MINISTRY OF HEALTH

KENYA NATIONAL GUIDELINES FOR CARDIOVASCULAR DISEASES MANAGEMENT

DIVISION OF NON-COMMUNICABLE DISEASES
MINISTRY OF HEALTH

Republic of Kenya

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FOR CARDIOVASCULAR DISEASES
MANAGEMENT

Developed by the Division of Non-Communicable Diseases - Ministry of Health

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Foreword

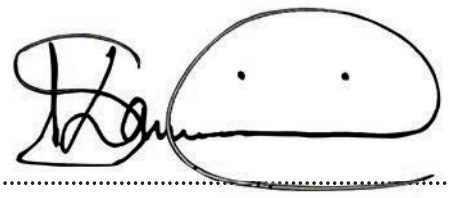
The Kenya Health Policy 2014-2030 aims at attaining the highest possible standard of health in a manner responsive to the health needs of our population. One of the major policy directions towards realizing the intentions of this policy is to halt and reverse the rising burden of non-communicable diseases.

Non communicable diseases (NCDs) are not just a serious setback to our social and economic development; they are a real threat to the integrity of our health system. Cardiovascular diseases (CVDs) in particular are a major public health concern with significant economic implications in terms of health care-needs, lost productivity and premature death as they are now affecting men and women at their most productive years necessary for national building, placing a heavy burden on our economy and reducing our economic growth.

These guidelines for the prevention and management of cardiovascular diseases are a critical ingredient for streamlining care across the entire health services provision continuum. They are a strategic component in achieving universal health coverage, securing affordable health care and improving the livelihood of all Kenyans which in turn will guarantee a healthy nation working towards sustainable development and prosperity.

These guidelines bring to the fore the need for availability of skilled human resource, sustained adequate funding and partnership building at all levels of governance. It provides clear roles for health workers at the different levels of our devolved system which will ensure a harmonized referral system with basic cardiovascular diseases treatment services available closest to the people while decongesting the county and national referral facilities.

To this end, we are committed to working with the county governments, CVD stakeholders, public and private sector healthcare providers and teaching institutions to ensure that these guidelines form part of the standard package of care.



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Sicily K. Kariuki (Mrs.), EGH
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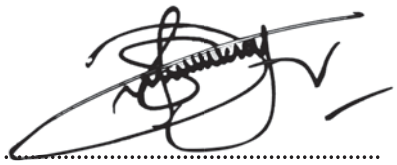
Preface

The prevalence of chronic non-communicable diseases such as cardiovascular diseases and cancers has been on the increase in Kenya in the recent past. This has been occasioned by changes in the social and demographic situation in the country. The life expectancy is improving, while the country is developing at a rapid pace. This has resulted in people living for more years and at the same time adopting lifestyles that have a negative impact on their health. This increase in cardiovascular disease (CVD) and other non-communicable diseases has given rise to a double burden of communicable and non-communicable diseases in Kenya.

CVDs are the number one cause of death globally with more people dying annually from CVDs than from any other cause. Over three quarters of CVD deaths take place in low- and middle-income countries. Most cardiovascular diseases can be prevented by addressing behavioral risk factors such as tobacco use, unhealthy diet and obesity, physical inactivity and harmful use of alcohol using population-wide strategies. People with cardiovascular disease or who are at high cardiovascular risk (due to the presence of one or more risk factors such as hypertension, diabetes, hyperlipidemia or already established disease) need early detection and management using counselling and medicines, as appropriate.

In response to this crisis, the Ministry of Health in collaboration with Non-Governmental Organizations, Regional and International Cardiac Support Bodies spearheaded the National Guidelines for the Management of Cardiovascular Diseases to provide a standardized way of managing cardiovascular diseases in the country.

A technical Working Group was established under the auspices of the Division of Non-Communicable Diseases (DNCD) and various stakeholders to develop these guidelines that are based on up to date and evidence based management of cardiovascular diseases. These Guidelines are a synthesis of information drawn from an extensive review of local and inter- national knowledge and experience. The Guidelines are suitable for use by all health workers and health institutions from both the public and private sectors. They give clear directions on what needs to be done for people living with cardiovascular diseases and provide a guide on the continuum of care required throughout their life course.



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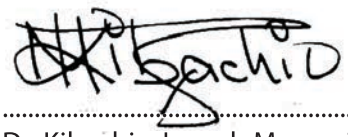
Acknowledgment

The Ministry of Health wishes to acknowledge the collaboration and participation of all individuals and institutions that dedicated their time and effort towards the development of these Guidelines.

We appreciate the immense support from the Cabinet Secretary Mrs. Sicily Kariuki, the Principal Secretary Mr. Peter Tum, Director of Medical Services Dr. Jackson Kioko, the Head of Department of Preventive and Promotive Services Dr. Peter Cherutich and the entire Division of Non Communicable Diseases for their support.

In a special way we wish to convey our gratitude to the technical working group that worked tirelessly to ensure the successful completion of this document. The team was led by Dr. Loise Nyanjau, the focal person for cardiovascular diseases at the Ministry of Health who provided coordination and guidance during the entire process. Others included Dr. Gladwell Gathecha and Dr. Ephantus Maree (MOH-DNCD), Dr. Joyce Nato (WHO), Prof. Fred Bukachi (UoN) and Dr. Bernard Gitura (KCS).

We sincerely thank our partners who participated in this exercise. In this regard, we would like to particularly recognize the Kenya Cardiac Society, Kenya Pediatric Association, University of Nairobi, AMPATH, Kenyatta University, NASCOP, Unit of Specialized Services (MOH), AMREF, CHAK, MSF-Belgium, Kenya Society of Thrombosis and Hemostasis, Kenyatta National Hospital and NCD Alliance-Kenya. We are very grateful to the Healthy Heart Africa project, WHO Kenya Country Office and AIHD for their financial and technical support. We are indebted to the various subject matter experts that reviewed the document and those that provided editorial services.
These Guidelines provide a big milestone in the country's response to CVDs. We urge all health workers and partners to adopt and implement them as we strive towards halting and reversing the burden of cardiovascular diseases in Kenya.



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1 Introduction

1 Introduction

The World Health Organization (WHO) estimates that CVDs are the number one cause of death globally. An estimated 17.7 million people died from CVDs in 2015, representing 31% of all global deaths. Of these deaths, an estimated 7.4 million were due to coronary heart disease and 6.7 million were due to stroke. Over three quarters of CVD deaths take place in low- and middle-income countries. Out of the 17 million premature deaths (under the age of 70) due to non-communicable diseases in 2015, 82% were in low- and middle-income countries, and 37% are caused by CVDs (1).

In Kenya, it is estimated that 25% of hospital admissions are due to CVD and 13% of autopsies revealed CVDs as cause of death (2), representing the second highest cause of death after infectious/maternal/perinatal causes (3). The CVDs are costly to diagnose and manage leading to premature death among the most productive individuals in the household and the society. They are key contributors to poverty due to catastrophic health spending and high out-of-pocket expenditure.

To this end, the National Cardiovascular Disease Management articulate key messages to assist health workers to deliver CVD care to the highest standard. The guidelines are recommended for use by policy makers, program designers and implementers of NCD interventions, health care workers, community educators and teaching institutions.

1:2 Organisation of the Guideline

This guideline begins with an introduction which highlights the need for this document as well as the outlining the roles of both levels of government in disseminating it.
Thereafter, the document describes in detail the prevention of cardiovascular diseases with specific advice for management of risk factors. This is followed by a detailed discussion on specific conditions and their management across the health system. The specific conditions focused on are 6 CVDs which are considered high burden and of public health concern in Kenya:

- Coronary heart disease
- Cerebrovascular disease
- Peripheral arterial disease
- Rheumatic fever, heart disease and infective endocarditis
- Congenital heart disease
- Venous thrombo-embolism

Hypertension is addressed in detail as it's the biggest risk factor to development of CVDs. Additionally, CVD occurring in special populations such as atheletes, older persons, PLHIV, in diabetes, kidney disease and pregnancy have been discussed.Guidance on palliative care follows this section and the document terminated with annexes.

1:3 Dissemination and Use of the National Guidelines for Prevention and Control of Cardiovascular Disease

Roles of the Different Levels of Care

Level one (Tier 1)

Community Health Volunteers (CHVs) are the core resource persons at this level; they should be well trained and equipped with a kit designed to assess CVD risk in the community.
The kit should include:

- A blood pressure machine
- Glucometer and strips
- A weighing scale
- A tape measure
- Waist/hip charts
- BMI charts

The CHVs are the link between the households, community and the health facility.
The community health extension workers (CHEWs) should be trained in the CVDs and coordinate the activities of the CHVs.

The main intervention entails carrying out public awareness campaigns on CVD risk management through mass media e.g. posters, mass media outlets e.g. vernacular radio stations, barazas, community health education forums, community dialogue and action days.

Level two and three (Tier 2)

The CVD package at this level includes:

- Provision of lifestyle intervention/advice
- CVD risk assessment and this entails blood pressure (BP) monitoring, blood sugar assessment, electrocardiography, measuring of lipid profiles, renal functions and urinalysis
- Provision of the initial treatment for type II diabetes mellitus and hypertension
- Referral and follow up for patients on management for CVD in higher levels of care.

Level four and five (Tier 3)

- The CVD package at this level entails a comprehensive CVD risk assessment using the appropriate risk assessment tools.
- Complete cardiovascular imaging and laboratory assessment (echocardiogram etc.)
- Comprehensive management of both primary and secondary prevention interventions
- Referral up (to level 6) for further management and down to lower levels for follow up after initiation of management.

Level six (Tier 4)

These facilities should offer advanced cardiovascular assessment and treatment including cardiac catheterization, angioplasty, bypass surgery, endarterectomy, prostheses and pacemakers, ventricular devices, heart transplant and rehabilitation services.

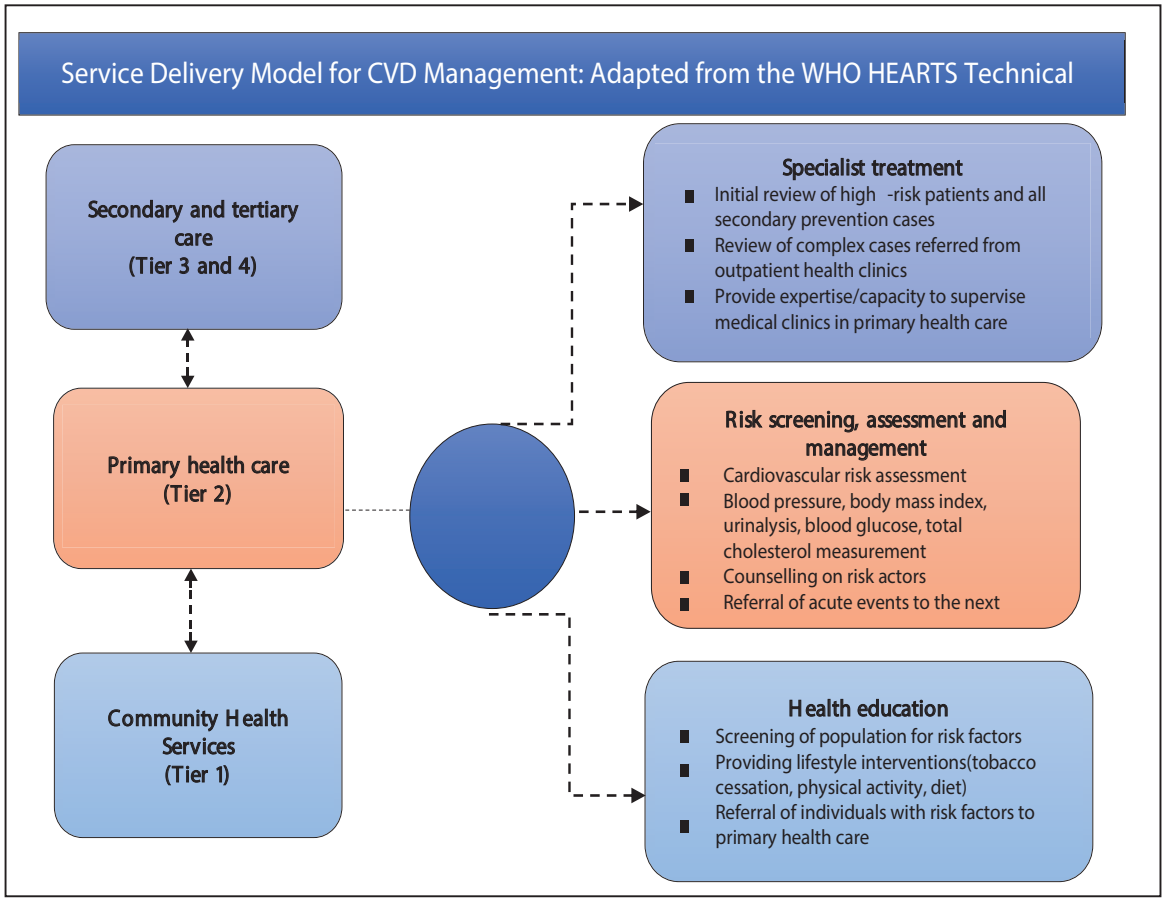

Figure 1.1 Service delivery model for CVD.

(Adapted from WHO, HEARTS Package)

1:4 The table below lists the recommended resources for management of CVD in health facilities:

Table 1:1 Resources Needed for CVD Care Delivery

Resource Needed	Level 2 and 3	Level 4	Level 5 and 6
Human Resources	o Nurses o Clinical officers o Nutritionist o Medical Officer o Lab personnel o Radiographers o Pharmacists o Pharmaceutical Technologists	o Cadres in level 2 and 3 o Physician o Paediatrician o Echocardiographer o Radiologist	o Cadres in Level 2,3,4 o Cardiologist o Pediatric Cardiologist o Perfusionists o Cardiac anaesthetists o Specialised nurses o Cardiothoracic surgeons o Clinical pharmacist o Pathologist
Diagnostic Equipment	o BP Machine o Stethoscope o Weighing scale o Height meter o Thermometer o CVD risk assessment tools o Strips for urinalysis o Glucometer o Hematology equipement and reagents o Biochemistry equipment and reagents o X-Ray o ECG machine	o Equipment in level 2 and 3 o Echo machine for screening o Biochemistry and hematological machines o Ophthalmoscope	o Equipment in level 2, 3 and 4 o Echo machine (high specification) o Blood analysis: fasting blood sugar, electrolytes, creatinine, cholesterol and lipoproteins o Cardiac catheterisation lab o Ambulatory BP o 24 hr Holter machine o Treadmill o Facilities for telemedicine o A critical care unit

Medications	o Thiazide-like* diuretic o Calcium-channel blocker* o ACEI/ARB* o Furosemide** o Statins** o Aspirin**	o Diuretics (including spironolactone and furosemide) o Beta-blockers*** o Digoxin**** o Warfarin o Clopidogrel	o Diuretics (including spironolactone and furosemide) o Beta blockers o Angiotensin converting enzyme inhibitors o Calcium channel blockers o Aspirin o Digoxin**** o Dopamine o Dobutamine o Sildenafil/tadalafil
Main Services	o Detection o Diagnosis o Initiate treatment of uncomplicated hypertension o Follow-up clinic for hypertension o Referral	o Services offered in level 2&3 o Treatment of general medical conditions Comprehensive diagnosis o Management of complications e.g heart failure as you prepare for referral o Referral o Rehabilitation and follow up o Training	o Services offered in level 4 o Cardiac catheterisation and open heart surgery o Treatment of non-cardiac and cardiac surgical complications o Management of pregnancy in a cardiac patient including safe delivery

KEY: *Medications can be initiated at level 2 or 3
**Medications not to be initiated, but prescription can be refilled at level 2/3
***Beta-blockers recommended for children are propranolol and carvedilol
*****Digoxin use limited to physicians and other specialists

Priority Research Areas

1. Regional mapping of the burden of priority CVDs at population level and health facilities in Kenya
2. Determinants of occurrence, the severity and outcome of priority CVDs in Kenya
3. Qualitative studies on quality of CVD care across counties in Kenya

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2. Ongeng'o J, Gatonga P, Olabu B. Cardiovascular causes of death in an east African country: an autopsy study. Cardiol J. 2011;8(1):67–72.
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2 Prevention of Atherosclerotic Cardiovascular Disease

2 Prevention of Atherosclerotic Cardiovascular Disease

List of Abbreviations

AFR E	Africa Region E
BMI	Body Mass Index
BP	Blood Pressure
CABG	Coronary Artery Bypass Grafting
CHEW	Community Health Extension Worker
CHV	Community Health Volunteer
CVD	Cardiovascular Disease
eGFR	Estimated Glomerular Filtration Rate
HDL-C	High Density Lipoprotein-Cholesterol
LDL-C	Low Density Lipoprotein-Cholesterol
MI	Myocardial Infarction
NCD	Non-Communcable Diseases
PCI	Percutaneous Coronary Intervention
PLHIV	Persons Living with HIV
TC	Total Cholesterol
TIA	Transient Ischeamic Attacks
WHO	World Health Organisation

2:1 Etiology and Risk Factors

The majority of cardiovascular diseases are preventable with control of risk factors, early detection and prompt management. There are many risk factors that contribute to CVDs. Hypertension is the single most important risk factor for CVDs.
A small proportion of the population are born with conditions that predispose them to CVDs, while the majority who develop them do so because of a combination of modifiable and non-modifiable risk factors as listed below:

Table 2:1 Risk factors for CVD

Modifiable Risk factors	Non-modifiable Risk factors
• Tobacco use and exposure to tobacco smoke • Unhealthy diet • Overweight/ obesity, • Physical inactivity • Harmful use of alcohol, • Hypertension, • Diabetes and hyperlipidemia • Infections e.g. Rheumatic fever/heart disease, HIV	• Sex • Age • Race, • Family history

Many of the risk factors for CVDs lead to atherosclerosis, which is the narrowing and thickening of arteries; and develop over many years without causing symptoms. The narrowing and thickening of arteries is due to deposition of fatty material, cholesterol and other substances in the walls of the vessels which in turn may reduce blood flow to end organs such as the heart, brain, kidneys and limbs.

The figure below demonstrates a proposed causal pathway for Atherosclerotic CVD.

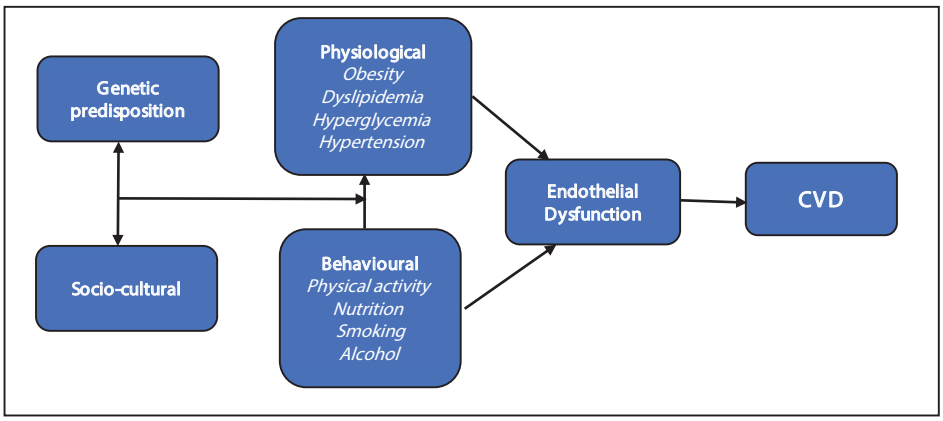


Figure 2.1 Causation pathway for CVD. Adapted from Stoner et al. 2014 (2)

2:2 Prevention

Most cardiovascular diseases are preventable through the reduction of behavioral risk factors such as tobacco use, unhealthy diet, physical inactivity and alcohol consumption (3). There are several strategies that target the modifiable risk factors:

Table 2:2 CVD prevention strategies

Prevention Strategy	Health Promotion/Primordial Prevention	Primary Prevention	Secondary Prevention	Tertiary Prevention
Target	Entire Population	People with one or more risk factors	People at early stage of disease	People with symptomatic or advanced disease
Effects	Prevent risk factors, lower population risk	Prevent development of disease at early age	Prevent disease progression or recurrence	Reduce complications or disability
Examples	• Public awareness campaigns • Encourage safe pregnancy by avoidance of exposure to risk factors and unnecessary and non-prescribed use of medicines. • Use folate and iron supplements in women of child bearing age.	Early detection, appropriate screening and surveillance, vaccination, together with lifestyle modification e.g. cessation of tobacco and alcohol use consumption of healthy diet low in saturated fat, salt and refined sugars and high in fruits and vegetables	Providing appropriate treatment and care, support groups, medication adherence together with lifestyle modification e.g. cessation of tobacco and alcohol use, and consumption of healthy diet low in saturated fat, salt and refined sugars and high in fruits and vegetables	Rehabilitation and palliative care at the various stages of the disease pathway. Revascularization i.e. coronary bypass surgery (CABG) or carotid artery endarterectomy

2:3 Cardiovascular risk assessment and reduction

Risk assessment provides evidence-based approach to helping the clinician and the patient to make appropriate decisions on effective prevention and management of CVD. The CVD risk assessment is based on key risk factors using the WHO/ISH assessment tools, specifically the AFRO E charts for the Kenyan context (4). Recommendations for prevention and care of CVD based on individual risk level are then made, focusing on tobacco and alcohol control, dietary modification, physical activity and pharmacological management as shown below.

Table 2:3 Recommended age to offer cardiovascular risk assessment

Population Group	Men	Women
1. Asymptomatic people without known risk factors*	40 years and above	40 years and above
2. Persons with other known cardiovascular risk factors or at high risk of developing diabetes	Age 30 years	Age 35 years
3. Family history risk factors • Diabetes in first-degree relative (parent, brother or sister) • Premature coronary heart disease or ischemic stroke in a first-degree relative (father or brother <55years, mother or sister <65 years)		
4. Personal history risk factors • People who smoke (or who have quit only in the last 12 months) • Gestational diabetes, polycystic ovary syndrome • Prior blood pressure (BP) ≥160/95 mm Hg (taken as a clinic BP), prior TC:HDL ratio ≥7 • HbA1c 41–49 mmol/mol -%! • BMI ≥30 or truncal obesity (waist circumference ≥100 cm in men or ≥90 cm in women) • eGFR<60 ml/min/1.73 m^2		
5. People with diabetes (type 1 or 2)	Annually from the time of diagnosis	Annually from the time of diagnosis

2:4 What to assess and document for in cardiovascular risk assessment

Table 2:4 Assessment for CVD

History	• Age • Gender • Ethnicity • Smoking status (if stopped smoking for <12 months, assess as a smoker)
Family history	• Premature coronary heart disease or ischemic stroke in a first -degree relative (father or brother <55 years, mother or sister <65 years) • Type 2 diabetes • Genetic lipid disorder
Past medical history	• Past history of CVD (MI, PCI, CABG, angina, ischemic stroke, TIA, peripheral vascular disease [PVD]) • Lipid disorder • Renal impairment (eGFR<60 if under age 75) • Diabetes • Atrial fibrillation
Measure	• Average of two sitting BP measurements – one sitting measurement if not above 160/95; two sitting measurements if the first is above 160/95 • BMI, waist circumference • Non-fasting lipid profile • Fasting Blood Glucose

* For asymptomatic Indo-Asians/Caucasians conduct CVD risk assessment at 30years for men and 35 years for women.

2:5 WHO/ISH Risk Prediction Charts for AFRO E region.

These are 10-year risk prediction tools for a fatal or non-fatal cardiovascular event by gender, age, systolic blood pressure, total blood cholesterol, smoking status and presence or absence of diabetes mellitus. The charts are shown below

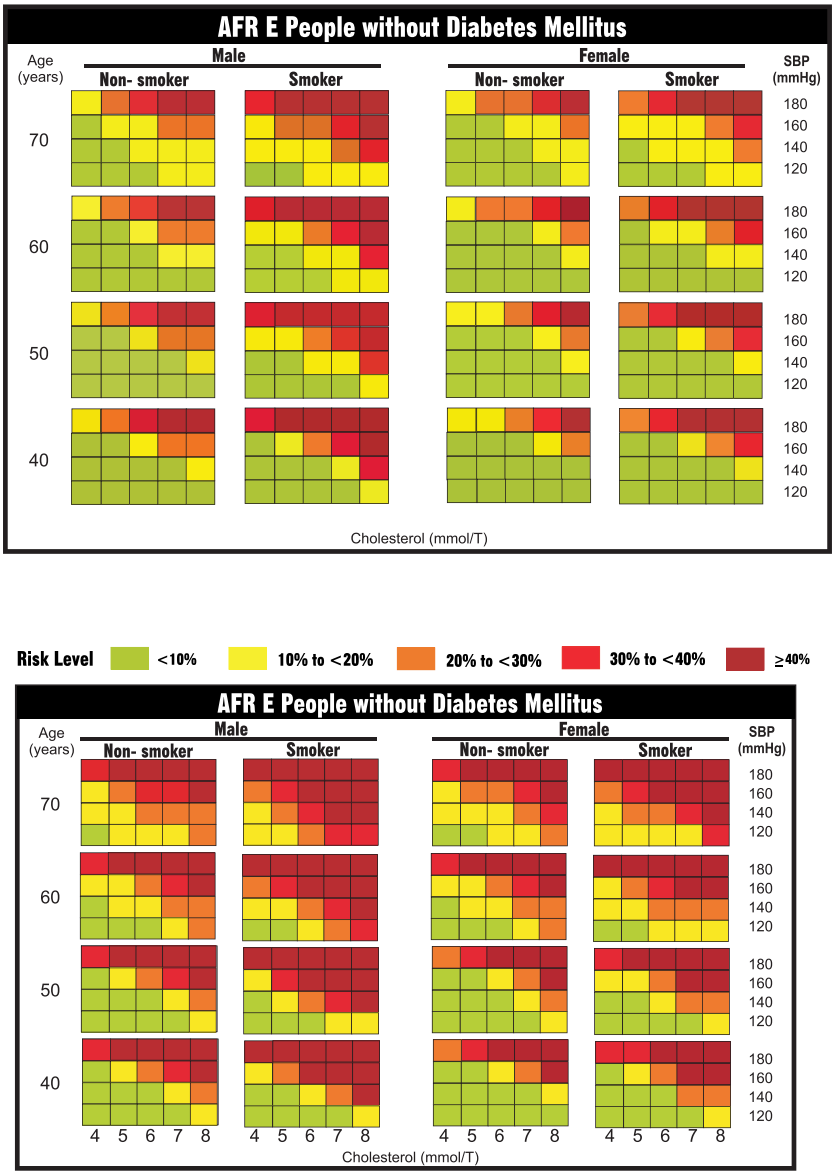


Figure 2.2: WHO/ISH CVD Risk Prediction Charts

2:6 Recommendations for prevention and care of CVD based on individual risk level (Adapted from WHO (5))

Table 2:6 Recommendations for prevention and care of CVD

10-year risk of cardiovascular event >30%	10-year risk of cardiovascular event 20–30%	10-year risk of cardiovascular event 10–20%	10-year risk of cardiovascular event <10%
● Individuals in this category are at veryhigh risk of fatal or non-fatal vascular events ● Monitor risk profile every 3–6 months	● Individuals in this category are at high risk of fatal or non-fatal vascular events ● Monitor risk profile every 3–6 months	● Individuals in this category are atmoderate risk offatal or non-fatalvascular events. ● Monitor risk profile every 6–12 months	● Individuals in this category are at lowrisk. Low risk does not mean"no" risk. ● Conservative management focusing on lifestyle Interventions is suggested
When resources are limited, individual counseling and provision of care may have to be prioritized according to cardiovascular risk.			

The aim of prevention and management is to reduce CVD risk, slow progression and prevent complications.

Cardiovascular disease prevention includes both non-pharmacological and pharmacological therapies.

Non-pharmacological therapy entails lifestyle interventions such as tobacco and alcohol control, consumption of healthy diets and adequate physical activity. Pharmacological therapy is disease specific, including lipid lowering agents, blood pressure lowering agents, control of diabetes and antiplatelet therapy as discussed in detail further in this document.

2:7 Principles of Non-Pharmacological Therapy

Non-pharmacological interventions are largely lifestyle interventions. Graded lifestyle advice is appropriate for everyone and needs to consider the individual's circumstances. Specific lifestyle interventions are based on a behavior counseling approach.

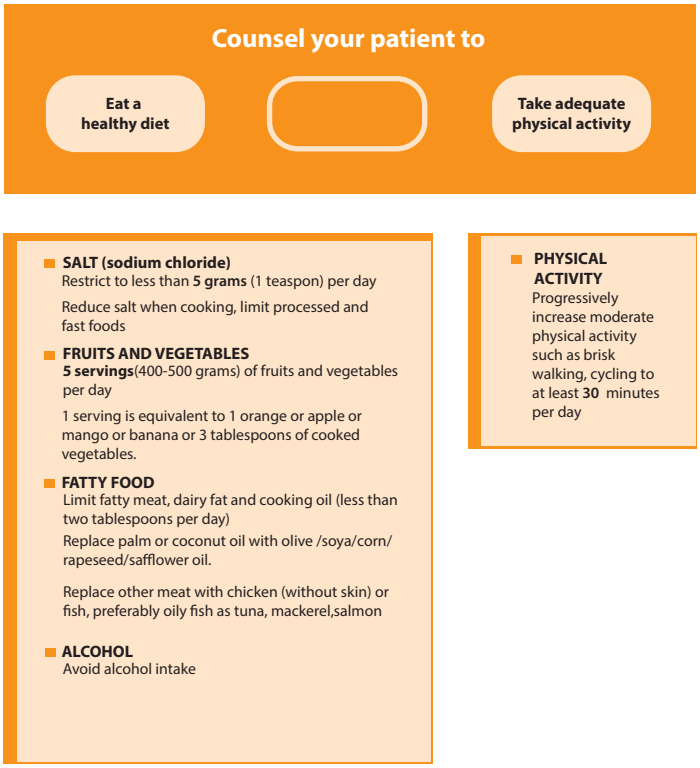
1. Nutrition and healthy diet

• Healthy dietary practices start early in life. A healthy pregnancy is likely to yield a healthy baby. Breastfeeding fosters healthy growth and improves cognitive development, and may have long-term health benefits like reducing the risk of becoming overweight or obese and developing CVDs later in life.

• Energy intake (calories) should be in balance with energy expenditure. Evidence indicates that total fat should not exceed 30% of total energy intake to avoid unhealthy weight gain, with a shift in fat consumption away from saturated fats to unsaturated fats, and towards the elimination of industrial trans-fats.

• A healthy diet helps protect against malnutrition in all its forms, as well as CVDs.

Figure 2.3: Protocol for counselling on diet and physical activity



• Limiting intake of refined sugars to less than 10% of total energy intake is part of a healthy diet. A further reduction to less than 5% of total energy intake is suggested for additional health benefits.

Recommendations for salt reduction

These guidelines are aligned to WHO recommendations (6):

• **Adults:** consume less than 5 g (just under a teaspoon) of salt per day.

• **Children:** consume less than 3g of salt per day

• All salt that is consumed should be iodized or "fortified" with iodine, which is essential for healthy brain development in the fetus and young child and optimizing people's mental function in general.

• Avoid processed foods such as bread, crisps

• Avoid adding salt at the table while eating.

Misperceptions about salt reduction

• "On a hot and humid day when you sweat, you need more salt in the diet:" There is little salt lost through sweat so there is no need for extra salt even on a hot and humid day, although it is important to drink a lot of water.

• "Sea salt is not 'better' than manufactured salt simply because it is 'natural.'" Regardless of the source of salt, it is the sodium in salt that causes bad health outcomes.

• "Food has no flavor without salt." Whilst this may be true at first, taste buds soon become accustomed to less salt and you are more likely to enjoy food with less salt, and more flavor.

2. Physical Activity and Cardiovascular Disease

Patients should be encouraged to engage in a variety of physical activities and to progressively increase their activity as tolerated. Individuals with a history of CVD should consult their healthcare provider before they undertake vigorous physical activity. Vigorous activity is generally not encouraged in people with impaired left ventricular function, severe coronary artery disease, recent MI, significant ventricular arrhythmias or stenotic valve disease. Moderate activity is however recommended under the guidance of a healthcare provider.

Recommendations (Adapted from WHO Physical Activity Fact Sheet (7):

• Children and adolescents aged 5-17 years should do at least 60 minutes of moderate to vigorous-intensity physical activity daily.

•Adults should do at least 150 minutes of moderate-intensity physical activity throughout the week.

•Those with poor mobility should perform physical activity to enhance balance and prevent falls, 3 or more days per week.

•Muscle-strengthening activities should be done involving major muscle groups, 2 or more days a week.

Table 2:2 Contraindications, Precautions and Indications to stop physical activity in patients with heart disease

Contraindications	Precautions	Indications to stop physical activity
● Unstable angina ● Symptoms (e.g chest discomfort, shortness of breath) on low activity ● Decompensated heart failure ● Severe aortic stenosis ● Uncontrolled hypertension (e.g. systolic BP≥180 mmHg; Diastolic BP ≥110 mmHg) ● Acute infection or fever ● Symptomatic uncontrolled diabetes (e.g. blood glucose <6mmol/L or >15mmol/L)	All patients should be provided with clear advice on risks and benefits of physical activity, warm-up and cool-down, limiting physical activity to low-moderate intensity, appropriate footwear and clothing, and the importance of following their symptom (chest pain, diabetes) management plans ● Resting tachycardia/arrhythmia	● Squeezing, discomfort or typical pain in the centre of the chest or behind the sternum, spreading to the shoulders, neck, jaw and/or arms ● Dizziness, lightheadedness or feeling faint, difficulty breathing,nausea, uncharacteristic excessive sweating ● Palpitations associated with feeling unwell, undue fatigue ● Shakiness, tingling lips, hunger, weakness or palpitations in people with diabetes

3. Weight Management

Overweight and/or obesity is a major risk factor for non-communicable diseases such as CVD. It is defined as excessive fat accumulation in the body. Body Mass Index (BMI) is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. It is defined as a person's weight in kilograms divided by the square of his height in meters (kg/m2). The fundamental cause of obesity and overweight is an energy imbalance between calories consumed and calories expended. Childhood obesity is associated with a higher chance of adult obesity, premature death and disability in adulthood. But in addition to increased future risks, obese children experience breathing difficulties, increased risk of fractures, hypertension, and early markers of cardiovascular disease, insulin resistance and psychological effects.

Table 2:3 Definition of overweight and obesity in children

Population Group	Overweight	Obesity
Children <5 years	Weight-for-height > 2 standard deviations above the WHO Child Growth Standards median*	Weight-for-height > 3 standard deviations above the WHO Child Growth Standards median*
Children 5-19 years	BMI-for-age > 1 standard deviation above the WHO Growth Reference median*	BMI-for-age >2 standard deviations above the WHO Growth Reference median *
Adults	BMI 25-30	BMI > 30

*Refer to WHO growth reference charts and tables for children aged < 5years between 5–19 years

Recommendations for Weight Management

- Limit energy intake from total fats and sugars;
- Increase consumption of fruit and vegetables, as well as legumes, whole grains and nuts; (refer to Nutrition and healthy diet section)
- Engage in regular physical activity. (Refer to physical activity section)

4. Tobacco Dependence Treatment, Cessation and Prevention

The tobacco epidemic is one of the biggest public health threats worldwide, killing more than 7 million people a year. More than 6 million of those deaths are the result of direct tobacco use while around 890 000 are the result of non-smokers being exposed to second-hand smoke. Tobacco is harmful in all its forms and stages of production. Thirteen percent of Kenyans currently consume some form of tobacco products witha significantly higher prevalence among men (23 %) than women (4.1 %).24% and 20.9 % of Kenyans are exposed to second hand smoke at home and work respectively (Kenya stepwise survey 2015).

Interventions for tobacco dependence

Health care professionals should provide regular and tailored counselling interventions for those who meet the criteria for tobacco dependence. Tobacco dependence treatment and cessation programs should combine behavioural support (such as psychological interventions, telephone support and self-help) with pharmacotherapy treatment where necessary. Before deciding on which intervention to use, it is essential to document tobacco use status and conduct screening. Healthcare providers of tobacco dependence treatment and cessation should receive suitable training.

There are three main categories of interventions:

a. Brief advice by a healthcare professional
b. Behavioural support
c. Pharmacotherapy

Brief advice (5As) by a healthcare professional

The 5As approach is an evidence based framework for structuring tobacco dependence treatment and cessation brief intervention in healthcare and community settings. The 5As for tobacco cessation are Ask, Advice, Assist, Assess and Arrange

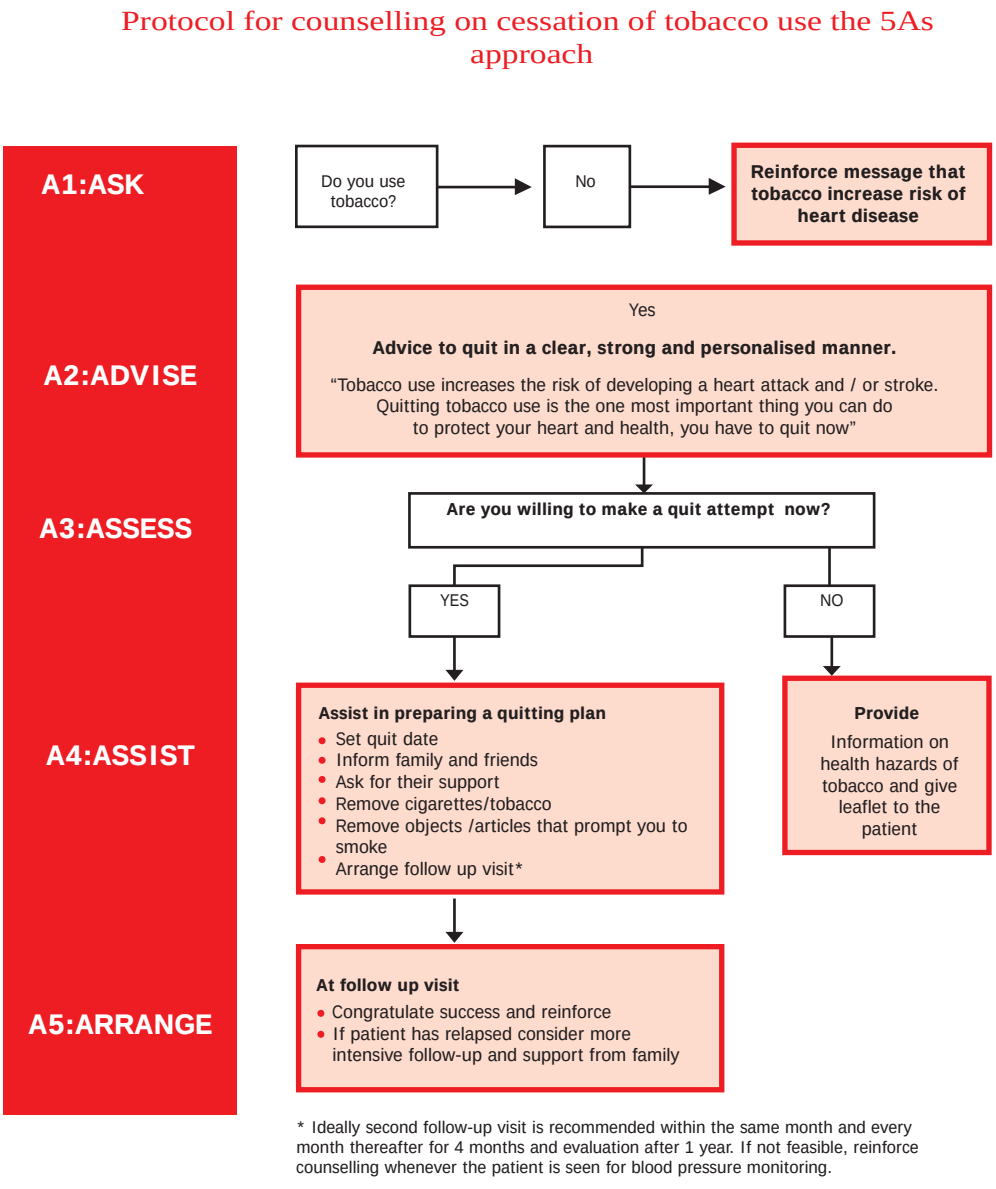


Figure 2.4: Counselling on Cessation of Tobacco Use. Adapted from WHO (8)

2:8 Behavioural support

Behavioural support aims at changing thought processes and beliefs. If one changes the way they feel about tobacco use, a change in behaviour should follow. The healthcare provider helps the person to deal with negative feelings and assists the clients in setting realistic goals to avoid failure.

Behavioural strategies that can support a client to cope with the triggers and high-risk situations for tobacco use include:

- Face to face support
- Individual behavioural counselling
- Group behaviour therapy
- Telephone counselling or quit lines
- Self-help materials

The pharmacological interventions include:

1. Nicotine replacement therapies
    - Nicotine gums
    - Nicotine patches
    - Nicotine lozenges/sublingual tablets
    - Nicotine inhalers
    - Nicotine nasal spray
2. Non-Nicotine replacement therapies
    - Bupropion
    - Varenicline

For further details on behavioral and pharmacological interventions, refer to the Kenya National Guidelines for Tobacco Dependence Treatment and Cessation.

2:9 Principles of Pharmacological Therapy

Pharmacological management is largely disease-specific and is majorly handled in chapter 2. Discussed below are general principles of lipid lowering and antiplatelet therapy.

a) Lipid Lowering Therapy

This therapy is given to patients depending on the individual's risk of developing CVD as per the assessment described in the previous chapter. However, in patients with diabetes, total Cholesterol (TC) ≥8 mmol/L or a TC:HDL-C ratio ≥8, lipid-lowering treatment is usually recommended irrespective of the combined CVD risk.

Lipid lowering for people with combined CVD risk between 10 % and 20 %

- For patients with combined CVD risk between about 10 %t and 20 %, discuss the benefits (and risks) of initiating statins.
- Following lifestyle management, repeat lipid profile (non-fasting) to recalcu late risk and use the results to inform shared treatment decision-making in 6–12 months.
- The aim is to achieve a moderate reduction in LDL-C; no target is required for those with a combined risk ratio under 20 %.
- Re-measurement can wait until the next formal combined risk assessment.

Before a person starts on medication, it is important to consider and exclude a treatable primary cause for a dyslipidemia. Such causes include a high saturated fat diet and excessive alcohol consumption, hypothyroidism, diabetes, liver disease, nephrotic syndrome and steroid treatment.

Lipid lowering for people with combined CVD risk > 20% or established cardiovascular disease

For people with known cardiovascular disease and those with a combined cardiovascular risk >20 %, statin treatment is strongly recommended.

Monitoring

Monitor non-fasting lipids every three to six months until the person is stable on their treatment regime and then no more than once a year. Measuring more frequently may mislead as the variation in day-to-day measurement may be greater than drift over time. The aim is to achieve a moderate reduction in LDL-C; no target is required for those with a combined risk ratio under 20 percent. Consider interactions of statins with other medications. Review with a pharmacist. Consider a simvastatin dose reduction for patients taking fibrates, systemic fusidic acid, colchicine or with renal impairment. Monitoring liver function tests with statin use is not considered necessary as the risk of liver toxicity appears negligible. Monitoring creatine kinase (CK) is not required in those who are asymptomatic. Check CK for unexplained muscle pain, tenderness or weakness. The risk of myopathy is usually dose-related and is increased in the elderly and with combination treatments. For muscle pain without CK rise, dose reduction or discontinuation may be required. With CK rise 3–10x normal with symptoms, dose reduction or discontinuation with regular weekly monitoring of symptoms and CK is appropriate. With CK rise >10x normal with symptoms, discontinue statin immediately.

b) Antiplatelet Therapy

- Aspirin and other antiplatelet agents are not generally recommended for people with a risk lower than 20 percent.
- Antiplatelet therapy is recommended for people with combined CVD risk over 20 % but without established cardiovascular disease.
- Low-dose Aspirin (75-150 mg) can be considered for primary prevention in high-risk populations, taking into account the harms and benefits.
- Antiplatelet therapy for people with established cardiovascular disease
- Antiplatelet therapy is strongly recommended for people with established cardiovascular disease.

