American Heart Association.

ACLS

ADVANCED CARDIOVASCULAR LIFE SUPPORT

PROVIDER MANUAL

Advanced Cardiovascular Life Support

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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 **heart.org/courseupdates.**

Contents

Appendix

Note on Medication Doses

Emergency cardiovascular care is a dynamic science. Advances in treatment and medication 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 medication and medical device and the course updates available on **heart.org/courseupdates**.

Part 1
Overview of ACLS

Introduction

Course Description and Goal

The Advanced Cardiovascular Life Support (ACLS) Provider Course is designed for health care professionals who either direct or participate in the management of cardiac arrest, stroke, or other cardiopulmonary emergencies. Through didactic instruction and active participation in simulated cases, you will enhance your skills in the recognition and time-sensitive intervention of cardiopulmonary arrest, post–cardiac arrest, acute dysrhythmia, stroke, and acute coronary syndromes (ACS). The goal of the ACLS Provider Course is to improve outcomes for adult cardiac arrest, stroke, and other cardiopulmonary emergencies through early recognition and time-sensitive intervention by high-performance teams.

Course Objectives

After successfully completing this course, you should be able to

- Apply the basic life support (BLS), primary, and secondary assessments sequence for a systematic evaluation of adult patients
- Discuss how the use of a rapid response team (RRT) or medical emergency team (MET) 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 bradycardias and tachycardias that may result in cardiac arrest or complicate resuscitation outcome
- Perform early management of bradycardias and tachycardias 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, including prioritizing early chest compressions, ventilation, and integrating early automated external defibrillator (AED) use
- Perform optimized management of cardiac arrest until termination of resuscitation or transfer of care, including post–cardiac arrest care
- Evaluate resuscitative efforts during a cardiac arrest through continuous assessment of cardiopulmonary resuscitation (CPR) quality, monitoring the patient's 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 (managing multiple rhythms) 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 medication therapy
- Performance as an effective leader and member of a high-performance team
- Achievement of objective measures, such as chest compression fraction (CCF)

Course Prerequisites and Preparation

The American Heart Association (AHA) limits enrollment in this course to health care professionals 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 prework in the 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 ACLS 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 score report and bring them with you to the course.**

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

- BLS skills
- Electrocardiogram (ECG) rhythm interpretation for core ACLS rhythms
- Knowledge of airway management and adjuncts
- Basic ACLS medication and pharmacology knowledge
- Practical application of ACLS rhythms and medications
- 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 you to recognize these ECG presentations:

- Sinus rhythm
- Atrial fibrillation and flutter
- Sinus bradycardia
- Sinus tachycardia
- Narrow-complex tachycardia
- Atrioventricular (AV) blocks
- Asystole
- Ventricular tachycardias (VTs)
- Ventricular fibrillation (VF)

You should also recognize the clinical condition called *pulseless electrical activity* (PEA). This is the presence of an organized rhythm (not VF or pulseless VT [pVT]) on an ECG without a corresponding pulse (no pulse).

The 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 Medication and Pharmacology Knowledge

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

The 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*, ACLS Student Resources, and 3 reference cards.

The computer icon directs you to additional supplementary information in the 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.** If you are using the eBook version, you should download the manual to your device's eReader app and bring it with you 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

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

- Precourse Self-Assessment (passing score 70% or greater)
- Prework (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 health care professionals.

- Precourse Preparation Checklist (used to ensure that you 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 Emergency Cardiovascular Care 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
- ACS and stroke
- Cardiac arrest in select special situations and neuroprognostication

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 (OPA) and nasopharyngeal airway (NPA) 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 BLS and advanced life support (ALS) to save a person's life. The *2025 AHA Guidelines for CPR and Emergency Cardiovascular Care* reviewed evidence that in both in-hospital and out-of-hospital settings, many patients with cardiac arrest 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. Figure 1 shows the recent survival trends in both IHCA and out-of-hospital cardiac arrest (OHCA) in the United States.[2]

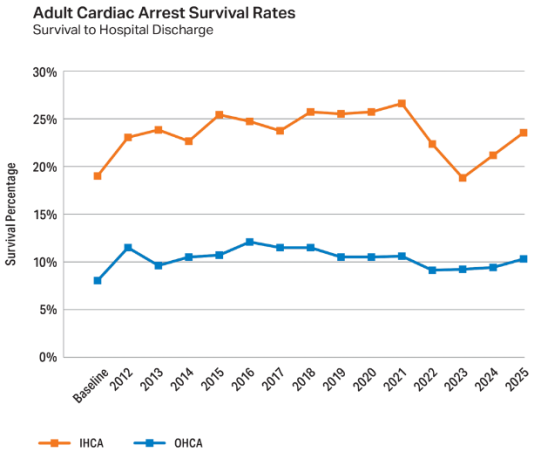


Figure 1. Recent adult cardiac arrest survival rates.

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 health care professionals to become more knowledgeable about what improves survival rates
- Proactive planning and simulation of cardiac arrest to provide the opportunity for a health care professional 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 ALS strategies (eg, defibrillation, medications, advanced airway).
- Studies have shown that reducing the interval between compressions and shock delivery can increase shock success. Proven strategies include limiting the number of interruptions in compressions to critical interventions (eg, rhythm analysis, shock delivery) and minimizing 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

Health care 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 2).

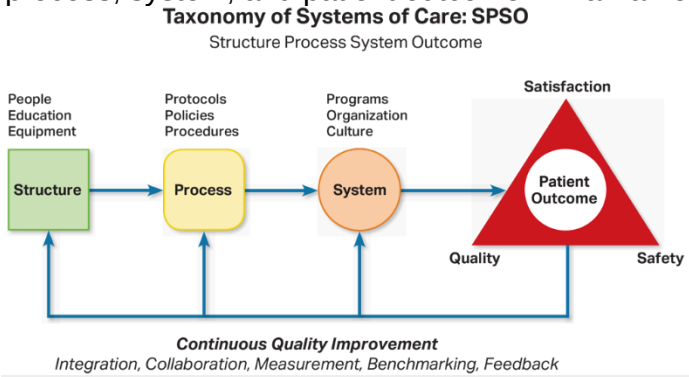


Figure 2. Taxonomy of systems of care.

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.

The key factors associated with improved survival from cardiac arrest involve all of the links in the chain with no gaps. Everything matters, from the education and training of health care professionals and lay rescuers, the design and availability of equipment, and community-wide recognition of cardiac arrest to the integration of different emergency teams, transport to well-

prepared centers for intervention, and 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 cardiac arrest Chain of Survival (Figure 3) represent these actions and include the following elements:

- Recognition and emergency activation, comprising early recognition of cardiac arrest and rapid response either in- or out-of-hospital
- High-quality CPR
- Defibrillation, including early defibrillation of VF and pVT
- Advanced resuscitation interventions, including medications, advanced airway interventions, and extracorporeal CPR (ECPR)
- Post–cardiac arrest care, including critical care interventions and targeted temperature management
- Recovery and survivorship, including effective support for the physical and cognitive care of the patient and the emotional needs of both the patient and the family

Figure 3. 2025 Cardiac Arrest Chain of Survival.

