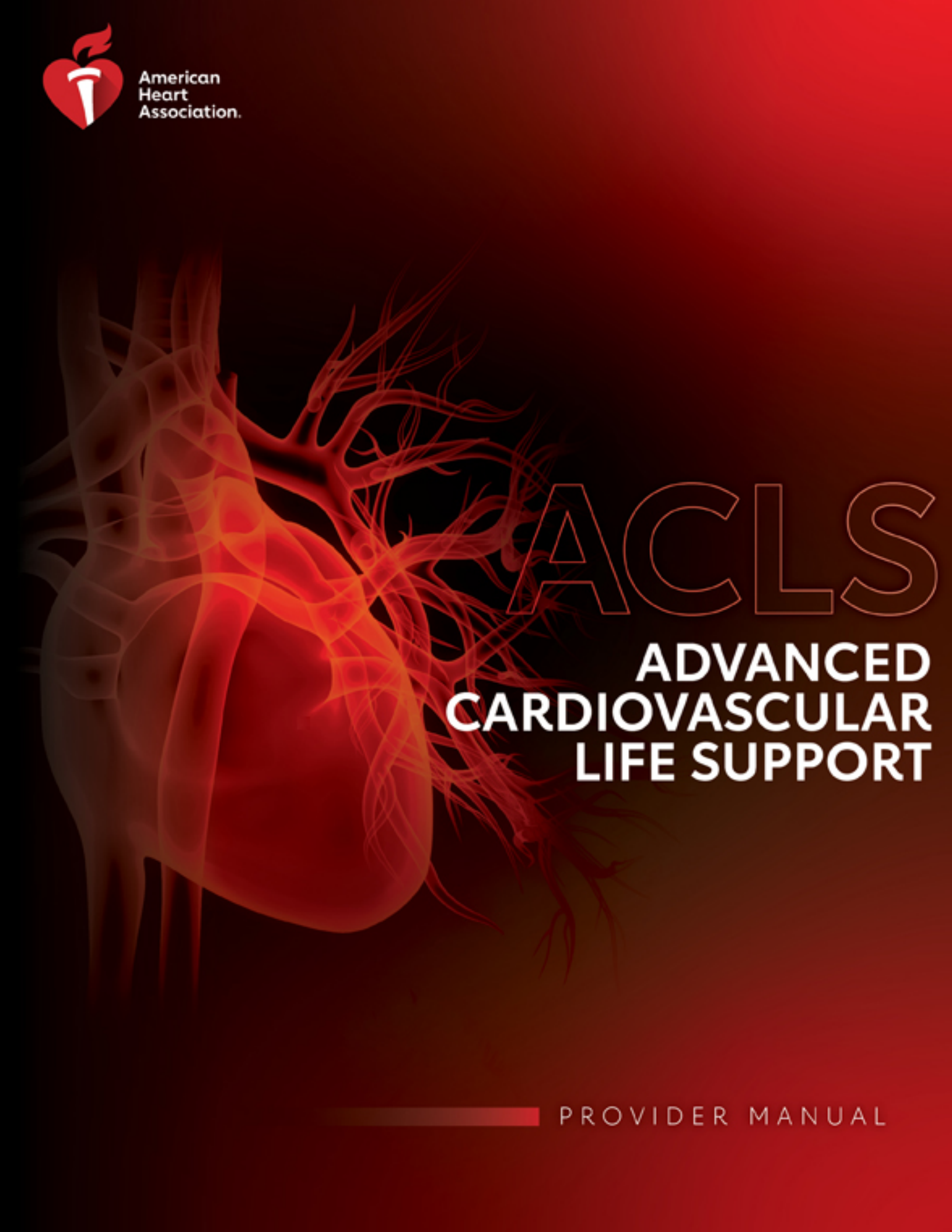
American Heart Association.



ACLS

ADVANCED CARDIOVASCULAR LIFE SUPPORT

PROVIDER MANUAL

Advanced Cardiovascular Life Support Provider Manual

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Acknowledgments

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ACLS Student Resources can be found at eLearning.heart.org. Contact your Training Center Coordinator for more information about accessing these before your course.

To find out about any updates or corrections to this text, visit www.heart.org/courseupdates.

Contents

Note on Medication Doses

Emergency cardiovascular care (ECC) is a dynamic science. Advances in treatment and drug therapies occur rapidly. Readers should use the following sources to check for changes in recommended doses, indications, and contraindications: the package insert product information sheet for each drug and medical device and the course updates available on **www.heart.org/courseupdates**.

Part 1: Overview of ACLS

Introduction

Course Description and Goal

The Advanced Cardiovascular Life Support (ACLS) Provider Course is designed for healthcare providers who either direct or participate in the management of cardiopulmonary arrest or other cardiovascular emergencies. Through didactic instruction and active participation in simulated cases, students will enhance their skills in the recognition and intervention of cardiopulmonary arrest, immediate post–cardiac arrest, acute dysrhythmia, stroke, and acute coronary syndromes (ACS). The goal of this course is to improve outcomes for adult patients of cardiopulmonary arrest and other cardiovascular emergencies through early recognition and interventions by high-performance teams.

Course Objectives

After successfully completing this course, you should be able to

- Define *systems of care*
- Apply the Basic Life Support (BLS) Assessment, Primary Assessment, and Secondary Assessment sequences for systematic evaluation of adult patients
- Discuss how using rapid response teams (RRTs) or medical emergency teams (METs) may improve patient outcomes
- Discuss early recognition and management of ACS, including appropriate disposition
- Discuss early recognition and management of stroke, including appropriate disposition
- Recognize bradycardia and tachycardia that may result in cardiac arrest or complicate resuscitation outcome
- Perform early management of bradycardia and tachycardia that may result in cardiac arrest or complicate resuscitation outcome
- Model effective communication as a member or leader of a high-performance team

- Recognize the impact of team dynamics on overall team performance
- Recognize respiratory arrest
- Perform early management of respiratory arrest
- Recognize cardiac arrest
- Perform prompt, high-quality BLS, which includes prioritizing early chest compressions and integrating early automated external defibrillator (AED) use
- Perform early management of cardiac arrest until termination of resuscitation or transfer of care, including immediate post–cardiac arrest care
- Evaluate resuscitative efforts during cardiac arrest by continually assessing cardiopulmonary resuscitation (CPR) quality, monitoring the patient's physiologic response, and delivering real-time feedback to the team

Course Design

To help you achieve these objectives, the ACLS Provider Course includes learning stations and a Megacode evaluation station. The learning stations provide activities such as

- Simulated clinical scenarios
- Video or instructor demonstrations
- Discussion and role-playing
- Group practice to achieve effective high-performance teams

In these learning stations, you will practice essential skills both individually and as part of a team. Because this course emphasizes effective team skills as a vital part of the resuscitative effort, you'll practice as both a team member and as Team Leader.

In the Megacode evaluation station at the end of the class, you will participate in a simulated cardiac arrest scenario to evaluate your

- Integration of core material and skills
- Application of algorithms
- Interpretation of arrhythmias
- Use of appropriate ACLS drug therapy
- Performance as an effective leader and member of a high-performance team
- Achieving objective measures such as chest compression fraction (CCF)

Course Prerequisites and Preparation

The American Heart Association (AHA) limits enrollment in this course to healthcare providers who

- Direct or participate in the resuscitation of patients in or out of hospital
- Have the basic knowledge and skills to participate actively with the instructor and other students

Before class, read the *ACLS Provider Manual*, complete the mandatory precourse work in ACLS Student Resources (accessed via **eLearning.heart.org**), identify any gaps in your knowledge, and remediate those gaps by studying the applicable content in the *ACLS Provider Manual* or other supplementary resources, including the ACLS Student Resources. You must pass the Precourse Self-Assessment with a minimum score of **70%**. You may retake the self-assessment as often as needed to pass. **Print your certificate of completion and bring it with you to the course.**

You will need the following knowledge and skills to successfully complete the course:

- BLS skills
- Electrocardiogram (ECG) rhythm interpretation for core ACLS rhythms
- Knowledge of airway management and adjuncts
- Basic ACLS drug and pharmacology knowledge
- Practical application of ACLS rhythms and drugs
- Effective high-performance team skills

BLS Skills

Strong BLS skills form the foundation of ACLS, so you must pass the high-quality BLS Testing Station to complete this course. *Make sure that you are proficient in BLS skills before attending the class*.

ECG Rhythm Interpretation for Core ACLS Rhythms

The basic cardiac arrest and periarrest algorithms require students to recognize these ECG rhythms:

- Sinus rhythm
- Atrial fibrillation and flutter
- Sinus bradycardia
- Sinus tachycardia

- Supraventricular tachycardia
- Atrioventricular blocks
- Asystole
- Pulseless electrical activity (PEA)
- Ventricular tachycardias (VTs)
- Ventricular fibrillation (VF)

The ACLS Precourse Self-Assessment contains an ECG rhythm identification section. Use your self-assessment score and feedback to help you identify your areas of strength and weakness before attending the class. You must be able to identify and interpret rhythms during course practice sessions and the final Megacode evaluation station.

Basic ACLS Drug and Pharmacology Knowledge

You must know the drugs and doses used in the ACLS algorithms. You will also need to know when to use which drug based on the clinical situation.

The ACLS Precourse Self-Assessment contains pharmacology questions. Use your self-assessment score and feedback to help you identify areas of strength and weakness before attending the class.

Course Materials

Course materials consist of the *ACLS Provider Manual*, in the ACLS Student Resources, and 3 reference cards.

The computer icon directs you to additional supplementary information ACLS Student Resources (accessed via **eLearning.heart.org**).

ACLS Provider Manual

The *ACLS Provider Manual* contains the basic information you will need to participate in the course, including the systematic approach to a cardiopulmonary emergency, information about effective high-performance team communication, and the ACLS cases and algorithms. **Review this manual before attending the class, and bring it with you to the class.** Students using the eBook version should download the manual to their device's eReader app and bring it with them, in case there is no internet connection.

The *ACLS Provider Manual* also contains important information presented in *Critical Concepts* and *Caution* callout boxes that require your attention:

Critical Concepts

These boxes contain the most important information you must know, including specific risks associated with certain interventions and additional background on key topics this course covers.

Caution

Caution boxes emphasize specific risks associated with interventions.

ACLS Student Resources

The ACLS Student Resources (accessed via **eLearning.heart.org**) contain mandatory precourse preparation and supplementary materials.

- Precourse Self-Assessment (passing score 70% or greater)
- Precourse work (complete interactive video lessons)

Use the following website resources to supplement basic concepts in the ACLS Course. Some information is supplementary; other areas provide additional information for interested students or advanced providers.

- Precourse Preparation Checklist (used to ensure that students are ready to attend the class).
- ACLS Supplementary Material
    - –Basic Airway Management
    - –Advanced Airway Management
    - –ACLS Core Rhythms
    - –Defibrillation
    - –Access for Medications
    - –Acute Coronary Syndromes
    - –Human, Ethical, and Legal Dimensions of ECC and ACLS
- Optional Videos
    - –Intraosseous Access
    - –Coping With Death

Reference Cards

The 3 stand-alone reference cards included with the *ACLS Provider Manual* (and sold individually packaged) provide quick reference for training in real emergencies on the following topics:

- Cardiac arrest, arrhythmias, and their treatment
    - –Adult Cardiac Arrest Algorithms
    - –Table with drugs and dosage reminders
    - –Adult Post–Cardiac Arrest Care Algorithm
    - –Adult Bradycardia Algorithm
    - –Adult Tachycardia With a Pulse Algorithm
- ACS and stroke
    - –Acute Coronary Syndromes Algorithm
    - –Fibrinolytic Contraindications for STEMI
    - –Adult Suspected Stroke Algorithm
    - –Emergency Medical Services Acute Stroke Routing Algorithm
    - –Hypertension Management in Acute Ischemic Stroke
- Cardiac arrest in select special situations and neuroprognostication
    - –Opioid-Associated Emergency for Healthcare Providers Algorithm
    - –Adult Ventricular Assist Device Algorithm
    - –Cardiac Arrest in Pregnancy In-Hospital ACLS Algorithm
    - –Neuroprognostication diagram

Use these cards as a reference when you are preparing for class, during the learning stations, and during real emergencies. You may refer to them during the Megacode and the cognitive exam.

Course Completion Requirements

To successfully complete the ACLS Provider Course and obtain your course completion card, you must

- Pass the Adult High-Quality BLS Skills Test
- Pass the Airway Management Skills Test, including oropharyngeal airway/nasopharyngeal airway insertion
- Demonstrate competency in learning station skills
- Pass the High-Performance Teams: Megacode Test
- Pass the open-resource exam with a minimum score of 84%

Advanced Cardiovascular Life Support

ACLS providers face an important challenge: functioning as a team that implements basic and advanced life support to save a person's life. The *2020 AHA Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care* reviewed evidence that in both in-hospital and out-of-hospital settings, many cardiac arrest patients do not receive

high-quality CPR, and most do not survive. One study of in-hospital cardiac arrest (IHCA) showed that CPR quality was inconsistent and did not always meet guidelines recommendations.[1] Over the years, however, patient outcomes after cardiac arrest have improved. Table 1 shows the recent survival trends in both IHCA and out-of-hospital cardiac arrest (OHCA) in the United States.[2]

Table 1. Recent Cardiac Arrest Survival Data

Statistical update	OHCA incidence, n	OHCA bystander CPR (overall), %	OHCA survival rate* (overall), %	IHCA incidence,† n	IHCA survival rate* (Adults), %
2020	356 461	41.6	10.4	209 000	25.8
2019	356 461	46.1	10.4	209 000	25.6
2018	347 322	46.1	11.4	209 000	25.8
2017	356 500	45.7	11.4	209 000	23.8
2016	356 500	46.1	12.0	209 000	24.8
2015	326 200	45.9	10.6	209 000	25.5
2014	424 000	40.8	10.4	209 000	22.7
2013	359 400	40.1	9.5	209 000	23.9
2012	382 800	41.0	11.4	209 000	23.1
Baseline		31.0	7.9		19.0

*Survival to hospital discharge.
†Extrapolated incidence based on the same 2011 Get With The Guidelines®-Resuscitation study.

Multiple evidence reviews have focused on the essentials of CPR, the links in the Chain of Survival, and the integration of BLS with ACLS. Minimizing the interval between stopping chest compressions and delivering a shock (ie, minimizing the preshock pause) improves the chances of shock success[3] and patient survival.[4] Experts believe that high survival rates from both in-hospital and out-of-hospital sudden cardiac death are possible with strong systems of care.

Several factors have been associated with improved survival in patients with cardiac arrest:

- Training healthcare providers to become more knowledgeable about what improves survival rates
- Proactive planning and simulation of cardiac arrest to provide the opportunity for a healthcare provider to practice and improve responding to cardiac arrest
- Rapidly recognizing sudden cardiac arrest
- Immediately providing high-quality CPR
- Defibrillating immediately, as soon as a defibrillator is available
- Providing goal-directed, time-sensitive post–cardiac arrest care

Rapid intervention by skilled people working within a strong system of care leads to the best outcomes.

Critical Concepts: Optimizing ACLS

Team Leaders can optimize ACLS by integrating high-quality CPR and minimal interruption of chest compressions with advanced life support strategies (eg, defibrillation, medications, advanced airway).

Studies have shown that reducing the interval between compressions and shock delivery can increase predicted shock success. Limit the number of interruptions in compressions to critical interventions (rhythm analysis, shock delivery, intubation, etc), and minimize the duration of necessary interruptions to 10 seconds or less.

Continuous Quality Improvement

Every emergency medical services (EMS) system and hospital system should assess its resuscitation interventions and outcomes through continuous quality improvement (CQI) with a defined process of data collection and review. The current consensus on the best way to improve both in-hospital and out-of-hospital survival after sudden cardiac arrest is to modify the standard quality improvement model according to the Chain of Survival metaphor. Each link in the chain comprises structural, process, and outcome variables that systems can examine, measure, and record. System managers can quickly identify gaps between observed processes and outcomes and local expectations or published standards. Individuals and teams who regularly review their performance in actual resuscitations will, on average, improve their performance in subsequent resuscitation events. Therefore, it is important for resuscitation teams to find the time to

debrief themselves at some time after every resuscitation, either immediately or later.

Systems of Care

A *system* is a group of interdependent components that regularly interact to form a whole. The system

- Provides the links for the Chain of Survival
- Determines the strength of each link and of the chain
- Determines the ultimate outcome
- Provides collective support and organization

Healthcare delivery requires **structure** (eg, people, equipment, education) and **processes** (eg, policies, protocols, procedures) that when integrated produce a **system** (eg, programs, organizations, cultures) leading to **outcomes** (eg, patient safety, quality, satisfaction). This integrated response, known as a *system of care*, comprises all of these elements—structure, process, system, and patient outcome—in a framework of CQI (Figure 1).

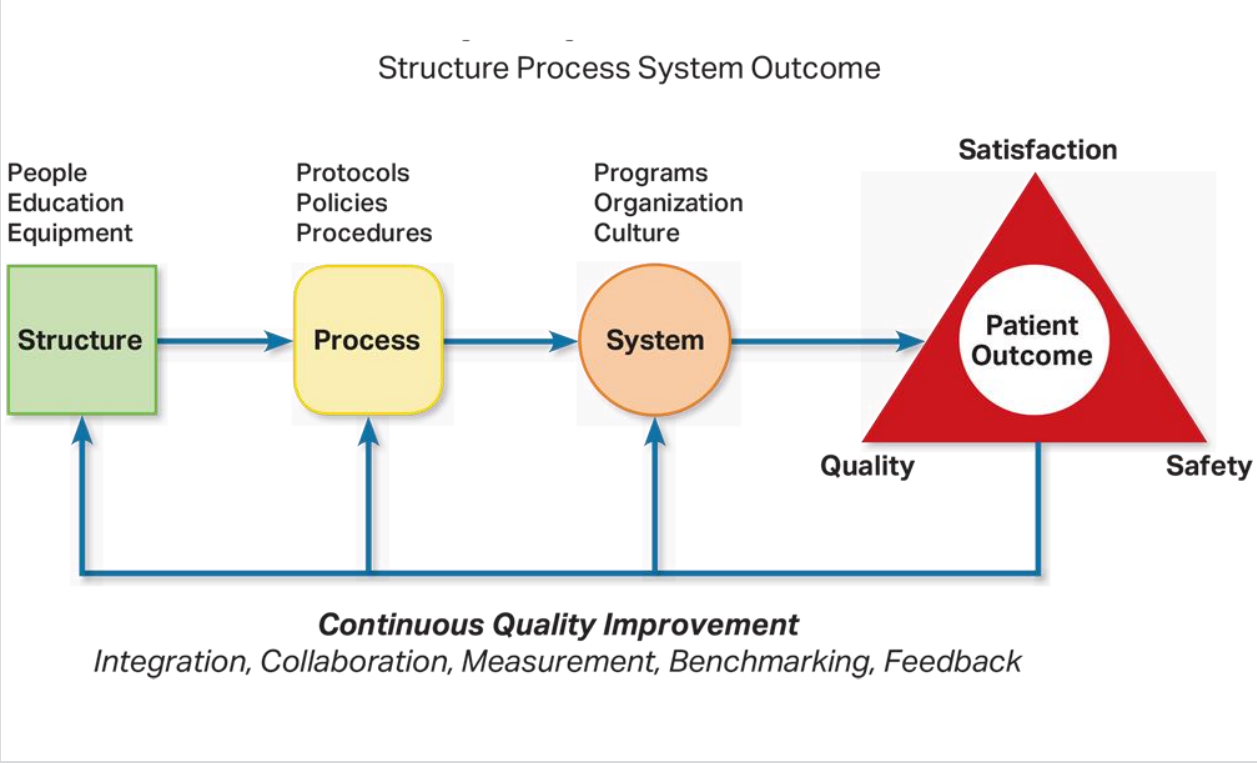


Figure 1. Taxonomy of systems of care.

These systems require individuals and groups to share information so that they can evaluate and improve their system. Leadership and accountability

are important components of this team approach. Participants and leaders in systems of care must continually assess the performance of each system component; only after this assessment can they effectively intervene to improve outcomes.

The CQI process consists of an iterative cycle of

- Systematically evaluating resuscitation care and outcome
- Creating benchmarks with stakeholder feedback
- Strategically addressing identified deficiencies

Cardiac Arrest and Post–Cardiac Arrest Systems of Care

Successful resuscitation requires integrated, coordinated actions. Experts believe that high survival rates from both in- and out-of-hospital sudden cardiac death are possible with strong systems of care.

Several factors have been associated with improved survival in patients with cardiac arrest:

- Training healthcare providers to become more knowledgeable about what improves survival rates
- Proactive planning and simulation of cardiac arrest to provide the opportunity for a healthcare provider to practice and improve responding to cardiac arrest
- Rapidly recognizing cardiac arrest
- Immediately providing high-quality CPR
- Early defibrillation, as soon as a defibrillator is available
- Providing goal-directed, time-sensitive post–cardiac arrest care

Rapid intervention by skilled people working within a strong system of care leads to the best outcomes.

The links in the system-specific Chains of Survival represent these actions. The chain of survival is a metaphor used to organize and describe the integrated set of time-sensitive coordinated actions necessary to maximize survival. The use of evidence-based education and implementation strategies can optimize the links in the chain. However, 2 separate chains (Figure 2) were created to reflect the differences in the steps needed for response to cardiac arrest in-hospital (IHCA) and out-of-hospital cardiac arrest (OHCA).

Figure 2. System-specific Chains of Survival.

Cardiac arrest can happen anywhere—on the street, at home, or in a hospital emergency department (ED), in-patient bed, or intensive care unit (ICU). Elements in the system of care and order of actions in the Chain of Survival differ based on the situation. Care will depend on whether the victim has the arrest outside the hospital or inside the hospital. Care also depends on whether the victim is an adult, child, or infant.

Chain of Survival Elements

Although there are slight differences in the Chains of Survival based on the age of the victim and location of the cardiac arrest, each includes the following elements:

- **Prevention and preparedness**, including responder training, early recognition of cardiac arrest, and rapid response
- **Activation of the emergency response system**, either outside of or within the hospital
- **High-quality CPR**, including **early defibrillation** of VF and pVT
- **Advanced resuscitation interventions**, including medications, advanced airway interventions, and extracorporeal CPR

- **Post–cardiac arrest care**, including critical care interventions and targeted temperature management
- **Recovery**, including effective support for physical, cognitive, emotional, and family needs

Regardless of where an arrest occurs, the care following resuscitation converges in the hospital, generally in an ED or ICU. This post–cardiac arrest care is depicted as the final link in both chains, symbolized by a hospital bed with a monitor and thermometer, which represent critical care interventions, advanced monitoring, and targeted temperature management. Patients who achieve ROSC after cardiac arrest in any setting have complex pathophysiologic processes called the *post–cardiac arrest syndrome*. This syndrome plays a significant role in patient mortality and includes

- Postarrest brain injury
- Postarrest myocardial dysfunction
- Systemic ischemia and reperfusion response
- Persistent acute and chronic pathology that may have precipitated the cardiac arrest[5]

Healthcare systems should implement a comprehensive, structured, consistent, multidisciplinary system of care for treating post–cardiac arrest patients. Programs should address ventilation and hemodynamic optimization, targeted temperature management (TTM), immediate coronary reperfusion with percutaneous coronary intervention (PCI) for eligible patients, neurologic care and prognostication, and other structured interventions. Individual hospitals that frequently treat cardiac arrest patients show an increased likelihood of patient survival when these interventions are provided.

As noted above, the structure and process elements before the convergence of the 2 chains vary significantly. Patients with OHCA depend on elements within the community for support. Lay rescuers must recognize the patient's arrest, call for help, and initiate CPR and early defibrillation (public-access defibrillation [PAD]) until a team of professionally trained emergency medical services (EMS) providers assumes responsibility and transports the patient to an ED and/or cardiac catheterization lab, followed by admission to an ICU for post–cardiac arrest care. Ideally, all victims of OHCA receive bystander CPR and defibrillation; if not, CPR and defibrillation won't occur until EMS personnel arrive,

making the victim's chance of survival much lower. It is vital to measure and improve dispatch-aided CPR rates.

In contrast, patients with IHCA depend on a system of appropriate early recognition and prevention of cardiac arrest, which is represented by a magnifying glass in the first link. When cardiac arrest occurs, prompt activation of the emergency response system with a response to the cardiac arrest that provides high-quality CPR, early defibrillation, and advanced cardiovascular life support should result in the smooth interaction of a multidisciplinary team of professional providers, including physicians, nurses, respiratory therapists, and others. The last link is recovery and includes considerations for the postarrest needs of the patient and family. The chain metaphor endures: in any resuscitation, the chain is no stronger than its weakest link.

The classic resuscitation Chain of Survival concept linked the community to EMS and EMS to hospitals, with hospital care as the destination.[6] But patients with a cardiac emergency may enter the system of care at any point (Figure 3), including on the street; at home; or in the hospital's ED, inpatient bed, ICU, operating suite, catheterization suite, or imaging department. The system of care must be able to manage cardiac emergencies wherever they occur.

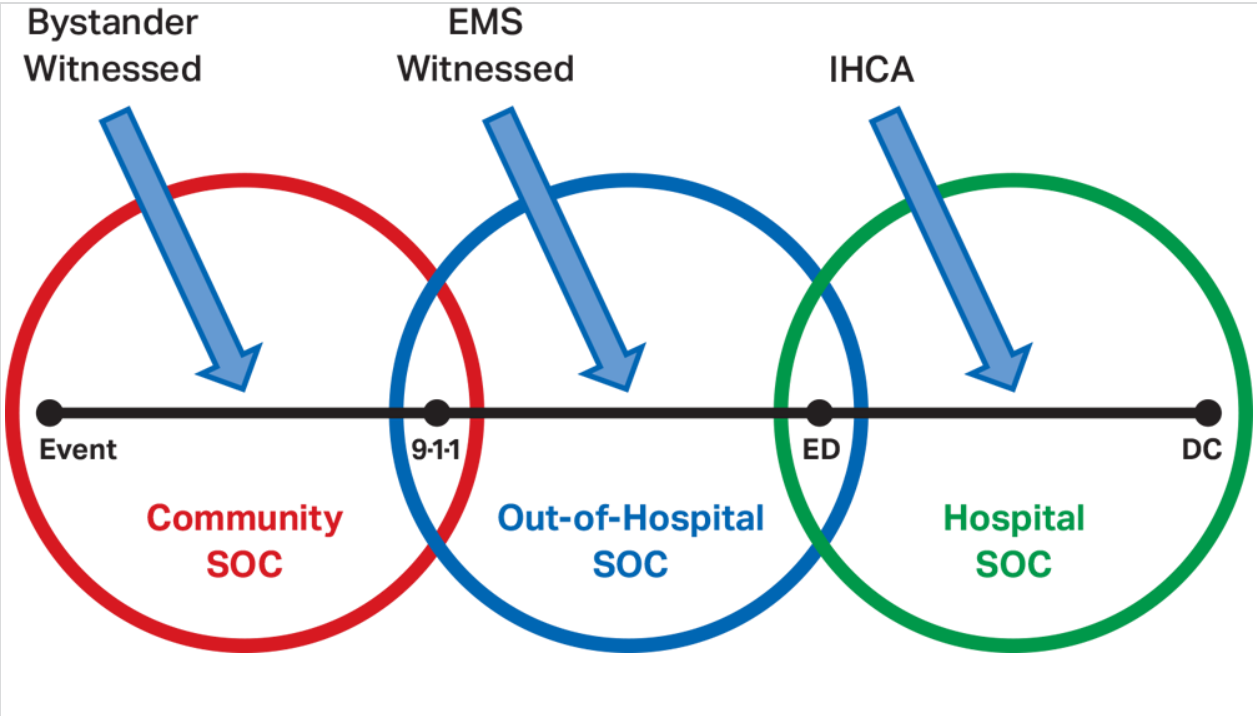


Figure 3. Patient's point of entry.

Abbreviations: DC, discharge; SOC, system of care.

Measurement

Systems that continually work to improve resuscitation outcomes capture and review data related to resuscitation education, processes, and outcomes to identify measures that can lead to better patient care. Quality improvement efforts rely on valid assessments of resuscitation performance and patient outcomes.

Utstein-style guidelines and templates allow for reporting resuscitation outcomes after trauma and drowning.[7] These OHCA guidelines[8] provide guidance for core performance measures, including

- Occurrence of bystander CPR
- Time interval from collapse to defibrillator placement
- Time interval to advanced airway management
- Time interval to first administration of resuscitation medication
- Survival to hospital discharge

These IHCA guidelines[9] provide guidance for core performance measures, including the following:

- Patient demographics
- Patient category (inpatient or outpatient) and illness category
- Details of arrest:
    - –Date/time/location
    - –Witnessed
    - –Resuscitation team activated
    - –Monitored arrest
    - –Chest compression
    - –AED or defibrillator
    - –Initial rhythm
    - –ECPR used
- Postresuscitation details:
    - –TTM
    - –Pyrexia
    - –Coronary angiography
    - –Coronary reperfusion
- Patient outcome:
    - –Date/time/reason CPR stopped
    - –ROSC achieved?
    - –Survival to discharge or to 30 days?

    - –Neurologic outcome
    - –Date/time of death
    - –Organ donation

Physiologic end points are generally considered the best indicators of resuscitation effectiveness, and outside of a hospital setting, $ETCO_2$ is typically available. During CPR, $ETCO_2$ is a relative indicator of cardiac output and can also signal return of spontaneous circulation (ROSC), so this should be used if possible. CPR performance monitors are now widely available,[10] and they provide invaluable real-time feedback on the quality of CPR that rescuers deliver during resuscitation attempts. Other monitors of CPR performance are increasingly available and can provide real-time feedback to healthcare providers. After a resuscitation attempt, these monitors also provide data for debriefing and information for system-wide CPR CQI programs. Without a way to measure CPR and understand their performance, providers cannot improve.

Some CPR performance characteristics are available as immediate feedback while other characteristics are available only after CPR or not at all, given current technology. The ability to receive immediate feedback on the following items will depend on the level of technology available:

- Immediately available feedback
    - –Chest compression rate
    - –Depth
    - –Recoil
- Feedback for review
    - –Chest compression fraction
    - –Preshock, perishock, and postshock pauses
    - –Feedback that cannot be assessed adequately
    - –Ventilation rate
    - –Airway pressure
    - –Tidal volume
    - –Inflation duration
    - –Other physiological end points, if available (ie, $ETCO_2$, intra-arterial blood pressure, cardiac ultrasound)

Current CPR monitoring devices do not always provide optimal feedback. For example, accelerometers do not sense mattress compression, and their rigid algorithms do not realistically prioritize the order of feedback—they cannot measure depth if the provider leans too much, so the devices prioritize feedback to correct leaning before depth. Although some solutions

currently exist, including software (automated algorithms) and hardware (smart backboard, dual accelerometers, reference markers, and others), continued improvements to performance require continued improvements to CPR monitoring.

Benchmarking and Feedback

Systems should review feedback data and compare the information to prior internal performance and similar external systems. Existing registries can help benchmark the data. Examples of these registries include

- Cardiac Arrest Registry to Enhance Survival (CARES) for OHCA
- Get With The Guidelines-Resuscitation program for IHCA

Change

Simply by measuring and benchmarking care, systems can positively influence outcome. However, they must also review and interpret the data to identify areas for improvement, such as

- Increased bystander CPR response rates
- Improved CPR performance
- Shortened time to defibrillation
- Citizen awareness
- Citizen and healthcare professional education and training

Over the past 50 years, rescuers have used the modern-era BLS fundamentals of early recognition and activation, early CPR, and early defibrillation to save hundreds of thousands of lives around the world. However, survival disparities identified years ago persist, so we must continue improving care to fulfill the potential of the Chain of Survival. Fortunately, we have the knowledge and tools—represented by the Chain of Survival—to address many of these care gaps, and future discoveries will offer opportunities to improve rates of survival.

STEMI Systems of Care

The goal of STEMI care is to minimize heart damage and maximize the patient's recovery. The STEMI links (Figure 4) indicate the actions that patients, family members, and healthcare providers can rapidly take to maximize STEMI recovery:

- Recognition and reaction to STEMI warning signs
- EMS dispatch and rapid EMS system transport and prearrival notification to the receiving hospital
- Assessment and diagnosis in the ED (or cath lab)

- Treatment

Figure 4. The STEMI Chain of Survival.

Starting With Dispatch

All dispatchers and EMS providers must train to recognize ACS symptoms along with the potential complications. When authorized by medical control or protocol, dispatchers should tell patients with no history of aspirin allergy or signs of active or recent gastrointestinal (GI) bleeding to chew aspirin (162 to 325 mg) while they wait for EMS providers to arrive.

Activating EMS

Prompt diagnosis and treatment offer the best chance for saving the heart, so healthcare providers must recognize patients with potential ACS and begin evaluation, triage, and management as quickly as possible.

EMS Components

- Obtain prehospital ECGs
- Notify the receiving facility of a patient with possible ST-segment elevation myocardial infarction (STEMI) (“STEMI alert”)
- Activate the cardiac catheterization team to shorten reperfusion time
- Continuously review and improve quality

Hospital-Based Components

- ED protocols
    - –Provide a streamlined cardiac catheterization team process
    - –Admit to the coronary ICU

    - –Provide quality assurance, real-time feedback, and healthcare provider education
- Emergency physician's role
    - –Select the most appropriate reperfusion strategy
    - –Activate the cardiac catheterization team when needed
- Hospital leadership role
    - –Must be involved in the process and committed to supporting rapid access to STEMI reperfusion therapy

Stroke Systems of Care

The goal of stroke care is to minimize brain injury and maximize the patient's recovery. The Stroke Chain of Survival (Figure 5) links the actions that patients, family members, and healthcare providers should take to maximize stroke recovery. These links are

- Rapid recognition of and reaction to stroke warning signs and symptoms
- Rapid use of 9-1-1 and EMS dispatch
- Rapid EMS recognition of stroke, triage, transport, and prehospital notification to the receiving hospital
- Rapid diagnosis and treatment in the hospital

Figure 5. The Stroke Chain of Survival.

Recent clinical trials suggest that all patients eligible for endovascular therapy (EVT) should be considered for that treatment in addition to IV alteplase. Regional stroke systems of care for acute ischemic stroke need to be in place so that eligible patients can be quickly transported from the field per local designation protocols or transferred from non-EVT centers to comprehensive or thrombectomy-capable stroke centers that offer these treatments.

Systematic Approach

For optimal care, healthcare providers use a systematic approach to assess and treat arrest and acutely ill or injured patients. For a patient in respiratory or cardiac arrest, high-performance teams aim to support and

restore effective oxygenation, ventilation, and circulation with return of intact neurologic function. An intermediate goal of resuscitation is ROSC. These teams guide their actions by using the following systematic approaches:

- **Initial assessment** (visualization and scene safety)
- **BLS Assessment**
- **Primary Assessment** (A, B, C, D, and E)
- **Secondary Assessment** (SAMPLE, H's and T's)

Before you approach any patient, rapidly verify scene safety (no threat to the provider). Once you've determined that the scene is safe, use the systematic approach (Figures 6 and 7) to determine the patient's level of consciousness.

- If the patient appears unconscious, use the BLS Assessment for the initial evaluation, and use the Primary and Secondary Assessments for more advanced evaluation and treatment.
- If the patient appears conscious, use the Primary Assessment for your initial evaluation.

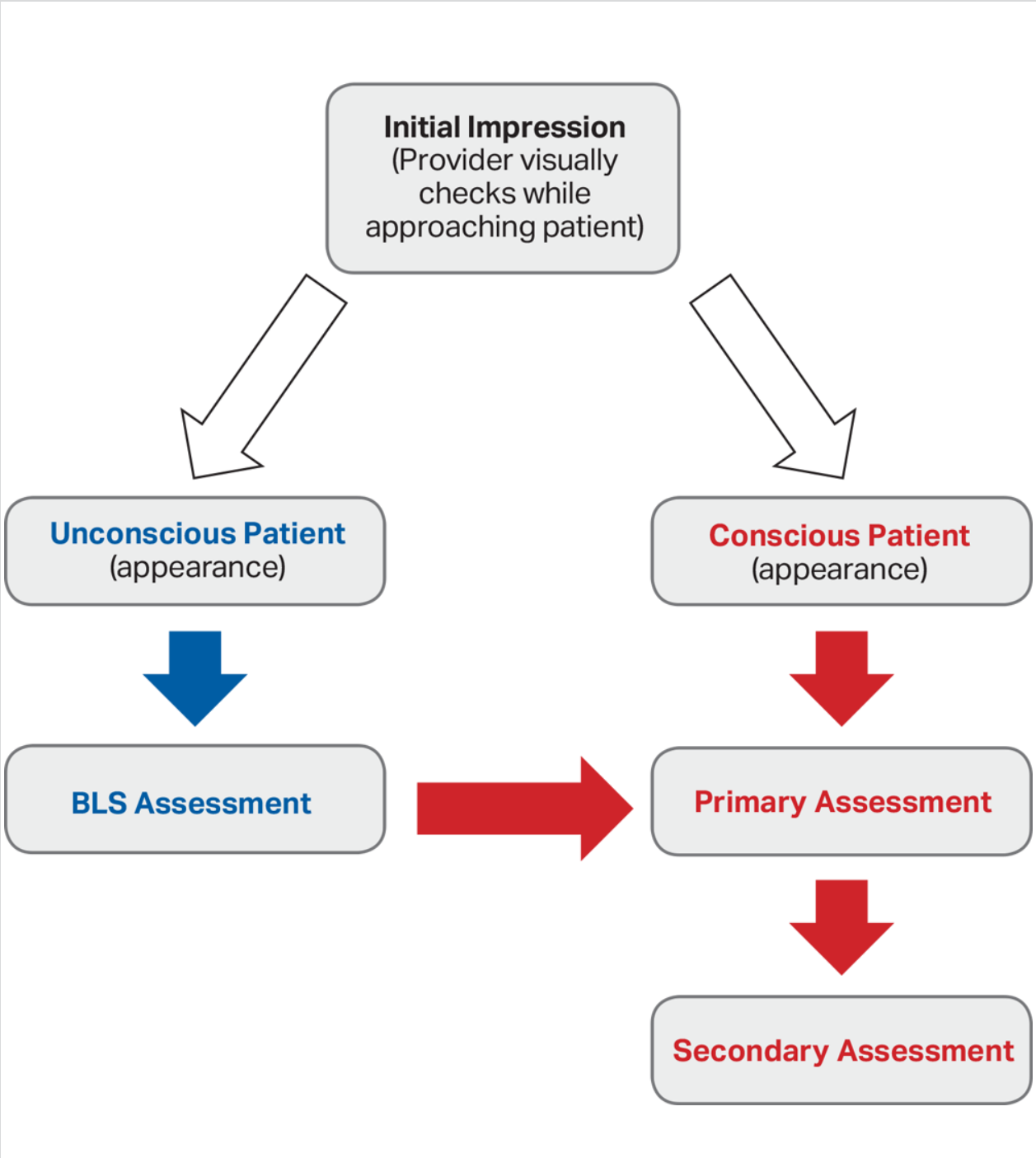


Figure 6. The systematic approach.

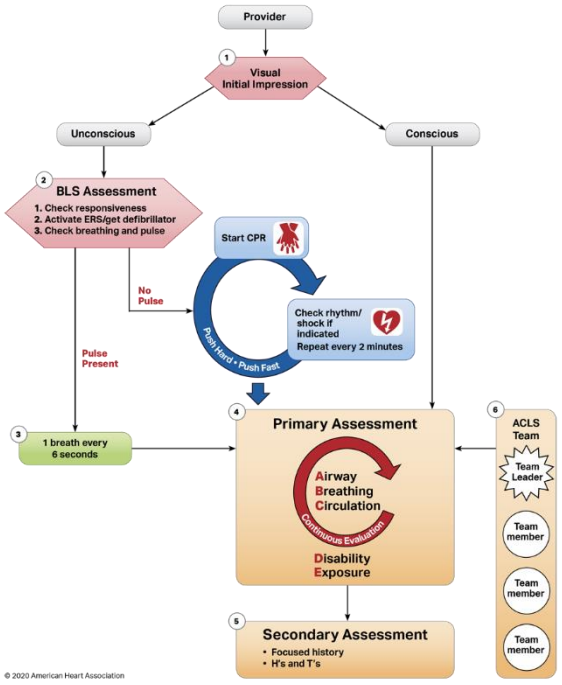


Figure 7. The expanded systematic approach.

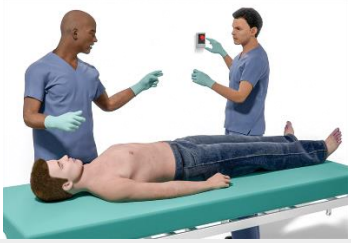
BLS Assessment

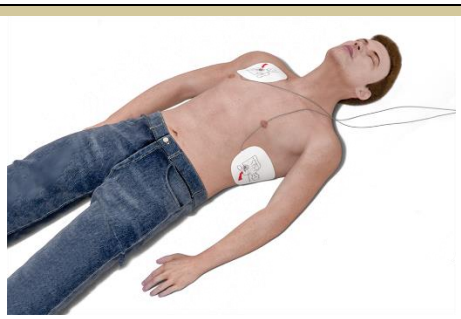
The BLS Assessment is a systematic approach to BLS for trained healthcare providers. This approach stresses **early CPR with basic airway management and defibrillation** but not advanced airway techniques or drug administration. By using the BLS Assessment, any healthcare provider can support or restore effective oxygenation, ventilation, and circulation until the patient achieves ROSC or advanced providers intervene. Performing the BLS Assessment substantially improves a patient's chance of survival and a good neurologic outcome.

Remember to assess first, and then perform the appropriate action.

Although the BLS Assessment requires no advanced equipment, you can use readily available supplies, such as a bag-mask ventilation device if it is available. Whenever possible, place the patient faceup on a firm, flat surface to maximize the effectiveness of chest compressions. Table 2 is an overview of the BLS Assessment, and Figures 8 through 12 illustrate the steps needed during the BLS Assessment.

Table 2. BLS Assessment

Assessment	Assessment technique and action	Supporting image
Check responsiveness.	• Tap and shout, “Are you OK?”	 **Figure 8. Check for responsiveness.**
Shout for nearby help/activate the emergency response system and get the AED/defibrillator.	• Shout for nearby help. • Activate the emergency response system. • Get an AED if one is available, or send someone to activate the emergency response system and get an AED or defibrillator.	 **Figure 9. Shout for nearby help/activate the emergency response system/get an AED.**
Check for breathing and pulse.	• **To check for absent or abnormal breathing** (no breathing or only gasping) **scan the chest for rise and fall** for at least 5 but no more than 10 seconds. • Feel for a pulse for at least 5 but no more than 10 seconds. • Perform the pulse check simultaneously with the breathing	 **Figure 10. Check breathing and pulse simultaneously.**

	check **within 10 seconds** to minimize delaying CPR. • If you find no breathing and no pulse within 10 seconds, start CPR, beginning with chest compressions. • If you find a pulse, start rescue breathing at 1 breath every 6 seconds. Check pulse about every 2 minutes.	 **Figure 11. Check for a carotid pulse.**
Defibrillate.	• If pulse is not felt, check for a shockable rhythm with an AED/defibrillator as soon as it arrives. • Provide shocks as indicated. • Follow each shock immediately with CPR, beginning with compressions.	 **Figure 12. Defibrillation.**

Critical Concepts: High-Quality CPR

To perform high-quality CPR, rescuers should

- *Compress the chest hard and fast at least 2 inches (5 cm) at a rate of 100 to 120/min (30:2 or another advanced protocol that maximizes CCF).*
- *Allow the chest to completely recoil after each compression.*
- *Switch compressors about every 2 minutes or earlier if fatigued—the switch should only take about 5 seconds.*
- *Minimize interruptions in compressions to 10 seconds or less (high CCF).*
- *Avoid excessive ventilation.*

Caution: Chest Compression Depth

Chest compressions are more often too shallow than too deep. However, research suggests that compressing deeper than 2.4 inches (6 cm) in adults may not be optimal for survival from cardiac arrest and may cause injuries. If you have a CPR quality feedback device, target your compression depth from 2 to 2.4 inches (5 to 6 cm).

Tailoring the Response

Single rescuers may tailor the sequence of rescue actions to the most likely cause of arrest. For example, a healthcare provider who sees an adolescent suddenly collapse (eg, after a blow to the chest) can assume that the patient has had a sudden cardiac arrest. In this case, the rescuer should activate the emergency response system via mobile device, get an AED if nearby, return to the patient to attach the AED, and then provide CPR. However, if the rescuer believes hypoxia caused the cardiac arrest (such as in a drowning victim), he or she may perform about 2 minutes of CPR, including breaths, before activating the emergency response system.

Critical Concepts: Minimizing Interruptions in Chest Compressions

When you stop chest compressions, blood flow to the brain and heart stops, so you must minimize the number of interruptions. Additionally, try to limit the duration of interruptions for defibrillation or rhythm analysis to no longer than 10 seconds unless you are moving the patient from a dangerous environment. Refer to Figure 13.

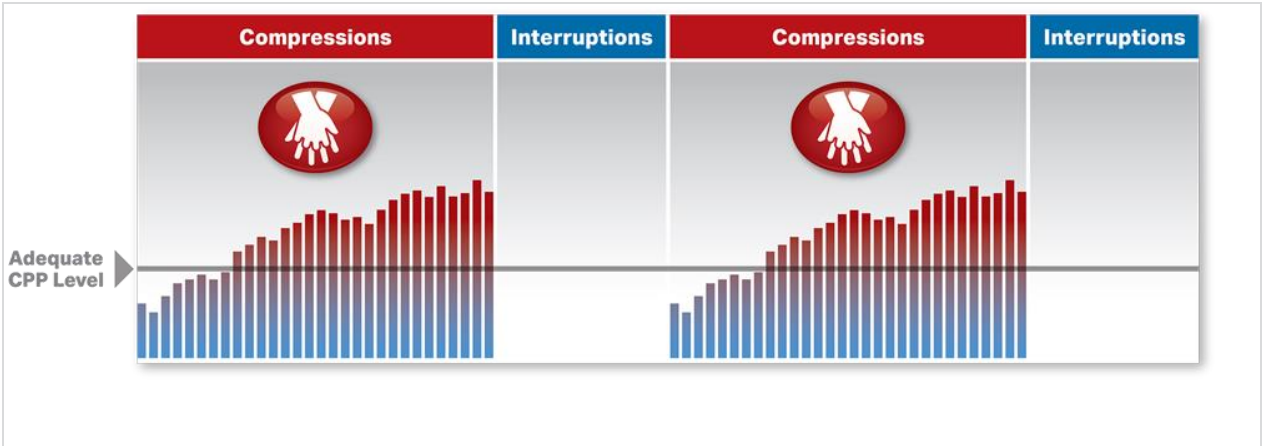


Figure 13. Relationship of quality CPR to coronary perfusion pressure demonstrating the need to minimize interruptions in compressions.

Avoid

- Prolonged rhythm analysis
- Frequent or inappropriate pulse checks
- Prolonged ventilation
- Unnecessary movement of the patient

Coronary perfusion pressure (CPP) is aortic relaxation (diastolic) pressure minus right atrial relaxation (diastolic) pressure. During CPR, CPP correlates with both myocardial blood flow and ROSC. In 1 human study, ROSC did not occur unless a CPP of 15 mm Hg or greater was achieved during CPR. Because $ETCO_2$ is related to cardiac output with chest compressions during cardiac arrest, ROSC is similarly unlikely with a persistent $ETCO_2$ of less than 10 mm Hg.

Starting CPR When You Are Not Sure About a Pulse

If you aren't sure whether you feel a pulse, start CPR. Unnecessary compressions are better than no compressions at all in a patient with no pulse, and delayed CPR reduces the chance of survival.

Agonal Gasps

You may see agonal gasps in the first minutes after sudden cardiac arrest, but agonal gasps are not normal breathing. They are a sign of cardiac arrest. A patient who gasps may appear to be drawing air in very quickly. The mouth may be open, and the jaw, head, or neck may move with gasps. These gasps may appear forceful or weak, and some time may pass between them because they usually happen at a slow, irregular rate. An agonal gasp may sound like a snort, snore, or groan. If you identify agonal gasps, begin chest compressions without delay.

Caution: Agonal Gasps

- *Agonal gasps may be present in the first minutes after sudden cardiac arrest.*
- *Agonal gasps are not normal breathing.*

Table 3. Primary Assessment

Assessment	Action

Airway
• Is the patient's airway patent?
• Is an advanced airway indicated?
• Have you confirmed proper placement of the airway device?
• Is the tube secured, and are you reconfirming placement frequently and with every transition?
	• Maintain an open airway in unconscious patients by using a head tilt–chin lift, an oropharyngeal airway, or a nasopharyngeal airway.
	• Use advanced airway management if needed (eg, laryngeal mask airway, laryngeal tube, endotracheal tube).
	  ○ –Weigh the benefits of placing an advanced airway against the adverse effects of interrupting chest compressions. If bag-mask ventilation is adequate, you may defer inserting an advanced airway until the patient does not respond to initial CPR and defibrillation or until ROSC. Advanced airway devices such as a laryngeal mask airway, a laryngeal tube, or an esophageal-tracheal tube can be placed while chest compressions continue.
	  ○ –If using advanced airway devices:
	  ▪ Confirm the proper integration of CPR and ventilation
	  ▪ Confirm the proper placement of advanced airway devices by physical examination and quantitative waveform capnography
	  ▪ Secure the device to prevent dislodgment
	  ▪ Monitor airway placement, effectiveness of CPR, and ROSC with continuous quantitative waveform capnography

Breathing
• Are ventilation and oxygenation adequate?
• Are quantitative waveform capnography
	• Give supplemental oxygen when indicated.
	  ○ –For cardiac arrest patients, administer 100% oxygen.
	  ○ –For others, adjust the oxygen administration to achieve oxygen saturation of 95% to 98% by pulse oximetry (90% for ACS and 92% to 98% for post–cardiac arrest care).

and oxyhemoglobin saturation monitored?	• Monitor the adequacy of ventilation and oxygenation by ◦ –Clinical criteria (chest rise and cyanosis) ◦ –Quantitative waveform capnography ◦ –Oxygen saturation ◦ –Avoid excessive ventilation
Circulation • Are chest compressions effective? • What is the cardiac rhythm? • Is defibrillation or cardioversion indicated? • Has intravenous (IV)/intraosseous (IO) access been established? • Is ROSC present? • Is the patient with a pulse unstable? • Are medications needed for rhythm or blood pressure? • Does the patient need volume (fluid) for resuscitation?	• Monitor CPR quality. ◦ –Quantitative waveform capnography (if the partial pressure of CO_2 in exhaled air at the end of the exhalation phase, or $PETCO_2$, is less than 10 mm Hg, attempt to improve CPR quality). Waveform capnography should be as high as possible with improved CPR quality. Continuous quantitative waveform capnography provides an indirect measure of cardiac output during chest compressions because the amount of carbon dioxide exhaled is associated with the amount of blood that passes through the lungs. An $ETCO_2$ less than 10 mm Hg during chest compressions rarely results in ROSC. ◦ –A sudden increase in $ETCO_2$ to more than 25 mm Hg may indicate ROSC. ◦ –Intra-arterial pressure (if relaxation phase [diastolic] pressure is less than 20 mm Hg, attempt to improve CPR quality). Inter-arterial pressure should be as high as possible with improved CPR quality. If intra-arterial pressure monitoring is available, strive to optimize blood pressure. Relaxation phase (diastolic) pressures less than 20 during chest compressions rarely results in ROSC. • Attach monitor/defibrillator for arrhythmias or cardiac arrest rhythms (eg, VF, pVT, asystole, PEA). • Provide defibrillation/cardioversion. • Obtain IV/IO access. • Give appropriate drugs to manage rhythm and blood pressure. • Give IV/IO fluids if needed.

	• Check glucose and temperature. • Check perfusion issues.
Disability	• Check for neurologic function. • Quickly assess for responsiveness, levels of consciousness, and pupil dilation. • AVPU: Alert, Voice, Painful, Unresponsive
Exposure	• Remove clothing to perform a physical examination. • Look for obvious signs of trauma, bleeding, burns, unusual markings, or medical alert bracelets.

The gasp may sound like a snort, snore, or groan. Gasping is a sign of cardiac arrest.

Primary Assessment

In the Primary Assessment, you continue to assess the patient and perform appropriate actions until the patient is transferred to the next level of care. Members of a high-performance team often perform assessments and actions in ACLS simultaneously.

For unconscious patients in arrest (cardiac or respiratory), complete the BLS Assessment before the Primary Assessment. For conscious patients who may need more advanced assessment and management, conduct the Primary Assessment first. Table 3 provides an overview of the Primary Assessment.

Remember to assess first, and then perform the appropriate action.

Secondary Assessment

The Secondary Assessment involves the differential diagnosis, including a focused medical history and searching for and treating underlying causes (H's and T's). Gather a focused history of the patient, if possible. Ask specific questions related to the patient's presentation.

SAMPLE

Consider using the memory aid SAMPLE:

- **S**igns and symptoms
    - –Breathing difficulty
    - –Tachypnea, tachycardia
    - –Fever, headache

    - –Abdominal pain
    - –Bleeding
- **A**llergies
    - –Medications, foods, latex, etc
    - –Associated reactions
- **M**edications (including the last dose taken)
    - –Patient medications, including over-the-counter, vitamins, inhalers, and herbal supplements
    - –Last dose and time of recent medications
    - –Medications that can be found in the patient's home
- **P**ast medical history (especially relating to the current illness)
    - –Health history (eg, previous illnesses, hospitalizations)
    - –Family health history (in cases of ACS or stroke)
    - –Significant underlying medical problems
    - –Past surgeries
    - –Immunization status
- **L**ast meal consumed
    - –Time and nature of last intake of liquid or food
- **E**vents
    - –Events leading to current illness or injury (eg, onset sudden or gradual, type of injury)
    - –Hazards at scene
    - –Treatment during interval from onset of disease or injury until evaluation
    - –Estimated time of onset (if out-of-hospital onset)

The answers to these questions can help you quickly identify likely or suspected diagnoses. Look for and treat the underlying cause by considering the H's and T's to ensure that you are not overlooking common possibilities. The H's and T's create a guide for possible diagnoses and interventions for your patient.

H's and T's

The H's and T's are a memory aid for potential reversible causes of cardiac arrest and emergency cardiopulmonary conditions. The ACLS cases provide details on these components:

H's

- Hypovolemia
- Hypoxia

- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia

T's

- Tension pneumothorax
- Tamponade (cardiac)
- Toxins
- Thrombosis (pulmonary)
- Thrombosis (coronary)

Critical Concepts: Common Underlying Causes of PEA

- *Hypovolemia and hypoxia are the 2 most common underlying and potentially reversible causes of PEA.*
- *Look for evidence of these problems as you assess the patient and treat immediately.*

Diagnosing and Treating Underlying Causes

Patients in cardiac arrest (VF/pVT/asystole/PEA) need rapid assessment and management to determine if an underlying, potentially reversible problem caused the arrest. If you can quickly identify a specific condition, you may achieve ROSC. Identifying the underlying cause is crucial in cases of cardiac arrest. Addressing the underlying cause will provide the best chance for a successful resuscitation. Ultrasound may help with identifying the underlying cause quickly and can also provide information to help determine the next step for treatment. Paying attention to the patient's response to interventions may also help you narrow the differential diagnosis.

To search for the underlying cause, do the following:

- Consider the underlying causes by recalling the H's and T's
- Analyze the ECG for clues to the underlying cause
- Recognize hypovolemia
- Recognize drug overdose/poisonings

Hypovolemia

Hypovolemia, a common cause of PEA, initially produces the classic physiologic response of a rapid, narrow-complex tachycardia (sinus tachycardia) and typically increases diastolic and decreases systolic

pressures. As loss of blood volume continues, blood pressure drops, eventually becoming undetectable, but the narrow QRS complexes and rapid rate continue (ie, PEA).

Consider hypovolemia as a cause of hypotension, which can deteriorate to PEA. Providing prompt treatment can reverse the pulseless state by rapidly correcting the hypovolemia. Common nontraumatic causes of hypovolemia include occult internal hemorrhage and severe dehydration. Consider volume infusion for PEA associated with a narrow-complex tachycardia.

Cardiac and Pulmonary Conditions

ACS that involve a large amount of heart muscle can present as PEA, VF, pVT, or asystole. That is, occlusion of the left main or proximal left anterior descending coronary artery can present with cardiogenic shock rapidly progressing to cardiac arrest and PEA. However, in patients with cardiac arrest and without known pulmonary embolism (PE) or suspected PE or STEMI, giving routine fibrinolytic treatment during CPR shows no benefit and is not recommended.

Massive or saddle PE obstructs flow to the pulmonary vasculature and causes acute right heart failure. In patients with cardiac arrest due to presumed or known PE, it is reasonable to administer fibrinolytics.

Pericardial tamponade may be reversible with pericardiocentesis, and during periarrest, volume infusion may help while definitive therapy is initiated. Once you recognize tension pneumothorax, you should effectively treat it with needle decompression and chest tube insertion.

You cannot treat cardiac tamponade, tension pneumothorax, and massive PE unless you recognize them. A skilled provider can perform bedside ultrasound to help rapidly identify tamponade, pneumothorax, and echocardiographic evidence of PE.

Drug Overdoses or Toxic Exposures

Certain drug overdoses and toxic exposures may lead to peripheral vascular dilatation and/or myocardial dysfunction with resultant hypotension and cardiovascular collapse. Treat poisoned patients aggressively because the toxic effects may progress rapidly, but during this time, the myocardial dysfunction and arrhythmias may be reversible.

Treatments that can provide support include

- Prolonged basic CPR in special resuscitation situations (such as accidental hypothermia)
- Extracorporeal CPR
- Intra-aortic balloon pump therapy
- Renal dialysis
- Intravenous lipid emulsion for lipid-soluble toxins
- Specific drug antidotes (digoxin immune Fab, glucagon, bicarbonate)
- Transcutaneous pacing
- Correction of severe electrolyte disturbances (potassium, magnesium, calcium, acidosis)
- Specific adjunctive agents

Remember, if the patient shows signs of ROSC, begin post–cardiac arrest care.