Aspirin contraindications

- Aspirin allergies/intolerance
- Active peptic ulceration
- Systolic BP >180
- Other major bleeding risks

Early detection and appropriate control of Hypertension is the hallmark of preventing atherosclerotic CVD in Kenya

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3 Hypertension

3 Hypertension

List of Abbreviations

ABPM	AmbulatoryBlood Pressure Monitoring
ACEI	Angiotensin Converting Enzyme Inhibitor
ACLS	Advanced Cardiac Life Support
ACS	Acute Coronary Syndrome
ARB	Angiotensin Receptor Blocker
BB	Beta Blockers
BLS	Basic Life Support
BMI	Body Mass Index
BP	Blood Pressure
CABG	Coronary Artery Bypass Graft
CAD	Coronary Artery Disease
CCB	Calcium Channel Blocker
CT	Computer Tomography
CVD	Cardiovascular Diseases
CXR	Chest Xray
DBP	Diastolic Blood Pressure
DVT	Deep Venous Thrombosis
ECG	Electrocardiogram
eGFR	Estimated Glomerular Filtration Rate
ER	Emergency Room
FBG	Fasting Blood Glucose
GERD	Gastro-esophageal Reflux Disease
HBA1c	Glycated Hemoglobin A1
HBPM	Home Blood Pressure Monitoring
HDL	High Density lipopr otein
HIV	Human Immunodeficiency Virus
HTN	Hypertension
IHD	Ischemic Heart Disease
LDL	Low Density lipoprotein
LDL	low Density Lipoprotein
LV	Left Ventricle
LVEF	Left Ventricular Ejection Fraction
LVH	Left Ventricular Hypertrophy
MAP	Mean Arterial Pressure
NSAIDS	Non-steroidal Anti-inflammatory Drugs
NSTEMI	None-ST Elevated Myocardial Infarction
OD	Once Daily
OGTT	Oral Glucose Tolerance Test
RBS	Random Blood Sugar
RV	Right Ventricle
SBP	Systolic Blood Pressure
SC	Subcutaneous
STEMI	ST Elevated Myocardial Infarction
TBC	Total Blood Count
TIA	Transient Ischemic Attack
UA	Unstable Angina
V/Q	Ventilation-perfusion Ratio
VF	Ventricular Fibrillation
VT	Ventricular Tachycardia

Highlights

- The prevalence of hypertension among adults is high in Africa. However awareness,treatment and control rates are very low.
- Hypertension is a life-long condition, which once diagnosed must be managed and monitored for life
- Hypertension is asymptomatic, therefore diagnosis and followup is only by measurement of BP
- The rationale for hypertension treatment is to prevent complications, especially cardiovascular and kidney disease
- Hypertension management includes combination of lifestyle modifications and drug therapy

3:1 Introduction

Hypertension is defined as persistently elevated, systolic and/or diastolic blood pressure (BP) of 140/90 mmHg or more in subjects aged 18 years and above. The definition also applies to those individuals who are already taking antihypertensive medications even if their current blood pressure is less than 140/90mmHg. The goal of treatment is to achieve blood pressure below 140/90mmhg as treatment of BP above this has been shown to significantly reduce risk of stroke, coronary heart disease, chronic kidney disease, heart failure and death. Modification of lifestyle factors can delay onset of hypertension, contribute to lowering of blood pressure in treated patients and in some cases abolish need for antihypertensive therapy (1).
(See last section for paediatric guideline).

Table 3:1 Definition and classification of hypertension (2)

Category	Systolic		Diastolic
Optimal	<120	and	<80
Normal	120-129	and/or	80-84
High normal	130-139	and/or	85-89
Grade 1 hypertension	140-159	and/or	90-99
Grade 2 hypertension	160-179	and/or	100-109
Grade 3 hypertension	≥180	and/or	≥100
Isolated systolic hypertension	≥140	and	<90

(Adapted from the ESH/ISH guidelines)

NB: The class is determined by whichever of the readings is highest.

3:2 Epidemiology

The global prevalence of raised blood pressure was around 31% in 2010; 28.5% in high income countries and 31.5 in low and middle income economies (3). In addition, the LMICs have low awareness, treatment levels and control (38%, 29% and 8%, respectively). The prevalence dramatically increases in patients older than 60 years and the prevalence may be as high as 50% in this age group. Across the WHO regions, the prevalence of raised blood pressure is highest in Africa, where it was 46% for both sexes combined(4). Treatment and control of hypertension are critically important for the prevention of consequent cardiovascular and kidney diseases (5).

According to the 2015 Kenya STEPS survey, 24% of Kenyans either had elevated blood pressure or were on treatment for hypertension, more than half (56 percent) of Kenyans have never been screened for hypertension. Overall, only 15% of those with hypertension are aware of their status, only 8% of kenyans living with hypertension are on treatment and only 4.6% of those on treatment are well controlled [6]

3:3 Causes of hypertension

Hypertension is broadly classified into 2 groups:

1. **Primary or Essential hypertension:** The cause is unknown, constitutes about 95% of cases in adults. Risk factors include:
    - Age above 45 (60 years in women)
    - Race (more in blacks)
    - Family history
    - Overweight/central obesity
    - Physical inactivity
    - Tobacco use
    - High dietary salt
    - Low dietary potassium
    - Low vitamin D
    - Stress
    - Chronic/heavy alcohol use
2. **Secondary hypertension:** cases where the cause of hypertension can be identified and sometimes treated, around 5% of the cases. Causes include:
    - Chronic parenchymal kidney disease
    - Renovascular disease (Renal artery stenosis)
    - Pheochromocytoma
    - Excessive aldosterone secretion
    - Excessive glucocorticoids
    - Thyroid disorders
    - Coarctation of the aorta
    - Drugs (anabolic steroids; oestrogen; NSAIDS; sympathomimetic drugs)
    - Sleep apnea
    - Psychoactive/recreational drugs: amphetamines, cocaine

3:4 Diagnosis

The accurate diagnosis of hypertension depends on the accurate measurement of BP. BP readings can be taken in the clinic (office) or out of the office i.e. ambulatory BP measurement (ABPM) or home measurement. Figure 1 gives details of office/health facility measurement.

Allow patient to sit for 3–5 minutes before commencing measurement

The SBP should be first estimated by palpation to avoid missing the auscultatory gap

Take two readings 1–2 minutes apart. If consecutive readings differ by > 5 mm, take additional readings

At initial consultation measure BP in both arms, and if discrepant use the higher arm for future estimations

The patient should be seated, back supported, arm bared and arm supported at heart level

Patients should not have smoked, ingested caffeine-containing beverages or food in previous 30 min

An appropriate size cuff should be used: a standard cuff (12 cm) for a normal arm and a larger cuff (15 cm) for an arm with a mid-upper circumference > 33 cm (the bladder within the cuff should encircle 80% of the arm)

Measure BP after 1 and 3 minutes of standing at first consultation in the elderly, diabetics and in patients where orthostatic hypotension is common

When adopting the auscultatory measurement use Korotkoff I (appearance) and V

(Disappearance) to identify SBP and DBP respectively

Take repeated measurements in patients with atrial fibrillation and other arthythmias to improve accuracy

Choice of cuff size in children

Newborns and premature infants: 4 × 8 cm

Infants: 6 × 12 cm

Older children: 9 × 18 cm

Figure 3.1 BP measurement procedure

Adapted from the South African hypertension practice guideline 2014 and AAFP 2005

3:5 Out of office/health facility BP measurement

a) Ambulatory blood pressure monitoring (ABPM)

ABPM is performed with the patient wearing a portable BP measuring device, usually on the non-dominant arm, for a 24–25 h period, so that it gives information on BP during daily activities and at night during sleep (2). Ambulatory BP is a better predictor of cardiovascular events such as myocardial infarction and stroke better then office/health facility BP measurement.

b) Home blood pressure monitoring (HBPM): This is self-measurement of BP at home. The same precautions outlined above should be observed. Frequency of measurement and recording will be as advised by health practitioner. HBPM has been correlated with hypertension-induced organ damage, cardiovascular morbidity and mortality than office/health facility BP measurement.

Table 3:2 Diagnostic cutoffs for office and out of office BP measurements (2)

Category	Systolic BP(mmHG)		Diastolic BP(mmHG)
Office BP	≥140	and	≥90
Ambulatory BP			
Daytime (or awake)	≥135	and/or	≥85
Nighttime (or asleep)	≥120	and/or	≥70
24-h	≥130	and/or	≥80
Home BP	≥135	and/or	≥85

(Adapted from the 2013 ESH/ESC Guidelines for the management of arterial hypertension)

NB:

White coat/ isolated office hypertension: It is persistently elevated BP in the clinic while BP is normal outside the clinic.

Masked/ isolated ambulatory Hypertension: BP is normal in the office and abnormally high out of the medical environment. It is usually associated with other risk factors, asymptomatic organ damage and increased risk of diabetes and sustained hypertension.

NB: These terms should only be used to define untreated patients.

3:6 Evaluation(7)

Hypertension is largely asymptomatic. The main purpose of evaluation is for assessment of complications, concomitant risk factors and secondary causes of hypertension.

History

- Ask about risk factors outlined above e.g age, smoking status, diet, alcohol intake, physical activity, family history of hypertension and use of medications.
- Ask about previous diagnosis of hypertension and medications being used
- Ask about any cardiovascular events, including stroke, TIA or dementia, coronary artery disease, heart failure, chronic kidney disease, peripheral artery disease, diabetes and sleep apnea. The presence of any of these conditions determine the choice of drugs for treatment as well as the overall cardiovascular risk score for this individual.

3:7 Physical examination and Investigations

- At the first visit it is important to perform a complete physical examination because often getting care for hypertension is the only contact that patients have with a medical practitioner.
- Measuring blood pressure (discussed earlier).
- Document the patient's weight and height and calculate body mass index: this helps to set targets for weight loss
- Waist circumference: this helps determine whether a patient has the metabolic syndrome or is at risk for type 2 diabetes. Risk is high when the measurement is >102 cm in men or >88 cm in women.
- Signs of heart failure: This diagnosis strongly influences the choice of hypertension therapy. Left ventricular hypertrophy can be suspected by chest palpation, and heart failure can be indicated by distended jugular veins, rales on chest examination, an enlarged liver, and peripheral edema.
- Neurologic examination: This may reveal signs of previous stroke and affect treatment selection.
- Eyes: If possible, the optic fundi should be checked for hypertensive or diabetic changes and the areas around the eyes for findings such as xanthomas.
- Pulse: It is important to check peripheral pulse rates; if they are diminished or absent, this can indicate peripheral artery disease. (Adapted from the ASH/ISH practice guidelines on management of hypertension in the community)

Table 3:3 Rationale for Physical Examination

Examination	Rationale
Weight and height (BMI)	Set targets for weight loss, choice of drugs,
Waist circumference	Diagnose metabolic syndrome and T2DM risk
Signs of heart failure (distended jugular veins, rales on chest auscultation, enlarged liver and peripheral edema)	Choice of medications
Neurologic examination	Signs of previous stroke and treatment selection
Peripheral Pulses	If diminished or absent, suspect peripheral artery disease If irregular, suspect cardiac disease

Table 3:4 Essential package of investigations

Investigation	Rationale
Urinalysis	Evidence of kidney disease or diabetes
Blood glucose	Diagnosis of diabetes.
Full blood count	Anaemia may indicate CKD
Creatinine, Electrolytes	Diagnosis of renal disease. Electrolytes imbalance may suggest renal or hormonal anomaly
Lipid profile	Dyslipidaemia is a cardiovascular disease risk factor
Electrocardiography (ECG)	Identify cardiac anomalies such as enlargement, infarction, ventricular dysfunction etc

Note: Additional tests may be ordered as needed, at the discretion of the health care team.

3:8 Cardiovascular Disease Risk factor stratification

The rationale for treatment of hypertension is to prevent complications, mainly cardiovascular. Patients should therefore undergo cardiovascular risk stratification based on level of blood pressure, concomitant risk factors, target organ damage and clinical complications as shown in table 5. Table 6 shows the risk stratification and how this guides the management.

Table 3:5 Major risk factors, target-organ damage (TOD) and complications. (8)

Major risk factors	TOD	Complications
• Levels of systolic and diastolic BP • Smoking • Dyslipidaemia: — total cholesterol > 5.1 mmol/l, OR — LDL > 3 mmol/l, OR — HDL men < 1 and women < 1.2 mmol/l • Diabetes mellitus • Men > 55 years • Women > 65 years • Family history of early onset of CVD: — Men aged < 55 years — Women aged < 65 years • Waist circumference: abdominal obesity: — Men ≥ 102 cm — Women ≥ 88 cm	• LVH: based on ECG • Microalbuminuria: albumin creatine ratio 3–30 mg/mmol preferably spot morning urine and eGFR > 60 ml/min	• Coronary heart disease • Heart failure • Chronic kidney disease: — macroalbuminuria > 30 mg/mmol — OR eGFR < 60 ml/min • Stroke or TIA • Peripheral arterial disease • Advanced retinopathy: — haemorrhages OR — exudates — papilloedema

Table 3:6 Cardiovascular Risk Stratification Tool

Other disease/risk factor history	BP				
	Normal SBP 120-129 Or DBP 80-84	**High normal SBP 130-139 Or DBP 85-89**	**Stage 1 Mild hypertension SBP 140-159 Or DBP 90-99**	**Stage 2 Moderate hypertension SBP 160-179 Or DBP 100-109**	**Stage 3 Severe hypertension SBP >180 Or DBP >110**
No other major risk factors	Average risk No BP intervention	Average risk No BP intervention	Low added risk • Lifestyle changes for several months • Then add BP drugs targeting <140/90	Moderate added risk • Lifestyle changes for several months • Then add BP drugs targeting <140/90	High added risk • Lifestyle changes • Immediate BP drugs targeting <140/90
1-2 major risk factors	Low added risk • Lifestyle changes • No BP intervention	Low added risk • Lifestyle changes • No BP intervention	Moderate added risk • Lifestyle changes for several months • Then add BP drugs targeting <140/90	Moderate added risk • Lifestyle changes for several months • Then add BP drugs targeting <140/90	Very high added risk • Lifestyle changes • Immediate BP drugs targeting <140/90
≥3 major risk factors or TOD or DM or metabolic syndrome	Moderate added risk • Lifestyle changes • No BP intervention	High added risk • Lifestyle changes • No BP intervention	High added risk • Lifestyle changes • BP drugs targeting <140/90	High added risk • Lifestyle changes • BP drugs targeting <140/90	Very high added risk • Lifestyle changes • Immediate BP drugs targeting <140/90
Symptomatic CVD, CKD stage ≥4 or diabetes with OD/RFs	High added risk • Lifestyle changes • No BP intervention	Very high added risk • Lifestyle changes • No BP intervention	Very high added risk • Lifestyle changes • BP drugs targeting <140/90	Very high added risk • Lifestyle changes • BP drugs targeting <140/90	Very high added risk • Lifestyle changes • Immediate BP drugs targeting <140/90

3:8 Management

The overall aim of the treatment of hypertension is the adequate control of blood pressure and the control of other risk factors with the overall aim of reducing morbidity and mortality from the complications.

Non-pharmacologic/Lifestyle Modification

At every clinic visit, all patients should receive advice about lifestyle modification. Healthy lifestyle choices can reduce blood pressure and cardiovascular risk and reduce the dose and number of antihypertensive medications required.

Table 3:7 Lifestyle advice for hypertension

- Avoidance of alcohol
- Avoidance of all forms of tobacco
- Daily adequate physical exercise: Hypertensive patients should be advised to participate in at least 30 min of moderate-intensity dynamic aerobic exercise (walking, jogging, cycling or swimming) on 5–7 days per week
- Consumption of a healthy diet: Hypertensive patients should be advised to eat vegetables, low-fat dairy products, dietary and soluble fibre, whole grains and protein from plant sources, reduced in saturated fat and cholesterol, Fresh fruits. Avoid added salt and high-salt food.
- Weight reduction: for overweight and obese hypertensive patients.

(Adapted from the ESH/ESC guidelines, 2013).

Pharmacologic

Threshold for initiation (When to initiate antihypertensive therapy and goals of treatment)
The diagnosis of hypertension and decision to begin antihypertensive medication requires elevated SBP and/or DBP measurements confirmed on at least 3 separate occasions over a 2-month period **as well as** the cardiovascular risk level of the patient.

The overall health and frailty of an elderly person should be assessed before making a decision to start antihypertensive therapy. If there is doubt or concern about the health status of the patient, they should be referred to a specialist for further management.

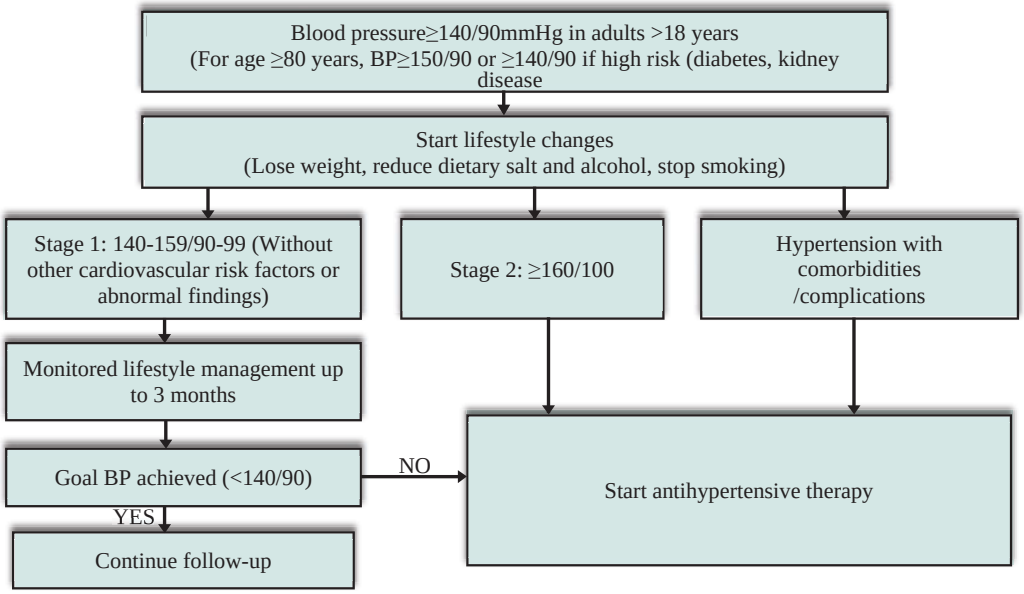

Figure 3.1: Threshold for treatment initiation

Antihypertensive Medications

There are six major classes of antihypertensive agents:

- **A**, Angiotensin Converting Enzyme Inhibitors (ACEIs) and Angiotensin receptor blockers (ARBs);
- **B**, β-blockers (BBs);
- **C**, Calcium Channel Blockers (CCBs);
- **D**, Thiazide or thiazide-like diuretics;
- Aldosterone antagonists/ mineralocorticoid receptor amtagonsosts: spironolactone
- **Z**, others (sympatholytics, α adrenergic blockers, centrally acting alpha 2- agonists and direct arterial vasodilators.

This last class contains agents that are rarely used, or are obsolete, and examples are as follows:

- Sympatholytics and alpha adrenergic blockers e.g. methyldopa and prazocin
- Direct arterial vasodilators e.g. hydralazine

Choice of medications

The figure below describes how medication should be initiated and combined depending on the co-morbidities of the patient.

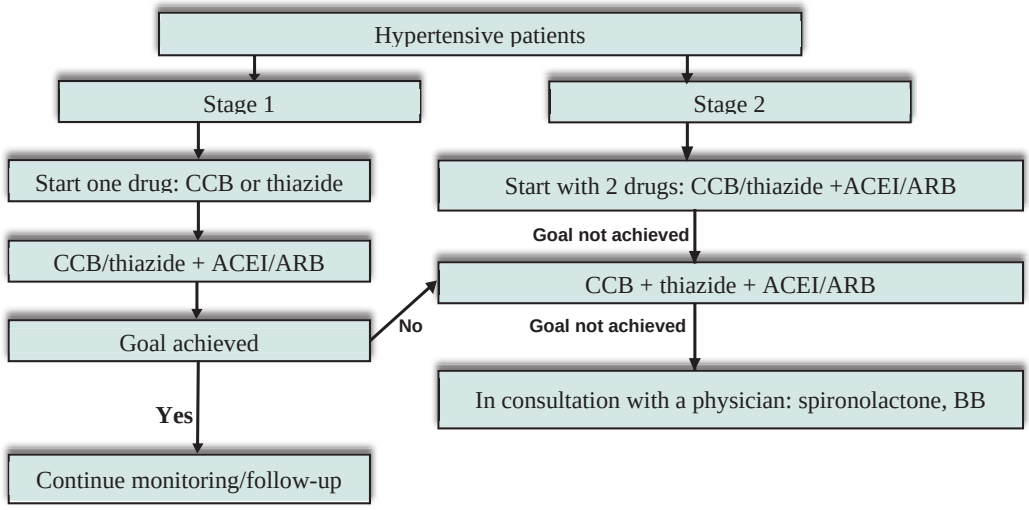

Figure 3:2 Choice of medications for lowering blood pressure

Combination therapy

Combination therapy including fixed dose combi- nations are recommended as it minimizes toxicity and therefore side effects as well as improves adherence to treatment. The recommended options for combinations therapy are shown in the figure beside:

Key: Green: Use, Red: Don't use, Black: consult a physician (Adapted from 2013 ESH/ESC Guidelines for the management of arterial hypertension).

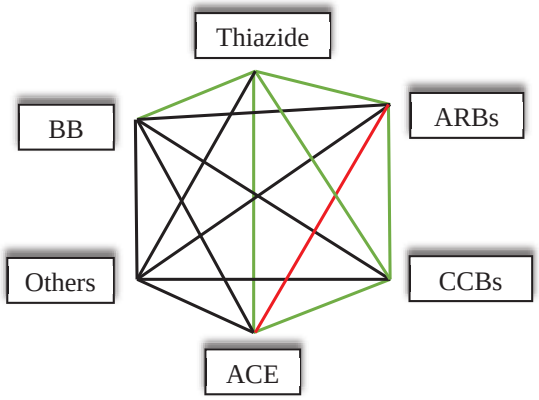

Figure 3.3: Choices for combination medications

The recommended dosages and common side effects of various antihypertensive medications is outlined below.

Table 3:7 Antihypertensive agents and their common side effects

Class	Examples	Usual monotherapy	Maximum daily dose	Possible side effects
Long-acting CCB	Amlodipine	5 mg OD	10 mg OD	• Oedema • Fatigue • Headache • Palpitations
	Felodipine	5 mg OD	10 mg OD	
	Nifedipine	Retard tabs: 10-20 mg BD LA tabs: 30 mg OD	Retard tabs: 30mg BD LA tabs: 90 mg OD	
Thiazide diuretic	Chlorthalidone	25 mg OD	50 mg OD	• Hypokalaemia • Hyponatraemia, • Hyperuricaemia • Hypocalciuria, • Hyperglycaemia • Rash • Dyslipidaemia
	Hydrochlorothiazide (HCTZ)	12.5 mg OD	25 mg OD	
Thiazide-like diuretic	Indapamide	2.5 mg OD	5 mg OD	
ACE inhibitor	Captopril	25-50 BD or TDS	50 mg TDS	• Cough (ACEI) • Hyperkalaemia • Increased serum creatinine • Angioedema
	Enalapril	5-20 mg daily in 1 or 2 divided doses	20 mg daily in 1 or 2 divided doses	
	Lisinopril	10 mg OD	40 mg OD	
	Perindopril	4 mg OD or 5 mg OD	8 mg OD or 10 mg OD	
	Ramipril	2.5 mg OD	10 mg OD	
Beta-blockers	Atenolol	25 mg OD	100 mg	
	Bisoprolol	2.5 mg OD	20 mg OD	
	Carvedilol	6.25mg BD	25 mg BD	
	Labetalol	100 mg BD	400 mg BD	
	Metoprolol succinate	25mg OD	100 mg OD	
	Nebivolol	5 mg OD	20 mg OD	
ARB	Candesartan	8 mg OD	32 mg OD	
	Irbesartan	150 mg OD	300 mg OD	
	Losartan	50 mg OD	100 mg OD	
	Telmisartan	40 mg OD	80 mg OD	
	Valsartan	80 mg OD	160 mg OD	

CCB: Calcium channel blocker; ACE: angiotensin converting enzyme; ARB: angiotensin receptor blocker
OD: administer once daily; BD: administer twice daily; TDS: administer 3 times daily

The table below outlines other less commonly used medications that prescribed by specialists depending on circumstances of the patient.

Table 3:8 Other anti-hypertensive medications

Other antihypertensive agents				
Class	**Examples**	**Usual monotherapy starting dose**	**Maximum daily dose**	**Possible side effects**
Centrally acting agents	Methyldopa	250mg BD or TDS	1000mg/day	• Angina • Orthostatic Hypotension • Gynaecomastia • Rash
	Clonidine	0.1mg BD	2.4mg/day	
	Phenoxybenzamine	0mg BD	40mg TDS	
Potassium sparing diuretics	Amiloride	5mg OD/ divided	10mg OD or divided dose	• Hyperkalaemia • Headache
	Triametrene	25mg OD or divided dose	100mg OD or divided dose	
Loop Diuretics	Torasemide	5 mg OD	20 mg OD	• Hyperuricaemia • Hypokalaemia
	Furosemide	20 mg OD	80mg OD or divided dose	
Vasodilators	Hydrallazine	25 mg BD or TDS	150 mg/day	Hypotension Palpitations
Alpha 1 Receptor Blocker	Prazosin	1mg BD-TDS	20mg/day	Hypotension, diarrhea, Tachycardia
	Terazosin	1mg OD	20mg/day	

Resistant hypertension

This is defined as BP ≥ 140/90 mmHg despite treatment with at least 3 drugs (including a diuretic) in adequate doses and after exclusion of false hypertension such as isolated office hypertension and failure to use large cuffs on large arms.

Table 3:9 Causes of resistant hypertension (8)

Category	Possible causes	Interventions
Non-adherence to therapy	• Instructions not understood • Side effects • Cost of medication and/or cost of attending at healthcare centre • Lack of consistent and continuous primary care • Inconvenient and chaotic dosing schedules • Organic brain syndrome (e.g. memory deficit)	• Adherence counselling • Ensure family/social support mechanism for the patient • Tailor dosing schedules to individual patients
Volume overload	• Excess salt intake • Inadequate diuretic therapy • Progressive renal damage (nephrosclerosis)	• Counsel on low salt diet, optimize diuretic therapy, refer as appropriate
Associated conditions	• Smoking • Increasing obesity • Sleep apnoea • Insulin resistance/hyperinsulinaemia • Ethanol intake of more than 30 g (three standard drinks) daily • Anxiety-induced hyperventilation or panic attacks • Chronic pain • Intense vasoconstriction (Raynaud's phenomenon), arteritis	• Manage the associated condition
Identifiable causes of hypertension	• Chronic kidney disease • Renovascular disease • Primary aldosteronism • Coarctation • Cushing's syndrome • Phaeochromocytoma	• Investigate and/or refer
Pseudoresistance	• 'Whitecoat hypertension' or office elevations • Pseudohypertension in older patients • Use of regular cuff in obese patients	• Out of office BP measurement • Ensure proper BP measurement technique
Drug-related causes	• Doses too low • Wrong type of diuretic • Inappropriate combinations • Rapid inactivation (e.g. hydralazine)	• Review treatment plan
Drug actions and	• Non-steroidal anti-inflammatory drugs (NSAIDs) • Sympathomimetics: nasal decongestants, appetite	• Remove offending drug, refer for specialist care

Follow-up for hypertensive patients on treatment

Initially, patients should be seen at 4 week intervals to assess antihypertensive efficacy, check for side effects and adjust medication as appropriate. The aim of therapy is to control BP without side effects. Patients should be advised to return earlier if they feel unwell or experience new symptoms (e.g., headache, persistent cough). Once goal BP has been achieved, the patient should be followed up every 4- 6 months.

NB:-in treating hypertension older age is defined as age greater or equal to 80 years.

What to do if goal BP is not achieved and when to step up therapy

i) Confirm that the patient is taking his/her medication as instructed (i.e., every day, the correct dose, the correct number of times per day, at the correct time of day). If necessary ask the spouse or another family member to confirm this information.

ii) If the patient has not been taking their medication as prescribed, determine reasons for this and address them appropriately.

iii) Ask about use of other prescribed medicines, over-the-counter medicines

Hypertensive crises and appropriate management and referral

Hypertensive emergencies: large elevations in SBP or DBP (>180mmHg or >120mmHg, respectively) associated with impending or progressive organ damage/dysfunction. They require immediate BP reduction (not necessarily to normal) to prevent or limit target organ damage. They must be managed on an inpatient with close monitoring and physician presence.

Hypertensive urgencies: are those situations associated with severe elevations in BP without progressive target organ dysfunction. The majority of these patients present as noncompliant or inadequately treated hypertensive individuals, often with little or no evidence of target organ damage. This can be managed in the outpatient setting by investigating the factors that may underlie the BP rise and dose adjustments as necessary.

Table 3:10 Hypertensive emergencies and urgencies (10)

Emergency	Management options
Acute ischemic stroke	Antihypertensive therapy is **not routinely recommended** for patients with acute stroke and HTN.
Acute Intracerebral Hemorrhage	BP lowering when the SBP is**>200mmHg** or the DBP is**>110mmHg.** If signs of **increased ICP**, maintain **MAP** just **below 130mmHg** (or **SBP <180mmHg**) for first 24 hours after onset. Patients **without increased ICP**, maintain **MAP < 110mmHg** (or **SBP < 160mmHg**) for first 24 hours after symptoms onset.
Subarachnoid Hemorrhage-	Maintain **SBP <160mmHg** until the aneurysm is treated or cerebral vasospasm occurs. Oral **nimodipine** is used to **prevent delayed ischemic neurological deficits**, but it is **NOT indicated** for treating acute hypertension.
Aortic dissection	**Immediately** reduce the **SBP < 110mmHg** and maintain it at this level unless signs of end-organ hypo perfusion are present. Preferred treatment includes a combination of; a) narcotic analgesics (morphine sulphate), b)β-blockers (labetalol, esmolol) or calcium channel blockers (verapamil, diltiazem); **Avoid β-blockers** if there is aortic valvular regurgitation or suspected cardiac tamponade.
Acute Coronary Syndrome-	Treat if **SBP>160 mmHg** and/or **DBP >100 mmHg**. Reduce BP by **20-30%**of baseline. Thrombolytics are **contraindicated** if BP is**>185/100 mmHg**. Preferred medications include **β-blockers**& **Nitroglycerin**
Acute Heart Failure-	Treatment with vasodilators (in addition to diuretics) for **SBP ≥140 mmHg**. **IV or sublingual nitroglycerin** is the preferred agent.
Preeclampsia/eclampsia	Prepartum and intrapartum: **SBP** should be **< 160 mmHg** and **DBP <110 mm Hg** If the **platelet count is <100,000 cells/mm, S**BP should be maintained below **150/100mmHg.** Patients with eclampsia or preeclampsia should also be loaded with **IV Magnesium sulphate 4gm**diluted in 100mL NS over 15 mins then with an **infusion of 2gm/hr** to avoid seizures. **Preferred medications**-Hydralazine,Labetalol, Nifedipine **Medications to avoid** -Nitroprusside, ACEIs, Esmolol

(Adopted from Emergency care algorithms, Kenya, 2017) (10)

Patient referral

The following patients require specialized care and should be referred if the requisite expertise is not available at the facility of initial contact:

- All pregnant women
- Pre-existing diabetes
- Fasting plasma glucose (FPG) indicates diagnosis of diabetes
- Heart failure
- BP >180 mmHg systolic and/or 110 mmHg diastolic BP
- Abnormal results on urine dipsticks or blood tests
- Patients not reaching goal BP after a reasonable trial of antihypertensive therapy (4 weeks)
- Hypertensive patients aged 18 years or younger
- Secondary cause of hypertension is suspected
- Associated clinical condition: coronary heart disease, heart failure, chronic kidney disease, stroke or transient ischaemic attack, peripheral arterial disease
- Consider referral for patients aged 80 years or older with a first diagnosis of hypertension

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3:9 Management of Hypertension in Children

Normal blood pressure values for children and adolescents are based on age, sex, and height based on standardized tables (1) available in annex. The table below details out the classification of hypertension in children based on the above criteria.

CLASSIFICATION	*SYSTOLIC OR DIASTOLIC BLOOD PRESSURE**
Normal	< 90th percentile
Prehypertension	90th to < 95th percentile or ≥ 120/80 mm Hg†
Stage 1 hypertension	95th to < 99th percentile plus 5 mm Hg
Stage 2 hypertension	> 99th percentile plus 5 mm Hg

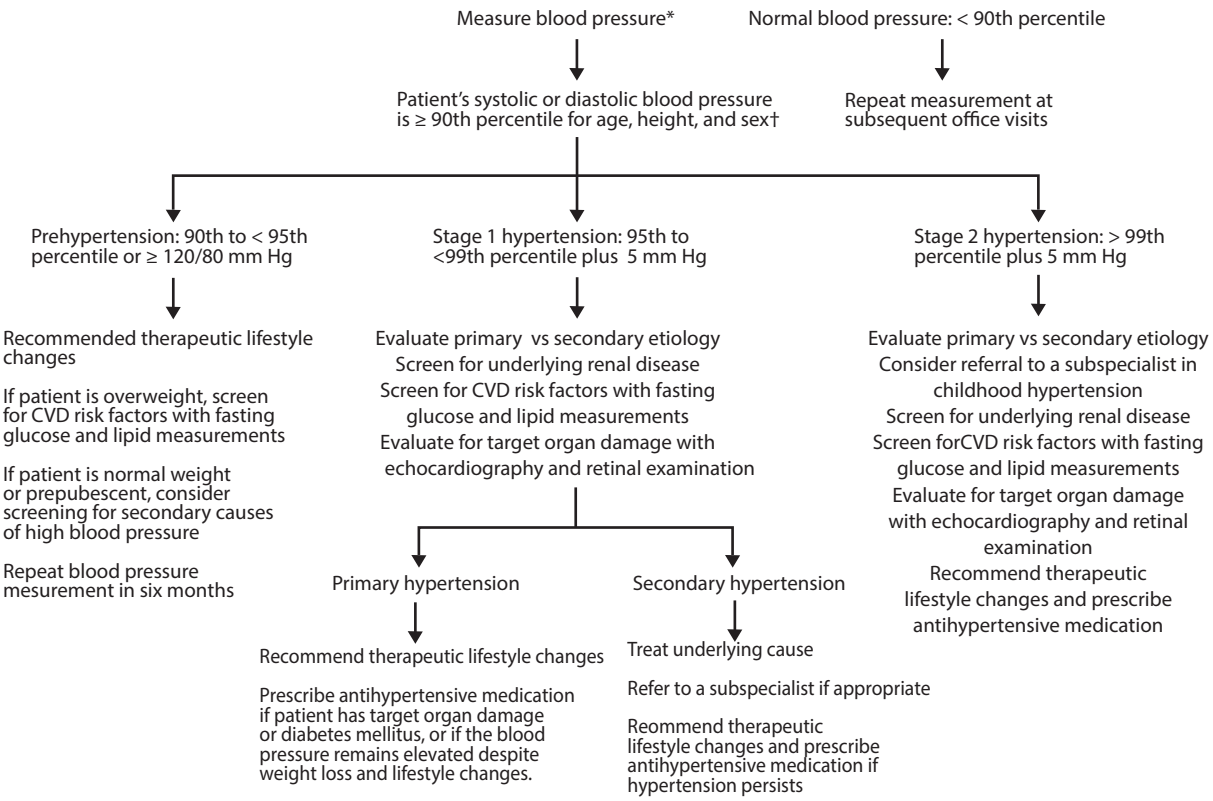
*—Based on sex, age, and height; measured on at least three separate occasions.
†—Blood pressure of 120/80 mm Hg or greater is prehypertension regardless of whether it is less than the 90th percentile. If 120/80 mm Hg is in the 95th percentile or greater, then the patient has hypertension.

Secondary hypertension is more common in children than in adults, with a renal aetiology being most likely (2).

Risk factors for childhood primary hypertension include:

- Overweight and obesity
- Family history of hypertension or CVD
- Male sex
- Maternal smoking during pregnancy

Algorithm for diagnosis of hypertension in children



**– Blood pressure should be mesured at every office visit beginning at tjhree years of age*
† –Remeasure during the same office visit, then confirm the elevation at three separate office visits.

Diagnosis and evaluation

a) Family history

Inquire about history of the following in the family: Hypertension, Cardiovascular and cerebrovascular Disease, Diabetes mellitus, Dyslipidaemia, Obesity, Hereditary renal disease (Polycystic kidney disease), Hereditary endocrine disease (pheochromocytoma, glucocorticoid-remediable aldosteronism, multiple-endocrine neoplasia type 2, von Hippel–Lindau), Syndromes associated with hypertension (neurofibromatosis)

b) Risk factor history

Physical exercise, dietary habits, Smoking, alcohol, Drug intake, Anti-hypertensives Steroids, cyclosporine, tacrolimus or other, Tricyclic anti-depressants, atypical antipsycotics, decongestants, Oral contraceptives, illegal drugs, Pregnancy

c) Perinatal history

Birth weight, gestational age, oligohydramnios, anoxia, umbilical artery catheterization

d) Previous medical history

Hypertension, Urinary tract infection, renal or urological disease, Cardiac, endocrine (including diabetes) or neurological disease, Growth restriction.

Symptoms suggestive of secondary hypertension

- Dysuria
- Thirst/polyuria
- Nocturia
- Hematuria
- Edema
- Weight loss
- Failure to thrive
- Palpitations
- Sweating
- Fever
- Pallor
- Flushing
- Cold extremities
- Intermittent claudication
- Virilization
- Primary amenorrhea
- Male pseudo-hermaphroditism

Symptoms suggestive of target organ damage

Headache, epistaxis, vertigo, visual impairment, Facial palsy, fits, strokes, Dyspnoea

e) Sleep history

Snoring, apnoea, daytime somnolence

f) Principles of physical exam

Findings are usually normal in most children with hypertension.

Body mass index should be calculated. Blood pressure measurement should be done in both arms while the child is seated and in one leg while the child is in a prone position. Blood pressure should be roughly equal in both arms and is normally 10 to 20 mm Hg higher in the leg.

Abnormal findings may indicate secondary aetiology as below:

- Coarctation of the aorta is suspected if leg blood pressure is lower than arm blood pressure, or if the femoral pulses are diminished.
- Abdominal bruit may point to reno-vascular disease such as renal artery stenosis
- Ambiguous genitalia is associated with mineralocorticoid excess.
- A low BMI and/or failure to thrive may indicate an underlying chronic illness.

Treatment

Treatment for hypertension in children should by initiated and periodically monitored by a paediatrician. Goal of treatment and drug choices in children (3):

- The 95th percentile is commonly used as a cut-off for defining hypertension in children and adolescents considering age, sex and height specific percentile
- Goal of treatment is to aim at a BP below the 90th percentile.
- In children with chronic kidney disease, it is recommended to achieve a stricter BP control preferably below the 50th percentile.
- Like in adults, choice of antihypertensive agents can include ACEIs, angiotensin receptor antagonists (ARBs), calcium antagonists, beta-blockers and diuretics. For dosages, reference should be made to relevant paediatric drug formularies.
- Lifestyle measures, where appropriate, should not only precede but also accompany pharmacological treatment.

Routine evaluation:

Evaluation should follow the adult algorithm previously discussed with special focus on excluding secondary causes of hypertension.

Ambulatory blood pressure monitoring can differentiate true hypertension from white coat hypertension, and can also determine the response to antihypertensive treatments

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4 Ischemic Heart Disease

4 Ischemic heart disease

List of Abbreviations

ACEI	Angiotensin Converting Enzyme Inhibitor
ACLS	Advanced Cardiac Life Support
ACS	Acute Coronary Syndrome
ARB	Angiotensin Receptor Blocker
BLS	Basic Life Support
BMI	Body Mass Index
BP	Blood Pressure
CABG	Coronary Artery Bypass Graft
CAD	Coronary Artery Disease
CT	Computer Tomography
CVD	Cardiovascular Diseases
CXR	Chest Xray
DVT	Deep Venous Thrombosis
ECG	Electrocardiogram
ER	Emergency Room
FBG	Fasting Blood Glucose
GERD	Gastro-esophageal Reflux Disease
HBA1c	Glycated Hemoglobin A1
HIV	Human Immunodeficiency Virus
HTN	Hypertension
IHD	Ischemic Heart Disease
LDL	Low Density lipoprotein
LVEF	Left Ventricular Ejection Fraction
NSAIDS	Non-steroidal Anti-inflammatory Drugs
NSTEMI	None-ST Elevated Myocardial Infarction
OGTT	Oral Glucose Tolerance Test
RBS	Random Blood Sugar
RV	Right Ventricle
SC	Subcutaneous
STEMI	ST Elevated Myocardial Infarction
TBC	Total Blood Count
UA	Unstable Angina
V/Q	Ventilation-perfusion Ratio
VF	Ventricular Fibrillation
VT	Ventricular Tachycardia

4:1 Introduction

Ischaemic heart disease (IHD), also known as coronary heart disease/coronary artery disease is the eventual manifestation of myocardial dysfunction following occlusion of the coronary arteries by cholesterol plaque.

4:2 Presentation of IHD

Patients with coronary artery disease can present acutely or insidiously with symptoms over months to years.

1. Stable disease

Stable angina is defined as myocardial ischaemia on exertion and relieved by rest in the absence of cardiomyocyte necrosis. The Canadian Cardiovascular Society Classification is usually used to grade angina as shown below:

Table 4:1 Canadian Cardiovascular Society Classification of Angina

Class I	**Angina with strenuous or prolonged exertion**
Class II	Angina on walking or climbing stairs rapidly, walking/stair climbing after meals, during the first few hours after awakening, walking more than 2 blocks on level or climbing more than one flight of ordinary stairs at a normal pace and conditions
Class III	Angina on walking 1 to 2 blocks on level or climbing one flight of stairs at normal pace and conditions
Class IV	Angina at rest

Unstable angina is defined as myocardial ischaemia at rest or minimal exertion in the absence of cardiomyocyte necrosis. Unstable angina can be classified according to Braunwald as shown below: -

Table 4:2 Braunwald classification of angina severity

Class/severity	Description
I	New onset severe angina, no rest pain
II	Angina at rest within preceding month, but not past 48 hours
III	Angina at rest within the preceding 48 hours

2. Acute Coronary Syndrome or Acute myocardial infarction (MI)

defines cardiomyocyte necrosis that is consistent with acute myocardial ischaemia.

3. Heart failure

Chronic ischemic heart disease may lead to heart failure.

4. Arrhythmia

Some patients may present with VT/VF commonly and less commonly with supraventricular arrhythmia such as atrial fibrillation. VF usually signifies acute disease whereas VT signifies scar in a previously infarcted territory.

5. Cardiac arrest and sudden cardiac death

Patients with severe disease affecting the left main stem or severe disease with a single remaining vessel may experience sudden cardiac death.

4:3 Diagnosis

Given broad spectrum of IHD, the presentation is quite varying. Therefore, a high index of suspicion is demanded from the clinician. Patients with chest pain and suspected angina should have full history and examination performed as part of their initial evaluation. This should include family history of coronary artery disease and sudden death.

Stable angina

The table below outlines the prediction tool developed by Diamond and Forrester.

Table 4:3 Angina risk prediction tool

Criteria **A: Sub-sternal chest pain with rest**	**B: Exertional chest pain**	**C: Chest pain relieved**	
Interpretation			
A: Typical angina (All 3 criteria)	**Age**	**Sex**	
		Male	**Female**
	30-39	Intermediate	Intermediate
	40-49	High	Intermediate
	50-59	High	Intermediate
	60-69	High	High
B: Atypical angina (2 criteria)	30-39	Intermediate	Low
	40-49	Intermediate	Low
	50-59	Intermediate	Intermediate
	60-69	Intermediate	Intermediate
C: Non-anginal chest pain (1 criteria)	30-39	Low	Low
	40-49	Intermediate	Low
	50-59	Intermediate	Low
	60-69	Intermediate	Intermediate
D: No criteria present	Risk is low for both men and women		
Interpretation			
	Risk	**Action**	
	Low	Investigate for non-cardiac causes	
	Intermediate	Perform/refer for stress testing	
	High	Perform/refer for coronary angiogram	

The following dynamic stress tests should be performed under supervision by an experienced cardiologist:

- Exercise ECG
- Exercise stress echocardiogram
- Dobutamine stress echo-cardiogram
- Nuclear Myocardial perfusion

Patients that have negative tests should be evaluated for other causes of chest pain.
Patients with borderline tests should be offered a CT coronary angiogram

Diagnosis of acute IHD

Acute IHD presents as acute coronary syndrome (ACS). The diagnosis of ACS is dependent of on 3 variables that include chest pain, ECG changes and elevated cardiac biomarkers. These can be utilized to score the patient to direct action.