The Chain of Survival is an infographic 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.

Regardless of where an arrest occurs, the care after resuscitation converges in the hospital, generally in an emergency department (ED) or intensive care unit (ICU). This post–cardiac arrest care is depicted as the second-to-last link in the chain, symbolized by a hospital bed with a monitor and thermometer, which represent critical care interventions, advanced monitoring, and temperature control. Patients who achieve return of spontaneous circulation (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]

Health care systems should implement a comprehensive, structured, consistent, multidisciplinary system of care for treating post–cardiac arrest patients. Programs should address oxygenation, ventilation, and hemodynamic optimization, temperature control, immediate coronary reperfusion with percutaneous coronary intervention (PCI) for eligible patients, and neurologic care and prognostication. The final link is recovery and survivorship. This link represents the important elements to consider for a survivor and their caregivers. More people survive cardiac arrest now than ever before. Unfortunately, some survivors of cardiac arrest experience emotional, social, physical, neurologic, and cognitive sequelae, some of which begin to manifest early whereas some may not become apparent until after hospital discharge.[6,7] Therefore, improving cardiac arrest survivorship—the journey from stabilization through rehabilitation, recovery, and societal reintegration—must be a systematic priority involving families, health care partners, and communities.

Optimizing survivorship for survivors and their caregivers involves in-hospital screening, treatment, referral, and postdischarge surveillance of postarrest sequelae.[6] Recent evidence has demonstrated that the quality of life of survivors improves when they are enrolled in an early cognitive-focused screening, self-management, and referral program.[8] Additionally, community reintegration and return to work or other daily activities may be slow and depend on availability of social support and degree of postarrest sequelae, highlighting the need for direction on managing postarrest challenges and return to daily activities.

Finally, survivorship also involves lay rescuers, families, and health care professionals. With burnout being common among health care professionals and many of them experiencing emotional distress while caring for patients with cardiac arrest, interventions to support these individuals are critical.

Key Considerations for Survivorship

- Cardiac arrest survivors should have multimodal rehabilitation assessment and treatment for cognitive, physical, neurologic, and cardiopulmonary impairments before hospital discharge.
- Cardiac arrest survivors and caregivers should have multidisciplinary discharge planning. This should include medical and rehabilitative treatment recommendations and return to activity and work expectations.
- Interventions to address health care worker burnout may be beneficial.
- Debriefing and referrals for emotional support may be beneficial for lay rescuers and health care professionals (in- and out-of-hospital) after cardiac arrest events.

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. **CQI efforts** rely on valid assessments of resuscitation performance and patient outcomes. Measuring patient care is key to CQI.

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

Change

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

- Increased lay rescuer CPR response rates
- Improved CPR performance
- Shortened time to defibrillation
- Lay rescuer awareness
- Lay rescuer and health care professional education and training

STEMI Systems of Care

The goal of ST-segment elevation myocardial infarction (STEMI) care is to minimize heart damage and maximize the quality of the patient's recovery. The STEMI links (Figure 4) indicate the actions that patients, family members, and health care professionals can rapidly take to maximize the quality of STEMI recovery:

- Rapid recognition of and reaction to STEMI warning signs and symptoms
- EMS telecommunicators and rapid transport and prearrival notification to the receiving ED
- Assessment and diagnosis in the ED (or cath lab)
- Rapid treatment

Figure 4. **STEMI Chain of Survival.**

When authorized by medical control or protocol, telecommunicators should tell patients with no history of aspirin allergy or signs of active or recent gastrointestinal (GI) bleeding to chew aspirin (162-325 mg) while they wait for emergency responders to arrive. Emergency responders should rapidly transport patients with STEMI to the appropriate ED with PCI capability, while hospital systems should diagnose and provide reperfusion interventions or transfer a patient to a capable facility as soon as possible for the best possible outcomes.

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 health care professionals should take to maximize stroke recovery. These links are

- Rapid recognition of and reaction to stroke warning signs and symptoms
- Rapid use of 911 and telecommunicators
- Rapid recognition of stroke, triage, transport, and prehospital notification to the receiving hospital by emergency responders
- Rapid diagnosis and treatment in the hospital

Figure 5. **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 intravenous (IV) alteplase. Regional stroke systems of care for acute ischemic stroke (AIS) 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, health care professionals 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 (there should be no threat to the health care professional). 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.

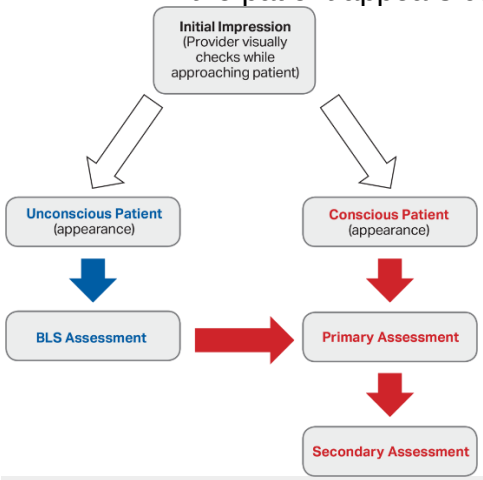


Figure 6. The systematic approach.

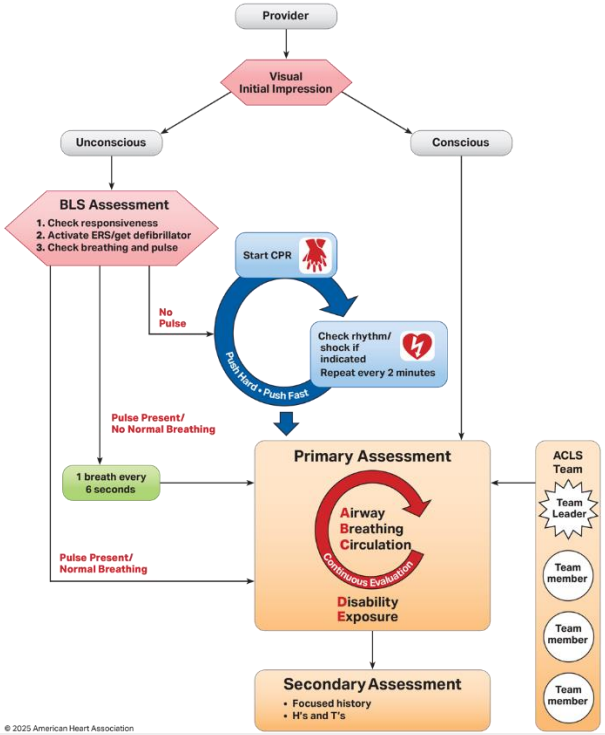


Figure 7. Expanded Systematic Approach Algorithm.

Abbreviation: ERS, emergency response system.

