by Galindo at the 2014 meeting of the American Geriatrics Society indicated the use of metformin in the very old (average age 87, n=400) was safe, a finding that contradicts common advice to avoid metformin use in patients 80 years and older. Current recommendations advise not to initiate metformin in adults more than 80 years old without normal renal function.

Care should be taken when patients who are taking metformin undergo IV contrast studies. It is important to withhold the medication for 1 to 2 days after the contrast agent has been administered to ensure normal renal function. Usually, metformin is started at 500 mg once a day, given before the evening meal, and increased gradually to a maximum dose of 2,000 mg daily in the older adult. Gastrointestinal (GI) side effects can result in patient intolerance and are minimized with this slow up-titration. When given with food, the GI side effects tend to be decreased and absorption is increased. An extended-release formulation is available and may be very useful for increasing adherence to therapy. When switching from regular release to extended release metformin, the total daily dose should remain the same. For some patients on 2,000 mg of extended release a day, dividing the dose into two 1,000 mg tablets may be more effective.

Sulfonylureas are insulin secretagogues and act by increasing pancreatic insulin secretion. They tend to lower Hb A1c by up to 1.25% (Cornell, 2014). The second-generation sulfonylureas include glipizide (Glucotrol, Glucotrol XL), glimepiride (Amaryl), and glyburide (DiaBeta, Micronase, Glynase). Glyburide is on the *Beers List: Potentially Inappropriate Drugs for Elderly* and, along with the first-generation agents (chlorpropamide, tolazamide, and tolbutamide), should be avoided and continued only in patients who are already adequately controlled on those medications and not experiencing side effects. Because sulfonylureas are renally cleared and renal function declines with aging, an increase of the level of sulfonylurea in the blood may result with an increase in the risk

of hypoglycemia. If sulfonylureas are used, choose ones with the shortest half-life (e.g., glimepiride or glipizide). Side effects of this class of medications include weight gain, nausea, hypoglycemia, weakness, and photosensitivity. These agents should not be used in patients who have had diabetic ketoacidosis and caution should be exercised in patients who are allergic to sulfonamides. The starting dose depends on the particular drug, but should be initiated at the lowest possible dose and increased as tolerated.

Meglitinides are another class of insulin secretagogues and include nateglinide (Starlix) and repaglinide (Prandin). They act quickly to increase the endogenous release of insulin from the pancreas and must be taken 1 to 30 minutes before a meal, typically three times a day. They have less effect on Hb A1c than sulfonylureas, with about a 1% reduction (Cornell, 2014). Though frequent dosing may be seen as a drawback, it can be an advantage in patients with irregular eating habits when the medications are not taken if a meal is skipped. Side effects include hypoglycemia, headache, upper respiratory infection, nausea, vomiting, constipation, diarrhea, muscle aches, and chest pain. Repaglinide is metabolized by the liver, with less than 10% renally excreted, and does not need dose adjustment in renal insufficiency. It is also somewhat better in lowering Hb A1c than nateglinide, so repaglinide can be initial therapy in patients who are not candidates for metformin or sulfonylureas. All insulin secretagogues should be used with caution in those with hepatic insufficiency.

Thiazolidinediones (TZDs), which include pioglitazone (Actos) and rosiglitazone (Avandia), are insulin sensitizers. They act by increasing sensitivity to insulin by acting on adipose, muscle, and liver to increase glucose utilization and decrease hepatic glucose production. TZDs have a favorable effect on lipids and can improve β cell function. They reduce Hb A1c by 1% to 1.5% (Cornell, 2014). Side effects include headache, weight gain, edema, and anemia. TZDs should not be used in patients with heart failure, bladder cancer, osteoporosis, or liver disease. Serious reactions include liver toxicity and worsening congestive heart failure. Liver function tests should be performed prior to initiation and periodically after that. TZDs have a very slow onset of action. Initial effects are noted in 2 weeks, but as monotherapy the maximum effect is not seen for 10 to 16 weeks.

Dipeptidyl peptidase-4 (DPP-4) inhibitors include sitagliptin (Januvia), linigliptin (Tradjenta), saxagliptin (Onglyza), and alogliptin (Nesina). All are once-daily oral medications that help the body reduce its own blood sugar levels. They have a unique action by inhibiting the enzyme DPP-4, which breaks down glucagon-like peptide–1 (GLP-1), thereby increasing the bioavailability of GLP-1. GLP-1 is secreted by the small intestinal L cell, and it is a potent anti-hyperglycemic hormone that works by glucose-dependent insulin stimulation (so the risk of hypoglycemia is low). It also suppresses inappropriate glucagon secretion, slows gastric emptying, and reduces food intake. DPP-4 inhibitors also improve β cell function. Hb A1c reduction is comparable to that of sulfonylureas and TZDs (Cornell, 2014). When used as monotherapy, DPP-4 inhibitors have a low risk of hypoglycemia, but may increase the risk of hypoglycemia when used with sulfonylureas. Side effects of DPP-4 inhibitors include upper respiratory tract infection, nasopharyngitis, and headache. Dose reduction in patients with moderate to severe renal insufficiency is required for all DPP-4 inhibitors except linigliptin, and they should not be used in patients with a past history of pancreatitis.

Alpha-glucosidase inhibitors, acarbose (Precose) and miglitol (Glyset), act to decrease or delay glucose absorption and lower blood glucose levels. Serum triglycerides tend to be lowered also, and this may be their greatest benefit. Side effects include flatulence, diarrhea, abdominal pain, and rash. Both are dosed three times a day and should be taken with the first bite of each meal. α-glucosidases are contraindicated in patients who have had ketoacidosis, inflammatory bowel disease, intestinal obstruction, or renal impairment. The primary side effect of this medication is flatulence and patients' lack of tolerance to this side effect results in this class rarely being used. There is only a minor reduction in Hb A1c, 0.8%, which further contributes to their minor clinical usefulness.

The newest addition to the oral options are the sodium-glucose cotransporter 2 (SGLT2) inhibitors, dapagliflozin (Forxiga), canagliflozin (Invokana), and empagliflozin (Jardiance), and ertugliflozin (Steglatro). They block glucose reabsorption in the proximal renal tubule by inhibiting the action of SGLT2, which reduces glucose reabsorption and increases urinary glucose excretion. The action of SGLT2 inhibitors is not dependent on insulin. They reduce Hb A1c by 0.5% to 1% (ADA, 2016). Specific to the brand, Invokana (canagliflozin), is a black box warning regarding a 2-fold increase of leg and foot amputation in those patients with type 2 diabetes and established cardiovascular disease. Risk factors include prior amputation, history of peripheral vascular disease, neuropathy, and history of diabetic foot ulcers. Recommendation is to monitor patients at risk for infection, new pain or tenderness, sores or ulcers on lower extremities and discontinue if complications occur (FDA, 2018).

The choice of an appropriate oral agent is a complex clinical decision. Consider the severity of the hyperglycemia and the need for Hb A1c reduction. Most guidelines agree that a biguanide (metformin) should be the first-line agent. If there are contraindications to the use of metformin, a sulfonylurea could be an alternative choice, keeping in mind that it carries the risk of hypoglycemia and often results in weight gain. DPP-4 inhibitors are another possibility for initial treatment with a similar effect on Hb A1c to that of sulfonylureas without the risk of hypoglycemia, but they are expensive. The newest agents, SGLT2 inhibitors, are also approved for monotherapy, but are also expensive and have less effect on Hb A1c. If monotherapy with any of these agents is unsuccessful after 3 months, combination therapy with metformin, sulfonylurea, thiazolidinedione, DPP-4 inhibitors, SGLT2 inhibitors, GLP-1 receptor agonists, or basal insulin should be considered. If irregular meals are an issue, adding a meglitinide to either metformin or a DPP-4 inhibitor may be a reasonable option. The injectable glucagon-like peptide–1 (GLP-1) receptor agonists are non-insulin options for combination therapy and have a side benefit of weight loss. Insulin therapy is a reasonable choice at any point in the patient diagnosed with type 2 diabetes, but it is usually reserved as second-line therapy when combination therapy fails. Insulin therapy may be required either as a single agent or in addition to a sulfonylurea, metformin, DPP-4 inhibitor, or GLP-1 agonist.

Injectables: Glucagon-like peptide–1 (GLP-1) receptor agonists are within the drug class called incretins. Exenatide

(Byetta), liraglutide (Victoza), lixisenatide (Lyxumia), albiglutide (Tanzeum), dulaglutide (Trulicity), and semaglutide (Ozempic) are approved by the FDA for patients with type 2 diabetes who have not achieved adequate blood glucose control while taking metformin or a combination of oral agents. Unlike endogenous GLP-1, these medications are resistant to degradation by the DPP-4 enzyme and have a longer half-life than endogenous GLP-1. These medications are effective in reducing glucose levels, as well as inducing weight loss with Hb A1c reduction of 0.5% to 1.5% and weight loss of 2% to 5%. They may also be prescribed for patients with impaired fasting glucose to delay the onset of type 2 diabetes. Side effects include nausea and a feeling of fullness that may dissipate after a few weeks of treatment. Other common side effects include hypoglycemia when used with sulfonylureas or insulin, vomiting, diarrhea, headache, nervousness, and stomach discomfort. Patients may also experience decreased appetite, acid reflux, and increased sweating. GLP-1 agonists are contraindicated for use in type 1 diabetes and patients with a history of diabetic ketoacidosis. A black box warning exists for patients with medullary thyroid carcinoma or a family history of that disease or with multiple endocrine neoplasia syndrome type 2 (MEN2). GLP-1 agonists should be used with caution in patients with a history of pancreatitis or gastroparesis. In choosing a GLP-1 agonist it is important to consider ease of administration, dosing frequency, side-effect profile, and patient preference. All of these drugs are expensive, but the benefits may outweigh the cost.

Insulin Therapy

After many years of having type 2 diabetes, almost all patients will require insulin therapy due to the decline of beta cells in the pancreas and endogenous insulin supply. The body's normal production of insulin is biphasic, with about one-half slowly released around the clock to suppress glucagon release from the liver between meals and while we sleep, and one-half released in response to a rise in blood sugar after food intake. Insulins are the most natural agents and are always effective in lowering blood sugar. They all work alike; the difference is in the onset and duration of action. Insulin therapy may be added when optimal glycemic control is not achieved with an oral regimen or the addition of a GLP-1 agonist. Recent guidelines from the ADA (2016) suggest initiating insulin earlier in the treatment plan.

Every type 2 diabetic needs to understand that the longer they live with diabetes the more likely it is that they will need to use insulin. The ADA Standards of Medical Care in Diabetes 2016 emphasize the importance of introducing the role of insulin from the beginning to avoid the implication that insulin is punishment for poor diabetic control. Insulin should always be used as first-line therapy in type 1 diabetes or with patients who have had diabetic ketoacidosis. A variety of regimens are used in the administration of insulin based on the time of onset of action, time of peak effects, and the duration of action. Currently, the most frequently used regimen is the basal/bolus insulin concept. This reflects the body's physiological insulin response. Basal insulins have an intermediate to long duration of action, while bolus insulins, immediate or rapid in their action, provide postprandial coverage.

Basal Insulins: Basal insulins suppress glucose production between meals and overnight, and provide nearly constant levels of insulin. Basal insulin includes the intermediate-acting insulin, neutral protein Hagedorn (NPH); long-acting insulins, glargine (Lantus, Toujeo, Basaglar) and insulin detemir (Levemir); and ultra-long-acting insulin, degludec (Tresiba). Head-to-head studies comparing insulin detemir and glargine to NPH found a higher incidence of hypoglycemia and increased weight gain with the NPH (Hamaty, 2011). Administration of insulin glargine and detemir in the morning had even less incidence of nighttime hypoglycemia. Whereas NPH insulin has a distinct peak of action at 4 to 12 hours, insulin glargine and detemir are considered to have no peak. NPH and insulin detemir both may be dosed twice a day.

Bolus Insulins: Mealtime bolus insulins include regular human insulin and the analogues aspart (NovoLog), glulisine (Apidra), or lispro (Humalog). Regular insulin is considered fast acting, with an onset and duration of action that exceeds that of the analogues. It is given 30 minutes before eating and has a peak in 2 to 4 hours. The analogues are considered immediate acting, with an onset within 15 minutes and a peak in 1 to 2 hours. The analogues should not be given until the person is eating and may be given immediately after a meal if the injection was forgotten. Bolus insulins have the advantage of being adaptable to the day. Less can be given if the person is exercising after a meal, and they may not be given at all if a meal will be missed. It is important to understand how to adjust the dose of bolus insulin. It is not sliding scale adjusted in response to the fasting glucose, but rather it should be adjusted based on the preprandial glucose of the next meal (so the dose given at breakfast would be changed based on the lunchtime preprandial glucose).

Premixed insulin combinations of long- and short-acting insulins are available and offer less control but more convenience than the basal/bolus concept. A classic mixture has been the combination of NPH and regular insulin 70/30, with the NPH being 70% of that mixture. By adding protamine to insulin aspart or insulin lispro the release of those insulins is delayed. Humalog mix is the delayed-acting insulin lispro protamine and the immediate-acting lispro and is available as 50/50 or 75/25. The protamine lispro is 75% of that mixture. Novolog mix 70/30 is the delayed-acting insulin aspart protamine with the immediate-acting aspart. The insulin protamine aspart is the 70% of that solution. These mixtures are often dosed twice a day before breakfast and again before dinner.

Although the ability to control mealtime spikes in blood glucose is decreased, the fact that fewer injections are required may be more agreeable to the patient. The participation of the patient in decision making is an essential part of individualized care. To further improve convenience of administration, insulin pens are available for all of these mixtures. Insulin pens are not self-explanatory, however, and taking the time to demonstrate their use with the patient returning the demonstration is essential for accurate insulin administration. Insulin pumps are available and most often used for type 1 diabetics with documentation of very low C-peptide levels often required for coverage by insurance companies. The types of insulin, their onset, peak, and the duration of action and how the insulin is normally used are delineated in Table 14-3.

The frequency and timing of self-monitoring of blood glucose levels (SMBGs) need to be individualized based on

TABLE 14-3 Insulin Types—Onset, Peak, Duration of Action, and Typical Use

TYPE OF INSULIN (INCLUDES BRAND NAMES)	ONSET OF ACTION	PEAK ACTION	DURATION OF ACTION	ROLE OF INSULIN IN MEAL PLANS
Rapid Acting				
Lispro (Humalog)	15–30 min	30–90 min	3–5 hr	Take at same time as meal along with longer-acting insulin.
Aspart (Novolog)	10–20 min	40–50 min	3–5 hr	
Glulisine (Apidra)	20–30 min	30–90 min	1–2.5 hr	
Short Acting				
Regular-Humulin or Novolin	30 min–1 hr	2–5 hr	5–8 hr	Take within 30–60 min of eating along with longer-acting insulin.
Vesosulin (pump)		2–3 hr	2–3 hr	
Intermediate Acting				
NPH (N)	1–2 hr	4–12 hr	18–24 hr	Covers insulin needs for one-half day or overnight. Often combined with rapid- or short-acting insulin.
Long Acting				
Glargine (Lantus, Toujeo)	1–1.5 hr	No peak	20–24 hr	Covers up to 1 full day. May be combined with short- or rapid-acting insulin.
detemir (Levemir)	1–2 hr	6–8 hr	Up to 24 hr	
Ultra Long Acting				
Degludec (Tresiba)				Greater than 24-hr coverage but dosed daily and may be combined with short or rapid acting insulin.
Premixed				
Humulin 70/30	30 min	2–4 hr	16–24 hr	Usually taken twice a day before meals. Premixed are combination intermediate- and short-acting insulin. The numbers indicate how much of each type of insulin.
Novolin 70/30	30 min	2–12 hr	Up to 24 hr	
Novolog 70/30	10–20 min	1–4 hr	Up to 24 hr	
Humulin 50/50	30 min	2–5 hr	18–24 hr	
Humalog mix 75/25	15 min	0.5–2.5 hr	16–20 hr	

Source: Adapted from American Diabetes Association. (2010). Insulin basics. Retrieved from http://forecast.diabetes.org/files/images/InsulinChart_4.pdf

several factors, including age, ability to adhere, and oral versus insulin therapies. SMBG results in promoting glycemic control and reinforcing adherence to therapy. Even patients maintaining glucose levels by nonpharmacological means may benefit from intermittent either postprandial or fasting SMBG. Patients requiring oral medications who are capable of SMBG should test glucose levels two to three times per week, preferably alternating before breakfast, the evening meal, and at bedtime, as well as an occasional 2-hour postprandial measurement. The postprandial measurement is particularly important for older adults who may not have increased hepatic glucose production when in the fasting state. Patients receiving insulin therapy should perform SMBG at least two to three times daily. For all diabetics, SMBG monitoring should increase during illness, changes in diet and exercise, or a change in medications.

Follow-Up: Patients with diabetes should be scheduled for appropriate follow-up to evaluate response, tolerability to therapy, goal reassessment, and management of acute and chronic complications. Response to treatment is judged based on home glucose monitoring results and Hb A1c. The Hb A1c reflects the average control over the past 3 months and is not useful for judging the response to recent treatment adjustments. Older patients and patients on insulin therapy may need to be seen every 3 months. When there is a sudden change in health status or treatment regimen, follow-up of 1 month or less may be indicated. For stable patients who are able to maintain treatment goals, follow-up may be every 3 to 6 months.

The following evaluations are recommended at follow-up visits: blood pressure, review records of home glucose monitoring, review of medications, examination of the feet for complications, annual eye examination including dilation, routine urinalysis including evaluation for albuminuria and albumin to creatinine ratio, serum creatinine levels and eGFR, annual electrocardiogram and fasting lipid profile, Hb A1c every 3 to 6 months, evaluation for neurovascular complications, self-management education, immunization update including influenza and pneumococcal vaccines, and a biannual oral examination. A special nutritional benefit is available for diabetics from Medicare and should be taken advantage of yearly. Medicare also covers the yearly ophthalmological examination for diabetics.

Sequelae: Acute complications requiring immediate attention include diabetic ketoacidosis, recurring fasting hyperglycemia of greater than 300 mg/dL, Hb A1c of greater than 13%, or severe hypoglycemia with changes in sensorium, altered behavior, seizures, or coma. Complications resulting from

prolonged hyperglycemia include renal failure, blindness, coronary artery disease, stroke, peripheral vascular disease, slow-healing wounds, autonomic neuropathies, hypertension, sexual problems, and genitourinary system disorders. Macrovascular complications from diabetes substantially increase the risk of morbidity and death from coronary artery disease, stroke, and peripheral vascular disease. Complications may result from either type 1 or type 2 diabetes. Evidence indicates that the degree of severity with respect to microvascular, neuropathic, and possibly macrovascular complications seems to be related to the number of years a patient has had hyperglycemia and the magnitude of the glucose elevation.

Prevention/Prophylaxis: The focus of prevention for developing type 2 diabetes is diet modification and exercise with subsequent weight loss. In obese adults with diabetes even a modest weight loss of 5% can improve glycemic control (ADA, 2016). When a patient is diagnosed with type 2 diabetes, the maintenance of glucose levels at as near-normal levels as possible is extremely beneficial in preventing complications from the disease. Monitoring and controlling serum lipid levels and blood pressure can greatly reduce complications from coronary artery disease and stroke. The ADA (2016) recommends considering 75 to 162 mg per day aspirin therapy in men 50 years old or older and women 60 years old or older, or in persons with one other risk factor such as hypertension, hyperlipidemia, or others at increased risk for cardiovascular disease. Equally important is maintaining routine examinations recommended by the primary care provider.

Referral: Patients with type 2 diabetes should be referred when acute complications requiring hospitalization develop, such as ketoacidosis, severe hyperglycemia, or hypoglycemia. Patients with fasting glucose levels that are consistently greater than 300 mg/dL or Hb A1c of greater than 13% also should be referred to an endocrinologist for further assessment. Annual eye and oral examinations should be referred to appropriate providers. Patients with uncontrolled hypertension, hyperlipidemia, unmanageable skin disorders, or renal insufficiency should be referred to appropriate providers. Consider referring all patients interested in greater control of self-management of the disease to diabetes educators.

Education: The cornerstone of education for patients with diabetes is disease self-management. Assess the patient's knowledge and understanding of the disease, treatment goals, management, and response to complications. Include in the assessment barriers to treatment, which include cultural influences, health beliefs and behaviors, socioeconomic status, psychological factors, and education and skills deficits that may preclude the ability to self-manage diabetes adequately. Explicit instructions should be given to patients who participate in daily SMBG monitoring. For optimal results, families of patients with diabetes should always be included in the education and management of the disease. Information and helpful tools that may be printed pertaining to diabetes can be obtained from the American Diabetes Association, 2451 Crystal Drive, Suite 900, Arlington, VA 22202, 1-800-DIABETES (1-800-342-2383), www.diabetes.org.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Diagnosis of type 2 diabetes mellitus: two fasting blood glucoses ≥126 mg/dL or two random blood glucoses ≥200 mg/dL.	C	ADA, 2016
In a systematic review of the evidence, support is given to the use of Hb A1c as a measure for screening. The ADA endorsed Hb A1c as a diagnostic test for diabetes at a cut-off of ≥6.5% with the provision that this be measured in a laboratory using a NGSP-certified assay aligned to the DCCT study, and that in the absence of unequivocal hyperglycemia the test should be repeated. It is important to understand that values <6.5 does not exclude the diagnosis. Using this criteria, one-third of patients will be missed that would have been diagnosed with the use of a fasting glucose of ≥126 mg/dL.	A	Florkowski, 2013 ADA, 2016
The 2-hour plasma glucose during an OGTT rises much more steeply than fasting glucose levels with aging.	A	Kalyani & Egan, 2013
For some patients, Hb A1c may not accurately reflect control. Other options include fructosamine and glycated albumin.	C	Danese et al., 2015
Counsel patients regarding lifestyle modification (weight loss, exercise) (expected decrease in Hb A1c 1%–2%).	C	ADA, 2016 Inzucchi et al., 2015 Cornell, 2014

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

FAILURE TO THRIVE

Signal Symptoms: The concept of Failure to Thrive in the elderly evolved from the syndrome long recognized in pediatrics. It began to be applied to older adults in the late 1980's and included symptoms of weight loss, decreased appetite, poor nutrition, dehydration, physical inactivity and functional decline, depression, impaired immune function, low cholesterol (Palmer, 1990, IOM, 1991, Osato, Stone, Phillips, Winne, 1993).

Description: Adult failure to thrive (FTT) is not considered a normal part of aging. The patient undergoes a process of functional decline, progressive apathy, and a loss of willingness to eat or drink that culminates in death. FTT is associated with "increased infections, diminished cell-mediated immunity, hip fractures, decubitus ulcers, and increased surgical mortality" (Robertson and Montagnini, 2004 p.343). This population of gravely ill and impaired older adult patients has experienced multiple hospitalizations, has multiple diagnoses, and can be viewed as a paradigm of the very sick and frail elders (Rossi et al., 2016; Luger et al., 2016). It can be concluded that patients given a diagnosis of FTT present with a complex picture of multifaceted problems across a spectrum of physiological ailments, psychological deficits, and social and environmental needs (Kumeliauskas, Fruetel, Holroyd-Leduc, 2013). Early assessment of the older adult patient is necessary to improve functioning or prevent further decline. The goal of early assessment is to identify the precise needs of the patient in determining appropriate intervention strategies, providing necessary supports, and optimizing rehabilitation (Luger et al., 2016B; Donini et al., 2016).

Etiology: FTT is associated with medical conditions that include cancer, heart failure, chronic obstructive pulmonary disease, chronic renal insufficiency, chronic steroid use, cirrhosis, stroke, depression, diabetes, hepatitis, hip or large bone fractures, inflammatory bowel disease, GI surgery, myocardial infarction, recurrent urinary tract infections, recurrent pneumonia, rheumatoid arthritis, lupus, systemic infections, and tuberculosis (Ali & Chawla, 2015).

Medications associated with FTT include anticholinergics, anti-epileptics, benzodiazepines, beta blockers, central alpha antagonists, diuretics in high-potency combinations, glucocorticoids, neuroleptics, opioids, serotonin reuptake inhibitors, tricyclic antidepressants, and any combination of more than four medications (Robertson & Montagnini, 2004).

Associated issues can be alcohol and substance abuse, cognitive impairment, functional impairment, social isolation, and nutritional problems. Nutritional marasmus decreases body fat stores indicated by a 75% fat loss and a 25% lean tissue loss. General catabolic responses are indicated by a 75% lean tissue loss and a 25% fat loss (Donini et al., 2016).

Occurrence: Occurs in 5% to 35% of community-dwelling elderly and 25% to 40% of nursing home residents (Robertson & Montagnini, 2004).

Age: Incidence increases with age (Kumeliauskas, Fruetel, Holroyd-Leduc, 2013).

Gender: Occurs equally in males and females.

Ethnicity: Occurs in all ethnic groups.

Contributing Factors: Unintentional weight loss, decreased appetite, poor nutrition, inactivity, dehydration, depression, impaired function, and cognitive impairment have been shown to contribute to the development of FTT in older adults. Patients with both acute conditions (infections, electrolyte and metabolic disorders) and exacerbations of chronic (cardiac or respiratory) and terminal (cancers and neurodegenerative conditions) diseases can develop the clinical characteristics of FTT (Kumeliauskas, Fruetel, Holroyd-Leduc, 2013; Rao et al., 2016).

Signs and Symptoms: Fever, orthostatic changes, weight loss, BMI less than 22, evidence of neglect, inability to transfer independently, bed bound, temporal wasting, dry mucosa, cheilosis, glossitis, dysphagia, palpable lymph nodes, loss of subcutaneous fat, dehydration, venous distention, osteoporosis, decreased breath sounds, signs of heart failure, organomegaly, ascites, abdominal masses, muscle wasting, peripheral edema, sacral edema, joint inflammation, focal neurological symptoms, depression, and decreased mental status (Rossi et al., 2016) Diminished scores on the Nutrition Risk Screening Tool and Mini Nutritional Assessment (MNA) can also point to FTT (Donini et al., 2016).

Diagnostic Tests: Consider ordering a CBC, serum albumin level and thyroid-stimulating hormone (TSH), cholesterol, and comprehensive metabolic panel initially in patients presenting with signs of malnutrition over time. For patients with suspected infection or inflammation associated with malnutrition consider blood cultures; chest x-ray; CT scan or MRI if injury, malignancy, or infection is suspected; sedimentation rate or C-reactive protein; growth hormone for patients with suspected endocrine disorder (HIV); rapid plasma reagin (RPR); purified protein derivative (PPD); or urinalysis for specific infection (Bharadwaj, Ginoya, Tandon, Gohel, Guirguis, Vallabh . . . , Hanouneh, 2016). Assess function using the Timed Up and Go Test (Rossi et al., 2016). Conduct a nutritional assessment with the MNA (score less than or equal to 17 indicates malnutrition). Depression screening with the Geriatric Depression Scale should be included, with a score of 5 to 9 suggestive of depression and greater than or equal to 10 significant for depression. St. Louis University Mental Status Exam should be administered if you suspect cognitive decline; an obtained score of 1 to 20 is indicative of dementia (Ali & Chawla, 2015). Calculate either a Karnofsky or Palliative Performance Scale, a value less than or equal to 40% is used in the consideration of the need for hospice care. (Brandon, 2014).

Differential Diagnosis: The differential diagnosis for FTT includes environmental and socioeconomic burdens, as well as underlying or concomitant psychological and debilitating conditions that can lead to frailty if left undetected. One way to remember this is to use the mnemonic introduced by Dr. Anne Egbert in 1993, DWINDLES:

- Diseases
- Dementia
- Delirium

- Drinking alcohol
- Drugs
- Dysphagia
- Deafness, sensory deficits
- Depression
- Desertion by family, friends
- Destitution
- Despair (Egbert, 1993)

Underlying medical conditions such as malabsorption syndrome, endocrine deficiency, anemia, infections (urinary tract, pneumonia, cellulitis), and COPD must be treated to prevent FTT (Ali & Chawla, 2015).

Treatment: Treat underlying medical conditions. Review medication list to determine if any drugs contribute to anorexia, delayed gastric emptying, and altered taste perception (Evans, 2005). With positive scores on depression scale, consider antidepressants (with underlying benefit of increase in appetite), psychotherapy, and environmental modification. For patients with malnutrition, evaluate the need for a speech therapy assessment if dysphagia is present. Treat oral pathologies with regular and/or specialized oral hygiene measures as needed. Review dietary restrictions and adjust as needed. Increase frequency of feedings with smaller meals and add nutritional supplements. For patients with cognitive impairment, optimize living conditions, treat infections, and administer anticholinesterase inhibitors. For patients with functional impairment, refer to physical and occupational therapy, modify environment, and provide assistive devices. Optimize chronic disease management and manage medications. Address advance directives and contingency planning (Sera, Holmes, & McPherson, 2016).

General recommendations for calorie needs: healthy maintenance for women is 25 to 30 kcal/kg/day and for men 30 to 40 kcal/kg/day. Patients who are under physiological stress with pressure ulcers should obtain 30 to 35 kcal/kg/day. For obese and critically ill patients, consider 21 kcal/kg/day. Protein recommendations: 1 to 1.2 g/kg in healthy older adults, 1.25 to 1.5 g/kg/day for those under stress and with pressure ulcers, and 0.8 g/kg/day for those with renal failure (Donini, 2016). The use of Megace and Marinol to stimulate appetite has not shown any significant effect in treating FTT. High-resistance exercise training counteracts muscle weakness and physical frailty (Sera et al., 2016; Luger et al., 2016A).

Follow-Up: The multidimensional causes of FTT in the elderly speak to the importance of coordinated efforts by all healthcare professionals in the care of elderly patients. Interdisciplinary assessments of the complex etiology related to age, disease, and reversibility of the syndrome provide the data needed to establish baselines and needs for follow-up (Ali & Chawla, 2015).

Sequelae: Increased infections, decreased immunity, fractures, decubitus ulcers, muscle wasting, malnutrition, and increased mortality.

Prevention/Prophylaxis: Perform early assessment of functional and cognitive decline in patients at risk. Closely manage chronic illness and medications. Avoid restrictive diets in patients with unintentional weight loss.

Referral: Further work-up of potential causes or for treatment of underlying conditions is warranted. Consider referral to hospice for patients with a BMI below 22 kg/m^2, the patient's Karnofsky or Palliative Performance Scale is less than or equal to 40% and the patient with family refusing a feeding tube or IV nutritional support or who has not responded to nutritional support despite adequate caloric intake (Sera et al., 2016). Failure to thrive however cannot be listed as the principal diagnosis for hospice consideration. (Centers for Medicare and Medicaid Services. Hospice Manual Update for Diagnosis Reporting and Filing Hospice Notice of Election (NOE) and Termination or Revocation of Election, 2018).

Education: Education should include early signs and symptoms of decline, appropriate muscle strengthening exercises, environmental modifications to enhance socialization and stimulate appetite, end-of-life decisions, and caregiver support (Rao et al., 2016; Luger et al., 2016).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCE
High-resistance exercise programs have been shown to positively affect both ADLs and instrumental ADLs of frail community-dwelling older adults.	A	Luger et al., 2016A
Consider referral to Hospice if a patient with a BMI < 22 kg/m^2 is not responding to nutritional support despite adequate caloric intake or refuses any nutritional support.	C	Sera, Holmes, & McPherson, 2016
Patients' nutritional health after hospitalization is often affected by functional status, evidence of depression, increased use of medication, decreased cognition, and poor oral health.	B	Rossi et al., 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

HYPERLIPIDEMIA

Signal Symptoms: BMI greater than 25, central obesity, metabolic syndrome, diabetes, familial hypercholesterolemia, thyroid dysfunction, corneal arcus, xanthelasma, xanthoma.

Description: Hyperlipidemia is a major cardiovascular risk factor that increases the rate of atherosclerotic disease in the general population, as well as in older adults. Other terms used to denote this condition are hypercholesterolemia, hypertriglyceridemia, and dyslipidemia, and it is generally classified as primary or secondary. Increasing age continues to be the strongest nonmodifiable risk factor for atherosclerotic cardiovascular disease (ASCVD). According to the World Health Organization (WHO, 2014), 80% of deaths from heart disease occur in the population more than 65 years of age.

Many factors contribute to elevated cholesterol but generally fall into the category of lifestyle or genetic causes. Cholesterol metabolism is complicated and treatment of high cholesterol in the older adult, with medication, has been a controversial topic. There are limited clinical trials that represent the older population (more than 75 years old) in the preventative care of clinical ASCVD. The PROSPER trail was the only study that was specifically designed to evaluate the efficacy of 3-hydroxy-3-methyglutaryl-coenzyme A (HMG-CoA) reductase inhibitors (statins) in the primary and secondary prevention in older adults ages 70 to 82 years (Shepard, Blauw & Murphy, 2002). Guidelines from the American College of Cardiology and American Heart Association (ACC/AHA) (Stone et al., 2014) and the National Lipid Association Part 2 (Jacobson et al., 2015) both support the pharmacological treatment of hyperlipidemia in the older adult with risk factors.

Since the publication of the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) in 2002 there have been several updates released from professional organizations. The ACC/AHA in 2013 and the NLA in 2015 updated their guidelines to include specific recommendations for management of dyslipidemia in older patients. The newer evidence uses more specific risk assessment equations that focus on major risk factors rather than age to calculate heart disease risk. Maintenance of a healthy diet and exercise regimen remain the cornerstone for risk reduction. An empathic, patient-centered approach to care is essential to facilitate optimal care of this patient population.

Etiology: Cholesterol is essential to the structure and function of cell membranes, as well as vitamin and hormone synthesis in the body. It is formed in the human body in three different ways: food absorption, bile acids produced by the liver and reabsorbed through the intestinal tract, and through cellular synthesis. The latter two have a strong genetic component. Cholesterol is transported in our circulation by lipoproteins. The four major types of lipoproteins are chylomicrons, very low-density lipoprotein (VLDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL). These lipoproteins are characterized by their densities. The degree of lipid in a lipoprotein affects its density—the lower the density of a lipoprotein, the more lipid it contains relative to protein (Felix-Redondo, Grau, & Fernandez-Berges, 2013). The total plasma cholesterol measured in a lipid panel is determined by the supply of cholesterol from VLDL, IDL, LDL, and HDL.

Approximately 60% of plasma cholesterol is carried in the LDL particle, which is predominantly responsible for the transport of cholesterol from the liver to the peripheral tissue. It is this mechanism, under certain conditions, that leads to fatty deposits in the intimal layer of the artery and is the foundation for atherosclerotic lesions. HDL contains and transports approximately 30% of plasma cholesterol. This lipoprotein is considered "good cholesterol" and is responsible for the reverse transport of cellular cholesterol and macrophages back to the liver and elimination from the body. The regulatory mechanisms of LDL and HDL cholesterol control factors that are responsible for the onset, progression, and complications of atherosclerosis and cardiovascular disease (CVD).

The pathological process of atherosclerosis is influenced by many other factors as well. Age (older than 45 years for men and 55 years for women); male gender; genetic abnormalities in lipoprotein metabolism; and comorbidities such as diabetes, chronic kidney disease (CKD), and hypertension (HTN) all contribute significantly to this disease process. Lifestyle factors, such as poor diet, physical inactivity, weight gain, smoking, and excess alcohol intake also promote the development of CVD (Jacobson et al., 2015).

Secondary causes of hypercholesterolemia include hypothyroidism, liver disease, and certain medications. Older adult patients are often on polypharmacy; therefore, medications should be evaluated thoroughly. Hernick and Ito (2015) did a review looking at medications that induced changes in lipid and lipoproteins. Medications listed in their report include:

- Antihypertensive drugs (diuretics and β-adrenergic blockers)
- Other cardiovascular drugs (amiodarone)
- Steroid hormones (estrogens and progestins)
- Anabolic steroids (Danazol)
- Androgens (testosterone replacement)
- Retinoids (oral isotretinoin)
- Antipsychotics (Clozapine)
- Immunosuppressives (corticosteroids, cyclosporine, and tacrolimus)
- HIV therapy (protease inhibitors)
- Interferons

Occurrence: According to data from the Heart Disease and Stroke Statistics (Mozaffarian et al., 2016), an estimated 30.9 million adults 20 years of age and older have serum total cholesterol levels greater than or equal to 240 mg/dL, with a prevalence of 13.1%. This data indicates a steady decline in the prevalence of high LDL cholesterol over the last four decades. Between 1976 to 1980 and 2007 to 2010, the prevalence of high LDL cholesterol decreased significantly in adults aged 40 to 64 years (56% to 27%) and 65 to 74 years (72% to 30%). This decline in cholesterol levels in recent years appears to be a result of an increase in the use of cholesterol-lowering medications rather than improvements in dietary patterns (Ford & Capewell, 2013).

Age: Hyperlipidemia can occur at any age due to genetic syndromes that are associated with accelerated atherosclerosis and early CVD. The effects of cholesterol levels on CAD mortality rates are much higher in the older adult. In men, the level of total cholesterol increases with age from puberty until age 45 to 55 years, followed by a plateau until age 70 years, at which time a mild decrease is observed. In women, the level continues to rise another 10 years before it begins to decrease. However, the levels in women usually remain higher than men of similar age group. This mild drop in total cholesterol with aging is thought to be a result of a decrease in LDL synthesis related to a decline in liver function (Felix-Redondo, 2013).

The burden of ASCVD is significant in the older population and is expected to worsen with the increasing life expectancy of patients. The Department of Economic and Social Affairs, Population Division (2015) predicts that the global number of older adults will double by the year 2050. The 2016 AHA Heart Disease and Stroke Statistics report showed the average age of first heart attack in men is at age 65 years and in women age 71.8 years. Stroke statistics show that nearly 70% of first strokes occur in patients 65 years of age and older. Older patients should continue to be screened for risk factors and treated for hypercholesterolemia and the potential cardiovascular sequelae.

Gender: Gender-specific differences should be considered in the screening and management of hyperlipidemia. Until recently, women have been underrepresented in randomized controlled trials (RCTs) of lipid-lowering therapies. The most recent data available indicate that more women than men die annually from CVD and women account for 60% of deaths due to stroke. CVD risk increases as women transition through menopause. The lipid profile adversely changes in postmenopausal women. It is believed that when menopausal women lose the protection of estrogen, the LDL cholesterol begins to increase and the protective HDL cholesterol decreases. The NLA Recommendations for Patient-Centered Care – Part 1 (Jacobson, 2015) highlight the need to perform ASCVD risk assessments on women with intensity of treatment corresponding to the level of risk. Clinical data indicates that women may be at higher risk for myalgia when taking statin medications.

Ethnicity: Treatment guidelines for patients of Hispanic/Latino, African American, and South Asian descent are outlined in the NLA Recommendations for Patient-Centered Management of Dyslipidemia – Part 2 (Jacobson, 2015). Special considerations for each group are discussed and include the following recommendations. Hispanics/Latinos are the fastest growing and largest minority population in the United States. This population is very diverse with risk factor burden tracing back to their country of origin. Hispanics/Latinos tend to have a greater occurrence of high TG and low HDL than non-Hispanic Caucasians, leading to higher levels of the more atherogenic non-HDL-cholesterol. These patients also have a higher prevalence of type 2 diabetes mellitus, obesity, and metabolic syndrome compared to non-Hispanic Caucasians, particularly among women.

African Americans as a group are at greater risk for ASCVD, however, this risk is less the result of hyperlipidemia then in the non-Hispanic Caucasian population. For this reason, when assessing ASCVD risk in African Americans, particular attention should be given to non-lipid risk factors, such as hypertension, overweight and obesity, type 2 diabetes mellitus, and physical inactivity.

South Asian Americans also have an increased risk for ASCVD. This population has a greater prevalence of insulin resistance than non-Hispanic Caucasians, as well as some of the metabolic disturbances that accompany this condition, including high TG, low HDL-C, and hyperglycemia. Because of the increased risk of metabolic syndrome in Asian patients, practitioners should be aware that the waist circumference cut points are different than those of Caucasians. A waist circumference of greater than or equal to 37 inches (94 cm) for men and 32 inches (80 cm) for women meets the criteria for metabolic syndrome in South Asian Americans. The NLA (2015) also recommends starting with a moderate intensity statin dose and titrating upward to reach cholesterol goals, or downward if intolerance occurs, due to the possibility of genetic variations in drug metabolism for patients of Asian ethnicity.

Contributing Factors: Both modifiable and nonmodifiable influences contribute to the risk for hyperlipidemia. Age, gender, and family history are risk factors that cannot be changed, however, diets high in saturated and trans fats, inactivity, and excess weight are strong controllable factors that have a significant impact on cholesterol parameters. Obesity is associated with increased total cholesterol, LDL, VLDL, and triglycerides, as well as with decreased levels of HDL. Other contributing factors to hyperlipidemia include alcoholism, steroid use, and smoking. Cigarette smoking lowers HDL levels and is an independent risk factor for cardiovascular disease. Secondary causes are discussed under etiology.

Signs and Symptoms: Hyperlipidemia is typically asymptomatic and is usually detected during a routine lipid screening. Occasionally, in patients with genetic disorders such as familial hypercholesterolemia, xanthelasma and xanthomas may be present. These are fatty deposits under the skin surface, on the hands, elbows, knees, heels, or eyelids, and found in patients with very high cholesterol. Ocular signs of hyperlipidemia may include arcus senilis and xanthelasma palpebrarum. Arcus senilis is a yellowish-white ring around the cornea that is caused by lipid deposition in the peripheral cornea. Xanthelasma palpebrarum is a plaque-like yellow lesion near the inner canthus of the eyelids that is caused by lipid deposition in the dermis (Leichter, Johnson, Ammerman, & Egbert, 2013)

Diagnostic Tests: The U.S. Preventive Services Task Force (USPSTF) (2014) strongly recommends that clinicians routinely screen men age 35 years and older and women age 45 years and older for lipid disorders and treat abnormal lipids in people who are at increased risk of coronary heart disease. There is no specific age recommendation for stopping screening. The screening should include a total cholesterol (TC), LDL, HDL, and triglyceride level, as well as a measurement of non-high-density lipoprotein cholesterol (non-HDL-C). This number is simply the difference between the total cholesterol concentration and the HDL cholesterol concentration. The non-HDL number provides a single index of atherogenic cholesterol particles and many researchers believe this provides a better assessment of heart disease risk (Jacobson, 2105).

TABLE 14-4 Treatment Goals and Levels to Consider Drug Therapy According to Risk Category

RISK CATEGORY	TREATMENT GOAL (Non-HDL-C mg/dL LDL-C mg/dL)	CONSIDER DRUG THERAPY (Non-HDL-C mg/dL LDL-C mg/dL)
Low	<130 <100	≥190 ≥160
Moderate	<30 <100	≥160 ≥130
High	<130 <100	≥130 ≥100
Very High	<100 <70	≥100 ≥70

Non-HDL-C testing is universally available, requires no additional cost, and may be measured in the non-fasting state.

Table 14-4 displays the NLA recommendations for lipid management according to risk category. The atherogenic cholesterol (non-HDL-C and LDL-C) levels are considered the appropriate targets of therapy. In patients 65 years of age and older, whom treatment with medication is questionable, a coronary artery calcium (CAC) score may be considered to obtained additional objective information for treatment decisions. A CAC score of zero in asymptomatic patients may indicate nonpharmacological treatment as an option.

Both the ACC/AHA and the NLA utilize risk calculation tools in their primary prevention risk assessment. These tools are used in select older adult individuals with one additional risk factor to further assess risk. However, the two organizations differ in their cut points of high risk and the use of specific treatment goals. Instead of the traditional NLA ATP III risk factor stratification, treat-to-goal approach, the ACC/AHA 2013 guidelines recommend the use of statins in four specific groups of patients. The new guideline eliminates treatment targets or goals and focuses on treatment for those "at risk" with a fixed dose of a moderate-intensity statin (defined as an LDL cholesterol reduction of 30% to less than 50%) or a high-intensity statin (LDL cholesterol reductions of greater than 50%). Statins will be discussed in more detail in the section on Treatment. The four "at risk" patient groups that were identified by the ACC/AHA task force include:

1. Patients with clinical ASCVD
2. Patients with primary elevations in LDL cholesterol greater than or equal to 190 mg/dL
3. Patients with type 1 or 2 diabetes between the ages of 40 and 75 years
4. Patients with an estimated 10-year ASCVD risk greater than or equal to 7.5%

The new 10-year risk estimator tool is called the Pooled Cohort Cardiovascular Risk Estimator and is recommended instead of the traditional Framingham Risk Score (FRS). Components assessed in the risk calculation include gender, age, race, TC, HDL, systolic blood pressure, treatment for hypertension, diabetes, and smoking status. The new tool adds diabetes and race into the risk calculation and includes stroke in the outcome, however, it can only be used to calculate the 10-year risk in patients between 40 and 79 years of age (ACC/AHA, 2013). The risk calculator can be found at http://tools.acc.org/ASCVD-Risk-Estimator/.

For secondary prevention, the ACC/AHA supports the use of moderate- but not high-intensity statins for patients older than 75 years. The data does not support the use of statins in this age group without clinical ASCVD or diabetes (ACC/AHA, 2013).

Recommendations for lipid management from the 2015 NLA Expert Panel on older patients differs somewhat from that of the ACC/AHA. A 10-year ASCVD risk greater than or equal to 15% is used instead of greater than or equal to 7.5% in the Pooled Cohort Equation and the ATP III Framingham Risk Calculator is recommended. The use of risk calculators should be used with caution in the older adult, as advanced age is often the major driver of increased ASCVD risk and could result in potential overtreatment of lower risk older adult individuals.

Primary prevention strategies in patients 65 to 79 years old is the same as younger adults and should follow the NLA Recommendations of Patient-Centered Management of Dyslipidemia-Part 1. The NLA expert recommendations for secondary prevention are:

1. Consideration of moderate- or high-intensity statin medication for patients 65 to 75 years of age with ASCVD or diabetes mellitus.
2. Similar statin doses are recommended for patients 75 to 80 years of age after careful risk-benefit ratio assessment.
3. For patients older than 80 years of age, moderate-intensity statin may be considered after careful evaluation of polypharmacy and drug-to-drug interaction, comorbidities, cost, and patient preference.

Differential Diagnosis: When evaluating older patients with hyperlipidemia it is important to think through other potential medical causes. Differential diagnosis is discussed here based on their lipoprotein abnormality. Hypercholesterolemia (elevated TC and LDL) can be attributed to hypothyroidism, nephrotic syndrome, and malnutrition. Hypertriglyceridemia (elevated triglycerides) can be secondary to diabetes mellitus, obesity, metabolic syndrome, alcohol use, oral estrogen, renal failure, hypothyroidism, retinoic acid, and lipodystrophies. Combined hyperlipidemia (high TC, LDL and triglycerides) can be caused from diabetes mellitus, obesity, metabolic syndrome, nephrotic syndrome, hypothyroidism, and lipodystrophies. Lastly, low HDL cholesterol can be linked to diabetes mellitus, obesity, metabolic syndrome, hypertriglyceridemia, smoking, and the use of anabolic steroids (Bhat, Dretler, Gdowski, Ramgopal, & Williams, 2016). Medication causes are discussed in the section on Etiology. Investigating and treating these secondary causes can often improve abnormal lipid disorders without aggressive statin therapy.

Treatment: Managing modifiable risk factors such as hyperlipidemia remains the primary method to prevent ASCVD. Hypercholesterolemia is common in the older population and decision making regarding treatment can be challenging. Ideally, patients at risk for ASCVD should begin treatment well before they reach older age. Treatment strategies in this

population require careful consideration and should be made on an individual patient basis.

Older adults should follow the general principals of a healthy lifestyle recommended by the 2015–2020 Dietary Guidelines for Americans (DGA). A balance between energy intake (calories in) and expenditure (calories out) is emphasized to maintain a healthy body weight. The following are key general recommendations that can help older individuals achieve healthy eating patterns within calorie limits:

- Consume less than 10% of calories per day from added sugars.
- Consume less than 10% of calories per day from saturated fats.
- Consume less than 2,300 mg per day of sodium.
- If alcohol is consumed, it should be consumed in moderation (7 drinks or less per week for women and 14 drinks or less per week for men)

A recent recommendation from the NLA expert panel, based on results from RCTs, indicates that dietary cholesterol has modest effects on levels of TC, LDL, and HDL cholesterol, and that saturated fatty acids and trans fatty acids have a more predictable impact on levels of atherogenic cholesterol (Jacobson et al., 2015).

Regular physical activity is essential for healthy aging. Adults age 65 years and older gain substantial health benefits from regular physical activity. According to the Office of Disease Prevention and Health Promotion (DPHP), older adults should follow these recommendations for exercise:

- All older adults should avoid inactivity. Some physical activity is better than none, and older adults who participate in any amount of physical activity gain some health benefits.
- For substantial health benefits, older adults should do at least 150 minutes (2 hours and 30 minutes) a week of moderate-intensity, or 75 minutes (1 hour and 15 minutes) a week of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity aerobic activity. Aerobic activity should be performed in episodes of at least 10 minutes, and preferably, it should be spread throughout the week.
- For additional and more extensive health benefits, older adults should increase their aerobic physical activity to 300 minutes (5 hours) a week of moderate-intensity, or 150 minutes a week of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity activity. Additional health benefits are gained by engaging in physical activity beyond this amount.
- Older adults should also do muscle-strengthening activities that are moderate or high intensity and involve all major muscle groups on two or more days a week, as these activities provide additional health benefits.
- When older adults cannot do 150 minutes of moderate-intensity aerobic activity a week because of chronic conditions, they should be as physically active as their abilities and conditions allow.
- Older adults should do exercises that maintain or improve balance if they are at risk of falling.
- Older adults should determine their level of effort for physical activity relative to their level of fitness.

TABLE 14-5 Intensity of Statin Therapy

HIGH INTENSITY DAILY DOSE ↓ LDL-C ≥50%	MODERATE INTENSITY DAILY DOSE ↓ LDL-C 30 TO <50%
Atorvastatin 40–80 mg	Atorvastatin 10–20 mg
Rosuvastatin 20–40 mg	Fluvastatin 40 mg bid
	Fluvastatin XL 80 mg
	Lovastatin 40 mg
	Pitavastatin 2–4 mg
	Pravastatin 40–80 mg
	Rosuvastatin 5–10 mg
	Simvastatin 20–40 mg

- Older adults with chronic conditions should understand whether and how their conditions affect their ability to do regular physical activity safely.

It has been well established in multiple clinical trials and in more than 27 years of clinical use that 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors, statins, are one of the most effective treatments to reduce ASCVD risk, particularly for secondary prevention. Statin use in the older adult as primary prevention is not as strong due to limitations in our risk estimation tools and lack of RCTs for this age group. In addition to their cholesterol-lowering properties, statins also have anti-inflammatory effects that are believed to contribute to plaque stabilization. There are currently seven statin medications available: rosuvastatin, atorvastatin, pitavastatin, simvastatin, lovastatin, pravastatin, and fluvastatin. Statins should be prescribed with regard to potency and intensity as recommended in both the NLA and ACC/AHA guidelines (see Table 14-5).

Safety of these agents, particularly in the older adult, remains a concern. Advancing age, multiple comorbidities, and polypharmacy can cause pharmacokinetic and pharmacodynamic variability, resulting in potential adverse effects (Grundy et al., 2014). In 2012, the FDA issued additional label changes addressing concerns associated with hepatic injury, the risk of developing diabetes, myopathy, and cognitive dysfunction. By far, the most frequently reported adverse effect of statin therapy is the associated muscle complaints or myopathy. It is often difficult for the practitioner to differentiate between what may be statin related and what may be an age-related musculoskeletal pain. Using the lowest effective dose of the statin or re-challenging patients with interval (every second or third day) dosing should be considered before discontinuation of the medication, as long as laboratory values remain stable. If these strategies are ineffective and myalgia symptoms persist, then non-statin therapies should be discussed.

Evidence supporting the relationship between statin therapy and cognitive changes is lacking, however, these complaints must be evaluated by the health-care provider. Other causes for cognitive changes should be investigated, including B_{12} deficiency, depression, delirium, thyroid dysfunction, and other medications. Again, decreasing the dose or switching to a hydrophilic statin, such as pravastatin or

rosuvastatin, may be considered before discontinuation of the medication (Thelen, 2006). As with any clinical decision in patient care, the medication benefit must outweigh the associated risk.

Evidence in three key meta-analyses (Sattar et al., 2010; Preiss et al., 2011; Waters et al., 2011) indicates that statin use is associated with a modest but statistically significant increase in the risk of new-onset diabetes. Patients at higher risk for diabetes before beginning statin therapy appeared to be at highest risk in these studies. It is important for the primary care provider to evaluate this diabetes risk related to the CVD benefit of statin therapy. These same meta-analyses demonstrated that several cardiovascular events are prevented for each new statin-related diabetes case.

Elevations in liver enzymes and hepatic damage have been a concern since the first statin was introduced in 1987. However, there is no evidence that routine monitoring of hepatic function is effective in identifying the rare case of liver injury. The FDA's update on statin labeling in 2012 confirmed this statement and determined that routine monitoring of liver enzymes was not necessary. Consequently, liver enzyme tests are recommended prior to beginning statin therapy and as clinically indicated thereafter.

There are many non-statin therapy regimens available to treat hyperlipidemia. No clinical trial has studied the effect of combination therapy on ASCVD risk reduction in the older population and met the primary endpoint. The ACC/AHA guidelines only recommend non-statin therapies in high-risk patients who either fail therapy on a maximum tolerated statin or are completely intolerant to statins. However, the NLA recommends treating to specific LDL and non-HDL cholesterol goals, making the addition of non-statin medication an option to reach these goals in high-risk patients.

Non-statin therapy includes ezetimibe, niacin, fibrates, bile acid sequestrants, and omega-3 fatty acids. When making clinical decisions regarding the use of non-statin or combination therapy, careful consideration of polypharmacy, drug–drug interactions, and adverse effects must be a priority. Equally, it is important to recognize that older adults are less likely to tolerate high-dose statins and some lower-dose combination therapies may be an excellent choice to achieve treatment goals with minimal side effects.

Ezetimibe is a cholesterol absorption inhibitor and is the only agent in its class. It can be used alone for a modest LDL lowering effect (18%) or in combination with a statin for a more robust lowering effect (54%). Results from the IMPROVE-IT trial showed that patients taking a combination of simvastatin with the non-statin ezetimibe experienced significantly fewer major cardiovascular events than patients treated with simvastatin alone. Several combinations are available.

Niacin, or nicotinic acid, is a form of vitamin B_3 and has been available for the treatment of cholesterol disorders since the early 1950s. Niacin's primary use has been in the treatment of low HDL cholesterol disorders, but has also been used to treat elevated LDL cholesterol. Its main mechanism of action is in the reverse transport of cellular cholesterol and macrophages from the body. Niacin is used with caution in older adults due to safety and tolerability concerns. In addition, recent secondary prevention studies have failed to show any additional benefit of using niacin with maximum dose statins (i.e., AIM-HIGH, HPS2-THRIVE).

Fibrates are a non-statin therapy that is primarily used to treat high triglycerides and VLDL cholesterol, but also has a modest effect on raising the HDL cholesterol. These medications work by enhancing the catabolism of triglyceride-rich particles and reducing the secretion of VLDL. Gemfibrozil use is contraindicated with simvastatin due to increased risk of myopathies. Fenofibrate is therefore preferred in combination with statins. However, gemfibrozil is preferred in patients with CKD because its effect on serum creatinine levels are more favorable when compared with fenofibrates (Davidson et al., 2007).

Bile acid sequestrants (BASs), sometimes referred to as resins, include cholestyramine, colesevelam, and colestipol. These drugs bind with cholesterol-containing bile acids in the intestines and are then eliminated in the stool. The primary effect of BASs is a 20% reduction in LDL cholesterol, however, these medications also have a beneficial clinical effect in the treatment of diabetes (Staels, Handelsman & Fonseca, 2010). These drugs can cause significant GI side effects and are therefore usually limited to those patients unable to tolerate a statin.

Omega-3 fatty acids (O3FAs) refer to the long-chain polyunsaturated fatty acids docosahexaenoic (DHA) and eicosapentaenoic (EPA). There are currently two prescription strength O3FAs on the market, Lovaza and Vascepa, which are indicated for the treatment of moderate to severe hypertriglyceridemia. These products are generally well tolerated, but can inhibit platelet aggregation and should be used with caution in patients taking antiplatelet or anticoagulation therapies (Kris-Etherton, Harris, & Appel, 2002).

Follow-Up: The NLA Recommendations for Patient-Centered Management of Dyslipidemia – Part 1 (Jacobson, 2015) advise a follow-up visit 6 weeks after the primary diagnosis of hyperlipidemia. If therapeutic lifestyle change (TLC) alone was the treatment of choice at the primary visit, then weight, dietary adherence, and exercise level should be evaluated. This visit should also include a lipid panel to measure atherogenic cholesterol response to TLC. If the patient is not at goal, these recommendations should be reinforced and a referral to a dietician for medical nutritional therapy should be considered. The patient should be reevaluated in 6 weeks to review response to therapy. If the patient is not to goal, then TLC should be intensified and drug therapy should be considered. For patients at high risk for ASCVD, drug therapy and TLC may be initiated concurrently. Patients on drug therapy should be evaluated every 4 to 6 months to monitor compliance and potential side effects.

Sequelae: Complications related to untreated or undertreated hypercholesterolemia include heart disease (acute coronary syndrome and ischemic heart disease), stroke, organ insufficiency (erectile dysfunction, CKD), peripheral vascular disease (PVD), and pancreatitis. Early screening and management of high cholesterol is the cornerstone of preventing these life-threatening conditions.

Prevention/Prophylaxis: Prevention focuses on a healthy lifestyle of good nutrition and physical activity. The NLA Expert Panel supports a cardioprotective eating pattern for the management of dyslipidemia. This includes minimal intake of trans fatty acids, viscous fiber intake (5 to 10 g/day), fiber-rich whole grains that have 1.1 g or more of fiber per 10 g

of carbohydrate, vegetables, nuts, and restriction of alcohol and refined carbohydrates. The recommended minimal frequency, intensity, and duration of exercise for improving the lipid profile (reduce TG and modestly raise high-density lipoprotein cholesterol [HDL-C]) are 5 or more days/week at 40% to 75% aerobic capacity, and at least 30 to 60 minutes of accumulated daily physical activity (at least 150 minutes per week). This level of physical activity is consistent with the NLA expert panel and public health recommendations.

Referral: The treatment of lipid disorders in older patients can generally be managed by the practitioner in primary care; however, a team-based collaborative approach to include a dietician for medical nutritional therapy and a personal trainer that specializes in cardiovascular fitness for seniors is an excellent approach. Patients with ASCVD should also be monitored by a cardiologist to track progression or stabilization of the disease. Lipid clinics are available if practitioners feel a lipid specialist is necessary.

Education: Education should focus on improving patient outcomes. When patients do not understand their disease or condition and the importance of the treatment regimen they are less likely to be compliant. Take the time to educate patients on what to expect from their treatment, as well as the risks versus benefits. Considerations in the older adult include cost, dosing frequency of medication, physical difficulties in taking the drug, and risk of adverse drug reactions as perceived by the patient. Identify and discuss any barriers to adherence and formulate strategies for overcoming them. Involve the patient in the treatment plan and establish a positive, supporting, trusting relationship.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
In treating patients more than 65 years to less than 80 years of age with ASCVD or diabetes mellitus, moderate- or high-intensity statin therapy should be considered after a careful consideration of the individual risk-benefit ratio.	A	Jacobson, Maki, Orringer, Jones, Kris-Etherton, Sikand, . . . Brown, 2015
Older, primary prevention patients who are statin-eligible should undergo a patient-centered discussion with their provider about the risks and benefits of statin therapy so that they can make a more informed decision about taking statins over the long term.	C	Jacobson, Maki, Orringer, Jones, Kris-Etherton, Sikand, . . . Brown, 2015 Dixon, Donohoe, Ogbonna, & Barden, 2015
For patients reporting statin intolerance, consider the use of alternate statin regimens such as low-intensity statin therapy or non-daily moderate-intensity statin therapy; low-dose statin combination therapy with ezetimibe, bile acid sequestrants, or niacin; or non-statin as a second-line option.	B	Jacobson, Maki, Orringer, Jones, Kris-Etherton, Sikand, . . . Brown, 2015 Dixon, Donohoe, Ogbonna, & Barden, 2015
When treating for secondary prevention of hyperlipidemia in patients >80 years of age, consider using one of the moderate-intensity statins based on a provider-patient discussion of the risks and benefits of such therapy, consideration of drug-drug interactions, polypharmacy, concomitant medical conditions including frailty, cost considerations, and patient preference.	B	Jacobson, Maki, Orringer, Jones, Kris-Etherton, Sikand, . . . Brown, 2015

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HYPERTHYROIDISM

Signal Symptoms: Apathy, confusion, weakness, course tremors, anorexia, palpitations, lid lag, lid retraction.

Description: Hyperthyroidism occurs when there is an excessive synthesis and secretion of thyroid hormone (Kravets, 2016). Whereas the clinical signs and symptoms of hyperthyroidism are generally well recognized in younger patients, older adults present atypically, often with new cardiac arrhythmias and apathetic mood changes (Boelaert, Torlinska, Holder & Franklyn, 2010; Trivalle et al., 1996).

Etiology: The most common cause of hyperthyroidism in adults is Graves' disease; however, the proportion of patients with toxic multinodular goiter (Plummer's disease) and toxic adenoma increases with age. Graves' disease is a familial autoimmune disorder of unknown origin in which thyrotropin receptor antibodies stimulate the TSH receptor, resulting in increased thyroid hormone production (Kravets, 2016). It is thought to be an HLA-related, organ-specific defect in suppressing T-lymphocyte function (Lee et al., 2010). The multiple nodules of a toxic multinodular goiter are thought to produce thyroid hormone without TSH, resulting in thyrotoxicosis.

Occurrence: Less than 5% of people in the United States have hyperthyroidism.

Age: Of all cases of hyperthyroidism, 15% to 25% occur in people 60 years and older (Papaleontiou & Haymart, 2012).

Gender: Women develop Graves' disease more frequently than men, although it has been shown that by age 70 years, the prevalence of the disease among men and women is almost equal (Boelaert et al., 2010). Older men who present with hyperthyroidism are more likely to develop atrial fibrillation from the untreated thyroid disorder than older women (Boelaert et al., 2010).

Ethnicity: A study of U.S. military personnel found that African Americans, as well as Asian/Pacific Islanders, had a higher incidence of Graves' disease than Caucasians (McLeod, Caturegli, Cooper, Matos, & Hutfless, 2014).

Contributing Factors: Toxic multinodular goiter occurs in a small percentage of people with hyperthyroidism in the United States; the number increases in countries known for iodine deficiency. TSH-producing pituitary tumors and metastatic follicular carcinoma are rare causes of hyperthyroidism (Gambert, Kant, & Miller, 2014). Patients with other autoimmune problems such as diabetes, rheumatoid arthritis, and myasthenia gravis are at risk for developing hyperthyroidism. A familial tendency for developing hyperthyroidism exists (Kravets, 2016). Known smokers have been found to have increased symptom presentation of hyperthyroidism (Osman, Franklyn, Holder, Sheppard, & Gammage, 2007). Patients prescribed amiodarone or radiographic contrast media can develop iodine-induced hyperthyroidism (Kravets, 2016; Samuels & Franklyn, 2015). Hyperthyroidism can occur in patients with metastatic follicular thyroid cancer, certain ovarian tumors, and TSH-secreting pituitary tumors. Patients who are known to consume diets high in excess iodine are at risk for developing hyperthyroidism (Leung & Braverman, 2014). Hyperthyroidism always needs to be considered in patients already prescribed thyroid replacement therapy, given that over time it may develop due to normal aging changes (Gambert et al., 2014).

Signs and Symptoms: Older patients may report fatigue, confusion, anorexia, weight loss, apathy, depression, heat intolerance, course tremor, insomnia, palpitations, chest pain, dyspnea, nervousness, and anxiety (Boelaert et al., 2010; Samuels & Franklyn, 2015). Older females were found to more frequently report weight gain, experience palpitations, and on examination, have more neck enlargement than older males (Boelaert et al., 2010). Older adults may experience diarrhea; prior history of constipation may resolve with the onset of undiagnosed hyperthyroidism. A reduction or increase in appetite may be revealed; patients with severe hyperthyroidism may become cachectic. Photophobia and blurred and/or double vision may be reported (Gambert et al., 2014).

Patients and/or family members may report a new onset of decreased attention span. Known as apathetic hyperthyroidism, patients and/or caregivers may report that the patient is withdrawn, anorexic, and experiencing new onset of proximal muscle weakness, all contributing to inactivity (Samuels & Franklyn, 2015). Lethargy and listlessness are more common than hyperactivity (Papaleontiou & Haymart, 2012). Older patients with untreated hyperthyroidism may exhibit a cachectic or chronically ill overall appearance. Assess for signs of anemia, such as pallor in conjunctiva.

On physical examination, ophthalmological changes in the elderly such as exophthalmos are not as common, however, one study did show that ophthalmopathy is more prevalent in older adults who smoke (Boelaert et al., 2010). Lid lag and lid retraction are more common findings. When tested, extraocular muscle palsy may be evident (Gambert et al., 2014).

An enlarged thyroid occurs in 20% to 40% of patients age 70 years and older. The examiner needs to remember that the thyroid is found lower in the neck and substernal area in older adults than in a younger person. Palpate the thyroid to determine presence or absence of tenderness.

Because cardiac findings are very prevalent in older adults with hyperthyroidism, a thorough cardiac examination is essential. Accurate measurement of pulse and blood pressure is important for the initial work-up and ongoing surveillance of patients with hyperthyroidism. Tachycardia, angina, new onset of atrial fibrillation, and heart failure are common manifestations of untreated hyperthyroidism (Kravets, 2016; Papaleontiou & Haymart, 2012). New onset of atrial fibrillation is much more common in the older adult that has thyrotoxicosis. Additionally, these patients may also exhibit worsening ischemic heart disease (Samuels & Franklyn, 2015). Look for evidence of peripheral nonpitting edema with asymmetrical plaques or nodules (pretibial myxedema).

A reduction in muscle mass may be detected (proximal weakness is common in hyperthyroidism), so muscle strength should be tested. Ask patients to demonstrate their ability to stand up from a seated position. If patients report recent falls, observe gait and assess the patient's ability to maintain balance. Test for postural instability (Gambert et al., 2014). A coarse tremor may be evident; note for presence in outstretched fingers. You may note brisk tendon reflexes, however, these are less common in older adults (Boelaert et al., 2010).

Diagnostic Tests: The TSH level is decreased in patients with hyperthyroidism. If the serum free T_4 result is normal following a decreased TSH, a serum T_3 with radioimmunoassay should be ordered (Kravets, 2016). This value is sometimes increased, indicating T_3 toxicosis (Gambert et al., 2014). If the T_3 result is also normal, the patient is said to have subclinical hyperthyroid disease (Grossman et al., 2016). Radioactive iodide uptake (RAIU) is ordered to differentiate the cause of the thyrotoxicosis. This test is usually elevated in the presence of hyperthyroidism. There is a diffuse uptake of RAIU in Graves' disease, whereas there is focal uptake in toxic nodular

thyroiditis. In older adults, an electrocardiograph (EKG) is warranted with associate cardiac arrhythmias. The use of ultrasound as a safe alternative to radioactive iodine uptake and scan is recommended for patients with amiodarone-induced thyrotoxicosis (Kravets, 2016). Routine laboratory tests of patients with hyperthyroidism may reveal impaired glucose tolerance, increase in serum calcium levels, and elevated liver enzymes (Gambert et al., 2014).

Differential Diagnosis:

- Anxiety
- Depression
- Diabetes mellitus (normal fasting blood sugar)
- Severe anemia
- Leukemia
- Atrial fibrillation
- Pheochromocytoma
- Certain endocrine malignancies (thyroid function studies are generally normal; fine-needle aspiration of nodule confirms diagnosis of malignancy) (Gambert et al., 2014)

Treatment: Hyperthyroidism needs immediate attention in older adults because it is usually associated with involvement of other organ systems, especially cardiac. Long-acting beta blockers are indicated in the early treatment of hyperthyroidism (unless contraindicated) to control the cardiac symptoms of palpitations and tachycardia, unless contraindicated by other concomitant conditions such as COPD, heart failure, and insulin-dependent diabetes mellitus (Papaleontiou & Haymart, 2012). Patients may report relief of tremors and emotional lability, along with an increase in exercise tolerance, once on a regular beta blocker regimen (Papaleontiou & Haymart, 2012). Older patients may also be treated with antithyroid agents, initially with methimazole or propythiouracil (Gambert et al., 2014). Radioisotope therapy is the treatment of choice for toxic thyroid nodules and Graves' disease; however, patients should be in a euthyroid state before receiving radioactive iodine treatment (Papaleontiou & Haymart, 2012).

Follow-Up: Patients with hyperthyroidism require monitoring during the course of treatment, including thyroid studies, cardiac status, and changes in mental status. The patient must be aware of changes to recognize impending hypothyroidism once treatment has been initiated. Diagnostic studies, usually ordered by the patient's endocrinologist, include free T_4 and serum T_3 monitored every 4 to 8 weeks following treatment for Graves' disease until a euthyroid state is achieved, and then TSH levels every 3 months for the first year, and twice yearly thereafter, until the patient's condition is stable and he or she is asymptomatic for thyroid disease (Gambert et al., 2014). Underlying anemia diagnosed at the same time the hyperthyroidism was diagnosed needs reevaluation. Following radioactive iodine ablation, most patients eventually develop hypothyroidism within 2 to 6 months and will require thyroid replacement (Kravets, 2016). For patients on medical therapy only, relapse is common and patients should be instructed to contact their provider if symptoms present again. Careful monitoring of thyroid function is required on a regular basis monthly to quarterly for the first year and as needed (Kravets, 2016).

Sequelae: Complications that can develop in patients with hyperthyroidism include ischemic heart disease, angina, cardiac arrhythmia, atrial fibrillation, myocardial infarction, and heart failure. Bone density may decrease also, rendering the patient susceptible to the development of osteoporosis (Samuels & Franklyn, 2015). Patients prescribed antithyroid medications may develop drug-induced agranulocytosis up to 3 months after the thyroid medication is initiated.

Prevention/Prophylaxis: Hyperthyroidism is not known to be preventable; however, because thyroid disease is prevalent in the elderly, regular screening for signs and symptoms of hyperthyroidism in patients with a history of rheumatoid arthritis and other collagen diseases, diabetes mellitus, and family history of thyroid disease should be considered. Additionally, patients who present with new onset of atrial fibrillation, dyspnea, and weight loss, but are found to have subclinical hyperthyroidism, should be monitored for thyroid studies more frequently to monitor for potential changes in thyroid function (Boelaert et al., 2010).

Referral: Patients need to be referred to a radiotherapist for calculation of the dose of ^{131}I. Patients with complicated hyperthyroidism should be treated by an endocrinologist, especially in the presence of atrial fibrillation and thyroid storm. These patients usually require hospitalization.