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Part 2: Preventing Arrest

Recognition: Signs of Clinical Deterioration

Rapid Response

Often, nurses, physicians, or family members who are concerned that the patient is deteriorating will activate the rapid response system in the hospital. Some rapid response systems weigh, combine, and score specific physiologic criteria to determine when to act. The following list gives examples of such criteria for adult patients:

- Airway compromise
- Respiratory rate less than 6/min or more than 30/min
- Heart rate less than 40/min or greater than 140/min
- Systolic blood pressure (SBP) less than 90 mm Hg
- Symptomatic hypertension
- Unexpected decrease in level of consciousness
- Unexplained agitation
- Seizure
- Significant decrease in urine output
- Subjective concern about the patient

The wide variability in incidence and location of IHCA suggests potential areas for standardizing quality and preventing some cardiac arrests. More than half of IHCAs result from respiratory failure or hypovolemic shock, and changes in physiology such as tachypnea, tachycardia, and hypotension foreshadow most of these events. Therefore, IHCA often represents the progression of physiologic instability and a failure to quickly identify and stabilize the patient. This scenario is more common on the general wards—outside of critical care and procedural areas—where patient-to-nurse ratios are higher and monitoring of patients is less intense. In this setting, intermittent manual vital sign monitoring with less frequent direct observation by clinicians may increase the likelihood of delayed recognition.

Over the past decade, hospitals in several countries have designed rapid response systems to identify and treat early clinical deterioration in patients, improving patient outcomes through critical care expertise. The rapid response system has several components:

- Event detection and response-triggering arm
- A planned response arm, such as an RRT or MET

- Quality monitoring
- Administrative support

RRTs and METs

Hospitals established RRTs or METs to provide early intervention in patients whose conditions are deteriorating, with the goal of preventing IHCA.[1,2] These teams can include physicians, nurses, and respiratory therapists who have the critical care experience and skills to intervene in life-threatening situations. They are typically asked to see any patient who is identified as deteriorating, no matter who reports that deterioration: staff members, the family, or the patient. These teams bring monitoring and resuscitation equipment to perform a rapid patient assessment, and they initiate appropriate treatment and drug therapies to reverse physiologic deterioration and prevent poor outcomes, much like EMS services intervening when called in the prehospital setting.

Published Studies

Although the ideal composition of METs or RRTs is not known, many published before-and-after studies of METs or RRTs have reported a drop in the rate of cardiac arrests after these teams intervene.[3,4] Although some studies have not reported a decrease in overall mortality with the introduction of these teams,[5] there may be other benefits, such as improved end-of-life care, because these teams may initiate discussions with patients and families before cardiac arrest, preventing unwanted interventions in critically ill patients.

Additional documented benefits of these systems include the following:

- Decreased unplanned emergency transfers to the ICU
- Decreased ICU and total hospital length of stay
- Reduced postoperative morbidity and mortality rates
- Improved rates of survival from cardiac arrest

Implementing a Rapid Response System

Implementing any type of rapid response system requires a significant cultural change in most hospitals. Those who design and manage the system must pay particular attention to issues that may prevent the hospital from using the system effectively. Examples of such issues are insufficient resources, poor education, fear of calling the team, fear of losing control over patient care, and resistance from team members.

Implementing a rapid response system requires ongoing education, impeccable data collection and review, and feedback. Developing and maintaining these programs requires a long-term cultural and financial commitment from the hospital administration. Hospital administrators and healthcare professionals need to reorient their approach to emergency medical events and develop a culture of patient safety with a primary goal of decreasing morbidity and mortality.

Acute Coronary Syndromes

The Acute Coronary Syndromes Algorithm (Figure 16) will help guide your clinical strategy when patients have signs and symptoms of ACS, including possible acute myocardial infarction (AMI). To apply this algorithm effectively, you must have the basic knowledge to assess and stabilize patients with ACS.

The initial 12-lead ECG is used to classify patients into 1 of 2 ECG categories of myocardial infarction, with different strategies of care and management needs. These 2 ECG categories are outlined in the Acute Coronary Syndromes Algorithm:

- ST-segment elevation (STEMI)
- Non-ST–segment elevation ACS (NSTE-ACS)
    - –ST-segment depression, T-wave inversion, transient ST-segment elevation
    - –Nondiagnostic or normal ECG

The following focuses on the first category: STEMI, with time-sensitive reperfusion strategies.

You will

- Identify, assess, and triage acute ischemic chest discomfort
- Provide initial treatment of possible ACS
- Emphasize early reperfusion of the patient with ACS/STEMI

Goals for ACS Patients

The primary goals are as follows:

- Prevention of major adverse cardiovascular events such as death, nonfatal MI, and the need for urgent postinfarction revascularization
- Identification of patients with STEMI and triage for early reperfusion therapy

- Relief of ischemic chest discomfort
- Treatment of acute, life-threatening complications of ACS, such as VF/pVT, unstable bradycardia, ventricular wall rupture, papillary muscle rupture, decompensated shock, and other unstable tachycardias

Reperfusion therapy opens an obstructed coronary artery with either mechanical means or drugs. PCI, performed in the cardiac catheterization laboratory after coronary angiography, allows for balloon dilation or stent placement—or both—for an obstructed coronary artery.

Rhythms for ACS

Sudden cardiac death, ventricular tachycardias, and hypotensive bradycardia may occur with acute ischemia. Anticipate these rhythms, and be prepared for immediate interventions, including defibrillation or cardioversion as well as administration of drugs or pacing for stable bradycardias.

Drugs for ACS

Drug therapy and treatment strategies continue to evolve rapidly in the field of ACS, so be sure to keep up with important changes.

To treat ACS, you'll initially use these drugs to relieve ischemic discomfort, dissolve clots, and inhibit thrombin and platelets:

- Oxygen
- Aspirin
- Nitroglycerin
- Opiates (eg, morphine)
- Fibrinolytic therapy (overview)
- Heparin (unfractionated, low-molecular-weight)

Additional adjunctive agents that will not be discussed in this course include the following:

- β-Blockers
- Bivalirudin
- $P2Y_{12}$ inhibitors (clopidogrel, prasugrel, ticagrelor)
- Angiotensin-converting enzyme inhibitors
- HMG-CoA reductase inhibitors (statin therapy)
- Glycoprotein IIb/IIIa inhibitors

STEMI Chain of Survival

The STEMI Chain of Survival (Figure 14) is similar to the Chain of Survival for sudden cardiac arrest. Its links indicate the actions that patients, family members, and healthcare providers can rapidly take to maximize STEMI recovery:

- Recognition and reaction to STEMI warning signs
- EMS dispatch and rapid EMS system transport and prearrival notification to the receiving hospital
- Assessment and diagnosis in the ED (or cath lab)
- Treatment

Figure 14. The STEMI Chain of Survival.

OHCA Response

Half of ACS deaths occur before the patient reaches the hospital, with VF or pVT as the precipitating rhythm in the majority of cases. VF is most likely to develop during the first 4 hours after onset of symptoms, so communities should develop EMS and prehospital programs to respond quickly to ACS. Such programs should focus on

- Recognizing symptoms of ACS
- Activating the EMS system, with EMS providing prehospital notification
- Providing early CPR if cardiac arrest occurs
- Providing early defibrillation with AEDs available through public-access defibrillation programs and first responders
- Providing a coordinated system of care among the EMS system, the ED, cath lab, and cardiac specialists

Pathophysiology of ACS

Patients with coronary atherosclerosis may develop a spectrum of clinical syndromes that represent varying degrees of coronary artery occlusion. These syndromes include NSTE-ACS and STEMI. Sudden cardiac death may occur with any of these syndromes. Figure 15 illustrates the pathophysiology of ACS.

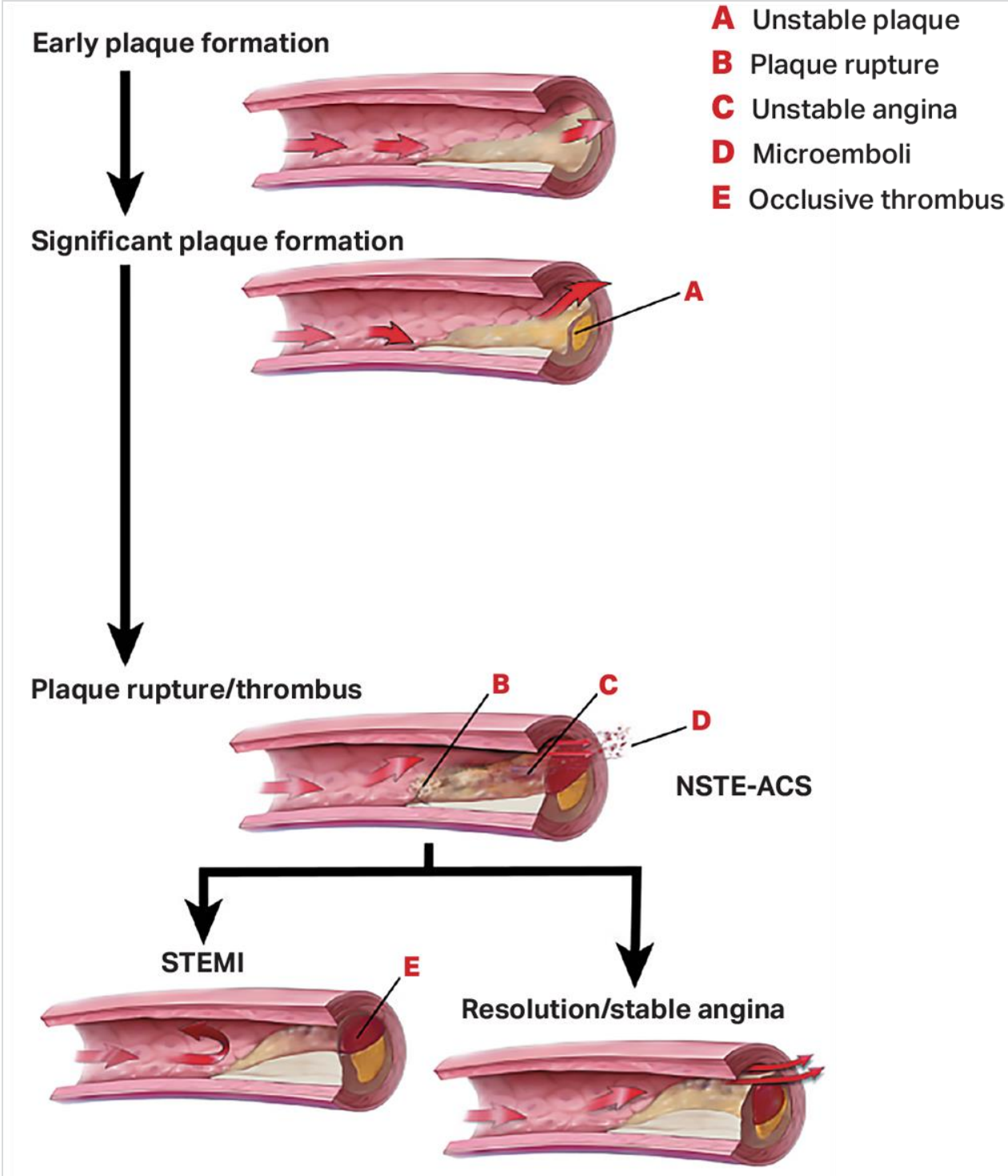


Figure 15. Pathophysiology of ACS.

Managing ACS: The Acute Coronary Syndromes Algorithm

The Acute Coronary Syndromes Algorithm (Figure 16) outlines the steps for assessing and managing a patient who has symptoms suggestive of ischemia or infarction (ACS symptoms, Step 1). The EMS assessment and care and hospital preparation should include (Step 2) the following:

- Assess ABC (airway, breathing, circulation). Be prepared to provide CPR and defibrillation.
- Administer aspirin and consider oxygen, nitroglycerin, and morphine if needed.
- Obtain a 12-lead ECG. If there is ST elevation, notify the receiving hospital with a transmission or interpretation; note time of onset and first medical contact.
- Provide prehospital notification; on arrival, transport to ED/cath lab per protocol.
- The notified hospital should mobilize hospital resources to respond to STEMI and activate STEMI alert.
- If considering prehospital fibrinolysis, use a fibrinolytic checklist.
- If out-of-hospital providers cannot complete these initial steps before the patient arrives at the hospital, the ED provider should do so.

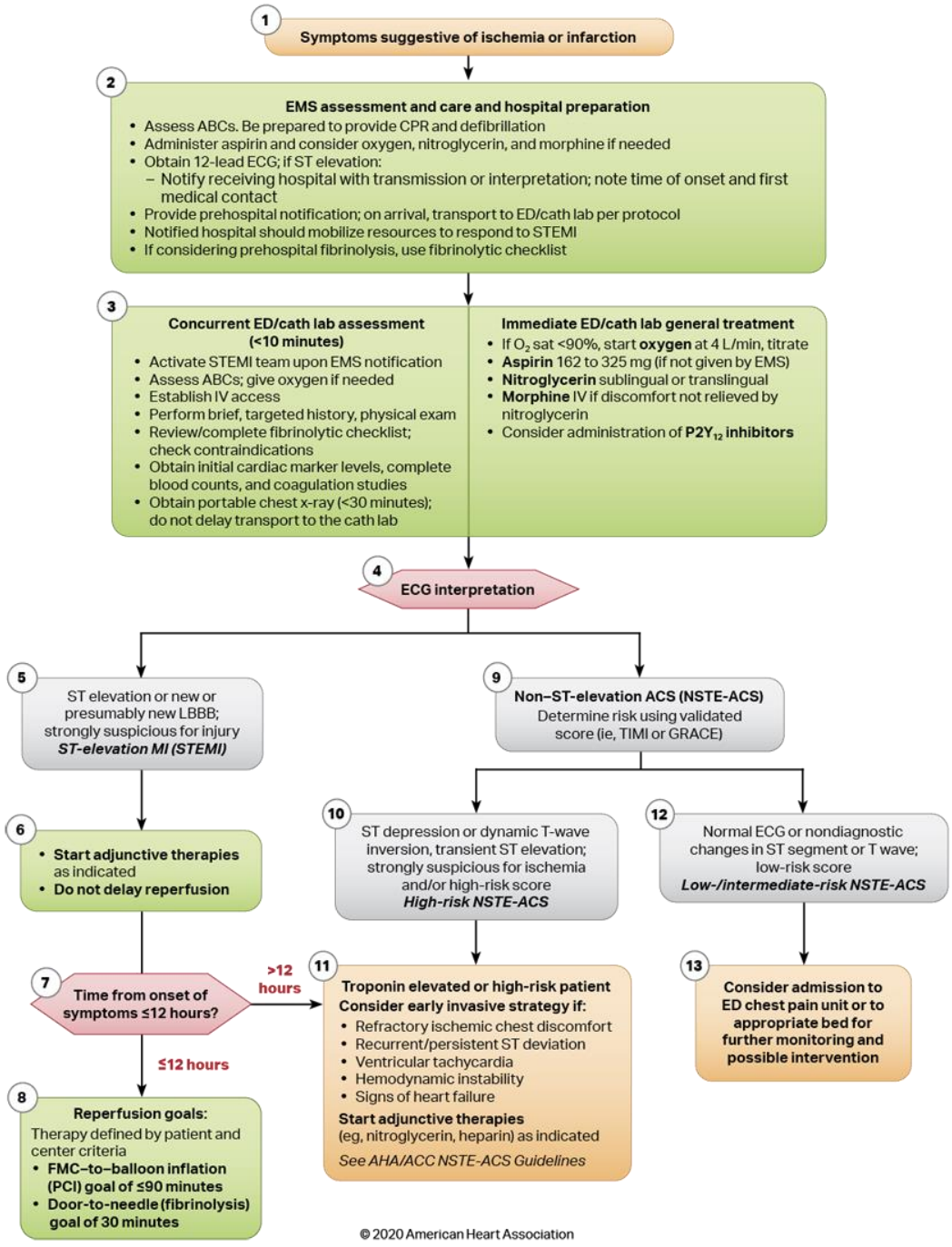


Figure 16. Acute Coronary Syndromes Algorithm.

Subsequent treatment may begin with EMS providers, based on local protocols, or it may begin when the patient arrives at the hospital. Concurrent ED or cath lab assessment (Step 3) should occur in less than 10 minutes and include

- Activate STEMI team upon EMS notification.
- Assess ABCs; give oxygen if needed.
- Establish IV access.
- Perform a brief, targeted history and a physical exam.
- Review and complete the fibrinolytic checklist; check contraindications.
- Obtain initial cardiac marker levels, complete blood counts, and coagulation studies.
- Obtain a portable chest x-ray (in less than 30 minutes); do not delay transport to the cath lab.

The immediate ED/cath lab general treatment (Step 3) includes the following:

- If oxygen saturation is less than 90%: Start oxygen at 4 L/min and titrate
- Aspirin 162 to 325 mg (if not given by EMS)
- Nitroglycerin sublingual or translingual
- Morphine IV if discomfort not relieved by nitroglycerin
- Consider administration of $P2Y_{12}$ inhibitors

Treatment recommendations are specific to each group:

- STEMI
- NSTE-ACS
    - –High-risk NSTE-ACS
    - –Low- to intermediate-risk NSTE-ACS

ACS management focuses on early reperfusion of the STEMI patient, emphasizing initial care and rapid triage for reperfusion therapy.

Important Considerations

The ACS Algorithm (Figure 16) provides general guidelines, based on patient symptoms and the 12-lead ECG, for the initial triage of patients. Healthcare personnel often obtain serial cardiac markers (CK-MB, cardiac troponins) in patients that allow additional risk stratification and treatment recommendations. Two important points for STEMI need emphasis:

- The ECG is central to the initial risk and treatment stratification process.

- For STEMI patients, you do not need evidence of elevated cardiac markers to decide to administer fibrinolytic therapy or perform diagnostic coronary angiography with coronary intervention (angioplasty/stenting).

Application of the ACS Algorithm

The steps in the algorithm guide assessment and treatment as follows:

- Step 1 is to identify symptoms suggestive of ischemia or infarction, such as chest or shoulder pain, dyspnea, or nausea.
- In Step 2, EMS assesses the patient and provides care, transport, and hospital prearrival notification. Prehospital ECG capture and evaluation is important.
- In Step 3, the ED/cath lab immediately assesses and treats the patient in less than 10 minutes. Next, the ED/cath lab provides immediate general treatment including oxygen and administration of drugs.
- After interpreting the ECG in Step 4, use Steps 5 and 9 to classify patients according to ST-segment analysis.
- If your analysis points to STEMI, use Steps 5 through 8 to treat the patient.

Symptoms Suggestive of Ischemia or Infarction

You should know how to identify symptoms that suggest cardiac ischemia (Step 1). Promptly conduct a targeted evaluation of every patient whose initial symptoms suggest possible ACS.

The most common symptom of myocardial ischemia and infarction is retrosternal chest discomfort. The patient may perceive this discomfort more as pressure or tightness than as actual pain.

Chest discomfort is the major symptom in most patients (both men and women) with ACS, but patients frequently deny or misinterpret this and other symptoms. The elderly, women, diabetic patients, and hypertensive patients are most likely to delay, in part because they are more likely to have atypical symptoms or presentations. The decision to call for an ambulance may also reduce delays in care. Other factors that can affect the interval between symptom onset and presentation to hospital include time of day, location (eg, work or home), and presence of a family member.

Symptoms that suggest ACS may also include

- Uncomfortable pressure, fullness, squeezing, or pain in the center of the chest lasting several minutes (usually more than a few minutes)
- Chest discomfort spreading to the shoulders, neck, one or both arms, or jaw
- Chest discomfort spreading into the back or between the shoulder blades
- Light-headedness, dizziness, fainting, syncope, sweating, nausea, or vomiting
- Unexplained, sudden shortness of breath, which may occur with or without chest discomfort
- Less commonly, the discomfort occurs in the epigastrium and is described as indigestion.

These symptoms may also suggest other life-threatening conditions, including aortic dissection, acute PE, acute pericardial effusion with tamponade, and tension pneumothorax.

Starting With Dispatch

All dispatchers and EMS providers must train to recognize ACS symptoms along with the potential complications. When authorized by medical control or protocol, dispatchers should tell patients with no history of aspirin allergy or signs of active or recent gastrointestinal (GI) bleeding to chew aspirin (162 to 325 mg) while they wait for EMS providers to arrive.

EMS Assessment, Care, and Hospital Preparation

Step 2 in the algorithm outlines EMS assessment, care, and hospital preparation. EMS responders may perform the following assessments and actions as they stabilize, triage, and transport the patient to an appropriate facility:

- Assess ABCs. Be prepared to provide CPR and defibrillation.
- Administer aspirin and consider oxygen, nitroglycerin, and morphine if needed.
- Obtain a 12-lead ECG. If there is ST elevation, notify the receiving hospital with a transmission or an interpretation; note time of onset and first medical contact.
- Provide prehospital notification; on arrival, transport to ED/cath lab per protocol.
- The notified hospital should mobilize hospital resources to respond to STEMI and activate STEMI alert.

- If considering prehospital fibrinolysis, use fibrinolytic checklist.

Assessing ABCs

Assessing ABCs includes

- Monitoring vital signs and cardiac rhythm
- Being prepared to provide CPR
- Using a defibrillator if needed

Administering Oxygen and Drugs

You should be familiar with the actions, indications, cautions, and treatment of side effects.

Oxygen

EMS providers should administer **oxygen** if the patient is dyspneic or hypoxemic, has obvious signs of heart failure, or has an arterial oxygen saturation that is less than 90% or unknown. Providers should adjust oxygen therapy to a noninvasively monitored oxyhemoglobin saturation 90% or greater. The usefulness of supplemental oxygen therapy has not been established in normoxic patients with suspected or confirmed ACS, so providers may consider withholding it in these patients.

Aspirin (Acetylsalicylic Acid)

A dose of 162 to 325 mg of non–enteric-coated or chewed aspirin causes immediate and near-total inhibition of thromboxane A_2 production by inhibiting platelet cyclooxygenase (COX-1). Platelets are one of the principal and earliest participants in thrombus formation. This rapid inhibition also reduces coronary reocclusion and other recurrent events independently and after fibrinolytic therapy.

If the patient has not taken **aspirin** and has no history of true aspirin allergy and no evidence of recent GI bleeding, administer aspirin (162 to 325 mg) to chew. In the initial hours of an ACS, aspirin is absorbed better when chewed than when swallowed, particularly if the patient has received morphine. Use rectal aspirin suppositories (300 mg) for patients with nausea, vomiting, active peptic ulcer disease, or other disorders of the upper GI tract. Aspirin is associated with a reduction in mortality for patients with ACS.

Nitroglycerin (Glyceryl Trinitrate)

Nitroglycerin effectively reduces ischemic chest discomfort, and it has beneficial hemodynamic effects. The physiologic effects of nitrates reduce left ventricular (LV) and right ventricular (RV) preload through peripheral arterial and venous dilation.

Give the patient 1 sublingual nitroglycerin tablet (or translingual dose) every 3 to 5 minutes for ongoing symptoms if permitted by medical control and no contraindications exist. You may repeat the dose twice (total of 3 doses). Administer nitroglycerin only if the patient remains hemodynamically stable: SBP is greater than 90 mm Hg or no lower than 30 mm Hg below baseline (if known) and the heart rate is 50 to 100/min.

Nitroglycerin is a venodilator; use it cautiously or not at all in patients with inadequate ventricular preload. These situations include

- **Inferior wall MI and RV infarction.** RV infarction may complicate an inferior wall MI. Patients with acute RV infarction depend on RV filling pressures to maintain cardiac output and blood pressure. If you cannot rule out RV infarction, use caution in administering nitrates to patients with inferior STEMI. If you confirm RV infarction by right-sided precordial leads, or if an experienced provider confirms it through clinical findings, then nitroglycerin and other vasodilators (morphine) or volume-depleting drugs (diuretics) are contraindicated as well.
- **Hypotension, bradycardia, or tachycardia.** Avoid using nitroglycerin in patients with hypotension (SBP less than 90 mm Hg), marked bradycardia (heart rate less than 50/min), or marked tachycardia.
- **Recent phosphodiesterase inhibitor use.** Avoid using nitroglycerin if you suspect or know that the patient has taken sildenafil or vardenafil within the previous 24 hours or tadalafil within 48 hours. These agents are generally used for erectile dysfunction or in cases of pulmonary hypertension, and in combination with nitrates, they may cause severe hypotension refractory to vasopressor agents.

There appears to be no association between nitroglycerin therapy and survival in patients with ACS.

Opiates (eg, Morphine)

Consider administering morphine for severe chest discomfort that does not respond to sublingual or translingual nitroglycerin if authorized by protocol

or medical control. Morphine is indicated in STEMI when chest discomfort does not respond to nitrates. Use morphine with caution in NSTE-ACS because of an association with increased mortality. In addition, morphine may mask symptoms of myocardial ischemia and decrease absorption of important orally administered drugs, such as antiplatelets ($P2Y_{12}$ inhibitors). Currently there are no data to suggest an association between morphine and survival advantages in patients with ACS.

Morphine may be used to manage ACS because it

- Produces central nervous system analgesia, which reduces the adverse effects of neurohumoral activation, catecholamine release, and heightened myocardial oxygen demand
- Alleviates dyspnea
- Produces venodilation, which reduces LV preload and oxygen requirement
- Decreases systemic vascular resistance, which reduces LV afterload
- Helps redistribute blood volume in patients with acute pulmonary edema

Remember, morphine is a venodilator. As with nitroglycerin, use smaller doses and carefully monitor physiologic response before administering additional doses in patients who may be preload dependent. If hypotension develops, administer fluids as a first line of therapy.

Critical Concepts: Pain Relief With Nitroglycerin

Pain relief with nitroglycerin is not useful for diagnosing the cause of symptoms in ED patients with chest pain or discomfort. GI and other causes of chest discomfort can improve with nitroglycerin administration, so a patient's response to nitrate therapy is not diagnostic of ACS.

Caution: Nonsteroidal Anti-inflammatory Drugs

Do not use nonsteroidal anti-inflammatory drugs (except for aspirin), including nonselective and COX-2 selective drugs, during hospitalization for STEMI because of the increased risk of mortality, reinfarction, hypertension, heart failure, and myocardial rupture associated with their use.

Obtaining a 12-Lead ECG

The AHA recommends out-of-hospital 12-lead ECG diagnostic programs in all EMS systems, and all EMS systems should take the actions outlined in Table 4.

Table 4. EMS Actions per AHA Recommendations

EMS action	Recommendation
Obtain a 12-Lead ECG if available.	The AHA recommends routine use of 12-lead out-of-hospital ECGs for patients with signs and symptoms of possible ACS.
Provide prearrival notification to the hospital.	Prearrival notification of the ED shortens the time to treatment (10 to 60 minutes has been achieved in clinical studies) and speeds reperfusion therapy with fibrinolytics or PCI or both, which may reduce mortality and minimize myocardial injury.
Complete a fibrinolytic checklist if appropriate.	If STEMI is identified on the 12-lead ECG, complete a fibrinolytic checklist if appropriate. Consider prehospital fibrinolysis per local protocol.

Immediate ED Assessment and Treatment

The ED and cath lab assessment should occur concurrently within the first 10 minutes. The high-performance team should quickly evaluate the patient with potential ACS on the patient's arrival and obtain a 12-lead ECG (if not already performed before arrival) and assess the patient.

The 12-lead ECG (example in Figure 17) is at the center of the decision pathway in managing ischemic chest discomfort and is the only way to identify STEMI.

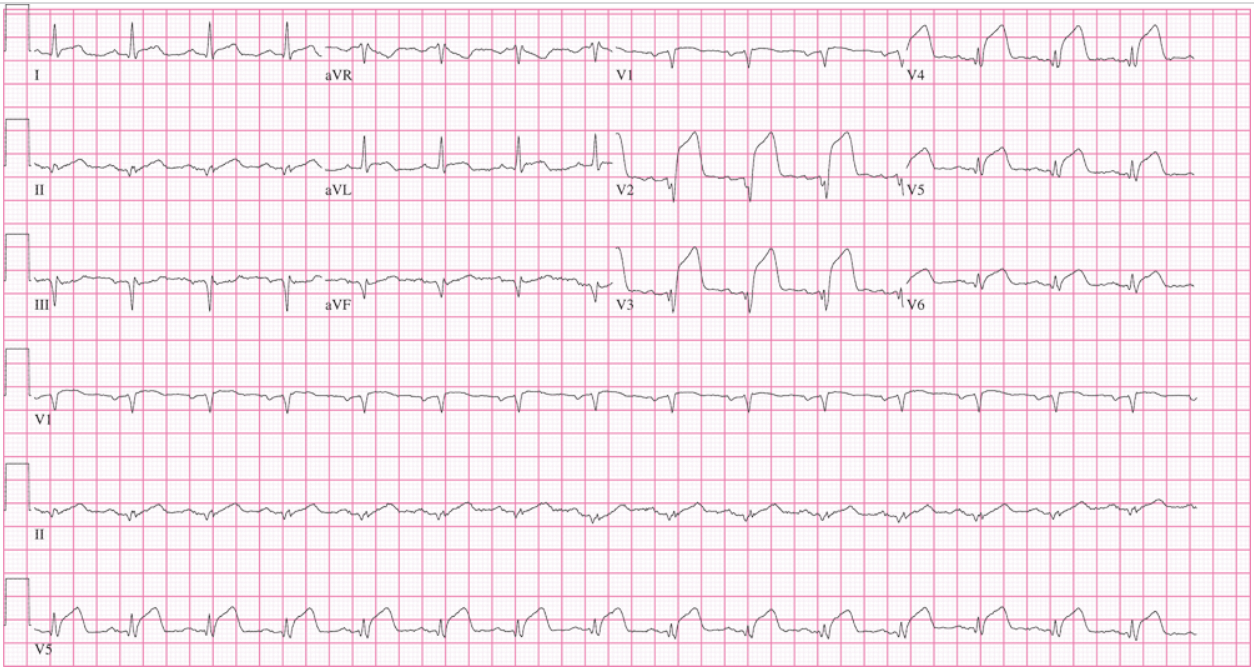


Figure 17. Anterior STEMI on a 12-lead ECG.

Assess ABCs, give oxygen (if needed), and establish IV access. Perform a brief targeted history and physical exam, focusing on chest discomfort, signs and symptoms of heart failure, cardiac history, risk factors for ACS, and historical features that may preclude the use of fibrinolytics. Review and complete fibrinolytic checklist and check contraindications. Obtain initial cardiac marker levels and complete blood counts and coagulation studies, and obtain a portable chest x-ray in less than 30 minutes (do not delay transport to the cath lab). For a patient with STEMI, the goals of reperfusion are

- PCI should begin within 90 minutes from first medical contact to balloon inflation
- Fibrinolytic administration should begin within 30 minutes of the patient's arrival in the ED

Figure 18 shows how to measure ST-segment deviation.

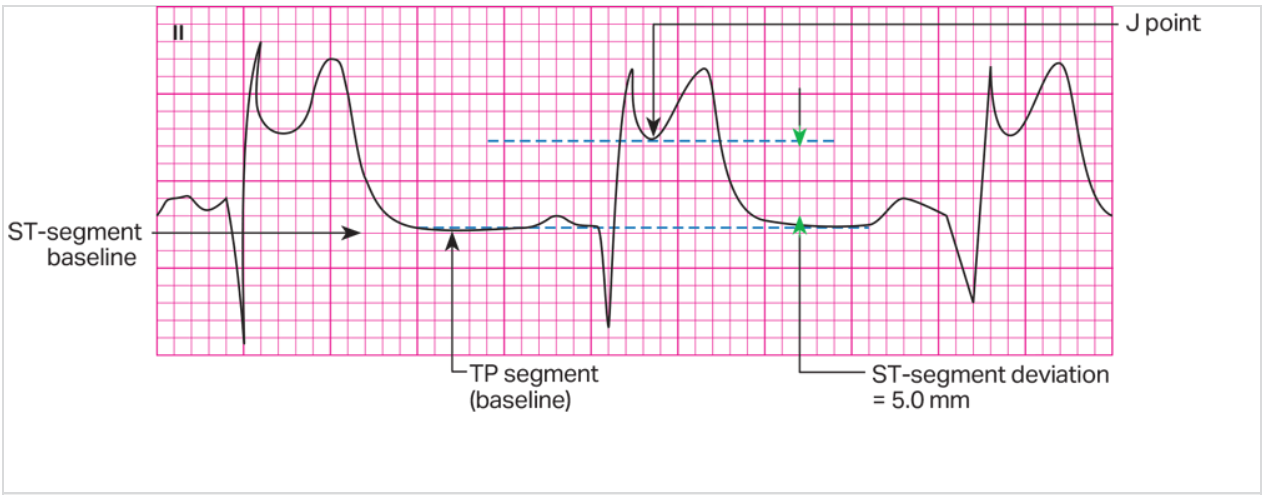


Figure 18A. How to measure ST-segment deviation. A, Inferior MI. The ST segment has no low point (it is coved or concave).

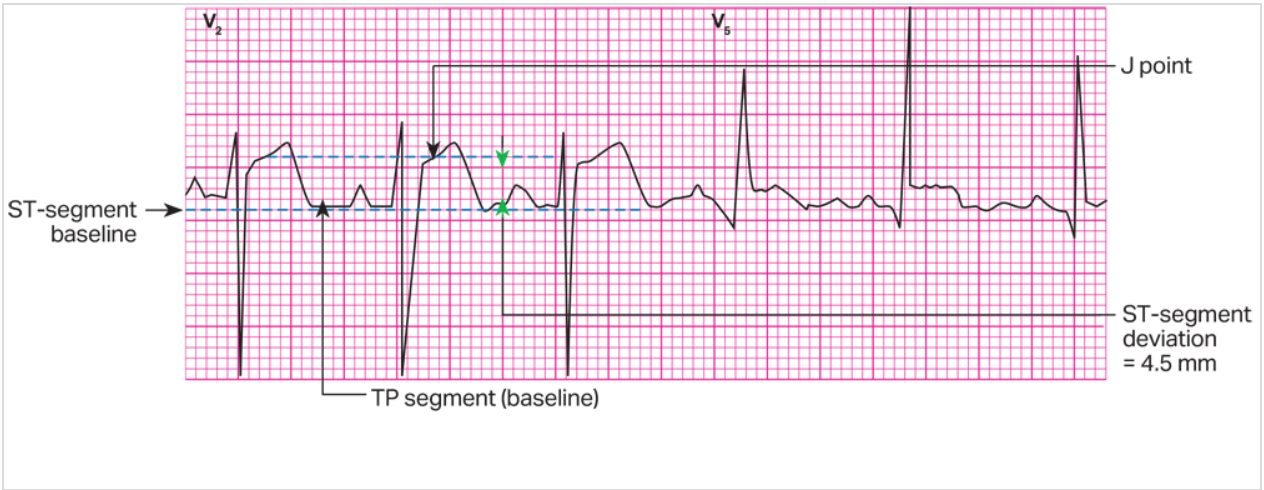


Figure 18B. Anterior MI.

The First 10 Minutes

Concurrent ED/cath lab assessment in the first 10 minutes:

- Activate STEMI team upon EMS notification.
- Assess ABCs; give oxygen if needed.
- Establish IV access.
- Perform brief, targeted history, physical exam.
- Review and complete fibrinolytic checklist; check contraindications.
- Obtain initial cardiac marker levels and complete blood counts and coagulation studies.
- Obtain a portable chest x-ray (in less than 30 minutes); do not delay transport to the cath lab. The results of cardiac markers, chest x-ray, and laboratory studies should not delay reperfusion

therapy unless clinically necessary, eg, suspected aortic dissection or coagulopathy.

Immediate ED and Cath Lab General Treatment

Unless allergies or contraindications exist, consider these 4 agents in patients with ischemic-type chest discomfort:

- If oxygen saturation is less than 90%: Start oxygen at 4 L/min and titrate.
- Aspirin 162 to 325 mg (if not given by EMS)
- Nitroglycerin sublingual or translingual
- Morphine IV if discomfort not relieved by nitroglycerin

Consider administration of $P2Y_{12}$ inhibitors. Because out-of-hospital providers may have given these agents already, administer initial or supplemental doses as indicated. (See the discussion of these drugs in EMS Assessment, Care, and Hospital Preparation.)

Critical Concepts: Oxygen, Aspirin, Nitrates, and Opiates

- *Unless contraindicated, initial therapy with aspirin, nitrates, and, if indicated, oxygen is recommended for all patients suspected of having severe ischemic chest discomfort. If pain is not controlled, consider morphine to minimize pain and the associated catecholamine release. However, morphine can decrease absorption of oral antiplatelet medications.*
- *The major contraindication to nitroglycerin and morphine is hypotension, including from an RV infarction. The major contraindications to aspirin are true aspirin allergy and active or recent GI bleeding.*

Classifying Patients by ST-Segment Deviation

Review the initial 12-lead ECG (Step 4) and classify patients into 1 of 2 following major clinical groups (Steps 5 and 9):

- *STEMI* is characterized by ST-segment elevation in 2 or more contiguous leads or new left bundle branch block (LBBB). Threshold values for ST-segment elevation consistent with STEMI are J-point elevation greater than 2 mm (0.2 mV) in leads V_2 and V_3 (2.5 mm in men younger than 40 years; 1.5 mm in all women)

and 1 mm or more in all other leads or by new or presumed new LBBB.

- NSTE-ACS (Step 9):
    - –*High-risk NSTE-ACS (Step 10)* is characterized by ischemic ST-segment depression 0.5 mm (0.05 mV) or greater or dynamic T-wave inversion with pain or discomfort. Nonpersistent or transient ST-segment elevation 0.5 mm or greater for less than 20 minutes is also included in this category. If troponin is elevated or if this is a high-risk patient, consider early invasive strategy if (Step 11)
        - Refractory ischemic chest discomfort
        - Recurrent/persistent ST-segment deviation
        - Ventricular tachycardia
        - Hemodynamic instability
        - Signs of heart failure

    - Start adjunctive therapies (eg, nitroglycerin, heparin) as indicated. Please refer to the “2014 AHA/ACC Guideline for the Management of Patients With Non–ST-Elevation Acute Coronary Syndromes: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines” for more information.[6]

    - –*Low- to intermediate-risk NSTE-ACS (Step 12)* is characterized by normal or nondiagnostic changes in the ST segment or T wave that are inconclusive and require further risk stratification. This classification includes patients with normal ECGs and those with ST-segment deviation in either direction of less than 0.5 mm (0.05 mV) or T-wave inversion of 2 mm (0.2 mV) or less. Serial cardiac studies and functional testing are appropriate. Note that additional information (troponin) may place the patient into a higher risk classification after initial classification. Consider admission to the ED chest pain

unit or to an appropriate bed for further monitoring and possible intervention (Step 13).

The ECG classification of ischemic syndromes is not exclusive—for example, a small percentage of patients with normal ECGs may have MI. If the initial ECG is nondiagnostic and clinical circumstances indicate (eg, ongoing chest discomfort), repeat the ECG. The use of a single ECG to classify patients with suspected ACS is not sufficient. Assessment of cardiac enzymes and serial ECGs in patients with ongoing symptoms is necessary to complete the acute assessment of patients suspected of having this condition.

STEMI

Patients with STEMI usually have complete occlusion of an epicardial coronary artery.

Treat STEMI by providing early reperfusion therapy achieved with primary PCI or fibrinolytics.

Reperfusion therapy for STEMI is perhaps the most important advancement for treating cardiovascular disease in recent years. Early fibrinolytic therapy or direct catheter-based reperfusion is an established standard of care for patients with STEMI who present within 12 hours after symptom onset with no contraindications. Reperfusion therapy reduces mortality and saves heart muscle; the shorter the time to reperfusion, the greater the benefit. In fact, providing fibrinolytic therapy in the first hour after symptom onset reduces mortality by 47%.

Critical Concepts: Delay of Therapy

- *Do not delay diagnosis and treatment to consult with a cardiologist or another physician except in equivocal or uncertain cases because delays are associated with increased hospital mortality rates.*
- *Potential delay during in-hospital evaluation may occur from door to data (ECG), from data to decision, and from decision to drug (or PCI). These 4 major points of in-hospital therapy are commonly referred to as the 4 D's.*
- *All providers must focus on minimizing delays at each of these points.*

Early Reperfusion Therapy

Rapidly identify patients with STEMI and use a fibrinolytic checklist to screen for indications and contraindications to fibrinolytic therapy, if appropriate.

The first qualified physician who encounters a patient with STEMI should interpret or confirm the 12-lead ECG, determine the risk/benefit of reperfusion therapy, and direct administration of fibrinolytic therapy or activation of the PCI team. Early activation of PCI may occur with established protocols. Use these recommended time frames:

- For PCI, the goal is first medical contact–to–balloon inflation time of 90 minutes or less. For patients at a non–PCI-capable hospital, time from first medical contact to device should be less than 120 minutes when considering primary PCI, but systems should strive to achieve the shortest time possible.
- If fibrinolysis is the intended reperfusion, the longest acceptable ED door-to-needle time (needle time is the beginning of infusion of a fibrinolytic agent) is 30 minutes, but systems should strive to achieve the shortest time possible.
- Consider patients who are ineligible for fibrinolytic therapy for transfer to a PCI facility, regardless of delay, but prepare for a door-to-departure time of 30 minutes.

Adjunctive treatments may also be indicated.

Choosing Primary PCI

The most common form of PCI is coronary angioplasty with stent placement, and primary PCI is preferred over fibrinolytic administration. Many studies have shown PCI to be superior to fibrinolysis in the combined end points of death, stroke, and reinfarction for patients presenting between 3 and 12 hours after onset.

Interventional strategies for the management of STEMI are as follows:

1. Primary PCI: The patient is taken to the catheterization laboratory for PCI immediately after hospital presentation.
2. Rescue PCI: The patient is initially treated with fibrinolytic therapy. The patient does not show signs of reperfusion (lack of ST resolution more than 50% after 1 hour of fibrinolytic therapy administration) and therefore is referred for rescue PCI.

3. Pharmacoinvasive strategy: The patient is initially treated with fibrinolytic therapy with the intention to perform coronary angiography and PCI, if appropriate.

Considerations for the use of primary PCI include the following:

- PCI is the treatment of choice for the management of STEMI when it can be performed effectively with first medical contact–to–balloon inflation time of 90 minutes or less by a skilled provider at a skilled PCI facility.
- Primary PCI may also be offered to patients presenting to non–PCI-capable centers if PCI can be initiated promptly within 120 minutes after first medical contact.
- For patients admitted to a non-PCI center, transferring for PCI vs administering on-site fibrinolytics may have some benefit in terms of reinfarction, stroke, and a trend to lower mortality when PCI is performed within 120 minutes after first medical contact.
- PCI is also preferred in patients with contraindications to fibrinolytics and is indicated in patients with high-risk features, heart failure complicating MI, or cardiogenic shock.

Using Fibrinolytic Therapy

Administer a fibrinolytic agent or “clot-buster” to patients with ST-segment elevation greater than 2 mm (0.2 mV) in leads V_2 and V_3 and 1 mm or more in all other leads or by new or presumed new LBBB (eg, leads III, aVF; leads V_3, V_4; leads I and aVL) without contraindications. Fibrin-specific agents achieve normal flow in about 50% of patients given these drugs. Examples of fibrin-specific drugs are alteplase, reteplase, and tenecteplase. Streptokinase was the first fibrinolytic used widely, but it is not fibrin specific.

Considerations for the use of fibrinolytic therapy are as follows:

- In the absence of contraindications and in the presence of a favorable risk-benefit ratio, fibrinolytic therapy is one option for reperfusion in patients with STEMI and *onset of symptoms within 12 hours after presentation* with qualifying ECG findings and if PCI is not available within 90 minutes after first medical contact.
- In the absence of contraindications, it is also reasonable to give fibrinolytics to patients with *onset of symptoms within the prior 12 hours* and ECG findings consistent with true posterior MI. Experienced providers will recognize this as a condition where ST-

segment depression in the early precordial leads is equivalent to ST-segment elevation in others. When these changes are associated with other ECG findings, it suggests a “STEMI” on the posterior wall of the heart.

- Fibrinolytics are generally not recommended for patients presenting *more than 12 hours after onset of symptoms*. But they may be considered if ischemic chest discomfort continues with persistent ST-segment elevation.
- Do not give fibrinolytics to the following patients:
    - –Those who present *more than 24 hours after the onset of symptoms*
    - –Those with ST-segment depression, unless a true posterior MI is suspected

Adjunctive Treatments

Other drugs are useful when indicated in addition to oxygen, sublingual or translingual nitroglycerin, aspirin, morphine, and fibrinolytic therapy. These include

- Unfractionated or low-molecular-weight heparin
- Bivalirudin
- $P2Y_{12}$ inhibitors (clopidogrel, prasugrel, and ticagrelor)
    - –Clopidogrel and prasugrel are thienopyridines that require liver biotransformation into active metabolites. Ticagrelor does not require liver biotransformation and is a reversible $P2Y_{12}$ inhibitor. The timing of administration of $P2Y_{12}$ should be at the discretion of local site practices.
- IV nitroglycerin
- β-Blockers
- Glycoprotein IIb/IIIa inhibitors

IV nitroglycerin and heparin are common for early management of patients with STEMI. We briefly discuss heparin and IV nitroglycerin, but we do not review bivalirudin, $P2Y_{12}$ inhibitors, β-blockers, and glycoprotein IIb/IIIa inhibitors. These agents require additional risk stratification skills and a detailed knowledge of the spectrum of ACS and, in some instances, continuing knowledge of the results of clinical trials.

Heparin (Unfractionated or Low-Molecular-Weight)

Heparin is a routine adjunct for PCI and fibrinolytic therapy with fibrin-specific agents (alteplase, reteplase, tenecteplase). If you use these drugs, you must be familiar with dosing schedules for specific clinical strategies.

Inappropriate dosing and monitoring of heparin therapy have caused excess intracerebral bleeding and major hemorrhage in STEMI patients. Providers using heparin must know the indications, dosing, and use in the specific ACS categories.

The dosing, use, and duration are derived from use in clinical trials. Specific patients may require dose modification. See the ECC Handbook for weight-based dosing guidelines, intervals of administration, and adjustment of low-molecular-weight heparin in renal function. See the American College of Cardiology/AHA guidelines for detailed discussion in specific categories.

IV Nitroglycerin

Routine use of IV nitroglycerin is not indicated and has not been shown to significantly reduce mortality in STEMI. However, IV nitroglycerin is indicated and used widely in ischemic syndromes and is preferred over topical or long-acting forms because it can be adjusted in a patient with potentially unstable hemodynamics and clinical condition. Indications for initiating IV nitroglycerin in STEMI are

- Recurrent or continuing chest discomfort unresponsive to sublingual or translingual nitroglycerin
- Pulmonary edema complicating STEMI
- Hypertension complicating STEMI

Treatment goals using IV nitroglycerin are as follows:

For relief of ischemic chest discomfort,

- Titrate to effect
- Keep SBP greater than 90 mm Hg
- Limit drop in SBP to 30 mm Hg below baseline in hypertensive patients

For improvement in pulmonary edema and hypertension,

- Titrate to effect
- Limit drop in SBP to 10% of baseline in normotensive patients

- Limit drop in SBP to 30 mm Hg below baseline in hypertensive patients

Acute Stroke

Overview

The identification and initial management of patients with acute stroke is within the scope of an ACLS provider.

Out-of-hospital acute stroke care focuses on critical EMS assessments and actions (Step 2):

- Assess ABCs; give oxygen if needed.
- Initiate stroke protocol.
- Perform physical exam.
- Perform validated prehospital stroke screen and stroke severity tool.
- Establish time of symptom onset (last known normal).
- Triage to most appropriate stroke center.
- Check glucose; treat if indicated.
- Provide prehospital notification; on arrival, transport to brain imaging suite.

Note: Refer to the Emergency Medical Services Acute Stroke Routing later in this section.

In-hospital acute stroke care occurs in the ED or brain imaging suite. It is best practice to bypass the ED and go straight to the brain imaging suite. The immediate general and neurologic assessment by the hospital or stroke team (Step 3) includes the following:

- Activate stroke team upon EMS notification.
- Prepare for emergent CT scan or MRI of brain upon arrival.
- Stroke team meets EMS on arrival.
- Assess ABCs; give oxygen if needed.
- Obtain IV access and perform laboratory assessments.
- Check glucose and treat if indicated.
- Review patient history, medications, and procedures.
- Establish time of symptom onset or last known normal.

Perform a physical exam and neurologic examination, including the NIH Stroke Scale or Canadian Neurological Scale. In 2010, the AHA/ASA launched Target: Stroke, a national quality improvement initiative with the

goal of reducing door-to-needle times for eligible patients with acute ischemic stroke. The most recent implementation of current best practices, Target Stroke: III, has established new, more aggressive target goals to give patients the best chance for a good recovery. Target Stroke: III time target goals for reperfusion strategies include the following:

- Achieve door-to-needle times within 60 minutes in 85% or more of acute ischemic stroke patients treated with IV thrombolytics.
- Achieve door-to-device times (arrival to first pass with thrombectomy device) within 90 minutes for direct-arriving patients and within 60 minutes for transfer patients in 50% or more of acute ischemic stroke patients treated with endovascular therapy (EVT).

Drugs for Stroke

Drugs for stroke include

- Approved fibrinolytic agent (alteplase)
- Glucose (D_{10}/D_{50})
- Labetalol
- Nicardipine
- Clevidipine
- Aspirin

Major Types of Stroke

Stroke is a general term. It refers to an acute neurologic impairment that follows interruption in blood supply to a specific area of the brain. Although expeditious stroke care is important for all patients, this section emphasizes reperfusion therapies for acute ischemic stroke.

The major types of stroke are

- Ischemic stroke: accounts for 87% of all strokes and is usually caused by an occlusion of an artery to a region of the brain (Figure 19)
- Hemorrhagic stroke: accounts for 13% of all strokes and occurs when a blood vessel in the brain suddenly ruptures into the surrounding tissue. Fibrinolytic therapy is contraindicated in this type of stroke. Avoid anticoagulants.

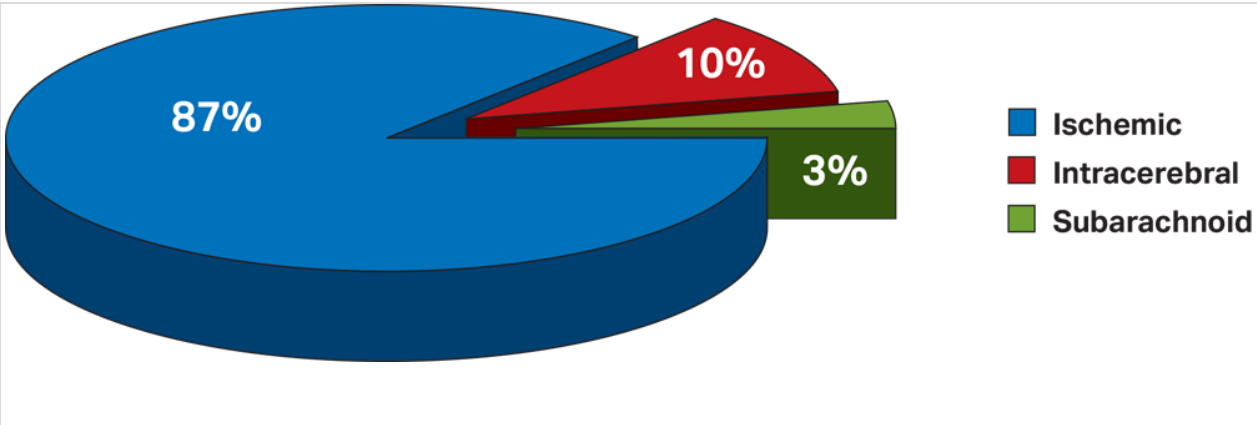


Figure 19. Types of stroke. Eighty-seven percent of strokes are ischemic and potentially eligible for reperfusion therapy if patients otherwise qualify. Thirteen percent of strokes are hemorrhagic, and the majority of these are intracerebral.

The male-to-female incidence ratio is 1.25 in persons 55 to 64 years of age, 1.50 in those 65 to 74, 1.07 in those 75 to 84, and 0.76 in those 85 and older. Blacks have almost twice the risk of first-ever stroke compared with Whites.

Approach to Stroke Care

Each year in the United States, about 795 000 people suffer a new or recurrent stroke. Stroke remains a leading cause of death as well as disability in the United States. Stroke risk increases with age, but approximately one third of people hospitalized for stroke are under age 65.[7]

Early recognition of acute ischemic stroke is critically important because time from symptom onset to reperfusion is key. IV fibrinolytic treatment should be provided as early as possible, generally within 3 hours after onset of symptoms, or within 4.5 hours after onset of symptoms for selected patients. EVT may be given within 24 hours after onset of symptoms in appropriately selected patients, but better outcomes are associated with shorter times to treatment. Although most strokes occur at home, only half of acute stroke patients use EMS for transport to the hospital, which delays time to evaluation and therapeutic interventions. Stroke patients often deny or try to rationalize their symptoms. Even high-risk patients, such as those with atrial fibrillation or hypertension, fail to recognize the signs of stroke. This delays activation of EMS and treatment, resulting in increased morbidity and mortality.

Community and professional education is essential, and it has been successful in increasing the proportion of eligible stroke patients treated with fibrinolytic therapy. Healthcare providers, hospitals, and communities must continue to develop regional stroke systems of care to improve the efficiency and effectiveness of stroke care.

Stroke Chain of Survival

The goal of stroke care is to minimize brain injury and maximize the patient's recovery. The Stroke Chain of Survival (Figure 20) described by the AHA and the American Stroke Association (ASA) is similar to the Chain of Survival for sudden cardiac arrest. It links the actions that patients, family members, and healthcare providers should take to maximize stroke recovery. These links are

- Rapid recognition of and reaction to stroke warning signs and symptoms
- Rapid use of 9-1-1 and EMS dispatch
- Rapid EMS recognition of stroke, triage, transport, and prehospital notification to the receiving hospital
- Rapid diagnosis and treatment in the hospital

Figure 20. The Stroke Chain of Survival.

The 8 D's of Stroke Care

The 8 D's of Stroke Care highlight the major steps in diagnosis and treatment of stroke and key points at which delays can occur:

- **D**etection: rapid recognition of stroke signs and symptoms
- **D**ispatch: early activation and dispatch of EMS by phoning 9-1-1
- **D**elivery: rapid EMS stroke identification, management, triage, transport, and prehospital notification
- **D**oor: emergent ED/imaging suite triage and immediate assessment by the stroke team
- **D**ata: rapid clinical evaluation, laboratory testing, and brain imaging
- **D**ecision: establishing stroke diagnosis and determining optimal therapy selection
- **D**rug/**D**evice: administration of fibrinolytic and/or EVT if eligible

- **D**isposition: rapid admission to the stroke unit or critical care unit, or emergent interfacility transfer for EVT

For more information on these critical elements, see the Adult Suspected Stroke Algorithm (Figure 21).

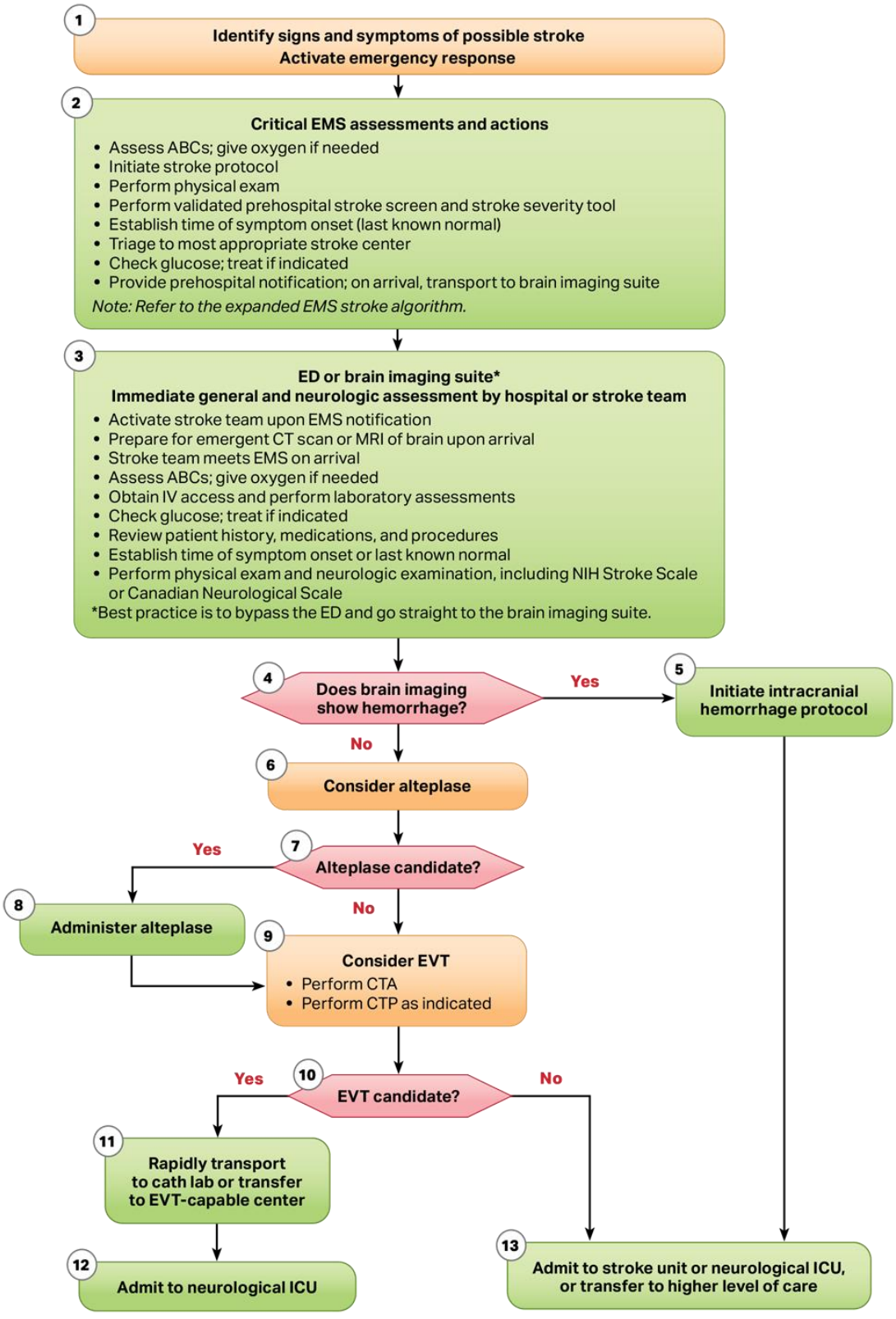


Figure 21. Adult Suspected Stroke Algorithm.