BLS Assessment

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

Critical Concepts

High-Quality CPR

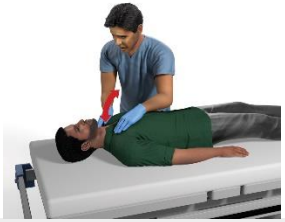
To perform high-quality CPR, emergency responders should

- Start compressions immediately, within 10 seconds after recognizing cardiac arrest
- Push hard and fast: Compress at a rate of 100 to 120/min with a depth of at least 2 inches
- Allow complete chest recoil after each compression; *avoid leaning on the chest between compressions*
- Minimize interruptions in compressions (limit interruption to 10 seconds or less)
- Avoid excessive ventilation; deliver each ventilation over 1 second, enough for visible chest rise

Remember to assess first and then perform the appropriate action.

The BLS assessment requires no advanced equipment. You can use readily available supplies, such as a bag-mask ventilation device if it is accessible. When possible, place the patient faceup on a firm, flat surface to maximize the effectiveness of chest compressions. Table 1 is an overview of the BLS assessment, and Figures 8 through 14 illustrate the steps needed during the BLS assessment, after verifying scene safety.

Table 1. BLS Assessment

Assessment	Assessment technique and actions	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 or 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, and get an AED.**

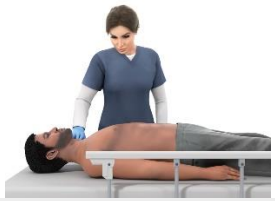
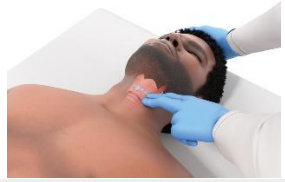
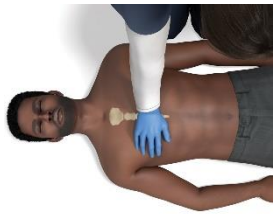
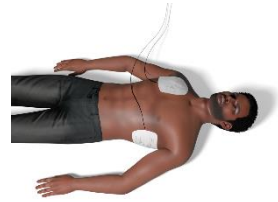
Assess for breathing and a pulse.	• Assess the patient for normal breathing and a pulse. • To minimize delay in starting CPR, you should assess breathing at the same time as you check the pulse. This should take at least 5 seconds but no more than 10 seconds. • To check for breathing, scan the person's chest for rise and fall for no more than 10 seconds. • **If the person is breathing:** Monitor the person until additional help arrives. • **If the person is not breathing or is only gasping and a pulse is not felt within 10 seconds:** Immediately begin high-quality CPR, starting with compressions. • **If you find a pulse:** Start ventilating at 1 every 6 seconds. Check the pulse about every 2 minutes.	 **Figure 10. Check breathing and pulse simultaneously.**  **Figure 11. Check for a carotid pulse.**
Begin CPR, starting with compressions.	• Push hard and fast: Compress at a rate of 100-120/min with a depth of at least 2 inches. • Allow complete chest recoil after each compression. • Limit interruptions to 10 seconds or less. • Avoid excessive ventilation. Deliver each ventilation over 1 second, enough for visible chest rise.	 **Figure 12A. Hand position.**  **Figure 12B. Hand position.**

		 **Figure 13A. Compressions.**  **Figure 13B. Compressions.**
Defibrillate.	• If a pulse is not felt, check for a shockable rhythm with an AED or defibrillator as soon as it arrives. • Provide shocks as indicated. • Follow each shock immediately with CPR, beginning with compressions.	 **Figure 14A. Defibrillation.**  **Figure 14B. Defibrillation.**

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 (5-6 cm).

Tailoring the Response

Single rescuers may tailor the sequence of rescue actions to the most likely cause of arrest. For example, an emergency responder who sees an adult suddenly collapse (eg, after a blow to the chest) can assume that the patient has had a sudden cardiac arrest. In this case, the emergency responder should activate the emergency response system via mobile device, get an AED if one is nearby, return to the patient to attach the AED, and then provide CPR. However, if the emergency responder believes hypoxia caused the cardiac arrest (eg, in a drowning patient), they 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 15.

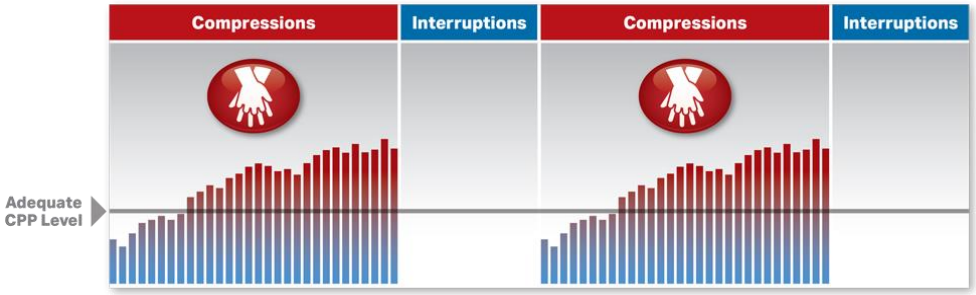


Figure 15. Relationship of quality CPR to coronary perfusion pressure, demonstrating the need to minimize interruptions in compressions.

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 partial pressure of end-tidal carbon dioxide (P_{ETCO_2}) is related to

cardiac output with chest compressions during cardiac arrest, ROSC is similarly unlikely with a persistent P_{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.

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

Caution

Seizure-Like Activity

During cardiac arrest, a person will become unresponsive and collapse. They can look like they are having a seizure for the first few minutes. They might stop breathing or may only gasp, which can sound like a snort, snore, or groan.

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 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 2 provides an overview of the primary assessment.

Remember to assess first and then perform the appropriate action.

Table 2 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 OPA, or an NPA.
	• Use advanced airway management if needed (eg, supraglottic airways, 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 and a laryngeal 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 and oxyhemoglobin saturation monitored?
	• Give supplemental oxygen when indicated.
	  ○ –For patients with cardiac arrest, administer 100% oxygen.
	  ○ –For other patients (not in cardiac arrest), adjust the oxygen administration to achieve oxygen saturation of 95%-98% by pulse oximetry (90% for ACS and 90%-98% for post–cardiac arrest care).
	• 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 IV or IO access been established?
• Is ROSC present?
• Is the patient with a pulse unstable?
• Are medications needed for rhythm or BP?
• Does the patient need volume (fluid) for resuscitation?
	• Monitor CPR quality.
	  ○ –Use a CPR feedback device.
	  ○ –Monitor quantitative waveform capnography (if P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality). Continuous 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 CO_2 exhaled is associated with the amount of blood that passes through the lungs. A P_{ETCO_2} of <10 mm Hg during chest compressions rarely results in ROSC.
	  ○ –A sudden increase in P_{ETCO_2} may indicate ROSC. The exact increase in P_{ETCO_2} has not been defined and is still under investigation.
	• Attach monitor or defibrillator for arrhythmias or cardiac arrest rhythms (eg, VF, pVT, asystole, PEA).
	• Provide defibrillation or cardioversion.
	• Obtain IV or IO access.
	• Give appropriate medications to manage rhythm and BP.
	• Give IV or IO fluids if needed.
	• Check temperature.
	• Check perfusion issues.