Table 4:4 Heart Score for possible ACS

Parameter	Category	Points
History	Highly suspicious	2
	Moderately suspicious	1
	Slightly/Non- suspicious	0
ECG	Significant ST deviation	2
	Non-specific repolarization	1
	Normal	0
Age (Years)	≥65	2
	46-64	1
	≤45	0
Risk factors	≥3 risk factors or history of CAD	2
	1 or 2 risk factors	1
	No risk factors	0
Troponin levels	≥3× normal limit	2
	>1 to <3× normal limit	1
	≤ normal limit	0
Risk factors: DM, current/recent 91 month) smoker, HTN, hyperlipidaemia, family history of CAD and obesity		
Interpretation and action		
Score	**Action**	
0-3	Discharge home	
4-6	Admit for observation	
7-10	Perform/refer for invasive strategies	

ECG

In patients with chest pain the ECG should be performed within 10 minutes of presentation.

Biomarkers for ACS

The gold standard biomarker for the diagnosis of ACS is cardiac troponin. Most assays now use the highly sensitive troponin I and T.

Blood for troponin should be drawn immediately after the ECG is performed. Usually it takes approximately 4 hours after onset of symptoms before a rise in troponin can be elicited in the peripheral blood.

It is recommended that all patients should have troponin performed at the time of presentation, and if the initial test is negative and the patients has suspicious symptoms another test should be repeated in 4 hours. A rise in troponin usually suggests a coronary cause of chest pain. A negative troponin in two serial tests and a normal ECG should trigger evaluation for non-cardiac causes of symptoms but where the index of suspicion remains high, non-invasive evaluation should be considered.

Differential Diagnosis of Acute Chest Pain

The table below outlines the other possible diagnosis of acute chest pain and the diagnostic elements.

Table 4:5 Differential diagnosis for acute chest pain

Diagnosis	History	Physical exam	Diagnostic tests
Aortic dissection	Tearing pain radiating to the back	New murmur, bruits, unequal pulses	CXR,CT-Angiogram, Echo
ACS	Pressure-like pain radiating to the arms/face, diaphoresis, dysponea, risk factors	Evidence of heart failure	ECG, biochemical markers
Pulmonary embolism	Sudden onset, pleuritic pain, dyspnoea, risks for DVT	Tachypnea, tachycardia, DVT	CXR, V/Q scan, CT angiogram, pulmonary angiogram
Esophageal rupture	Constant severe retrosternal/epigastric pain, inciting event	Mediastinal rub/crunch	CXR
Pneumothorax	Pleuritic pain, dyspnea	Diminished breath sounds over hemi-thorax	CXR
Pneumonia	Cough, fever, dyspnea, pleuritic pain	Abnormal breath sounds, fever, hypoxia, tachypnea	CXR
Pericarditis	Positional ache, dyspnea	Rub	ECG, CXR, sonogram
GI causes	Associated abdominal/ GERD symptoms	Abdominal tenderness, guarding	Amylase, lipase, KUB, ultrasound
Musculoskeletal causes	Pain increased with minimal muscular activity or movement	Chest wall tenderness to palpation	Normal

Adapted from Green G, Hill P. Approach to chest pain and possible myocardial ischemia. Emergency medicine: A comprehensive study guide. New York: McGraw Hill; 2000: 341-352

4:4 Treatment of IHD

Acute Disease

Acute IHD presents as ACS. This is usually STEMI, NSTEMI or UA as defined above
The following are recommendations to institutions that offer care for patients with ACS

1. There should be clear point of contact for patients and health care professionals where help can be obtained immediately
2. There should be ambulances that are appropriately equipped to support ACLS care to patients with ACS
3. The ambulances should be equipped with an ECG machine
4. There should be a system that coordinates ambulances to enhance patient care
5. The triage system in the hospital should identify chest pain as one of the symptoms whose care is expedited
6. There should exist a link with cardiologist to enhance diagnostic capability and management of patients with acute coronary syndromes
7. There should exist a protocol for care for patients with ACS including care pathways, care bundles and care teams to expedite care for patients with ACS
8. There needs to have a collaborative effort that includes hospitals and cardiologists to improve care and outcomes for ACS patients

In the hospital

1. Patients with ACS should be evaluated in the acute room of the health facility
2. These patients should be connected to the ECG monitor and vitals assessed every 15 minutes.
3. There should be resuscitation trolley in all hospitals that take care of STEMI patients
4. There should be resuscitation teams in all hospitals that take care of STEMI patients
5. ECG should be performed and interpreted within 10 minutes of arrival to the hospital
6. ACS patients should managed by the EMERGENCY team in consultation with an attending cardiologist
7. The following tests should be performed on patients with ACS
    a. TBC
    b. Renal function and electrolytes
    c. RBS
    d. Portable CXR
8. On admission, the following tests should be considered
    a. OGTT/FBG
    b. HBA1c
    c. Lipid profile
    d. Thyroid screen
    e. HIV

9. Other tests should be performed depending on the complexity of the patient as guided by the clinical team. These include blood gas analysis in patient with severe dyspnea and lactate levels in patients with hypotension and shock.
10. Pain should be managed with morphine or morphine derivatives such as fentanyl. The recommended doses are as follows: -
    Morphine – 2.5-5mgmg iv or SC
    Fentanyl 25mcg -100mcg iv
    Caution is given in patients with respiratory distress where these drugs can cause respiratory failure and therefore airway support should be available
11. Drugs to avoid: These include NSAIDS and steroids. These have been shown to have deleterious effects in patients with ACS
12. Fluids: Patients with ACS should not be given fluids without a clear indication. This may precipitate heart failure in the vulnerable patient. Fluids are useful in a subset of patients with inferior MI and RV infarction. Consult the cardiologist on this.

STEMI

The treatment for STEMI underscore the need for system preparedness, anticipation and collaboration amongst different entities and institutions. Therefore, the patient must be identified early, diagnosis made swiftly and treatment/transfer offered immediately.
The following are recommended for patients with STEMI

1. There should a care box for STEMI patients that consists of the following drugs
    a. Aspirin 300mg orally stat
    b. Clopidogrel 300mg stat
    c. Enoxaparin 1mg/kgsc stat
2. Patient should be connected to ECG monitors immediately on arrival to the health facility
3. Oxygen therapy should be considered to those patients with saturation of less than 92% at room air. Routine oxygen utilization is not recommended
4. Intravenous access should be obtained as soon as possible once the patient is in the health facility
5. Thrombolysis should be administered within 30 minutes of diagnosis being made after consultation with the attending cardiologist.

STEMI should be treated in consultation with an interventional cardiologist. Where the cardiologist is not accessible, immediate transfer should be done to a facility with a cardiologist after administering oxygen as appropriate, aspirin and clopidogrel and heparin (LMWH).

NSTEMI

Patients with NSTEMI will require the same treatment as patients with STEMI, only that they do not require thrombolysis as it has not been shown to be beneficial.
The following recommendations are made:

1. There should a care box for NSTEMI patients that consists of the following drugs
   a. Aspirin 300mg orally stat
   b. Clopidogrel 300mg stat
   c. Enoxaparin 1mg/kg sc stat
2. Patient should be connected to ECG monitors immediately on arrival to the health facility
3. Oxygen therapy should be considered to those patients with saturation of less than 92% at room temperature. Routine oxygen utilization is not recommended
4. Intravenous access should be obtained as soon as possible once the patient is in the health facility
5. Prompt referral to a center with a cardiologist.

Unstable angina

1. There should a care box for NSTEMI patients that consists of the following drugs
   a. Aspirin 300mg orally stat
   b. Clopidogrel 300mg stat
   c. Enoxaparin 1mg/kg sc stat
2. Patient should be connected to ECG monitors immediately on arrival to the health facility
3. Oxygen therapy should be considered to those patients with saturation of less than 92% at room. Routine oxygen utilization is not recommended
4. Intravenous access should be obtained as soon as possible once the patient is in the health facility
5. An attending cardiologists should be informed immediately to guide care in the patient.

Treatment for chronic IHD

The purpose of treatment for chronic stable IHD is geared towards alleviation of symptoms and prevention of death
Treatment is therefore dependent on making of correct diagnosis using coronary angiography.
The medical management of stable IHD consist of the following

1. Statin
2. Aspirin
3. Short and Long acting nitrates
4. Beta blockers
5. If these are insufficient then the following can be considered for symptom control.
    a. Nicoradil
    b. Ranolazine
    c. Trimetazidine
    d. Procorolan

These patients should undergo coronary angiography to confirm diagnosis and determine extend and severity of disease.
Further treatment depends on severity of disease but medical therapy applies to all patients. This is guided by a cardiologist on an ongoing process.

Complications of myocardial infarction

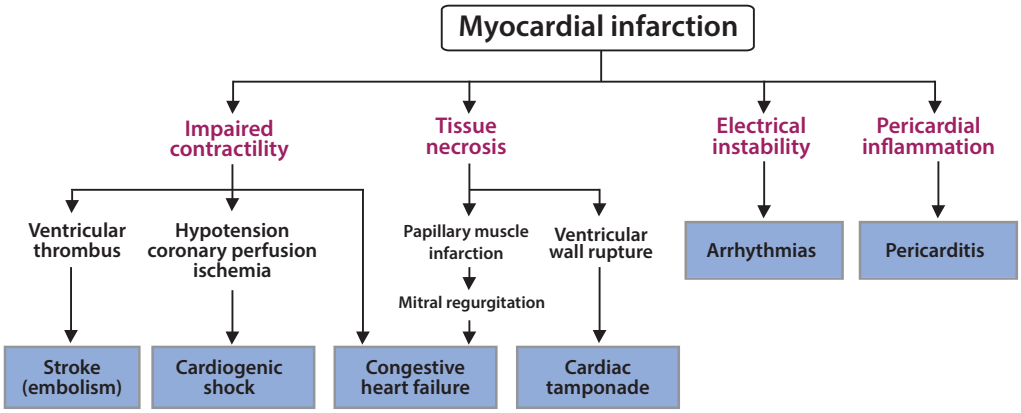

Figure 4.1: Common complications of IHD

Secondary Prevention of IHD

Secondary prevention of IHD begins in the hospital. Measures that have been shown to be useful include: -

1. Cardiac rehabilitation
2. Health education
   Physical activity - Thirty minutes of moderate intensity aerobic exercise at least five times per week is recommended.
   Dietary advise – for all patients with and without diabetes
   Medication adherence
3. Tobacco cessation. See details in chapter 2: Prevention of CVD
4. Aggressive risk factors modification
   - Blood pressure control – Aim for BP < 130/85. Avoid hypotension in patients with IHD. Medicine to be utilised include ACEI/ARB and beta blockers particularly in patients with impaired LV dysfunction. Other anti-hypertensives may be utilized to optimize BP.
   - Diabetes control – aim for HBA1c <7.1%
   - Body weight management– aim for normal BMI
   - Lipids – aim for LDL <2mmol/L. This should be achieved with high intensity statin and if necessary addition of ezetimibe. Newly licensed PCSK9 inhibitors may playrole in patients that do not achieve this target.
   - Anti-platelets - Aspirin for is recommended for all patients for secondary prevention of IHD. Patients that are intolerant to aspirin should be given clopidogrel.
5. Management of heart failure
   Patients with IHD require treatment for heart failure and specialized heart failure clinics are recommended.
6. Use of devices
   Patients that survive acute myocardial infarction and have heart failure may require primary prevention ICDs.

Health Systems Recommendations for the Management of IHD

These guidelines come to fore at a time when competing interests of disease burden and resource allocation abound. The writers of the guidelines therefore felt there is a great need for system organization to optimize care and resource allocation. In order to do this the following recommendations are suggested in order to help health care organisations deliver the best care to their patients

1. **Shared resources:**

Health institutions should plan to share resources in order to reduce pressure on resources and also to concentrate expertise. Most benefit is achieved through having shared ambulances and ambulance systems, shared critical areas (HDU/ICU/CCU), cath labs and diagnostic and rehabilitation centres. Systems of transfer can be put in place and health system exchanges optimized in order to improve care.

2. **Shared manpower:**
   By sharing resources then sharing of manpower becomes easier. Therefore experts will be located in given centres where patient care be optimized. These include physicians, nurses and other health care professionals.
3. **Medical records:**
   By sharing health records in a secure and confidential platform health care systems can optimise patient care and save meagre resources

Screening for asymptomatic IHD

Generally there is no blanket screening for people for occult IHD. However, in some people this becomes of essence for a variety of reasons.
Populations for whom screening for asymptomatic IHD is recommended include:

1. People with abnormal ECG – particularly pathological Q-waves and ST depression and have risk factors for CAD
2. People with diabetes
3. People with strong family history of IHD presenting with atypical symptoms
4. People with 3 or more cardiovascular risk factors who are due for major surgery
5. People over 50 years who are to undergo heart surgery or over 40 years and with two CVD risk factors that require heart surgery e.g. mitral valve replacement
6. People being worked up for major organ transplant e.g. renal transplant, lung or liver transplant
7. People with heart failure of unclear aetiology may require screening for IHD
8. People with VT/VF
9. Resuscitated cardiac arrest

Testing for Asymptomatic IHD

The best test is chosen depending on the patient's primary diagnosis and underlying comorbidities and locally available resources. An experienced cardiologist should be involved in testing of these patients in view of the risk for missing disease and prescription of more expensive downstream tests
The following tests are recommended: -

1. Exercise ECG
2. Exercise stress echocardiogram
3. Dobutamine stress echocardiogram
4. CT calcium score and CT coronary angiogram
5. Nuclear myocardial perfusion study
6. Cardiac MRI with gadolinium enhancement
7. Invasive coronary angiography

Lab tests for patients with IHD

In patients with ACS the most important laboratory tests have been specified above. Other tests may be required in complex patient scenarios.
However, in chronic IHD tests performed are geared towards consolidating secondary prevention and evaluating for complications. These include renal function tests and electrolytes, TBC, random lipid profile (in contrast to fating), FBG or OGTT if necessary and HBA1c. Urinalysis should be performed in patients with diabetes and hypertension.

Other tests for patients with IHD

The single most important non-laboratory test for patients with IHD is an echocardiogram. An echocardiogram is key is showing the LVEF and LV dimensions. Patients with ACS require an echocardiogram before leaving hospital to assess LV function. An echocardiogram is generally not recommended for the diagnosis of ACS as it lack sensitivity and specificity but can be used in patients as part of evaluation of chest pain differentials and for evaluation of complications of MI.
Patients with reduced EF and IHD are recommended to have complete revascularization as this can potentially improve LV function. Studies have showed that the best improvement in these patients is achieved through CABG.

4:5 Management of Cardiac arrest in IHD

Many deaths occur early during the first few hours after STEMI, due to ventricular fibrillation (VF). Since this arrhythmia occurs most frequently at an early stage, these deaths usually happen out of hospital. Therefore, it is crucial that all medical and paramedical personnel caring for suspected myocardial infarction have access to defibrillation equipment and are trained in cardiac life support and that, at the point of first medical contact, ECG monitoring be immediately implemented in all patients with suspected myocardial infarction.

Care providers in ambulances and hospitals managing ACS patients should be ACLS certified. It is recommended for this guideline that ACLS re-certification should be performed every 5 years.
Hospitals taking care of ACS patients should put in place methods to ensure staff certification and re-certification in BLS and ACLS through work plan schemes that should be freely available.

IHD in special populations

Women tend to present late and may have somewhat atypical symptoms more frequently than men. Yet myocardial infarction remains a leading cause of death in women and it is therefore important to maintain a high degree of awareness for myocardial infarction in women with potential symptoms of IHD.

Elderly patients often present with atypical or mild symptoms, which may result in delayed or missed diagnoses of MI. The elderly are at particular risk of bleeding and other complications from acute therapies because bleeding risk increases with age, renal function tends to decrease and prevalence of co-morbidities is high.

Renal dysfunction is present in approximately 30–40% of patients with ACS and is associated with a worse prognosis and increased bleeding risk. In patients with known or anticipated reduction of renal function, several antithrombotic agents should be either withheld or their doses reduced appropriately.

Diabetic patients are at higher risk of death and complications, but selection of antithrombotic therapies and reperfusion therapy is the same as in non-diabetics.
Hyperglycaemia on admission is common in patients with an ACS and is a powerful predictor of mortality and in-hospital complications. In the acute phase, it is reasonable to manage hyperglycaemia (i.e. maintain a blood glucose concentration ≤11.0 mmol/L) but absolutely avoid hypoglycaemia. This may require a dose-adjusted insulin infusion with monitoring of glycaemia in some patients.

Given the frequency of unrecognised diabetes and impaired glucose metabolism in STEMI patients, it is reasonable to measure HbA1c and fasting blood glucose in all patients without known diabetes, who developed hyperglycaemia during the acute phase.

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5 Heart Failure

5 Heart Failure

List of Abbreviations

CAD	Coronary Artery Disease
CathLab	Cardiac Catheterization Laboratory
HF	Heart Failure
ECG	Electrocardiogram
HFpEF	Heart Failure with Preserved Ejection Fraction
HFrEF	Heart Failure with Reduced Ejection Fraction
HTN	Hypertension
ICD	Implantable Cardioverter Defibrillator
JVP	Jugular Venous Pressures
LVEF	Left Ventricular Ejection Fraction
MR	Mitral Regurgitation
MRA	Mineralocorticoid Receptor Anatagonist
MS	Mitral Stenosis
NTproBNP	N-Terminal Pro-Brain Natriuretic Peptide
PND	Paroxysmal Nocturnal Dyspnea
RHD	Rheumatic Heart Disease
SCD	Sudden Cardiac Death
SOBOE	Shortness of Breath on Exertion

5:1 Introduction

HF is a clinical syndrome which may be caused by varied underlying conditions including hypertension, valvular disease including rheumatic heart disease, coronary artery disease or primary myocardial disease. It has typical symptoms, which may be accompanied by signs resulting from abnormality in cardiac structure and function, resulting in a reduced cardiac output and/or elevated intra-cardiac pressures (1).

5:2 Burden and public health implications

It is estimated that HF affects over 26 million people worldwide (1). The magnitude of HF in Kenya is not well established due to lack of population-based data, but the prevalence worldwide is around 1-2%. Among hospital patients, HF is the 3rd most common cause of acute admission at about 8% (2). The prevalence of HF depends on the burden of comorbidities but increases with age (3).

In Sub-Saharan Africa (SSA), unlike in high-income countries, HF is predominantly caused by hypertension, dilated cardiomyopathies and rheumatic heart disease (4). Right heart failure due pulmonary hypertension secondary to non-smoking related chronic lung disease and HIV-related disease, remain other significant causes of heart failure in the region(5). HF has a prognosis equivalent to many cancers with a 5-year mortality of 50% (3).

5:3 Aetiology

Table 5:1 Common morphological changes and their causes of heart failure

Broad category	Morphological findings	Common aetiology
Hypertrophic	Left ventricular hypertrophy (LVH) – concentric.	Hypertensive heart disease with normal or with reduced left ventricular ejection fraction, coarctation or aortic stenosis.
	LVH – severe concentric >15 mm, (out of proportion with hypertension) or asymmetric.	Hypertrophic cardiomyopathy (HCM).
Dilated	Dilated cardiomyopathy. Classically dilated spherical left ventricle with reduced ejection fraction. In come conditions with regional changes	- Unknown cause (idiopathic) - Associated with HIV, alcohol, anthracyclines. Viral or autoimmune myocarditis/reactive
	Reduced EF with regional changes. Often dilated.	- Ischemic cardiomyopathy
Right ventricular	Right ventricular thinning and reduced RVEF	- Arrhythmogenic right ventricular cardiomyopathy (ARVC)
	Right ventricular dilatation and/or hypertrophy in the setting of pulmonary hypertension	- Secondary to LV dysfunction - Chronic pulmonary disease - Acute or chronic PE - Primary pulmonary arterial hypertension - PHTN associated with other systemic diseases (autoimmune, HIV, infiltrative, toxic)
	RV dilation and/or RV hypertrophy. Large left atrium ± smoke, and small left ventricle.	Rheumatic mitral stenosis
Congenital heart disease	Enlarged RA and RV	Atrial septum defect
	Enlarged LV and LA, if uncorrected also RV and RA dilation.	Ventricular septum defect -
	Highly abnormal anatomy - ± cyanosis	Complex congenital disease

5:4 Prevention and screening

Development of HF can be delayed or prevented by treating risk factors for HF early. Many trials show that control of hypertension will delay the onset of HF and some also show that it will prolong life (6).

The mainstay of prevention is screening for and treating the underlying conditions such as cardio-metabolic conditions (diabetes, hypertension, dyslipidaemia) and avoiding acute rheumatic fever. Combating behavioural risk factors such as tobacco use, alcohol use, sedentary lifestyle and unhealthy diet also play an important role.

Major international society guidelines (AHA/ACC/ESC) recommend Initiation of an ACEI, a beta-blocker and a mineralocorticoid receptor antagonist(MRA) immediately after a myocardial infarction or in all causes of HF with reduced LVEF and this forms the cornerstone of HF treatment and reduces the rate of HF hospitalization and mortality (7–9).

Recommendations to prevent or delay the development of overt heart failure or prevent death before the onset of symptoms is considered highly cost-beneficial both for the individual patient, but also reduces the overall cost of healthcare in HF patients. These mainly address modifiable risk factor reduction such as control of hypertension and diabetes, treatment of dyslipidemias, weight reduction, smoking cessation, avoidance of excessive alcohol ingestion and emotional stress.

The classification of HF from the aspect of objective assessment of the patient at risk of developing HF or with manifest HF sensitizes health personnel to treat risk factors that may lead to the development of HF at an early stage.

Class	Objective Assessment
A	No objective evidence of cardiovascular disease. No symptoms and no limitation in ordinary physical activity.
B	Objective evidence of minimal cardiovascular disease. Mild symptoms and slight limitation during ordinary activity. Comfortable at rest.
C	Objective evidence of moderately severe cardiovascular disease. Marked limitation in activity due to symptoms, even during less-than-ordinary activity. Comfortable only at rest.
D	Objective evidence of severe cardiovascular disease. Severe limitations. Experiences symptoms even while at rest.

5:4 Diagnosis

Symptoms in HF are often non-specific and do not, therefore, help discriminate between HF and other problems. Symptoms may also fluctuating depending on fluid status. Signs, such as elevated JVP and S3 may be difficult to reproduce. Symptoms and signs are particularly difficult to interpret in obese individuals. In our setting HF often have a more varied aetiology, clinical presentation and outcome warranting a higher index of suspicion (1). Once a diagnosis of HF has been established, the physician should always ask the question – "why HF?", as specific HF aetiologies have very different prognosis, and sometimes treating the underlying factor may completely reverse the syndrome.

Table 5:2 Symptoms and signs of HF

Symptoms	**Signs**
Typical	**More specific**
• Breathlessness • Orthopnea • Paroxysmal nocturnal dyspnea • Reduced exercise tolerance • Fatigue, tiredness, increased time to recover after exercise • Ankle swelling	• Elevated jugular venous pressure • Hepatojugular reflex • Third heart sound (gallop rhythm) • Lateral displaced apical impulse
• Less typical symptoms	• Less specific
• Nocturnal cough • Wheezing • Bloated feeling • Loss of appetite • Confusion (especially in the elderly) • Depression • Palpitations • Dizziness • Syncope • Bendopnea (dyspnea on bending over)	• Weight gain(>2kg/week) • Cardiac murmur • Peripheral edema (ankle, sacral, scrotal) • Pulmonary crackles • Dullness on percussion at the lung bases (pleural effusion) • Tachycardia • Irregular pulse • Tachypnoea • Cheyne-Stokes respiration (late) • Hepatomegaly/Ascites • Cold extremities • Oliguria • Narrow pulse pressure

5:5 Heart failure diagnosis

The diagnosis of HF-REF requires three conditions to be satisfied:
1. Symptoms typical of HF
2. Signs typical of HF
3. Reduced LVEF
The diagnosis of HF-PEF requires four conditions to be satisfied:
1. Symptoms typical of HF
2. Signs typical of HF
3. Normal or only mildly reduced LVEF and LV not dilated
4. Relevant structural heart disease (LV hypertrophy/LA enlargement) and or/or diastolic dysfunction (see Sectoin 4.1.2)

HF=heart failure; HF-PEF = heart failure with 'preserved' ejection fractions; HF-REF= heart failure and a reduced ejection fraction; LA = left atrial; LV = left ventricular; LVEF = left ventricular ejection fraction.
[a]Signs may not be present in the early stages of HF (especially in HF-PEF) and in patients treated with diuretics.

Figure 5.1: Distinction of diagnosis between HF-REF and HF-PEF

1. Clinical history – History of common risk factors (HTN, RHD or CAD), family history of cardiac disease or SCD. Exposition to cardiotoxicity (alcohol, drugs/radiation). Typical symptoms Shortness of breath on exertion. Good response to diuretic therapy.
2. Physical examination – On auscultation: Bilateral crackles, third heart sound, murmurs. Signs of RV failure: Elevated JVP, hepatomegaly/ascites or bilateral pedal oedema.
3. 12-lead ECG
4. Echocardiography
5. (Natriuretic peptides: NTproBNP or BNP) – useful in HFpEF when no clear echocardiographic signs of HF have been established.

Investigations

The specific tests chosen may depend in part upon the patient presentation, cost and equipment available. These investigations are meant to determine the cause of HF as well as evaluate for precipitants for acute decompensation.

Chest radiograph

Important radiographic abnormalities associated with HF include pulmonary vascular redistribution, cardiomegaly, pleural effusions and interstitial oedema. However in clinical practice the chest x-ray will more often be used to rule out differential diagnoses like pneumonia, COPD or pulmonary TB as illustrated below. In children cardiomegaly/cardiac shadow.

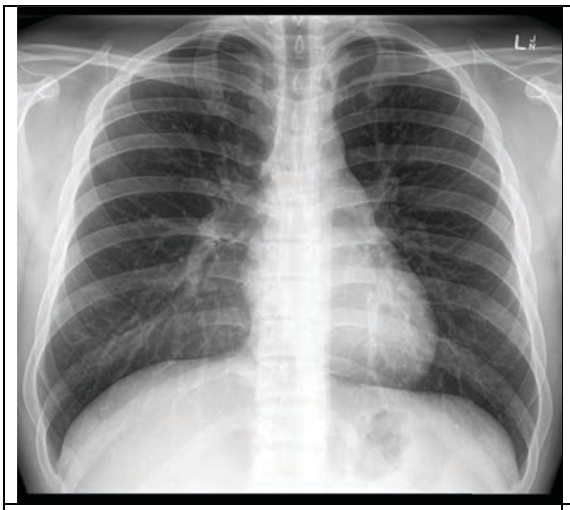	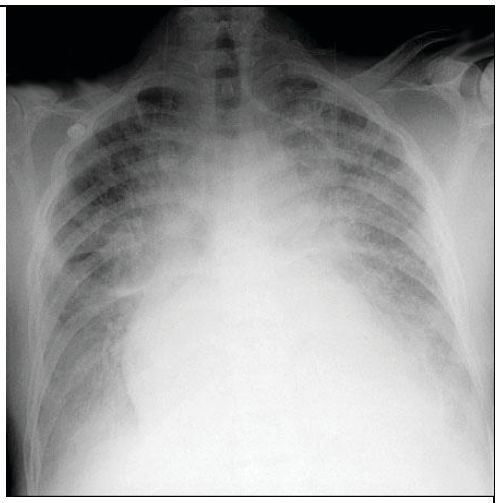
Normal chest radiograph.	Chest radiograph of a patient with heart failure. • Note the enlarged cardiac shadow(cardiomegaly),increased vascular markings, blunting of the costo-phrenic and cardio-phrenic angles.

Figure 5.2: Chest radiograph showing normal and abnormal findings

Electrocardiogram (ECG)

A normal ECG makes the diagnosis of HF highly unlikely. However, ECG alone cannot be used to confirm the diagnosis of HF. Major features on the ECG seen in patients with HF include poor r-wave progression, axis deviation, atrial fibrillation, bundle branch blocks, left or right ventricular hypertrophy and pathological Q-waves.

Echocardiography

Echocardiography is the Gold standard test for characterizing cardiac dysfunction in HF as it: -

- Determines systolic LV performance through determining left ventricular ejection fraction (LVEF), cardiac output, chamber quantification and regional wall motion abnormalities.
- Determines end-diastolic LV filling pressures for assessment of diastolic dysfunction and systolic pulmonary artery pressure to diagnose pulmonary hypertension.
- identifies clinically important valvular and pericardial disease.

Laboratory investigations

Other laboratory investigations are required both to assist in the diagnosis of HF, evaluate for underlying aetiologies, and to monitor patients' clinical status during therapeutic interventions.
At baseline these should include:

- Natriuretic peptides (BNP or NT-proBNP): Are useful in both diagnosis with very high sensitivity for ruling out HF at the lowest cut off point.
- Full blood count: HF due to, or aggravated by, anaemia or infections; HF associated anaemia which is associated with poorer prognosis.
- Urinalysis: proteinuria due to nephropathy/nephritic syndrome, or red blood cells/casts due to glomerulonephritis.
- Serum urea, electrolytes and creatinine (UECs): renal dysfunction, a major determinant of disease progression. (baseline)
- Serum albumin: rule out oedema secondary to low serum albumin in nephrotic syndrome(baseline)
- Lipid profile, thyroid function tests (TFTs): reveal potential cardiovascular or thyroid disease as a cause of HF. (baseline)
- Liver function tests: may indicate liver dysfunction in liver failure or disease progression in liver congestion. (baseline)
- Fasting blood sugar or HbA1c: Type 2 diabetes mellitus is more common in HF.
- Thyroid Function test,
- Blood cultures: Are useful in suspected infectious endocarditis.
- Serum magnesium and calcium: Low levels may precipitate reversible conditions like malignant arrhythmias (Torsade de Pointes) or poor contractility respectively.
- Troponin T or I: Out of proportion levels may help guide the whether to do further investigations for ischemic aetiology.

Functional classification of heart failure

The New York Heart Association (NYHA) Functional Classification is based on the degree of limitation of the patient's day-to-day activities of living and is a strong predictor of hospitalization and mortality. The American College of Cardiology/American Heart Association (ACC/AHA) staging is an important tool to identify patients at risk of developing HF.

Table 5:3 NYHA classification of HF

NYHA Functional Classification	
None	
I	No limitations of physical activity. No HF symptoms
I	No limitations of physical activity. No HF symptoms
II	Slight limitations of physical activity. Comfortable at rest, but ordinary results in HF symptoms
III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes HF symptoms
IV	Unable to carry on any physical activity without HF symptoms

5:6 Management

Management interventions for patients with heart failure commonly involve acute care, patient education, lifestyle intervention, pharmacological therapy and more advanced intervention such as device therapy.

Acute Care

This is indicated for decompensated or exacerbation of chronic HF (NYHA III and IV). Exacerbations may occur as a result of cardiac problems, treatment non-compliance and co-morbidities. Cardiac issues that may worsen HF include ischaemia, arrhythmias (most commonly AF) and valvular dysfunction. Patient's non-compliance refers to cessation of medicines (especially furosemide), non-adherence to salt and fluid restriction. Treatment involves:

Oxygen

During decompensation, oxygen administration in semi-recumbent position will relieve symptoms of dyspnoea and increase tissue oxygen delivery. On occasions, oxygen therapy may have independent beneficial effects, for example in myocardial ischaemia if SpO2<90%.

Diuretics

Loop diuretics, such as furosemide, are the mainstay medications as they result in increased fluid excretion. Patients will often require an increase in their usual oral or intravenous dose of furosemide to clear the fluid overload. Intravenous dosage plays a critical role in acute management due to the occurrence of gut wall oedema that limits absorption of oral medication. Short-term thiazide diuretics (including metolazone) may be used in instances of furosemide resistance to augment diuresis.

Caution must be exercised to avoid excessive diuresis leading to hypovolaemia and its consequences (acute renal failure, and electrolyte imbalance).

Regular assessment of hydration status and monitoring of UECs are therefore necessary.

Vasodilators

Nitrates are predominantly venodilators and often relieve symptoms of pulmonary congestion, particularly at night when the heart is exposed to increased filling pressures due to the recumbent position.

Long-term vasodilators, such as ACEIs and ARB's can be continued, increased or added throughout the tretament period, particularly if blood pressure is elevated.

Beta-blockers

These should not be commenced or increased during the acute decompensation episode, as the acute effect of these agents at a time of fluid overload may worsen clinical status.

Inotropic agents

Dobutamine, norepinephrine and dopamine and other inotropic agents have not been shown to decrease mortality and should only be used when there is protracted hypotension

Multidisciplinary care team

Cardiac rehabilitation and palliative care, must be integrated into the overall provision for patients with HF. Fundamental to the delivery of this complete package of care are multidisciplinary management programs designed to improve outcomes through structured follow-up with patient education, optimization of medical treatment, psychosocial support and improved access to care. Such strategies reduce HF hospitalization and mortality in patients discharged from the hospital (10). These are class I recommendations.

Patient education with the intent to promote self-care is related to the maintenance of the appropriate level of physical and psychological well-being, decrease in morbidity and mortality and in the use and cost of healthcare, increase in patient's satisfaction, improvement of control sense and life quality.

Discharge planning, the vulnerable period and follow up

Discharge planning should commence as soon as the patient's condition is stable. Hospitals with early physician follow-up after discharge show reduced 30-day readmission (1,3,6).

Self-Care and Symptom recognition

- Provide educational material that helps the patient understand their condition and related symptoms.
- Sodium and fluid management with 3 to 6-montly UECs and preferably weekly/ daily weight is a proven cost-effective way of avoiding undue hospitalization for worsening HF, hyponatremia and reduces mortality.
- Patients should weigh themselves once to twice a week, preferably daily and at the same time of day.
- Patient should seek medical help with worsening symptoms such as weight gain, shortness of breath, peripheral edema, dizziness or fainting episodes.

Adherence to drug treatment

- All patients should know all their drugs and keep a list of what there are actually taking whenever visiting a physician.
- Patients should be asked for (any adverse side effects, non-adherence, concurrent ingestion of traditional/herbal drugs).
- Advised about the use of analgesics, particular against the use of nonsteroidal anti-inflammatory drugs (NSAIDs).

Smoking

- All patients should be asked if they smoke, and if so, strongly encouraged to quit.

Diet, nutrition and fluids

- Strict fluid restriction in patients with mild to moderate heart failure does not appear to confer clinical benefit. However, patients should be advised to avoid excessive fluid intake (recommended daily fluid intake less than1.5day) including fruits.
- Patients should restrict foods high in salt by avoiding commercially processed foods, avoid the use of table salt and restrict salt when cooking, but should also have serum-sodium measured 3-monthly if there was a previously low reading.
- Patients should avoid alcohol intake and be encouraged to eat a varied balanced diet high in whole grains, vegetables and fruit.

Physical activity/Cardiac Rehabilitation

- Exercise training improves exercise tolerance, health-related quality of life and HF hospitalization rates in patients with HF.
- Ideally, the exercise programme should be supervised by cardiac rehabilitation team and individualized to every patient. If this is not possible, patients are encouraged to participate in activities of daily living with regular follow-up by primary health provider.

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Table 5:3 Contraindications to exercise in heart failure

Absolute contraindications
• **NYHA class IV** • **Progressive worsening of exercise tolerance or shortness of breath at rest or on exertion over the prior 3-5 days;** • **Significant ischaemia at rest;** • **Uncontrolled diabetes;** • **Acute systemic illness or fever;** • **Recent embolism;** • **Thrombophlebitis;** • **Active pericarditis or myocarditis;** • **Severe aortic stenosis;** • **Regurgitant valvular heart disease requiring surgery;** • **Myocardial infarction within previous 3 weeks;** • **New-onset atrial fibrillation**

5:7 Pharmacotherapy

This is dependent on the LVEF:

- HF with reduced ejection fraction (HFrEF):
  HF with LVEF<40%. (From a clinical perspective, it is reasonable to include patients with LVEF 40 – 50% in this group).
- HF with preserved ejection fraction (HFpEF):
  HF with LVEF ≥50%.
- identifies clinically important valvular and pericardial disease.

Pharmacotherapy of HFrEF

Management algorithm or HF patients with reduced LVEF

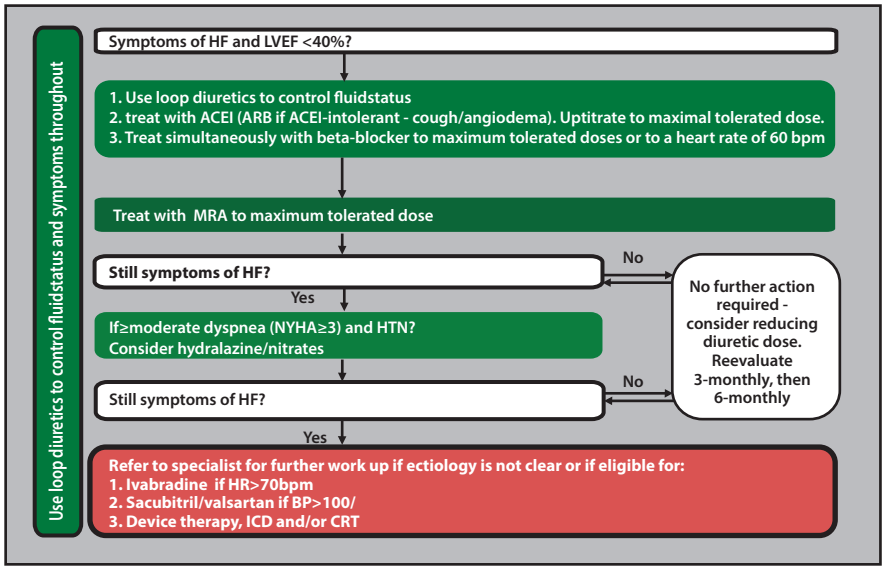

Figure 5.3: HF algorithm for symptomatic HFrEF. Modified from the HFA/ESC Guidelines 2016(1). Symptomatic = NYHA Class II-IV.

For patients with a diagnosis of HF with reduced LVEF <40% (HFrEF), pharmacotherapy should in most instances entail the use of ACEI or if ACEI-intolerant, ARBs, beta-blockers; and aldosterone antagonists. Ivabradine which exclusively inhibits the sinus node without any lowering of the BP can be used as an adjunct to beta-blocker in any patient with a heart rate >70 bpm in sinus rhythm where beta-blocker is either contraindicated, or the dose cannot be increased due to hypotension

Only after having fully optimized these dosages, and after careful evaluation by a cardiologist (1), may further therapies including the use of angiotensin receptor-neprilysin inhibitors (so-called ARNIs) be considered in patients with systolic BP >100 mmHg instead of RAS blockade.

Recommended Pharmacotherapy for patients with heart failure and normal LVEF

A significant amount of patients (up to half in other populations) with HF will have relatively normal or subnormal (LVEF ≥50%); so called - HF with preserved LVEF. As no evidence based management of these patients exists, treatment remains individualized targeting the underlying cause, most often controlling risk factors such as hypertension, obesity, obstructive sleep apnea (OSA) in order to lower left ventricular end-diastolic pressures. A substantial amount of patient will also have HF secondary to isolated valvular disease, either due to rheumatic valvular heart disease, infectious endocarditis or pericardial disase. This patient group will not benefit from the treatment outlined above.

If no high-level echocardiography is available, these patients should be referred to a cardiologist

Table 5:4 Pharmacotherapy dosage for disease-modifying drugs in HF with reduced ejection fraction

	Starting dose	Target dose
ACEI		
Enalapril	2.5 mg b.id.	10 – 20 mg b.i.d.
Ramipril	1.25 mg b.i.d.	5mg b.i.d.
Lisonopril	2.5 – 5 mg o.d.	20 – 35 mg o.d.
Beta-blockers		
Bisoprolol	1.25 mg o.d.	10 mg o.d.
Carvedilol	3.125 mg b.i.d.	25 mg b.i.d.
Metoprolol Succinate	12.5 – 25 mg b.i.d.	200 mg o.d.
Nebivolol*	1.25 – 2.5 mg o.d.	10 mg o.d.
ARBs		
Candesartan	4 mg o.d.	32 mg o.d.
Losartan	50mg o.d.	150mg o.d.
Valsartan	40 mg o.d.	160 mg o.d.
MRAs		
Eplerenone	25 mg o.d.	50 mg o.d.
Spironolactone	12.5mg o.d.	25 – 50 mg o.d.
If-channel inhibitor		
Ivabradine	5 mg b.d.	7.5 mg b.d.
ARNI		
Sacubitril/valsartan	49/51 mg b.i.d.	97/103 mg b.i.d.

***Has not been shown to reduce mortality.**

Table 5:5 Pharmacotherapy dosages for diuretics used in HF.

Diuretics	Starting dose	Usual dose
Loop diuretics		
Furosemide	20 – 40 mg	40 – 240 mg o.d.
Torasemide	5 – 10 mg	10 – 20 mg o.d.
Thiazides		
Hydrochlorthiazide	25 mg	12.5 – 100 mg o.d.
Metolazone	2.5 x 2/week	2.5 – 10 mg o.d.
Others		
Hydralazine/ISD(M)N	25/10 mg t.d.s.	50/20 mg t.d.s.
Digoxin	0.0625 mg o.d	0.125 mg o.d.

Table 5:6 Clinical consideration when using HF medication.

Drug class	Clinical use considerations
Loop diuretics	Use as first line therapy for immediate volume and symptom control in the acute decompensated patient. Monitor renal function and electrolytes (including calcium and magnesium) frequently especially with IV administration. Overly aggressive diuresis may cause renal dysfunction or hyperkalaemia. Titrate doses based on fluid status. Monitor weight closely (daily if inpatient and at each visit if outpatient).
ACE-I/ARB	May all cause renal dysfunction with hyperkalaemia. Monitor UECs 3 – 6 monthly. Hold if creatinine increases more than 30% from baseline, or if K+ >5.5 mmol/L, and re-start at a lower dose once K drops < 5. Titrate to target doses, but watch out for hypotension which may worsen kidney dysfunction.
ACE-I	ACEI intolerance may lead to a dry cough or angioedema. The most common cause of cough in HF, however, remains cardiac decompensation.
Beta-blockers	May cause bradycardia and cardiac decompensation. Consider holding or reducing dose if HR <50 bpm. Wait 2 – 3 weeks between titrations. Start at lowest dose. Do not give if in acute. decompensation. Reduce, but don't stop entirely during admissions for worsening HF.
MRAs	May cause hyperkalaemia or creatinine rise. Monitor UECs 3-6 monthly. Hold if K+ >5.5 mmol/L and re-start at a lower dose once K drops < 5. Hyperkalaemia more likely in renal dysfunction, older age, or patients also on ACE-I/ARB. Counsel patients to reduce intake of foods with high K+ e.g. bananas and avocados. Spironolactone may cause gynecomastia and such patients can be switched to eplerenone which is a selective MRA blocker and has less androgen side effects.
If-channel inhibitors	Use only if heart rate >70 bpm despite maximally tolerated beta-blocker dose. May precipitate atrial fibrillation. Do not use in atrial fibrillation.
Digoxin	Use primarily in atrial fibrillation when beta-blocker is contraindicated. Digoxin toxicity may occur at high doses or at normal doses in elderly patients or those with renal dysfunction. Digoxin therapy in conjunction with electrolyte abnormalities may lead to malignant arrhythmias. Correct K+, Mg+ and Ca++ for patients on Digoxin. Use max maintenance doses of 0.125 mg per day. In low body weight patients, elderly and those with eGFR < 60, consider lower doses of 0.125mg every 48-72 hours. Digoxin use in sinus rhythm can be attempted in

Device Therapy

In secondary prevention, i.e. in patients with proven malignant arrhythmia (VT/VF), or in primary prevention in patients with reduced LVEF <35% trials have demonstrated a clear survival advantage when an (implantable cardioverter defibrillator) ICD is added onto optimal medical treatment. For patients with LVEF <35% and QRS-complex >140 milliseconds (130ms if in LBBB), the further addition of biventricular pacing (Cardiac Resynchronization Therapy – CRT) with and without a defibrillator, has been shown to confer significant cost-benefit (1).

Any patient who meets the above criteria should be referred to a cardiologist with expertise in device therapy.

5:8 Treament of other forms of HF (Physician must be consulted)

Rheumatic Heart Disease (Please see chapter on RHD for details)

- Penicillin prophylaxis is necessary to prevent recurrences of acute rheumatic fever. Use azithromycin or sulphonamides in case of penicillin allergy. Diuretics are recommended for fluid management.
- Digoxin, Beta-blockers or CCBs are recommended for heart rate control in those with tachyarrhythmias.
- Afterload reduction with ACEIs or hydralazine may be beneficial in those with regurgitant lesions and/or reduced LVEF.
- Anticoagulation for those in atrial fibrillation/flutter.
- Percutaneous balloon mitral valvuloplasty should be considered in MS without MR.
- Early surgical evaluation should be considered in all patients with severe MS, even if asymptomatic.

Right Heart Failure

Diuretics are recommended for relief of venous congestion. No proven therapies have been shown to impact on mortality. Heart rate control is appropriate for those in tachyarrhythmias plus anticoagulation as appropriate. Treatment of the primary cause if known is indicated e.g. COPD.