Goals of Stroke Care

Initial time goals were based on the National Institute of Neurological Disorders and Stroke consensus conference held in 1997, shortly after the approval of alteplase. Over the past 2 decades, AHA process improvement projects have led to new and updated goals. Each stroke center should adopt the best practices identified in the Target: Stroke programs as they apply to that center's unique settings. The overall goal remains to minimize delays to reperfusion. The Adult Suspected Stroke Algorithm reviews the critical in-hospital time periods for patient assessment and treatment:

1. Immediate general and neurologic assessment by the hospital or stroke team, emergency physician, or another expert, ideally upon arrival and within 10 minutes after arrival; activate stroke team upon EMS notification; prepare for emergent CT scan or MRI of brain upon arrival; stroke team meets EMS on arrival; assess ABCs and give oxygen if needed; obtain IV access and perform laboratory assessments; check glucose and treat if indicated; review patient history, medications, and procedures; establish time of symptom onset or last known normal; perform physical exam and neurologic examination, including NIH Stroke Scale or Canadian Neurological Scale (Step 3).
2. Neurologic assessment by the stroke team or designee and noncontrast computed tomography (NCCT) scan or MRI performed within 20 minutes after hospital arrival (ideally EMS goes directly to computed tomography (CT)/MRI suite from the field) (Step 3)
3. Interpretation of the NCCT/MRI within 45 minutes after ED/brain imaging suite arrival (box 4)
4. Initiation of fibrinolytic therapy in appropriate patients (those without contraindications) within 45 minutes after hospital arrival (Steps 6 through 8)
5. Door-to-device times within 90 minutes for direct arriving patients and 60 minutes for transfer patients (Step 9)
6. Door-in to door-out times for patients being transferred for possible EVT within 60 minutes (Steps 9 through 11)
7. Door-to-admission (stroke unit or neurocritical care unit) time of 3 hours (Steps 12 and 13)

Critical Time Periods

Patients with acute ischemic stroke have a time-dependent benefit for reperfusion therapy similar to that of patients with STEMI, but this time-dependent benefit is much shorter. The critical time period for administration of reperfusion therapies begin with the onset of symptoms. Critical time periods from hospital arrival are summarized here and represent maximum times:

- Immediate general assessment: **within 10 minutes**
- Immediate neurologic assessment: **within 20 minutes**
- Acquisition of CT/MRI of the head: **within 20 minutes**
- Interpretation of the CT/MRI scan: **within 45 minutes**
- Administration of fibrinolytic therapy, timed from ED/brain imaging suite arrival: **within 60 minutes**
- Administration of fibrinolytic therapy, timed from onset of symptoms: **within 3 hours, or 4.5 hours in selected patients**
- Administration of EVT, timed from onset of symptoms: **up to 24 hours for patients with large vessel occlusion (LVO): 0 to 6 hours requires eligible NCCT scan; 6 to 24 hours requires eligible penumbral imaging**
- Admission to a monitored bed: **3 hours**
- Interfacility transfers for EVT (door-in-door-out): **1 hour**

The Adult Suspected Stroke Algorithm (Figure 21) emphasizes important elements of out-of-hospital and in-hospital care for patients with possible stroke. In addition, the Emergency Medical Services Acute Stroke Routing (Figure 22) emphasizes an important evaluation to determine the best hospital to take the patient with a suspected stroke to. These actions include using a stroke screen and severity tool, and rapid transport to the hospital. As with ACS, notifying the receiving hospital in advance speeds the care of the stroke patient upon arrival.

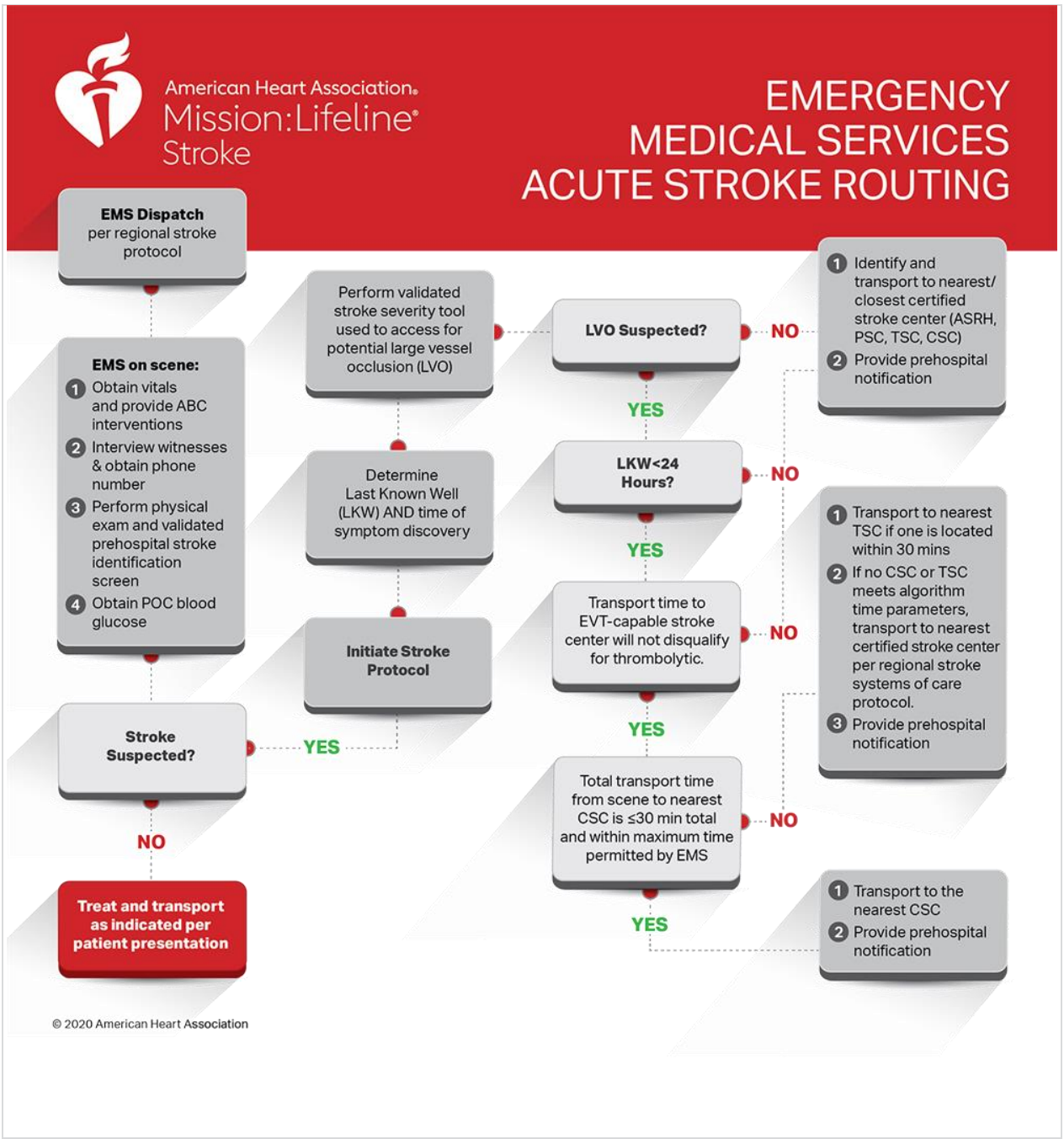


Figure 22. Emergency Medical Services Acute Stroke Routing Algorithm.

Application of the Adult Suspected Stroke Algorithm

We will now discuss the steps in the algorithm (as well as other related topics):

- Identification of signs and symptoms of possible stroke and activation of emergency response (Step 1)

- Critical EMS assessments and actions (Step 2)
- Immediate general and neurologic assessment by hospital or stroke team (ED or brain imaging suite) (Step 3)
- Brain imaging (CT/MRI scan) (Step 4): Does brain imaging show hemorrhage?
- Alteplase candidate? (Fibrinolytic therapy risk stratification of candidate) (Step 7)
- Considering EVT and patient's qualification as a candidate (Steps 9 and 10)
- Rapid transport to cath lab or transfer to EVT-capable center (Step 11)
- Admittance to neurologic ICU or stroke unit, or transfer to higher level of care (Steps 12 and 13)
- Additional imaging for presence of LVO and penumbra when indicated (Steps 9 and 10)
- General stroke care (Steps 12 and 13)

Identify Signs of Possible Stroke and Activate Emergency Response

Warning Signs and Symptoms

The signs and symptoms of a stroke may be subtle. They include

- Sudden weakness or numbness of the face, arm, or leg, especially on one side of the body
- Trouble speaking or understanding
- Sudden trouble seeing in one or both eyes
- Sudden trouble walking
- Dizziness or loss of balance or coordination
- Sudden severe headache with no known cause
- Sudden confusion

Activate EMS System Immediately

Currently half of all stroke patients are driven to the ED by family or friends. Thus, stroke patients and their families must be educated on potential signs or symptoms of stroke and the need to phone 9-1-1 and activate EMS as soon as they detect a possible stroke.

EMS provides the safest and most efficient method of emergency transport to the most appropriate stroke hospital. The advantages of EMS transport include the following:

- Emergency medical dispatchers play a critical role in timely treatment of potential stroke by
    - –Identifying possible stroke patients
    - –Providing high-priority dispatch
    - –Instructing bystanders in lifesaving CPR skills or other supportive care if needed while EMS providers are on the way
- Responding providers can assess ABCs and give oxygen as needed.
- EMS personnel can initiate stroke protocol, perform a physical exam, establish time of symptom onset (last known normal), and check glucose and treat if indicated.
- EMS can triage to the most appropriate stroke center on the basis of a validated prehospital stroke screen and a stroke severity tool and on patient characteristics following regional destination protocols.
- EMS can provide prehospital notification, enabling the hospital to prepare to evaluate and manage the patient more efficiently, and on arrival transport to the brain imaging suite.

Provide Critical EMS Assessments and Actions

Prehospital EMS providers must minimize the interval between the onset of symptoms and patient arrival in the ED/brain imaging suite. Specific stroke therapy can be provided only in the appropriate receiving hospital ED, so time in the field only delays (and may prevent) definitive therapy. More extensive assessments and initiation of supportive therapies can continue en route to the hospital or in the ED/brain imaging suite.

Critical EMS Assessments and Actions

To provide the best outcome for the patient with potential stroke, EMS providers should identify the signs and symptoms of possible stroke (Step 1). These include the following:

- Assess ABCs and give oxygen if needed to hypoxemic stroke patients (ie, whose oxygen saturation is 94% or less) or to those patients with unknown oxygen saturation.
- Initiate stroke protocol.
- Perform physical exam.
- Perform validated prehospital stroke severity tool. Perform a rapid prehospital stroke screen (eg, CPSS) and stroke severity

assessment for possible large vessel occlusion (eg, Los Angeles Motor Scale [LAMS], Rapid Arterial Occlusion Evaluation [RACE], Cincinnati Stroke Triage Assessment Tool [CSTAT], Field Assessment Stroke Triage for Emergency Destination [FAST-ED]).

- Establish time of symptom onset (last known normal). Determine the time of symptom onset or when the patient was last known normal or at neurologic baseline. This represents time zero. If the patient wakes from sleep with symptoms of stroke, time zero is the last time the patient was seen to be normal.
- Triage to most appropriate stroke center. Transport the patient rapidly and triage to an appropriate stroke center based on last known well, stroke severity tool, and regional stroke destination protocol. Support cardiopulmonary function during transport. If possible, bring a witness, family member, or caregiver with the patient to confirm time of onset of stroke symptoms.
- Check glucose if indicated. During transport, check blood glucose if protocols or medical control allows.
- Provide prehospital notification to the receiving hospital, and on arrival, transport to the brain imaging suite.

Table 5. The Cincinnati Prehospital Stroke Scale

Test	Findings
Facial droop: have the patient show teeth or smile (Figure 23).	**Normal**—both sides of the face move equally **Abnormal**—one side of the face does not move as well as the other side
Arm drift: patient closes eyes and extends both arms straight out, with palms up, for 10 seconds (Figure 24).	**Normal**—both arms move the same *or* both arms do not move at all (other findings, such as pronator drift, may be helpful) **Abnormal**—one arm does not move *or* one arm drifts down compared with the other
Abnormal speech: have the patient say, “you can’t teach an old dog new tricks.”	**Normal**—patient uses correct words with no slurring

	Abnormal—patient slurs words, uses the wrong words, or is unable to speak
Interpretation: if any 1 of these 3 signs is abnormal, the probability of a stroke is 72%.	

- The patient with acute stroke is at risk for respiratory compromise from aspiration, upper airway obstruction, hypoventilation, and (rarely) neurogenic pulmonary edema. The combination of poor perfusion and hypoxemia will exacerbate and extend ischemic brain injury, and it has been associated with worse outcome from stroke.

Both out-of-hospital and in-hospital medical personnel should provide supplemental oxygen to hypoxemic stroke patients (ie, those whose oxygen saturation is 94% or less) or patients for whom oxygen saturation is unknown.

Stroke Assessment Tools

The AHA recommends that all EMS personnel be trained to recognize stroke by using a validated, abbreviated out-of-hospital neurologic evaluation tool such as the Cincinnati Prehospital Stroke Scale (CPSS) (Table 5) or the Los Angeles Prehospital Stroke Screen.
Modified from Kothari RU, Pancioli A, Liu T, Brott T, Broderick J. Cincinnati Prehospital Stroke Scale: reproducibility and validity. *Ann Emerg Med.* 1999;33(4):373-378. With permission from Elsevier.

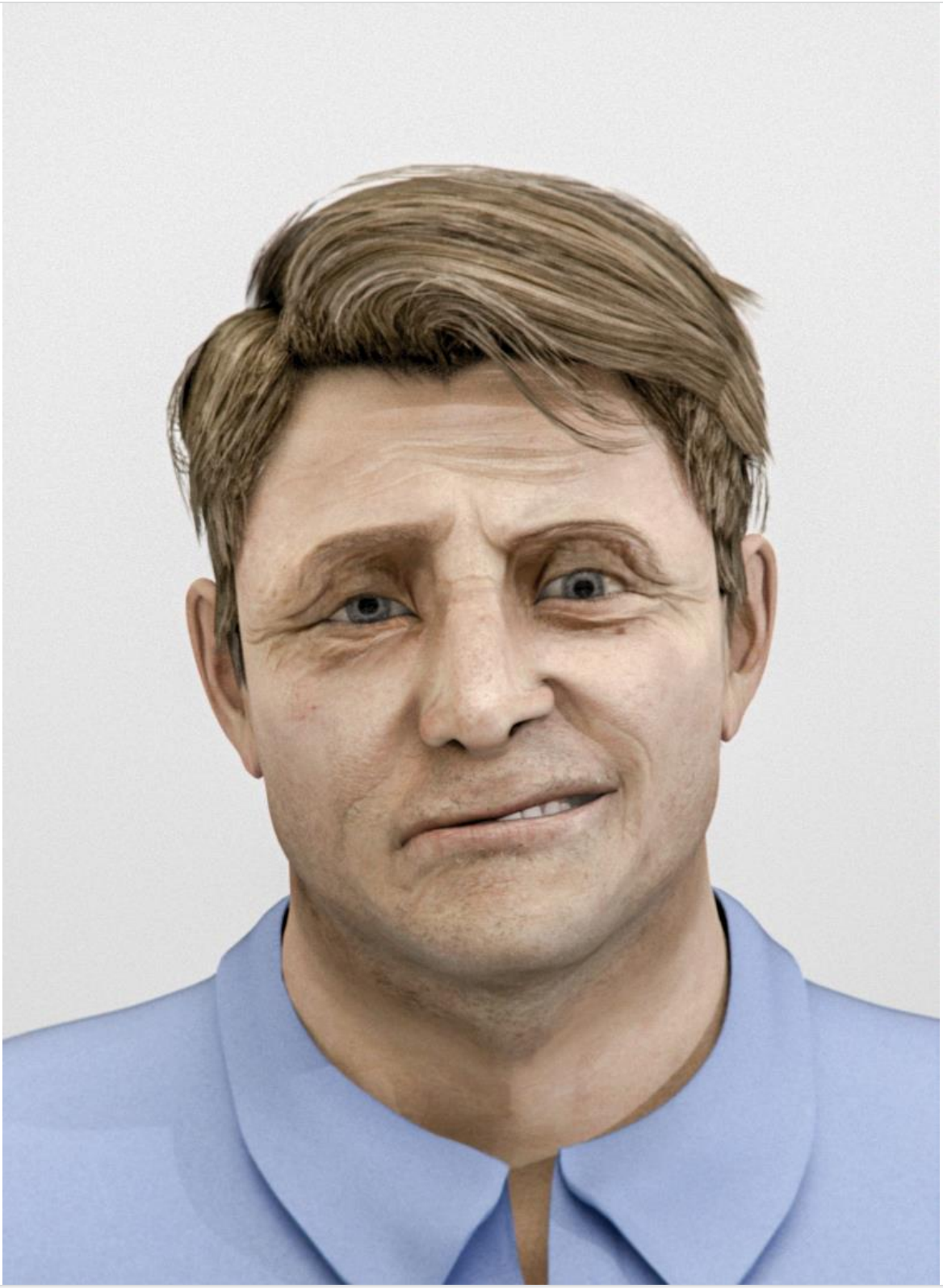


Figure 23. Facial droop.

Figure 24. One-sided motor weakness (right arm).

Cincinnati Prehospital Stroke Scale

The CPSS identifies stroke on the basis of 3 physical findings:

- Facial droop (have the patient smile or try to show teeth)
- Arm drift (have the patient close eyes and hold both arms out, with palms up)
- Abnormal speech (have the patient say, “You can’t teach an old dog new tricks”)

By using the CPSS, medical personnel can evaluate the patient in less than 1 minute. The presence of 1 finding on the CPSS has an estimated probability of stroke of 72% when scored by prehospital providers.

The following list includes examples of prehospital stroke screens and stroke severity scores.

Prehospital stroke screens:

- Cincinnati Prehospital Stroke Scale (CPSS/FAST)
- Los Angeles Prehospital Stroke Screen (LAPSS)
- Melbourne Ambulance Stroke Screen (MASS)
- Miami Emergency Neurologic Deficit Score (MENDS)
- Recognition of Stroke in the Emergency Room Score (ROSIER)

Stroke severity score:

- National Institutes of Health (NIH) Stroke Scale
- Shortened National Institutes of Health Stroke Scale 5 and 8 (sNIHSS-5 and sNIHSS-8)
- Cincinnati Prehospital Stroke Severity Screen (CPSSS)
- Field Assessment Stroke Triage for Emergency Destination (FAST-ED)
- Los Angeles Motor Scale (LAMS)
- Rapid Arterial Occlusion Evaluation Score (RACE)
- Three Item Stroke Scale (3ISS)

Stroke Centers and Stroke Units

Evidence indicates a benefit from triage of stroke patients directly to designated certified stroke centers. Local stakeholders should create a stroke destination protocol based on regional stroke resources.

As stated in the “Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke,” “Certification of stroke centers by

an independent external body, such as Center for Improvement in Healthcare Quality, Det Norske Veritas, Healthcare Facilities Accreditation Program, and The Joint Commission, or a state health department, is recommended." This recommendation is supported by data that demonstrate that the development of stroke centers improves patient care and clinical outcomes. Table 6 shows the different levels and capabilities of hospital stroke designation. Currently, 4 levels of stroke certification exist, and certification is given on the basis of a hospital's specific capabilities.

Table 6. Levels and Capabilities of Hospital Stroke Designation

Hospital attributes	ASRH	PSC	TSC	CSC
Location	**Likely rural**	**Likely urban/suburban**	**Likely urban**	**Likely urban**
Stroke team accessible/available 24 hours/day, 7 days/week	Yes	Yes	Yes	Yes
Noncontrast CT available 24 hours/day, 7 days/week	Yes	Yes	Yes	Yes
Advanced imaging (CTA/CTP/MRI/MRA/MRP) available 24 hours/day, 7 days/week	No	Yes	Yes	Yes
Intravenous alteplase capable	Yes	Yes	Yes	Yes
Thrombectomy capable	No	Possibly	Yes	Yes
Diagnoses stroke pathogenesis/manages poststroke complications	Unlikely	Yes	Yes	Yes
Admits hemorrhagic stroke	No	Possibly	Possibly	Yes
Clips/coils ruptured aneurysms	No	Possibly	Possibly	Yes
Dedicated stroke unit	No	Yes	Yes	Yes
Dedicated neurocritical care unit/ICU	No	Possibly	Possibly	Yes

Abbreviations: ASRH, Acute Stroke Ready Hospital; CSC, Comprehensive Stroke Center; CTA, computed tomography angiography; CTP, computed tomography perfusion; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; MRP, magnetic resonance perfusion; PSC, Primary Stroke Center; TSC, Thrombectomy-Capable Stroke Center.

Acute Stroke Ready Hospital

Acute Stroke Ready Hospitals typically serve rural and under-resourced areas. Emergent identification and treatment of patients with alteplase, when indicated, is typically facilitated by telemedicine to provide access to acute neurologic expertise. Typically, patients are later transferred for admission to a stroke unit or for a higher level of care, as indicated.

Primary Stroke Center

The Primary Stroke Center is the cornerstone of stroke systems of care. These centers comprise a wide range of hospitals able to quickly identify stroke patients, provide alteplase therapy if indicated, and admit patients to a dedicated stroke unit. Roughly half of all stroke patients in the United States receive care in a Primary Stroke Center.

Thrombectomy-Capable Stroke Center

The Thrombectomy-Capable Stroke Center certification was jointly created by the AHA and the Joint Commission to recognize stroke centers that meet the same high-quality standards as a primary stroke center but are also capable of providing EVT for patients with LVO. The Thrombectomy-Capable Stroke Center designation was created to recognize these EVT-capable facilities in areas where a Comprehensive Stroke Center was not available.

Comprehensive Stroke Center

Hospitals achieving Comprehensive Stroke Center certification are capable of managing all forms and severities of stroke, both ischemic and hemorrhagic, and can provide 24/7 access to specialty care, such as neurosurgery, EVT, and neurocritical care. A Comprehensive Stroke Center typically serves as the hub of a regional stroke system of care, providing receiving capabilities for transferred patients and providing feedback and education for transferring sites.

Hospitals in a region should achieve stroke center certification to the highest level possible, and then use these levels of capabilities to design a regional stroke system of care. The hospitals' capabilities should be communicated to the regional EMS system and the community.

Once a patient arrives in the ED, a number of assessments and management activities must occur quickly. Protocols should be used to

minimize delay in definitive diagnosis and therapy. Incorporating best practices from the Target Stroke programs has been shown to reduce overall door-to-needle times and improve clinical outcomes while maintaining overall safety. These practices have also been shown to decrease the various interval times first established by the 1997 National Institute of Neurological Disorders and Stroke consensus conference.

The goal of the stroke team, emergency physician, or other experts should be to assess the patient with suspected stroke within* 10 minutes *after arrival in the ED/brain imaging suite (Step 3): "time is brain."

Target: Stroke II Best-Practice Strategies

1. **EMS Prenotification:** EMS providers should provide early prenotification to the receiving hospital when stroke is recognized in the field.
2. **Use stroke tools:** A stroke toolkit containing rapid triage protocol, clinical decision support, stroke-specific order sets, guidelines, hospital-specific algorithms, critical pathways, NIH Stroke Scale, and other stroke tools should be available and used for each patient.
3. **Employ rapid triage protocol and stroke team notification:** Acute triage protocols facilitate the timely recognition of stroke and reduce time to treatment. Acute stroke teams enhance stroke care and should be activated as soon as there is hospital prenotification from EMS personnel of a stroke patient or the stroke patient is identified in the ED.
4. **Use a single-call activation system:** A single call should activate the entire stroke team.
5. **Attach a timer or clock to chart, clipboard, or patient bed:** Acute ischemic stroke care requires an accurate, timely, coordinated, and systematic evaluation of the patient. A universal clock visible to the healthcare providers is an enabling tool for improving the quality of care.
6. **Ensure EMS transfer directly to CT or MRI scanner:** Guided by prespecified protocols, providers can transport eligible stroke patients, if appropriate, from the ED triage area directly to the CT/MRI scanner for initial neurologic examination and brain imaging to determine tissue plasminogen activator eligibility, bypassing the ED bed.

7. **Rapidly acquire and interpret brain imaging:** It is essential to initiate a brain CT scan (or MRI) as soon as possible after patient arrival. Consider initial CT interpretation by a stroke neurologist, reserving advanced imaging for unclear cases only. Additional brain imaging may be acquired after alteplase consideration to determine the presence of an LVO and salvageable penumbra.
8. **Conduct rapid laboratory testing (including point-of-care testing if indicated):** When indicated, order laboratory tests such as glucose and testing for patients in whom coagulation parameters should be assessed because of suspicion of coagulopathy or warfarin treatment. International normalized ratio (prothrombin time)/partial thromboplastin time results should be available as quickly as possible and no later than 30 minutes after ED arrival.
9. **Prepare alteplase in advance:** Mix the drug and set up the bolus dose and 1-hour infusion pump as soon as a patient is recognized as a possible alteplase candidate, even before brain imaging.
10. **Provide rapid access and administration of intravenous alteplase:** Once eligibility has been determined and intracranial hemorrhage has been excluded, intravenous alteplase should be promptly administered without delay.
11. **Use a team-based approach:** The team approach based on standardized stroke pathways and protocols has proven to be effective in enhancing the number of eligible patients treated and reducing time to treatment in stroke.
12. **Provide prompt data feedback:** Accurately measuring and tracking prehospital times, door-to-needle times, IV alteplase and EVT treatment rates in eligible patients, other time intervals, and performance on other stroke performance/quality measures equip the stroke team to identify areas for improvement. A data monitoring and feedback system includes the use of the Get With The Guidelines-Stroke Patient Management Tool.

Immediate General and Neurologic Assessment

Table 7 show the steps by the hospital or stroke team in the ED or brain imaging suite (best practice is to bypass the ED and go straight to the brain imaging suite).

Table 7. Critical Actions in the Evaluation of Potential Acute Stroke	
Step	**Action**
Activate stroke team	Activate stroke team upon EMS notification.
Obtain a CT brain scan or MRI scan	Prepare for emergent CT scan or MRI of brain upon arrival. Upon prehospital notification, order an emergent CT scan or MRI scan of the brain and have the patient taken directly to the CT/MRI suite. Have the CT/MRI read promptly by a qualified physician.
Meet stroke team	Stroke team meets EMS on arrival. Upon prehospital notification or arrival, activate the stroke team or arrange consultation with a stroke expert based on predetermined protocols.
Assess ABCs	Assess the ABCs and evaluate baseline vital signs; give oxygen if needed.
Obtain IV access	Obtain IV access and perform laboratory assessments. Do not let this delay obtaining a CT scan of the brain or administering alteplase.
Check glucose	Check glucose and promptly treat hypoglycemia (<60 mg/dL).
Obtain patient's history	Review patient history, medications, and procedures
Establish symptom onset	Establish time of symptom onset or last known normal
Perform physical and neurologic examinations	Perform physical exam and neurologic examination, including NIH Stroke Scale or Canadian Neurological Scale
Obtain a 12-lead ECG	Obtain a 12-lead ECG, which may identify a recent or ongoing AMI or arrhythmias (eg, atrial fibrillation) as a cause of embolic stroke. A small percentage of patients with acute stroke or transient ischemic attack have coexisting myocardial ischemia or other abnormalities. There is general agreement to recommend cardiac monitoring during the first 24 hours of evaluation in patients with acute ischemic stroke to detect atrial fibrillation and potentially life-threatening arrhythmias.

	Life-threatening arrhythmias can follow or accompany stroke, particularly intracerebral hemorrhage. If the patient is hemodynamically stable, treatment of non–life-threatening arrhythmias (bradycardia, VT, and atrioventricular [AV] conduction blocks) may not be necessary. ***Do not delay the CT/MRI scan to obtain the ECG.***

Immediate Neurologic Assessment by Hospital or Stroke Team

The stroke team, neurovascular consultant, or emergency physician does the following:

- Reviews the patient's history, medications, and procedures and establishes time of symptom onset or last known normal
- Performs a physical and neurologic examination, including the NIH Stroke Scale or Canadian Neurological Scale

The goal for neurologic assessment is within* 20 minutes *after the patient's arrival in the ED/brain imaging suite: "time is brain."

Establish Symptom Onset

Establishing the time of symptom onset or last known well may require interviewing out-of-hospital providers, witnesses, and family members.

Conduct Neurologic Examination

Assess the patient's neurologic deficits by using an established stroke scale, preferably the NIH Stroke Scale or Canadian Neurological Scale. The NIH Stroke Scale uses 15 items to assess and quantify neurologic deficits of a stroke patient. This is a validated measure of stroke severity based on a detailed neurologic examination.

Perform Brain Imaging (CT/MRI): Does Brain Imaging Show Hemorrhage?

A critical decision point in the assessment of the patient with acute stroke is the performance and interpretation of an NCCT/MRI to differentiate ischemic from hemorrhagic stroke. Assessment also includes identifying other structural abnormalities that may be responsible for the patient's symptoms or that represent contraindication to fibrinolytic therapy. The initial NCCT/MRI scan is the most important test for a patient with acute stroke.

- If an NCCT/MRI scan is not readily available, stabilize and promptly transfer the patient to a facility with this capability.
- The presence of intracranial hemorrhage is an absolute contraindication to alteplase and EVT.

Systems should be established so that brain-imaging studies can be performed within* 20 minutes *after the patient arrives in the ED or brain imaging suite.

Decision Point: Hemorrhage or No Hemorrhage

Additional imaging techniques such as CT perfusion, CT angiography, or MRI scans of patients with suspected stroke should be promptly interpreted by a physician skilled in neuroimaging interpretation. Obtaining these additional studies should not delay initiation of IV alteplase in eligible patients. The presence of hemorrhage vs no hemorrhage determines the next steps in treatment.

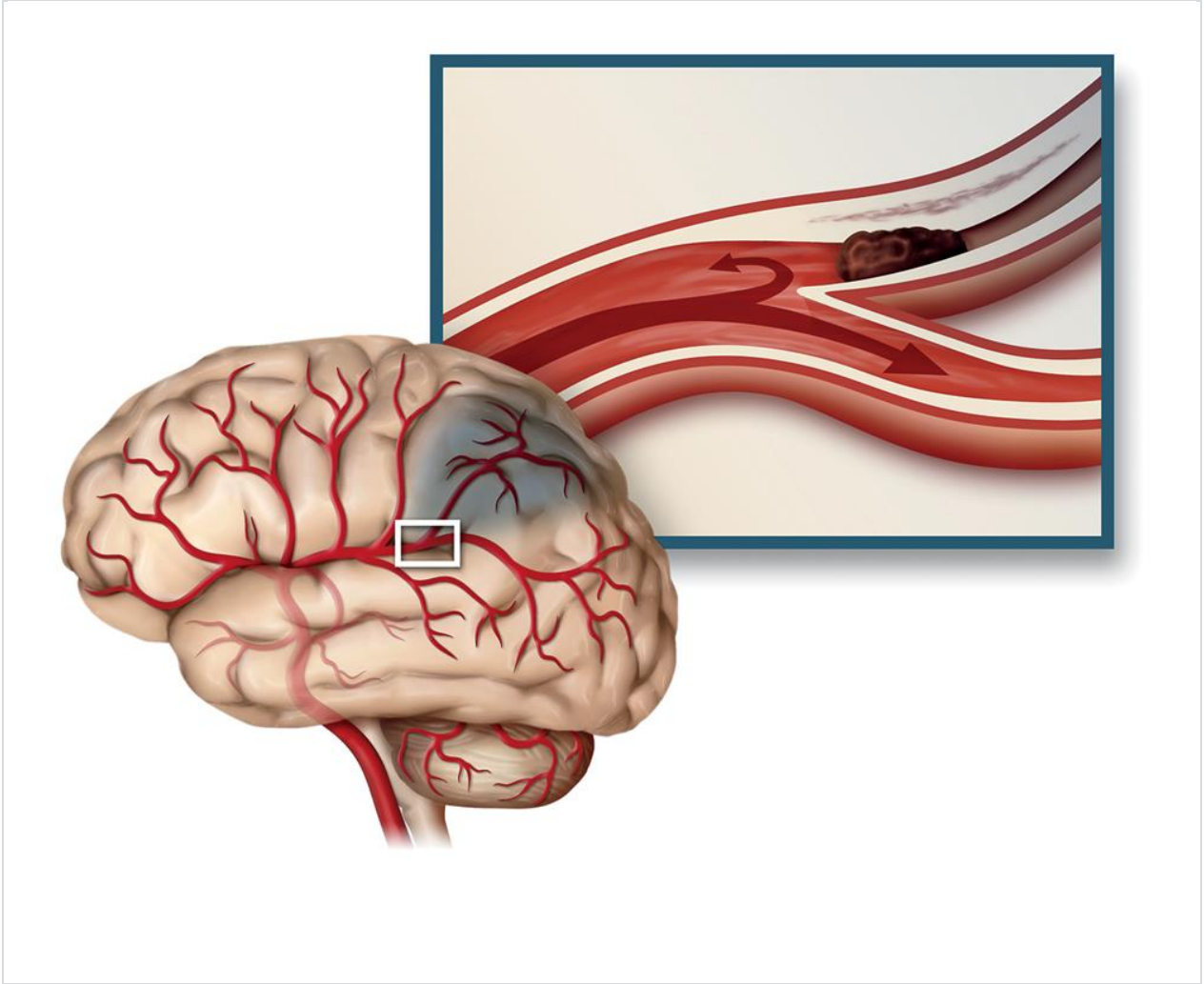


Figure 25A. Occlusion in a cerebral artery by a thrombus. A, Area of infarction surrounding immediate site and distal portion of brain tissue after occlusion.

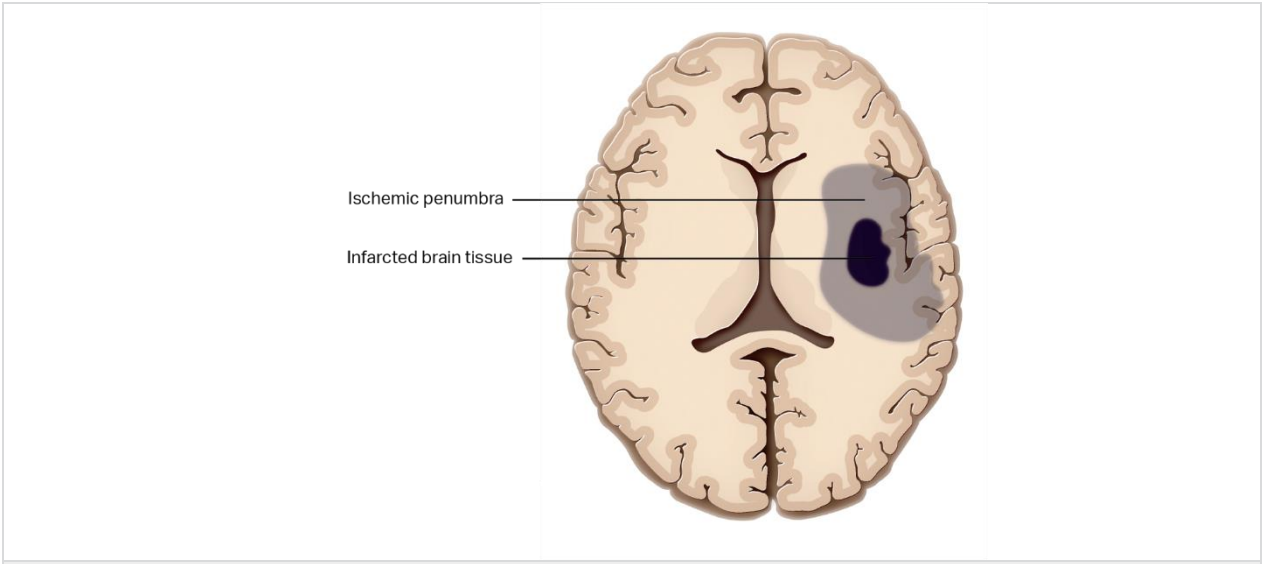


Figure 25B. Area of ischemic penumbra (ischemic, but not yet infarcted [dead] brain tissue) surrounding areas of infarction.

Hemorrhage is present (Steps 5 and 13). If hemorrhage is noted on the NCCT/MRI scan, the patient is not a candidate for fibrinolytics. Initiate intracranial hemorrhage protocol. Admit to the stroke unit or neurologic ICU, or transfer to a higher level of care.

Hemorrhage is not present (Step 6 and 8). If the NCCT/MRI scan shows no evidence of hemorrhage and no sign of other abnormality (eg, tumor, recent stroke), the patient may be a candidate for fibrinolytic therapy.

For patients with a suspected LVO, additional imaging is required. CT angiography will determine if an LVO is present. Less than 6 hours from symptom onset, penumbral imaging is not required. More than 6 hours from symptom onset, penumbral imaging (CT perfusion or multimodal MRI) is required to identify patients with salvageable penumbra. Advanced imaging, including perfusion imaging, should not delay administration of IV alteplase.

Figures 25A and B show an ischemic penumbra that is alive but dysfunctional because of altered membrane potentials. The dysfunction is potentially reversible. The goal of current stroke reperfusion treatments is to minimize the area of permanent brain infarction by preventing the areas of reversible brain ischemia in the penumbra from transforming into larger areas of irreversible brain infarction.

Fibrinolytic Therapy

Studies have demonstrated that there is a higher likelihood of good to excellent functional outcome when alteplase is given to adults with acute ischemic stroke within 3 hours after onset of symptoms, or within 4.5 hours after onset of symptoms for selected patients. Evidence from prospective randomized studies in adults also documents a greater likelihood of benefit the earlier treatment begins.

Table 8. Inclusion and Exclusion Characteristics of Patients With Ischemic Stroke Who Could Be Treated With Alteplase Within 3 Hours After Symptom Onset and Extended Window for Select Patient From 3 to 4.5 Hours*

Indications (COR 1)	
Within 3 hours‡	IV alteplase (0.9 mg/kg, maximum dose 90 mg over 60 minutes with initial 10% of dose given as bolus over 1 minute) is recommended for selected patients who may be treated within 3 hours of ischemic stroke symptom onset or patient last known well or at baseline state. Physicians should review the criteria outlined in this table to determine patient eligibility.‡ (COR 1; LOE A)
Within 3 hours—Age	For otherwise medically eligible patients ≥18 years of age, IV alteplase administration within 3 hours is equally recommended for patients ≤80 and >80 years of age.‡ (COR 1; LOE A)
Within 3 hours—Severe stroke	For severe stroke, IV alteplase is indicated within 3 hours from symptom onset of ischemic stroke. Despite increased risk of hemorrhagic transformation, there is still proven clinical benefit for patients with severe stroke symptoms.‡ (COR 1; LOE A)
Within 3 hours—Mild disabling stroke	For otherwise eligible patients with mild but disabling stroke symptoms, IV alteplase is recommended for patients who can be treated within 3 hours of ischemic stroke symptom onset or patient last known well or at baseline state. (COR 1; LOE B-R)§
BP	IV alteplase is recommended in patients with BP <185/110 mm Hg and in those patients whose BP can be lowered safely to this level with

	antihypertensive agents, with the physician assessing the stability of the BP before starting IV alteplase.‡ (COR 1; LOE B-NR)‖
CT	IV alteplase administration is recommended in the setting of early ischemic changes on NCCT of mild to moderate extent (other than frank hypodensity).‡ (COR 1; LOE A)

The AHA/ASA 2019 Guidelines for the Early Management of Patients With Acute Ischemic Stroke recommends giving IV alteplase to patients with acute ischemic stroke who meet the current eligibility criteria, if it is given by

- Physicians using a clearly defined institutional protocol
- A knowledgeable interdisciplinary team familiar with stroke care
- An institution with a commitment to quality stroke care

Evaluate for Fibrinolytic Therapy

If the CT/MRI scan is negative for hemorrhage, the patient may be a candidate for fibrinolytic therapy. Immediately perform further eligibility and risk stratification:

Additional recommendations for treatment with IV alteplase for patients with AIS (COR 2a)	**And (COR 2b)**
Wake-up and unknown time of onset	IV alteplase (0.9 mg/kg, maximum dose 90 mg over 60 minutes with initial 10% of dose given as bolus over 1 minute) administered within 4.5 hours of stroke symptom recognition can be beneficial in patients with AIS who awake with stroke symptoms or have unclear time of onset >4.5 hours from last known well or at baseline state and who have a DW-MRI lesion smaller than one third of the MCA territory and no visible signal change on FLAIR. (COR 2a; LOE B-R)§
Early improvement	IV alteplase treatment is reasonable for patients who present with moderate to severe ischemic stroke and demonstrate early improvement but remain moderately impaired and potentially disabled in the judgment of the examiner.‡ (COR 2a; LOE A)

Stroke mimics	The risk of symptomatic intracranial hemorrhage in the stroke mimic population is quite low; thus, starting IV alteplase is probably recommended in preference over delaying treatment to pursue additional diagnostic studies.‡ (COR 2a; LOE B-NR)‖

Contraindications (COR 3: No Benefit)*	And (COR 3: Harm)
0 to 4.5-hour window—Mild nondisabling stroke	For otherwise eligible patients with mild nondisabling stroke (NIHSS score 0–5), IV alteplase is not recommended for patients who could be treated within 3 and 4.5 hours of ischemic stroke symptom onset or patient last known well or at baseline state. (COR 3: No Benefit, LOE B-R)§
CT	There remains insufficient evidence to identify a threshold of hypoattenuation severity or extent that affects treatment response to alteplase. However, administering IV alteplase to patients whose CT brain imaging exhibits extensive regions of clear hypoattenuation is not recommended. These patients have a poor prognosis despite IV alteplase, and severe hypoattenuation defined as obvious hypodensity represents irreversible injury.‡ (COR 3: No Benefit; LOE A)
ICH	IV alteplase should not be administered to a patient whose CT reveals an acute intracranial hemorrhage.‡ (COR 3: Harm; LOE C-EO)‖
Ischemic stroke within 3 months	Use of IV alteplase in patients presenting with AIS who have had a prior ischemic stroke within 3 months may be harmful.‡ (COR 3: Harm; LOE B-NR)‖
Severe head trauma within 3 months	In AIS patients with recent severe head trauma (within 3 months), IV alteplase is contraindicated.‡ (COR 3: Harm; LOE C-EO)‖
Acute head trauma	Given the possibility of bleeding complications from the underlying severe head trauma, IV alteplase should not be administered in posttraumatic infarction that occurs during the acute in-hospital phase.‡ (COR 3: Harm; LOE C-EO)‖ (Recommendation wording modified to match COR 3 stratifications.)
Intracranial/intraspinal surgery within 3 months	For patients with AIS and a history of intracranial/spinal surgery within the prior 3 months, IV alteplase is potentially harmful.‡ (COR 3: Harm; LOE C-EO)‖
History of intracranial hemorrhage	IV alteplase administration in patients who have a history of intracranial hemorrhage is potentially harmful.‡ (COR 3: Harm; LOE C-EO)‖

Subarachnoid hemorrhage	IV alteplase is contraindicated in patients presenting with symptoms and signs most consistent with an SAH.‡ (COR 3: Harm; LOE C-EO)‖
GI malignancy or GI bleed within 21 days	Patients with a structural GI malignancy or recent bleeding event within 21 days of their stroke event should be considered high risk, and IV alteplase administration is potentially harmful.‡ (COR 3: Harm; LOE C-EO)‖
Coagulopathy	The safety and efficacy of IV alteplase for acute stroke patients with platelets <100 000/mm^3, INR >1.7, aPTT >40 seconds, or PT >15 seconds are unknown, and IV alteplase should not be administered.‡ (COR 3: Harm; LOE C-EO)‖ (In patients without history of thrombocytopenia, treatment with IV alteplase can be initiated before availability of platelet count but should be discontinued if platelet count is <100 000/mm^3. In patients without recent use of OACs or heparin, treatment with IV alteplase can be initiated before availability of coagulation test results but should be discontinued if INR is >1.7 or PT is abnormally elevated by local laboratory standards.) (Recommendation wording modified to match COR 3 stratifications.)
LMWH	IV alteplase should not be administered to patients who have received a full treatment dose of LMWH within the previous 24 hours.‡ (COR 3: Harm; LOE B-NR)§‖ (Recommendation wording modified to match COR 3 stratifications.)
Thrombin inhibitors or factor Xa inhibitors	The use of IV alteplase in patients taking direct thrombin inhibitors or direct factor Xa inhibitors has not been firmly established but may be harmful.‡ (COR 3: Harm; LOE C-EO)‖ IV alteplase should not be administered to patients taking direct thrombin inhibitors or direct factor Xa inhibitors unless laboratory tests such as aPTT, INR, platelet count, ecarin clotting time, thrombin time, or appropriate direct factor Xa activity assays are normal or the patient has not received a dose of these agents for >48 hours (assuming normal renal metabolizing function).

	(Alteplase could be considered when appropriate laboratory tests such as aPTT, INR, ecarin clotting time, thrombin time, or direct factor Xa activity assays are normal or when the patient has not taken a dose of these ACs for >48 hours and renal function is normal.) (Recommendation wording modified to match COR 3 stratifications.)
Concomitant Abciximab	Abciximab should not be administered concurrently with IV alteplase. (COR 3: Harm; LOE B-R)§

- If the CT/MRI scan shows no hemorrhage, the probability of acute ischemic stroke remains. *Review inclusion and exclusion criteria for IV fibrinolytic therapy (Table 8) and repeat the neurologic exam (NIH Stroke Scale or Canadian Neurological Scale).*
- If the patient's neurologic function is rapidly improving to normal, fibrinolytics may be unnecessary.

Alteplase Considerations in the 3- to 4.5-Hour Time Window in Addition to Those in the 0- to 3-Hour Window*

Indications (COR 1)	
3-4.5 hours‡	IV alteplase (0.9 mg/kg, maximum dose 90 mg over 60 min with initial 10% of dose given as bolus over 1 min) is also recommended for selected patients who can be treated within 3 and 4.5 hours of ischemic stroke symptom onset or patient last known well. Physicians should review the criteria outlined in this table to determine patient eligibility.‡ (COR 1; LOE B-R)‖
3-4.5 hours–Age	IV alteplase treatment in the 3- to 4.5-hour time window is recommended for those patients ≤80 years of age, without a history of both diabetes mellitus and prior stroke, NIHSS score ≤25, not taking any OACs, and without imaging evidence of ischemic injury involving more than one third of the MCA territory.‡ (COR 1; LOE B-R)‖
Additional recommendations for treatment with IV alteplase for patients with AIS (COR 2a)	**And (COR 2b)**
3-4.5 hours–Age	For patients >80 years of age presenting in the 3- to 4.5-hour window, IV alteplase is safe and can be as effective as in younger patients.‡ (COR 2a; LOE B-NR)‖

3-4.5 hours—Diabetes mellitus and prior stroke	In AIS patients with prior stroke and diabetes mellitus presenting in the 3- to 4.5- hour window, IV alteplase may be as effective as treatment in the 0- to 3-hour window and may be a reasonable option.‡ (COR 2b; LOE B-NR)‖
3-4.5 hours—Severe stroke	The benefit of IV alteplase between 3 and 4.5 hours from symptom onset for patients with very severe stroke symptoms (NIHSS score >25) is uncertain.‡ (COR 2b; LOE C-LD)‖
3-4.5 hours—Mild disabling stroke	For otherwise eligible patients with mild disabling stroke, IV alteplase may be reasonable for patients who can be treated within 3 and 4.5 hours of ischemic stroke symptom onset or patient last known well or at baseline state. (COR 2b; LOE B-NR)§

Abbreviations: AC, anticoagulants; AIS, acute ischemic stroke; aPTT, activated partial thromboplastin time; BP, blood pressure; COR, Class of Recommendation; CT, computed tomography; DW-MRI, diffusion-weighted magnetic resonance imaging; FLAIR, fluid-attenuated inversion recovery; GI, gastrointestinal; ICH, intracerebral hemorrhage; INR, international normalized ratio; IV, intravenous; LMWH, low-molecular-weight heparin; LOE, Level of Evidence; MCA, middle cerebral artery; NCCT, noncontrast computed tomography; NIHSS, National Institutes of Health Stroke Scale; OAC, oral anticoagulant; PT, prothromboplastin time.

*The relative contraindications are abbreviated. Modified from Table 8 in the "Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke: a Guideline for Healthcare Professionals from the American Heart Association/American Stroke Association."[8] Please see Table 8 for a full listing of specific considerations.

‡When uncertain, the time of onset time should be considered the time when the patient was last known to be normal or at baseline neurological condition.

‡Recommendation unchanged or reworded for clarity from 2015 IV Alteplase. See Table XCV in online Data Supplement 1 for original wording.

§See also the text of these guidelines for additional information on these recommendations.

‖LOE amended to conform with American College of Cardiology/AHA 2015 Recommendation Classification System.

COR amended to conform with American College of Cardiology/AHA 2015 Recommendation Classification System.

Unless otherwise specified, these eligibility recommendations apply to patients who can be treated within 0 to 4.5 hours of ischemic stroke symptom onset or patient last known well or at baseline state.

Clinicians should also be informed of the indications and contraindications from local regulatory agencies (for current information from the US Food and Drug Administration refer to http://www.accessdata.fda.gov/drugsatfda_docs/label/2015/103172s5203lbl.pdf).

For a detailed discussion of this topic and evidence supporting these recommendations, refer to the AHA scientific statement on the rationale for inclusion and exclusion criteria for IV alteplase in AIS.

Potential Adverse Effects

As with all drugs, fibrinolytics have potential adverse effects. At this point, weigh the patient's risk for adverse events against the potential benefit and discuss with the patient and family.

- Confirm that no exclusion criteria are present (Table 8).
- Consider risks and benefits.
- Be prepared to monitor and treat any potential complications.
- The major complication of IV alteplase for stroke is intracranial hemorrhage. Other bleeding complications may occur and may range from minor to major. Angioedema and transient hypotension may occur.

Patient Is a Candidate for Fibrinolytic Therapy

If the patient remains a candidate for fibrinolytic therapy (Step 8), discuss the risks and potential benefits with the patient or family if available. After this discussion, if the patient or family members decide to proceed with

fibrinolytic therapy, give the patient alteplase. Begin your institution's stroke alteplase protocol, often called a *postalteplase pathway of care*.

Alteplase is considered the standard of care for eligible patients with acute ischemic stroke. Because of this treatment's proven benefit and the need to expedite it, healthcare providers are justified to proceed with IV thrombolysis in an otherwise eligible adult patient with a disabling acute ischemic stroke in situations where that patient cannot provide consent (eg, due to aphasia or confusion) and a legally authorized representative is not immediately available to provide proxy consent.

Do not administer anticoagulants or antiplatelet treatment for 24 hours after administration of alteplase, typically until a follow-up CT scan at 24 hours shows no intracranial hemorrhage.

Extended IV Alteplase Window: 3 to 4.5 Hours

Treatment of carefully selected patients with acute ischemic stroke with IV alteplase between 3 and 4.5 hours after onset of symptoms has also been shown to improve clinical outcome, although the degree of clinical benefit is smaller than that achieved with treatment within 3 hours. Data supporting treatment in this time window come from a large, randomized trial (ECASS-3 [European Cooperative Acute Stroke Study]) that specifically enrolled patients between 3 and 4.5 hours after symptom onset, as well as from a meta-analysis of prior trials.

The use of IV alteplase within the 3- to 4.5-hour window has not been approved by the US Food and Drug Administration (FDA), although it is recommended by the 2019 AHA acute ischemic stroke guidelines for those who meet the ECASS-3 eligibility criteria (Table 8).

Endovascular Therapy

Substantial new high-quality research on the clinical efficacy of endovascular treatments of acute ischemic stroke was published in 2015. In light of that research, although IV alteplase remains as a first-line treatment, the AHA now recommends EVT for select patients with acute ischemic stroke due to an LVO.

As with fibrinolytic therapy, patients must meet inclusion criteria to be considered for this treatment. Similarly, better clinical outcomes are associated with reduced times from symptom onset to reperfusion, but these new treatment options offer the added benefit of expanding the

treatment window up to 24 hours from the onset of symptoms. Once you determine the patient is an EVT candidate, rapidly transport to cath lab or transfer to an EVT-capable center, followed by admittance to a neurologic ICU.

Mechanical Thrombectomy With Stent Retrievers

Mechanical thrombectomy has been demonstrated to provide clinical benefit in selected patients with acute ischemic stroke.

Patients arriving within 6 hours after symptom onset should receive EVT with a stent retriever if they meet all of the following criteria:

- Prestroke modified Rankin Score of 0 to 1
- Causative LVO of the internal carotid artery or proximal middle cerebral artery demonstrated on cerebrovascular imaging
- Age 18 years or older
- NIH Stroke Scale score of 6 or greater
- Alberta Stroke Program Early CT Score (ASPECTS) of 6 or greater (ASPECTS is an early, reliable tool that uses a 10-point quantitative topographic CT scan score to determine early ischemic changes.)
- Treatment can be initiated (groin puncture) within 6 hours after symptom onset or last known normal

In selected patients with acute ischemic stroke within 6 to 16 hours after last known normal who have LVO in the anterior circulation and meet other DAWN (Clinical Mismatch in the Triage of Wake Up and Late Presenting Strokes Undergoing Neurointervention With Trevo) or DEFUSE 3 (Endovascular Therapy Following Imaging Evaluation for Ischemic Stroke) eligibility criteria, mechanical thrombectomy is recommended.

In selected patients with acute ischemic stroke within 16 to 24 hours after last known normal who have LVO in the anterior circulation and meet other DAWN eligibility criteria, mechanical thrombectomy is reasonable.

Intra-Arterial Alteplase

Initial treatment with intra-arterial thrombolysis is beneficial for carefully selected patients with major ischemic strokes of less than 6 hours' duration caused by occlusions of the middle cerebral artery. Regarding the previous recommendations for intra-arterial thrombolysis, those data were derived from clinical trials that no longer reflect current practice, including the use of fibrinolytic drugs that are not available. A clinically beneficial dose of intra-

arterial alteplase is not established, and alteplase does not have FDA approval for intra-arterial use. As a consequence, mechanical thrombectomy with stent retrievers is recommended over intra-arterial thrombolysis as first-line therapy. Intra-arterial thrombolysis initiated within 6 hours after stroke onset in carefully selected patients who have contraindications to the use of IV alteplase might be considered, but the consequences are unknown.

Stroke Systems of Care

Recent clinical trials suggest that all patients eligible for EVT should be considered for that treatment in addition to IV alteplase. Regional stroke systems of care for acute ischemic stroke need to be in place so that eligible patients can be quickly transported from the field per local designation protocols or transferred from non-EVT centers to comprehensive or thrombectomy-capable stroke centers that offer these treatments.

Begin General Stroke Care

After being considered for reperfusion strategies, all patients should be placed on an acute stroke pathway. The general care of all patients with stroke includes the following actions:

- Begin acute stroke pathway.
- Assess ABCs, and give oxygen if needed.
- Monitor blood glucose.
- Monitor blood pressure.
- Monitor temperature.
- Perform dysphagia screening.
- Monitor for complications of stroke and fibrinolytic therapy.
- Transfer to a higher level of care (EVT, neurological ICU) if indicated.

Begin Stroke Pathway

Admit patients to a stroke unit (if available) for careful observation, including monitoring of blood pressure and neurologic status. If neurologic status worsens, order an emergent CT scan. Determine if cerebral edema or hemorrhage is the cause; consult neurosurgery as appropriate.

Additional stroke care includes support of the airway, oxygenation, ventilation, and nutrition. Provide normal saline to maintain intravascular volume (eg, approximately 75 to 100 mL/h) if needed.

Monitor Blood Glucose

Hyperglycemia is associated with worse clinical outcome in patients with acute ischemic stroke. Although there is no direct evidence that active glucose control improves clinical outcome, here is evidence that insulin treatment of hyperglycemia in other critically ill patients improves survival rates. For this reason, consider giving IV or subcutaneous insulin to lower blood glucose in patients with acute ischemic stroke when the serum glucose level is greater than 180 mg/dL.

Monitor for Complications of Stroke and Fibrinolytic Therapy

Prophylaxis for seizures is not recommended. But treatment of acute seizures followed by administration of anticonvulsants to prevent further seizures is recommended. Monitor the patient for signs of increased intracranial pressure such as increasing lethargy or decreasing level of consciousness or increased blood pressure with a concurrent decrease in heart rate. Continue to control blood pressure to reduce the potential risk of bleeding.

Table 9. Options to Treat Arterial Hypertension in Patients With Acute Ischemic Stroke Who Are Candidates for Emergency Reperfusion Therapy[8]

COR 2b	LOE C-EO
Patient otherwise eligible for emergency reperfusion therapy except that BP is >185/110 mm Hg: • Labetalol 10-20 mg IV over 1-2 minutes, may repeat 1 time; or • Nicardipine 5 mg/h IV, titrate up by 2.5 mg/h every 5-15 minutes, maximum 15 mg/h; when desired BP reached, adjust to maintain proper BP limits; or • Clevidipine 1-2 mg/h IV, titrate by doubling the dose every 2-5 minutes until desired BP reached; maximum 21 mg/h • Other agents (eg, hydralazine, enalaprilat) may also be considered If BP is not maintained ≤185/110 mm Hg, do not administer alteplase.	
Management of BP during and after alteplase or other emergency reperfusion therapy to maintain BP ≤180/105 mm Hg: • Monitor BP every 15 minutes for 2 hours from the start of alteplase therapy, then every 30 minutes for 6 hours, and then every hour for 16 hours.	
If systolic BP >180-230 mm Hg or diastolic BP >105-120 mm Hg: • Labetalol 10 mg IV followed by continuous IV infusion 2-8 mg/min; or	

- Nicardipine 5 mg/h IV, titrate up to desired effect by 2.5 mg/h every 5-15 minutes, maximum 15 mg/h; or
- Clevidipine 1-2 mg/h IV, titrate by doubling the dose every 2-5 minutes until desired BP reached; maximum 21 mg/h

If BP not controlled or diastolic BP >140 mm Hg, consider IV sodium nitroprusside.

Hypertension Management in Alteplase Candidates

Although management of hypertension in the stroke patient is controversial, patients who are candidates for fibrinolytic therapy should have their blood pressure controlled to lower the risk of intracerebral hemorrhage after administration of alteplase. General guidelines for the management of hypertension are outlined in Table 9.

Abbreviations: AIS, acute ischemic stroke; BP, blood pressure; COR, Class of Recommendation; IV, intravenous; LOE, Level of Evidence.

Different treatment options may be appropriate in patients who have comorbid conditions that may benefit from rapid reductions in BP, such as acute coronary heart failure, aortic dissection, or preeclampsia/eclampsia.

Data derived from Jauch et al.[9]

If a patient is eligible for fibrinolytic therapy, blood pressure must be 185 mm Hg or less systolic and 110 mm Hg or less diastolic to limit the risk of bleeding complications. Because the maximum interval from the onset of stroke until effective treatment of stroke with alteplase is limited, most patients with sustained hypertension above these levels will not be eligible for IV alteplase.

Managing arterial hypertension in patients not undergoing reperfusion strategies remains challenging. Data to guide recommendations for treatment are inconclusive or conflicting. Many patients have spontaneous declines in blood pressure during the first 24 hours after onset of stroke. Until more definitive data are available, the benefit of treating arterial hypertension in the setting of acute ischemic stroke is not well established (Class 2b; Level of Evidence C).[9] Patients who have malignant hypertension or other medical indications for aggressive treatment of blood pressure should be treated accordingly (revised from the previous guideline).[10]

Bradycardia

Overview

Bradycardia is generally defined as any rhythm disorder with a heart rate less than 60/min but for assessment and management of a patient with symptomatic bradycardia, it is typically defined as having a heart rate less than 50/min.