Disability	• Check for neurologic function. • Check glucose. • Quickly assess for responsiveness, levels of consciousness, and pupil dilation. • Remember 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 jewelry.

Abbreviations: BP, blood pressure; IO, intraosseous.

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 underlying causes of cardiac arrest and emergency cardiopulmonary conditions:

H’s

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypokalemia/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 them immediately.

Diagnosing and Treating Underlying Causes

Patients in cardiac arrest (VF, pVT, asystole, or PEA) need rapid assessment and management to determine if an underlying, potentially reversible problem caused the arrest. If you can quickly identify and treat 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. When done by an experienced health care professional, ultrasound may help with identifying the underlying cause quickly, as long as it does not delay or interrupt high-quality CPR. Ultrasound might provide information to help determine the next step for treatment, but the benefit remains uncertain. 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 medication overdose or 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 (BP) 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 of hypovolemia can reverse the pulseless state. Common nontraumatic causes of hypovolemia include occult internal hemorrhage and severe dehydration. Consider volume infusion for PEA associated with a narrow-complex tachycardia in conjunction with simultaneous compression, ventilations, and other appropriate ALS interventions.

Cardiac and Pulmonary Conditions

ACS that involves 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 health care professional can perform bedside ultrasound to help rapidly identify tamponade, pneumothorax, and echocardiographic evidence of PE.

Medication Overdoses or Toxic Exposures

Certain medication 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 during medication and toxic exposure include

- Prolonged CPR in special resuscitation situations
- ECPR
- Intra-aortic balloon pump therapy
- Renal dialysis
- IV lipid emulsion for lipid-soluble toxins
- Specific medication antidotes (digoxin immune Fab, glucagon, bicarbonate, naloxone)
- Transcutaneous pacing
- Correction of severe electrolyte disturbances (potassium, magnesium, calcium, acidosis)
- Specific adjunctive agents (eg, calcium, glucagon)

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, health care professionals 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 BP (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.

Although the ideal composition of RRTs or METs is not known, many published before-and-after studies of RRTs or METs 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

Acute Coronary Syndromes

Overview

While substantial progress has been achieved in the diagnosis and treatment of ACS, cardiovascular disease is still the leading cause of death in the world.[6] ACS is defined as a sudden reduction of blood flow to the heart. Half of ACS deaths occur before the patient reaches the ED, with VF or pVT as the precipitating rhythm in most cases. VF is most likely to develop during the

first 4 hours after onset of symptoms. Therefore, it is important that communities develop EMS and hospital programs to respond quickly to ACS. Such programs should focus on the STEMI Chain of Survival (Figure 4), considering

- Recognizing signs and symptoms of ACS
- Activating the EMS system, with rapid transport and prearrival notification to the receiving ED/cath lab by emergency responders; 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 emergency responders, ED, cath lab, and cardiac specialists
- Providing time-sensitive treatment[7]

The primary goals of ACS care are prevention of major adverse cardiovascular events, such as heart failure, low left ventricular ejection fraction, and death. Additional goals are identification of patients with STEMI, triage for early reperfusion therapy, relief of ischemic chest discomfort, and treatment of acute, life-threatening complications of ACS.

Rhythms for ACS

VF, VTs, and hypotensive bradycardia (including heart blocks) may occur with acute ischemia.

Medications for ACS

Medication 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 the following medications to relieve ischemic discomfort, dissolve clots, and inhibit thrombin and platelets:

- Oxygen
- Aspirin
- Nitroglycerin
- Opiates (eg, morphine, fentanyl)
- Fibrinolytic therapy (overview)
- Anticoagulants (eg, unfractionated heparin, bivalirudin, enoxaparin, fondaparinux)

Additional adjunctive agents may include the following:

- β-Blockers (if no contraindications)
- P2Y12 inhibitors (prasugrel, ticagrelor)
- Angiotensin-converting enzyme inhibitors

- High-intensity statin therapy
- Glycoprotein IIb/IIIa inhibitors

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 3 related clinical conditions—unstable angina, non–ST-segment elevation ACS (NSTE-ACS) or non–ST-segment elevation myocardial infarction (NSTEMI), and STEMI. Sudden cardiac death may occur with any of these syndromes. Figure 16 illustrates the pathophysiology of ACS.

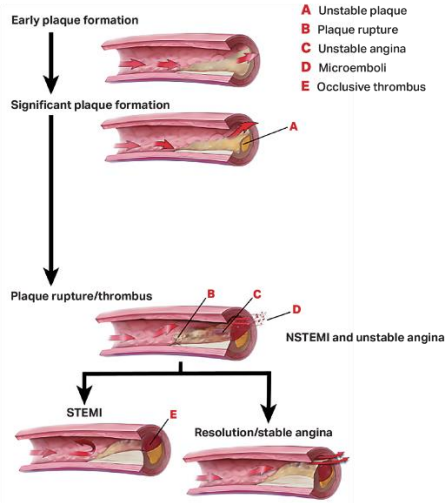

Figure 16. Pathophysiology of ACS.

Managing ACS

Managing ACS is time-sensitive and requires a systematic approach for best outcomes. The ACS Algorithm (Figure 17) will help guide your assessment and clinical strategy when patients have signs and symptoms of ACS, including possible STEMI. To apply this algorithm effectively, you must have the basic knowledge to assess and stabilize patients with ACS.[7]

The initial 12-lead ECG is used to classify patients into 1 of 2 major ECG categories of ACS—STEMI and NSTEMI or unstable angina—each with different strategies of care and management needs.

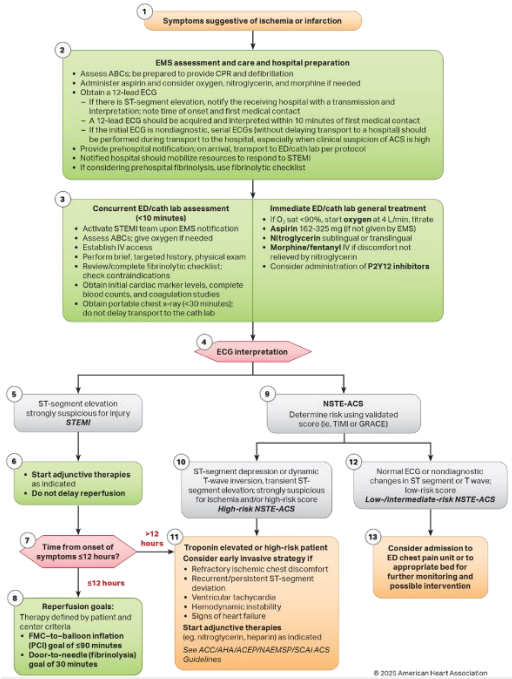

Figure 17. ACS Algorithm.