Ischemic heart disease

Exclude ischemic heart disease. Please see chapter on IHD.
Immunization- pneumococcal and influenza. Most HF patients should receive this intervention, especially if older than 65 years of age.

Immunization- pneumococcal and influenza. Most HF patients should receive this intervention, especially if older than 65 years of age.

Palliative Care in Patients with Heart Failure

Patients with advanced HF may have frequent symptoms and poor quality of life (QoL). Palliative care should be offered in parallel with optimal treatment strategies at an early point. Aside from sudden death, strong markers of impending mortality in patients with HF are:

- Advanced age;
- Recurrent hospitalisation for decompensated HF and/or a related diagnosis;
- NYHA class IV symptoms;
- Advanced renal dysfunction
- Cardiac cachexia
- Hyponatraemia
- Refractory hypotension necessitating withdrawal of medical therapy

Management of symptoms associated with heart failure at the end of life is an important aspect of palliative care: Symptoms such as: -

- Breathlessness: diuretics will generally still be required to optimise management of fluid-overload. Opioids such as morphine can also be effective in managing shortness of breath.
- Fatigue: look for potentially reversible causes such an infection, anaemia and drug side effects. Measures such as gentle exercise, relaxation, visualisation techniques and pacing of activities may be helpful.
- Thirst: sucking on frozen juice cubes, using mouth washes or artificial saliva/saliva stimulants (such as chewing gum) may be effective in managing xerostomia.
- Anorexia/cachexia: look for potentially reversible causes such as oral candidiasis, untreated nausea, constipation and ill-fitting dentures.
- The indications for ongoing medical heart failure therapies should be reviewed regularly. Some therapies may assist with symptom management at end of life and should be continued, whilst others may not be required. Therapeutic decisions should be individualised for each patient.
- Patients with an Intra-cardiac device (ICD) may require specific counselling regarding switching off the ICD during end-of-life care.

5:9 Follow up and referral criteria

Clinicians should rely on their clinical judgement and **when in doubt should err on the side of referral.** Undertaking specialist referral should not delay initiation of appropriate treatment (most often diuretic treatment) for patients with symptomatic HF.

Specialist referral should be considered in the following situations (see figure 1): -

1. In any transferable patient with HF refractory to treatment where identification of a reversible cause may change the course of HF.
2. Those whose history suggests severe myocardial ischemia where further investigation and intervention (revascularization) may be indicated.
3. If valve intervention or surgery may reverse or eliminate the cause of HF f6 (rheumatic or degenerative valve disease).
4. The diagnosis or aetiology is uncertain despite all tests available at the site of presentation.
5. Arrhythmia (such as atrial fibrillation or ventricular arrhythmias) are apparent and the ejection fraction is low or the patient has refractory symptoms.
6. The indication for anticoagulation (warfarin) is uncertain.
7. If the QRS is >130ms and the patient is still symptomatic despite optimized treatment.
8. If the patient is at high risk of SCD i.e. low EF or symptoms and signs of malignant arrhythmias (VT).

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Guidelines to the diagnosis and management of paediatric heart failure

5:10 Introduction

The definition of Paediatric Heart Failure (PHF) is not different to that applied to adult patients and can be defined broadly as the failure of the heart to supply blood to either systemic or pulmonary circulation at an appropriate rate of flow, or to receive venous return at an inappropriate filling pressure, resulting in adverse effects on the heart, the circulation, and the patient.

Hsu and Pearson have given a good working definition of HF in children as a progressive clinical and pathophysiological syndrome caused by cardiovascular and non-cardiovascular abnormalities that result in characteristic signs and symptoms including oedema, respiratory distress, growth failure, and exercise intolerance, and accompanied by circulatory, neurohormonal, and molecular derangements.

5:11 Causes of paediatric heart failure

Unlike adults, paediatric heart failure can occur from birth to childhood (aged 0 -18 years) and may develop at any stage of this age spectrum. Paediatric heart failure in the low-income countries is characterised by additional burden, low resources, late diagnosis in a setting of tropical disease like malaria, tuberculosis, HIV, rickets and protein energy malnutrition. The most common cause of heart failure in this setting includes structural congenital heart diseases, while rheumatic fever and rheumatic heart diseases, cardiomyopathies are common causes in structurally normal heart (table 1 & 2). Paediatric cardiomyopathies in this setting are predominantly acquired from inflammatory and metabolic causes and present with poor systolic function or pump failure (table 3).

Table 5.7
Cardiac malformations leading to heart failure

Shunt Lesions
- Ventricular septal defect
- Patent ductus arteriosus
- Aortopulmonary window
- Atrioventricular septal defect
- Single ventricle without pulmonary stenosis
- Atrial septal defect (rare), Total/Partial Anomalous Pulmonary Venous Connection & coronary artery anomalies

Valvular Regurgitation
- Mitral regurgitation ● Aortic regurgitation

Inflow Obstruction
- Cor triatriatum
- Pulmonary vein stenosis & congenital Mitral stenosis

Outflow Obstruction
- Aortic valvular, subvalvular and supravalvular stenosis
- Aortic coarctation

Table 5.8
Sources of heart failure with a structurally normal heart

Primary Cardiac
- Cardiomyopathy
- Myocarditis
- Myocardial infarction
- Acquired valve disorders- rheumatic fever and rheumatic heart disease
- Hypertension
- Kawasaki syndrome
- Arrhythmia (bradycardia or tachycardia)

Non-Cardiac
- Anaemia
- Sepsis
- Hypoglycaemias
- Diabetic ketoacidosis
- Hypothyroidism
- Other endocrinopathies
- Arteriovenous fistula
- Renal failure
- Muscular dystrophies

Table 5.9: Types and causes of cardiomyopathies associated with paediatric heart failure

Dilated cardiomyopathy (systolic dysfunction)	Hypertrophic cardiomyopathy Diastolic dysfunction	Restrictive cardiomyopathy
- **Inflammatory** (viral myocarditis like HIV related and influenza, protozoal like Chagas disease and rickettsia) **Endocrine/metabolic** (hypothyroidism, diabetic, excessive catecholamine, rickets& hypocalcaemia, - **Nutritional deficiency** (kwashiorkor, beriberi, carnitine deficiency) **- Drugs** (doxorubicin, adriamycin) Barth syndrome -Neuromuscular disorder (i.e., Becker dystrophy, Duchenne dystrophy) -Familial DCM	Pompe's diseases Noonan syndrome Maternal diabetes -Mitochondrial diseases -Familial hypertrophic cardiomyopathy and	Endomyocardial fibrosis Idiopathic restrictive cardiomyopathy

Paediatric heart failure is unique due to the age-related aetiology.

- From birth to one week of age: Heart failure can be caused by metabolic conditions like severe hypoxia, acidosis, sepsis, hypoglycaemia and hypocalcaemia or severe anaemia and polycythaemia from twin to twin transfusions with few cardiac lesions like critical aortic and pulmonary stenosis, coarctation of aorta, transposition of the great vessels ,and hypoplastic left and right heart syndrome. These are typically duct dependent lesions.

-By six weeks of age: Large left to shunts of ventricular septal defects (VSD) Patent ductus arteriosus and atrioventricular septal defect. Viral myocarditis is commoner in small children older than 1yr. Rheumatic heart disease starts by 5yrs and is predominantly volume loading lesions of mitral and aortic regurgitation. Idiopathic dilated cardiomyopathy causes heart failure during childhood and adolescence while Adriamycin and doxorubicin toxicity for treatment of malignancies can occur months to years after completion of chemotherapy. Sickle cell anaemia may cause heart failure at older age while cor-pulmonale due to adenotonsillar hypertrophy occurs during early childhood. Heart failure in adolescence is more related to rheumatic heart disease and cardiomyopathies

5:12 Clinical manifestation of paediatric heart failure

Clinical presentation is uniquely related to age:

• New-borns – Clinical recognition during this period requires a high index of suspicion with subtle features of tachypnoea- RR> 50b/min and tachycardia- HR> 150/min with hepatomegaly commonly encountered. Duct dependent pulmonary circulation or right sided lesions presents with deep cyanosis and acidosis, whereas duct dependent systemic (left sided lesions) present with heart failure, hypotension and shock.

• Infants primarily having respiratory and feeding difficulties characterised by prolonged feeding time> 20minutes, decreased volume intake, irritability with feeding, vomiting after feeds and refusal to feed and diaphoresis.

• Older children& adolescence: Fatigue and shortness of breath, tachypnoea and exercise intolerance, abdominal pain and oedema increased metabolic demands

At any age, unequal pulses in upper and lower limb should be looked for in a child with unexplained heart failure. The severity of heart failure in children must be classified according to Ross modified classification table 4 which recognises the functional classification with increasing severity

Table 5.10: The Ross Classification of symptomatic severity in paediatric heart failure

Class of symptoms	Symptoms noted on history
I	Asymptomatic
II	Infants: mild tachypnoea or diaphoresis with feeding; no growth failure Older children: dyspnoea on moderate exertion
III	Infants: marked tachypnoea or diaphoresis with feeding; growth failure Older children: dyspnoea on mild or minimal exertion
IV	Tachypnoea, diaphoresis or respiratory distress at rest

5:13 Diagnostic Approach

Clinical Examinations

This involves stepwise guided examinations

Step 1: Identify abnormal clinical findings based on abnormal perfusion or increased fluid congestion as depicted in figure 5.4

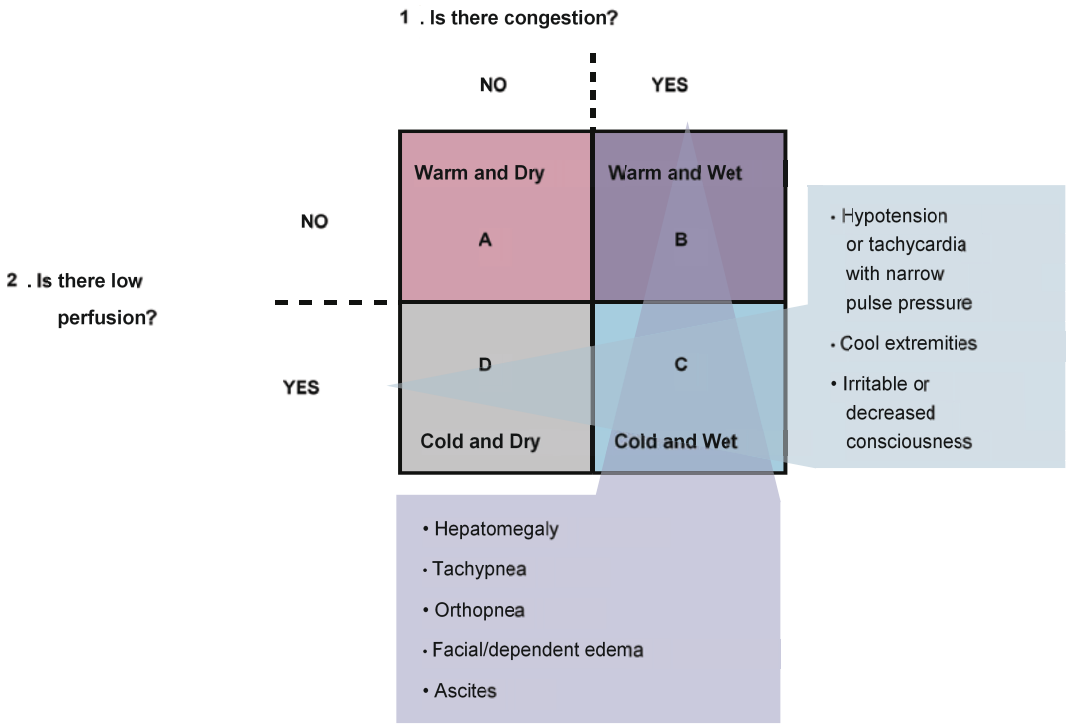

Figure 5.4: Patterns of presentation of recognised in acute decompensated heart failure

Step 2: Rapidly determine the hemodynamic status (Figure 5.4)

Step 3: Identify any reversible causes of heart failure

- Assessment of electrolyte (Na+ K, Cl, Ca, glucose acid base status, urea creatinine, thyroid hormone, hepatic transaminase
- Screening for hypoxia and sepsis should be done in new-born with HF severe anaemia

Step 4: Investigate for specific underlying disease

Chest Radiography – This is indicated as a first line investigation for children with suspected heart disease to

a) Assess for heart size- Cardiomegaly on chest X ray is highly predictive of ventricular dilatation

b) Check the typical heart shapes like egg on side for transposition of great arteries, wall to wall heart in cardiomyopathy and Ebstein's anomaly, snowman's sign and figure of eight in total anomalous pulmonary venous return

c) Signs of heart failure- Kerley- B lines and pleural effusions

Electrocardiography- Can confirm the tachycardia and type of rhythm, chamber enlargement, and myocardial infarction patterns

Echocardiography - Most useful and widely available and low cost. It should be prioritised.

- It provides immediate data on cardiac morphology and structure, assessment of right and left heart contractile and filling functions
- Assessment of pulmonary pressures
- Provides a guide to appropriate therapy
- Serial echocardiography is useful in surveillance of disease progression and to the response to therapy

Cardiac catheterization – indicated for:

- Accurate diagnosis of complex congenital heart disease
- Accurate evaluation of pressure gradients in patients with complex valve disease and multiple obstructive lesions
- Evaluation of hemodynamic parameter in children with late referral shunt lesions for pulmonary and systemic resistance
- Useful in evaluation of grown up unoperated and palliated patients with congenital heart disease

Biomarkers - Brain natriuretic peptide (BNP) or NT-proBNP levels are useful in distinguishing heart failure from respiratory or other non-cardiac diseases and should be used as a confirmatory test in acute evaluation of paediatric heart failure

Metabolic and genetic testing-- Recommended for children with unexplained cardiomyopathy (dilated, hypertrophic or non-compaction type.

5:14 Therapeutic Approach to Paediatric Heart Failure.

Treatment of PHF consists of

1) Timely treating the specific underlying cause

- Timely surgical correction of the structural cardiac abnormality like closing a ventricular septal defect or patent ductus arteriosus, repairing a coarctation, or relieving a valve obstruction, repairing or replacing the regurgitant valve in rheumatic
- Correction and treatment of anaemia or and correcting endocrinopathies like hypothyroidism
- Stopping the episode of supraventricular tachycardia, pacing for the patient who has heart block

2) Elimination of the precipitating or contributing cause –Fever precipitating events such as intercurrent infections, anaemia, electrolyte imbalances, arrhythmia, rheumatic reactivation, infective endocarditis, drug interactions, drug toxicity, or drug noncompliance should be identified and corrected if present. Acute HF patients can have symptoms related to fluid overload, under perfusion, or both. The early management of children with HF should address these problems
In neonates, acute circulatory collapse or progress to shock if not recognized early. Many of these conditions require maintenance of duct patency with prostaglandin infusion or emergency procedures such as ductal stenting and balloon atrial septostomy

3) Control of symptoms and disease progression- general measures

-Bed rest and limit activities
-Nurse propped up or in sitting position
-Fever control
-Expressed breast milk for small infants
-Fluid restriction in volume overloaded and in children with hyponatremia or unresponsive to diuretics
-Optimal sedation
-Correction of anemia, acidosis, hypoglycemia and hypocalcemia if present
-Oxygen –caution in LT-RT shunt as pulmonary vasodilation may increase shunt
-In patients with cyanotic congenital heart disease oxygen has little effect in raising SPO2 and is contraindicated
-CPAP or mechanical ventilation as necessary

4) Nutritional support - Nutritional support is as important as medical therapy, particularly in infants.

1) Sodium intake should be 2-3meq/kg/day. Sodium restriction is not recommended in infants and young children and because sodium is an important growth factor. Sodium restriction can result in impaired body and brain growth.
2) In infants, nutritional support must ensure a caloric intake about of 120-150 kcal/kg/d. This is achieved using dietary supplements, preferring small and frequent meals that are better tolerated.
3) Carbohydrates should not exceed 6 g/kg/d and lipids should not exceed 2.5 g/kg/d.
4) The provision of essential amino acids is necessary in the critically ill. Evidence suggests that 1.2 to 1.5 g/kg/d of protein is needed

5:14 Drug Therapy in Paediatric Heart Failure

Drug treatment in PHF aims to decrease of pulmonary wedge pressures, increase of cardiac output and the improvement of end organ perfusion and finally to delay of disease progression.

Scenario 1: No structural heart disease with left ventricular systolic dysfunction such as dilated cardiomyopathy

Digoxin – it is useful in symptomatic patients with left and/ or right ventricular systolic dysfunction. Rapid digitalization is not indicated when using digoxin for heart failure but can be used in tachyarrhythmias to slow the heart rate. The oral dose is twice daily for children under 10 years and once daily for children over 10yrs at (8-10ug/kg/day)

ACE inhibitors - they decrease afterload by antagonising the renin angiotensin/ aldosterone system. Should be started at low dose with up titration to the target dose. Captopril is the first choice in infants staring dose 0.1mg/kg/dose gradually increase to 0.5-1mg/kg/dose three times per day, maximum dose 2mg/kg/dose.

Enalapril is useful in older children at a dose 0.1-0.5mg/kg /dose twice daily.

> *Children treated with ACEIs should be watched for deterioration in renal function and hypotension. Other adverse effects include cough and angioedema. Angiotensin receptor blockers are generally reserved for those children with systemic ventricular systolic dysfunction who would benefit from renin-angiotensin-aldosterone system blockade but are intolerant of ACEIs.*

Beta blockers- the addition of beta blocker to above therapy may be useful in patients with left ventricular systolic dysfunction. Low dose therapy should be started with progressive upward titration.

Carvedilol started at **0.05mg/kg/dose** twice daily increased to **0.4-0.5mg/kg/dose** twice daily.

Metoprolol 0.1-0.2mg/kg dose twice daily to be escalated to 1mg twice daily

Scenario 2: Congenital Heart Disease: Volume overload such as large ventricular septal defects (VSDs), patent ductus arteriosus, or endocardial cushion defects

a) Loop diuretics such as furosemide, is recommended for patients with HF and signs and symptoms of congestion. An initial starting dose of **0.5-1 mg/kg** intravenously or orally every 6-12 hours, is safe and effective with a maximum dose of **4mg/kg/day**

b) Patients who are unresponsive to loop diuretics alone may benefit from addition of a thiazide agent

c) Aldosterone antagonist (spironolactone) therapy is used in children with chronic systolic HF. The starting dose of spironolactone is **1 mg/kg/day,** and the target maximum dose is **2 mg/kg/day**. Male gynecomastia can occur with spironolactone requiring discontinuation.

Scenario 3: Congenital Heart Disease: Pressure Overload

Includes critical aortic stenosis, severe pulmonary stenosis, coarctation of aorta.

In the new-born period presents as duct dependent lesions and requires prostaglandins to maintain the ductus as a bridge to catheter interventions. Prostaglandin E1 is given by intravenous infusion starting 0.05-0.1ug/kg/min and once desired dose achieved can be reduced to 0.05 to 0.01ug/kg

Table 5:11 Drugs used in paediatric heart failure.

Drugs	Routes of administration	Doses
Furosemide	Oral	1-2 mg/kg q6-12h
Furosemide	Intermittent bolus	0.5-2 mg/kg q6-12h
Furosemide	Continuous infusion	0.1-0.4 mg/kg/h
Captopril	Oral	0.3-2 mg/kg q8h
Enalapril	Oral	0.05-0.25 mg/kg q12h
Losartan	Oral	0.5-1.5 mg/kg/d
Carvedilol	Oral	0.05 mg/kg/d q12h
Metoprolol	Oral	0.25 mg/kg/d q12h
Spironolactone Oral		0.5-1.5 mg/kg q12h
Nitroglycerin	Continuous infusion	0.5-10 mg/kg/min
Nitroprusside	Continuous infusion	0.5-4 mg/kg/min
Hydralazine	Intermittent bolus	0.1-0.2 mg/kg every 4-6 h
Hydralazine	Oral	0.3-1 mg/kg/d in q8 -12h
Digoxin	Oral	5-10 mg/kg/d
Dobutamine	Continuous Infusion	2.5-10 mg/kg/min
Epinephrine	Continuous Infusion	0.01 - 0.1 mg/kg/min
Epinephrine	Intermittent bolus	0.01 mg/kg
Milrinone	Continuous Infusion	0.5 -1 mg/kg/min
Levosimendan	Continuous Infusion	0.05 -0.2 mg/kg/min

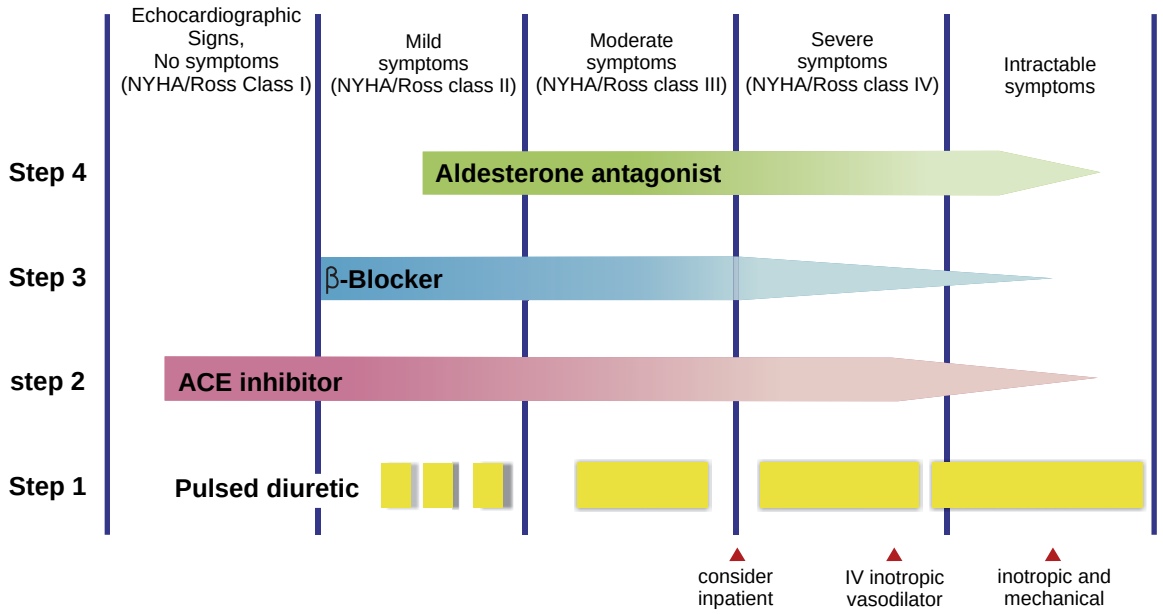

Figure 5.6: Stepwise introduction of medical therapy in heart failure

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6 Rheumatic Fever and Rheumatic Heart Disease

6 Rheumatic Fever, Rheumatic Heart Disease

List of Abbreviations

ARF	Acute Rheumatic Fever
CRP	C-reactive protein
ESR	Eryhtrocyte sedimentation rate
GAS	Group A streptococcal infection
RHD	Rheumatic Heart Disease

6:1 Introduction

Rheumatic Heart Disease (RHD) is the most common acquired heart disease in children in developing countries [1]. It is a chronic heart condition caused by acute rheumatic fever (ARF) which can be prevented and controlled. The Risk of RF following untreated GAS pharyngitis is between 0.3 – 3% [6]. For individuals with a history of previous RF the risk rises to 50%. Upto 30% of sore throats in children and young people are caused by a bacteria called group A streptococci (GAS) [2]. Without antibiotic treatment, some of these children will develop rheumatic fever (RF) a few weeks after their sore throat [3].

Repeated episodes of GAS infection and RF causes progressive heart valve damage and scarring [5]. This persistent valve scarring is called rheumatic heart disease (RHD).
The most important determinant of disease progression appears to be the number of times RF recurs in an individual. Only some people are susceptible to RF and RHD. A triad of environmental, genetic and bacterial factors appear to be important in the development of clinically significant disease [7].

6:2 Epidemiology and Burden of disease

Acute rheumatic fever and rheumatic heart disease are rare in the high income countries [8]. This is attributed in part to improvement in socioeconomic conditions and the widespread use of penicillin G benzathine to treat streptococcal pharyngitis [9]. The remaining burden of rheumatic heart disease is found mostly in low – income and middle income countries and among immigrants and older adults in high income countries [10].

Death and disability from RHD continues to extract an enormous social, economic and cultural toll on young adults and their communities. The burden is greatest in the most productive years of life for those who can least afford it.

It has been estimated that most children develop at least one episode of pharyngitis per year, 15–20% of which are caused by group A streptococci and nearly 80% by viral pathogens [11]. Group A streptococcal pharyngitis has a peak incidence in children 5–15 years of age [12, 13]. It is less frequent among children in the first three years of life and among adults.
Globally, over 15 million people suffer from RHD [14]. The prevalence of RHD in Kenya ranges from 1.7% to 2.7% in hospitalized surgical patients aged 5 to 35 years. The most devastating effects are on children and young adults in their most productive years.

6:3 Aetiology and pathogenesis

Rheumatic fever is an autoimmune inflammatory response to Group A streptococcal infection that tends to occur a few weeks after the infection. Persons who have experienced an episode of RF are predisposed to recurrence following subsequent group A streptococcal infections. The body's immune defenses are unable to distinguish between the streptococcus and the

host tissues, causing an inflammatory reaction. It affects the heart, subcutaneous tissues, joints and the central nervous system. Rheumatic heart disease is the most severe sequelae of [RF], and it mostly manifests as destruction of the heart valves after the initial or repeated inflammation of RF.

Determinants of the disease burden of rheumatic fever and rheumatic heart disease

The table below outlines the key socioeconomic and health-care related risk factors that are associated with development and course of RF and RHD.

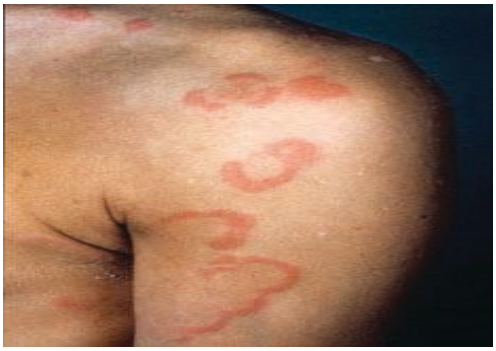
Table 6:1 Direct and indirect results of environmental and health-system determinants on rheumatic fever and rheumatic heart disease

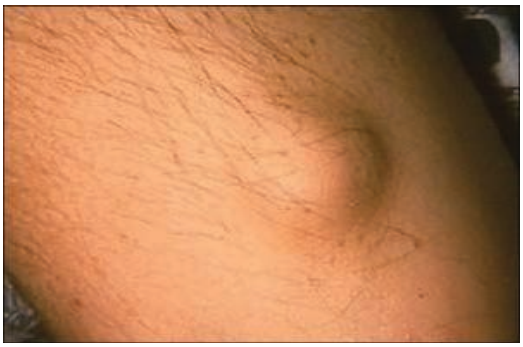
Determinants	Effects	Impact on RF and RHD burden
Socioeconomic and environmental factors • Poverty, undernutrition, overcrowding, poor housing)	Rapid spread of group A streptococcal strains	Higher incidence of acute streptococcal-pharyngitis and suppurative complications
	Difficulties in accessing health care	Higher incidence of acute RF Higher rates of recurrent attacks
Health-system related factors: • Shortage of resources for health care • Inadequate expertise of health-care providers • Low-level awareness of the disease in the community	Inadequate diagnosis and treatment of streptococcal pharyngitis	Higher incidence of acute RF and its recurrence
	Misdiagnosis or late diagnosis of acute RF	Patients unaware of the first RF episode More severe evolution of disease Untimely initiation or lack of secondary prophylaxis
	Inadequate secondary prophylaxis and/or non-compliance with secondary prophylaxis	Higher rates of recurrent attacks with more frequent and severe heart valve involvement, and higher rates of repeated hospital admissions and expensive surgical interventions

Clinical Manifestations

RF causes joint pains, fever, skin changes and sometimes abnormal movements. In most cases the heart also becomes inflamed during [RF. However, when other symptoms of RF resolve, changes to the heart valves persist [4]. Further features are shown in the table below:

Table 6:2 Manifestations of Acute Rheumatic Fever

Manifestation	Description
Carditis	Is the only manifestation that has the potential for long-term complications. It usually manifests as a pancarditis involving the endocardium, myocardium and pericardium. It presents as a new murmur, cardiac enlargement, congestive heart failure, pericardial friction rub, and/or pericardial effusion
Arthritis	Inflammation of the synovial membranes of several joints characterized by swelling, redness, warmth and pain. Usually presents as polyarthritis which is migratory in nature. Mostly affects the larger joints, including the knee, ankles and elbows and wrists. It rapidly improves on NSAIDS. Usually runs a self-limited course lasting ≈4 weeks Monoarthritis may be a presenting feature in high-risk populations
Sydenham's chorea	Chorea in ARF is characterized by purposeless, involuntary, nonstereotypical movements of the trunk or extremities. It often is associated with muscle weakness and emotional lability.
Erythema marginatum	Distinctive rash marked by the presence of pink rings in the torso or upper(proximal) parts of the body; can appear and disappear within minutes 

Subcutaneous nodules	Small painless lumps under the skin. Usually present over the elbows, wrists, knees, ankles, achilles tendon, occiput and posterior spinal processes of the vertebrae. These are uncommon, and represent severe carditis. 
Minor manifestations	
Clinical	Fever, polyarthralgia
Laboratory	Elevated acute phase reactants (erythrocyte sedimentation rate or leukocyte count)
Electrocardiogram	prolonged P-R interval
Supporting evidence of a preceding streptococcal infection within the last 45 days	
— elevated or rising antistreptolysin-O or other streptococcal antibody, or — a positive throat culture, or — rapid antigen test for group A streptococci, or — recent scarlet fever	

6:4 Diagnosis

An accurate diagnosis of ARF is important. Over diagnosis results in unnecessary treatment over a long time, while under diagnoses leads to further attacks of ARF, cardiac damage and premature death. Diagnosis remains a clinical decision, as there is no specific laboratory test. The diagnosis of ARF is usually guided by the Jones criteria and the more recent World Health Organization (WHO) criteria. The clinical diagnosis of carditis usually depends on detecting: (i). myocarditis (ii). Pericarditis, and (iii). Valve regurgitation. Carditis as a major manifestation of ARF has been a clinical diagnosis based on the auscultation of typical murmurs that indicate mitral or aortic valve regurgitation at either valve or both valves.

To increase sensitivity of ARF diagnosis current evidence now support the use of echocardiography/Doppler as part of the diagnostic criteria for confirmation of the presence of carditis in patients with suspected ARF. Accordingly, in the Revised Jones Criteria for the diagnosis of ARF in the era of Doppler Echocardiography recommends: -

i. Echocardiography with Doppler should be performed in all cases of confirmed and suspected ARF.
ii. It is reasonable to consider performing serial echocardiography/Doppler studies in any patient with diagnosed or suspected ARF even if documented carditis is not present on diagnosis.
iii. Echocardiography/Doppler testing should be performed to assess whether carditis is present in the absence of auscultatory findings, particularly in moderate- to high-risk populations and when ARF is considered likely.
iv. Echocardiography/Doppler findings not consistent with carditis should exclude that diagnosis in patients with a heart murmur otherwise thought to indicate rheumatic carditis.

Diagnostic criteria for Rheumatic Fever and RHD

The revised Jone's criteria was adopted to facilitate standardization of diagnosis. In principle, the Criteria prescribes various combinations of major and minor manifestations.

Any 1 of the following can serve as evidence of a preceding GAS infection, per a recent AHA statement:

1. Increased or rising anti-streptolysin O titer or other streptococcal antibodies (anti-DNASE B). A rise in titer is better evidence than a single titer result.
2. A positive throat culture for group A β-hemolytic streptococci.
3. A positive rapid GAS carbohydrate antigen test in a child whose clinical Presentation suggests a high pretest probability of streptococcal pharyngitis.

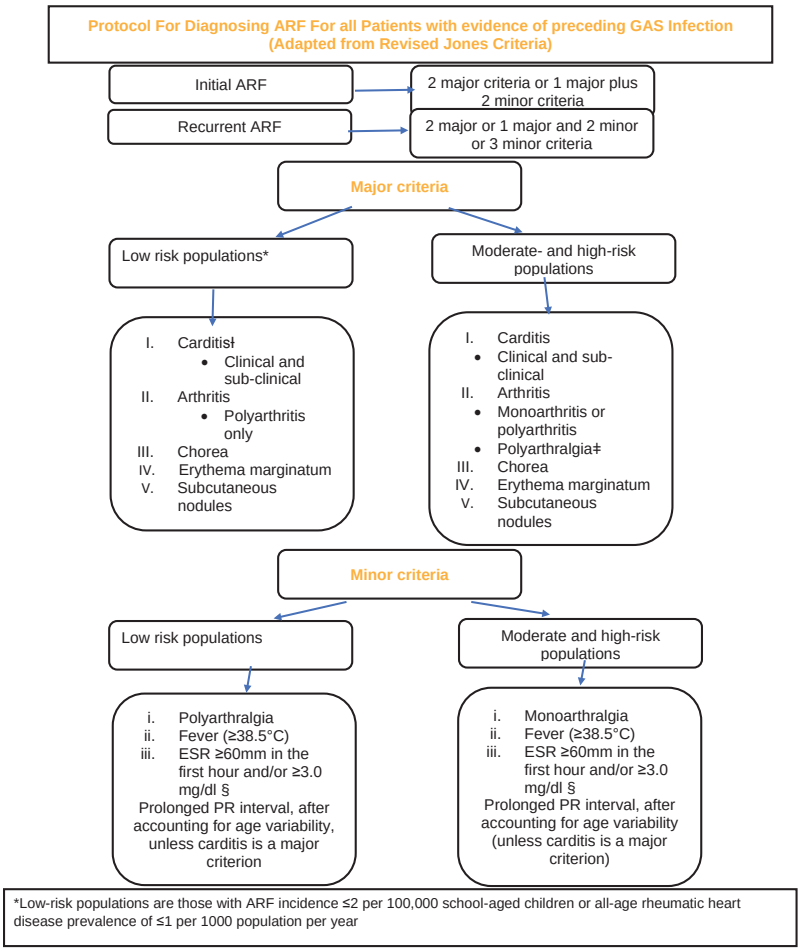


Figure 6.1: Diagnosis strategy for acute rheumatic fever with Echocardiography findings. *Subclinical carditis can be considered. Alt - alternative; ARF - acute rheumatic fever; echo - echocardiography; GAS - group A streptococcal; neg - negative

Differential diagnosis

Table 6:3 Differential diagnosis of Arthritis, Carditis and Chorea

Arthritis	Carditis	Chorea
• Septic arthritis (including gonococcal) • Connective tissue and other autoimmune diseases such as juvenile idiopathic arthritis • Viral arthropathy • Reactive arthropathy • Lyme disease • Sickle cell anaemia • Infective endocarditis • Leukaemia or lymphoma • Gout and pseudogout • Poststreptococcal reactive arthritis • Henoch-Schonlein purpura	• Physiological mitral regurgitation • Mitral valve prolapse • Myxomatous mitral valve • Fibroelastoma • Congenital mitral valve disease • Congenital aortic valve disease • Infective endocarditis • Cardiomyopathy • Myocarditis, viral or idiopathic Kawasaki disease	• Drug intoxication • Wilson disease • Tic disorder • Choreoathetoid cerebral palsy • Encephalitis • Familial chorea (including Huntington disease) • Intracranial tumor • Lyme disease • Hormonal • Metabolic (e.g. Lesch-Nyhan, hyperalaninemia, ataxia teleangiectasia) • Antiphospholipid syndrome • Autoimmune: Systemic lupus erythematosus, systemic vasculitis • Sarcoidosis • Hyperthyroidism

6:5 Management of Acute Rheumatic Fever

Rheumatic carditis is primarily a valvulitis rather than a myocarditis.

- The mainstay of treatment is supportive
- Bed rest, decreased physical activity, high nutrition
- Medical management
- Anti-inflammatory treatment with steroids in severe cases. The ubiquitous use of anti-inflammatory agents is thought to have masked many cases of ARF. There is still no convincing evidence that this changes the course nor natural history of the disease
- Symptomatic treatment of cardiac failure using anti-failure medication is required in severe cases. In the worst case scenario, surgery may need to form part of acute treatment
- Adequate penicillin for eradication for GAS

Management of RF/RHD involves four main steps:-

i. Primary prevention- eradication of streptococci and prevention of new infections
ii. Anti-inflammatory treatment
iii. Supportive treatment and management of complications
iv. Secondary prevention- prevention of recurrent attacks

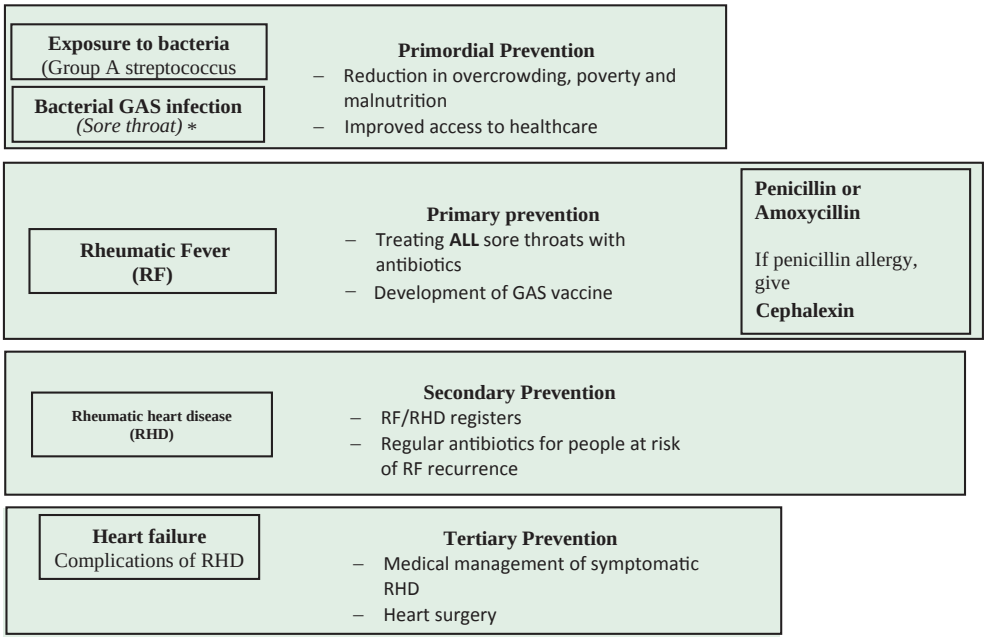


Figure 6.2: Opportunities for intervention for RF and RHD

1. Primary prevention activities

Primary prevention is based on the early detection, correct diagnosis and appropriate treatment of individual patients with Group A streptococcal pharyngitis. This RF and RHD prevention/ control programme needs to be part of the routine medical care available and should be integrated into the existing health infrastructure.
Health education to the public, teachers and health personnel would enhance the impact of a primary prevention programme.

Health education activities

Health education activities should address both primary and secondary prevention. The activities may be organized by trained doctors, nurses or teachers and should be directed at the public, teachers and parents of school-age children.
Health education activities should focus on the:-

- importance of recognizing and reporting sore throats early;
- methods that minimize and avoid the spread of infection;
- the benefits of treating sore throats properly; and
- the importance of complying with prescribed treatment regimes

Treatment of streptococcal pharyngitis/Antimicrobial therapy

The goal of therapy is eradication of the pharyngeal streptococcal infection which is mandatory to avoid chronic repetitive exposure to streptococcal antigens. Ideally, two throat cultures should be performed before starting antibiotics. However, antibiotic therapy is warranted even if the throat cultures are negative. Antibiotic therapy does not alter the course, frequency and severity of cardiac involvement (3).

Table 6: 4 Medications for streptococcal pharyngitis

Agent	Pediatric dosage	Adult Dosage	Route of administration	Duration
Benzathine penicillin	<27kg: 0.6 MU STAT	1.2 MU stat	IM	
Amoxycillin	Mild to moderate pharyngitis 12.25 mg/kg BD Or 10mg/kg TDS Severe pharyngitis 22.5 mg/kg BD Or 13.3 mg/kg TDS	500mg TDS	PO	10 days
Erythromycin ***(if penicillin allergies)***	30-50mg/kg per day in 2-4 divided doses	500mg QID	PO	10 days
Azithromycin		500mg OD	PO	5 days

Anti-inflammatory therapy

The cornerstone of management of ARF is the suppression of the inflammatory process. Anti-inflammatory agents are used to control the carditis, arthritis, fever, and other acute symptoms. Salicylates are the preferred agents, although other nonsteroidal agents are probably equally efficacious. Steroids are also effective but should probably be reserved for patients in whom salicylates fail.

Table 6:5 Priorities in managing ARF

Admission to hospital
Admit all patients suspected to have ARF
Confirmation of the diagnosis
Observation prior to anti-inflammatory treatment; Paracetamol (first line) or codeine for fever or joint pain
Investigations
Treatment
All cases
Single-dose IM BPG (preferable) or 10 days' oral penicillin V (IV not needed; oral erythromycin if allergic to penicillin)
Arthritis and fever
Paracetamol (first line) or codeine until diagnosis confirmed
Aspirin, naproxen or ibuprofen once diagnosis confirmed, if arthritis or severe arthralgia present
Mild arthralgia and fever may respond to paracetamol alone
Influenza vaccine for children receiving aspirin during the influenza season (autumn/winter)
Chorea
No treatment for most cases
Carbamazepine or valproic acid if treatment necessary
Carditis/heart failure
Bed rest, with mobilization as symptoms permit
Urgent echocardiogram
Antifailure medication
• diuretics/fluid restriction for mild to moderate failure • ACE inhibitors for more severe failure, particularly if AR present. Glucocorticoids optional for severe carditis (consider treating for possible opportunistic infections) • digoxin, if AF present
Valve surgery for life - threatening acute carditis (rare)
Long - term preventive measures
First dose of secondary prophylaxis
Notify case to ARF/RHD register, if available
Contact local primary care staff to ensure follow-up
Referral to a medical specialist
Provide culturally - appropriate education to patient and family
Arrange dental review and ongoing dental care to reduce risk of endocarditis

ACE, angiotensin converting enzyme; AF, atrial fibrillation; AR, aortic regurgitation; BPG, benzathine penicillin G; IM, intramuscular; IV, intravenous

Table 6:6 Medications Used In Management Of Acute Rheumatic Fever In Children

Medication	Indication	Regimen	Duration
BPG, im	Treat streptococcal infection	900 mg (1,200,000 U) ≥20kg 450 mg (600,000 U) <20kg	Single dose
Or phenoxymethylpenicillin po (penicillin V)	Initial treatment of streptococcal infection	Child: 250 mg, bd Adolescents and adults: 500 mg/bd	10 days
Or erythromycin ethyl succinate, po **(only if allergic to penicillin)**	Initial treatment of streptococcal infection	Child: 20 mg/kg up to 800 mg, bd Adult: 800 mg, bd	10 days
Or Erythromycin, po **(only if allergic to penicillin)**	Initial treatment of streptococcal infection	Child: 12.5 mg/kg upto 500 mg, bd Adult: 500 mg, bd	10 days
Paracetamol, po	Arthritis or until diagnosis confirmed Arthralgia	60 mg/kg/day (max 4 g) given 4-6 doses/day; may increase to 90 mg/kg/day, if needed, under medical supervision	Until symptoms relieved or NSAID started
Aspirin, po	Arthritis or severe arthralgia (when ARF diagnosis confirmed)	Begin with 50-60 mg/kg/day, increasing, if needed, up to 80-100 mg/kg/day (4-8 g/day in adults) given in 4-5 doses/day If higher doses required, reduce to 50-60 mg/kg/day when symptoms improve, and cease when symptom free for 1-2 weeks Consider ceasing in the presence of acute viral illness, and consider influenza	Until joint symptoms relieved

Naproxen, po	Arthritis or severe arthralgia (when ARF diagnosis confirmed)	10-20 mg/kg/day (max 1250 mg) given, bd	As for aspirin
Ibuprofen, po	Arthritis or severe arthralgia (when ARF diagnosis confirmed)	30 mg/kg/day (max 1600 mg) given tds	As for aspirin
Prednisone or prednisolone, po	Severe carditis, heart failure, pericarditis with effusion	1-2mg/kg/day (max. 80 mg); if used > 1 week, taper by 20-25% per week	Usually 1-3 weeks
Frusemide, po/iv (can also be given im)	Heart failure	Child: 1-2mg/kg stat, then 0.5-1mg/kg/dose 6-24 hourly(max 6mg/kg/day Adult: 20-40mg/dose, 6-24 hourly, up to 250-500 mg/day	Until failure controlled and carditis improved
Spironolactone,po	Heart failure	1-3mg/kg/day (max 100-200 mg/day) in 1-3 doses; round dose to multiple of 6.25mg (1/4 of a tablet)	As for frusemide
Enalapril. Po	Heart failure	Child: 0.1 mg/kg/day in 1-2 doses, increased gradually over 2 weeks to max of 1 mg/kg/day in 1-2 doses Adult: initial dose 2.5 mg daily, maintenance dose 10-20mg daily (max 40mg)	As for frusemide
Captopril, po	Heat failure	Child: initial dose 0.1 mg/kg/dose. Beware of hypotension. Increase gradually over 2 weeks to 0.5-	As for frusemide
Valproic acid, po	Severe chorea (may affect salicylate metabolism)	Usually 15-20mg/kg/day (can increase to 30mg/kg/day) given tds	As for carbamazepine

Bd - twice daily; BPG - benzathine penicillin G; im - intramuscular; iv - intravenous; NSAID - non-steroidal anti-inflammatory drug; po - per oral; tds - three times daily

The optimal aspirin dose should ensure an adequate response but avoid toxicity. If symptoms of toxicity are present, they may subside after a few days despite continuation of the medication, but salicylate blood levels could be monitored if facilities are available
However, in patients who are intolerant or allergic to aspirin, naproxen (10–20mg/ kg-day) can be used.