Managing bradycardia requires

- Differentiating between signs and symptoms caused by the slow rate vs those that are unrelated
- Correctly diagnosing the presence and type of atrioventricular (AV) block
- Using atropine as the drug intervention of first choice
- Deciding when to initiate transcutaneous pacing (TCP)
- Deciding when to start epinephrine or dopamine to maintain heart rate and blood pressure
- Knowing when to seek expert consultation about complicated rhythm interpretation, drugs, or management decisions or when to consider transvenous pacing
- Knowing the techniques and cautions for using TCP

Rhythms for Bradycardia

- Sinus bradycardia
- First-degree AV block
- Second-degree AV block: block of some, but not all, atrial impulses before they reach the ventricles. This block can be further classified as Mobitz type I or Mobitz type II second-degree AV block.
    - –Mobitz type I AV block:
        - Also known as *Wenckebach phenomenon*, typically occurs at the AV node. It is characterized by successive prolongation of the PR interval until an atrial impulse is not conducted to the ventricles (Figure 26B). The P wave corresponding to that atrial impulse is not followed by a QRS complex. The cycle of progressive lengthening of the PR interval until failure of conduction of the atrial impulse to the ventricles often repeats.
    - –Mobitz type II second-degree AV block (Figure 26C):

    - Occurs below the level of the AV node. It is characterized by intermittent nonconduction of P waves (atrial impulses to the ventricle) with a constant PR interval on conducted beats. There can be a consistent ratio of atrial to ventricular depolarizations, eg, 2 P waves to 1 QRS complex.
- Third-degree AV block

You should know the major AV blocks because important treatment decisions are based on the type of block (Figure 26). Complete (or third-degree) AV block is generally the most clinically significant block because it is most likely to cause cardiovascular collapse and require immediate pacing. Recognizing a stable bradycardia due to AV block is a primary goal, and recognizing the type of AV block is secondary.

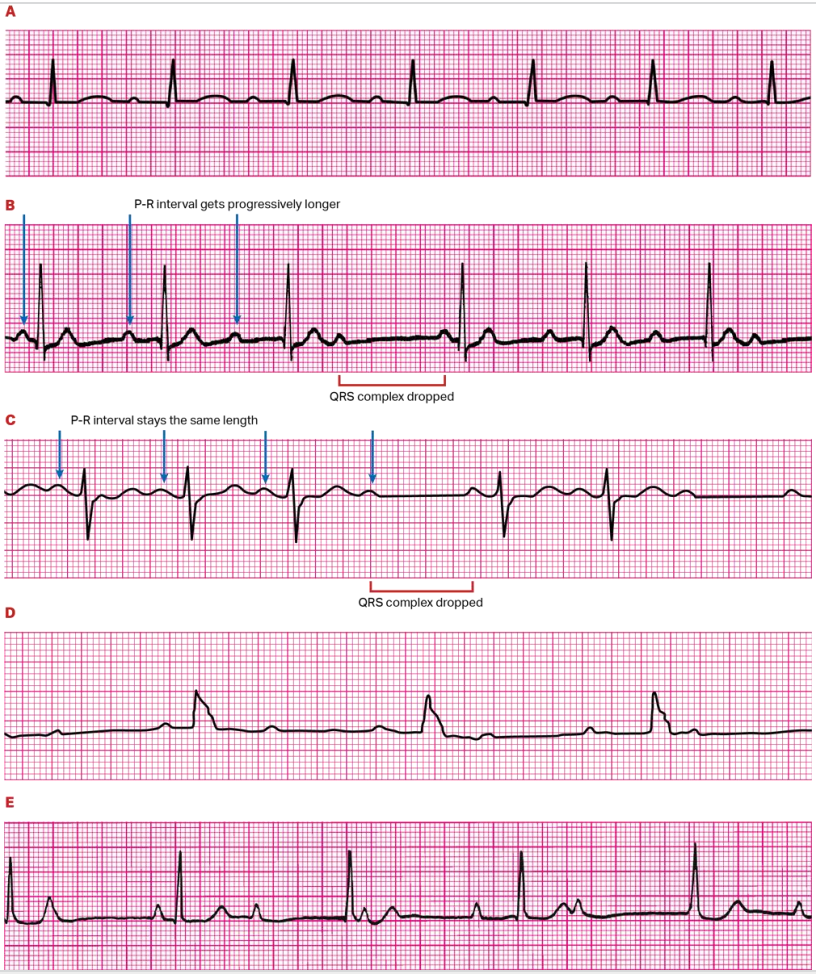


Figure 26. Examples of AV block. A, Sinus bradycardia with first-degree AV block. B, Second-degree AV block type I. C, Second-degree AV block type II. D, Complete AV block with a ventricular escape

pacemaker (wide QRS: 0.12 to 0.14 second). E, Third-degree AV block with a junctional escape pacemaker (narrow QRS: less than 0.12 second).

Drugs for Bradycardia

Drugs for bradycardia include

- Atropine
- Dopamine (infusion)
- Epinephrine (infusion)

Symptomatic Bradycardia

Bradycardia may have multiple causes, including some that are physiologic and require no assessment or therapy. For example, a healthy, well-trained athlete may have a resting heart rate less than 50/min.

In contrast, some patients have heart rates in the normal range, but these rates are inappropriate or insufficient for them. This is called a *functional* or *relative bradycardia*. For example, a heart rate of 70/min may be relatively too slow for a patient in cardiogenic or septic shock.

The key to managing symptomatic bradycardia is determining which signs or symptoms are due to the decreased heart rate. An unstable bradycardia exists clinically when 3 criteria are present:

1. The heart rate is slow.
2. The patient has symptoms.
3. The symptoms are due to the slow heart rate.

Signs and Symptoms

Unstable bradycardia leads to serious signs and symptoms that include

- Hypotension
- Acutely altered mental status
- Signs of shock
- Ischemic chest discomfort
- Acute heart failure
-

Managing Bradycardia: The Bradycardia Algorithm

The Adult Bradycardia Algorithm (Figure 27) outlines the steps for assessing and managing a patient who presents with unstable bradycardia with a pulse. Implementing this algorithm begins with identifying bradycardia (Step 1), which is typically when the heart rate is less than 50/min. First steps include the components of the BLS Assessment and the Primary Assessment.

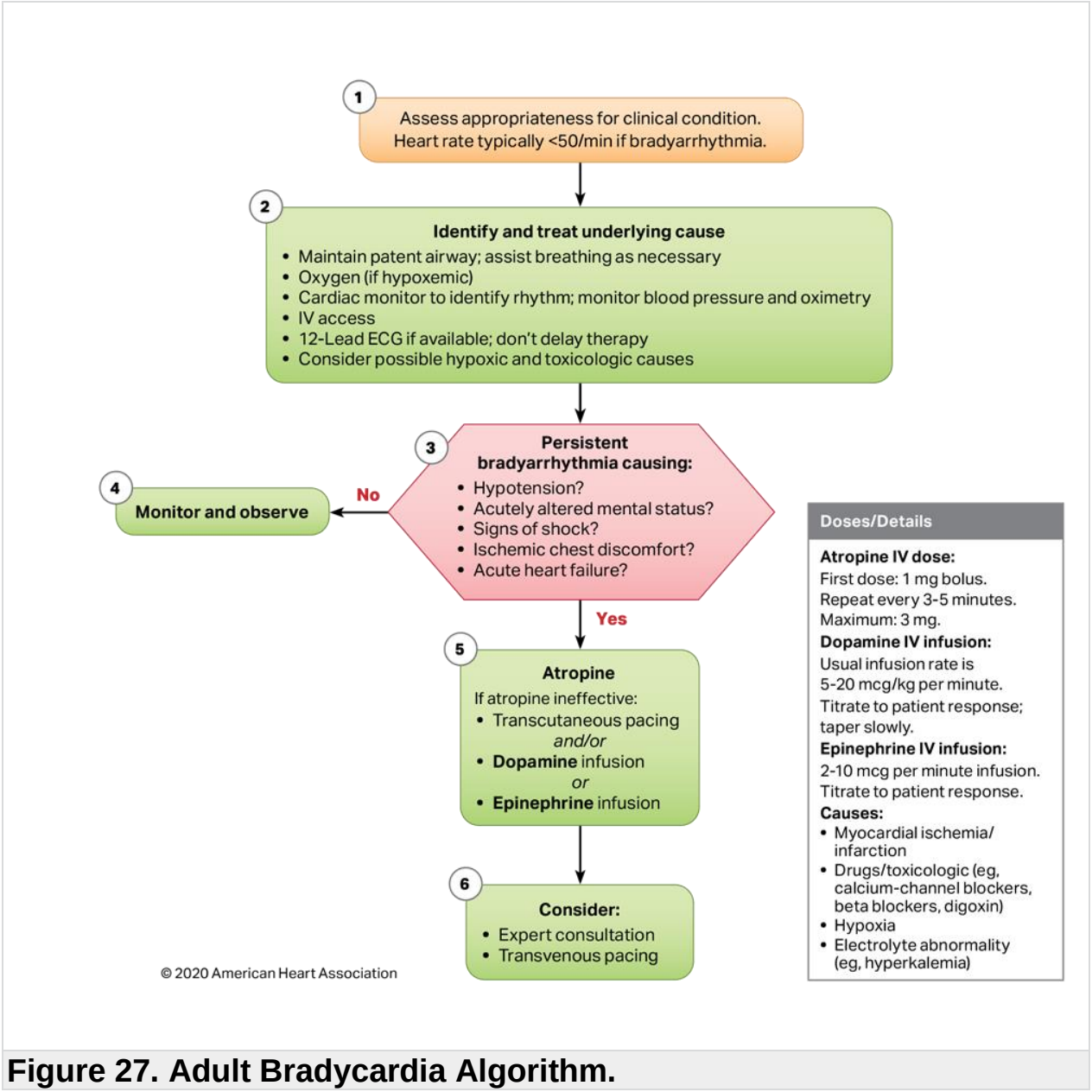


Figure 27. Adult Bradycardia Algorithm.

Identify and treat underlying causes (Step 2):

- Maintain patent airway; assist breathing as necessary.
- Give oxygen (if hypoxemic).
- Use a cardiac monitor to identify rhythm. Monitor blood pressure and oximetry.
- Establish IV access.
- Obtain a 12-lead ECG if available (Step 2).
- Consider possible hypoxic and toxicologic causes.

In the differential diagnosis, the primary decision point in the algorithm is to determine if the patient has signs or symptoms of poor perfusion and if these are caused by the bradycardia (Step 3). If there are no signs of poor perfusion, monitor and observe (Step 4). If there are signs of poor perfusion, administer atropine (Step 5). If atropine is ineffective, prepare for TCP and/or consider dopamine or epinephrine infusion (Step 5). If indicated, seek expert consultation and consider transvenous pacing (Step 6).

The severity of the patient's condition determines the treatment sequence in the algorithm, and you may need to implement multiple interventions simultaneously. If cardiac arrest develops, go to the Adult Cardiac Arrest Algorithm.

Applying the Adult Bradycardia Algorithm

In this case, a patient presents with symptoms of bradycardia. You conduct appropriate assessment and interventions as outlined in the Adult Bradycardia Algorithm while searching for and treating possible contributing factors.

Identify Bradycardia

Identify whether the heart rate is

- Bradycardia by definition, ie, heart rate typically less than 50/min
- Inadequate for the patient's condition (functional or relative)

Identify and Treat Underlying Causes

Perform the Primary Assessment, including the following:

- **A:** Maintain patent airway.
- **B:** Assist breathing as necessary; give oxygen in case of hypoxia; monitor oxygen saturation.
- **C:** Monitor blood pressure, oximetry, and heart rate; obtain and review a 12-lead ECG; establish IV access.

- **D and E:** Conduct a problem-focused history and physical examination; search for possible hypoxic and toxicologic causes, and treat possible contributing factors.

Critical Concepts: Bradycardia

- *Bradycardia can be a sign of life-threatening hypoxia.*
- *Bradycardia associated with hypertension can be a sign of a life-threatening increase in intracranial pressure, especially in the setting of stroke or brain injury.*

Are Signs or Symptoms Caused by Persistent Bradyarrhythmia

Look for these adverse signs and symptoms of the bradycardia:

- **Symptoms: acutely altered mental status, signs of shock, ischemic chest discomfort**
- **Signs: hypotension, acute heart failure**
- Are the signs and symptoms related to the slow heart rate?

Sometimes the symptom is not due to the bradycardia. For example, hypotension associated with bradycardia may be due to myocardial dysfunction rather than the bradycardia. Keep this in mind when you reassess the patient's response to treatment.

Critical Concepts: Bradycardia

The key clinical question is whether the bradycardia is causing the patient's symptoms or some other illness is causing the bradycardia.

Assess for Adequate Perfusion?

You must now decide if the patient has adequate or poor perfusion.

- If the patient has **adequate perfusion**, monitor and observe (Step 4).
- If the patient has persistent bradyarrhythmia causing **poor perfusion**, proceed to Step 5.

Treatment Sequence Summary

If the patient has poor perfusion secondary to bradycardia, treat as follows:

- Give atropine as first-line treatment: atropine 1 mg IV—may repeat to a total dose of 3 mg IV.

- *If atropine is ineffective,* provide transcutaneous pacing and/or dopamine 5 to 20 mcg/kg per minute infusion (chronotropic or heart rate dose) or epinephrine 2 to 10 mcg/min infusion.

The severity of the patient's clinical presentation determines the treatment sequence. For patients with unstable bradycardia, move quickly through this sequence. These patients may be in pre–cardiac arrest and may need multiple interventions simultaneously.

Avoid relying on atropine in type II second-degree or third-degree AV block or in patients with third-degree AV block with a new wide QRS complex where the location of the block is likely to be in infranodal tissue (such as in the bundle of His or more distal conduction system).

Treatment Sequence: Atropine

If you find no immediately reversible causes, atropine remains the first-line drug for acute stable bradycardia. Atropine sulfate acts by reversing cholinergic-mediated decreases in the heart rate and AV node conduction. Dopamine and epinephrine may be successful as an alternative to TCP.

For bradycardia, give atropine 1 mg IV every 3 to 5 minutes (maximum total dose of 3 mg IV). Note that atropine doses of less than 0.5 mg IV may further slow the heart rate.

Use atropine cautiously in the presence of acute coronary ischemia or myocardial infarction (MI). An atropine-mediated increase in heart rate may worsen ischemia or increase infarct size.

Do not rely on atropine in Mobitz type II second-degree or third-degree AV block or in patients with third-degree AV block with a new wide QRS complex. These bradycardias likely will not respond to reversal of cholinergic effects by atropine; preferably, treat them with TCP or β-adrenergic support as temporizing measures while the patient is prepared for transvenous pacing. Atropine administration should not delay external pacing or β-adrenergic infusion for patients with impending cardiac arrest.

A β-adrenergic infusion (ie, dopamine, epinephrine) is not a first-line agent for treating unstable bradycardia, but it can be used as an alternative when a bradycardia is unresponsive to treatment with atropine. You can also use a β-adrenergic infusion as a temporizing measure while the patient is prepared for transvenous pacing.

Vasopressors do not increase survival from bradycardia. Because these medications can improve aortic diastolic blood pressure, coronary artery perfusion pressure, and the rate of ROSC, the AHA continues to recommend their use.

Alternative drugs may also be appropriate in special circumstances such as the overdose of a β-blocker or calcium channel blocker. Do not wait for a maximum dose of atropine if the patient presents with second-degree or third-degree block; rather, move to a second-line treatment after 2 to 3 doses of atropine.

Treatment Sequence: TCP

TCP may be useful to treat unstable bradycardia. TCP is noninvasive and can be performed by ACLS providers. Consider immediate pacing in unstable patients with high-degree heart block when IV access is not available. It is reasonable to initiate TCP in unstable patients who do not respond to atropine.

After initiating TCP, confirm electrical and mechanical capture (Figure 28). Because heart rate is a major determinant of myocardial oxygen consumption, set the pacing to the lowest effective rate based on clinical assessment and symptom resolution. Reassess the patient for symptom improvement and hemodynamic stability. Give analgesics and sedatives for pain control. Note that many of these drugs may further decrease blood pressure and affect the patient's mental status. Try to identify and correct the cause of the bradycardia.

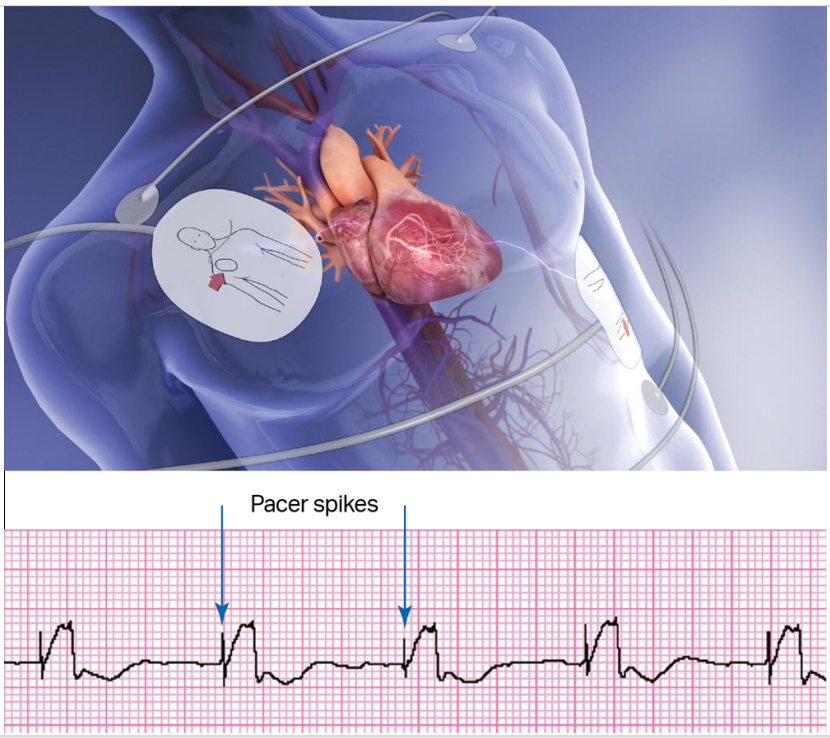


Figure 28. Transcutaneous pacing.

TCP has its limitations—it can be painful and may not produce effective electrical and mechanical capture. If bradycardia is not causing the symptoms, TCP may be ineffective despite capture. For these reasons, consider TCP as an emergent bridge to transvenous pacing in patients with significant sinus bradycardia or AV block.

If you chose TCP as the second-line treatment and it is also ineffective (eg, inconsistent capture), begin an infusion of dopamine or epinephrine and prepare for possible transvenous pacing by obtaining expert consultation.

Sedation and Pacing

Most conscious patients should be sedated before pacing. If the patient is in cardiovascular collapse or rapidly deteriorating, you may need to start pacing without prior sedation, particularly if sedation drugs are not immediately available. Evaluate the need for sedation in light of the patient's condition and need for immediate pacing. A review of sedation drugs is beyond the scope of this course, but the general approach could include the following:

- Give a parenteral narcotic for analgesia.
- Give parenteral benzodiazepine for anxiety and muscle contractions.

- Use a chronotropic infusion once available.
- Obtain expert consultation for transvenous pacing.

Treatment Sequence: Epinephrine, Dopamine

Although β-adrenergic agonists with rate-accelerating effects are not first-line agents for treating stable bradycardia, they are alternatives to TCP or in special circumstances, such as overdose with a β-blocker or calcium channel blocker.

Because epinephrine and dopamine are vasoconstrictors as well as chronotropes, healthcare providers must assess the patient's intravascular volume status and avoid hypovolemia when using these drugs. Dobutamine (a β-adrenergic agonist) is appropriate when vasoconstriction is not desired.

Either epinephrine infusions or dopamine infusions may be used for patients with stable bradycardia, particularly if associated with hypotension, for whom atropine may be inappropriate or after atropine fails.

Begin epinephrine infusion at a dose of 2 to 10 mcg/min and titrate to patient response; begin dopamine infusion at 5 to 20 mcg/kg per minute and titrate to patient response. At lower doses, dopamine has a more selective effect on inotropy and heart rate; at higher doses (greater than 10 mcg/kg per minute infusion), it also has vasoconstrictive effects.

Next Actions

After considering the treatment sequence in Step 5, you may need to

- Consider expert consultation—but do not delay treatment if the patient is unstable or potentially unstable.
- Prepare the patient for transvenous pacing.

Transcutaneous Pacing

Many devices can pace the heart by delivering an electrical stimulus, causing electrical depolarization and subsequent cardiac contraction, and TCP delivers pacing impulses to the heart through the skin via cutaneous electrodes. Most defibrillator manufacturers have added a pacing mode to manual defibrillators. Performing TCP is often as close as the nearest defibrillator, but you should know the indications, techniques, and hazards for using TCP.

Indications and Precautions

Indications for TCP are as follows:

- Hemodynamically unstable bradycardia (eg, hypotension, acutely altered mental status, signs of shock, ischemic chest discomfort, acute heart failure hypotension)
    - –Unstable clinical condition likely due to the bradycardia
- Bradycardia with stable ventricular escape rhythms

Precautions for TCP are as follows:

- TCP is contraindicated in severe hypothermia.
- Conscious patients require analgesia for discomfort unless delay for sedation will cause or contribute to deterioration.
- Do not assess the carotid pulse to confirm mechanical capture; electrical stimulation causes muscular jerking that may mimic the carotid pulse.

Technique

Perform TCP by following these steps:

1. Place pacing electrodes on the chest according to package instructions.
2. Turn the pacer on.
3. Set the demand rate to 60 to 80/min. You can adjust this rate up or down (based on patient clinical response) once pacing is established.
4. Set the current milliamperes output 2 mA above the dose at which consistent capture is observed (safety margin).

External pacemakers have either *fixed* rates (asynchronous mode) or *demand* rates.

Assess Response to Treatment

Signs of hemodynamic impairment include hypotension, acutely altered mental status, signs of shock, ischemic chest discomfort, acute heart failure, or other signs of shock related to the bradycardia. The goal of therapy is to improve these signs and symptoms rather than target a precise heart rate. Start pacing at a rate of 60 to 80/min. Once pacing is initiated, adjust the rate based on the patient's clinical response.

Consider giving atropine before pacing in mildly symptomatic patients. Do not delay pacing for unstable patients, particularly those with high-degree

AV block. Atropine may increase heart rate, improve hemodynamics, and eliminate the need for pacing. If atropine is ineffective or likely to be ineffective, or if IV access or atropine administration is delayed, begin pacing as soon as it is available.

Patients with ACS should be paced at the lowest heart rate that allows clinical stability. Higher heart rates can worsen ischemia because heart rate is a major determinant of myocardial oxygen demand. Ischemia, in turn, can precipitate arrhythmias.

If unstable bradycardia does not respond to atropine, consider a chronotropic drug infusion to stimulate heart rate as an alternative to pacing:

- Epinephrine: administer at 2 to 10 mcg/min infusion and titrate to patient response.
- Dopamine: administer at 5 to 20 mcg/kg per minute infusion and titrate to patient response.

Bradycardia With Escape Rhythms

A bradycardia may lead to secondary bradycardia-dependent ventricular rhythms. When a patient's heart rate falls, an electrically unstable ventricular area may "escape" suppression by higher and faster pacemakers (eg, sinus node), especially in the setting of acute ischemia. These ventricular rhythms often fail to respond to drugs. With severe bradycardia, some patients will develop wide-complex ventricular beats that can precipitate VT or VF. Pacing may increase the heart rate and eliminate bradycardia-dependent ventricular rhythms. However, an accelerated idioventricular rhythm (sometimes called AIVR) may occur in the setting of inferior wall MI. This rhythm is usually stable and does not require pacing.

Patients with ventricular escape rhythms may have normal myocardium with disturbed conduction. After correcting electrolyte abnormalities or acidosis, use pacing to stimulate effective myocardial contractions until the conduction system recovers.

Standby Pacing

Acute ischemia of conduction tissue and pacing centers can cause several bradycardic rhythms in ACS. Patients who are clinically stable may decompensate suddenly or become unstable over minutes to hours due to worsening conduction abnormalities, and these bradycardias may deteriorate to complete AV block and cardiovascular collapse. To prepare

for this clinical deterioration, place TCP electrodes on any patient with acute myocardial ischemia or infarction associated with the following rhythms:

- Symptomatic sinus node dysfunction with severe and symptomatic sinus bradycardia
- Asymptomatic Mobitz type II second-degree AV block
- Asymptomatic third-degree AV block
- Newly acquired left, right, or alternating bundle branch block or bifascicular block in the setting of AMI

Tachycardia: Stable and Unstable

Overview

The Team Leader in this case will assess and manage a patient with a rapid, unstable heart rate. You must be able to classify the tachycardia and intervene appropriately as outlined in the Adult Tachycardia With a Pulse Algorithm. You will be evaluated on your knowledge of the factors involved in safe and effective synchronized cardioversion as well as your performance of the procedure.

Rhythms for Unstable Tachycardia

This case involves these ECG rhythms (examples in Figure 29):

- Sinus tachycardia
- Atrial fibrillation
- Atrial flutter
- Supraventricular tachycardia (SVT)
- Monomorphic VT
- Polymorphic VT
- Wide-complex tachycardia of uncertain type

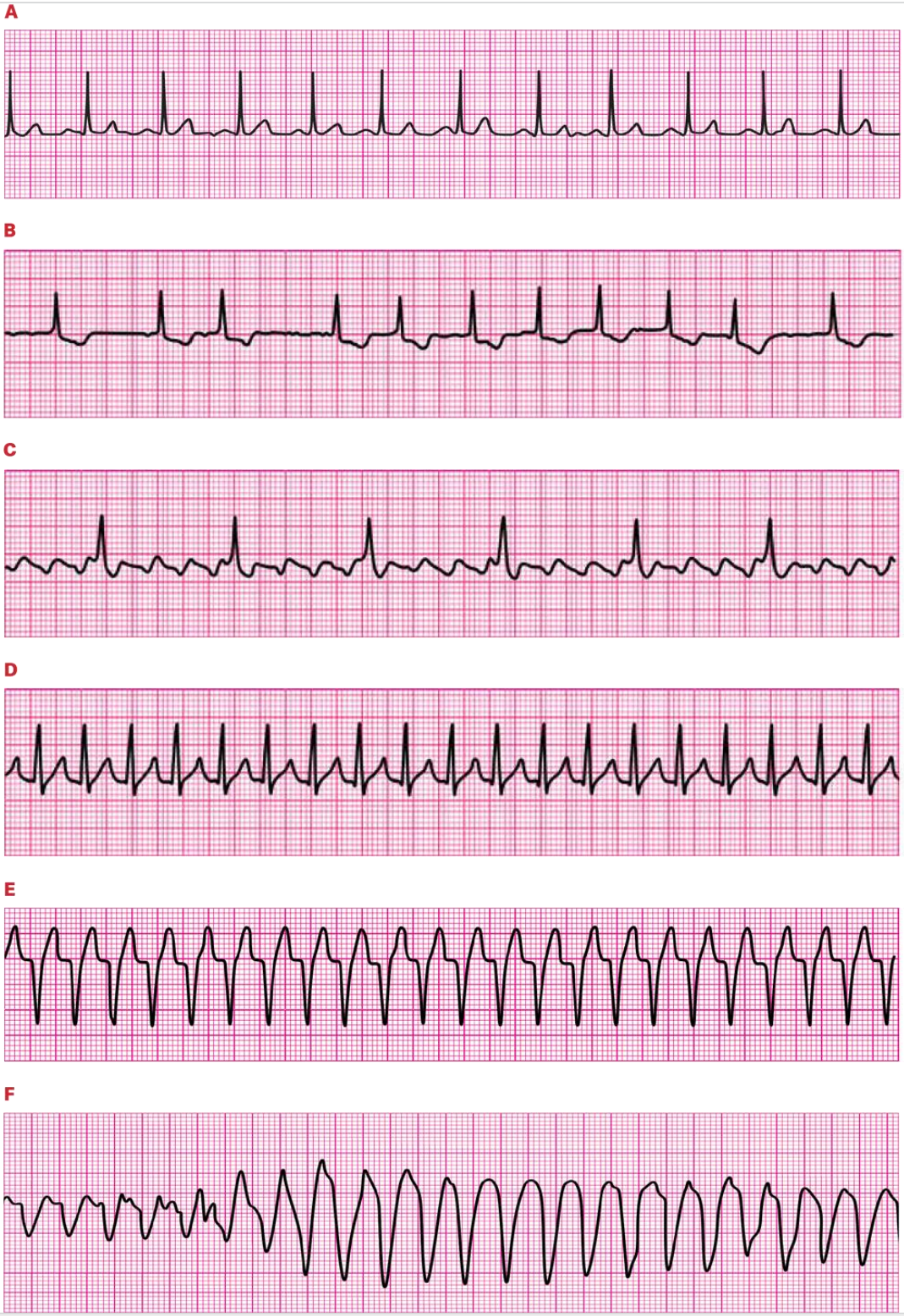


Figure 29. Examples of tachycardias. A, Sinus tachycardia. B, Atrial fibrillation. C, Atrial flutter. D, Supraventricular tachycardia. E, Monomorphic ventricular tachycardia. F, Polymorphic ventricular tachycardia.

Drugs for Unstable Tachycardia

Drugs are generally not used to manage patients with unstable tachycardia; rather, immediate cardioversion is recommended. Consider administering sedative drugs in conscious patients, but do not delay immediate cardioversion in unstable patients.

Approach to Unstable Tachycardia

A tachycardia—that is, a heart rate greater than 100/min—has many potential causes and may be symptomatic or asymptomatic. The key to managing a patient with any tachycardia is to assess the appropriateness for the clinical condition and determine whether pulses are present. If pulses are present, determine whether the patient is stable or unstable, and then provide treatment based on the patient's condition and rhythm.

If the tachycardia is sinus tachycardia, conduct a diligent search for the cause of the tachycardia. Treating and correcting this cause will improve the patient's signs and symptoms. Cardioversion is not indicated for tachycardia.

Definitions

Definitions used in this case are as follows:

- *Tachycardia:* defined as an arrhythmia with a heart rate typically 100/min or greater
- *Symptomatic tachycardia:* signs and symptoms due to the rapid heart rate
- The rate takes on clinical significance at its extremes and is more likely attributable to an arrhythmia if the heart rate is 150/min or greater.
- It is unlikely that symptoms of instability are caused primarily by the tachycardia when the heart rate is less than 150/min unless the patient has impaired ventricular function.

Pathophysiology of Unstable Tachycardia

Unstable tachycardia exists when the heart rate is too fast for the patient's clinical condition. This excessive heart rate causes symptoms or an unstable condition because the heart is

- *Beating so fast* that cardiac output is reduced; this can cause pulmonary edema, coronary ischemia, and hypotension with reduced blood flow to vital organs (eg, brain, kidneys)

- *Beating ineffectively* so that coordination between the atrium and ventricles or the ventricles themselves reduces cardiac output

Signs and Symptoms

Unstable tachycardia leads to serious signs and symptoms that include

- Hypotension
- Acutely altered mental status
- Signs of shock
- Ischemic chest discomfort
- Acute heart failure

Rapid Recognition

The 2 keys to managing unstable tachycardia are rapidly recognizing that

1. The patient is significantly symptomatic or even unstable
2. The signs and symptoms are caused by the tachycardia

Quickly determine whether the tachycardia is producing hemodynamic instability and the serious signs and symptoms *or* the serious signs and symptoms (eg, the pain and distress of an AMI) are the cause of the tachycardia.

Making this determination can be difficult. Many experts suggest that when a heart rate is less than 150/min, the symptoms of instability are not likely caused primarily by the tachycardia unless ventricular function is impaired. A heart rate typically less than 150/min is usually an appropriate response to physiologic stress (eg, fever, dehydration) or other underlying conditions.

Assess frequently for the presence or absence of signs and symptoms and for their severity.

Indications for Cardioversion

Rapidly identifying symptomatic tachycardia will help you determine whether to prepare for immediate cardioversion:

- At heart rates typically 150/min or greater, symptoms are often present and cardioversion is often required in unstable patients.
- If the patient is seriously ill or has underlying cardiovascular disease, symptoms may be present at lower rates.

You must know when cardioversion is indicated, how to prepare the patient for it (including appropriate medication), and how to switch the defibrillator/monitor to operate as a cardioverter.

Caution: Sinus Tachycardia

Never cardiovert a patient who has a sinus rhythm.

Managing Unstable Tachycardia: The Adult Tachycardia With a Pulse Algorithm

The Adult Tachycardia With a Pulse Algorithm simplifies initial management of tachycardia. The presence or absence of pulses is considered the key to managing patients with any tachycardia. If a pulseless tachycardia is present, then manage the patient according to the PEA pathway of the Adult Cardiac Arrest Algorithm (Figure 41). If pulses are present, assess appropriateness for the clinical condition and determine whether the patient is stable or unstable, and then provide treatment based on the patient's condition and rhythm Step 1). Identify and treat underlying causes by doing the following (Step 2):

- Maintain patent airway; assist breathing as necessary.
- Give oxygen (if hypoxemic).
- Use a cardiac monitor to identify rhythm; monitor blood pressure and oximetry.
- Obtain IV access.
- Obtain a 12-lead ECG (if available).

Determine if the persistent tachyarrhythmia is causing (Step 3)

- Hypotension
- Acutely altered mental status
- Signs of shock
- Ischemic chest discomfort
- Acute heart failure

To manage unstable tachycardia, ACLS providers should consider synchronized cardioversion and sedation, and, if regular narrow complex, adenosine 6 mg IV (follow with saline flush) (Step 4). If these interventions are not successful and if the tachycardia is refractory, providers should look for any underlying causes and consider the need to increase the energy level for the next cardioversion and add antiarrhythmic drugs. Providers should also obtain expert consultation (Step 5). Actions in the steps require advanced knowledge of ECG rhythm interpretation and antiarrhythmic therapy; these actions should take place in-hospital with expert consultation available.

The Adult Tachycardia With a Pulse Algorithm (Figure 30) outlines the steps for assessing and managing a patient presenting with symptomatic tachycardia with pulses. Implementation of this algorithm begins with the identification of tachycardia with pulses (Step 1). If a tachycardia and a pulse are present, identify and treat underlying causes and perform assessment and management steps guided by the BLS, Primary, and Secondary Assessments (Step 2). The key in this assessment is to decide whether the tachycardia is stable or unstable.

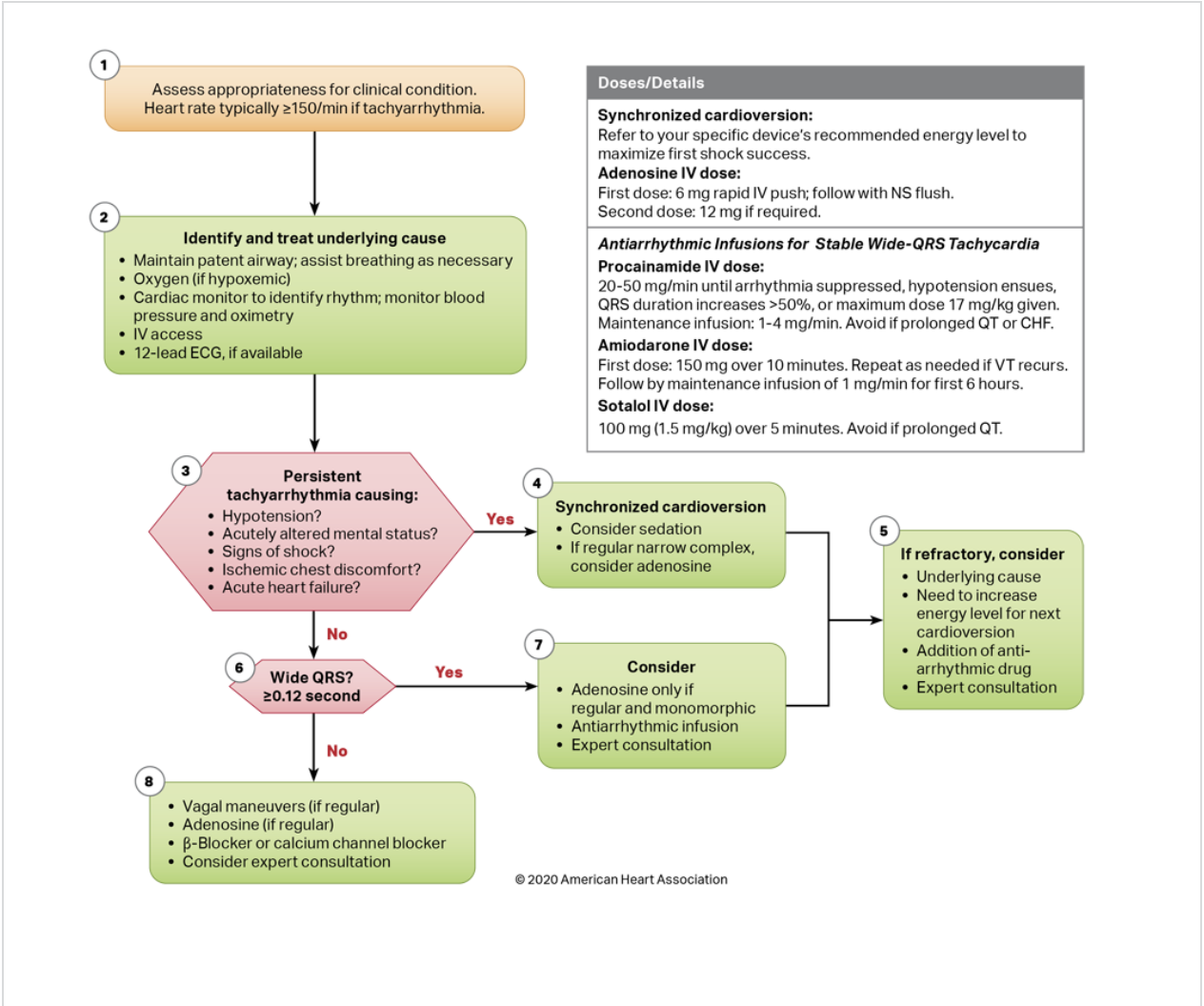


Figure 30. Adult Tachycardia With a Pulse Algorithm.

The tachycardia is unstable if signs and symptoms persist after maintaining the patent airway, assisting with breathing as necessary, the patient receives supplemental oxygen, *and* if significant signs or symptoms are due to the tachycardia (Step 3). In this case, immediate synchronized

cardioversion is indicated (Step 4). If cardioversion is unsuccessful, consider next steps (Step 5).

If the patient is stable, evaluate the ECG and determine if the QRS complex is wide (0.12 second or greater) and whether it is regular or irregular (Step 6). (*Note:* the treatment of stable tachycardia is presented in the next case.)

Serious Signs and Symptoms, Unstable Condition

Intervention is determined by the presence of serious signs and symptoms or by an unstable condition resulting from the tachycardia. Serious signs and symptoms include hypotension, acutely altered mental status, signs of shock, ischemic chest discomfort, and acute heart failure. Ventricular rates less than 150/min usually do not cause serious signs or symptoms.

These key questions in the Adult Tachycardia With a Pulse Algorithm will guide your assessment of this patient and help determine your next steps:

- Are symptoms present or absent?
- Is the patient stable or unstable?
- Is there a wide QRS (0.12 second or greater)?
- Is the rhythm regular or irregular?
- Is the QRS monomorphic or polymorphic?

Applying the Adult Tachycardia With a Pulse Algorithm to Unstable Patients

In this case, you have a patient with tachycardia and a pulse. Conduct the steps in the Adult Tachycardia With a Pulse Algorithm to evaluate and manage the patient.

Assess Clinical Condition

Use the BLS, Primary, and Secondary Assessments to guide your approach.

- Assess appropriateness for clinical condition (Step 1):
    - –Look for signs of increased work of breathing (tachypnea, intercostal retractions, suprasternal retractions, paradoxical abdominal breathing), and hypoxemia as determined by pulse oximetry.

Identify and Treat the Underlying Cause

Identify and treat underlying cause (Step 2).

- Maintain patent airway; assist breathing as necessary.
- Give oxygen (if hypoxemic).
- Use a cardiac monitor to identify rhythm; monitor blood pressure and oximetry.
- Establish IV access.
- Obtain a 12-lead ECG if available.

If symptoms persist despite support of adequate oxygenation and ventilation, proceed to Step 3.

Critical Concepts: Unstable Patients

- *Obtain a 12-lead ECG (if available) early in the assessment to better define the rhythm.*
- *However, unstable patients require immediate cardioversion.*
- *Do not delay immediate cardioversion to acquire the 12-lead ECG if the patient is unstable.*

Decision Point: Is the Persistent Tachycardia Causing Serious Signs or Symptoms?

Assess the patient's degree of instability and determine if it is related to the tachycardia (Step 3).

Unstable

If the persistent tachyarrhythmia is causing the patient to demonstrate rate-related cardiovascular compromise with serious signs and symptoms, proceed to immediate synchronized cardioversion (Step 4).

Serious signs and symptoms are unlikely if the ventricular rate is less than 150/min in patients with a healthy heart. However, if the patient is seriously ill or has significant underlying heart disease or other conditions, symptoms may be present at a lower heart rate.

Stable

If the patient does not have rate-related cardiovascular compromise, proceed to Step 6. You'll have time to obtain a 12-lead ECG, evaluate the rhythm, determine the width of the QRS, and determine treatment options. For stable patients, seek expert consultation because treatment has the potential for harm.

Treatment Based on Type of Tachycardia

You may not always be able to distinguish between supraventricular and ventricular rhythms. Most wide-complex tachycardias are ventricular in origin, especially if the patient has underlying heart disease or is older. If the patient is pulseless, treat the rhythm as VF and follow the Adult Cardiac Arrest Algorithm.

If the patient has a wide-complex tachycardia and is unstable, assume it is VT until proven otherwise. The amount of energy required for cardioversion of VT is determined by the specific device's recommended energy level to maximize first shock success.

- If the patient is unstable but has a pulse with regular uniform wide-complex VT (monomorphic VT), treat with synchronized cardioversion. Follow your device's specific recommended energy level to maximize the success of the first shock. If the patient does not respond to the first shock, increasing the dose stepwise is reasonable. (This recommendation represents expert opinion.)
- Arrhythmias with a polymorphic QRS appearance (polymorphic VT), such as torsades de pointes, will usually not permit synchronization. If the patient has polymorphic VT, treat as VF with high-energy unsynchronized shocks (eg, defibrillation doses).
- If you have any doubt about whether an unstable patient has monomorphic or polymorphic VT, do not delay treatment for further rhythm analysis. Provide high-energy, unsynchronized shocks (defibrillation doses).

Perform Immediate Synchronized Cardioversion

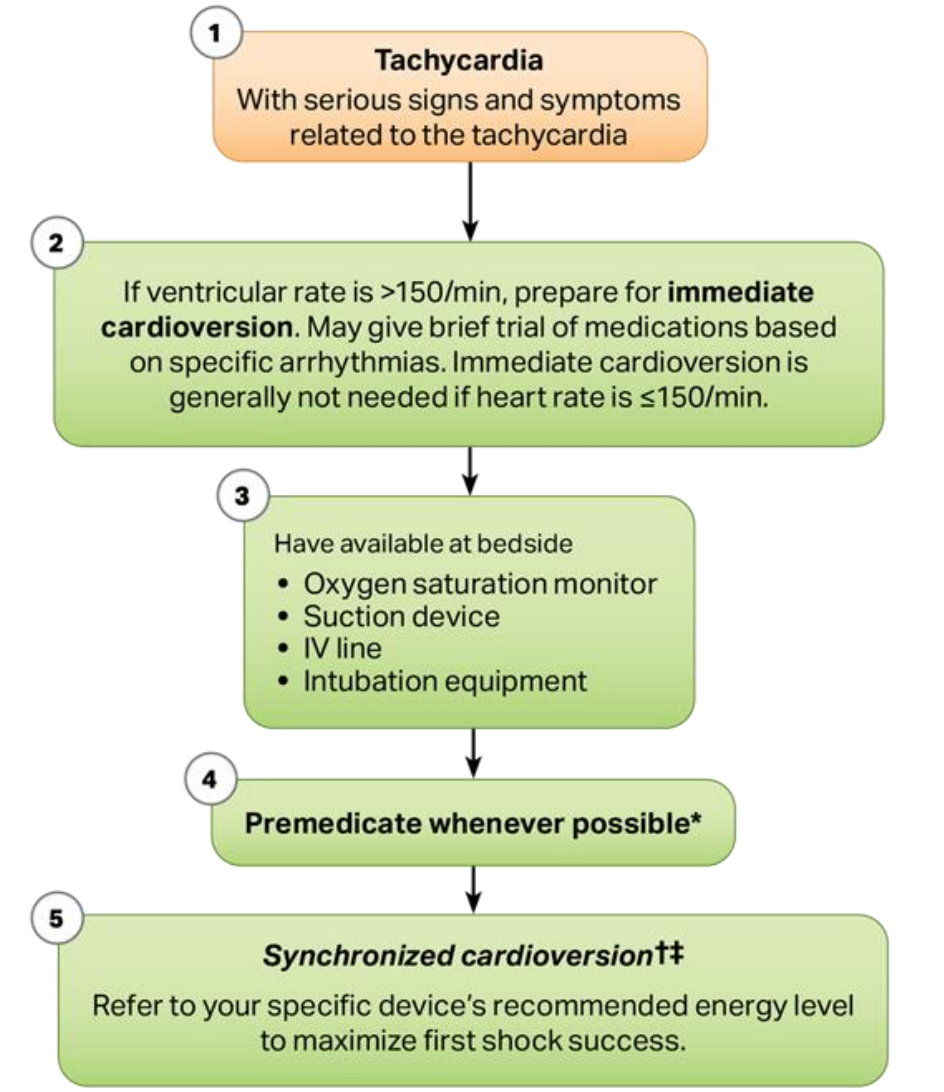
- If possible, establish IV access before cardioversion and administer sedation if the patient is conscious.
- Do not delay cardioversion if the patient is extremely unstable.

If the patient with a regular narrow-complex SVT or a monomorphic wide-complex tachycardia is not hypotensive, healthcare providers may administer adenosine 6 mg IV (follow with saline flush) while preparing for synchronized cardioversion.

If cardiac arrest develops, see the Adult Cardiac Arrest Algorithm.

Cardioversion

You must know when cardioversion is indicated and what type of shock to administer (Figure 31). Before cardioversion, establish IV access and sedate the responsive patient if possible, but do not delay cardioversion in unstable or deteriorating patients.



Notes

*Effective regimens have included a sedative **(eg, diazepam, midazolam, etomidate, methohexital, propofol)** with or without an analgesic agent **(eg, fentanyl, morphine)**. Many experts recommend anesthesia if service is readily available.

†Note possible need to resynchronize after each cardioversion.

‡If delays in synchronization occur and clinical condition is critical, go immediately to unsynchronized shocks.

Figure 31. Electrical Cardioversion Algorithm.

This section discusses the difference between unsynchronized and synchronized shocks, potential problems with synchronization, and energy doses for specific rhythms.

Unsynchronized vs Synchronized Shocks

Modern defibrillators and cardioverters can deliver unsynchronized or synchronized shocks. An *unsynchronized shock* means that the electrical shock is delivered as soon as you push the shock button on the device. These shocks may fall randomly anywhere within the cardiac cycle and use higher energy levels than synchronized shocks. *Synchronized cardioversion* uses a sensor to deliver a shock that is synchronized with a peak of the QRS complex. When you engage the sync option, pressing the shock button can result in a delay before shocking because the device synchronizes the shock to the peak of the R wave, and this may require analysis of several complexes. Synchronization avoids delivering a shock during cardiac repolarization (represented on the surface ECG as the T wave), a period of vulnerability in which a shock can precipitate VF. Synchronized shocks also use a lower energy level than attempted defibrillation. Always deliver synchronized shocks in patients with a pulse unless there is polymorphic VT, synchronization is impossible, or there is a delay to treatment in the unstable patient.

Potential Problems With Synchronization

In theory, synchronization is simple: just push the sync control on the face of the defibrillator/cardioverter. In practice, however, synchronization has potential problems:

- If the R-wave peaks of a tachycardia are undifferentiated or of low amplitude, the monitor sensors may be unable to identify an R-wave peak and therefore will not deliver the shock.
- Many cardioverters will not synchronize through the handheld quick-look paddles. An unwary practitioner may try to synchronize—unsuccessfully in that the machine will not discharge—and may not recognize the problem.
- Synchronization can take extra time (eg, if you need to attach electrodes or are unfamiliar with the equipment).

Recommendations

Synchronized shocks are recommended for patients with a pulse and tachycardias such as

- Unstable SVT
- Unstable atrial fibrillation
- Unstable atrial flutter
- Unstable regular monomorphic tachycardia with pulses

Unsynchronized high-energy shocks are recommended

- For a patient with no pulse (VF/pVT)
- For clinical deterioration (in prearrest), such as those with severe shock or polymorphic VT, when you think a delay in converting the rhythm will result in cardiac arrest
- For patients who are unstable or deteriorating and synchronization cannot be immediately accomplished
- When you are unsure whether monomorphic or polymorphic VT is present in the unstable patient

If the shock causes VF (occurring in only a very small minority of patients despite the theoretical risk), immediately attempt defibrillation.

Energy Doses for Specific Rhythms

For dosing, follow your specific device's recommended energy level to maximize the success of the first shock.

Synchronized Cardioversion

Synchronized cardioversion is the treatment of choice when a patient has a symptomatic (unstable) reentry SVT or VT with pulses and is recommended to treat unstable atrial fibrillation and flutter.

Cardioversion is unlikely to be effective for treating junctional tachycardia or ectopic or multifocal atrial tachycardia because these rhythms have an automatic focus arising from cells that are spontaneously depolarizing at a rapid rate. Delivering a shock generally cannot stop these rhythms and may actually increase the rate of the tachyarrhythmia.

In synchronized cardioversion, shocks are administered through adhesive electrodes or handheld paddles with the defibrillator/monitor in synchronized (sync) mode. The sync mode delivers energy just after the R wave of the QRS complex.

Follow these steps to perform synchronized cardioversion, modifying the steps for your specific device.

1. Sedate all conscious patients unless unstable or deteriorating rapidly.
2. Turn on the defibrillator (monophasic or biphasic).
3. Attach monitor leads to the patient and ensure proper display of the patient's rhythm. Position adhesive electrode (conductor) pads on the patient.
4. Press the sync control button to engage the synchronization mode.
5. Look for markers on the R wave indicating sync mode.
6. Adjust monitor gain if necessary until sync markers occur with each R wave.
7. Select the appropriate energy level. Deliver synchronized shocks according to your device's recommended energy level to maximize the success of the first shock.
8. Announce to team members: "Charging defibrillator—stand clear!"
9. Press the charge button.
10. Clear the patient when the defibrillator is charged.
11. Press the shock button(s).
12. Check the monitor. If tachycardia persists, increase the energy level (joules) according to the device manufacturer's recommendations.
13. Activate the sync mode after delivery of each synchronized shock. Most defibrillators default back to the unsynchronized mode after delivery of a synchronized shock. This default allows an immediate shock if cardioversion produces VF.

Figure 31 shows the steps to perform electrical cardioversion. First, determine if the patient has serious signs and symptoms related to tachycardia (Step 1). If the heart rate is greater than 150/min, prepare for immediate cardioversion and consider giving a brief trial of medications on the basis of the specific arrhythmias. Immediate cardioversion is generally not needed if heart rate is 150/min or less (Step 2).

At the bedside, the provider should have the following available (Step 3):

- Oxygen saturation monitor
- Suction device
- IV line
- Intubation equipment

Next, premedicate whenever possible (Step 4). Effective regimens have included a sedative (eg, diazepam, midazolam, etomidate, methohexital,

propofol) with or without an analgesic agent (eg, fentanyl, morphine). Many experts recommend anesthesia if service is readily available.

Perform synchronized cardioversion (Step 5). Refer to your specific device's recommended energy level to maximize first shock success. Note possible need to resynchronize after each cardioversion. If delays in synchronization occur and the patient's clinical condition is critical, go immediately to unsynchronized shocks.

Stable Tachycardias

If the patient does not have rate-related cardiovascular compromise, proceed to Step 6. You'll have time to obtain a 12-lead ECG, evaluate the rhythm, determine if width of the QRS is 0.12 second or greater. In this case, consider adenosine only if the rhythm is regular and monomorphic, and consider antiarrhythmic infusion. Seek expert consultation because treatment has the potential for harm. If the rhythm is refractory, consider the underlying cause, the need to increase energy level for the next cardioversion, additional antiarrhythmic drugs, and additional expert consultation.

Determine the Width of the QRS Complex

- If the width of the QRS complex is 0.12 second or more, go to Step 7.
- If the width of the QRS complex is less than 0.12 second, go to Step 8.

In some cases, a "stable" tachycardia is actually an early sign that the patient is becoming unstable, and you should initiate a search for underlying causes early to avoid further deterioration.

You must be able to classify the type of tachycardia (wide or narrow; regular or irregular) and intervene appropriately as outlined in the Adult Tachycardia With a Pulse Algorithm. During this case, you will perform initial assessment and management of regular narrow-complex rhythms (except sinus tachycardia), and you'll treat them with vagal maneuvers, adenosine, β-blocker or calcium channel blocker.

If the rhythm does not convert, consider expert consultation. If the patient becomes clinically unstable, prepare for immediate unsynchronized shock or synchronized cardioversion.

Understanding Sinus Tachycardia

Sinus tachycardia is a heart rate that is greater than 100/min, has P waves, and is generated by sinus node discharge. The heart rate in tachycardia typically does not exceed 220/min and is age-related. Sinus tachycardia usually does not exceed 120 to 130/min, and it has a gradual onset and gradual termination. Reentry SVT has an abrupt onset and termination.

Note that sinus tachycardia is excluded from the Adult Tachycardia With a Pulse Algorithm. Sinus tachycardia is caused by external influences on the heart, such as fever, anemia, hypotension, blood loss, or exercise—systemic, not cardiac, conditions. Sinus tachycardia is a regular rhythm, although the rate may be slowed by vagal maneuvers. *In sinus tachycardia, the goal is to identify and correct the underlying systemic cause, and cardioversion is contraindicated.*

β-Blockers may cause clinical deterioration if the cardiac output falls when a compensatory tachycardia is blocked. This is because cardiac output is determined by the volume of blood ejected by the ventricles with each contraction (stroke volume) and the heart rate.

Cardiac output (CO) = Stroke volume (SV) × Heart rate

If a condition such as a large AMI limits ventricular function (severe heart failure or cardiogenic shock), the heart compensates by increasing the heart rate. If you attempt to reduce the heart rate in patients with a compensatory tachycardia, cardiac output will fall, and the patient's condition will likely deteriorate.

Rhythms for Stable Tachycardia

Tachycardia classifications include the appearance of the QRS complex, heart rate, and whether they are regular or irregular:

- Narrow–QRS complex (SVT) tachycardias (QRS less than 0.12 second) in order of frequency
    - –Sinus tachycardia
    - –Atrial fibrillation
    - –Atrial flutter
    - –AV nodal reentry
- Wide–QRS complex tachycardias (QRS 0.12 second or more)
    - –Monomorphic VT
    - –Polymorphic VT
    - –SVT with aberrancy

- Regular or irregular tachycardias
    - –Irregular narrow-complex tachycardias are probably atrial fibrillation

Drugs for Stable Tachycardia

Drugs for tachycardia include

- Adenosine 6 mg IV (follow with saline flush); second dose (if required) 12 mg IV (follow with saline flush)
- Several analgesic and sedative agents are also used during electrical cardioversion, but those agents are not covered in this course.

Approach to Stable Tachycardia

A *stable tachycardia* refers to a condition in which the patient has

- A heart rate greater than 100/min
- No significant signs or symptoms caused by the increased rate
- A potential underlying cardiac electrical abnormality that generates the rhythm

Questions to Determine Classification

Classification of the tachycardia requires the careful clinical evaluation of these questions:

- Are symptoms present or absent?
- Are symptoms due to the tachycardia?
- Is the patient stable or unstable?
- Is the QRS complex narrow or wide?
- Is the rhythm regular or irregular?
- Is the QRS monomorphic or polymorphic?
- Is the rhythm sinus tachycardia?

The answers guide subsequent diagnosis and treatment.

Managing Stable Tachycardia: The Adult Tachycardia With a Pulse Algorithm

As noted in the Unstable Tachycardia Case, the keys to managing a patient with any tachycardia are assessing the appropriateness for the clinical condition, identifying and treating underlying causes (Step 1), and determining whether pulses are present and, if so, whether the patient is stable or unstable and then providing treatment based on the patient's

condition and rhythm. If the patient is pulseless, manage the patient according to the Adult Cardiac Arrest Algorithm (Figure 41). If the patient has pulses, manage the patient according to the Adult Tachycardia With a Pulse Algorithm (Figure 30).

If a tachycardia and a pulse are present, perform the steps of the BLS, Primary, and Secondary Assessments. Determine if serious signs or symptoms are present and due to the tachycardia. This will direct you to either the stable or unstable section of the algorithm.

- If significant signs or symptoms are due to the tachycardia, immediate cardioversion is indicated (see the Unstable Tachycardia Case).
- If the patient develops pVT or VF, deliver unsynchronized high-energy shocks (defibrillation energy) and follow the Adult Cardiac Arrest Algorithm.
- If the patient has polymorphic VT, treat the rhythm as VF and deliver high-energy unsynchronized shocks (ie, defibrillation energy).

In this case, the patient is stable, so you will manage according to the stable section of the Adult Tachycardia With a Pulse Algorithm (Figure 30). A precise identification of the rhythm (eg, reentry SVT, atrial flutter) may not be possible at this time.

Applying the Adult Tachycardia With a Pulse Algorithm to Stable Patients

In this case, a patient has stable tachycardia with a pulse. Conduct the steps in the Adult Tachycardia With a Pulse Algorithm to evaluate and manage the patient.

Patient Assessment

Step 1 directs you to assess the appropriateness for the patient's clinical condition. Typically, a heart rate greater than 150/min at rest is due to tachyarrhythmias other than sinus tachycardia.

BLS and ACLS Assessments

Using the BLS, Primary, and Secondary Assessments to guide your approach, identify and treat underlying causes (Step 2):

- Maintain patent airway; assist breathing as necessary.
- Give oxygen (if hypoxemic).

- Use a cardiac monitor to identify rhythm; monitor blood pressure and oximetry.
- Obtain IV access.
- Obtain 12-lead ECG if available.

If symptoms persist, proceed to Step 3. If the patient is stable, go to Step 8.