Abbreviations: ACC, American College of Cardiology; ACEP, American College of Emergency Physicians; GRACE, Global Registry of Acute Coronary Events; NAEMSP, National Association of EMS Physicians; SCAI, Society for Cardiovascular Angiography and Interventions; TIMI, thrombolysis in myocardial infarction.

The ACS Algorithm

The ACS Algorithm (Figure 17) outlines the steps for assessing and managing a patient who has symptoms suggestive of ischemia or infarction (ACS symptoms, Step 1). The emergency responder assessment and care and hospital preparation (Step 2) should include the following:

- Assess ABCs (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 and interpretation; note time of onset and first medical contact (FMC).
- A 12-lead ECG should be acquired and interpreted within 10 minutes after FMC.

    - –If the initial ECG is nondiagnostic, serial ECGs (without delaying transport to a hospital) should be performed during transport to the hospital, especially when clinical suspicion of ACS is high.
- Provide prehospital notification; upon arrival, transport to ED or cath lab per protocol.
- The notified ED should mobilize hospital resources to respond to STEMI and activate STEMI alert.
- If considering prehospital fibrinolysis, use a fibrinolytic checklist.
- If out-of-hospital emergency responders cannot complete these initial steps before the patient arrives at the ED/cath lab, the ED/cath lab professionals should do so.

Starting With Telecommunicators

All telecommunicators and emergency responders must train to recognize ACS symptoms along with the potential complications. When authorized by medical control or protocol, telecommunicators should tell patients with no history of aspirin allergy or signs of active or recent GI bleeding to chew aspirin (162-325 mg) while they wait for emergency responders to arrive. If STEMI is suspected, emergency responders should immediately transport the patients to a PCI-capable hospital for primary PCI, with an FMC-to-first-device (balloon inflation) time goal of 90 minutes or less.[5-8]

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

- Activate STEMI team upon notification by emergency responders.
- Assess ABCs; give oxygen if needed.
- Establish IV access.
- A nondiagnostic 12-lead ECG should be compared to prior ECGs, and a repeat ECG should be obtained during the ED/cath lab course to assess for evolving changes.
- 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 or 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 (162-325 mg), if not given by emergency responders
- Nitroglycerin, sublingual or translingual
- Morphine or fentanyl IV if discomfort is not relieved by nitroglycerin

- Consider administration of P2Y12 inhibitors

You should know how to identify symptoms that suggest cardiac ischemia (Figure 18) (Step 1). Promptly conduct a targeted evaluation of every patient whose initial symptoms suggest possible ACS.

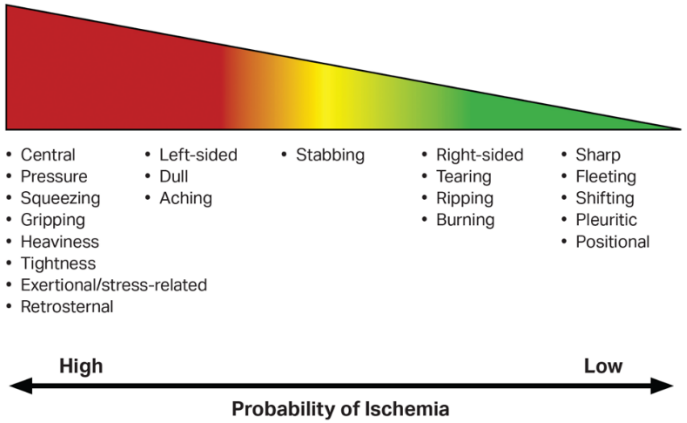

Figure 18. Index of suspicion that chest discomfort is ischemic in origin on the basis of commonly used descriptors.

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 male and female) with ACS, but patients frequently deny or misinterpret this and other symptoms. Older, female, diabetic patients and hypertensive patients are most likely to delay, in part because they are more likely to have symptoms or presentations that are less common. 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 ED/cath lab 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 longer 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 (more commonly in women) and is described as indigestion. Diabetic patients may present without chest discomfort but with complaints of weakness, fatigue, or severe exhaustion.
- Symptoms on the left or right side of the chest; stabbing, sharp pain; or discomfort in the throat or abdomen may occur in women, older patients, and patients with diabetes.[8,9]
- Chest pain remains the predominant symptom reported by women ultimately diagnosed with ACS, occurring with a frequency equal to that of men, and ACS should be considered in both women and men presenting with chest pain.
- Women may also present with accompanying symptoms (eg, nausea, fatigue, shortness of breath) more often than men.

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

Administering Oxygen and Medications

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

Oxygen

EMS professionals 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. EMS professionals 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 health care professionals may consider withholding it in these patients.

Aspirin (Acetylsalicylic Acid)

A dose of 162 to 325 mg of chewed aspirin (non–enteric-coated preferred) causes immediate and near-total inhibition of thromboxane A2 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 (162-325 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 5 minutes (total of 3 doses max) for ongoing symptoms if permitted by medical control and no contraindications exist. 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 myocardial infarction (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 health care professional 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), and with caution (relative contraindication) with marked bradycardia (heart rate less than 50/min), or marked tachycardia (heart rate greater than 110).
- **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.

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.

Opiates (Morphine, Fentanyl)

Consider administering morphine or fentanyl 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 (P2Y12 inhibitors). Currently, there is 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.

Dosage: 2 to 4 mg; may repeat if needed every 5 to 15 minutes. Doses up to 10 mg may be considered.

Fentanyl is indicated in STEMI when chest discomfort does not respond to nitrates. Use fentanyl for relief of pain that is resistant to other maximally tolerated anti-ischemic medications. May delay the effects of oral P2Y12 therapy.[8] Monitor closely for adverse effects.

Dosage: 25 to 50 mcg; may repeat if needed. Doses up to 100 mcg may be considered.

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.

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 and fentanyl 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.

Important Considerations

The ACS Algorithm (Figure 17) provides general guidelines, on the basis of patient symptoms and the 12-lead ECG, for the initial triage of patients. Health care professionals often obtain serial cardiac markers (cardiac troponins [cTn]) in patients who 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 patients with STEMI, you do not need evidence of elevated cardiac markers to decide to administer fibrinolytic therapy or perform diagnostic coronary angiography with coronary intervention (angioplasty or stenting).

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.

Obtaining a 12-Lead ECG

The AHA recommends out-of-hospital 12-lead ECG diagnostic programs in all EMS systems, and all emergency responders should take the actions outlined in Table 3 if appropriate.

Table 3. Emergency Responder Actions per AHA Recommendations

Emergency responder 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 (10-60 minutes has been achieved in clinical studies) and speeds reperfusion therapy with fibrinolytics, PCI, or both, which may reduce mortality and minimize myocardial injury. Qualified and specially trained emergency responders can accurately identify typical ST-segment elevation in the 12-lead ECG. If emergency responders are not trained to interpret the 12-lead ECG, field transmission of the ECG or a computer report to the receiving hospital is recommended.
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.