6:6 The role of surgery in active rheumatic carditis

Surgical option in acute RF is usually limited to instances of intractable heart failure. Usually, deferral of surgery till active inflammation has subsided is the preferred option.

Secondary prevention and rheumatic heart disease control

Secondary prevention refers to the early detection of disease and implementation of measures to prevent recurrent and worsening disease. Secondary prophylaxis with benzathine penicillin G (BPG) is the only RHD control strategy shown to be clinically effective and cost-effective at both community and population levels. Randomized, controlled trials (RCT) have shown that regular administration is required to prevent recurrent ARF.

Secondary Prophylaxis

Guidelines for Secondary Prophylaxis

Length of time for secondary prophylaxis depends on a number of factors including

- Age at first diagnosis of ARF (or RHD)
- Severity of disease
- If carditis was present with first ARF
- Time (years) since last ARF illness
- Ongoing risk factors (e.g. level of poverty)
- If medication is received regularly

World Health Organisation guidelines for secondary prophylaxis duration:

Disease Classification	Duration of secondary prophylaxis
ARF (no carditis)	Minimum of 5 years after last ARF, or Until age 18 years (whichever is longer)
Mild-moderate RHD (or healed carditis)	Minimum of 10 years after last ARF, or Until age 25 years (whichever is longer)
Severe RHD and after Surgery	Continue for life

** ***Secondary prophylaxis guidelines may vary*** **

In pregnant patients, penicillin prophylaxis should continue for the duration of pregnancy to prevent recurrent ARF. There is no evidence of teratogenicity. Erythromycin is also considered safe in pregnancy.

Table 6:6 Selection of therapy for secondary prevention of rheumatic fever

Agent	Dose	Mode of administration
Benzathine Penicillin	Patients weighing 27 kg (60 lb) or less: 600,000 units IM every 4 weeks Patients weighing more than 27 kg: 1,200,000 units IM every 4 weeks	IM
Penicillin V	250 mg BD	PO
Azithromycin ***(if penicillin allergies)***	250mg OD	PO
Erythromycin	250mg BD	PO

6:7 Management of Rheumatic Fever at Different Levels of Care

Table 8: Management of Rheumatic Fever by level of care

Level of Service	Services offered	Requirements
Level 1	1. Health education 2. Behavior change communication 3. Surveillance and referral of children with fever and sore throat	1. IEC materials 2. Sensitization and support of CHW activities
Level two and three	1. Detect streptococcal sore throat 2. Treat sore throat /streptococcal URTI as per the guidelines 3. Provide secondary prophylaxis for patients with RF to prevent recurrence 4. Detect ARF using the revised Jones criteria and refer 5. Detect heart conditions and refer 6. Carry out epidemiological surveillance	1. Training of health care providers on the guidelines 2. Job aids 3. Reliable light source (e.g. solar torches) and spatulas 4. Essential drugs such as penicillin and alternatives such as erythromycin 5. Lab support- Throat swabs 6. Data collection tools
Level four and five	1. All activities under level two and three 2. Detection, treatment and follow up RF and RHD 3. Development of a comprehensive management plan including referral instructions upwards and downwards 4. Epidemiological surveillance 5. Definitive cardiac surgery (at level five)	1. Training of personnel on the guidelines 2. A functional laboratory Imaging services; 2D echo with appropriate probes, TEE probe 3. CT scans and MRI, Catheterization laboratory. 4. Job Aids 5. Essential drugs 6. Consumables both surgical and medical
Level six	1. All activities in level four and five 2. Interventional cardiology	1. Case specific surgical management and referral management protocols 2. Training of accredited courses in cardiovascular disease 3. Research in cardiovascular diseases

6:8 Chronic Rheumatic Heart Disease

Chronic RHD is sequelae of poorly-managed or undiagnosed RF. It evolves over 2-10 years following repeated episodes of RF. The hallmark of this condition is the development of valvular heart lesions.

Clinical presentation

The various presentations of chronic RHD are summarized in table 8.

Table 9: Valvular lesions in chronic RHD with recommendation

.l	Symptoms	Signs	Recommendation
Mitral stenosis	-Fatigue -Dyspnoea -Palpitation -Cough Hemoptysis Edema Ascites Chest pain	Atrial fibrillation -Mitral facies (flushed cheeks) -Crepitations -Diastolic murmur -Loud P2	Maintain airway, breathing and circulation. Then refer to a physician/ Paediatrician for definitive diagnosis and management plan.
Mitral regurgitation	-Fatigue -Cough -Palpitation -Edema -Ascites -Dyspnoea	-Atrial fibrillation -Cardiomegaly -Apical pansystolic murmur -Crepitations -Signs of RHF (edema, Ascites,hepatomegaly)	Maintain airway, breathing and circulation. Then refer to a physician/ paediatrician for definitive diagnosis and management plan.
Aortic regurgitation	-if mild to moderate it is asymptomatic -Palpitation -Breathlessness -Angina	-Large volume or collapsing pulse -Femoral Bruit (doroziez's sign) -Capillary pulsation (quincke's) -Bounding periphery pulse -Head nodding (demusset's signs) -Murmurs sytolic murmurs (soft mid diastolic murmur (Austin flint murmur)	Maintain airway, breathing and circulation. Then refer to a physician/paediatrician for definitive diagnosis and management plan.

Aortic Stenosis	Mild to moderate –asymptomatic -Dyspnoea -Angina -Exertion syncope -Angina -Episodes of acute pulmonary oedema	-Injection systemic murmur -Slow carotid pulse -Narrow pulse pressure -Thrusting apex beat -Crepitations	Maintain airway, breathing and circulation. Then refer to a physician/paediatrician for definitive diagnosis and management plan.
Tricuspid regurgitation/ Tricuspid Stenosis	Edema Ascites Exercise intolerance→ Angina (rare; due to RV overload and strain) Symptoms of heart failure if is underlying cause	- S3 gallop - Jugular venous distention with a prominent V wave -In some patients, a pansystolic murmur -Diminished peripheral pulse volume secondary to impaired forward blood flow; -Right ventricular heave and S 4 gallop that increases with inspiration -Ascites -Peripheral edema -Cachexia and jaundice -Atrial fibrillation -Pulmonary rales, if TR is associated with LV dysfunction or MS	Maintain airway, breathing and circulation. Then refer to a physician/paediatrician for definitive diagnosis and management plan.

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7 Infective Endocarditis

7 Infective Endocarditis

List of Abbreviations

IE	Infective endocarditis
NVE	Native valve endocarditis
PVE	Prosthetic valve endocarditis
IVDA	Intravenous drug abuse

7:1 Introduction

Infective endocarditis (IE) is defined as an infection of the endocardial surface of the heart, which may include one or more heart valves, the mural endocardium, or a septal defect.
Infective endocarditis poses a special threat for individuals with chronic rheumatic valvular disease, or who have had prosthetic valves implanted because of rheumatic heart disease (RHD). Infective endocarditis rarely occurs without underlying cardiac pathology, either congenital or acquired. Infective endocarditis may also occur in IV drug users, patients with pacemaker leads or conduits.

7:2 Epidemiology

Globally, the incidence of IE ranged between 1.5 and 11.6 cases per 100,000 people [1]
In the Unites States, there has been a steady increase in the incidence of IE hospitalizations over the past decade. This has been attributed to increase in at-risk populations (such as older, diabetic and hemodialysis patients) and increase in number of invasive procedures leading to transient bacteremia [2, 3]

In high-income countries, there has been a significant decline in IE caused by rheumatic heart disease. Instead, most cases are attributed to degenerative valvulopathies, prosthetic valves, and cardiovascular implantable electronic devices, with an increase in the proportion of IE caused by staphylococci. In low-income countries, however, most cases are caused by streptococcus, with rheumatic heart disease being the underlying risk factor disease remains the main risk factor, and streptococci the most frequent causative agents [4]

7:3 Risk factors for infective endocarditis

Cardiac conditions at high risk of endocarditis for which prophylaxis should be considered prior to a high-risk procedure include:

Patients with any prosthetic valve, including a transcatheter valve, or those in whom any prosthetic material was used for cardiac valve repair
Patients with a previous episode of IE
Patients with CHD a. Any type of cyanotic CHD b. Any type of CHD repaired with a prosthetic material, whether placed surgically or by percutaneous techniques, up to 6 months after the procedure or lifelong if residual shunt or valvular regurgitation remains

7:4 Classification of infective endocarditis

There are five main types of infective endocarditis

- Native valve endocarditis (NVE)
- Prosthetic valve endocarditis (PVE)
- Intravenous drug abuse (IVDA) endocarditis
- Fungal endocarditis
- Nosocomial/healthcare-associated endocarditis

Table 7:1 Classification/types of infective endocarditis

Classification		Aetiology/characteristics	Organisms
Native valve endocarditis	Complications arise due to slowly progressive **valvular** destruction, **embolic**or **immunologic** phenomena	**Causes**i. Rheumatic valvular disease -Primarily involves the mitral valve followed by the aortic valveii. Cyanotic congenital heart diseases e.g. Tetralogy of Fallot, transposition of great arteries (TGA)	Streptococcus spp (70% of cases) • S. viridans • S. bovis • Enterococci Staphylococcus spp (25% of cases)
Prosthetic valve endocarditis	Early (less than 1 year)	Shortly after surgery Runs an acute and aggressive course with local abscess and fistula formation and valve dehiscence, leading to shock and heart failure, pericardial tamponade and emboli Nosocomially-acquired	-Staph. aureus(17% early; 12% late) -Corynebacterium -Non-enterococcal streptococci-Fungi e.g Candida spp, Aspergillus spp) -Legionella -HACEK (**H**aemophilus aphrophilus, **A**ctinobacillus actinomycetemcomitans, **C**ardiobacterium hominis, **E**ikenella corrodens, **K**ingella kingae)
	Late(>1 year)	Runs a sub-acute course	
Intravenous drug abuse(IVDA) endocarditis			- Staph.aureus(< 50% of cases) - Groups A, C, and G streptococci enterococci Gram-negative organisms(P aeruginosa and the HACEK)
Nosocomial/health-care-associated endocarditis		Intravascular devices e.g. central lines/catheters, peripheral intravenous catheters Intracardiac devices e.g. pacemakers	Gram-positive cocci (ie,*S aureus,*CoNS, entero-cocci, nonenterococcal streptococci) are the most common pathogens

7:5 Clinical manifestations of infective endocarditis

In patients with infective endocarditis (IE), the present illness history is highly variable. Symptoms commonly are vague, emphasizing constitutional complaints, or complaints may focus on primary cardiac effects or secondary embolic phenomena.

Table 7:2 Clinical presentation of infective endocarditis

Symptoms	Signs

• Common symptoms
  • Fever and chills (most common);
  • Anorexia
  • weight loss
  • malaise
  • headache
  • myalgias
  • night sweats
  • shortness of breath
  • cough
  • joint pains
• Primary cardiac disease may present with signs of congestive heart failure due to valvular insufficiency.
• Secondary embolic phenomena causing vascular obsctruction (in upto 20% cases). Could include:-
  • Acute meningitis
  • focal neurologic complaints(e.g. hemiplegia) due to an embolic stroke or
  • back pain associated with vertebral osteomyelitis
  • unilateral blindness
  • Painless hematuria
  • angina/MI
• Dyspnea, cough, and chest pain are common complaints of intravenous drug users
(This is likely related to the predominance of tricuspid valve endocarditis in this group and secondary emboli to the pulmonary vasculature)
	• Fever (90% of cases)
	• Heart murmurs (85%)
	• Change in a pre-existing murmur (10%)
	• Classical symptoms (50%)
	  • Petechiae
	  • Subungual (splinter) hemorrhages - Dark red linear lesions in the nailbeds
	  • Osler nodes - Tender subcutaneous nodules usually found on the distal pads of the digits
	  • Janeway lesions – Non-tender maculae on the palms and soles
	  • Roth spots - Retinal hemorrhages with small, clear centers; rare and observed in only 5% of patients.
	• Signs of congestive heart failure, such as distended neck veins, frequently are due to acute left-sided valvular insufficiency
	• Splenomegaly may be present
	• Other signs include the following:
	  • Stiff neck
	  • Delirium
	  • Paralysis, hemiparesis, aphasia
	  • Conjunctival hemorrhage
	  • Pallor
	  • Gallops
	  • Rales
	  • Cardiac arrhythmia
	  • Pericardial rub
	  • Pleural friction rub

7:6 Investigations

i. **Blood cultures:** Positive blood cultures remain the cornerstone of diagnosis and provide live bacteria for both identification and susceptibility testing. At least three sets are taken at 30-min intervals, each containing 10 mL of blood, and should be incubated in both aerobic and anaerobic atmospheres. Sampling should be obtained from a peripheral vein rather than from a central venous catheter (because of the risk of contamination and misleading interpretation), using a meticulous sterile technique

ii. Full blood count- anaemia, leukocytosis
iii. Erythrocyte sedimentation rate (ESR)- elevated in 90% cases
iv. Urinalysis- proteinuria, microscopic hematuria (50%)
v. Echocardiography- visible vegetations, abscesses
vi. Two-dimensional Doppler ultrasonography- vegetations, valvular thrombi
vii. Chest radiography-pulmonary pyogenic lesions due to emboli may be visualized
viii. Other studies: CT scan of the head for patients with CNS manifestations

Table 7: 3 Modified duke criteria for diagnosis of infective endocarditis

Major criteria	Minor criteria
Major blood culture criteria • **Two blood cultures positive for organisms typically found in patients with IE (ie, *S viridans, Streptococcus bovis,* a HACEK group organism, community-acquired *S aureus,* or enterococci in the absence of a primary focus)** • **Blood cultures persistently positive for one of the above organisms from cultures drawn more than 12 hours apart** • **Three or more separate blood cultures drawn at least 1 hour apart**	• Predisposing heart condition or intravenous drug use • Fever of 38°C (100.4°F) or higher • Vascular phenomenon, including major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhage, or Janeway lesions • Immunological phenomenon such as glomerulonephritis, Osler nodes, Roth spots, and rheumatoid factor • Positive blood culture results not meeting major criteria or serologic evidence of active infection with an organism consistent with IE (e.g., *Brucella, C burnetii* [ie, Q fever],*Legionella*) • Echocardiogram results consistent with IE but not meeting major echocardiographic criteria
Major echocardiographic criteria • **Echocardiogram positive for IE, documented by presence of an intracardiac vegetation** • **Myocardial abscess** • **Development of partial dehiscence of a prosthetic valve** • **New-onset valvular regurgitation**	

7:7 Management of infective endocarditis

The primary goals of therapy for infective endocarditis (IE) are to:-

- Eradicate the infection, including sterilizing vegetations
- Address the complications of valvular infection
- Intracardiac
- Extracardiac consequences of IE.

Emergent care should focus on making the correct diagnosis and stabilizing the patient with acute disease and cardiovascular instability. General measures include the following:

- Treatment of congestive heart failure
- Oxygen
- Hemodialysis (may be required in patients with renal failure)
- No special diets are recommended for patients with endocarditis; however, if the patient has congestive heart failure, administer a sodium-restricted diet.
- Activity limitations are determined by the severity of the illness, complications (e.g., stroke), and the presence of significant congestive heart failure.

Antibiotics

Antibiotics remain the mainstay of treatment for IE. In the setting of acute IE, institute antibiotic therapy as soon as possible to minimize valvular damage. Three sets of blood cultures are obtained, followed by the infusion of the appropriate antibiotic regimen. By necessity, the initial antibiotic choice is empiric/broad spectrum in nature, determined by clinical history and physical examination findings.

Treatment is essentially always parenteral; oral therapy is less desirable because of the potential for suboptimal patient compliance and possibility of irregular absorption from the gastrointestinal tract. In addition to antimicrobial therapy, supportive care for complications such as heart failure is important.

Table 7:4 Antibiotic treatment of inective endocardiditis due to streptococcus group

Antibiotic	Dosage and route	Comments
Strains penicillin-susceptible (MIC ≤ 0.125 mg/L) oral and digestive streptococci		
Standard treatment: 4-week duration		
Penicillin G Or Cefriaxone	12-18 million U/day i.v either in 4-6 doses or continuously 2g/day i.v. or i.m. in 1 dose **Pediatric doses** Penicillin G 200,000 U/kg/day i.v. in 4-6 doses Cefriaxone 100 mg/kg/day i.v. or i.m. in 1 dose	Preferred in patients. 65 years or with impaired renal or VIII (Vestibulocochlear) cranial nerve functions. 6-week therapy recommended for patients with PVE
Standard treatment: 2-week duration		
Penicillin G or Ceftriaxone combined with Gentamicin	12–18 million U/day i.v. either in 4–6 doses or continuously 2 g/day i.v. or i.m. in 1 dose 3 mg/kg/day i.v. or i.m. in 1 dose **Paediatric doses** Penicillin G and ceftriaxone as above Gentamicin 3 mg/kg/day i.v. or i.m. in 1 dose or 3 equally divided doses	Only recommended in patients with non-complicated NVE with normal renal function.
In beta-lactam allergic patients		
Vancomycin	30 mg/kg/day i.v. in 2 doses **Paediatric doses:** Vancomycin 40 mg/kg/day i.v. in 2 or 3 equally divided doses	6-week therapy recommended for patients with PVE
Strains relatively resistant to penicillin (MIC 0.250–2 mg/l)		
Standard treatment		
Penicillin G or	24 million U/day i.v. either in 4–6 doses or continuously	6-week therapy recommended for patients with PVE

Indications for Surgery

Indications for surgery	Timing
1. Heart failure	
Aortic or mitral NVE or PVE with severe acute regurgitation, obstruction or fistula causing refractory pulmonary oedema or cardiogenic shock	Emergency
Aortic or mitral NVE or PVE with severe regurgitation or obstruction causing symptoms of HF or echocardiographic signs of poor haemodynamic tolerance	Urgent
2. Uncontrolled infection	
Locally uncontrolled infection (abscess, false aneurysm, fistula, enlarging vegetation)	Urgent
Infection caused by fungi or multi-resistant organisms	Urgent/elective
Persisting positive blood cultures despite appropriate antibiotic therapy and adequate control of septic metastatic foci	Urgent
PVE caused by staphylococci or non-HACEK gram-negative bacteria	Urgent/elective
3. Prevention of embolism	
Aortic or mitral NVE or PVE with persistent vegetations .10 mm after one or more embolic episode despite appropriate antibiotic therapy	Urgent
Aortic or mitral NVE with vegetations .10 mm, associated with severe valve stenosis or regurgitation, and low operative risk	Urgent
Aortic or mitral NVE or PVE with isolated very large vegetations (.30 mm)	Urgent
Aortic or mitral NVE or PVE with isolated large vegetations (.15 mm) and no other indication for surgery[a]	Urgent
HACEK = Haemophilus parainfluenzae, Haemophilus aphrophilus, Haemophilus paraphrophilus, Haemophilus influenzae, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae and Kingella denitrificans;	

Indications for antibiotic prophylaxis

In patients with infective endocarditis (IE), the present illness history is highly variable. Symptoms commonly are vague, emphasizing constitutional complaints, or complaints may focus on primary cardiac effects or secondary embolic phenomena.

Table 7:5 Recommendations for prophylaxis of infective endocarditis in the highest-risk patients according to the type of at-risk procedure [5]

A. Dental procedures
Antibiotic prophylaxis should only be considered for dental procedures requiring manipulation of the gingival or periapical region of the teeth or perforation of the oral mucosa
Antibiotic prophylaxis is **not** recommended for local anaesthetic injections in non infected tissues, treatment of superficial caries, removal of sutures, dental X-rays, placement or adjustment of removable prosthodontic or orthodontic appliances or braces or following the shedding of deciduous teeth or trauma to the lips and oral mucosa
B. Respiratory tract procedures[c]
Antibiotic prophylaxis is not recommended for respiratory tract procedures, including bronchoscopy or laryngoscopy, or transnasal or endotracheal intubation
C. Gastrointestinal or urogenital procedures or TOE[c]
Antibiotic prophylaxis is not recommended for gastroscopy, colonoscopy, cystoscopy, vaginal or caesarean delivery or TOE
D. Skin and soft tissue procedures[c]
Antibiotic prophylaxis is not recommended for any procedure
E. Cardiac or vascular procedures
Perioperative prophylaxis is recommended before placement of a pacemaker or implantable cardioverter defibrillator
Perioperative antibiotic prophylaxis should be considered in patients undergoing surgical or transcatheter implantation of a prosthetic valve, intravascular prosthetic or other foreign material
[c]*For patients undergoing surgical procedures involving infected skin (including oral abscesses), skin structure or musculoskeletal tissue, it is reasonable that the therapeutic regimen contains an agent active against staphylococci and beta-haemolytic streptococci.*

Regimen for Prophylaxis

Table 7:6 Recommended prophylaxis for high-risk dental procedres in high-risk patients[5]

Situation	Antibiotic	Single-dose 30-60 minutes before procedure	
		Adults	**Children**
No allergy to penicillin	**Amoxicillin** **or** **Ceftriaxone**	**2g orally or i.v.** **1 g i.v.**	**50mg/kg orally** **50 mg/kg i.v.**
Allergy to penicillin	**Clindamycin**	**600 mg orally or i.v.**	**20 mg/kg orally or i.v.**

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8 Congenital Heart Disease in Children and Adults

8 Congenital Heart Disease in Children and Adults

List of Abbreviations

ASD	Atria Septal Defect
CHD	Congenital Heart Disease
HCTZ	Hydrochorothiazide
VSD	Ventricle Septal Defect

8:1 Definition of Congenital Heart Disease

A range of birth defects that affect the normal workings of the heart. It may be referred to as a structural anomaly of the heart or great vessels that is or could be of functional significance. It is the most common congenital disorder in newborns.

8:2 Aetiology

In most cases its aetiology is unknown but it is associated with an increased incidence in the following conditions or substances affecting the mother in pregnancy.

Table 8:1 Conditions occurring in pregnancy

Infectious Causes	Non-infectious causes
Toxoplasmosis	*Chromosomal abnormalities (e.g Downs syndromes)
Rubella	Phenylketonuria (PKU)
Syphilis	Alcohol abuse
Herpes	Anti-convulsion medications such as phenytoin sodium, benzodiazepines
Cytomegalovirus	Acne medications such as topical retinoid and isotretinoin
Varicella	Organic solvents such as nail polish and glue
Parvovirus B19	Poorly controlled diabetes

***Babies with chromosomal abnormalities have a higher incidence of CHD as compared to normal babies**

It is thought that these defects occur mostly at about week five of pregnancy during the development of the heart from a simple tube like structure into a shape resembling a fully formed heart.

8:3 Epidemiology and Scope of the Problem

It is estimated that 8 in 1000 live births worldwide have a congenital heart disease. About 50% of these children die within one month of age from critical congenital heart disease (1). In Africa, the prevalence of CHD in different countries showed; Cameroon (13.1%) Mozambique (2.3 per 1000) Nigeria (9.3%) predominantly VSD and TOF (1).

The most common acyanotic congenital heart defects are patent ductus arteriosus (PDA), ventricular septal defects (VSDs) and secundum atrial septal defects (ASDs). Tetralogy of Fallot (TOF) is the most common of the cyanotic heart defects identified (2).

In Kenya, there's hardly any data on the prevalence of CHD. However, there was a study published in 1996 where echocardiography done on 1115 school going children found 2 of them with CHD. The lesions found were secundum ASD and VSD with pulmonary stenosis (3).

8:4 Classification of CHD

CHD is generally classified based on how the patients present. This is true for both adults and children. They are broadly classified into 2 groups:

- Acyanotic
- Cyanotic

Please see the figure 14 below.

The adult congenital heart disease population includes those who have

- Never undergone cardiac surgery,
- Have undergone cardiac surgery and require no further operation,
- Have had palliative surgery with or without anticipation of reparative surgery
- Inoperable apart from organ transplant.

Majority of congenital heart diseases are simple and correctable hence the need for a national screening and care program.

8:5 Clinical Presentation

Patients often present with recurrent respiratory tract infections, excessive sweating, easy fatiguability, poor growth or cyanosis depending on the heart defect present.

The illustration below outlines the common symptoms and signs at presentation.

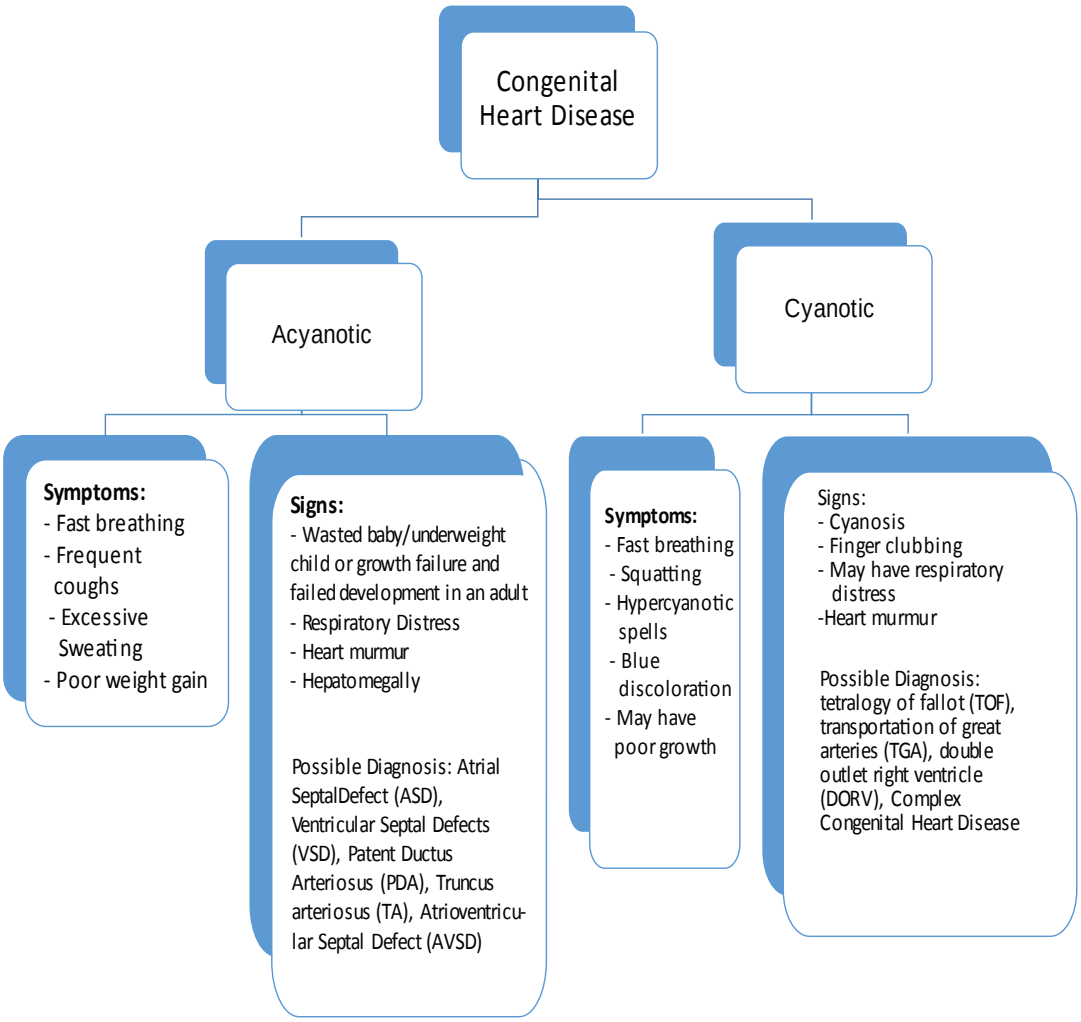

Figure 8.1: Classification of CHD

Recommended action when a potential case is detected is to refer to level 4 facility to see Pediatrician for definitive diagnosis and management plan.

Infants with critical cardiac lesions, the risk of morbidity and mortality increases when there is a delay in diagnosis and timely referral to a tertiary center with expertise in treating these patients.

8:6 Recommendations for Delivery of Care

These patients require prompt diagnosis and referral to specialists in tertiary levels for development of a management plan (both short term and long term). There is room for follow up in lower levels of care depending on the individual case management plan.

Table 8:2 Management by level of care

Resource Needed	Level 2 and 3	Level 4	Level 5 and 6
Human Resources	o Nurses o Clinical officers o Nutritionist o Medical Officer	o Cadres in level 2 and 3 o Physician o Paediatrician o Clinical Pharmacist o Echo-cardiographers	o Cadres in Level 2,3,4 o Paediatric Cardiologist o Perfusionists o Cardiac anaesthetists o Specialised nurses o Cardiothoracic surgeons
Diagnostic Equipment	o BP Machine o Stethoscope o Weighing scale o Height meter o Thermometer o CVD risk assess ment tools o Strips for urinalysi s o Glucom eter o X Ray	o Equipment in level 2 and 3 o ECG machine o ***Echo screening machine o Blood analysis: -Biochemistry: fasting blood sugar, electrolytes, creatinine, lipid profile -Hematological	o Equipment in level 2, 3 and 4 o High specification Echo machine o Ophthalmoscope o Cardiac catheterisation lab o Ambulatory BP o 24 hr Holter machine o Treadmill o Facilities for telemedicine o A critical care unit
Medicatio ns	o Furosemide o Spironolactone o Digoxin*	o All in level 2 and 3 o Beta blockers o Angiotensin converting enzyme inhibitors o Calcium channel blockers (sustained release formulations) o Aspirin o Warfarin	o All in level 2, 3 and 4 o Dopamine o Dobutamine o Sildenafil o Labetalol o Adenosine o Adrenaline o Nitroglycerine o **Others
Main Services	o Detection o Referral o Prophylaxis services	o Services in level 2 and 3 o Treatment of co-morbid general medical conditions o Comprehensive	o Diagnostic / Intervention Cardiac catheterisation and open heart surgery o Treatment of

*prescribed by paediatrician

**any other medications as deemed fit by cardiac teams in various facilities

***a machine that is not elaborate but functions adequately for screening of heart lesions.

There is need to build capacity across all levels of care. This entails training of more cardiologists, specialized nurses and echo-cardiographers. In addition, there needs to be adequate infrastructure (specifically open heart surgery, Interventional cardiology and imaging services) to support

Table 8:3 Heart Failure Medications for Children

Drugs for Management of Heart Failure in children and their common side effects				
Class	**Examples**	**Usual starting dose**	**Maximum daily dose**	**Possible side effects**
Diuretic	Furosemide	1mg/kg/dose	QID	Hypokalaemia Hyponatraemia Hypotension Ototoxicity
	HCTZ	2mg/kg/d divided bd	QID	
Mineralocorticoid Receptor Antagonist	Spironolactone	1mg/kg OD	3.3mg/kg	Hypotension Hyperkalaemia Hyponatraemia Gynaecomastia
ACE inhibitor	Captopril	0.1mg/kg/d PO divided q8h	0.5 mg/kg/d	Cough (ACEI) Hyperkalaemia Hypotension Increased serum creatinine Angioedema Cardiac Arrythmia Bradycardia
	Enalapril	0.1 mg/kg/d PO divided qd/bid	0.5 mg/kg/d	
Digitalis	*Digoxin	From preterm infants: 0.005 mg/kg/d PO divided bid or 75% of this dose IV From age 10yr: 0.005 mg/kg/d PO QID or 75% of this dose IV		
Beta blockers	Carvedilol	0.2mg/kg/dose PO bid; initiate with lower dose and gradually increase dose q2-3wk to therapeutic range	0.4 mg/kg/dose	Aggrevated CHF Malaise Hypotension Bradycardia
	Propanolol	0.5mg/kg/dose given bd	1mg/kg/dose	Bradycardia Hair loss Malaise Diarrhoea
CCB: Calcium channel blocker; ACE: angiotensin converting enzyme; HCTZ: Hydrochlorothiazide OD: administer once daily; BD: administer twice daily; TDS: administer 3 times daily				

8:8 Further Recommendations

1. Infant screening for CHD.

All infants should be subjected to a simple screening process as follows:

a) Midwife to auscultate the babies heart with a stethoscope

b) Pulse oximetry – see below protocol

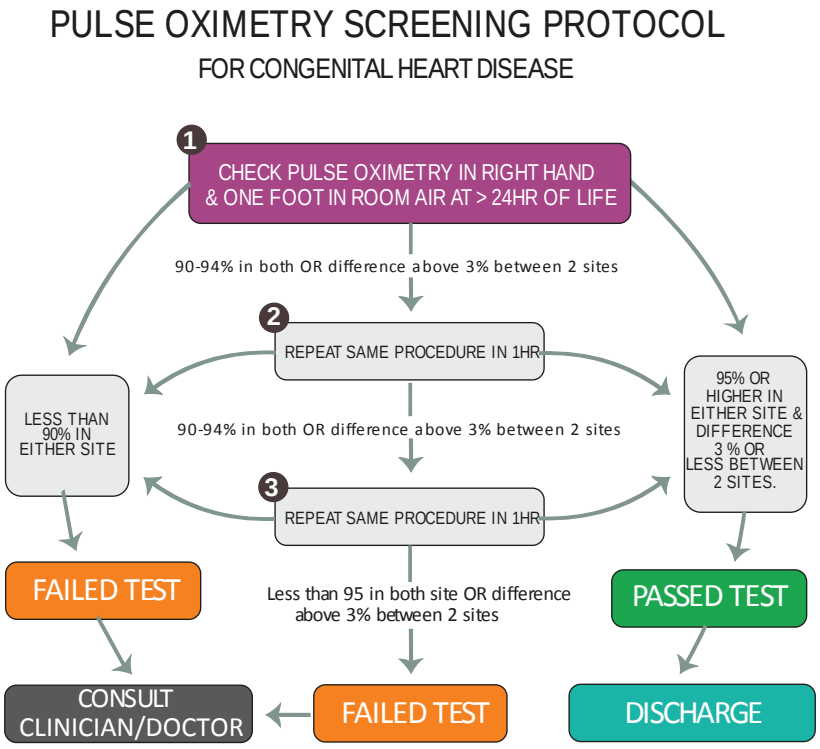

Figure 8.2: Protocol for screening CHD in neaonates

Babies suspected to have CHD should be reviewed by the clinical officer, medical officer or pediatrician/cardiologist (depending on level of facility) before discharge.

2. Counselling of the patient and family by health care provider should include education on:

a. Endocarditis prophylaxis measures where necessary

- Antibiotic prophylaxis is recommended before urethral instrumentation and/or dental procedures that involve manipulation of gingival tissue, the peri-apical region of teeth or perforation of the oral mucosa. This is in patients with CHD with the highest risk for adverse outcome from IE, such as:
  - i. Prosthetic cardiac valve or prosthetic material used for cardiac valve repair
  - ii. Previous IE.
  - iii. Unrepaired and palliated cyanotic CHD, including surgically constructed palliative shunts and conduits.
  - iv. Completely repaired CHD with prosthetic materials, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure.

b. Guidelines for physical exertion (1).

Participation in regular exercise has a well documented benefit for fitness, psychological well-being, and social interaction, as well as having a positive effect on the future risk of acquired heart disease. Recommendations for exercise and sports need to be based on the patient's ability, the impact on underlying haemo-dynamics, and the risk of acute decompensation and arrhythmias. Counselling should be based on the type of sport and the anticipated effort levels. As a general recommendation, dynamic exercise is more suitable than static exercise. In patients with known cardiac conditions, sudden death during exercise is very rare.

c. Contraception and pregnancy information (1).

All women with CHD whether treated or untreated should seek medical advice before getting pregnant. Counseling before pregnancy is important and should include genetic evaluation for the couple. Specifically for women, assessment of potential fetal risk, risk of prematurity or low birth weight in the offspring, review of medications that may be harmful to the fetus, appropriate management of antico agulation, and discussion of potential maternal complications should be done before pregnancy. Additionally, pre-pregnancy counseling is recommended for women receiving chronic anticoagulation with warfarin to enable them to make an informed decision about maternal and fetal risks.

If pregnancy occurs, fetal echocardiography should be obtained and its consequences discussed. Breast feeding is safe in women with CHD. Women requiring cardiovascular medications should be aware that many of the medications will cross into breast milk and should clarify the potential effect of medications on the infant with a pediatrician.

Health providers managing pregnant patients with CHD should have a plan for management of labor and the postpartum period that includes consideration of the appropriate response to potential complications. This care plan should be made available to the patient and to all the health providers.

d. Advice on healthy lifestyle (smoking cessation, weight loss/maintenance, hypertension/lipid screening). Please see chapter xxx for more details.

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9 Venous Thromboembolism

9 Venous Thromboembolism

List of Abbreviations

DVT	Deep Venous Thrombosis
LMWH	Low Molecular Weight Heparin
PE	Pulmonary embolism
UFH	Unfractionated Heparin
VKA	Vitamin K Antagonist
VTE	Venous thromboembolism

9:1 Introduction

Venous thromboembolism constitutes two serious medical conditions, Deep Venous Thrombosis and Pulmonary Embolism. VTE is important because missed diagnosis or delayed treatment can lead to death or debilitating long-term complications, including pulmonary hypertension and post-thrombotic syndrome(1). Following symptomatic DVT, the incidence of severe post-thrombotic syndrome is 10% after 5 years but is as high as 80% following pregnancy associated VTE, mostly occurring within the first 2 years(2). Chronic thromboembolic pulmonary hypertension occurs in 5% of patients with treated PE(2). Cancer associated thrombosis is also an important consideration; age-appropriate cancer screening is recommended for patients presenting with first unprovoked VTE. Certain cancers also predispose to VTE(3,4).

9:2 Epidemiology

1. 1 in 4 deaths globally is due to VTE. 60% of VTE occurs in hospitalized patients (5).

2. PE has been shown to be a frequent cause of cardiovascular mortality, accounting for up to 14.2% of these deaths in Kenya (6). Among those admitted to hospital with PE, there is a 28.1% mortality, while 18.8% of the admissions develop cor-pulmonale (7).

9:3 Risk factors

Lifestyle-related conditions, inherited and acquired disorders can all predispose to VTE. Virchow proposed three major pathophysiologic determinants of VTE including venous stasis, endothelial injury and hypercoagulability. VTE risk factors may be categorized as shown in table 1 below.

Table 8:1 Classification of Risk Factors of VTE(8)

Strong	Moderate	Weak
Fracture (hip or leg)	Arthroscopic knee surgery	Bed rest >3 days
Hip or knee replacement	Central venous lines	Immobility due to sitting (e.g. prolonged car or air travel)
Major general surgery	Chemotherapy	Increasing age
Major trauma	Congestive heart or respiratory failure	Laparoscopic surgery (e.g. cholecystectomy)
Spinal cord injury	Hormone replacement therapy	Obesity
	Malignancy	Pregnancy/ antepartum
	Oral contraceptive therapy	Varicose veins
	Paralytic stroke	
	Pregnancy/, postpartum	
	Previous venous thromboembolism	
	Thrombophilia	

9:4 Diagnosis of DVT

1. Clinical Signs and Symptoms

DVT most commonly develops in the leg veins, but can occasionally also occur in the upper extremities, especially if there is an in-dwelling central venous catheter or a hemodialysis catheter. Common symptoms of proximal lower limb DVT include:

- Sudden onset of unilateral leg pain or tenderness of the thigh or calf.
- Leg swelling (oedema)
- Skin that feels warm to the touch

2. Clinical Probability Testing in suspected DVT

There are many scoring systems to assist diagnosis, but many are difficult to implement. The Wells' Score is a simplified prediction tool for DVT to help the busy clinician working in limited resource settings.

Table 9:2 Wells' Score for suspected DVT (2,9,10)

Clinical features	Score
Active cancer (treatment within last 6 months or palliative)	1 point
Paralysis, paresis, or recent plaster immobilization of leg	1 point
Recently bed-ridden for >3d or major surgery in last 12wks	1 point
Local tenderness along distribution of deep venous system	1 point
Entire leg swollen	1 point
Calf swelling >3cm compared with asymptomatic leg (measured 10cm below tibial tuberosity)	1 point
Pitting edema (greater in the symptomatic leg)	1 point
Collateral superficial veins (non-varicose)	1 point
Previously documented DVT	1 point
Alternative diagnosis** at least as likely as DVT	-2 points
Note: In patients with symptoms in both legs, the more symptomatic leg is used **≥*2 points = DVT likely:*** Perform ultrasound; if positive, treat as DVT. If ultrasound is unequivocal, do D-dimer test. If D-dimer positive and ultrasound negative, repeat ultrasound in 1 week. If both D-dimer and ultrasound negative, DVT excluded ***<2 point = DVT unlikely:*** Perform D-dimer (where available). If negative, DVT excluded. If positive, proceed to Ultrasound (if ultrasound negative, DVT excluded; if positive, treat as DVT).	

** An alternative diagnosis may include: superficial phlebitis, post-thrombotic syndrome, cellulitis, muscle strain or tear, leg swelling in paralyzed limb, venous insufficiency, edema due to CCF or cirrhosis, external venous obstruction (e.g. due to tumor), lymphangitis or lymphedema, popliteal (Baker's) cyst, hematoma, pseudo aneurysm or knee abnormality

3. Imaging in DVT

The gold standard test for diagnosing lower or upper extremity DVT is venous compression ultrasonography (CUS). Failure to compress the vein is diagnostic of DVT.

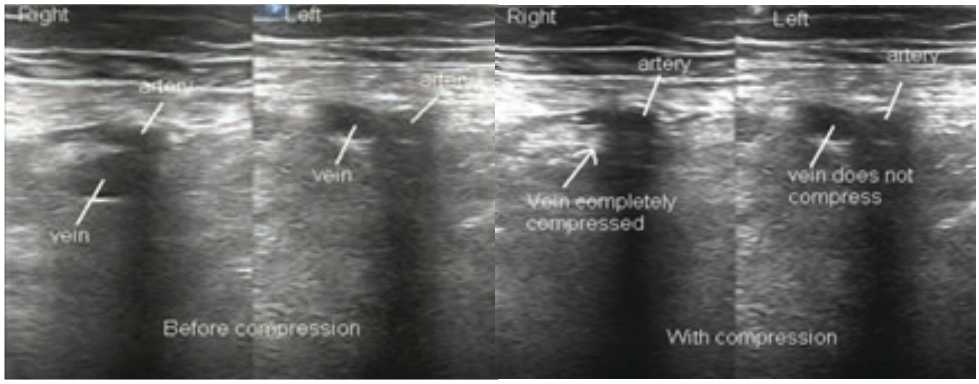

Figure 9.1: Ultrasonography images of femoral vessels before and with compression (11)

Alternative imaging using CT, MRI and contrast venography are reserved for imaging segments that are not easily assessed by CUS, e.g. pelvic veins, or subclavian vein DVT.