Education: Patients should note a change in their overall behavior and function following the initial treatment. Thus, patients must realize that they will need to take the prescribed medication daily and continue with the radioactive iodine treatment. After patients have achieved the euthyroid state and received the radioactive iodine treatment, have them report to their health-care provider if they experience signs and symptoms of hypothyroidism or hyperthyroidism.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Older patients tend to present with atypical or subtle symptoms of hyperthyroidism, with atrial fibrillation a common finding. Older patients with new cases of atrial fibrillation may be found to have subclinical hyperthyroidism.		
The use of beta-adrenergic blockade agents should be prescribed for older adults with symptomatic thyrotoxicosis, presenting with heart rates >90 beats per minute.	B	Bahn, Burch, Cooper, Garber, Greenlee, Klein, . . . Ross, 2011

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Older patients with new cases of atrial fibrillation may be found to have subclinical hyperthyroidism.	B	Boelaert et al., 2010
Older men with subclinical hyperthyroidism were found to be at a slightly higher risk for hip fracture than men who were found to be euthyroid.	B	Lee et al., 2010

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HYPOTHYROIDISM

Signal Symptoms: Weakness, cold intolerance, myalgias, depression, apathy, impaired memory, fatigue, hair thinning and/or loss.

Description: Hypothyroidism occurs when the body tissues are subjected to subnormal amounts of thyroid hormone. Hypothyroidism is further classified as primary hypothyroidism, which is the failure of the thyroid gland to produce hormones; secondary hypothyroidism, which occurs when the pituitary gland fails to secrete adequate amounts of thyrotropin; and tertiary hypothyroidism, which is the failure of the thyroid to secrete thyrotropin-releasing hormone. It is a challenging disorder at times to recognize, given the often nonspecific symptoms the patients report. Progression is often slow and insidious.

Etiology: Autoimmune thyroiditis, known as Hashimoto's thyroiditis, is the most common cause of hypothyroidism in older adults. Persons who have had prior thyroid surgery or ablation of the thyroid are also susceptible to hypothyroidism (Pearson, 2013). Rarely, infection or infiltration of the thyroid will cause hypothyroidism (Guha, Krishnaswamy, & Peiris, 2002).

Occurrence: Approximately 11 million people in the United States have hypothyroidism (Papaleontiou & Haymart, 2012). Approximately 10% are adults 65 years and older; however, up to 10% of patients ages 80 years or older have subclinical hypothyroidism (Bensenor, Olmos, & Lotufo, 2012; Pearson, 2013).

Age: Predominantly begins at age 40 years, although the age of onset can continue through old age.

Gender: More prevalent in women than in men; women are up to 10 times as likely to be diagnosed with the condition (Bensenor et al., 2012).

Ethnicity: Most common in Caucasians. In a study of U.S. military personnel, African Americans and Asian Americans were found to have a lower incidence of hypothyroidism than Caucasians (McLeod et al., 2014).

Contributing Factors: Increased age and female gender are risk factors for developing hypothyroidism. Patients with previous thyroid dysfunctions, Hashimoto's thyroiditis, and goiter may develop hypothyroidism. There is an association between leukotrichia and also vitiligo and developing hypothyroidism. Patients who have had extensive neck surgery or radiation or prior thyroid surgery without proper follow-up often develop hypothyroidism. Hypothyroidism can also result from pituitary disease and certain infiltrative diseases such as sarcoidosis and scleroderma (Guha et al., 2002). A relationship exists between contact with some environmental pollutants (e.g., fire retardation materials), fungicides, and coal conversion products and the development of hypothyroidism (Bharaktiya et al., 2011). Long-term lithium use can also be a contributor to the disease, as can taking amiodarone. Additional medications known to decrease TSH levels include glucocorticoids, opioids, and dopamine (Pearson, 2013). Patients with first-degree relatives with thyroid disease are also at risk. Patients with type 1 and type 2 diabetes mellitus, rheumatoid arthritis, pseudogout, or Addison's disease should be routinely screened for thyroid disease (Gupta & Nagri, 2012). In patients with elevated lipids, consider hypothyroidism as an underlying condition. Practitioners should suspect underlying hypothyroidism in patients who are difficult to wean off of mechanical ventilation (Gambert et al., 2014; Pearson, 2013).

Signs and Symptoms: Patients often present with weakness, myalgias, arthralgias, fatigue, urticaria, decreased perspiration, cold intolerance, constipation, hair loss, leg cramps, hoarseness, tinnitus with decreased hearing reported, snoring, paresthesias, numbness, stiffness, headaches, and reported weight changes. Patients or family members may report depression, impaired memory, changes in personality, or the inability to concentrate on tasks such as calculating math (Bensenor et al., 2012). If the disease has progressed untreated, patients appear apathetic and debilitated, with possible frank psychosis (Pearson, 2013). Additionally, patients with undiagnosed hypothyroidism may begin to experience dyspnea and sleep apnea (Jonklaas et al., 2014). On physical examination, the overall appearance of the patient may reveal brittle nails, puffiness of the face and eyelids, thinning of the outer halves of the eyebrows, and xerosis (Bensenor et al., 2012). During the thyroid examination, the examiner should first observe the thyroid while the patient swallows and then proceed to the hands-on examination. If the thyroid

gland is very tender to touch, the patient may have thyroiditis. A goiter may be present that feels rubbery, is not tender, and is possibly nodular. Thyroid nodules, which are very common in older adults, are benign if they feel smooth and easy to manipulate, whereas malignant nodules are hard, irregular, fixed, and tender on palpation.

Physical examination may also reveal macroglossia (Bharaktiya et al, 2011). Bradycardia and cardiac enlargement may be detected during the cardiac examination; the diastolic blood pressure may be elevated (Mauk, 2005). Bowel sounds may be diminished. A change in reflexes may be present, notably normal upstroke with a delay in the relaxation phase. Nonpitting edema may be found in the lower extremities (pretibial myxedema). Patients should be examined for signs of carpal tunnel disease (often bilaterally) and cerebellar dysfunction to check for ataxia (Gambert et al., 2014). Myopathy may be a dominant presenting clinical sign in hypothyroidism; on physical examination, proximal weakness is noted (Madhu, Jain, Kant, Prakash, & Kumar, 2010). Signs of peripheral neuropathy may present in patients with hypothyroidism. Patients with secondary hypothyroidism may have diminished body hair and postural hypotension. A screening mental status examination should be performed.

Diagnostic Tests: Patients may be asymptomatic, with hypothyroidism discovered only during diagnostic testing. Elevation of TSH (greater than 4.0 μIU/L) and decreased free T_4 is indicative of hypothyroidism (Pearson, 2013). An elevated TSH (greater than 5.0 to 10.0 μIU/L) and a normal free T_4 occurs in subclinical hypothyroidism (Gambert et al., 2014). The presence of thyroid antibodies is useful in the diagnoses of subclinical hypothyroidism or goiter and Hashimoto's thyroiditis. If a recent CBC with indices has not been checked, evaluation for an underlying anemia is warranted (Gambert et al., 2014; Pearson, 2013). A creatine kinase level would be elevated in patients with hypothyroid myopathy, but the level should return to normal once they are treated for the hypothyroidism (Madhu et al., 2010). In patients with subclinical hypothyroidism, review the patient's last lipid levels because it is common to see an increase in LDL cholesterol levels (Fenstemacher & Winn, 2011). In one study of thyroid patients, patients with elevated TSH also had elevated triglyceride levels (Wang et al., 2010). An EKG typically shows sinus bradycardia, prolonged QT intervals, and possibly atrioventricular block and conduction disturbances (Bharaktiya el al., 2011). A thyroid ultrasound is warranted in patients with thyroid nodule(s) or goiter (Fenstemacher & Winn, 2011).

Differential Diagnosis: In determining whether or not a patient has hypothyroidism, the first thing to consider is whether the disease is primary or secondary hypothyroidism. Hashimoto's thyroiditis, post-irradiation disease, subacute thyroiditis, iodide deficiency, and subtotal thyroidectomy can cause primary hypothyroidism. People who have pituitary hyposecretion, pituitary tumors, and some infiltrative diseases (e.g., sarcoidosis) are susceptible to secondary hypothyroidism. In the older adult, when there are numerous signs and symptoms of hypothyroidism, an increased number of these clinical findings point to thyroid disease:

- Dementia
- Anemia
- Depression
- Heart failure
- Ascites
- Chronic fatigue syndrome
- Fibromyalgia
- Sleep disorder
- Chronic megacolon (Bharaktiya et al., 2011; Gambert et al., 2014).

Treatment: Older adults with an underactive thyroid are prescribed levothyroxine for lifelong treatment. The starting dose of levothyroxine in older adults is 25 mcg/day (Almandoz & Gharib, 2012). However, patients with severe coronary artery disease may need to be started on a lower dose of 12.5 mcg (Almandoz & Gharib, 2012; Chakera, Pearce, & Vaidya, 2012). For patients taking synthetic levothyroxine, it is recommended that older adults begin on 12.5 mcg or 25 mcg (Almandoz & Gharib, 2012). Patients should then be reevaluated in 4 to 6 weeks for assessment of clinical presentation and TSH level. The dose can then be adjusted upward by 12.5- to 25-mcg intervals every 4 to 6 weeks (Papaleontiou & Haymart, 2012). Usual replacement doses for older adults in the absence of cardiac disease eventually reach 100 to 125 mcg/day. The TSH level for treatment of hypothyroidism for older adults is 1.0 to 4.0 μIU/L (Papaleontiou & Haymart, 2012)

Clinical presentation of hypothyroidism in older adults often mimics normal aging changes. Patients may continue to have increased TSH levels yet show signs of clinical improvement. Excessive doses of levothyroxine induce osteoporosis. Older adults with cardiac disease should receive no more than 0.025 mg/day; evaluate blood pressure and pulse before increasing dose (Fenstemacher & Winn, 2011). A thorough medication history is necessary when prescribing levothyroxine. Cholestyramine, ferrous sulfate, sucralfate, and antacids containing aluminum hydroxide can reduce the effectiveness of this medication. Phenytoin, carbamazepine, rifampin, and anticoagulants may increase the drug metabolism. Patients taking any of these medications with levothyroxine should allow a 4- to 6-hour interval between the medications (Pearson, 2013). Dietary fiber and soy can also interfere with absorption, so levothyroxine needs to be taken on an empty stomach in the morning. Older patients who have lower serum protein levels may require reductions in their maintenance dosage over time, given the protein-bound nature of levothyroxine (Gambert et al., 2014).

Follow-Up: Patients should have a routine TSH evaluation every 6 to 12 months after stabilization. Patients with secondary hypothyroidism need a free T_4 test. Monitor clinical signs in all patients. Patients found to have subclinical hypothyroidism (TSH between 5 and 10 μIU/L) should be reevaluated every 3 to 6 months to determine if hypothyroidism is clinically indicated at this time (Jonklaas et al., 2014).

Sequelae: Patients with untreated hypothyroidism may develop coronary artery disease because of the increase in LDL and triglyceride levels associated with this disorder. Megacolon may occur in patients with a long history of untreated hypothyroidism. Myxedema coma with hypothermia and hypotension is a complication of severe untreated hypothyroidism (Bharaktiya el al., 2011; Gambert et al., 2014; Pearson 2013).

Prevention/Prophylaxis: Although the value of regular screening for thyroid disease has been debated, it is advocated for older adults with insulin-dependent diabetes, hyperlipidemia, CKD, unexplained depression, rheumatoid arthritis, other collagen-related disorders, and a family history of thyroid disease and any other condition that is known to contribute to the development of hypothyroidism (Fenstemacher & Winn, 2011).

Referral: If the patient has numerous complications or is not responding to treatment despite compliance, refer him or her to an endocrinologist.

Education: Remind patients about the potential for drug interactions that have been shown to interfere with L-thyroxine absorption, including OTC products that contain iron supplements, aluminum-containing antacids, and calcium carbonate. Tell patients not to increase their dosage even if they are experiencing symptoms of hypothyroidism; instead, they should contact their health-care provider. Any unexplained weight gain of 5 pounds or more should be reported. Encourage patients to increase their activity level. Alert patients to contact their health-care provider if they experience signs and symptoms of hypothyroidism or hyperthyroidism.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Levothyroxine is recommended as the preparation of choice for the treatment of hypothyroidism.	A	Jonklaas et al, 2014
Levothyroxine needs to be consistently taken either 60 minutes before breakfast or at bedtime (3 or more hours after the evening meal) for optimal, consistent absorption.	B	Jonklaas et al, 2014
Levothyroxine should be separated from other potentially interacting medications and nutritional supplements (e.g., calcium carbonate and ferrous sulfate).	C	Jonklaas et al., 2014

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MALNUTRITION

Signal Symptoms: Deficit in muscle mass, depleted visceral protein.

Description: Malnutrition is defined in terms of nutritional imbalance, thus patients are categorized first as having either undernutrition or overnutrition (White, Guenter, Jensen, Malone, & Schofield, 2012). Overnutrition is discussed later in the obesity section in this chapter. Although undernutrition and malnutrition are often used interchangeably and represent the same nutritional deficit, there are varying types of malnutrition depending on the initial insult. In recent years, the role of inflammation has been identified as a key component of malnutrition. Starvation-related malnutrition is a result of chronic starvation but lacks an inflammatory component (e.g., anorexia nervosa); chronic disease-related malnutrition is a result of the presence of mild to moderate chronic inflammation (e.g., organ failure or rheumatoid arthritis); and acute disease/injury-related malnutrition is attributed to acute and severe inflammation (e.g., burns, trauma, or acute systemic infection) (Guenter et al., 2015; White et al., 2012). Protein-calorie malnutrition is diagnosed by a combination of clinical factors. Generally, an unexplained involuntary weight loss and a drop in the serum albumin level to less than 3.5 g/dL may be indicative of a nutritional deficit. A decrease in serum albumin and prealbumin levels is also understood to be a reflection of the inflammatory response (White et al., 2012). Undernutrition in older adults is multifactorial: age-related organ changes; psychosocial factors such as depression, social isolation, and poverty; and functional decline pose risks for developing this often underrecognized medical condition (Ritchie, 2016). Despite the increased prevalence of malnutrition in older adults, it often remains underdiagnosed (Mauldin & O'Leary-Kelley, 2015).

Etiology: Although a number of factors can lead to malnutrition in older adults, certain age-associated factors are precursors to malnutrition, including reduced food and micronutrient intake, decreased absorption of ingested food, and the increased bodily demands for protein, calories, or micronutrients because of physiological stressors (Mueller, 2015). Decreases in metabolic rate, physical activity, and sensory input can also contribute to malnutrition, often referred to as the anorexia of aging (Soenen & Chapman, 2013). The relationship between inflammation and nutritional deficits has only recently been studied and is identified as a risk factor for malnutrition due to the metabolic stressors on the body during an inflammatory state. Inflammation in response to acute injury or illness hinders nutritional interventions in the healing process, contributing to the development or worsening of malnutrition (White et al., 2012).

Occurrence: More than 6 million older adults are at high risk for malnutrition. Reports indicate that approximately 16%

of community-dwelling elderly, 30% to 65% of acute care hospital patients, and 25% to 60% of nursing home residents experience protein-energy malnutrition (John, Bullock, Brenner, McGraw, & Scolapio, 2013).

Age: Malnutrition can occur at any age. Frail older adults and people more than 80 years old are at the highest risk for malnutrition.

Gender: Malnutrition in people 85 years old and older is more prevalent in women because of the higher number of women than men in this age bracket.

Ethnicity: Because the reasons for malnutrition varies, no specific ethnic prevalence is known.

Contributing Factors: Any of several factors may contribute to malnutrition in older adults: decreased olfactory sensitivity; loss of taste buds; being edentulous; gastroesophageal reflux disease; dysphagia; poor dietary habits; certain chronic diseases with or without an inflammatory component such as intestinal ischemia, hyperthyroidism, depression, chronic pain, COPD, stroke, alcoholism, constipation, chronic impaction, malabsorption syndromes, dementia, obesity, and cholelithiasis (Soenen & Chapman, 2013). One study confirmed that patients requiring walking aids and patients with urinary incontinence, depression, and lower educational preparation were found to be malnourished compared with patients who had higher scores on the MNA scale (vanBokhorst-de van der Schueren, 2013).

Review medications because a number of medications can decrease appetite (digoxin), alter taste and smell (antibiotics, antihistamines, antifungals, chemotherapeutic agents), decrease salivary gland production contributing to dry mouth (anticholinergic agents), and cause malabsorption of nutrients (colchicine, neomycin, methotrexate, methyldopa, allopurinol) (Flaherty & Resnick, 2014). Patients who are less educated exhibit more depressive symptoms (Wu, Courtney, & Isenring, 2015) and have lower cognitive and functional status are at risk to be malnourished. Social isolation and loneliness are additional risk factors for malnutrition among older adults (Boulos, Salameh, & Barberger-Gateau, 2016). Older adults who have difficulty with chewing and frequent episodes of nausea and vomiting are at risk for malnutrition. Limitations in the ability to not only prepare food but also to eat, due to tremors and arthritic upper extremity conditions, can lead to malnutrition. Hospitalization with a restricted diet, drug-nutrient interactions related to polypharmacy, social isolation, and ultimately, poverty, also may contribute to malnutrition (Moreira, 2016).

Signs and Symptoms: Identification of elders at risk for malnutrition is a complex process, however, unexplained weight loss is identified as the best factor for predicting increased risk of malnutrition. Assessment of malnutrition is complex because it involves investigation of physiological, psychological, pathological, functional, and financial parameters to determine the possible causes of the malnutrition. Comprehensive assessment of the following diagnostic characteristics is essential in identifying at-risk and presently malnourished patients: history and clinical diagnoses, physical examination/clinical signs, anthropometric data, laboratory data, food/nutrient intake, and functional assessment (White et al., 2012). In patients with suspected malnutrition, all risk factors for poor nutritional status should be assessed using objective measures as indicated (i.e., depression scales, mental status scales, functional status tests) (Flaherty & Resnick, 2014). Several nutritional screening tools have been developed and are available for use; however, the MNA is the most commonly used and has been validated as a nutritional screening device for the elderly (Ritchie, 2016; Sarikaya et al., 2016)

A thorough review of prescribed and OTC medications is indicated in order to determine if the patient is taking any substance that can cause anorexia such as digoxin, quinidine, hydralazine, amiodarone, levodopa, fluoxetine, lithium, colchicine, NSAIDs, proton pump inhibitors, and many of the chemotherapeutic agents (Flaherty & Resnick, 2014). During the physical examination, determine if the malnutrition is related to cardiac, respiratory, intestinal, endocrine, hepatic, neurological, or renal impairment. Look for specific signs of nutritional deficiencies such as nail abnormalities, brittle hair, bruises, skin color (jaundice, pallor), cheilosis, glossitis, loss of subcutaneous body fat, muscle wasting, and edema. Explore any unexplained weight loss to rule out treatable causes of malnutrition. If a patient is found to be 15% or more below ideal body weight or had a recent loss of 10% under baseline weight, consider protein-calorie malnutrition (although dehydration should be ruled out).

BMI is calculated using the formula weight in kilograms divided by height in meters squared. BMI is an effective measurement for obesity, but is generally not as sensitive in indicating malnutrition (John et al., 2013). In the normal healthy older adult, the BMI should range from 18.5 to 24.9 kg/m^2; BMI less than 18.5 kg/m^2 generally indicates a weight deficit and is an indication that nutritional intervention is required. In older adults, mid–upper arm circumference (MUAC) and triceps skinfold (TSF) measurement is not an accurate means for testing for malnutrition; however, as an estimate, an MUAC or TSF measurement below the tenth percentile indicates poor nutritional status (less than 22 cm in women and less than 23 cm in men) (Ritchie, 2016). Hand grip strength should also be assessed over time; a decrease in strength can indicate nutritional deficits related to the loss of muscle function. Clinical signs of inflammation such as fever, hypothermia, or tachycardia should also be considered when evaluating the patient at risk for malnutrition (White et al., 2012).

Diagnostic Tests: There is no gold standard test for diagnosis of malnutrition. Diagnostic studies help identify the severity and define the type of malnutrition. In screening for malnutrition, laboratory values should be considered only as an indirect measurement or as a tool to evaluate treatment. A CBC should be ordered to determine anemia and rule out infection and immunocompromised status. Inflammatory markers such as leukocytosis, elevated blood glucose, or C-reactive protein level may be indicators of a nutritional deficit; however, they are not yet considered diagnostically specific for malnutrition (White et al., 2012). Total lymphocyte counts less than 1,500 cells/mm^3 are found in mild-to-moderate malnutrition; a total of less than 1,000 cells/mm^3 is associated with immune paralysis. Protein, iron, folate, and/or vitamin B_{12} deficiencies should be assessed for to further identify deficiencies.

A serum albumin level less than 3.5 mg/dL and especially less than 3.0 mg/dL predicts protein depletion and in the past has been the most common biochemical parameter indicated

when considering malnutrition (John et al., 2013). However, serum albumin levels can be skewed in patients experiencing urinary loss from nephrotic syndrome or in those receiving IV fluids. A dilutional effect on albumin is also seen when the patient is bedridden, which can produce up to a 0.5 mg/dL decrease. Because the half-life of albumin is 21 days, serum albumin levels rise slowly following nutritional supplementation. Prealbumin with an average life span of 2 to 3 days is highly dependent on iron levels and is not indicated as an evaluation tool unless the individual is hospitalized. A decrease in the serum transferrin levels also points to malnutrition. In patients with coexisting iron-deficiency anemia, however, the results will be misleading (normal to elevated serum transferrin). If the elderly individual is not on a lipid-lowering agent, a TC level less than 160 mg/dL is also considered a marker for malnutrition and has been associated with increased mortality (Flaherty & Resnick, 2014).

Differential Diagnosis: When determining if a person is suffering from malnutrition, discern if any of the following conditions coexist:

- Anorexia
- Dehydration
- Dementia—alcohol related
- Dementia—Alzheimer's related
- Depression—major
- Dysphagia
- Eating disorder
- Failure to thrive
- Feeding problem/older adult
- Nutritional deficiency not otherwise specified (NOS)

Treatment: Identifying the contributing factors to malnutrition in each individual patient is essential to treating this condition. When possible, a 3-day diet intake should be obtained to determine the severity of the nutritional deprivation. All medications that can cause drug-induced malnutrition should be discontinued or reconsidered or an alternative should be used (Flaherty & Resnick, 2014). Any fecal impaction should be identified and removed. Because not all factors, such as alterations in sensory input, can be treated, dietary consultation should be ordered to assist in the diagnosis of malnutrition and planning of diet supplementation. Any older person experiencing a physiological stressor such as surgery, infection, or trauma requires an increase in protein, calories, and micronutrients. If the patient is diagnosed with protein-energy malnutrition (PEM), nutritional support will depend on the patient's medical condition and the degree of the PEM. Enteral nutritional support is considered when the patient's nutritional intake is inadequate to meet physiological requirements for more than 7 days (John et al., 2013) or when the weight loss is more than 10% of the patient's pre-illness weight. A recent study by Lee et al. (2013) found that a soy-based nutritional supplement provided to patients with MNA scores of 24 or less and BMI scores of 24 kg/m^2 or less resulted in significant improvements in body weight, BMI, and clinical markers of malnutrition, serum albumin, and cholesterol (Lee et al., 2013).

Follow-Up: Patients who are hospitalized for malnutrition or who develop malnutrition secondary to an acute-care hospitalization should be monitored daily for response to the nutritional supplementation (Deutz et al., 2016). The patient's weight should be monitored weekly until the malnutrition has been corrected. If a psychosocial factor contributed to the malnutrition, periodic review of the patient's improvement is warranted.

Sequelae: An older person's initial reaction to prescribed nutrient supplements may worsen malnutrition when used as a meal replacement instead of supplementation (John et al., 2013). Patients who are malnourished can develop sarcopenia and/or cachexia. Sarcopenia, defined as the progressive and generalized loss of skeletal muscle mass and function (Eglseer, Eminovic, & Lohrmann, 2016), has been associated with inadequate intake of protein from a variety of sources. Cachexia is defined as "a complex metabolic syndrome associated with underlying illness and characterized by loss of muscle with or without loss of fat mass . . . clinical feature of cachexia is weight loss" (Baylis et al., 2014). Long-term malnutrition increases the risk of morbidity and mortality. Careful assessment is necessary to determine if a patient is suffering from FTT (Kumeliauskas, Fruetel, & Holroyd-Leduc, 2013).

Prevention/Prophylaxis: Given the number of older adults who are at risk for malnutrition, the health-care provider must periodically screen patients for nutritional deficits. A two-step approach for identifying malnutrition is suggested using the MNA. A shortened version of the MNA (MNA-SF) has been studied and approved as an initial screening tool when undernutrition is suspected in the geriatric population. The MNA-SF is highly correlated with the MNA, with a diagnostic accuracy of 98.7% for predicting undernutrition. The six-item MNA-SF can be easily used in the first step of the process because it does not require anthropometric testing. For the second level of screening, the MNA is considered the most reliable and validated nutritional tool. It is appropriate for use in the outpatient and nursing home settings. It does not require invasive laboratory testing and is cost effective. The tool consists of an anthropometric assessment, general assessment, dietary assessment, and a self-assessment that engages the older adult person to assess his or her nutritional status. International studies have validated that an MNA score between 17 and 23.5 indicates risk of malnutrition and intervention is indicated. Soderstrom and colleagues (2013) found that the MNA is a useful tool for screening for nutritional status and predicting preterm death in older adults. The MNA-SF contains six questions; a score between 8 and 11 points indicates risk for malnutrition (Marshall, Young, Baeur, & Isenring, 2016).

Referral: Referring patients who are malnourished to a specialist depends on the identifiable cause of the nutrition depletion. A dietitian should be consulted for a nutritional support evaluation. Refer patients who need long-term enteral support to a gastroenterologist for consideration of a percutaneous endoscopic gastrostomy or, in some cases, a feeding jejunostomy. Arrangements can be made for the community-dwelling older adult to receive Meals on Wheels (Rubin et al., 2014) or attend a Title III meal site. Provide information on the location of centers for congregate meals sites. Patients with swallowing difficulties need to be referred to a speech therapist, and those with difficulty with food preparation or eating can be referred to an occupational therapist for evaluation for adaptive equipment and retraining. For the patient

diagnosed with a terminal illness, discuss the patient's and family's decision on nutritional support before beginning any intervention. If social isolation, low income, and/or functional status contributed to the development of malnutrition, the patient should be referred for social services and/or discharge planning. Patients with financial needs should be evaluated to see if they can qualify for food assistance and could be enrolled in the Supplemental Nutrition Assistance Program (SNAP) (Pomeranz, 2017) and/or participate in community-based food bank programs.

Education: For the alert ambulatory older adult, review the Dietary Guidelines for Americans and My Plate guidelines (www.choosemyplate.gov/dietary-guidelines.html). Teach family members caring for cognitively impaired or physically disabled older adults about dietary requirements and nutritional supplementation. Caregivers and older adult persons can improve nutritional status by simple measures, such as preparation of an adequate diet, hand feeding, adequate-fitting dentures and oral hygiene, and adding liquid nutritional supplements between meals.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The MNA tool has been used successfully globally to assess for malnutrition risk in older adults.	B	Marshall et al., 2016 Sarikaya et al., 2015
Malnutrition as measured by the MNA was found to be a predictor of preterm death in older adults.	B	Soderstrom et al., 2013
Patients admitted to the hospital for malnutrition should be monitored daily for their response to nutritional supplements.	A	Deutz et al., 2016
All medications that can cause drug-induced malnutrition should be discontinued or reconsidered, or an alternative should be used.	C	Flaherty & Resnick, 2014

OBESITY

Signal Symptoms: Elevated BMI, central obesity, generalized obesity.

Description: Obesity can be defined as the acquisition of enough body fat such that there is a negative effect on health. There are a variety of methods to determine if an individual is obese. Simply weighing a patient does not distinguish the effect of muscle versus body fat on weight; that is, a very muscular patient may weigh the same as a very obese patient. Also, taller patients will have a higher normal weight than shorter patients. The use of height to weight tables may be problematic in that there are many different tables in use with many different acceptable weight ranges, such as those for individuals with Asian origins (Hsu, Araneta, Kanaya, Chiang, & Fujimoto, 2015).

Although none of these methods distinguishes differences in gender, ethnicity, or the effects of fat versus muscle, the most clinically practical and accurate method is the measurement of the BMI, shown in Box 14-1 (CDC, 2015a; Tsai & Wadden, 2013). This latter point is important to recognize, as recent research has emphasized that body composition should be considered when assessing an individual's health and risk of death; a higher percentage of body fat has been independently associated with reduced survival (Padwal, Leslie, Lix, & Majumdar, 2016). Standardized nomograms or online calculators easily determine BMI; obesity can be further classified into progressive grades that reflect increased risk of developing disease.

In addition, the distribution of body fat is important as well. Central obesity, in which there is an increase in abdominal fat, appears to be a risk factor in the development of diabetes mellitus, hypertension, and the metabolic syndrome. A female patient with a waist measurement of more than 88 cm (35 inches) or a male patient with a waist measurement of more than 102 cm (40 inches) is defined as having central obesity. The National Heart, Lung, and Blood Institute

BOX 14-1
Body Mass Index

BMI = (Weight in pounds/height in inches squared) × 703

Obesity is defined as a BMI >30 with morbid obesity as a BMI >40. Overweight is defined as a BMI of 25 to 29. The CDC provides a BMI calculator on their Healthy Weight Web site (CDC, 2015b).

BMI	DEFINITION
<18.5	Underweight
18.5–24.9	Normal
25.0–29.9	Overweight
30.0–34.9	Class I obesity
35.0–39.9	Class II obesity
>40.0	Class III extreme obesity

(NHLBI, 2016) recommends waist circumference as a comparable measurement of central obesity. Taken with the subject standing, waist circumference is measured from the uppermost lateral border of the iliac crest. Waist circumference cut points can be standardized across all adult ethnic populations except for individuals less than 5 feet tall or individuals with a BMI more than 35. The NHLBI (2016) recommends that waist circumference in these cases be adjusted by age and BMI.

Etiology: Obesity occurs when an individual consumes more calories than he or she expends. The etiology of obesity is genetic as well as environmental, however, it is difficult to separate the influence of genetics versus the influence of similar lifestyle habits that individuals and families share. Certainly, environmental, social, and cultural influences also determine the risk of obesity. Obesity tends to occur more often when individuals eat high-calorie, high-fat, processed foods or are unable to participate in enough physical activity or exercise. Some medications may cause weight gain by stimulating appetite, affecting how the body absorbs and stores glucose, slows overall metabolism, and/or causes fatigue (i.e., making physical activity challenging). Examples of these types of medications include insulin, thiazolidinediones, sulfonylureas, tricyclic antidepressants, selective serotonin reuptake inhibitors, lithium, antipsychotics, antiseizure, oral and inhaled corticosteroids, estrogen/progestogen hormone therapy, antihistamines and beta-adrenergic blockers (Kyle & Kuehl, n.d.). Often, weight gain is seen as patients quit smoking. In addition, a lack of sleep has been associated with the development of obesity (Beccuti & Pannanin, 2011).

Occurrence: Obesity rates in the United States are on an upward trajectory. Currently, more than two-thirds of Americans are overweight (defined as BMI between 25 and 30 kg/m^2) or obese (defined as BMI greater than 30 kg/m^2) (Hruby & Hu, 2015). Recent statistical models estimate that by 2030, 42% of adults will be obese, and 11% will be severely obese (Finkelstein et al., 2012). According to the CDC (2015a), obesity occurs in approximately 33.8% of the older adult population in the United States, with the South having the highest prevalence at 29.4%.

Age: According to CDC (2015a), between 2011 and 2014, the prevalence of obesity was just over 36% in adults, with rates higher among women (38.3%) compared to men (34.4%). Obesity was higher among middle-aged adults ages 40 to 59 years (40.2%) and older adults age 60 years and older (37.0%) than younger adults ages 20 to 39 years (32.3%).

Gender: The overall rate for obesity is similar in men and women, but the rate of obesity in women ages 65 to 74 years is higher than in women ages 75 years and older in all racial and ethnic groups, except for those indicating non-Hispanic African American (Fakhouri, Ogden, Carroll, Kit, & Flegal, 2012).

Ethnicity: Obesity affects some groups more than others. According to the CDC (2015a), non-Hispanic African Americans have the highest age-adjusted rates of obesity (47.8%), followed by Hispanics (42.5%), non-Hispanic Caucasians (32.6%), and non-Hispanic Asians (10.8%).

Contributing Factors: Many factors contribute to the development of obesity. These factors include genetic makeup, environmental and social factors, cultural factors, psychological factors, and certain medical conditions. Environmental factors that result in a lack of exercise or the increase in the use of a high-fat, high-calorie diet will result in the increased chance of obesity. Psychological influences such as depression, anger, or boredom may result in an unhealthy eating response, such as binge eating, which contributes to an increase in caloric consumption (Toups et al., 2013; *Medical News Today,* 2011).

Signs and Symptoms: Usually, obesity can be recognized by the initial physical examination. A height and weight measurement should be obtained when medically necessary at each visit to the care provider. The use of any number of standardized height-to-weight tables can distinguish the normal-weight patient from the overweight or obese patient. The use of the BMI is the most practical and accurate method to quantify obesity in the office setting. Measurement of waist circumference can predict the development of the metabolic syndrome. Skin calipers can be used to measure subcutaneous fat thickness by "pinching" and measuring skin fold thickness. Unfortunately, this assessment technique requires training and strict adherence to measurement protocols that may be impractical to use in a clinical setting.

Associated symptoms that older adults with obesity often present with include feelings of worthlessness, sadness, and hopelessness. Obese older adults should be questioned on associated functional limitations, joint pain, decreased mobility, and activity intolerance (Starr & Bales, 2015). Evidence of loss of muscle strength due to sarcopenia in sedentary older obese adults should be assessed and functional ability to perform ADLs should be measured (Starr, McDonald & Bales, 2014). Skin conditions prevalent in obese older adults include perspiration and friction. Assess for areas of skin excoriation and pressure ulcers (Hyun et al., 2014; Yap & Kennerly, 2011).

Diagnostic Tests: Lipid profile and fasting glucose levels should be obtained in all obese patients. If hypothyroidism is suspected, a TSH level should be ordered. If Cushing's syndrome is suspected, practitioners should order one of the following: a late-night salivary cortisol level, a 24-hour urine free cortisol, or an overnight low-dose dexamethasone suppression test.

Differential Diagnosis: Determine the ideal body weight (IBW) for height. Patients 20% above are considered obese; those 40% above are morbidly obese.

Treatment: In June 2013, the American Medical Association adopted a policy that recognized obesity as a disease requiring a range of medical interventions to advance obesity treatment and prevention. This policy comes after decades of various weight-loss-targeted (or bariatric) surgeries with variable outcomes and health insurance coverage. Obesity management includes a comprehensive nutritional evaluation and a complete history and physical examination. Important considerations include social determinants of health, dietary and physical activity patterns, and medication review for medications that may promote weight gain. There are many comorbidities associated with obesity, including but not limited to diabetes, hypertension, obstructive sleep apnea, and osteoarthritis (Medscape, 2016). The basic treatment for obesity involves a reduction of caloric intake while increasing

physical activity. The goal of a standard weight loss regimen should be about 1 to 2 pounds per week; however, it is also important to have a clear understanding of a patient's expectations and degree of motivation (CDC, 2015a, 2015b; Hamdy, Citkowitz, Uwaifo, & Oral, 2011).

The initial diet should aim at reducing the caloric intake for the female patient to 1,200 to 1,500 calories per day and the male patient to 1,500 to 1,800 calories per day. This goal of caloric reduction can often be obtained by decreasing portion size and limiting high-calorie but poorly nutritious foods. Keeping a daily food diary and counting calories will often help patients to see how much they really consume each day. Other commercial programs such as Weight Watchers, Jenny Craig, or Take Off Pounds Sensibly involve prepackaged meals or meal replacement entities with a calorie count that has been predetermined. Very low calorie diets that reduce the caloric intake to less than 800 to 1,200 calories per day require strict medical monitoring through a structured program. There are few data to support the use of low-carbohydrate diets such as Atkins or South Beach over the typical balanced but calorie-restricted diet in terms of safety or efficacy (CDC, 2015a, 2015b; Hamdy et al., 2016). Starr & Bales (2015) recommend a combined approach of a weight-reduction diet plus a program of exercise. In patients with sarcopenic obesity, an increase in dietary protein and protein supplements should be encouraged, unless contraindicated by other concomitant conditions that restrict protein intake (Starr & Bales, 2015; Benton et al., 2011). Weight loss therapy in an older patient with sarcopenic obesity should be tailored to minimize loss to bone and muscle mass (Mathus-Vliegen et al., 2012).

Increased activity and exercise are important strategies to increase caloric expenditure. Patients should undergo cardiovascular and pulmonary evaluation for safety before starting an exercise program. Han and colleagues (2011) recommend modest calorie restriction combined with exercise for patients with sarcopenic obesity. Per the CDC (2015c), older adults need at least 2 hours and 30 minutes (150 minutes) of moderate-intensity aerobic activity (i.e., brisk walking) every week and muscle-strengthening activities on 2 or more days a week that work all major muscle groups (i.e., legs, hips, back, abdomen, chest, shoulders, and arms) (CDC, 2015c; Hamdy et al., 2016). For patients with sarcopenic obesity, resistance training is highly encouraged to protect muscle and bone while undergoing intentional weight loss (Starr & Bales, 2015; Benton et al., 2011).

Currently, there are three major groups of drugs used to manage obesity. Drugs work either centrally to limit dietary intake, peripherally to impair dietary absorption, or increase energy expenditure. The only medications approved by the FDA for chronic weight management are Xenical (orlistat), Belviq (lorcaserin hydrochloride), Qsymia (phentermine and topiramate extended-release topiramate), Saxenda (liraglutide injection), and Contrave (naltrexone hydrochloride and bupropion hydrochloride) (FDA, 2016). Prescribers of these medications should carefully review prescribing information to avoid medication-related side effects and drug–drug interactions. Other pharmacological medications include appetite suppressants/anorexiants (noradrenergic/sympathomimetic agents) and botanicals (herbals), which may be contraindicated among older adults with multimorbidities (Balkon, Balkon, & Zitkus, 2011).

Follow-Up: The treatment of obesity should focus on the short-term weight loss phase and the long-term maintenance phase. Weight loss has been associated with reduced risk of developing type 2 diabetes, improvement in lipid profile, and reduced blood pressure (Jensen et al., 2014). Patients should be followed closely as the diet and exercise program is begun. As patients lose weight, there should be close monitoring of potentially harmful side effects of weight loss, including cardiac arrhythmias, exacerbation of gout, electrolyte abnormalities such as hypokalemia, development of gallstones (cholelithiasis), depression, or eating disorders such as binge eating or induced vomiting. The goal is to promote the long-term management of weight with a healthy lifestyle (Hamdy et al., 2016).

Sequelae: Obesity is considered a risk factor for the development of a number of illnesses or diseases. In a study with nearly 2 million individuals worldwide, being overweight or obese explained almost 50% of cardiovascular outcomes (i.e., coronary heart disease, stroke) and contributions of blood pressure, dyslipidemia, and glucose concentration (Global Burden of Metabolic Risk Factors for Chronic Diseases Collaboration, 2014). The obese patient is more likely to develop coronary artery disease, hypertension, and hyperlipidemia. There is an increased risk of developing type 2 diabetes mellitus, cerebrovascular disease, and CKD (Chung, Kang, Lee, Lee, & Lee, 2012; Gabbay, Slotki, & Shavit, 2015). Additionally, in obese older adults, there is an increased risk for physical disability, sexual dysfunction, lower urinary tract symptoms, and impaired cognitive function and dementia (Semins, Shore, Makary, Weiner, & Matlaga, 2012; Hans, Tajar, & Lean, 2011). Certain types of cancer such as colon, breast, endometrium, liver, kidney, esophagus, gastric, pancreatic, gallbladder, and leukemia are also associated with obesity (Vucenik & Stains, 2012). The obese patient is more likely to develop obstructive sleep apnea, gallbladder disease, fatty liver disease, and osteoarthritis. Often, the obese patient will have symptomatic varicose veins or gastroesophageal reflux. In older adults who experience the loss of muscle mass combined with the increase in body fat, the loss of muscle strength contributes to reduction in function and overall quality of life (Starr & Bales, 2015; Benton et al., 2011).

Prevention/Prophylaxis: Prevention of obesity must start in childhood with the promotion of a nutritionally balanced diet and proper exercise. Weight should be monitored when medically needed at each visit to the primary care provider and education provided to support a healthy lifestyle with proper exercise (CDC, 2015b; U.S. Department of Health and Human Services, National Institutes of Health [NIDDK NIH], 2011). Medicare patients are now eligible to receive counseling services for obesity in patients with a BMI over 30 (DiSantostefano, 2012).

Referral: The management of obesity is challenging. Patients who fail the typical diets can often be referred to more structured programs such as Weight Watchers, Jenny Craig, NutriSystem, and many others. Medically supervised, structured diets that use meal replacements or energy restriction (i.e., low or very low energy) can be effective not only in weight loss but also in improvement in comorbid conditions, depending on the intensity and duration of the intervention

(Anderson & Luan, 2004; MacLaughlin et al., 2012). Referral to an endocrinologist is appropriate when dealing with such illnesses as hypothyroidism, Cushing's disease, and a history of polycystic ovary syndrome (PCOS). Finally, referral to a bariatric surgeon may be considered for individuals who have a BMI of 40 or more or are more than 100 pounds overweight; have a BMI of 35 or more and at least two obesity-related comorbidities (i.e., type 2 diabetes, hypertension, sleep apnea, nonalcoholic fatty liver disease, hyperlipidemia, osteoarthritis, or heart disease); or are unable to achieve a healthy weight loss for a period of time with prior weight loss efforts (American Society for Metabolic and Bariatric Surgery, 2016). The goal of bariatric operations is either restriction of food intake, restriction of food absorption, or both. The most common types of bariatric surgeries performed include the laparoscopic adjustable gastric band (LAGB), the Roux-en-Y gastric bypass, and the gastric sleeve. Emerging evidence suggests that bariatric surgery may establish a new body-weight set point by altering the physiological mechanisms of body-weight regulation, thereby causing sustained weight loss.

Education: Teach patients and significant others meal preparation, calorie counts, and fat gram calculation. There are commercial weight loss programs that are available for individuals who prefer individualized, group, or online methods to assist with weight loss. They range in costs so that everyone should be able to find a weight loss program that suits their individual needs. To sustain the clinical benefits of weight loss (i.e., improved insulin sensitivity), exercise training needs to be added to the weight loss intervention (Bouchonville et al., 2013). Older adults are encouraged to include resistance, flexibility, and strength training in their aerobic exercise programs (Bocalini et al., 2012).

It is important to encourage patients to begin a safe exercise regimen like walking, which is the most recommended exercise program (CDC, 2015b; NIDDK NIH, 2011; WebMD, 2011). One study found that older women benefit from participation in exercise programs; however, the participants in the study experienced some musculoskeletal injuries while exercising (Rossen, Milsom, Middleton, Daniels, & Perri, 2013). Providers should provide the older adult with numerous safe options for exercising. Older adults need to be educated on the importance of including micronutrients, including vitamins D and B_{12}, as well as dietary fiber, in their diet.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Caloric restriction combined with exercise.	A	CDC, 2015a, 2015b Hamdy et al., 2016 Laddu et al., 2011
High-protein, low-carbohydrate diets are more effective at weight loss than low-fat diets.	A	CDC, 2015a, 2015b Hamdy et al., 2016 Laddu et al., 2011
Mediterranean diets consisting of increased fruits and vegetables are more effective at weight loss than low-fat diets.	A	Laddu et al., 2011
Evidence suggests that commercial weight loss programs that require attendance are more effective in weight loss than the usual do-it-yourself programs.	A	CDC, 2015a, 2015b Hamdy et al., 2016 Laddu et al., 2011
Evidence suggests that Internet weight loss programs and mobile health apps combined with personalized feedback were more effective in weight loss than Internet programs that did not provide personal feedback.	A	Bakken et al., 2014 Laddu et al., 2011
Current physical activity recommendations call for approximately 30 minutes of moderate activity 5–7 days per week; for weight loss without calorie restriction, 1 hour of moderate activity is required. More than 300 minutes a week of moderate-intensity activity, or 150 minutes a week of vigorous-intensity activity, results in greater health benefits.	A	CDC, 2015c Hamdy et al., 2016 Laddu et al., 2011
Regular exercise is the primary predictor of maintenance of weight after weight loss.	A	Laddu et al., 2011 Tsai & Wadden, 2013

Continued

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Individuals receiving standard behavioral weight loss treatment (SBT) and SBT plus individual self-efficacy enhancement sessions both achieved clinically significant weight loss.	A	Burke et al., 2015
Those receiving motivational interviewing lost more weight and had less attrition than those receiving guided self-help without motivational counselling.	A	Laddu et al., 2011 Burke et al., 2015
Weight reduction supplements usually resulted in repeated, unsuccessful attempts at weight loss.	A	CDC, 2015a, 2015b Hamdy et al., 2016 Laddu et al., 2011
FDA-approved medications for chronic weight management work best with other behavior modification techniques. If deemed ineffective (weight loss less than 5% at 3 months) or there are safety or tolerability issues at any time, it is recommended to discontinue the medication, consider an alternative medication, or consider an alternative treatment approach.	A	CDC, 2015a, 2015b Hamdy et al., 2016 Laddu et al., 2011 FDA, 2016
Bariatric surgery is the only available therapeutic modality associated with clinically significant and relatively sustained weight loss. Roux-en-y-gastric bypass (RYGB), most often used in the United States, causes 50%–80% loss of excess weight and improves glucose control outside of normal weight loss. May be used to normalize glucose in non–insulin-dependent diabetics.	A	Hamdy et al., 2016 Laddu et al., 2011
Individuals who achieve weight loss of 2%–5% are more likely to have clinically meaningful improvement in health outcomes (i.e., reduced risk of developing type 2 diabetes, improvement in lipid profile, and reduced blood pressure).	A	Jensen et al., 2014

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

OSTEOPOROSIS

Signal Symptoms: Kyphosis, decreased height, vertebral fractures, severe back pain.

Description: Osteoporosis is the most common bone disease in humans, representing a major public health problem. The National Osteoporosis Foundation (NOF, 2014) defines osteoporosis as a metabolic skeletal disease characterized by low bone mass, deterioration of bone tissue, disruption of bone microarchitecture, and compromised bone strength with a consequent increase in bone fragility and susceptibility to fracture. The WHO defines osteoporosis as a bone mineral density (BMD) less than 2.5 standard deviations (SDs) or more below the mean of the young adult peak bone mass (20-year-old healthy, T-score of less than −2.5). When bone density is between 1.0 and 2.5 SD below the mean, the patient is said to have low bone mass or osteopenia (T-score less than −1 and greater than −2.5). A T-score greater than −1 is normal BMD. The T-score is an important predictor of fracture risk, with the highest risk in those with the lowest BMD (NOF, 2014). Osteopenia confers a moderate risk for fractures and osteoporosis a high risk for fractures. However, most fractures occur in people with osteopenia because of the larger number of individuals with bone mass in this range (NOF, 2014). Although osteoporosis has been called the silent disease because fractures, occurring late in the disease process, are often the first symptoms, an increase in screening has resulted in earlier diagnosis and treatment to prevent or minimize fractures. Even after the first fracture has occurred, there are effective treatments to reduce the risk of additional fractures.

Etiology: BMD results from both the peak bone mass achieved by ages 18 to 25 years and the amount of bone loss after maturity. Although osteoporosis is considered a disease of older adults, the origins of the disease begin in adolescence when there is a failure to achieve a high quality of bone.

Peak bone mass is determined by genetic factors, with contributions from nutrition, endocrine status, physical activity, and health during growth. Healthy bone is maintained by the bone remodeling unit's (basic multicellular unit [BMU]) removal of old bone and replacement with new bone. This action repairs microdamage to maintain strength and maintains serum calcium. Osteocytes, osteoblasts, and osteoclasts make up the BMU. Osteocytes make up 95% of the total bone cell number and trigger the remodeling sequence. Osteoblasts are the bone-forming units, and osteoclasts are responsible for bone resorption. Recently, RANK ligand (RANKL) has been identified as the cytokine responsible for the components of the BMU. This has resulted in the development of new treatment options. Remodeling is regulated by hormones, including estrogen, androgen, vitamin D, parathyroid hormone, locally produced growth factors, and immunoreactive growth hormone. Estrogen loss effects trabecular bone in preference to cortical bone. The loss of estrogen increases RANKL. Vitamin D plays a crucial role in calcium homeostasis. It acts on the kidney to decrease calcium and phosphorus excretion. Vitamin D works with parathyroid hormone to regulate the release of calcium from the bone. Approximately 99% of calcium is found in the skeleton. Calcium absorption is affected by age, as well as by a decline in estrogen. When calcium is low it results in an increase in parathyroid hormonal activity, which increases bone remodeling to maintain serum calcium levels. Secondary hyperparathyroidism results from low levels of either calcium or vitamin D.

Osteoporosis results from an imbalance between bone resorption and bone formation, causing a reduction in bone tissue. Fractures occur because of both the qualitative and quantitative character of bone. The quality of bone is not measurable by current screening techniques; it encompasses the microstructure and macrostructure, biochemical composition, distribution and integrity of material components within the bone, turnover, and microdamage accumulation. The importance of bone quality explains the fact that a 50-year-old woman with the same measured BMD as an 80-year-old woman has a much lower risk of fracture (Kolata, 2003). That BMD is not the only factor is also illustrated in the statistic that more than one-half of hip fractures occur in patients with BMD above 2.5 SD below the mean (the WHO definition of osteoporosis). Primary osteoporosis is a decrease in BMD unrelated to other diseases. It occurs with aging in both men and women, as well as with the decline in hormones after menopause in women. Secondary osteoporosis may result from the use of systemic steroids, phenytoin, and diseases that cause malabsorption of calcium and vitamin D, chronic diseases such as rheumatoid arthritis, hyperparathyroidism, hyperthyroidism, overmedication of hypothyroidism, and celiac disease. Approximately 15% of those with celiac disease have osteoporosis as their only clinical sign (Stenson, Newberry, Lorenz, Baldus, & Civitelli, 2005).

Occurrence: In the United States, more than 10 million people have osteoporosis, with an additional 43.1 million experiencing low bone mass (NOF, 2014). Of the 10 million, 8 million are female and 2 million are male. The NOF (2014) estimates that 50% of women and 20% of men will suffer an osteoporosis-related fracture in their lifetime, costing an estimated $17 billion in direct expenditures. Each year in the United States, there are about 2 million osteoporotic fractures, approximately 300,000 hip fractures, close to 1 million vertebral fractures, and 200,000 wrist fractures. The occurrence of fractures has an interesting distribution with Colles (wrist) fractures peaking at age 60 years, while the risk for hip fracture doubles every 5 years after age 70 years. A 50-year-old woman has a risk of death from a hip fracture that is equal to her risk of death from breast cancer (Strom et al., 2011). Although the statistics on fractures are sobering, they do not define the whole problem. The NOF notes that in North America it is likely that 45% of vertebral fractures go unnoticed.

Age: Age is as great a risk factor for osteoporosis as low BMD. Osteoporosis is due to age-related changes, as well as extrinsic and intrinsic factors. Osteoporosis is estimated to affect 200 million women worldwide, approximately one-tenth of women age 60 years, one-fifth of women age 70 years, two-fifths of women age 80 years, and two-thirds of women age 90 years (Strom et al., 2011). With the increasing number of older adults in the United States, the prevalence of osteoporosis is also expected to increase. When an adult more than 50 years old suffers any fracture, the possibility of the diagnosis of osteoporosis should be considered.

Gender: Women have a lower peak bone mass, an increased rate of bone loss after menopause, and an increased risk of falling. In the United States, the risk of fracture in women is twice that in men, but the mortality rate after a fracture is higher in men. This disparity between women and men seen regarding fracture risk in the United States is not the same in cultures where both sexes primarily engage in manual labor. Incidence of hip fracture increases in both men and women with age, but in men the incidence occurs 5 to 10 years later than in women. The incidences of osteoporotic fractures resulting from secondary osteoporosis differ in men and women, causing few of the fractures that occur in women and more than 50% of vertebral fractures in men.

Ethnicity: Peak bone mass and bone density are influenced by diet and lifestyle, but primarily by genetics. From 3.9% of Caucasian women 50 to 59 years old to 47.5% of those older than 80 years suffer from osteoporosis (WHO, 2003). The risk for osteoporosis is twice as great in Caucasian women compared with African American women. Asian American women are also at a high risk, and about 10% of Hispanic women have osteoporosis, while 49% have osteopenia. Caucasian men are at greater risk for developing osteoporosis than African American men. Although African Americans have a lower rate of osteoporosis, their risk of fracture is equal to their Caucasian counterparts when they have a decreased BMD, and African American women are more likely than Caucasian women to die after a hip fracture.

Contributing Factors: Many risk factors predispose individuals to osteoporosis. Some of these risk factors cannot be changed, including age, family history of the disease, low peak bone mass, female gender, Caucasian or Asian race, small body structure, light hair and complexion, certain chronic disease states, and history of medication use. Chronic diseases that increase risk include hyperthyroidism, diabetes mellitus, rheumatoid arthritis, CKD, past or present history of Cushing's syndrome, previous gastric surgery, major organ transplantation, liver disease, epilepsy, alcoholism, malabsorption states, anorexia nervosa, hyperparathyroidism, and women

having menopause before age 40 years without hormonal replacement. A history of fracture after age 50 years or a history of falling puts a person at increased risk for sustaining an osteoporotic fracture. Medications may be an unmodifiable as well as a modifiable risk factor. Patients taking medications such as corticosteroids, thyroid hormones, anticonvulsants (e.g., phenytoin), anticoagulants, lithium, chemotherapeutic agents, aluminum antacids, and tetracycline are at increased risk, and these medications need to be reviewed to determine if alternatives are possible.

Other modifiable risk factors are tobacco use, excessive alcohol use, sedentary lifestyle, low gonadal hormone levels, and diet. The relationship of fat to osteoporosis is complex. Low BMI is known to increase risk of osteoporosis; this is assumed to be a result of less insulation in the event of a fall, as well as decreased peripheral conversion of testosterone to estrogen, which depends on fat deposits. A recent study of weight loss in elderly men found that when at least 2% of weight was lost there was a decline in BMD, concluding that maintaining weight is important for bone health in elderly men (Bleicher et al., 2011). Diet is a key, with calcium and vitamin D supporting good bone health, but large quantities of protein and sources of phosphorus such as carbonated sodas (due to their high phosphate content, which results in excessive urinary excretion of calcium) contributing to bone loss. Vitamin D deficiency in adults results in osteomalacia, a failure to mineralize the bone matrix. Those at highest risk are the older adults; those living in the northern latitudes; those suffering from poor nutrition, malabsorption, and chronic liver or kidney disease; and dark-skinned individuals.

Signs and Symptoms: Patients with osteoporosis may be asymptomatic. All patients more than 50 years old should be screened for fracture risk. Patients need to be questioned about all the risk factors for osteoporosis. Inquire about a personal history of fractures. A fracture at any major skeletal site in an adult older than 50 years should be considered a significant risk factor for osteoporosis. The most common fractures are those of the spine, hip, wrist, and distal forearm. Most fractures that occur after the age of 50 years are due at least in part to low bone mass, even when they result from considerable trauma (NOF, 2014). The exceptions are fractures of the fingers, toes, face, and skull, which tend to be more related to trauma than low bone mass (NOF, 2014). Inquire about a family history of fractures and obtain a thorough menstrual history in women; questions about libido and potency in men are important to determine secondary gonadal issues. After a vertebral fracture, there may be acute pain that may resolve or may become chronic. There may be side pain due to iliocostalis syndrome, resulting from vertebral fractures that result in the lower ribs rubbing against the iliac crest.

Two clinical tools using computer-based algorithms have been developed to assist clinicians in the identification of patients at high risk for fractures. The most widely used is the FRAX: Fracture Risk Assessment Tool developed by the WHO in 2008, accessible at www.shef.ac.uk/FRAX/tool.jsp. The tool is intended to screen patients with low bone density who are not currently receiving treatment to help determine need for treatment. FRAX integrates validated clinical risk factors and BMD of the femoral neck to calculate the 10-year probability of hip fracture and the 10-year probability of a major osteoporotic fracture (clinical spine, forearm, hip, or shoulder). Treatment of osteoporosis should be considered for patients with low BMD, as well as a 10-year risk of hip fracture of 3% or more or a 10-year risk of a major osteoporosis-related fracture of 20% or more. Japanese women were evaluated with the FRAX, and their fracture rate after 10 years was the same as the rate predicted by the FRAX (Tamaki et al., 2011). The FRAX is recommended for screening in most of the current clinical guidelines and the results are often incorporated into the standard bone density test results. It is not appropriate to use FRAX to monitor treatment response (ISCD, 2015). The more recent Q-fracture developed in the United Kingdom is accessible at www.qfracture.org; it includes additional factors intended to improve the estimate of risk.

Physical examination may reveal loss of height with associated kyphosis of the spine. Height should be measured at the initial visit and yearly using a wall mounted stadiometer. Height loss (difference between the current height and peak height at age 20 years) of 1.5 inches (4 cm) or more is significant. If the patient is uncertain of what their peak height was, the arm span measurement is known to be an equivalent to adult height. Gait should be assessed, and the patient's body mechanics should be observed at the same time. The mouth should be examined to assess dentition and any evidence of oral bone loss. The thyroid gland should be palpated. Observe for restrictive respiratory problems due to decreased volume of the thoracic cage and poor expansion with breathing. The spine should be examined in detail, including configuration. The most common site for vertebral fractures in patients with osteoporosis is the lower thoracic (T12) or upper lumbar (L1) region. Any tenderness to palpation over the spinous processes and evidence of swelling, tenderness, and ecchymosis present at the sight of an injury should be noted. Range of motion should be determined, noting limitations or painful movement. An abdominal examination reveals whether the abdomen is protuberant from spinal changes. The distance between the rib cage and the anterior iliac crest should be recorded.

Diagnostic Tests: Osteoporosis is defined based on the BMD measurement. BMD can be measured by several methods, including dual-energy x-ray absorptiometry (DEXA or DXA), quantitative computed tomography (QCT), and quantitative ultrasound (QUS). The results of the DEXA are reported as T- and Z- scores. The WHO T-score compares the bone mass of the patient to the mean of a young adult (20-year-old healthy woman). Recommendations apply to postmenopausal women and men age 50 years and older. In premenopausal women, men less than age 50 years, and children, the International Society for Clinical Densitometry (ISCD) recommends the diagnosis of osteoporosis be made based on ethnic- or race-adjusted Z-score (NOF, 2014). A Z-score of −2 or less is defined as low BMD for chronological age and those above −2 are within the expected range for age. QCT is three-dimensional and provides a true density measurement of the spine and hip. It can also determine trabecular and cortical bone separately. It is used less often due to the increased expense, greater exposure to radiation, and less reproducibility (meaning difficulty comparing results over time). Ultrasound of the heel is FDA approved for screening, but positive results should be confirmed with DEXA. The reproducibility of the

DEXA makes it the test of choice to follow up on response to treatment. The percent change in BMD (from initial or previous) should be used to monitor treatment, not the T-score (ISCD, 2015).

Signs and symptoms identified on physical examination should guide which tests to include. Currently, there is investigation into the use of serum markers of bone turnover to help guide treatment of osteoporosis, but they are not recommended as a substitute for BMD measurement in diagnosis. Other tests are useful in determining secondary causes of osteoporosis. Vitamin D levels should be at least 77 µmol/L or 30 ng/mL because lower levels can result in secondary hyperparathyroidism and have been linked to an increase in other chronic diseases. When interpreting serum calcium level in older adults, it is important to correct for albumin level because 30% to 55% of calcium is bound to albumin. A falsely low measurement results when albumin is low. Every 1 g/dL of albumin binds 0.8 mg/dL of calcium. The correction adds 0.8 mg/dL for every 1 g/dL decrease in albumin. Ionized calcium measures free calcium, but it is an expensive test that is difficult to interpret; consultation before requesting may be helpful.

Differential Diagnosis:

- Multiple myeloma (bone marrow infiltrated; lytic lesions common in the axial skeleton)
- Hyperparathyroidism (serum calcium greater than 10.5 mg/dL, intact PTH)
- Osteomalacia (abnormal serum calcium, alkaline phosphatase, and phosphate levels)
- Hyperthyroidism (decreased TSH levels, T_3, T_4, and the free thyroxine index [FTI])
- Cushing's syndrome (serum cortisol levels greater than 7.5 g/dL, 24-hour free urine cortisol)
- Paget's disease (serum alkaline phosphate is distinctly elevated)
- Hypogonadism (testosterone in men; estradiol, luteinizing hormone, follicle-stimulating hormone in women)

Treatment: Good management of osteoporosis requires a comprehensive approach. The goal of treatment is to prevent fractures. The basic level of prevention and treatment includes diet, exercise, and fall prevention strategies. Adequate intake of calcium and vitamin D is essential to decrease bone loss and bone turnover. Women older than 50 years and men older than 70 old years need 1,200 mg of calcium a day to maintain bone health. There is limited benefit in increasing calcium intake above 1,500 mg, and the risk of renal stones is increased. Conflicting reports have suggested that calcium intake, particularly from supplements, may have either beneficial or harmful effects on cardiovascular outcomes. In response, the NOF and American Society for Preventive Cardiology convened an expert panel to evaluate the effects of dietary and supplemental calcium on cardiovascular disease based on the existing peer-reviewed scientific literature. The organizations ended up adopting the position that there is moderate-quality evidence (B level) that calcium with or without vitamin D intake from food or supplements has no relationship (beneficial or harmful) to the risk for cardiovascular and cerebrovascular disease, mortality, or all-cause mortality in generally healthy adults at this time (Kopecky et al., 2016). Considering the evidence available to date, calcium intake from food and supplements that does not exceed the tolerable upper level of intake (defined by the National Academy of Medicine as 2,000 to 2,500 mg/d) should be considered safe from a cardiovascular standpoint (Ross et al., 2011).

Older women treated with calcium tablets have been shown to be at risk for hypercalcemia. To avoid overtreatment but reach the required amount of daily calcium, all sources should be evaluated. Food sources are preferable because they provide other nutrients as well. When supplements are prescribed, they should not exceed 600 mg in a single dose because absorption is decreased at higher doses. Several formulations of calcium are available in tablets and chews, with and without vitamin D. Calcium carbonate is 40% elemental calcium by weight, but it should be taken with food because stomach acid is required to increase the solubility. Side effects include intestinal gas and constipation. Calcium citrate, which is 21% elemental calcium by weight, is better absorbed and has fewer GI side effects. Tribasic calcium phosphate offers a third option, with 39% elemental calcium by weight; absorption is like calcium citrate, with a price point between calcium carbonate and calcium citrate. Options for calcium treatment are identified in Table 14-6.

Vitamin D replacement is available in two forms, ergocalciferol (vitamin D_2) or cholecalciferol (vitamin D_3). Ergocalciferol is a prescription preparation and carries the potential advantage of coverage by insurance plans; cholecalciferol is OTC. In a head-to-head comparison of the two forms of vitamin D, cholecalciferol was more effective in increasing serum 25-hydroxy vitamin D (25 OH D) than the same dose of ergocalciferol (Glendenning et al., 2009). However, there

TABLE 14-6 Calcium Treatment

FORM OF CALCIUM	ELEMENTAL CALCIUM	PROS/CONS	BRAND NAMES
Carbonate	40%	Cheap Readily available May not be as readily absorbed, take with food GI side effects include gas, bloating, constipation Tablet, liquid, chewable	Tums, Rolaids, Caltrate, Viactiv
Citrate	21%	Better absorbed May be taken on empty stomach Less GI upset Tablet and liquid	Citracal
Tribasic calcium phosphate	39%	Better absorbed Less expensive than calcium citrate	Posture
Food sources	1 cup milk 330 mg 1 oz cheese 100–200 mg ½ cup broccoli 50 mg ½ cup beans 100 mg	Include other nutrients Have the most bioavailable source of calcium	

Abbreviation: GI = gastrointestinal

was no difference in the effect on parathyroid hormone, so the authors question the clinical significance of the difference between the two forms. The choice of which to recommend remains unclear, and both are effective in raising the level of serum vitamin D. Determining the proper vitamin D requirement for a given patient is based on expert recommendations in conjunction with clinical judgment. The American Association of Clinical Endocrinologists (AACE) recommends basing a dose required to maintain a 25 (OH) level at 30 to 50 ng/ml (75 to 125 nmol/L) or above for an optimal and safe range (ISCD, 2015). This often requires 1,000 to 2,000 IU of vitamin D daily.

Lifestyle recommendations include the elimination of tobacco, excessive alcohol and caffeine, and a reduction in the amount of high-phosphorus foods and carbonated beverages. Patients should engage in regular weight-bearing exercises, which help to reduce the rate of bone loss and increase strength. Muscle-strengthening exercises improve strength, balance, and flexibility, which are important in preventing falls. The NOF offers advice on exercise at www.nof.org. Measures to enhance safety in the home and environment should be emphasized. Personal risk factors should be minimized, such as compromised vision, orthostatic blood pressure drop, and drug or alcohol use. Consider a referral to physical or occupational therapy to improve gait and strength, and instruct the patient on proper body mechanics to prevent fractures.

The NOF's (2014) *Clinicians Guide to Prevention and Treatment of Osteoporosis* recommends pharmacological treatment for postmenopausal women and men with a hip or vertebral fracture, a T-score of −2.5 or less or a T-score between −1.0 and −2.5, and high 10-year risk of fracture as indicated by the score on the FRAX. Pharmacological measures to treat osteoporosis in postmenopausal women include antiresorptives, which decrease bone resorption, and anabolic therapy, which promotes bone formation. Antiresorptives include bisphosphonates, a RANKL antibody, a selective estrogen receptor modulator (SERM), calcitonin, and estrogen/hormone therapy. Recent clinical guidelines from the AACE include the newest antiresorptive treatment, denosumab, a monoclonal antibody that targets RANKL (Watts et al., 2010). The parathyroid hormone, teriparatide, is approved for the highest-risk individuals (both postmenopausal women and men). Bisphosphonates require adequate calcium to be affective. The AACE guidelines recommend for first-line treatment one of these bisphosphonates: alendronate, risedronate, zoledronic acid, or the RANKL antibody denosumab. Second-line therapies include the bisphosphonate ibandronate and the SERM raloxifine, which can also be used as third-line therapy. Because of the risk of hypersensitivity, calcitonin, available as an injection or a nasal spray, is recommended as the last line of therapy. Some of the available therapies are also a possibility for use in prevention. The details regarding their use are outlined in Table 14-7.

Follow-Up: For persons at risk for osteoporosis or those already diagnosed, follow-up should include monitoring of height, exercise recommendations, lifestyle concerns, and diet, including intake of calcium and vitamin D. Safety measures for the home and proper body mechanics should be reviewed and the need for adaptive equipment should be reassessed. The timing of repeat DEXA has recently been better defined by results from a longitudinal study. The conclusions of the study were that women with normal bone density or mild osteopenia should be rescreened in 15 years. Those with moderate osteopenia should be rescreened in 5 years, and for those with advanced osteopenia, yearly screening is recommended (Gourlay et al., 2012). For patients receiving pharmacological therapy, review the medication regimen, assess for side effects, and repeat laboratory tests as indicated. Repeating the DEXA in 2 years may be useful to monitor response to treatment, although the true measure of success is the prevention of fractures. Serum and urinary markers of bone turnover are being investigated for monitoring of response to treatment, and clear guidelines regarding their use are likely to be available soon. The patient should also be assessed for fractures and the need for analgesics. Patients with osteoporosis should be enrolled in a fall prevention program.

Sequelae: Osteoporosis-related fractures are major complications. Most fractures resulting from osteoporosis are fractures of the spine. Many of these are initially clinically asymptomatic, but may result in chronic pain, disability, deformity, a decline in function, and mortality. Multiple thoracic fractures may result in restrictive lung disease, and lumbar fractures may alter abdominal anatomy, leading to constipation, abdominal pain, distention, anorexia, and early satiety (NOF, 2014). Kyphosis and other postural changes may affect the way clothes fit and limit activity, including bending and reaching. Vertebral fractures are major predictors of future fracture risk, with a five-fold for subsequent vertebra fracture and two- to three-fold for other fractures (NOF, 2014). By far, the most devastating consequence of osteoporosis is a hip fracture. Of the 250,000 people who sustain a hip fracture yearly, 8% to 36% die within the first year after the injury, approximately 20% of patients require long-term care, and only 40% fully regain their previous level of independence (NOF, 2014). A hip fracture increases the risk of future fractures by two and one-half-fold (NOF, 2014). A fear of falling and injury can develop, which may reduce activity or alter gait to be more careful, which may result in a loss of social and physical function and increase the risk of falls. Fractures can also cause psychosocial symptoms like depression and loss of self-esteem. Wrist fractures are less disabling but can affect the ability to do ADLs. Pelvic and humerus fractures also contribute to increased morbidity and mortality.

Prevention/Prophylaxis: As an adolescent disease with geriatric consequences, prevention needs to begin at an early age. All older adults need to be screened to determine their level of risk for the development of osteoporosis and, using the FRAX, determine the 10-year likelihood of fracture. Dietary history, exercise patterns, and habits should be reviewed with patients. A measurement of height should be recorded at least yearly for all adults. The benefits of changing lifestyle patterns should be emphasized to patients even in advanced age. The USPSTF recommends screening women more than 65 years old and women less than 65 years old who have a 10-year fracture risk equal to that of a woman 65 years old. The USPSTF indicated there were insufficient data to make a recommendation for screening men. The NOF recommends screening men older than 70 years regardless of risk factors (www.qfracture.org).