IV Access and 12-Lead ECG

If the patient with tachycardia is stable (ie, no serious signs or symptoms related to the tachycardia), you have time to evaluate the rhythm and decide on treatment options. Establish IV access if not already obtained. Obtain a 12-lead ECG (if available) or rhythm strip to determine if the QRS is narrow (less than 0.12 second) or wide (0.12 second or more).

Decision Point: Wide or Narrow

The path of treatment is now determined by whether the QRS is wide or narrow and whether the rhythm is regular or irregular. If a monomorphic wide-complex rhythm is present and the patient is stable, consider adenosine (only if regular and monomorphic), consider antiarrhythmic infusion, and seek expert consultation. Treat polymorphic wide-complex tachycardia with immediate unsynchronized shock.

Wide-Complex Tachycardias

Wide-complex tachycardias are defined as a QRS of 0.12 second or more, but consider seeking expert consultation for help identifying the rhythm. The most common forms of life-threatening wide-complex tachycardias likely to deteriorate to VF are

- Monomorphic VT
- Polymorphic VT

Determine if the rhythm is regular or irregular.

- A regular wide-complex tachycardia is presumed to be VT or SVT with aberrancy.
- An irregular wide-complex tachycardia may be atrial fibrillation with aberrancy, pre-excited atrial fibrillation (atrial fibrillation using an accessory pathway for antegrade conduction), or polymorphic VT/torsades de pointes. These advanced rhythms require additional expertise or expert consultation. In addition, consider adenosine (only if regular and monomorphic) and antiarrhythmic infusion.

If the rhythm is likely VT or SVT in a stable patient, treat based on the algorithm for that rhythm.

Recent evidence suggests that if the rhythm etiology cannot be determined and is regular in its rate and monomorphic, IV adenosine is relatively safe for both treatment and diagnosis. IV antiarrhythmic drugs may be effective. We recommend:

- Procainamide 20 to 50 mg/min IV until arrhythmia suppressed, hypotension ensues, QRS duration increases more than 50%, or maximum dose 17 mg/kg IV is given. Maintenance infusion: 1 to 4 mg/min IV. Avoid if prolonged QT or congestive heart failure.
- Amiodarone (first dose) 150 mg IV over 10 minutes. Repeat as needed if VT recurs. Follow by maintenance infusion of 1 mg/min IV for first 6 hours.
- Sotalol 100 mg (1.5 mg/kg) IV over 5 minutes. Avoid if prolonged QT.

In the case of irregular wide-complex tachycardia, management focuses on control of the rapid ventricular rate (rate control), conversion of hemodynamically unstable atrial fibrillation to sinus rhythm (rhythm control), or both. Seek expert consultation.

Treating Tachycardia

You may not always be able to distinguish between supraventricular (aberrant) and ventricular wide-complex rhythms, so be aware that most wide-complex (broad-complex) tachycardias are ventricular in origin.

If a patient is pulseless, follow the Adult Cardiac Arrest Algorithm.

If a patient becomes unstable, do not delay treatment for further rhythm analysis. For stable patients with wide-complex tachycardias, consider expert consultation because treatment has the potential for harm.

Critical Concepts: Drugs to Avoid in Patients With Irregular Wide-Complex Tachycardia

Avoid AV nodal blocking agents such as adenosine, calcium channel blockers, digoxin, and possibly β-blockers in patients with pre-excitation atrial fibrillation, because these drugs may cause a paradoxical increase in the ventricular response.

Narrow QRS, Regular Rhythm

The therapy for narrow QRS with regular rhythm is to attempt vagal maneuvers, give adenosine, give a β-blocker or calcium channel blocker, and consider expert consultation. Vagal maneuvers, adenosine, and β-blocker or calcium channel blockers are the preferred initial interventions for terminating narrow-complex tachycardias that are symptomatic (but stable) and supraventricular in origin. Valsalva maneuvers or carotid sinus massage alone will terminate about 25% of SVT, and adenosine is required for the remainder.

- If SVT does not respond to vagal maneuvers, give **adenosine** 6 mg IV (follow with saline flush) in a large (eg, antecubital) vein over 1 second, and elevate the arm immediately.
- If SVT does not convert within 1 to 2 minutes, give a second dose of **adenosine** 12 mg IV (follow with saline flush) following the same procedure above.

Adenosine increases AV block and will terminate approximately 90% of reentry arrhythmias within 2 minutes. Adenosine will not terminate atrial flutter or atrial fibrillation but will slow AV conduction, allowing you to identify flutter or fibrillation waves.

Adenosine is safe and effective in pregnancy, but it has several important drug interactions. Patients with significant blood levels of theophylline, caffeine, or theobromine may require larger doses, and you should reduce the initial dose to 3 mg IV for patients taking dipyridamole or carbamazepine. Due to recent case reports of prolonged asystole after adenosine administration to patients with transplanted hearts or after central venous administration, you may consider lower doses such as 3 mg IV in these situations.

Adenosine may cause bronchospasm, so generally, you should not give adenosine to patients with asthma or chronic obstructive pulmonary disease, particularly if patients are actively bronchospastic.

If the rhythm converts with adenosine, it is probable reentry SVT. Observe patients for recurrence, and treat any recurrence with adenosine or longer-acting AV nodal blocking agents, such as the non-dihydropyridine calcium channel blockers (verapamil and diltiazem) or β-blockers. Typically, you should obtain expert consultation if the tachycardia recurs.

If the rhythm does not convert with adenosine, it is possible atrial flutter, ectopic atrial tachycardia, sinus tachycardia, or junctional tachycardia, and you should obtain expert consultation about diagnosis and treatment.

Critical Concepts: What to Avoid With AV Nodal Blocking Agents

Do not use AV nodal blocking drugs for pre-excited atrial fibrillation or flutter because these drugs are unlikely to slow the ventricular rate and may even accelerate the ventricular response. Also, be careful when combining AV nodal blocking agents of varying duration, such as calcium channel blockers or β-blockers, because their actions may overlap if given serially and provoke profound bradycardia.

Tachycardia Algorithm: Advanced Management Steps

As an ACLS provider, you should be able to recognize a stable narrow-complex or wide-complex tachycardia, classify the rhythm as regular or irregular, and provide initial management. You may treat regular narrow-complex tachycardias initially with vagal maneuvers, adenosine, and β-blocker or calcium channel blocker, but if these are unsuccessful, you'll need to consider expert consultation.

If you have experience with the differential diagnosis and therapy of stable tachycardias that do not respond to initial treatment, you can review the Adult Tachycardia With a Pulse Algorithm for additional steps and pharmacologic agents used in the treatment of these arrhythmias, both for rate control and for termination of the arrhythmia.

If at any point you become uncertain or uncomfortable while treating a stable patient, seek expert consultation because treatment has the potential for harm.

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Part 3: High-Performance Teams

High-performance teams are essential to successful resuscitation attempts. High-performance teams carry out their roles in highly effective manners, resulting in superior performance and timing, which can translate to improved survival for patients in cardiac arrest. What distinguishes high-performance teams from others is that each team member is committed to ensuring the highest-quality performance of the team rather than simply following orders. To function effectively, a high-performance team needs to focus on

- **Timing:** time to first compression, time to first shock, CCF ideally greater than 80%,* minimizing preshock pause, and early emergency medical services (EMS) response time
- **Quality:** rate, depth, complete recoil, minimizing interruptions, switching compressors every 2 minutes or sooner if fatigued, avoiding excessive ventilation, and using a feedback device
- **Coordination:** team dynamics: team members working together seamlessly toward a common goal, proficient in their roles
- **Administration:** leadership, measurement, continuous quality improvement, and number of participating code team members

* High-performing systems target at least 60%, with 80% or higher being a frequent goal.

High-performance teams (Figure 32) will need to incorporate timing, quality, coordination, and administration of the appropriate procedures during a cardiac arrest. The team will need to consider their overall purpose and goals, skills each team member possesses, appropriate motivation and efficacy, as well as appropriate conflict resolution and communication needs of the team. In addition, high-performance teams measure their performance, evaluate the data, and look for ways to improve performance and implement the revised strategy.

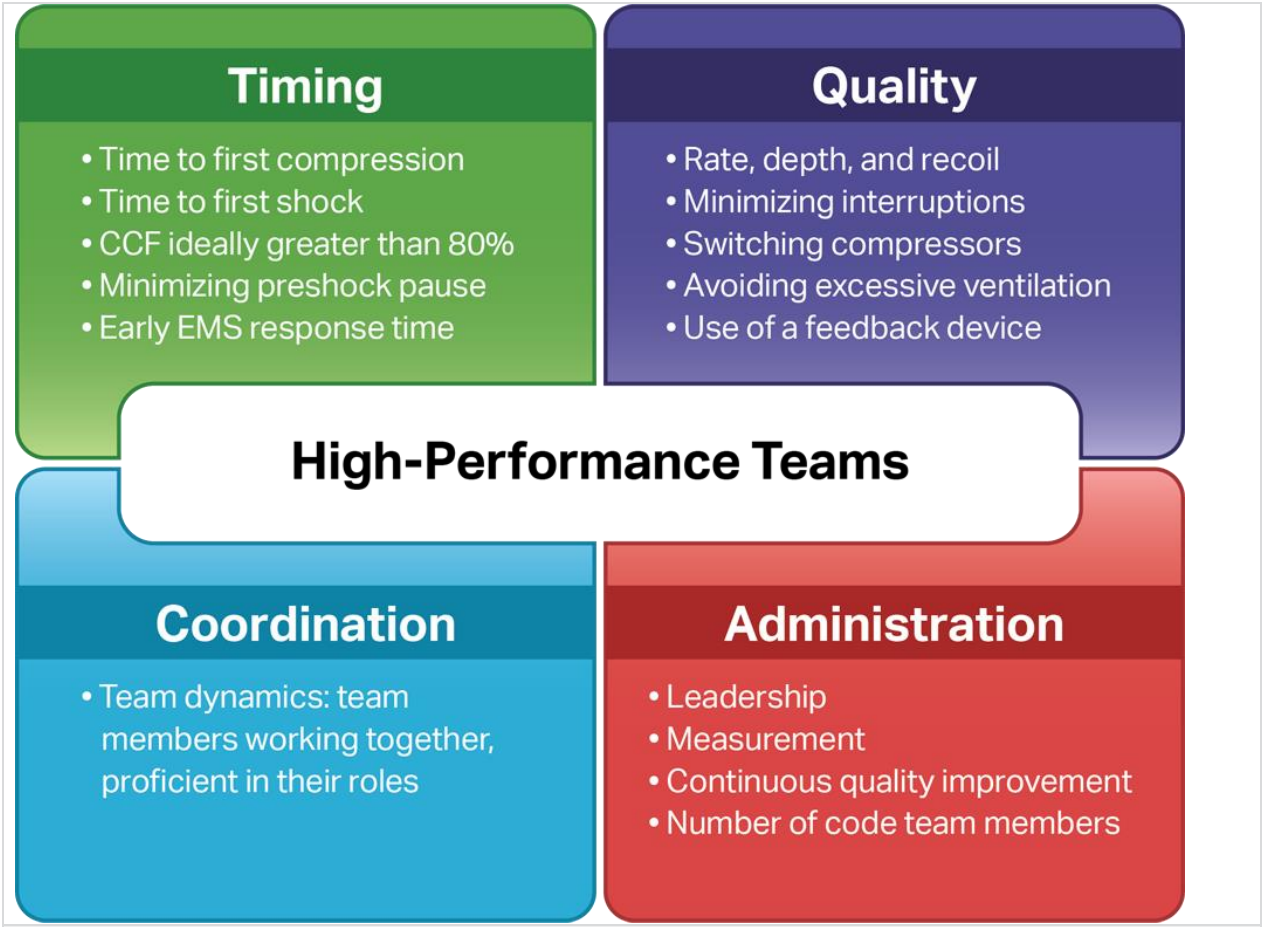


Figure 32. Key areas of focus for high-performance teams to increase survival rates.

Critical Concepts: Ways to Increase Chest Compression Fraction

Whether you are a team member or the Team Leader during a resuscitation attempt, you should understand how a high-performance team can maximize CCF when performing CPR during a cardiac arrest. The team can achieve key metrics and increase CCF by doing the following:

- **Precharge the defibrillator** *15 seconds before a 2-minute rhythm analysis (deliver shock immediately if VF or pVT on the monitor). This makes it possible to conduct a rhythm analysis and give a shock (if needed) within 10 seconds or less.*
- *Perform a pulse check during the precharge phase in anticipation of an organized rhythm during analysis (a pulse check during compressions is not a reliable indicator of CPR quality).*

- *Compressor* **hovers over the chest** *(not touching it), ready to start chest compressions immediately after a shock, a rhythm analysis, or other necessary pauses in compressions.*
- **Have the next compressor ready to take over immediately.**
- *Intubate without pausing compressions.*
- *Deliver medications during compressions.*
- *Consider CPR protocols that deliver fewer pauses (eg, continuous compressions with asynchronous ventilation using a bag-mask device).*

High-Performance Team Roles and Dynamics

Successful resuscitation attempts often require healthcare providers to simultaneously perform a variety of interventions. Although a CPR-trained bystander working alone can resuscitate a patient within the first moments after collapse, most attempts require multiple healthcare providers. Effective teamwork divides the tasks while multiplying the chances of a successful outcome.

Successful high-performance teams not only have medical expertise and mastery of resuscitation skills but also demonstrate effective communication and team dynamics. This section discusses the importance of team roles, behaviors of effective Team Leaders and team members, and elements of effective high-performance team dynamics.

Critical Concepts: Understanding Team Roles

Whether you are a team member or a Team Leader during a resuscitation attempt, you should understand your role and the roles of other members. This awareness will help you anticipate

- *What actions will be performed next*
- *How to communicate and work as a member or as a leader of a high-performance team*

Roles in a High-Performance Team

Team Leader Role

Every high-performance team needs a leader to organize the efforts of the group. The Team Leader

- Organizes the group

- Monitors individual performance of team members
- Backs up team members
- Models excellent team behavior
- Trains and coaches
- Facilitates understanding
- Focuses on comprehensive patient care
- Temporarily designates another team member to take over as Team Leader if an advanced procedure is required (eg, advanced airway placement)

The Team Leader is responsible for making sure everything is done at the right time in the right way by monitoring and integrating individual performance of team members. The Team Leader should also help train future Team Leaders and improve team effectiveness. After resuscitation, the Team Leader can help analyze, critique, and practice for the next resuscitation attempt.

The Team Leader also helps team members understand why they must perform certain tasks in a specific way. The Team Leader should be able to explain why it is essential to

- Push hard and fast in the center of the chest
- Ensure complete chest recoil
- Minimize interruptions in chest compressions
- Avoid excessive ventilation

Whereas members of a high-performance team should focus on their individual tasks, the Team Leader must focus on comprehensive patient care.

Team Member Roles

For a successful resuscitation attempt, high-performance team members must be

- Proficient in performing the skills in their scope of practice
- Clear about role assignments
- Prepared to fulfill their role responsibilities
- Well-practiced in resuscitation skills
- Knowledgeable about the algorithms
- Committed to success

Team Member Role: CPR Coach

Many resuscitation teams now include the role of CPR Coach. The CPR Coach supports performance of high-quality BLS skills, allowing the Team Leader to focus on other aspects of clinical care. Studies have shown that resuscitation teams with a CPR Coach perform higher-quality CPR with higher CCF and shorter pause durations compared with teams that don't use a CPR Coach.

The CPR Coach does not need to be a separate role; they can be blended into the current responsibilities of the Monitor/Defibrillator. The CPR Coach's main responsibilities are to help team members provide high-quality CPR and minimize pauses in compressions. The CPR Coach needs a direct line of sight to the Compressor, so they should stand next to the Defibrillator. Below is a description of the CPR Coach's actions.

Coordinate the start of CPR: As soon as a patient is identified as having no pulse, the CPR Coach says, "I am the CPR Coach," and tells providers to begin chest compressions. The CPR Coach can adjust the environment to help ensure high-quality CPR. They can lower the bedrails or the bed, get a step stool, or roll the victim to place a backboard and defibrillator pads.

Coach to improve the quality of chest compressions: The CPR Coach gives feedback about performance of compression depth, rate, and chest recoil. They state the CPR feedback device's data to help the Compressor improve performance. This is useful because visual assessment of CPR quality is often inaccurate.

State the midrange targets: The CPR Coach states the specific midrange targets so that compressions and ventilation are within the recommended range. For example, they should tell the Compressor to compress at a rate of 110/min instead of a rate between 100 and 120/min.

Coach to the midrange targets: The CPR Coach gives team members feedback about their ventilation rate and volume. If needed, they also remind the team about compression-to-ventilation ratio.

Help minimize the length of pauses in compressions: The CPR Coach communicates with the team to help minimize the length of pauses in compressions. Pauses happen when the team defibrillates, switches Compressors, and places an advanced airway.

Critical Concepts: CPR Coach Role

The CPR Coach role is designed to help a high-performance team achieve the key metrics of high-quality CPR by providing feedback about

- *The Compressor's rate, depth, and recoil*
- *Delivery of ventilations (rate and volume)*
- *Compression pauses*

Working closely with the Team Leader, the CPR Coach should facilitate all compression pauses, including intubation. The CPR Coach should be integrated into the existing role of Monitor/Defibrillator on a high-performance team.

Elements of Effective Team Dynamics as Part of a High-Performance Team

Roles

Clear Roles and Responsibilities

Every member of the team should know his or her role and responsibilities because each team member's role is important to the performance of the team. Figure 33 identifies 6 team roles for resuscitation. When fewer than 6 people are present, Team Leaders must prioritize these tasks and assign them to the healthcare providers present.

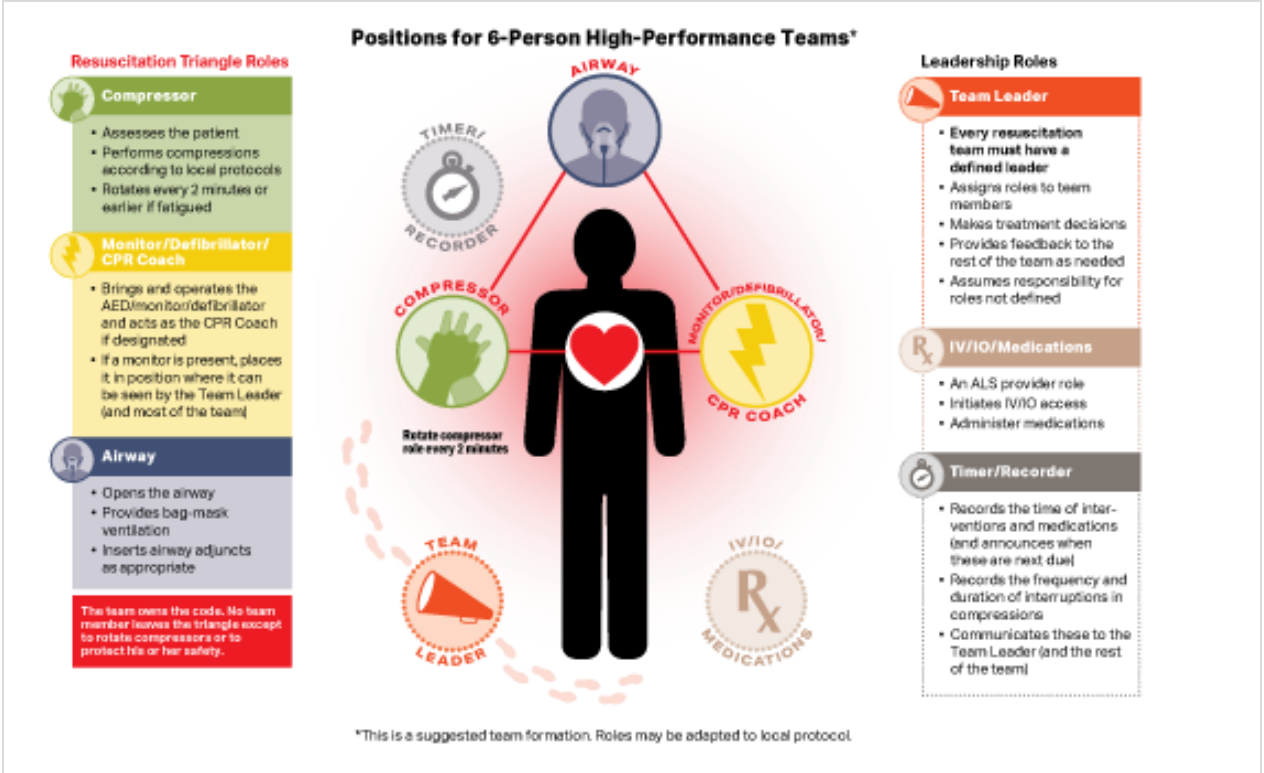


Figure 33. Suggested locations for the Team Leader and team members during case simulations and clinical events.

When roles are unclear, team performance suffers. Signs of unclear roles include

- Performing the same task more than once
- Missing essential tasks
- Assigning team members multiple roles when additional providers are available

For efficiency, the Team Leader must clearly delegate tasks. Team members should communicate when they can handle additional responsibilities. The Team Leader should encourage team members to participate actively and not simply follow directions. Table 10 lists some additional information about roles.

Table 10. Clear Roles and Responsibilities

Team member	Task
Team Leader	• Clearly define all team member roles in the clinical setting

	• Distribute tasks evenly to all available team members who are sure of their responsibilities
Team members	• Seek out and perform clearly defined tasks appropriate to their abilities • Ask for a new task or role if an assignment is beyond their level of expertise • Accept only assignments that are within their level of expertise

Knowing Your Limitations

Everyone on the team should know his or her own limitations and capabilities, including the Team Leader. This allows the Team Leader to evaluate resources and call for backup when necessary. High-performance team members should anticipate situations in which they need help and inform the Team Leader.

During the stress of an attempted resuscitation, do not practice or explore a new skill, especially without seeking advice from more experienced personnel. If you need extra help, request it early rather than waiting until the patient deteriorates further. Asking for help is not a sign of weakness or incompetence; it is better to have more help than needed rather than not enough help, which might negatively affect patient outcome. Table 11 lists some additional information about knowing your limitations.

Table 11. Knowing Your Limitations

Team member	Task
Team Leader and team members	• Call for assistance early rather than waiting until the patient deteriorates • Seek advice from more experienced personnel when th patient's condition worsens despite primary treatment • Allow others to carry out assigned tasks, especially if th task is essential to treatment
Team members	• Seek advice from more experienced personnel before starting an unfamiliar treatment or therapy • Accept assistance from others when it is readily availal

Constructive Interventions

During a resuscitation attempt, anyone on a high-performance team may need to intervene tactfully if a team member is about to take an inappropriate action. Team Leaders should avoid confrontation with team members and instead debrief afterward if needed. Table 12 lists some additional information about constructive interventions.

Table 12. Constructive Interventions

Team member	Task
Team Leader	• Ask that a different intervention be started if it has a higher priority • Reassign a team member who is trying to function beyond his or her level of skill
Team members	• Suggest an alternative drug or dose confidently • Question a colleague who is about to make a mistake • Intervene if a team member is about to administer a drug incorrectly

What to Communicate

Knowledge Sharing

Sharing information is critical to effective team performance. Team Leaders may become fixated on a specific treatment or diagnostic approach. Examples of these types of *fixation errors* are

- "Everything is OK."
- "This and only this is the correct path."
- "Do anything but this."

Table 13. Knowledge Sharing

Team member	Task
Team Leader	• Encourage information sharing

	• Ask for suggestions about interventions, differential diagnoses, and possible overlooked treatments (eg, intravenous access or drug treatments) • Look for clinical signs that are relevant to the treatment
Team members	• Share information with each other • Accept information that will improve their roles

When resuscitative efforts are ineffective, go back to the basics and talk as a team. Have conversations like, "Well, we've observed the following on the Primary Assessment…. Have we missed something?" High-performance team members should provide all available information about changes in the patient's condition to ensure that the Team Leader makes appropriate decisions. Table 13 lists some additional information about knowledge sharing.

Summarizing and Reevaluating

An essential role of the Team Leader is monitoring and reevaluating interventions, assessment findings, and the patient's status.

Team Leaders should periodically state this information to the team and announce the plan for the next few steps. Remember that the patient's condition can change. Be flexible to changing treatment plans, and ask for information and summaries from the Timer/Recorder as well. Table 14 lists some additional information about summarizing and reevaluating.

Table 14. Summarizing and Reevaluating

Team member	Task
Team Leader	• Continuously revisit decisions about differential diagnoses • Maintain an ongoing record of treatments and the patient's response • Change a treatment strategy when new information supports it • Inform arriving personnel of the current status and plans for further action
Team Leader and team members	• Note significant changes in the patient's clinical condition • Increase monitoring if patient's condition deteriorates (eg, frequency of respirations and blood pressure)

How to Communicate

Closed-Loop Communications

When communicating with high-performance team members, the Team Leader should use these closed-loop communication steps:

1. Give a message, order, or assignment to a team member.
2. Request a clear response and eye contact from the team member to ensure that he or she understood the message.
3. Confirm that the team member completed the task before you assign him or her another task.

Table 15 lists some additional information about closed-loop communications.

Table 15. Closed-Loop Communications

Team member	Task
Team members	• After receiving a task, close the loop by informing the Team Leader when the task begins or ends, such as, “The IV is in” • Only give drugs after verbally confirming the order with the Team Leader
Team Leader	• Always assign tasks by using closed-loop communication such as, “Give 1 milligram of epinephrine and let me know when it has been given” • Assign additional tasks to a team member *only* after receiving confirmation of a completed assignment

Clear Messages

Clear messages means concise communication spoken with distinctive speech in a controlled voice. All healthcare providers should deliver clear messages calmly and directly, without yelling or shouting. Distinct, concise messages are crucial for clear communication because unclear communication can delay treatment or cause medication errors. Yelling or shouting can also impair effective high-performance team interaction. Table 16 lists some additional information about clear messages.

Table 16. Clear Messages

Team member	Task
Team Leader	• Encourage all team members to speak clearly and use complete sentences
Team Leader and team members	• Repeat orders, and question them if the slightest doubt exists • Be careful not to mumble, yell, scream, or shout • Ensure that only 1 person talks at a time

Mutual Respect

The best high-performance team members mutually respect each other and work together in a collegial, supportive manner. Everyone in a high-performance team must abandon ego and show respect during the resuscitation attempt, regardless of any additional training or experience that specific team members may have. Table 17 lists some additional information about mutual respect.

Table 17. Mutual Respect[1]

Team member	Task
Team Leader	• Acknowledge correctly completed assignments by saying, “Thanks—good job!”
Team Leader and team members	• Show interest and listen to what others say • Speak in a friendly, controlled tone of voice • Avoid displaying aggression if teammates do not initially understand each other

	• Understand that when one person raises his voice, others will respond similarly • Try not to confuse directive behavior with aggression

Respiratory Arrest

Overview

For respiratory arrest, this patient is unconscious and unresponsive and has a pulse, but respirations are completely absent or clearly inadequate to maintain effective oxygenation and ventilation. Do not confuse agonal gasps with adequate respirations. Use the BLS, Primary, and Secondary Assessments even though the patient is in respiratory but not cardiac arrest.

Drugs for Respiratory Arrest

Drugs for respiratory arrest include oxygen. Systems or facilities that use rapid sequence intubation may consider additional drugs.

Normal and Abnormal Breathing

The average respiratory rate for an adult at rest is about 12 to 20/min. Typically, a tidal volume of 6 to 8 mL/kg maintains normal oxygenation and elimination of CO_2.

Tachypnea is a respiratory rate above 20/min and *bradypnea* is a respiratory rate below 12/min. A respiratory rate below 6/min (*hypoventilation*), requires assisted ventilation with a bag-mask device or advanced airway with 100% oxygen.

Identifying Respiratory Problems by Severity

Identifying the severity of a respiratory problem will help you decide the most appropriate interventions. Be alert for signs of respiratory distress and respiratory failure.

Respiratory Distress

Respiratory distress is a clinical state characterized by abnormal respiratory rate or effort—either increased (eg, tachypnea, nasal flaring, retractions, and use of accessory muscles) or inadequate (eg, hypoventilation or bradypnea).

Respiratory distress can range from mild to severe. For example, a patient with mild tachypnea and a mild increase in respiratory effort with changes in airway sounds is in mild respiratory distress. A patient with marked tachypnea, significantly increased respiratory effort, deterioration in skin color, and changes in mental status is in severe respiratory distress. Severe respiratory distress can indicate respiratory failure.

Respiratory distress typically includes some or all of these signs in varying severity:

- Tachypnea
- Increased respiratory effort (eg, nasal flaring, retractions)
- Inadequate respiratory effort (eg, hypoventilation or bradypnea)
- Abnormal airway sounds (eg, stridor, wheezing, grunting)
- Tachycardia
- Pale, cool skin (however, some causes of respiratory distress, like sepsis, may cause warm, red, and diaphoretic skin)
- Changes in level of consciousness/agitation
- Use of abdominal muscles to help breathe

Respiratory distress is apparent when a patient tries to maintain adequate gas exchange despite airway obstruction, reduced lung compliance, lung tissue disease, or increase in metabolic demand (sepsis or ketoacidosis). As these patients tire or their respiratory function, effort, or both deteriorate, they cannot maintain adequate gas exchange and develop clinical signs of respiratory failure.

Respiratory Failure

Respiratory failure is a clinical state of inadequate oxygenation, ventilation, or both. Respiratory failure is often the end stage of respiratory distress. If the patient has abnormal central nervous system control of breathing or muscle weakness, she may show little or no respiratory effort despite being in respiratory failure. In these situations, you may need to identify respiratory failure based on clinical findings. Confirm the diagnosis with objective measurements, such as pulse oximetry or blood gas analysis.

Suspect *probable respiratory failure* if some of the following signs are present:

- Marked tachypnea
- Bradypnea, apnea
- No respiratory effort
- Poor to absent distal air movement

- Tachycardia (early); bradycardia (late)
- Cyanosis
- Stupor, coma (late)

Respiratory failure can result from upper or lower airway obstruction, lung tissue disease, and disordered control of breathing (eg, apnea or shallow, slow respirations). When respiratory effort is inadequate, respiratory failure can occur without typical signs of respiratory distress. Respiratory failure requires intervention to prevent deterioration to cardiac arrest. Respiratory failure can occur with a rise in arterial carbon dioxide levels (hypercapnia), a drop in blood oxygenation (hypoxemia), or both.

Respiratory distress can lead to respiratory failure, and respiratory failure can lead to respiratory arrest.

Respiratory Arrest

Respiratory arrest is the absence of breathing, usually caused by an event such as drowning or head injury. For an adult in respiratory arrest, provide a tidal volume of approximately 500 to 600 mL (6 to 7 mL/kg), or enough to produce visible chest rise.

Patients with airway obstruction or poor lung compliance may need higher pressures to produce visible chest rise. A pressure-relief valve on a resuscitation bag-mask device may prevent sufficient tidal volume in these patients, so ensure that you can bypass the device's pressure-relief valve and use high pressures, if necessary, to produce visible chest rise.

Caution: Tidal Volume

Most adult bag-mask devices provide a higher tidal volume than is recommended. Caution is advised. Consider using a pediatric bag-mask device.

Critical Concepts: Avoiding Excessive Ventilation

Avoid excessive ventilation (too many breaths or too large a volume) during respiratory arrest and cardiac arrest. Excessive ventilation can cause gastric inflation and complications such as regurgitation and aspiration. More important, excessive ventilation can be harmful because it

- *Increases intrathoracic pressure*
- *Decreases venous return to the heart*

- *Diminishes cardiac output and survival*
- *May cause cerebral vasoconstriction, reducing blood flow to the brain*

BLS Assessment

When evaluating a patient, proceed with the BLS Assessment after you verify scene safety.

Assess and Reassess the Patient

The systematic approach is assessment, and then action, for each step in the sequence. Check for responsiveness, shout for nearby help, and activate the emergency response system via a mobile device (if appropriate). Get an AED and emergency equipment (or send someone to do so). Look for no breathing or only gasping and check pulse (simultaneously) within 10 seconds.

Remember to assess first, and then perform the appropriate action.

Initial actions should include

- Checking for responsiveness
- Calling for additional help
- Assessing ABCs

Ventilation and Pulse Check

If a patient has respiratory arrest with a pulse, deliver 1 breath every 6 seconds, or 10 breaths/min using a bag-mask device or any advanced airway device. Each breath should be delivered for 1 second and achieve a visible chest rise. Be careful to avoid excessive ventilation. Check the pulse about every 2 minutes, taking between 5 and 10 seconds to check. If no pulse, start CPR.

If possible opioid overdose, administer naloxone, if available, per protocol.

Primary Assessment

Airway Management in Respiratory Arrest

If bag-mask ventilation is adequate, you may defer the decision to place an advanced airway until the Primary Assessment. Advanced airways include laryngeal mask airways, laryngeal tubes, and endotracheal (ET) tubes. If

advanced airways are within your scope of practice, you may use them when appropriate and available.

Note: Ongoing quantitative waveform capnography will confirm and monitor placement of the advanced airway while the patient is intubated.

Managing Respiratory Arrest

Management of respiratory arrest includes both BLS and ACLS interventions. These interventions may include

- Giving supplemental oxygen
- Opening the airway
- Providing basic ventilation
- Using basic airway adjuncts (oropharyngeal airway [OPA] and nasopharyngeal airway [NPA])
- Suctioning the airway

Remember, for patients with a perfusing rhythm, deliver breaths once every 6 seconds.

Giving Supplemental Oxygen

Give oxygen to patients with acute cardiac symptoms or respiratory distress. Monitor their oxygen saturation, and adjust the supplemental oxygen to maintain at least 95% saturation (90% for ACS, and 92% to 98% for post–cardiac arrest care). Use 100% oxygen when treating patients in respiratory or cardiac arrest.

See the ACLS Student Resources for details on using oxygen for patients who are not in respiratory or cardiac arrest.

Opening the Airway

Common Cause of Airway Obstruction

The most common cause of upper airway obstruction in an unresponsive patient is loss of tone in the throat muscles (Figure 34 shows the airway anatomy). In this case, the patient's tongue falls back and obstructs the airway at the pharynx (Figure 35A).

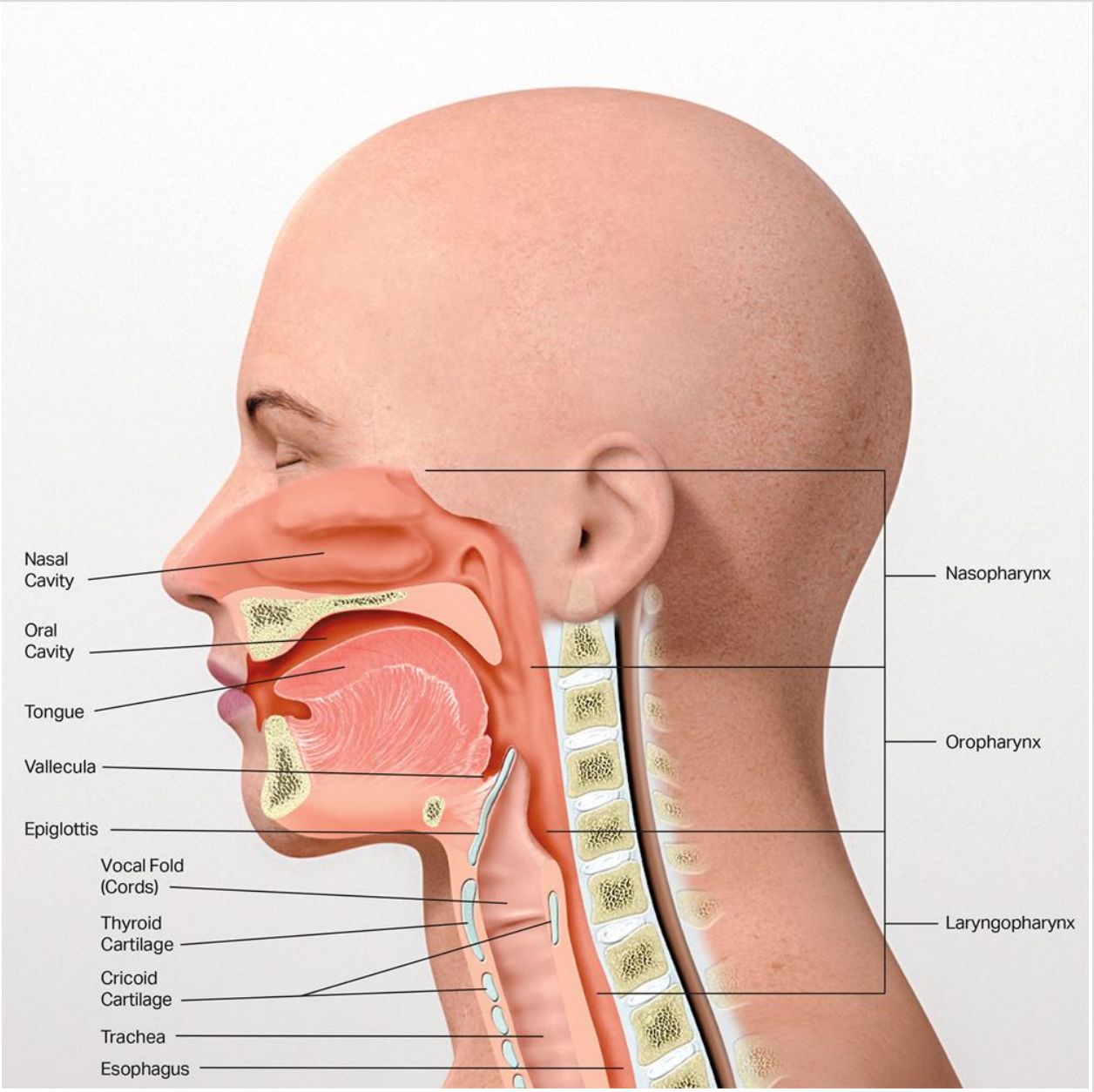


Figure 34. Airway anatomy.

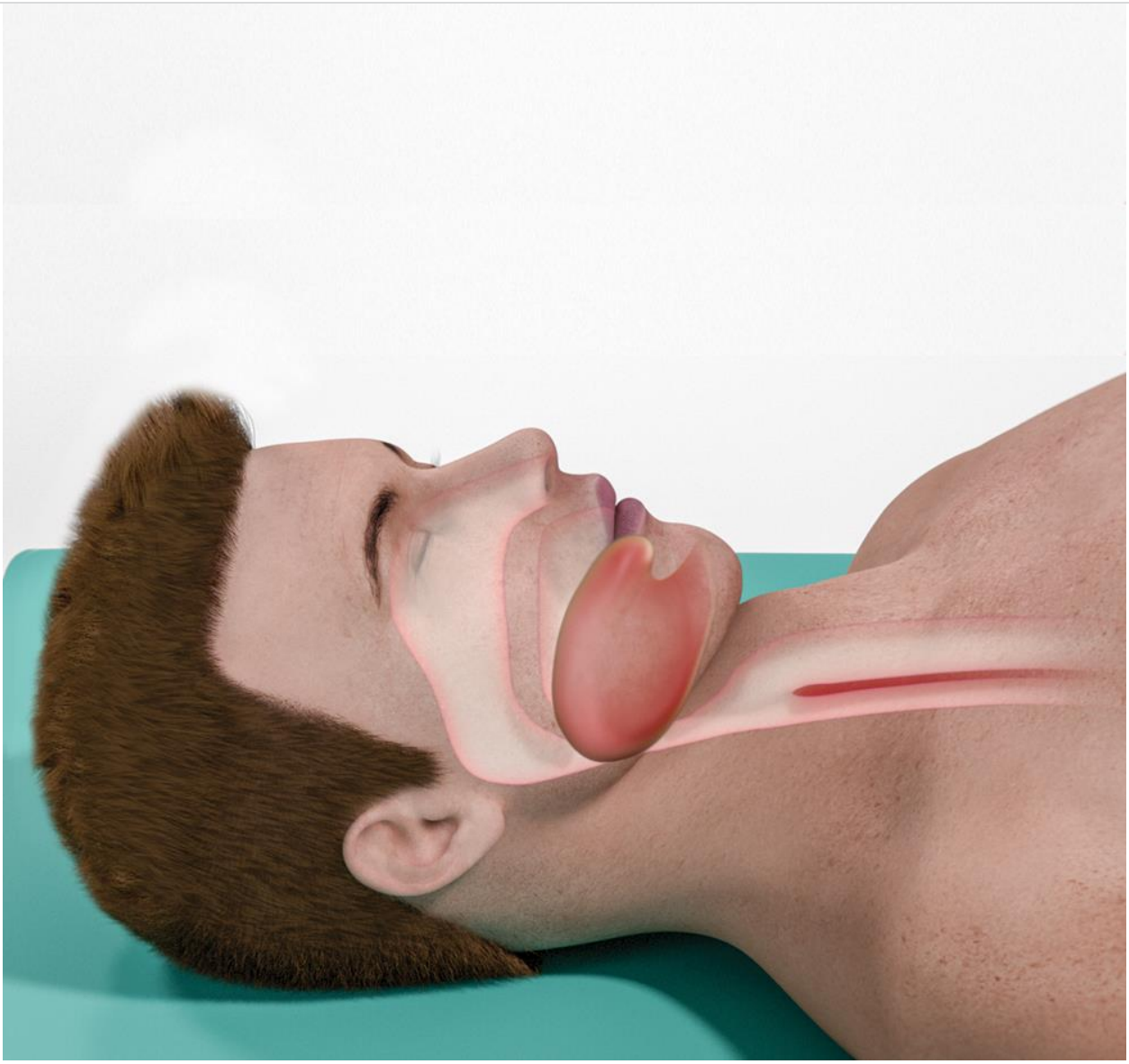


Figure 35A. Obstruction of the airway by the tongue and epiglottis. When a patient is unresponsive, the tongue can obstruct the airway. The head tilt–chin lift relieves obstruction in the unresponsive patient. A, The tongue is obstructing the airway.

Basic Airway Opening Techniques

Basic airway opening techniques relieve airway obstruction by the tongue or from relaxed upper airway muscles. One such technique requires tilting the head and lifting the chin: *the head tilt–chin lift* (Figure 35B).

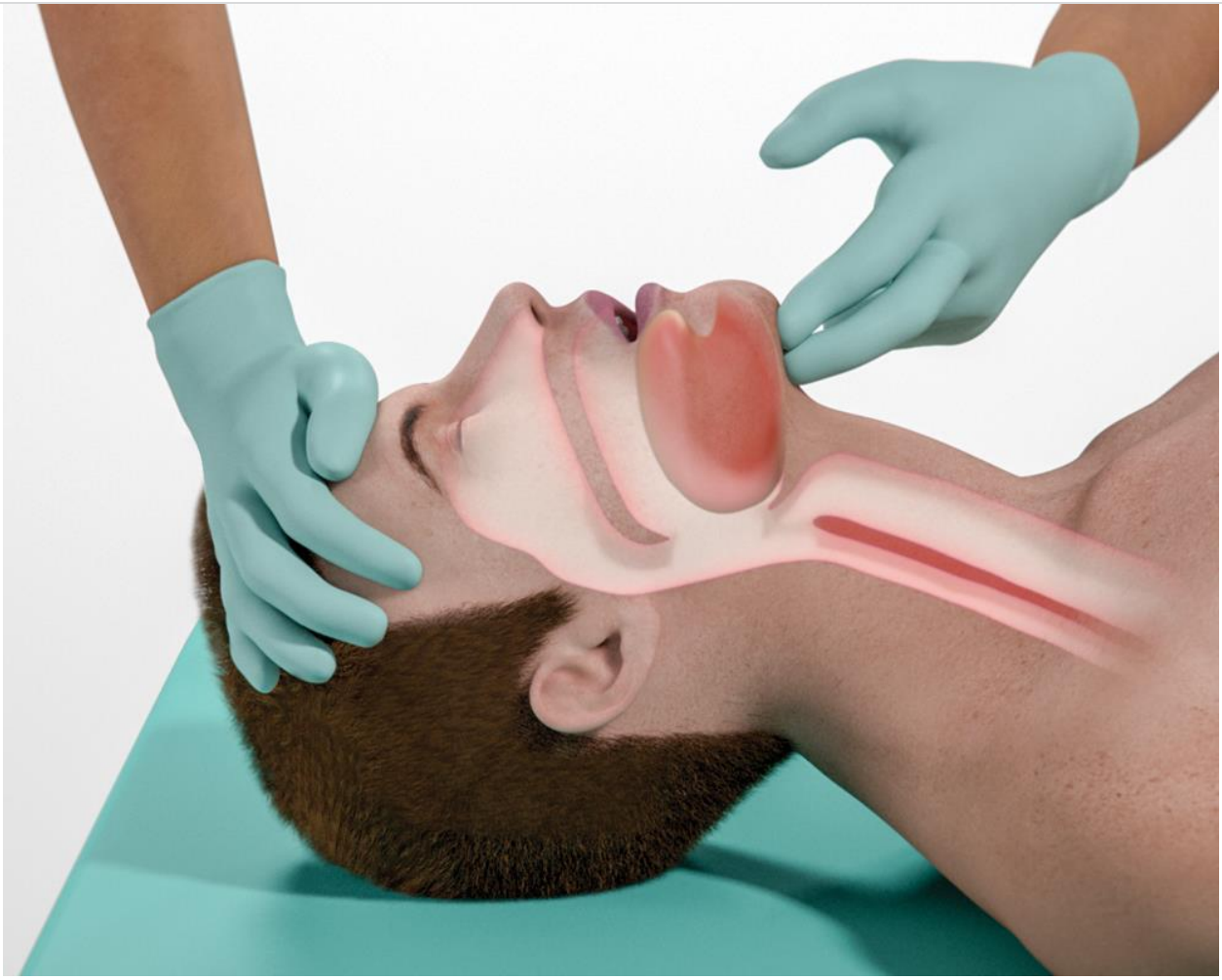


Figure 35B. The head tilt–chin lift lifts the tongue, relieving the obstruction.

In a trauma patient with suspected neck injury, try using a jaw-thrust technique that doesn't extend the head (Figure 35C). But because maintaining an open airway and providing ventilation is a priority, use the head tilt–chin lift if the jaw thrust does not open the airway.

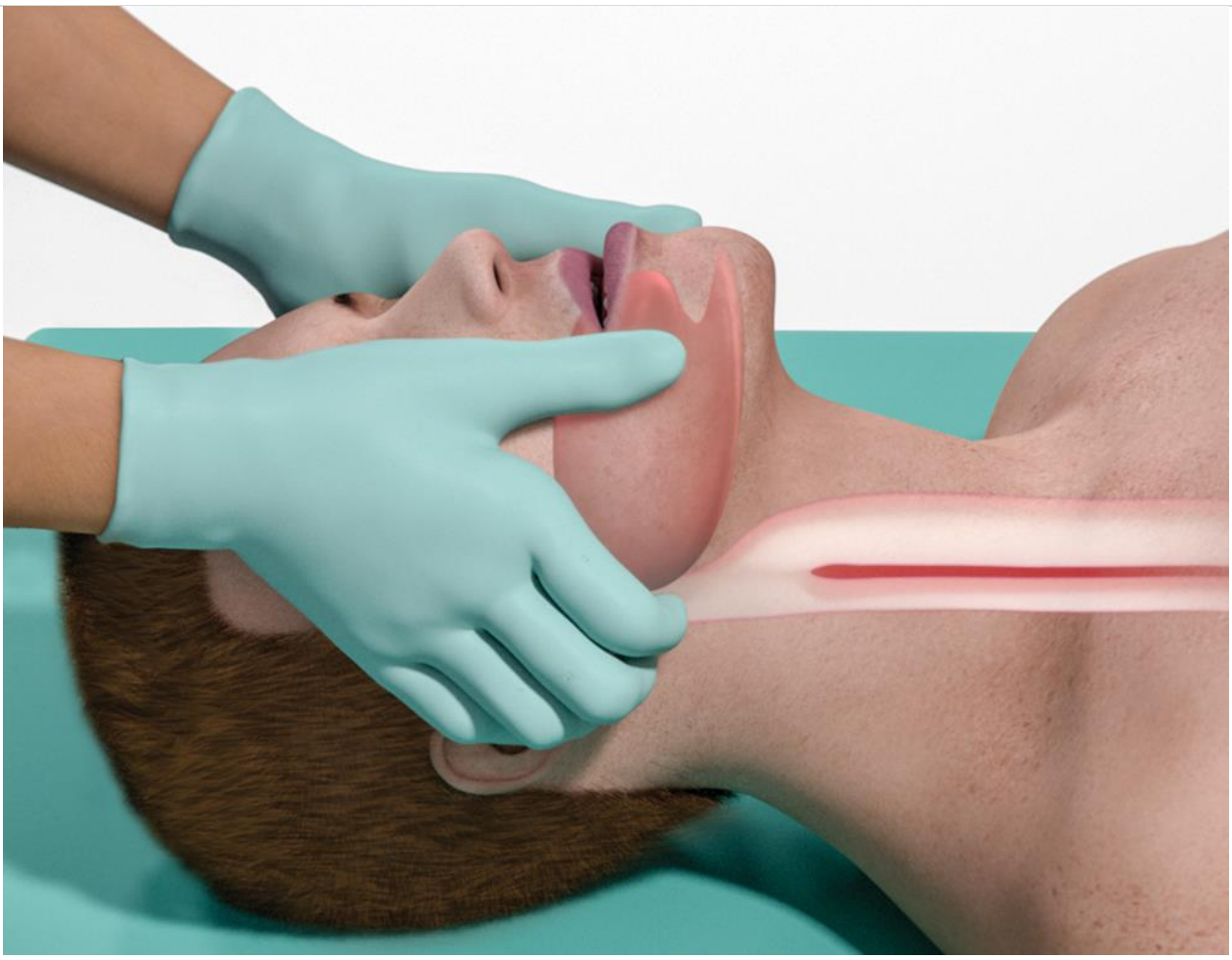


Figure 35C. If cervical spine trauma is suspected, use the jaw thrust without head extension.

Airway Management

Properly positioning the airway may be all you need to do for patients who can breathe spontaneously. In patients who are unconscious with no cough or gag reflex, insert an OPA or NPA to maintain an open airway.

If you find an unresponsive patient who was choking and is now in respiratory arrest, open the mouth wide and look for a foreign object. If you see one, remove it with your fingers. If you do not see a foreign object, start CPR. Each time you open the airway to give breaths, open the mouth wide, and look for and remove any foreign object. If you see no foreign object, resume CPR.

Providing Basic Ventilation

Basic airway skills used to ventilate a patient are

- Head tilt–chin lift
- Jaw thrust without head extension (suspected cervical spine trauma)
- Mouth-to-mouth ventilation
- Mouth-to-nose ventilation
- Mouth-to–barrier device ventilation (using a pocket mask)
- Bag-mask ventilation

Bag-Mask Ventilation

Bag-mask devices—which consist of a ventilation bag attached to a face mask—have been part of emergency ventilation for decades. These devices are the most common way to provide positive-pressure ventilation. When you use a bag-mask device, deliver approximately 500 to 600 mL tidal volume sufficient to produce chest rise over 1 second. Bag-mask ventilation is not the recommended method of ventilation for single rescuers during CPR. (A single rescuer should use a pocket mask for ventilation, if available.)

Providers can use the following techniques to hold the bag-mask device, depending on the number of rescuers:

- Use of the bag-mask device by 1 rescuer (Figure 36): The rescuer gets into position at the patient's head and circles the thumb and first finger around the top of the mask (forming a "C") while using the third, fourth, and fifth fingers (forming an "E") to lift the jaw. This is called the *E-C clamp technique*.
- Use of the bag-mask device by 2 rescuers (Figure 37): Two trained and experienced rescuers can more easily provide bag-mask ventilation. The rescuer at the patient's head tilts the patient's head and seals the mask against the patient's face, with the thumb and first finger of each hand creating a "C," to provide a complete seal around the edges of the mask. The rescuer uses the remaining 3 fingers (the "E") to lift the jaw (this holds the airway open). The second rescuer slowly squeezes the bag (over 1 second) until the chest rises. Both providers should observe chest rise.

Figure 36. E-C clamp technique for holding the mask while lifting the jaw.

Figure 37. Two-rescuer use of the bag-mask device.

The universal connections on all airway devices allow you to connect any ventilation bag to numerous adjuncts. Valves and ports may include

- One-way valves to prevent the patient from rebreathing exhaled air
- Oxygen ports to administer supplemental oxygen
- Medication ports to administer liquid and other medications
- Suction ports to clear the airway
- Ports to provide quantitative sampling of $ETCO_2$

You can attach other adjuncts to the patient end of the valve, including a pocket face mask, laryngeal mask airway, laryngeal tube, esophageal-tracheal tube, and ET tube. Ongoing quantitative waveform capnography can also be attached to a bag-valve apparatus to confirm and monitor the effectiveness of the ventilation. An obstructed airway with no air exchange will not produce exhaled carbon dioxide, even if the patient still has a pulse.

See the ACLS Student Resources for more information on bag-mask ventilation.

Basic Airway Adjuncts: OPA

The OPA is a J-shaped device (Figure 38A) that fits over the tongue to hold both it and the soft hypopharyngeal structures away from the posterior wall of the pharynx. Use this device for

- Patients at risk of developing airway obstruction from the tongue or from relaxed upper airway muscles
- Unconscious patients when other procedures (eg, head tilt–chin lift or jaw thrust) fail to maintain a clear, unobstructed airway
- Facilitating suctioning of intubated patients' mouths and throats
- Preventing patients from biting and obstructing the ET tube

Figure 38A. Oropharyngeal airways. A, Oropharyngeal airway devices.

You may also use an OPA during bag-mask ventilation when a rescuer might unknowingly push down on the chin, blocking the airway.

However, do not use an OPA with a conscious or semiconscious patient because it may stimulate gagging and vomiting. Before using an OPA, check whether the patient has an intact cough and gag reflex. If so, do not use an OPA.

Technique of OPA Insertion

- Clear the mouth and pharynx of secretions, blood, or vomit by using a rigid pharyngeal suction tip if possible.
- Select the proper size OPA, and place it against the side of the face (Figure 38B). When the flange of the OPA is at the corner of the mouth, the tip is at the angle of the mandible. Insert the OPA so that it curves upward toward the hard palate as it enters the mouth.
- As the OPA passes through the oral cavity and approaches the posterior wall of the pharynx, rotate the device 180° into the proper position (Figure 38C). You can also insert the OPA at a 90° angle to the mouth and then turn it down toward the posterior pharynx as you advance the device.

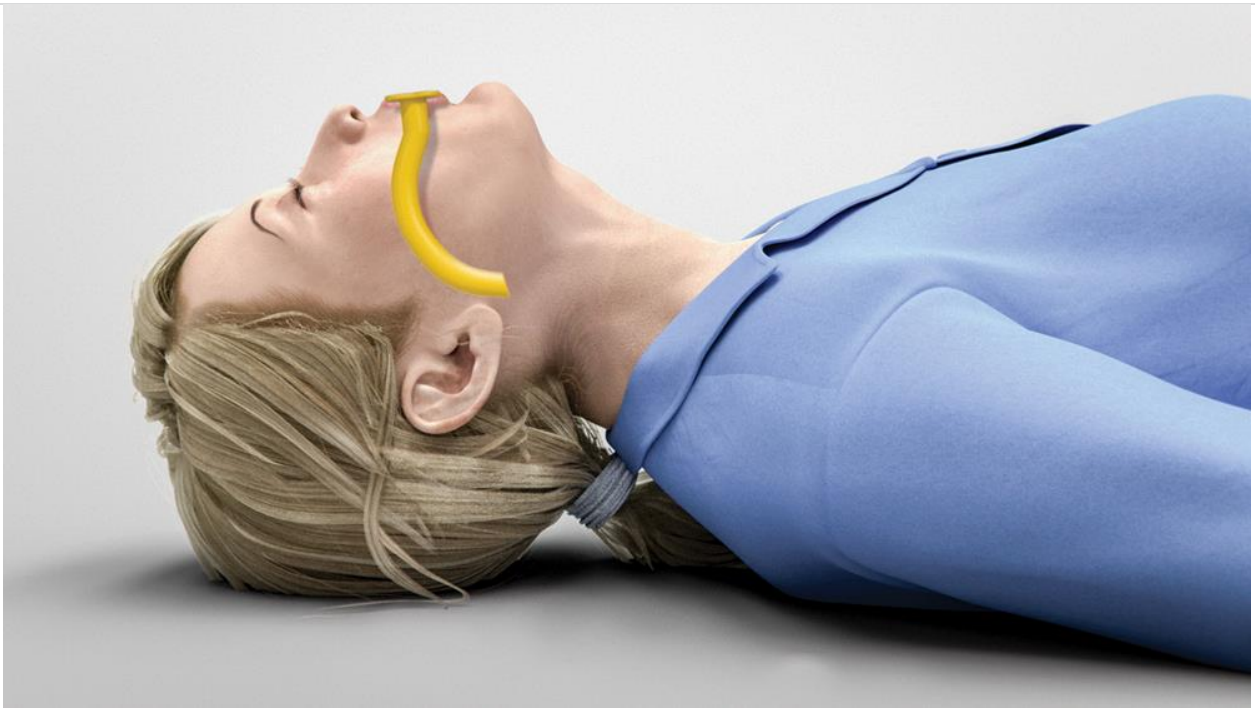


Figure 38B. Oropharyngeal airway device measurement.

Figure 38C. Oropharyngeal airway device inserted.

In both methods, the goal is to curve the device around the tongue so that you don't inadvertently push the tongue back into the pharynx rather than pull it forward. Alternatively, you can insert the OPA straight in while using a tongue depressor or similar device to hold the tongue forward as you advance the OPA.

If you have properly sized and inserted the OPA, it will align with the glottic opening. After inserting an OPA, monitor the patient. Keep the head and jaw positioned properly to maintain a patent airway. Suction the airway as needed.

Caution: Using an OPA

- *OPAs that are too large may obstruct the larynx or cause trauma to the laryngeal structures.*
- *OPAs that are too small or inserted improperly may push the base of the tongue back and obstruct the airway.*
- *Insert the OPA carefully to avoid soft tissue trauma to the lips and tongue.*

- *Remember to use the OPA only in the unresponsive patient with no cough or gag reflex. If the patient has a cough or gag reflex, the OPA may stimulate vomiting and laryngospasm.*

Basic Airway Adjuncts: NPA

The NPA is used as an alternative to an OPA in patients who need a basic airway adjunct. The NPA is a soft rubber or plastic uncuffed tube (Figure 39A) that provides a conduit for airflow between the nostrils and the pharynx.

Figure 39A. Nasopharyngeal airways. A, Nasopharyngeal airway devices.

Unlike oral airways, NPAs may be used in conscious, semiconscious, or unconscious patients (patients with an intact cough and gag reflex). Use an NPA when inserting an OPA is technically difficult or dangerous, such as for patients with a gag reflex, trismus, massive trauma around the mouth, or wired jaws. You may also use NPAs in patients who are neurologically impaired with poor pharyngeal tone or coordination leading to upper airway obstruction.