4. Laboratory testing in DVT

Plasma D-dimer is a non-specific marker of fibrin lysis anywhere in the body. It may be elevated in VTE but also in many other conditions, including Myocardial Infarction (MI), heart failure, infection, surgery, aortic dissection and pregnancy. The D-dimer test is very useful in the evaluation of outpatients or in casualty setting in patients with suspected VTE. The D-dimer test may be positive but CUS shows no evidence of proximal DVT. In this subgroup of patients, it is advised to repeat CUS in 7 days to rule out DVT

5. Diagnostic algorithm for suspected DVT

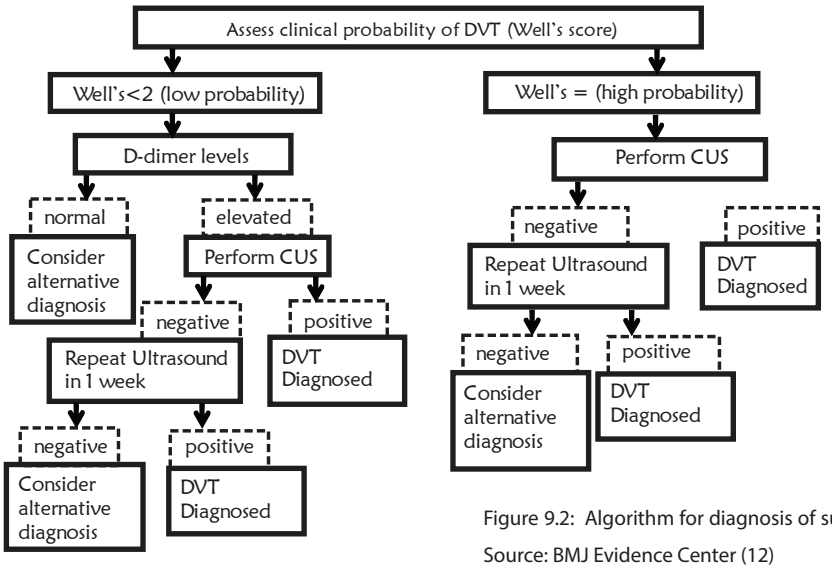

Figure 9.2: Algorithm for diagnosis of suspected DVT);
Source: BMJ Evidence Center (12)

9:5 Diagnosis of Acute Pulmonary Embolism

1. Clinical Presentation

The clinical presentation of acute PE may vary widely and the signs and symptoms are often non-specific. Common features include dyspnea, cyanosis or fainting in massive acute PE. Large thrombi obstruct the pulmonary arterial tree and cause hemodynamic and gas exchange abnormalities, resulting in hypotension, hypoxemia and increased Right Ventricular afterload. Clinical features may include the following:-

- Unexplained sudden onset of dyspnea
- Lightheaded sensation or syncope
- Pleuritic chest pain
- Pulse≥ 100 beats/minute
- Heart murmur of tricuspid regurgitation, loud P2
- Distended neck veins.

2. Assessment of clinical probability in acute PE

The combination of clinical findings and the use of prediction rules can help to classify patients with suspected PE into categories of probability that correspond to an increasing actual prevalence of confirmed PE on further testing.

Clinical Feature	Score
Previous PE or DVT	1
Pulse ≥ 100 beats/minute	1
Surgery or immobilization within the past 4 weeks	1
Hemoptysis	1
Active cancer	1
Clinical signs of DVT	1
Alternative diagnosis less likely than PE	1
Clinical Probability	
PE likely	≥2
PE unlikely	0-1

3. Radiologic imaging in Acute PE

Multi-detector computed tomographic angiography (CT pulmonary angiography) is recommended in outpatients with an elevated plasma D-dimer and in patients with a high clinical probability. The CTPA is diagnostic for acute PE when it shows a thrombus in the pulmonary arterial tree. With regards to prognosis, a dilated right ventricle on CT imaging points to Right Ventricular dysfunction and increased severity of PE with worse prognosis.

4. Laboratory Testing in suspected Acute PE

Plasma D-dimer testing combined with clinical probability assessment should be carried out in patients with suspected acute PE in the absence of shock. D-dimer should not be measured in patients with a high clinical probability or in hospitalized patients.

In patients with hypotension of suspected massive acute PE, cardiac biomarker testing of NT-Pro BNP or Troponin may be useful in risk stratification of severity of acute PE. Elevated cardiac biomarkers on admission have been reported and are associated with worse prognosis.

5. Predicting early mortality in acute PE

In patients with acute PE it is important to consider their prognosis as certain patients carry a very higher mortality risk and will need admission to critical care departments for monitoring and treatment. Other patients may have a very low risk of 30-day mortality, supporting admission to hospital in a general ward or even treatment at home with very close supervision. The following features indicate higher 30-day mortality in acute PE:

- Shock
- Pulmonary Embolism Severity Index (PESI) Class(14)
- Signs of Right Ventricular dysfunction on echocardiography or CT imaging
- Elevated cardiac biomarkers.

9:6 Prevention of Venous Thromboembolism

The use of VTE prophylaxis should be considered in all hospitalized patients but implementation of mechanical or pharmacological measures is inconsistent in many institutions and among practitioners. It is recommended that health facilities have an admission policy that assesses VTE risk for all medical and surgical patients, in order to identify at-risk patients in whom VTE prophylaxis can commence. The risk of VTE persists after hospital discharge in many patients. VTE mortality has been shown to rise between 10 - 35 days following hospital admission among acutely ill medical patients. A sample of a risk evaluation and recommendation tool (20) is provided in annex 2 .

1. Pharmacological Thrombo-prophylaxis

Recommendations for hospitalized patients at risk of VTE include:

- Enoxaparin 40mg SQ OD
- Unfractionated Heparin 5000 SQ BD
- Fondaparinux (where available) 2.5mg SQ OD

2. Mechanical Thromboprophylaxis

Graduated compression stockings/Anti-embolism stockings and intermittent pneumatic compression devices (where available) should be considered for at-risk patients who are NOT candidates for pharmacological thromboprophylaxis due to high risk of bleeding pre-operatively, intra-operatively and post operatively. Mechanical prophylaxis is effective when used in combination with early ambulation.

Standards for the use of anti-embolism stockings(15)

- Patients should have their legs measured and the correct size of stocking provided.
- Use anti-embolism stockings that provide graduated compression and produce a calf pressure of 14–15 mmHg.
- Encourage patients to wear their anti-embolism stockings day and night until they no longer have significantly reduced mobility
- Remove anti-embolism stockings daily for hygiene purposes and to inspect skin condition.
- In patients with a significant reduction in mobility, poor skin integrity or any sensory loss, inspect the skin two or three times per day, particularly over the heels and bony prominences
- Discontinue the use of the stockings if there is marking, blistering or discoloration of the skin, particularly over the heels and bony prominences, or if the patient experiences pain or discomfort. If suitable, offer intermittent pneumatic compression (IPC) device as an alternative.

Standards for Intermittent Pneumatic Compression

- Proper fitting of the device
- Compression pressure and cycling time: IPC pressure of 120–140mmHg applied to foot and calf with a one-second delay, and at a frequency of three to four impulses per minute(16).

Contraindications to mechanical prophylaxis(17)

- Peripheral artery disease, including history of peripheral arterial bypass grafting
- Severe peripheral neuropathy or other cause of sensory impairment
- Allergy to stocking material
- Massive leg edema or pulmonary edema from congestive cardiac failure
- Local skin or soft-tissue condition, including recent skin graft, fragile skin, gangrene, severe dermatitis and cellulitis
- Extreme deformity of the leg, or unusual leg shape or size preventing correct fit
- Documented DVT

9:7 VTE Treatment & Patient Management

1. **Anticoagulation**

Anticoagulation is the treatment foundation for patients with VTE. It is administered sequentially as follows:

- Initial anticoagulation in patients with acute DVT and PE
- Long-term anticoagulation (usually up to 3 months)
- Extended- treatment (beyond 3 months in select patients)

Current options include the following:-

- Low Molecular Weight Heparin
- Fondaparinux
- Unfractionated heparin
- Warfarin
- Direct Oral Anti-Coagulants

Low Molecular Weight Heparin

LMWHs are dosed according to weight and are administered subcutaneously; they offer the advantage of no routine laboratory monitoring while in hospital. They are as safe and effective as unfractionated heparin when starting anticoagulation in a patient with VTE. For patients with active cancer who develop VTE, treatment with LMWH is advised over warfarin.

- Dose: Enoxaparin 1 mg/kg SQBD
- Renal Dosing: A dose reduction is recommended in patients with severe renal insufficiency (GFR < 30 ml/min): 1mg/kg SQ OD

Fondaparinux

Fondaparinux is a synthetic pentasaccharide that has been shown to be safe for DVT and acute PE. It is administered once-daily according to weight and does not require routine monitoring or dose adjustment.

Dose:

- 5 mg SQ once daily (wt.<50kg)x 5 days
- 7.5mg SQ once daily (wt. 50-100kg)x 5 days
- 10mg SQ once daily (wt.> 100kg) x 5 days

Fondaparinux should not be used in patients with renal impairment(2)

Unfractionated Heparin

- Intravenous (IV) infusion regimen: Give heparin 60IU/Kg IV bolusTHEN infuse at a rate of 18 units/kg/h. Check APTT at 6h, aim for APTT of 60-85 seconds or APTT ratio of 1.5–2.5.Measure APTT daily or 10h after dose change(10,18).
- Subcutaneous Regimen-17,500IU SQ BD
  UFH is recommended in patients with a CrCl<30 mL/min with dose adjustments based on the APTT(2).

NOTE: Parenteral anticoagulants (e.g. LMWH, fondaparinux or UFH, in that order of preference) are used in the initial/acute phase of treatment during hospitalization. If planning to use VKA or long-term anticoagulation, warfarin can be started from day 1. Heparin should be continued until INR has reached target therapeutic range and until day 5, as warfarin has an initial prothrombotic effect(10). There is risk of bleeding and thrombocytopenia when using heparin so monitoring the hemoglobin and platelet count is important when commencing therapy.

Vitamin K Antagonists - Warfarin

- Recommended Starting Dose: 5 mg PO OD
  Initiate warfarin on day 1 or 2 of parenteral anticoagulation therapy. Do INR daily and adjust dose accordingly. Overlap warfarin and parenteral anticoagulant until desired INRis maintained for 24 hours, and then discontinue parenteral therapy.
- Typical maintenance dose: 2 to 10 mg orally once a day. Dosage must be individualized according to the patient's INR.
- Target INR: 2.5 (range: 2 to 3)
- Renal Dose: No dosage adjustment is necessary for patients with renal failure. However, some studies suggest lower warfarin doses; in those with renal impairment(19)
- Important drug-drug interactions: Warfarin has interactions with many drugs including some antibiotics (including some anti-TBs), antifungals, antiretrovirals, cardiovascular drugs, corticosteroids, anticonvulsants, contraceptives among others(2,10). Consult with pharmacist/pharmaceutical technologist before prescribing other medications that may alter the efficacy/potentiate the effects of warfarin.
- Contraindications: Peptic ulcers, bleeding disorders, severe hypertension, pregnancy. Use with caution in the elderly and those with past gastrointestinal bleeds(10).
- Dietary restrictions: Warfarin has many food interactions. Consult a dietitian nutritionist as part of the initial patient counseling for warfarin therapy.

Direct Oral Anti-Coagulants (DOACs)

Factor Xa inhibitors (rivaroxaban and apixaban) and direct thrombin inhibitors (dabigatran) are newer oral anticoagulants. They offer the advantage of fixed dosing without need for intense coagulation monitoring. Treatment for VTE can commence immediately with these drugs in patients with DVT or acute PE without the need for monitoring, though it is important to consider bleeding risk an all patients starting anticoagulation.

Dose:

- Rivaroxaban 15 mg BD as the loading dose for 21days followed by 20 mg daily.
- Dabigatran: 150mg BD after 5 days of parenteral therapy

2. Duration of Anticoagulation Therapy in VTE

The importance of anticoagulation therapy in patients with VTE is to prevent recurrence. VKAs are used in most cases, while LMWH are preferred in patients with VTE and active cancer. The DOACs have been utilized as an alternative to VKA for long term treatment of VTE.

Recommendations for duration of Anticoagulation after VTE

- For patients with VTE secondary to a transient (reversible) risk factor, oral anticoagulation is recommended for 3 months.
- For patients with first time unprovoked VTE, oral anticoagulation is recommended for at least 3 months.
- Anticoagulation treatment in patients with a second episode of unprovoked VTE should have anticoagulation treatment of indefinite duration as guided by a Physician. The risk-benefit ratio of continuing extended anticoagulation should be reassessed at regular intervals.

3. Antidotes(10)

- If UFH overdose: stop infusion. If there is bleeding, administer protamine sulfate in consultation with a physician.
- Warfarin: In case of any major bleed (including intracranial hemorrhage), stop warfarin. Give prothrombin complex concentrate 50units/kg (where available) in consultation with a Physician. If unavailable, give Fresh Frozen Plasma (FFP) (15mL/kg). Also give 5–10mg vitamin K IV slowly.

9:8 Important considerations

- Monitoring of bleeding- take note of gastrointestinal, brain, skin and urological bleeding
- Dosing, especially in renal disease
- Special groups-Pregnancy, HIV, elderly and children, sickle cell disease (refer to appropriate experts)
- Precautions: Drug interactions when on antiplatelets; warfarin and its interactions
- Follow-up- with regards to the efficacy of anticoagulation and recurrence of VTE

9:9 Practice recommendations

- Thrombo-prophylaxis assessment on admission of patients to medical and surgical wards
- Anticoagulation monitoring- multidisciplinary anticoagulation clinics with designated staff e.g. anticoagulation nurses and pharmaceutical technologists
- Strengthening of Research and Health Information Systems in relation with VTE.
- Training of specialists in thrombosis and hemostasis

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10 Stroke

10 Stroke

List of Abbreviations

ABC	Airway, Breathing, Circulation
DVT	Deep Venous Thrombosis
LMWH	Low Molecular Weight Heparin
IHD	Ischemic Heart Disease
NGT	Naso-gastric Tube
TIA	Transient Ischemic Attack
UFH	Unfractionated Heparin

10:1 Introduction

Stroke: Rapidly developing episode of focal or at times global loss of cerebral functions with symptoms lasting more than 24 hours or leading to death, and with no apparent cause other than that of a vascular origin. This cuts off the supply of oxygen and nutrients, causing damage to the brain tissue (1).

Transient Ischemic Attack (TIA): a focal neurological or visual deficit caused by interruption in blood supply to the brain (or retina), whose cause is presumed to be vascular, in which all symptoms resolve within 24 hours.

Burden (Public health implications, Economic implications)

According to the World Health Organization (WHO), around 15 million people suffer from stroke each year globally and an estimated 5•8 million deaths are attributed to stroke (2). Two-thirds occurred in low-income and middle-income countries whereas burden is reducing in developed countries, it is increasing in developing countries where it is the 3rd leading cause of mortality and 2nd leading causing of disability. Sub-Saharan Africa is undergoing epidemiological transition. Stroke and other vascular diseases increasingly contribute to the burden of disease.

Pathophysiology

The etiologies of stroke are varied, but as shown , they can be broadly categorized into:

- Ischemic
- Hemorrhagic

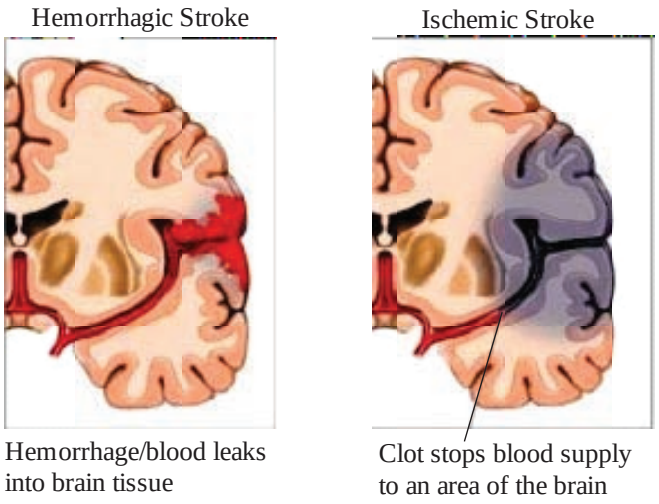


Figure 10.1 Types of stroke lesions

10:2 Ischemic stroke

Approximately 80-87% of strokes are from ischemic infarction caused by thrombotic or embolic cerebrovascular occlusion. These may result from thrombosis in brain artery, embolus from heart or major arteries like carotid artery.

10:3 Hemorrhagic Stroke

This results from a weakened vessel that ruptures or defects in coagulation leading to bleeding into the surrounding brain. The blood accumulates and compresses the surrounding brain tissue. The two types of hemorrhagic strokes are intracerebral (within the brain) hemorrhage or subarachnoid hemorrhage. Hemorrhagic stroke accounts for 13% -20% stroke.

10:4 Risk factors for Stroke

Local studies have shown 80% of stroke patients had hypertension and 34% had diabetes mellitus2. Other risk factors included dyslipideamia, atrial fibrillation, obesity and HIV infection. The global interstroke study reported hypertension as the most significant risk factor for stroke occurrence. Other risk factors included current smoking, waist-to-hip ratio and diabetes mellitus (3).

Table 10:1 Risk factors for stroke

Risk factors for ischemic stroke

Non-modifiable
• Advanced age
• Race
• Male sex
• Previous history of stroke
 	
Modifiable
• Hypertension (the most important)
• Diabetes mellitus
• Cardiac disease
  • Valvular heart diseases
  • Heart failure
  • Arrhythmias
• Tobacco use
• Excessive alcohol use
• Hypercholesterolemia
• TIAs
• Obesity/Sedentary lifestyle
• Oral contraceptive use
• Sickle cell disease
• HIV infection

Risk factors for hemorrhagic stroke
• Hypertension • Alcohol use • Advanced age • Coagulopathies • Eclampsia • Arteriovenous malformation (AVM), aneurysms, and other vascular malformations (venous and cavernous angiomas) • Dural sinus thrombosis • Cerebral amyloidosis • Vasculitis • Intracranial neoplasm • Use of illicit drugs (e.g. cocaine, other sympathomimetic drugs) • Anticoagulant therapy • Thrombolytic therapy

10:5 Prevention

Primary stroke prevention refers to the treatment of individuals with no history of stroke. Secondary stroke prevention refers to the treatment of individuals who have already had a stroke or transient ischemic attack (4).

Primary Prevention of Stroke

Risk-reduction measures in primary stroke prevention include:

- Optimise treatment for diabetes, hypertension, obesity and lipidemia
- Mitigate behavioral risk factors e.g tobacco use, alcohol use, physical inactivity, unhealthy diet
- Screen for hypertension, obesity, diabetes, lipidemia in the community and refer cases to health facilities for care

  Refer to chapter on prevention

Secondary Prevention of Stroke

Preventing recurrent stroke in patients with ischaemic stroke or TIA is crucial to minimize damage to brain tissue and therefore degree of disability.

Measures may include use of the following:

- Anti-platelet agents
- Anti-hypertensives
- Statins
- Anti-coagulants
- Lifestyle interventions

Low-dose aspirin (75 mg daily) or Clopidogrel (75 mg daily) should be prescribed after ischaemic stroke or TIA for secondary prevention of recurrent vascular events.

Secondary prevention can be summarized by the mnemonic A, B, C, D, E, as follows:

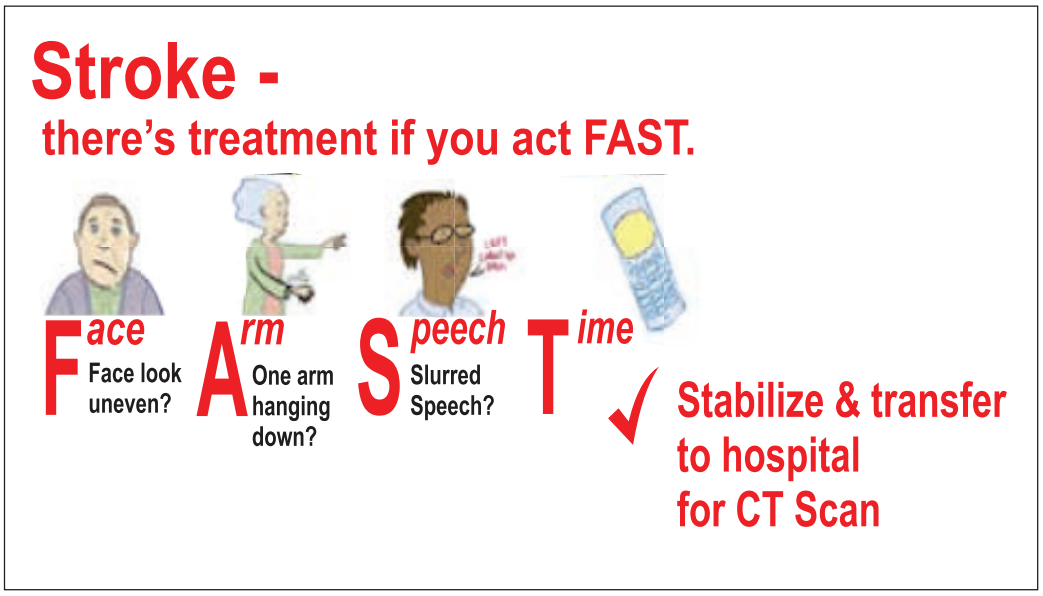
A Antiaggregants (aspirin, clopidogrel, extended-release dipyridamole, ticlopidine) and anticoagulants (apixaban, dabigatran, edoxaban, rivaroxaban, warfarin)
B Blood pressure–lowering medications
C Cessation of cigarette smoking, cholesterol-lowering medications, carotid revascularization
D Diet
E Exercise

Smoking cessation, blood pressure control, diabetes control, a healthy diet, weight loss, and regular exercise should be encouraged.

10:6 Diagnosis

TIME IS BRAIN – This is a popular phrase that call for urgent action when managing stroke. Permanent damage to brain tissue occurs very quickly and therefore to minimize the level of disability following stroke, treatment must be initiated as soon as possible (5).

Below is a simplified criteria for detecting stroke.



The three signs most diagnostic of stroke are (FAST)

- Facial paresis
- Arm drift
- Abnormal Speech
- Time (Symptoms persist for >24hr)

Pre-hospital assessment

Standard assessment tools as shown are used to improve the speed and accuracy of diagnosis in patients with suspected stroke, although there is a small risk of wrong diagnosis. The aim of using these tools is to minimize delay in initiating treatment.

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Diagnosis of Stroke

Table 10:2 Diagnosis of Stroke

Component	Details
Proper history taking	Consider stroke in any patient presenting with acute neurologic deficit or any alteration in level of consciousness. Determine onset time Progression of presenting complaints Determine risk factors and co-morbidities Family and social characteristics eg occupation Common stroke symptoms include the following: • Abrupt onset of extremity weakness • Hemi sensory disturbance • Visual disturbance • Abnormal speech • Facial droop • Abnormal gait or posture • Dizziness and loss of balance • Sudden decrease in level of consciousness Although such symptoms can occur alone, they are more likely to occur in combination. No historical feature distinguishes ischemic from hemorrhagic stroke, although nausea, vomiting, headache, and sudden change in level of consciousness are more common in hemorrhagic strokes.
Physical examination	• Assessment of the ABCs, take vitals - BP, temperature, pulses • General examination: Examination of the head and neck may reveal signs of trauma or seizure activity (e.g., contusions, tongue lacerations), carotid disease (bruits), and jugular venous distention. • Cardiac examination focuses on identifying concurrent myocardial ischemia, valvular conditions, irregular rhythm, or congestive heart failure • Respiratory examinations • Neurological Examination - use Glascow Coma Scale or stroke scale The goals of the neurologic examination include the following: • Confirming the presence of a stroke syndrome • Distinguishing stroke from stroke mimics • Establishing a neurologic baseline, should the patient's condition improve or deteriorate • Establishing stroke severity, using a structured neurologic exam and score (National Institutes of Health Stroke Scale [NIHSS]) to assist in prognosis and therapeutic selection – Check in annex Essential components of the neurologic examination include the following evaluations: • Cranial nerves • Motor function

10:6 Management

Emergency medical services should be redesigned to facilitate rapid access to specialist stroke services; Patients with suspected stroke should have:

- Rapid referral to appropriate centre; ambulance priority in appropriate cases (functional ambulance),
- Rapid triage on arrival at hospital,
- Immediate access to specialist stroke services,
- Rapid brain imaging
- Rapid specialist assessment.

Initial supportive care

Table 10:3 Recommended supportive care

Step by Step Procedure
1) Manage ABCs
2) Monitor Blood Pressure and other vital signs every 15 min for the first hour and hourly thereafter
3) Gain large bore intravenous access 4) Oxygen (as required O_2 saturation 92%) 5) Assess for hypoglycemia or hyperglycemia 6) Take blood sample for lab analysis (refer above) *7)* Maintain *Nil per oral* (NPO) and insert NGT to prevent aspiration 8) Get CT scan of the brain as soon as possible 9)Admit patient or organise for Referral to closest appropriate facility capable of treating acute stroke 10) Alert receiving Hospital/Emergency Department

Avoid

1. Dextrose-containing fluids in non-hypoglycemic patients
2. Hypotension/excessive blood pressure reduction
3. Excessive intravenous fluids

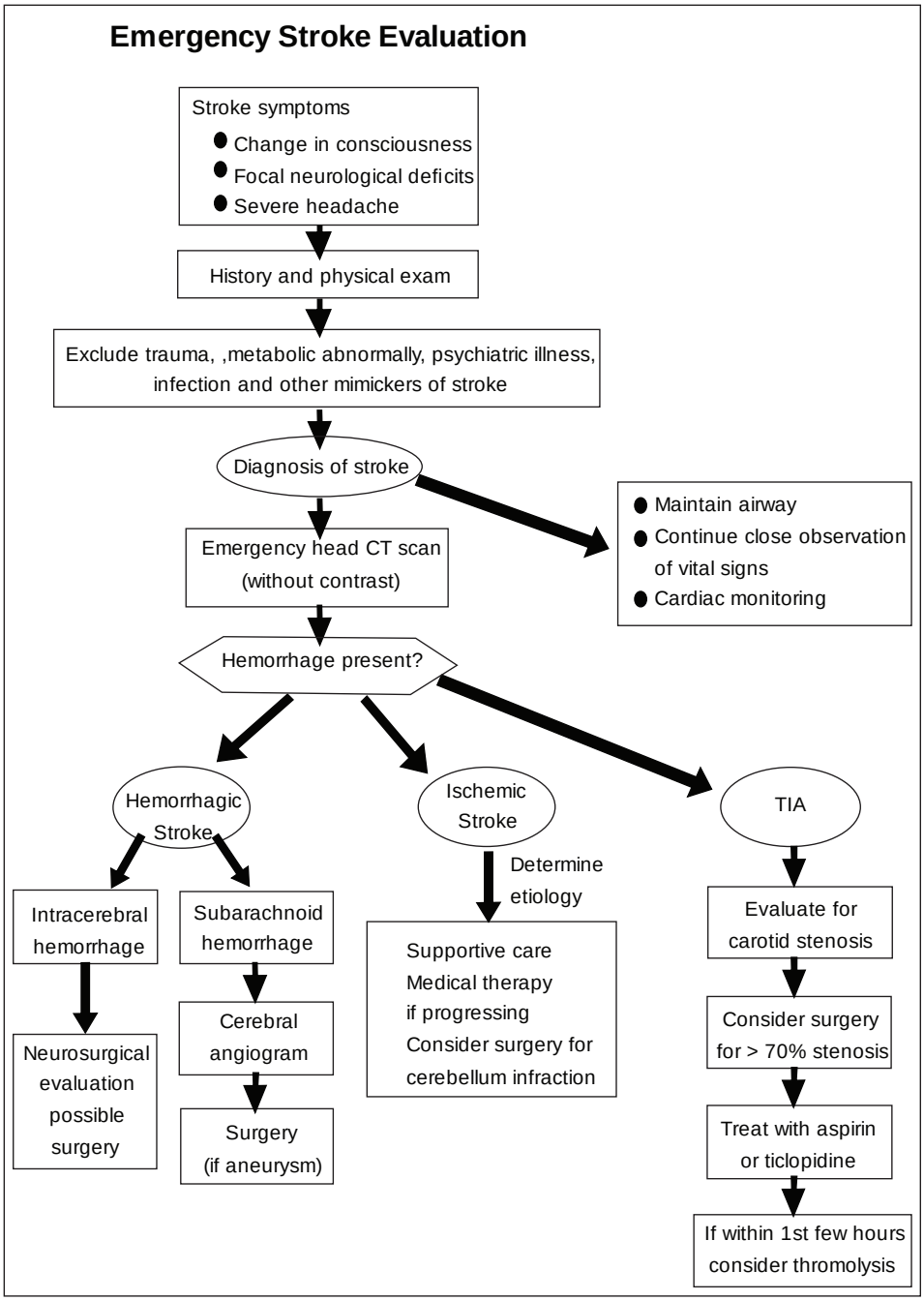

Figure 10.2: Protocol for diagnosis of stroke

10:7 Treatment of ischemic stroke

Table 10:4 Medications used for ischemic stroke

Thrombolysis	Patients admitted with stroke within four and a half hours of definite onset of symptoms, who are consider table, should be treated with 0.9 mg/kg (up to maximum 90 mg) intravenous rt-PA. NB: Emphasis is on early diagnosis and referral for possible thrombolytic therapy. Thrombolytic therapy with rt-PA (alteplase 0.9 mg/kg up to maximum 90 mg) administered within four and a half hours of stroke onset according to protocols stated in the product licence significantly reduces death and disability at 90 days. The odds of a favourable outcome (full or nearly full recovery from stroke) are strongly related to the time to treatment and are significantly greater the earlier that treatment is delivered. I***ntra-arterial thrombolysis should only be carried out by an appropriately trained interventional neuro-radiologist.***
Antiplatelet Agents	Aspirin 75mg or clopidogrel 75mg daily started immediately where thrombolysis is not available.
Anticoagulants	Anticoagulant therapy is recommended in cardio-embolic event. Refer to chapter xx (CAD section) for details.
Statins	Statins should be prescribed to patients who have had an ischaemic stroke, irrespective of cholesterol level.

10:8 Management of Haemorrhagic Stroke

The treatment and management of patients with acute intracerebral hemorrhage depends on the cause and severity of the bleeding.

1. Basic life support
2. Nil by mouth and NGT insertion to prevent aspiration pneumonia
3. Control of bleeding, seizures, blood pressure (BP), and intracranial pressure, are critical
4. Medications used in the treatment of acute stroke include the following:
   - Anticonvulsants - To prevent seizure recurrence
   - Antihypertensive agents - To reduce BP and other risk factors of heart disease
   - Osmotic diuretics - To decrease intracranial pressure in the subarachnoid space

A potential treatment for hemorrhagic stroke is surgical evacuation of the hematoma. However, the role of surgical treatment for supratentorial intracranial hemorrhage remains controversial.

10:8 Treatment of comorbid conditions

May include the following:

- Reduce fever
- Correct hypotension/significant hypertension
- Correct hypoxia
- Correct hypoglycemia
- Manage cardiac arrhythmias
- Manage myocardial ischemia

10:9 Inpatient Supportive Care

1. **Mobilise early to prevent complications** such as pneumonia, deep vein thrombosis, pulmonary embolism, and pressure sores
2. **Nutrition and Hydration**
   a. Assess ability to swallow by performing a water swallowing test at the bed side. Patients able to swallow are encouraged to feed orally. Patients not able to swallow are inserted a nasogastric tube for feeding and oral medication where necessary.
   b. Dehydration is a potential cause of deep vein thrombosis after stroke. Hydration is sustained orally for those able to swallow or intra-venously for those unable to swallow. Hydration status is monitored by maintenance of an input-output chart.
   c. Bowel management to avoid constipation and fecal impaction or diarrhea is required from the outset.
3. **Infection Prevention**
   a. Pneumonia, which is most likely to occur in seriously affected, immobile patients and those who are unable to cough, is an important cause of death after stroke.
      - Protect the airway and carry out suctioning as required to lower the risk of aspiration.
      - Treat nausea and vomiting to lower the risk of aspiration pneumonia.
      - Exercise and encouragement to take deep breaths to lessen the development of atelectasis
      - Monitor vital signs for fever and tachypnoea and treat for pneumonia upon diagnosis.

   b. Urinary tract infections are relatively common among patients with stroke with bacteremia or sepsis as a potential complication.
      - Screen urine for infection in the occurrence of fever after stroke

4. **Deep Vein Thrombosis and Pulmonary Embolism Prevention**
   The risk of deep vein thrombosis is highest among immobilized and older patients with severe stroke. Symptomatic deep vein thrombosis also slows recovery and rehabilitation after stroke. Pulmonary emboli generally arise from venous thrombi that develop in a paralyzed lower extremity or pelvis.

10:10 Long Term Care

Psychosocial support:

Providing information and support: Information should be offered to patients and carers in a variety of formats, including easy access in the living environment. Caregivers should be offered ongoing practical information and training individualised for the needs of the person for whom they are caring for.

- Clinical care referral for investigations and/or further management
- Rehabilitation in terms of feeding, toileting, walking, writing etc

Assessment of degree of dependency: Once a patient has been admitted, the use of standard impairment scales, such as Canadian neurological scale (CaNS), Scandinavian stroke scale (SSS) score or the Los Angeles motor scale (LAMS), can be predictive of the degree of dependency and length of hospital stay.

Other components of long term care should be applied as part of the management plan depending on the individual needs of the patient. These include:

- Palliative care
- Pain management
- Physiotherapy
- Occupational therapy
- Speech therapy
- Psychotherapy
- Nutritional support
- Treatment of contracture, urinary bladder problems including infections and catheterization, bed sore management etc

10:11 Management of Stroke at different Levels of care

Table 10:5 Recommended stroke services by levels of care

Level of Service Delivery	Action Taken
Community level	Refer to nearest facility with basic emergency services
Level 2 – Dispensary	Basic emergency – ABCs Detection and emergency care Refer to specialist or county referral
Level 3 – Health centres	Basic emergency – ABCs Detection and emergency care Refer to specialist or county referral
Level 4 – sub county hospital	Basic emergency – ABCs Diagnosis and initiation of treatment Refer to specialist or county referral if necessary
Level 5 – County Referral Hospital	Basic emergency – ABCs Diagnosis and initiation of treatment Definitive management Rehabilitation and palliation Training
Level 6 – National Referral Hospital	Definitive Management Rehabilitation and Palliation Training Research

Referral criteria:

- Rapidly deteriorating Glasgow Coma Scale (GCS) or Rapidly deteriorating Glasgow Coma Scale (GCS) or GCS of of 8 or below
- Onset of symptoms < 4.5 hours for all ischemic CVAs for possible thrombolysis in Centre capable for this for all ischemic CVAs for possible thrombolysis in Centre capable for this
- Evidence of subdural hemorrhage on Brain CT scan
- Evidence of subarachnoid hemorrhage
- Deranged UECs
- If unable to carry out basic lab tests or to obtain CT scan Brain
- If unable to manage comorbid conditions
- Evidence of subarachnoid hemorrhage on Brain CT scan
- CT scan evidence of brain edema or increased intracranial pressure

Recommendations for Health System Strengthening

Certain interventions have been shown to be effective:

- Education programmes to improve the general public's recognition of symptoms of stroke
- Training paramedics to diagnose stroke more accurately and decrease time to hospital
- Urgent ambulance transfer
- Training emergency medical staff in acute stroke care
- Multifaceted interventions (including telemedicine systems and tele-radiology).
- Reorganisation of hospital systems to allow for dedicated stroke management teams. There is evidence of better clinical outcomes and shorter hospital stay in patients managed in a 'stroke unit' rather than admitted to a general ward or remaining at home. Nb: in a resource constraint set up, a section of the general ward can be dedicated for stroke patients only.

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11 Cardiovascular Diseases among the elderly

11 Cardiovascular disease among the elderly

List of Abbreviations

YLD	Years lost to disability

11:1 Introduction

Adults above 65 years of age currently constitute about 2.7% of the total Kenyan population as of early 2017, or 1, 285, 585 persons [1]. Elderly people are at a higher risk of developing cardiovascular disease [2], and is associated with higher rates of mortality among this age bracket. Management of cardiovascular disease among elderly persons has to incorporate special considerations due to presence of comorbidities, organ deficiencies and impaired cognitive abilities to direct their own health care.

11:2 Epidemiology

The mortality from cardiovascular disease in elderly people above 60 years in Kenya ranges from 346.3 per 100,000 in those between 60-65 years to 4264.6 per 100000 in those above 80 years, with a combined 122.8 years lost to disability (YLD) per 100,000 population (3). The main risk factors for the elderly population include high systolic blood pressure, dietary patterns, high body mass index, air pollution and tobacco smoke. The leading causes of cardiovascular morbidity and mortality in this age group include ischemic heart disease, stroke and hypertensive heart disease [3]. Management of the risk factors has a major role in reducing the burden of cardiovascular disease among older people globally.

11:3 Pathophysiology [4]

Table 11:1 Age associated changes and cardiovascular disease in older people

Organ	Age-associated changes	Cardiovascular disease
Vasculature	Increased intimal (wall) thickness	Systolic hypertension Early atherosclerosis
	Arterial stiffening	Coronary artery obstruction Systolic hypertension Left ventricular wall thickening Stroke Atherosclerosis
	Increased pulse pressure	Peripheral artery obstruction
	Increased pulse wave velocity	Carotid artery obstruction
Atria	Increased left atrial size Atrial premature complexes	Atrial fibrillation
Sinus node	Decreased maximal heart rate Decreased heart rate variability	Sinus node dysfunction
Atrioventricular node	Increased conduction time	Heart block
Valves	Sclerosis, calcification	Stenosis, regurgitation
Ventricle	Increased left ventricular wall tension	Left ventricular hypertrophy
	Prolonged myocardial contraction Prolonged early diastolic filling rate Decreased maximal cardiac output Right bundle branch block	Heart failure
	Ventricular premature complexes	Ventricular tachycardia, fibrillation
	Increased cardiovascular reserve	Heart failure

11:4 Common comorbidities

Older adults not only have coronary risk factors and coronary artery disease but are also at a higher risk of cerebrovascular disease, peripheral artery disease, congestive heart failure, chronic kidney disease, atrial fibrillation, chronic obstructive pulmonary disease, arthritis, dyslipidemia, hypertension and diabetes [5].

11:5 Special considerations in management [6–11]

1. Careful monitoring of treatment since drug toxicities are common.
2. Slow, careful titration of medications.
3. Avoid atenolol in adults over 60 years of age, unless they have coronary artery disease.
4. Avoid short-acting calcium channel blockers, e.g. Sublingual nifedipine
5. Compelling indications for hypertension treatment

Table 11:2 Compelling indications for antihypertensive medications in the elderly patient

Compelling Indication	Recommendation
Congestive Heart Failure	Use ACE Inhibitors or ARB's as first-line agents. B-blockers are also beneficial.
Myocardial Infarction	Beta-blockers and/or ACE inhibitors (or ARB's) should be considered as first-line agents. If an anti-anginal is necessary, the use of calcium channel blockers can be considered.
Nephropathy	Use ACE inhibitors (or ARB's) when the serum creatinine is greater than 1.5 mg/dL or the 24-hour urine protein is greater than 1 gram.
Gout	Avoid thiazide diuretics in patients with gout.
Hyperlipidemia	Use calcium channel blockers or ACE inhibitors
Erectile dysfunction	Use chlorthalidone with caution

11:6 Treatment cutoffs and targets

Table 11:3 Hypertension treatment indications and targets

Hypertension treatment in the elderly patient over 80 years		
	BP levels	
	SBP	**DBP**
Treatment indication	≥150 mmHg	≥ 90 mmHg
Treatment targets	<150 mmHg	< 90 mmHg
Hypertension treatment in the elderly patient 60-79 years		
	BP levels	
	SBP	**DBP**
Treatment indication	≥140 mmHg	≥ 90 mmHg
Treatment targets	<140 mmHg	< 90 mmHg
Hypertension treatment in the elderly patient over 60 years, with diabetes		
	BP levels	
	SBP	**DBP**
Treatment indication	≥135 mmHg	≥ 85 mmHg
Treatment targets	<135 mmHg	< 85 mmHg

11:7 Preventive therapy

Table 11:4 Indications for statins and anticoagulants in patients above 60 years of age

Treatment	Indications	Remarks
Statins	**1.** Patients with atherosclerotic CVD 2. Patients with LDL cholesterol above 190mg/dl 3. Diabetic patients between 40-75 years 4. Estimated 10 year risk of CVD of 7.5% or higher	No evidence of benefit for patients over 75 years
Anticoagulant therapy	**Primary prevention**: for 60-69 years olds: High CVD risk (DM, HTN, male sex, smokers, dyslipidemia), low risk for bleeding (GI ulcers, upper GI pain, other anticoagulant medications, uncontrolled hypertension) >70 year old: not recommended **Secondary prevention**: Patients with atrial fibrillation, left ventricular thrombus, cerebral embolism, venous embolism, pulmonary embolism)	Patients above 70 years and those without a bleeding risk with a firm indication can receive warfarin with low dose clopidogrel and aspirin

11:8 Treatment approach for heart failure in the elderly

DEFEAT [12]

Diagnosis: careful history taking can enable one to make a clinical diagnosis of heart failure in the elderly

Etiology: It is important to determine the underlying etiology of the heart failure

Fluid volume: by careful examination of the external jugular veins in all visits

Ejection Fraction: should be assessed to guide therapy

Therapy: When ejection fraction cannot be determined, all heart failure in the elderly should be managed as systolic heart failure with an ACE inhibitor, beta blocker and aldosterone antagonists for advanced heart failure. Diuretics should be used judiciously to achieve euvolemia.

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12 Cardiovascular Disease in Diabetes

12 Cardiovascular disease in diabetes

List of Abbreviations

ACEI	Angiotensin Enzyme Inhibitor
ACLS	Advaced Cardiac Life Support
ARB	Angiotensin Receptor Blocker
ARV	Anti-retroviral Drugs
CAD	Coronary Artery Diease
CVD	Cardiovascular Diseases
DBP	Diastolic Blood Pressure
SBP	Systolc Blood Pressure
T2DM	Type 2 Diabetes Mellitus

12:1 Introduction

CVD is a major complication of DM and causes premature death[1] . About 65% of people with DM die from heart disease and stroke [2]. Adults with DM are 2-4 times more likely to have heart disease or suffer a stroke than people without diabetes[2]. Hyperglycaemia in adults with diabetes increases the risk for Myocardial Infarction, Stroke, Angina, and Coronary Artery Disease. People with T2 DM have high rates of high BP, Dyslipidaemia, and Obesity, which contribute to high rates of CVD [3]. Smoking doubles the risk of CVD in people with diabetes[2]. HIV and ARV therapy increases the risk of Diabetes and CVD [4].

12:2 Mechanisms of Developing CVD in Diabetes Setting[5]

Diabetes affects the heart and the blood vessels. Effects on the arteries are due to atherosclerosis, while effects in the heart are due to ventricular hypertrophy. Left Ventricular Hypertrophy (LVH) is a characteristic of hypertensive and/or diabetic heart disease, which is an important cause of heart failure in Africa. Heart failure, a serious condition associated with repeated hospitalizations and high in-hospital mortality. LVH is associated with susceptibility to athero-thrombosis and therefore stroke or CAD, increased albuminuria and therefore Chronic Kidney Disease - a marker of microvascular disease and endothelial dysfunction.

The most common cardiovascular conditions found in DM patients are:

- Hypertension
- Heart Disease –LVH, Coronary Artery Disease, Heart Failure
- Stroke

The management of CVD in diabetes follows the principles outlined in the previous sections of this document with the exception of Hypertension.

12:3 Principles of management of hypertension in diabetes mellitus [6]

All persons whether hypertensive or not should be counselled on the following at each visit:

- Lifestyle modifications (physical exercise, diet and weightloss) and setting health goals
- Diet should be low in sodium, rich in vegetables and fruits, and use of low fat dairy products
- Alcohol consumption and tobacco cessation

Determine blood pressure in people with Type2 diabetes at every visit, using standard techniques (measure with a well calibrated BP machine and with the right-sized cuff with the patient seated after 5 minutes).

Table 12:1 Hypertension treatment target for various categories of diabetic patients [6]

Hypertension treatment in patients with diabetes mellitus		
	BP levels	
	SBP	**DBP**
Treatment indication	≥140 mmHg	≥ 90 mmHg
Treatment targets	<140 mmHg	< 85 mmHg
Hypertension treatment in diabetic patients with established cardiovascular disease		
	BP levels	
	SBP	**DBP**
Treatment indication	≥140 mmHg	≥ 90 mmHg
Treatment targets	<140 mmHg	< 90 mmHg
Hypertension treatment in patients with diabetes, and renal impairment (serum creatinine >133µmol/L), GFR <60 and microalbuminuria)		
	BP levels	
	SBP	**DBP**
Treatment indication	≥140 mmHg	≥ 90 mmHg
Treatment targets	<140 mmHg	< 90 mmHg

12:4 Pharmacologic Management of Hypertension

- Pharmacologic management should be considered in all patients with diabetes and a documented sustained Blood Pressure of above 140/90mmMg. Lifestyle modification should be developed in all patients with diabetes and/or hypertension.
- Individualize hypertensive therapy to achieve good control.
- Multiple agents are frequently required. Fixed dose combinations should be used when Blood pressures are stable and response to individual agents is known.
- Monitor serum creatinine and potassium once a year and more frequently if there is evidence of renal impairment.
- Note the potential problems with certain anti-hypertensives:
- Diuretics in large doses inhibit insulin release
- Betablockers may blunt or mask symptoms of hypoglycaemia and exacerbate peripheral vascular disease
- Dyslipidaemias may be worsened by betablockers and diuretics
- Impotence and postural hypotension may be precipitated or aggravated by alpha blockers and centrally acting agents (e.g.methyldopa).
- Angiotension converting enzyme (ACE) inhibitors may induce hyperkalaemia, renal failure, apersistent cough

Avoid sublingual antihypertensives and hydrallazine that have the potential to dramatically and catastrophically reduce blood pressure which can cause renal injury.