TABLE 14-7 Pharmaceutical Prevention and Treatment

MEDICATION	ROUTE	DOSE	TREATMENT/ PREVENTION	EFFECTIVENESS
ANTIRESORPTIVES				
Bisphosphonates				
Alendronate	PO	5 mg qd or 35 mg qwk	Prevention	Increase spine and hip BMD. Reduce fractures of spine, hip, and wrist by 50%.
		10 mg qd or 70 mg qwk	Treatment	
Risedronate	PO	5 mg qd or 35 mg qwk	Both	Reduce fractures of spine by 41%. Others: 39%.
Zoledronic acid	IV	5 mg every 2 years	Prevention	Increase BMD spine and hip. Reduce fractures of spine by 70%, hip 41%, others 25%.
		5 mg yearly	Treatment	
Ibandronate	PO	150 mg monthly	Both	Reduce fractures of spine by 40%. Not effective in reducing others.
	IV	3 mg q3mo	Treatment	
SERM				
Raloxifene	PO	60 mg qd	Both	Increase BMD. Reduce fractures of spine by 35%. Reduce invasive breast cancer.
RANKL Antibody				
Denosumab	SC	60 mg q6mo	Both	Increase BMD in the spine, hip, and forearm. Reduce fractures of the spine by 70%, hip 40%, others 20%.
Calcium Metabolism Modifier				
Calcitonin salmon	SC/IM	100 U qod	Treatment	Small increase in BMD. Small reduction in spine fractures. Not effective in reducing others. May reduce bone pain.
	Nasal	1 spray (200 U) qd		
ANABOLIC				
Parathyroid Hormone				
Teriparatide	SC	20 mcg qd	Treatment	Increase in BMD. Reduce fractures of spine by 77%, others by 68%.
			Maximum 2 years	

Abbreviations: BMD = bone mineral density; IM = intramuscular; IV = intravenous; PO = per os (by mouth); qd = every day; qod = every other day; qwk = every week; RANKL = RANK ligand; SC = subcutaneous; SERM = selective estrogen receptor modulator

Referral: The high prevalence of osteoporotic fractures and their major effect on morbidity and mortality emphasizes the critical need to optimize bone health care. Patients presenting with fragility fractures are at high risk of subsequent fracture, but treatment rates have remained low for these patients. A significant care gap exists in the management of osteoporotic fractures. Primary care providers may be knowledgeable about prevention and screening of fractures, but may refer to an orthopedic surgeon for management of a fracture or to an endocrinologist for management of osteoporosis. Fracture liaison service (FLS) programs, a model of care where patients with recent fractures may be referred for secondary fracture prevention, have demonstrated improvement in the quality of care delivered, increased osteoporosis treatment, improved patient outcomes, and cost savings (NOF, 2014). FLS programs have been implemented successfully in both hospital and outpatient settings in the United States and abroad. They are typically coordinated by a nurse practitioner, physician's assistant, nurse or other health professional, and require support from a qualified physician or physician team. The coordinator(s) ensures that individuals who suffer a fracture receive appropriate diagnosis, treatment, and support. Patients may require referral for physical therapy to be evaluated for weight-bearing exercises and for demonstration of safe transferring, lifting, and bending. Patients with functional limitations need instruction on the use of adaptive equipment (e.g., walkers, grabbers).

Education: Recommend patient groups for persons with osteoporosis, senior exercise classes, and dietary counseling. Patients can contact the National Osteoporosis Foundation at 1232 22nd Street NW, Washington, DC 20037, 202-223-2226, www.nof.org, for educational materials. Patients should also be given information about local support groups.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
In a population-based study, CHAMP, of 1705 men ages 70–97 years, 368 subjects lost at least 2% of their body weight. Weight loss was associated with a decline in BMD. Conclusions from the results of the study were that maintaining body weight is important for bone health in elderly men.	A	Bleicher et al., 2011
A study to determine if cholecalciferol and ergocalciferol were equally effective in treating vitamin D deficiency included 95 hip fracture inpatients of whom 70 completed the study. They were randomized to treatment with either cholecalciferol or ergocalciferol, and both 25 OH D levels and intact parathyroid hormone (iPTH) levels were used as outcome measures. Although the cholecalciferol group had a significantly greater increase in 25 OH D than the group taking ergocalciferol, both increased to therapeutic levels. Because there was not a significant difference in measured iPTH, the authors questioned the biological importance of the finding that cholecalciferol was more effective in increasing vitamin D.	B	Glendenning et al., 2009
Denosumab was studied in 7,686 women enrolled in the 3-year FREEDOM study, with WHO-defined osteoporosis. The double-blind, placebo-controlled trial recorded new vertebral fractures as the primary endpoint but also evaluated hip and other fractures. Overall, the denosumab arm experienced 68% fewer spine fractures, 40% fewer hip fractures, and 20% fewer nonvertebral fractures. There were no significant adverse events in the patients treated with denosumab.	A	Cummings et al., 2009
In the 7-year FREEDOM Extension study, 4,550 women were studied to evaluate the long-term benefit/risk of denosumab. Treatment for up to 8 years was associated with persistent reduction of bone turnover, continued increases in BMD, low fracture incidence, and a favorable benefit/risk profile. Eight cases of osteonecrosis of the jaw and two atypical femoral fractures were confirmed.	A	Papapoulos et al., 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

PANCREATIC CANCER

Signal Symptoms: Pancreatic cancer is called the "silent disease" because often the disease is advanced before any symptoms begin. Symptoms include weight loss, pain in the upper abdomen spreading to the back that may come and go and be worse after eating and at night, jaundice of the skin or eyes, dark urine and light-colored stools, bowel movements that look greasy and float in the toilet, weakness, loss of appetite, and nausea and vomiting (De La Cruz, Young, & Ruffin, 2014).

Description: Pancreatic cancer is the fourth leading cause of cancer deaths in the United States. It is difficult to detect, hard to diagnose, early to metastasize, and resistant to treatment. Pancreatic tumors can be characterized as either endocrine or exocrine, depending on the cells of origin. Exocrine pancreatic tumors are the most common, with adenocarcinoma being the most common subtype. Five-year survival rates for pancreatic cancer range from approximately 12% to 14% for stages 1A and 1B, whereas the 5-year survival rate for stage

IV pancreatic cancer is about 1% (American Cancer Society, 2016). Cancers can arise from both exocrine and endocrine portions of the pancreas, though 95% develop in the exocrine portion. Approximately 75% of pancreatic cancers occur within the head or neck, 20% in the body, and 5% to 10% in the tail of the pancreas. The most common type of pancreatic cancer is pancreatic duct adenocarcinoma. Generally, it metastasizes to regional lymph nodes, then to the liver or, less commonly, the lungs (National Comprehensive Cancer Network [NCCN], 2016).

Etiology: Pancreatic cancer is fundamentally a disease caused by damage to the DNA. These mutations can be inherited, or they can be acquired as one ages. DNA can also be damaged by certain behaviors, such as alcohol or smoking, or simply by chance (American Cancer Society, 2016; NCCN, 2016).

Occurrence: There are approximately 53,000 new cases of pancreatic cancer per year and approximately 41,000 deaths per year due to this disease. It is the fourth leading cause of cancer deaths in both men and women in the United States (National Cancer Institute, 2016).

Age: The disease is rare in patients under the age of 45 years. The average age of diagnosis is 71 years. Approximately two-thirds of patients are at least 65 years old (American Cancer Society, 2016).

Gender: Incidence is slightly higher in men than in women.

Ethnicity: African Americans are more likely to have pancreatic cancer than Caucasians, however, recent data suggests that these racial differences are diminishing (Ma, Siegel, & Jemal, 2013).

Contributing Factors: Pancreatic cancer has been linked to smoking and heavy alcohol consumption. Exposure to chemicals and heavy metals including beta-naphthylamine, benzidine, pesticides, asbestos, benzene, and chlorinated hydrocarbons is associated with pancreatic cancer (De La Cruz et al., 2014). An increased BMI during early adulthood is associated with increased pancreatic cancer mortality. There is some association that increased consumption of red and processed meats has been associated with the development of pancreatic cancer. Pancreatic cancer occurs more often in those with diabetes. Certain diabetes medications, including insulin and sulfonylureas, have also been associated with this increased risk. Chronic pancreatitis due to alcohol is associated with a higher incidence and earlier onset of pancreatic carcinoma. Certain hereditary conditions are associated with pancreatic cancer, including hereditary pancreatitis, hereditary nonpolyposis colon cancer, von Hippel–Lindau syndrome, ataxia-telangiectasia syndrome, familial atypical multiple mole melanoma, and familial breast-ovarian cancer (BRCA1 and BRCA2 mutations), among others (American Cancer Society, 2016; NCCN, 2016).

Signs and Symptoms: Patients should be questioned about abdominal and midback pain, anorexia, depression, nausea, vomiting, and changes in bowel movements. Along with significant weight loss and moderate epigastric tenderness, the patient may have a palpable gallbladder (Courvoisier sign) and skin excoriation from pruritus. One of the most characteristic signs of pancreatic cancer of the head of the pancreas is painless obstructive jaundice (De La Cruz et al., 2014). However, patients may notice a darkening of the urine and lightening of stools before the jaundice is visually evident. When questioned, they may indicate that their stools float. They may also have ascites, and an abdominal mass may be noted in those with advanced pancreatic cancer. Left supraclavicular lymphadenopathy (Virchow's node) may be palpated in some patients with widespread disease. Many patients with advanced disease have Trousseau's syndrome (a hypercoagulable state) and there is a high incidence of thromboembolic events as well (NCCN, 2016).

Diagnostic Tests: Liver function testing may show an elevated bilirubin and alkaline phosphatase. Bilirubin may be noted in urine and stool samples as well. There are several tumor markers being evaluated for pancreatic cancer, the most useful being antigen 19-9; the reported sensitivity and specificity are 80% to 90%. The imaging studies that can be done include contrast-enhanced multi-slice helical CT (the preferred method to diagnose and stage pancreatic cancer where available); CT scan, transcutaneous ultrasonography, EUS, MRI, ERCP, or positron emission tomography (PET) may also be used to look at the structures and vessels in the abdomen. Percutaneous transhepatic cholangiography can also be done, in which dye is injected into the liver and shows the bile ducts so they can be seen to detect a blockage. A biopsy will be done using fine-needle aspiration after a CT scan or EUS is done. Where expert EUS is available, it has proven to be the most specific and sensitive diagnostic test, with detection rates of 99% to 100%. The most difficult clinical situation to diagnose pancreatic cancer is the person with underlying chronic pancreatitis because all of the previous imaging studies may show abnormalities and may not help differentiate between carcinoma and chronic pancreatitis.

Differential Diagnosis:

- Aortic aneurysm
- Duodenal or gastric ulcers
- Ampullary carcinoma
- Bile duct strictures or tumors
- Cholangitis
- Cholecystitis
- Choledocholithiasis
- Cholelithiasis (De La Cruz et al., 2014)

Treatment: The extent of surgery depends on the location and size of the tumor, the stage of the disease, and the patient's general health. The Whipple procedure is the most common surgery for pancreatic cancer, especially if the tumor is in the head of the pancreas. A distal pancreatectomy removes the body and the tail of the pancreas, and a total pancreatectomy removes the entire pancreas, part of the small intestine, a portion of the stomach, the common bile duct, the gallbladder, the spleen, and nearby lymph nodes. After surgery the patient may have radiation therapy, chemotherapy, or a combination of both. Gemcitabine remains the standard of care for the disease, either alone or in conjunction with other drugs (NCCN, 2016). Pain relief is crucial in palliative care. This can be done with narcotic analgesics alone or by combining them with tricyclic antidepressants or antiemetics. Radiation therapy can also palliate pain, but does not affect patient survival. Obstructive jaundice warrants relief if the patient has pruritus or right upper quadrant pain. Anorexia may also improve after relief of biliary obstruction.

Pancreatic insufficiency and subsequent malabsorption is treated with pancreatic enzyme replacement.

Follow-Up: Follow-up after surgery and/or treatment modalities is important to ensure that any changes are seen and dealt with as early as possible. This will include visits to the primary care provider, surgeon if surgery was done, and oncologist if that treatment modality was selected (De La Cruz et al., 2014).

Sequelae: Patients with pancreatic carcinoma are often anorexic. Patients with malabsorption diarrhea and weight loss may benefit from pancreatic enzyme supplementation. Visits to a dietitian may also be useful for nutritional assistance (De La Cruz et al., 2014).

Prevention/Prophylaxis: Although pancreatic cancer is not completely preventable, patients need to be aware of probable modifiable risks associated with the development of pancreatic cancer, such as smoking and excessive alcohol intake (a contributor to chronic pancreatitis). It remains controversial whether people with given risk factors should be screened for pancreatic cancer. EUS was found to be superior to CT scan or MRI for detection of pancreatic abnormalities in asymptomatic high-risk patients (Canto et al., 2012).

Referral: Current guidelines recommend that decisions about treatment and resectability involve input from a multidisciplinary group of specialists. Patients selected for surgery should be based on the probability of cure (NCCN, 2016). Other factors include comorbidities, overall performance, and age. In advanced pancreatic cancer, patients and family should be informed of availability of palliative care measures and hospice as indicated by the patient's progression of disease.

Education: The best decisions are made with a solid knowledge base, so one needs to learn as much about pancreatic cancer as possible. The patient and family will need end-of-life education. Although some patients may live up to 2 years or more, most with advanced pancreatic cancer die within months of diagnosis. There are support groups that can give information on possible clinical trials pertinent to the patient's condition (De La Cruz et al., 2014).

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Gemcitabine is recommended as first-line chemotherapy for patients with metastatic pancreatic cancer.	A	NCCN, 2016
Asymptomatic, high-risk patients should be considered for pancreatic cancer screening using EUS.	C	Canto et al., 2012 NCCN, 2016
Dual-phase helical CT scan is the best initial imaging test for diagnosis and staging of suspected pancreatic cancer.	B	NCCN, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

A 65-year-old man presents with pain in the left-upper quadrant and epigastric area of his abdomen. He states that he had an episode of acute pancreatitis 10 years ago without any real sequelae. He does say that he has had episodes of abdominal pain in the epigastric and upper abdominal area over the years, but generally over time it goes away within hours or days and he does not seek medical care.

On further questioning, he admits to drinking anywhere from two to six beers at least 3 days a week. He does not feel this is a problem; he does remember being told to watch his alcohol intake when he had acute pancreatitis, but after several years without pain he decided that alcohol must not be a factor in his abdominal pain. Further questioning about his symptoms has him telling you that his pain radiates to the left scapular area of his back. He admits to being thirstier recently and has been urinating more frequently as well. He has had to notch his belt two holes within the last year. He has not been trying to lose weight. He denies diarrhea but has had nausea without vomiting. Eating aggravates the pain but bending over at the waist helps the pain lessen.

Objective: Blood pressure (BP) 135/80 mm Hg, heart rate (HR) 80 beats/min, BMI 21.

1. What additional subjective data are you seeking?
2. What additional objective data will you be assessing for?
3. What are the differential diagnoses that you are considering?
4. What laboratory tests will help you rule out some of the differential diagnoses?
5. What imaging studies are generally ordered for a patient with these symptoms?
6. What is your treatment plan for this patient?
7. Will you be looking for a consultation?

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CHAPTER 15

Hematological and Immune System Disorders

Lori Martin-Plank

Many factors affect patients' hematological and immune status, and because blood and lymph circulate throughout the body, manifestations of these disorders may appear in different organ systems.

ASSESSMENT

The assessment process includes a detailed history, physical examination, and diagnostic testing. The presenting symptoms of hematological deficiency can include excessive bruising, petechiae, unexplained bleeding, epistaxis, or inflamed lymph nodes. Older adult patients with severe anemia may complain of fatigue, dizziness, heart palpitations, headache, shortness of breath with exertion, and exercise intolerance. Confusion, depression, and cold intolerance are also common. In many cases, symptoms are absent, subtle, or erroneously attributed to the aging process.

The medical history obtained from the patient should include past history of anemia, hemoglobin abnormalities (thalassemia), vitamin C or K deficiency, recent surgery or blood transfusion, lymphadenopathy, and clotting disorders. Also inquire about any occupational or other known exposure to toxic agents or radiation, family history of anemia, current medications, and history of chronic disease. Some of the most common causes of hematological disease in the older adult include iron-deficiency anemia (IDA), anemia of chronic disease (ACD), and pernicious anemia, which has an increased incidence in patients over the age of 60 years. Myelodysplastic syndromes and leukemias also are becoming more common. Systemic disease and severe clotting disorders need to be included in the differential diagnosis.

Physical Examination and Diagnostics

The physical examination should be complete and thorough. Assess the eyes for pale conjunctiva and icteric sclera, which occur with some anemias. Inspect the color of the skin, noting any ashen or yellow changes; pallor is difficult to assess in older adults. Check for petechiae, bruising, and pale mucous membranes. Assess the nails for the presence of concave spoon nails, seen with iron deficiency. A red, painful, beefy tongue can be present with pernicious anemia. Oral lesions may occur. Palpate the abdomen for any splenomegaly or tenderness. Tachypnea and tachycardia can be seen with severe anemia. Auscultate the heart for any systolic murmurs. If blood loss is suspected—and the history warrants—a rectal examination to assess for guaiac-positive stools is performed. Finally, determine whether any mental status changes have occurred; do a complete neurological examination, checking cranial nerve function and deep tendon reflexes. The absence or presence of physical findings on examination helps to narrow the clinical diagnosis. Diagnostic tests done to complete the hematological assessment include:

- Complete blood count (CBC) with differential; check red blood cells (RBCs), hemoglobin (Hgb), hematocrit (Hct), mean corpuscular volume (MCV), and mean corpuscular hemoglobin concentration (MCHC)
- Iron, total iron-binding capacity (TIBC)
- Ferritin if iron-deficiency or microcytic anemia is considered
- Reticulocyte count to assess for blood loss or blood cell destruction
- Hgb electrophoresis to assess for Hgb abnormalities or thalassemia
- Vitamin B_{12} and folate levels to rule out pernicious anemia

The general physical examination should note any weight loss or wasting, skin changes, or lymphadenopathy. The history and physical examination provide the baseline for the plan of care and necessary diagnostic testing. The general laboratory tests ordered should include:

- CBC with differential (platelet count)
- Urinalysis
- Electrolyte panel with Hg A1c or fasting glucose levels to assess for diabetes
- Erythrocyte sedimentation rate (ESR)
- Rapid plasma reagin (RPR)

Tests to rule out more specific disease include titers for HIV, Lyme disease, Epstein-Barr virus (EBV), toxoplasmosis, and cytomegalovirus (CMV). Clinical consultation or referral to a specialty physician or nurse practitioner may be necessary to work up the older adult patient with suspected immunological suppression or disease and to ensure that the appropriate diagnosis is made.

Immunological Assessment

The presentation or complaints of immunological disease may be vague and nonspecific. Older adult patients may present with chronic fatigue, frequent or recurrent illness, weight loss, lymphadenopathy, depression, or recurrent infections. As stated earlier, a complete medical history should be obtained. A complete sexual history also should be obtained, with specific focus on the number of partners the patient has had in the past 6 months; encounters with men, women, or both; any history of sex with prostitutes; sexually transmitted infections; and protection used by the patient and his or her partners. Inquire about recent travel, tick bites, risk factors for HIV, recent immunization or surgery, and medication use. Ask about illicit or recreational drug use, including IV drug use.

ANEMIA OF CHRONIC DISEASE

Signal Symptoms: Minimal to none; nonspecific fatigue.

Description: ACD is the most common form of anemia in the older adult, followed by IDA. The percentage of anemia in noninstitutionalized persons 65 years and older, with respect to ACD, is 19.7%, compared to 16.6% with IDA (Bross, Soch, & Smith-Knuppel, 2010). ACD has been described in the literature as "anemia of chronic inflammation (AI)," and "anemia of chronic disorders," alluding to its etiology. ACD is seen in a variety of conditions, including infections, malignancies, and autoimmune conditions. Frequently, association is seen with kidney disease, heart failure, and transplantation.

ACD has an insidious onset. Signs and symptoms can be vague or absent, and laboratory data can present an even more daunting challenge. ACD is generally normocytic and normochromic, although about 25% to 35% may be microcytic in advanced stages; it coexists with IDA in some older persons. Anemia in patients with renal, hepatic, or endocrine disorders is categorized separately, because it manifests with a different hematological profile.

Anemias of any form in the elderly must never be attributed solely to old age and should be recognized as a sign of physiological decline. ACD in particular must be differentiated from IDA, because their treatments can vary significantly. Recognition and appropriate treatment are important, as in hospitalized elderly patients anemia has been associated with increased length of stay, readmissions, and mortality (De Amici et al., 2015; Nathavitharana et al., 2012). A link between anemia in older adults and an increased risk for developing dementia has also been reported (Hong et al., 2013).

Etiology: ACD, as its name implies, is associated with a chronic disorder, usually infection, inflammation, malignancy, or trauma, and, in many cases, the cause is unknown. ACD can be classified as a normochromic, normocytic anemia, but approximately one-third of patients with ACD have microcytosis. The pathophysiology in the development of ACD includes a shortening in RBC survival, impaired erythropoietin production, and a blunted bone marrow response to erythropoietin (Cullis, 2011). A pathophysiological hallmark of ACD is an immune activation, with an increase in inflammatory cytokines resulting in a decrease in hepcidin levels. This leads to disregulation of iron homeostasis, characterized by an increased uptake and retention of iron within the cells of the reticuloendothelial system (liver/spleen), resulting in decreased RBC production (Gangat & Wolanskyj, 2013). Essentially, iron is present but inaccessible for use in the production of Hgb with the erythrocytes (Santosh, Nagarag, & Sasidaran, 2015). A shortened RBC survival is also a contributing factor to ACD. In addition, aging is associated with increased inflammation, and elderly patients with anemia have higher levels of inflammatory markers, such as C-reactive protein and IL-6 (Price & Schrier, 2010; Weiss, 2015). Lower erythropoietin levels have also been found in elderly patients with ACD (Gowanlock et al., 2016).

Occurrence: Due in part to the fact that elderly persons frequently encounter multiple chronic comorbidities, ACD is a common cause of geriatric anemia, accounting for approximately more than one-third of the cases. The prevalence of anemia increases two-fold over the general population in patients with chronic kidney disease (CKD). In those with CKD, the prevalence further increases with the stage up to 53.4% at Stage 5 (Stauffer & Fan, 2014).

Age: Although anemia can be present in all age groups, ACD is seen frequently in the older adult due to the presence of chronic disease states, many involving inflammatory processes.

Gender: Sex distribution varies based on the underlying cause of anemias. In contrast to anemia in younger individuals, anemia in elderly persons is more common in men than in women. Some of the difference may be due to the fact that the World Health Organization's (WHO's) sex-specific value of less than 13 g/dL is used for males across all ages (as opposed to less than 12 g/dL for females). Because Hgb declines slightly and anemia prevalence rises with aging, the prevalence rate for men is higher with advancing age (Cappellini & Motta, 2015).

Ethnicity: Not significant.

Contributing Factors: The presence of one or more chronic conditions, such as cancer, chronic infection, chronic inflammation, or autoimmune conditions, is a contributing factor to the development of ACD.

Signs and Symptoms: Onset of signs and symptoms in ACD can be confounding and is usually masked by the underlying conditions. The typical symptoms of anemia, such as fatigue,

weakness, dyspnea, and palpitations, may be present, but are nonspecific, and in older adult patients tend to be attributed to the aging process. Additionally, many older patients adjust their activities as their bodies make physiological adaptations for the underlying disease. Although it may be difficult to assess in the geriatric population, conjunctival pallor is a reliable sign. Its absence, however, cannot exclude anemia. Other signs may be discovered through incidental laboratory testing. Cognitive decline, depression, and decreased quality of life may also accompany ACD. The clinician should have a high index of suspicion of ACD in older adult patients with chronic conditions, and any presentation of the previous symptoms should prompt further evaluation.

Diagnostic Tests/Tools: In discussing diagnostic testing for ACD, it is important to reiterate that ACD is classified as a normocytic, normochromic anemia with less than 25% presenting with microcytosis (Gangat & Wolanskyj, 2013). This is important to consider when interpreting laboratory tests to determine whether there is a coexisting IDA.

Diagnosis of ACD is one of exclusion. Supportive findings include adequate iron stores, an erythropoietin level that is not increased (in those with preserved kidney function), and no other cause of the anemia (DeLoughery, 2014). In ACD, serum ferritin is a key test in differentiating ACD from IDA. Serum ferritin is a measure of iron stores, and the main abnormality in ACD is the impaired ability to use iron stores in the reticuloendothelial system (liver/spleen). Values in ACD are reflected in a normal to high ferritin level (i.e., iron is present but inaccessible for use). In contrast, ferritin levels in IDA are low (below 30 ng/ml).

Other laboratory tests include CBC, RBC indices, serum iron, TIBC, transferrin saturation, reticulocyte count, peripheral blood smear, and reticulocyte production index (RPI), which corrects for the degree of anemia. Table 15-1 compares the laboratory data in ACD and IDA. Research is being directed to look at new serological markers, such as hepcidin, that may be used in the future to better distinguish ACD from IDA.

TABLE 15-1 Laboratory Data in ACD and IDA[1]

LABORATORY TEST	ACD	IDA
CBC		
• Hemoglobin	<12 g/dL (120 g/L) women <13 g/dL (130 g/L) men Rarely <10 g/dL (100 g/L)	<12 g/dL (120 g/L) women <13 g/dL (130 g/L) men
• Mean corpuscular volume: (MCV)[2]	80–96 mcm^3 (normocytic)	70–80 mcm^3 (microcytic)
• RBC distribution width: (RDW)	Normal	Increased
Serum ferritin	Normal or increased	Decreased
Serum iron	Decreased	Decreased
Total iron-binding capacity	Decreased	Increased
Transferrin	Normal to low	Increased
Transferrin saturation	Reduced	Reduced
Reticulocyte production index	Decreased	Decreased
Reticulocyte count	Decreased	Decreased
Peripheral smear	Normochromic	Microcytosis, hypochromic

[1] Changes in relation to respective normal values
[2] Overlap, may be normal with ACD + IDA
Abbreviations: ACD = anemia of chronic disease; CBC = complete blood count; IDA = iron deficiency anemia; RBC = red blood cell; g/dl = grams per deciliter; L = liter

Differential Diagnosis: Early or partially treated IDA is the main differential diagnosis for ACD, because microcytosis may be present or absent in either disorder. Hypoproliferative anemias (e.g., renal disease, endocrine disorders) and thalassemia are also considerations.

Treatment: Treatment of ACD focuses on management of the underlying disorder. A therapeutic trial of oral iron supplementation may be useful in delineating between ACD and IDA. Iron supplementation is of limited benefit in ACD, except in cases of coexisting IDA or in patients with hyporesponsiveness to erythropoiesis-stimulating agents (ESAs). IV iron may be preferable in these cases because increased hepcidin levels inhibit intestinal absorption of oral iron supplements (Gangat & Wolanskyj, 2013).

Although there is no specific treatment for ACD, if anemia is severe or underlying disease is resistant to treatment, ESAs (e.g., Epogen, Procrit) may be used (Cullis, 2011). Coverage for ESAs by Medicare and other third-party reimbursement requires documentation of laboratory test findings indicative of ACD. Due to inherent risks, transfusion should be reserved for treating severe or life-threatening anemias. The American College of Physicians recommends against use of ESAs in patients with mild to moderate anemia and heart failure or coronary heart disease due to an increased risk for thromboembolic events and possible stroke (Qaseem et al., 2013). The U.S. Food and Drug Administration (FDA) Medwatch (2011) also urged caution in using ESAs in patients with renal disease due to the potential for cardiovascular events. Dosage for a patient with renal disease should be individualized and be as low as possible while reducing the need for transfusion.

A study by Perlstein and colleagues (2011) demonstrated an association between vitamin D deficiency and ACD in the elderly. Vitamin D replacement is thought to decrease hepcidin levels. However, more studies are needed to see if there is an etiological link between ACD and vitamin D deficiency, and whether replenishment has any significant effect.

Follow-Up: Follow-up is individualized depending on underlying cause and extent of the anemia.

Sequelae: Variable depending on the underlying condition.

Prevention/Prophylaxis: Early education in healthy lifestyle may prevent or delay onset of some chronic conditions.

Referral: Referral is indicated for progression of anemia and/or resistance to treatment of underlying condition; significant anemia of unknown causation; and severe, symptomatic anemias.

Education: Teaching the patient and family about the importance of lifestyle modifications and adherence to a treatment plan for management of underlying conditions may be a

factor in preventing or slowing the progression of chronic diseases. A healthy balanced diet is indicated to prevent or correct dietary deficiencies that can contribute to other forms of coexisting anemias.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Anemia is an independent risk factor for increased morbidity and mortality and decreased quality of life in community-dwelling older persons.	B	Goodnough & Schrier, 2013 Bross et al., 2010
Anemia is associated with increased length of stay, hospital readmissions, and mortality in older adults.	B	De Amici et al., 2015 Nathavitharana et al., 2012
IV iron therapy may have a beneficial effect in treating ACD.	C	Gangat & Wolanskyj, 2013 Goodnough & Schrier, 2013 Cullis, 2011
ESAs should not be used to treat patients with mild to moderate anemia and heart failure or coronary heart disease.	B	Qaseem et al., 2013
When possible, treatment of the underlying disease is the therapeutic approach of choice for anemia of chronic disease.	C	Weiss, 2015 Cullis, 2011

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

ANEMIA, IRON DEFICIENCY

Signal Symptoms: Minimal to none; nonspecific fatigue and/or weakness, dizziness, shortness of breath.

Description: IDA is classified as a microcytic, hypochromic anemia and is the second most common anemia in the elderly, preceded by ACD. Anemia is defined by the WHO as an Hgb concentration of less than 12 g/dL for women and less than 13 g/dL for men (de Benoit, McLean, Egli, & Cogswell, 2008). IDA can be acute (i.e., from rapid blood loss, as in hemorrhage) or chronic (reflecting occult blood loss or poor nutrition).

Etiology: Multifactorial; IDA can be caused by inadequate intake, decreased absorption, increased demand, or increased loss of iron. In the United States, IDA in this population is less frequently solely a result of dietary deficiency. Although the National Health and Nutrition Examination Survey III (NHANES III) classified iron-deficiency anemia with other nutritional anemias, in the United States iron deficiency is more often caused by blood loss (Pang & Schrier, 2012). The majority of older adults with IDA have an underlying gastrointestinal (GI) tract abnormality (Hempel & Bollard, 2016).

Occurrence: Anemia affects 27% of the population worldwide, with iron deficiency as the dominant cause (greater than 60%) (Kassebaum et al., 2016). IDA occurs in 2% to 5% of adult men and postmenopausal women in developed countries (Goddard, James, McIntyre, & Scott, 2011; Liu & Kaffes, 2012).

Age: Most common in children and younger females of childbearing age; in older adults, incidence increases with age.

Gender: In ages 65 years and older, IDA affects more men than women.

Ethnicity: Not significant; certain cultural groups may have underlying genetic factors predisposing them to anemias, such as thalassemia.

Contributing Factors: In the older adult patient, decreased oral intake or inadequate diet, GI-related surgery, malabsorption syndromes, low socioeconomic status, medications (i.e., salicylates, NSAIDs, proton pump inhibitors, H_2 blockers, and antacids), combination of medication and alcohol, and chronic blood loss, most frequently from the GI tract, are contributing factors for IDA. Some GI causes include peptic ulcer disease, gastritis, hiatal hernia with mucosal ulceration, neoplasms, diverticular disease, or bleeding caused by inflammatory bowel disease. *Helicobacter pylori* infection can decrease iron absorption and can also lead to micro erosions that cause bleeding. Surgeries such as gastrectomy, duodenal bypass, or bariatric surgery (Roux-en-Y gastric bypass) can result in decreased iron absorption (Camaschella, 2015).

Recent evidence suggests that obesity may also play a role in IDA. Obesity-related inflammation can cause impaired duodenal absorption and an increase in hepcidin, a peptide that regulates iron homeostasis. Higher hepcidin levels may reduce iron absorption and blunt the effects of iron fortification (Aigner, Feldman, & Datz, 2014). In patients with prosthetic heart valves, intravascular hemolysis may lead to IDA related to increased hemosiderin loss in the urine. Epistaxis, hematuria, dysfunctional uterine bleeding, and bleeding diathesis are other possible contributing factors in older adults.

Signs and Symptoms: Diagnosis of IDA may be the result of an incidental blood study finding, requiring further investigation. Clinical presentation of IDA can be vague, unreliable, and, in many cases, asymptomatic. In the older adult, symptoms may be incorrectly attributed to the aging process or chronic comorbid conditions. Anemia that develops slowly tends to present with fewer signs and symptoms then anemia that is of a rapid onset (e.g., acute bleeding) (Vanasse, 2014). Fatigue, weakness, lethargy, tachycardia, palpitations, dyspnea on exertion, headache, irritability, inability to concentrate, neuralgia, sore tongue, paresthesias, restless legs syndrome, alopecia, and susceptibility to infection are possible symptoms. Dizziness, faintness, claudication, exercise intolerance, or angina also may be present (Lopez, Cacoub, Macdougall, & Peyrin-Biroulet, 2016).

A thorough history should include questions about diet, symptoms (especially GI-related), signs or sources of blood loss, surgeries, and family history of GI malignancies such as stomach or colon cancer. Pica or pagophagia (i.e., compulsive consumption of ice or iced drinks) may be reported (Short & Domagalski, 2013). Medication history, including over-the-counter (OTC) drugs, is especially important to rule out NSAID-induced bleeding.

Physical examination may be unremarkable. Pallor, common in patients with significant anemia, is also common in aging and may be discounted as a normal age-related finding. Conjunctival pallor, bluish discoloration of the sclera, cheilosis, glossitis, brittle ridged nails, or "spoon" nails (koilonychia) may also be present. Cardiovascular and respiratory examination may reveal tachycardia, systolic murmur, or signs of heart failure (HF). In some patients, splenomegaly may be present, owing to hemolysis of iron-deficient RBCs. Lymphadenopathy, weight loss, bruising, and jaundice are physical signs that require further evaluation.

Diagnostic Tests/Tools:

- CBC will reveal Hgb of less than 12 g/dL. Note that patients who are smokers and those with chronic hypoxemia have a higher premorbid Hgb and therefore can be anemic at higher Hgb levels (Domino, 2016).
- RBC indices reveal an MCV/RBC size that will be decreased to less than 80 fL in adults; mean corpuscular hemoglobin (MCH)/RBC color will show hypochromia or pale cells; RBC distribution width (RDW)/volume variation will be increased.
- Serum ferritin level is the most accurate initial test for IDA; patients with IDA will test with low levels. A ferritin level of less than 25 ng/mL is highly predicative of IDA, whereas a level of greater than 100 ng/mL indicates adequate iron stores with a decreased likelihood of IDA. Note that in some patients, deficiency may be missed, because ferritin is an acute-phase reactant and ferritin levels may be falsely elevated in inflammatory conditions (Domino, 2016). The abnormal laboratory cut-off may be higher in these situations.
- TIBC: increased
- Serum iron: decreased
- Transferrin saturation: decreased. This finding indicates that there is a decreased amount of iron available to bind to transferrin, which is an iron-carrying protein.
- Peripheral smear: reveals microcytosis, hypochromia, and poikilocytosis
- Bone marrow iron stain: usually not necessary

Once IDA is confirmed, further diagnostic studies to determine the cause must be undertaken. The presence of IDA in adults, especially men and postmenopausal women, is presumed to be the result of blood loss in the GI tract, and the possibility of malignancy must be ruled out. Endoscopic evaluation usually begins with colonoscopy, if indicated, and when the patient is 50 years or older to identify any lower GI bleeding. Esophagogastroduodenoscopy **(**EGD**)** investigates for upper GI bleeding. Evaluation of mid and distal small bowel can be done through several methods, including capsule endoscopy, and should be done if the colonoscopy and EGD findings are negative (Raju, Gerson, Das & Lewis, 2007). The choice and sequence of procedures is dependent on clinical suspicion and presenting signs and/or symptoms (Bull-Henry & Al-Kawas, 2013).

Differential Diagnosis: Differential diagnosis for microcytic, hypochromic anemias by etiology includes:

- Defective iron utilization: thalassemia trait, sideroblastosis, G6PD deficiency
- Defective iron reutilization: infection, inflammation, cancer, and other chronic diseases
- Hypoproliferation: decreased erythropoietin from renal failure, hypothyroidism, and other hypoproliferative states (Domino, 2016)

In adult men and postmenopausal women, differential diagnosis for IDA includes the following in decreasing order of frequency: ACD, unexplained anemias, and thalassemia trait (Stauder & Thein, 2014).

Treatment: Oral iron therapy is usually first-line treatment for patients with IDA. An initial medication of choice is ferrous sulfate 325 mg (65 mg of elemental iron) BID–TID. Depending on the severity of the IDA, adults will usually require a daily dose in the range of 100 to 200 mg of elemental iron (Camaschella, 2015). Older adults have an increased risk for toxicity so may need treatment with lower doses. Although no specific guidelines exist for treating older adults, one study of hospitalized older adults demonstrated that lower doses achieved the same results as higher doses with significantly fewer side effects (Rimon et al., 2005). In a study of iron-depleted younger women, results showed that twice-daily iron supplements seemed to have limited additional effect on total iron absorption compared to daily administration (Moretti et al., 2015). Further research, especially with older adults, is needed to determine optimal dosing efficacy.

Ideally, a dose is administered on an empty stomach, 1 hour before meals. Absorption of iron is enhanced with ascorbic acid (vitamin C). Other measures to improve iron

absorption include avoidance of foods high in tannate (tea, bran, cereal) and medications that increase gastric pH (milk, antacids, proton pump inhibitors, H_2-histamine blockers, quinolones, and tetracycline). Liquid iron preparations are recommended for those who are unable to dissolve the coating of iron pills, which leads to decreased iron absorption. Sustained-release formulations (enteric coated) of iron for initial therapy should be avoided because they reduce the amount of iron presented for absorption by the GI tract (Domino, 2016). The patient should be educated that oral iron may cause dark stools.

Oral iron therapy may cause significant GI side effects, including constipation, diarrhea, nausea, and abdominal cramping. Interventions to reduce GI symptoms include dose reduction of iron or starting with one-half the dose and gradually increasing; switching to ferrous gluconate 325 mg (35 mg of elemental iron) or ferrous fumarate 325 mg (99 to 108 mg of elemental iron), because these preparations may be better tolerated than ferrous sulfate; or taking the iron with food, although not recommended, which may reduce absorption by 50% but may increase patient compliance with therapy. Measures to alleviate constipating effects of iron therapy include gentle laxatives, stool softeners, fiber, and adequate liquid intake. Medication effect should be demonstrated after 3 to 4 weeks of therapy.

Parenteral iron therapy is indicated in the following situations: chronic uncorrectable bleeding, intestinal malabsorption, intolerance to oral therapy, nonadherence, or Hgb level less than 6 g/dL with signs of poor perfusion in those who would otherwise receive transfusions (e.g., those with religious objections), and coexisting inflammatory conditions that can interfere with iron homeostasis. Several different IV iron products are available in the United States.

Blood transfusions may be necessary initially (e.g., acute bleeding) and should be considered for patients who are symptomatic (i.e., fatigue, dyspnea on exertion), as well as for asymptomatic cardiac patients with an Hgb level less than 7 g/dL (Cappellini & Motta, 2015; Qaseem et al., 2013). The decision to transfuse should consider the clinical status and comorbidities (Carson et al., 2016).

In addition to medical therapy, a healthy diet with foods rich in iron should be encouraged. Dietary sources of iron include meat, fish, poultry, fruits, green leafy vegetables, beans, nuts, and grain products.

Follow-Up: The restoration of iron stores is the primary goal of treatment. The Hgb level should increase by 1 g/dL every 2 to 3 weeks, but restoration of iron stores may take 3 months after Hgb is corrected, during which time iron therapy should be continued (Cappellini & Motta, 2015; Goddard, James, McIntyre, & Scott, 2011). The patient should be seen at regular intervals to reassess symptoms or side effects from the treatment plan. Patients should be evaluated in 2 to 4 weeks for examination and Hgb after the initiation of therapy. Referral should be considered if failure to respond to a 4- to 6-week trial of oral iron occurs (Domino, 2016). Subsequent evaluations are at the discretion of the health-care provider, but some sources recommend rechecking the CBC every 3 months for a year (Primack & Mahaniah, 2015; Short & Domagalski, 2013).

Sequelae: Possible complications include failure to identify an occult bleeding source, particularly one related to malignancy.

Prevention/Prophylaxis: IDA can be prevented by promoting proper nutrition with adequate iron intake, identifying those at high risk, and prompt evaluation and treatment of any symptoms or bleeding.

Referral: Referral to a specialist (e.g., gastroenterology for diagnostic studies, hematology, oncology if malignancy is suspected) is indicated to aid in the diagnosis and treatment of the causation. Collaborative management is appropriate for complex cases. Referral to a nutritionist can assist with the evaluation of dietary inadequacies and with meal planning or with congregate or home-delivered meals.

Education: The patient should be educated about the mechanism of anemia, the expected course of therapy with respect to frequency of office appointments and blood tests, any referrals, the importance of adhering to the treatment regimen, and the need to notify the patient's health-care provider about problems with the regimen. A guide to the dietary sources of iron to supplement oral iron medication is helpful to both the patient and any person responsible for meal planning and preparation.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Serum ferritin is the best test for iron deficiency anemia.	C	Miller, 2013 Goddard et al., 2011
Adult men and postmenopausal women with iron deficiency anemia should be screened for GI malignancy.	C	Liu & Kaffes, 2012
Treatment of iron deficiency once per day is as efficacious as a two to three per day regimen.	B	Moretti et al., 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

IMMUNE THROMBOCYTOPENIC PURPURA (IDIOPATHIC THROMBOCYTOPENIC PURPURA)

Signal Symptoms: Petechiae or ecchymoses; oral mucocutaneous bleeding. Some have no symptoms, and thrombocytopenia is an incidental finding on blood work.

Description: Immune thrombocytopenic purpura (ITP) (also called idiopathic thrombocytopenic purpura) is an acquired condition manifested by thrombocytopenia from immune-mediated, accelerated platelet destruction combined with inadequate platelet production and T-cell–mediated effects (Arnold, 2015). ITP is commonly defined as having a platelet count less than 100×10^9/L with mucocutaneous bleeding of an idiopathic origin (Grainger et al., 2016). ITP is classified as primary (idiopathic) or secondary. Secondary ITP occurs in association with other autoimmune diseases, such as systemic lupus erythematosus, HIV, or HCV, among others. The next level of ITP delineation includes newly diagnosed (less than 3 months), persistent (between 3 and 12 months duration), and chronic (more than 1 year duration) (Konkle, 2012; Sultan et al., 2016).

Etiology: Etiology is not fully understood, but current evidence indicates the increased platelet destruction by the immune system and megakaryocytopoiesis abnormalities are associated with genetic and acquired factors (Raj, 2017).

Occurrence: Approximately 2 to 4 in 100,000 adults (Lambert & Gernsheimer, 2017).

Age: Increases with age; patients over 65 years old have a higher risk of bleeding and mortality.

Gender: Women are affected more than men.

Ethnicity: Not significant.

Contributing Factors: The condition is idiopathic or occurs after a virus in conjunction with certain drugs (e.g., heparin, salicylates, sulfonamides, quinine) or autoimmune conditions.

Signs and Symptoms: Epistaxis, purpuric or ecchymotic lesions, mucocutaneous oral bleeding, possibly bleeding with aspirin use or other NSAID use. There may be no symptoms, but a routine blood test may show thrombocytopenia with no other abnormalities. Severe bleeding is usually only a concern if platelet count falls under $10 \times 10(9)$/L (Arnold, 2015). The concurrent presence of an autoimmune disorder favors secondary ITP (George, 2012; Rodeghiero et al., 2009).

Diagnostic Tests: CBC with peripheral blood smear (PBS) will reflect thrombocytopenia but otherwise be normal. PBS may show larger than normal platelets but normal RBC and WBC morphology. If there are other abnormalities in the PBS, a bone marrow biopsy is required to rule out other problems. In patients over 60 years old, bone marrow biopsy is realistic due to an increase in myelodysplastic syndromes.

Differential Diagnosis:

- Acute leukemia
- Thrombotic thrombocytopenic purpura
- Other autoimmune diseases
- Drug-induced thrombocytopenia
- Infection (HIV, EBV, hepatitis, CMV)
- Vasculitis
- Vitamin K deficiency
- Disseminated intravascular coagulation (DIC)
- Hemolytic uremic syndrome (HUS)
- Hypersplenism
- Primary ITP is a diagnosis of exclusion (Raj, 2017; Smith, 2007; Snyderman & Herman, 2011).

Treatment: The goal of treatment is to sustain a safe (not normal) platelet count to avoid bleeding. This may include withholding certain drug therapy, where risks outweigh benefits. For patients with platelet counts less than 10,000/mm^3, platelet transfusions may be indicated. Prednisone 1 mg/kg/day orally or IV immunoglobulin (IVIG) are used initially; a 4-day burst of dexamethasone (40 mg/day) may also be initiated or used in lieu of steroid treatment. In cases where platelet counts are less than 20,000/mm^3 with severe mucocutaneous bleeding or any significant bleeding, hospitalization is indicated to stabilize the patient. For RhD-positive patients who have not had a splenectomy, anti-D products, such as WinRho and Rhophylac, are as effective as IVIG in refractory cases (Cines & Bussel, 2005; Raj, 2017).

Splenectomy is a second-line option; the risks and benefits of surgery must be weighed. If splenectomy is planned, immunize the patient 2 to 4 weeks before the procedure against *Streptococcus pneumoniae, Haemophilus influenzae* type b, and *Neisseria meningitidis,* encapsulated organisms that can be fatal if they infect an asplenic individual (Provan et al., 2010; Vesely, Perdue, Rizvi, Terrell, & George, 2004; Weledji, 2013). Newer therapies include the monoclonal antibody rituximab and thrombopoietic growth factors for recalcitrant cases (Arnold, 2016; Konkle, 2012; Lambert & Gernsheimer, 2017; Palau et al., 2010). Other drugs that have been used successfully include cyclosporine, antifibrinolytics, and certain chemotherapy agents.

For many adult patients with ITP and a platelet count 30,000 to 40,000, watchful monitoring is all that is required if they are asymptomatic. The course of ITP in older adults tends to be chronic with initial responses to treatment, with a reversal back to baseline after a few months.

Follow-Up: Follow-up is variable dependent on symptoms, platelet levels, and response to treatment, if needed. Patients with moderate or severe ITP will be followed by hematology.

Sequelae: Mortality rate is less than 1%, usually related to infections, bleeding, or refractory disease. There are some spontaneous remissions.

Prevention/Prophylaxis: Unless the ITP is secondary to a known autoimmune disease, there is no prevention, because it is idiopathic.

Referral: Consultation with or referral to a hematologist is indicated in cases of severe bleeding or cases refractory to treatment. Initial hematology consultation may aid in accurate diagnosis.

Education: Patients should be instructed to avoid invasive dental procedures and other activities that could precipitate bleeding episodes. Low-impact exercise is recommended.

Aspirin and NSAIDs should be avoided; alcohol intake should be limited to an occasional drink. Patients with hypertension should be encouraged to take their medications as ordered and to have frequent monitoring of their blood pressure to avoid a stroke. Periodic monitoring of platelets will be necessary.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Treatment decisions are influenced by platelet count and degree of bleeding; therapy is seldom required for platelet counts <30,000/mm^3.	C	Arnold, 2015 Palau et al., 2010
The goal of treatment is to prevent bleeding, not necessarily to return platelet count to normal.	C	Arnold, 2015 Cines & Bussel, 2005
Initial management includes treatment with oral prednisone. Other choices for treatment include high-dose dexamethasone, IVIG, and WinRho.	C	Raj, 2017 Provan et al., 2010
Chronic refractory ITP is defined as persistence for >3 months, lack of response to splenectomy, and platelet count <50,000/mm^3. Treatment options include glucocorticoids, rituximab, vincristine, cyclophosphamide, and methylprednisone.	C	Raj, 2017 Rodeghiero et al., 2009

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LEUKEMIAS

The leukemias are malignancies of the bone marrow and blood, and are classified into four main groups depending on the cell type and rate of growth: acute lymphoblastic leukemia (ALL), acute myeloid leukemia (AML), chronic lymphocytic leukemia (CLL), and chronic myeloid leukemia (CML).

ACUTE LYMPHOBLASTIC LEUKEMIA

Signal Symptoms: Bleeding from gums, bone pain, fever, frequent infections, frequent or severe nosebleeds, lymphadenopathy, paleness, shortness of breath, fatigue, weakness.

Description: ALL is an aggressive leukemia that is characterized by the presence of an increased number of lymphoblasts or lymphocytes in the bone marrow and peripheral blood (Litzow & Ferrando, 2015). The abnormal cells can also be found in the lymph nodes, spleen, liver, central nervous system (CNS), and other organs. Adult patients with ALL are at risk of developing CNS involvement. ALL usually progresses quickly. It can arise from either B or T cells, where approximately 70% to 75% of patients are diagnosed with B-cell ALL and 20% to 25% with T-cell ALL (Borowitz & Chan, 2008). ALL is divided into three subtypes by the French-American-British system: L1, L2, and L3. However, in the 2008 WHO classification, ALL was classified by the lymphoid cell of origin and the presence of cytogenetic abnormalities (Borowitz & Chan, 2008). The three main categories are: B lymphoblastic leukemia/lymphoma (B-ALL), B lymphoblastic leukemia/lymphoma with recurrent genetic abnormalities, and T lymphoblastic leukemia/lymphoma (T-ALL). It is frequently accompanied by the suppression of normal hematopoiesis. There is no clear-cut staging system for adult ALL; therefore, the division of ALL is untreated, in remission, or recurrent.

ALL expression is different based on the cell of origin (T or B cell). B-cell ALL cells typically express CD10, CD19, and CD34 on their surface, along with terminal deoxynucleotide transferase (TdT). Precursor T-cell ALL cells commonly express CD2, CD3, CD7, CD34, and TdT.

In children, there is a high cure rate from treatment; however, in adults, the prognosis is not as positive. The factors that influence the prognosis of individuals with ALL include age, WBC count at diagnosis, rapidity of remission induction, and cytogenetics (NCCN, 2016). Patients under the age of 25 years have been shown to have improved prognosis over older patients; however, a separate study has indicated that patients younger than 35 years have a better prognosis (NCCN, 2016). Certain chromosomal abnormalities may correlate with prognosis, and karyotype has been noted to be the most important predictor of disease-free survival. Philadelphia positive t (9;22) ALL is noted in more than 30% of ALL patients and has a poor prognosis. Patients with

acute leukemia may have a cytogenetic abnormality that is cytogenetically indistinguishable from the Philadelphia chromosome. The Philadelphia chromosome only occurs in 1% to 2% of patients with ALL, but occurs in about 20% of adult patients with ALL. Very few children with ALL exhibit the Philadelphia chromosome. However, this molecular abnormality is different from the Philadelphia chromosome that is characteristic of CML. While many of the patients with ALL may have the molecular evidence of the BCR-ABL fusion gene that is characteristic of the Philadelphia chromosome, there is no evidence of the abnormal chromosome by cytogenetics in those with ALL. Many of the patients with ALL will have a different fusion protein than the one found in CML.

Those leukemias that have BCR-ABL rearrangement but do not demonstrate the classical Philadelphia chromosome carry a poor prognosis that is similar to those that are Philadelphia chromosome positive. Additional chromosomal abnormalities with poor prognoses include t (4;11) and deletion of chromosome 7 or trisomy 8; these have a lower probability of survival at 5 years (NCCN, 2016).

Occurrence: The American Cancer Society (ACS) (2017) estimates that there will be approximately 5,970 new cases in 2017 and approximately 1,440 deaths from ALL in 2017.

Age: Occurs in both children and adults, but is more common in children and is the most common type of cancer in children. Four out of 10 cases occur in adults.

Gender: Incidence is slightly higher in males than in females.

Ethnicity: Incidence is higher in Caucasians than African Americans.

Contributing Factors: Risk factors include radiation exposure, chemical exposure (e.g., benzene), viral infections (e.g., HTLV-1, EBV), inherited syndromes (Down syndrome, Klinefelter syndrome, Bloom syndrome, Ataxia-telangiectasia, neurofibromatosis). Someone with an identical twin diagnosed with ALL during their first year of life has a higher risk of contracting ALL (ACS, 2017d).

Signs and Symptoms: Signs and symptoms of ALL are nonspecific and may include weakness or fatigue; lethargy; constitutional symptoms such as fever, night sweats, or weight loss; bruises or bleeding that can include the gums; purplish patches in the skin or petechia; shortness of breath; dizziness; and infection. Additional symptoms include pain in the bones or joints, enlarged lymph nodes, and swelling or discomfort in the abdomen (NCCN, 2016). Patients may present with pancytopenia, while others may have hyperleukocytosis.

Diagnostic Tests: The work-up of an individual who is thought to have ALL includes laboratory testing, including CBC with differential and platelet count, comprehensive chemistry panel, and fibrinogen and coagulation testing to screen for disseminated intravascular coagulation. A history and physical examination should be completed, including a careful screen for evidence of active infection. A bone marrow biopsy and aspirate are routinely completed to determine the extent of marrow involvement. RT-PCR and FISH should be completed in the work-up of those suspected of having ALL, especially those with B-cell lineage disease to assess for the presence of any of fusion proteins. Testing for *c-myc* should be completed because ALL is associated with a variety of translocations that involve the translocation of the *c-myc* proto-oncogene (NCCN, 2016). Tumor lysis syndrome panel, including LDH, uric acid, potassium, and phosphorus should be completed. Additional tests should be completed if the individual exhibits neurological symptoms including either a computed tomography (CT) scan or magnetic resonance imaging (MRI) of the head and a lumbar puncture. An echocardiogram is completed in patients because it is likely they will receive an anthracycline. Human leukocyte antigen (HLA) typing should be completed if the patient is expected to proceed to an allogeneic transplant during his or her treatment (NCCN, 2016).

Differential Diagnosis:

- AML
- Hairy cell leukemia
- Malignant lymphomas (e.g., high grade immunoblastic lymphoma, Mantle cell lymphoma)

Treatment: In the National Comprehensive Cancer Network (NCCN) guidelines, treatment is stratified based on the evidence of the Philadelphia chromosome and the age of the patient. The treatment of ALL consists of the control of bone marrow and systemic disease, and the treatment of disease in sanctuary sites, especially the CNS. This usually includes systemic combined chemotherapy with CNS preventive therapy. CNS prophylaxis is achieved with intrathecal chemotherapy, or high-dose chemotherapy, and in some situations, cranial radiation. Treatment is divided into three phases that include remission induction, CNS prophylaxis, and post-remission or maintenance. Treatment on a clinical trial is preferred in this population (NCCN, 2017).

Combinations such as HyperCVAD, which was developed for the treatment of aggressive lymphomas, and other combinations such as CALGB 9511, E2993, B-NHL-86, and GMALL 07/2003 are also used for front-line treatment of ALL. Sixty percent to 80% of adults with ALL achieve complete remission following induction therapy. Initial treatment usually consists of a regimen that includes vincristine, prednisone, and an anthracycline with or without asparaginase with complete response rate of up to 80%. In patients who have Philadelphia positive ALL, remission rate is higher than 90% when standard induction regimens are combined with BCR-ABL tyrosine kinase inhibitors. Remission induction in Philadelphia positive ALL includes combination chemotherapy, imatinib mesylate for patients with Philadelphia chromosome positive ALL, along with combination chemotherapy and supportive care.

The goal of consolidation therapy (also called intensification) is to eliminate any leukemic cells that are potentially remaining after induction therapy. The combination of drugs and the duration of therapy during this phase vary largely, but can include the combination of drugs that are similar to those used during the induction phase, including high-dose methotrexate, cytarabine, 6-MP, and l-asparaginase (NCCN, 2016).

Maintenance therapy is administered to prevent the relapse of the disease after post-remission induction and consolidation therapy. Most maintenance regimens are based on the backbone of 6-MP and weekly methotrexate for 2 years in adults. In patients with mature B-cell ALL, maintenance therapy is omitted because relapses are rarely seen beyond 12 months (NCCN, 2016). Supportive care during the induction

of remission should routinely include RBC and platelet transfusions, when appropriate. Prophylactic oral antibiotics may be appropriate in patients with expected prolonged, profound granulocytopenia. The use of myeloid growth factors during remission induction therapy appears to decrease the time to hematopoietic reconstitution.

CNS prophylaxis is critical to achieve control of the disease within a sanctuary site. CNS prophylaxis therapy includes cranial radiation therapy plus intrathecal methotrexate, high-dose systemic methotrexate, and intrathecal methotrexate without cranial radiation or intrathecal chemotherapy alone (NCCN, 2016; PDQ, 2016). CNS prophylaxis is given to all patients throughout the course of treatment, including induction, consolidation, and maintenance (NCCN, 2016).

When patients relapse, the response from additional treatment is low, and if a complete response is obtained, it usually is of very short duration (Douer et al., 2014). Post-remission treatment for adult ALL includes short-term, relatively intense chemotherapy followed by longer-term therapy at lower doses called maintenance therapy or an allogeneic bone marrow transplant. Optimal post-remission therapy for ALL remains unclear and, therefore, participation in a clinical trial is recommended. Treatments including the use of CAR T cells are under clinical trial currently for the treatment of patients with relapsed ALL. Blinatumomab was approved by the FDA in 2014 for the treatment of relapsed or refractory Philadelphia-negative precursor B-cell ALL (NCCN, 2016; Douer, 2014).

Patients who experience a relapse of their ALL following chemotherapy and maintenance therapy are unlikely to be cured with further chemotherapy alone. Therefore, these patients should be considered for reinduction chemotherapy followed by an allogeneic bone marrow transplantation or palliative radiation therapy. The low-dose radiation therapy may be considered in patients with symptomatic recurrence either within or outside the CNS. Patients with Philadelphia-positive ALL will often be taking imatinib at the time of relapse and are expected to have imatinib-resistant disease. Dasatinib can be used for patients with Philadelphia chromosome positive ALL that is either resistant to or intolerant of imatinib (PDQ, 2016; NCCN, 2016).

Follow-Up: Treatment for ALL can last multiple years and will have close follow-up with the oncologist while on treatment. Once treatment is concluded, the patient is followed monthly for at least 3 months, every 2 months for 6 months, and then every 3 months for up to 3 years. If the patient develops any signs of symptoms of recurrence of ALL, he or she should be referred back to their oncologist quickly.

Sequelae: Long-term myelosuppression, second malignancies, fatigue, altered quality of life, decreased concentration related to CNS treatment and prophylaxis.

Prevention/Prophylaxis: Avoid exposure to benzenes; management of associated viruses (e.g., HTLV-1).

Referral: Because of the rapid rate of progression, patients should be referred to an oncologist experienced in the care of patients with ALL as quickly as possible once abnormal blood counts are noted.

Role of the Primary Care Provider: The primary role of the primary care provider (PCP) in the care of the individual with ALL is to recognize abnormal CBC and refer the individual to an oncologist who is experienced in the care of ALL. During induction therapy, the patient usually has frequent follow-up with the oncologist and may not be seen by the PCP during that time frame. However, once patients achieve a remission, they may also be seen by the PCP. The PCP needs to be aware of potential long-term side effects of treatment. For example, osteonecrosis involves bone destruction with pain and morbidity that can be caused by glucocorticoid use. Complaints of pain in the joints, weakness, or changes in the individual's gait should be assessed immediately. A bone scan or MRI can be useful in the diagnosis. Important goals include the appropriate management of pain and the prevention of further deconditioning that are caused by immobility associated with osteonecrosis.

Some of the agents used to treat ALL are oral agents and lack of adherence can affect the outcome of treatment in this population, and is especially an issue in the young adult. The PCP needs to assess the individual for other long-term effects from treatment that can include cardiopulmonary issues associated with the chemotherapy agents. Ongoing communication between the oncologist and the PCP can assist each provider in the identification of long-term effects from treatment.

Education: Patients need to be educated regarding the potential diagnosis of leukemia and the importance of prompt referral to a hematologist. In addition, they should be educated regarding the need for ongoing follow-up. They should also be informed of the importance for ongoing follow-up with their PCP to diagnose and the timely management of comorbidities.

Clinical Recommendation: Management of ALL-Level 2

ACUTE MYELOID LEUKEMIA

Description: AML is a rare heterogeneous group of diseases that are characterized by both a defect in the differentiation of the myeloid cells, as well an uncontrolled growth of the abnormal myeloid cells that leads to the accumulation of blasts in the bone marrow (Sanz et al, 2016). The outcome is poor, with less than 5% of older patients being alive 5 years after diagnosis, as compared to 40% in the young. The reason for the poor outcome in older adults include both patient-related and disease-related issues. Patients who are older are often frail and have comorbidities that have an important impact on the tolerance of these individuals to more aggressive treatments for AML. Not only do older adults have lower rates of complete remission than the young, but the issues with intolerance to treatment are compounded by the belief that older adults have a different disease biology than younger adults. Approximately 50% of older patients

who are able to receive intensive chemotherapy will achieve a complete remission, however, they have a higher relapse rate than younger patients and, overall, the survival rates for older adults are low (Sanz et al., 2016). Older adults are more likely to have secondary AML from other diseases such as myelodysplastic disease or myeloproliferative cancers, however, secondary AML in older adults does not carry a poor prognosis in older adults as it does in younger adults. The older patient with AML is more likely to have multiple chromosomal abnormalities than the younger patient (Almeida & Ramos, 2016). There has been an improvement in supportive care, as well as changes in the treatment of AML that has affected the outcomes for younger patients; however, these changes have not improved the outcome for older patients with AML, regardless of the treatment they receive (Sanz et al., 2016).

Acute promyelocytic leukemia (APL) is a particularly aggressive subtype of AML. It has a distinct morphology and clinical presentation that may have a high early death rate due to coagulopathy. APL may be de novo or therapy related and is associated with breast cancer, hematologic malignancy, multiple sclerosis, and genitourinary malignancy.

Occurrence: It is estimated that there will be approximately 21,380 new cases and 10,590 deaths predicted for 2017 (ACS, 2017a).

Age: The median age at diagnosis is 67 years and it is more common in older adults. It is uncommon before the age of 45 years.

Gender: Slightly more common in males. Average lifetime risk for males and females is 0.5%.

Ethnicity: Not significant.

Contributing Factors: Increased risk due to:

- Tobacco smoking
- Chemical exposures (e.g., benzene)
- Chemotherapy peaks approximately 8 years after treatment with alkylating agents (e.g., cyclophosphamide, mechlorethamine, procarbazine, chlorambucil, melphalan, busulfan, carmustine) or topoisomerase II inhibitors occurring a few years after treatment (e.g., etoposide, teniposide, mitoxantrone, epirubicin, doxorubicin)
- Radiation exposure
- Blood disorders such as myeloproliferative disorders (i.e., polycythemia vera, essential thrombocythemia, idiopathic myelofibrosis) and myelodysplastic syndrome
- Family history: While no strong genetic link, there is evidence that someone with an identical twin with diagnosis of AML before 1 year of age has very high risk of AML.
- Genetic syndromes (i.e., Fanconi anemia, Bloom syndrome, ataxia-telangiectasia, Diamond-Blackfan anemia, Schwachman-Diamond syndrome, Li-Fraumeni syndrome, neurofibrosis type 1, severe congenital neutropenia, Down syndrome, Trisomy 8)

Signs and Symptoms: The symptoms of AML are often nonspecific and include fatigue, malaise, unusual bleeding, and bruising (Sanz et al., 2016). In addition, patients may experience frequent and persistent infections and bone pain. Additional signs and symptoms relate to the effect of leukemic infiltration on the body that includes angina and shortness of breath on exertion due to anemia, altered mental status, ocular effects, patchy or diffuse infiltrates of the lungs, and retinal hemorrhage due to leukostasis, bone pain, gingival leukemia, hepatomegaly, leukemia cutis, lymphadenopathy, or splenomegaly due to organ infiltration and bleeding from any site due to thrombocytopenia (Person et al., 2013).

Diagnostic Tests: The initial work-up of AML is to assess and characterize the disease based on prior exposures, antecedent myelodysplasia and karyotype, or molecular abnormalities that may provide prognostic information of AML. In addition, it is important to identify the individual's comorbidities that can affect the individual's ability to tolerate chemotherapy. The work-up includes a comprehensive medical history and physical examination. Laboratory evaluations include a CBC including platelets and differential, as well as a blood chemistry panel. Bone marrow biopsy with cytogenetics is important for risk stratification and to guide the therapy of AML. Several molecular markers (e.g., FLT3, NPM, CDBPA, KIT) can assist in outlining the prognosis of the individual with AML and assist in the treatment (NCCN, 2016). Individuals with neurological signs indicate the potential for CNS involvement of the AML. In those cases, a CT scan or MRI is an important component of the work-up; whereas a spinal tap may be done to assist in determining if CNS involvement is present. In addition, in patients who are at high risk for CNS involvement (monocytic differentiation or high WBC count), a spinal tap should be completed.

Evaluation for a coagulopathy is also important because it frequently is present at diagnosis of the disease. Echocardiograms are frequently a component of the work-up because anthracyclines are commonly included in the treatment. HLA typing is also completed if an allogeneic transplant is being considered as part of the treatment. Although cytogenetics is often not available at the time of starting treatment for AML, the karyotype is the most important prognostic factor in AML. There are also molecular markers that can help refine the prognostic groups. The tests for these molecular markers are becoming more common in commercial laboratories and in referral centers. See Table 15-2 for the risk of AML based on cytogenetics.

Differential Diagnosis:

- ALL
- Anemia
- Aplastic anemia
- B-cell lymphoma
- Bone marrow failure

TABLE 15-2 AML Risk Based on Cytogenetics

RISK GROUP	CYTOGENETIC ABNORMALITY
Better Risk	Inv(16), t(16;16), t(8;21), t(15;17)
Intermediate Risk	Normal cytogenetics, +8, t(9;11); other chromosomal abnormalities
Poor Risk	–5, 5q-, –7, 7q-, 11q23 other than t(9;11), inv(3), t(6;9), t(9;22), complex findings (≥3 clonal chromosomal abnormalities)

Data from Medscape (2016). Genetics of acute myeloid leukemia. http://emedicine.medscape.com/article/1936033-overview#a1. Retrieved 10/1/2016.

- CML
- Lymphoblastoid lymphoma
- Myelodysplastic syndrome
- Myelophthisis
- Primary myelofibrosis

Treatment: The treatment of AML is divided into induction and post-remission or consolidation therapy. The important component of induction therapy is for the individual to achieve a remission; however, the patient must complete the induction therapy in good enough shape to be able to withstand the consolidation therapy. While cytogenetic and molecular abnormalities are the most important prognostic indicators, failure to achieve remission after one cycle of induction therapy or high tumor burden, which is defined as a WBC greater than 40,000/mcL, are also included as poor risk factors. To determine if the individual has achieved remission, bone marrow biopsies are assessed at periodic time frames during treatment. The most favorable situation is in a patient with acute promyelocytic leukemia (APML) who can be treated conventionally with rather optimistic expectations.

The NCCN recommends enrollment in a clinical trial for treatment of patients with AML as the first choice of therapy (see Table 15-3). Outside the clinical trial, the NCCN guidelines divide treatment options based on the individual's age, history of prior myelodysplasia or cytotoxic therapy, and performance status. The age cut-off is 60 years of age and the potential options are divided into those who are younger than 60 years of age and those who are older (NCCN, 2016). The standard of care for patients with AML other than APML who are thought to be able to tolerate more aggressive treatment includes the "3 + 7" schedule that includes 3 days of an anthracycline (usually daunorubicin or idarubicin) and 7 days of cytarabine (Sanz et al., 2016; NCCN, 2016). It is estimated that approximately 50% of older patients will achieve a complete recovery with this regimen. Survival of the older patients who do not achieve a complete recovery from induction therapy is approximately 4 months (Burnett et al., 2011). Patients who benefit from high-dose cytarabine (HIDAC) include those who are high risk, including those with very poor risk cytogenetic abnormalities and/or FLT3-ITD mutation or with secondary AML. See Table 15-3 for treatment induction.

A bone marrow biopsy is completed 14 to 21 days after the initiation of induction therapy to determine if the patient has achieved remission from the induction therapy. In patients who received standard-dose cytarabine induction and have significant residual disease without hypoplasia, additional treatment with standard dose cytarabine and anthracycline needs to be considered. If residual blasts are noted in the marrow following treatment with cytarabine combined with daunorubicin and cladribine, a second cycle of induction should be considered. Treatment with HIDAC may be considered for the additional induction treatment. If the marrow shows significant cytoreduction and a low percentage of residual blasts, standard-dose cytarabine with an anthracycline is recommended. If the marrow is hypoplastic (cellularity less than 10% to 20% and residual blasts less than 5% to 10%), additional treatment selection can be deferred until the marrow recovers (NCCN, 2016).

In patients who achieve a complete recovery after induction therapy, consolidation can be initiated to reduce the abnormal cells to a level that can be contained by immune surveillance (NCCN, 2016). For individuals less than 60 years of age with intermediate risk disease and/or molecular abnormalities, the options of clinical trial or HIDAC are recommended with the option of proceeding to an allogeneic transplant. In those with treatment-related disease or those having poor-risk cytogenetics and/or molecular abnormalities, a clinical trial is an option or an allogeneic transplant. In patients older than 60 years, clinical trial remains the best option. Other potential options include standard dose cytarabine of 100 to 200 mg/m two times 7 days with an anthracycline or low intensity therapy such as low-dose cytarabine, 5-azacytidine, or decitabine. In patients who are not candidates for intensive anthracycline and cytarabine remission induction, clinical trial, lower-intensity therapy with 5-azacitidine, decitabine or low-dose cytarabine, or clofarabine plus cytarabine or best supportive care are options for treatment.

Older patients face a higher relapse rate than younger patients, with estimates that approximately 85% of those who achieve remission will relapse from their disease (Sanz et al., 2016). In the remainder of elderly patients with AML, intensive chemotherapy has not been shown to improve overall survival (Almeida & Ramos, 2016). Evidence indicates that patients treated with low-intensity treatments tend to live longer than those who only receive supportive care. Whenever possible, the aim should be to proceed to conventional intensive chemotherapy followed by hematopoietic stem cell transplantation. A predictive scoring system for use in patients with AML who are treated by azacytidine has been developed. Azacytidine may result in superior overall survival as compared to intensive chemotherapy or low-dose cytarabine in patients with high-risk cytogenetics, secondary AML from myelodysplastic syndrome, or patients with high-risk cytogenetics.

Hematopoietic cell transplantation (HCT) is generally recommended for AML with intermediate or high-risk cytogenetics when the aim of treatment is cure. The primary cause of death of the elderly who undergo a transplant is relapse of disease rather than toxicity. However, of those who are transplanted, an approximately 40% 3-year relapse-free and overall survival is reported (Sengsayadeth et al., 2015). It is believed that all elderly patients with good performance status and with any disease risk other than the good prognostic group should be considered for HCT, depending on the individual's HCT specific comorbidity index and the European Group for Blood and Marrow Transplantation (EBMT) risk score (Michelis et al., 2015; Sorror & Estey, 2014).

TABLE 15-3 AML Treatment Induction for Patients Over the Age of 60 Years

60 years or older (de novo AML without unfavorable cytogenetics/ molecular markers
Clinical trial
Standard-dose cytarabine (100–200 mg/m^2 continuous infusion × 7 days) with idarubicin 12 mg/m^2 × 3 days or mitoxantrone 12 mg/ m^2 × 3 days
Lower intensity therapy (low-dose cytarabine) (5-Azacytidine, decitabine)

Treatment for relapsed disease is based on the age of the patient. Patients older than 60 years who are physically fit and wish to pursue treatment may be offered a clinical trial, chemotherapy followed by a reduced intensity allogeneic stem cell transplant, or retreatment with the initial induction regimen. Best supportive care is always an option for patients who cannot tolerate or do not wish to pursue further intensive treatment (NCCN, 2016).