The 12-lead ECG (example in Figure 19) is at the center of the decision pathway in managing ischemic chest discomfort and is the only way to identify STEMI.

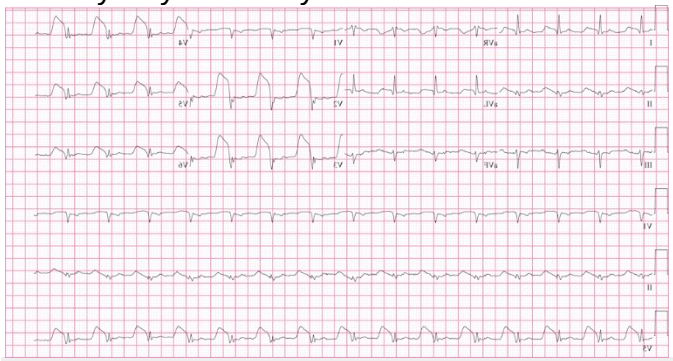

Figure 19. Example of Anterior STEMI on a 12-lead ECG.

Figure 20 shows how to measure ST-segment deviation.

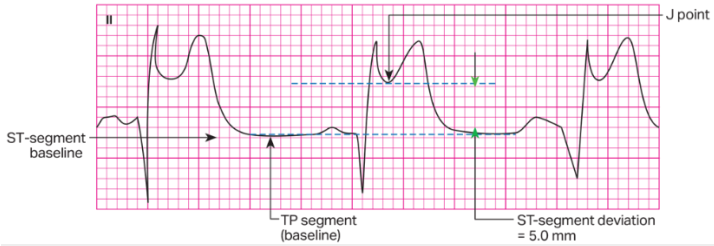

Figure 20A. How to measure ST-segment deviation. A, Inferior myocardial infarction—the ST segment has no low point (it is coved or concave).

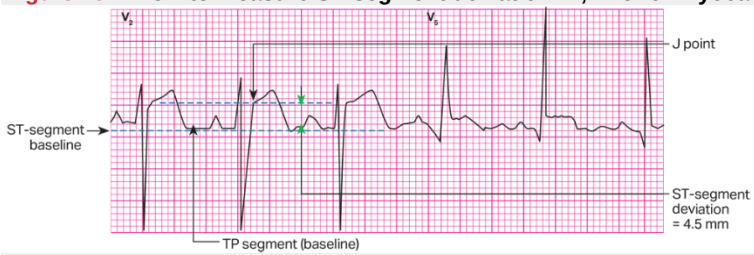

Figure 20B. Anterior myocardial infarction.

Classifying Patients by ST-Segment Deviation

Treatment recommendations are specific to each major group[8]:

- STEMI
- NSTE-ACS/NSTEMI and unstable angina
    - –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. Primary PCI is the preferred method of reperfusion for patients with STEMI.

Figure 21 illustrates the breakdown of information in both major categories of ACS.

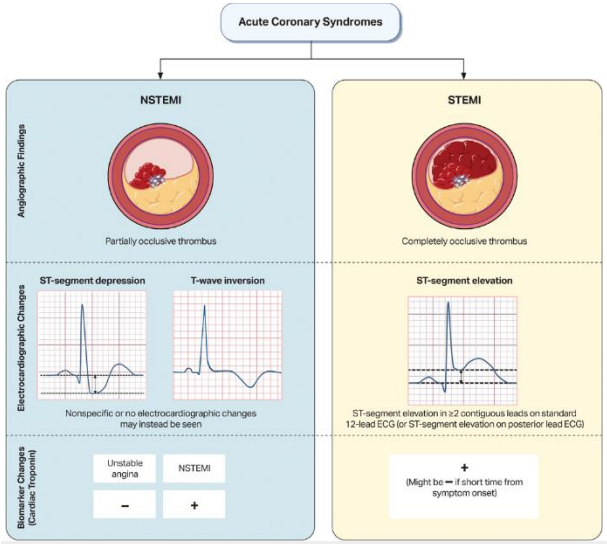

Figure 21. ACS broken down into 2 major categories.

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 new or presumably new ST-segment elevation in 2 or more anatomically contiguous leads. Threshold values for ST-segment elevation consistent with STEMI are J-point elevation of 1 mm or more in all leads other than V2 and V3 in all men and women; greater than 2 mm (0.2 mV) in leads V2 and V3 in men (2.5 mm in men younger than 40 years); and 1.5 mm in all women.
    - –New or presumably new left bundle branch block (LBBB) in isolation should not be considered diagnostic for acute myocardial infarction (AMI); clinical correlation is required.
    - –A new LBBB in an asymptomatic patient does not constitute a STEMI equivalent.
    - –Posterior leads (V7-V9) should be obtained in patients with suspected left circumflex occlusion, particularly in the setting of isolated ST-segment depression 0.5 mm or greater in leads V1 through V3.
- **NSTE-ACS/NSTEMI** including unstable angina (Step 9):
    - –High-risk NSTE-ACS (Step 10) is characterized by new or presumably new and usually dynamic horizontal or down-sloping ST-depression 0.5 mm or greater in 2 or more contiguous leads or T wave inversion greater than 1 mm in 2 or more contiguous leads with prominent R wave or R/S ratio greater than 1 or transient ST-segment elevation.
        - Many patients with NSTE-ACS have either nonspecific ST-segment or T-wave changes or a normal ECG. The absence of ECG evidence of ischemia does not exclude ACS. If troponin is

elevated or if this is a high-risk patient, consider early invasive strategy if there is any of the following (Step 11):

- Refractory ischemic chest discomfort
- Recurrent or persistent ST-segment deviation
- VT
- Hemodynamic instability
- Signs of heart failure

- Start adjunctive therapies (eg, nitroglycerin, heparin) as indicated. Refer to the "2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines."[8]

- –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 (cTn) 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.

Caution

12-Lead ST-Segment Changes

ST-segment changes may be observed in other conditions, including acute pericarditis, left ventricular hypertrophy, LBBB, Brugada syndrome, right ventricular pacing, Takotsubo syndrome, and early repolarization that may obscure the diagnosis of STEMI.

If STEMI is present, review and complete fibrinolytic checklist and check contraindications. Obtain and review initial cardiac marker levels (cTn I or T—preferably high-sensitivity cTn, if available) 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). Because the sensitivity and negative predictive values of high-sensitivity cTn are greater, high-sensitivity cTn assays are preferred over conventional cTn assays. If initial cardiac markers are nondiagnostic, repeat measurements after the initial sample collection (time zero) at 1 to 2 hours for high-sensitivity cTn and 3 to 6 hours for conventional cTn assays.

For a patient with STEMI, the goals of reperfusion include the following:

- PCI should begin within 90 minutes after FMC to balloon inflation.
    - –*Note*: An arterial approach to PCI is preferred over a femoral approach.
- Fibrinolytic administration should begin within 30 minutes after the patient's arrival in the ED.