A Multifacted Management approach is essential to attainment of good outcomes in care for CVD in DM. This approach includes:

- Healthy behavior optimisation
- Blood glucose, pressure and lipid control
  Use of vascular protection mechanisms outlined

12:5 Multi-faceted Vascular Protection Checklist

Table 12:2 Vascular protection checklist

Acronym	Parameter	Remarks
A	HbA1C	Optimal glycemic control (usually ≤7%)
B	BP	Optimal blood pressure control (<140/90)
C	Cholesterol	LDL ≤2.0 mmol/L if decided to treat
D	Drugs to protect the heart (regardless of baseline BP or LDL	**A** – ACEi or ARB **S** – Statin **A** – ASA if indicated-evidence suggests no benefit to routine use in DM. Prescribe to those with established CVD or with additional CV risk factors (Age over 40, HTN, Dyslipidemia) [7,8]
E	Exercise / Eating healthily	Regular physical activity, achieve and maintain healthy body weight
S	Smoking cessation	

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13 Cardiovascular Diseases in People Living with HIV/AIDS (PLHIV)

13 Cardiovascular Disease in People Living with HIV/AIDS (PLHIV)

List of Abbreviations

ATV/r	Atazanavir/ritonavir
CKD	Chronic Kidney Disease
CrCl	Creatinine Clearance
CVD	Cardiovascular Disease
EFV	Efavirenz
HAART	Highly Active Retroviral Therapy
HBV	Hepatitis B vaccine
HIV	Human Immunodeficiency Virus
INSTI	Integrase Strand Transfer Inhibitor
LDL	Low Density Lipoproten
LPV/r	Lopinavir/Ritonavir
NNRTIs	Non-Nucleoside Reverse Transcriptase Inhibitor
NRTIs	Nucleoside Reverse Transcriptase Inhibitor
OD	Once Daily
PI	Protease Inhibitor
PI/r	Protease Inhibitor/Ritonavir

13:1 Introduction

Kenya currently has approximately 1.5 million people living with HIV infection; 1.4million adults and 98,200 children (1). . Kenya has approximately 1 million PLHIV on ART with annual new infections having reduced to approximately 77,000 from 98,000 in 2013 (2).

132:2 Epidemiology

Prevalence of CVD and its risk factors is higher among PLHIV (3). Gerald and colleagues found a prevalence of hypertension of 9.3% among all adult HIV positive patients in a study in Western Kenya5, while a systematic review and metanalysis put the prevalence at 14.5 and 10.5% in patients on HAART and HARRT-naïve patients respectively (4) .

13:3 Pathophysiology of CVD (hypertension, CKD, Heart failure) in HIV

Occurrence of CVD in HIV patients utilizes various pathogenetic mechanisms including persistent inflammation, immune activation, metabolic derangements from ART (e.g dyslipidemia from PIs), higher rates of behavioural risk factors including smoking and hypertension.

Pathogenesis and risk factors of CVD in PLHIV

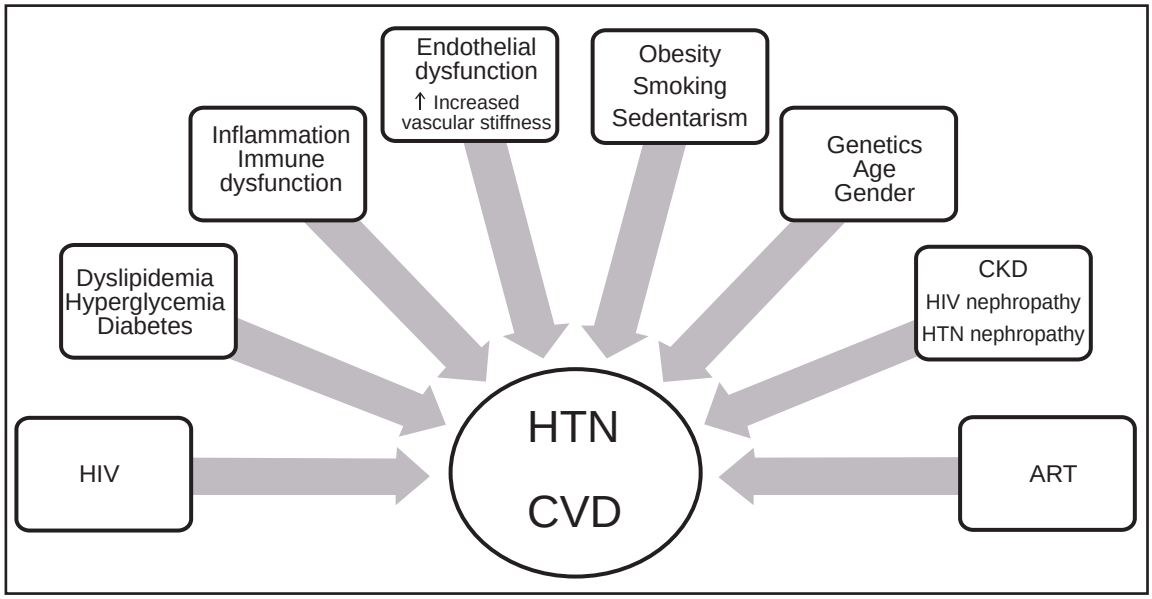

Figure13:1 Causes of CVD in HIV Infection. (Source: Costa AL, 2014)

13:4 Diagnosis and Mandatory investigations to be done to prevent CVD in HIV

Table 13:1 Additional investigations for PLHIV for CVD screening (in addition to routine HIV monitoring tests)

Test category	Recommended tests
HIV specific	Refer to National ART Guidelines for details
Others (HIV related)	• Creatinine (baseline then annually on TDF) • Plasma Lipid profile (for all at initial then annual) • HBsAg (Baseline) • Glucose (baseline then annually) • HCV (for those who inject drugs)
Dyslipidaemia	• Fasting lipid profile (Total cholesterol , LDL cholesterol , Triglycerides)
Hypertension	• Urinalysis (Urine dipstick for protein, blood and sugar) • Random blood sugar (finger prick) • Electrocardiogram (ECG)
Chronic Kidney disease	• Urinalysis • Creatinine

13:5 Treatment and prevention of CVD in HIV

1) Lifestyle modification

This is recommended for the initial step of prevention and management for cardiovascular diseases and should be integrated into routine HIV treatment and prevention 10

2) Screening, diagnosis and initial management of PLHIV

2.1. Dyslipidaemia

Table 13 :2 Dyslipidaemia screening, diagnosis and initial management of PLHIV .

Screening
• Fasting lipid profile should be evaluated at baseline for all PLHIV, then annually if baseline screening is normal
Diagnosis
• Dyslipidaemia is defined as high fasting total cholesterol (>5.2 mmol/L), LDL (>3.4 mmol/L) or triglycerides (>2.2 mmol/L)
Management
• Lifestyle modification for 3-6 months • If the patient is on an ARV known to cause or exacerbate dyslipidaemia (primarily LPV/r & EFV) thenconsider a single-drug substitution to a more lipid-friendly drug (such as from LPV/r or EFV to ATV/ror DTG) as the treatment of choice before adding a lipid-lowering drug. -Rule out treatment failure beforemaking single-drug substitutions (Figure 6.2 in Section 6) • If does not meet treatment target with lifestyle modifications then add drugs:Atorvastatin: starting dose of 10 mg OD (maximum dose 20 mg if patient is on a PI/r; maximum dose 80 mg once daily if not on a PI/r) -Allow at least 3 months before repeating fasting lipids and titrating dose • Once targets achieved can monitor lipids every 6-12 months

2.2 Chronic Kidney disease

Table13:3 Chronic kidney disease screening, diagnosis and management among the PLHIV

Screening
• Urinalysis (for protein) and serum creatinine should be evaluated at baseline for all PLHIV
Diagnosis
• Impaired renal function is defined as creatinine clearance < 90 ml/min, or dipstick proteinuria ≥ 1 • Abnormal results should be repeated to confirm diagnosis
Management
• Management depends on the cause of the renal impairment; additional investigations and/or specialist consultation may be required • Treat dehydration promptly and aggressively • If on TDF-containing regimen, substitute with another ARV if CrCl<50 ml/min (see Section 6.4 ART Guidelines 2016), with the exception of patients with HBV/HIV co-infection (see Table 9.3 ART Guidelines 2016) • Avoid nephrotoxic drugs e.g. aminoglycosides, non-steroidal anti-inflammatory drugs (NSAIDS) • Evaluate for and treat hypertension • All NRTIs except ABC require dose adjustments for renal impairment, depending on the severity (see Table 6.5 in Section 6 ART Guidelines 2016 for specific dose adjustments). NNRTIs, PIs, and INSTIs do not require dose adjustments for impaired renal function

2.3. Hypertension

Screening, diagnosis and initial management of hypertension in PLHIV is similar to that in the general population, except for a few considerations as outlined in the table below:

Table 13:4 Important considerations when treating hypertension in PLHIV

Precaution	Action
Metabolism of CCBs is induced by EFV or NVP, blunting antihypertensive effect.	Higher starting doses may be required. Monitor closely for response.
PIs (such as LPV/r or ATV/r) inhibit metabolism of CCBs.	Monitor closely for excessive reduction in BP and reduce the dose of CCB accordingly
ACEI and Thiazide diuretics do not have significant interactions with ARVs.	N/A
Patients on PIs (e.g. LPV/r or ATV/r) may experience oedema, dizziness, fatigue and orthostatic hypotension within the first week of initiation of CCB therapy[11]	Counsel patient accordingly

13:6 ARV commonly used in Kenya

For ARV drugs commonly used in Kenya, please refer to ARV guidelines Kenya 2016

13:7 Potential drug-drug interaction

Table 13:5 Potential drug interactions – antihypertensive and ARV drugs

Antihypertensive class	Potential interactions with ARV drugs	Corrective measures/ Precautions
A, Angiotensin-converting enzyme inhibitors (ACEIs): E.g. **- Enalapril**, **Lisinopri**l, Perindopril, and Ramipril	None with NRTIs, NNRTIs and PIs	None
A, Angiotensin II receptor blockers (ARBs): E.g. **Losartan**, **Telmisartan**, and Candesartan	Telmisartan, Candesartan: None with PIs, NNRTIs, and NRTIs **Losartan**: Potential interactions with all PIs, NNRTIs and Zidovudine. None with other NRTIs	Net effect of Losartan interactions: difficult to predict.
B, Beta blockers: E.g. **Atenolol**, Carvedilol and **Propranolol**	**Atenolol**: Potential interactions with Atazanavir, Lopinavir, and Ritonavir. None with Saquinavir.	Potential increase in B-blocker effect. Careful dose† adjustment and ECG where indicated.
C, Calcium channel blockers (CCBs): E.g. **Nifedipine**, **Amlodipine** and **Felodipine**	Potential interaction with all NNRTIs. • Metabolism of CCBs is induced by EFV or NVP, blunting antihypertensive effect. None with NRTIs	***Higher starting doses may be required. Monitor closely for response and a***djust dose according to BP readings.
	Potential interaction with all PIs • PIs (e.g. LPV/r or ATV/r) inhibit metabolism of CCBs.	***Monitor closely for excessive reduction in BP and reduce the dose of CCB accordingly***.
D, Diuretics: E.g. Bendroflumethiazide; **HCTZ** , **Indapamide. Furosemide** and **Spironolactone**	No significant interactions with PIs, NNRTIs, NRTIs Indapamide: Potential interactions with Lamivudine, and Tenofovir. No interactions with other NRTIs, PIs and NRTIs.	No effect on all the listed diuretics except Indapamide (levels may fluctuate). Adjust dose if necessary

Dose†: for adjustments refer to the hypertension treatment protocol.
Listed drugs in bold: commonly used in Kenya/HHA project sites.
Similar precautions are indicated in patients using post-exposure prophylaxis (PEP) and pre-exposure prophylaxis (PrEP)

Patients with HTN and TB and/or Diabetes should be referred to a facility with capacity to monitor and manage potential drug interactions.

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14 Cardiovascular Diseases in Chronic Kidney Disease

14 Cardiovascular Disease in Chronic Kidney Disease

List of Abbreviations

ACEI	Angiotensin Enzyme Inhibitor
ARB	Angiotensin Receptor Blocker
CAD	Coronary Artery Diease
CVD	Cardiovascular Diseases
BP	Blood Pressure
DBP	Diastolic Blood Pressure
SBP	Systolic Blood Pressure
YLD	Years Lost due to Disabilty

14:1 Introduction

When compared with the general population, patients with CKD are at a higher risk of developing cardiovascular disease and its complications, with the commonest conditions being ischemic heart disease, congestive heart failure, arrhythmias (commonly atrial fibrillation) and peripheral vascular disease (1). Several measures directed at preventing the progression of CKD can have impact on CVD prevention as well.

14:2 Economic and public health burden

CKD is an important contributor to both morbidity and mortality in Kenya, with an estimated annual mortality rate of 3.5 per 100,000 people, healthy years lost due to disability (YLD) 163.5 per 100,000 people while the years of life lost due to premature mortality from the condition is 133.5 per 100,000 population according to the Global Burden of Disease, 2013 (2). Cardiovascular disease has been found to complicate approximately 10% of stable CKD patients (3) and contribute to at least 50% of deaths in patients with end-stage renal disease (4). CKD has a major economic impact on individuals, their families and health care systems, especially the advanced stages, and cardiovascular disease compounds this effect, especially because CVD is the leading cause of hospitalization in CKD patients (5).

14:3 Pathophysiology

Traditional risk factors for CVD such as increasing age, dyslipidemia, hypertension, diabetes, smoking and obesity are risk factors for CKD as well, hence they are common in CKD patients. In addition, other risk factors are common in CKD patients as compared to the general population and include albuminuria, anaemia, hyperparathyroidism, metabolic bone disease, malnutrition, hyperhomocystinemia, inflammation, endothelial dysfunction and oxidative stress (1). The relationship between these two set of factors (traditional vs non-traditional CVD risk factors) are illustrated in the figure below (figure 1)

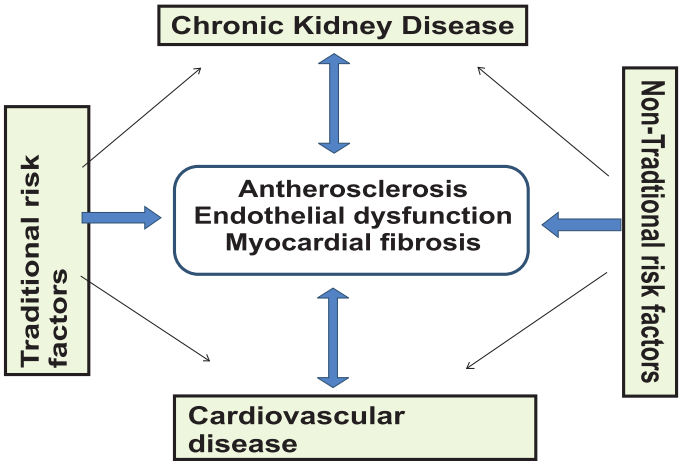

Figure 14.1: Interaction of CVD risk factors.
(Source: Heart Asia. 2016; 8(2): 56–61).

14:4 Important considerations in management

1. **Diagnosis** requires high index of suspicion since the classic triad of ischæmic symptoms, elevated cardiac biomarkers and ECG changes may be absent in CKD patients, data on diagnostic modalities is sparse since mostly CKD patients are excluded from trials and diagnostic tests have low negative predictive value die to the relatively high prevalence of CVD in CKD patients (6)

2. **Management of specific conditions (7, 8)**

CVD condition	Management	Caution
Coronary artery disease and myocardial infarction (heart attack)	Aspirin, clopidogrel, ARBs, ACEIs, reperfusion therapy, statins	CKD patients are at a higher risk of bleeding on antiplatelet therapy
Congestive heart failure	Dietary salt restriction, ACEIs, ARBs, carvediolol and bisoprolol,though limited evidence	Use aldosterone antagonists with caution, specialist supervision for GFR below 30ml/min/1.73m^2
Stroke	Low dose antiplatelet therapy for both primary and secondary prevention, statins, intravenous thrombolysis	Avoid intravenous tissue plasminogen activator for patients undergoing dialysis (due to use of heparin in these patients)
Atrial fibrillation	Warfarin for those at high risk for stroke (those with CHF, previous stroke, LVH, hypertension)	Risk stratification excluded ESRD patients, hence best to avoid warfarin in these patients
Peripheral artery disease	Smoking cessation, aspirin, revascularization or amputation	CKD patients are at a higher risk of bleeding on antiplatelet therapy
Sudden cardiac death		Avoid digoxin, benefit of slow dialysis, low potassium dialysate, beta blockers (In dilated cardiomyopathy) not supported by evidence.
Hypertension	ACEI, ARB as a compelling indication, targets BP below 130/80mmHG	A limited rise in creatinine of up to 35% on ACEIs and ARBs should not preclude therapy unless hyperkalemia debelops

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15 Cardiovascular Diseases in Pregnancy

15 Cardiovascular Disease in Pregnancy

List of Abbreviations

ACEI	Angiotensin Enzyme Inhibitor
ACLS	Advaced Cardiac Life Support
ACS	Acute Coronary Syndrome
ARB	Angiotensin Receptor Blocker
BP	Blood Pressure
CAD	Coronary Artery Disease
CT	Computer Tomography
CVD	Cardiovascular Diseases
CXR	Chest Xray
DVT	Deep Venous Thrombosis
ECG	Electrocardiogram
FBG	Fasting Blood Glucose
GERD	Gastroesophageal Reflus Disease
HBA1c	Glycated Hemoglobin A1
HIV	Human Immunodeficiency Virus
HTN	Hypertension
IHD	Ischemic Heart Disease
LDL	Low Density lipoprotein
LV	Left Ventricle
NSAIDS	Non-steroidal Anti-inflammatory Drugs
OGTT	Oral Glucose Tolerance Test
RBS	Random Blood Sugar
RV	Right Ventricle
TBC	Total Blood Count
UA	Unstable Agina
V/Q	Ventilation-perfusion Ratio
VF	Ventricular Failure
VT	Ventricular Tachycardia

15:1 Introduction

Presently, 1-4% of pregnancies are complicated by cardiovascular disease. This is associated with significant morbidity and mortality, both for the mother and the fetus.

Hemodynamic and Metabolic Changes in Pregnancy

During pregnancy, the maternal circulation undergoes physiological changes to meet the increased metabolic demands of the mother and fetus. They include increases in blood volume and cardiac output (CO), and reductions in systemic vascular resistance and blood pressure(BP). Plasma volume reaches a maximum of 40% above baseline at 24 weeks gestation. A 30–50% increase in CO occurs in normal pregnancy. CO increases by 15% in early labour, by 25% during 1st stage , and by 50% during 2nd stage. It reaches an increase of 80% early post-partum due to autotransfusion associated with contraction and involution of the uterus, and resorption of oedema in the lower limbs. This increased stress on the heart can cause significant deterioration of heart function where there is already disease, or cause symptoms in previously undiagnosed CVD. Other factors that cause hemodynamic changes are uterine contractions, positioning (left lateral vs. supine), pain, anxiety, exertion, bleeding, anaesthesia and infection.

Blood pressure (BP) typically falls early in gestation and diastolic BP (DBP) is usually 10 mmHg below baseline in the second trimester. This decrease in BP is caused by active vasodilatation.
In the third trimester, the DBP gradually increases and may normalize to non-pregnant values by term. In view of this, serialized blood pressure measurements provide the best opportunity to pick up a rising trend in BP, even when not overtly elevated above the traditional 140/90mmHg.
Pregnancy causes a hyper-coagulable state, resulting in an increased risk of thrombo-embolic events. There is an increase in the concentration of coagulation factors such as fibrinogen, and platelet adhesiveness, as well as a reduction in fibrinolysis. In addition, the enlarging uterus causes obstruction to venous return, resulting in stasis and a further rise in risk of thrombo-embolism.

Maternal glucose homeostasis may change and cholesterol levels increase in adaptation to fetal–maternal needs. Increased levels of anti-insulin hormones peak at 24-28weeeks gestation. This may result in maternal hyperglycemia that results in poor fetal and maternal outcomes.

Cardiovascular diseases in pregnancy include:

1. Hypertensive disorders of pregnancy
2. Cardiac disease in pregnancy
3. Venous Thromboembolism

15:2 General Reproductive Health Considerations in Cardiovascular Disease

Preconception Care:

Girls with congenital heart disease should be referred to a facility where they can access both cardiac and obstetric care, once in puberty (Age 12-15). Newly diagnosed older women should also be referred for the same. Counseling offered should include information on increased cardiovascular risk in pregnancy, contraceptive options, risk to the fetus especially with congenital lesions and increased need for surveillance in pregnancy.

Antepartum:

Because of the diversity in cardiovascular disease, often with differing implications, it is important that a risk assessment of any woman with a heart murmur or a history of any cardiac defect or vascular disease be carried out early in pregnancy. They should be reviewed jointly by a cardiologist and an obstetrician. Women at low risk can be identified and returned to routine care. Women at significant risk of adverse events during pregnancy should be seen regularly in the antenatal clinic, whenever possible by the same obstetrician. Cardiovascular assessment should be carried out at every antenatal clinic visit. This should include:

- Blood pressure measurement
- Measurement of pulse rate and rhythm is also mandatory as it may be the first sign of volume overload
- Auscultation to assess any change in murmur or any lung changes associated with pulmonary oedema
- Women with cyanotic heart disease should have their oxygen saturations checked periodically (each trimester or more often if there are any clinical signs of deterioration).
- All women with congenital heart disease should be offered a fetal echocardiogram during the second trimester to be carried out by fetal cardiologist (as distinct from the standard four-chamber view offered to all women as part of routine antenatal screening
- A multidisciplinary meeting (obstetrics, cardiology, neonatology, anaesthesia) should take place at 32–34 weeks of gestation to establish a plan of management for delivery

Intrapartum:

The general principle of intrapartum management is to minimise cardiovascular stress. This is achieved by providing adequate analgesia/anesthesia and shortening 2nd stage of labour by performing an assisted vaginal delivery. Caesarean section is done for obstetric indications. High risk patients should deliver in level 6 facilities which have high-dependency and intensive care units, suitable for the care of pregnant women with significant heart disease. For more details, lease refer to session 3 (page 126) of complications/conditions during ante-natal period in the National Guidelines on Quality Obstretics and Perinatal Care.

Postpartum:

For patients with cardiac lesions the standard bolus IM dose of 10IU units of oxytocin for active management of 3rd stage of labour SHOULD NOT be given. Instead uterine massage, and a low dose infusion with limited fluids should be given. The postpartum period is associated with a high level of complications due to the increased cardiac output. These patients should therefore remain in hospital for at least 10 days post partum for monitoring.

15:3 Hypertensive disorders in pregnancy

Hypertension complicates 5 to 7% of all pregnancies. It is a major cause of maternal, fetal, and neonatal morbidity and mortality in developing and in developed countries. This is due to severe complications such as abruptio placentae, cerebrovascular accidents, organ failure, and disseminated intravascular coagulation. Hypertensive disorders are classified as follows:

- Preeclampsia: Hypertension with onset after 20weeeks gestation with proteinuria or end-organ damage.
- Eclampsia: Pre-eclampsia with generalized tonic-clonic convulsions
- Chronic hypertension: Hypertension that ante-dates pregnancy
- Preeclampsia superimposed on chronic hypertension.
- Gestational hypertension: Hypertension in pregnancy with no proteinuria

1.1 Pre eclampsia

Preeclampsia is a disorder of widespread vascular endothelial malfunction and vasospasm that occurs after 20 weeks' gestation, and can present as late as 4-6 weeks postpartum. It results in significant maternal and fetal morbidity and is a marker for future cardiac and metabolic disease.
Aetiology
The aetiological factors are poorly understood. The risk factors are listed in the table below:

Table 15:1 Risk factors for pre-eclampsia

Maternal specific	Pregnancy related
Extremes of age **<18 yrs >40yrs**	Pre-eclampsia in previous pregnancy
Black Race	Multiple gestation
Family History of pre-eclampsia	Hydatidiform mole
Change of male partner	Chromosomal abnormalities
Primiparity	In vitro fertilization
Pregnancy interval > 10 years	
Obesity	
Comorbidities: Chronic hypertension, renal disease, diabetes, autoimmune disease (APS, SLE)	

Diagnostic criteria

To make a diagnosis of Pre-eclampsia, the following criteria is used.

Table 15:2 Diagnostic criteria for Pre-eclampsia

Blood pressure	• Greater than or equal to 140 mm Hg systolic or greater than or equal to 90mm Hg diastolic on two occasions at least 4 hours apart after 20weeks of gestation in a woman with a previously normal blood pressure • Greater than or equal to 160 mm Hg systolic or greater than or equal to 110 mm Hg diastolic, hypertension can be confirmed with a short interval (minutes) to facilitate timely antihypertensive therapy
and	
Proteinuria	• Greater or equal to 300 mg per 24 hour urine collection (or this amount extrapolated from a times collection) or • Protein/creatinine ratio greater than or equal to 0.3* • Dipstick reading of 1+ (used only if other quantitative methods not available)
Or in the absence of proteinuria, new-onset hypertension with the new onset of any of the following	
Thrombocytopenia	• Platelet count less than 100,000/microliter
Renal insufficiency	• Serum creatanine concentrations greater than 1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease
Impaired liver function	• Elevated blood concentration of liver transaminases to twice normal concentration
Pulmonary edema	
Cerebral or visual symptoms	

Each measure as mg/dL

Classification of Pre-eclampsia

Pre-eclampsia is further classified as having severe features or without severe features. Features of severity are listed in the table below:

Table 15:3 Features of Severity in pre-eclampsia

Severe Features of Preeclampsia (Any other findings)
Systolic blood pressure of 160 mm Hg or higher, or diastolic blood pressure of 110 mm Hg or higher on two occasions at least 4 hours apart while the patient is on bed rest (unless antihypertensive therapy in initiated before this time)
• Thrombocytopenia (platelet count less than 100,000/microliter)
• Impaired live function as indicated by the abnormally elevated blood concentrations of liver enzymes (to twice normal concentration), severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by alternative diagnoses or both
• Progressive renal insufficiency (serum creatinine concentration greater than 1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease)
• Pulmonary edema
• New-onset cerebral on visual disturbances

1:2 HELLP Syndrome

This is an acronym for the clinical picture found in some patients who have pre-eclampsia that includes Hemolysis, Elevated liver enzymes and Low Platelets. A patient with this syndrome is classified as having pre-eclampsia with severe features. It usually will progress rapidly to cause both maternal and fetal deterioration and is a marker for immediate delivery.

Prevention and screening

- All women should have BP measured at each clinic visit
- Weight should be measured at each clinic visit. An increase of >1kg in a week should be flagged and the patient closely monitored for any BP changes
- Sudden onset lower limb and facial oedema should be noted and patient closely monitored for BP changes
- Women who are high risk should take Aspirin 75mg daily from 13 weeks gestation to time of delivery
- In populations where dietary calcium is low, Calcium supplementation has been shown to reduce occurrence of pre-eclampsia
- Bed rest and use of Vitamin C, E and fish oil has not been shown to be beneficial

Clinical Presentation

Pre-eclampsia may be asymptomatic. Many cases are detected through routine screening by checking BP. Presenting symptoms and signs include:

Table 15:4 Symptoms and signs of Pre-eclampsia

System	Symptoms (Presentation)	Signs (on examination)
General	Lower limb or facial swelling	Oedema, sudden weight gain
Cardiovascular	Often asymptomatic	BP >140/90mmHg
Central Nervous System	Drowsiness, headache, convulsions, visual disturbances	Altered consciousness, hyper reflexia, seizures, focal neurological deficit, visual stocomata
Pulmonary	Difficulty breathing, Frothy cough	Basal crepitations on auscultation
Hepatic	Epigastric pain	Right upper quadrant tenderness
Renal	Reduced urine output	Oliguria
Fetal	Reduced/absent fetal movement	Non reassuring fetal status, fetal growth restriction, fetal demise
Hematological	Dark coloured urine, easy bruising	Hemoglobinuria, Ecchymoses

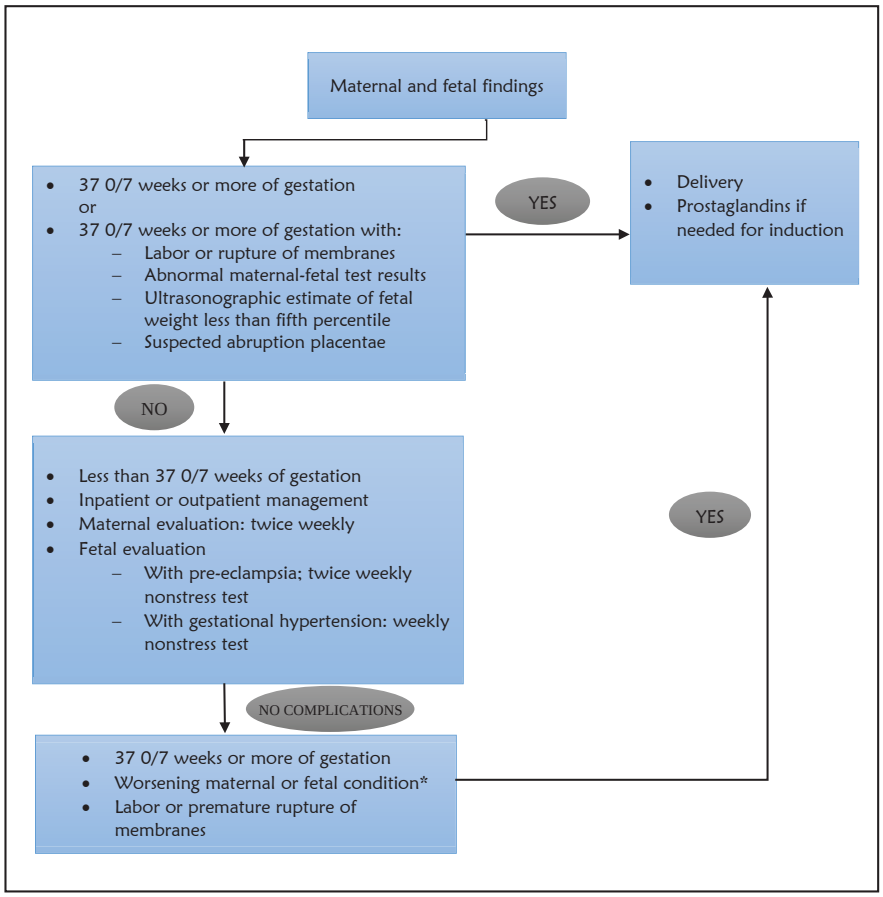
Investigations:

1. **Laboratory Studies :** Urinalysis or 24 hour urine, Hemoglobin concentration, platelets, coagulation profile, Serum creatinine, AST, ALT
2. **Imaging:** Obstetric ultrasound- assess fetal viability, Gestational age, amniotic fluid index, Resisitive index of the umbilical artery and middle cerebral artery and Bio-physical profile

5:4 Management

1.1a Pre-eclampsia WITHOUT severe features

The figures below outlines the management of pre-eclampsia in women with and without severe features of the disease.



*BP Targets: Systolic 140-160 mmHg, Diastolic 90-100 mHg

Figure 15.1: Management of pre-eclampsia without severe features or gestational hypertension

1.1b Pre-eclampsia WITH severe features

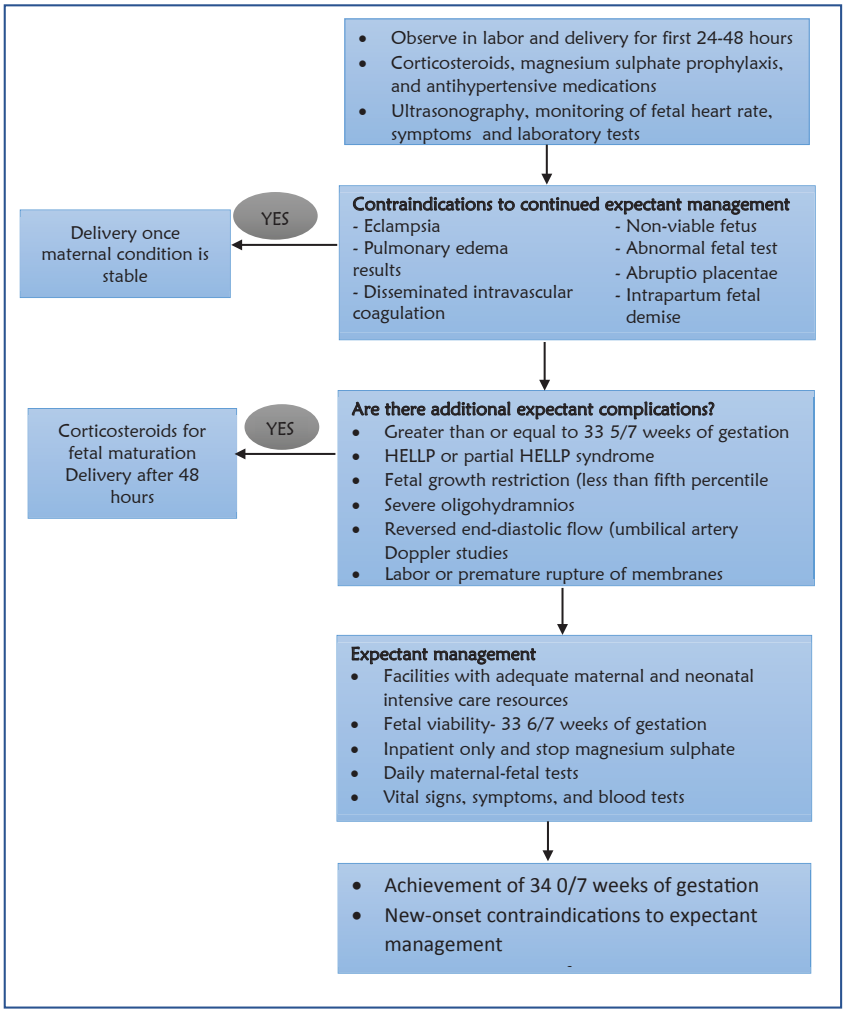


Fig 15.2: Management of pre-eclampsia with severe features and HELLP Syndrome

1.2 Eclampsia

This is the presence of new onset generalized tonic-clonic convulsions in a woman with pre-eclampsia. It is due to cerebral oedema and hypoxia and may occur ante partum or post partum. It is an indication for immediate delivery, after stabilization of the mother.

Differential diagnosis of Eclampsia

Eclampsia must be differentiated from other conditions that may be associated with convulsions and coma, e.g. epilepsy, cerebral malaria, meningitis, head injury, cerebrovascular accident, intoxication (alcohol, drugs, and poisons), drug withdrawal, metabolic disorders, water intoxication, encephalitis, hypertensive encephalopathy, hysteria.

Management of Eclampsiai.

Anti-convulsants

a. Magnesium Sulphate (MgSO4)

MgSO4 is given in eclampsia and pre-eclampsia with severe features to prevent convulsions. It works by antagonizing calcium channels of smooth muscle. Various regimens can be used safely:

Table 15:5 Magnesium Sulphate Regimens

Intravenous (Zuspan or Sibai)	Intramuscular (Pritchard)
✓ Loading dose of 4 g should be given intravenously over 5 minutes Followed by: ✓ Maintenance dose (infusion) of 1-2 g/hour ✓ Give until 24 hours after the last convulsion or delivery, whichever comes 1st	✓ Intramuscular Magnesium sulphate 20 % solution, 4 g by deep intramuscular injection over a period of 5 minutes, Followed by : ✓ Two deep intramuscular injections of 5 g magnesium sulphate 50 % solution into each buttock (Total dose of 10 g) ✓ Maintenance dose is 5 g magnesium sulphate 50 %, given by deep intramuscular injection, every 4 hours. ✓ Alternate the buttocks in which the injection is administered ✓ Give until 24 hours after the last convulsion or delivery, whichever comes 1st

MgSo4 Toxicity:

Every patient should be assessed before repeat dosing. The drug should be withheld if the following signs of MgSO4 toxicity are present:

- Respiratory rate falls below 16 per minute
- Patellar reflexes are absent
- Urinary output falls below 30ml per hour over the preceding 4 hours

b. Phenytoin:

Phenytoin is an effective anticonvulsant. It stabilizes neuronal activity by decreasing the ion flux across depolarizing membranes. It has no known neonatal adverse effects in the short term.

Dosage:

10 mg/kg loading dose infused IV no faster than 50 mg/min, followed by maintenance dose started 2 hrs later at 5 mg/kg .

c. Diazepam:

- In the absence of MgSO4, diazepam is used following the regime below : Intravenous administration:
- Loading dose Diazepam 20mg IV slowly over 2 minutes If convulsions recur, repeat loading dose.
- Maintenance dose Diazepam 40mg in 500ml IV fluids (normal saline or Ringer's Lactate) titrated to keep the woman sedated but can be aroused.
- Maternal respiratory depression may occur when dose exceeds 30mgs in 1 hour. Assist ventilation (mask and bag, anaesthesia apparatus, intubation), if necessary.
- Do not give more than 100mg in 24 hours.

Rectal Administration: Give Diazepam rectally when IV access is not possible. The loading dose is 20mg in 10ml syringe or a catheter may be used.
If convulsions are not controlled within 10 minutes administer an additional 10mg per hour

1.3 Chronic Hypertension

This is defined as hypertension that pre-dates the pregnancy or that is diagnosed before 20 weeks gestation.

Effects of chronic hypertension on pregnancy

- Premature birth (two thirds); worse with preeclampsia
- Intrauterine growth retardation (one third)
- Fetal demise: 2-4 times compared to the general population
- Without preeclampsia (5 per 1,000)
- With preeclampsia (28 per 1,000)
- Placental abruption
- Caesarian section
- Incidence of these events depend on severity and duration of hypertension and associated target organ damage

Effects of pregnancy on chronic hypertension

- Increase in blood volume and decrease in oncotic pressure-may lead to heart failure
- Physiologic decrease in blood pressure; from 12 wks, peaks at 16-18wks: masks detection of chronic hypertension
- Progression to preeclampsia and eclampsia
- Peripartum cardiomyopathy
- Renal failure; especially if baseline creatinine >124 mmol/L

Treatment of Hypertension in Pregnancy

ii. Anti-Hypertensives

The tables below indicate the medications used for management of hypertension in pregnancy.

Table 15:6 Oral anti-hypertensives in pregnancy aQW3

Drug	Dosage	Comments
Labetalol	200-2,400 mg/d orally in two to three divided doses	Well tolerated Potential brochoconstrictive effects Avoid in patients with asthma and congestive heart failure
Nifedipine	30-120 mg/d orally of a slow-release preparation	Do not use sublingual form
Methyldopa	0.5-3 g/d orally in two to three divided doses	Childhood safety data up to 7 years of age May not be as effective in control of severe hypertension
Thiazide diuretics	Depends on agent	Second-line agent
Angiotensin-converting enzyme inhibitors/ angiotensin receptor blockers		Associated with fetal anomalies Contraindicated in pregnancy and pre-conception period

Table 15:7 Anti-hypertensives for urgent BP control

Drug	Dosage	Comments
Labetalol	10-20 mg IV then 20-80 mg every 20-30 min to a maximum dose of 300 mg or constant infusion 1-2 mg/min IV	Considered a first time-line agent Tachycardia is less common and fewer adverse effects Contraindicated in patients with asthma, heart disease or congestive heart failure
Hydralazine	5 mg IV or Im than 5-10 mg IV every 20- 40 min or Constant infusion 0.5-10mg/h	Higher or frequent dosage associated with maternal hypertension, headaches and fetal distress-may be more common than other agents
Methyldopa	0.5-3 g/d orally in two to three divided doses	Childhood safety data up to 7 years of age May not be as effective in control of severe hypertension
Nifedipine	10-20 mg orally repeat in 30 minutes if needed; than 10-20 mg every 2-6 hours	May observe reflex tachycardia and headaches

2. Cardiac Diseases in Pregnancy

a) Peri-partum cardiomyopathy and Heart Failure in Pregnancy

PPCM is an idiopathic cardiomyopathy, where patients present with symptoms of heart failure due to left ventricular dysfunction. It is a diagnosis of exclusion when no other cause of heart failure is found. The LV may or may not be dilated, but the EF is nearly always reduced below 45%. It usually occurs late in pregnancy or in the peuperium. The cause of PPCM is unknown. Associated factors include multiparity and multiple childbirths, family history, ethnicity, smoking, diabetes, hypertension, pre-eclampsia, malnutrition, advanced age of mothers or teenage pregnancy, and prolonged use of β-agonists It usually presents in late pregnancy or early in the pueperium, but it can occur up to 6 months after delivery.

Presentation

- Pregnant or in peuperium
- Shortness of breath especially on lying flat or at night
- Nocturnal cough
- Lower limb and/or facial oedema

Diagnosis

All such women should have an electrocardiogram and an echocardiogram. A chest X-ray should be done that may show cardiomegaly, as well as help exclude other chest pathology.

Clinical Evaluation of patient with heart failure

Many pregnant women will experience deterioration of as pregnancy progresses, and they should be warned about this. They may need to take more rest than usual during pregnancy, although it is also important for them to maintain their fitness as much as possible.

A rising pulse rate can be one of the first signs of cardiac decompensation. The pulse rate is best measured using a stethoscope and auscultating the heart, because when the pulse becomes fast, irregular or faint, the radial pulse is often difficult to detect accurately.

The woman's blood pressure should be checked carefully using a manual sphygmomanometer.

The heart sounds should be auscultated carefully at each visit in a standard place, commonly the left sternal edge, to check if there has been any substantial change from the previous visit. Heart murmurs are graded from one (extremely soft) to six (the loudest one has ever heard). It is usual for a murmur to increase by one grade as pregnancy progresses because of the increase in cardiac output. A sudden increase in the loudness of a heart murmur can suggest the development of vegetations from endocarditis. The appearance of a new murmur isnearly always significant.

Management of PostPartum Cardiomyopathy(PPCM) Complications

i. Acute heart failure

Managing acute heart failure in pregnancy is similar to that applied to acute HF arising from any other cause

- Oxygen - in order to achieve an arterial oxygen saturation of ≥95 %, using, where necessary, noninvasive ventilation with a positive end-expiratory pressure (PEEP) of 5-7.5 cm H2O.
- Intravenous diuretics, when there is congestion and volume overload with an initial bolus of furosemide 20-40 mg i.v., is recommended.
- Intravenous nitrate is recommended (e.g. nitroglycerine starting at 10-20 µg/min up to 200 µg/min) in patients with a systolic blood pressure > 110 mmHg and may be used with caution in patients with SBP between 90-110 mmHg.
- Inotropic agents should be considered in patients with a low output state, indicated by signs of hypo-perfusion

ii. chronic heart failure

After delivery, heart failure should be treated in accordance with the current guidelines
During pregnancy the following restrictions to these guidelines apply:

- Anticoagulation is recommended in patients with intra-cardiac thrombus detected by imaging or with evidence of systemic embolism
- Therapeutic anticoagulation with LMWH or vitamin K antagonists according to stage of pregnancy is recommended for patients with atrial fibrillation.
- ACE-inhibitors and Angiotensin-II receptor blocker (ARBs) are Contraindicated because of serious renal and other foetal toxicity (I-C).
- Beta-blockers Not shown to have teratogenic effects. Beta-1 selective drugs preferred because beta-2 receptor blockade can have an anti-tocolytic action.
- Furosemide and hydrochlorothiazide- caution is adviced on loop diuretics use in chronic HF due to Ototoxicity

Breast feeding

Due to high metabolic demands of lactation and breastfeeding, preventing lactation may be considered in PPCM.
Several ACE-inhibitors (captopril, enalapril and quinapril) have been adequately tested inbreastfeeding women.