Treatment of APL: The major causes of death during induction treatment of APL is coagulopathy. To minimize the early induction mortality, patients who are presumed to have APL based on morphology, immunophenotyped, and/or coagulopathy with a positive disseminated intravascular coagulation (DIC) screen should promptly start all-trans-retinoic acid (ATRA) without waiting for molecular testing or cytogenetics to confirm the diagnosis. The NCCN guidelines divide patients into those with high-risk disease (WBC greater than 10,000/mcL) or low-risk disease (WBC less than or equal to 10,000/mcL) (NCCN, 2017). The patients with high-risk disease are then divided into those who are thought to be able to tolerate anthracyclines versus those who cannot. Those who are believed to not tolerate the anthracyclines are administered ATRA along with arsenic trioxide. At the time of count recovery, they are then consolidated with arsenic trioxide and ATRA on a different schedule than during induction. Those who can tolerate anthracyclines are treated with ATRA along with daunorubicin and cytarabine, ATRA with idarubicin plus arsenic trioxide. Consolidation for the individuals with high-risk disease includes ATRA plus arsenic trioxide with or without daunorubicin or daunorubicin plus or ATRA, cytarabine plus mitoxantrone plus idarubicin. The specific induction regimen guides which consolidation regimen the patient will be treated with.

Follow-Up: Following treatment for AML, patients will need to be monitored routinely. The monitoring includes CBCs, including platelet counts every 1 to 3 months for the first 2 years after patients have completed the consolidation therapy. Thereafter, they will require follow-up CBCs every 3 to 6 months for up to 5 years. Bone marrow biopsies are only performed if the CBC is abnormal (NCCN, 2016).

Sequelae: Fatigue, altered sexual function, increased risk of cardiovascular disease and death from cardiovascular events, increased risk of infectious complications (Cheng et al., 2014).

Prevention/Prophylaxis: Supportive care for patients with AML includes the use of blood products that should be leukocyte-depleted and irradiated. Patients who are expected to potentially receive an HCT at some time should have CMV screening and transfused CMV-matched blood. Tumor lysis prophylaxis includes hydration and allopurinol or rasburicase treatment. Patients who are receiving HIDAC need to be closely monitored for changes in renal function. Renal dysfunction is highly correlated with increased risk of cerebellar toxicity. Some institutions prescribe antibiotics to minimize the patient's risk of infection. Growth factors are not recommended during induction treatment for APL but may be considered during induction in AML for patients who are septic or have a life-threatening infection. Lumbar punctures are not recommended routinely in the work-up of individuals with AML; it is considered in those who exhibit neurological symptoms when CNS involvement is possible.

Referral: Obtain a CBC in patients who appear anemic and/or have extreme fatigue, unexplained high fevers, infections that are difficult to control or recur, lymphadenopathy, bruising or bleeding or petechiae, or an enlarged spleen. If symptoms persist or blood counts are normal, refer to a hematologist for further work-up.

Role of the PCP: The main role of the PCP is in the early components of diagnostic work-up. In any individual with an abnormal CBC, rapid referral to a hematologist should be considered. Treatment of an individual with AML is considered an urgent need and patients will usually be admitted to a referral center as soon as the potential diagnosis is made. After patients are discharged from the hospital after their induction treatment they are usually carefully monitored by the oncology team. If the patient contacts the PCP's office during or shortly after treatment, the PCP should communicate with the oncology team to ensure that any issues are not related to the disease or due to the treatment of AML. PCPs may be involved in the supportive care that may need to be provided to the patient. Clear delineation of roles of the care providers is necessary when patients are receiving supportive care to ensure needed care of the complications of AML are addressed.

Education: Patients need to be educated regarding the need for the CBC. In addition, the patient should be informed regarding the suspicion of the diagnosis of leukemia. Long-term survivors of AML need to be educated regarding the need for ongoing follow-up for long-term sequelae after successful treatment of AML, such as cardiovascular disease and immunizations.

Clinical Recommendation: Management of AML-Level 2

CHRONIC LYMPHOCYTIC LEUKEMIA

Signal Symptoms: Frequently asymptomatic; fatigue, feeling of fullness in abdomen, weight loss.

Description: CLL is characterized by the progressive accumulation of lymphocytes that are morphologically indistinguishable from a normal mature lymphocyte. The lymphocytes in CLL do not rapidly proliferate and are functionally inert. They have a longer life span than of normal lymphocytes (Rai & Jain, 2015). CLL and small lymphocytic lymphoma (SLL) are considered to be different manifestations of the same disease and are managed in much the same way. The difference between the two entities is that in CLL, a significant number of the abnormal lymphocytes are found in the bone marrow and blood, while in SLL, the abnormal lymphocytes are primarily found in the lymph nodes and bone marrow (NCCN, 2016). See Tables 15-4 and 15-5 for staging.

TABLE 15-4 Rai Staging System for CLL

STAGE	DESCRIPTION
0	Lymphocytosis, lymphocytes in blood >15,000/mcL and 40% lymphocytes in the bone marrow
I	Stage 0 with enlarged nodes(s)
II	Stage 0–I with splenomegaly, hepatomegaly, or both
III	Stage 0–II with hemoglobin <11.0 g/dL or hematocrit <33%
IV	Stage 0–III with platelets <100,000/mcL

TABLE 15-5 Binet Staging System for CLL

STAGE	DESCRIPTION
A	Hemoglobin ≥10 g/dL and Platelets ≥100,000/mm^3 and <three enlarged areas
B	Hemoglobin ≥10 g/dL and Platelets ≥100,000/mm^3 and ≥three enlarged areas
C	Hemoglobin <10 g/dL and/or Platelets <100,000/mm^3 and Any number of enlarged areas

Occurrence: CLL is a disease of the elderly and is the most common adult leukemia in the Western world. It accounts for approximately 25% of adult leukemias and approximately 25% of non-Hodgkin's lymphoma (NHL). It is estimated that there will be 20,110 new cases of CLL in 2017 and 4,660 deaths from the disease (ACS, 2016).

Age: Average age is 71 years; rare in those under 40 years; extremely rare in children (ACS, 2017c).

Gender: Men are more likely to contract CLL than women (ACS, 2017c).

Ethnicity: More common in North America and Europe than in Asia. Asian people who live in the United States do not have a higher risk than those living in Asia (ACS, 2017c).

Contributing Factors: The incidence of CLL is higher in individuals who have a family history (of a first-degree relative) of CLL, patients who are Caucasian, and those who have had prior Agent Orange exposure. Gender may play a role because there is a slightly higher risk in males than in females (ACS, 2017c).

Signs and Symptoms: Usually patients present with asymptomatic peripheral blood lymphocytosis or with leukocytosis that is predominantly lymphocytosis, lymphadenopathy, hepatosplenomegaly, bone marrow failure, and recurrent infections. Often, patients will have autoimmune hemolytic anemia or autoimmune thrombocytopenia (Rai & Jain, 2016).

Diagnostic Tests: According to the guidelines of the International Working Group of CLL, the criteria for the diagnosis of CLL include monoclonal B lymphocytes greater than or equal to 5,000 lymphocytes/ul in the peripheral blood for at least 3 months and less than or equal to 55% prolymphocytes and flow cytometry showing co-expression of CD5 and B-cell surface antigens CD19, CD20, CD10, and CD23 (NCCN, 2016). If flow cytometry is used to establish diagnosis, other tests to assist in making the diagnosis include cyclin D1 or FISH for t(11;14; t11q;v). In addition, CD200 and LEF1 may be useful to distinguish CLL from mantle cell lymphoma. If the diagnosis is not made by flow cytometry, then a lymph node biopsy is needed to assist in making the diagnosis. When there are fewer B cells and no other clinical features of a lymphoproliferative disorder, the diagnosis is monoclonal B-lymphocytosis, which is a relatively recent diagnostic category (NCCN, 2016).

The SLL diagnosis requires the presence of lymphadenopathy and/or splenomegaly with B lymphocytes of less than 5×10^9/L in the peripheral blood. Additional tests can be done for assistance in determining the prognosis of the individual's disease and/or to assist in determining the proper therapy for the patient. FISH is useful to detect +12, del(11q), del(13), or del(17p). The most common abnormality del(13q) is associated with favorable prognosis and the longest median survival. Del(11q) is often associated with extensive lymphadenopathy that is associated with shorter median survival. Previously untreated patients with del(11q) have been noted to respond well to combination therapy with fludarabine and cyclophosphamide, indicating that the use of an alkylating agent added to fludarabine may help to overcome the adverse prognosis. The worse prognosis is associated with TP53 and indicates short treatment-free interval, short median survival, and poor response to chemotherapy. Determination of the karyotype can help delineate complex karyotype that determines a worse prognosis.

Molecular analysis to detect IgVH mutational status is an important predictor of survival outcomes in CLL, with the unmutated IgVH being associated with poor prognosis and significantly decreased survival as compared to those with mutated IgVH. Determination of CD38 and ZAP-70 expression by flow cytometry is also useful in determining prognosis and have been associated with shorter progression-free survival and overall survival. CD38 and ZAP-70 correlated with unmutated IgVH mutational status; however, the CD38 status may vary over the course of the disease. Elevated levels of serum beta-2 microglobulin have been shown to be a strong independent prognostic indicator for the treatment-free interval, as well as overall survival. Factors that have been identified as predictors of shorter time to first treatment include increased size of cervical lymph nodes, three involved nodal sites, del(17p) or del(11q), unmutated IgVH status, and elevated serum LDH levels (NCCN, 2016).

CLL can transform into clonally related or unrelated diffuse large B cell lymphoma called Richter's transformation or Hodgkin lymphoma or interdigitating dendritic cell sarcoma (Rai et al., 2016). In patients who have transformed disease, the disease follows an aggressive course with poor response to treatment (Rai & Jain, 2016). There are two staging systems, including the Rai and Binet staging systems, that are based on the extent of involvement of the peripheral blood, lymph nodes, spleen, liver, and bone marrow by CLL (see Tables 15-4 and 15-5). These systems provide a tool to identify those patients who are suitable for observation alone versus those who require systemic therapy.

Differential Diagnosis: Infectious causes of lymphocytosis, monoclonal B cell lymphocytosis, prolymphocytic leukemia, mangle cell lymphoma, lymphoplasmacytic lymphoma,

hairy cell leukemia, follicular lymphoma, splenic marginal zone lymphoma.

Treatment: The issues of the treatment of CLL include when to treat and how to treat (Ghia & Hallek, 2014). Multiple guidelines indicate that the indication for treatment is based on the presence of active disease. Symptoms of "active disease" include progressive bone marrow failure that is demonstrated by anemia and/or thrombocytopenia, bulky disease with massive or rapidly progressive lymphadenopathy (10 cm or greater than 6 cm below the left costal margin), or splenomegaly that is rapidly progressive; uncontrolled autoimmune cytopenia that does not respond to steroids; rapid lymphocyte doubling time (less than 6 months); and the presence of B symptoms (Scarfo et al., 2016). Patients who have CLL and are asymptomatic with early stage disease should be monitored (Eichhorst et al., 2016).

Patients with CLL are divided into fit or "go-go" versus unfit or "slow-go." Patients in the fit or "go-go" category are candidates to receive a chemoimmunotherapy combination with fludarabine, cyclophosphamide, and rituximab (FCR) if they are experiencing progressive disease (Scarfo et al., 2016). Alternative options for treatment includes bendamustine with rituximab, which is better tolerated than FCR but has a lower rate of complete responses. Individuals who have relevant comorbidities and/or impaired renal function are considered unfit or "slow-go." This category represents the majority of patients with CLL. FCR-lite with decreased doses of fludarabine and cyclophosphamide over FCR, bendamustine as single-agent therapy, or bendamustine with rituximab are potential options. Patients who have high-risk categories include those with del(17p) and/or TP53. Some of these patients may not initially require therapy because they may initially have indolent disease. When they are noted to have progressive disease, they are felt to have a more adverse clinical course. It is recommended that these patients be enrolled in clinical trials that includes novel agents that work independent of the TP53 pathway.

Recently, novel agents have been approved by the FDA, including ibrutinib and idelalisib. Ibrutinib inhibits the bruton-tyrosine kinase (BTK) pathway that is a key component downstream to the B-cell receptor pathway (Scarfo et al., 2016). Ibrutinib irreversibly binds to BTK and causes kinase function inactivation. Overall response rates of 90% and progression-free survival of 75% at 26 months have been reported with the use of ibrutinib in CLL. The response has been independent of many of the disease risk factors. Ibrutinib is overall well tolerated. The plan of treatment is long-term and patients need to be educated for the need to remain on the drug until progression when deciding on treatment. Following initiation of treatment with ibrutinib, patients can develop significant lymphocytosis that is due to redistribution of lymphocytes from the lymph nodes into the peripheral blood. It can take several months to have normalization of the lymphocyte count. The presence of the lymphocytosis and its severity is not a prognostic factor in the success of controlling the CLL. Potential side effects of ibrutinib include diarrhea, infection, arthralgia, and fatigue. Because ibrutinib also inhibits other kinases, this can result in impaired platelet function and lead to an increased risk of bleeding. In prior studies, concomitant use of warfarin and ibrutinib was contraindicated, although the use of other anticoagulants and antiplatelet agents was fairly common and has been found to have little effect on major hemorrhagic complications with ibrutinib (Routledge & Bloor, 2016). There has been a report of increased risk of atrial fibrillation in about 5% of patients. Ibrutinib is now being combined with other agents to manage CLL.

Idelalisib is a potent inhibitor of PI3K delta isoform that is predominantly expressed in hematopoietic cells, especially B cells, and is highly effective in managing CLL. Overall response of 72% has been reported using idelalisib for the treatment of relapsed CLL and has been combined with anti-CD20 antibodies such as rituximab and ofatumumab. However, there have been no studies comparing the use of idelalisib alone versus idelalisib and rituximab. Idelalisib has also been combined with chemotherapy agents. As is seen with ibrutinib, many patients show a progressive lymphocytosis after starting idelalisib. Treatment is overall well tolerated, with the most common toxicities including fever, fatigue, neutropenia, and altered liver function studies. Immune-mediated diarrhea/colitis and pneumonitis can complicate the treatment. The onset of these toxicities can be delayed for several months after starting treatment. Many patients respond to steroids and are able to continue treatment (Routledge & Bloor, 2016). The usefulness of idelalisib as first-line therapy is unclear at this time.

The management of CLL may include the target of the microenvironment with the use of such agents as lenalidomide and ventoclax. The use of lenalidomide has been shown to be effective in relapsed/refractory CLL with overall response rate 32% to 47%. Higher response rates have been reported with lenalidomide in combination with anti-CD20 antibodies in untreated (overall response rate 79% to 85%) and relapsed disease (overall response rate 48% to 62%). Toxicities from lenalidomide include neutropenia and tumor flare reaction. The side effects seem to be dose related.

Venetoclax promotes apoptosis. It has been shown to have an overall response rate of 70% in patients who have disease that is resistant to fludarabine and those with TP53 dysregulation. Responses have been shown in both the bone marrow, as well as in the lymph nodes. Tumor lysis has been noted that leads to the recommendation to use a lower starting dose with stepwise dose escalation along with prophylaxis for tumor lysis syndrome.

Deciding on the specific treatment in patients with CLL depends on the frailty of the patient, as well as the specific risk factors of the disease, including del(11q) or del(17p)/TP53 (NCCN, 2016). Also, age is counted into the factors that are important when selecting the treatment for CLL. There are many treatment options available to manage this disease. An additional factor to consider when determining the best treatment at any given time is to consider the patient's comorbidities (e.g., atrial fibrillation, anticoagulation). Transplant is considered as a potential option for patients with CLL, but usually is reserved for patients who are young or those who have recurrent disease and have previously received the majority of the treatments available. See Table 15-6 for treatment options.

Follow-Up: Patients with CLL should be followed at least every 3 months by an oncologist to monitor their blood counts. At times, if the patient has monoclonal lymphocytosis (absolute lymphocyte count [ALC] less than 5,000), the oncologist may

TABLE 15-6 Potential Treatment Options for First-Line Treatment of CLL

Obinutuzumab + chlorambucil
Ibrutinib
Ofatumumab + chlorambucil
Rituximab + chlorambucil
Bendaumstine ± rituximab (starting 70–90 mg/m^2)
Obinutuzumab
Fludarabine ± rituximab
Chlorambucil
Rituximab
Pulse corticosteroids
FCR (fludarabine, cyclophosphamide, rituximab)
FR (fludarabine, rituximab)
PCR (pentostatin, cyclophosphamide, rituximab)
Bendamustine ± rituximab
Idelalisib
Fludarabine + alemtuzumab
RCHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, prednisone)
OFAR (oxaliplatin, fludarabine, cytarabine, rituximab)
Obinutuzumab
Lenalidomide ± rituximab
Alemtuzumab ± rituximab
HDMP + rituximab
Dose Dense Rituximab

Data from NCCN (2016). NCCN Guidelines Version 3.2016. Non-Hodgkin lymphoma. Retrieved from www.nccn.org.

refer the patient to the PCP for follow-up with the expectation that if the ALC goes over 5,000, they would be referred back to the hematologist for work-up and treatment. Patients with CLL are often treated with antibiotics when having symptoms of infection, and if their IgG is below 400 they may also require IVIG infusions. The PCP also needs to be cognizant of the patient's potential for infections as a component of their CLL (especially upper respiratory infections).

Sequelae: Recurrent infections; autoimmune hemolytic anemia; autoimmune thrombocytopenia; myelosuppression related to treatment; atrial fibrillation from ibrutinib; immune-mediated diarrhea/colitis from idelalisib; tumor lysis from treatment including rituximab, ofatumumab, venetoclax, or obinutuzumab; reactivation of hepatitis B from rituximab, ofatumumab or obinutuzumab.

Prevention/Prophylaxis: There is no evidence of methods to prevent CLL.

Referral: If patients are noted to have an abnormal blood count manifested by increased lymphocyte count, they should be referred to a hematologist for evaluation and work-up. In addition, patients with lymphadenopathy, hepatomegaly, or splenomegaly also should be referred for evaluation. Usually, they should be referred to a hematologist; however, for the work-up of lymphadenopathy, a referral to a surgeon or interventional radiologist is a potential alternative.

Role of the Primary Care Provider: Patients with CLL frequently see their PCP during the management of their CLL. Most often, it is the PCP who first notes the evidence of lymphocytosis and will need to refer patients to the oncologist for additional work-up. Many patients will initially be observed early in the trajectory of their disease. The PCP needs to understand the treatment plan of each patient. Patients may report potential symptoms that can be related to progression of their disease and the PCP needs to inform the oncologist of these symptoms, as it may indicate that the patient needs to initiate treatment. When on treatment, the PCP needs to be aware of any potential comorbidities that may impact the individual's ability to remain on certain treatments (e.g., anticoagulants). Patients with the diagnosis of CLL are recommended to receive immunizations, regardless of their age, such as annual influenza vaccine and pneumococcal vaccine every 5 years (NCCN, 2016). All live vaccines should be avoided.

Patients with a prior history of hepatitis B are at increased risk of reactivation of the hepatitis B if they receive rituximab or other CD20-targeted therapies. All patients should be tested for their immune status to hepatitis B before starting CD20-targeted therapy. If they test positive for prior hepatitis B, some patients may require hepatitis B virus (HBV) prophylaxis and monitoring. Patients with CLL should receive blood products that are irradiated to avoid transfusion-associated graft-versus-host disease (GVHD) (NCCN, 2016). Patients who are receiving certain agents (e.g., fludarabine and/or alemtuzumab) should receive anti-infective prophylaxis for herpes and pneumocystis pneumonia (PCP).

Education: Patients need to be educated regarding the potential for recurrent infections related to the diagnosis of CLL. Due to the potential of myelosuppression, patients should be educated to report fevers over 100.4°F as soon as they happen so that a CBC can be completed to determine if they are also neutropenic, which would require prompt management with systemic antibiotics. They should also be aware to notify their oncologist for the development of any new comorbidities (e.g., atrial fibrillation), as they may be related to their treatment. They also should be aware to avoid live vaccines, but other vaccines are acceptable and vaccines such as flu and pneumococcal vaccines are recommended.

Clinical Recommendation: Treatment of CLL-Level 2

CHRONIC MYELOID LEUKEMIA

Signal Symptoms: Asymptomatic; frequently diagnosed following routine laboratory work with abnormal results. Manifestation of symptomatology is dependent upon the phase; see Description and Signs and Symptoms for details.

Description: CML is a myeloproliferative neoplasm. It is classified into three phases: chronic phase (CP), accelerated phase (AP), and blast phase (BP). The majority of patients (90% to 95%) present in CP. CML can transform to a more aggressive form of disease (AP or BP). Most patients evolve into AP prior to BP; however, about 20% transform to BP without signs of AP. CML-BP presents as an acute leukemia, including 60% myeloid, 30% lymphoid, and megakaryocytic or undifferentiated in 10% (Jabbour & Kantarjian, 2016).

Etiology: Expression of oncoprotein BCR-ABL is caused by the fusion of the Abelson murine leukemia gene (ABL1) on chromosome 9 with the breakpoint cluster region (BCR) gene on chromosome 22. This rearrangement is known as the Philadelphia chromosome (Jabbour & Kantarjian, 2016). Increased risk occurs with radiation exposure and increased age.

Occurrence: Incidence of CML is 8,960 estimated for 2016 (ACS, 2016; Jabbour & Kantarjian, 2016). CML accounts for an estimated 15% of newly diagnosed cases of leukemia in adults. There are an estimated 1,070 deaths from CML expected in the same year (ACS, 2016). The rate of death from CML has been noted to decrease dramatically since the introduction of imatinib in 2000 from 10% to 20% to 1% to 2% (Jabbour & Kantarjian, 2016).

Age: The average age at diagnosis is 64 years; almost half are 65 years and older at diagnosis (ACS, 2017b).

Gender: About equal, but various references indicate CML is more common in females (ACS, 2017b), while others indicate it is more common in males (SEER, 2010–2014).

Ethnicity: SEER data indicates that CML is most common in Caucasians and least common in Asians/Pacific Islanders (SEER, 2010–2014).

Signs and Symptoms: Symptoms of CML are often dependent on the stage of the disease: CP, AP, or BP. When symptoms are present during CP, they result from anemia and splenomegaly, which include fatigue, weight loss, malaise, early satiety, and left upper quadrant fullness or pain. Rare manifestations of CP include bleeding (from a low platelet count and/or platelet dysfunction), thrombosis (associated with thrombocytosis and/or marked leukocytosis), gouty arthritis (from elevated uric acid levels), priapism (usually with marked leukocytosis or thrombocytosis), retinal hemorrhages, and upper GI ulceration and bleeding (from elevated histamine levels due to basophilia). Symptoms can also include those due to leukemic cells sludging in the pulmonary or cerebral vessels, such as dyspnea, drowsiness, loss of coordination, and confusion, and are uncommon in individuals diagnosed with CML-CP.

In approximately 40% to 50% of cases, splenomegaly is the most consistent physical sign, while hepatomegaly is less common. It is rare for patients to present with lymphadenopathy and infiltration of the skin or other tissues. However, when lymphadenopathy or infiltration of the skin or tissues is present at the time of diagnosis, the individual usually has Philadelphia-negative CML or CML-AP or CML-BP. Headaches, bone pain, arthralgias, pain from splenic infarction, and fever are more frequent with transformation of CML. CML-AP can present with worsening anemia, splenomegaly, and organ infiltration. CML-BP presents as an acute leukemia with worsening constitutional symptoms, bleeding, fever, and infections (Jabbour & Kantarjian, 2016).

Diagnostic Tests: Routine cytogenetics or by fluorescence *in situ* hybridization (FISH); quantitative reverse transcriptase-polymerase chain reaction (RT-PCR); bone marrow biopsy mandatory for diagnostic work-up and provides information needed for staging (CP, AP, or BP) (Jabbour & Kantarjian, 2016).

Differential Diagnosis: Must be differentiated from leukemoid reactions, effect from corticosteroids, myeloproliferative disorders or myelodysplastic syndromes, agnogenic myeloid metaplasia, polycythemia vera with associated iron deficiency, Philadelphia-negative CML, chronic myelomonocytic leukemia, myeloid hyperplasia, and essential thrombocythemia (Jabbour & Kantarjian, 2016).

Treatment: The presence of the BCR-ABL1 gene causes deregulation of tyrosine kinase activity. Key to the improved management of CML is the finding that turning the BCR-ABL1 gene off would eliminate the leukemic cells while sparing normal cells. In 2002, imatinib became the gold standard for front-line treatment of CML (Soverini et al., 2016; Johnson et al., 2003). There are currently three tyrosine kinase inhibitors (TKIs) available for front-line therapy of CML-CP: imatinib, dasatinib, and nilotinib. Imatinib inhibits the BCR-ABL1 kinase along with blocking the platelet-derived growth factor receptor and the c-KIT tyrosine kinase (Jabbour & Kantarjian, 2016). The standard dose is 400 mg/day. With reports of 8-year follow-up of the IRIS trial, the estimated event-free survival rate was 81%, with an overall survival rate of 93%. Common side effects with imatinib include peripheral edema, GI toxicities such as nausea and diarrhea, myalgias, musculoskeletal symptoms, and rash (Bhalla et al., 2016).

Dasatinib is an oral, second generation TKI that is 350 times more potent than imatinib and also inhibits the Src family of kinases. Dasatinib is approved as salvage therapy for those who have inadequate response to imatinib, as well as for front-line treatment. Patients receiving dasatinib achieved complete cytogenetic response at 12 months more often (77%) than those who were receiving imatinib (66%). Dasatinib was also showed to induce more rapid and deeper response as compared to imatinib. At the dosing schedule of 100 mg/day of dasatinib, pleural effusions occur more frequently that with imatinib. Other side effects of dasatinib include myelosuppression and rare pulmonary hypertension (Jabbour & Kantarjian, 2016).

Nilotinib is a structural analog of imatinib with an increased affinity for the ATP-binding site on BCR-ABL1 increased 30 to 50 times over imatinib. Nilotinib has been shown to have the ability to induce hematologic and cytogenetic responses in patients who have not obtained adequate

TABLE 15-7 Side Effects of Tyrosine Kinase Inhibitors in CML

Myelosuppression
Edema
Nausea
Muscle cramps
Musculoskeletal pain
Diarrhea
Skin problems
Fatigue
Abdominal pain
Headache
Joint pain
GI bleed
Elevated pancreatic enzymes
Pancreatitis
Elevated liver function tests
Decline of GFR
Hypophosphatemia
Altered thyroid hormones
Altered testosterone level
QT prolongation
Venous and arterial thrombotic events
Pleural and pericardial effusions
Pulmonary arterial hypertension
Secondary malignancies

Data from Caldemeyer, L., Dugan, M., Edwards, J., & Akard, L. (2016). Long-term side effects of tyrosine kinase inhibitors in chronic myeloid leukemia. *Current Hematolic Malignancy Reports, 11,* 71–79.

response to imatinib. As with dasatinib, nilotinib has been compared to imatinib in a large, international, randomized study (ENEST-nd) and was found to achieve higher rates of major molecular response at 12 months (43%) than those receiving imatinib (22%). Overall, nilotinib is well tolerated; however, there has been shown to be an increased risk of accumulated vascular events in patients who receive nilotinib. Other side effects are headache, skin rashes, self-limiting elevation of indirect bilirubin, elevations of blood sugar, and rare pancreatitis (Jabbour & Kantarjian, 2016). See Table 15-7 for side effects of TKIs.

Current guidelines recommend any of the three TKIs for first-line therapy: imatinib, dasatinib, or nilotinib for the treatment of CML-CP (NCCN, 2016). While second generation TKIs have been shown to produce higher rates of early optimal responses, second generation TKIs have not yet been shown to effect long-term survival. The main advantage of the second generation TKIs is in patients with high-risk disease, with a decrease in the rate of transformation to AP-CML or BP-CML (Jabbour & Kantarjian, 2016). Allogeneic stem cell transplant or other chemotherapy agents are not recommended for first-line therapy for CML-CP due to the excellent outcomes and long-term survival with the TKIs. Selection of front-line therapy is based on the patient's age, comorbidities, and the TKI toxicity profile. In addition, cost needs to be considered, as imatinib is now available in generic formulation.

Bosutinib is another TKI that is approved in the treatment of CML. It appears to retain activity across most known mutations that confer resistance to imatinib, except for T325I. The most common toxicities were diarrhea, nausea, vomiting, and rash. Diarrhea occurs in approximately 84% of patients. In addition, additional side effects include myelosuppression and alterations in liver function tests (Cortes et al., 2011).

Ponatinib is a third-generation TKI and exhibits activity against CML with the T315I mutation. It is 500 times as potent as imatinib at the inhibition of BCR-ABL1. The response rates indicate that 56% of patients who were either resistant to or intolerant to either dasatinib or nilotinib achieved major cytogenetic response by 12 months (Cortes et al., 2014).

There are distinct differences in the occurrence of specific side effects of the various TKIs. Nilotinib has little incidence of fluid retention and has the least myelosuppression among the TKIs; however, it has more rash, headache, pancreatitis, and cardiovascular effects than does imatinib. Dasatinib has less peripheral edema, but more GI toxicity and pleural effusions as compared to imatinib. Bosutinib has more diarrhea, vomiting, and abdominal pain, but less edema and muscle pain than reported of imatinib. Ponatinib has more rash, abdominal pain, headache, and pancreatitis, but less nausea, muscle pain, diarrhea, and cardiovascular effects than imatinib (Jabbour & Kantarjian, 2016).

Cardiac arrhythmias have been reported with QT prolongation. The frequency of QT prolongation appears highest with nilotinib. However, based on reported studies, there has not been a clear association of CML with increased venous or arterial thrombotic events. There has been a recent study from Sweden that individuals treated for CML-CP had a higher risk of arterial and venous thrombotic events, and the risk of myocardial infarction was higher for nilotinib and dasatinib as compared to imatinib. All BCR-ABL1 TKIs have been associated with the development of pleural or pericardial effusions; dasatinib has had the highest likelihood of these specific cardiac and pulmonary toxicities, including pulmonary arterial hypertension. Pulmonary arterial hypertension has been reported in patients receiving dasatinib. Additional side effects include hypophosphatemia, altered LFTs, altered thyroid function tests, decline of GFR, elevated pancreatic enzymes and pancreatitis, GI bleeds, and altered testosterone levels.

There are varied reports about the potential increased risk of second cancers in patients who have received TKIs as treatment for CML (Caldemeyer et al., 2016). At times, the side effects can be managed or may require a reduction in the dose of the medication. If not controlled by these methods, a change in the TKI may be necessary. While approximately 27% of patients diagnosed with CML are under the age of 50 years, fertility and pregnancy issues have not been adequately studied. This makes giving patients recommendations regarding the desire to become pregnant a difficult one, with no clear information to outline the risks to the fetus or to the

TABLE 15-8 Measurement of Response of Treatment with TKI for CML

LEVEL OF RESPONSE	DEFINITION	TEST
Hematologic	Complete hematologic response is noted when the peripheral blood counts are normal with no immature blood cells, leukocyte count is less than 10×10^9/L, and platelet count is less than $450{,}000 \times 10^9$/L.	CBC
Cytogenetic	Cytogenetic response is noted when there is a decrease in the number of Philadelphia-positive metaphases, as determined by bone marrow aspirate and cytogenetics.	Bone Marrow Cytogenetics
Molecular	Decrease in the amount of BCR-ABL1 chimeric mRNA as measured by the RT-PCR.	Quantitative Real Time-Polymerase Chain Reaction (RT-PCR)

Adapted from NCCN. (2016). Chronic myelogenous leukemia. Version 1.2016. Retrieved from www.nccn.org.

individual who questions the risks of stopping treatment to allow child bearing.

Patients receiving TKI therapy must be aware of potential drug–drug interactions. Patients must be advised to discuss with their pharmacists the potential for potential drug interactions for any of the medications or supplements they are taking or are prescribed after starting the TKI (Bhalla et al., 2016).

Follow-Up: There are three components to response from treatment of CML: hematologic response, cytogenetic response, and molecular response. See Table 15-8 for response description. Monitoring for response when on treatment with a TKI requires a RQ-PCR that is commonly repeated monthly for the first 12 months and at least every 3 months thereafter (Bhalla et al., 2016). Currently, the recommended length of treatment is lifelong; however, there may be a select group of patients who are able to maintain their response even after their treatment is discontinued. Almost all patents who stop treatment with imatinib and had recurrence of their disease were able to achieve CMR once the imatinib was restarted. While the results of these trials are encouraging, the current recommendation is that patients not stop treatment unless under a clinical trial (Jabbour & Kantarjian, 2016).

Sequelae: Adherence is key to the individual diagnosed with CML. Multiple studies have demonstrated a clear correlation between adherence and clinical outcome in CML. In a recent study by Anderson and colleagues (2015), approximately 31% of patients on imatinib were noted to be nonadherent with their prescribed medication regimen. This group indicated that the patients who were more likely to be nonadherent included younger patients (less than 50 years old), those who were not taking other medications, and patients on imatinib. One factor related to adherence is the dietary requirements that are associated with each of the specific TKIs. Imatinib has to be taken with a big meal, while nilotinib is to be taken twice a day on an empty stomach. Dasatinib has to be taken with a full glass of water. In addition, there are certain other restrictions that can also be problematic for patients who are taking a TKI. The use of a proton pump inhibitor for GERD is contraindicated in those taking a TKI. Other medications that can cause a prolonged QT interval should be either discontinued or require increased monitoring. These two issues can affect the individual's ability to be adherent to their medication regimen (Flynn & Atallah, 2016).

Prevention/Prophylaxis: There are no known methods to prevent the occurrence of CML.

Referral: Patients who are noted to have abnormal blood counts should be referred to a hematologist for work-up. A bone marrow biopsy is key to making the diagnosis of CML or determining if the cause of the abnormal counts is from an alternate diagnosis. Patients with CML are often also followed by their PCP while receiving what is thought to be lifelong treatment. The PCP needs to be knowledgeable about the treatments for CML and their possible side effects.

Role of the Primary Care Provider: In many cases, the patients are without symptoms and recognition of abnormal blood counts can lead to the referral for work-up. Additionally, communication with the oncologist related to side effects the patient may be experiencing is important to assist the patient to be adherent in the treatment. Encouraging patients to discuss issues they may be having taking the medication may assist the patient to maintain adherence to the treatment plan. If the side effects are not manageable, the patient may need to have the treatment changed to an alternate medication. In addition, if the patient develops additional comorbidities, the treatment of the comorbidities may need specific medications that are less likely to interact with the TKI due to the multiple potential drug–drug interactions with the TKIs. Continued discussion with the patient about long-term goals and quality of life as it relates to and is impacted by CML is key to ensuring the patient will continue to adhere to the treatment.

Education: Patients need to be educated regarding the importance in adhering to the treatment plan and taking the prescribed medication as directed. They also need to be educated regarding the potential side effects of the specific drug they are taking for managing the CML. Clear communication between the patient, the PCP, the pharmacist, and the oncologist is essential to prevent potential drug–drug interactions. Patients need to understand the importance of lifelong treatment until clear data exists regarding the ability to discontinue treatment safely.

Clinical Recommendations: Use of first and second generation TKIs: Level A.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Suspect ALL in patients with symptoms and signs related to cytopenias.	C	Fava & Jabbour, 2017
Initial diagnostic testing for ALL should include CBC, chemistries, coagulation studies, and bone marrow with cytochemistry and immunophenotyping.	C	Fava & Jabbour, 2017
Current management strategies for adult patients with ALL require a careful assessment of relapse risk at treatment initiation.	B	Fava & Jabbour, 2017
CML is a myeloproliferative disorder characterized by the presence of the Philadelphia chromosome. Patients with CML typically present with vague symptoms, such as fatigue or weight loss, or may be asymptomatic. WBC count reveals severe leukocytosis with left shift, basophilia, and/or thrombocytosis.	C	Ghanem & Jabbour, 2017a
Imatinib, an oral TKI, is recommended as a standard treatment for chronic phase CML; it delays the progression of the disease.	A	Ghanem & Jabbour, 2017a
AML is the most common form of acute leukemia in adults. Patients present with insidious symptoms related to variable degrees of bone marrow failure.	C	Ghanem & Jabbour, 2017b
The diagnosis of AML is demonstrated by increased number of myeloblasts (>20%) in the bone marrow or the peripheral blood. The cornerstone of treatment is chemotherapy given in two phases: induction and consolidation. Bone marrow transplantation and investigational drugs may be used in selected patients.	B	Ghanem & Jabbour, 2017b

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

Mrs. L., a 68-year-old woman, presents to your office today with a complaint of feeling tired all the time and now, more recently, feeling weak and like "I can't catch my breath sometimes." She has been healthy except for high cholesterol, managed by Lipitor. Her husband died 9 months ago, and she has attributed her fatigue to dealing with his death, but realizes that she is feeling worse and not better as time passes.

No known drug allergies, takes only Lipitor. Past surgical history: Appendectomy in childhood; hysterectomy for uterine myoma 10 years ago. No significant medical history. Has two daughters living nearby. Blood pressure (BP) 106/70 mm Hg, heart rate (HR) 98 beats/min and regular, respiratory rate 18 breaths/min and afebrile, body mass index (BMI) 22 (10-pound weight loss since death of husband).

Slender, quiet-spoken older woman appearing tired. Conjunctiva pale, mucous membranes moist. No lymphadenopathy of neck or femoral area. Chest CT angiogram (CTA), good air movement. Heart tachyarrhythmic with regular rate, soft midsystolic murmur. Abdomen soft, bowel sounds × 4. Urine dipstick negative.

1. What additional subjective data are you seeking?
2. What additional objective data will you be assessing for?
3. What national guidelines are appropriate to consider?

CASE STUDY—cont'd

4. What tests will you order?
5. Are there any screening tools that you want to use?
6. What are the differential diagnoses that you are considering?
7. What is your plan of care?
8. Are there any *Healthy People 2020* objectives that you should consider?
9. What additional patient teaching may be needed?
10. Will you be looking for a consultation?

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Acute Myelogenous Leukemia

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CHAPTER 16

Psychosocial Disorders

Lori Martin-Plank

ASSESSMENT

Psychosocial assessment of the older adult is the systematic review and evaluation of patient, family, and environment. The psychosocial assessment provides the foundation for developing and implementing a comprehensive plan of care and a means for evaluating its effectiveness. The practitioner must understand what happens during the aging process, because this knowledge allows differentiation between symptoms that are normal and those considered abnormal. For the older adult, physical health, mental health, spirituality, and environmental and social problems all interact to complicate the life and function of the older patient, caregiver, and family.

The psychosocial assessment of the older adult entails evaluation of the following basic needs:

- Autonomy and independence
- Dignity, credibility, and respect
- Identity and individuality
- Communication and belonging
- Touch

An accurate psychosocial assessment is an interactive process between the practitioner and the older adult that creates an awareness of risks, limitations, and functional changes. This enables the older adult to make appropriate lifestyle changes to accommodate these changes and maintain autonomy, independence, and dignity. The psychosocial assessment should be performed at the initial encounter and annually thereafter for all older adults. Reassessment is appropriate whenever a patient's health status changes.

Ethnicity: Ethnicity may be defined as affiliation with a group whose members share a common social and cultural heritage that is passed on to successive generations and provides a sense of identity. To provide culturally competent care, the nurse practitioner should perform a cultural assessment within the psychosocial assessment for a multicultural society. Specific to older adults, ethnic traditions and expectations are factors that should be addressed in the psychosocial assessment.

Contributing Factors: A positive correlation exists between social supports and the use of health-care services by older adults. Older adults who are alone and lonely, depressed, or having difficulty adapting to change appear in their practitioner's office more frequently than those with adequate support systems. Social networks are an important aspect of a patient's social functions, as are work, hobbies, and interests. Older adults may be reluctant to reveal social or emotional concerns and, in some cultures, may feel that it is unacceptable to share personal problems with outsiders. However, they may feel comfortable discussing a physical manifestation, such as pain or sleeplessness, with a health-care provider.

Risk Factors: The older adult population is not a homogeneous group, rather, the population demonstrates a diversity in health, social and environmental supports, financial security, and cultural and personal philosophies. Some distinctive occurrences in the lives of older adults may include:

- Developmental milestones that have an impact on most older adults (e.g., retirement, loss of a spouse)
- Changes in financial and social resources that allow the older adult to cope effectively with age-related phenomena

Relocation, financial concerns, and lack of social supports may be the impetus for physical and mental health problems. The most common life events that place the older adult at risk for psychosocial dysfunction include:

- Retirement/role loss
- Loss of spouse
- Deaths of close friends
- Family problems
- Relocation
- Financial problems

Psychological Health

Because older adult patients may hesitate to discuss social or emotional problems with a health-care provider, physical symptoms may be the chief expression of underlying psychosocial dysfunction. Therefore, measurement of psychological health adds an important component to the older adult assessment. Psychological health is measured based on the two subdomains of cognition (mental status) and affect (anxiety and depression). A variety of tools, mentioned

throughout this text, can be used for cognitive and emotional health assessment. Although helpful, screening tools should not replace the interactive relationship between practitioner and patient.

Socioenvironmental Tools

The heterogeneity of socioenvironmental factors precludes the use of a single tool for evaluating the older adult; however, to evaluate this domain, the environmental and safety checklist may be helpful (see Internet Home Safety Resources for Older Adults), and it may also be used for patient and family education. The information derived from this evaluation will direct the practitioner in areas of education for the patient and family.

Expected Outcomes: Chronic disease, physical disability, pain and suffering, cognitive impairment, accumulated losses, and social isolation may occur when an individual is least able to cope with change. Therefore, a psychosocial assessment provides a foundation for developing and implementing a comprehensive plan of care. The expected outcome for the older adult is an enhanced quality of life.

Internet Home Safety Resources for Older Adults

www.aota.org/documentvault/documents/41878.aspx
www.cpsc.gov/cpscpub/pubs/701.pdf
www.cpsc.gov/cpscpub/pubs/older.html
http://michigan.gov/mdch/0,4612,7-132-8347-261897—,00.html
www.ces.ncsu.edu/depts/fcs/pdfs/FCS-461.pdf
http://orthoinfo.aaos.org/topic.cfm?topic<\#61>A00123
www.seniorresource.com/Senior_Home_Safety_Checklist.htm#chklst
www.choosehomecare.com/home_safety_checklist.html

AGITATION

Signal Symptoms: Change in behavior, also known as neuropsychiatric symptoms of dementia.

Description: Occur in clusters or syndromes identified as psychosis and manifested as delusions, hallucinations, aggression, anxiety, nighttime awakening, and socially inappropriate behavior.

Etiology: Symptoms are related to changes in the brain structure and function, particularly in the brain circuits and networks that are involved in motivated behavior, emotions, and cognitive processing (Nowrangi, Lyketsos, & Rosenberg, 2015).

Occurrence: Approximately 80% to 90% of patients with dementia will have episodes of agitation. Patients with Parkinson's disease, stroke, and other brain disorders may also experience agitation (Kales, Gitlin, & Lyketsos, 2015; Nowrangi, Lyketsos, & Rosenberg, 2015).

Age: Predominantly in older adults.

Gender: Both, but a higher incidence in females.

Ethnicity: African American and Latino community-dwelling patients with moderate to severe dementia have a higher prevalence of dementia-related behaviors than Caucasians (Lines, Sherif, & Wiener, 2014).

Contributing Factors: Cognitive impairment, sensory impairment, social isolation, chronic bedrest, pain, and hunger can precipitate episodes of agitation. Environmental triggers, such as noise, light, and visual cues from television and physical surroundings, can also lead to agitation. Psychosocial triggers, such as the approach taken by staff, interaction with other residents, and anxiety during personal care, may cause a person to become agitated (Kales, Gitlin, & Lyketsos, 2015).

Signs and Symptoms: Delusions and hallucinations, especially new in onset. Behaviors may be displayed in multiple ways. Common behaviors include repeating questions, arguing, pacing, screaming, crying out, making disruptive sounds, physical or verbal aggression, and shadowing a caregiver (Kales, Gitlin, & Lyketsos, 2015).

Diagnostic Tests: Infection panel, complete metabolic panel (CMP), vitamin B_{12} level, and thyroid-stimulating hormone (TSH) to rule out medical cause of agitation (Cummings et al., 2015).

Differential Diagnosis:

- Delirium
- Psychosis
- Mania
- Infection
- Medication side effects
- Late-life delusional disorder
- Depression
- Pain
- Seizures

Treatment: There are two types of treatment for agitation: psychotropic medications and behavioral interventions. Psychotropic medications include antipsychotics, anxiolytics, and antidepressants. All three medications fall on the Beers criteria list, where they are described as "requiring caution when prescribing for the older adult." Antipsychotics should be used only if there is evidence of psychosis. Antiseizure drugs are used for manic-like symptoms. Evidence supports the use of antiseizure medications in lieu of antipsychotics. Anxiolytics treat the symptoms of anxiety that often accompany agitation. If there is evidence of depression, an antidepressant may be indicated. Behavioral interventions fall under the categories of individual approach, environmental, alternative modalities, and staff and caregiver education (Kales, Gitlin, & Lyketsos, 2015; Nowrangi, Lyketsos, & Rosenberg, 2015). Tables 16-1 and 16-2 outline the most commonly prescribed psychotropic medications and behavioral interventions.

Follow-Up: Review laboratory reports, treat any infections or acute medical conditions, and review medications. If antipsychotics were prescribed, monitor for extrapyramidal symptoms, falls, and lethargy. If indicated, attempt a gradual dose reduction of any psychotropic medications after the acute episode and further reduction at least once every 3 months.

TABLE 16-1 Psychotropic Drug Management for Agitation

Antipsychotics (Always Start at Lowest Dose)

DRUG	DOSE	FORM	CAUTIONS
Aripiprazole (Abilify)	2.5 mg	Tab, dissolvable	Decreased WBCs
	Max 30 mg	Liquid, IM	Potentiates antihypertensives
Risperidone (Risperdal)	0.25–1.0 mg	Tab, liquid, IM	Dose-related EPS
			Potentiated by proton inhibitors
			May be affected by Prozac, Paxil
Saphris	5–10 mg	SL	Weight gain, anxiety, depression

Antiseizure Agents (Off Label Use)

Carbamazepine (Tegretol)	200–1,000 mg	Tab	Poor tolerability in older adults
			Monitor CBC, electrolytes, liver profile
Lamotrigine (Lamictal)	25–200 mg	Tab	Slow dose titration
			Stevens-Johnson syndrome
Divalproex sodium (Depakote)	250–2,000 mg	Tab, liquid, sprinkles	Monitor CBC, platelets, liver function
			Better tolerated by the older adults

Abbreviations: CBC = complete blood count; EPS = extrapyramidal symptoms; IM = intramuscular; SL = sublingual; WBCs = white blood cells.

TABLE 16-2 Behavioral Interventions for Agitation

Individual approach	Cognitive training Redirection and reassurance Validation Cognitive stimulation Increased activity and exercise Pain management Multisensory stimulation Reminiscence Reality orientation
Environmental	Cuing with signage and room layout Decreased noise levels Ambient lighting Person-centered care model Availability of activities and supplies
Alternative modalities	Music therapy Pet therapy Massage Therapeutic touch Snoezlen Acupuncture Aromatherapy Art therapy
Staff/caregiver education	Approach to patient Behavioral intervention skills

Sequelae: Injury to the patient and/or caregivers, falls, need for specialized care, and hospitalization.

Prevention/Prophylaxis: Identify antecedents, avoid triggers, educate staff and caregivers, and introduce behavior modification. Data also support the use of the person-centered care model in long-term care facilities and assisted living communities, along with the use of the DICE approach (Kales, Gitlin, & Lyketsos, 2014):

- Describe: Describe the context, environment, and degree of distress.
- Investigate: Rule out treatable causes: medications, pain, medical conditions, poor sleep hygiene, sensory changes, boredom, comorbid psychiatric conditions.
- Create: Develop a treatment plan.
- Evaluate: Review implementation of plan and revise as needed.

Referral: In refractory cases, a specialist, such as a geriatric psychiatrist, a geriatrician, or a neurologist with specific expertise in pharmacological management, should be consulted.

Education: Patient safety and fall prevention, identification of triggers, reassurance and redirection techniques, simplification of activities, establishment of routine and structure, promotion of rest and sleep, and consistent caregiver assignments.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Assessment of symptoms	A	Kales, Gitlin, & Lyketsos, 2015 Nowrangi, Lyketsos, & Rosenberg, 2015
Etiology and occurrence	A	Kales, Gitlin, & Lyketsos, 2015 Nowrangi, Lyketsos, & Rosenberg, 2015
Identification of triggers	A	Kales, Gitlin, & Lyketsos, 2015 Nowrangi, Lyketsos, & Rosenberg, 2015

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Psychotropic drug management	A	Kales, Gitlin, & Lyketsos, 2014 Cummings et al., 2015
Nonpharmacological interventions	A	Kales, Gitlin, & Lyketsos, 2015 Nowrangi, Lyketsos, & Rosenberg, 2015 Ellis-Smith et al., 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

ALCOHOL MISUSE (HAZARDOUS OR RISKY DRINKERS)

Signal Symptoms: It is time to ask questions when cognitive changes are apparent, such as new onset anxiety, depression, social isolation, or memory loss; or physical health changes, such as poor hygiene, falls, bruises, poor nutrition, or sleep problems. Also, if the patient is taking medications, look for the presence of unusual medication response (Taylor, Jones, & Dening, 2014).

Description: Alcohol misuse is a maladaptive pattern of alcohol use often resulting in variable levels of social, occupational, or functional impairment that can be described as occurring regularly and over a period of time. Older adults with alcohol problems often do not meet the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5) criteria for alcohol use disorder, but are impaired nonetheless. Alcohol misuse is part of a continuum of alcohol use disorders (Coogle & Owens, 2015).

Etiology: It is unclear whether risky drinking in older people represents a pattern of continued use, a return to use after a period of abstinence, new onset of use, or a combination of all these patterns. As adults move into the retirement years, many life changes occur. For instance, stressful late-life events, loss of productive social roles, loneliness, and the absence of supportive social relationships, to name a few. Additionally, drinking habits acquired in early life continue to be maintained through the life span. Several genetic markers have been studied, and a clear case for biological heritability has been established as one factor. Neurotransmitter effects from alcohol and alterations in brain anatomy in patients with alcohol use disorders serve to reinforce the biological connection (Gold & Aronson, 2012). Consistent with the complexity of alcohol use disorders, multiple pathways appear to be involved.

Occurrence: Alcohol is by far the most common substance problem in older people. Approximately half of American adults aged 65 years and over drink alcohol regularly, and estimates of late-onset alcoholism have been reported as high as 68%. Rates of misuse vary widely depending on the operational definitions of older adults, problem drinking, the methodology used in obtaining the statistics, and the definition used to describe misuse. Community surveys have estimated the prevalence of risky or hazardous drinking among older adults to range from 1% to 16% (Barnes et al., 2010; Hasin, Stinson, Ogburn, & Grant, 2007; Menninger, 2002; Substance Abuse and Mental Health Services Administration [SAMHSA], 2015).

Age: The National Survey on Drug Use and Health (Center for Behavioral Health Statistics and Quality, 2015) found that, for older individuals (50+), 12.2% were heavy drinkers, and 3.2% were binge drinkers (more than four drinks on a drinking occasion).

Gender: Men are affected more than women (Hasin et al., 2007; Zhang et al., 2008).

Ethnicity: Rates for risky drinking leading to Alcohol Use Disorder are greater among Native Americans and Alaska Natives (12.1%) than among Caucasians (8.9%), Hispanics (7.9%), African Americans (6.9%), and Asian Americans and Pacific Islanders (4.5%) (American Psychiatric Association, 2013).

Contributing Factors: Male gender, major life changes, and losses are contributing factors. Additionally, aging itself is often associated with an increased risk of medical conditions involving pain, and alcohol is often used to cope with pain. This practice itself results in poorer health outcomes. Other contributing factors include:

- Cultural attitudes toward drinking and intoxication
- The availability of alcohol
- Acquired personal experiences with alcohol
- Exaggerated positive expectations of the effects of alcohol
- Persistent stress levels

Concerns about alcohol consumption in the older adult are directed primarily toward the physiological changes that accompany aging and the problems posed by regular alcohol consumption. Chemical breakdown of alcohol does not seem to change with aging; however, the changes associated with aging may increase the concentration of alcohol in the blood. These age-related changes include decreased lean muscle mass, decreased amount of body water, changes in liver function, and increased nervous system sensitivity to alcohol. After drinking one ounce of 80-proof alcohol, a 60 year old would have a 20% higher blood alcohol level than a 20 year old, and a 90 year old would have a 50% higher

TABLE 16-3 Clinical Characteristics of Early and Late Onset Problem Drinkers

Variable	Early onset	Late onset
Age at onset	Usually <25	Usually >55
Gender	Higher proportion of men than women	Higher proportion of women than men
Socioeconomic status	Tends to be lower	Tends to be higher
Family history of alcohol use disorder	More prevalent	Less prevalent
Problems associated with alcohol use	More psychosocial; greater severity	Fewer psychosocial; lesser severity
Alcohol-related chronic illness	More common	Less common
Cognitive problems	More severe; less reversible	Less severe; more reversible

Adapted from: Substance abuse and mental health services administration. (1998). *Substance abuse among older adults.* HHS Publication No. SMA 98-3179. Rockville, MD: Substance Abuse and Mental Health Services Administration.

TABLE 16-4 Potential Laboratory Test Alterations With Identified Problem/Hazardous Drinking

TEST	EXPECTED RESULTS WITH ALCOHOL ABUSE
Complete blood count (CBC)	Increased mean corpuscular volume (MCV) with normal hemoglobin, possible decreased hemoglobin
Aspartate aminotransferase-to-alanine aminotransferase ratio	>2 suggests alcoholic liver disease
Gamma glutamyltransferase	Increased in all liver disease, including alcoholic; may remain increased for weeks after cessation of chronic alcohol intake
Carbohydrate-deficient transferring (CDT)	Increased in heavy drinking, most accurate biomarker for alcohol abuse

Physical findings may include hepatomegaly, ascites (late stage), jaundice (with pancreatitis), and spider angiomata; men may have gynecomastia, testicular atrophy, and loss of pubic and axillary hair. A complete neurological examination that includes cranial nerves, gait, sensory, motor, reflexes, Romberg's sign, and tandem walking should be included.

blood alcohol level than a 20 year old (Gilbertson, Ceballos, Prather, & Nixon, 2009).

Another major concern associated with alcohol misuse in the older adult is the increased occurrence of drug–alcohol interactions. The decreased metabolism of drugs by the liver in older adults yields significantly higher-than-normal drug levels, and alcohol increases this effect. Alcohol diminishes the effect of many medications and unpredictably strengthens the effects of sedatives. Six categories of alcohol-interactive (AI) drugs have been identified as being problematic: benzodiazepines, antidepressants, sleep medications, muscle relaxants, antipsychotics, and prescription narcotics (Jalbert, Quilliam, & Lapane, 2008; Pringle, Ahern, & Heller, 2006). Patterns of alcohol use disorder in older adults have been divided into two categories: early onset and late onset. See Table 16-3 for a comparison of the two categories.

Signs and Symptoms: Alcohol misuse is often overlooked in older adults because medical problems, psychosocial problems, and medication use may obscure the signs of alcoholism (Ferri, 2013). In addition, many older adults are solitary, so the drinking is hidden. One way that alcohol misuse comes to light is when older adults are brought to the emergency department due to events related to their problem drinking. Physical signs that may be present on examination include tachycardia, peripheral neuropathy, physical trauma, tremulousness, hepatosplenomegaly, rhinophyma, inconsistent or mild hypertension, and telangiectasias (Gold & Aronson, 2012). Other symptoms possibly related to alcohol misuse include falls, confusion, anxiety, insomnia, paranoid ideation, and pancreatitis without stones.

Diagnostic Tests: The contemporary approach to the screening and intervention with individuals who may have at-risk or problem use of alcohol is called SBIRT (Screening, Brief Interventions, and Referral to Treatment), which is an evidence-based practice used to identify, reduce, and prevent problematic use, abuse, and dependence on alcohol and illicit drugs (Substance Abuse and Mental Health Services Administration, n.d.). Diagnostic assessment depends on a thorough history. The U.S. Preventive Services Task Force (USPSTF) recommends screening for alcohol misuse problems in primary care. Numerous screening instruments with acceptable sensitivity and specificity can detect alcohol misuse in adults. The USPSTF prefers the following tools for alcohol misuse screening in the primary care setting: the AUDIT (Alcohol Use Disorders Identification Test) found at www.addictionsandrecovery.org/addiction-self-test.htm; the AUDIT-C, an abbreviated version of the AUDIT, which has been incorporated into several electronic health record (EHR) systems (www.thenationalcouncil.org/galleries/business-practice%20files/tool_auditc.pdf); and a single-question screening, such as asking, "How many times in the past year have you had five (for men) or four (for women and all adults older than 65 years) or more drinks in a day?" (USPSTF, 2014). The AUDIT-C and the AUDIT have been shown to have higher sensitivity than the other screening instruments, and to be gender and culture neutral (Frank et al., 2008).

Other tools available are the Brief Michigan Alcohol Screening Test (G-MAST)–Geriatric Version (www.the-alcoholism-guide.org/michigan-alcohol-screening-test.html) and TWEAK, a five-item scale developed originally to screen for risk drinking during pregnancy, which is an acronym for questions related to Tolerance, Worry, Eye-opener, Amnesia, and (K)Cutting down usage. This tool can be found at http://pubs.niaaa.nih.gov/publications/AssessingAlcohol/InstrumentPDFs/74_TWEAK.pdf.

A psychosocial assessment that includes a mental status examination and a geriatric depression scale should also be performed. Additionally, consider a cultural assessment if appropriate. Table 16-4 details expected results of blood chemistry and hematology tests with patients who abuse alcohol.

Physical findings may include hepatomegaly, ascites (late stage), jaundice (with pancreatitis), and spider angiomata; men may have gynecomastia, testicular atrophy, and loss of pubic and axillary hair. A complete neurological examination that includes cranial nerves, gait, sensory, motor, reflexes, Romberg's sign, and tandem walking should be included.

Differential Diagnosis:

- Nonpathological use of alcohol. Drinking, even daily, in low doses and occasional intoxication do not by themselves, identify misuse.
- Misuse or a use disorder of other psychoactive substances such as opiates, benzodiazepines, or other psychoactive drugs.
- Dementia
- Cerebrovascular accident
- Urinary tract infection
- Gastritis
- Pancreatitis also should be considered

Treatment: The goal of treatment is sobriety or total abstinence from alcohol (Willenbring, Massey, & Gardner, 2009; Wilson, 2009). The level and severity of alcohol misuse determines the treatment (see Referral). For patients who are heavy drinkers, education and brief motivational intervention by the primary care provider or cognitive behavioral therapy (CBT) may suffice (Duru et al., 2010; Fink, Elliott, Tsai, & Beck, 2005; Lin et al., 2010). Patients with symptoms of alcohol withdrawal should be hospitalized. Uncomplicated or mild alcohol use disorder can be treated in the outpatient setting.

Alcoholics Anonymous (AA) is the most successful group in encouraging ongoing sobriety; however, the self-sufficient spirit, often characteristic of older adults, reduces the probability of participation. An AA volunteer of the same gender and of an age comparable to the patient's age is usually available to meet with an individual at the clinic site and can assume the role of the patient's sponsor, reducing fear and providing the support that may encourage group participation. For older adults, people who are important in their lives need to be instructed by counselors in ways to encourage the treatment process and decrease behaviors that enable the older adult to misuse alcohol.

After consultation with an addictions specialist, naltrexone may be given to help maintain abstinence in healthy patients; naltrexone cannot be used if the patient is taking an opioid (Anton, 2008; Anton et al., 2006). For chronic alcoholism, the diet should be supplemented with multivitamins containing folic acid and thiamine, 100 mg/day. The patient should also be evaluated for electrolyte problems and anemia.

Follow-Up: Initially, the older adult should be seen weekly to provide continuity in the practitioner–patient relationship and to monitor treatment effectiveness. When the patient is participating in the treatment protocol, monthly visits should be adequate to monitor progress.

Sequelae: Alcohol misuse can lead to gastrointestinal (GI) bleeding, especially if the patient is taking aspirin or arthritis medications. More than two alcoholic drinks daily can contribute to hypertension. Gait disturbances, peripheral neuropathy, and decreased functional ability are consequences of alcohol misuse. Malnutrition, cirrhosis, urinary incontinence, decline in cognitive status, insomnia, anxiety, addiction, and tolerance with concomitant withdrawal symptoms may occur. Problematic drinking contributes to disinhibition and feelings of sadness and irritability, which contribute to suicide attempts and completed suicides, and is also associated with a significant increase in the risk of accidents and violence (American Psychiatric Association, 2013, p. 496).

Prevention/Prophylaxis: Older adults have age-related risks for continued use that should be considered. Barrick and Connors (2002) reviewed the literature regarding prevention among older adults and identified common antecedents to continued alcohol use, such as social isolation, loneliness, loss and grief, and depression, and how older drinkers report using alcohol to alleviate these negative emotional states. Taking a brief drinking history with the annual wellness visit and administering the AUDIT-C or other screening tools provides the practitioner with an opportunity for patient education. If a problem is suspected, brief interventional counseling at each visit is warranted. Although results vary in their long-term efficacy (Wutzke, Conigrave, Saunders, & Halt, 2002), brief motivational counseling is effective in some older problem drinkers (Fink et al., 2005; Lin et al., 2010). Educating patients on what constitutes "a drink" is helpful (see Education).

Referral: A specialty referral to an addictions treatment specialist is warranted for suspected complications, comorbid substance use, psychiatric diagnosis, or a substance use disorder. Consider an inpatient detoxification program for alcohol use disorder. Refer the patient to a mental health professional as indicated for suspected psychiatric comorbidity and to a community-based, peer mentoring program such as AA.

Education: Patients must be encouraged to continue participation in a treatment program, and family members should participate in a support group such as Al-Anon. Early intervention with family members who mistakenly believe that "a few drinks can't hurt" may prevent progression. Educate patients on possible consequences of continued use. Dietary supplements should be taken as ordered. Providers and patients must be educated about what constitutes "a standard drink." Standard drinks are measured as follows: 1.5 ounces of 80-proof distilled spirits, 12 ounces of beer or wine cooler, or 5 ounces of wine. Education should include clarification about the risks and benefits of moderate drinking and specific limits in quantity, frequency, and duration for older adults (Merrick et al., 2008).

Resources:

- www.samhsa.org
- www.aa.org
- www.niaaa.nih.gov
- www.hazelden.org
- www.ncadd.org
- www.discus.org

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
The USPSTF recommends screening and behavioral counseling interventions to reduce alcohol misuse by adults.	B	USPSTF, 2014
Alcohol-related brief interventions and counseling are effective in the short term in reducing unsafe drinking compared to usual care. Significant long-term reductions in drinking behavior cannot be sustained without regular follow-up and reinforcement.	A	Wutzke et al., 2002
Naltrexone (NTX) has some benefits for patients with alcohol dependence, but patients' adherence to treatment should be of concern. Psychosocial treatments may help patients to maintain adherence to NTX treatment.	A	World Health Organization, 2009

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

ANXIETY

Signal Symptoms: Excessive worrying that is difficult to control and interferes with daily life; can also manifest with somatic symptoms such as chest tightness, shortness of breath, upset stomach.

Description: Anxiety disorders are common in older adults and continue to rise as the older population increases. The 2010 Census (U.S. Census Bureau, 2011) reported that more than 40 million people were age 65 years or older, representing 13% of the total population, with about 3% residing in skilled nursing facilities. Aging may protect or contribute to anxiety depending on the circumstances (Lenze, Mohlman, & Wetherell, 2015). For example, contributing factors include changes in brain regions such as the locus coeruleus, or new stressors such as loss of a spouse, failing health, financial concerns, fear of falling, and loss of independence, while protective factors include enhanced emotional regulation and acceptance (Lenze et al., 2015).

Anxiety disorders are often under-recognized and undertreated (Andreescu & Varon, 2015). Most anxious older adults are seen in primary or specialty care, rather than in mental health clinics, where providers may mistakenly attribute symptoms to normal aging or physical illness (Tampi & Tampi, 2014). Anxiety seen in older adults is usually an exacerbation of anxiety diagnosed earlier in life, although some patients experience new-onset late-life anxiety. Predictors of late-onset anxiety include female gender, recent adverse life events, illness, cognitive impairment, and mental illness comorbidities, while poverty and poor psychological support during earlier years also contribute (Zhang et al., 2015). Comorbid dementia or depression are not uncommon in older patients experiencing anxiety (Tampi & Tampi, 2014).

According to the DSM-5 (APA, 2013), anxiety disorders referencing older adults generally include generalized anxiety disorder (GAD), social anxiety, specific phobia, and anxiety disorder related to substance use, medication, or another medical condition. Panic disorder and agoraphobia are less common in older adults (Lenze et al., 2015). A diagnosis of GAD according to the DSM-5 requires excessive anxiety, difficulty controlling worry, and associated symptoms (at least three) including restlessness, easy fatigability, difficulty concentrating, irritability, muscle tension, difficulty falling or staying asleep, or restlessness (APA, 2013). Medical conditions should be ruled out before making a diagnosis.

Etiology: Largely unknown. Risk factors include female gender, anxious personality, serious life events (military, motor vehicle accident, abuse), stress early in life, and disability (Clifford, Duncan, Heinrich, & Shaw, 2015). Evidence suggests hyper-activation of the amygdala in GAD, and also the insula in specific phobia and social anxiety, while elevated cortisol levels in older adults with GAD suggest hypothalamic-pituitary-adrenal (HPA) axis dysfunction (Andreescu & Varon, 2015). Anxiety has been linked to increased risk for cardiovascular events (Lambiase, Kubzansky, & Thurston, 2014).

Occurrence: The exact prevalence of anxiety in older adults is unknown; however, it is higher than previously recognized. Anxiety symptoms are more common than diagnosed disorders. Occurrence ranges from 15% to 50% depending on the population and criteria used, with chronically medically ill persons having higher reported rates (Kaiser, Wachen, Potter, Moye, & Davison, 2013; Tampi & Tampi, 2014).

Age: Between 3% to 14% of older adults have a diagnosed anxiety disorder.

Gender: There are no statistics on gender differences for older adults; however, it is well documented that females experience anxiety more than men. Studies show that females are one and one-half to two times more likely to develop anxiety as males (Texner & Schuth, 2016).

Ethnicity: There are limited national data. Studies have shown that those from minority groups or those who are foreign born are less likely to meet criteria for anxiety disorders than their counterparts born in the United States (Asnaani, Richey, Dimaite, Hinton, & Hofmann, 2010; Budhwani, Hearld, & Chavez-Yenter, 2015). African Americans are less likely identified with anxiety and less likely to receive treatment (Assari, 2016).

Contributing Factors: Multiple factors including poor physical health, financial insecurity, loss of significant others, fear of disability and dependence, sleep problems, and inadequate social support to name a few.

Signs and Symptoms: May include a sense of impending doom, trembling, breathlessness, and tachycardia. Anxiety may impair working memory, attention, and problem-solving skills (Andreescu & Varon, 2015). In older adults, somatic complaints are more common, such as constipation, nausea, and sleep disturbance. Worries about health, disability, and finances are also common. One is more likely to learn of a patient's anxiety by asking the question, "How do you feel when you are under stress?" than by asking, "Are you anxious?" (National Institute of Mental Health, n.d.). Patients with specific phobias may have an irrational fear to something that poses little danger, such as fear of crowds or natural phenomena (heights, lightening). Specific phobias may occur following a traumatic event, such as falling. Symptoms of anxiety in older adults often overlap with symptoms of physical disorders, depression, and dementia (Koychev & Klaus 2016).

Diagnostic Tests: Complete a history and physical examination. Laboratory tests can rule out medical conditions with anxiety symptoms, including complete blood count (CBC), CMP, and TSH. Order additional tests based on the findings of the history and physical examination. Valid assessment scales to help diagnosis and assess older adults for anxiety include the Geriatric Anxiety Inventory (GAI) and the Geriatric Assessment Scales (Clifford et al., 2015; Gould et al., 2014).

Differential Diagnosis: Includes medical conditions (hypoglycemia, hyperthyroidism, pain, brain tumor, chronic obstructive pulmonary disease [COPD], etc.) and substance use that precede new-onset anxiety symptoms. Many cardiac, respiratory, endocrine, hematologic, and neurological conditions may be associated with anxiety (Andreescu & Varon, 2015; Allahverdipour, Asghari-Jafarabadi, Heshmati, & Hashemiparast, 2013; Uchmanowicz, Jankowska-Polanska, Motowidlo, Uchmanowicz, & Chabowski, 2016). Medications, including anticholinergic drugs, dopamine agonists, levothyroxine, steroids, psychostimulants, and over-the-counter (OTC) sympathomimetics may be anxiogenic. Depression and dementia commonly overlap with anxiety in older adults (Andreescu & Varon, 2015; Lenze et al., 2015; Vasiliadis et al., 2013).

Treatment: Treatment for anxiety should reduce symptoms and improve functioning. Simply listening, being compassionate, and showing respect are important to improving outcomes. Comorbid depression and medical conditions should be treated. There are no large-scale studies of pharmacotherapy for late-life anxiety disorders to guide treatment decisions, as randomized controlled trials largely exclude those more than 65 years old. Evidence from studies with younger patients suggests both pharmacotherapy and psychotherapy, especially CBT, are effective.

"Start low and go slow" with medication dosing to avoid risks from drug interactions. Older adults are more likely to take many medications and may have side effects from aging changes in absorption, metabolism, distribution, and excretion of medication. Doses are often started at half the usual adult starting dose and titrated slowly upward. Evaluate and manage side effects, because as many as 25% of patients stop taking medication in the first 6 months due to side effects. First-line treatment includes the selective serotonin reuptake inhibitors (SSRIs) because they have the least risk of drug interactions, side effects, or worsening existing medical conditions. Escitalopram, sertraline, and citalopram are commonly used in older adults (Clifford et al.; Shaw, 2015; Koychev & Klaus, 2016), although citalopram should not be used routinely in doses above 20 mg daily due to prolongation of QT interval precautions. GI disturbances, sexual dysfunction, and altered mental status due to hyponatremia may occur. Maintenance SSRI use has been shown to reduce relapse of anxiety in older adults (Baldwin et al., 2014; Lenze et al., 2015). SSRIs can increase anxiety if started at higher doses. It may take several weeks for full effect to occur. Serotonin-norepinephrine reuptake inhibitors (SNRIs), including Venlafaxine and Duloxetine, have been shown to be effective in older adults with anxiety (Andreescu & Varon, 2015). Blood pressure should be monitored with high doses of SNRIs. Benzodiazepines, including lorazepam, alprazolam, and clonazepam, are effective (Andreescu & Varon, 2015) and may be used as a bridge until the SSRI takes effect. They are not the first choice due to the risk of falls and confusion.