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 medication (or PCI). These 4 major points of in-hospital therapy are commonly referred to as the 4 D's.
- All health care professionals 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 or 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 FMC-to-first-device (balloon inflation) time of 90 minutes or less. For patients at a non–PCI-capable hospital, time from FMC 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 cath lab for PCI immediately after ED presentation or will go straight to the cath lab.
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 FMC-to-balloon inflation time of 90 minutes or less by a skilled health care professional at a skilled PCI facility.
- Primary PCI may also be offered to patients presenting to non–PCI-capable hospitals if PCI can be initiated promptly within 120 minutes after FMC.

- 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 FMC.
- 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 V2 and V3 and 1 mm or more in all other leads without contraindications. Fibrin-specific agents achieve normal flow in about 50% of patients given these medications. Examples of fibrin-specific medications are alteplase, reteplase, and tenecteplase.

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 FMC.
- 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 health care professionals 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 or confirmed

Adjunctive Treatments

Other medications are useful when indicated in addition to oxygen, sublingual or translingual nitroglycerin, aspirin, morphine, and fibrinolytic therapy.[7] These include

- Anticoagulants (eg, unfractionated heparin, bivalirudin, enoxaparin, fondaparinux)
- P2Y12 inhibitors (prasugrel and ticagrelor)

    - –Prasugrel is a thienopyridine that requires liver biotransformation into active metabolites. Ticagrelor does not require liver biotransformation and is a reversible P2Y12 inhibitor. The timing of administration of P2Y12 inhibitors should be at the discretion of local site practices.
- IV nitroglycerin
- β-blockers
- Glycoprotein IIb/IIIa inhibitors
- Angiotensin-converting enzyme inhibitors
- High-intensity statin therapy

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, enoxaparin, fondaparinux, P2Y12 inhibitors, β-blockers, glycoprotein IIb/IIIa inhibitors, angiotensin-converting enzyme inhibitor, and high-intensity statin therapy. 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 medications, 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. Health care professionals 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 Handbook of Emergency Cardiovascular Care for Health Care Professionals for weight-based dosing guidelines, intervals of administration, and adjustment of low-molecular-weight heparin in renal function. See the AHA/American College of Cardiology (ACC) 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 for relief of ischemic chest discomfort are as follows:

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

Treatment goals using IV nitroglycerin for improvement in pulmonary edema and hypertension are as follows:

- 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

Stroke is a global health problem. It is the world's second leading cause of death and the third leading cause of death and disability combined.[10,11] *Stroke* is a general term that refers to an acute neurologic impairment that follows either an interruption in blood supply to a specific area of the brain or a hemorrhage (bleeding) directly into the brain itself. Although expeditious stroke care is important for all patients, this section emphasizes diagnosis and treatment of the most common type of stroke—AIS. Time is brain, so communities should develop EMS and hospital programs that respond quickly to stroke. Such programs focus on the Stroke Chain of Survival (Figure 5), considering

- Rapid recognition of and reaction to stroke warning signs and symptoms
- Rapid use of 911 and telecommunicators
- Rapid emergency responder recognition of stroke, triage, transport, and prehospital notification to the receiving ED
- Rapid diagnosis and treatment in the ED

The goal of stroke care is to minimize brain injury and maximize the patient's recovery. Early recognition of AIS is critically important because time from symptom onset to reperfusion is key. IV thrombolytic treatment as well as EVT should be provided as early as possible within the parameters of indications and contraindications. Although most strokes occur at home, only half of patients with acute stroke use emergency responders for transport to the hospital, which delays time to evaluation and therapeutic interventions. Patients with stroke 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 emergency responders and treatment, resulting in increased morbidity and mortality.

Medications for Stroke

Medications for stroke include

- Thrombolytic agents (alteplase and tenecteplase)
- Glucose (D_{10}/D_{50})
- Labetalol
- Nicardipine
- Clevidipine
- Aspirin
- Clopidogrel

Pathophysiology

Interruption of blood supply to a section of the brain happens either through a blockage of a vessel in the brain (ischemic; Figure 22) or dissection of a vessel in the brain that leads to blood leaking out into the brain cavity (hemorrhagic; Figure 23). The major types of strokes are

- **Ischemic stroke:** accounts for a majority of all strokes both in the United States and globally (Figures 24 and 25) and is usually caused by an occlusion of an artery to a region of the brain
- **Hemorrhagic stroke:** accounts for a minority of all strokes (Figure 23) and occurs when a blood vessel in the brain suddenly ruptures into the surrounding tissue; thrombolytic therapy is contraindicated in this type of stroke; avoid anticoagulants

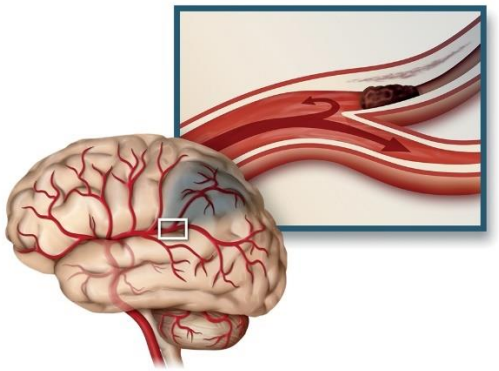

Figure 22. **Ischemic stroke: occlusion in a cerebral artery by a thrombus; area of infarction surrounding immediate site and distal portion of brain tissue after occlusion.**

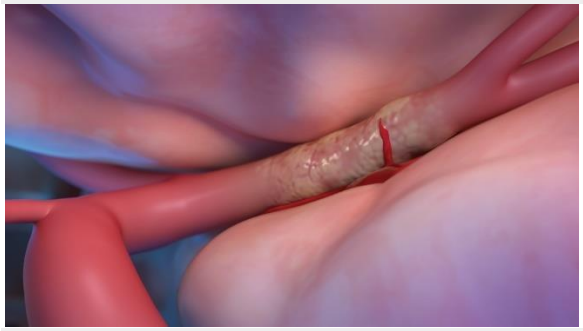

Figure 23. **Hemorrhagic stroke: a blood vessel in the brain suddenly ruptures into the surrounding tissue.**

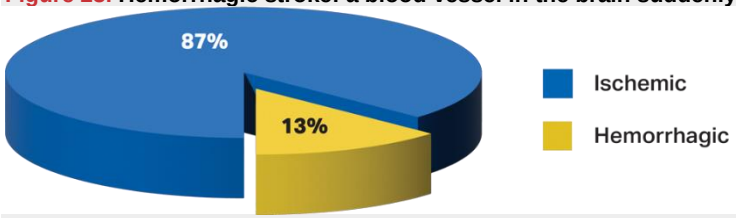

Figure 24. **Types of stroke in the United States: 87% of strokes are ischemic and potentially eligible for reperfusion therapy if patients otherwise qualify[13]; 13% of strokes are hemorrhagic, and the majority of these (10%) are intracerebral.**

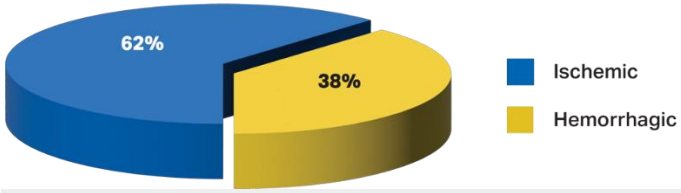

Figure 25. Types of stroke globally: 62% of strokes are ischemic and potentially eligible for reperfusion therapy if patients otherwise qualify; 38% of strokes are hemorrhagic.