Timing and mode of delivery

A team comprising a cardiologist, obstetrician, anaesthesiologist, neonatologist and intensive care physician should discuss the planned mode and conduct of delivery in each case. The primary consideration should be maternal cardiovascular benefit.
In general, spontaneous vaginal birth is preferable in women whose cardiac condition is well controlled, with an apparently healthy fetus. Planned Caesarean section is preferred for women who are critically ill and in need of inotropic therapy or mechanical support.

b) Ischaemic Heart Disease

Pregnancy by itself increases the risk of myocardial infarction 3-4 times, when compared to the incident rate in non pregnant women. This risk is further increased in women above 40 years of age. Up to 1/13 of pregnant women with a myocardial infarction will die.
Risk factors include chronic hypertension, pre-eclampsia, diabetes, smoking, obesity, hyperlipidaemia and a positive family history. While these factors are increasingly prevalent in the Kenyan population, most women will be asymptomatic prior to pregnancy. Hence a high index of suspicion is needed in making an accurate diagnosis.

Presentation
Chest pain characterized as a crushing pain radiating to the left arm. In pregnancy this pain may also be experienced as epigastric pain.

Diagnosis
All women with chest pain in pregnancy must have an electrocardiogram done, and interpreted by a cardiologist with the requisite skill. ST elevation or depression as well as T wave changes may be present. Serum cardiac troponin levels should be measured.

Management

Acute:

- 100% Oxygen by mask
- Analgesia: Opioids preferred e.g. Morphine
- Immediate referral to a Level 6 facility for coronary angiography and possible percutaneous coronary intervention (PCI).
- Thrombolytic therapy may cause sub-placental hemorrhage, therefore should only be used as a life saving measure when PCI is unavailable

Medical therapy: ACE inhibitors and angiotensin receptor blockers (ARBs), are contraindicated during pregnancy . β-Blockers and low dose acetylsalicylic acid are considered to be safe
Other management as in non-pregnant state: Lifestyle modification (stop alcohol, smoking, weight control) BP control and glucose control

c) Arrythmias

It has been observed that premature extra beats and sustained tachyarrhythmias increase in frequency or may be evident for the first time during pregnancy. Most palpitations are not of major concern, however new onset of VT should be for evidence of an underlying structural cause.
The use of anti-arrythmic drugs should be done with caution due to the risk of feto-toxicity, more so in the 1st trimester, where the risk of teratogenicity is highest.

i. Arrhythmias associated with structural and congenital heart disease

Supraventricular and ventricular arrhythmias that require treatment have been observed in up to 15% of patients with congenital heart diseases during pregnancy. Episodes of sustained tachycardia are poorly tolerated, and can have adverse fetal effects due to hypo-perfusion in structural heart disease.

Management

- Sinus rhythm should be restored by direct current conversion.
- Digoxin can be used to control ventricular rate, however it has no prophylactic antiarrhythmic effect.
- β-Blocking agents, class I antiarrhythmic drugs, and sotalol should be used with caution if the LV or RV function is impaired.
- Amiodarone is reserved for use in exceptional cases when other therapy has failed. The lowest effective dose should be used.

ii. Supraventricular tachycardias

Atrioventricular nodal re-entry tachycardia and atrioventricular re-entry tachycardia
These re-entry tachycardias involving an accessory pathway and paroxysmal SVT can be terminated by vagal manoeuvres. If that is unsuccessful IV adenosine is the first drug of choice IV. metoprolol is recommended if adenosine fails to terminate a tachycardia. Prophylactic antiarrhythmic drug therapy are used when symptoms are intolerable, or if the tachycardia causes haemodynamic compromise. Then digoxin or a selective b-blocking agent (metoprolol) are the first-line agents, followed by sotalol, flecainide, or propafenone. AV nodal blocking agents should not be used in patients with manifest pre-excitation on resting ECG. Catheter ablation should be considered only in special cases if necessary during pregnancy.

iii. Atrial flutter and atrial fibrillation

Atrial flutter and AF are rare during pregnancy unless structural heart disease or hyperthyroidism is present. Diagnosis and treatment of the underlying condition are the first priorities.

Management

- Electrical cardioversion should be performed in the case of haemodynamic instability.
- In haemodynamically stable patients with no structural abnormalities, pharmacological termination of atrial flutter and AF should be considered. I.v. ibutilide or flecainide is usually effective, and may be considered, but the experience during pregnancy is very limited. Amiodarone is not recommended, unless other options fail, due to its fetotoxic effects.
- Cardioversion of atrial flutter and AF, whether electrically or by drugs, requires prior anticoagulation therapy and/or transoesophageal echocardiographic examination to exclude left atrial thrombus formation
- Anticoagulation (warfarin, substituted with UFH or LMWH in the first and last trimester) is considered mandatory for at least 3 weeks before elective cardioversion for AF or atrial flutter of 48 h duration or longer, or when the duration of AF is unknown, and should be continued for at least 4 weeks after cardioversion because of the risk of thrombo-embolism related to so-called 'atrial stunning'. For patients with AF duration, 48 h and no thrombo-embolic risk factors, i.v. heparin or weight-adjusted therapeutic dose LMWH may be considered pericardioversion, without the need for post-cardioversion oral anticoagulation.
- Indications for prophylactic antiarrhythmic drugs and anticoagulation relate to the presence of symptoms and the presence of risk factors for thrombo-embolism, respectively.
- In patients with risk factors for stroke or AF recurrence, antithrombotic treatment should continue lifelong irrespective of apparent maintenance of sinus rhythm following cardioversion.

Anticoagulation in atrial fibrillation

The risk for thrombo-embolism in AF depends on the presence of risk factors. Patients without structural heart disease or risk factors ('lone atrial fibrillation') have the lowest risk of thrombo-embolic events and do not require anticoagulation or antiplatelet therapy outside of or during pregnancy. In pregnant patients, thromboprophylaxis is recommended in high risk patients. The choice of the anticoagulant is made according to the stage of pregnancy.

- Vitamin K antagonists are recommended in most cases from the second trimester until 1 month before expected delivery.
- Weight-adjusted therapeutic doses of LMWH administered subcutaneously is recommended during the first trimester and during the last month of pregnancy.
- Either single or dual antiplatelet therapy (clopidogrel and acetylsalicylic acid) were not as effective as warfarin in high risk patients with atrial fibrillation.

Rate control

AV nodal blocking drugs including digoxin, β-blocking agents, and non-dihydropyridine calcium channel antagonists (verapamil, diltiazem) are used. For heart rate control of AF, β-blockers are recommended as first choice. Verapamil should be the drug of second choice. Digoxin can also be used but is less effective during strenuous exercise. Prophylactic antiarrhythmic drugs (sotalol, flecainide, or propafenone) may be considered in the case of severe symptoms despite rate-controlling drugs.

Other Interventions
These include:

- Catheter ablation: This is used where tachycardias are refractory to drugs and symptoms are poorly tolerated. Due to the radiation exposure, this is generally done after the 1st trimester
- Implantation of a cardioverter-defibrillator

15:4 Venous Thromboembolism

Pregnancy and puerperium are associated with an 4-5 fold increased risk of venous thromboembolism. This risk is highest in the immediate post partum period, especially post cesarean delivery. VTE includes pulmonary embolism and deep venous thrombosis. Risk factors include:

Table 15:8 Risk factors for venous thromboembolism (Adapted from the Royal College of Obstetricians and Gynaecologists)

Pre-existing risk factors
Previous recurrent VTE[a]
Previous VTE-unprovoked or oestrogen related[b]
Previous VTE-provoked
Family history of VTE
Known thrombophilia
Medical co-morbodities e.g. heart or lung diseases, SLE, cancer, inflammatory conditions, nephritic syndrome, sickle cell disease, i.v. drug use
Age > 35 years[c]
Obesity, BMI > 30kg/m^2
Parity ≥3
Smoker
Gross varicose veins
Obstetric risk factors
Pre-eclampsia
Dehydration/hyperemesis/ovarian hyperstmulation syndrome
Multiple pregnancy or assisted reproductive therapy
Emergency caesarean section
Elective caesarean section
Mid-cavity or rotational forceps
Prolonged labour (>24 hours)
Peripartum hemorrhage (>1 L or transfusion)
Transient risk factors
Current systemic infection
Immobility
Surgical procedure in pregnancy or <6 weeks post-partum
[a]Patients with previous recurrent VTE).1) or those with [b] a previous unprovoked or oestrogen related VTE belong to the high risk group [c]Obesity based on booking weight

Preventative measures for VTE according to risk

Table 15:9 Risk groups according to risk factors, definition and preventive measures modified according to the Royal College of Obstetricians and Gynaecologists

Risk groups	**Definition according to risk factors listed in table 6**	**Preventive measures according to risk group**
High risk	Patient with: Previous recurrent VTE(>1) or VTE unprovoked/oestrogen-related Or Single previous VTE + thrombophilia or family history	High risk patients should receive antenatal prrophylaxis with LMWH as well as post-partum for the duration of 6 weeks Graduated compression stockings are also recommended during pregnancy and post-partum
Intermediate risk	Patients with: 3 or more risk factors other than thse listed above as high risk 2 or more risk factors other than those listed as high risk if patient is admitted to hospital	In intermediate risk patients antenatal prophylaxis with LMWH should be considered. Prophylaxis is recommended postpartum for at least 7 dys or longer, if > 3 risk factors persist. Graduated compression stockings should be considered during pregnancy and postpartum.
Low risk	Patients with: <3 risk factors	In low risk patients early mobilization and avoidance of dehydration is recommended.
LMWH=Low molecular weight heparin; VTE= venous thromboembolism		

Deep venous thrombosis

Clinical presentation

Patients may present with unilateral or bilateral lower limb swelling. DVT is left sided in 85% of cases, due to compression of the left iliac vein by the right iliac artery and the gravid uterus. Iliac vein thrombosis may manifest with isolated pain in the buttock, groin, flank, or abdomen.

Diagnostic pathway for DVT

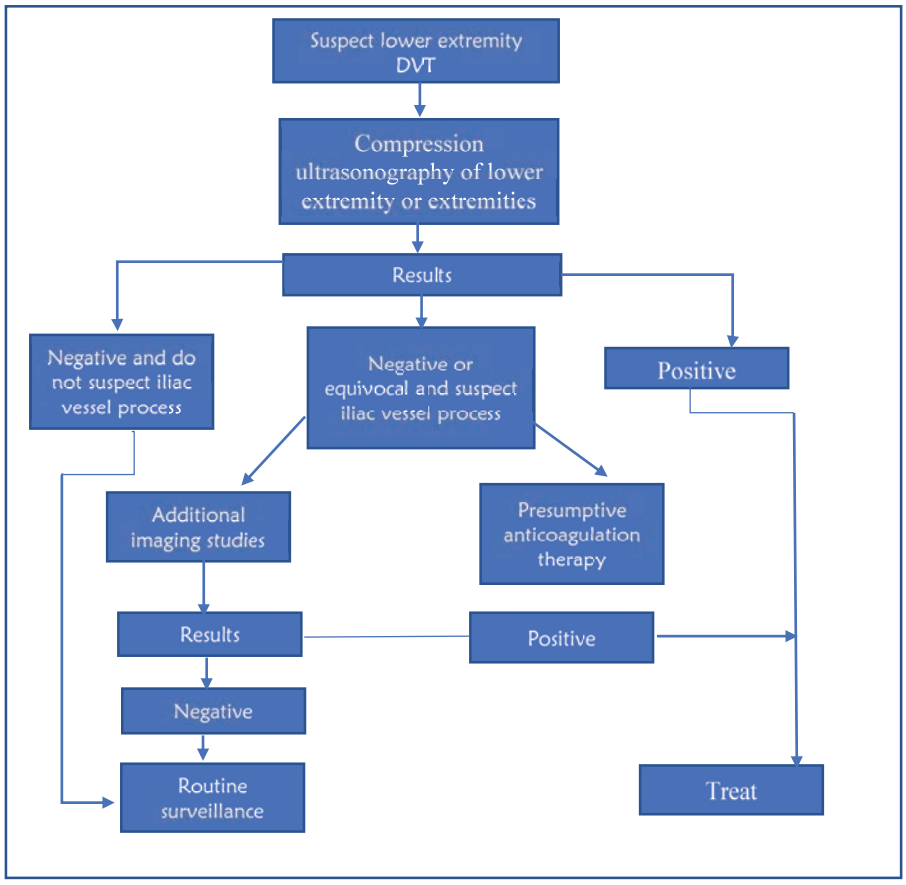


Figure 15:3 Diagnosis of DVT

Pulmonary Embolism

The clinical symptoms and signs of pulmonary embolism during pregnancy are the similar to the non-pregnant state (dyspnoea, chest pain, tachycardia, haemoptysis, and collapse). Subjective clinical assessment of pulmonary embolism may be difficult, because dyspnoea and tachycardia are not uncommon in normal pregnancy.

Simplified Diagnostic Pathway

Table 15:10 Simplified Wells' score for clinical probability prediction of P.E

Clinical Feature	Score
Previous PE or DVT	1
Pulse ≥ 100 beats/minute	1
Surgery or immobilization within the past 4 weeks	1
Hemoptysis	1
Active cancer	1
Clinical signs of DVT	1
Alternative diagnosis less likely than PE	1
Clinical Probability	
PE likely	≥2
PE unlikely	0-1

Baseline Investigations

1. Imaging studies: Duplex Doppler, V/Q scan, CTPA
2. Laboratory studies: Full blood count, coagulation screen, urea and electrolytes, and liver function tests.

Treatment of Venous thromboembolism

a. Low Molecular Weight Heparin (LMWH)

LMWH is the drug of choice for the treatment of VTE in pregnancy and puerperium

Dosage

Enoxaparin 1mg/kg body weight twice daily

Dalteparin 100 IU/kg body weight twice daily

b. Unfractionated Heparin (UFH)

It does not cross the placenta, however it is associated with more thrombocytopenia and osteoporosis. It is indicated in the acute treatment of massive pulmonary emboli or in renal failure when urgent reversal of anticoagulation by protamine may be needed.

Dosage

In patients with acute pulmonary embolism with haemodynamic compromise, intravenous administration of UFH is recommended (loading dose of 80 U/kg, followed by a continuous i.v infusion of 18 U/kg/h).

Monitoring

The aPTT has to be determined 4–6 h after the loading dose, 6 h after any dose change, and then at least daily when in the therapeutic range. The therapeutic target aPTT ratio is usually 1.5– 2.5 times the average laboratory control value. The dose is then titrated to achieve a therapeutic aPTT, defined as the aPTT that corresponds to an anti-Xa level of 0.3–0.7 IU/mL. When haemodynamics are improved and the patient is stabilized, UFH can be switched to LMWH in therapeutic doses and maintained during pregnancy.

LMWH should be switched to i.v. UFH at least 36 h before the induction of labour or caesarean delivery. UFH should be discontinued 4–6 h before anticipated delivery, and restarted 6 h after delivery if there are no bleeding complications.

Neither UFH nor LWMH is found in breast milk in any significant amount and they do not represent a contraindication to breastfeeding.

c. Warfarin

This is a vitamin K antagonist. It may be used where LMWH is not feasible from the second trimester, however it has been observed to be fetotoxic in some cases. It should be stopped at 37 0/7 weeks, and the patient started on LMWH in anticipation of delivery.

- Dose is titrated to achieve a therapeutic INR of 2-3
- Warfarin does not enter breast milk and is therefore safe for use while breastfeeding

Post-partum management

- In patients with VTE, pre-partum heparin treatment should be restarted 6 h after a vaginal birth and 12 h after a caesarean delivery, if no significant bleeding has occurred.
- Women should be offered the option to continue with heparin or warfarin, with the attendant risks, costs and monitoring protocols explained
- If a woman opts for warfarin, it should be started on the second day post partum to overlap with heparin treatment for 3-5 days, until a therapeutic INR of 2-3 is achieved.
- Anticoagulation should be continued until at least 6 weeks post partum, or until 3 months of treatment have been completed. Before stopping treatment, the on-going risk of VTE should be assessed.

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16 Cardiovascular Disease in Athletes

16 Cardiovascular Disease in Athletes

List of Abbreviations

AV	Atrial-ventricular
LV	Left Ventricle
ECG	Electrocardiogram
LVH	Left Ventricular Hypertrophy
RBBB	Right Bundle Branch Block
PPE	Pre-participation Assessment
BP	Blood Pressure
CMR	Cardiac Magnetic Resonance Imaging
VSD	Ventricular septal Defect
ASD	Atrial septal Defect

16:1 Introduction

In this document, we define athlete broadly and include participants at all levels of sport, including:

- Competitive athletes
- Scholastic (e.g. high school)
- Club
- Collegiate
- Professional
- Recreational athletes
- Persons who exercise for fitness and health

The main conditions of focus are athlete's heart, hypertension in athletes, heart failure in athletes and sudden cardiac death

1. Athletes Heart

Physiology

Intensive, prolonged endurance and strength training causes many physiologic adaptations including:

- Volume and pressure loads in the left ventricle (LV) increase, which, over time, increase LV muscle mass, wall thickness, and chamber size.
- Maximal stroke volume and cardiac output increase, contributing to a lower resting heart rate and longer diastolic filling time.
- Lower heart rate results primarily from increased vagal tone. Bradycardia decreases myocardial oxygen demand; at the same time, increases in total Hb and blood volume enhance oxygen transport. Despite these changes, systolic function and diastolic function remain normal. Structural changes in women are typically less than those in men of the same age, body size, and level of training.

Symptoms and Signs

There are usually no symptoms

Signs vary but may include:

- Bradycardia
- LV impulse that is laterally displaced, enlarged, and increased in amplitude
- Systolic ejection (flow) murmur at the left lower sternal border
- A 3rd heart sound (S3) due to early, rapid diastolic ventricular filling
- A 4th heart sound (S4), heard best during resting bradycardia because diastolic filling time is increased
- Hyperdynamic carotid pulses

These signs reflect structural cardiac changes that are adaptive for prolonged intense exercise.

Diagnosis

- Clinical evaluation
- Usually ECG
- Sometimes echocardiography
- Rarely, stress testing

Findings are typically detected during routine screening or during evaluation of unrelated symptoms. Most athletes do not require extensive testing, although ECG is often warranted. If symptoms suggest a cardiac disorder (e.g., palpitations, chest pain), ECG, echocardiography, and exercise stress testing are done.

It is important for any ECG to be interpreted by a clinician familiar with changes in the ECG commonly associated with athlete's heart. Athletes often manifest findings on the ECG that might suggest pathology in non-athletes but are normal variants in this population. These findings are shown in the figure below. LVH criteria can also differ in athletes.

Table 16:1 Classification of ECG abnormalities in athletes

Classification of abnormalities of the athlete's electrocardiogram
Sinus bradycardia.
First-degree AV block.
Incomplete RBBB.
Early repolarization.
Isolated QRS voltage criteria for left ventricular hypertrophy.

Athlete's heart is a diagnosis of exclusion; it must be distinguished from disorders that cause similar findings but are life threatening (e.g., hypertrophic or dilated cardiomyopathies, ischemic heart disease, arrhythmogenic right ventricular dysplasia).

Summary

Athlete's heart is a constellation of structural and functional changes that occur in the heart of people who train for > 1 h most days. The changes are asymptomatic; signs include bradycardia, a systolic murmur, and extra heart sounds. ECG abnormalities are common. Diagnosis is clinical or by echocardiography. No treatment is necessary other than reassurance. Athlete's heart is significant because it must be distinguished from serious cardiac disorders.

2. Hypertension in Athletes

Introduction

Hypertension is among the most common conditions seen in primary care and the most common cardiovascular condition affecting athletes(1,2). However, the management of hypertension in athletes can differ from standard approaches, primarily due to the potential side effects of some medications that may impair training and performance.

The fundamental parameters defining hypertension in both adults and children do not differ in athletes

Systolic blood pressure rises during endurance (e.g. weight lifting and wrestling) and dynamic (e.g. swimming, running, soccer) exercise; both systolic and diastolic blood pressures rise during endurance/resistance/static exercise. These increases in blood pressure reflect the body's efforts to increase cardiac output in order to meet the metabolic requirements of working muscles.

Pre-Participation Examination and Blood Pressure Screening

Pre-Participation Examinations (PPEs) are focused medical assessments for individuals engaged in competitive sports. Hypertension is the most common cardiovascular abnormality identified during the PPE. A blood pressure (BP) measurement must be performed during the pre-participation examination (PPE)(1). Diagnosis of Hypertension is as per the recommendations outlined in another section of this document.

If an athlete is found to have an elevated BP during the PPE they are not allowed to participate in sport until their follow-up BP evaluation. The diagnosis of hypertension is not established until a second elevated measurement is obtained at a subsequent visit. No workup is required following the initial elevated measurement (Refer to Chapter On hypertension)

Evaluation and Clearance for Sports Participation

We concur with the management recommendations for hypertension in athletes found in international guidelines (1) as shown in the table below.

Table 16:2 Recommendations for Hypertension in athletes

Stage of Hypertension	Recommendations
Prehypertension or Stage 1 hypertension without signs of end organ damage	• No restrictions on their participation in sport • Athletes with prehypertension should have their BP rechecked annually • Athletes diagnosed with Stage 1 hypertension should be treated and have their BP monitored according to standard guidelines.
Stage 1 hypertension (BP between 140/90 and 159/99) accompanied by signs of end organ damage	• **Should not** participate in sport until their BP is well-controlled
Stage 2 hypertension (BP >160/100)	• **Should not** participate in sport until their BP is well-controlled

Workup of athlete diagnosed with hypertension

All athletes with a new diagnosis of hypertension or prehypertension should receive an appropriate clinical, laboratory and other workup as would be performed for any patient.

In athletes younger than 25 years diagnosed with hypertension, the examination should include a lower-extremity BP to assess for coarctation of the aorta (1). Specifically, diminished or delayed femoral pulses (brachial femoral delay) and low or unobtainable blood pressure in a lower extremity raise concern and indicate the need for further investigation of possible coarctation.

A thorough review of the athlete's diet and use of supplements, including ergogenics, recreational drugs, alcohol and other performance-enhancing substances which may be related to hypertension should be done.

Table 16:3 Possible causes of elevated blood pressure

Medication/Supplement Category	Examples
Ergogenics	• Caffeine
Androgen hormones	• Testosterone • Testosterone analogs
Recreational drugs and Stimulants	• Nicotine • Ephedra-containing medications and supplements • Amphetamines • Cocaine • Phencyclidine (PCP) • Alcohol
Non-Steroidal Anti-Inflammatory drugs (NSAIDs)	
Other performance-enhancing substances associated with hypertension	• Human growth hormone, • Erythropoietin • Creatine

Special considerations for child athletes

Renal ultrasound is recommended for child athletes with established hypertension(1). In addition, an echocardiogram is recommended for child athletes with diabetes or renal disease associated with a BP between the 90th and 94th percentiles. An echocardiogram is part of the initial evaluation of all children with a BP in the 95th percentile or higher. When indicated, these imaging studies are performed in addition to the thorough history and physical examination, including a retinal examination, performed in any child with unexplained hypertension.

Treatment Strategies

Non-pharmacologic interventions — A number of lifestyle modifications can be used effectively to lower blood pressure. These non-pharmacologic interventions are discussed in greater detail in Chapter…..

Medical therapy — If non-pharmacologic modifications fail to reduce BP adequately in an athlete with persistent hypertension, medical therapy is required. The ideal medication controls BP without compromising exercise capacity. In addition, the medication selected should be permissible under the rules of the governing body for their sport (i.e., not a banned substance). The general principle of initiating treatment with monotherapy still holds, but the choice of medications requires caution. We suggest avoiding both diuretics and beta blockers. In athletes, diuretics are relatively contraindicated, as they can impair athletic performance due to volume depletion, and they are prohibited by many of the governing bodies in sport because they may be used to prevent detection of performance enhancing drugs. In addition, beta blockers decrease heart rate and thus can reduce exercise tolerance; they too are prohibited in some sports.

Angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), and long-acting calcium channel blockers are better choices for athletes as they do not impede athletic performance and are permissible under the rules of most governing bodies in sport. Starting doses for athletes are the same as for members of the general population, as is the approach to increasing the dose as needed based on patient response. As treatment is initiated and modified based on response, follow-up on a biweekly basis is performed to monitor BP, treatment compliance, general adverse effects, and effects on sport performance.

Persistent hypertension — Athletes with hypertension that has persisted for a longer period (6 to 12 months) or that has not responded to lifestyle modifications and pharmacotherapy should be assessed with an echocardiogram(2,3). Concentric LV hypertrophy with diminished LV volume is a form of end-organ damage and requires modification of activity (strenuous athletic activity is restricted) and implementation of appropriate medical therapy.
Although hypertension must still be treated appropriately, patients with non-pathologic concentric LV hypertrophy (i.e., normal LV volume and function) or eccentric hypertrophy consistent with athlete's heart do not require long-term activity modification once their hypertension is adequately controlled.

Competition
Generally, antihypertensive medications should be continued during competition.

Prognosis
The potential long-term complications from hypertension in athletes are thought to be similar to those in the general population, and include myocardial infarction, stroke, renal failure, and death(4). Hypertension is not recognized as a cause of exertional sudden cardiac arrest.

Left ventricular hypertrophy deserves close attention in athletes, as the degree of hypertrophy can affect exercise capacity. It is important for an experienced clinician to review any echocardiographic studies obtained to distinguish between athletic heart (eccentric or concentric hypertrophy with an increase in left ventricular volume) and left ventricular hypertrophy of hypertension (concentric hypertrophy with a diminished left ventricular volume).

3. Heart Failure in Athletes

Pathophysiology, inducing factors and clinical consequences
Exercise hemodynamics
Normal response to incremental exercise is characterized by:

- Doubling in heart rate (HR)
- Threefold or fourfold increase in cardiac output (CO)
- Moderate increase in mean arterial pressure (MAP)
- Markedly decreased total peripheral resistance.

This adaptive physiology is needed to meet the greatly increased demand for oxygen by a large mass of muscles.

From athlete's heart to heart failure

A. PHYSIOLOGIC VERSUS PATHOLOGIC HYPERTROPHY
Normal response (physiologic) to exercise leads to enlargement (hypertrophy) of the heart muscle cells with preserved cardiac function. In pathological hypertrophy, there is inadequate amount of nutrients and oxygen reaching the heart muscle due to excessive growth in response to stressors such as hypertension. Initially there may be features of Left Ventricular Outflow Tract obstruction, giving a murmur similar to that of aortic stenosis, and might also lead to syncope. Subsequently there is negative remodeling, fibrosis, cell death and cardiac dysfunction/heart failure.

B. Concentric and Eccentric Hypertrophy
As depicted in the diagram below, exercise with a predominantly static component is characterized by sustained periods of increased mean arterial pressure and peripheral resistance, and only a moderate increase in oxygen consumption. Physiological response to elevated mean pressure is concentric left ventricular wall thickening with relative contraction of the chamber volume (red panel).

Dynamic exercise is typically associated with a marked increase in oxygen consumption and cardiac output, a moderate increase in mean arterial pressure and a marked drop in peripheral resistance. Such a volume load results in eccentric left ventricular enlargement (blue panel).

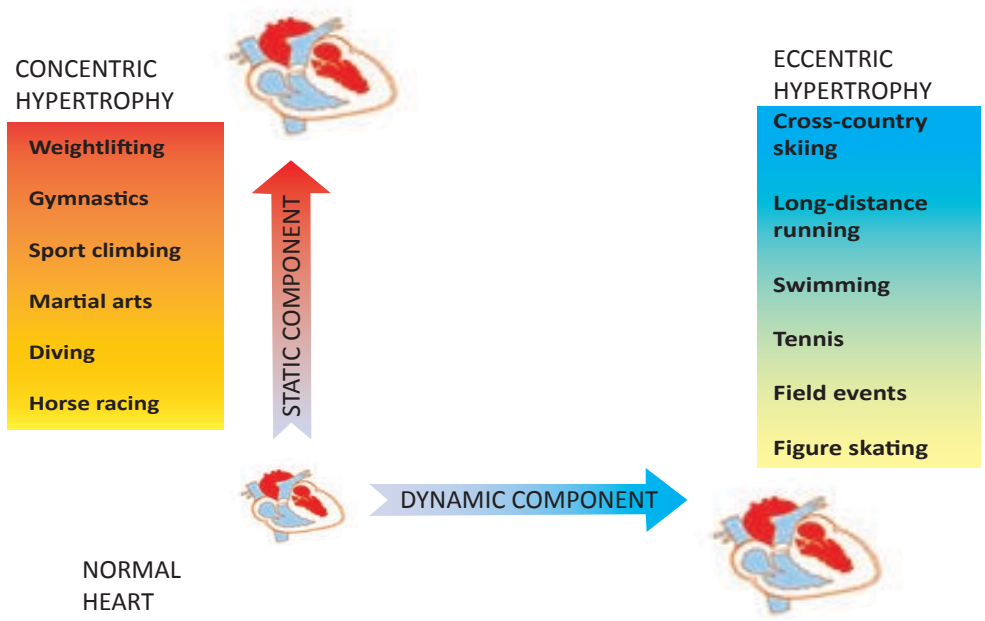

Figure 16.1: Cardiovascular adaptation to exercise

Symptoms of heart failure in athletes

Ambitious athletes are inclined to dissimulate symptoms or blame them on non-cardiac causes. The most challenging group is elderly athletes who often attribute their exertional dyspnea or fatigue to ageing.

The following symptoms may indicate heart failure in athletes:

- Shortness of breath, unexpected drop in performance, dyspnea on exertion
- Persistent fatigue and muscle pains of uncertain etiology, refractory to anti-in flammatory medications, physiotherapy, reduction in training intensity or exercise cessation
- Mental disorder – especially when combined with fatigue
- Pre-syncope or dizziness
- Persistent cough
- Nocturia or oliguria
- Lower extremities swelling
- Anginal pains
- Recent history of respiratory tract infection and drop in performance
- Persistent heartbeat irregularities

Diagnosis and appropriate management of chronic Heart Failure (HF) in this group can in addition be compounded by other comorbidities such as Chronic Obstructive Pulmonary Disease (COPD), diabetes mellitus or depression. Moreover, the fatigue or drop in performance is often ignored and attributed to overtraining. It should be noted that electrocardiography, basic laboratory tests, and biomarkers (N-terminal proBNP, BNP), can be falsely negative in some groups of athletes suffering from HF. Hence, the persistence of symptoms in cases with normal physical examination should always be supplemented by cardiac imaging, prolonged electrocardiographic monitoring (Holter-ECG) and cardiopulmonary exercise test (CPET).

Causes of heart failure in athletes

Heart failure in athletes may be secondary to various conditions as shown in table xx below:

Table 16:4 Causes of heart failure in athletes

Causes	Clinical consideration	Diagnostic tools
Myocarditis	• Mainly athletes below 35 years • Viral or rarely parasitic origin • Sometimes subclinical; usually preceded /precipitated by a respiratory tract infection. • Athletes with a history of acute myocarditis should be regularly screened for symptoms of HF, and diastolic global and regional LV dysfunction on echocardiography.	It is advisable that athletes who have suffered a recent respiratory tract infection undergo medical check-up (detailed physical examination, resting ECG - negative T-waves, pathological Q-waves, intraventricular conduction disturbances can indicate acute myocarditis even in athletes with normal echocardiogram) before they resume intensive physical training. CMR can help differentiate between inflammatory and ischemic LV dysfunction
Arterial hypertension	High static and high dynamic sports can contribute to hypertensive diastolic and/or systolic HF	Echocardiography, CMR
Drug abuse or doping	Cocaine or amphetamine addiction, anabolic abuse	Echocardiography, CMR
Iatrogenic myocardial damage	Chemotherapy (anthracyclines), radiotherapy (chest irradiation) – dose-dependent. Ventricular function testing necessary prior to and during chemotherapy. Responds well to ACE-Inhibitor treatment.	Echocardiography, CMR
Inherited dilated cardiomyopathy	Genetically conditioned (e.g., deletion in dystrophin gene)	Echocardiography, CMR
Ischemic cardiomyopathy	Post-myocardial infarction contractile dysfunction with lowered EF (below 50%)	Echocardiography, CMR
Hypertrophic cardiomyopathy	Diastolic and/or systolic HF	Echocardiography, CMR, genetic tests
Left ventricle non-compaction (LVNC)	Genetically conditioned, differential diagnosis with exercise-induced hypertrabeculations in athletes	Echocardiography, CMR – the most reliable diagnostic tool
Acquired or congenitalvalve defects	Mitral or aortic insufficiency, aortic stenosis, VSD, ASD, Ebstein anomaly	Echocardiography, CMR

Athletes suffering or recovering from any of these causes should be on close follow up and screening for Heart Failure

4. Sudden Cardiac Death in Athletes

Sudden cardiac death (SCD) resulting from undetected cardiac abnormalities in athletes is a tragic and potentially avoidable event. There is overwhelming evidence that exercise can trigger ventricular arrhythmias and cardiac arrest in individuals with preexisting heart conditions, even in well-conditioned young athletes(5). The vast majority of these sudden deaths are caused by previously unidentified and asymptomatic underlying cardiovascular conditions.

Pathophysiology of SCD

The cardiovascular conditions triggering SCD in athletes vary in their prevalence in different cultures. The common final pathway of SCD in athletes is the result of ventricular tachyarrhythmias. Below are details of common causes of SCD.

i. Hypertrophic Cardiomyopathy

HCM is the most common genetic cardiac disease, inherited as an autosomal dominant trait(6). Findings that suggest HCM may include a heart murmur, family history, an abnormal ECG (increased precordial or limb lead voltages, alterations in ST segments, T-wave inversion, or deep and narrow Q waves), or new cardiac-related symptoms such as shortness of breath on exertion, dizziness, and/or syncope.

ECG abnormalities may be significant but do not reliably reflect the severity of disease(7). The gold standard for diagnosing HCM is with two-dimensional echocardiography to identify a thickened LV wall (>12 mm) in the absence of a dilated chamber(8)

ii. Arrhythmogenic right ventricular cardiomyopathy (ARVC)

Many different cardiac conditions can be grouped as ARVCs and share a common pathology of structural abnormalities(9), resulting in a thin and dilated right ventricular wall. ARVC leads to arrhythmias that are exacerbated during exercise and it should be considered during cardiovascular screenings of athletes. Early diagnosis in the absence of symptoms is often difficult and the initial presentation in 30% of patients is syncope(10).

iii. Congenital coronary artery anomalies

Normal coronary artery anatomy for the majority of individuals includes a left main coronary artery and a right coronary artery that originate at their respective sinuses of Valsalva. A diagnosis of anomalous coronary artery origin is considered when any deviation from this normal anatomy occurs and this may cause SCD in individuals by causing transient myocardial ischemia during vigorous exercise(11). Coronary artery angiography has traditionally been considered the gold standard for diagnosis, but newer noninvasive techniques, such as magnetic resonance imagery and computed tomography, are replacing angiography.

iv. Arrhythmias

Non-pathologic arrhythmias do occur among athletes. However, all individuals in whom arrhythmias are suspected (palpitations, irregular pulse/heart rate or abnormal heart sounds) require urgent cardiovascular evaluation a physician or cardiologist.

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17 Palliative Care

17 Palliative Care

17:1 Introduction

Palliative Care for both children and adults is an approach that improves the Quality of Life of patients and their families facing the problems associated with a life-threatening illness, through 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 (WHO, 2002)
We should not just focus on preventing avoidable deaths but also on preventing avoidable suffering. Positioning palliative care within a continuum of care for CVDs is recommended.
Palliative care is required throughout the course of illness regardless of access to disease modifying treatment.

Benefits of palliative care

- Causes patients to spend more time at home and reduces the number of hospital inpatients days
- Improves symptom management
- Provides patient, family and care takers satisfaction
- Reduces overall cost of disease
- Prolongs survival
- Improves quality of life of patients and family

17:2 Provision of Palliative Care Services

Table 17:1 Palliative care services

Aspect of Palliative/ Supportive Care	Definition and Scope
Palliative care plan	Palliative care should be provided by a multidisciplinary team A patient should have a detailed holistic assessment and care plan developed by the palliative care provider in collaboration with the patient and family in order of priority
Pain control	Effective pain control is central to palliative care using both pharmacological and non-pharmacological measures. ***Pharmacological Measures*** The WHO analgesic ladder is used for all types of pain including nociceptive and neuropathic pain, and should be used as the standard approach to the management of pain Pain control drugs should be administered regularly –by the clock, by the ladder, by the mouth and for the individual patient • Opioids for control of moderate-to-severe pain • Non-steroidal anti-inflammatory drugs such as aspirin, paracetamol, ibuprofen and diclofenac for mild pain ***Non Pharmacological Measures*** • Education of the patient and family/carer on the condition to provide insight and support • Psychosocial therapy/care - companionship, music, art, aromatherapy, yoga, drama, and group therapy • Physical care: Exercises, heat/cold application, lotions/massage therapy, positioning, occupational therapy, physiotherapy, etc • Spiritual care: meditation, prayer and religious counseling • Palliative surgery and chemotherapy.

Symptom Control	• Assessment for the cause and severity of the symptom • Anticipate multiple symptoms- which may include physical, psychological, social and spiritual • Treatment of reversible causes • Initiation of disease/symptom-specific medicines and non-drug measures • Involvement of the patient and family on the management plan
Nutrition	Successful management of medicine-food interactions requires understanding clients' individual food access as well as eating habits Patient and caregivers shall be counseled on balanced healthy diet and appropriate feeding according to the stage of the illness
Infection Prevention and Control	• Hand hygiene • Personal protective equipment • Isolation precautions • Aseptic technique • Cleaning and disinfection and • Sterilization
Psychosocial care of patients and their families	Psychosocial interventions are an integral part of CVD management Information of common psychosocial issues including anxiety, depression, care of children, finances, will preparation, community support, and family relationships will be provided Psychosocial needs of the patient and family will be prioritized and these will be included in the care plan

Spiritual Care	Involves being a compassionate presence to patients even as they suffer. It recognizes that emotional and spiritual healing can take place even though a physical cure is impossible Areas of life that can generate spiritual peace or spiritual distress include a relationship with God/Creator/Higher Being, with self, with others, and with the world around them
End of life care	Health care providers shall prepare both the patient and the family on the impending death Care provider shall be honest, attend to emotional responses and spiritual needs, shall maintain presence and talk to the patient even if he/she is unconscious, give guidance as well as enquire about the presence of an ethical will Comfort measures shall be provided depending on the presenting signs and symptoms of impending death End-of-life concerns, hopes, fears, and expectations shall be openly and honestly addressed in the context of social and cultural customs in a developmentally appropriate manner Palliative care practice shall be guided by the medical-ethical principles of autonomy, beneficence, non-maleficence and justice
Grief and Bereavement	Grief and bereavement risk assessment shall be done routinely throughout the illness trajectory Care providers shall offer a safe, comforting place to the bereaved family to enable them express their feelings, thoughts and needs as they are going through bereavement

Pediatric Palliative Care (0- 16 YEARS)

Palliative care for children shall focus on pain assessment & management, psychosocial & emotional support and communication, which shall be appropriate for the age and developmental stage of the child.

Pediatric Pain Control

- Pain assessment tools should be age appropriate (Refer to Annex 8, National Palliative Care Guidelines – 2013).
- Aspirin is contraindicated in children less than 12 years.
- Dosages shall be calculated in kilogram per body weight (Annex 7, National Palliative Care Guidelines)

Special needs for children

- Special needs shall be identified through comprehensive assessment and addressed holistically.
- Children shall be involved in decisions about their own care. Appropriate information according to age shall be communicated in clear and simple language at their pace
- Children shall be allowed to lead a normal life that includes access to education within the limitation of their illness. School teachers, community members including other children shall be encouraged to support and deal sensitively with the affected child
- Recreation activities shall be encouraged like play activities, drawings, poems or songs.
- Palliative care providers shall take into consideration the needs of orphans and vulnerable children and shall refer them to appropriate services for care and support

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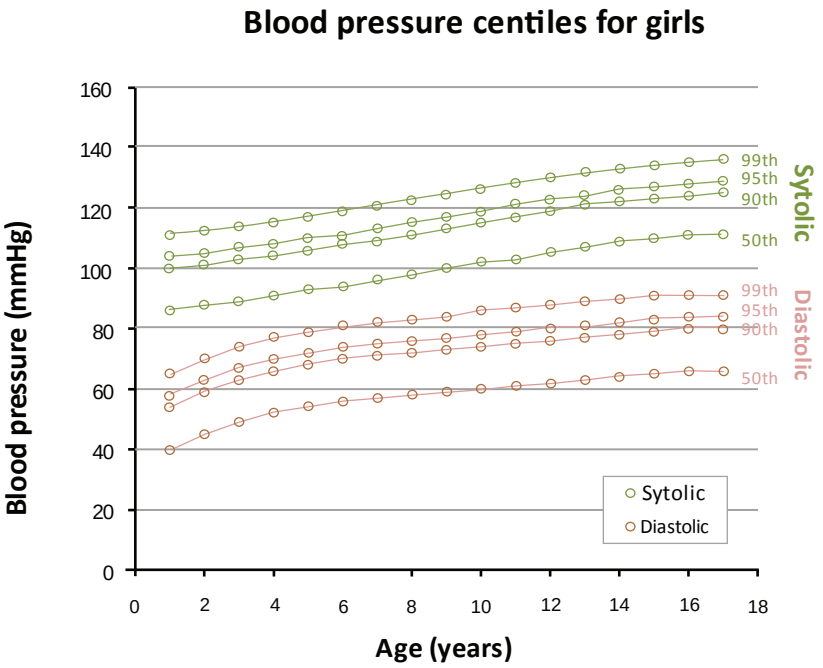
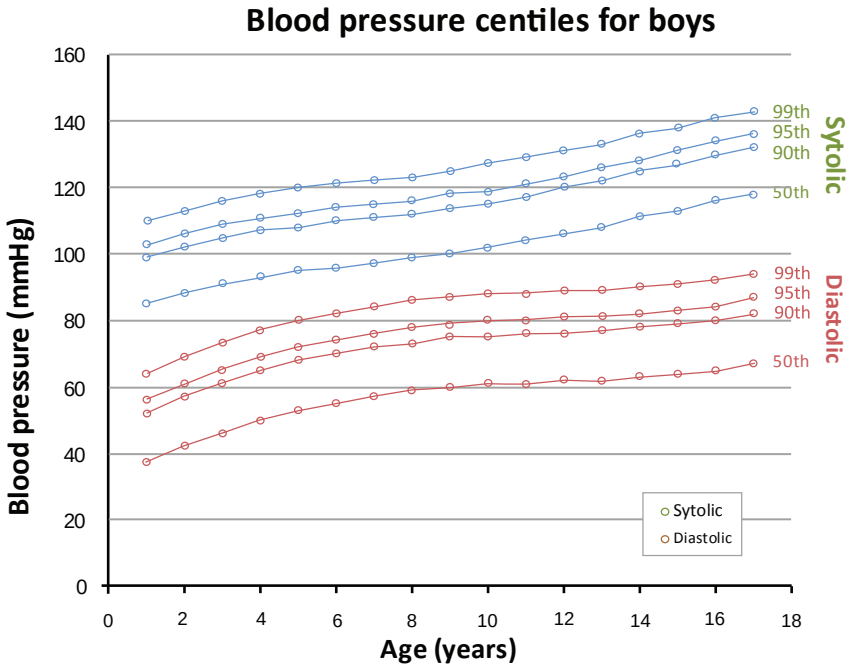
18 Annexes

Annex 1

Blood Pressure Centiles for Boys and Girls:





Annex 2

Caprini risk assessment model for Venous Thromboembolism

One point each	Two points each	Three points each	Five points
Age 41-60 years	Age 61-74 years	Age ≥ 75 years	Stroke (< 1 mo)
Minor surgery	Arthroscopic surgery	History of VTE	Elective arthroplasty
BMI > 25 kg/m^2	Major open surgery (> 45 min)	Factor V Leiden	Hip, pelvis, or leg fracture
Swollen legs	Laparoscopic surgery (> 45 min)	Lupus anticoagulant	Acute spinal cord injury (< 1 mo)
Varicose veins	Malignancy	Anticardiolipin antibodies	
Pregnancy or postpartum	Confined to bed (> 72 hour)	Elevated serum homocysteine	
History of unexplained or recurrent spontaneous abortion	Immobilizing cast	Heparin-induced thrombocytopenia	
Oral contraceptives or hormone replacement	Central venous access	Other congenital or acquired thrombophilia	
Sepsis (< 1 mo)		Family History of VTE	
Serious lung disease, including pneumonia (< 1 mo)			
Abnormal pulmonary function			
Acute myocardial infarction			

VTE - Risk Stratification and Recommendations		
Risk class	**Caprini Score**	**Recommendations**
Very low	0	No specific prophylaxis
Low	1-2	Mechanical prophylaxis
Moderate	3-4	LMWH/LDUH *or* mechanical prophylaxis
High	≥ 5	LMWH/LDUH *plus* mechanical prophylaxis
High-risk cancer surgery		LMWH/LDUH *plus* mechanical prophylaxis *and* extended-duration prophylaxis with LMWH post-discharge
High risk, LDUH and LMWHcontraindicated or not available		Fondaparinux *or* low-dose aspirin (160 mg); mechanical prophylaxis or both

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