Buspirone and gabapentin are also used as secondary agents when first-line therapy fails and anxiolytic therapy is warranted (Andreescu & Varon, 2015; Baldwin et al., 2014). Research supports psychotherapy, especially CBT, for older adults with GAD (American Psychiatric Association, n.d.; Baldwin et al., 2014; National Institute of Mental Health, n.d.). CBT may augment SSRIs to further reduce worry symptoms and relapse (Lenze et al., 2015). However, in some cases, CBT may be counterproductive because cognitive reappraisal can trigger additional worry due to over-engagement of the amygdala and under-engagement of the prefrontal cortex in older adults with anxiety (Andreescu et al., 2015). Preliminary findings on mindfulness-based relaxation therapy in older adults is promising in reducing worry (Lenze et al., 2014), although further study is necessary.

Follow-Up: Evaluation of the effectiveness and tolerability of treatment depends on the severity of symptoms and impairment in functioning. Older adults may not be able to describe terms consistent with anxiety, thus it is necessary to allow them to use their own words, allow extra time for questioning, and avoid complex terminology.

Sequelae: Higher morbidity, mortality, disability, and poor quality of life (Tully, Cosh, & Baune, 2013). Anxiety has been linked to subsequent cognitive impairment (Yang et al., 2015), however, causation remains unproven.

Prevention/Prophylaxis: Includes management of stress, using social supports, and maintenance of daily routines as much as possible.

Referral: Any patient with suicide intent should be referred immediately to psychiatry or an inpatient facility if safety is a concern. Refer to a mental health professional if symptoms and impaired functioning persist, or to the appropriate medical specialist, if necessary, to evaluate and treat underlying medical conditions.

Education: Educate patients and caregivers to recognize and manage anxiety disorders. Because older adults have vision and cognitive changes due to normal aging, offer large-print handouts, explain things simply, repeat things, and ask them to repeat what was taught.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Anxiety disorders are associated with an elevated risk of a range of different cardiovascular events, including stroke, coronary heart disease, heart failure, and cardiovascular death.	A	Emdin, Odutayo, Wong, Tran, Hsiao, & Hunn, 2016
CBT or relaxation training can be used to treat anxiety in older adults.	A	Klainin-Yobas, Oo, Suzanne-Yew, & Lau, 2015
The SSRIs are generally considered first-line pharmacological treatments for GAD; escitalopram and sertraline are best studied.	A	Clifford et al., 2015 Kapczinski, dos Santos Souza, Batista Miralha da Cunha, & Schmitt, 2016
Venlafaxine, duloxetine, tricyclic antidepressants, and pregabalin are alternative drug therapies.	A	Kapczinski, dos Santos Souza, Batista Miralha da Cunha, & Schmitt, 2016
Augmenting SSRIs with CBT results in reduced anxiety among older adults.	A	Wetherell, Petkus, & White, 2013
Exercise, mindfulness, and relaxation training have been shown to reduce chronic anxiety in older adults.	B	Klainin-Yobas, Oo, Suzanne-Yew, & Lau, 2015 Lenze et al., 2014 Shearer, 2016
Listening to music reduces anxiety in older adults.	B	Bradt, Dileo, & Potvin, 2013 Eells, 2014

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

BIPOLAR DISORDER

Signal Symptoms: Variable presentation ranging from depression to mania or hypomania, feelings of grandiosity, rapid speech, or irritability. Up to 22% of older adults with bipolar disorders experience anxiety symptoms (Dols et al., 2014). Cognitive deficits affecting verbal fluency and memory are common in older adults (Martino, Strejilevich, & Manes, 2013; Sajatovic & Chen, 2016; Wu et al., 2013). The depressive symptoms often include trouble with eating and sleeping (Kontis, Theochari, & Tsalta, 2013).

Description: Bipolar disorders, according to the APA (2013), are classified as bipolar 1, bipolar II, cyclothymic disorder, and other specified bipolar and related disorders. Each depends on the presentation and intensity of symptoms. A diagnosis of bipolar I disorder requires an individual to have experienced at least one manic episode. A manic episode involves a change in mood that may be expansive, euphoric, or irritable, and accompanied by an increase in energy level. Most patients also have depressive episodes, but this is not a required component. A diagnosis of bipolar II requires at least one prior episode of major depression and at least one hypomanic episode, a milder form of mania. Cyclothymic disorder is characterized by milder mood alterations that occur over a longer period of time, while unspecified bipolar disorder consists of symptoms that cause clinical impairment but do not meet criteria for the previously mentioned listings (APA, 2013). It is important to distinguish bipolar disorder from major depression, as treatment differs (Beyer, 2015).

Etiology: The precise etiology of bipolar disorder is unknown. Geriatric bipolar disorder has been studied only recently, and studies point to a multiplicity of possible causes and heterogeneous clinical presentations. Genetic vulnerabilities, comorbid conditions, lifestyle, and psychosocial factors are

all potential contributors (Craddock & Sklar, 2013; Kermer, 2015; Young & Grunze, 2013). Experts differ on the clinical significance of late-life emergent bipolar disorder versus the expression of bipolar disease manifesting in early adulthood and continuing into old age (Al Jurdi et al., 2012; Pose et al., 2013).

Occurrence: A recent meta-analysis found the prevalence of bipolar disorder to be approximately 1% in older adults, slightly less than the general population (Rej, Al Jurdi, & Sajatovic, 2014; Volkert, Schulza, Hartera, Wlodarczyka, & Andreas, 2013), although others have reported a 7% occurrence rate (Sheeran et al., 2012; Volkert et al., 2013). Approximately 25% of all patients with bipolar disorder are older adults and the number of older adults with bipolar disorders is expected to increase due to the aging population (Rej et al., 2014; Sajatovic et al., 2005).

Age: Bipolar disorder can occur at any age; peak age of onset is between 15 and 30 years. About 90% of older adults with bipolar disorder developed a bipolar spectrum disorder as a young adult and have had a continuation of the disorder lifelong. Less commonly, the condition begins after the age of 50 years (Pose et al., 2013; Rej et al., 2014).

Gender: Both sexes are affected similarly (APA, 2013); however, it remains controversial, as some studies report males are affected more and other studies report females are affected more (Cotton et al., 2013).

Ethnicity: Bipolar disorder manifests across all ethnic and cultural groups (APA, 2013; Warren, 2015).

Contributing Factors: Genetic vulnerability in families is strong in younger individuals who develop bipolar disorder, while older adults are less likely to have a family history of bipolar disorder (Ipekcioglu et al., 2015; Pose et al., 2013). Neurocognitive changes and cerebral vascular comorbidities are more common in older adults with bipolar disorder (Martino et al., 2013; Pose et al., 2013). For older adults with new-onset bipolar disorder, stresses from life are postulated to be a contributing factor, including possible childhood incidents, while older adults tend to have more medical illnesses, including obesity, diabetes, and cardiovascular and cerebrovascular conditions (Abraham, Miller, Birgenheir, Lai, & Kilbourne, 2014; Cosci, Fava, & Sonino, 2015; Sajatovic et al., 2015).

Signs and Symptoms: Elevated mood, presenting as euphoria or irritability, is common. Dysphoria, manifesting with depression alone or with irritability, is another presentation. Rapid cycling includes back-and-forth shifts from mania to depression. Inquiring about suicide ideation or intent should be addressed at every visit. Psychotic symptoms can present in either manic or depressed states, and cognitive impairment is common (Cosci et al., 2015; Dols et al., 2014). The acronym DIGFAST has been used to describe signs and symptoms during a manic or hypomanic phase. According to the DSM-5, the individual must also experience increased energy while having these symptoms (APA, 2013):

- **D**istractibility
- **I**nsomnia
- **G**randiosity
- **F**light of ideas
- **A**ctivities (hyperactive, does not require rest)
- **S**peech (rapid, can be garbled)
- **T**houghtlessness (impulsivity)

Symptoms during the depressive phase are similar to those of major depression. Use the acronym SIGECAPS:

- **S**leep disturbance
- **I**nterest/pleasure reduction
- **G**uilt feelings, thoughts of worthlessness
- **E**nergy changes/fatigue
- **C**oncentration/attention impairment
- **A**ppetite/weight changes
- **P**sychomotor disturbances
- **S**uicidal thoughts

Diagnostic Tests: The Mood Disorder Questionnaire (MDQ) is a validated (Hirschfeld et al., 2003, 2000) screening tool to assess for bipolar spectrum disorder, however, is not specific to older adults. The tool can be accessed at www.integration.samhsa.gov/images/res/MDQ.pdf. For patients with depressive features, the Geriatric Depression Scale, regular (www.stanford.edu/~yesavage/GDS.english.long.html) or short form (www.stanford.edu/~yesavage/GDS.english.short.score.html), is recommended as a screening tool.

Diagnostic studies include a CBC and comprehensive metabolic panel (CMP), toxicology screen, urinalysis, thyroid function tests, rapid plasma reagin (RPR), HIV, electrocardiogram (EKG), and other individualized testing as indicated by the individual patient presentation and anticipation of treatment modalities (Rej et al., 2014; Sajatovic et al., 2015). In patients with new onset of psychosis, an electroencephalogram (EEG) and magnetic resonance imaging (MRI) or computed tomography (CT) scan may be appropriate to rule out medical pathologies. Other screening tests for cognitive disorders, such as the Mini-Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975) or the Saint Louis University Mental Status (SLUMS), which is more sensitive to detecting cognitive impairment, may be useful in assessing comorbid neurocognitive deficits (Feliciano et al., 2013).

Differential Diagnosis: Bipolar disorder in older individuals is often undetected or misdiagnosed (Rise, Hara, & Gjervan, 2016). The etiology of new onset mania may be an underlying illness. Medical conditions include stroke (Antelmi, Fabbri, Cretella, Guarino, & Stracciari, 2014), brain tumor, multiple sclerosis, hyperthyroidism, mild traumatic brain injury, medication reaction, vascular dementia, or delirium (Rej et al., 2014). Substance use disorder, schizophrenia, schizoaffective disorder, major depressive disorder (MDD) (unipolar), impulsive disorders, and anxiety include psychiatric diagnoses (APA, 2013). A complete medication review should be done to rule out medication-induced mania and to assess polypharmacy.

Treatment: The goal is remission of symptoms. There are no specific guidelines specific to older adults, however, practice guidelines generally suggest similar pharmacological treatment for older adults as with younger adults (National Institute for Health and Care Excellence [NICE], 2014). Patients with bipolar disorder are often challenging to manage because of the fluctuating and chronic nature of bipolar disorder. Depending on the presentation and severity, inpatient treatment may be required to stabilize the patient.

First-line treatment for late-life mania includes the mood stabilizers lithium and valproic acid, or the antipsychotics, quetiapine and olanzapine (Rej et al., 2014; Sajatovic & Chen, 2016), but there are few studies specific to older adults. Because older adults are frequently on multiple medications for other comorbid conditions, monotherapy has been recommended as a starting point (Sajatovic & Chen, 2016; Young et al., 2010), with a backup plan for adding other drugs as indicated. Patients with coexisting dementia require individualized treatment, and co-management by a geriatric psychiatrist is advised. There is early evidence of a neuro-protective affect for developing dementia in those prescribed lithium (Mauer, Vergne, & Ghaemi, 2014).

Drug therapy is specific to different bipolar disorder states (Bjørklund, Horsdal, Mors, Østergaard, & Gasse, 2016; McIntyre, 2015).

Bipolar Mania U.S. Food and Drug Administration (FDA)-Approved Drugs

- Anticonvulsant mood stabilizers: Lithium, valproic acid, divalproex, or carbamazepine (second line)
- Antipsychotics: Olanzapine, risperidone, quetiapine, ziprasidone, aripiprazole, asenapine

Bipolar Acute Depression FDA-Approved Drugs

- Anticonvulsants: Lithium
- Antipsychotics: Quetiapine, lurasidone, olanzapine-fluoxetine combination.

Bipolar Maintenance FDA-Approved Drugs

- Mood stabilizers: Lithium, lamotrigine, valproic acid
- Antipsychotics: Olanzapine, aripiprazole, quetiapine, risperidone, ziprasidone

Dosing should begin at the lowest dose and be slowly increased, while monitoring comorbidities and adverse effects. Benzodiazepines are sometimes used for acute agitation in mania. SSRIs are generally not recommended for bipolar depression, as they are often ineffective and can induce mania (Price & Marzani-Nissen, 2012); however, they are used in selective, resistant cases. Treatment may require a combination of the previously mentioned medications (Stahl, 2013). Electroconvulsive therapy (ECT) is highly effective in resistant cases of bipolar depression and should be considered if drug therapy is ineffective (Oldani, Altamura, Abelghani, & Young, 2014; Schoeyen et al., 2015).

A collaborative care model has been successful for patients with combined chronic medical and mental health problems (Reilly et al., 2013). Establishing a therapeutic alliance is key to management; psychotherapy and psycho-education are also an important part of treatment (Haugh, 2014; Parikh et al., 2015; Xu, Chomutare, & Iyengar, 2014).

Follow-Up: Regular follow-up, particularly during active periods of mania or depression, is essential and should include monitoring for suicide. Family members are usually included because bipolar disorder affects the entire family. Patients on lithium require initial evaluation of renal, cardiac, and thyroid function before initiating therapy, and then periodically during therapy. Lithium levels also need close monitoring during the initial period and periodic monitoring once stabilized. Concurrent use of NSAIDs, thiazide or loop diuretics, and angiotensin-converting enzyme (ACE) inhibitors may adversely affect lithium levels (Rej, Elie, Mucsi, Looper, & Segal, 2015). Adverse effects of lithium include tremor, hypothyroidism, weight gain, and cognitive and renal impairment Patients being treated with valproic acid also require close monitoring; drug levels, liver function tests (LFTs), and CBC should be checked. Adverse effects include weight gain, hepatotoxicity, pancreatitis, and thrombocytopenia.

Those on atypical antipsychotics should have weight, glucose, and lipids monitored. QT interval prolongation can also occur. Atypical antipsychotics prescribed for older adults with dementia have been found to increase mortality; this could be translated to those with bipolar disorder (Park et al., 2015).

Sequelae: Bipolar disorder is a chronic health condition with both physical and mental health sequelae. Medications used to treat bipolar disorder often cause adverse effects. Concurrent substance abuse or sexual hyperactivity can result in legal consequences and can complicate bipolar disorder. Comorbidities often confound and determine the course of bipolar disorder in older adults (Rise et al., 2016).

Prevention/Prophylaxis: Teach patients, caregivers, and families to avoid unnecessary stress that could precipitate an acute manic episode. Educate caregivers and families to recognize early signs of mania, such as decreased sleep or pressured speech, so they can act to obtain intervention before a full-blown episode occurs. Work with the patient and his or her family to develop a safety strategy if the patient demonstrates injurious behavior directed toward self or others. Educate the patient and family on the importance of regular sleep patterns, medication adherence, and routine follow-up with a designated health-care provider (Saito-Tanji, Tsujimoto, Taketani, Yamamoto, & Ono, 2016).

Referral: Initial referral to a psychiatrist skilled in the assessment and management of bipolar disorder is important, particularly in older adults with cognitive deficits or medical comorbidities. Any patient with suicide intent should be referred immediately to psychiatry or an inpatient facility if safety is a concern. Referral of the patient and his or her family to support services is important.

Education: Patients who have had bipolar disorder from early adulthood may require reinforcement or re-education. Those with new-onset bipolar disorder require extensive education on the disease. Poor insight is part of the clinical picture, thus frequent re-education is indicated. For patients who are in a group living setting, education of caregiving staff is also essential so that manic and hypomanic episodes in particular are not misinterpreted as deliberate aggression. Spouse and family, if present, also require education in the disease process, the chronic nature of the disorder, and the importance of medication adherence. Avoidance of alcohol and other addictive substances is an important part of the education.

Resources:

- National Institute of Mental Health: www.nimh.nih.gov
- Depression and Bipolar Support Alliance: www.dbsalliance.org
- National Alliance on Mental Illness: www.nami.org
- Mental Health America: www.nmha.org
- American Psychiatric Association: www.psych.org

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Patients older than 50 years who present with new-onset mania should be evaluated for medical and neurological and cardiovascular disorders and psychiatric comorbidities, including substance use, anxiety, and personality disorders.	B	National Institute for Heath and Care Excellence (NICE), 2014
Assess safety of patient and others and determine the need for hospitalization, intensive outpatient therapy, and/or close monitoring.	A	Brennan & Rapport, 2016
Lithium has been shown to decrease the risk for suicide in patients with bipolar depression.	A	Benard, Vaiva, Masson, & Geoffroy, 2016 Cipriani, Hawton, Stockton, & Geddes, 2013
Drug therapy is essential during acute episodes and in the maintenance phase to reduce symptoms and improve functioning.	A	APA guidelines, n.d. Brennan & Rapport, 2016 Sajatovic & Chen, 2016
For geriatric patients with acute mania or hypomania, monotherapy with lithium, valproate, olanzapine, or quetiapine is suggested as initial treatment.	B	Sajatovic & Chen, 2016
For geriatric patients with bipolar major depression, initial treatment with quetiapine or lurasidone is suggested.	B	Rajagopalan, Bacci, Ng-Mak, Wyrwich, Pikalov, & Loebel, 2016 Sajatovic et al., 2015 Sajatovic & Chen, 2016
Currently, there is no convincing evidence to suggest that lithium should be avoided in elderly patients for fear of renal adverse effects.	A	Brennan & Rapport, 2016 Rej, Elie, Mucsi, Looper, & Segal, 2015
ECT is considered an important treatment option in treatment-resistant bipolar depression.	B	Sienaert, Lambrichts, Dols, & De Fruyt, 2013

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DELIRIUM

Signal Symptoms: Change in mental status, confusion, disorientation (time, place, person), agitation.

Description: Delirium is a neurocognitive disorder that presents as a disturbance "in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) and awareness (reduced orientation to the environment)" (DSM-5 Criterion). The onset of the disturbance is rapid (hours to days) and typically fluctuates over the course of the day (DSM-5 Criterion B). Cognitive changes (poor memory, disorientation, speech disturbance) and/or perceptual disturbances are distinct from a pre-existing, established, or evolving neurocognitive disorder (DSM-5 Criterion C and D). Delirium frequently represents a sudden and significant decline from a previous level of functioning, and there is usually evidence from the history, physical examination, or laboratory tests of a direct physiological etiology of a general medical condition, substance intoxication or withdrawal, use of a medication, toxin exposure, or a combination of these factors (DSM-5 Criterion D and E) (APA, 2013).

The DSM-5 differentiates delirium into the following categories:

1. Substance intoxication delirium
2. Substance withdrawal delirium
3. Medication-induced delirium
4. Delirium due to another medical condition
5. Delirium due to multiple etiologies

Delirium should also be specified as acute or persistent, as well as hyperactive, hypoactive, or mixed level activity.

Etiology: Causes of delirium are numerous, and in older adult hospitalized patients there are often multiple etiologies:

- Metabolic: renal failure, hepatic failure, anemia, hypoxia, hypoglycemia, thiamine deficiency, electrolyte abnormalities
- Infection: meningitis, encephalitis, sepsis, urinary tract infection (UTI), respiratory infection
- Cardiac: myocardial infarction, congestive heart failure, arrhythmia
- Neurological: stroke, intracranial hemorrhage, head trauma, seizures, undiagnosed pain
- Pulmonary: respiratory failure, COPD causing hypoxia
- Sensory impairment: visual and/or hearing deficits
- Medications: benzodiazepines, sedative-hypnotics, opioids, anticholinergics, antihypertensives, corticosteroids, lithium
- Toxins: alcohol, amphetamines, cocaine, substance intoxication or withdrawal

Because the cause of delirium is multifactorial, no single neuropathology of delirium has been identified (Inouye, Westendorp, & Saczynski, 2014). Possible neurological pathways may involve oxidative stress, inflammatory responses, and neurotransmitter deficiencies in acetylcholine and melatonin; excesses of norepinephrine, dopamine, and glutamate; and abnormalities in serotonin, histamine, and y-aminobutyric acid (Hatta et al., 2014; Inouye et al., 2014; Maldonado, 2013).

A clinical review of studies on biomarkers for delirium was inconclusive but implicated cytokines, along with insulin-like growth factor and the presence of S-100B, a substance expressed on astrocytes, an indicator of neuronal injury, and found in traumatic brain injury and stroke; S-100B was found in high levels in patients with delirium (Androsaova, Krause, Wintera, & Reinhard, 2015; Khan, Zawahiri, Campbell, & Boustani, 2011).

Changes in brain function, multiple general medical problems, polypharmacy, reduced hepatic metabolism of medications, multisensory declines, and brain disorders, such as dementia, make the elderly particularly vulnerable to delirium. A careful medical evaluation that includes attention to level of oxygenation, possible occult infection (e.g., UTI), and the role of medications is essential. Although many medications can be a causative factor, those with anticholinergic effects are frequently responsible (Saczynski & Inouye, 2015; Tullmann, Blevins, & Fletcher, 2016).

Occurrence: Prevalence in the community is 1% to 2%, but increases to 14% in those age 85 years and older. The prevalence of delirium ranges from 10% to 30% of older persons in the emergency department, 15% to 53% postoperatively, and 70% to 87% in the intensive care unit. In long-term care residents ages 75 years and older, the prevalence of delirium may be as high as 60% at any given time. Approximately 14% to 24% of persons exhibit delirium on admission, and 6% to 56% may develop delirium during their hospital stay. Prevalence varies depending on the underlying condition(s), procedures and surgeries performed, and other medical interventions provided (APA, 2013).

It is not uncommon for hospitalized cancer patients (25%) and hospitalized AIDS patients (30% to 40%) to develop delirium. Approximately half of postoperative patients develop delirium, and the majority of those with terminal illness (up to 83%) develop delirium as they approach death (APA, 2013; Trzepacz et al., 2006). Symptoms of subclinical delirium, such as restlessness, anxiety, irritability, distractibility, or sleep disturbance, may be manifested in the days before the onset of overt delirium and may progress to full-blown delirium over the course of a few days. The duration of delirium can range from less than 1 week to more than 2 months and typically resolves within 10 to 12 days. However, for some older persons persistent delirium can last for years. Although delirium was once thought to be reversible, evidence supports that delirium may result in permanent neuronal damage to the brain and increased mortality (APA, 2013; Cole, 2010; Flaherty, 2016; Fong, Davis, Growdon, Albuquerque, & Inouye, 2015; Trzepacz et al., 2006).

Age: Any medically ill patient can develop delirium; however, older adults are more prone to delirium due to pre-existing conditions, aging processes, and greater vulnerability to multiple precipitating factors (Saczynski & Inouye, 2015).

Gender: Males tend to have a higher incidence rate, and the male gender appears to be an independent risk factor for delirium.

Ethnicity: Culture and ethnicity should be taken into consideration when evaluating an individual's mental status and capacity. Some patients may not be familiar with information used in cognitive rating scales (general knowledge, geographical information, memory, and orientation/location), and many scales adjust for these factors, as well as educational level, in the interpretation of scores.

Contributing Factors: See Etiology, Occurrence, and Age. Frailty and delirium are related entities (Saczynski & Inouye, 2015).

Signs and Symptoms: The clinical presentation may include confusion; difficulty sustaining and shifting attention; extreme distractibility; disorganized thinking; rambling, irrelevant, pressured, and incoherent speech; impaired reasoning ability and goal-directed behavior; disorientation to time and place; impairment of recent memory; misperceptions about the environment, including illusions and hallucinations; emotional instability; and psychomotor activity that fluctuates between agitation, purposeless movements, and a vegetative state (Saczynski & Inouye, 2015). Disorientation to other persons occurs commonly. Dysarthria is a frequent speech and language disturbance, and dysnomia (impaired ability to name objects), dysgraphia (impaired ability to write), or aphasia may be observed.

Commonly associated features of delirium include disturbances in the sleep–wake cycle, such as daytime sleepiness, nighttime agitation, and disturbances in sleep continuity. Complete reversal of the sleep–wake cycle or fragmentation of the circadian sleep–wake pattern can occur. Emotional disturbances may include anxiety, fear, depression, irritability, anger, euphoria, and apathy. Affective lability (rapid and unpredictable shifts from one emotional state to another) may occur. Possible autonomic signs associated with delirium include tachycardia, sweating, flushed face, dilated pupils,

and elevated blood pressure (Saczynski & Inouye, 2015; Tullmann et al., 2016).

The diagnosis of delirium superimposed on dementia presents challenges for clinicians (Morandi et al., 2016) and results in poor outcomes for older persons, including increased cognitive decline, institutionalization, prolonged hospitalizations, and mortality (Fick, Steis, Waller, & Inouye, 2013; Inouye et al., 2014; Morandi et al., 2014). Delirium is an independent risk factor for dementia and dementia is the leading risk factor for delirium (Fong et al., 2015).

Diagnostic Tests: Laboratory work will assist in identifying potential causative factors, as will rating scales that measure cognition and establish diagnostic data such as the Confusion Assessment Method (CAM), the Delirium Rating Scale – Revised, or the Delirium Observation Screening Scale. These scales assist in tracking progress and in the patient's return to baseline functioning (Saczynski & Inouye, 2015; Tullmann et al., 2016). However, because of fluctuating levels of consciousness and cognition, it may be difficult to assess mental status and cognitive function. When possible, obtain information from the medical record, medical staff, and others, especially family members.

Differential Diagnosis: See Etiology. The most common differential diagnostic challenge is whether the patient has dementia or delirium, has delirium alone, or has a delirium superimposed on a pre-existing dementia. Although there are common cognitive disturbances in delirium and dementia, a primary difference is that the patient with dementia usually is alert, whereas the patient with delirium manifests overt disturbances of consciousness or arousal. The rapid onset and course of cognitive impairments and the reversibility of symptoms are helpful in distinguishing between delirium and dementia. The severity of delirium symptoms typically fluctuates over the course of a day, whereas dementia symptoms generally do not fluctuate (Inouye et al., 2014). Information from medical records, caregivers, and family members may help determine whether dementia was present before the onset of delirium. Depression is another differential diagnosis, because patients often manifest cognitive and psychomotor symptoms common to dementia and delirium (Saczynski & Inouye, 2015).

Treatment: Appropriate treatment for delirium involves discovering the causes, many of which are reversible, and preventing complications through prompt treatment of specific, identified disorders. A thorough, comprehensive assessment; evaluation of medications, interactions, and contraindications; and ordering of laboratory work will assist in ruling out/in the many etiologies of delirium. While assessing for probable etiology and definitive treatment, management should focus on ensuring safety from behavioral disturbances by combining environmental, behavioral, and pharmacological therapies.

Quality improvement models of care, such as the Hospitalized Elder Life Program (HELP), a prevention model, use a multicomponent strategy with the overall purpose of promoting independence in hospitalized older persons. Protocols are implemented to screen for delirium, treat the underlying cause, and prevent cognitive and functional decline (Flaherty, 2016; Yue et al., 2015; Zaubler et al., 2013). Acute care for the elderly units in the hospital setting and the designation of delirium beds within these units (Flaherty, 2016) is another strategy to manage delirium. The framework for using the ABCDE (Awakening and Breathing coordinating, Choice of sedatives, Delirium identification, and Early exercise and mobility) Bundle is also used in critical care settings (Bassett et al., 2015). Specially trained nursing staff assess and monitor patients with delirium (Kratz, Heinrich, Schauls, & Diefenbacher, 2015; Layne, Haas, Davidson, & Klopp, 2015). Further research is needed to determine the efficacy of these innovative models of care.

Nonpharmacological Interventions: A therapeutic environment would include frequent reassurance and reality orientation; clear communication; caregiver consistency; decreased stimuli (noise reduction, adequate lighting, sufficient time to perform tasks); decreased stress and anxiety through frequent reassurance and provision of a daily routine; comfort maintenance (eyeglasses, hearing aids, personal belongings); reestablishment of a sleep–wake cycle by controlling nighttime noise and unnecessary disruptions; guarantee of adequate daily fluid intake; assurance that elimination needs are met; provision of space and programs for physical activity, ambulation, and range of motion; and avoidance of chemical or physical restraint. Medication should be used as a last resort (Tullmann et al., 2016).

Pharmacotherapy: Data support the use of first-generation (e.g., haloperidol) and second-generation (e.g., olanzapine, risperidone, ziprasidone, and quetiapine) antipsychotic medications to control behavioral symptoms of delirium and prevent injury to self or others. Antipsychotic medications have significant side effects, especially for older persons with dementia, and should be prescribed at the lowest effective dose and only 1 to 2 days or the shortest interval possible depending upon the setting, such as surgery or intensive care (Inouye, 2015; Inouye, Marcantonio, & Metzger, 2014; Reus et al., 2016). The avoidance of benzodiazepines except for specific indications (e.g., alcohol or g-hydroxybutyric acid [GHA] withdrawal delirium, delirium related to seizures) continues to be a recommendation (Inouye, 2015; Inouye et al., 2014).

It is important to periodically reassess the patient's mental status and other psychiatric symptoms and behaviors such as depression, suicidal ideation or behavior, hallucinations, delusions, aggression, agitation, anxiety, disinhibition, affective lability, cognitive deficits, and sleep disturbances, because these symptoms can fluctuate rapidly. Regular monitoring and serial assessments of mental status and symptoms will allow for the adjustment of treatment strategies and may indicate the effectiveness of interventions and new or worsening medical conditions (Inouye, 2015; Tullman et al., 2016).

Follow-Up: Close follow-up for treatment efficacy, appropriate laboratory and diagnostic studies to monitor resolution of the underlying cause, and monitoring of mental status and cognitive functioning are essential to ensure full recovery.

Sequelae: During overt periods of delirium, there is risk of injury to self or others due to confusion, altered perception, and impaired insight and judgment. Fear is often a precipitant to injury in patients with delirium and may result in attacking others; falling out of bed; or pulling on IV lines, oxygen tubing, tracheotomy or GI tubes, urinary catheters, or

other medical equipment. Although the majority of patients recover fully, delirium may progress to stupor, coma, seizures, or death. Full recovery is less likely in the elderly, and persistent cognitive deficits are common (Fong et al., 2015). Such deficits may be due to pre-existing dementia that was not clearly established.

The elderly have a significantly increased risk of developing complications, such as pneumonia and decubitus ulcers, which may result in longer hospital stays. In postoperative patients, delirium may limit recovery and contribute to poorer long-term outcomes. Increased risk for postoperative complications, longer postoperative recuperation periods, longer hospital stays, and long-term disability are associated with delirium. Delirious patients with alcohol or sedative-hypnotic withdrawal, cocaine intoxication, head trauma, hypoglycemia, strokes, or extensive burns are at increased risk for seizures. Delirium in the medically ill is associated with an increased mortality rate, and patients who develop delirium during a hospitalization also have a very high rate of death during the months following discharge (Inouye et al., 2014; Morandi et al., 2014).

Prevention/Prophylaxis: Preventive measures to lessen the likelihood of delirium include elimination or minimization of risk factors. These measures include judicious use of high-risk medications (Beers list; STOPP/START), timely management and good control of acute and chronic medical disease processes, correction of sensory deficits (eyeglasses, magnifying glasses, adequate lighting, hearing aids, cerumen removal), promotion of normal sleep patterns through good sleep hygiene measures, provision of adequate nutrition and hydration with oral/parenteral supplementation as necessary, prompt attention to elimination needs, participation in activities that maintain and stimulate cognitive and physical functioning, and provision of general supportive measures (environmental modifications, reality orientation, control of external stimuli) (Layne et al., 2015). For hospitalized elders and long-term care residents, encourage frequent visits by family members to provide familiarity, reality orientation, reassurance, and comfort.

Referral: Patients with delirium should be hospitalized so that diagnostic testing, identification of underlying causes, and management can occur in a rapid, coordinated manner during concurrent treatment of acute symptoms to ensure patient safety and comfort. Care of the patient with delirium should be coordinated by the primary care provider and managed jointly with internal medicine, psychiatry, neurology, and other specialty physicians to ensure appropriate comprehensive evaluation and care.

Education: Patients and families should be educated about the etiology of delirium and the expected course of illness, while being provided reassurance that delirium is usually temporary and that the symptoms are part of a medical condition. The American Delirium Society (www.americandeliriumsociety.org) is a multidisciplinary professional group dedicated to delirium research and the education of professionals and families of patients with delirium.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Assessment of predisposing/vulnerability factors includes findings from the patient's history and physical assessment. Pre-existing cognitive impairment, severity of presenting illness, and age are the most consistent risk factors identified for the development of delirium.	B	Saczynski & Inouye, 2015
Research is needed on Framework for Improving the Diagnosis of Delirium Superimposed on Dementia: • Improving attention testing • Impact of arousal/awareness/motor fluctuations • Clinical evaluation of delirium superimposed on dementia • Clinical examination • Laboratory testing • Neuroimaging	B	Morandi et al., 2016
Nonemergency antipsychotic medications should only be used for agitation or psychosis in persons with dementia if the symptoms are severe, dangerous, or are distressing to the patient.	A	Reus et al., 2016
Nurses' recognition and assessment of delirium can be enhanced with education on assessing cognition, cognitive impairment, features of delirium, and factors associated with poor recognition of delirium.	B	Layne et al., 2015
In older persons not taking cholinesterase inhibitors, cholinesterase inhibitors should not be prescribed to prevent or treat delirium.	C	Inouye et al., 2015

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Nursing admission and daily assessment processes should incorporate the use of standardized instruments for assessing cognition and the presence of delirium.	B	Inouye et al., 2015
Clinicians' implementation or adherence to multicomponent intervention strategies is essential to improve patient outcomes.	B	Yue et al., 2015; Zaubler et al., 2013
Early mobilization is an intervention strategy aimed at prevention of delirium for hospitalized older persons.	B	Bassett et al., 2015

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

DEMENTIA

Alzheimer's disease (AD) is the most common cause of dementia and is the primary focus of this section. Dementia is a neurocognitive disorder (APA, 2013). Dementia is defined as a clinical syndrome with global cognitive decline from a previous level of baseline function that interferes with activities of daily living (ADLs) (APA, 2013; Kimchi & Lyketsos, 2015; Tay et al., 2015). In the differential diagnosis, it is important to ascertain whether an individual has cognitive impairment or an illness with similar or overlapping signs and symptoms such as delirium, depression schizophrenia, bipolar disorder, or other neurological disorder (Lyketsos, 2016a). If there is cognitive impairment, then developmental delay, borderline intellectual functioning, mild cognitive impairment, and other related diagnoses must be ruled out (Lyketsos, 2016b). To meet the criteria for minor or major neurocognitive disorders according to the DSM-5 (APA, 2013), cognitive decline must be in at least one of the following cognitive domains including "complex attention, executive function, learning and memory, language, perceptual motor or social cognition" (Kimchi & Lyketsos, 2015, p. 243; Tay et al., 2015). Once dementia is ruled in, the type of dementia can then be determined (Lyketsos, 2016b).

Signal Symptoms: Cognitive changes including confusion, disorientation (as to time, place, person), and impaired short-term memory. Personality changes, psychiatric symptoms, problem behaviors, and changes in daily functioning (Lyketsos, 2016b).

Description: Clinicians must consider normal aging processes and factors that may contribute to the overall clinical picture when assessing a patient who presents with signs or symptoms of dementia (Kimchi & Lyketsos, 2015). AD has a gradual onset, and the course of illness and progression is typically slow (Kimchi & Lyketsos, 2015). The duration of AD ranges from 3 to 20 years and averages 10 years as comorbidities complicate the course of illness (APA, 2013). Symptoms vary from person to person, and cognitive deficits cause significant impairment in social and occupational functioning, impaired ability to care for oneself, and altered behavioral patterns. Signs and symptoms progress from memory loss to impaired executive functioning, language deficits, coordination, and perception (Rosenberg, Pontone, & Onyike, 2016) with total or partial loss of the ability to recognize familiar people or objects. Impairment in memory and learning (amnestic) is the typical presentation for AD (APA, 2013) and neuropsychiatric symptoms almost always occur (Kimchi & Lyketsos, 2015).

The National Institute on Aging and the Alzheimer's Association workgroup introduced diagnostic staging for AD, including preclinical, mild cognitive impairment due to AD, and dementia due to AD (Albert et al., 2011; McKhann et al., 2011; Sperling et al., 2011).

Etiology: The etiology of dementia includes numerous systemic disorders; however, most cases of dementia are irreversible because dementia is a progressive disease process unto itself.

- Central nervous system (CNS) disorders: mild cognitive impairment (MCI), AD (most common type of dementia [Alzheimer's Association, 2016], 60% to 80%; one-half of cases are mixed dementia related to other pathology), Lewy body dementia (10% to 25%), Parkinson's disease (incidence one-tenth of AD), vascular dementia (10%), primary degenerative dementia, frontotemporal dementia or Pick's disease (10% of persons 45 to 60 years old), Huntington's disease, normal pressure hydrocephalus (5%)
- Cardiovascular disease: cerebral hypoxia/anoxia, vascular insults to brain, cardiac arrhythmias, inflammatory blood vessel disease
- Infectious processes: AIDS, Creutzfeldt-Jakob syndrome, neurosyphilis
- Liver disease: chronic progressive hepatic encephalopathy
- Neoplastic conditions: intracranial lesions, primary or metastasis
- Pulmonary disease: respiratory encephalopathy, COPD/CO_2 toxicity
- Urinary tract disease: UTI, chronic or progressive uremic encephalopathy

Occurrence: Currently, 5.4 million Americans are living with AD, and one in nine people age 65 years and older (11%) have AD (Alzheimer's Association, 2016). For every 5-year age group after age 65 years the percentage of AD doubles (Rosenburg, Pontone, & Onyike, 2016). By 2050, the number of older persons 65 years and older with AD is expected to almost triple, and the estimated number of AD cases by 2050 will be 959,000 new cases (Alzheimer's Association, 2016).

Age: Early-onset AD affects those 65 years old or younger, is usually familial, and represents less than 5% of AD cases. Late-onset AD affects those age 65 years and older and may or may not be related to family history (Alzheimer's Association, 2016).

Gender: More women than men have dementia, primarily because women live longer.

Ethnicity: African Americans and Hispanics are at highest risk of developing AD. African Americans are approximately twice as likely to develop AD than Caucasians, and Hispanics have approximately one and one-half times the risk of their Caucasian counterparts.

Contributing Factors: Risk factors for AD are designated as actual and probable.

Actual Risk Factors

- Age
- Genetics/family history (NIA, 2015; Ridge, Ebbert, & Kauwe, 2013)
  - Gene mutations chromosomes 1, 14, 21
  - A 50/50 chance of developing early-onset AD if one parent had AD
  - Apolipoprotein E (ApoE) gene on chromosome 19
  - ApoE alleles e2, e3, e4
  - Possibly chromosomes 9, 10, 12
  - Microglial TREM2 gene triples the risk of AD (Guerreiro et al., 2013)
  - Down syndrome
- Less than 5% of AD cases are caused by rare genetic variations found in a small number of families worldwide. In these inherited forms of AD, the disease tends to develop before age 65 years, sometimes in people as young as 30 years old. The genetic mutations involve the following chromosomes:
  - Chromosome 21 on the gene for the amyloid precursor protein
  - Chromosome 14 on the gene for the presenilin 1 protein
  - Chromosome 1 on the gene for presenilin 2 (APA, 2013; NIA, 2015; Ridge, Ebbert, & Kauwe, 2013)

Probable Risk Factors

- Low educational level, low lifetime achievements
  - More years of education (versus fewer years) provides a "cognitive reserve" that enables compensation for symptoms of AD/other dementia
  - Differences in education and dementia risk may reflect increased risk for disease in general and less access to medical care in lower socioeconomic groups
- Female gender, low estrogen levels
  - Framingham Study: Lifetime risk for any dementia in females who reached age 55 years = 21% and for males = 14%
  - Depression, brain injury
  - Cardiovascular disease, hypertension, type 2 diabetes mellitus, smoking, obesity

TABLE 16-5 Stages of Alzheimer's Disease and Associated Symptoms

STAGE	ASSOCIATED SYMPTOMS	DURATION
Preclinical	Impaired memory, excused or covered Insidious instrumental ADLs losses (money handling, bills) Preserved basic ADLs Poor judgment and decisions Subtle personality changes Decreased spontaneity, sense of initiative Increased anxiety, socially normal	2–4 years or longer
Mild–moderate	Obvious memory impairment Overt instrumental ADL impairment Basic ADLs failing Prominent behavioral difficulties Shortened attention span Language difficulty Variable social skills Supervision required	2–10 years
Severe	Memory fragments only No recognition of familiar people Assistance with basic ADLs required Fewer troublesome behaviors Reduced mobility Weight loss, infections Seizures, dysphagia Incontinence Groaning, moaning, grunting	1–2 years or longer

Signs and Symptoms: Signs and symptoms vary according to the stage of dementia and disease progression. AD is a multiyear brain disease; it is thought to begin well before clinical manifestations appear. Preclinical changes in the brain can begin 10 to 20 years before symptoms present. These changes include diffuse cerebral plaques, neuritic plaques and tangles, neuron and synapse loss, and some cognitive impairment (Ariga, Miyatake, & Yu, 2010; Sperling et al., 2011). The onset of clinical symptoms typically begins with memory loss. The duration of each stage varies, and functional changes usually occur late in the disease process (see Table 16-5).

Many patients manifest noncognitive behavioral symptoms (NCBSs) years before being diagnosed with dementia (see Box 16-1). Once dementia is diagnosed, NCBSs may continue to manifest similarly or progress to symptoms more difficult to manage.

Diagnostic Tests: A thorough diagnostic evaluation aimed at identifying the specific etiology of dementia is necessary and will guide treatment decisions. The evaluation should determine if any treatable psychiatric or general medical conditions might be causing or exacerbating the symptoms manifested by the patient. The clinician must obtain a comprehensive history, and because of questionable reliability secondary to cognitive deficits, a family member or caregiver

BOX 16-1

Noncognitive Behavioral Symptoms of Dementia

Apathy
Agitation, aggression
Combativeness
Delusions, hallucinations
Depression, anxiety
Disinhibition/sexual behaviors
Emotional lability
Irritability
Wandering
Sleep disturbances
Sundowning

should be present to validate and obtain collateral information. Take care to note the patient's attitude toward the family member (e.g., friendly, positive, suspicious, paranoid, angry, hostile) who is answering the examiner's questions. If the family member is frequently "correcting" the patient's answer to a question, consider patient and/or family denial, compensation, inconsistency, and fabrication of information (Lyketsos, 2016b). The history should include:

- Family history, past medical history, occupation, current health status
- Onset and frequency of memory and cognitive lapses
- Word-finding difficulties
- Ability to perform ADLs, instrumental ADLs
- Sleep patterns
- Ability to drive without getting lost
- Recognition of known others and recall of names
- Wandering

Diagnostic and Screening Tests: Evidence-based studies support the following routine laboratory studies: CBC, electrolytes, glucose, blood urea nitrogen (BUN), creatinine, LFTs, TSH, serum B_{12}, folate, syphilis serology, and urinalysis (Lyketsos, 2016b). A noncontrast CT scan will detect vascular insults (infarcts, stroke), and based on history and physical findings and clinical suspicion, an MRI may assist in ruling out medical/neurological processes and assist in differential diagnosis. Tests for early diagnosis of AD are in investigative stages and include a positron emission tomography (PET) scan and biological markers (Lyketsos, 2016b).

Mental Status Examination and Cognitive Testing: A full mental status examination may uncover mood and neurovegetative symptoms that indicate treatable comorbid psychiatric conditions (e.g., depression, anxiety) that may contribute to cognitive difficulties. Instruments that assess orientation, attention, calculation, and memory should be initiated early to assist in diagnosis and establish baseline, and should be routinely repeated to objectively document and measure changes over time.

Although commonly used, the MMSE has poor specificity for dementia and may exhibit poor sensitivity in highly intelligent or well-educated patients; copyright limitations also apply. Additionally, sensory loss and physical frailty may lower scores. Other cognitive instruments may unmask deficits in executive function, abstraction, praxis, visuospatial performance, and test clock-drawing ability, word fluency, proverb interpretation, and praxis (e.g., ability to brush teeth or comb hair). The Montreal Cognitive Assessment (MoCA) and the St. Louis University Mental Status Exam (SLUMS) are well-validated instruments that may be used in the assessment of dementia. The MoCA is a screening test to detect MCI and has greater sensitivity and specificity in distinguishing normal controls from MCI when compared to the MMSE. The MoCA assesses attention and concentration, executive functions, memory, language, visuoconstructional skills, conceptual thinking, calculations, and orientation (Doerflinger, 2012). The SLUMS is useful for detecting MCI and dementia, is more sensitive than the MMSE, and tests orientation, short-term memory, word fluency, attention, concentration and recall, clock drawing, and identification (Tariq, Tumosa, Chibnall, Perry, & Morley, 2006).

The Clock Drawing Test (CDT) can be administered alone or within other cognitive screening instruments (e.g., SLUMS) and screens for AD and other types of dementia. The CDT offers clues regarding areas of cognitive change or damage and provides information about general cognitive and adaptive functioning (memory, information processing, vision, visual-spatial skills). Of note, the CDT lacks sensitivity for MCI. The patient is asked to draw a clock with all of the numbers and set the hands to read a certain time (e.g., 10 past 8). Certain errors, such as grossly distorted contour or erroneous markings, are rarely produced by intact cognition (Ehreke et al., 2009).

Physical Examination: A general medical assessment should be completed with emphasis on the cardiovascular, respiratory, neurological, and musculoskeletal systems. Assess for endocrine, inflammatory, and infectious processes that may contribute to cognitive symptoms, and note focal motor or sensory signs and altered reflexes, gait, or coordination. The presence of tremor, rigidity, or cogwheeling may indicate a parkinsonian process and vertical gaze paralysis may suggest supranuclear palsy. Look for funduscopic changes that may indicate vascular damage or intracranial pressure, and vision and hearing loss that can mimic or worsen cognitive decline. Unless there are comorbid medical disease processes, the physical examination does not typically reveal many findings.

Diagnosis: Definitive diagnosis of AD is only possible on autopsy and upon finding disease-specific pathology in the brain. As such, the diagnosis of AD is presumptive and based upon the following diagnostic criteria:

Major or Mild Neurocognitive Disorder Due to AD

DIAGNOSTIC CRITERIA

A. The criteria are met for major or mild neurocognitive disorder.

B. There is insidious onset and gradual progression of impairment in one or more cognitive domains (for major neurocognitive disorder, at least two domains must be impaired).

C. Criteria are met for either probable or possible AD disease as follows:

TABLE 16-6 Differential Diagnosis of Dementia, Delirium, and Depression

CLINICAL FEATURES	DEMENTIA	DELIRIUM	DEPRESSION
Onset	Insidious	Rapid	May be abrupt, with life changes
Course	Long, progressive	Short, diurnal variation	Situational
Duration	Months to years	Hours to 1 month	2 weeks, months, years
Awareness	Clear	Reduced	Clear
Alertness	Normal	Impaired	Normal
Orientation	Impaired	Impaired	Selective
Thought process	Poor, abstract thinking; diminished thoughts; poor judgment; difficulty with word finding/verbalizing	Disorganized, distorted, fragmented, diminished or expansive thoughts, incoherence	Intact, linear; themes of hopelessness, helplessness, poor self-esteem
Perception	Frequent misperceptions	Distorted with illusions, delusions, hallucinations	Intact
Psychomotor behavior	Normal, apraxia	Varies/mixed; hypokinetic, hyperkinetic	Varies with restlessness, agitation, retardation
Sleep–wake cycle	Fragmented, disturbed, reversed	Disturbed, reversed	Disturbed sleep patterns (increased/decreased); early, mid, late insomnia; napping
Associated features	Affect superficial, labile, inappropriate; may be in attempt to conceal deficits	Variable affective changes, increased arousal, personality exaggeration	Affect and mood depressed, increased somatic complaints, preoccupation, rumination
Mental status testing	Increased effort to find appropriate replies, frequent near-miss answers, word searching	Distracted from task, inability to focus	Inability to focus/concentrate; makes little effort; gives up; shows indifference, apathy

For Major Neurocognitive Disorder:

Probable Alzheimer's disease is diagnosed if either of the following is present; otherwise, **possible Alzheimer's disease** should be diagnosed.

1. Evidence of a causative AD genetic mutation from family history or genetic testing.
2. All three of the following are present:
   a. Clear evidence of decline in memory and learning and at least one other cognitive domain (based on detailed history or serial neuropsychological testing)
   b. Steadily progressive, gradual decline in cognition, without extended plateaus
   c. No evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease, or another neurological, mental, or systemic disease or condition likely contributing to cognitive decline)

For Mild Neurocognitive Disorder:

Probable Alzheimer's disease is diagnosed if there is evidence of a causative AD genetic mutation from either genetic testing or family history.

Possible Alzheimer's disease is diagnosed if there is no evidence of a causative AD genetic mutation from either genetic testing or family history, and all three of the following are present:

1. Clear evidence of decline in memory and learning
2. Steadily progressive, gradual decline in cognition, without extended plateaus
3. No evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease, or another neurological or systemic disease or condition likely contributing to cognitive decline)

D. The disturbance is not better explained by cerebrovascular disease, another neurodegenerative disease, the effects of a substance, or another mental, neurological, or systemic disorder.

Differential Diagnosis: Numerous disorders and disease processes have similar or overlapping symptoms to dementia (see Etiology), and a comprehensive assessment is necessary for accurate diagnosis. The primary focus of differential diagnoses for dementia is depression and delirium, because symptoms may be very difficult to differentiate in an acutely ill patient (see Table 16-6). Additional differential diagnoses include:

- Psychiatric: depression, delirium, mild cognitive impairment, vascular cognitive impairment, amnesic disorder
- Neurological: multiple sclerosis, normal-pressure hydrocephalus, intracranial tumors, subdural hematoma, dementia pugilistica
- Infection: HIV, neurosyphilis
- Inflammatory: rheumatoid cerebrovasculitis, lupus cerebrovasculitis, neurosarcoidosis
- Endocrine: hypothyroidism, hypoparathyroidism, Cushing's disease, Addison's disease, hyperthyroidism, hyperparathyroidism

- Metabolic: vitamin B deficiency
- Toxins: alcohol/substance abuse/dependence

Once depression, delirium, and systemic causes of dementia are eliminated from the differential, it is important to differentiate the type of dementia manifested by the patient. Dementia that is primarily caused by degenerative CNS processes or sequelae includes vascular dementia, Lewy body dementia (associated with Parkinson's disease), frontotemporal lobe dementia (Pick's disease), AD, and mixed dementia. There are similar/overlapping symptoms and distinct symptoms of each type, which help with the differential diagnosis.

Differentiation of Dementia: Dementia can be classified as vascular, Lewy body/Parkinson's, frontotemporal lobe/Pick's disease, or mixed (Alzheimer's Association, 2016; APA, 2013).

Vascular Dementia

- Sequelae from transient ischemic attacks, mini-strokes, cerebrovascular accidents
- No or little cortical shrinkage
- Cognitive, behavioral, and functional losses defined by area of infarct
- Stepwise deterioration over time

Clinical Presentation:

- Abnormal executive functioning
- Impaired psychomotor performance
- Changes in personality and mood
- Disturbances in gait (slow and unsteady)
- Hyper-reflexia, extensor plantar response
- Urinary incontinence
- Hemiparesis, including lower facial weakness
- Hemisensory deficits
- Visual problems (field defect, diplopia)
- Pseudobulbar syndrome (e.g., dysarthria, dysphagia, emotional incontinence)
- Focal deficits

Lewy Body/Parkinson's Dementia

- Diffuse presence of Lewy body proteins in brain, including cerebral cortex
- Lewy bodies deplete dopamine
- Acetylcholine is depleted, causing disruption of perception, thinking, and behavior
- Resultant parkinsonian symptoms: stiff, shuffling gait, stiffness in arms and legs, tremors, frequent falls, mask-like facies with blank stare, flat affect, stooped posture, drooling, runny nose

Clinical Presentation:

- Parkinsonian signs
- Symptoms may fluctuate as often as moment to moment, hour to hour, or day-to-day
- Fluctuating cognition, varying degrees of alertness and attention
- Progressive memory loss
- Visual hallucinations
- Rapid eye movement (REM) sleep difficulties

Frontotemporal Lobe Dementia/Pick's Disease

- Gradual and progressive changes in behavior—socially inappropriate, disinhibition, easily frustrated, impulsive, compulsive behaviors; or
- Gradual and progressive language dysfunction—problems with expression of language, incorrect words, naming objects
- Difficulties with reading and writing

Mixed Dementia

- More than one type of dementia (e.g., combination of AD and vascular dementia)

Treatment: A comprehensive, multidimensional treatment plan for dementia includes biological, psychotherapeutic, social, family, and pharmacological interventions (Lyketsos, 2016b).

Biological Interventions: Treat underlying medical disorders with medications, medical or surgical procedures, and ongoing evaluation and management as indicated.

Psychotherapeutic Interventions: Include behavioral management, reminiscence therapy, validation therapy, supportive psychotherapy, sensory integration, simulated presence therapy, reality orientation, skills training, recreation and art therapy, exercise, and aromatherapy.

Social Interventions: Include a functional and safety assessment, environmental modifications, assessment for abuse and neglect, provision of supervision and home health care, cleaning and meal services, assessment for appropriate level of care, financial and estate planning, and legal provisions for power of attorney.

Family Interventions: Include caregiver education, training and support, respite care, and support groups.

Pharmacotherapy: Cholinesterase inhibitors (ChEIs) are the cornerstone of pharmacological therapy, with the aim to enhance or preserve cognitive and behavioral status. Acetylcholine is important for the functioning of brain cells involved in memory, thought, and judgment, and brain levels are significantly decreased in those with AD. ChEIs inhibit breakdown of acetylcholine, which increases levels within the brain; this mechanism of action may improve or delay a decline in memory. ChEIs are effective in approximately 30% of patients and are not curative, preventive, or disease-reversing agents. The choice of ChEI is based on the patient's clinical presentation and comorbid conditions.

The three commonly prescribed ChEIs are donepezil, rivastigmine, and galantamine (Birks, 2016; NIA, 2016). Clinical and safety issues for ChEIs include medical and psychosocial factors before drug initiation, consideration of when to initiate the medication, and side effects, which are fewer with slower dose titration. Careful monitoring for efficacy and side effects is necessary. Common side effects of ChEIs are nausea, vomiting, dyspepsia, anorexia, diarrhea, insomnia, vivid dreams, fatigue, increased urination, and cramps. Uncommon side effects of ChEIs are syncope, bradycardia, confusion, depression, and agitation. Use cautiously in patients with liver or gastric disease, COPD, bradycardia, and inadequate supervision.

Considerations regarding when to stop ChEIs may include intolerable side effects, new medical contraindications, poor compliance, lack of supervision, and rapid cognitive and/or functional decline. Any benefits of treatment are rapidly lost upon discontinuation. Long-term treatment may continue to offer advantages such as slowing of cognitive decline,

TABLE 16-7 FDA-Approved Medications for Alzheimer's Disease

	DONEPEZIL (ARICEPT) ChEI	RIVASTIGMINE (EXCELON) ChEI	GALANTAMINE (REMINYL) AND EXTENDED RELEASE (RAZADYNE) ChEI	MEMANTINE (NAMENDA) (NMDA RECEPTOR AGONIST)
FDA approval	1996; mild, moderate, severe AD	2000; mild, moderate AD	2001; mild, moderate AD	2004; moderate, severe AD
Benefit	Typically well tolerated; improves cognitive and behavioral status, caregiver burden, and capacity for ADLs	Improves cognitive, behavioral, and functional impairments; is more selective for central processes and regions critical for cognition and memory	Significantly improves cognitive, behavioral, and functional symptoms of AD	Delays loss of daily functions, cognition, and global performance; decreases agitation
Dosage strengths (mg)	5, 10	1.5, 3, 4.5, 6	4, 8, 12 Extended Release: 8, 16, 24	Titrate to 10 mg bid over 4 wks
Oral solution	1 mg/mL	2 mg/mL	4 mg/mL	
Starting dose	5 mg qd	1.5 mg bid	4 mg bid	
Maximum recommended dose	10 mg qd	6 mg bid	8–10 mg bid Extended Release: 16–24 mg qd	10 mg bid
T1/2 (hours)	73	5	6–8	60–80
Plasma protein binding	96%	40%	18%	
CYP450 substrate of	2D63A4	NA	2D63A4	
CYP450 inhibitor of	NA	NA	NA	NA

continued ability to perform ADLs, fewer noncognitive behavioral symptoms, and decreased caregiver burden (NIA, 2016).

Another medication approved to treat dementia is memantine (Namenda), an *N*-methyl-D-aspartate (NMDA) receptor antagonist. Memantine assists in regulating high levels of glutamate in the brain, typically found in AD. Common side effects include headache and constipation, and an uncommon side effect is confusion. Memantine is excreted through the kidneys, and caution is advised in patients with renal impairment. Combination therapy of memantine with a ChEI is a good strategy, because these medications work differently (NIA, 2016). Usually medications are started when AD severity is moderate, but they can be initiated earlier depending on individual patient clinical presentations (see Table 16-7).

Management of Noncognitive Behavioral Symptoms (NCBSs) of Dementia: Includes determination and management of other potential or influencing factors, including (Tampi et al., 2011):

- Environmental: external (noise) or internal (UTI, constipation)
- Situational: time of day, unknown trigger
- Psychiatric: depression, anxiety, panic, fear
- Medical: medication, pain, metabolic, infection, sensory deficits, cardiopulmonary

Nonpharmacological Approaches for NCBSs Include (Carnahan, 2010):

- Ensure safety and provision of adequate supervision.
- Analyze behavior(s) for clues to potential causes.
- Use environmental interventions.
- Provide structure—usual routines and predictability are important to allay anxiety.
- Provide pleasurable experiences.
- Do not rely on learning or memory.
- Educate caregivers and other support systems.
- Use of physical or chemical restraint is discouraged and may be used as last resort only, in order to ensure safety to self and others.
- Provide caregiver education for managing agitation (see Table 16-8).

Treatment Strategies and Pharmacotherapy for NCBS and Psychiatric Comorbidities: It is estimated that up to 90% of patients with dementia have psychiatric comorbidities (Trivedi et al., 2013). According to the Alzheimer's Association, depression affects up to 40% of persons with dementia, and prevalence is higher in vascular dementia than in AD. Depressive symptoms may present as initial manifestations of dementia and may fluctuate over time. There are more reports of decreased ability to concentrate and indecisiveness, and fewer reports of insomnia or hypersomnia, feelings of worthlessness and guilt, or thoughts of suicide and death. Diagnostic criteria for comorbid depression in dementia include the addition of irritability, social withdrawal, and isolation with frequent, concurrent apathy and anxiety.

The Cornell Scale for Depression in Dementia (CSDD) is a validated screening tool designed for use in the assessment of those who can communicate basic needs. The CSDD differentiates between the diagnostic categories and severity of depression (Alexopoulos, Abrams, Young, & Shamoian, 1988; Hancock & Larner, 2014). Other diagnostic instruments include the PHQ-9 and the GDS (see Depression). First-line

TABLE 16-8 Managing Agitation: Caregiver Education

DO	DON'T	PREVENTION	SAFETY
Ask permission to approach/ provide assistance. Use a calm approach. Slow down (do not rush patient or self). Use visual and verbal cues. Provide time and space. Limit stimuli.	Raise voice. Take offense. Corner or crowd. Rush or pressure. Criticize or condescend. Argue or attempt reason. Shame or ignore. Demand, force. Make sudden movements. Restrain.	Create a calm environment. Follow a routine with set structure and tasks. Use verbal cueing. Redirect attention. Use a clock and calendar. Monitor heat, cold, hunger, sleep, elimination, and comfort.	Use alarms and/or locks on doors and gates. Remove or secure firearms.

treatment strategies for comorbid depression include supportive psychotherapy and an SSRI such as citalopram (Celexa), fluoxetine (Prozac), or sertraline (Zoloft). If interventions are ineffective and/or suffering is severe and persistent, patients may be referred to psychiatry for consideration of ECT, a well-established and effective treatment option.

The prevalence of anxiety in those with dementia is 5% to 21% (Gellis, Kim, & McCracken, 2014; SAMHSA, 2012). GAD occurs in 5% of patients with AD. There is a higher percentage of anxiety with vascular and frontotemporal dementia, which is reflected in estimates of clinically significant anxiety as high as 70% (Ballard, 2000; Gellis, Kim, & McCracken, 2014). Notably, there is a high comorbidity of anxiety with major depression in AD (more than 75%). Manifestations of anxiety in dementia include restlessness, irritability, muscle tension, and fear. Respiratory symptoms, such as hyperventilation or difficulty catching one's breath, correlate with excessive anxiety and worry. Diagnostic instruments to assist in diagnosing anxiety in those with dementia include the Worry Scale and the Rating Anxiety in Dementia Scale. First-line treatment options include psychosocial interventions and an SSRI or buspirone (Buspar). Use of benzodiazepines (e.g., lorazepam) is discouraged due to sedating side effects and increased risk for falls.

Psychosis in AD is frequently present with other cognitive symptoms (global deficits, anosognosia), affective symptoms (depression, elevated mood), and behavioral symptoms (agitation and overt aggression). Approximately 18% of dementia patients present with delusions that typically manifest as persecutory and misidentification delusions (IOM, 2012). Hallucinations are present in approximately 14% of dementia patients, with the occurrence of visual hallucinations more than twice that of auditory hallucinations (IOM, 2012). Capgras syndrome (belief that a close relative or friend has been replaced by an impostor) and "phantom boarder syndrome" (belief that strangers are living in the home) may be associated with agnosia. Instruments to assist in diagnosing psychosis in dementia include the Behavioral Pathology in Alzheimer's Disease (BEHAVE-AD) scale and the Dementia Psychosis Scale. Behavioral and environmental interventions are first-line treatment strategies.

Before initiating pharmacological treatment with antipsychotic medications, the clinician should carefully analyze the risks and benefits of the use of such medications for each patient. Based on studies demonstrating increased risk for stroke and mortality in the elderly, the FDA has issued a black box warning for use of antipsychotic medications in this population. Judicial and cautious use of antipsychotic medication should be reserved for hallucinations, delusions, aggression, agitation, hostility, and uncooperative behavior that significantly interferes with care. Antipsychotics effective at low doses include aripiprazole (Abilify), haloperidol (Haldol), olanzapine (Zyprexa), quetiapine (Seroquel), risperidone (Risperdal), and ziprasidone (Geodon). Research evidence, FDA warnings, and clinical guidelines recommend use of antipsychotic medication only when 1) behavioral symptoms are due to mania or psychosis, 2) symptoms present a danger to the patient or others, or 3) the patient is experiencing inconsolable or persistent distress, a significant decline in function, or substantial difficulty receiving needed care (Ballard & Waite, 2006; Reus et al., 2016; Steinberg & Lyketsos, 2012; Wulsin, 2005).

Antipsychotic medication should not be used for sedation or restraint and should be used at the minimum dosage for the minimum amount of time possible. Careful monitoring for efficacy and side effects is required. Patients, caregivers, and those with power of attorney (POA) should be educated and informed about the risks and benefits of antipsychotic use, and the clinician may wish to consider obtaining written informed consent before initiating treatment with this type of medication (Reus et al., 2016).

Approximately 27% of dementia patients exhibit agitation and/or aggression. Prevalence increases as dementia progresses (13% in mild dementia, 24% in moderate dementia, and 29% in severe dementia). *Agitation* is defined as intermittent psychomotor hyperactivity, disinhibition, screaming, physical aggression, and combativeness. There are many potential causes for agitation, including the underlying pathophysiology of dementia (e.g., serotonergic deficiency) and an inability to communicate needs (e.g., hunger) or physical discomfort (e.g., pain, constipation). Psychosocial stressors that may induce agitation include changes in one's living situation, caregiver(s), or environment. Medical evaluation is needed to rule out occult medical problems, medication side effects, and delirium. Instruments that may assist in accurate diagnosis are the Abbey Pain Scale for those who cannot verbalize discomfort (Abbey et al., 2004), the BEHAVE-AD Scale, and the Cohen-Mansfield Agitation Inventory (Kalapatapu, 2010).

Interventions include evaluation and management of antecedents and/or causes of agitation, behavioral approaches (e.g., distraction, pleasurable activities), and calming activities or gentle sensory stimulation (e.g., music, art, routine/known physical activities such as folding towels). Pharmacotherapy options might include off-label use of mood stabilizers, such as carbamazepine or Depakote, to effectively manage agitation. Antipsychotic medications are not considered to be first-line treatment, because they are less effective than first believed; however, they should be considered when behaviors present a safety risk to self or others (Ballard & Waite, 2006; Reus et al., 2016). For sleep problems, low-dose doxepin may be effective and is FDA approved for insomnia.

Follow-Up: After the presumptive diagnosis of AD is made, the patient should be followed closely with pharmacotherapy initiation and following any adjustments for efficacy and side effects. Thereafter, follow-up visits every 3 to 6 months to assess physical, mental, and emotional status should be individualized to the patient. Reevaluation of cognitive function with the same scale used at diagnosis/baseline is useful to track cognitive stability or decline. The patient's functional status, ability to perform ADLs and instrumental ADLs, behavior, sleep, appetite, weight, elimination, communication, and signs of anxiety or depression should routinely be evaluated at follow-up visits. The physical examination is individualized to the patient and any episodic or chronic illnesses should be well managed to maintain optimum quality of life and avoid complications or worsening of cognitive function.

The ability to drive competently and safely is a frequent concern of family members, and clinicians are often asked to assist in making the difficult decision to revoke the patient's driver's license. Dementia exacerbates age-related changes in driving ability and poses a substantial risk to safe driving. The American Medical Association (AMA) National Highway Safety Administration guidelines state that a dementia diagnosis is not sufficient to withdraw driving privileges and recommend basing decisions on an individual's driving ability. Determining whether a patient with mild dementia is fit to drive presents a challenge to the clinician and begins with discussion of driving history, safety, and cessation with the patient. Data from physical and cognitive assessments, family and caregiver reports, and, when available, on-road testing will assist in the decision-making process. For patients deemed to be safe, discuss the necessity of future driving cessation and suggest driving training and self-limitation. A history of significant traffic problems, inability to find the way home, inattention, and psychosis are indications of progressive cognitive decline and driving skill deterioration. When driving safety is uncertain and the patient wishes to continue driving, a referral for on-road driving evaluation is needed. Those with mild dementia who pass an on-road driving test should be reevaluated and retested at least every 6 months. If driving safety is uncertain and the patient decides to stop driving, or if the patient is deemed unsafe to drive, the Department of Motor Vehicles and the patient should be notified by letter, and a copy of the letter should be placed in the patient's medical record. International consensus groups agree that a diagnosis of moderate-to-severe dementia precludes driving, and the driver's license should be revoked. Driving is essential to autonomy, and the vast majority of older adults rely on driving as their primary mode of transportation. Although the clinician's recommendation for driving cessation is distressing to patients and families, it is critical for patient and public safety (Pomidor, 2015).

Caregiver burden regarding patient care, household and financial responsibilities, and ability to provide adequate supervision and care at home should be determined at each follow-up visit. When the patient loses bowel and/or bladder control, his or her behavior becomes too difficult to manage, or the patient's or caregiver's safety is compromised, institutional care should be discussed.

Sequelae: Complications from severe dementia include immobility, swallowing disorders, malnutrition, and significantly increased risk of developing pneumonia, which is the most commonly identified cause of death among the elderly with AD and other dementias. Although there is a blurred distinction between death *with dementia* and death *from dementia*, since 2000 the AD mortality rate has been on the rise. AD is the sixth leading cause of death in the United States and the fifth leading cause of death among those 65 years of age and older (Alzheimer's Association, 2016; Xu, Murphy, Kochanek, & Bastain, 2016).

Prevention/Prophylaxis: A recent comprehensive evidence review commissioned by the Agency for Healthcare Research and Quality (AHRQ) was undertaken by the Duke Center for Evidence-Based Practice to identify factors associated with prevention of cognitive decline and AD. Factors such as nutrition, medical conditions, social/economic/behavioral factors, prescription and nonprescription medications, genetics, and environment were studied. The project included 25 systematic reviews and 250 primary research studies, both observational and randomized clinical trials (RCTs). The primary conclusion was that there was a dearth of evidence for any strong recommendations for preventive interventions for AD (Williams, Plassman, Burke, Holsinger, & Benjamin, 2010). Physical activity and cognitive engagement were factors that demonstrated fairly constant association with prevention of cognitive decline and AD; diabetes, depression, smoking, and APOE e4 were associated with increased risk of cognitive impairment and AD.

Referral: Neurology, geriatric psychiatrist, geriatric nurse practitioner, psychiatric mental health nurse practitioner or clinical nurse specialist, psychology, support groups for patients and caregivers.

Education: The impact of AD on caregivers expands as the course of illness progresses. Caregivers, family, and friends of those with AD experience new or changing responsibilities such as shopping, meal preparation, cleaning, financial management, provision of ADLs, and managing behavioral issues. Caregivers also experience financial burden, physical and emotional stress, fatigue, and depression. It is estimated that 34% of caregivers are 65 years or older, and 23% are within the "sandwich" generation, providing care for family members (Alzheimer's Association, 2016).

Patient and caregiver education and support needs include:

- Disease process, course of illness, staging
- Anticipatory guidance, life planning, advance directives, POA

Preservation of the following:

- Safety at all times
- Dignity, respect, and self-esteem
- Independence and autonomy for as long as possible/practical/safe
- Daily schedules and routines
- Least restrictive environment
- Cognitive, behavioral, and pharmacological therapies

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Dementia with Lewy bodies should be suspected in patients presenting with dementia accompanied by hallucinations, fluctuating cognition, and with the onset of spontaneous parkinsonism subsequent to cognitive decline.	C	Donaghy & McKeith, 2014 Neef, 2016
The diagnosis rests on clinical criteria, but evaluation should include a validated brief cognitive test and laboratory tests focused on treatable causes.	C	Hagen, 2016
Laboratory evaluation should include CBC, TSH, serum calcium and electrolytes, vitamin B_{12}, and fasting glucose levels to examine for treatable causes of cognitive decline. Consider serum folate, tests for syphilis, and HIV serology if patient is at risk for those conditions.	C	Lyketsos, 2016b
There is currently no adequate, robust evidence to recommend the use of any nonpharmacological intervention to reduce wandering in dementia.	A	Futrell, Melillo, & Remington, 2014
No evidence has been found of any significant general improvement in manifestations of agitation, other than aggression, among demented patients treated with haloperidol, compared with controls.	A	Lonergan, Luxenberg, Colford, & Birks, 2002
The use of atypical antipsychotics for behavioral and psychological symptoms of dementia is associated with increased mortality.	A	Maust et al., 2015 Reese, Thiel, & Cocker, 2016 Schneider, Dagerman, & Insel, 2005
Memantine has a small, clinically detectable effect on cognitive function and clinical decline measured at 6 months in patients with moderate-to-severe AD.	A	McShane, Areosa Sastre, & Minakaran, 2006
ChEIs, donepezil, galantamine, and rivastigmine are efficacious for mild to moderate AD.	A	Birks, 2006

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

DEPRESSION

Signal Symptoms: Pervasive and sustained mood of sadness, discouragement, lack of pleasure in usual activities, guilt, loss of motivation, low energy, and sleep and/or appetite disturbances.