Technique of NPA Insertion

1. Select the proper size NPA.
    a. Compare the outer circumference of the NPA with the inner opening of the nostrils. The NPA should not be so large that it causes sustained blanching of the nostrils. You can use the diameter of the patient's smallest finger as a guide for the proper size.
    b. The NPA should be as long as the distance from the tip of the patient's nose to the earlobe (Figure 39B).
2. Lubricate the airway with a water-soluble lubricant or anesthetic jelly.
3. Insert the airway through the nostril in a posterior direction perpendicular to the plane of the face. Pass it gently along the floor of the nasopharynx (Figure 39C). If you encounter resistance
    a. Slightly rotate the NPA to insert at the angle of the nasal passage and nasopharynx
    b. Attempt to place through the other nostril (the size of a patient's nasal passages varies)
4. Reevaluate often, and maintain head tilt by using a chin lift or jaw thrust. Mucus, blood, vomit, or the soft tissues of the pharynx can obstruct the NPA, which has a small internal diameter. Frequently evaluate, and suction the airway if needed to ensure patency.

Figure 39B. Nasopharyngeal airway device measurement.

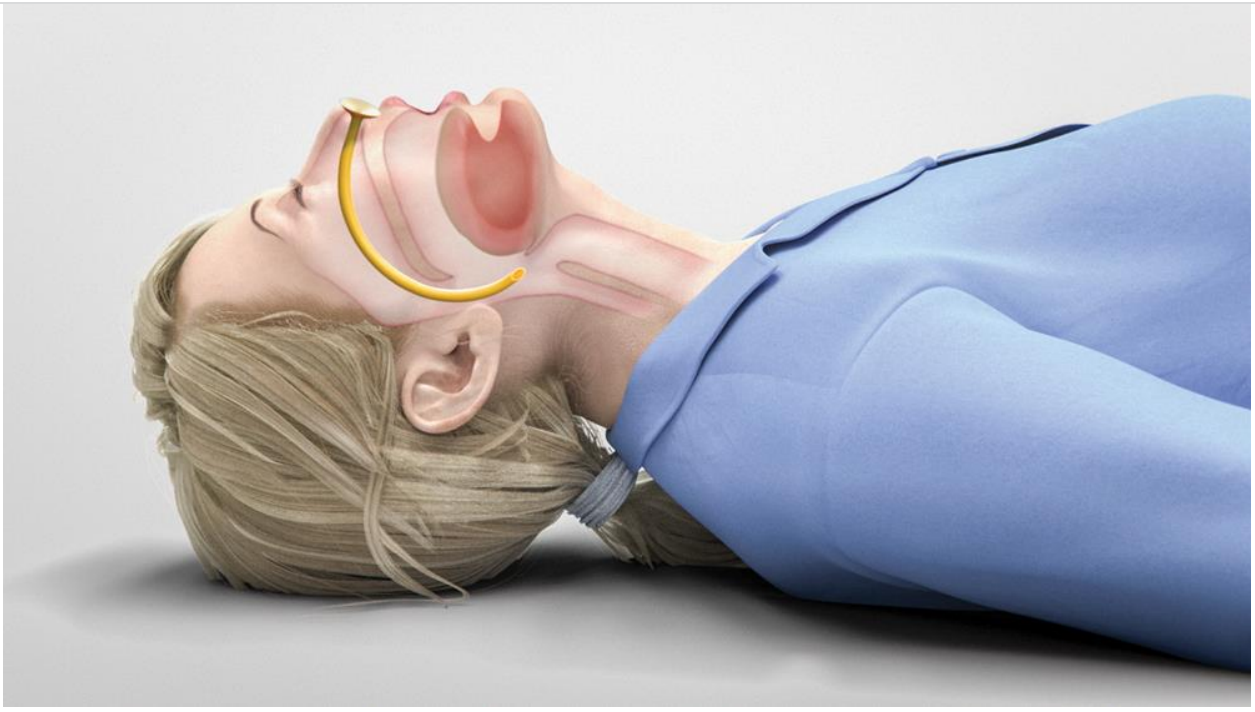


Figure 39C. Nasopharyngeal airway device inserted.

Caution: Using an NPA

- *Insert the airway gently to avoid complications. The airway can irritate the mucosa or lacerate adenoidal tissue and cause bleeding, and the patient could aspirate blood clots. You may need to suction to remove blood or secretions.*
- *An improperly sized NPA may enter the esophagus. With active ventilation, such as bag-mask ventilation, an NPA in the esophagus may cause gastric inflation and possible hypoventilation.*
- *An NPA may cause laryngospasm and vomiting, even though it is commonly tolerated by semiconscious patients.*
- *Use caution in patients with facial trauma because of the risk of misplacement into the cranial cavity through a fractured cribriform plate.*

Caution: Using an OPA or NPA Airway Adjunct

Take the following precautions when using an OPA or NPA:

- *Always check spontaneous respirations immediately after inserting an OPA or an NPA.*
- *If respirations are absent or inadequate, start positive-pressure ventilation at once with an appropriate device.*
- *If OPA, NPA, or other adjuncts are unavailable, provide mouth-to–barrier device ventilations.*

Suctioning

Suctioning is essential to maintain a patient's airway. Suction devices include portable and wall-mounted units.

- Portable suction devices are easy to transport but may not provide adequate suction power.
- Wall-mounted suction units should be able to provide an airflow of more than 40 L/min at the end of the delivery tube and a vacuum of more than −300 mm Hg when the tube is clamped at full suction.
- Suction the airway immediately if the patient has copious secretions, blood, or vomit.

Soft vs Rigid Catheters

For suctioning, you'll use both *soft flexible* and *rigid* catheters.

Use soft flexible catheters (available in sterile wrappers)

- In the mouth or nose
- For ET tube deep suctioning
- For aspiration of thin secretions from the oropharynx and nasopharynx
- To perform intratracheal suctioning
- To suction through an in-place airway (ie, NPA) to access the back of the pharynx in a patient with clenched teeth

Use rigid catheters (eg, Yankauer)

- To suction the oropharynx
- For suctioning thick secretions and particulate matter
- For more effective suctioning of the oropharynx

Oropharyngeal Suctioning Procedure

Follow these steps to perform oropharyngeal suctioning:

- Measure the catheter before suctioning.
- Gently insert the suction catheter or device into the oropharynx beyond the tongue. Do not insert it any further than the distance from the tip of the nose to the earlobe.
- Apply suction by occluding the side opening of the catheter while withdrawing with a rotating or twisting motion.
- If using a rigid suction device, place the tip gently into the oral cavity. Advance by pushing the tongue down to reach the oropharynx if necessary.
- Limit each suction attempt to 10 seconds or less.

ET Tube Suctioning Procedure

Patients with pulmonary secretions may require suctioning even after ET intubation. Follow these steps to perform ET tube suctioning:

- Use a sterile technique to reduce the likelihood of airway contamination.
- Gently insert the catheter into the ET tube but no further because it may injure the ET mucosa or stimulate coughing or bronchospasm. Be sure the side opening is not occluded during insertion.
- Apply suction by occluding the side opening only while withdrawing the catheter with a rotating or twisting motion.

- Do not exceed 10 seconds for a suction attempt. To avoid hypoxemia, precede and follow suctioning attempts with a short period of administration of 100% oxygen.

Monitor the patient's heart rate, pulse, oxygen saturation, and clinical appearance during suctioning. If bradycardia develops, oxygen saturation drops, or clinical appearance deteriorates, interrupt suctioning at once. Administer high-flow oxygen until the heart rate returns to normal and the clinical condition improves. Assist ventilation as needed.

Using Quantitative Waveform Capnography With a Bag-Mask Device

The AHA recommends using quantitative waveform capnography with a bag-mask device to confirm and monitor CPR quality. In addition to using feedback devices for CPR quality, using quantitative waveform capnography can help with real-time adjustment of CPR quality.

Pulse Oximetry

Oxygen saturation can be monitored noninvasively through pulse oximetry. This is a rapid tool to measure and monitor the amount of peripheral oxygen saturation (SpO_2), or oxygen in the blood. Normal pulse oximetry readings should be between 95% and 98%. Give supplemental oxygen when indicated. For cardiac arrest patients, give 100% oxygen. For other clinical conditions, adjust the oxygen administration to achieve an oxygen saturation as follows:

- ACS: 90%
- Stroke: 95% to 98%
- Post–cardiac arrest care: 92% to 98%

Providing Ventilation With an Advanced Airway

Selecting an advanced airway device depends on the high-performance team's training, scope of practice, and equipment. Advanced airways include

- ET tube
- Laryngeal tube
- Laryngeal mask airway

This course will familiarize you with these types of advanced airways but will not discuss how to place them. You will practice ventilating with an advanced airway already in place, and you'll integrate ventilation with chest compressions. To be proficient in using advanced airway devices, you must

have adequate initial training and ongoing experience. Providers who insert advanced airways must participate in a process of CQI to document and minimize complications.

Caution: Advanced Airways

- *Some patients cannot be ventilated with a laryngeal mask airway, so be sure to have an alternative airway management strategy, such as a bag-mask device.*
- *For any advanced airway device, the ventilation rate is once every 6 seconds for respiratory or cardiac arrest.*
- *We* **do not recommend** *the routine use of cricoid pressure in cardiac arrest. Although cricoid pressure in nonarrest patients may protect the airway from aspiration and gastric insufflation during bag-mask ventilation, it also may impede ventilation and interfere with placing a tube or supraglottic airway.*

Only experienced providers should insert these advanced airways.

See ACLS Student Resources for more information about all the advanced airways listed here.

Endotracheal Tube

If you are assisting with *ET intubation*, refer to these basic steps for performing the procedure:

- Prepare for intubation by assembling the necessary equipment.
- Perform ET intubation (see ACLS Student Resources).
- Inflate the cuff or cuffs on the tube.
- Attach the ventilation bag.
- Confirm correct placement by physically examining the patient and using a confirmation device.
    - –Continuous waveform capnography is recommended (in addition to clinical assessment) as the most reliable method of confirming and monitoring correct placement of an ET tube. However, you may use colorimetric and nonwaveform carbon dioxide detectors when waveform capnography is not available.
- Secure the tube in place and monitor for displacement. Use the DOPE mnemonic (displacement, obstruction, pneumothorax, equipment failure) to help you troubleshoot.

Laryngeal Tube

The advantages of the *laryngeal tube* are similar to those of the esophageal-tracheal tube; however, the laryngeal tube is more compact and less complicated to insert. If you are trained to use a laryngeal tube, you may consider it as an alternative to bag-mask ventilation or ET intubation for airway management in cardiac arrest.

Laryngeal Mask Airway

The *laryngeal mask* airway is an advanced airway alternative to ET intubation and provides comparable ventilation for airway management in cardiac arrest.

Precautions for Trauma Patients

When you help ventilate patients with known or suspected cervical spine trauma, avoid moving their head, neck, or spine. This movement can irreversibly injure the spinal cord or worsen a minor spinal cord injury. Approximately 2% of patients with blunt trauma serious enough to require spinal imaging in the ED have a spinal injury, and the risk triples if the patient has a head or facial injury. Assume that any patient with multiple trauma, head injury, or facial trauma has a spine injury, and be particularly cautious if you suspect a cervical spine injury (eg, patients who were in a high-speed crash, fell from a height, or were injured while diving).

Follow these precautions if you suspect cervical spine trauma:

- Open the patient's airway by using a jaw thrust without head extension. But remember that maintaining a patent airway and providing adequate ventilation are your priorities, so use a head tilt–chin lift maneuver if the jaw thrust is not effective.
- Have another team member stabilize the patient's head in a neutral position as you manipulate the airway. *Restrict spinal motion manually rather than with immobilization devices.* Manual spinal immobilization is safer, and cervical collars may complicate airway management or even interfere with airway patency.

Spinal immobilization devices are helpful during transport.

Cardiac Arrest: VF/pVT

Overview

To be successful, any resuscitation attempt needs a strong base of high-quality CPR and defibrillation when the patient's ECG rhythm requires it. Leaders must also assess the performance of each system component, ensuring that system participants can effectively intervene to improve care. This process of quality improvement consists of an iterative and continuous cycle of

- Systematic evaluation of resuscitation care and outcome
- Benchmarking with stakeholder feedback
- Strategic efforts to address identified deficiencies

Another characteristic of high-quality CPR is minimal interruptions in chest compressions. Studies demonstrate that healthcare providers interrupt compressions far too often and for too long, in some cases spending 25% to 50% of a resuscitation attempt *without* delivering chest compressions.

Chest compression fraction (CCF) is the proportion of time during cardiac arrest resuscitation when the rescuer is performing chest compressions. CCF should be as high as possible: ideally greater than 80%. Data suggest lower CCF is associated with decreased ROSC and survival to hospital discharge.

Measurement

Quality improvement relies on valid assessment of resuscitation performance and outcome (refer to the Utstein guidelines in Part 1: Systems of Care).

- Share information among all links in the system of care, including
    - –Dispatch records
    - –EMS patient care report
    - –Hospital records

Benchmarking and Feedback

Systematically review and compare data internally to previous performance and externally to similar systems. Existing registries can help this benchmarking effort. Examples include the

- CARES for OHCA
- Get With The Guidelines®-Resuscitation program for IHCA

Change

By simply measuring and benchmarking care, systems can positively influence outcome, but they'll also need ongoing review and interpretation to identify areas for improvement, such as

- Citizen awareness
- Citizen and healthcare professional education and training
- Increased bystander CPR response rates
- Improved CPR performance
- Shortened time to defibrillation

Rhythms for VF/pVT

- VF (example in Figure 40)
- VT
- ECG artifact that looks like VF
- New LBBB

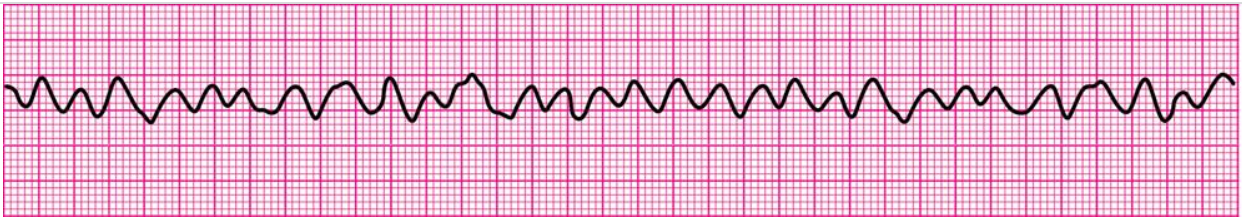


Figure 40. Example of VF.

Drugs for VF/pVT

- Drugs for VF/pVT include
- Epinephrine
- Amiodarone
- Lidocaine
- Magnesium sulfate
- Dopamine
- Oxygen
- Other medications, depending on the cause of the VT/pVT arrest

Managing VF/pVT: The Adult Cardiac Arrest Algorithm

You must know the most important algorithm for adult resuscitation: the Adult Cardiac Arrest Algorithm (Figure 41). This algorithm outlines all the steps to assess and manage a pulseless patient who does not initially

respond to BLS interventions, including a first shock from an AED. The algorithm consists of the 2 pathways for cardiac arrest:

- A shockable rhythm, displayed on the VF/pVT pathway of the algorithm
- A nonshockable rhythm, displayed on the asystole/PEA pathway of the algorithm

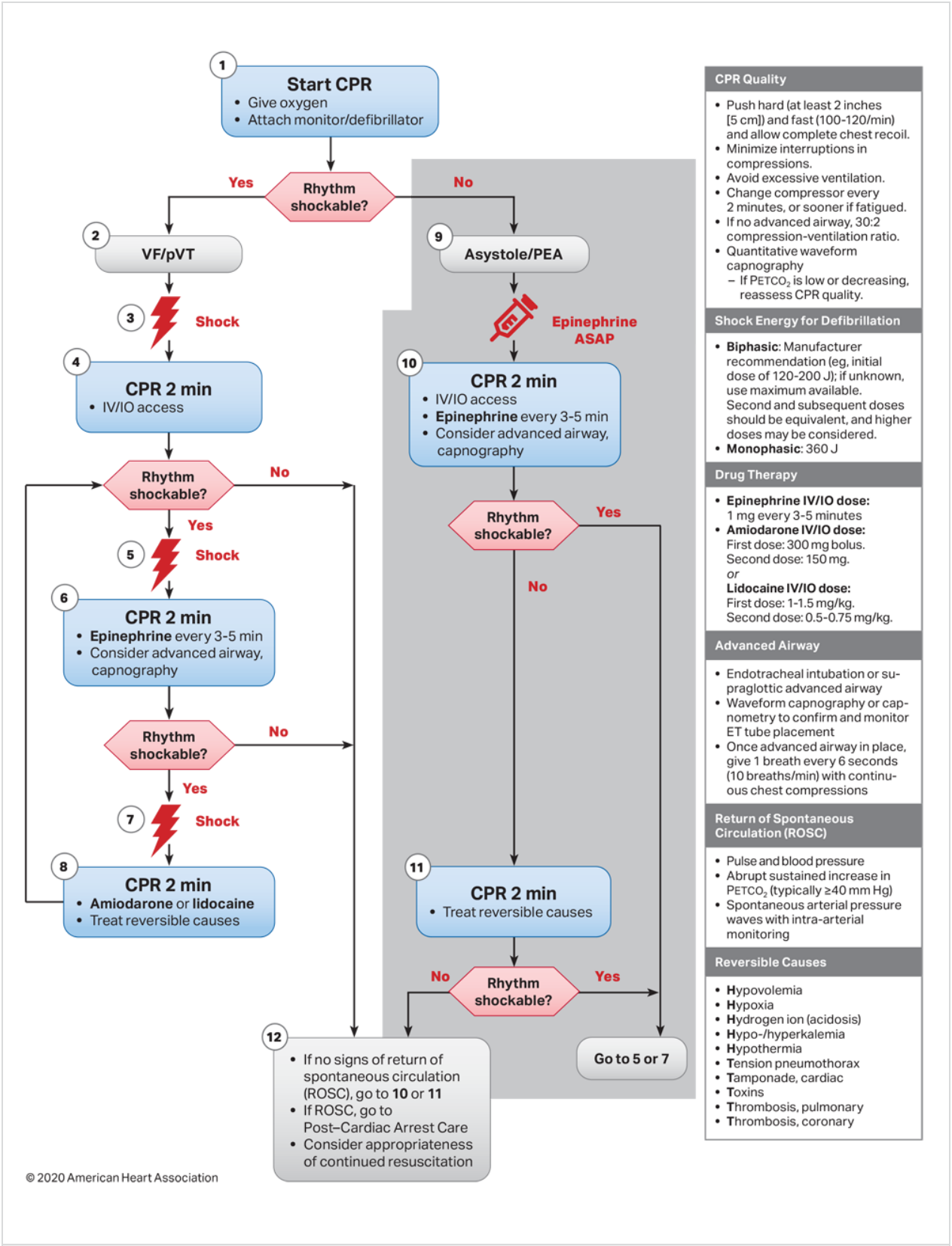


Figure 41. Adult Cardiac Arrest Algorithm, VF/pVT pathway.

Throughout the case discussion of the Adult Cardiac Arrest Algorithm, we will refer to Steps 1 through 12. These are the numbers assigned to the steps in the algorithm.

VF/pVT Path

Because many patients with sudden cardiac arrest demonstrate VF at some point in their arrest, most ACLS providers will often follow the VF/pVT pathway of the Adult Cardiac Arrest Algorithm (Figure 41). Rapidly treating VF according to this sequence is the best approach to restoring spontaneous circulation.

The algorithm includes pVT because it is treated as VF. VF and pVT require CPR until a defibrillator is available to deliver high-energy unsynchronized shocks.

Asystole/PEA Path

The asystole/PEA pathway of the algorithm outlines the sequence of actions to perform if the rhythm is nonshockable. You will practice this sequence in the Asystole and PEA Cases.

During the VF/pVT Case, you will practice performing rapid treatment on the VF/pVT pathway in the Adult Cardiac Arrest Algorithm.

Applying the Adult Cardiac Arrest Algorithm: VF/pVT Pathway

For this algorithm, healthcare providers should have already completed the BLS Assessment, including activating the emergency response system, performing high-quality CPR, attaching the manual defibrillator, and delivering the first shock (Steps 1 through 4). Now, the ACLS high-performance team intervenes and conducts the Primary Assessment. In this case, the team assesses the patient and takes actions as needed. The Team Leader coordinates the efforts of the high-performance team as they complete the steps listed in the VF/pVT pathway of the Adult Cardiac Arrest Algorithm.

Caution: Agonal Gasps

- *Agonal gasps may be present in the first minutes after sudden cardiac arrest.*
- *Agonal gasps are not normal breathing.*

A patient who gasps usually appears to be drawing air in very quickly. The mouth may be open and the jaw, head, or neck may move with gasps. Gasps may appear forceful or weak. Some time may pass between gasps because they usually happen at a slow, irregular rate. The gasp may sound like a snort, snore, or groan.
Gasping is a sign of cardiac arrest.

Start CPR

- Start CPR (Step 1)

The initial step in the Adult Cardiac Arrest Algorithm is to start CPR. As soon as the patient is found to be unresponsive with no breathing (or only gasping), shout for nearby help and activate the emergency response system, send for a defibrillator, check for a pulse, and start CPR, beginning with chest compressions. Attach the ECG monitor or AED pads as soon as they are available. Throughout the resuscitation attempt, provide high-quality CPR (give chest compressions of adequate rate and depth, allow complete chest recoil after each compression, minimize interruptions in compressions, and avoid excessive ventilation).

- Give oxygen.
- Attach the monitor/defibrillator.

Once the monitor/defibrillator is attached, check the rhythm to determine whether it is shockable (VF/pVT) or nonshockable (asystole/PEA) and follow the appropriate cardiac arrest pathway.

Minimize Interruption of Chest Compressions

A team member should continue to perform high-quality CPR until someone brings the defibrillator and attaches it to the patient. The Team Leader assigns roles and responsibilities and organizes interventions to minimize interruptions in chest compressions. This accomplishes the most critical interventions for VF or pVT: CPR with minimal interruptions in chest compressions and defibrillation during the first minutes of arrest. CPR quality should be measured in real time with an audiovisual feedback device, including CCF and quantitative waveform capnography, that captures the following information:

- Rate: 100 to 120/min
- Depth: at least 2 inches (5 cm)
- Chest recoil
- CCF: ideally greater than 80%

- Time to first defibrillation
- Time to first compression

Calculating CCF

Healthcare providers can calculate CCF using a feedback device, or they can calculate it manually by using 2 timers. Use one timer to measure the total code time, from code start until code stop, or until ROSC. Use a second timer to measure the total chest compression time. Each time chest compressions are stopped, pause the second timer until chest compressions are resumed. To calculate CCF, divide chest compression time by the total code time.

CCF = Actual chest compression time ÷ Total code time

The AHA does not recommend continued use of an AED (or the automatic mode) when a manual defibrillator is available and providers can adequately interpret rhythms. Rhythm analysis and shock administration with an AED may prolong the interruptions in chest compressions.

Additionally, while the manual defibrillator is charging, providers should resume CPR. Shortening the interval between the last compression and the shock by even a few seconds can improve shock success (defibrillation and ROSC), so practice efficient coordination between CPR and defibrillation.

For example, after you verify a shockable rhythm and initiate the charging sequence on the defibrillator, another provider should resume chest compressions and continue until the defibrillator is fully charged. You should deliver the shock as soon as the compressor removes his or her hands from the patient's chest and all providers are "clear" of contact with the patient. The same compressor should resume compressions immediately after the shock is delivered.

Note: although manual defibrillators can shorten the interruption needed for rhythm analysis, providers who are inexperienced with rhythm analysis should use an AED instead to avoid delays or inappropriate shocks.

Figure 42 illustrates the need to minimize interruptions in compressions. Coronary perfusion pressure (CPP) is aortic relaxation ("diastolic") pressure minus right atrial relaxation ("diastolic") pressure. During CPR, CPP correlates with both myocardial blood flow and ROSC. In 1 human study,

ROSC did not occur unless a CPP 15 mm Hg or greater was achieved during CPR.

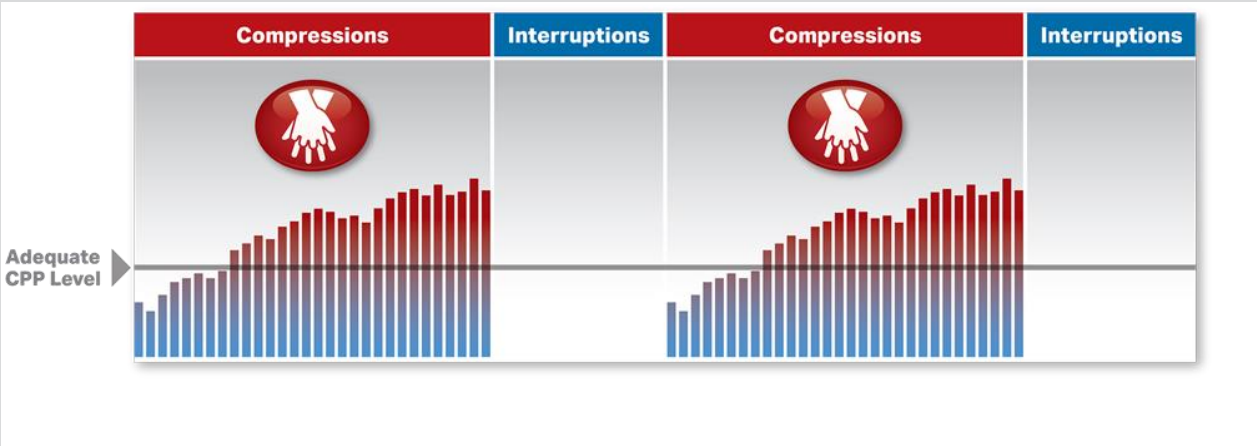


Figure 42. Relationship of quality CPR to CPP demonstrating the need to minimize interruptions in compressions.

Defibrillate (Shockable Rhythm: VF/pVT)

As soon as you determine that the rhythm is shockable (VF or pVT), deliver 1 shock. The appropriate energy dose is determined by the identity of the defibrillator—monophasic or biphasic.

If you are using a *monophasic* defibrillator, give a single 360-J shock. Use the same energy dose for subsequent shocks.

Biphasic defibrillators use various waveforms that effectively terminate VF over a specific dose range. When using biphasic defibrillators, providers should use the manufacturer's recommended energy dose (eg, initial dose of 120 to 200 J). Many biphasic defibrillator manufacturers display the effective energy dose range on the face of the device. If you do not know the effective dose range, deliver the maximal energy dose for the first and all subsequent shocks.

If the initial shock terminates VF but the arrhythmia recurs later in the resuscitation attempt, deliver subsequent shocks at the previously successful energy level.

For an AED, follow the device's prompts or know your device-specific manufacturer's recommendations. Healthcare providers should know how their defibrillator operates and limit pauses in chest compressions to rhythm analysis and shock delivery.

Immediately after the shock, resume CPR, beginning with chest compressions. Give 2 minutes of CPR. If there are available providers, IV or IO access should be established.

In adults experiencing sudden cardiac arrest due to VF or pVT, the heart is quivering but is not effectively pumping blood to vital organs. These patients have a much higher survival rate if they receive immediate chest compressions and early defibrillation. **Timing is critical.** Defibrillation stuns the heart—it doesn't restart the heart—to briefly terminate all electrical activity, including VF and pVT. If the heart is still viable, defibrillation may help the heart's normal pacemakers eventually resume electrical activity (return of spontaneous rhythm) that ultimately results in a perfusing rhythm (ROSC).

In the first 4 to 6 minutes after cardiac arrest, referred to as *clinical death*, no damage occurs to the brain. In the 6- to 10-minute period (biological death) after cardiac arrest, damage is likely to occur to the brain. Brain damage is usually irreversible after 10 minutes, except in special circumstances such as accidental hypothermia and cold-water drowning. Starting chest compressions immediately can delay these effects, and defibrillation can restore a perfusing rhythm. Again, time is critical. A defibrillator should be used as soon as it is available. If there are 2 or more providers present, CPR should be performed while the defibrillator pads are being attached to the patient's chest.

In the first minutes after successful defibrillation, any spontaneous rhythm is typically slow and may not create pulses or adequate perfusion. The patient needs CPR (beginning with chest compressions) for several minutes until adequate heart function resumes. Moreover, not all shocks will lead to successful defibrillation, so resume high-quality CPR beginning with chest compressions immediately after a shock.

The interval from collapse to defibrillation is one of the most important determinants of survival from cardiac arrest, and early defibrillation is critical:

- A common initial rhythm in out-of-hospital witnessed sudden cardiac arrest is VF.
- pVT rapidly deteriorates to VF, and then the heart quivers and does not pump blood.
- Electrical defibrillation is the most effective way to treat VF (delivery of a shock to stop the VF) and pVT.

- The probability of successful defibrillation decreases quickly over time.
- VF deteriorates to asystole if not treated.

The earlier defibrillation occurs, the higher the survival rate. When VF is present, CPR can provide a small amount of blood flow to the heart and brain but cannot directly restore an organized rhythm. Restoring a perfusing rhythm is more likely with immediate CPR and defibrillation within a few minutes after the initial arrest (Figure 43).

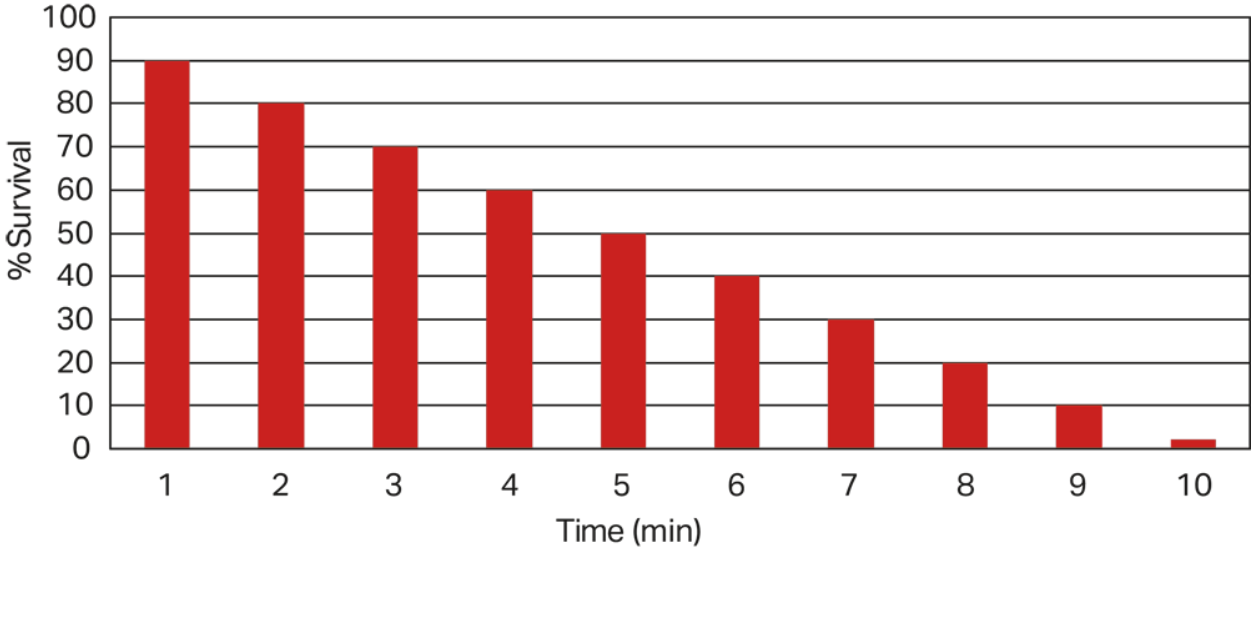


Figure 43. Relationship between survival from VF sudden cardiac arrest and time from collapse to defibrillation.

For every minute that passes between collapse and defibrillation, the chance of survival from a witnessed VF sudden cardiac arrest declines by 7% to 10% per minute without bystander CPR.[2] When bystanders perform CPR, the decline is more gradual and averages 3% to 4% per minute.[2-5] Early CPR can double[2,6] or triple[7] survival from witnessed sudden cardiac arrest at most defibrillation intervals.

Lay rescuer AED programs increase the likelihood of early CPR and attempted defibrillation and shorten the time between collapse and defibrillation for more patients with sudden cardiac arrest.

To ensure safety during defibrillation, always announce the shock warning. State the warning firmly and in a forceful voice before delivering each shock (this entire sequence should take less than 5 seconds):

- “Clear. Shocking.” You do not need to use these exact words, but you must warn others that you are about to deliver shocks and that everyone must stand clear of the patient.
    - –Check to make sure you are clear of contact with the patient, the stretcher, or other equipment.
    - –Make a visual check to ensure that no one is touching the patient or stretcher.
    - –Be sure oxygen is not flowing across the patient’s chest.
- When pressing the shock button, the defibrillator operator should face the patient, not the machine. This helps to ensure coordination with the chest compressor and to verify that no one resumed contact with the patient.

Resume CPR, Establish IV/IO Access, and Check Rhythm

- Perform CPR for 2 minutes.
    - –Immediately resume CPR, beginning with chest compressions. Do not perform a rhythm or pulse check at this point unless the patient is showing signs of life, such as ROSC.
- Establish IV/IO access.
    - –While CPR is being performed, if you do not already have vascular access (IV/IO), another member of the resuscitation team should establish vascular access to get ready for medications.

The Guidelines recommend that healthcare providers tailor the sequence of rescue actions based on the presumed etiology of the arrest. Moreover, ACLS providers can choose the best approach (functioning within a 2-minute cycle) for their high-performance team to minimize interruptions in chest compressions and improve CCF, including protocols such as

- Continuous chest compressions with asynchronous ventilation once every 6 seconds with the use of a bag-mask device
- Compression-only CPR in the first few minutes after arrest

Use a default compression-to-ventilation ratio of 30:2 for less-trained healthcare providers or if 30:2 is the established protocol. Figure 44 shows the progression from lay rescuers to highly trained and proficient healthcare providers.

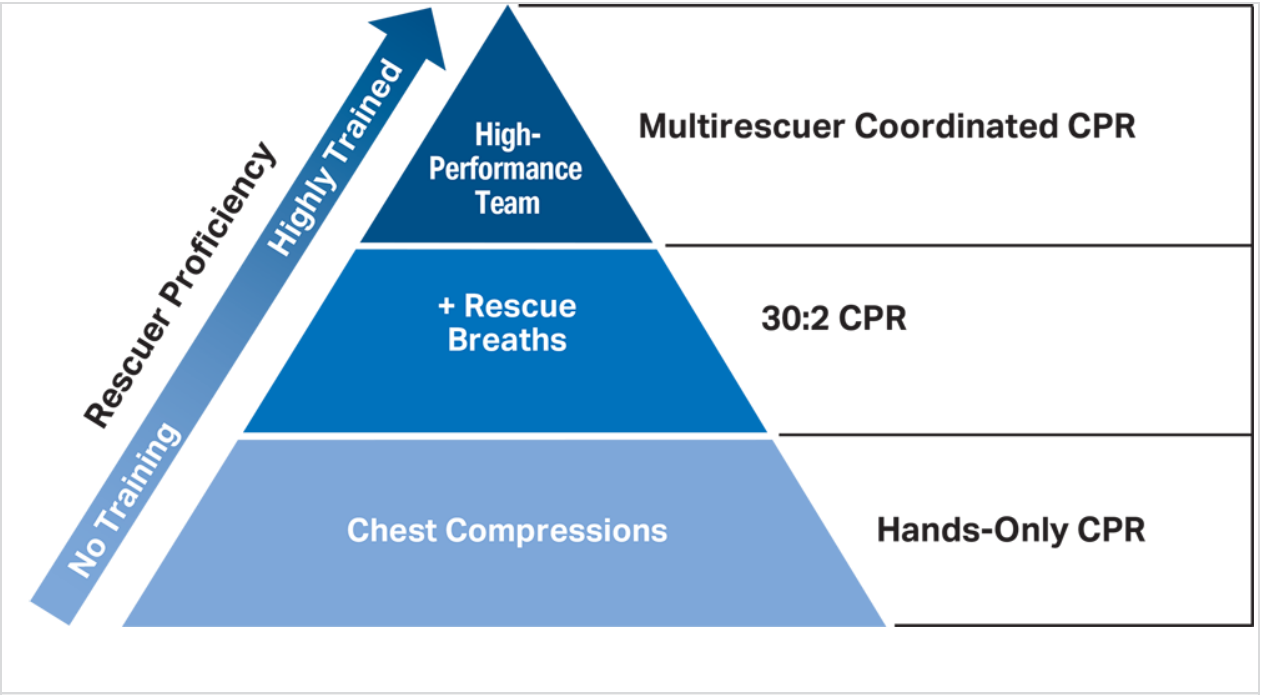


Figure 44. Progression from lay rescuers to highly trained healthcare providers for CPR delivery.

Perform a Rhythm Check

Check the rhythm after 2 minutes of CPR, but be careful to minimize interruptions in chest compressions.

Do not exceed 10 seconds for the pause in chest compressions to check the rhythm.

- If the rhythm is nonshockable and organized, try to find a pulse. If you have any doubt about the presence of a pulse, immediately resume CPR.

Remember to perform a pulse check—preferably during rhythm analysis—**only** if an organized rhythm is present.

- If the rhythm is organized and a pulse is felt, proceed to post–cardiac arrest care.
- If the rhythm is nonshockable and a pulse is not felt, proceed along the asystole/PEA pathway of the Adult Cardiac Arrest Algorithm (Steps 9 through 11).
- If the rhythm is shockable, give 1 shock and immediately resume CPR for 2 minutes after the shock.

Note: the AHA recommends routinely using self-adhesive pads during defibrillation because conductive materials (gel pads or self-adhesive pads)

reduce transthoracic impedance—the resistance that the chest has on electrical current.

For persistent VF/pVT, give 1 shock and immediately resume CPR for 2 minutes, beginning with chest compressions.

Vasopressors

Vasopressors optimize cardiac output and blood pressure, and evidence shows that using vasopressors favors initial resuscitation with ROSC. However, research is still lacking on the effect that routinely using vasopressors during cardiac arrest has on the rates of survival to hospital discharge.

Epinephrine hydrochloride is used during resuscitation primarily for its α-adrenergic effects, ie, vasoconstriction. Vasoconstriction increases cerebral and coronary blood flow during CPR by increasing mean arterial pressure and aortic diastolic pressure. In previous studies, escalating and high-dose epinephrine administration did not improve survival to discharge or neurologic outcome after resuscitation from cardiac arrest.

When IV/IO access is available, give **epinephrine** 1 mg IV/IO during CPR after the second shock, and repeat every 3 to 5 minutes, or every 4 minutes as a midrange (ie, every other rhythm check). If additional team members are available, they should anticipate the need for drugs and prepare them in advance.

No known vasopressor (epinephrine) increases survival from VF/pVT. But because these medications can improve aortic diastolic blood pressure, coronary artery perfusion pressure, and the rate of ROSC, the AHA continues to recommend their use.

Perform a Rhythm Check

Check the rhythm after 2 minutes of CPR, but be careful to minimize interruptions in chest compressions. If the rhythm is shockable, give 1 shock and immediately resume CPR for 2 minutes after the shock.

Antiarrhythmics

Healthcare providers may consider giving antiarrhythmic drugs, either before or after the shock. The focus should be on administering medications quickly and so that defibrillation is not delayed. Evidence is still lacking on whether giving antiarrhythmic drugs during cardiac arrest is

associated with improved survival to hospital discharge. Amiodarone or lidocaine may be considered for VF/pVT that is unresponsive to defibrillation. These drugs may be particularly useful for patients with witnessed arrest, for whom time to drug administration may be shorter.[8]

In ROC-ALPS (Resuscitation Outcomes Consortium–Amiodarone, Lidocaine or Placebo Study), a large out-of-hospital randomized controlled trial that compared captisol-based amiodarone with lidocaine or placebo for patients with VF/pVT refractory after at least 1 shock, there was no overall statistically significant difference in survival with good neurologic outcome or survival to hospital discharge.[9] In that study, ROSC was higher in patients receiving lidocaine compared with those receiving placebo but not for patients receiving amiodarone compared with patients receiving placebo. Among the subgroup of patients with bystander-witnessed cardiac arrest, survival to hospital discharge was higher for patients given amiodarone or lidocaine compared with those given placebo.[8]

Amiodarone or lidocaine may be considered for VF/pVT that is unresponsive to defibrillation. These drugs may be particularly useful for patients with witnessed arrest, for whom time to drug administration may be shorter.[8]

- **Amiodarone:** 300 mg IV/IO bolus, then consider 1 additional 150 mg IV/IO
    - –Amiodarone is considered a class III antiarrhythmic drug, but it possesses electrophysiologic characteristics of the other classes. Amiodarone blocks sodium channels at rapid pacing frequencies (class I effect) and exerts a noncompetitive antisympathetic action (class II effect). One of the main effects of prolonged amiodarone administration is lengthening of the cardiac action potential (class III effect).
- **Lidocaine:** 1 to 1.5 mg/kg IV/IO first dose, then 0.5 to 0.75 mg/kg IV/IO at 5- to 10-minute intervals, to a maximum dose of 3 mg/kg
    - –Lidocaine suppresses automaticity of conduction tissue in the heart by increasing the electrical stimulation threshold of the ventricle, His-Purkinje system, and spontaneous depolarization of the ventricles during diastole by a direct action on the tissues.
    - –Lidocaine blocks permeability of the neuronal membrane to sodium ions, which inhibits depolarization and the blockade of conduction.

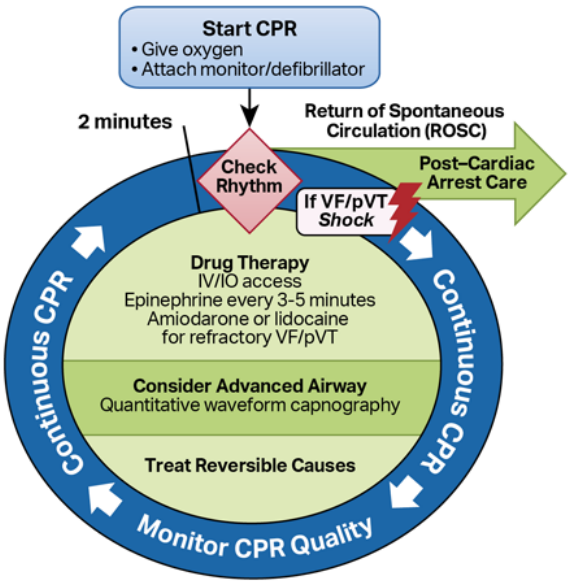
Providers should consider magnesium sulfate for torsades de pointes associated with a long QT interval.

- **Magnesium sulfate** for torsades de pointes, loading dose 1 to 2 g IV/IO diluted in 10 mL (eg, D_5W, normal saline) given as IV/IO bolus, typically over 20 minutes
    - –Magnesium can be classified as a sodium/potassium pump agonist. Magnesium has several electrophysiological effects, including suppression of atrial L- and T-type calcium channels, and ventricular after-depolarizations. Routinely administering magnesium sulfate in cardiac arrest is not recommended unless torsades de pointes is present.

Search for and treat any treatable underlying cause of cardiac arrest, such as the H's and T's.

Cardiac Arrest Treatment Sequences

The Adult Cardiac Arrest Circular Algorithm (Figure 45) summarizes the recommended sequence of CPR, rhythm checks, shocks, and delivery of drugs based on expert consensus. We don't yet know the optimal number of CPR cycles and shocks to perform before starting pharmacologic therapy, but rhythm checks and shocks are organized around 5 cycles of CPR, or 2 minutes if a provider is timing the arrest. Do not delay shock. Continue CPR while preparing and administering drugs and charging the defibrillator. Interrupt chest compressions for only the minimum time required for ventilation (until an advanced airway is placed), rhythm check, and actual shock delivery.



CPR Quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
  - If P_{ETCO_2} is low or decreasing, reassess CPR quality.

Shock Energy for Defibrillation

- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.
  or
- **Lidocaine IV/IO dose:** First dose: 1-1.5 mg/kg. Second dose: 0.5-0.75 mg/kg.

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Figure 45. Adult Cardiac Arrest Circular Algorithm.

Physiologic Monitoring During CPR

For intubated patients, the AHA recommends using quantitative waveform capnography to monitor CPR quality (Figure 46), optimize chest compressions, and detect ROSC during chest compressions (Figure 47). The capnography tracing in Figure 47 displays $PETCO_2$ in millimeters of mercury on the vertical axis over time. This patient is intubated and receiving CPR. Note that the ventilation rate is approximately 10/min. Chest compressions are given continuously at a rate slightly faster than 100/min but are not visible with this tracing. The initial $PETCO_2$ is less than 12.5 mm Hg during the first minute, indicating very low blood flow.

$PETCO_2$ increases to between 12.5 and 25 mm Hg during the second and third minutes, consistent with the increase in blood flow with ongoing resuscitation. ROSC occurs during the fourth minute. ROSC is evident from the abrupt increase in $PETCO_2$ (visible just after the fourth vertical line) to greater than 50 mm Hg, which is consistent with a substantial improvement in blood flow.

Figure 46A. Physiologic monitoring during CPR. A, High-quality compressions are shown through waveform capnography and intra-arterial relaxation pressure.

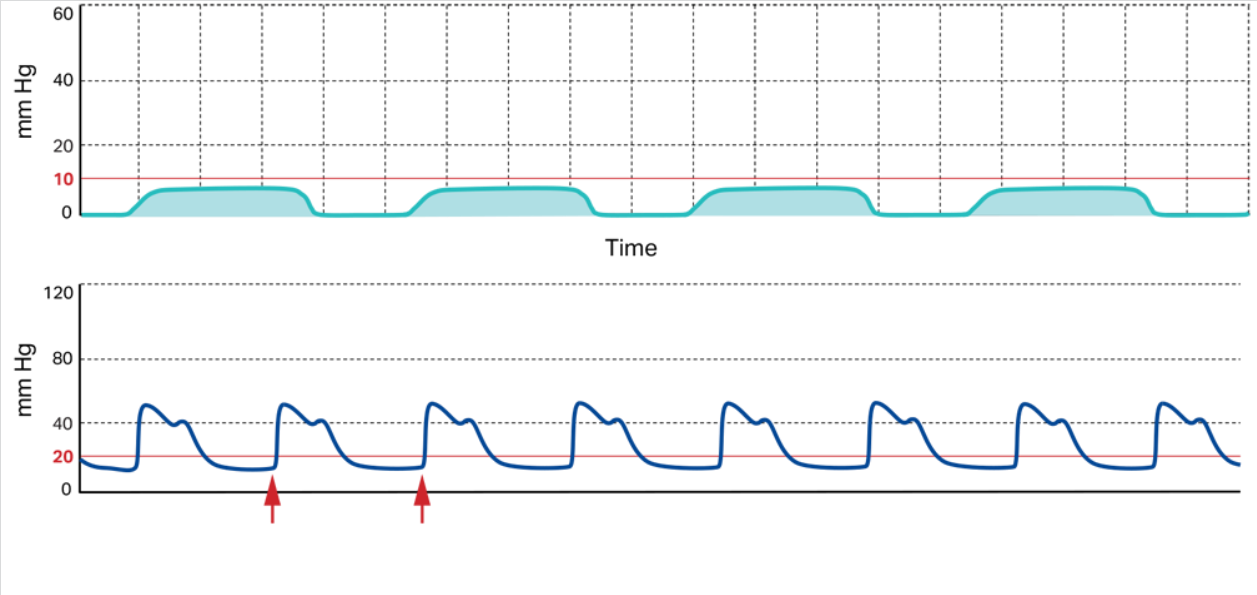


Figure 46B. Ineffective CPR compressions shown through intra-arterial relaxation pressure and waveform capnography.

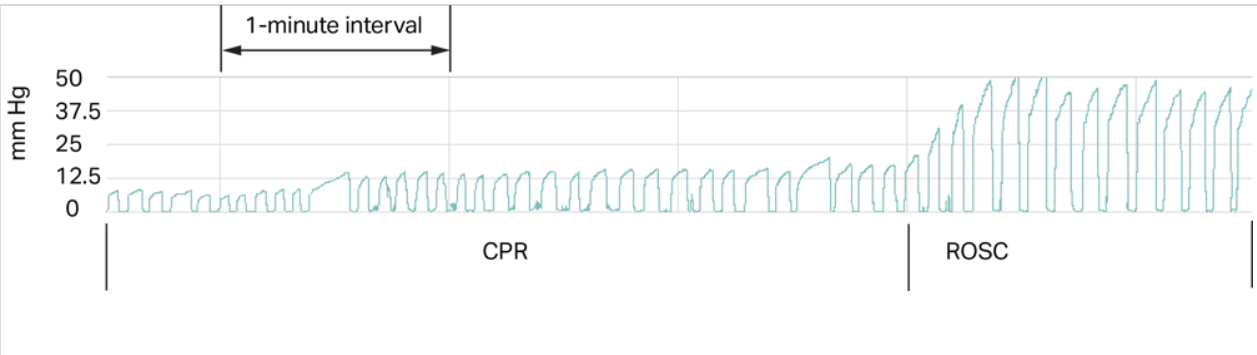


Figure 47. Waveform capnography during CPR with ROSC.

Although invasive monitors are usually not needed during CPR, physiologic parameters such as intra-arterial relaxation pressures (Figures 46A and B) and central venous oxygen saturation ($SCVO_2$) may help optimize CPR and detect ROSC.

Animal and human studies indicate that monitoring $PETCO_2$, CPP, and $SCVO_2$ provides valuable information on the patient's condition and response to therapy.[10-16] These physiologic parameters also correlate with cardiac output and myocardial blood flow during CPR, and when chest compressions fail to achieve identified threshold values, the patient rarely achieves ROSC. Furthermore, an abrupt increase in any of these parameters is a sensitive indicator of ROSC that you can monitor without interrupting chest compressions.

Although no clinical study has examined whether adjusting resuscitative efforts on the basis of physiologic parameters improves outcome, it is reasonable to use these parameters to optimize compressions and guide vasopressor therapy during cardiac arrest.

End-Tidal CO_2

The main determinant of $PETCO_2$ during CPR is blood delivery (cardiac output) to the lungs. Persistently low $PETCO_2$ values less than 10 mm Hg during CPR in intubated patients (Figure 46B) suggest that ROSC is unlikely, and it is reasonable to try to improve chest compressions and vasopressor therapy. If $PETCO_2$ abruptly increases to a normal value of 35 to 40 mm Hg or higher, it is reasonable to consider this an indicator of ROSC.

Coronary Perfusion Pressure or Arterial Relaxation Pressure

Increased coronary perfusion pressure (CPP) correlates with both myocardial blood flow and ROSC. A reasonable substitute for CPP during CPR is arterial relaxation ("diastolic") pressure, which you can measure with an intra-arterial catheter. If the arterial relaxation pressure is less than 20 mm Hg (Figure 46B), it is reasonable to try to improve chest compressions and vasopressor therapy.

Routes of Access for Drugs

The first priority during cardiac arrest is providing high-quality CPR and early defibrillation; inserting an advanced airway and administering drugs are secondary. There is no evidence that any drug used during cardiac arrest improves survival to hospital discharge with improved neurologic function.

Historically in ACLS, providers have administered drugs via IV or ET, but ET absorption of drugs is poor and optimal drug dosing is unknown. For these reasons, the IV route is preferred. When IV access is unsuccessful or not feasible, IO access may be considered.

Intravenous Route

Use a peripheral IV for drug and fluid administration unless central line access is already available. Central line access is not necessary during most resuscitation attempts, and it may cause interruptions in CPR and complications during insertion. These complications include vascular

laceration, hematomas, bleeding, thrombosis, and infection. Inserting a central line in a noncompressible vessel is a relative (not absolute) contraindication to fibrinolytic therapy in patients with ACS.

You do not need to interrupt CPR to establish a peripheral IV line, but drugs typically take 1 to 2 minutes to reach the central circulation via the peripheral IV route. If you give a drug by the peripheral IV route, administer it as follows:

- Give the drug by bolus injection unless otherwise specified.
- Follow with a 20-mL bolus of IV fluid.
- Elevate the extremity for about 10 to 20 seconds to help deliver the drug to the central circulation.

Intraosseous Route

If IV access is not successful or feasible, you can safely and effectively deliver drugs and fluids during resuscitation via the IO route. Important points about IO access are that

- You can establish it in all age groups
- You can achieve it in 30 to 60 seconds
- It is preferable to ET and may be easier to establish in cardiac arrest
- Any ACLS drug or fluid that you administer via IV can be given IO

IO cannulation provides access to a noncollapsible marrow venous plexus, which serves as a rapid, safe, and reliable route during resuscitation for administering drugs, crystalloids, colloids, and blood. The technique requires a rigid needle, preferably a specially designed IO or bone marrow needle from an IO access kit. For more information on IO access, see the Access for Medications section in the ACLS Student Resources.

Endotracheal Route

IV and IO routes are preferred over ET, but if you consider administering drugs via the ET route during CPR, keep these concepts in mind:

- The optimal dose of most drugs given by the ET route is unknown.
- The typical dose of drugs administered via the ET route is 2 to 2½ times the IV route.
- You will need to stop CPR briefly so that the drug does not regurgitate up the ET tube.
- Drugs like epinephrine can negatively affect the colorimetric CO_2 detector’s functionality.

Studies demonstrate that the circulatory system absorbs epinephrine, vasopressin, and lidocaine after administration via the ET route. When giving drugs via the ET route, dilute the dose in 5 to 10 mL of sterile water or normal saline, and inject it directly into the ET tube.

Fluid Administration

Adjust fluid administration and vasoactive or inotropic agents as needed to optimize blood pressure, cardiac output, and systemic perfusion. The optimal post–cardiac arrest blood pressure remains unknown; however, a mean arterial pressure 65 mm Hg or greater is a reasonable goal.

In hypovolemic patients, the ECF volume is typically restored with normal saline or lactated Ringer's solution, and avoid D_5W because it will reduce serum sodium too rapidly. Monitor serum electrolytes as appropriate.

Ultrasound for VF/pVT/Asystole/PEA

Ultrasound may be applied to patients receiving CPR to help assess myocardial contractility and identify potentially treatable causes of cardiac arrest, such as hypovolemia, pneumothorax, pulmonary thromboembolism, or pericardial tamponade. However, it is unclear whether routinely using ultrasound among patients experiencing cardiac arrest affects important clinical outcomes. If a qualified sonographer is present and use of ultrasound does not interfere with the standard cardiac arrest treatment protocol, then consider ultrasound as an adjunct to standard patient evaluation.

Return of Spontaneous Circulation

If resuscitative efforts successfully restore an organized rhythm (or you find other evidence of return of spontaneous circulation (ROSC), such as pulse and blood pressure, an abrupt and sustained increase in $PETCO_2$ (typically 40 mm Hg or higher) or spontaneous arterial pressure waves with intra-arterial monitoring, go to the Adult Healthcare Provider Post–Cardiac Arrest Care Algorithm.

If no signs of ROSC, resume CPR, administer epinephrine, and treat reversible causes. Consider appropriateness of continued resuscitation.

Cardiac Arrest: PEA and Asystole

Overview

During the BLS Assessment, high-performance team members will demonstrate high-quality CPR with effective chest compressions and ventilation with a bag-mask device. In the Primary Assessment, the Team Leader will recognize PEA or asystole and implement the appropriate interventions outlined in the Adult Cardiac Arrest Algorithm. Because correcting an underlying cause of PEA or asystole, if present and identified, is critical to patient outcome, the Team Leader will state the differential diagnosis while leading the high-performance team to find and treat reversible causes.

Rhythms for PEA

You will need to recognize the following rhythms:

- Rate—too fast or too slow
- Width of QRS complexes—wide vs narrow

Rhythms for Asystole (Lack of Rhythm)

You will need to recognize asystole (Figure 48) and slow PEA terminating in a bradyasystolic rhythm.

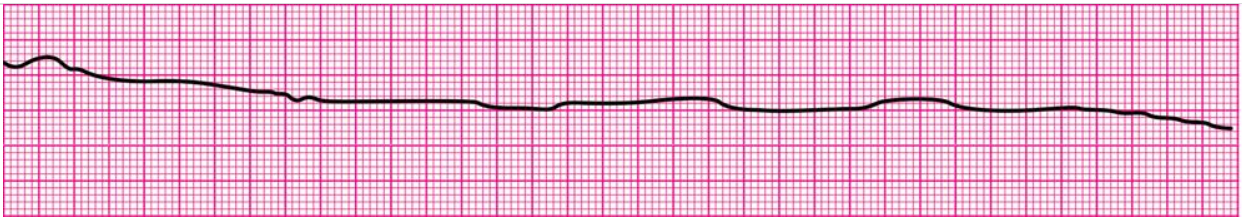


Figure 48. Example of asystole.

Drugs for PEA and Asystole

Drugs for PEA and asystole include

- Epinephrine
- Other medications, depending on the cause of the PEA and asystole arrest

Description of PEA

PEA refers to a situation where the heart generates electrical activity that should correspond to a pulse but no pulse can be palpated. PEA encompasses a heterogeneous group of rhythms that are organized or semiorganized. An organized rhythm consists of QRS complexes that are similar in appearance from beat to beat (ie, each has a uniform QRS configuration). Organized rhythms may have narrow or wide QRS complexes, they may occur at rapid or slow rates, and they may be regular or irregular.

Any organized rhythm without a pulse is defined as PEA, including sinus rhythm, atrial fibrillation or flutter, bundle branch blocks, and idioventricular or ventricular escape rhythms, etc. The heart is not pumping enough blood to sustain cardiac perfusion and the primary therapeutic approach relies on addressing the underlying cause of the arrest rather than converting to a different cardiac rhythm. Pulseless rhythms that are excluded include VF and pVT, which respond best to immediate electrical therapy, and asystole, which is treated similarly to PEA but is excluded by definition.

Differential Diagnosis in PEA

Previously, high-performance teams used the term *electromechanical dissociation* to describe patients who displayed electrical activity on the cardiac monitor but lacked apparent contractile function because of an undetectable pulse. That is, weak contractile function is present—detectable by invasive monitoring or echocardiography—but the cardiac function is too weak to produce a pulse or effective cardiac output. This is the most common initial condition present after successful defibrillation.

PEA also includes other conditions where the left ventricle of the heart is empty because of inadequate preload. In this case, the contractile function of the heart is adequate, but there is inadequate volume for the ventricle to eject. This may occur as a result of severe hypovolemia or as a result of decreased venous return from PE, cardiac tamponade, or tension pneumothorax.