Description: Transient symptoms of depression are normal, healthy responses to life stressors and disappointments. Pathological depression manifests when a patient's coping skills are inadequate and adaptation to stressful life events or loss is ineffective. Depression is described as a pervasive feeling of sadness or a lack of interest or pleasure in previously enjoyed or usual activities. Feelings of guilt, low self-esteem, sleep and appetite disturbances, low energy, and poor concentration are common. Late-life depression is defined as a new onset of depression occurring in one's sixties. Depression may be categorized as a single episode or recurrent, and further qualified as mild, moderate, or severe, with or without features such as melancholy, mood-congruent, catatonia, peripartum onset, or with seasonal pattern. Types of geriatric depression include MDD, vascular depression, dysthymia, and depression that manifests as a comorbid condition in dementia, bipolar disorder, and executive dysfunction (Glover & Srinivasan, 2013; Taylor, 2014).

Etiology: Depression is not a normal part of the aging process. Causative factors for depression include physiological

influences such as medication side effects, neurological disorders, cardiac disease, neuroendocrine disturbances, electrolyte and hormonal disturbances, and nutritional deficiencies in vitamin B_{12}. Vitamin D deficiency strongly correlates with depression, raising the risk of depression two-fold (Lapid, Cha, & Takahashi, 2013).

Psychosocial and cognitive theories postulate internalized loss with ego dysfunction, learned helplessness, loss and bereavement, and cognitive distortions with negative attitudes and thoughts as contributors to depression (Aziz & Steffens, 2013). Difficulty accomplishing developmental tasks associated with life stages may contribute to and/or be a consequence of depression. Environmental and social factors contribute to depression when major stressors occur and social support systems are inadequate. Depression can be triggered by a single major life event or a combination of events and stressors. Loss of any kind may contribute to depression and in the older adult may include loss of a spouse/loved one or loss of mobility, independence, one's home, and financial security along with deterioration of physical health and social isolation.

Biological theories point to impaired synthesis, deficiencies, increased uptake, and increased metabolism or breakdown of the neurotransmitters serotonin, norepinephrine, and dopamine as causative factors in depression (McKinney & Sibille, 2013). The comorbidity of depression, vascular disease, vascular risk factors, and the association of ischemic cerebral lesions with distinctive behavioral symptoms supports the "vascular depression" hypothesis. This hypothesis proposes that cerebrovascular disease may predispose, precipitate, perpetuate, or exacerbate some geriatric depressive syndromes (Gleason, Pierce, Walker, & Warnock, 2013).

Occurrence: Aging adults are at increased risk of depression. There is a 10% to 44% prevalence of *depressive symptoms* in the elderly, and the percentage is higher in those with disabilities or physical illness. Approximately 2% to 16% of elders have MDD, and there is an increase in recurrence with increased age. The prevalence of depression in older adults varies depending on the setting in which they live: 2% to 4% in the community, 13% to 24% in assisted living or residential care facilities, and 12% to 20% in nursing home residents. Nursing home residents are three to four times more likely to suffer from depression compared to older adults living in the community (Taylor, 2014).

Unfortunately, up to 85% of older adults with depression remain untreated, because it is often underdiagnosed, misdiagnosed, or obscured by comorbidities or somatic complaints that are considered to be physical problems (Ismail, Fischer, & McCall, 2013). Additionally, older adults and some cultural groups may be unwilling to discuss emotional distress and suffering due to social stigmas attached to mental illness (Sirey, Franklin, McKenzie, Ghosh, & Raue, 2014). An environmental barrier to accurate diagnosis and treatment of depression is ineffective communication among the treatment team in residential care facilities, where residents who are more vocal or agitated are more likely to receive psychiatric consultation than those who may be depressed.

Age: Age 65 years and older.

Gender: Statistics reveal that depression is more common in women than men; however, it is thought that although men experience and suffer from depression, they either do not report or under-report their depressive symptoms, therefore skewing the epidemiological data.

Ethnicity: Those considered members of a minority culture living among the dominant majority have higher risk of depression, possibly due to less resources, greater health burdens, and lack of health insurance.

Contributing Factors:

Genetics: Higher risk of depression among those with siblings or parents with depression.

Psychosocial: Single or multiple losses: loved ones, independence, mobility, function, financial security, autonomy, privacy, social network; changes in environment; admission to health-care facility; limited social support, loneliness, negative life events.

Physical Illnesses: Diabetes, cancer, Parkinson's disease, AD, cerebrovascular accident, obstructive sleep apnea, vascular brain lesions, heart disease, arthritis, hypothyroidism, vitamin deficiencies, anemia, COPD, any chronic illness/pain syndromes, disabilities.

Psychiatric Illnesses: Alcohol, substance abuse, family or personal history of psychiatric illness or depression

Medications: Anxiolytics, sedatives/hypnotics, antipsychotics, cardiac medications, antihypertensives, beta blockers, H_2 blockers, narcotic analgesics, and steroids, either alone or in combination with other medications (monotherapy, polypharmacy, drug–drug interactions), can cause behavioral, affective, and cognitive changes. The Beers list and the Centers for Medicare and Medicaid Services (CMS) unnecessary medications list indicate medications to avoid prescribing for the elderly (American Geriatrics Society, 2015). Special attention should be paid to medications affecting kidney function and the potential for drug-to-drug interactions.

Signs and Symptoms: Symptoms of depression may encompass four domains: affect/mood, cognition, physiological, and behavior. Sadness, anhedonia, apathy, helplessness, hopelessness, worthlessness, and loneliness are common affective symptoms. Ambivalence, uncertainty, inability to concentrate, confusion, poor memory, slowed speech, lack of motivation, a pessimistic outlook, negative thoughts, self-criticism, and poor self-esteem are common cognitive symptoms. Assess for perceptual disturbances, such as hallucinations, illusions, and delusions, to determine if psychotic features of an agitated depressive episode are present.

Sleep, appetite, and energy disturbances; weight change; constipation; pain; headache; decreased libido; sexual nonresponsiveness; and exaggerated concerns over bodily functions are common physiological presentations in depression. Note behavioral symptoms such as psychomotor retardation or agitation, irritability, poor personal hygiene, tearfulness, and social withdrawal. It is of paramount importance to question the patient about suicidal ideation and/or plans and assess accessibility to lethal weapons. Some patients experience excessive worry, preoccupation, and generalized anxiety. Clinical presentation may include inattention to personal appearance, poor hygiene, poor eye contact, and a blunted or flat affect; many of these manifestations appear similar to dementia or delirium (Downing, Caprio, & Lyness, 2013).

The DSM-5 (APA, 2013) diagnostic criteria for MDD include sustained, disruptive, and pervasive depressed mood or loss of interest or pleasure. With the exception of suicidal ideation and weight change, five or more of the following symptoms must be present for most of the day, on most days, over a 2-week period:

- Depressed mood as reported in subjective report or as observed by others
- Obsessive rumination or worry
- Change in appetite and significant change in weight
- Change in sleep pattern: insomnia (difficulty falling asleep or staying asleep, waking earlier than necessary, inability to fall back asleep), hypersomnia, frequent napping
- Psychomotor agitation or retardation (including as observed by others)
- Fatigue or loss of energy
- Feelings of hopelessness
- Feelings of worthlessness, excessive guilt
- Diminished concentration, indecisiveness
- Recurrent thoughts of death, suicidal ideation or gestures, suicide attempt, suicide plan

Distinct clinical manifestations in older adults include:

- Report of lack of emotions (versus depressed mood)
- Excessive concern with bodily functions
- Seeking reassurance and support
- Isolative, withdrawn behavior
- Change in previous level of function, decline in ADLs
- Feeling overwhelmed, easily frustrated, excessive crying
- Irritability, fearfulness, agitation, anxiety
- Transient, recurring symptoms, diurnal fluctuations or pattern
- Minimizing expressed death wishes or passive suicidal behavior

At the first encounter, the patient should be interviewed individually. Goals of the interaction should include establishing a therapeutic, trusting alliance, which can then enhance the discussion of sensitive topics such as sexuality, abuse, and suicidal thoughts. The subjective history should begin with open-ended questions, which can then be followed by specific and focused questions. The history should include present illness, past psychiatric history, family history including mental health, medical history, prescription and OTC medication use, social history (education, work history, important relationships, sexual activity, spirituality, and current living arrangements), and functional assessment. Assessment should include a comprehensive physical examination to look for secondary causes of depressive symptoms, a full mental health assessment, and a mental status examination if confusion or cognitive impairment is manifested.

Diagnostic Tests: Diagnostics to assess for underlying or undiagnosed medical causes of depressive symptoms should be ordered. Standard blood work includes CBC with differential, CMP, lipid panel, thyroid function studies (TSH with reflex T_4), and serum vitamin B_{12} and vitamin D levels. Clinical evaluation instruments and scales are useful in assessing symptoms and severity of depression, cognitive impairment, and disability. Such instruments provide objective measures of signs and symptoms of illness and help in establishing baseline information with which future assessments can be compared in evaluating efficacy of treatment and interventions. Instruments also provide guides to dialogue, and allow the practitioner to further discuss key concerns with the patient.

Depression Scales

- Geriatric Depression Scale (GDS, long and short forms)
- Beck Depression Inventory (BDI)
- Patient Health Questionnaire (PHQ–9)
- Hamilton Depression Scale (HAM-D)
- Quick Inventory of Depressive Symptomology—Self-Report (QIDS-SR)
- Scale for Suicide Ideation

Overall Cognitive Functioning

- Folstein's Mini-Mental Status Exam (MMSE)

Differential Diagnosis:

- Mood disorder due to general medical condition such as diabetes, cancer, epilepsy, stroke, multiple sclerosis, Parkinson's disease, cardiac disease, acute prolonged illness, or injury
- Substance-induced mood disorder from alcohol, illicit drugs, prescription drug abuse, or side effects
- Dysthymic disorder that manifests symptoms of mild depression and may have an early or late onset and is chronic in nature, lasting more than 2 years
- Bipolar affective disorder in which bipolar depression is often misdiagnosed as unipolar depression
- Delirium
- Dementia
- Grief and bereavement
- Somatization disorder
- Sleep disorder, sleep apnea

Treatment: The treatment goal for depression is full remission and recovery. Additional goals of treating late-life depression include: prevent or reduce relapse or recurrence, improve quality of life and functioning, improve medical health, and reduce mortality. The initial step in treating depression in older adults is to evaluate the present medication regimen and remove or change any medications that may contribute to symptoms. Treat any systemic disorder that may have predisposed the patient to depression. Ensure adequate nutrition, elimination, sleep, and physical comfort (Karlsson, Johnell, Sigstrom, Sjobert, & Fratiglioni, 2016; Taylor, 2014). Optimum depression treatment for older adults should not be managed with medications alone and should include social interventions and possibly psychological modalities (Kok, 2013).

Pharmacotherapy: The strategy for pharmacological management is to achieve complete and sustained relief or remission of depressive symptoms with the least amount of distressing side effects. The most widely prescribed antidepressants and with greatest evidence for achieving remission are the SSRIs. Other types of antidepressants that are recommended as first-line treatment include SNRIs, serotonin partial agonist/reuptake inhibitors (SPARIs), norepinephrine-dopamine reuptake inhibitors (NDRIs), selective norepinephrine reuptake inhibitors (NRIs), and alpha-2 antagonists (see Table 16-9).

While the tricyclics (TCAs) and mono-amine oxidase inhibitors (MAOIs) are still available, these are no longer considered first-line recommendations. With an array of

TABLE 16-9 First-Line Treatment With Antidepressant Medications

TYPE OF ANTIDEPRESSANT	MEDICATION—GENERIC: TRADE NAMES
SSRI	citalopram: Celexa escitalopram: Lexapro fluoxetine: Prozac, Sarafem fluvoxamine: Luvox paroxetine: Paxil, Pexeva, Brisdelle sertraline: Zoloft
SNRI	desvenlafaxine: Pristiq, Khedezla duloxetine: Cymbalta milnacipran: Savella levomilnacipran: Fetzima venlaxafine: Effexor, Effexor XR
NDRI	bupropion: Wellbutrin
Serotonin modulator	nefazodone: Dutonin, Nefadar, Serzone trazodone: Desyrel, Oleptro
Norepinephrine-serotonin modulator	mirtazapine: Remeron

TABLE 16-10 Common Side Effects and Potentially Serious/Dangerous Side Effects of Antidepressants

SSRIs, NDRIs, SNRIs: common side effects	Mild nausea, loose bowel movements, anxiety, headache, insomnia, increased sweating, weight gain/loss, sexual dysfunction, vivid dreams, rash, syndrome of inappropriate SIADH
Serotonin modulators: side effects	Sedation, weight gain, mild anticholinergic symptoms; priapism, a side effect of trazodone, is a medical emergency
Serotonin syndrome (excessive serotonin)	Agitation, restlessness, diarrhea, diaphoresis, muscle twitching or rigidity, confusion, hyperpyrexia, seizures, tachycardia, death
SSRI discontinuation syndrome	Dizziness, headache, paresthesia, nausea, diarrhea, insomnia, and irritability; seen with missed doses and abrupt discontinuation of antidepressants with a short half-life

Abbreviations: NDRIs = norepinephrine-dopamine reuptake inhibitors; SIADH = secretion of antidiuretic hormone; SSRIs = selective serotonin reuptake inhibitors

antidepressant medications available, the specific choice should be based on the uniqueness of each individual. Considerations include short- and long-term side effects, least-desirable side effects, safety, and cognition. Additional factors in selection are the patient's primary symptoms, symptom clusters, current medications, past history of medications used, efficacy and side effect tolerance, and family history of effective medication use. In older adults, it is generally best to start with a low dose and titrate upward slowly, depending on the patient's response (Cleare et al., 2015; Stahl, 2013).

It is very important to maximize the dose of an antidepressant for an adequate period of time to attain efficacy and full remission. Monitor and evaluate therapeutic response to antidepressant therapy, and observe for side effects, tolerance, and unremitting symptoms of depression. Studies show that the majority of patients on a single agent (monotherapy) do not tolerate it, have limited or no response, stop the medication within the first 3 months, or never receive an adequate dose or trial of medication. Consequently, monotherapy is effective in approximately one-third of patients, and the great majority do not reach remission. For those patients who do not respond adequately to monotherapy, other treatment strategies may be employed. Switching agents within the same class or combining different types of antidepressants (e.g., a combination of sertraline and bupropion) may result in symptom remission. With multiple failed trials of monotherapy or combination therapy, the patient may be considered to have treatment-resistant depression. Several second-generation antipsychotic agents are FDA approved for augmentation to antidepressant therapy in treatment resistant depression: aripiprizole (Abilify), quetiapine extended release (Seroquel XR), and olanzapine (Zyprexa). Providers who are not familiar or comfortable with switching, combination, and augmentation strategies or off-label use of mood stabilizers, other second-generation antipsychotics, or stimulant medications to augment antidepressants should refer patients to a psychiatric mental health nurse practitioner or a psychiatrist (Han et al., 2013).

Promoting adherence is important to achieve full recovery. Partnering with the patient through inquiry into prior antidepressant use, shared decision making, and education regarding expected therapeutic response time will positively affect patient adherence to the therapeutic regimen. Discussion regarding common and potentially bothersome side effects and the likelihood that they will wane over time promotes adherence. The sexual side effects of SSRIs may not wane over time and should be addressed as depressive symptoms remit and the patient expresses concern about and/or regains desire for sexual activity. Educate patients about serious or potentially dangerous effects of antidepressant medication and instruct them when it is necessary to notify their prescriber (see Table 16-10). As patients begin to feel better, advise them to continue the medication and explain the risk of stopping medication abruptly or too soon. Patients should be educated about the dangers of combining herbal remedies such as St. John's wort (hypericum) with SSRIs, as this raises the risk of serotonin syndrome.

Length of Pharmacotherapy Treatment and Follow-Up: When initiating or changing the dose of antidepressant medication, it is critical to monitor the patient closely for efficacy and side effects. Observe for worsening of depression, suicidal thoughts or actions, unusual changes in behavior, agitation, and irritability. Educate patients, families, and caregivers to pay close attention to sudden changes in mood, behaviors, thoughts, or feelings, as well as suicidal thoughts, and to report these concerns immediately to the provider. It is well established that as depression begins to remit, patients may become more energized and may be able to follow through on prevailing suicidal ideation and plans. Factors that increase risk for suicide include severe insomnia, being a member of a sexual or cultural minority, chronic pain or disability, a family history of suicide (especially in the same-sex parent), previous suicide attempts, loss of a loved one, lack of employment, and increased financial burden. Every patient should be screened for suicidal thoughts during pharmacological

initiation, dosing phases, and at every subsequent encounter. A return office visit every 2 to 4 weeks is strongly recommended; if a physical visit is not possible, telephone contact should be made (Kok, 2013; U.S. Administration on Aging, 2013).

Length of antidepressant treatment varies depending on whether it is a first or recurrent episode. With each recurrence, there is an increased risk of future recurrence if medication is discontinued, and as a result, antidepressant therapy must be maintained for longer periods of time if not indefinitely. The *acute phase* of treatment lasts up to 3 months after the start of medication, with the goal to achieve remission. Close follow-up is critical and highly individualized; however, there should be patient contact within the first month and additional contact every 4 weeks or sooner with dosage adjustments, switching, combination, or augmentation. The *continuation phase* of treatment begins once remission is achieved, and medication should be continued for at least another 4 to 12 months. With one lifetime episode of MDD, a trial discontinuation of medication is optional if the patient has remained asymptomatic for the duration of the continuation phase of treatment. Patients in the continuation phase of treatment should be seen every 5 or 6 months or sooner. The *maintenance phase* of antidepressant therapy is specifically for patients with two or more lifetime episodes of MDD. These patients should be maintained on medication at the same dosage for another 15 months to 5 years. Patients in the maintenance phase of treatment should be seen every 6 to 12 months or sooner.

Psychotherapy: Many patients continue to experience distressing symptoms of depression despite one or even several pharmacological interventions. Research studies demonstrate that supportive counseling, psychotherapy, cognitive behavioral therapy, problem-solving therapy, interpersonal psychotherapy, reminiscence therapy, group therapy, and support groups improve remission and recovery rates when patients actively participate (Francis & Kumar, 2013). For patients with mild depression, therapy alone may be sufficient as a treatment strategy. Those with moderate to severe depression experience significantly better outcomes with concurrent psychotherapy and pharmacotherapy.

Complementary and Alternative Therapies: Complementary or alternative treatment modalities may be effective at preventing or reduce depressive symptoms. Evidence supports dietary or supplemental intake of omega-3 fatty acids (Grosso et al., 2014) and vitamin D (Lapid et al., 2013). St. John's wort has been shown to reduce depressive symptoms among elder patients. Additional minerals, vitamins, and herbs that may help to reduce depression include SAMe and folic acid. However, these alternative modalities are not without risk and should not be recommended lightly. It is important to consider which supplements may induce the metabolism of other medications, which inhibit metabolism, which are metabolized by the cytochrome P450 enzymes, and which may increase the risk of vascular bleeding (Nyer et al., 2013).

Patients should be encouraged to engage in activities that promote physical and mental wellness. Activities such as exercise, tai chi, massage, and yoga improve physical and mental well-being and may reduce or even prevent depression. Evidence supports aerobic exercise for reducing the somatic symptoms of depression, although moderate exercise may not be as effective for older frail adults residing in long-term care facilities (Underwood et al., 2013). Some older adults benefit from spiritual or religious therapy. Engaging with music actively (playing or composing) and listening has several benefits, including promoting mental well-being, facilitating expressions of emotions, and enhancing antidepressant medication therapy (Nyer et al., 2013).

Integrative Health: While there is strong evidence that integrative health-care teams optimize health-care delivery and provide sustained improvements, older populations have been of less focus. Emerging evidence does support integrative team management for depression among older adults, including a significant reduction of depressive symptoms, improved medication adherence, and remission. There is also evidence that patients find collaborative care models to be acceptable and useful (Cartier, 2013).

Follow-Up:

- Use diagnostic scales/instruments (symptom and severity tools) to objectively assess progress compared to baseline evaluation.
- Assess for therapy and medication adherence, response, and side effects.
- Assess suicidal ideation.
- Titrate medication dose for total remission.
- If trial discontinuation, taper over 2 to 4 weeks.
- Monitor for early signs of recurrence.

Sequelae: There are many potential negative outcomes if depression is not identified and treated appropriately, and full remission and recovery is not reached. Complications of depression include impaired interpersonal relationships, interpersonal problems, increased medical comorbidity and medical risk factors over time, worsening of cognitive impairment, self-neglect, decreased mobility, increased disability, and increased mortality from suicide and interactions with medical conditions such as cardiovascular and cerebrovascular disease. Additionally, there is an increased risk of homicide/suicide (murder of loved one/spouse before suicide). Depression diminishes health and overall life span.

Prevention/Prophylaxis: Prevention of depression encompasses the initial episode, recurrent episodes, and protection from complications. Studies show that minimizing risk factors has demonstrated a reduction in the incidence of depression (Kiosses, 2016). Major risk factors for depression include stress, stressful life events, responses to stress, unhealthy coping styles, and living with chronic pain. Stress management, relaxation exercises, and problem-solving and decision-making skills can be taught and maintained as a prevention strategy. Another major risk factor for depression is persistent negative thinking, which can be modified with cognitive restructuring, self-monitoring of thought processes, and strategies such as increased awareness and mindfulness.

Poor health, loss of independence, and physical disability or pain place elders at high risk for depression. Self-care strategies, such as a nutritious diet, exercise, weight management, social interaction, cognitive stimulation, counseling, and a positive attitude toward life and aging, aid in maximizing physical and mental health. Prevention of vascular causes of late-life depression may be accomplished through identification and modification of shared risk factors, which include vascular inflammation, atherosclerosis, dyslipidemia,

hypertension, diabetes, alcohol consumption, obesity, and smoking. The goals of prevention are to decrease complications of late-life depression, thereby increasing overall health and quality of life, and prolonging the life span (Karp et al., 2015).

Referral: Patients who should be referred to psychiatry are those who have prominent suicide/self-harm thoughts or previous suicide attempts, a questionable diagnosis, possible bipolar disorder or comorbid psychiatric conditions, persistent residual symptoms, and partial or no response to monotherapy, combination, augmentation, or adjunctive medications. For counseling, refer the patient to a psychiatric mental health nurse practitioner, a psychiatric clinical nurse specialist, or a licensed clinical psychologist, and provide resources for support groups and appropriate service professionals and agencies.

Education: Patient and family education should include information about the disease process; causative factors; heritability; risk of relapse or recurrence; suicide risk; treatment strategies and recommendations; psychotropic medication use; side effects; length of time for medication efficacy; and use of herbs, vitamins, and supplements.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
No significant difference in performance of screening instruments; implement screening only if plan includes diagnosis, treatment, follow-up.	B	Siu & USPSTF, 2016
Best diagnostic tool is a detailed history performed by the practitioner in context of DSM-5 criteria and clinical judgment.	C	Glover & Srinivasan, 2013
Antidepressant medications and psychotherapy are equally effective; SSRIs raise risk of upper GI bleed.	A	Siu & USPSTF, 2016
Only half of patients respond to the first medication used regardless of class selected, so reevaluate effectiveness at least monthly until symptom remission.	A	Siu & USPSTF, 2016 Han et al., 2013
Risk factors for depression in older adults include chronic pain, comorbid illness, poor self-perceived health, functional disability, personality traits, inadequate coping strategies, previous psychopathology, small network size, being unmarried, qualitative aspects of social network, stressful life events, and female gender.	A	RNAO, 2016

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ELDER ABUSE

Signal Symptoms: Elder abuse is a global public health problem that can be difficult to detect (Dong, 2015). Tell-tale signs indicative of abuse may include bruises, broken bones, poor personal hygiene, abrupt changes in finances, sudden withdrawal in normal activities, unexplained weight loss, and excessive power or control by a close family member or friend (Administration on Aging, 2016).

Description: According to the National Adult Protective Service Association (NAPSA), elder abuse is a form of mistreatment resulting in harm to a vulnerable person (NAPSA, 2016). The Department of Health and Human Services Administration on Aging states that the term *elder abuse* describes any knowing, intentional, or negligent act performed by a caregiver or another person that may cause harm or risk of harm to an older adult who is vulnerable (Administration on Aging, 2016). Types of abuse include:

- Physical abuse: causing physical pain or injuring a vulnerable elder
- Sexual abuse: sexual contact with a vulnerable elder without his or her consent
- Neglect: failing to provide food, shelter, health care, or protection for a vulnerable elder
- Exploitation: the taking of funds, property, or any assets of a vulnerable elder without legal consent and not for the benefit of the elder

- Emotional abuse: using verbal or nonverbal means to cause mental pain, anguish, or distress in an elder
- Abandonment: deserting the vulnerable elder once someone has assumed responsibility for that individual
- Self-neglect: the elder fails to perform the needed activities to protect his or her own health and safety (lacks food/utilities, refuses medications, hoards, lives in unsafe conditions, neglects his or her grooming/appearance, is unable to handle finances, is isolated, is disoriented, develops a dependence on drugs and/or alcohol) (Administration on Aging, 2016; NAPSA, 2016)

Elder abuse is highly under-reported to authorities, but is more common in the community setting compared to institutional settings. It is estimated that 1% to 2% of the elderly population experiences abuse (Powers, 2014).

Etiology: Abuse of vulnerable elders can happen in many settings, from their private home environment to that of a community nursing home or even a hospital setting. The days when the family lived close by and took responsibility for its members are fading. Families are spread out and lack the close communication of past years. This leaves the one member residing close to the elder adult to be fully responsible. This is a stress to the responsible family member, and little support is generally provided for this individual's efforts. Common features of perpetrators committing elder abuse are partners or spouses living with the victim, history of alcohol or drug abuse, history of mental illness, history of unemployment, and being socially isolated (National Institute of Justice, 2009).

Lachs and Pillemer (2015) suggest that the abused elder is more likely an older female. She generally has a physical impairment and is in poor health. She may either live alone, with the abuser, or in a household with many members. The abuser is generally found to be a male who has a history of past or current substance abuse, has mental health issues, is socially isolated, and has a history of past trouble with law enforcement. He may be financially dependent on the elder and be under major stress. The Patient Page on Elder Abuse in the Journal of the American Medical Association (Hildreth, Burke, & Glass, 2009) states that in 90% of cases of abuse of an adult age 60 years or older, the culprit is a family member. These cases result in an increased risk of death for the vulnerable elder involved in the abusive situation.

Occurrence: The actual numbers of cases of elder abuse are difficult to quantify due to the variance in mandatory state reporting laws. Estimates include 1 in 10 Americans 60 years of age and older have experienced abuse, although others estimate the number to be 5 million. One study reported only 1 in 14 cases of elder abuse are reported to authorities (National Council on Aging, 2016). The National Elder Abuse Incidence Study states that for every one reported case of abuse there are about five that go unreported, thus we may be seeing just the tip of the iceberg (Administration on Aging, 2014). These numbers are expected to increase with the burgeoning of the elder population.

Age: One out of every 10 seniors over age 60 years is a victim of elder abuse. Sixty percent of abusers are close family members.

Gender: Women are more likely to be victims of elder abuse than men.

Ethnicity: Occurs in all ethnic groups and socioeconomic classes.

Contributing Factors: Social isolation, frailty, physical or mental disability, and dependency are some of the contributing factors. Living with others can also be a risk factor.

Signs and Symptoms: The Administration on Aging provides several examples of potential abuse to observe in older patients. Physical signs include slap marks and unexplained burns or blisters, especially circular, as from a cigarette. Unusual bruising in areas that are not usually thought to be accidental can raise suspicion of possible abuse. Any bruising around breasts or genital area and/or unexplained sexually transmitted diseases might indicate sexual abuse. Emotional abuse can be suspected if a patient withdraws from his or her normal activities or has an unusual change in his or her level of alertness or any other change in behavior that has not been previously observed. Furthermore, the inability to interview the elder alone may provide suspicion of elder abuse. Often, the affected victim fears placement in long-term care or being abandoned and, thus, tolerates the abuse (AOA, 2016; Powers, 2014).

The National Institute of Justice warns sudden changes in finances, unexplained changes to wills or trusts, unexplained bank withdrawals, and loss of property may indicate a problem of exploitation of an elder. Signs of neglect can be decubitus that is untreated, evidence of restraint use, suspicious bruising, medication nonadherence, missed medical appointments, untreated medical conditions, unkempt appearance, refusal of care, and unusual weight loss. These are all concerns that need to be followed up as possible signs of caregiver neglect or self-neglect, if no caregiver is involved (Lachs, 2000; Powers, 2014).

Diagnostic Tests: The basis to establishing a baseline of health is a head-to-toe examination of the patient in question. Having the patient unclothe to a patient gown will allow examination of areas that may otherwise be hidden by clothing, socks, or shoes. It is important to examine the patient alone without the caregiver present. This individual time at first allows the practitioner to ask indirect and nonthreatening questions of abuse, as most older adults do not self-report abuse, followed by more direct questioning (Burnett, Achenbuam, & Murphy, 2014; Lachs & Pillemer, 2015). Interviewing the caregiver separately may provide clues to differences in the victim and the alleged perpetrator's story regarding suspicious physical findings. The baseline examination will dictate the needed laboratory tests and any imaging required to establish a diagnosis. If memory or alertness is suspected, a formal assessment of cognition and mood needs to be performed (Lachs & Pillemer, 2015). In complex cases of suspected elder abuse, a referral to a geriatric team for a comprehensive geriatric assessment may be warranted (Burnett et al., 2014).

Differential Diagnosis:

- Unintentional injury
- Falls
- Thin, friable skin with easy bruising

Treatment: If elder abuse is suspected, it is the health-care professional's responsibility—and in most cases his or her legal obligation—to report this to either 911 or the state elder

abuse hotline (National Council on Child Abuse and Family Violence, 2015). Carefully collect information regarding the patient, using physical findings, patient's functional abilities, testing results, and verbal information from the patient and his or her caregivers. Use the interdisciplinary team and speak with social workers, nursing staff, and others who may have interacted with the patient and caregiver. Document all findings, because they may be required to be presented in court later. Especially document any differences in verbal accounts between the patient and his or her caregiver. It is also prudent to photograph suspicious injuries, as well as measuring or comparing size of injury with a familiar object if a ruler is not available (Cooper, Selwood, & Livingston, 2009; Gibbs, 2014). When a patient has dementia, a history can be difficult to evaluate. This collection of information will assist the adult protective services case workers in their case investigation. The information may protect the patient from further exploitation, neglect, or abuse. Be sure to follow up with the case workers to determine the outcome of the case. It sometimes takes several reports before the true picture of neglect, exploitation, or abuse can be investigated thoroughly, and the elder individual moved to a safe environment.

Follow-Up: Once the elder abuse is reported, adult protective services will become involved and take action to protect the elder from further abuse. Contact them to obtain information on the outcome.

Sequelae: The elder may need to be moved to a safe environment, such as a long-term care setting or the residence of another family member. There may be legal proceedings that can involve the health-care professional's testimony based on the observations and findings that have been carefully documented. The abuser may be incarcerated for his or her acts. Sometimes what is required is an evaluation of support in resources to assist the caregiver in improving his or her knowledge of the caregiving role. This requires education and counseling from community resources (Reuben, 2013). Unfortunately, risk of death increases in elder abuse cases (Lachs & Pillemer, 2015).

Prevention/Prophylaxis: Prevention can come in the form of education at multiple levels. The caregiver especially requires education on the elder's care. This can be done in part on regular clinical visits with the primary care provider, in community offerings, at Area Agency on Aging seminars, and with other local resources. In some communities, there are social workers and nurses who provide support services to families through private agencies. Respite programs can be accessed to lessen the stress on the caregiver by providing much-needed time for themselves. Adult day-care services are available in larger communities, as are meal services and other resources that can assist the caregiver in lessening the burden of providing care.

Health-care professionals need to incorporate routine screening of elder abuse in at-risk populations (Dong, 2015). Education of health-care professionals is also needed so that there is an understanding of those responsible for the care of older adults. Providing education on normal aging body expectations found in the older patient and what to do when there are red flags that require further investigation can help professionals provide better care for elders.

Referral: See Treatment.

Education: See Prevention.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
One in four vulnerable elders are at risk of abuse, and only a small proportion of this is currently detected. Elders, family, and professional caregivers are willing to report abuse and should be asked about it routinely. Valid, reliable measures and consensus on what constitutes an adequate standard for validity of abuse measures are needed.	A	Cooper, Selwood, & Livingston, 2008
Findings of this systematic review of elder abuse suggest elder abuse is common, particularly in minority community-dwelling older adults. This review also confirms knowledge gaps, inconsistent definitions, and insufficient findings regarding intervention and prevention research in cases of elder abuse.	A	Dong, 2015
The state of current elder abuse research is comprised primarily of descriptive, observational case studies, no meta-analyses, and a few intervention trials. Little evidence is available that supports any intervention to prevent elder abuse. More rigorous elder abuse research and more investigators are needed.	A	Baker, Francis, Hairi, Othman, & Choo, 2016

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GRIEF AND BEREAVEMENT

Signal Symptoms: May be none; may have feelings of depression with associated symptoms, crying, insomnia, fatigue, anger, sadness, withdrawal, emptiness, decreased appetite, difficulty with concentration, yearning. Distressing memories, feeling attracted to things associated with the deceased, (Powell, 2016; Shear, Reynolds, Simon, & Zisook, 2017).

Description: Grief is a natural emotional response to loss; grieving, bereavement, or mourning is the process of dealing with loss. Complicated grief refers to the inability to cope with the loss or becoming "stuck." When the patient is unable to work through his or her grief it intensifies and physical or psychological illness may occur; this type of grief is more intense and prolonged (Shear, Reynolds, Simon, & Zisook, 2017).

Etiology: Loss or change perceived as loss; for older adults, the loss of a spouse or life partner is the most common and significant event (Nseir & Larkey, 2013). Other losses include loss of a parent, child, sibling, or close friend.

Occurrence: Approximately 50% of women over the age of 65 years are widows; 13% of men over the age of 65 years are widowers.

Age: May occur at any age.

Gender: Affects more women than men.

Ethnicity: Not significant.

Contributing Factors: Length of illness, amount of suffering, relationship quality, survivor guilt, financial burden of illness, caregiver burden, personality attributes of deceased and survivor, and religious and cultural expectations all may be related to the variety of grief responses (Keyes et al., 2014; Stahl & Schulz, 2013). Factors present in the anticipatory grieving stage include attachment style, relationship to the departed, prior loss, previous psychiatric problems, or traumatic exposure. Being unprepared for the death, violent death, marital intimacy, and caregiving quality were features associated with the actual death that foretold complicated grieving (Keyes et al., 2014; Shah et al., 2013; Stahl & Schulz, 2014). Widows and widowers with recent disabilities, few friends, and poor relationships with their children were more apt to require counseling (Keyes et al., 2014; Supiano & Luptak, 2014; Powell, 2016). Current studies have questioned the validity of the "grief work" theory and demonstrate that there is great variability in individual responses to grief, as well as differences in the timeline for bereavement (Shear, Reynolds, Simon, & Zizook, 2017; Moayedoddin & Markowitz, 2015; Powell, 2016; Tol, Barbui, & van Ommeren, 2013).

Signs and Symptoms: Grief is characterized by feelings of depression with associated symptoms of poor appetite and weight loss or compulsive eating and weight gain, sleep disturbance, tearfulness, lack of interest, withdrawal and isolation, emptiness, indecisiveness, and guilt feelings. Some grieving individuals may respond by making dramatic changes in a short time to avoid dealing with feelings. Somatic symptoms are common in the early months of bereavement and may require referrals outside of the primary care arena (Powell, 2016; Shear, Reynolds, Simon, & Zisook, 2017; Simon, 2013).

Diagnostic Tests: None.

Differential Diagnosis:

- Social phobia
- MDD
- Adjustment reaction
- Persistent complex bereavement disorder

Treatment: Provide emotional support, allowing the older adult to express feelings. Contact close family members who were not present at the bedside of the deceased immediately after the event and offer condolences, answer any questions, and allow them to view the body if feasible. A card or letter from professionals who cared for the patient is also recommended. Reaching out to the surviving spouse in the early bereavement period is also helpful. Sleep disruptions are common in the first 2 weeks; education on sleep hygiene and assessing for pre-existing conditions such as obstructive sleep apnea may be necessary (Shear et al., 2017).

Reminiscence is helpful to many. Encourage patients to return to their normal routine as soon as possible. Daily physical exercise can help patients cope with the depression that accompanies grief. Referral to a bereavement support group may help some individuals, but not all (Shear et al., 2017); for most older adults with a support system of family, friends, clergy, etc., counseling is unnecessary. In cases of complicated grief, psychotherapy and/or antidepressant medication may be indicated (Keyes et al., 2014; Simon, 2013; Shear et al., 2017). Those with pre-existing chronic medical conditions, particularly cardiac, will need to be monitored for adverse events; studies have demonstrated the occurrence of acute coronary syndrome (ACS/MI) after the death of a significant person (Moon et al., 2011; Pini et al., 2015; Shah et al., 2013).

Follow-Up: Guidelines from palliative care and other sources speak of a need for bereavement visits or follow-up. This needs to be individualized depending on the situation (ISCI, 2013; Gippsland Region Palliative Care Consortium, 2016; Moayedoddin & Markowitz, 2015; National Consensus Project for Quality Palliative Care, 2013).

Sequelae: In the first 3 months after the death of a spouse for adults over 65 years, the mortality rate increases 48% in men and 22% in women. This increase in mortality is across the board and is unrelated to prior health status and socioeconomic level (Moon, Kondo, Glymour, & Subramanian, 2011; Shah et al., 2013). Practitioners must be alert to older adults who do not improve in 3 months after the loss. Additionally, a major depressive syndrome that occurs for 2 or more weeks early in the course of bereavement should be taken seriously and managed by the nurse practitioner or by referral to mental health/psychiatry (Shear et al., 2017; Simon, 2013).

Prevention/Prophylaxis: The goal is to encourage and support the patient in the normal grieving process and prevent dysfunctional grieving. Activation of social and spiritual support

networks, discussion of anticipated loss by participants, and involvement in group activities or volunteerism may be helpful.

Referral: Older adults experiencing abnormal grieving (complicated grief and depression) may benefit from a mental health referral (Keyes et al., 2014). Referral to support groups, such as Widow-to-Widow, may be helpful in the grieving process, however, it is recommended that the survivor self-initiates contact with a resource if they feel the need (Shear et al., 2017).

Education: Make patients and their support networks aware of the variability of normal grieving; alert them to signs of dysfunctional grieving and resources for help. A helpful resource for survivors and families is www.nlm.nih.gov/medlineplus/bereavement.html. This site has information on usual and complicated grief written for the general public.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
At the time of admission to a hospice or palliative care program, an initial, developmentally appropriate professional assessment is completed to identify patients and families at risk for complicated grief, bereavement, or comorbid complications, particularly among older adults.	C	Gippsland Region Palliative Care Consortium, 2016 National Consensus Project for Quality Palliative Care, 2013
Following the death of the patient, it is essential to allow the patient's loved ones to perform any customs or rituals that are important to them, within the policy guidelines of the facility. Clinicians should be available to answer questions and offer support.	C	ICSI, 2013 National Consensus Project for Quality Palliative Care, 2013
Bereavement services and follow-up are made available to the family for at least 12 months or for as long as is needed after the death of the patient.	C	Gippsland Region Palliative Care Consortium, 2016 National Consensus Project for Quality Palliative Care, 2013
A post-death bereavement plan is activated. An interdisciplinary team member is assigned to the family in the post-death period to help with religious practices, funeral arrangements, and burial planning.	C	Gippsland Region Palliative Care Consortium, 2016 National Consensus Project for Quality Palliative Care, 2013
For bereaved individuals who do not have mental disorders, we suggest not prescribing psychotropic medications such as benzodiazepines.	B	Shear, Reynolds, Simon, & Zisook, 2017 Tol, Barbui, & van Ommeren, 2013
Acute grief typically does **not** require treatment. For bereaved individuals who do not have mental disorders, we suggest not routinely administering grief counseling or other psychotherapies. However, grief counseling can be helpful for bereaved individuals who request it and may also be helpful when it is coupled with other efforts that are focused upon new activities.	B	Shear, Reynolds, Simon, & Zisook, 2017 Tol, Barbui, & van Ommeren, 2013

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INSOMNIA

Signal Symptoms: Patient report of not sleeping, excessive daytime sleepiness, loud snoring (sleep apnea), restless legs, difficulty falling asleep and staying asleep, irritability, difficulty concentrating, sleep that is not refreshing and restful (Shochat & Ancoli-Israel, 2017; Sondheimer, 2016).

Description: Insomnia is difficulty falling asleep or staying asleep despite the desire to do so and regardless of adequate conditions to promote sleep; poor-quality sleep and difficulty with daytime functioning are also part of insomnia. This sleep–wake disorder is not attributed to another physical disease, substance use/abuse, or mental disorder (DSM-5).

Etiology: Insomnia can have several etiologies, including medical, behavioral, circadian, or psychiatric. Sleeping states and sleep schedules change with age. Sleep efficiency (time actually sleeping versus time in bed) is less than 80% in older adults, and time to fall asleep is extended. The most important age-related changes in sleep include:

- Decreased continuity of sleep with an increase in the number of arousals
- Tendency for the major period of sleep and rapid eye movement (REM) sleep to occur earlier in the night
- A decrease in the deepest parts of non–rapid eye movement (NREM) sleep
- Increased napping during the day
- Tendency to spend more time in bed

Chronic insomnia lasts more than 1 month and results from age-related changes in sleep and chronic stressors (Bonnet & Arand, 2017a).

Occurrence: Approximately 50% of people more than 65 years of age experience and regularly complain of poor sleep quality. Poor health confounds this problem, with older adults with respiratory problems reporting 40% greater difficulty with insomnia, and those with psychiatric problems being two and one-half times more likely to experience insomnia (Shochat & Ancoli-Israel, 2017).

Age: Insomnia can occur at any age; however, older adults have greater difficulty falling asleep and staying asleep.

Gender: Women who are widowed, separated, or divorced have more insomnia up to age 85 years; men have more insomnia in the over-85-years age group (Bonnet & Arand, 2017a).

Ethnicity: Not significant.

Contributing Factors: Factors that may contribute to insomnia include:

- Restless legs syndrome
- Periodic limb movement disorder
- Sleep apnea
- Dementia
- Depression
- Anxiety
- Drugs, including caffeine, alcohol, nicotine, antipsychotics, beta blockers, stimulant decongestants, sedative-hypnotics, sympathomimetic bronchodilators, diuretics, carbidopa-levodopa, H_2 blockers, and centrally acting α-agonist antihypertensives, stimulant antidepressants, corticosteroids, calcium channel blockers, anticholinergics, SSRIs, and SNRIs (Shochat & Ancoli-Israel, 2017).

Many chronic medical conditions can cause insomnia, including musculoskeletal, cardiac, respiratory, GI, renal, endocrine, and neurological (Shochat & Ancoli-Israel, 2017). Insomnia is associated with an increase in hypertension and atherosclerosis (Rangaraj & Knutson, 2015; Shochat & Ancoli-Israel, 2017), as well as increase in mortality (Li et al., 2014).

Signs and Symptoms: A complete history should reveal a full description of the problems. Patients may complain about difficulty falling asleep and staying asleep, frequent awakenings, early morning awakening and inability to return to sleep, daytime fatigue with unwanted naps, irritability, or difficulty concentrating. Additionally, an older adult may spend 10 to 12 hours in bed at night trying to sleep. A pertinent physical examination should evaluate the systems associated with any medical conditions listed here. Falling may be a sign of insomnia. A mental status examination is useful in detecting cognitive or psychiatric disease (Bonnet & Arand, 2017a).

Diagnostic Tests: None, unless indicated by history and physical examination. Insomnia is a clinical diagnosis, verified by a sleep history or sleep log. The sleep history should include an assessment of daytime sleepiness, fatigue, or sleep disturbance; the sleep environment; and the duration of symptoms. Additionally, information on frequency and duration of awakenings, sleep times, nap times, and lengths is important (Bonnet & Arand, 2017a; Shochat & Ancoli-Israel, 2017).

Differential Diagnosis:

- Anxiety
- Inadequate sleep hygiene
- Medical problems
- Medication-related sleep disorder
- Depression
- Alcohol-related sleep disorder or primary sleep disorder

Treatment: For transient insomnia, patients should avoid caffeine for 12 hours before bedtime and discontinue alcohol and unnecessary sleep-interrupting drugs. OTC melatonin or prescription ramelteon can be tried. If ineffective, initiate a short-acting sedative-hypnotic, such as zolpidem (Ambien) or zaleplon (Sonata), at lowest dosage before desired bedtime for 1 week or less. Suggest spacing dosing to every other day to avoid side effects. If a benzodiazepine is used, temazepam (Restoril) is relatively short-acting. If this is ineffective, reevaluate the diagnosis and restructure the treatment modalities.

For chronic insomnia, the treatment is more complex. A complete medical and psychiatric history is indicated, including any family history of sleep problems. A validated self-administered instrument, such as the Epworth Sleepiness Scale (www.stanford.edu/~dement/epworth.html) or Stanford Sleepiness Scale (www.stanford.edu/~dement/sss.html), can be used to focus on specific aspects of the problem. The University of Pittsburgh Sleep Center also has

several instruments that require permission for use (http://www.sleep.pitt.edu/research/instruments.html). The patient should keep a sleep diary and bring it to the next office visit. If the patient has a bed partner, this person should be interviewed as well. If sleep apnea is suspected, refer for polysomnography. Review sleep hygiene tips. CBT, even that delivered by Internet (Seyffert et al., 2016), focused on reducing time in bed, correcting false beliefs and expectations regarding sleep, and decreasing exposure to stimuli that deter sleep has been shown to be superior and long-lasting compared with short-term sedative-hypnotic use (Trauer et al., 2015). Combined, sleep hygiene instruction and cognitive behavioral therapy are more effective than either modality alone or usual treatment (Trauer et al., 2015). Music therapy with patient-selected music is also effective (Wang et al., 2016). Medications should be evaluated in light of ability to interfere with sleep and modifications should be made where possible. Gradual increase from sedentary to moderate aerobic exercise combined with sleep hygiene is also an effective intervention (Varrasse, Li, & Gooneratne, 2015). Mindfulness meditation is a successful treatment in motivated patients, and results have been sustained over a long period (Black et al., 2015; Gong et al., 2016). Treat underlying or coexisting disorders.

Pharmacological therapy includes temazepam (Restoril) for sleep onset insomnia, eszopiclone (Lunesta) for sleep onset and sleep maintenance, zolpidem CR and zolpidem (AmbienCR and Ambien) and zolpidem sublingual (Intermezzo) for sleep maintenance, and zaleplon (Sonata) and ramelteon (Rozerem) for sleep onset insomnia. The FDA (2013) has issued a drug safety communication requiring lower doses of zolpidem products to avoid risk of next-day impairment. Intermezzo was not included in this warning. All of these drugs are listed as potentially inappropriate medications (PIMs) on the Beers list (2015) to be avoided in older adults. Other drugs used for insomnia include suvorexant (Belsomra) and low-dose doxepin (Silenor). Suvorexant was not available when the most recent Beers list (2015) published; doxepin more than 6 mg is listed as a PIM, but Silenor is 6 mg; doxepin is also available as a liquid for oral use in low doses (Edmonds & Swanoski, 2017). Melatonin may be effective in low doses. Diphenhydramine (Benadryl) is on the Beers list (2015) as a PIM and should be avoided; patients frequently use this as Tylenol PM. The use of trazodone is common but off-label. Benzodiazepines are not recommended and are also on the list of PIMs.

Follow-Up: Patients should return in 2 weeks. Examine the patient's sleep diary and evaluate the effectiveness of the treatment. If indicated, reevaluate the diagnosis and restructure the treatment. Pharmacological therapy should be considered a last choice and every effort should be made to wean the patient from these or avoid long-term use.

Sequelae: Includes reduced quality of life, depression, increased risk for falls/injury, and potential for drug dependence or drug interactions resulting from use of OTC sleep aids.

Prevention/Prophylaxis: Sleep hygiene suggestions may include:

- Establish a regular bedtime and wake-up time.
- Set aside a time each evening for relaxation and thinking.
- Avoid caffeine, alcohol, and nicotine because they all interrupt sleep.
- Minimize awake time in bed, reserving bed for sleep and sexual activity.
- Create an optimal sleep environment.
- Establish regular eating habits because hunger can interrupt sleep.
- Avoid napping.
- Exercise daily to the extent possible, but avoid exercise just before bedtime.
- Maximize daytime exposure to bright light.

Referral: If therapy brings no improvement and other underlying causes have been eliminated, refer the patient to a sleep laboratory for evaluation.

Education: Explain age-related sleep changes to the patient and his or her family. Teach the patient to follow the guidelines listed under Prevention/prophylaxis. Educate the patient to avoid OTC antihistamines because they can have dangerous side effects, including dry mouth and a "hangover" effect. Teach the patient to avoid alcohol use with prescribed sleep medication to prevent accidental overdose. Provider education is also needed, because many providers do not recognize insomnia in the older adult as a problem.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
CBT is an effective treatment for insomnia in older adults.	A	Sontheimer, 2016
Sedative-hypnotics (zolpidem, ramelteon, zaleplon, and eszopiclone) are effective but also have adverse effects such as depression and rebound insomnia.	A	Sontheimer, 2016
Most patients do not require diagnostic testing; the practitioner should consider performing a polysomnography if obstructive sleep apnea or periodic limb movement disorder is suspected.	C	Sontheimer, 2016

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

PRESCRIPTION DRUG MISUSE (HAZARDOUS OR RISKY USERS)

Signal Symptoms: Variable depending on abused substance. Opioids and benzodiazepines are among the most frequent categories. Pinpoint pupils, constipation, confusion, memory loss, depression, behavioral changes, drowsiness, difficulty breathing, other behavioral symptoms, including personality changes.

Description: Older people use and misuse a variety of substances. For instance, a vast majority of the elderly take prescribed medications (sometimes not as prescribed) for physical and psychiatric ailments; they buy OTC medications (which they may not always take according to instructions); they smoke cigarettes, drink alcohol, and use illicit drugs, such as marijuana, and there is a growing number of elderly using heroin and cocaine (Grover et al., 2008). Another problematic use of prescribed medications involves "borrowing" a medication from a friend or relative instead of seeking medical advice. The obvious problem is that all medications and drugs of abuse may also produce toxicity, cause withdrawal symptoms, cause physical and/or psychological harm after short- or long-term use, and they may (and generally do) interact with each other. Ultimately, there are further adverse effects, which usually cause the person to take even more medication to neutralize these nasty side effects. Other prescription drug abuse or misuse includes the deliberate use of prescribed drugs for nonmedical purposes, accidental misuse due to inaccurate dosing, or accepting drugs prescribed for another person for one's own use (Sehgal, Manchikanti, & Smith, 2012). Willful or accidental mixing of prescription drugs with other psychoactive substances, such as alcohol, is another form of prescription drug abuse, as is the deliberate diversion of prescription drugs for personal gain (Manchikanti, 2006; Office of National Drug Control Policy, n.d.).

Etiology: Multiple factors influence the misuse of psychoactive prescription drugs and OTC medications. The aging process, poor physical health, and female gender are the most consistently documented correlates. Other factors include genetic predisposition to addiction, unrealistic expectations for pain relief, coexisting mental health problem (often undiagnosed) or substance use problem, undiagnosed cognitive impairment, and socioeconomic hardship, to name a few. Recent evidence shows that family and friends are the primary source for opiate diversion, and most prescriptions are obtained from a single source, as opposed to shopping around to multiple providers. Among older women, use of psychoactive drugs is correlated with middle- and late-life divorce, widowhood, less education, poorer health and chronic somatic problems, higher stress, and lower income (SAMHSA, 2014).

Occurrence: The United States consumes 80% of the world's opioids and 99% of the hydrocodone supply globally, despite comprising only 4.6% of the population worldwide (Manchikanti & Singh, 2008). Over the last two decades, opioid analgesic prescribing has increased four-fold, adding to the dramatic rise in opioid-analgesic related misuse among older people (Hedegaard et al., 2015). A cross-sectional community-based study of individuals age 57 to 85 years found that a significant number of men and women used at least five prescription medications concurrently (Qato et al., 2008). Benzodiazepines are the most commonly prescribed psychiatric medication among all adults, and despite contraindications for use with older adults, they are widely prescribed and are disproportionately prescribed to older adults (Achildi et al., 2013).

Age: Multiple studies show a higher rate of prescription drug abuse in the 50- to 64-year age group (Blazer & Wu, 2009; Gossop & Moos, 2008; Wu & Blazer, 2011). Unfortunately, health-care professionals often make assumptions about their older patients on the basis of age rather than functional status, which is believed to be caused by the limited training clinicians receive in the care and management of this population (Borkowski, 2016).

Gender: A national survey found that overall more men have prescription drug abuse problems, but women who abuse prescription drugs are less likely to receive treatment (Back, Payne, Simpson, & Brady, 2010). In two studies specific to older adults (Culberson & Ziska, 2008; Simoni-Wastila & Yang, 2006), female gender was cited as a risk factor for prescription drug abuse.

Ethnicity: All races are affected.

Contributing Factors: Factors vary considerably by substance and the specific clinical presentation of a patient; for example, age, medical comorbidities, current medications, and health history. Lack of perception by the patient that prescription drug abuse is problematic (Wu & Blazer, 2011), unmet need for chronic pain relief (Sjøgren, Ekholm, Peuckmann, & Grønbaek, 2009), coexisting mental health problems or substance use disorder (Lofwall, Schuster, & Strain, 2008; Sehgal et al., 2012), genetic predisposition to addiction, and availability of abused substances from trusted sources such as family and friends (Hernandez & Nelson, 2010) are seen frequently. Another common factor is that they may see multiple providers, each of whom may prescribe the patient medications that interact with each other and/or with alcohol or other substances. See Table 16-11 for a summary of risk factors related to drug misuse in the older adult.

Signs and Symptoms: Vary depending on class of drug. Can often be mistaken for a medical, cognitive, or psychosocial problem in an older adult. Family members are quick to conclude that behavioral changes are the result of processes such as aging, empty-nest syndrome, loss of spouse and/or close friends, etc. Drug-seeking behavior may be the initial sign and can include reporting lost prescriptions or unmanageable pain despite being given an appropriate dose. Other objective signs include pinpoint pupils, constipation, confusion or disorientation in familiar surroundings, memory loss, slurred speech, ataxia, depression, behavioral changes, drowsiness, difficulty breathing, irritability, and personality changes.

Diagnostic Tests: As a general rule, older adults are less likely to be screened for substance use, and this population is known to have difficulty identifying their own risky behaviors around misuse of substances (Duru et al., 2010). Urine drug screening at random intervals to detect the presence

TABLE 16-11 Risk Factors for Substance Misuse in the Older Adult

Physical risk factors	Male sex (for alcohol); female sex (for prescription drugs) Caucasian Chronic pain from physical disabilities or reduced mobility Poor health status Polypharmacy
Psychiatric risk factors	Use of avoidance as a coping mechanism Previous/current substance use disorder Previous/current psychiatric illness
Social risk factors	High socioeconomic status Presence of bereavement Unexpected or forced retirement Living alone

Adapted from Kuerbis et al. (2014). Substance abuse among older adults. *Journal of Clinical Geriatric Medicine, 30,* 629–654. Retrieved from http://dx.doi.org/10.1016/j.cger.2014.04.008

of prescribed drug and any other illicit drugs should be considered. There are several excellent screening tools that can be utilized with this population. Examples include the Michigan Alcohol Screening Test – Geriatric Version (MAST-G); Alcohol Use Disorders Identification Test (AUDIT); and Alcohol, Smoking, and Substance Involvement Screening Test (ASSIST), which was developed by the World Health Organization, just to name a few. These tools have been validated for use in the geriatric population. SBIRT (Screening, Brief Intervention, and Referral to Treatment) is another approach that can be used in alcohol and substance use disorders (Babor et al., 2007).

Differential Diagnosis:

- Dementia
- Delirium
- Personality disorder
- Inadequate pain management
- Neurocognitive problem
- Depression
- Alcohol use disorder

Treatment: Treatment depends on the extent of the misuse and the particular class of drug abused, other medical and/or psychiatric comorbidities, and presentation. The possibility of multiple substances being misused simultaneously must be assessed. The two most commonly abused classes are addressed here.

Acute Opioid Intoxication: Pharmacodynamically, the opioids, as a class of drugs, act on the respiratory centers and, even in therapeutic doses in the elderly, they can cause respiratory depression. In an intoxicating state, respiratory depression is expected and treatment requires an acute care setting. All opioids should be discontinued and the patient should be thoroughly examined for the presence of any transdermal patches. Because multiple substance use is common, obtain a comprehensive toxicology screen, monitor the patient, and provide supportive care, including management of any medical comorbidities. Administer naloxone, an opioid antagonist, with dosage determined by patient's respiratory status. Management of opioid use disorder usually occurs on an outpatient basis. Methadone maintenance should be considered in the absence of impulse control and poor supports; however, the patient must go on a daily basis to a methadone-dispensing clinic, which may cause undue distress to the elderly patient. Buprenorphine (Suboxone) may be more appropriate because the patient can get a prescription and be monitored monthly. Buprenorphine cannot be administered until the patient is showing objective signs of opioid withdrawal, which looks like a bad case of the flu. Naltrexone injection (Vivitrol) is a monthly injectable that is an opioid antagonist that requires a monthly visit to the prescriber. Unfortunately, this medication is a thick, viscous substance of which 4 ml must be administered deep into the gluteal muscle and many older patients do not have the muscle mass to tolerate monthly injections. Naltrexone cannot be administered until at least 10 days after the last use of the full-agonist opioid drug (Maldonado, 2010).

Acute Benzodiazepine Intoxication: This is more likely to occur in the presence of concurrent alcohol use or abuse because alcohol has a synergistic effect on the benzodiazepines. Offer supportive care and close monitoring in the acute care setting, as abrupt discontinuation can cause seizures. Substituting a longer-acting benzodiazepine and tapering the dose over several days is indicated. Also, obtain a comprehensive toxicology screen to check for other substances. Treat medical comorbidities. Flumazenil, a benzodiazepine antagonist, may be used in severe cases, but the patient must have an IV access point and be cardiac monitored because Flumazenil itself can cause seizures (Maldonado, 2010). In addition to pharmacological management, psychotherapy or counseling is indicated. A meta-analysis of current treatment approaches to discontinuation of benzodiazepines found that gradual dose reduction (GDR) combined with a psychological modality was more effective than either of those alone. There was insufficient evidence to support other pharmacological substitutes (Parr, Kavanagh, Cahill, Mitchell, & McD. Young, 2008). The SBIRT approach is also recommended (Babor et al., 2007).

Follow-Up: Patients with prescription drug misuse may require individualized follow-up in an outpatient treatment program and should also be monitored by their primary care provider.

Sequelae: Deaths from prescription drug abuse and misuse have risen steadily. Alcohol and drug interactions are common in older adults, leading to significant morbidity and mortality (Pringle, Ahern, Heller, Gold, & Brown, 2005). Legal and criminal prosecution can result from drug diversion.

Prevention/Prophylaxis: Educate providers, pharmacists, patients, families, and the public about the use and misuse of prescription drugs. Make an assessment of pain and abuse potential of patients when prescribing and allow for the ongoing monitoring of patients' medication use, using patient contracts and random drug testing as indicated. The FDA and the Department of Justice Drug Enforcement Administration (DEA) have collaborated to develop a Risk Evaluation and Mitigation Strategy (REMS) for extended-release and long-acting opioid analgesics. This REMS program will require education of providers and pharmacists, education of patients and families by the provider, and continued

education at the site of prescription dispensing by the pharmacist (www.ER-LA-opioidREMS.com).

Prescription drug monitoring programs are also in place in most states, and the programs allow prescribers and dispensers to access records of controlled substance prescriptions filled for a particular patient in live time. These safety measures are a beginning step, but the responsibility rests with the provider and the patient for ongoing monitoring and reevaluation of a continued need for the prescribed drug. To date, there is insufficient evidence on the efficacy of opioids for noncancer chronic pain (Manchikanti, Fellows, Ailinani, & Pampati, 2010), but the preponderance of prescriptions are not for that purpose. Providers should regularly monitor for clinical guideline updates on prescription drug abuse and safe prescribing of commonly abused drugs (Manchikanti et al., 2012a, 2012b).

Prevention prophylaxis with older patients requires planning for the potential psychosocial and physical health factors that place them at risk for relapsing. Approaches such as CBT, group and family therapies, self-help groups, and pharmacological adjuncts, as discussed previously, should be considered (Barrick & Connors, 2002).

Referral: Older adults who regularly abuse or misuse prescription drugs should be referred to an addictions specialist or treatment program for co-management and follow-up. Refer addicted adults to community-based support groups, such as Narcotics Anonymous, for mentoring.

Education: Educate providers, patients, families, and the public (see Prevention/prophylaxis). For patients, families, and the public, education on the dangers of using or sharing drugs that are not prescribed for the individual is essential.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Primary attention in opioid overdose is airway and ventilation management.	C	Kraft, 2011
The use of naloxone is a key diagnostic and therapeutic intervention; naloxone reversal should be titrated to ventilation, rather than to the level of arousal, to prevent withdrawal symptomatology.	C	Kraft, 2011
Intoxication with longer-acting opioids predicts recurrence of respiratory depression after naloxone reversal.	B	Kraft, 2011
There is current evidence supporting that case management can enhance linkage with other services. However, evidence that case management reduces drug use or produces other beneficial outcomes is not conclusive.	A	Hesse, Vanderplasschen, Rapp, Broekaert, & Fridell, 2007
Psychosocial treatments offered in addition to pharmacological detoxification treatments are effective in terms of completion of treatment, use of opiate, participants abstinent at follow-up, and clinical attendance.	A	Amato, Minozzi, Davoli, & Vecchi, 2011
Currently, there is not enough evidence to conclude that psychosocial treatments alone are adequate to treat people with opiate abuse and dependence.	A	Mayet, Farrell, Ferri, Amato, & Davoli, 2004
SBIRT yields short-term improvements in individuals' health; long-term effects on population health have not yet been demonstrated, but simulation models suggest that the benefits could be substantial.	B	Babor et al., 2007

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

Mrs. C. is a 69-year-old married woman who presents to your practice with a chief complaint of fatigue. She tells you that she is tired all the time, despite sleeping through the night. She is concerned she may be coming down with something. You ask her about other symptoms, and she confesses that she is under a lot of stress. Her husband has terminal cancer and is also depressed. He is still functional but has refused all interventions. Mrs. C. relates that she quit her job to take care of him, but it is "dragging her down" because he is so depressed. She has four children, three of whom are close by, but she feels like she is bearing the burden herself, and the children do not realize what is happening. She tells you tearfully, "I want to have a life, too, before it is all over."

Review of her chart shows recent CBC and chemistry profiles that are normal, normal TSH, and normal EKG. There is a family history of alcohol abuse (her father) and suicide (a sibling).

1. What additional subjective data are you seeking?
2. What additional objective data will you be assessing for?
3. What national guidelines are appropriate to consider?
4. What tests will you order?
5. Are there any screening tools that you want to use?
6. What are the differential diagnoses that you are considering?
7. What is your plan of care?
8. Are there any *Healthy People 2020* objectives that you should consider?
9. What additional patient teaching may be needed?
10. Will you be looking for a consultation?

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unit IV

Complex Illness

CHAPTER 17

Polypharmacy

Lori Martin-Plank

Polypharmacy has many definitions, including prescribing many drugs, prescribing five or more drugs, or prescribing potentially inappropriate medications (PIMs), among others. This chapter will examine polypharmacy trends and consequences in older adults, including underprescribing, overprescribing, and misprescribing; adverse drug reactions (ADRs) as an outcome of polypharmacy; specific areas of concern in prescribing for older adults; pharmacodynamics and pharmacokinetics that may affect safe prescribing in older adults; recommendations for safe prescribing in older adults, and recommendations for deprescribing.

A recent study (Charlesworth et al., 2015) examined trends in polypharmacy from 1988 to 2010 using National Health and Nutrition Examination Survey (NHANES) data, and found that median use of prescribed medications doubled in that time period from two to four medications in adults age 65 years and older. The percentage of older adults using five or more prescribed medications increased from 12.8% to 39.0%, which was attributed primarily to the use of antidepressant and cardioprotective drugs (Charlesworth et al., 2015). As more sophisticated, evidence-based guidelines have evolved, providers focusing on following the guidelines strictly have added medications, often without considering patient goals and preferences, but striving to satisfy the expectations of insurers and pay-for-performance mandates (Anderson et al., 2014; Maher, Hanlon, & Hajjar, 2014). Utilization of the American Geriatrics Society (AGS) Principles for the Care of Patients with Multimorbidities can assist the provider in determining priorities of care, including pharmacotherapeutics (AGS, 2012).

Polypharmacy and the potential for an ADR remain major concerns in geriatric health care (Kalisch Ellett et al., 2014). Although older adults represent 14.9% of the population, they are the biggest consumers of medications. One-third of community-dwelling elderly take more than 5 prescribed medications, and nearly 20% take more than 10 prescribed medications. Forty-two percent of older adults take more than one over-the-counter (OTC) drug, and 49% take more than one nutritional supplement (Slone Epidemiology Center at Boston University, 2006). Polypharmacy is a primary predictor for an ADR, which is any undesired or unwanted consequence that occurs as a result of taking medications (Ferrah, Lovell, & Ibrahim, 2017; Hyttinen, Jyrkkä, & Valtonen, 2016; Kalisch Ellett et al., 2014; Maher, Hanson, & Hajjar, 2014).

The magnitude of the problem is reflected in some current statistics. ADRs account for approximately 10% of all emergency department visits and up to 17% of all hospital admissions (Hanlon, Sloane, Pieper, & Schmader, 2011; Moss et al., 2016; Nothelle et al., 2017; Rochon, Gill, & Gurwitz, 2017). The most significant factor contributing to the risk of an ADR in older individuals is the number of drugs taken (Scott et al., 2015). Factors contributing to the increased ADR risk in older adults include not only polypharmacy, but also pharmacokinetics and pharmacodynamics that are altered with aging, adherence problems, inappropriate health-care provider prescribing, and self-prescribing. In general, the therapeutic window narrows with age, so the potential for benefiting the patient measured against the risk of doing harm becomes more significant for the prescribing professional.

PHARMACOKINETIC/ PHARMACODYNAMIC CHANGES

Certain changes occurring with the aging process alter the dynamic processes that drugs undergo to produce therapeutic effects. These changes involve the processes of pharmacokinetics (what the body does to the drug) and pharmacodynamics (what the drug does to the body).

Absorption

Drug absorption is generally thought to have a less significant impact on pharmacokinetics than drug distribution, metabolism, or excretion (Katzung, 2015). Gastric acidity declines with age because of decreased intestinal blood flow and fewer absorbing cells in the gastrointestinal (GI) tract. These changes appear to be offset, however, by the longer contact time that occurs as transit time slows (in aging, this slowing is more functional than physiological). Another factor that affects drug absorption is the presence of food and other drugs in the stomach at the same time. Antacids and

iron, for example, inhibit the absorption of tetracycline; antacids can significantly decrease the bioavailability of digoxin. Anticholinergic medications cause a slowing of colonic motility and can result in greater absorption rates. Metabolic diseases, such as thyroid disease or diabetes, can cause an increase or decrease in transit time and, therefore, can cause either increased or decreased drug absorption (Rochon, Gill, & Gurwitz, 2018).

Another consideration is whether an oral medication actually passes through the esophagus before it is absorbed. Many older adults take medications without adequate fluid, increasing the potential for esophageal damage from the dissolution of drugs in the esophagus. Esophageal erosions have been noted with caustic drugs such as alendronate potassium and tetracycline. Drug–drug, drug–disease, and drug–food interactions are likely to influence drug absorption (Sera & McPherson, 2012).

Distribution

Drug distribution is affected by aging, particularly in individuals of smaller body size, those who have decreased body water, and those with higher body fat. Drugs distributed in water (e.g., alcohol and lithium) have a higher concentration in elderly persons, thereby exerting a more profound effect. Drugs distributed in fat (e.g., most psychoactive drugs) have a wider distribution and a less intense effect, but a more prolonged action, particularly in those individuals with more adipose tissue. Medications with a higher protein-binding rate (e.g., phenytoin, warfarin, salicylates) have a greater potential to cause an ADR in those with less lean body mass. Because these individuals have fewer receptor sites and less albumin for binding, plasma concentration is greater, and more free drug is available for tissue distribution, pharmacological activity, and elimination. Protein-bound drugs in particular may reach toxic levels if the patient is not monitored closely. Distribution influences are marked in the patient who is malnourished and dehydrated (Katzung, 2015; Sera & McPherson, 2012).

Drug distribution also relies on the bioavailability of the drug. The amount of the drug that reaches the systemic circulation may be increased or decreased, depending on certain influencing factors: 1) route of administration is important to consider, because inhalants and drugs given intravenously and topically are usually more readily available than drugs administered intramuscularly, subcutaneously, orally, or rectally; 2) solubility of the drug is influential, because aqueous solutions are available more quickly than oily ones; and 3) general circulation to the site of drug administration has an impact, because blood flow increases with massage or heat or in the presence of an occlusive dressing and decreases during shock or during the administration of vasoconstrictors.

Metabolism

Biotransformation occurs in all body tissues, but primarily in the liver, where enzymatic activity (cytochrome P [CYP] system) alters and detoxifies the drug and prepares it for excretion. With advancing age, the ability of the liver to metabolize drugs does not decline similarly for all pharmacological agents. Although liver size and blood flow do decline with age, routine liver function test results are typically normal when no disease exists. Decreased liver size and blood flow can result in decreased first-pass metabolism; drug activity for some medications is prolonged, because drugs are metabolized and eliminated more slowly (Katzung, 2015; Sera & McPherson, 2012).

Being familiar with the age-related pharmacokinetics of drugs is of the utmost importance when determining the initial and maintenance dosages. When prescribing for an older adult patient, it is critical to understand whether the drug inhibits or induces the CYP enzymes. Other factors influence biotransformation also. Men have faster and more efficient biotransformation, presumably because of serum testosterone. Conditions of increased or decreased liver perfusion alter the overall level of the drug that is absorbed and how it is metabolized.

Elimination

The most profound pharmacokinetic change is reduced elimination of drugs. Most drugs are excreted in the urine via the renal system, although some are excreted in the feces via the biliary system. Water-soluble drugs are excreted directly by the kidneys, and fat-soluble drugs are converted to water-soluble drugs by the liver first. Changes in kidney function begin in the fourth decade of life and continue to decline with each subsequent decade (Katzung, 2015; Sera & McPherson, 2012). Therefore, by age 70 years an individual might reasonably have a 40% to 50% decrease in renal function, even in the absence of disease. These kidney function changes may prolong the half-lives of drugs. This is particularly important for drugs that are excreted unchanged in the urine and for drug categories known to be particularly nephrotoxic in older persons (e.g., radiocontrast materials, aminoglycosides, angiotensin-converting enzyme inhibitors, and NSAIDs).

Pharmacodynamics

It is important to know the pharmacodynamic influences of the drug (what the drug does to the body). A drug's pharmacodynamics describe the effect at the site of action and the time and intensity of the drug effect. A good example is that the older adult tends to exhibit enhanced responses to drugs affecting the central nervous system (CNS) (e.g., benzodiazepines), and this is attributed to greater tissue sensitivity caused by aging. Older adults often experience more sedation from CNS drugs than younger persons at the same concentration. Older adults given opiates are more likely to experience ataxia, and those taking haloperidol are more likely to experience extrapyramidal symptoms.

Some studies demonstrate that older individuals may have increased tissue sensitivity for oral anticoagulants (Rochon et al., 2018). In certain instances, patients may exhibit a decreased, rather than an exaggerated, response at this tissue level; this appears to be the case with beta blockers, in which an increased dosage may be required to have a desired effect. Drug responsiveness may vary depending on the patient's activity and stress levels and on the environment. These factors have not yet been adequately studied. There is a general trend of assuming greater pharmacodynamic sensitivity in the elderly; however, this sensitivity is not universal, and age-related changes need to be investigated agent by agent until further research yields greater understanding of the aging process (Rochon et al., 2018).

Inappropriate Prescribing by Health-Care Professionals

Most older adults have at least two comorbidities and see several health-care providers, in addition to a primary care provider. Prescribing by several providers is a risk factor for polypharmacy and ADRs, and lack of timely communication, or any communication between specialists and primary care providers, further complicates the problem (Pretorius et al., 2013; Shade, Berger, & Chaperon, 2014). The expectation that the older adult patient will be the communicator is flawed and unrealistic. HIPPA permissions for release of information can allow timely faxing of medication changes by a specialist to the primary care provider to facilitate communication. All prescribing should be vetted through the primary care provider (Scott et al., 2015).

Underprescribing, overprescribing, and prescribing of PIMs are all challenges faced by health-care providers dealing with older adults. Utilization of the AGS Stepwise Approach to the Care of Patients with Multimorbidities (2012) can be helpful in guiding the prescriber. Consideration of patient goals and preferences of care, risk–benefit ratio of the drug, quality of life, cost–benefit ratio, narrow therapeutic index of the drug, and pill burden (number of pills taken per day) are all factors to weigh before prescribing. Consultation with a pharmacist, especially a geriatric consulting pharmacist, can be beneficial (Coggins, 2017; Stevens et al., 2017).

Clinical inertia can occur when a patient is started on a low dose of a drug for safety but is never advanced to a therapeutic dose or is maintained on a drug that has no therapeutic benefit while another drug is added. Both of these situations contribute to polypharmacy and the potential for ADRs. Another circumstance that is a red flag for ADRs is when a patient presents with an acute condition and is started on a drug to treat that condition without reviewing other drugs that he or she is on for chronic health conditions—antibiotics are an example of this. Using a macrolide such as clarithromycin in a patient who is on digoxin will increase the potential for digoxin toxicity.

Whenever a patient presents with a new chief complaint, a change in cognitive or functional status, or a newly recognized health problem, evaluate for an ADR as the cause before prescribing (Pretorius et al., 2013; Scott et al., 2015). Failure to do this can result in prescribing another drug for an unrecognized ADR, causing a prescribing cascade where unnecessary drugs are added, increasing the chance for further ADRs. An example includes the prescribing of a high-dose NSAID for an acute musculoskeletal problem and putting the patient on a proton pump inhibitor (PPI) to avoid GI effects of the NSAID. When the patient returns, he has an elevated blood pressure and is diagnosed with hypertension. He is placed on an antihypertensive, has orthostasis, and falls, breaking a hip. While hospitalized, he is continued on the PPI and develops *Clostridium difficile,* which requires further treatment with antibiotics (Coggins, 2017).

Several categories of drugs have been listed as problematic in older adults, including antipsychotics, benzodiazepines, opioids, PPIs, and anticholinergic drugs (anticholinergic burden scale can be found at http://indydiscoverynetwork.org/resources/idnd-developed-clinical-tools/) (AGS, 2015). Prescribers should consult current resources for precautions before prescribing.

TOOLS TO ASSIST PROVIDERS TO AVOID PIMS AND POLYPHARMACY

There are over 35 assessment tools currently available to assist providers with PIMs (Kaufmann, Tremp, Hersberger, & Lampert, 2014). Two of the most well-known and frequently used are STOPP/START and the BEERS criteria.

STOPP/START (Lavan, Gallagher, & O'Mahony, 2016; O'Mahony et al., 2015) is a tool designed to detect prescribing of PIMs. This screening tool of older persons' potentially inappropriate prescriptions (STOPP) consists of 65 clinically important criteria that relate to PIMs and can contribute to hospitalization for ADRs, including a brief explanation of each. This is used in conjunction with the screening tool to alert doctors to right treatment (START), which contains 22 criteria, supported by evidence, to remind prescribers to consider certain drugs that are appropriate for specific conditions but may be omitted.

The Beers criteria (AGS, 2015) consists of a list of PIMs to be avoided in older adults; they are listed by drug category and also by diagnosis. An addition in the 2015 edition is the listing of some drugs that require renal dosing or should be avoided in patients with renal problems, as well as a listing of harmful drug–drug interactions. There is an additional article (Hanlon, Semla, & Schmader, 2015) that details potential alternative drugs to those listed in the Beers criteria, that can be safely used in older adults. The Beers criteria is not appropriate for patients on palliative care or hospice (AGS, 2015).

The Beers criteria (AGS, 2015) and the STOPP/START (O'Mahony et al., 2015) are both valuable tools for prescribers, but they are not meant to be mandates for prescribing. Clinical judgment, knowledge of the patient who has the disease, clinician and patient shared goals, risk–benefit considerations, and quality of life all factor into decision making for individual patients. There may be times when drugs on these lists need to be prescribed; however, this should be done with full awareness of the aforementioned factors and the need for monitoring.

PREVENTING POLYPHARMACY, ADDRESSING POLYPHARMACY

Polypharmacy can be prevented by the use of parsimonious prescribing; that is, prescribing only necessary drugs after consultation with the patient and/or family, consideration of goals of treatment and expected outcomes, and regular reevaluation of the plan of treatment. Initial and ongoing assessment should include asking about drug allergies, use of herbals, dietary supplements and OTC preparations, sharing others' medications, alcohol use, and use of any recreational drugs, including marijuana and drugs for sexual enhancement. Consider any nonpharmacological interventions that may be effective before prescribing. Designating and training office staff to do the initial medication review can save time, but still requires review by the provider. "Brown bag" reviews where the patient brings in all current medications are helpful, but time consuming.

The patient should be seen as soon as possible after discharge in the case of a hospitalization, as transitions in care such as hospitalization can increase the potential for confusion by the patient, resulting in taking too many drugs or the wrong drugs. Also caution the patient against hoarding old drugs, which may be expired and have lost their efficacy. Avoid prescribing new drugs when an older generic drug has the same effect and is more cost-effective. Patients will not fill the prescription if they cannot afford the drug, but may be reluctant to disclose that to their provider.

Educate the patient about his or her role in taking the medications as prescribed, what each drug is for, not to self-discontinue any drug without speaking with the provider, and to report any unusual reactions or side effects. In the case of a cognitively impaired patient, work with family or nursing home staff on these issues. Consider ordering home health services, if needed, to educate patient and family, and monitor response to any new drug therapy. Print out a list of current drugs for the patient at each office visit and tell him or her to destroy old lists. Some areas have a program, such as Vial of Life, where current medication lists are stored in or on the refrigerator for easy access in case of an emergency. Use the annual Medicare wellness visit (AMW) to reevaluate the patient's condition and goals, and adjust drug therapy accordingly. Before making an adjustment in a drug that appears to not be effective, check with the patient to see if he or she is taking it, and if yes, taking it as ordered. For certain classes of drugs, including those for pain, behavior, or cognition, introduce the drug as a trial and set a date and time to reevaluate the efficacy before continuing. If you "inherit" a patient in whom polypharmacy is evident, address this and work with the patient on a plan to make adjustments, realizing that this will occur over time with deprescribing (Machado-Alba, Gaviria-Mendoza, Machado-Duque, & Chica, 2017).

Deprescribing is the reevaluation of the efficacy and necessity of a drug for a specific patient, done in conjunction with the patient and family, with the plan to discontinue or reduce the dosage of the medication in a safe manner (Page, Potter, Clifford, & Etherton-Beer, 2016; Scott et al., 2015; Stevens et al., 2017). Providers may consult with a pharmacist as part of the plan (Coggins, 2017). This concept originated in Europe (Reeve et al., 2015) and has expanded to Canada (see http://deprescribing.org; Frank & Weir, 2014) and the United States after several systematic reviews highlighted the dangers of polypharmacy in older adults. Although the concept has been adopted, there are currently few guidelines on the "how to" of deprescribing and also some questions/challenges within the prescribing community about what constitutes polypharmacy and "rational polypharmacy" (polypharmacy that is justified). Scott and colleagues (2015) suggest five steps to safe deprescribing:

- Identify all drugs that the patient is presently taking and the rationale for each one (creating a drug/disease grid is helpful for this step).
- Consider the overall risk of drug-induced harm to the patient in designing the extent and aggressiveness of the deprescribing effort.
- Review each drug individually in terms of present/future potential good versus present/future harm (risk/benefit ratio) to the patient.
- Prioritize deprescribing in terms of drugs with least benefit/harm ratio and least chance of problems from withdrawal or chance of disease exacerbation when withdrawn.
- Develop a plan for discontinuation or dose reductions. Monitor the patient closely during this period for adverse events or improvement in outcomes (Scott et al., 2015). Include the patient and family in the deprescribing process.

The Canadian group (http://deprescribing.org) has developed algorithms for deprescribing of PPIs, benzodiazepines, and antipsychotics. As evidence accumulates for safe deprescribing, this has the potential to decrease polypharmacy and ADRs (Coggins, 2017; Frank & Weir, 2014).

CHAPTER 18

Chronic Illness and the APRN

M. Catherine Wollman

Evidence-based management of common chronic diseases is an essential skill for advanced practice registered nurses (APRNs) in order to provide quality care for patients. It is also essential to understand and manage complex challenges of older adults with chronic disease. This chapter reviews the effect of chronic illness on older patients, their families, providers, and the overall health-care system. Specific topics include the demographics of chronic disease, lifestyle risk factors, the significance of multiple chronic conditions (MCCs), new models of care that reorganize health care in an effort to improve outcomes for complex patients with chronic disease, reimbursement mechanisms that support chronic disease, and the role of the APRN in chronic illness.

DEFINITIONS OF CHRONIC DISEASE AND CHRONIC ILLNESS

The literature does not support a single uniform definition for chronic disease, but the concept of chronicity includes the knowledge that patients experience persistent and recurring health problems. Definitions of chronic disease also reflect the pathophysiology of disease and, more importantly, consider the meaning of chronic illness and the experience of the patient, family, and provider as they struggle to cope with the range of mildly complicated to extreme challenges. Curtin and Lubkin (1995, pp. 6–7) defined *chronic illness* as "the irreversible presence, accumulation, or latency of disease states or impairments that involve the total human environment for supportive-care and self-care, maintenance of function, and prevention of further disability." Anderson and Horvath (2004) defined *chronic conditions* as conditions lasting 1 year or more and requiring ongoing medical attention and/or the limiting of activities of daily living (ADLs). *Chronic disease* or a *chronic condition* is also defined as any condition that requires ongoing adjustments by the affected person and requires periodic interaction with the health-care system (Improving Chronic Illness Care, 2016).

DEMOGRAPHICS OF CHRONIC ILLNESS

Increased life expectancy and health-care advances are the main reasons for the overwhelming increase in numbers of patients with chronic illness. The population of individuals age 65 years and older is now 46.2 million or 14.5% of the population. That number is projected to increase to 21.7% of the population in 2040 (Administration on Aging [AOA], Administration for Community Living [ACL], U.S. Department for Health and Human Services [USDHHS], 2016).

As of 2012, about half of adults, or 117 million people, had one or more chronic health conditions. The most common chronic diseases in the population age 65 years and older include hypertension (60%), dyslipidemia (41%), arthritis (28%), cardiac disease (25%), and eye disease (23%) (Robert Wood Johnson Foundation [RWJF], 2010).

Seven of every 10 deaths in the United States are caused by chronic conditions. Heart disease, cancer, chronic obstructive pulmonary disease (COPD), and stroke are the leading causes of death, with heart disease as the number one cause of death among both men and women. Other major diseases that contribute to the 70% death rate from chronic disease include diabetes and Alzheimer's disease (Centers for Disease Control and Prevention [CDC], 2016). Heart disease and cancer together accounted for nearly 48% of all deaths (CDC, 2016).

Notable Changes in Incidence and Prevalence of Chronic Disease

In a major change to prior trends, Stage 3 and 4 chronic kidney disease (CKD) in the United States has not increased in prevalence over the past 10 years, likely related to better treatment of type 2 diabetes (Murphy et al., 2016). From 1999 to 2009, cardiovascular disease (CVD) deaths declined by 32.7%, but the burden of disease remains high, with CVD still accounting for 32.3% of all deaths, or one of every three deaths in the United States (Go et al., 2013). The prevalence of diagnosed diabetes in the United States increased by 382% from 1988 to 2014. In the United States, 29.1 million or

9.3% of the population has diabetes. Over 8 million individuals have undiagnosed type 2 diabetes (American Diabetes Association, 2015).

Multiple studies indicate that the risk of Alzheimer's disease and other dementias in the United States and other developed Western countries may have declined in the past 25 years. That change is hypothesized to be due to increasing levels of education and improved control of cardiovascular risk factors. The total number of people in the United States with Alzheimer's disease and other dementias is still, however, expected to continue to increase dramatically because of the population's aging population. By 2025, the number of people age 65 years and older with Alzheimer's disease is estimated to reach 7.1 million—almost a 40% increase from the 5.2 million affected in 2016 (Alzheimer's Association, 2016, p. 25).

The requirements of care for specific chronic diseases, such as heart failure (HF) or HIV, have changed dramatically in the last two decades. HIV has evolved from an untreatable and usually fatal disease to a chronic illness with a life expectancy of several decades (Losina & Freedberg, 2011). Eighty percent of HF patients are over age 65 years, with the incidence of HF doubling in each decade of life, with an eventual increase to 20% in patients over 80 years of age (Go et al., 2013). As a result, most patients with HF are very old, with increasing debility and very complex health-care regimens.

Health Risk Behaviors

According to the CDC, four health risk behaviors are responsible for the majority of chronic disease and death, including lack of physical activity, poor nutrition, tobacco use, and excessive use of alcohol. The prevalence of cigarette smoking is still close to 20% of adults (USDHHS, 2014). The American Heart Association tells us that there is a sharp increase in CVD risk with even low levels of exposure to cigarette smoke, including secondhand smoke. On average, male smokers die 13.2 years earlier than male nonsmokers, and female smokers die 14.5 years earlier than female nonsmokers (Go et al., 2013).

Binge or heavy drinking can lead to high risk sexual behavior, unintentional injuries (e.g., motor vehicle crashes), falls, violence, and suicide. Excessive alcohol consumption can also lead to development of high blood pressure, liver disease, some cancers, dementia, and alcohol dependence (Chowdhury et al., 2016).

The age-adjusted prevalence of obesity in 2013 to 2014 was 35.0% among men and 40.4% among women (Flegel, Kruszon-Moran, Carroll, Fryar & Ogden, 2016). Individuals who are obese can suffer serious health problems, face discrimination, and have a reduced life expectancy. The greater the body mass index (BMI) (and waist circumference), the greater the risk of CVD, including hypertension, coronary artery disease, and stroke. In addition, obese individuals have an increased incidence of type 2 diabetes, sleep apnea, osteoarthritis, gallbladder disease, respiratory problems, some types of cancer, and depression (Jenson et al., 2014). It is known that 85.2% of people with type 2 diabetes are overweight or obese (American Diabetes Association, 2015). There is strong evidence that modest weight loss (5% to 15%) can greatly reduce the risk of these conditions. An expert panel (Jensen et al., 2014) also recommends intensive management of CVD risk factors (hypertension, dyslipidemia, prediabetes, diabetes, and sleep apnea) in addition to weight loss measures.

Prevention

The goals of national health promotion and disease prevention are to prevent or delay disease, decrease premature mortality, and improve health-related quality of life. Modification of behavioral risk factors and using preventive services such as screenings and immunizations can substantially reduce morbidity and mortality. The Behavioral Risk Factor Surveillance System (BRFSS), part of the USDHHS, monitors health risk behaviors, chronic diseases or conditions, and the use of preventive services to help identify high-risk groups with the greatest need for intervention (Chowdury et al., 2016).

The 2010 passage of the Patient Protection and Affordable Care Act (PPACA), or the ACA, increased the opportunities for disease prevention. Ongoing efforts are aimed at redesigning the health system to support overall population health. One successful prevention initiative removed out-of-pocket costs and increased access to evidence-based clinical preventive services recommended by the U.S. Preventive Services Task Force (USPSTF). Results have demonstrated increased use of those preventive services, including tobacco counseling, cervical cancer screenings, and colorectal cancer screenings (Koh, Rajkumar, & McDonough, 2016).

In the United States, diabetes affects more than 25% of people age 65 years or older and its prevalence is projected to increase approximately two-fold for all U.S. adults (ages 18 to 79 years) by 2050, if current trends continue (Centers for Medicare and Medicaid Services [CMS], 2016). Another successful intervention is the CMS Diabetes Prevention Program (DPP), an evidence-based intervention targeted to individuals with prediabetes, with the primary goal of weight loss and behavior change. The DPP uses lifestyle coaches to deliver evidence-based counseling interventions to Medicare beneficiaries with prediabetes. The results of the program demonstrated that participants lost at least a modest amount of weight through dietary changes and increased physical activity, which reduced their chances of developing diabetes. The DPP will be greatly expanded in 2018 to further prevent type 2 diabetes with appropriate lifestyle changes (CMS, 2016).

MULTIPLE CHRONIC CONDITIONS

The U.S. health system was created to manage and pay for acute illness with a focus on one disease or health-related problem at a time. As the number of chronic conditions in an individual increases, there is a parallel risk for poor outcomes, often related to the unintended consequences of a single disease approach. Those outcomes include poor functional status, unnecessary hospitalization, adverse drug events, conflicting medical advice from multiple providers, and even death. There is an increasing awareness that the health system and health-care providers require a new paradigm of care that recognizes patients with MCCs. To explore the public health problem of MCCs, the USDHHS created the Multiple Chronic Conditions Interagency Workgroup. One of the main goals of the framework is to generate research

to bridge knowledge gaps about individuals with MCCs (USDHHS, 2010).

Data from the 2012 National Health Interview Survey (NHIS) were used to generate estimates of MCCs for U.S. adults. Approximately half (117 million) of U.S. adults have at least 1 of the 10 most common chronic conditions, which include hypertension, coronary heart disease, stroke, diabetes, cancer, arthritis, hepatitis, CKD, asthma, or COPD. In addition, one in four adults has more than one chronic condition or MCC, and among the more than 20 million Medicare beneficiaries, 37% have five or more chronic conditions (CMS, 2012). Overall, the 2012 estimates show that 25.5% of U.S. adults have MCCs. The numbers from the NHIS do not include mental health issues or institutionalized older adults, which would substantially increase the estimates (Ward, Schiller, & Goodman, 2014).

MCCs can contribute to frailty and disability, and older persons who are frail or disabled typically have MCCs. Functional limitations can often complicate access to health care, interfere with self-management, necessitate reliance on caregivers, and produce high levels of spending. Older adults with MCCs often require long-term services and supports (LTSS). LTSS include the broad range of paid and unpaid medical and personal care assistance that people may need when they cannot complete self-care tasks as a result of aging, chronic illness, or disability (Reaves & Musumeci, 2015).

With the potential for continued growth of older adults requiring LTSS, it is especially important to increase knowledge about the high-risk factors related to MCCs that can increase costs or the risk of hospitalizations. One recent study determined that among older adults receiving LTSS, the MCC group with cardiopulmonary chronic conditions had the highest use of hospitalization (Van Cleave, Egleston, Abbott, Hirschman, & Naylor, 2016).

Other high-risk individuals with MCC include those with serious mental illness or behavioral health problems. Co-occurrence of mental and medical disorders is a disabling combination and occurs in approximately 30% of individuals with MCCs. Physical illness is often accompanied by mental health problems, and mental disorders are associated with an increased risk of a wide range of chronic physical conditions (Barnett et al., 2012; Walter & Druss, 2016). The number of older adults who require management of addictions is projected to double from 1.7 to 2.8 million in 2000 to 4.4 to 5.6 million in 2020 (Han, Gfroerer, Colliver, & Penne, 2009).

Self-Care Management

Patients coping with MCCs are especially vulnerable to a sense of burden with their treatment regimen because they are required to engage in a complex array of self-care activities on a daily basis. Treatment burden is defined as "the workload of health care and its impact on patient functioning and well-being" (Eton et al., 2013, p. 7). That "work" might include medication management, self-monitoring of blood pressure or blood sugars, visits to providers, laboratory tests, and lifestyle changes. Treatment burden is not always addressed or appreciated, and can contribute to reduced adherence and quality of life (Goodman, Posner, Huang, Parekh, & Koh, 2013).

Many chronic conditions, such as heart disease, diabetes, and arthritis share common challenges associated with their self-care management, including dealing with symptoms; monitoring physical signs; managing complex medication regimens; maintaining proper levels of nutrition, diet, and exercise; adjusting to the psychological and social demands; and coping with decreased energy and function, while trying to engage in effective interactions with health-care providers (Grady & Gough, 2014). It is critical for health-care providers to be aware of ways in which their patients are managing, or failing to manage, the chronic conditions in their everyday lives.

New models of care are essential to provide tools to improve self-management and quality support systems. The National Institute of Nursing Research (NINR) at the National Institutes of Health (NIH) has promoted self-management science as one of its core areas of research to improve and manage symptoms of acute and chronic illness (NINR, 2016). Two of the most successful and well-known self-management programs are the Arthritis Self-Management Program (ASMP), developed by Margaret Grey, and the Chronic Disease Self-Management Program (CDSMP), developed by Kate Lorig. Both programs were supported by NINR and NIH funding (Grady & Gough, 2014).

The CDSMP assists with five core skills of self-management that include problem solving, decision making, resource utilization, the patient–provider partnership, and taking action (Lorig & Holman, 2003). It is a weekly two-and-a-half hour workshop given for 6 weeks in community settings. Workshops are facilitated by trained leaders who may be health-care professionals or patients with chronic diseases. Subjects covered include "1) techniques to deal with problems such as frustration, fatigue, pain and isolation, 2) appropriate exercise for maintaining and improving strength, flexibility, and endurance, 3) appropriate use of medications, 4) communicating effectively with family, friends, and health professionals, 5) nutrition, 6) decision making, and, 7) how to evaluate new treatments" (Stanford Medicine, 2016).

The ACA provided $32.5 million to support the implementation of the CDSMP in 45 states. Workshops supported adults with MCCs of different ages and from different cultural backgrounds. The initial 100,000 participants demonstrated that the program was successful and that large numbers of participants can be reached when evidence-based programs are offered in convenient community settings (Ory et al., 2013).

Caregivers

As older adults become more functionally limited, caregivers become central members of the care team and play a major role in supporting the daily self-care management and maintaining the health and well-being of the care recipient. The estimated prevalence of caring for an adult is 16.6% of the population, or 39.8 million people in the United States. The majority of caregivers are female (60%), with an average age of 49 years. About 49% provide care for a parent or parent-in-law, and 10% provide care for a spouse. The typical care recipient is female (65%), with an average age of 69 years. Nearly half of caregivers provide assistance to someone 75 years old or older (National Alliance for Caregiving, American Association of Retired Persons [NAC & AARP], 2015).

Caregivers' tasks can include assistance with ADLs and instrumental ADLs (IADLs), medication and treatment management, monitoring signs and symptoms, or navigating health-care appointments and health-care systems. The

demands of caregiving can create additional stress in the caregiver's life, commonly referred to as caregiver burden. Caregiver burden is associated with anxiety, depression, insomnia, poor self-care management, poor self-rated health, and greater mortality (NAC & AARP, 2015). Caregiver burden also directly affects decisions to place care recipients in long-term care facilities. About 40% of caregivers report high burden, 18% report moderate burden, and 41% report a relatively low burden (NAC & AARP, 2015).

Caregiving has traditionally been seen as a family obligation, but little attention has been paid to the type of formal support, guidance, and skills needed to perform caregiving. There are a few sporadic programs that provide educational support for caregivers. The Red Cross offers caregiver education with topics related to home safety, providing personal care, legal and financial issues, nutrition, and positioning and physical skills (www.programsforelderly.com, n.d.). Because prevalence of burden is not specific to stage of illness and is relatively stable over time, health-care teams should assess caregiver burden and refer burdened caregivers to supportive resources early in the course of chronic illness (Sautter et al., 2014). Ongoing caregiver education should be provided by multiple members of the health-care team and should include caregiver involvement in decision making and guidance prior to any potential health-care crisis (Leff, Kao, & Ritchie, 2015).

Health Literacy

The ability to use and correctly interpret health information is important for anyone with one or more chronic diseases. Health literacy involves using reading, writing, verbal, and numerical skills in order to obtain, process, and understand basic health information to make appropriate decisions (Brega et al., 2015, p. 1). A seminal national survey showed that 88% of U.S. adults do not have the health literacy skills needed to manage all the demands of the current health-care system and 36% have limited health literacy (Kutner, Greenberg, Jin, & Paulsen, 2006).

Research indicates that low health literacy contributes to a number of health-related problems, including a poor understanding about health conditions and services, the inability to provide self-care, difficulty understanding medication instructions, poor medication adherence, and low use of preventive services (USDHHS, n.d.). Individuals with limited health literacy also experience more negative outcomes. Due to the limited knowledge of their health problems, they make more medication errors, use more inpatient and emergency department care, and have worse overall health status and higher health-care costs (Brega et al., 2015).

Research additionally shows that clinicians have trouble identifying patients with limited health literacy, so it is suggested that universal precautions are used regardless of the complexity of any information. Universal precautions defined in the AHRQ Health Literacy Universal Precautions Toolkit include: simplifying communication with and confirming comprehension for all patients, making the office environment and health-care system easier to navigate, and supporting any and all patient efforts to improve their health. Studies have shown that 40% to 80% of the medical information patients are told during office visits is forgotten immediately, and nearly half of the information retained is incorrect. The teach-back method is a way of checking understanding by asking patients to state in their own words what they need to know or do about their health. Teach-back is a way to confirm that the provider has explained things in a manner that the patient understands (Brega et al., 2015, p. 18).

ECONOMIC BURDEN OF CHRONIC DISEASE

Eighty-six percent of all health care spending in 2010 was for people with one or more chronic medical conditions (CDC, 2016). The latest economic reports of chronic disease indicate that the cost burden associated with five of the most common chronic diseases was $28 billion greater than had been predicted. This unexpected increase relates to the prevalence of chronic, preventable conditions among people in the United States, largely due to the effects of obesity. In all diseases other than CVD, the numbers of patients with chronic disease rose above projections, and actual treatment costs and productivity losses exceeded estimates. Total overall treatment costs and lost productivity in the United States presently amounts to $1.3 trillion (Chatterjee, Kubendran, King, & Devol, 2014).

A few of the more common chronic diseases have substantial cost estimates. The estimated total cost of diagnosed diabetes in 2012 was $245 billion, a 41% increase from the previous estimate of $174 billion (American Diabetes Association, 2013). Total payments in 2016 for individuals with Alzheimer's disease and other dementias are estimated at $236 billion, with Medicare and Medicaid payments of $160 billion, or 68%, and out-of-pocket spending expected to be $46 billion, or 19% of total payments (Alzheimer's Association, 2016, p. 45).

The total costs of heart disease and stroke in 2010 were estimated to be $315.4 billion with $193.4 billion, respectively, for direct medical costs, excluding costs of nursing home care (CDC, 2016).

According to the CMS, Medicare spending for fee for service (FFS) beneficiaries in 2010 was over 300 billion dollars. The one-third of beneficiaries with 0 or 1 chronic conditions accounted for only 7% of Medicare spending, while the 14% with six or more chronic conditions accounted for 46% of Medicare spending (2012, pp. 22, 24). Medicare data also clearly reflect the most costly chronic conditions. Per capita costs for patients with stroke, CKD, and asthma or COPD were seven times higher than the average spending for Medicare FFS beneficiaries (CMS, p. 28).

Additional costs of chronic disease relate to the multiple acute care hospitalizations experienced by the seriously chronically ill population. In a 2004 classic study, almost one-fifth of Medicare patients had unplanned rehospitalizations within 30 days of release, with a cost of $17.4 billion (Jencks, Williams, & Coleman, 2009). Cost and complexity of care are greater for individuals with multiple chronic diseases, who account for 75% of overall health-care spending (Thorpe, Ogden, & Galactionova, 2010).

HF is the highest cause of hospital readmission. The increased prevalence and life expectancy of HF is expected to contribute to an increase in annual direct medical costs from the present estimate of $24.7 billion to approximately $77.7 billion over the next two decades. Indirect costs due to

reduced productivity are expected to increase from $9.7 to $17.4 billion (Konstam, 2012).

The medical care costs of obesity in the United States continue to be overwhelming. In 2008, those costs were estimated to be about $147 billion dollars (Finkelstein, Trogdon, Cohen, & Dietz, 2009). More recent estimates suggest that the annual health-care costs of obesity-related illnesses are a staggering $190.2 billion, or more than 20% of annual medical spending in the United States (Cawley & Meyerhoefer, 2012).

In a study of over 2,600 patients with dementia, individuals with cognitive impairment had greater Medicare and Medicaid costs, including nursing home use; greater hospital and home health use; more transitions per person per year of follow-up; and more mean total transitions than those without cognitive impairment (Callahan et al., 2012).

The costs to address MCCs are significant, with 66% of total health-care spending directed toward care for the approximately 27% of people in the United States with MCCs. Individuals with MCCs also have substantial personal economic challenges related to higher costs for prescription drugs and total out-of-pocket costs (USDHHS, 2010).

MINORITIES AND CHRONIC DISEASE

The older population in the United States continues to be more racially and ethnically diverse as the minority population grows. Racial and ethnic minority populations have increased from 6.3 million in 2003 (17.5% of older adults) to 9.5 million in 2013 (21.2% of older adults) and are projected to increase to 21.1 million in 2030 (28.5% of older adults). African Americans (9%) and Hispanics (8%) comprise the largest groups of minority populations (AOA, DHHS, 2015).

Chronic conditions can be more common or severe for minority groups. Non-Hispanic African Americans are 40% more likely to have high blood pressure and with poorer control than are non-Hispanic Caucasians. Diagnosed diabetes is 77% higher among non-Hispanic African Americans, 66% higher among Hispanics, and 18% higher among Asians than among non-Hispanic Caucasians. Native Americans and Alaska Natives are 60% more likely to be obese than non-Hispanic Caucasians. As a result, life expectancy for non-Hispanic African Americans is 75.1 years, compared to 78.9 years for non-Hispanic Caucasians (CDC, 2015).

There are noticeable racial/ethnic differences in the prevalence of many chronic conditions and treatment for those illnesses. Although CKD has not increased in overall prevalence, there remains a continued increase of CKD in non-Hispanic African Americans (Murphy et al., 2016). The literature shows consistent disparities among African Americans and Hispanics compared to non-Hispanic Caucasians in the prevalence and incidence of Alzheimer's disease, mortality, participation in clinical trials, use of medications and other interventions, use of long-term services and supports, expenditures, and quality of care (Lines, Sherif, & Wiener, 2014). African Americans have the highest death rate and shortest survival of any racial/ethnic group in the United States for most cancers. The cancer death rate among African American men is 27% higher compared to non-Hispanic Caucasian men and 11% higher for African American women (American Cancer Society, 2016).

FUNCTION AND FRAILTY

Aging is a highly variable process that leaves some older adults with strength and vitality, and others with increased vulnerability to illness, injury, and disease. That reduced physiological capacity contributes to the development of geriatric syndromes such as falls, delirium, and frailty (Leff, Kao, & Ritchie, 2015). Consideration and documentation of geriatric syndromes and overall function is as important in the care of older adults with chronic disease as a medical diagnosis. ADLs and IADLs are used to measure functional status. ADLs are measures of functions that people perform on a daily basis and include feeding, bathing, dressing, grooming, toileting, ambulating, and transferring (Katz et al., 1963). IADLs are indicators of more complex functions required on a regular basis, including use of the telephone, shopping, housekeeping, laundry, transportation, management of medications, and handling of finances (Lawton & Brody, 1969).

Older adults are often more concerned about the impact of their illness on daily function than they are about the specific diagnosis. Despite the importance of function to alert providers to changes in health or prognosis and to determine appropriate interventions, mobility and function are not routinely measured or monitored in clinical practice. It is important to systematically include mobility and functional assessment in clinical documentation.

Frailty is not synonymous with comorbidity or disability, but comorbidity is a risk factor for frailty, and disability is an outcome of frailty. Frailty has been defined as a clinical syndrome when three or more of the following criteria are present: unintentional weight loss of 10 pounds in the past year, self-reported exhaustion, weakness based on grip strength, slow walking speed, and low physical activity (Fried et al., 2001). Awareness of frailty will also help to develop accurate assessments, help patients and families with anticipatory planning, and make appropriate decisions about interventions and treatment.

Geriatricians and other aging experts additionally defined physical frailty as "a medical syndrome with multiple causes and contributors that is characterized by diminished strength, endurance, and reduced physiologic function that increases an individual's vulnerability for developing increased dependency and/or death" (Morley et al., 2013, p. 392). That group of experts recommended that everyone older than 70 years should be checked for frailty using a screening questionnaire known as FRAIL that was originally developed by Dr. Fried in 2001 (Morley et al., 2013).

The FRAIL tool asks five screening questions about fatigue, walking, number of chronic diseases, and weight loss of more than 5% in 6 months to identify those at high risk of being frail. Those who answer yes to at least three of the screening questions are likely to be frail and should be further evaluated (American Geriatrics Society, 2012). The likelihood of poor outcomes related to frailty is very high. There is a higher frequency of primary care visits, home care and nursing home services, and a higher likelihood of rehospitalization (Kahlon et al., 2015). In many cases, once frailty is identified it can be treated with aerobic and resistance exercise, protein and

caloric supplements, vitamin D, and reduced polypharmacy (Morley et al., 2013).

EVIDENCE-BASED PRACTICE AND CHRONIC DISEASE

Available evidence to manage chronic disease is found within clinical practice guidelines (CPGs). Guidelines, however, are almost exclusively focused on one specific disease. It is essential to evaluate those guidelines to determine if they are appropriate for frail, older adults with MCCs. There is wide diversity in the older adult population in life expectancy, functional status, and individual priorities and preferences related to health care. Those characteristics limit the use of specific disease-oriented models of care. Complex comorbid conditions and geriatric syndromes are common, and usual signs and symptoms, such as fatigue, may be due to one or several chronic diseases, problems, or syndromes, and require different or multiple interventions.

When CPGs have been evaluated for content that considers unique issues of complex older adults with multiple comorbidities, it has been determined that only a few CPGs address those issues. The neglected content includes identifying patient and caregiver concerns, setting clinical priorities based on life expectancy, managing expectations around prognosis, maintaining communication, and considering patient, provider, or system barriers to implementation (Mutasingwa, Ge, & Upshur, 2011).

Older adults with MCCs have poorer outcomes when treated according to disease-specific guidelines. Those negative outcomes may be due to decreased benefit from therapy directed at any one chronic disease, the presence of polypharmacy, or the increased likelihood that the high-risk older adult will have a poor response to any specific intervention (Fried, Tinetti, & Iannone, 2011). To improve decision making, clinicians require unique data about the frail older adults and alternative approaches that include patient and family values; communication and coordination with specialty providers, and other members of the team; and a restructured reimbursement system (Fried et al., 2001).

Experts continue to study the complex relationship between patients with MCCs and available evidence. In an effort to guide understanding about CPGs and MCCs, researchers developed a patient-centered and context-sensitive model of care called minimally disruptive medicine (MDM). MDM acknowledges the limitations of disease-centered treatment recommendations for patients with MCC. MDM suggests caveats to clinicians caring for complex patients. For example, following an evidence-based recommendation for a frail older adult with MCCs, especially when the recommendation is not strong, may do more harm than good (Leppin & Montori, 2015). In other national attention to this issue, principles are being developed to strengthen the content of guidelines to support patients with MCCs and to increase focus on patient-centered care (Goodman et al., 2014).

CHRONIC CARE MODEL OF QUALITY IMPROVEMENT

A majority of people in the United States with major chronic illnesses are not receiving appropriate or effective management. The consequences of inadequate care are poor disease control, exacerbations, and complications that far exceed those seen with appropriate care. The Institute of Medicine (IOM, 2001) has described this difference between usual and appropriate care as the "quality chasm."

The Chronic Care Model (CCM) (see Figure 18-1) was developed through funds from the Robert Wood Johnson Foundation to improve care for people who suffer from chronic illness. The CCM recognizes the significance of increasing chronic disease and MCCs. The CCM is an

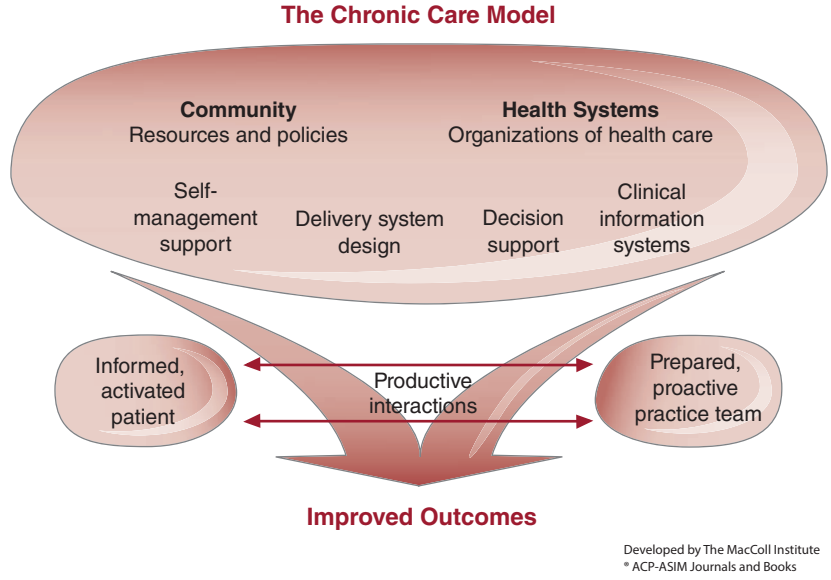

FIGURE 18-1. The chronic care model (CCM). *(Wagner, E. H. [1998]. Chronic disease management: What will it take to improve care for chronic illness?* Effective Clinical Practice, 1, *2–4, Figure 1. With permission from the American College of Physicians.)*

evidence-based policy response to reduce the problems inherent in a health-care system that is currently not designed to meet the needs of chronically ill patients and their families. The CCM is also used to direct quality improvement and overall systems change for patients with chronic illnesses (Improving Chronic Illness Care, 2016).

The CCM approaches care of the chronically ill away from the reactive acute care visit to a planned patient-centered encounter. The CCM involves six elements considered essential to effective chronic care management and depicted in the model. Those elements are community resources and policies, organization of health-care systems, self-management support, delivery system design, decision support, and clinical information systems.

LEGISLATION AND CHRONIC DISEASE

Accountable Care Organizations

The ACA included multiple payment and delivery system reforms with incentives for health systems to better manage the care of patients with chronic disease. Beginning in 2012, the law authorized Medicare to contract with accountable care organizations (ACOs) in a Medicare Shared Savings Program. ACOs provide financial incentives to improve the coordination and quality of care for Medicare beneficiaries while reducing costs. ACOs are groups of doctors, hospitals, and other health-care providers who come together voluntarily to give coordinated high-quality care to their Medicare patients. The primary goal of coordinated care is to ensure that patients with chronic disease receive quality care, while avoiding unnecessary duplication of services and preventing medical errors. When the ACO succeeds in delivering high-quality care and reduced spending, it will share in the savings it achieves (CMS, 2015).

A key element of ACOs will relate to the effective discharge of patients from acute care settings and initiatives to reduce admissions. All members of the ACO are incentivized to coordinate care of patients across the continuum in order to achieve savings. In 2015, almost 400 ACOs participated in the MSSP. They have demonstrated positive trends in savings, but with major variation in performance (Health Affairs Blog, September 9, 2016). Organizations require time to create major transformation and determine new paths to affect positive changes in health-care delivery.

Hospital Readmission Reduction Program

A major provision of the ACA is the Hospital Readmissions Reduction Program (HRRP) that financially penalizes hospitals for excess readmissions within 30 days of discharge. The HRRP motivates hospitals to conduct interventions that reduce the likelihood of readmission, and has drastically changed how hospitals manage complex patients at high risk for readmission (CMS, 2016).

Before 2012, hospitals had little direct financial incentive to reduce readmission. The HRRP requires CMS to reduce payments to hospitals who experience excess readmissions. The HRRP initially targeted select diagnoses of HF, pneumonia, and acute myocardial infarction. In 2015, the additional diagnoses of COPD, total hip arthroplasty, and total knee arthroplasty were added (CMS, 2016). As of 2015, penalties for excess hospitalizations increased to 3% of base payments by Medicare, and readmissions for these diagnoses are also treated as an indicator of the quality of a hospital's care and are publicly reported and available to consumers.

High rates of preventable hospitalizations and emergency department visits are still frequent poor outcomes. In a recent Medicare Payment Advisory Commission (MedPAC) report to Congress, all-cause 30-day rehospitalization rates for Medicare beneficiaries decreased from an average of 19% to below 18%, at least in part due to major changes in incentives (MedPAC, 2015). However, among Medicare beneficiaries with four or more MCCs, the 30-day rehospitalization rate was still 36% (Lochner, Goodman, Posner, & Parekh, 2013).

Many hospital readmissions are not preventable, but evidence and model programs are available to assist hospitals with system-wide changes that will decrease readmission rates. The HRRP has helped create collaborative relationship within hospitals, between medical institutions, and with surrounding communities that focus on improving the overall patient experience through hospitalization and post-discharge.

Patient-Centered Medical Home

The 2011 National Committee for Quality Assurance (NCQA) has defined standards for the patient-centered medical home (PCMH), which reinforce the central components of primary care. Those core components include an ongoing relationship with a provider, person-centered care, enhanced access to care, coordinated care, quality and safety, and a payment structure (NCQA, n.d.). Additional requirements for PCMH designation include patient tracking and registry, a care management component, patient self-management support, advanced electronic communication (including electronic prescribing), and performance reporting and improvement. Outcome measures include patient and family satisfaction, decreased emergency department use, decreased hospitalization, improved health parameters (e.g., blood pressure, hemoglobin [Hb] A1c), provider satisfaction, decreased cost, improved preventive care, and patient language preference.

Initial data report improved outcomes for the PCMH, including reduction in emergency department visits and hospitalizations, improved patient satisfaction, and increased use of measures such as recommended diabetes services (NCQA, 2015). APRNs are involved within PCMHs in providing primary care, as well as care coordination, health system navigation, and as community team members.

TRANSITIONS OF CARE

Coordination of Care

Improving care coordination has been identified by payers and policymakers as a priority to improve quality and lower costs of health care. The National Quality Forum (2010) identified important performance measures of care coordination, including prevention of hospital admissions and readmissions, care transitions, the patient experience of care coordination, the importance of cultural and linguistically appropriate resources and services, and communication to support patient self-management. Others have identified

continuity of care as an important measure of care coordination. The recent chronic care reimbursement codes from the CMS (2016) require continuity of care with a "designated practitioner or member of the care team with whom the patient is able to get successive routine appointments." The PCMH also requires that the patient have an ongoing relationship with a specific provider.

Care coordination has always been central to the role of the registered nurse and the APRN. Multiple research about nursing roles demonstrates that nurses are key in facilitating communication between the patient, the patient's family, and other health-care providers (Yang & Meiners, 2014).

Transitional Care Models

Medicare's attention to preventing hospital readmissions places a high premium on care continuity and coordination, with a central focus on transitions across providers and sites of care. Health-care services for older adults exist on a complex continuum that includes primary care, acute care, post-acute care, long-term care, home health care, and community-based services. Each of the care settings along the continuum serves a specific purpose and meets the needs of specific patients, but also generates health-care transitions that occur as patients move across multiple providers, payers, and settings for different acute episodes or for the same episode of care. Efforts to improve care transitions are intended to improve patient safety and quality of care; decrease significant burden on patients, families, and caregivers; and decrease costs to patients, providers, and payers.

Transitional care is defined as a set of actions necessary to ensure the coordination and continuity of care as patients transfer between different sites of care or from one level of care to another within the same site (Coleman & Berenson, 2004). The transitional care model (TCM) defines transitional care as a "broad range of time-limited services designed to ensure health care continuity, avoid preventable poor outcomes among at-risk populations, and promote the safe and timely transfer of patients from one level of care to another or from one type of setting to another." Transitional care functions in a complementary role to other health services, is time-limited, and has a specific focus on managing adverse outcomes of transitions (Naylor, Aiken, Kurtzman, Olds, & Hirschman, 2011, p. 747).

As a response to challenges facing the health-care system, APRNs were the first to develop and study transitional care, an empirically supported method to reduce readmissions post hospitalization (Verhaegh et al., 2014). The most consistently tested models of transitional care are the Care Transitions Intervention created by Dr. Eric Coleman (The Care Transitions Program, n.d.) and the Transitional Care Model, which has consistently shown reductions in readmissions, emergency department visits, and costs of care (Hirschman, Shaid, McCauley, Pauly, & Naylor, 2015).

Technology plays a significant role in communication and coordination of care to improve access to care and quality, especially with the implementation of electronic medical records (EMRs). Because hospital readmission is a common and costly problem, and a target of recent health-care system reform, efforts to use technology to advance knowledge of high-risk patients and standardize evidence-based discharge information are priorities. Recently, a team of nurse researchers created an evidence-based decision support tool that integrates with hospital EMR systems to identify high-risk patients in order to prevent readmissions. Outcomes were dramatically improved, with 37% reduction in 60-day readmissions (Bowles et al., 2015).

Palliative Care

Chronic illnesses are marked by fluctuations over time, with the potential for worsening symptoms, decrease in function, and caregiver burden. Palliative care consists of specialized care for people living with serious illness. It focuses on providing relief from the symptoms and the stress of the serious illness, with the goal is to improve quality of life for both the patient and the family. Palliative care can serve as an intervention to manage chronic illness, not just at the end of life, but also in the early phases of illness to support patients and caregivers. Patients who receive early palliative care show significant improvements in quality of life and mood, and may survive longer (Center to Advance Palliative Care, n.d.).

The Center to Advance Palliative Care emphasizes that palliative care is commonly used among people living with serious, complex, and chronic illnesses. The most common clinical conditions relevant to this discussion include cancer, congestive HF, COPD, and CKD and/or end-stage renal disease. Palliative care should be considered for close to half (45%) of all Medicare beneficiaries who have four or more chronic conditions for which palliative care services may be clinically indicated. Palliative care is used to alleviate symptoms, either in combination with or instead of curative treatment (Effiong & Effiong, 2012).

Interprofessional Education and Interprofessional Teams

Prevention and management of complex older adults with chronic illness are best implemented by interdisciplinary teams in primary care and in most other sites of care. It is suggested that a larger interdisciplinary workforce is needed, and payment for primary care should reward practices that incorporate interdisciplinary teams. Interprofessional education (IPE) occurs when learners of two or more health and/or social care professions engage in learning with, from, and about each other to improve collaboration and the delivery of care (IOM, 2015).

Researchers and educators continue to evaluate the impact of IPE on outcomes, including patient safety, patient and provider satisfaction, quality of care, health promotion, population health, and the cost of care. Core competencies for interprofessional practice were recently updated by the Interprofessional Education Collaborative, which is now comprised of 16 health-care professional groups. Those competencies relate to values and ethics for interprofessional practice, roles, and responsibilities; interprofessional communication; and teams and teamwork (Interprofessional Education Collaborative, 2016).

Interprofessional practice continues to validate approaches to complex patient management. In one study, using a team consisting of a physician, nurse practitioner, clinical pharmacist, nurse, dietary specialist, and behaviorist, clinical outcomes were optimized in a low-income diabetic population. Interprofessional diabetic care improved outcomes related to the Hb A1c, triglycerides, and blood pressure values (Hutchison, 2014).

Improved interprofessional teamwork and team-based care play critical roles in many of the new primary care approaches, including the PCMH, chronic care coordination, and transitions of care.

PROVIDER REIMBURSEMENT FOR CHRONIC ILLNESS CARE

Chronic Care Management Services

There is widespread awareness that frail geriatric patients and others with complex physical and functional problems often experience worsening disease, decreased function, unwanted transfers to skilled nursing facilities, and potentially costly emergency department visits or preventable hospital admissions or readmissions. Typically, those complex issues are not well addressed within the traditional outpatient office visit. As previously discussed, strategies and programs that include care coordination are known to lower the rates of adverse outcomes.

As of January 2015, Medicare started reimbursing physicians, nurse practitioners, and other non-physician providers for non–face-to-face care coordination for patients with two or more chronic conditions associated with significant risk of exacerbation, decompensation, functional decline, or death. Those services are known as chronic care management (CCM) services (CMS, 2016).

The CMS stipulates that the clinician must provide at least 20 minutes of coordination for each 30-day billing period and lists seven components of CCM. Three components involve the patient and clinician, including continuous access to care management (including use of EMRs and 24/7 telephone coverage); continuous care with a designated health-care professional; and multiple ways for the patient or caregiver to communicate with that clinician (Aronson, Bautista, & Covinsky, 2015). The next two requirements relate to the clinician's plan of care. Initially, there must be a comprehensive assessment and management of the patient's medical, cognitive, functional, and psychosocial needs, as well as the patient's environment, resources, and support. The second requirement requires creation of a comprehensive care plan. The third and fourth requirements include the management of transitions among clinicians and settings, and coordination with home- and community-based health-care professionals (Aronson et al., 2015).

Some practices have the ability to incorporate all of the requirements for chronic care management, while others will need to make additional investments in technology, staff training, and the care management protocols. Patient care benefits of CCM services include increased round-the-clock access to providers, enhanced care continuity across settings, stronger community support services, and the ability to develop a patient-centered care plan with providers.

Transitional Care Management Services

Transitional care management codes offer providers reimbursement for the non–face-to-face care furnished when patients transition from an acute care setting back into the community. The two care transition codes cover communication with the patient within 2 business days of discharge. Communication can occur by phone, email, or in person (CMS, 2016). Patient care benefits include enhanced care continuity during their transition from the hospital to home, with increased access to providers throughout the transition.

The Annual Wellness Visit

The annual wellness visit (AWV) is a yearly preventive care visit offered at no cost to all Medicare Part B beneficiaries and separate from any physical examination. The purpose of the visit is to identify patient risk factors and plan for future preventative service needs. The visit is well reimbursed and can be conducted by a provider or another supervised professional. There are significant patient care benefits, including a yearly opportunity for a health-risk assessment, which includes psychosocial and behavioral risks, functional and cognitive assessments, as well as a general safety check. Older adults also have the ability to work with providers to update a preventative care plan and be connected to necessary community resources.

Advance Care Planning Discussions

In 2016, Medicare began covering advance care planning discussions as a separate and billable service. Physicians, APRNs, and other health professionals are encouraged to have discussions with patients regarding end-of-life care preferences. Communication about serious illness care goals is now an intervention that is systematically integrated into our clinical care structures and processes. The CPT codes allow the clinician to explain and discuss advance directives and assist with the completion of approved forms. The codes are time-based and allow for a basic code, as well as an add-on code for each additional 30 minutes of time spent with patients (CMS, 2016).

End-of-life conversations are associated with better quality of life, reduced use of life-sustaining treatments near death, earlier hospice referrals, and care that is more consistent with patient preferences. Patients who engage in advance care planning are more likely to have their wishes known and followed. Best practice in communication about advance care planning should include discussions about individual goals of care, prognosis, decision-making preferences, and wishes for family involvement (Bernacki & Block, 2014).

THE ROLE OF APRNS IN CHRONIC DISEASE

The effect of health-care reform as part of the ACA of 2010, the rise in complex chronic diseases, and the continuing growth of the aging population combine to create a huge demand for health-care services. Individuals with multiple chronic disease, including frail older adults, will interact frequently with the health-care system. APRNs will continue to play an essential role in individual patient quality of care, as well as health system reform.

National attention has been placed on the removal of all scope-of-practice restrictions on APRNs as a way to drive down costs, while increasing high-quality access to primary health care. Numerous studies have shown the importance of APRNs as key components of an overall health-care system strategy to address chronic disease (Yang & Meiners, 2014).

APRNs are ideally suited to incorporate elements of the CCM into practice, as they excel in patient education and patient empowerment, with the goal of supporting the patient's self-management skills. APRNs are involved in technology and clinical information systems with shared medical appointments, telehealth, electronic documentation, and developing and maintaining data for quality improvement. APRNs embrace evidence-based care that is maximized with decision support tools and algorithms.

APRNs appreciate the diversity of the older population, including diversity of health trajectory, functional ability, cultural background, support systems, as well as individual values and personal preferences. The APRN's holistic approach to care includes prevention and support of healthy aging, education, care coordination, management and monitoring of complex medication and treatment regimens, counseling for family issues related to caregiving and finances, access to community resources, advance care planning, and the vigilant management of all comorbid conditions.

New health-care models and alternatives to traditional care address discontinuities in health care. APRNs are presently involved in unique strategies that target high-risk older adults across systems of care, partnerships with families and caregivers, identification of resources for individuals with chronic disease, and involvement in key policy decisions that affect those populations. APRNs are committed to providing affordable, accessible, and high-quality care through clinical practice, education, leadership, and research.

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Develops and operationalizes a phenotype of frailty in older adults and assesses concurrent and predictive validity.	B	Fried et al., 2001
Comparison of three interventions to enhance outcomes of hospitalized cognitively impaired older adults. Prospective nonrandomized study.	B	Naylor et al., 2014
Analysis of hospital discharge data to examine hospital referral regions and variations in readmission rates and the extent to which multiple chronic illnesses contribute to the variations.	B	Basu, Avila, & Ricciardi, 2016
Systematic review to investigate effectiveness of chronic care management for HF.	B	Drewes et al., 2012
Systematic review of the effects of community-based health worker interventions to improve chronic disease management.	B	Kim et al., 2016
A narrative literature review synthesis of current research findings related to self-management to support improved priority setting and decision making among adults with multimorbidities.	C	Bratzke et al., 2015
A systematic review of patient-reported measures of burden of treatment in diabetes, CKD, and HF.	B	Eton et al., 2013

A = consistent, good-quality, patient-oriented evidence; B = inconsistent or limited-quality, patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to www.aafp.org/afpsort.xml.

CASE STUDY

Mrs. A. is an 82-year-old Caucasian woman. She lives with her 86-year-old husband, who has mild-to-moderate dementia. Mrs. A. has a primary care provider, endocrinologist, cardiologist, ophthalmologist, and podiatrist. She has a daughter nearby who assists with shopping and housekeeping. Mrs. A. is overweight and has hypertension, type 2 diabetes, CKD, macular degeneration, incontinence, and peripheral neuropathy with chronic pain. She has trouble sleeping, complains of chronic fatigue, and has fallen twice in the past 2 months. She is on nine prescription medications. Her self-care regimen includes monitoring her glucose, weighing herself daily, taking medications twice daily, and managing her nutrition and exercise. She is also responsible for managing her husband's meals and medications.

Additional comprehensive assessment is critical to developing a person-centered plan of care for Mrs. A. A

Continued

CASE STUDY—cont'd

multidisciplinary and continuity-of-care focus will prevent poor outcomes.

1. What multiple factors place Mrs. A. at high risk for poor outcomes?
2. What additional assessment data are essential to determine if Mrs. A. can safely manage her complex health-care regimen at home?
3. How do Mrs. A.'s multiple chronic diseases contribute to the complexity of her care?
4. Are there caveats when using evidence for individual chronic diseases in light of her MCCs?
5. What communication is necessary to provide ongoing coordination of care?
6. What community resources may be available to support Mrs. A.?
7. What additional team members would be appropriate to contribute to a safe and person-centered plan of care?
8. What new models of care can potentially contribute to Mrs. A.'s quality of care while she is at home or in the event of a hospitalization?

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CHAPTER 19

Palliative Care and End-Of-Life Care

M. Jane Griffith and Christine Coletta Hansen

OVERVIEW OF PALLIATIVE CARE

Description: Palliative care is an interdisciplinary approach to care aimed at improving the quality of life of patients and their families facing a life-threatening illness. The goal of palliative care is the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems, physical, psychosocial, and spiritual. Palliative care:

- Provides relief from pain and other distressing symptoms
- Affirms life and regards dying as a normal process
- Intends to neither hasten nor postpone death
- Integrates the psychological and spiritual aspects of patient care
- Offers a support system to help patients live as actively as possible until death
- Offers a support system to help the family cope during the patient's illness and in their own bereavement
- Uses a team approach to address the needs of patients and their families, including bereavement counseling, if indicated
- Enhances quality of life, and may also positively influence the course of illness
- Is applicable early in the course of illness, in conjunction with other therapies that are intended to prolong life, such as chemotherapy or radiation therapy, and includes those investigations needed to better understand and manage distressing clinical complications (World Health Organization [WHO], 2016)

Families are included in the care planning. Patients and families benefit from the availability of palliative care services early in the disease process, particularly when symptoms affect their quality of life. As the disease advances, hospice care becomes an option. The National Hospice and Palliative Care Organization (NHPCO) defines hospice as, "a team-oriented approach to providing specialized care for people facing a life-limiting illness or injury. It includes expert medical care, pain management and emotional support for patients and their families" (NHPCO, 2014). Hospice is a program of care designed for the last 6 months of a person's life. It uses the principles of palliative care, focusing on quality of life, to support patients and their families through the dying process, and includes bereavement services. Hospice services are covered by Medicare, Medicaid, and most private insurance providers.

Etiology: The five leading causes of death in 2014 were heart disease, malignant neoplasms, chronic respiratory disease, accidents, and cerebrovascular disease (Kochanek et al., 2016) (see Box 19-1).

Numerous medical advances in the management of these illnesses have led to people living longer with chronic health conditions. Accurate prognostication of chronic illnesses is difficult, and the difficulty of prognostication can result in overuse of acute care interventions at the end of life and delays in referral to hospice care. In 2014, the median length of stay in a hospice program was 17.4 days, meaning one-half of patients died within 2.5 weeks of enrollment in a hospice program. The average length of stay in a hospice program was 71.3 days (NHPCO Facts and Figures, 2015). Thus, most people are not benefiting fully from the Hospice Medicare Benefit, which is designed to provide care for patients in the last 6 months of life.

There are several obstacles to implementing palliative care, including confusion around terminology (hospice versus palliative care), misperception about the intent and scope of care, concerns regarding cost and insurance coverage, potential mistrust with perceived economic motive, prognostic uncertainty, provider discomfort with end-of-life discussions, and the psychiatry of decision makers with patients and providers having a bias toward optimism, as well as provider fear of doing harm but failing to provide an intervention (McAteer & Wellbery, 2013). The question, "Would I be surprised if this patient died in the next year?" as well as frequent hospitalizations; admissions prompted by physical or psychological symptoms that are difficult to treat; those with complex care requirements; and those with functional decline, feeding intolerance, and/or unintended weight loss are triggers that could indicate need for palliative care (Weissman & Meier, 2011). Additional screening criteria include hospital admission from a long-term care facility; elderly with cognitive impairment and acute hip fracture; metastatic cancer;

BOX 19-1

1. Heart disease	6. Alzheimer's disease	11. Septicemia
2. Malignant neoplasm	7. Diabetes mellitus	12. Chronic liver disease and cirrhosis
3. Chronic lower respiratory diseases	8. Influenza and pneumonia	13. Essential hypertension and hypertensive renal disease
4. Accidents	9. Kidney disease	14. Parkinson's disease
5. Cerebrovascular accidents	10. Suicide	15. Pneumonitis due to solids and liquids

Adapted from Kochanek et al., 2016.

chronic home oxygen use; cardiac arrest occurring outside of the hospital; current or past hospice enrollment; limited social support; and no documented advanced care planning discussion (Weissman & Meier, 2011). Cardona-Morrell and Hillman (2015) are investigating a new screening tool: Criteria for Screening and Triaging to Appropriate aLternative Care (CriSTAL).

Occurrence: Seventy-five percent of hospital deaths are those who are greater than age 65 years; 27% are those greater than age 85 years (AACN & COH, ELNEC Core, 2016). Of those 75 years and older, 33% die in a nursing home, and of those 85 years and older, 42% die in a nursing home. It is important to provide access to hospice and palliative care services in all settings. The goal is to ensure a peaceful, dignified death with symptoms well-managed and support for the family and caregivers. Establishing the goals of care in treating older adults with life-limiting illnesses is essential (O'Neill & Morrison, 2015).

Age: Average life expectancy is 78.8 years, according to National Centers for Health Statistics (Vu et al., 2016). The fastest growing segment of the population is the 85 years and older age group, with that population expected to double by 2036 and triple by 2049 (Ortman, Velkoff, & Hogan, 2014). Palliative care is beneficial for the frail elderly with symptom burden, functional limitations, cognitive impairment, and lack of family or social support (Wajnberg et al., 2013).

Caregiving needs increase with advanced age and are typically met by the family. As caregivers, frail elderly spouses face challenges in meeting both the patient's needs and the caregiver's own needs. Caregivers are at risk for physical and psychological illness, including depression, insomnia, fatigue, and anxiety, as well as physiological effects, which can lead to early death (Bevans & Sternberg, 2012).

Gender: The average overall life expectancy for females is 81.2 years; the average life expectancy for males is 76.4 years (Xu et al., 2016).

Ethnicity: The average life expectancy for Caucasians is 79.1 years; the average life expectancy for African Americans is 75.5 years; and the average life expectancy for Hispanics is 81.6 years (Xu et al., 2016). Cultural influences affect how people define health and illness and shape their beliefs and practices in end-of-life care (AACN & COH, ELNEC Core, 2016).

Contributing Factors: Contributing factors include advanced age and multiple comorbidities.

Signs and Symptoms: See Symptom Management.

Differential Diagnosis: Not applicable.

SYMPTOM MANAGEMENT

A few of the most prevalent symptoms in end-of-life care in the geriatric population are delirium, dyspnea, and pain (AACN & COH, ELNEC Core, 2016). Additional symptoms such as nausea and vomiting, constipation, diarrhea, fatigue, depression, anxiety, and insomnia are also common. These latter symptoms are described in detail elsewhere in this book. When addressing pharmacological symptom management in the elderly it is important to consider physiological changes, such as decreased renal and hepatic function and altered body fat distribution, that may alter drug metabolism, leading to higher serum drug levels. Elderly people are more susceptible to adverse reactions, thus, begin with lower initial doses and titrate cautiously (O'Neill & Morrison, 2015).

DELIRIUM

Description: Delirium is disturbance of attention and awareness accompanied by a change in baseline cognition, developing over a short period of time, fluctuating during the course of a day, and often worsening at night (APA, 2013). Those with delirium have:

- A reduced awareness of their environment
- Disturbance of consciousness with impaired ability to focus, sustain, or shift attention
- Cognitive changes involving memory impairment, disorientation, and language disturbance
- Perceptual disturbances, including delusions and hallucinations

Delirium can be hyperactive with symptoms of hyper-alertness, hypervigilance, and agitation, or delirium may be hypoactive and characterized by somnolence (Quill et al., 2014).

Etiology: There are numerous potential causes of delirium in the dying patient related to the terminal disease itself, treatment of the disease, metabolic effects of organ failure, electrolyte imbalance, including hypoglycemia, infection, hematological disorders, infection, nutritional deficiencies,

dehydration, hypoxia, uncontrolled pain, sensory deprivation, sleep disturbance, alcohol or drug withdrawal, diarrhea, constipation, and urinary retention. Many medications, particularly benzodiazepines and anticholinergic medications, can cause delirium. Opioids, particularly meperidine, may increase risk for delirium as well (AACN & COH, ELNEC APRN, 2016; Francis, 2014; AGS, 2015).

Occurrence: Delirium is common at end of life and can be very distressing to the family, as well as to the patient. Prevalence ranges from 13% to 80%. It is often unrecognized, particularly hypoactive delirium (Quill et al., 2014). Terminal delirium is common as death approaches, affecting up to 90% (Szarpa et al., 2013).

Age: Advancing age is a risk factor for delirium (AACN & COH, ELNEC Core, 2016). Older patients presenting with delirium have up to 50% mortality at 1 year (Quill et al., 2014).

Gender: No significance.

Ethnicity: Delirium can affect all people. Utilize culturally sensitive screening tools.

Contributing Factors: The frail elderly with less physiological reserve, multiple comorbidities, polypharmacy, pre-existing dementia, previous history of delirium, sensory impairment, and who are in an unfamiliar environment are more vulnerable to delirium. Patients admitted to the hospital, in particular to an intensive care unit, are at increased risk for delirium (Fletcher, 2014).

Signs and Symptoms: Acute onset and fluctuating course of inattention, altered level of consciousness, and disorganized thinking are signs of delirium. Disorientation, memory impairment, perceptual disturbances, psychomotor agitation or retardation, emotional instability, and impaired sleep–wake cycle are common findings (Fletcher, 2014).

Diagnostic Tests: Patient history and assessment, including a review of the patient's medications, are the first steps in diagnosing delirium. Disorientation, memory impairment, perceptual disturbances, psychomotor agitation or retardation, and impaired sleep–wake cycle are common findings. The Confusion Assessment Method (CAM) has been validated as an effective tool to diagnose delirium (Inouye et al., 1990). CAM assesses for the presence of acute onset and fluctuating course, as well as inattention and either disorganized thinking or altered levels of consciousness. These findings are diagnostic of delirium. Other tools include the Memorial Delirium Assessment Scale and the Delirium Rating Scale (AACN & COH, ELNEC Core, 2106). More invasive diagnostic tools may be indicated to determine the etiology if further evaluation is consistent with goals of care. In the terminally ill, the most common cause of delirium is medications, including opioids, anticholinergics, and benzodiazepines. The second most common cause of delirium is metabolic insufficiency due to organ failure (AACN & COH, ELNEC Core, 2016; Quill et al., 2014).

Differential Diagnosis: Consider the possibility of dementia, depression, or the coexistence of either of these with delirium (Fletcher, 2014).

Treatment: The first step in treating delirium is to identify the cause(s) if possible. Make appropriate changes to the plan of care based on the etiology. Perform regular medication reviews to eliminate unnecessary agents. Consider inadequate pain control as a cause of delirium, but remember that opiates can cause delirium and dose reduction may be necessary (Francis, 2014). Deprescribing of psychoactive medications is also important (Lawlor & Bush, 2014). Evaluate fluid and electrolyte imbalances, infections, organ failure, hypoglycemia, drug toxicity, as well as withdrawal from alcohol and sedatives (Francis, 2014). Attention should also be paid to providing a therapeutic environment, as well as a supportive and safe environment focusing on clear communication and a structured daily routine with consistent caregivers and structured sleep–wake cycle, avoiding sensory overstimulation and providing for glasses and hearing aids if indicated, and providing physical activity and attention to oral intake and elimination (Fletcher, 2014). Frequent reassurance, touch, and verbal orientation may lessen behavior disturbances (Francis, 2014).

Nonpharmacological interventions are the mainstay of managing delirium. However, in the acutely agitated patient, a trial of pharmacological agents may be indicated. Neuroleptic agents are generally used, but there continues to be limited data to support their use (Francis, 2014). Atypical antipsychotics, such as olanzapine, risperidone, quetiapine, and ziprasidone, can be considered for the agitated patient. However, these agents carry an increased risk of QT prolongation, with increased risk for cardiovascular events and death in the elderly with dementia (AACN & COH, ELNEC Core, 2016; Francis, 2014; Quill et al., 2014). In the palliative care literature, haloperidol is considered the mainstay of delirium management, beginning with low doses, 0.5 mg intravenously or subcutaneously, or 1 mg orally (Bailey & Harman, 2016; Quill et al., 2014). Titrate slowly in the elderly. Chlorpromazine 25 to 50 mg is an alternative if more sedation is needed, but it may cause hypotension (Quill et al., 2014).

Benzodiazepines, such as lorazepam, may benefit patients with a seizure disorder or alcohol withdrawal and those who cannot tolerate antipsychotic medications. However, benzodiazepines may lead to paradoxical agitation, causing symptoms of delirium to worsen. Therefore, these agents should be used cautiously (AACN & COH, ELNEC Core, 2016; Quill et al., 2014).

Anticholinergic medications should be avoided. Cholinesterase inhibitors do not have a role in treating delirium. In fact, a trial comparing rivastigmine to placebo in intensive care unit patients was stopped early due to higher mortality in the rivastigmine arm (Francis, 2014). On the other hand, melatonin and the melatonin agonist ramelteon are showing promise in the prevention of delirium (Francis, 2014; Lawlor & Bush, 2014).

Follow-Up: Provide adequate treatment of underlying cause. Due to multimodal causes, an interdisciplinary approach is preferred.

Sequelae: Prompt evaluation and treatment can improve outcomes. Delirium is distressing to patients, their families, and health-care providers. Delirium contributes to increased morbidity and mortality. Older patients presenting with delirium have up to 50% mortality at 1 year (Quill et al., 2014). Patients are at risk for falls and potential injury. In end-of-life care, the goal is supportive care to promote a peaceful, dignified death.

Prevention/Prophylaxis: Avoid polypharmacy. Discontinue unnecessary medications and use high-risk medications cautiously. Maintain adequate hydration and nutrition, as appropriate, with prompt attention to elimination needs. Minimize invasive devices such as catheters. Correct sensory deficits. Promote normal sleep patterns. Avoid physical restraints. Use music and massage. Provide a therapeutic milieu with appropriate environmental stimulation (Fletcher, 2014; Francis, 2104; AGS, 2015).

Referral: Refer the patient to a geriatric psychiatrist, geriatrician, geriatric nurse practitioner, and/or palliative medicine practitioner as needed.

Education: Teach the patient's family signs of delirium, and ask that they report the signs promptly to the medical team. Teach nonpharmacological methods of providing a supportive environment. Avoid over-the-counter (OTC) medications.

DYSPNEA

Description: Dyspnea, also called breathlessness, has been defined by the American Thoracic Society as a subjective experience of breathing sensations that vary in intensity. The experience of dyspnea derives from interactions of physiological, psychological, social, and environmental factors that may induce secondary physiological and behavioral responses (Parshall et al., 2012). It is a distressing symptom at end of life and affects quality of life.

Etiology: Dyspnea can have multiple causes, such as infection, cancer, heart failure, chronic obstructive pulmonary disease (COPD), asthma, pulmonary fibrosis, pneumothorax, pleural effusion, anemia, and amyotrophic lateral sclerosis (Brogolio, 2016).