If good CPR produces a strong pulse, relatively high $ETCO_2$ or blood pressure, it is more likely that the left ventricle is full and the cause of PEA is a poorly contractile left ventricle; conversely, if good CPR still does not produce evidence of good cardiac output, it is more likely that the left ventricle is relatively empty. This may help to focus the differential on the more likely causes when considering the H’s & T’s.

Approach to Asystole

Asystole is a cardiac arrest rhythm associated with no discernible electrical activity on the ECG (also called *flat line*). You should confirm that the flat line on the monitor is indeed true asystole by validating that the flat line is

- Not some other rhythm (eg, fine VF) masquerading as a flat line
- Not the result of an artifact associated with a disconnected lead or incorrect lead setting (eg, lead set to the pads when they are not on the patient)

Asystole and Technical Problems

Asystole is a specific diagnosis, but the term *flat line* is nonspecific and can result from several possible conditions, including absence of cardiac electrical activity, equipment failure, and operator error. Some defibrillators and monitors signal the operator when a lead or other equipment failure occurs, but some do not.

For a patient with cardiac arrest and asystole, quickly rule out any other causes of an isoelectric ECG, such as loose leads or leads that are not connected to the patient or defibrillator or monitor; no power; or amplitude or signal strength that is too low.

Asystole as an End Point

Often, asystole represents the final rhythm, including for a patient initially in VF or pVT. Cardiac function diminishes until electrical and functional cardiac activity finally stop and the patient dies.

Prolonged efforts are unnecessary and futile unless special resuscitation situations exist, such as hypothermia and drug overdose. Consider stopping if $ETCO_2$ is less than 10 mm Hg after 20 minutes of CPR and all reversible causes of cardiac arrest have been addressed.

Asystole: An Agonal Rhythm?

You will see asystole most often in 2 situations:

- As a terminal rhythm in a resuscitation attempt that started with another rhythm
- As the first rhythm identified in a patient with unwitnessed or prolonged arrest

Persistent asystole represents extensive myocardial ischemia and damage from prolonged periods of inadequate coronary perfusion. Prognosis is

poor unless a special resuscitation circumstance or immediately reversible cause is present.

Managing Asystole/PEA: The Adult Cardiac Arrest Algorithm

The Adult Cardiac Arrest Algorithm consists of 2 cardiac arrest pathways (Figure 49): the treatment for a shockable rhythm (VF/pVT), and the treatment for a nonshockable rhythm (asystole/PEA). Because of the similarity in causes and management, the Adult Cardiac Arrest Algorithm combines the asystole and PEA pathways, but we will review these rhythms in separate cases. In both pathways, therapies are organized around 2-minute periods of uninterrupted, high-quality CPR.

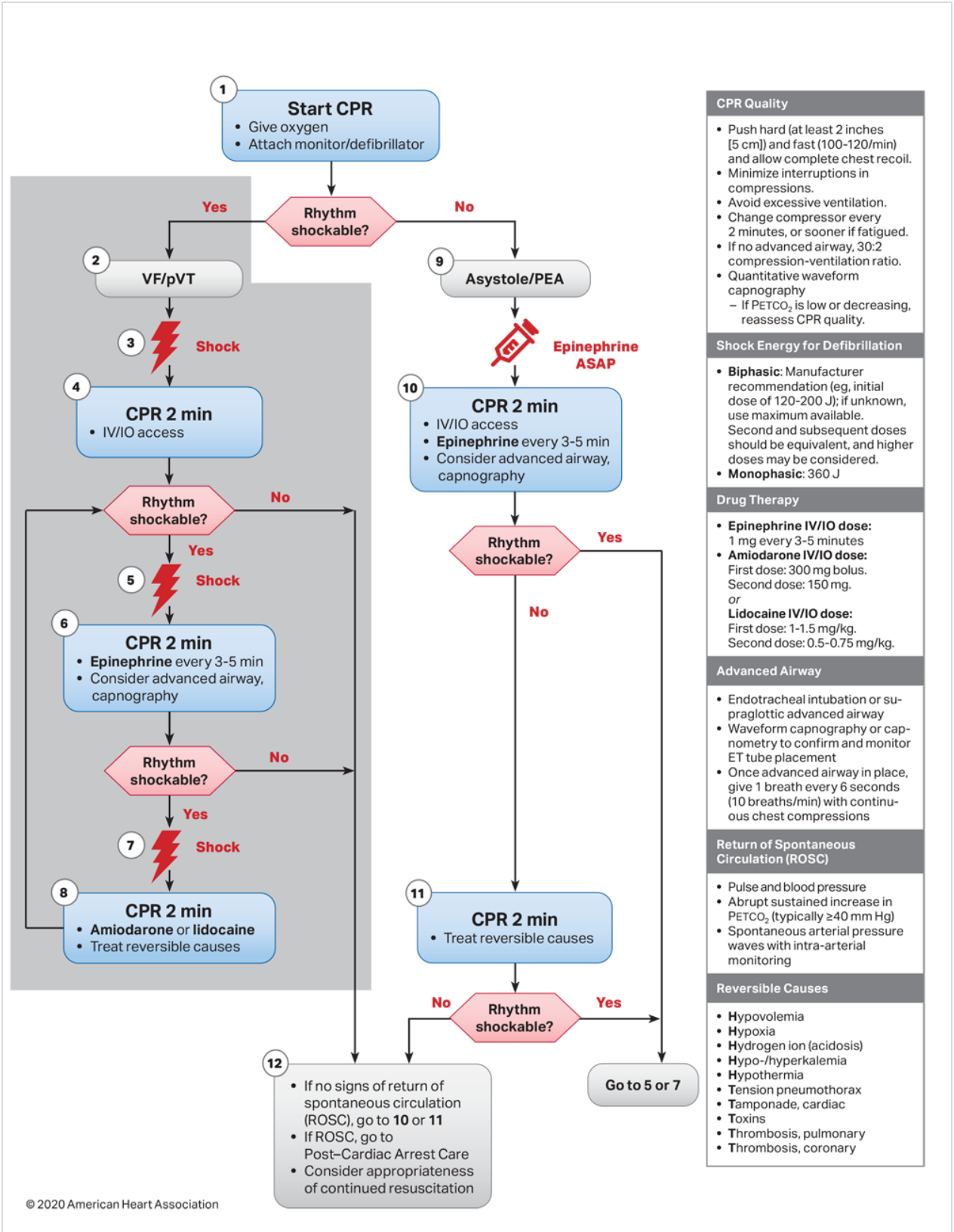


Figure 49. Adult Cardiac Arrest Algorithm, asystole/PEA pathway.

A good resuscitation outcome with return of a perfusing rhythm and spontaneous respirations requires the high-performance team to provide effective CPR and identify and correct the cause of PEA if present.

A high-performance team must seamlessly carry out the steps outlined in the algorithm while simultaneously working to identify and treat reversible causes of the arrest.

The Asystole/PEA Pathway of the Cardiac Arrest Algorithm

In this case, *the patient is in cardiac arrest*. High-performance team members initiate and perform high-quality CPR throughout the BLS, Primary, and Secondary Assessments. The team interrupts CPR for 10 seconds or less for rhythm and pulse checks.

- Start CPR (Step 1)

The initial step in the Adult Cardiac Arrest Algorithm is to start CPR. As soon as the patient is found to be unresponsive with no breathing (or only gasping), shout for nearby help and activate the emergency response system, send for a defibrillator, check for a pulse, and start CPR, beginning with chest compressions. Attach the ECG monitor or AED pads as soon as they are available. Throughout the resuscitation attempt, provide high-quality CPR (give chest compressions of adequate rate and depth, allow complete chest recoil after each compression, minimize interruptions in compressions, and avoid excessive ventilation).

- Give oxygen.
- Attach the monitor/defibrillator.

Once the monitor/defibrillator is attached, check the rhythm to determine whether it is shockable (VF/pVT) or nonshockable (asystole/PEA) and follow the appropriate cardiac arrest pathway.

Managing Asystole/PEA

This patient has an organized rhythm on the monitor but no pulse. The condition is PEA (Step 9). Resume chest compressions immediately. The Team Leader now directs the team in the steps outlined in the asystole/PEA pathway of the Adult Cardiac Arrest Algorithm (Figure 49).

Critical Concepts: Administer Epinephrine

Give epinephrine as soon as IV/IO access becomes available.

- *With respect to timing, for cardiac arrest with a nonshockable rhythm, it is reasonable to administer epinephrine as soon as feasible.*
- *Epinephrine 1 mg IV/IO—repeat every 3 to 5 minutes, or every 4 minutes as a midrange (ie, every other rhythm check)*

Administer drugs during CPR. Do not stop CPR to administer drugs. *With respect to timing, for cardiac arrest with a nonshockable rhythm, it is reasonable to administer epinephrine as soon as feasible. A recent systematic review found an association between earlier epinephrine and ROSC for patients with nonshockable rhythms, although improvements in survival were not universally seen.*[17]

Prioritize establishing IV/IO access over managing an advanced airway unless bag-mask ventilation is ineffective or hypoxia caused the arrest. All high-performance team members must search for an underlying and treatable cause of the PEA while they perform their assigned roles.

Perform a Rhythm Check

Check the rhythm and give 2 minutes of CPR after administering the drugs, but be careful to minimize interruptions in chest compressions.

Do not exceed 10 seconds for the pause in chest compressions to check the rhythm.

Consider advanced airway and capnography.

Nonshockable Rhythm

- If *no electrical activity is present* (asystole), repeat the sequence.
- If organized electrical activity is present, try to feel for a pulse. Take at least 5 seconds but no more than 10 seconds to check for a pulse.
- If *no pulse is present*, or if you have any doubt about the presence of a pulse, immediately resume CPR for 2 minutes, starting with chest compressions, and then repeat the sequence.
- If a pulse is present and the rhythm is organized, begin post–cardiac arrest care.
- Depending on the patient’s status and the length of time you have been performing CPR, consider the appropriateness of continuing the resuscitation attempt.

Decision Point: Shockable Rhythm

- If the rhythm check reveals a shockable rhythm, resume CPR with chest compressions while the defibrillator is charging.
- Switch to the VF/pVT sequence in the algorithm, starting with Step 5 or 7.

Asystole/PEA Treatment Sequences

Figure 49 summarizes experts' recommended sequence of CPR, rhythm checks, and delivery of drugs for PEA and asystole.

Identifying and Correcting Underlying Causes

Treating asystole/PEA goes beyond the interventions in the algorithm. As you assess the patient, try to identify evidence of an underlying cause and correct it if present. Stop, think, and ask, "Why did this person have this cardiac arrest at this time?" You must search for and treat reversible causes of asystole/PEA for resuscitative efforts to be potentially successful. Use the H's and T's to recall conditions that could have contributed to asystole/PEA, and remember that hypovolemia and hypoxia are the 2 most common underlying, potentially reversible causes of asystole/PEA.

When in Doubt

If it is unclear whether the rhythm is fine VF or asystole/PEA, an initial attempt at defibrillation may be warranted. Fine VF may result from a prolonged arrest. At this time, the benefit of delaying defibrillation to perform CPR first is unclear. EMS system medical directors may consider implementing a protocol that allows EMS responders to provide CPR while preparing for defibrillation of patients who EMS personnel identify as being in VF.

Patients With DNAR Orders

During the BLS, Primary, and Secondary Assessments, you should be aware of reasons to stop or withhold resuscitative efforts. Some of these are

- Rigor mortis
- Indicators of do-not-attempt-resuscitation (DNAR) status (eg, bracelet, anklet, written documentation)
- Threat to the safety of providers

Out-of-hospital providers need to know EMS-specific policies and protocols applicable to these situations. In-hospital providers and high-performance

teams should know of any advance directives or specific limits to resuscitation attempts that are in place. For example, a patient may consent to CPR and defibrillation but not to intubation or invasive procedures, and many hospitals will record this in the medical record. If the DNAR order is unclear or uncertain, resuscitation should be initiated and continued until it can be clarified.

Terminating Resuscitative Efforts

In-Hospital

If healthcare providers cannot rapidly identify an underlying cause and the patient does not respond to the BLS and ACLS interventions, consider terminating all resuscitative efforts.

The decision to terminate resuscitative efforts rests with the treating physician in the hospital and is based on many factors, including

- Time from collapse to CPR
- Time from collapse to first defibrillation attempt
- Comorbid disease
- Prearrest state
- Initial arrest rhythm
- Response to resuscitative measures
- $ETCO_2$ less than 10 after 20 minutes of high-quality CPR

None of these factors alone or in combination clearly predicts outcome, but the duration of resuscitative efforts is an important factor associated with poor outcome. The chance that the patient will survive to hospital discharge neurologically intact diminishes as resuscitation time increases. *Extracorporeal CPR* (ECPR) refers to the initiation of cardiopulmonary bypass during the resuscitation of a patient in cardiac arrest, with the goal of supporting end-organ perfusion while potentially reversible conditions are addressed. Consider the appropriateness of continued resuscitative efforts and stop the resuscitation attempt when you determine with a high degree of certainty that the patient will not respond to further ACLS and ECPR is not indicated or not available.

Out-of-Hospital

Continue out-of-hospital resuscitative efforts until 1 of the following occurs:

- Restoration of effective, spontaneous circulation and ventilation
- Transfer of care to a senior emergency medical professional
- Reliable criteria indicate irreversible death

- Exhaustion or dangerous environmental hazards prevent the healthcare provider from continuing
- Continued resuscitation places the lives of others in jeopardy
- A valid DNAR order is presented
- Online authorization comes from the medical control physician or there is prior medical protocol for termination of resuscitation

Duration of Resuscitative Efforts

The final decision to stop resuscitative efforts can never be as simple as an isolated time interval. If ROSC of any duration occurs, it may be appropriate to consider extending the resuscitative effort.

Experts have developed clinical rules to help decide whether to terminate resuscitative efforts for in-hospital and out-of-hospital arrests. Familiarize yourself with the established policy or protocols for your hospital or EMS system.

When deciding whether to extend resuscitative efforts, it may be appropriate to consider other issues, including drug overdose and severe prearrest hypothermia (eg, submersion in icy water). Special resuscitation interventions (such as ECPR) and prolonged resuscitative efforts may be indicated for patients with hypothermia, drug overdose, or other potentially reversible causes of arrest.

Ethical Considerations

High-performance teams must make a conscientious and competent effort to give patients a trial of CPR and ACLS, if the patient did not express a decision to forego resuscitative efforts and is not obviously dead (eg, rigor mortis, decomposition, hemisection, decapitation) (see the DNAR discussion in the ACLS Student Resources). The final decision to stop resuscitative efforts can never be as simple as an isolated time interval.

Human, Ethical, and Legal Dimensions of CPR in the ACLS Student Resources provides additional information on these considerations.

Transporting Patients in Cardiac Arrest

Emergency medical response systems should not require field personnel to transport every cardiac arrest patient to a hospital or an ED. However, transportation with continuing CPR is justified if personnel cannot perform interventions out-of-hospital that are available in the hospital and that are

needed for special circumstances (ie, cardiopulmonary bypass or extracorporeal circulation for patients with severe hypothermia).

After OHCA with ROSC, transport the patient to an appropriate hospital with a comprehensive post–cardiac arrest treatment system of care that includes acute coronary interventions, neurologic care, critical care, and hypothermia. Transport in-hospital post–cardiac arrest patients to an appropriate critical care unit that can provide comprehensive post–cardiac arrest care.

Cardiac Arrest: Selected Special Situations

Treating VF/pVT in Accidental Hypothermia

Defibrillation is appropriate for cardiac arrest patients in VF/pVT with severe accidental hypothermia (a body temperature of less than 30°C [86°F]). If a patient does not respond to the initial shock, it is reasonable to perform additional defibrillation attempts by using BLS guidelines while actively rewarming. Hypothermic patients may have a reduced rate of drug metabolism, and drugs may accumulate to toxic levels with standard dosing regimens. It is reasonable to consider administering a vasopressor according to the standard ACLS algorithm while rewarming, although evidence does not support using antiarrhythmic drug therapy for hypothermic patients in cardiac arrest.

For patients in cardiac arrest with severe accidental hypothermia in-hospital, aim ACLS treatment at rapid core rewarming.

For patients in cardiac arrest with moderate hypothermia (30°C to 34°C [86°F to 93.2°F]), start CPR, attempt defibrillation, give medications according to local protocols, and, if in-hospital, provide active core rewarming.

Respiratory or Cardiac Arrest Associated With Opioid Overdose

- In the United States, between 2000 and 2014, there was a 200% increase in the rate of overdose deaths involving opioids.[18] In 2018, approximately 46 800 people died of opioid toxicity in the United States.[19] Most of these deaths were associated with prescription opioids. In Canada, more than 12 800 deaths were attributed to opioids between January 2016 and March 2019.[20]
- Isolated opioid toxicity is associated with central nervous system (CNS) and respiratory depression that can progress to respiratory

and cardiac arrest. Most opioid deaths involve ingesting multiple drugs or having medical and mental health comorbidities.[21-23] In addition, methadone and propoxyphene can cause torsades de pointes, and cardiotoxicity has been reported with other opioids.[24-30] Except in specific clinical settings (eg, unintended opioid overdose during a medical procedure), rescuers cannot be certain that the patient's clinical condition is due to opioid-induced CNS and respiratory depression toxicity alone.

- Naloxone is a potent opioid receptor antagonist in the brain, spinal cord, and GI system. Naloxone has an excellent safety profile and can rapidly reverse CNS and respiratory depression in a patient with an opioid-associated resuscitative emergency. Depending on their training and clinical circumstance, rescuers can administer naloxone intravenously,[31-34] intramuscularly,[31,32,35] intranasally,[33,35-39] or subcutaneously;[40] nebulize it for inhalation;[41,42] or instill it into the bronchial tree via ET tube.[43]

Management of Opioid-Associated Life-Threatening Emergency

Refer to the following steps in the Opioid-Associated Emergency for Healthcare Providers Algorithm (Figure 50) to manage an opioid-associated life-threatening emergency.

- Suspected opioid poisoning (Step 1):
    - –Check for responsiveness.
    - –Shout for nearby help.
    - –Activate the emergency response system.
    - –Get naloxone and an AED if available.
- Is the person breathing normally (Step 2)?
    - –Yes:
        - Prevent deterioration by checking for responsiveness (tap and shout), open airway and reposition, consider naloxone, transport to the hospital (Step 3).
        - Perform an ongoing assessment of responsiveness and breathing (Step 4).
    - –No. Does the person have a pulse (assess for 10 seconds or less) (Step 5)?

- If the person has a pulse, the provider should support ventilation by opening the airway and repositioning, providing rescue breathing or bag-mask ventilation, and giving naloxone (Step 6).
- If the person does not have a pulse, the provider should start CPR, use an AED, consider naloxone, and refer to BLS and ALS protocols (Step 7).

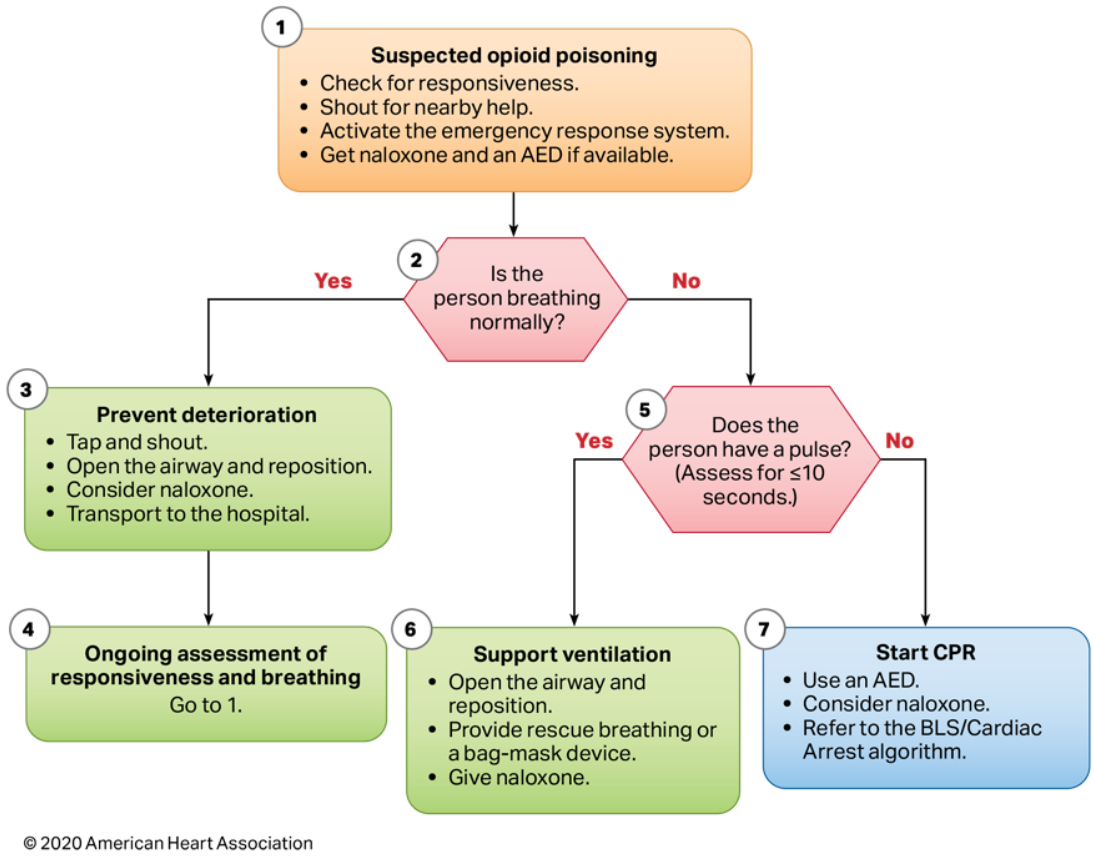


Figure 50. Opioid-Associated Emergency for Healthcare Providers Algorithm.

ECPR (for VF/pVT/Asystole/PEA)

ECPR refers to venoarterial extracorporeal membrane oxygenation during cardiac arrest. ECPR techniques require adequate vascular access and specialized equipment (Figure 51). By using ECPR, providers may support

vital organs with perfusion and gas exchange while reversible causes of cardiac arrest (eg, acute coronary artery occlusion, PE, refractory VF, profound hypothermia, cardiac injury, myocarditis, cardiomyopathy, congestive heart failure, drug intoxication) are treated. ECPR can also serve as a bridge for LV assist device implantation or cardiac transplantation.

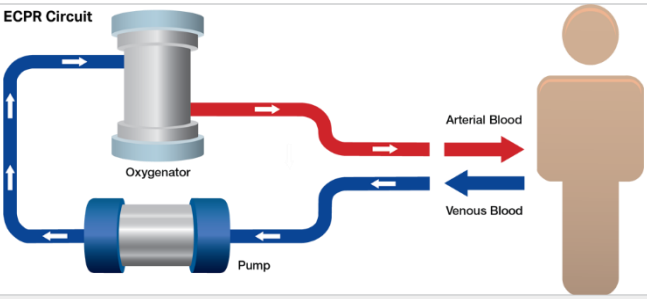


Figure 51. Schematic depiction of components of ECMO circuit as used for ECPR. Components include a venous cannula, a pump, an oxygenator, and an arterial cannula.

Abbreviations: ECMO, extracorporeal membrane oxygenation; ECPR, extracorporeal cardiopulmonary resuscitation.

Currently, ECPR requires vascular access with large bore cannulas inserted into the central vasculature, specialized equipment, and expertise in using ECMO, but evidence suggests a benefit to survival and favorable neurologic outcome with the use of ECPR when compared with conventional CPR in patients with refractory cardiac arrest.

Consider ECPR in settings where the necessary equipment and trained personnel can be deployed rapidly for select cardiac arrest patients with known or suspected reversible causes of cardiac arrest in whom conventional ACLS has failed**.**

Ventricular Assist Devices

Mechanical circulatory support devices, also called *ventricular assist devices* (VADs), can support the function of the ventricles with[44]

- A left ventricle with a left ventricular assist device (LVAD)
- A right ventricle with a right ventricular assist device (RVAD)
- Both ventricles with a biventricular assist device

Figure 52 shows the support intended with an LVAD, an RVAD, and a biventricular assist device. Most VADs are implanted inside the thoracic/abdominal cavity (intracorporeal, Figure 53). These devices pump

blood from the weakened ventricle back into circulation. With an LVAD, blood enters the device from the left ventricle and is pumped to the central aortic circulation, assisting the heart.[44]

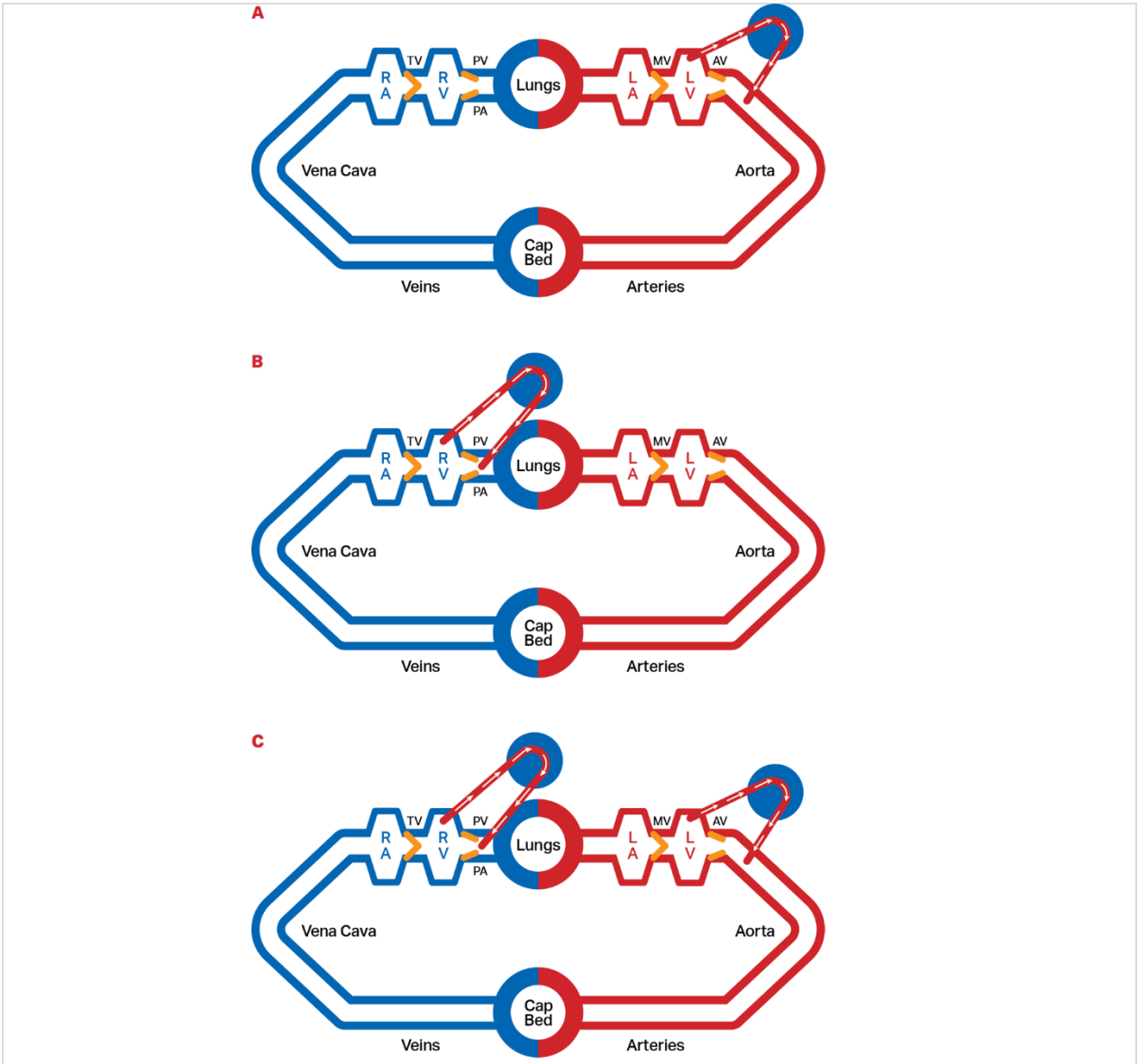


Figure 52. Ventricular assist device configurations of A, an LVAD; B, an RVAD; and C, a biventricular assist device.

Abbreviations: LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

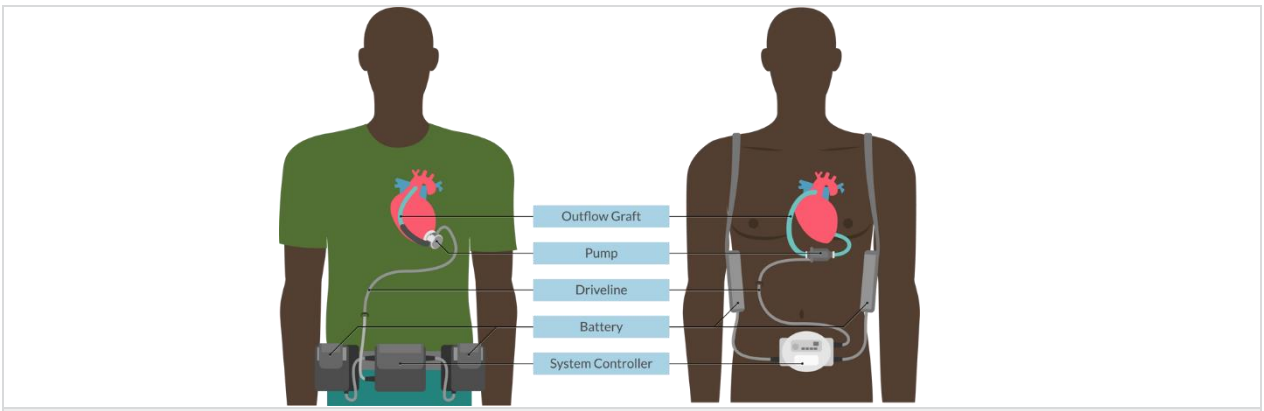


Figure 53. Intracorporeal pumps.

With an RVAD, the inflow is the right ventricle or atrium, and the outflow is the main pulmonary artery, just distal to the pulmonic valve. When an LVAD and an RVAD are used in the same patient simultaneously, the patient is referred to as having *biventricular support* or a *biventricular assist device*, indicating that both ventricles are supported mechanically.

A total artificial heart replaces the heart itself. Most patients who are discharged home with mechanical circulatory support currently have a durable LVAD.

LVADs can have 2 distinctly different mechanisms of blood flow and, therefore, are different physiologically:

- Pulsatile-flow LVADs (older technology, rarely used)
- Continuous-flow LVADs (the current generation of devices)

Because palpable pulses are often absent in patients with continuous-flow LVADs, it is important to understand the differences in the physical exam and in methods that can help rescuers determine if an unresponsive or mentally altered patient is, in fact, in cardiac arrest or circulatory collapse.

The 2 most common causes of pump failure are disconnection of the power or of the driveline. Therefore, the first step in assessing an unresponsive, mentally altered, or hypotensive VAD patient is to ensure that all connections are secure and an adequate power source is connected. Controller malfunction, damage, or disconnection can also lead to pump dysfunction or stoppage. All patients should have a backup controller with them, as well as backup batteries for emergency replacement, in case of damage or malfunction. EMS providers must keep patients and their backup equipment together at all times because replacement equipment may be limited or nonexistent at receiving hospitals, particularly at non-

VAD centers. To reiterate, when a mechanical circulatory support patient is transported by EMS, all of the patient's VAD equipment must accompany him or her to the hospital to ensure continued mechanical support.

The driveline that connects the controller to the device is a potentially vulnerable component and is subject to wear, damage, or kinking, which can result in device malfunction. Although driveline wiring has built-in redundancy as a safety measure, driveline trauma can cause internal damage and lead to pump failure. Damage can be acute, such as a cutting or crush injury, or it can be the result of chronic stress or fatigue on the line. In these settings, there will often be alarms preceding or accompanying the pump stoppage, but alarms will cease once the batteries are drained.

Management of the Patient With an LVAD (Figure 53)

Refer to the following steps in the Adult Ventricular Assist Device Algorithm (Figure 54) to manage a patient with an LVAD.

- Assist ventilation if necessary and assess perfusion (Step 1).
    - –Normal skin color and temperature?
    - –Normal capillary refill?
- Adequate perfusion (Step 2)?
    - –If yes, assess and treat non-LVAD causes for altered mental status, such as hypoxia, blood glucose, overdose, and stroke (Step 3).
        - Follow local EMS and ACLS protocols and notify VAD center and/or medical control and transport (Steps 4 and 5)
    - –If no, assess LVAD function by looking and listening for alarms and listening for the LVAD hum (step 6).
- Is the LVAD functioning (Step 7)?
    - –If yes, is the mean arterial pressure more than 50 mm Hg and or $PETCO_2$ more than 20 mm Hg (Step 8)?
        - If yes, do not perform chest compressions (Step 9); follow local EMS and ACLS protocols (Step 4) and notify VAD center and/or medical control and transport (Step 5).
        - If no, perform external chest compressions (Step 10) and follow local EMS and ACLS protocols (Step 4) and notify VAD center and/or medical control and transport (Step 5).

- –If no, attempt to restart LVAD and consider if the driveline and power source are connected. Do you need to replace the system controller (Step 11)?
    - If LVAD is not restarted, perform external chest compressions (Step 10) and follow local EMS and ACLS protocols (Step 4) and notify VAD center and/or medical control and transport (Step 5).
    - If LVAD has been restarted, follow local EMS and ACLS protocols (Step 4) and notify VAD center and/or medical control and transport (Step 5).

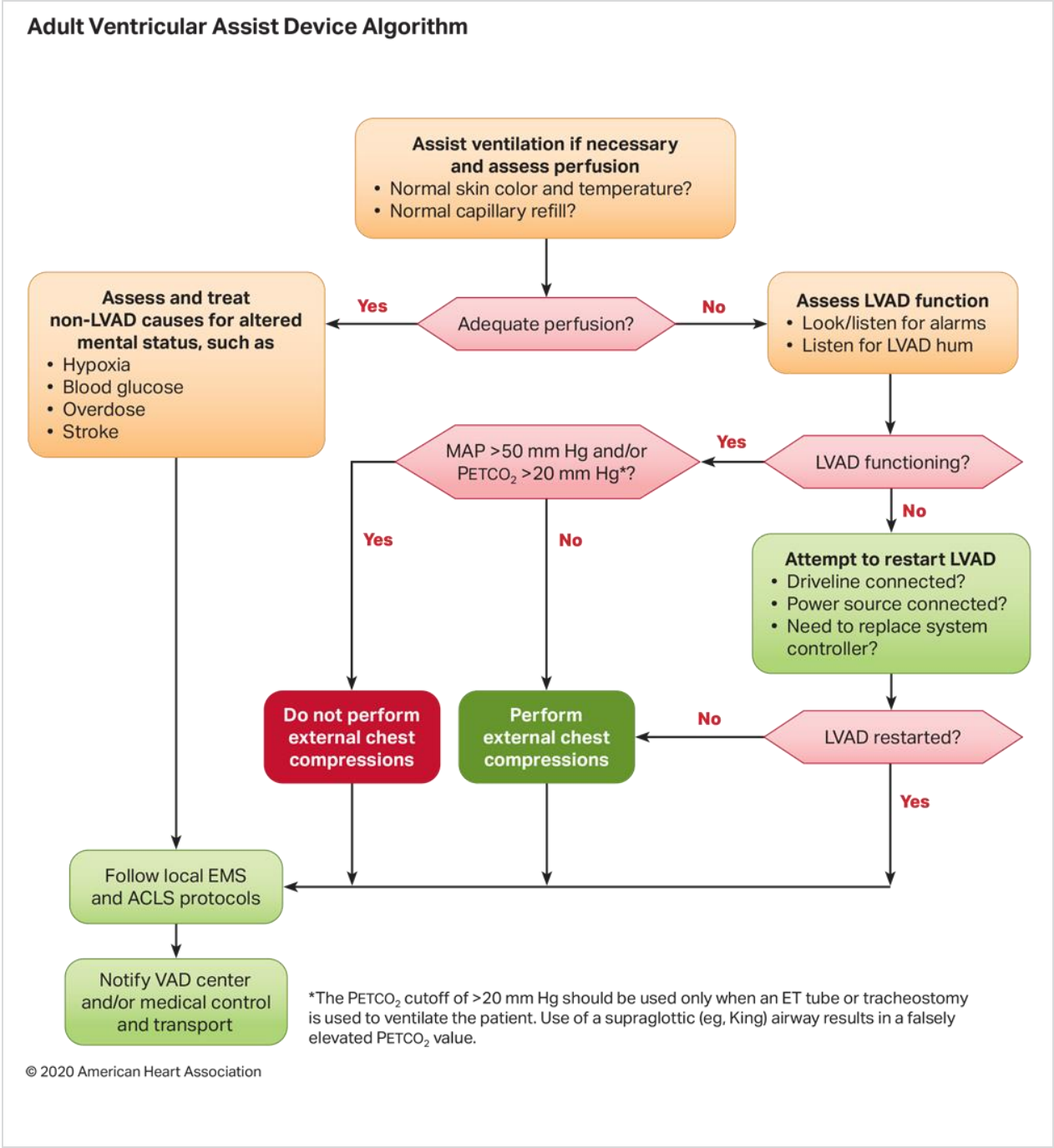


Figure 54. Adult Ventricular Assist Device Algorithm.

Abbreviation: MAP, mean arterial pressure.

Identifying the presence of mechanical circulatory support and code status is of initial importance. Some destination therapy patients with LVADs will have a legally executed, valid DNAR status and should be treated as any other patient with such a request. Obtain information from caregivers and medical alert identifications or wallet cards to ensure definitive patient

identification. It seems reasonable for VAD centers to standardize their approach to patient identification. Medical alert bracelets and necklaces can help to identify VAD patients and their code or intubation status, and such medical jewelry should be kept with the patient during transport to the hospital.

If it is unclear whether the patient is an LVAD patient, establish care with standard BLS and ACLS protocols. Breathing should be supported as needed with supplemental oxygen, airway adjuncts, and intubation as indicated.

Once a patient is identified as an LVAD patient, EMS providers must recognize that their patient may be in a state of pseudo-PEA and not have a palpable pulse or measurable blood pressure yet have adequate perfusion. If there is adequate mental status, a provider should assess the VAD for function by auscultating for a VAD hum over the left chest/left upper abdominal quadrant, looking and listening for VAD alarms, ensuring secure connections to the VAD controller, and ensuring sufficient power for the VAD. Prompt notification of the VAD center and its personnel (eg, VAD coordinator) is strongly recommended.

Clinical emergencies in LVAD patients—as well as LVAD alarms, such as low flow, power spikes, suction events, and pulsatility alarms—most often occur as a result of processes that are extrinsic to the LVAD itself. Events within the LVAD also occur, but less frequently. By providing comprehensive assessment of cardiac anatomy and function, along with evaluation of LVAD function, echocardiography can provide critical information for physicians caring for acutely ill patients who have LVADs.

Cardiac Arrest Associated With Pregnancy

Background

During attempted resuscitation of a pregnant woman, providers have 2 potential patients: the mother and the fetus. The best hope for fetal survival is maternal survival. For the critically ill pregnant patient, rescuers must provide appropriate resuscitation with consideration of the physiologic changes due to pregnancy.

The Second Patient

A cardiovascular emergency in a pregnant woman creates a special situation for the ACLS provider. You must always consider the fetus when

an adverse cardiovascular event occurs in a pregnant woman. At approximately 20 weeks or more of pregnancy (and possibly earlier), the size of the uterus begins to adversely affect the attempted resuscitation. At approximately 24 to 25 weeks of gestational age, the fetus may be able to survive outside the womb.

Decisions About Cesarean Delivery

The decision about whether to perform an emergency cesarean delivery must be made quickly when the mother is in cardiac arrest. Emergency cesarean delivery—also known as *hysterotomy*—may improve the outcome for both mother and child.

Key Interventions: Prevention of Cardiac Arrest in Pregnancy

To treat the critically ill pregnant patient:

- Place the patient in the left-lateral decubitus position to relieve possible compression of the inferior vena cava. Uterine obstruction of venous return can produce hypotension and could precipitate arrest in the critically ill patient.[45,46]
- Two methods of supporting the patient in the left-lateral decubitus position are (1) to use the angled backs of 2 or 3 chairs or (2) to use the angled thighs of several providers. Overturn a 4-legged chair so that the top of the chair back touches the floor. Align 1 or 2 more overturned chairs on either side of the first so that all are tilted in the same manner. Place the woman on her left side and align her torso parallel with the chair backs (Figure 55). Remember that this position will not be practical if chest compressions are needed.

Figure 55. Supporting the patient in the left-lateral decubitus position.

If cardiac arrest occurs, refer to the following steps in the Cardiac Arrest in

Pregnancy In-Hospital ACLS Algorithm (Figure 57):

- Continue BLS and ACLS (Step 1):
    - –High-quality CPR
    - –Defibrillation when indicated
    - –ACLS interventions (eg, epinephrine)
- Assemble the maternal cardiac arrest team (Step 2).
- Consider the etiology of the arrest (Step 3).
    - –Perform maternal interventions (Step 4):
        - Perform airway management.
        - Administer 100% oxygen; avoid excessive ventilation.
        - Place IV access above the diaphragm.
        - If the patient is receiving IV magnesium, stop it and give calcium chloride or gluconate.
- Continue BLS/ACLS (Step 5).

    - –High-quality CPR
    - –Defibrillation when indicated
    - –Other ACLS interventions (epinephrine)
- Perform obstetric interventions (Step 6)
    - –Provide continuous lateral uterine displacement.
    - –Detach fetal monitors.
    - –Prepare for perimortem cesarean delivery.
- Perform perimortem cesarean delivery (Step 7).
    - –If no ROSC in 5 minutes, consider immediate perimortem cesarean delivery.
- Neonatal team to receive neonate (Step 8).

Team planning should be done in collaboration with the obstetric, neonatal, emergency, anesthesiology, intensive care, and cardiac arrest services. Priorities for pregnant women in cardiac arrest should include provision of high-quality CPR and relief of aortocaval compression with lateral uterine displacement.

The goal of perimortem cesarean delivery is to improve maternal and fetal outcomes. Ideally, perform perimortem cesarean delivery in 5 minutes, depending on provider resources and skill sets.

Assess for hypotension; maternal hypotension that warrants therapy is defined as an SBP less than 100 mm Hg or less than 80% of baseline.[47,48] Maternal hypotension can cause a reduction in placental perfusion.[49-51] In the patient who is not in arrest, both crystalloid and colloid solutions increase preload.[52]

Consider potential etiology and reversible causes of cardiac arrest, and identify any preexisting medical conditions that may be complicating the resuscitation.

- Anesthetic complications
- Bleeding
- Cardiovascular
- Drugs
- Embolic
- Fever
- General nonobstetric causes of cardiac arrest (H’s and T’s)
- Hypertension

Advanced Airway

In pregnancy, a difficult airway is common. Use the most experienced provider. Provide endotracheal intubation or supraglottic advanced airway. Perform quantitative waveform capnography or capnometry to confirm and monitor ET tube placement. Once an advanced airway is in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions.

Techniques to Improve Maternal Hemodynamics

Shifting the Gravid Uterus

In cardiac arrest, the reduced venous return and cardiac output caused by the gravid uterus puts the mother at a hemodynamic disadvantage, thereby potentially reducing the effective coronary and cerebral perfusion produced by standard chest compressions. Therefore, when there is aortocaval compression, the effectiveness of the chest compressions may be limited.

Patient positioning has emerged as an important strategy to improve the quality of CPR and the resultant compression force and cardiac output.[53]

Patient Positioning During CPR

The gravid uterus can compress the inferior vena cava, impeding venous return, thereby reducing stroke volume and cardiac output. In general, aortocaval compression can occur for singleton pregnancies at approximately 20 weeks of gestational age,[54] at about the time when the fundus is at or above the umbilicus. Although chest compressions in the left lateral tilt position are feasible in a manikin study,[55] they result in decreased CPR quality (less forceful chest compressions) than is possible in the supine position.[56] Manual left lateral uterine displacement effectively relieves aortocaval pressure in patients with hypotension (Figure 56).[57]

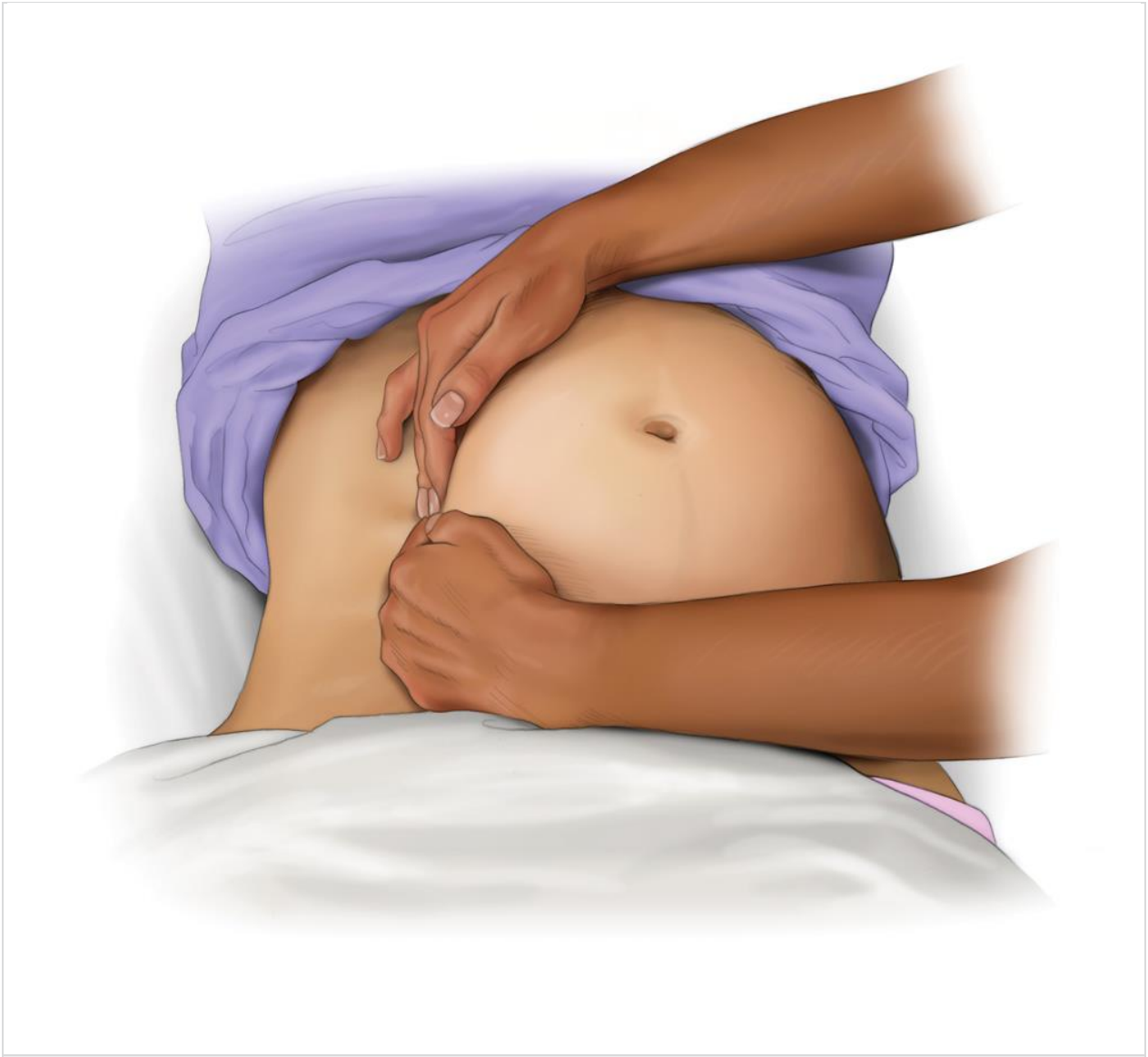


Figure 56A. Manual left uterine displacement, performed with the 2-handed technique.

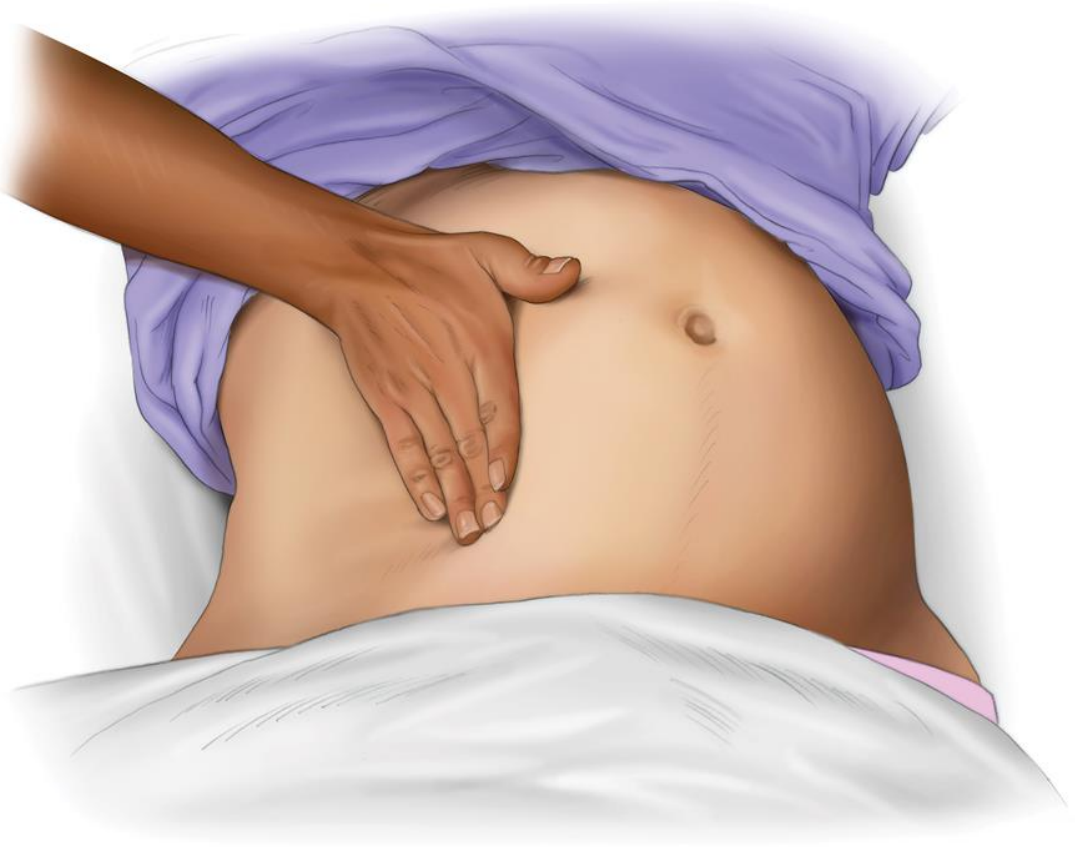


Figure 56B. One-handed technique during resuscitation.

Manual Left Uterine Displacement

Relieve compression of the inferior vena cava and the aorta by shifting the gravid uterus left and upward off the maternal vessels:

- Stand on the left side of the patient, level with the top of the uterus.
- Reach across the midline with both hands (Figure 56A) and pull the gravid uterus leftward and upward toward your abdomen.
- If it is not possible to stand to the left of the patient, use one hand to push the gravid uterus (Figure 56B) to the patient's left and upward.

Chest Compressions in the Left-Lateral Tilt

In cardiac arrest, the reduced venous return caused by the gravid uterus puts the mother at a hemodynamic disadvantage, reducing the cardiac output produced by chest compressions. Therefore, when there is aortocaval compression, the effectiveness of the chest compressions may be limited.

Chest compressions performed while the patient is tilted are not ideal. Although it is feasible to perform chest compressions in the tilted patient,[55] chest compressions performed in the tilted position are less forceful when compared with the supine position.[56] However, there are no physiologic data available for chest compressions in the tilted position. High-quality chest compressions are essential to maximize the chance of a successful resuscitation. An alternative method of relieving aortocaval compression, such as manual displacement, may be more practical and ideal during resuscitation because it allows for continuous and easier delivery of all other aspects of resuscitation, including high-quality chest compressions, defibrillation, IV access, and intubation.

ACLS for Pregnant Women

Because immediate ROSC cannot always be achieved, local resources for a perimortem caesarean delivery should be summoned as soon as cardiac arrest is recognized in a woman in the second half of pregnancy.[58] Systematic preparation and training are the keys to a successful response to such rare and complex events. Care teams that may be called upon to manage these situations should develop and practice standard institutional responses to allow for smooth delivery of resuscitative care.[53]

The treatments listed in the Cardiac Arrest in Pregnancy In-Hospital ACLS Algorithm include recommendations for defibrillation, medications, and intubation (Figure 57). The algorithm is divided into 2 focuses (maternal interventions and obstetric interventions) to reflect the simultaneous resuscitation interventions of both the maternal resuscitation team and the obstetrical/neonatal team to improve team performance, efficiency, and success.

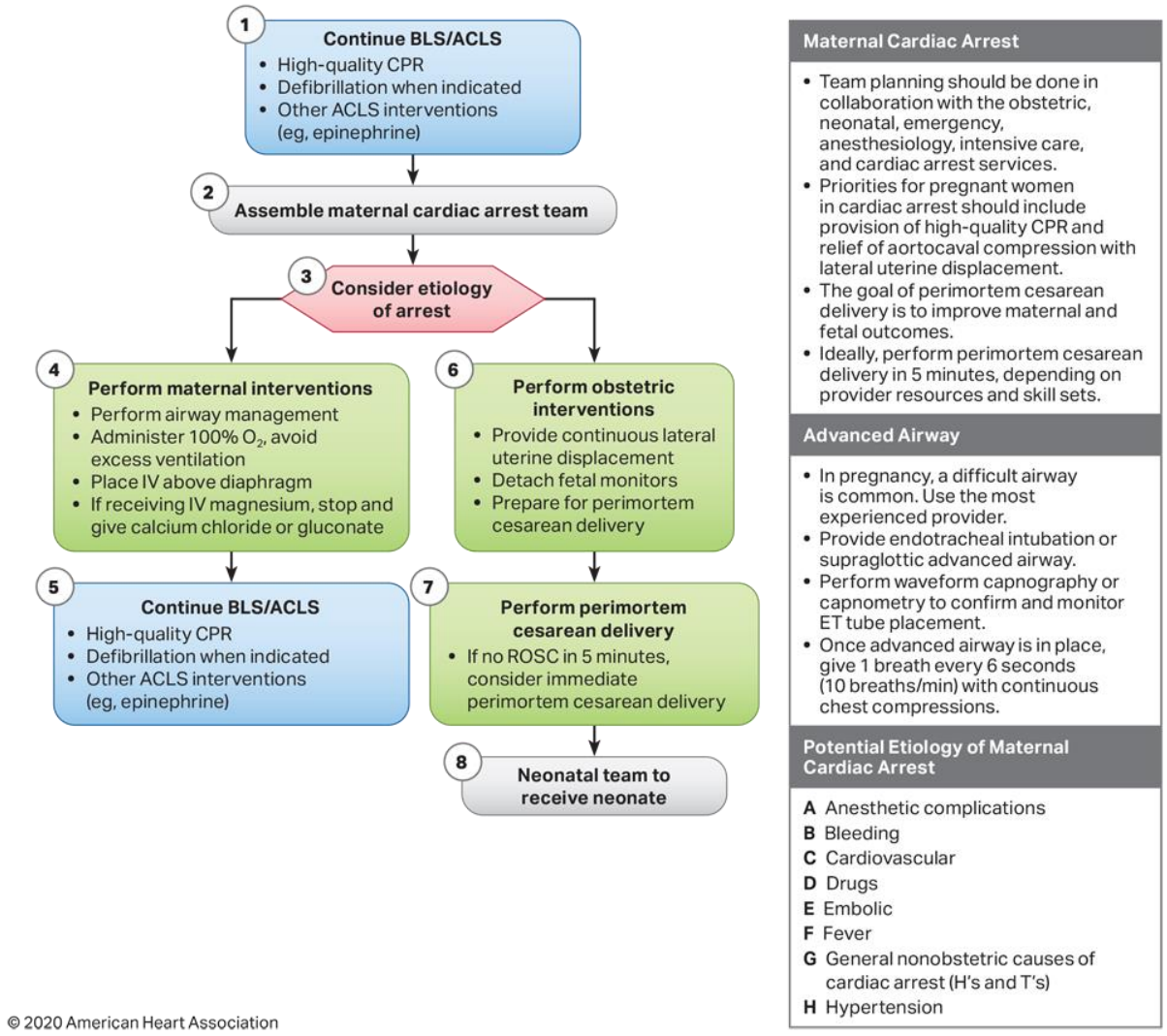


Figure 57. Cardiac Arrest in Pregnancy In-Hospital ACLS Algorithm.

Post–Cardiac Arrest Care

Overview

ACLS providers increasingly recognize that systematic post–cardiac arrest care after ROSC can improve the likelihood of patient survival with good quality of life. In fact, studies have found positive correlations between the likelihood of survival and the number of cardiac arrest cases treated at any individual hospital.[59,60] Studies also show that most deaths occur during the first 24 hours after resuscitation from cardiac arrest,[61,62] so post–cardiac arrest care has a significant potential to reduce early mortality caused by hemodynamic instability as well as later morbidity and mortality caused by multiorgan failure and brain injury.[63,64]

A growing body of research focuses on identifying and optimizing practices that improve the outcomes of patients who achieve ROSC after cardiac arrest.[65] Merely restoring blood pressure and gas exchange does not ensure survival and functional recovery, and significant cardiovascular dysfunction can develop after ROSC. These dysfunctions can require active support of blood flow and ventilation, including intravascular volume expansion, vasoactive and inotropic drugs, and invasive devices. In addition, TTM and treating the underlying cause of cardiac arrest can impact survival and neurologic outcome, and hemodynamic optimization protocols also serve as part of a bundle of care to improve survival.[66-68] Overall, the data suggest that proactively managing post–cardiac arrest physiology can improve patient outcomes by ensuring organ oxygenation and perfusion and by avoiding and managing complications.

This case focuses on managing and optimizing cardiopulmonary function and perfusing vital organs after ROSC.

To ensure the success of post–cardiac arrest care, you must consider what interventions are needed for the initial stabilization phase as well as the continued management with additional emergent activities. Refer to the steps in the Adult Post–Cardiac Arrest Care Algorithm (Figure 58), as described below.

Initial stabilization phase: Resuscitation is ongoing during the post-ROSC phase (Step 1), and many of these activities can occur concurrently depending on the resources available.

However, if prioritization is necessary, follow this order (Step 2):

- Manage the airway: Place an ET tube early, and use quantitative waveform capnography or capnometry to confirm and monitor endotracheal tube placement.
- Manage respiratory parameters: Start 10 breaths/min (1 breath every 6 seconds); SpO_2 92% to 98%; $PaCO_2$ of 35 to 45 mm Hg.
- Manage hemodynamic parameters: Administer crystalloid and/or vasopressor or inotrope for goal systolic blood pressure of greater than 90 mm Hg or mean arterial pressure greater than 65 mm Hg.
- Obtain a 12-lead ECG (Step 3).