Occurrence: Dyspnea is the most reported symptom as patients approach the end of life, occurring in 94% of patients with serious, advanced illness (AACN & COH, ELNEC Core, 2016). Dyspnea is also one of the most common reasons for visits to the emergency department for individuals with advanced disease (Vandyk, 2012). Approximately 60% to 95% of those with advanced heart disease and advanced COPD report breathlessness. Cancer patients have a 10% to 75% prevalence, and 11% to 62% of those with renal disease report breathlessness (AACN & COH, ELNEC Core, 2016). Compared to patients with lung cancer, patients with COPD have similar or worse dyspnea symptom burden and health related quality of life (Wysham et al., 2015).

Age: Certain causes, such as heart failure, cancer, and chronic lung disease, are more common in the elderly.

Gender: No gender-specific differences.

Ethnicity: Not applicable.

Contributing Factors: Smoking and comorbidities can contribute to the development of dyspnea.

Signs and Symptoms: Shortness of breath at rest and/or exertion, tachypnea, use of accessory muscles, hypoxia, adventitious breath sounds, cyanosis, and pursed-lip breathing are all signs of dyspnea.

Diagnostic Tests: The first step in diagnosing dyspnea is the clinical patient interview and physical examination. The physical examination should include respiratory rate, oxygen saturation, and assessment of breath sounds, heart rate, heart sounds, presence or absence of jugular venous distension, pallor, or ascites. The clinical interview should at a minimum include lifestyle assessment (such as smoke or irritant exposure), modifying factors or self-interventions, and use of a dyspnea assessment tool (Parshall et al., 2012). Multiple dyspnea assessment tools are validated, however, many are one-dimensional, disease-specific, or do not address the affective component in dyspnea in advanced disease. The American Thoracic Society and American College of Chest Physicians do not endorse one specific tool, however, use of these tools in combination with the clinical interview, physical examination, and diagnostic testing, if warranted, while keeping in mind the patient's goals for care, can help determine the appropriate course of dyspnea treatment (Parshall et al., 2012).

Examples of dyspnea assessment tools are the Modified Borg Scale, Visual Analog Scale, Numeric Rating Scale, Memorial Symptom Assessment Scale, Edmonton Symptom Assessment Scale, Dyspnea-12 (for congestive heart failure [CHF], COPD, interstitial lung disease [ILD]), Cancer Dyspnea Scale (cancer-specific and addresses the affective component of dyspnea as well), and Respiratory Distress Observation Scale for those patients who are unable to self-report (Brogolio, 2016).

Diagnostic tools should be used as appropriate, considering the patient's goals of care. Diagnostic tests include laboratory tests, such as complete blood count to determine anemia, basic metabolic panel to evaluate renal function, chest x-ray, and electrocardiogram (EKG). Computed tomography (CT) scan to evaluate for malignancy and/or pulmonary embolism may be indicated. Echocardiogram may also be performed to evaluate for poor cardiac ejection fraction or pericardial effusion (AACN & COH, ELNEC Core, 2016).

Differential Diagnosis: Consider all etiologies as outlined previously. If cough is present, consider pneumonia; pleural effusion; side effects of medications, such as angiotensin-converting enzyme inhibitors; and gastroesophageal reflux disease.

Treatment: Treatments should be aimed at the underlying etiology. Pharmacological interventions might include diuretics, bronchodilators, vasodilators, steroids, antibiotics, opioids, and/or sedatives (AACN & COH, ELNEC Core, 2016; Brogolio, 2016) (see Table 19-1). In a systematic review and several smaller studies, patients reported dyspnea relief with opioids (Currow, 2013; Horton, 2013; Simon et al., 2013). For patients with advanced disease, opioids are the first-line treatment for dyspnea. The use of opioids improves dyspnea, provides little impact on respiratory blood gases, and appears

TABLE 19-1 Pharmacological Interventions for Dyspnea

Opioids	Morphine 0.5 to 10 mg orally every 3–4 hours prn SOB
Nebulized opioids	Further research is needed
Benzodiazepines	Studies reveal no significant benefit
Nebulized furosemide	Potential benefit, further research is needed
Bronchodilators, diuretics, steroids, antibiotics, vasodilators	As indicated based on etiology

to have no significant impact on patient's life expectancy (AACN & COH, ELNEC Core, 2016). Morphine is the most extensively studied opiate and has been shown to be effective for treatment of dyspnea (Currow et al, 2013). There is no consensus regarding dosing of opioids for dyspnea (Anwar & Case, 2016). The proposed range for morphine in opioid-naïve patients with dyspnea is from 0.5 to 0.1 mg orally q 4 hours (Rocker, 2009) to 5 to 10 mg orally q 3 to 4 hours (Lanken, 2008), up to trialing extended release morphine with titration to 30 mg per day for those with chronic dyspnea (Currow et al., 2011).

When using opioids in the elderly, a "start low, go slow" approach should be adopted. Benzodiazepines have been used for the affective component of dyspnea, however, analysis of several studies in patients with advanced cancer or COPD did not show significant benefit, regardless of type, dose, or route (Simon et al., 2012). Inhaled/nebulized opioids and anesthetics have not proved to effectively treat dyspnea. Inhaled furosemide showed potential benefit for refractory dyspnea (Anwar & Case, 2016). Further research is needed before nebulized fentanyl can be recommended (Bausewein & Simon, 2014). Higher level reviews have failed to show positive effects of nebulized opioids for treatment of dyspnea and it is recommended that further research with more rigorous designs and larger sample sizes occur (Simon, 2013; Bausewein, 2014). It is also recommended to reduce excessive secretions with scopolamine, hyoscyamine, atropine, or glycopyrrolate (AACN & COH, ELNEC Core, 2016).

Nonpharmacological interventions include the use of compressed air or a fan for comfort, oxygen if hypoxic, elevation of the head of the patient's bed, relaxation, and breathing exercises, cooler temperatures, relaxation, and stress reduction techniques (Brogolio, 2016). Temporary ventilator support can be explored if clinically indicated for severe reversible conditions. The routine application of oxygen to patients who are near death is not supported (Campbell, Yarandi, & Doke-Meadows, 2013). Oxygen therapy for those who are hypoxic has shown to be beneficial for managing dyspnea. Implement oxygen therapy if there is a subjective report of relief. Abernathy (2010) found that the subjective feeling of dyspnea was relieved in those who were treated by air via nasal cannula at the same rate of those with oxygen therapy, thus questioning the need for oxygen therapy. Ameer and colleagues (2014) concluded that due to the quality of studies the evidence is inconclusive to recommend oxygen for long-term use in those with COPD who are not hypoxemic at rest. A study looking at the use of a different gas mixture (heliox 28%) in nonhypoxic dyspneic patients undergoing exercise indicated that there may be some benefit of this gas mixture (American Thoracic Society, 1999, as cited in Wysham et al., 2015). Noninvasive ventilation is suggested to be more effective compared with oxygen in reducing dyspnea and the need for higher morphine doses in patients with refractory dyspnea in end-stage cancer (Nava et al., 2013). Temporary ventilator support can be utilized if clinically indicated for severe reversible conditions.

Interventional therapies to be considered for dyspnea management related to refractory pleural effusions include thoracentesis, pleurodesis, and tunneled catheter to drainage. Other interventional options for refractory dyspnea related to obstruction can be bronchoscopy or stenting. Discontinue fluid support and consider low-dose diuretics if fluid overload may be a contributing factor. Although explored as a potential efficacious treatment for dyspnea, results from 25 systematic reviews did not find enough evidence to recommend acupuncture (Ben-Aharon et al., 2012).

Follow-Up: Follow-up is dependent on etiology. Ongoing assessment, reevaluation, and adjustment of plan of care should be used as needed. Monitor for therapeutic effect, as well as adverse effects from interventions.

Sequelae: Dependent on etiology and goals of care, dyspnea should improve when the cause is treated. However, dyspnea is also common in the dying person, and the goal is comfort measures when death is an anticipated outcome.

Prevention/Prophylaxis: Preventive measures are dependent on the cause. Smoking cessation is beneficial, as is the avoidance of smoke exposure. Use energy conservation and durable medical equipment for the severely dyspneic patient with advanced illness. If the patient is an aspiration risk, modifying food textures and a speech therapy consult may be beneficial.

Referral: Referral may be indicated based on etiology and goals of care. Appropriate referrals may include visits to a palliative care specialist, cardiologist, pulmonologist, and/or oncologist.

Education: Smoking cessation; the proper use of medications, including inhalers and nebulizers; the use of fans; energy conservation; and potential adverse effects of medications.

PAIN

Description: Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage (IASP definition at www.iasp-pain.org/taxonomy, 2015). Pain is whatever the experiencing person says it is, whenever they are experiencing it (Pasero & McCaffery, 2011). According to the Institute of Medicine's (IOM's) 2014 report *Dying in America: Improving Quality and Honoring Patient Preferences near End-of-Life,* 46% of older adults suffer pain in the last month of life. According to estimates, the prevalence of pain in cancer survivors is reported to be as high as 40% (Paice, 2016). The patient's own report is the best indicator of pain (American Geriatrics Society [AGS], 2009). Most elderly, including those with dementia, can effectively communicate pain, but a more detailed assessment of pain can be challenging. Several pain assessment tools are available, including the Visual Analogue Scale (VAS), Numerical Analogue Scale (NAS), Wong-Baker FACES Pain Rating Scale, Pain Assessment in Advanced Dementia (PAIN-AD) Scale, NOPPAIN, and Checklist of Non-Verbal Pain Indicators (AACN & COH, ELNEC Core, 2016; Venable, 2015).

Etiology: Pain is categorized as somatic, visceral, neuropathic, or mixed (AACN & COH, ELNEC APRN, 2016). Somatic pain originates from cutaneous, bone, and musculoskeletal tissues. Examples include muscle pain, such as fibromyalgia or myofascial pain; inflammatory pain, such as an acute injury or chronic inflammatory pain, including arthritis; and mechanical pain related to fractures or dislocations or compression of tissue by bony structures. Somatic pain is typically well localized and described as aching. Visceral pain originates in the thoracic, abdominal, and pelvic viscera. It is diffuse pain and often radiates. Neuropathic pain is nerve pain due to dysfunction of the nervous system. Examples include diabetic neuropathy, neuropathy due to herpes zoster, and peripheral vascular disease (Byrd, 2013). Psychological pain should be considered as well, related to depression, anxiety, personality disorders, somatization disorders, and post-traumatic stress disorder. Common causes of pain in the elderly are low back pain, cancer pain, and neurogenic pain syndromes (Byrd, 2013). Patients also experience nonmalignant pain from cardiac, renal, pulmonary, hepatic, neurological, or immune disorders (AACN & COH, ELNEC Core, 2016; Paice, 2015).

Occurrence: Pain is a common end-of-life symptom in the elderly. Chronic pain in the elderly has been estimated at 57% by the IOM, according to the *Prescribing Guidelines for Pennsylvania Geriatric Pain: Opioid Use and Safe Prescribing Guidelines* (2016). About 53% of cancer patients experience pain (Syrjala et al., 2014) and about 45% to 80% of nursing home residents experience pain (Byrd, 2013).

Age: The comorbidities experienced with aging make pain more likely. The elderly, particularly those with dementia, are at risk for undertreatment of pain (AACN & COH, ELNEC APRN, 2016). Many of the interventions to treat these challenging symptoms are pharmacological interventions. It is important to consider geriatric pharmacology when formulating treatment plans. Several physiological factors affect drug distribution, including cardiac output, regional blood flow, body mass and composition (which affect hydrophilic and lipophilic drug distribution), and plasma protein concentration. These changes make an elderly person more susceptible to adverse drug effects. Drug metabolism and clearance in the elderly is often slowed due to declining renal and liver function. Drug interactions, particularly with drugs metabolized through the P-450 enzyme system, also put an elderly person at risk. The health-care provider needs to be mindful of these effects when prescribing. Begin with low doses and titrate carefully, with frequent reassessment, to optimize pain relief and minimize adverse effects (AGS, 2009).

Gender: Women and men are at equal risk for undertreatment of pain (AACN & COH, ELNEC Core, 2016).

Ethnicity: Nondominant cultures are at risk for undertreatment of pain; biases exist in pain assessment and management. Culture impacts access to medical care, including access to medications (AACN & COH, ELNEC APRN, 2016).

Contributing Factors: Multiple comorbidities, as stated earlier in the text.

Signs and Symptoms: Complete a thorough pain assessment, including history, location, intensity (using an appropriate pain assessment tool), quality, pattern, aggravating/relieving factors, temporal pattern medication history, and cultural influences (AACN & COH, ELNEC Core, 2016). Clinicians conducting the comprehensive pain assessment should utilize one of the validated pain tools and also assess the holistic impact of pain, including physical, psychological, social, and spiritual factors, as well as capturing medical diagnoses, comorbid conditions, psychiatric history (including substance abuse history), and prior treatments for pain (Paice et al., 2016).

Diagnostic Tests: Patient history and physical examination should direct appropriate diagnostic testing to assess the underlying cause.

Differential Diagnosis: Determine somatic, visceral, and/or neuropathic pain, or treatment-related pain.

Treatment: Treatment of pain should include a multidimensional approach consisting of both pharmacological (see Table 19-2) and nonpharmacological interventions. The WHO estimates that its ladder can achieve adequate pain control in 80% to 90% of cancer patients (WHO, 2015, as cited in AACN & COH, ELNEC Core, 2016). Though AGS guidelines for treatment of pain in the geriatric population has not been updated since 2009, much of the data is still relevant today. The first step of the WHO ladder recommendation is the use of NSAIDs and acetaminophen for mild pain. Acetaminophen is still considered an effective agent for pain associated with osteoarthritis and low back pain. Many experts recommend limiting intake of acetaminophen to 3,000 mg per 24 hours to prevent overdose and decrease liver risks associated with its use (Byrd, 2013). In those with hepatic insufficiency or alcohol abuse, acetaminophen dose should be reduced by 50% to 75% (moderate quality of evidence, strong recommendation; AGS, 2009). Liver failure is an absolute contraindication (high quality of evidence, strong recommendation).

TABLE 19-2 Pharmacological Interventions for Pain Management

Acetaminophen	Limit to 3,000 mg or less in the elderly
NSAIDs	Avoid routine use in the elderly
Opioids: morphine, oxycodone, hydromorphone, fentanyl	Severe pain, start low go slow
Tramadol	Weak opioid antagonist and selective norepinephrine reuptake inhibitor (SNRI)
Methadone	Opioid agonist and NMDA antagonist
Buprenorphine	Opioid agonist-antagonist

Recent guidelines from the AGS advise against the routine use of NSAIDs in the elderly due to the significant potential for adverse effects. These include gastrointestinal (GI) toxicity, renal toxicity, and platelet dysfunction, as well as adverse effects on blood pressure control and heart failure management. The risk of myocardial infarction or stroke is increased with the use of NSAIDs (U.S. Food and Drug Administration [FDA], 2015). The risk increases with concurrent use of corticosteroids. Concurrent use of daily acetylsalicylic acid (ASA) and/or selective serotonin reuptake inhibitors (SSRIs) also may pose additional risk for GI bleeding. Administering NSAIDs with food, proton pump inhibitors, or histamine-2 blockers can help prevent GI challenges (Byrd, 2013). The COX-2 inhibitors may place a person at greater risk for cardiovascular events, including myocardial infarction (AGS, 2009). When using NSAIDs, the maximum daily doses (MDDs) for specific NSAIDs are: ASA, 4,000 mg/day; ibuprofen, 2,400 mg/day; Naprosyn, 1,375 mg/day; and Toradol, 120 mg in 24 hours (AACN & COH, ELNEC Core, 2016).

Patients with moderate-to-severe pain with functional impairment and/or decreased quality of life due to pain can be considered for opioid therapy (low quality of evidence, strong recommendation) (AGS, 2009). Step 2 of the WHO ladder adds opioids, typically in combination with acetaminophen, which limits the amount of total opioid given. Examples include codeine, hydrocodone with acetaminophen, and oxycodone with acetaminophen. These are used for moderate pain.

For severe pain, advance to step 3, which includes opioid pain medications. Opioid administration in the elderly population should consist of a "start low and go slow" approach. Consider long-term opioids in those patients who do not respond to more conservative management and who continue to experience pain-related distress or functional impairment and demonstrate benefit from the opioid medication. According to the American Society of Clinical Oncology (as cited in Paice et al., 2016), clinicians may prescribe a trial of opioids in cancer survivors with chronic pain. The only absolute contraindication to the use of opioids is if there is a history of rash, wheezing, or edema that was experienced in the past (Paice, 2016). Common opioids include morphine, oxycodone, and hydromorphone. Tramadol is a weak opioid agonist and a serotonin-norepinephrine reuptake inhibitor. It has a ceiling dose and can lower the seizure threshold, as well as contribute to hypoglycemia, and can lead to serotonin syndrome (AACN & COH, ELNEC Core, 2016). Monitor the patient on short-acting pain medication, and if the patient requires regular dosing every 4 to 6 hours, consider a long-acting formulation, but monitor closely for adverse effects (including altered mental status). Patients with continuous pain may be treated with around-the-clock dosing to achieve steady state (low quality of evidence, weak recommendation) (AGS, 2009).

Controlled-release opiates are appropriate for chronic pain (AACN & COH, ELNEC Core, 2016). Examples of long-acting opioids for pain control include morphine sulfate sustained release, oxycodone sustained release, hydromorphone sustained release, fentanyl patch, and the Butrans patch (Epocrates, 2016). Methadone is a unique opioid in that it appears to also act as an N-methyl-D-aspartate (NMDA) antagonist in addition to opioid receptor bonding. This unique element helps in the treatment of neuropathic pain management (AACN & COH, ELNEC Core, 2016; Quill et al., 2014). Methadone is also a long-acting pain medication, but it requires careful dosing and monitoring due to its variable half-life, potential prolongation of QT interval, and numerous drug interactions (McPherson, 2010).

Be cautious with opioid rotation and use an appropriate equianalgesic dosing chart (McPherson, 2010). Pay attention to renal and hepatic impairment. Choose an opioid with less active metabolites (fentanyl, hydromorphone, methadone) if renal function is declining (Portenoy & Ahmed, 2014, as cited in AACN & COH, ELNEC Core, 2016). Monitor for signs of drug accumulation, which can include delirium, sedation, and myoclonus. Also monitor polypharmacy due to potential for drug interactions. If opioids are no longer necessary, they should not be stopped abruptly. They should be tapered, as abrupt withdrawal could potentiate withdrawal symptoms (McPherson, 2010).

Anticipate, assess for, and identify potential adverse effects (moderate quality of evidence, strong recommendation) (AGS, 2009). Common adverse effects include constipation, nausea, sedation, urinary retention, and pruritis (AACN & COH, ELNEC Core, 2016). Long-term adverse effects include constipation, mental clouding, fatigue, osteoporosis, osteopenia, reduced libido, risk of myoclonus, mood changes, risk of opioid-induced hyperalgesia, and increased risk of sleep-disordered breathing (American Society of Clinical Oncology [ASCO], as cited in Paice et al., 2016).

Unless a patient is in the dying process, if a patient becomes unarousable or experiences opioid-induced respiratory depression, the opioid can be reversed by using an opioid antagonist such as naloxone (AACN & COH, ELNEC Core, 2016). Sedation precedes respiratory depression, so monitor closely, particularly in the opioid-naive patient. Begin with short-acting medications before adding a long-acting formulation (AGS, 2009). Sedation can occur during the inception of opioid treatment, yet tolerance generally develops to the effect. If sedation persists, consider rotating to another opioid. Methylphenidate can also be used to counteract sedation and can be given at doses of 5 to 10 mg in the morning and at 12:00 noon (Epocrates, 2016).

Most people develop a tolerance to all of the adverse effects, with the exception of constipation, which typically requires an aggressive bowel regimen with regular dosing of a laxative and stool softener and/or osmotic agent, and most

often should be started at the same time that the opioid is prescribed (AACN & COH, ELNEC Core, 2016). Methylnaltrexone is approved for severe opioid-induced constipation, and naloxone given orally has also demonstrated effectiveness (Economou, 2010). In addition, the FDA recently approved lubiprostone for treating opioid-induced constipation (International Foundation for Functional Gastrointestinal Disorders [IFFGD], 2013). Monitor bowel function closely to avoid obstipation, but if fecal impaction occurs, administer a lubricant, such as a glycerin suppository or mineral oil enema, and disimpact manually. Suppositories and enemas should be avoided in patients with neutropenia and thrombocytopenia. Because disimpaction can be a painful procedure, the patient may require premedication with an appropriate analgesic (Economou, 2010).

Clinicians should educate patients and families about potential risks and benefits when initiating treatment, including adopting a universal precautions approach to minimize abuse, addiction, diversion, as well as clarify myths and misconceptions about opioid therapy. Clinicians should educate patients and families regarding safe storage, use, and disposal of controlled substances, as well as caution with using alcohol or sedating OTC medications (AACN & COH, ELNEC APRN, 2016).

Adjuvant medications may improve pain control and can be added at any step of the WHO Step Ladder (WHO, 2011) (see Table 19-3). Tricyclic antidepressants may improve neuropathic pain. Adverse effects due to anticholinergic activity need to be considered. Nortriptyline and desipramine may be better tolerated in the elderly (Derby et al., 2010). Duloxetine, an inhibitor of norepinephrine and serotonin reuptake, is another option, indicated for diabetic neuropathy and chronic musculoskeletal pain, as well as for fibromyalgia (Epocrates, 2013). Anticonvulsants can be beneficial for lancinating neuropathic pain, which is often described as burning, shooting, and electrical. Gabapentin and pregabalin are commonly used (Paice et al., 2016). Topical analgesics, such as lidocaine 5% patch, can be beneficial for postherpetic neuropathy. Capsaicin cream has shown benefit in neuropathic and non-neuropathic pain syndromes; however, about 30% of people do not tolerate the adverse burning sensation when treatment is initiated (AGS, 2009). According to the ASCO special panel (as cited in Paice et al., 2016), the use of topical agents, such as anti-inflammatories, anesthetics, compounded creams or gels with baclofen, tricarboxylic acids (TCAs), ketamine, or other combinations, show evidence that benefits outweigh harms; evidence quality is intermediate, and strength of recommendation is moderate. Current evidence on the effectiveness of ketamine is insufficient to make a recommendation for routine clinical use (Paice et al., 2016).

Corticosteroids improve the pain of nerve or spinal cord compression. They also reduce edema and inflammation, resulting in improved pain control (Derby et al., 2010). Corticosteroids also may improve pain management related to bowel obstruction and headaches caused by increased intracranial pressure (AGS, 2009). Caution is advised due to the adverse effects of corticosteroids. ASCO (as cited in Paice et al., 2016) does not recommend the use of long-term corticosteroids in cancer survivors to relieve chronic pain. The panel also states clinicians can follow state-specific regulations to allow access to medical cannabis or cannabinoids for patients with chronic pain after consideration of the potential benefits and risks of available formulations (evidence-based benefits outweigh harms; evidence quality intermediate, strength of recommendation moderate) (AGS, 2009). According to ASCO Clinical Practice Guidelines (as cited in Paice et al., 2016), the use of cannabis offered modest analgesia with minimal adverse mild effects. Statistically significant improvement in pain was reported for nabilone, oral mucosal cannabis spray and extract, and smoked or vaped cannabis compared to placebo. Calcitonin may be beneficial for bone pain due to vertebral compression fracture, pelvic fractures, and pain due to bony metastatic disease (AGS, 2009). Baclofen, a muscle relaxant, can be used for spasticity due to central nervous system injury and neuromuscular disorders. Start with a low dose and increase slowly to minimize dizziness, sedation, and GI disorders. Baclofen should be tapered slowly to avoid potential delirium and seizure (AGS, 2009).

TABLE 19-3 Adjuvant Pain Medications

Tricyclic antidepressants: desipramine, nortriptyline, amitriptyline	Neuropathic pain
SNRI: duloxetine	Diabetic peripheral neuropathy, fibromyalgia, and musculoskeletal pain
Anticonvulsants: gabapentin, pregabalin	Neuropathic pain
Capsaicin topical	Neuropathic and non-neuropathic pain
Lidocaine topical	Postherpetic neuralgia
Corticosteroids	Pain due to nerve compression; anti-inflammatory
Cannabinoids	State-specific regulations

Nonpharmacological approaches include behavioral therapies such as relaxation, mindfulness, distraction, imagery, prayer, and cognitive reframing (AACN & COH, ELNEC Core, 2016). Physical measures include heat and/or cold application, repositioning, massage, and other complementary therapies such as acupuncture (Ferrell et al., ELNEC Core 2016). According to the ASCO (as cited in Paice et al., 2016) guidelines, on review of meta-analysis and randomized controlled trials, exercise and physical therapy showed small but significant impact on pain; acupuncture and massage were also helpful in improving pain (strength of recommendations weak; evidence quality low).

Radiation therapy is effective in the relief of pain related to tumor or masses. Radionucleotides also can be used for bone pain related to bone cancer. Chemotherapy, biological response modifiers, hormone therapy, and bisphosphonates all can reduce pain in patients with malignancies (AACN & COH, ELNEC APRN, 2016). Interventional procedures such as neuroablation, nerve blocks, celiac-plexus block, implantable drug delivery systems, vertebroplasty, and kyphoplasty can also help in the treatment of chronic pain (Paice et al., 2016).

Follow-Up: Perform ongoing assessment, reevaluation, and adjustment of plan of care as needed. Monitor for therapeutic effect, as well as adverse effects. The elderly are more susceptible to the adverse effects of opioids and other nonopioid

analgesics and adjuvant medications and, thus, require close follow-up (AGS, 2009).

Sequelae: Unrelieved pain is associated with functional impairment, falls, mood disorders (including depression and anxiety), sleep and appetite disturbances, and decreased socialization (AGS, 2009). Improved pain control improves function and quality of life. Potential negative outcomes of opioids can include sedation, respiratory depression, coma, and death. These adverse events can typically be avoided by choosing the appropriate medication for the patient, starting at a low dose, and titrating slowly to effect while monitoring for adverse effects. Addiction can occur and should be monitored for (AACN & CON, ELNEC APRN, 2016).

Prevention/Prophylaxis: Prevention/prophylaxis is dependent on etiology. Pain escalation may indicate advancing disease, particularly in the cancer patient; a new clinical finding; or tolerance to current opioid drug dose. Monitor with ongoing assessment. Anticipate painful procedures, such as wound care, and treat appropriately. Clinicians should incorporate a universal precautions approach to minimize abuse, addiction, and adverse consequences of opioid use, and should be cautious in co-prescribing other centrally acting drugs (AACN & COH, ELNEC APRN, 2016).

Referral: Refer the patient to a palliative care or pain management specialist for complicated pain syndromes or intolerance to common analgesics.

Education: Explain causes of pain syndromes, treatment strategies, and importance of regular follow-up for reassessment of pain and possible adverse effects. Educate patient and family about the risks of opioids and appropriate interventions for common adverse effects such as nausea and constipation. Advise patient, family, and staff that sedation precedes respiratory depression and to report excessive sedation promptly. Educate the patient and family on the risk also of drug diversion and stress safe-keeping of opioids. Educate patients and families on risks and benefits of nonopioid analgesics and the need to report to the clinician any changes regarding their pain management needs. Also instruct the patient and family on safe disposal of opioid medications once they are discontinued.

THE DYING PATIENT

Description: Death and dying are mostly considered medical events in today's society, but the growing acceptance of hospice and palliative care is creating a more natural dying experience. According to NHPCO Facts and Figures (2015), in 2011, 46% of deaths occurred under hospice care. Of the patients enrolled in hospice care, 58.9% died at home, including 35.7% in their private residence, 14.5% in a nursing home, and 8.7% in a residential care facility. Thirty-one percent of hospice patients died in a hospice inpatient facility and 9.3% died in an acute care hospital.

Etiology: Physiological changes of aging and multiple comorbidities influence the dying process.

Occurrence: Mortality rate is 100%. All people will die.

Age: Risk of death increases with advancing age.

Gender: No gender differences.

Ethnicity: The meaning and experience of death are often influenced by one's culture and ethnicity. Honor the patient's cultural beliefs, traditions, rights, and rituals (AACN & COH, ELNEC Core, 2016).

Contributing Factors: Advancing age, terminal illness, and comorbidities.

Signs and Symptoms: During the last hours to days of life, the most common symptoms include dyspnea, delirium, anxiety, noisy respiratory secretions, worsening pain, and nausea, all generally accompanied by functional decline (Bailey & Harman, 2016). Other symptoms observed in the actively dying are disorientation, agitation, restlessness, generalized weakness, drowsiness, sleeping more, decreased oral intake, dysphagia, fever, bowel changes, and incontinence. Though most people sleep more as death approaches with only brief periods of interactions, some experience a surge of energy. A dying person may also experience near-death awareness (AACN & COH, ELNEC Core, 2016).

Diagnostic Tests: Usually not appropriate in the dying patient.

Differential Diagnosis: Not applicable.

Treatment: Assess the dying person frequently for objective signs of distress, such as grimacing and moaning, and treat distressing symptoms as follows: morphine liquid concentrate for pain and/or shortness of breath/air hunger; oxygen may be beneficial if the person is hypoxic; lorazepam liquid concentrate for restlessness and anxiety; haloperidol liquid concentrate for agitation/delirium or nausea; atropine ophthalmic drops given orally, scopolamine patch, glycopyrulate, or hyoscyamine for upper airway secretions; acetaminophen suppository for fever; and haloperidol liquid concentrate or prochlorperazine suppository for nausea and vomiting. For seizures, lorazepam can be given subcutaneously or IV, and diazepam or phenobarbital rectally (AACN & COH, ELNEC Core, 2016; Quill et al., 2014). In the dying patient, glucocorticoids such as dexamethasone are often effective adjunctive medications for pain, anorexia, nausea, and asthenia (AACN & COH, ELNEC Core, 2016; Bailey & Harman, 2016; Quill et al., 2014). Focus on comfort measures such as oral care, skin care, continence care, and repositioning as appropriate; provide a calm environment (Bailey & Harman, 2016; Ferrell et al.; ELNEC Core, 2016). A copy of *ELNEC's Quick Reference Guide for Symptoms* is shown in Table 19-4 (AACN & COH, ELNEC Core, 2016).

Include the interdisciplinary team when addressing the spiritual, psychosocial, and emotional needs of the patient and the family. For most people, being "fully present" with the dying person is important. When death is inevitable, the dying person's attention shifts to the spiritual process of withdrawing from life. This process can create fear of abandonment, fear of the unknown, and fear of the dying process itself. Thus, it is essential for active involvement of the interdisciplinary team to support the patient and family through this process (AACN & COH, ELNEC APRN, 2016).

TABLE 19-4 Quick Reference Guide for Symptom Management

SYMPTOM	TREATMENT
Fatigue	• The most prevalent of symptoms reported in advanced disease • Rule out possible causative factors and evaluate which might be treatable given goals of care: anemia, iron deficiency, electrolyte imbalances, hypothyroidism, hypoxia, nutrition deficiencies, medications, anxiety/depression, sleep abnormalities • Exercise, physical therapy, occupational therapy • Assistive devices, caregiving support (hygiene, cleaning, meals) • Stimulants such as methylphenidate (Ritalin®) 2.5–0.5 mg PO QD or BID to start, then titrate prn • Dexamethasone (Decadron®) 2–8 mg PO QD, do not give in the evening • Mirtazapine (Remeron®) 15 mg PO QHS to enhance sleep, also improves appetite and mood
Insomnia/Sleep Disorders	• Evaluate sleep patterns current and prior to diagnosis • Suggest sleep hygiene measures: reduce caffeine in afternoon/evening, do not watch TV/tablets in bed, limit alcohol intake, cool room, warm bath before bed • Relaxation therapy such as mindfulness exercises, meditation, guided imagery • For some, pharmacological therapies ineffective if used daily • Zolpidem (Ambien®) 5–10 mg PO QHS; lower doses for women; safety concerns–sleep walking/eating • Mirtazapine (Remeron®) 15 mg PO QHS to enhance sleep, also improves appetite and mood • Buspirone (Buspar®) 5–20 mg PO TID • Trazodone (Desyrel®) 25–50 mg PO QHS • Avoid antihistamines for sleeping aid, especially in elderly or frail
Constipation	• Assess frequency, volume, consistency and normal patterns of bowel movements • Diarrhea may be due to impaction; rectal examination indicated • Goal ⊕ 3/week without straining, pain, tenesmus • Identify potential causative factors that can be addressed: opioids, anticholinergics, antihistamines, phenothiazines, tricyclic antidepressants, diuretics, iron, chemotherapy, ondansetron, antacids, dehydration, inactivity, hypercalcemia, hypokalemia, partial bowel obstruction, spinal cord compression, autonomic neuropathy, depression, anorexia, hypothyroidism • First evacuate bowel with bisacodyl 2–3 tabs PO QD or 10 mg suppository or Fleet's Enema→ (nothing per rectum if patient thrombocytopenic (<50,000 platelets) or neutropenic (ANC <500–1000); limit Fleet's and other sodium phosphate agents in renal dysfunction; if these are ineffective, give: • Methylnaltrexone (Relistor®) SQ (for opioid-induced constipation only); dosing is weight-based; contraindicated in obstruction
Constipation – Ongoing Prevention	• All patients on opioids should have an order for a stimulant laxative and softener • Add stimulant and softener combination (e.g., senna/docusate) and titrate to effect (max 8 tabs/day) • Increase with upward titration of opioid dose • If persistent, consider adding bisacodyl 2–3 tabs PO QD or 1 rectal suppository QD; lactulose 30–60 mL PO QD; metoclopramide (Reglan→) 10–20 mg PO QID; Milk of Magnesia 30 mL PO QD • When constipation is related to opioids or in debilitated patient, changing the diet or adding fiber supplements is rarely helpful • Educate patients/families; there is much stigma about discussing bowel function *Even when not eating, patients should have bowel movements every 1–2 days. Untreated constipation can lead to discomfort and increased pain, as well as agitation in the cognitively impaired patient.*
Diarrhea	• Evaluate for potential causes of diarrhea common in palliative care and correct/treat when feasible: medications (overuse of laxatives, antibiotics, magnesium, chemotherapy), infection, diet, herbal products (e.g., milk thistle, cayenne, ginger), fecal impaction, malabsorption syndromes from surgery or tumor, radiotherapy that includes abdomen in treatment field, inflammatory bowel disease, and other comorbid disorders • Loperamide (Imodium→) 2 mg PO; start with 4 mg, followed by 2 mg after each bowel movement, not to exceed 8 capsules/24 hours • Diphenoxylate/atropine (Lomotil→) 1–2 tabs PO QID, maximum 8 per 24 hours • Tincture of opium: 0.6 mL PO q 4–6 hours prn • Methylcellulose (e.g., Metamucil→) or pectin can help provide bulk to liquid stools • Octreotide (Sandostatin→) 50 mcg SQ/IV q 8 hours, maximum 1,500 mcg/day • Cholestyramine: 2–4 g PO/day before meals (especially for *C. difficile* diarrhea) • Pancrelipase (Creon→, Pancreaze→) 500–2,500 lipase units/kg PO with meals
Dyspnea (Shortness of Breath; Air Hunger)	• Identify and treat reversible causes: airway obstruction (e.g., bronchodilators and/or corticosteroids), infection (e.g. antibiotics), CHF or fluid overload (e.g., diuretics), anxiety (e.g., anxiolytics) • Opioids are first-line therapy; start with morphine 2.5–0.5 mg PO every hour (any opioid can be used); titrate upward aggressively • Liquids may be easier to swallow or can be placed sublingually (although absorbed enterally): morphine liquid; oxycodone liquid • Parenteral (IV or SQ) opioids – morphine or hydromorphone; Dilaudid→ can be used if patient unable to swallow • Add anxiolytics (benzodiazepines) if anxiety is present (e.g., lorazepam every 4 hours as needed) • Elevate head of bed (can use a fan for comfort) • Consider oxygen only if patient is hypoxemic

TABLE 19-4 Quick Reference Guide for Symptom Management—cont'd

SYMPTOM	TREATMENT
Anorexia	• Educate and counsel patient/family regarding anorexia as a natural response to disease; interventions below only when loss of appetite bothersome to patient • Environmental alterations: small, frequent meals; moist foods or those with sauce/gravy take less energy to eat; assistance with meal preparation to improve energy for eating • Megestrol acetate (Megace→) 400 mg PO QD; limit if concern regarding deep vein thrombosis, particularly in cancer patients • Dexamethasone (Decadron®) 4 mg PO QD or prednisone 20 mg PO QD, especially when prognosis <6 weeks • Dronabinol (Marinol®) 2–10 mg PO every 4 hours, use with caution in the older adult • Mirtazapine (Remeron®) 15 mg PO QHS to enhance sleep, also improves appetite
Nausea and Vomiting *Not intended to prevent or treat chemotherapy-induced nausea and vomiting*	• Rule out potentially reversible causes: constipation, central nervous system disease, pain, altered electrolytes, ↑ICP, obstruction, antibiotics, chemotherapy, radiation therapy, opioids, digoxin *If nausea and vomiting due to activation of chemoreceptor trigger zone (CTZ) (e.g., medication-induced):* • Prochlorperazine (Compazine→) 10 mg PO q 6 hours or 25 mg PR q 8 hours • Haloperidol (Haldol→) 0.5–0.4 mg PO or IV/SQ q 6 hours • Ondansetron (Zofran→) 4–8 mg PO or IV q 8 hours (best when used for chemotherapy or RT-induced nausea/vomiting; less effective when treating opioid-induced nausea and vomiting) • Olanzapine (Zyprexa®) 2.5–10 mg PO QD–BID • Promethazine (Phenergan®) 12.5–25 mg IV q 6 hours or 25 mg PO or PR q 6 hours *If nausea and vomiting due to gastric stasis causing early satiety, GI tract spasm:* • Metoclopramide (Reglan→) 10–20 mg PO or IV TID AC & HS (not with bowel obstruction) • Hyoscyamine (Levsin®) 0.125–0.25 mg PO/SL q 4 hours prn *If nausea and vomiting due to vestibular effects (nausea exacerbated by movement):* • Scopolamine transdermal patch 1.5 mg q 3 days (especially if underlying mechanism is vestibular; increased nausea or dizziness with ambulation) • Cyclizine (Meclizine®) 25–50 mg PO every 8 hours; best for motion sickness or increased intracranial pressure *If mechanism of nausea and vomiting is unclear, or unresponsive to other therapies:* • Dexamethasone (Decadron®) 4–8 mg PO/IV daily • Dronabinol (Marinol®) 2–10 mg PO every 4 hours *Administer antiemetics around the clock (scheduled). If nausea is controlled, then try reducing after 2–3 days.*
Pain in the Final Hours of Life	• Observe for escalating pain and increase medications accordingly • May need to change route if swallowing is diminished; alternatives include transdermal, concentrated liquids taken orally in small volumes, parenteral • Abruptly discontinuing opioids or benzodiazepines may precipitate withdrawal syndrome; reduce dose 25% daily if no sign of pain in comatose patient; return to previous dose if any sign of return of pain • Myoclonus may occur; treat with Clonazepam (Klonopin®) 0.5 mg PO TID, MAX 20 mg/day or Lorazepam (Ativan®) 0.5–2.0 mg PO/IV q 4 hours if patient unable to swallow; may require Midazolam (Versed®); IV/SQ; rotate opioids
Delirium and Agitation	• Identify and treat reversible causes: full bladder, fecal impaction, pain, dyspnea (hypoxemia, secretions, pulmonary edema), severe anxiety, nausea, pruritus, medications (e.g., corticosteroids, neuroleptics, anticholinergics), dehydration, infection • Haloperidol (Haldol→) 0.5–0.4 mg PO or IV/SQ q 6 hours (may repeat q 1 hour PRN in severe delirium) • Lorazepam (Ativan→) 0.5–0.2 mg PO/SL/IV q 4 hours PRN, then schedule ATC once effective dose is determined (not recommended as SQ) • Olanzapine (Zyprexa→) 2.5–20 mg PO QHS or Zyprexa (Zydis→) (orally disintegrating tablet) 5–20 mg PO QHS • Risperidone (Risperdal®) 0.5 mg PO q PM, increase by 0.25–0.5 mg q 2–7 days • Quetiapine (Seroquel®) 12.5–100 mg PO q 12–24 hours • Chlorpromazine (Thorazine→) 12.5–25 mg PO/SQ q 4–12 hours, or 25 mg pr q 4–12 hours (IV can cause hypotension; avoid unless other agents ineffective and oral/rectal route unavailable)
Excessive Secretions ("Death Rattle")	• Atropine 0.4 mg SQ q 15 minutes PRN • Scopolamine transdermal patch 1.5 mg TOP, start with 1 mg (about 4-hour onset), increase to 2 mg after 24 hours. If insufficient, begin scopolamine 50 mcg/hour IV or SQ; double every hour to maximum of 200 mcg/hour • Glycopyrrolate (Robinul→) 1–2 mg PO or 0.1 mg–0.2 mg IV/SQ q 4 hours PRN or 0.4–1.2 mg/day continuous IV/SQ infusion (this agent does not cross the blood brain barrier; less likely to cause confusion) • Hyoscyamine (Levsin®) 0.125–0.25 mg PO q 4 hours (liquid can be placed sublingually) • Change patient's position • D/C IV and/or enteral fluids as they may increase discomfort (e.g., cough, pulmonary congestion, sensations of choking/drowning, vomiting, edema, pleural effusions, ascites) • If fluids not discontinued, IV or SQ rate ought not exceed 500 mL/24 hours • Furosemide (Lasix→) PRN to control overhydration. • Control thirst by moistening lips and mouth with substitute saliva (Oral Balance Moisture Gel→ or Salivart→, at bedside; apply as frequently as needed) *Patients may be too weak to expectorate. This is not painful, but distressing to family. Suctioning is traumatic, can cause bleeding, and is painful. Do not suction beyond the oral cavity.*

From City of Hope Pain/Palliative Resource Center Index, http://prc.coh.org. Permission granted, City of Hope, September 2016 (AACN & COH, 2016).

Utilization of hospice services for the terminally ill has a beneficial impact on bereavement-related morbidity and mortality (Shear et al., 2015).

Follow-Up: Ensure 24-hour access to care for the dying patient and his or her family. Provide bereavement care for the family.

Sequelae: Death of the patient; bereavement follow-up for the family. Ensure adequate symptom management and family support during the terminal phase.

Prevention/Prophylaxis: An important consideration for dying persons is the ability to communicate their end-of-life wishes to their family and to their medical team. As discussed in *Dying in America: Improving Quality and Honoring Individual Preferences Near End of Life* (2015), effective communication and advance care planning are essential. Too often these discussions are held when death is imminent. Rather, these discussions should occur at a time in the person's life when he or she can clearly express his or her essential wishes (Bailey & Harman, 2016). A useful tool for delivering bad news and discussing goals of care is Buckman's S-P-I-K-E-S protocol, with its focus on Setting, Perception, Invitation, Knowledge, Empathy, and Strategy/summary (Baile et al., 2000; Buckman, 1992, 2005). Before delivering bad news, give a warning that bad news is coming, and respond to emotions. It is also important to summarize the meeting and agreed-on goals of care (Buckman, 2005).

Ensuring a private setting for the discussion of goals of care is important, as is inviting patients and family members to participate. Before discussing the plan of care, assess what the patient already understands about his or her condition and how much detail the patient would like to know about his or her illness and prognosis. Answer questions as clearly as possible, avoiding medical jargon. Explore goals of care. Ask about patient/family concerns/worries/fears. Ask about unacceptable states; that is, loss of critical functioning that a patient would want to avoid, such as mechanical ventilation (Blinderman & Billings, 2015).

"I am worried" conveys the seriousness of the situation, as well as provides support and allows for the possibilities of hope (Guwande, 2014). Dr. Guwande follows the advice of Dr. Susan Block when having these difficult conversations: "If time becomes short, what is most important to you?" (Guwande, 2014, p. 182). Back and colleagues (2009) recommend the Ask-Tell-Ask approach: "Ask" what the person/family understands about the current situation; "tell": provide an explanation considering their knowledge level and information preferences; then, with this new information, "ask" what they now understand about their condition and goals.

Referral: Timely referral palliative care and hospice care.

Education: Teach the patient's family the signs of impending death, and ask them to notify the medical team of distressing symptoms. Educate on comfort measures such as oral care, repositioning, and incontinence care. Prepare the family for the patient's possible near-death experience. Educate the patient's family on the signs of death and care at the time of death. Educate on bereavement resources (AACN & COH, ELNEC Core, 2016).

GRIEF AND BEREAVEMENT

Description: Bereavement is the process of experiencing the death of a loved one and adjusting to a world without the deceased. Bereavement includes grief, the normal response to a loss, and mourning, the outward, social expression of the loss, a process by which people adapt to the loss (AACN & COH, ELNEC Core, 2016; Quill et al., 2014; Shear et al., 2015). Grief and bereavement are affected by the circumstances at the time of death (Quill et al., 2014). There are three different types of grief: anticipatory, normal, and complicated grief (AACN & COH, ELNEC Core, 2016; Quill et al., 2014; Shear et al., 2015). Anticipatory grief is experienced before the death and can be experienced by everyone involved, including the patient. Normal grief encompasses the typical emotional, physical, cognitive, and spiritual reactions to a loss. Common grief reactions include somatic symptoms; sleep and appetite disturbances; memory loss and impaired concentration; social withdrawal and disinterest in prior activities; a sense of the presence of the deceased person, and auditory or visual hallucinations; questioning of spiritual and religious beliefs; and emotional reactions, which may include relief, numbness, helplessness, self-reproach, sadness, guilt, and despair.

Complicated grief is prolonged, intense, and disabling, with troubling thoughts, dysfunctional behaviors, dysregulated emotions, and/or serious psychosocial problems impeding adaptation to the loss (Shear et al., 2015). Complicated grief includes chronic, delayed, exaggerated, and masked grief (AACN & COH, ELNEC, Core 2016). In chronic grief, grief reactions continue over a prolonged period of time. Delayed grief occurs when the normal grief reaction is suppressed or postponed, with the survivor avoiding the pain of loss either consciously or unconsciously. Exaggerated grief consists of intense reactions such as nightmares, delinquent behavior, phobias, and suicidal thoughts. Masked grief occurs when the survivor is unaware that behaviors interfering with normal functioning are the result of loss.

An additional type of grief is disenfranchised grief, grief that occurs when a loss in not socially recognized (AACN & COH, ELNEC Core, 2016). Examples include partners of HIV/AIDS patients, ex-spouses and ex-partners, children experiencing the death of a stepparent, and women and their husband/partner experiencing a terminated pregnancy or stillbirth.

Etiology: Death of a loved one as experienced by family and caregivers.

Occurrence: Loss of relationships and role identity culminating in death, the ultimate loss experience. Complicated grief occurs in 10% to 20% of survivors (Quill et al., 2014).

Age: Affects people of all ages. Consider multiple cumulative losses in the elderly. Consider developmental stages of children in bereavement care.

Gender: Men and women express grief differently, with women typically more expressive and men more restrained (California State University, Fullerton, www.Fullerton.edu, accessed 8/21/16).

Ethnicity: Grief is influenced by one's culture, ethnicity, and religion (AACN & COH, ELNEC Core, 2016).

Contributing Factors: Contributing factors for complicated grief include a sudden or untimely death, a violent death, death of a child, multiple losses, difficult terminal illness and/or lack of social support, pre-existing mental health issues, or substance abuse (AACN & COH, ELNEC Core, 2016; Quill et al., 2014).

Signs and Symptoms: Normal grieving includes physical, emotional, cognitive, and behavioral responses. Common grief reactions include somatic symptoms; sleep and appetite disturbances; memory loss and impaired concentration; social withdrawal and disinterest in prior activities; a sense of the presence of the deceased person, and auditory or visual hallucinations; questioning of spiritual and religious beliefs; and emotional reactions, which may include relief, numbness, helplessness, self-reproach, sadness, guilt, and despair (AACN & COH, ELNEC, Core 2016; Quill et al., 2014; Shear et al., 2015).

Complicated grief typically includes intense shock, anger and denial, feelings of numbness or hopelessness, a blunted emotional expression and anxiety, panic, and chronic depression, as well as a risk for self-harm, such as suicide or substance abuse (AACN & COH, ELNEC Core, 2016). Those with complicated grief often have ruminating thoughts, excessive avoidance of reminders of the loss, and difficulty regulating emotion. Symptoms often persist for at least 6 months and interfere with functioning (Shear et al., 2015).

Diagnostic Tests: None usually; screening for depression with patient health questionnaire (PHQ-9) may be indicated.

Differential Diagnosis: Depression, including risk of suicide.

Treatment: William Worden (2008) describes the four tasks of mourning: first, to accept the reality of the loss; second, to process the pain of grief; third, to adjust to the world without the deceased; and fourth, to find an enduring connection with the deceased in the midst of embarking on a new life. The goal is to facilitate the bereaved actualization of the loss and facilitate living without the deceased. "Grief work is never completely finished. Healing occurs when the pain is less" (AACN & COH, ELNEC Core, Module 7, slide 26, 2016).

Interventions to support grief work include (AACN & COH, ELNEC Core, 2016; Quill et al., 2014):

- Support grieving as a normal experience. Being present and actively listening are essential. Allow the person to tell his or her story.
- Assist with identifying support systems such as extended family, friends, and support groups, such as local hospice bereavement support groups.
- Provide emotional and spiritual support. Consider referral for bereavement support groups and pastoral counseling. Provide written materials about grief. Encourage expression of feelings, and encourage reminiscence and life review. Journal writing, letter writing, and drawing pictures can be beneficial.
- For complicated grief, refer to a bereavement specialist, health-care provider, and/or psychologist/psychiatrist.
- Caregivers can support the bereaved by sending a note, attending the funeral or memorial service, or making a supportive phone call.
- Staff bereavement may include encouraging staff to express their grief, reviewing deaths and their effects on staff, enabling staff to attend memorial services, creating staff mourning rituals, and encouraging self-care.

Follow-Up: Bereavement is associated with increased morbidity and mortality (see Sequelae). Ensure regular health-care appointments with the primary care provider for appropriate assessment, screening, and interventions.

Sequelae: Cardiovascular and cancer deaths are high in the bereaved, as are deaths due to alcohol and suicide. Psychiatric disorders including depression, anxiety, and post-traumatic stress disorder. Substance abuse disorders are common as well (Shear et al., 2015). Screen and treat appropriately.

Prevention: Grief assessment begins at the time of a patient's admission to a hospital, long-term care facility, or hospice, as well as at the time of diagnosis of an acute, chronic, or terminal illness. Assess grief frequently during the bereavement period (AACN & COH, ELNEC Core, 2016). Allow expression of grief through appropriate support.

Referral: Refer surviving family and friends to bereavement support groups, grief counseling, and psychiatric care, if indicated. Follow-up with the health-care provider is essential as well.

Education: Instruct on normal grief reactions, bereavement resources in the community, and awareness of signs of complicated grief and depression.

CASE STUDY

Mrs. M. is an 85-year-old woman with breast cancer with metastasis to the lungs and bone. She is receiving palliative chemotherapy. Her husband died 5 years ago, and she is currently living with her daughter, who works outside the home. A private caregiver has been hired through a community agency. Mrs. M. enjoys visits from her family and fellow church members. Mrs. M. is seen by the nurse practitioner when she comes to her follow-up appointment in the oncology clinic. She is experiencing moderate-to-severe right chest wall pain. She also is

Continued

CASE STUDY—cont'd

experiencing dyspnea on exertion, nausea, constipation, and fatigue. A recent CT scan revealed disease progression on third-line chemotherapy.

Her past medical history includes metastatic breast cancer, hypertension, and hypothyroidism. Current medications: hydrochlorothiazide 12.5 mg PO daily and levothyroxine 50 mcg PO daily; and senna PO bid for constipation/APAP. She has no known allergies.

Exploring her symptoms: She rates her right chest wall pain at 7/10. It is a dull, aching pain and worsens with activity. The pain has been present for 2 weeks. Hydrocodone/APAP had initially helped, but is no longer effective. Because of the pain and dyspnea, she spends most of her time sitting in a reclining chair or lying in bed. Her appetite has decreased. She has not moved her bowels in 2 days. She complains of anorexia, fatigue, and insomnia.

Focused physical examination: temperature 98.2°F, heart rate (HR) 75 beats per minute, respiratory rate (RR) 22 breaths per minute, blood pressure (BP) 120/80 mm Hg, oxygen saturation 94%, weight 105 pounds, height 5 feet 4 inches, body mass index (BMI) 18 kg/m^2.

Mrs. M. is in moderate distress. She appears thin and frail. Her color is sallow, and her heart sounds reveal regular rate and rhythm, no murmur, rub, or gallop. Her lungs have bibasilar crackles, greater on the right, no wheezing. Her abdomen is soft with active bowel sounds, nontender to palpation.

What would you recommend for her symptom management?

appendix A

Physiological Influences of the Aging Process

AGE-RELATED CHANGE	APPEARANCE OR FUNCTIONAL CHANGE	IMPLICATION
	Integumentary System	
Loss of dermal and epidermal thickness	Paper-thin skin	Prone to skin tears
Flattening of papillae	Shearing and friction force more readily peels off the epidermis	Prone to skin breakdown and injury
	Diminished cell-mediated immunity in the skin	
Atrophy of the sebaceous glands	Decreased production of oil and cerumen	Frequent pruritus and xerosis
Atrophy of the eccrine glands	Decreased sweating ability	Impaired thermoregulation
Decreased vascularity	Slower recruitment of sweat glands by thermal stimulation	Alteration in thermoregularity response; diminished ability to adapt to temperature changes
		Fluid requirements may change seasonally
	Decreased body odor	Loss of skin water
	Decreased heat loss	Increased risk of heat stroke
	Dryness	
Collagen cross-linking	Increased wrinkling	Potential effect on one's morale and feeling of self-worth
Elastin regression	Laxity of skin	
Loss of subcutaneous fat	Intraosseous atrophy, especially to the back of hands and to the face	Loss of fat tissue on soles of feet—trauma of walking increases foot problems
Decreased elasticity		Difficulty assessing skin turgor
Loss of subcutaneous tissue	Purpuric patches after minor surgery	Reduced insulation against cold temperatures; prone to hypothermia
		Check why injury is occurring; be alert for potential abuse or falls
Decreased number of melanocytes	Loss of pigment	Teach the importance of using sun block creams; refer to a dermatologist as needed
	Pigment plaque appears	
Decreased turnover rate of keratinocytes	Increased exposure of the epidermal cells to the environment to include UV radiation	Increased risk of nonmalignant skin cancers and malignant melanoma
Decline in fibroblast proliferation	Decreased epidermal growth rate	Decreased tissue repair response
	Slower re-epithelialization	
	Decreased vitamin D production and synthesis	Increased risk for developing osteoporosis and other conditions associated with vitamin D deficiency
Decreased hair follicle density	Loss of body hair	

Continued

AGE-RELATED CHANGE	APPEARANCE OR FUNCTIONAL CHANGE	IMPLICATION
Decreased growth phase of individual fibers	Thin, short villus hairs predominate	
	Slower hair growth	
Loss of melanocytes from the hair bulb	Graying of the hair	Potential effect on self-esteem
Alternating hyperplasia and hypoplasia of nail matrix	Longitudinal ridges	Nails prone to splitting
	Thinner nails of the fingers	Advise patient to wear gloves, keep nails short, and avoid nail polish remover (causes dryness); refer patient to podiatrist
	Thickened, curled toenails or claw-like nails known as onychogryphosis	May cause discomfort
Respiratory System		
Decreased lung tissue elasticity	Decreased vital capacity	Reduced overall efficiency of ventilatory exchange
	Increased residual volume	
	Decreased maximum breath capacity	
Thoracic wall calcification	Increased anteroposterior diameter of chest	Obscuration of heart and lung sounds
	Displacement of apical impulse	
Cilia atrophy	Change in mucociliary transport; mucous-producing cells increase	Increased susceptibility to infection
Decreased respiratory muscle strength	Reduced ability to handle secretions and reduced effectiveness against noxious foreign particles	Prone to atelectasis
	Partial inflation of lungs at rest	
Less sensitivity to hypoxia; impaired ability to recognize bronchoconstriction	Increased respiratory distress	Increased risk of mortality from acute respiratory conditions
Cardiovascular System		
Heart valves fibrose and thicken	Reduced stroke volume; cardiac output may be altered	Decreased responsiveness to stress; heart rate and blood pressure take longer to return to normal resting rate following exertion
	Slight left ventricular hypertrophy	Increased incidence of murmurs, particularly aortic stenosis and mitral regurgitation
Mucoid degeneration of mitral valve	S_4 sound commonly heard	
	Valve less dense; mitral leaflet stretches with intrathoracic pressure	
Fibroelastic thickening of the sinoatrial node; decreased number of pacemaker cells	Slower heart rate	Increased prevalence of arrhythmias and extra heart beats become more common
	Irregular heart rate	
Increased subpericardial fat		
Collagen accumulation around heart muscle		
Elongation of tortuosity and calcification of arteries	Increased rigidity of arterial wall	Aneurysms may form
Elastin and collagen cause progressive thickening and loss of arterial wall resiliency	Increased peripheral vascular resistance	Decreased blood flow to body organs
		Altered distribution of blood flow
Loss of elasticity of the aorta dilation		Increased systolic blood pressure, contributing to coronary artery disease
Increased lipid content in artery wall	Lipid deposits form	Increased incidence of atherosclerotic events such as angina pectoris, stroke, gangrene
Decreased baroreceptor sensitivity (stretch receptors)	Decreased sensitivity to change in blood pressure	Prone to loss of balance—potential for falls
	Decreased baroreceptor mediation to straining	Valsalva maneuver may cause sudden drop in blood pressure, orthostatic hypotension, and dizziness when the patient changes from a lying or sitting position to standing
Gastrointestinal System		
Liver becomes smaller	Decreased storage capacity; decreased efficiency in metabolizing drugs that pass through the liver	

AGE-RELATED CHANGE	APPEARANCE OR FUNCTIONAL CHANGE	IMPLICATION
Less efficient cholesterol stabilization absorption	Increased evidence of gallstones	
Atrophy of muscles and bones of the jaw	Difficulty with mastication	Ability to thoroughly chew food is impaired and can contribute to dysphagia with solid foods
Dental enamel thins	Staining of tooth surface occurs	Tooth and gum decay; tooth loss
Gums recede	Teeth deprived of nutrients	
Fibrosis and atrophy of salivary glands	Prone to dry mucous membranes	Shift to mouth breathing is common; frequent complaints of dry mouth are expressed
	Decreased salivary ptyalin	Membrane more susceptible to injury and infection
		May interfere with breakdown of starches
Atrophy and decrease in number of taste buds	Decreased taste sensation	Altered ability to taste sweet, sour, and bitter
		Change in nutritional intake
		Excessive seasoning of foods
Delay in esophageal emptying	Decline in esophageal peristalsis	Occasional discomfort as food stays in esophagus longer
	Stiffening of the esophageal wall	
Decreased hydrochloric acid secretion	Reduction in amount of iron and vitamin B_{12} that can be absorbed	Possible delay in vitamin and drug absorption, especially calcium and iron
Decrease in gastric acid secretion		Altered drug effect fewer cases of gastric ulcers
Decreased muscle tone	Altered motility	Prone to constipation, functional bowel syndrome, esophageal spasm, diverticular disease
	Decreased colonic peristalsis	
Atrophy of mucosal lining	Decreased hunger sensations and emptying time	
Decreased proportion of dietary calcium absorbed	Altered bone formation, muscle contractility, hormone activity, enzyme activation, clotting time, immune response	Symptoms more marked in women than in men
Decreased basal metabolic rate (rate at which fuel is converted into energy)		May need fewer calories
		Possible effect on life span
	Genitourinary and Reproductive Systems	
Reduced renal mass	Decreased sodium-conserving ability	Administration and dosage of drugs may need to be modified
Loss of glomeruli	Decreased glomerular filtration rate	
	Decreased creatinine clearance	
	Increased blood urea nitrogen concentration	
Histological changes in small vessel walls	Decreased renal blood flow	
Sclerosis of supportive circulatory system		
Decline in number of functioning nephrons	Decreased ability to dilute urine concentrate	Altered response to reduced fluid load or increased fluid volume
Reduced bladder muscular tone	Decreased bladder capacity or increased residual urine	Sensation of urge to urinate may not occur until bladder is full
Atrophy and fibrosis of cervical and uterine walls	Menopause; decline in fertility	Urination at night may increase
Reduced number and viability of oocytes in the aging ovary	Narrowing of cervical canal	
Decreased vaginal wall elasticity	Vaginal lining thin, pale, friable	Potential for discomfort in sexual intercourse
	Narrowing of vaginal canal	
Decreased levels of circulating hormones	Reduced lubrication during arousal state	Increased frequency of sexual dysfunction
Degeneration of seminiferous tubules	Decreased seminal fluid volume	
	Decreased force of ejaculation	
	Reduced elevation of testes	

Continued

AGE-RELATED CHANGE	APPEARANCE OR FUNCTIONAL CHANGE	IMPLICATION
Proliferation of stromal and glandular tissue	Prostatic hypertrophy	Potentially compromised genitourinary function; urinary frequency and increased risk of malignancy
Involution of mammary gland tissue	Connective tissue replaced by adipose tissue	Easier to assess breast lesions
	Neuromuscular System	
Decreased muscle mass	Decreased muscle strength	Increased muscle cramping
	Tendons shrink and sclerose	Decreased tendon jerks
Decreased myosin adenosine triphosphatase activity	Prolonged contraction time, latency period, relaxation period	Decreased motor function and overall strength
Deterioration of joint cartilage	Bone makes contact with bone	Potential for pain, crepitation, and limitation of movement
Loss of water from the cartilage	Narrowing of joint spaces	Loss of height
Decreased bone mass	Decreased bone formation and increased bone resorption, leading to osteoporosis	More rapid and earlier changes in women
Decreased osteoblastic activity		Greater risk of fractures
Osteoclasts resorb bone	Hormonal changes	Gait and posture accommodate to changes
Increased proportion of body fat	Centripetal distribution of fat and invasion of fat in large muscle groups	Anthropometric measurements required
Regional changes in fat distribution		Increased relative adiposity
Thickened leptomeninges in spinal cord	Loss of anterior horn cells in the lumbosacral area	Leg weakness may be correlated
Accumulation of lipofuscin	Altered RNA function and resultant cell death	
Loss of neurons and nerve fibers	Decreased processing speed and vibration sense	Increased time to perform and learn
	Altered pain response	Possible postural hypotension
	Decreased deep tendon, Achilles tendon	Safety hazard
Decreased conduction of nerve fibers	Decreased psychomotor performance	Alteration in pain response
Few neuritic plaques		Possible cognitive and memory changes
Neurofibrillary tangles in hippocampal neurons		Heavy tangle formation and neuritic plaques in cortex of patients with Alzheimer's disease
Changes in sleep-wake cycle	Decreased stage 4, stage 3, and rapid eye movement phases	Increased or decreased time spent sleeping
	Deterioration of circadian organization	Increased nighttime awakenings
		Changed hormonal activity
Slower stimulus identification and registration	Delayed reaction time	Prone to falls
Decreased brain weight and volume		May be present in absence of mental impairments
	Sensory System	
Morphological changes in choroid, epithelium, retina	Decreased visual acuity	Corrective lenses required
	Visual field narrows	Increased possibility of disorientation and social isolation
Decreased rod and cone function		Slower light and dark adaptation
Pigment accumulation		
Decreased speed of eye movements	Difficulty in gazing upward and maintaining convergence	
Sclerosis of pupil sphincter	Difficulty in adapting to lighting changes	Glare may pose an environmental hazard
	Increased threshold for light perception	Dark rooms may be hazardous
Increased intraocular pressure	Increased incidence of glaucoma	
Distorted depth perception		Incorrect assessment of height of curbs and steps; potential for falls
Ciliary muscle atrophy	Altered refractive powers	Corrective lenses often required
Nuclear sclerosis (lens)	Presbyopia	Near work and reading may become difficult

AGE-RELATED CHANGE	APPEARANCE OR FUNCTIONAL CHANGE	IMPLICATION
Reduced accommodation	Hyperopia	
Increased lens size	Myopia	
Accumulation of lens fibers		
Lens yellows	Color vision may be impaired	Less able to differentiate low color tones: blues, greens, violets
Diminished tear secretion	Dullness and dryness of the eyes	Irritation and discomfort may result
		Intactness of corneal surface jeopardized
Loss of auditory neurons	Decreased tone discrimination and voice localization	Suspiciousness may be increased because of paranoid dimensions secondary to hearing loss
	High-frequency sounds lost first	Social isolation
Angiosclerosis calcification of inner ear membrane	Progressive hearing loss, especially at high frequency	Difficulty hearing, particularly under certain conditions such as background noise, rapid speech, poor acoustics
	Presbycusis	
Decreased number of olfactory nerve fibers	Decreased sensitivity to odors	May not detect harmful odors
		Potential safety hazard
Alteration in taste sensation		Possible changes in food preferences and eating patterns
Reduced tactile sensation	Decreased ability to sense pressure, pain, temperature	Misperceptions of environment and safety risk
Endocrine System		
Decline in secretion of testosterone, growth hormone, insulin, adrenal androgens, aldosterone, thyroid hormone	Decreased hormone clearance rates	Increased mortality associated with certain stresses (burns, surgery); increased prevalence of hormonal disease
Defects in thermoregulation	Shivering less intense	Susceptibility to temperature extremes (hypothermia/ hyperthermia)
Reduction of febrile responses	Poor perceptions of changes in ambient temperature	Unrecognized infectious process operative
	Reduced sweating; increased threshold for the onset of sweating	
	Fever not always present with infectious process	
Alteration in tissue sensitivity to hormones	Decreased insulin response, glucose tolerance, and sensitivity of renal tubules to antidiuretic hormone	
Enhanced sympathetic responsivity		
Increased nodularity and fibrosis of thyroid		Increased frequency of thyroid disease
Decreased basal metabolic rate	Alteration in carbohydrate tolerance	Increased incidence of obesity
Hematological System		
Decreased percentage of marrow space occupied by hematopoietic tissue	Ineffective erythropoiesis	Risky for patients who lose blood
Immune System		
Thymic involution and decreased serum thymic hormone activity	Decreased number of T cells	Less vigorous and/or delayed hypersensitivity reactions
	Production of anti–self-reactive T cells	
Decreased T-cell function	Impairment in cell-mediated immune responses	Increased risk of mortality
Appearance of autoantibodies	Decreased cyclic adenosine monophosphate and glucose monophosphate	Increased incidence of infection
	Decreased ability to reject foreign tissue	Reactivation of latent infectious diseases
	Increased laboratory autoimmune parameters	Increased prevalence of autoimmune disorders
Redistribution of lymphocytes	Impaired immune reactivity	
Changes in serum immunoglobulin	Increased immunoglobulin A levels	Increased prevalence of infection
	Decreased immunoglobulin G levels	

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appendix B

Laboratory Values in the Older Adult

LABORATORY TEST	NORMAL VALUES	CHANGES WITH AGE	COMMENTS
		Urinalysis	
Protein	0–5 mg/100 mL	Rises Slightly	May be due to kidney changes with age, urinary tract infection, or renal pathology
Glucose	0–15 mg/100 mL	Declines Slightly	Glycosuria appears after high plasma level; unreliable
Specific gravity	1.005–1.020	Lower Maximum In Elderly, 1.016–1.022	Decline in nephrons impairs ability to concentrate urine
		Hematology	
Erythrocyte sedimentation rate	Male: 0–20 mm/hour Female: 0–30 mm/hour	Slight increase in normal aging. Elevated in inflammatory conditions. May be decreased during heart failure	Neither sensitive nor specific in the aged
Iron	50–160 mcg/dL	Slight decrease	
Iron binding	230–410 mcg/dL	Decrease	
Hemoglobin	Male: 13 gm/dL Female: 11.0 gm/dL	Male: 11.5 gm/dL Female: 11.0 gm/dL	Anemia common in the elderly
Hematocrit	Male: 40%–54% Female: 36%–46%	Slight decrease speculated	Decrease in anemias, multiple myeloma, protein malnutrition, CDK, rheumatoid arthritis, increased dehydration, severe diarrhea
Leukocytes	4,300–10,800/mm^3	Drop to 3,100–9,000/mm^3	Decrease may be due to drugs or sepsis and should not be attributed immediately to age
Lymphocytes	500–2,400 T cells/mm^3 50–200 B cells/mm^3	T-cell and B-cell levels fall	Risk of infection is higher; immunization is encouraged
Platelets	150,000–350,000/mm^3	No change in number	
		Blood Chemistry	
Albumin	3.5–5.0 gm/dL	Decline	Related to decrease in liver size and enzymes; protein-energy malnutrition common
Globulin	2.3–3.5 g/100 mL	Slight increase	
Total serum protein	6.0–8.4 g/100 mL	No change in number	Decreases may indicate malnutrition, infection, liver disease
Blood urea nitrogen	Men: 10–25 mg/100 mL Women: 8–20 mg/100 mL	Increases significantly up to 69 mg/100 mL BUN may be decreased in patients with renal disease	Decline in glomerular filtration rate; decreased cardiac output All electrolytes need to be evaluated when a concern for renal disease exists
Creatinine	0.6–1.5 mg/100 mL	Increases to 1.9 mg/100 mL seen	Related to decreases in lean body mass Unreliable indicator of abnormalities in renal function, especially in the frail older adult and/or those with sarcopenia

Continued

LABORATORY TEST	NORMAL VALUES	CHANGES WITH AGE	COMMENTS
Creatinine clearance	104–124 mL/min	Decreases 10%/decade after 40 years of age	Decreased renal impairment, hyperthyroidism, thiazides, increased hypothyroidism
Glucose tolerance	62–110 mg/dL after fasting; <120 mg/dL after 2 hours postprandial	Slight increase of 10 mg/dL/ decade after 30 years of age	Diabetes increasingly prevalent; drugs may cause glucose intolerance
Triglycerides	40–150 mg/100 mL	20–200 mg/100 mL	Risk of coronary artery disease
Cholesterol	120–220 mg/100 mL	Males: increase to 50 mg/100 mL, then decrease Females: increase postmenopausally	Risk of cardiovascular disease
Thyroxine	4.5–13.5 mcg/dL	3.3–8.6 mcg/dL	Changes suggest thyroid disease; may be seen in euthyroid patients with acute or chronic illness or caloric deficiencies
Triiodothyronine	90–220 ng/dL	Decrease 25%	May impact metabolism, body temperature, or heart rate
Thyroid-stimulating hormone	0.5–5.0 mcg/mL	Slight increase	Sensitive indicator for diagnosing thyroid disease
Alkaline phosphatase	13–39 IU/L	Increase by 8–10 IU/L	Elevations (20% usually due to disease); elevations may be found with bone abnormalities, drugs (e.g., narcotics), and consumption of a fatty meal For best results, this test should be ordered during fasting

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