Continued management and additional emergent activities: These evaluations should be done concurrently so that decisions on targeted temperature management (TTM) receive high priority as cardiac interventions. Other critical care management activities include continuously monitoring core temperature (esophageal, rectal, bladder); maintaining normoxia, normocapnia, euglycemia; providing continuous or intermittent electroencephalogram (EEG) monitoring; and providing lung-protective ventilation.

- Consider emergent cardiac intervention if (Step 4)
    - –STEMI is present
    - –The patient has unstable cardiogenic shock
    - –Mechanical circulatory support is required
- Does the patient follow commands (Step 5)?
    - –Comatose (Step 6):
        - TTM: If the patient is not following commands, start TTM as soon as possible. Begin at 32°C to 36°C for 24 hours, using a cooling device with feedback loop.
        - Obtain brain CT.
        - Perform EEG monitoring.
        - Provide other critical care management, such as continuously monitoring core temperature; maintaining normoxia, normocapnia, and euglycemia; providing continuous or intermittent EEG monitoring, and lung-protective ventilation.
    - –Awake (step 7): Consider other critical care management.
- Evaluate and treat rapidly reversible etiologies and involve expert consultation for continued management (Step 8).

Consider the H's and T's:

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypokalemia/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Rhythms for Post–Cardiac Arrest Care

You will need to recognize the following rhythms:

- Rate—too fast or too slow
- Width of QRS complexes—wide vs narrow

Drugs for Post–Cardiac Arrest Care

Drugs for post–cardiac arrest care include

- Epinephrine
- Dopamine
- Norepinephrine infusions

Multiple System Approach to Post–Cardiac Arrest Care

To treat post–cardiac arrest patients, implement a consistent, comprehensive, structured, and multidisciplinary system of care. Programs should include management of airway and respiratory and hemodynamic parameters, TTM, immediate coronary reperfusion when indicated for restoration of coronary blood flow with PCI, neurologic diagnosis, critical care management, and prognostication.

Treat the precipitating cause of cardiac arrest after ROSC, and initiate or request studies that will further help identify and treat any cardiac, electrolyte, toxicologic, pulmonary, and neurologic precipitants of arrest.

Ensure an adequate airway and support breathing immediately after ROSC because unconscious patients usually require an advanced airway for mechanical support of breathing. Also, elevate the head of the bed 30° if tolerated to reduce the incidence of cerebral edema, aspiration, and ventilatory-associated pneumonia. Monitor the placement of an advanced

airway, particularly during patient transport, by waveform capnography as described in the *2015 AHA Guidelines Update for CPR and ECC* and the *2020 AHA Guidelines for CPR and ECC*, and continuously monitor the patient's oxygenation with pulse oximetry.

Although 100% oxygen may have been used during initial resuscitation, adjust inspired oxygen to the lowest level required for an arterial oxygen saturation of 92% to 98% so that you avoid potential oxygen toxicity. Avoid hyperventilation, which is common during resuscitation attempts and can increase intrathoracic pressure, which decreases preload and lowers cardiac output. The decrease in $PaCO_2$ from hyperventilation can also decrease cerebral blood flow directly. Start ventilation at 10/min and adjust to achieve a $PaCO_2$ of 35 to 45 mm Hg.

Frequently reassess vital signs and monitor for recurrent cardiac arrhythmias by using continuous ECG monitoring. If the patient is hypotensive (SBP less than 90 mm Hg or mean arterial pressure of greater than 65 mm Hg), you can administer fluid boluses. If the patient's volume status is adequate, you may initiate infusions of vasoactive agents and adjust them to achieve a minimum SBP of 90 mm Hg or more or a mean arterial pressure of 65 mm Hg or more. Some experts advocate higher mean arterial pressures to promote cerebral blood flow.

Brain injury and cardiovascular instability are the major factors that determine survival after cardiac arrest.[69] Because TTM is currently the only intervention demonstrated to improve neurologic recovery, consider TTM for any patient who is comatose and unresponsive to verbal commands after ROSC. Obtain a CT brain scan, have EEG monitoring, and consider other critical care management. Transport the patient to a location that reliably provides this therapy in addition to coronary reperfusion (eg, PCI) and other goal-directed postarrest care therapies.

Treat the precipitating cause of cardiac arrest after ROSC and initiate or request studies that will further help evaluate the patient. You must identify and treat any cardiac, electrolyte, toxicologic, pulmonary, and neurologic precipitants of arrest. Overall, the most common cause of cardiac arrest is cardiovascular disease and associated coronary ischemia,[70,71] so obtain a 12-lead ECG as soon as possible to detect ST-segment elevation or LBBB. Perform coronary angiography right away (rather than later in the hospital stay or not at all) for OHCA patients with suspected cardiac etiology of arrest and ST-segment elevation on ECG. When you highly suspect AMI,

activate local protocols for treatment and coronary reperfusion. Coronary angiography, if indicated, can be beneficial in post–cardiac arrest patients regardless of whether they are awake or comatose. It is unclear whether emergent coronary angiography is beneficial for post–cardiac arrest patients without STEMI. In the absence of evidence identifying the optimal timing for coronary angiography and PCI in post–cardiac arrest patients suspected of having ACS as the cause of their cardiac arrest but without ST-segment elevation, an interventional cardiologist should be consulted for each patient to determine timing of angiography and PCI based on local protocols. Concurrent PCI and TTM are safe, with good outcomes reported for some comatose patients who have undergone PCI.

Critical care facilities that treat patients after cardiac arrest should use a comprehensive care plan that includes acute cardiovascular interventions, use of TTM, standardized medical goal-directed therapies, and advanced neurologic monitoring and care. Determining neurologic prognosis is inaccurate during the first 72 hours after resuscitation in patients not treated with TTM. For those treated with TTM, you should wait 72 hours after the patient returns to normothermia. Prognostication using clinical examination may be confounded by sedation or paralysis, so these factors must be considered carefully before considering a withdrawal of life-sustaining therapy on the basis of neuroprognostication. Many initially comatose survivors of cardiac arrest have the potential for full recovery,[66,72,73] so it is important to place patients in a hospital critical care unit where experts can perform neurologic evaluation and appropriate testing to aid prognosis in a timely manner.

Managing Post–Cardiac Arrest Care: The Adult Post–Cardiac Arrest Care Algorithm

The Adult Post–Cardiac Arrest Care Algorithm (Figure 58) outlines the steps to immediately assess and manage post–cardiac arrest patients with ROSC. In this case, team members will continue to maintain good ventilation and oxygenation with a bag-mask device or advanced airway. You'll also use the H's and T's to recall conditions that could have contributed to the cardiac arrest. Throughout the case discussion of the Adult Post–Cardiac Arrest Care Algorithm, we will refer to Steps 1 through 8, the numbers assigned to the steps in the algorithm.

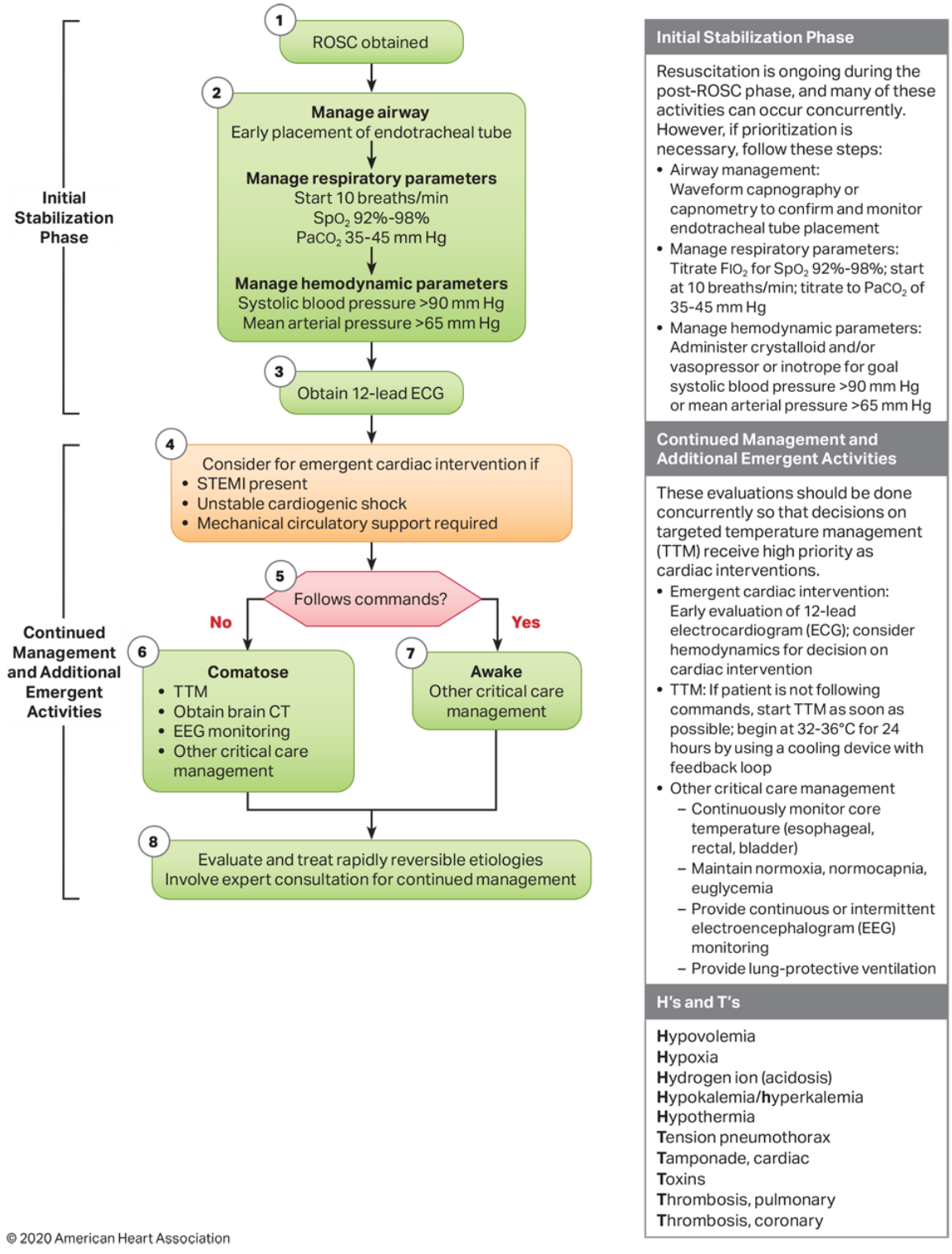


Figure 58. Adult Post–Cardiac Arrest Care Algorithm.

Application of the Adult Post–Cardiac Arrest Care Algorithm

ACLS providers will assess and treat a patient who had cardiac arrest and was resuscitated with the use of the BLS, Primary, and Secondary Assessments. During rhythm check in the Primary Assessment, the patient's rhythm was organized and a pulse was detected (Figure 58). The Team Leader will coordinate the efforts of the high-performance post–cardiac arrest care team as they perform the steps of the Adult Post–Cardiac Arrest Care Algorithm.

Optimize Ventilation and Oxygenation

Step 2 directs you to ensure an adequate airway and support breathing immediately after ROSC. An unconscious/unresponsive patient requires an advanced airway to mechanically support breathing.

- Use continuous quantitative waveform capnography to confirm and monitor correct placement of the ET tube (Figures 59 and 60).
- Use the lowest inspired oxygen concentration that will maintain arterial oxyhemoglobin saturation 92% to 98%. When titrating inspired oxygen is not feasible (eg, in an out-of-hospital setting), it is reasonable to use 100% oxygen until the patient arrives at the ED.
- Avoid excessive ventilation of the patient (do not ventilate too fast or too much). You may begin ventilation at 10/min and adjust to achieve a $PaCO_2$ of 35 to 45 mm Hg.

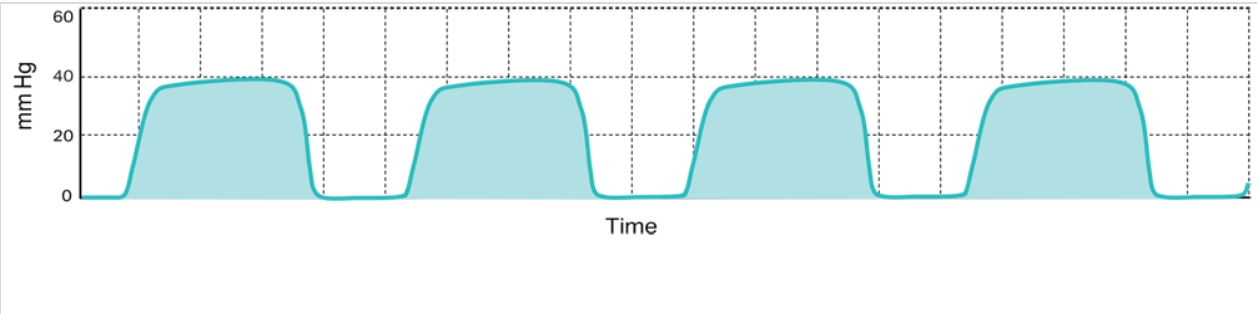


Figure 59A. Waveform capnography. A, Normal range of 35 to 45 mm Hg.

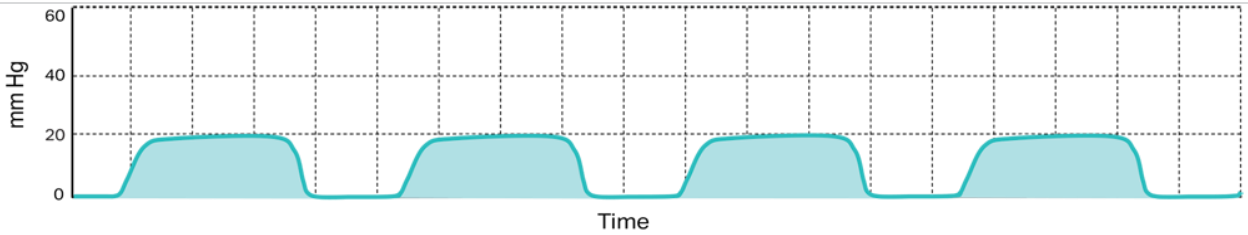


Figure 59B. 20 mm Hg.

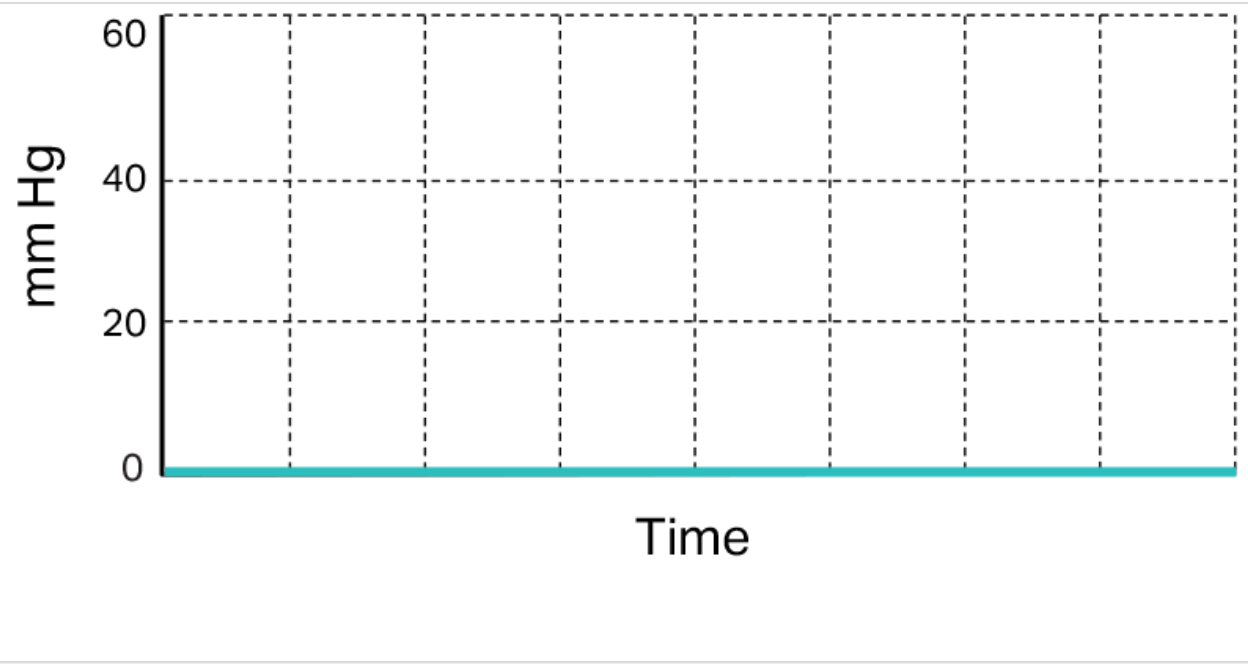


Figure 59C. 0 mm Hg.

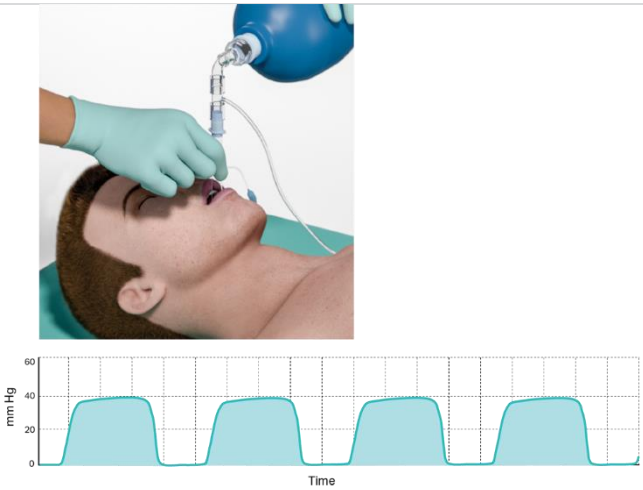


Figure 60. Waveform capnography with an ET showing normal (adequate) ventilation pattern: $PETCO_2$ 30 to 40 mm Hg.

To avoid hypoxia in adults with ROSC after cardiac arrest, you may use the highest available oxygen concentration until you can measure the arterial oxyhemoglobin saturation or the partial pressure of arterial oxygen, if the appropriate equipment is available. Decrease the fraction of inspired oxygen (FiO_2) when oxyhemoglobin saturation is 100% if you can maintain the oxyhemoglobin saturation at 92% to 98%.

Because an oxygen saturation of 99% or greater may correspond to a PaO_2 between approximately 145 and 500 mm Hg, in general, it is appropriate to wean FiO_2 for a saturation of 98% or greater to avoid hyperoxia as long as the patient can maintain oxyhemoglobin saturation of 92% to 98%.

Critical Concepts: Quantitative Waveform Capnography

In addition to monitoring ET tube position, quantitative waveform capnography allows healthcare personnel to monitor CPR quality, optimize chest compressions, and detect ROSC during chest compressions or when a rhythm check reveals an organized rhythm.

Caution: Things to Avoid During Ventilation

When securing an advanced airway, avoid using ties that encircle the patient's neck and can obstruct venous return from the brain.
Avoid excessive ventilation, which may lead to both adverse hemodynamic effects when intrathoracic pressures are increased and decreased cerebral blood flow when $PaCO_2$ decreases.

Quantitative Waveform Capnography

$ETCO_2$ is the concentration of carbon dioxide in exhaled air at the end of expiration, typically expressed as a partial pressure in millimeters of mercury ($PETCO_2$). There are 2 types of capnography devices: mainstream and sidestream. Mainstream measures the CO_2 directly on the airway and sends the signal back to the device to display. Sidestream samples the gas from the airway and measures the CO_2 within the device. Because CO_2 is a trace gas in atmospheric air, CO_2 that capnography detects in exhaled air is produced in the body and delivered to the lungs by circulating blood.

Cardiac output is the major determinant of CO_2 delivery to the lungs. If ventilation is relatively constant, $PETCO_2$ correlates well with cardiac output during CPR.

Observe a persistent capnographic waveform with ventilation to confirm and monitor ET tube placement in the field, in the transport vehicle, on arrival at the hospital, and after any patient transfer to reduce the risk of unrecognized tube misplacement or displacement.

Although researchers have not studied capnography to confirm and monitor correct placement of supraglottic airways (eg, laryngeal mask airway, laryngeal tube, or esophageal-tracheal tube), effective ventilation through a supraglottic airway device should result in a capnography waveform during CPR and after ROSC.

Treat Hypotension (SBP Less Than 90 mm Hg)

Step 2 directs you to treat hypotension when SBP is less than 90 mm Hg. Obtain IV access if not already established, and verify that any IV lines are open. Continue ECG monitoring after ROSC, during transport, and throughout ICU care until deemed clinically not necessary. At this stage, consider treating any reversible causes that might have precipitated the cardiac arrest but persist after ROSC.

Treat hypotension as follows:

- **IV bolus:** 1 to 2 L normal saline or lactated Ringer's solution
- **Norepinephrine:** 0.1 to 0.5 mcg/kg per minute (in 70-kg adult: 7 to 35 mcg per minute) IV infusion adjusted to achieve a minimum SBP of greater than 90 mm Hg or a mean arterial pressure of greater than 65 mm Hg
    - –Norepinephrine (levarterenol), a naturally occurring potent vasoconstrictor and inotropic agent, may be effective for managing patients with severe hypotension (eg, SBP less than 70 mm Hg) and a low total peripheral resistance who do not respond to less potent adrenergic drugs such as dopamine, phenylephrine, or methoxamine.
- **Epinephrine:** 2 to 10 mcg per minute IV infusion adjusted to achieve a minimum SBP of greater than 90 mm Hg or a mean arterial pressure of greater than 65 mm Hg
    - –Epinephrine can be used in patients who are not in cardiac arrest but who require inotropic or vasopressor support.

- **Dopamine:** 5 to 20 mcg/kg per minute IV infusion adjusted to achieve a minimum SBP of greater than 90 mm Hg or a mean arterial pressure of greater than 65 mm Hg
    - –Dopamine hydrochloride is a catecholamine-like agent and a chemical precursor of norepinephrine that stimulates the heart through both α- and β-adrenergic receptors.

STEMI Is Present or High Suspicion of AMI

Both in-hospital and out-of-hospital medical personnel should obtain a 12-lead ECG as soon as possible after ROSC to identify those patients with STEMI or a high suspicion of AMI.

EMS personnel should transport these patients to a facility that reliably provides this therapy (Step 4).

Coronary Reperfusion

Begin aggressive treatment, including coronary reperfusion with PCI, if you detect STEMI after ROSC, regardless of coma or TTM. In cases of out-of-hospital STEMI, provide advance notification to receiving facilities.

Following Commands

Step 5 directs you to examine the patient's ability to follow verbal commands. If the patient does not follow commands, the high-performance team should consider implementing TTM, obtaining a brain CT, performing EEG monitoring, and providing other critical-care management (Step 6). If the patient can follow verbal commands, move to Step 7.

Targeted Temperature Management

TTM is the only intervention demonstrated to improve neurologic recovery after cardiac arrest. The optimal duration of TTM is at least 24 hours, and although comparative studies of the duration of TTM have not been performed in adults, hypothermia for up to 72 hours was used safely in newborns.

During TTM, monitor the patient's core temperature by using an esophageal thermometer, bladder catheter in nonanuric patients, or a pulmonary artery catheter if one is already in place for other indications. Axillary, oral, and rectal temperatures do not adequately measure core temperature changes.

TTM should not affect the decision to perform PCI, because concurrent PCI and hypothermia are reported to be feasible and safe.

To protect the brain and other organs, the high-performance team should start TTM in patients who remain comatose with ROSC after cardiac arrest.

For TTM, healthcare providers should select and maintain a constant target temperature between 32°C and 36°C for at least 24 hours. Although the optimal method of achieving the target temperature is unknown, any combination of rapid infusion of ice-cold, isotonic, non–glucose-containing fluid (30 mL/kg), endovascular catheters, surface cooling devices, or simple surface interventions (eg, ice bags) appears to be safe and effective.

Specific patient features may necessitate selecting one temperature over another for TTM. Higher temperatures might be preferable in patients for whom lower temperatures convey some risk (eg, bleeding), and lower temperatures might be preferable when patients have clinical features that worsen at higher temperatures (eg, seizures, cerebral edema). Of note, temperature control between 32°C and 36°C is not contraindicated in any patients, so all patients who require intensive care are eligible.

In the prehospital setting, do not routinely cool patients after ROSC with rapid infusion of cold IV fluids. Current evidence indicates no direct outcome benefit from these interventions, and IV fluid administration in the prehospital setting may increase pulmonary edema and rearrest. We don’t yet know whether different methods or devices for temperature control outside of the hospital are beneficial.

Advanced Critical Care

After coronary reperfusion interventions, or if the post–cardiac arrest patient has no ECG evidence or suspicion of MI, the high-performance team should transfer the patient to an ICU.

Post–Cardiac Arrest Maintenance Therapy

No evidence supports continued prophylactic administration of antiarrhythmic medications once the patient achieves ROSC.

Other Postresuscitation Care

- Glucose management: The benefit of any specific target range of glucose management is uncertain in adults with ROSC after cardiac arrest. It is reasonable to manage blood glucose levels in

post-arrest patients using the same approach for the general critically ill population (eg, insulin therapy when needed to maintain a blood glucose of 150 to 180 mg/dL).

- Prophylactic antibiotics: The routine use of prophylactic antibiotics in postarrest patients is of uncertain benefit.
- Neuroprotective agents: The effectiveness of agents to mitigate neurologic injury in patients who remain comatose after ROSC is uncertain. There is no difference in any clinical outcomes with use of neuroprotective agents studied.
- Routine use of steroids: The routine use of steroids for patients with shock after ROSC is of uncertain value. There is no definitive evidence of benefit from steroids after ROSC.

Neuroprognostication

Hypoxic-ischemic brain injury is the leading cause of morbidity and mortality in survivors of OHCA, and it accounts for a smaller but significant portion of poor outcomes after resuscitation from IHCA.[69,74] Most deaths attributable to post-arrest brain injury are due to active withdrawal of life-sustaining treatment on the basis of a predicted poor neurologic outcome. Accurate neurologic prognostication is important to avoid inappropriate withdrawal of life-sustaining treatment in patients who may otherwise achieve meaningful neurologic recovery and also to avoid ineffective treatment when poor outcome is inevitable.[75]

Neuroprognostication relies on interpreting the results of diagnostic tests and correlating those results with outcome (Figure 61). Because a false positive test for poor neurologic outcome could lead to inappropriate withdrawal of life support from a patient who otherwise might have recovered, the most important test characteristic is specificity. Many of the tests considered are subject to error due to the effects of medications, organ dysfunction, and temperature. Furthermore, many research studies have methodological limitations, including small sample sizes, single-center design, lack of blinding, the potential for self-fulfilling prophecies, and the use of outcome at hospital discharge rather than a timepoint associated with maximal recovery (typically 3–6 months after an arrest).[75]

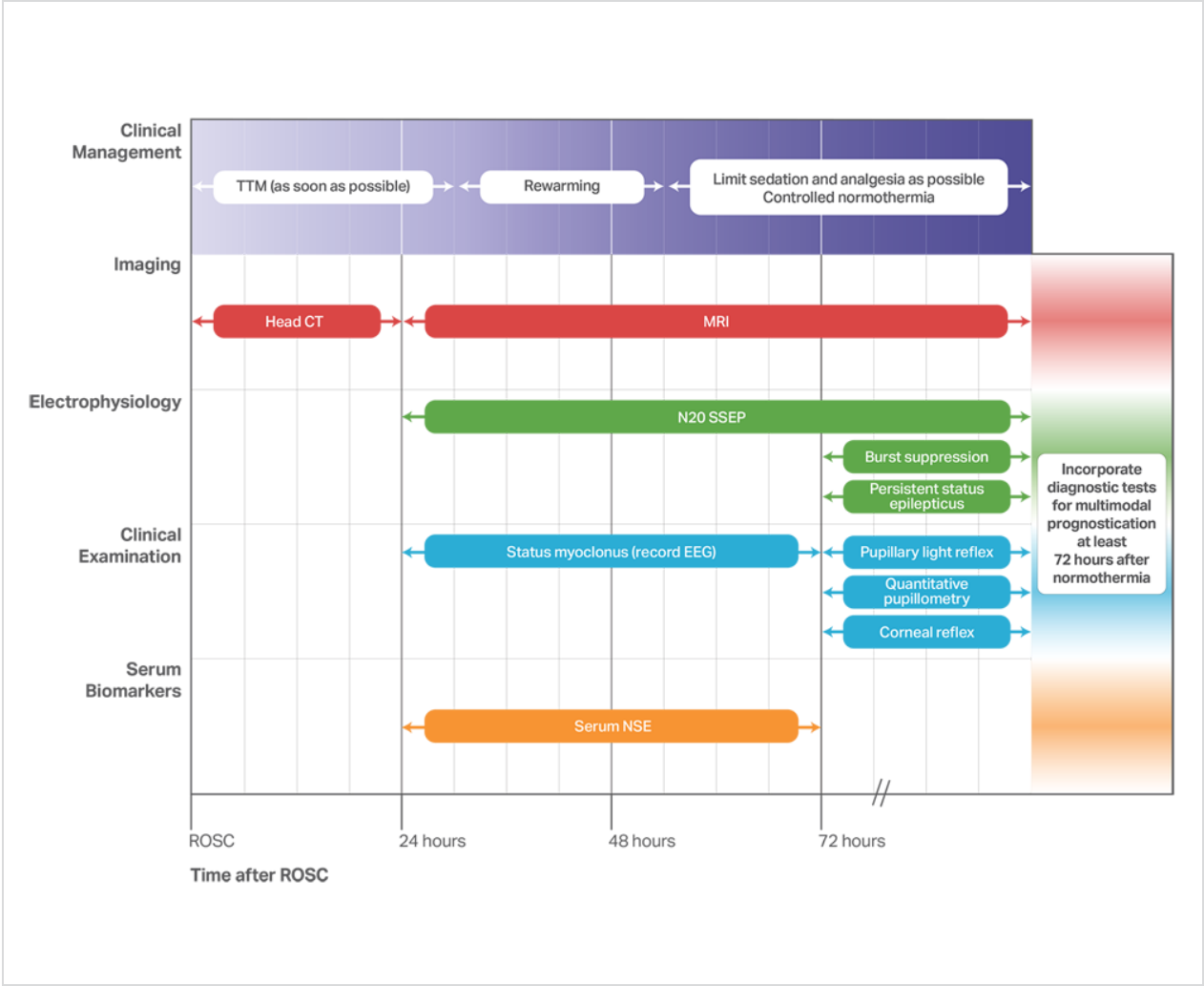


Figure 61. Schematic overview of a multimodal approach to neuroprognostication.

Because any single method of neuroprognostication has an intrinsic error rate and may be subject to confounding, multiple modalities should be used to improve decision-making accuracy.

General Considerations for Neuroprognostication

- In patients who remain comatose after cardiac arrest, neuroprognostication should involve a multimodal approach and not be based on any single finding.
- In patients who remain comatose after cardiac arrest, neuroprognostication should be delayed until adequate time has passed to ensure avoidance of confounding by medication effect or a transiently poor exam in the early postinjury period.

- Teams caring for comatose cardiac arrest survivors should have regular and transparent multidisciplinary discussions with surrogates about the anticipated time course for and uncertainties around neuroprognostication.
- For patients who remain comatose after cardiac arrest, it is reasonable to perform multimodal neuroprognostication at a minimum of 72 hours after normothermia, though individual prognostic tests may be obtained earlier than this.

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Appendix

Testing Checklists and Learning Station Checklists

Advanced Cardiovascular Life Support

Adult High-Quality BLS Skills Testing Checklist

American Heart Association.

Student Name _____ Date of Test _____

Hospital Scenario: "You are working in a hospital or clinic, and you see a person who has suddenly collapsed in the hallway. You check that the scene is safe and then approach the patient. Demonstrate what you would do next."

Prehospital Scenario: "You arrive on the scene for a suspected cardiac arrest. No bystander CPR has been provided. You approach the scene and ensure that it is safe. Demonstrate what you would do next."

Assessment and Activation

- ☐ Checks responsiveness
- ☐ Shouts for help/Activates emergency response system/Sends for AED
- ☐ Checks breathing
- ☐ Checks pulse

Once student shouts for help, instructor says, "I am going to get the AED."

Compressions ***Audio/visual feedback device required for accuracy***

- ☐ Hand placement on lower half of sternum
- ☐ Perform continuous compressions for 2 minutes (100-120/min)
- ☐ Compresses at least 2 inches (5 cm)
- ☐ Complete chest recoil. (Optional, check if using a feedback device that measures chest recoil)

Rescuer 2 says, "Here is the AED. I'll take over compressions, and you use the AED."

AED (follows prompts of AED)

- ☐ Powers on AED
- ☐ Correctly attaches pads
- ☐ Clears for analysis
- ☐ Clears to safely deliver a shock
- ☐ Safely delivers a shock
- ☐ Shocks within 45 seconds of AED arrival

Resumes Compressions

- ☐ Ensures compressions are resumed immediately after shock delivery
  - Student directs instructor to resume compressions *or*
  - Second student resumes compressions

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

Airway Management Skills Testing Checklist

American Heart Association.

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
BLS Assessment and Interventions	
Checks for responsiveness • Taps and shouts, "Are you OK?"	
Activates the emergency response system • Shouts for nearby help/Activates the emergency response system and gets the AED *or* • Directs second rescuer to activate the emergency response system and get the AED	
Checks breathing • Scans chest for movement (5-10 seconds)	
Checks pulse (5-10 seconds) **Breathing and pulse check can be done simultaneously** Notes that pulse is present and does not initiate chest compressions or attach AED	
Inserts oropharyngeal or nasopharyngeal airway	
Administers oxygen	
Performs effective bag-mask ventilation for 1 minute • Gives proper ventilation rate (once every 6 seconds) • Gives proper ventilation speed (over 1 second) • Gives proper ventilation volume (~half a bag)	

STOP TEST

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**
Instructor Initials _____ Instructor Number _____ Date _____		

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to Instructor Manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**
Instructor Initials _____ Instructor Number _____ Date _____		

Megacode Testing Checklist: Scenarios 1/3/8
Bradycardia → Pulseless VT → PEA → PCAC

American Heart Association.

Student Name _____ Date of Test _____

Critical Performance Steps						Check if done correctly
Team Leader						
Assigns team member roles						
Ensures high-quality CPR at all times	Compression rate 100-120/min ☐	Compression depth of ≥2 inches ☐	Chest compression fraction >80% ☐	Chest recoil (optional) ☐	Ventilation (optional) ☐	
Ensures that team members communicate well						
Bradycardia Management						
Starts oxygen if needed, places monitor, starts IV						
Places monitor leads in proper position						
Recognizes symptomatic bradycardia						
Administers correct dose of atropine						
Prepares for second-line treatment						
Pulseless VT Management						
Recognizes pVT						
Clears before analyze and shock						
Immediately resumes CPR after shocks						
Appropriate airway management						
Appropriate cycles of drug–rhythm check/shock–CPR						
Administers appropriate drug(s) and doses						
PEA Management						
Recognizes PEA						
Verbalizes potential reversible causes of PEA (H's and T's)						
Administers appropriate drug(s) and doses						
Immediately resumes CPR after rhythm checks						
Post–Cardiac Arrest Care						
Identifies ROSC						
Ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, verbalizes need for endotracheal intubation and waveform capnography, and orders laboratory tests						
Considers targeted temperature management						

STOP TEST

Test Results	Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**
Instructor Initials _____	Instructor Number _____	Date _____	

Learning Station Competency
☐ Bradycardia ☐ Tachycardia ☐ Cardiac Arrest/Post–Cardiac Arrest Care ☐ Megacode Practice

Megacode Testing Checklist: Scenario 12
Bradycardia → VF → Asystole/PEA → PCAC

American Heart Association.

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Ensures high-quality CPR at all times: Compression rate 100-120/min ☐; Compression depth of ≥2 inches ☐; Chest compression fraction >80% ☐; Chest recoil (optional) ☐; Ventilation (optional) ☐	
Ensures that team members communicate well	
Bradycardia Management	
Starts oxygen if needed, places monitor, starts IV	
Places monitor leads in proper position	
Recognizes symptomatic bradycardia	
Administers correct dose of atropine	
Prepares for second-line treatment	
VF Management	
Recognizes VF	
Clears before analyze and shock	
Immediately resumes CPR after shocks	
Appropriate airway management	
Appropriate cycles of drug–rhythm check/shock–CPR	
Administers appropriate drug(s) and doses	
Asystole and PEA Management	
Recognizes asystole and PEA	
Verbalizes potential reversible causes of asystole and PEA (H's and T's)	
Administers appropriate drug(s) and doses	
Immediately resumes CPR after rhythm checks	
Post–Cardiac Arrest Care	
Identifies ROSC	
Ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, verbalizes need for endotracheal intubation and waveform capnography, and orders laboratory tests	
Considers targeted temperature management	

STOP TEST

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

Learning Station Competency
☐ Bradycardia ☐ Tachycardia ☐ Cardiac Arrest/Post–Cardiac Arrest Care ☐ Megacode Practice

Megacode Testing Checklist: Scenario 9
Tachycardia → PEA → VF → PCAC

American Heart Association.

Student Name _____ Date of Test _____

Critical Performance Steps						Check if done correctly
Team Leader						
Assigns team member roles						
Ensures high-quality CPR at all times	Compression rate 100-120/min ☐	Compression depth of ≥2 inches ☐	Chest compression fraction >80% ☐	Chest recoil (optional) ☐	Ventilation (optional) ☐	
Ensures that team members communicate well						
Tachycardia Management						
Starts oxygen if needed, places monitor, starts IV						
Places monitor leads in proper position						
Recognizes tachycardia (specific diagnosis)						
Recognizes no symptoms due to tachycardia						
Considers appropriate initial drug therapy						
PEA Management						
Recognizes PEA						
Verbalizes potential reversible causes of PEA (H's and T's)						
Administers appropriate drug(s) and doses						
Immediately resumes CPR after rhythm check and pulse checks						
VF Management						
Recognizes VF						
Clears before analyze and shock						
Immediately resumes CPR after shocks						
Appropriate airway management						
Appropriate cycles of drug–rhythm check/shock–CPR						
Administers appropriate drug(s) and doses						
Post–Cardiac Arrest Care						
Identifies ROSC						
Ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, verbalizes need for endotracheal intubation and waveform capnography, and orders laboratory tests						
Considers targeted temperature management						

STOP TEST

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**
Instructor Initials _____ Instructor Number _____ Date _____		

Learning Station Competency
☐ Bradycardia ☐ Tachycardia ☐ Cardiac Arrest/Post–Cardiac Arrest Care ☐ Megacode Practice

Megacode Testing Checklist: Scenarios 6/11
Bradycardia → VF → PEA → PCAC

American Heart Association.

Student Name _____ Date of Test _____

Critical Performance Steps						Check if done correctly
Team Leader						
Assigns team member roles						
Ensures high-quality CPR at all times	Compression rate 100-120/min ☐	Compression depth of ≥2 inches ☐	Chest compression fraction >80% ☐	Chest recoil (optional) ☐	Ventilation (optional) ☐	
Ensures that team members communicate well						
Bradycardia Management						
Starts oxygen if needed, places monitor, starts IV						
Places monitor leads in proper position						
Recognizes symptomatic bradycardia						
Administers correct dose of atropine						
Prepares for second-line treatment						
VF Management						
Recognizes VF						
Clears before analyze and shock						
Immediately resumes CPR after shocks						
Appropriate airway management						
Appropriate cycles of drug–rhythm check/shock–CPR						
Administers appropriate drug(s) and doses						
PEA Management						
Recognizes PEA						
Verbalizes potential reversible causes of PEA (H's and T's)						
Administers appropriate drug(s) and doses						
Immediately resumes CPR after rhythm checks						
Post–Cardiac Arrest Care						
Identifies ROSC						
Ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, verbalizes need for endotracheal intubation and waveform capnography, and orders laboratory tests						
Considers targeted temperature management						

STOP TEST

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**
Instructor Initials _____ Instructor Number _____ Date _____		

Learning Station Competency
☐ Bradycardia ☐ Tachycardia ☐ Cardiac Arrest/Post–Cardiac Arrest Care ☐ Megacode Practice

Megacode Testing Checklist: Scenarios 4/7/10
Tachycardia → VF → PEA → PCAC

American Heart Association.

Student Name _____ Date of Test _____

Critical Performance Steps						Check if done correctly
Team Leader						
Assigns team member roles						
Ensures high-quality CPR at all times	Compression rate 100-120/min ☐	Compression depth of ≥2 inches ☐	Chest compression fraction >80% ☐	Chest recoil (optional) ☐	Ventilation (optional) ☐	
Ensures that team members communicate well						
Tachycardia Management						
Starts oxygen if needed, places monitor, starts IV						
Places monitor leads in proper position						
Recognizes unstable tachycardia						
Recognizes symptoms due to tachycardia						
Performs immediate synchronized cardioversion						
VF Management						
Recognizes VF						
Clears before analyze and shock						
Immediately resumes CPR after shocks						
Appropriate airway management						
Appropriate cycles of drug–rhythm check/shock–CPR						
Administers appropriate drug(s) and doses						
PEA Management						
Recognizes PEA						
Verbalizes potential reversible causes of PEA (H's and T's)						
Administers appropriate drug(s) and doses						
Immediately resumes CPR after rhythm checks						
Post–Cardiac Arrest Care						
Identifies ROSC						
Ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, verbalizes need for endotracheal intubation and waveform capnography, and orders laboratory tests						
Considers targeted temperature management						

STOP TEST

Test Results	Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**
Instructor Initials _____	Instructor Number _____	Date _____	

Learning Station Competency
☐ Bradycardia ☐ Tachycardia ☐ Cardiac Arrest/Post–Cardiac Arrest Care ☐ Megacode Practice

Megacode Testing Checklist: Scenarios 2/5
Bradycardia → VF → Asystole → PCAC

American Heart Association.

Student Name _____ Date of Test _____

Critical Performance Steps						Check if done correctly
Team Leader						
Assigns team member roles						
Ensures high-quality CPR at all times	Compression rate 100-120/min ☐	Compression depth of ≥2 inches ☐	Chest compression fraction >80% ☐	Chest recoil (optional) ☐	Ventilation (optional) ☐	
Ensures that team members communicate well						
Bradycardia Management						
Starts oxygen if needed, places monitor, starts IV						
Places monitor leads in proper position						
Recognizes symptomatic bradycardia						
Administers correct dose of atropine						
Prepares for second-line treatment						
VF Management						
Recognizes VF						
Clears before analyze and shock						
Immediately resumes CPR after shocks						
Appropriate airway management						
Appropriate cycles of drug–rhythm check/shock–CPR						
Administers appropriate drug(s) and doses						
Asystole Management						
Recognizes asystole						
Verbalizes potential reversible causes of asystole (H's and T's)						
Administers appropriate drug(s) and doses						
Immediately resumes CPR after rhythm checks						
Post–Cardiac Arrest Care						
Identifies ROSC						
Ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, verbalizes need for endotracheal intubation and waveform capnography, and orders laboratory tests						
Considers targeted temperature management						

STOP TEST

Test Results	Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**
Instructor Initials _____	Instructor Number _____	Date _____	

Learning Station Competency
☐ Bradycardia ☐ Tachycardia ☐ Cardiac Arrest/Post–Cardiac Arrest Care ☐ Megacode Practice

ACLS Pharmacology Summary Table

This table provides information about common drugs used in ACLS.

Drug	Indications	Precautions and contraindications	Adult dosage
Adenosine	• First drug for most forms of stable narrow-complex SVT; effective in terminating those due to reentry involving AV node or sinus node • May consider for unstable narrow-complex reentry tachycardia while preparations are made for cardioversion • Regular and monomorphi	• Contraindicated in poison/ drug-induced tachycardia or second- or third-degree heart block • Transient side effects include flushing, chest pain or tightness, brief periods of asystole or bradycardia, ventricular ectopy • Less effective (larger doses may be	**IV Rapid Push** • Place patient in mild reverse Trendelenburg position before administration of drug • Initial bolus of 6 mg given rapidly over 1 to 3 seconds followed by NS bolus of 20 mL; then elevate the extremity • A second dose (12 mg) can be given in 1 to 2 minutes if needed **Injection Technique** • Record rhythm strip during administration • Draw up adenosine dose in one syringe and flush in another; attach both syringes to the same or immediately adjacent IV injection ports nearest patient, with adenosine closest to patient; clamp IV tubing above injection port • Push IV adenosine as quickly as possible (1 to 3 seconds) • While maintaining pressure on adenosine plunger, push NS flush as rapidly as possible after adenosine • Unclamp IV tubing

	c wide-complex tachycardia, thought to be or previously defined to be reentry SVT • Does not convert atrial fibrillation, atrial flutter, or VT • Diagnostic maneuver: stable narrow-complex SVT	required) in patients taking theophylline or caffeine • Reduce initial dose to 3 mg in patients receiving dipyridamole or carbamazepine, in heart transplant patients, or if given by central venous access • If administered for irregular, polymorphic wide-complex tachycardia/VT, may cause deterioration (including hypotension) • Transient periods of sinus	

		bradycardia and ventricular ectopy are common after termination of SVT • Safe and effective in pregnancy	
Amiodarone	Because its use is associated with toxicity, amiodarone is indicated for use in patients with life-threatening arrhythmias when administered with appropriate monitoring: • VF/pVT unresponsive to shock delivery, CPR, and a vasopressor • Recurrent, hemodynamically unstable VT With expert consultation, amiodarone may be used for treatment of some atrial and ventricular arrhythmias.	• Rapid infusion may lead to hypotension • With multiple dosing, cumulative doses >2.2 g over 24 hours are associated with significant hypotension in clinical trials • Do not administer with other drugs that prolong QT interval (eg, procainamide)	**VF/pVT Cardiac Arrest Unresponsive to CPR, Shock, and Vasopressor** • First dose: 300 mg IV/IO push • Second dose (if needed): 150 mg IV/IO push **Life-Threatening Arrhythmias** **Maximum cumulative dose:** 2.2 g IV over 24 hours. May be administered as follows: • **Rapid infusion**: 150 mg IV over first 10 minutes (15 mg/min). May repeat rapid infusion (150 mg IV) every 10 minutes as needed • **Slow infusion**: 360 mg IV over 6 hours (1 mg/min) • **Maintenance infusion**: 540 mg IV over 18 hours (0.5 mg/min)

	***Caution*: Multiple complex drug interactions**	• Terminal elimination is extremely long (half-life lasts up to 40 days)	
Atropine sulfate Can be given via endotracheal tube	• First drug for symptomatic sinus bradycardia • May be beneficial in presence of AV nodal block; **not likely to be effective for type II second-degree or third-degree AV block or a block in non-nodal tissue** • Routine use during PEA or asystole is unlikely to have a therapeutic benefit • Organophosphate (eg, nerve agent) poisoning:	• Use with caution in presence of myocardial ischemia and hypoxia. Increases myocardial oxygen demand • Unlikely to be effective for hypothermic bradycardia • May not be effective for infranodal (type II) AV block and new third-degree block with wide QRS complexes (in these patients	**Bradycardia (With or Without ACS)** • 1 mg IV every 3 to 5 minutes as needed, not to exceed total dose of 0.04 mg/kg (total 3 mg) **Organophosphate Poisoning** Extremely large doses (2 to 4 mg or higher) may be needed

	extremely large doses may be needed	may cause paradoxical slowing; be prepared to pace or give catecholamines)	
Dopamine IV infusion	• Second-line drug for symptomatic bradycardia (after atropine) • Use for hypotension (systolic blood pressure ≤70-100 mm Hg) with signs and symptoms of shock	• Correct hypovolemia with volume replacement before initiating dopamine • Use with caution in cardiogenic shock with accompanying CHF • May cause tachyarrhythmias, excessive vasoconstriction • Do not mix with sodium bicarbonate	**IV Administration** Usual infusion rate is 5-20 mcg/kg per minute. Titrate to patient response; taper slowly
Epinephrine Can be given via	• **Cardiac arrest:** VF, pulseless VT,	• Raising blood pressure and increasing	**Cardiac Arrest** • **IV/IO dose:** 1 mg (10 mL of 0.1 mg/mL solution) administered every 3-5 minutes during resuscitation; follow each dose

endotracheal tube Available in 1:10 000 and 1:1000 concentrations	asystole, PEA • **Symptomatic bradycardia:** Can be considered after atropine as an alternative infusion to dopamine • **Severe hypotension:** Can be used when pacing and atropine fail, when hypotension accompanies bradycardia, or with phosphodiesterase enzyme inhibitor • **Anaphylaxis, severe allergic reactions:** Combine with large fluid volume, corticosteroids,	heart rate may cause myocardial ischemia, angina, and increased myocardial oxygen demand • High doses do not improve survival or neurologic outcome and may contribute to postresuscitation myocardial dysfunction • Higher doses may be required to treat poison/drug-induced shock	with 20 mL flush, elevate arm for 10-20 seconds after dose • **Higher dose:** Higher doses (up to 0.2 mg/kg) may be used for specific indications (β-blocker or calcium channel blocker overdose) • **Continuous infusion:** Initial rate: 0.1-0.5 mcg/kg per minute (for 70-kg patient: 7-35 mcg/min); titrate to response • **Endotracheal route:** 2–2.5 mg diluted in 10 mL NS **Profound Bradycardia or Hypotension** 2-10 mcg/min infusion; titrate to patient response

	antihistamines		
Lidocaine Can be given via endotracheal tube	• Alternative to amiodarone in cardiac arrest from VF/pVT • Stable monomorphic VT with preserved ventricular function • Stable polymorphic VT with normal baseline QT interval and preserved LV function when ischemia is treated and electrolyte balance is corrected • Can be used for stable polymorphic VT with baseline QT-interval prolongation if torsades suspected	• Contraindication: Prophylactic use in AMI is contraindicated • Reduce maintenance dose (not loading dose) in presence of impaired liver function or LV dysfunction • Discontinue infusion immediately if signs of toxicity develop	**Cardiac Arrest From VF/pVT** • Initial dose: 1-1.5 mg/kg IV/IO • For refractory VF, may give additional 0.5-0.75 mg/kg IV push and repeat in 5-10 minutes; maximum 3 doses or total of 3 mg/kg **Perfusing Arrhythmia** For stable VT, wide-complex tachycardia of uncertain type, significant ectopy: • Doses ranging from 0.5-0.75 mg/kg and up to 1-1.5 mg/kg may be used • Repeat 0.5-0.75 mg/kg every 5-10 minutes; maximum total dose: 3 mg/kg **Maintenance Infusion** 1-4 mg/min (30-50 mcg/kg per minute)

Magnesium sulfate	• Recommended for use in cardiac arrest only if torsades de pointes or suspected hypomagnesemia is present • Life-threatening ventricular arrhythmias due to digitalis toxicity • Routine administration in hospitalized patients with AMI is not recommended	• Occasional fall in blood pressure with rapid administration • Use with caution if renal failure is present	**Cardiac Arrest (Due to Hypomagnesemia or Torsades de Pointes)** 1-2 g (2-4 mL of a 50% solution diluted in 10 mL [eg, D5W, normal saline] given IV/IO) **Torsades de Pointes With a Pulse or AMI With Hypomagnesemia** • Loading dose of 1-2 g mixed in 50-100 mL of diluent (eg, D5W, normal saline) over 5-60 minutes IV • Follow with 0.5-1 g per hour IV (titrate to control torsades)

Science Summary Table

This table compares topics from 2015 with 2020, providing a quick reference to what has changed and what is new in the science of advanced cardiovascular life support.

ACLS topic	2015	2020
Ventilation	• 1 breath every 5 to 6 seconds for respiratory arrest, with a bag-mask device • 1 breath every 6 seconds for ventilation with an advanced airway in place	• 1 breath every 6 seconds for respiratory arrest with or without an advanced airway and also for cardiac arrest with an advanced airway (use this rate with a bag-mask device if your local protocol is continuous compressions and asynchronous ventilations for cardiac arrest)
Bradycardia	• Atropine dose: 0.5 mg • Dopamine dosing: 2 to 20 mcg/kg per minute	• Atropine dose: 1 mg • Dopamine dosing: 5 to 20 mcg/kg per minute
Tachycardia	• Synchronized cardioversion initial recommended doses: ○ –Narrow QRS complex, regular rhythm: 50 to 100 J ○ –Narrow QRS complex, irregular rhythm: 120 to 200 J ○ –Wide QRS	• Follow your specific device's recommended energy level to maximize the success of the first shock • Wide QRS complex, irregular rhythm: defibrillation dose (not synchronized)

	complex, regular rhythm: 100 J • Wide QRS complex, irregular rhythm: defibrillation dose (not synchronized)	
Post–Cardiac Arrest Care	• Titrate oxygen saturation to 94% or higher	• Titrate oxygen saturation to 92% to 98%
Adult Chain of Survival	• 5 links for both chains (IHCA and OHCA)	• 6 links for both chains (IHCA and OHCA): added a Recovery link to the end of both chains
IV/IO Access	• IV access and IO access are equivalent	• IV preferred over IO access, unless IV fails (then OK to proceed to IO)
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Cardiac Arrest	• Epinephrine 1 mg every 3 to 5 minutes or every 4 minutes as a midrange (ie, every other 2-minute rhythm check) • Amiodarone and lidocaine are equivalent for treatment (ie, either may be used) • Added maternal cardiac arrest information and algorithms (in-hospital) • Added ventricular assist device information (VAD: LVAD and RVAD) and algorithm • Added new prognostication diagram and information • Recommend using waveform capnography with a bag-mask device	
Stroke	• Revised stroke algorithm • New stroke triage algorithm for EMS destination • Focus on large vessel occlusion (LVO) for all healthcare providers • Endovascular therapy: treatment window up to 24 hours (previously up to 6 hours)	

	• Both alteplase and endovascular therapy can be given/performed if time criteria and inclusion criteria are met • Consider having EMS bypass the emergency department and go straight to the imaging suite (CT/MRI); initial assessment can be performed there to save time • Titrate oxygen saturation to >94%

Glossary

This table defines some common terms used in ACLS.

A	
Acute	Having a sudden onset and short course
Acute myocardial infarction (AMI)	The early critical stage of necrosis of heart muscle tissue caused by blockage of a coronary artery
Advanced cardiovascular life support (ACLS)	Emergency medical procedures in which basic life support efforts of CPR are supplemented with drug administration, IV fluids, etc
Asystole	Absence of electrical and mechanical activity in the heart
Atrial fibrillation	In atrial fibrillation the atria “quiver” chaotically and the ventricles beat irregularly
Atrial flutter	Rapid, irregular atrial contractions due to an abnormality of atrial excitation
Atrioventricular (AV) block	A delay in the normal flow of electrical impulses that cause the heart to beat
Automated external defibrillator (AED)	A portable device used to restart a heart that has stopped
B	
Basic life support (BLS)	Emergency treatment of a victim of cardiac or respiratory arrest through cardiopulmonary resuscitation and emergency cardiovascular care
Bradycardia	Slow heart rate, whether physiologically or pathologically
C	
Capnography	The measurement and graphic display of CO_2 levels in the airways, which can be performed by infrared spectroscopy
Cardiac arrest	Temporary or permanent cessation of the heartbeat
Cardiopulmonary resuscitation (CPR)	A basic emergency procedure for life support, consisting of mainly manual external cardiac massage and some artificial respiration
Coronary syndrome	A group of clinical symptoms compatible with acute myocardial ischemia. Also called coronary heart disease.
Coronary thrombosis	The blocking of the coronary artery of the heart by a thrombus

E	
Electrocardiogram (ECG)	A test that provides a typical record of normal heart action
Endotracheal (ET) intubation	The passage of a tube through the nose or mouth into the trachea for maintenance of the airway
Esophageal-tracheal tube	A double-lumen tube with inflatable balloon cuffs that seal off the hypopharynx from the oropharynx and esophagus; used for airway management
H	
Hydrogen ion (acidosis)	The accumulation of acid and hydrogen ions or depletion of the alkaline reserve (bicarbonate content) in the blood and body tissues, decreasing the pH
Hyperkalemia	An abnormally high concentration of potassium ions in the blood. Also called hyperpotassemia.
Hypoglycemia	An abnormally low concentration of glucose in the blood
Hypokalemia	An abnormally low concentration of potassium ions in the blood. Also called hypopotassemia.
Hypothermia	When the patient's core body temperature is below 96.8°F (36°C)
Hypovolemia	A decrease in the volume of circulating blood
Hypoxia	A deficiency of oxygen reaching the tissues of the body
I	
Intraosseous (IO)	Within a bone
Intravenous (IV)	Within a vein
M	
Mild hypothermia	When the patient's core body temperature is between 93.2°F and 96.8°F
Moderate hypothermia	When the patient's core body temperature is from 86°F and 93.2°F
N	
Nasopharyngeal (NPA)	Pertaining to the nose and pharynx

O	
Oropharyngeal airway (OPA)	A tube used to provide free passage of air between the mouth and pharynx
P	
Perfusion	The passage of fluid (such as blood) through a specific organ or area of the body (such as the heart)
Prophylaxis	Prevention of or protection against disease
Pulmonary edema	A condition in which fluid accumulates in the lungs
Pulseless electrical activity (PEA)	Continued electrical rhythmicity of the heart in the absence of effective mechanical function
R	
Recombinant tissue plasminogen activator (rtPA)	A clot-dissolving substance produced naturally by cells in the walls of blood vessels
S	
Severe hypothermia	When the patient's core body temperature is below 86°F
Sinus rhythm	The rhythm of the heart produced by impulses from the sinoatrial node
Supraglottic	Situated or occurring above the glottis
Synchronized cardioversion	Uses a sensor to deliver a shock that is synchronized with a peak in the QRS complex
Syncope	A loss of consciousness over a short period of time, caused by a temporary lack of oxygen in the brain
T	
Tachycardia	Increased heart rate, usually ≥100/min
Tamponade (cardiac)	A condition caused by accumulation of fluid between the heart and the pericardium, resulting in excess pressure on the heart. This impairs the heart's ability to pump sufficient blood.
Tension pneumothorax	Pneumothorax resulting from a wound in the chest wall which acts as a valve that permits air to enter the pleural cavity but prevents its escape

Thrombus	A blood clot formed within a blood vessel
U	
Unsynchronized shock	An electrical shock that will be delivered as soon as the operator pushes the shock button to discharge the defibrillator. Thus, the shock can fall anywhere within the cardiac cycle.
V	
Ventricular fibrillation (VF)	Very rapid uncoordinated fluttering contractions of the ventricles
Ventricular tachycardia (VT)	A rapid heart rate that originates in one of the lower chambers (ventricles) of the heart