The 2 common cerebral small vessel pathologies that account for the overwhelming majority of primary intracerebral hemorrhage are arteriolosclerosis and cerebral amyloid angiopathy.[12] Arteriolosclerosis is detected as concentric hyalinized vascular wall thickening most often in the penetrating arterioles of the basal ganglia, thalamus, brainstem, and deep cerebellar nuclei (collectively referred to as *deep territories*).[12]

Managing Stroke

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 emergency responders by phoning 911
- **D**elivery: rapid stroke identification, management, triage, transport, and prehospital notification to the hospital by emergency responders
- **D**oor: 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 thrombolytic and/or EVT if eligible
- **D**isposition: rapid admission to the stroke unit or critical care unit or emergency interfacility transfer for EVT

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 (Figure 26) reviews the critical in-hospital time periods for patient assessment and treatment:

1. Immediate general and neurologic assessment by the ED or stroke team, emergency medicine physician, or another expert, ideally upon arrival and within 10 minutes after arrival; activate stroke team upon notification by emergency responders; prepare for emergency computed tomography (CT) scan or magnetic resonance imaging (MRI) of brain upon arrival; stroke team meets emergency responders upon 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 the National Institutes of Health Stroke Scale or Canadian Neurological Scale (Step 3)
2. Neurologic assessment by the stroke team or designee and noncontrast CT (NCCT) scan or MRI performed within 20 minutes after arrival at hospital (ideally emergency responders take the patient directly to CT/MRI suite from the field) (Step 3)
3. Interpretation of the NCCT/MRI within 45 minutes after ED/brain imaging suite arrival (Step 4)
4. Initiation of thrombolytic therapy in appropriate patients (those without contraindications) within 60 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)

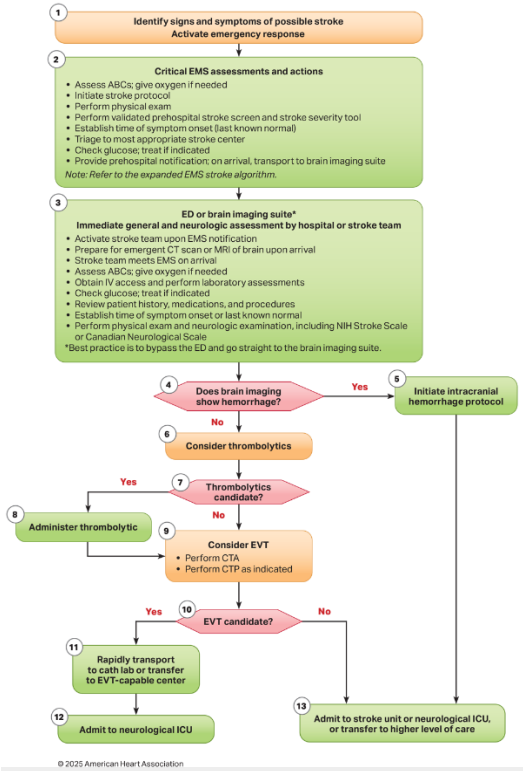

Figure 26. Adult Suspected Stroke Algorithm.

Abbreviations: CTA, computed tomography angiography; CTP, computed tomography perfusion; NIH, National Institutes of Health.

Critical Time Periods

Patients with AIS 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 begins with the onset of symptoms. Critical time periods from hospital arrival are summarized here and represent maximum goal 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 thrombolytic therapy, timed from ED/brain imaging suite arrival: **within 60 minutes**

- Administration of thrombolytic 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**

Application of the Adult Suspected Stroke Algorithm

The Adult Suspected Stroke Algorithm (Figure 26) emphasizes important elements of out-of-hospital and in-hospital care for patients with possible stroke. In addition, the EMS Acute Stroke Routing Algorithm (Figure 27) 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 patient with stroke upon arrival.

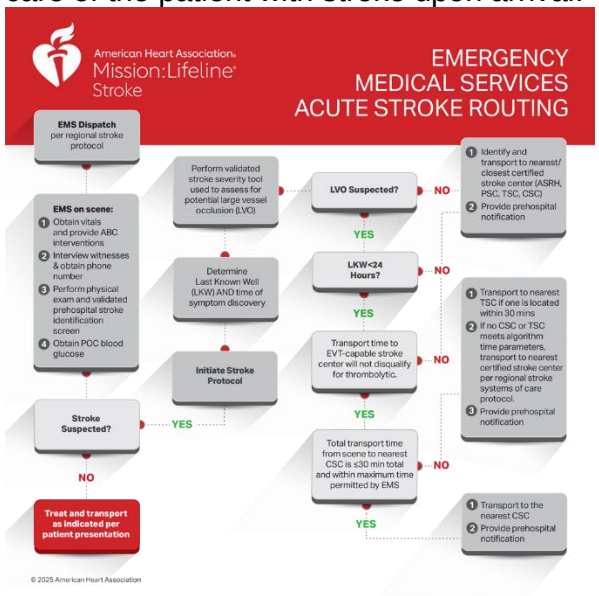

Figure 27. The EMS Acute Stroke Routing Algorithm.

Abbreviations: ASRH, Acute Stroke Ready Hospital; CSC, Comprehensive Stroke Center; MRA, magnetic resonance angiography; MRP, magnetic resonance perfusion; POC, point-of-care; PSC, Primary Stroke Center; TSC, Thrombectomy-Capable Stroke Center.

The stroke algorithm outlines identification and treatment of all strokes. Major identified parts of the algorithm include

- Identification of signs and symptoms of possible stroke and activation of emergency response (Step 1)
- Critical emergency responder 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?
- Thrombolytic candidate? (Thrombolytic therapy risk stratification of candidate) (Step 7)
- Considering EVT and the 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 patients with stroke are driven to the ED by family or friends. Thus, patients with stroke and their families must be educated on potential signs or symptoms of stroke and the need to phone 911 and activate emergency responders as soon as they detect a possible stroke.

Emergency responders provide the safest and most efficient method of emergency transport to the most appropriate stroke hospital. The advantages of EMS system activation and emergency responder transport include the following:

- EMS telecommunicators play a critical role in timely treatment of potential stroke by
    - –Identifying patients with possible stroke

    - –Providing high-priority dispatch
    - –Instructing lay rescuers in lifesaving CPR skills or other supportive care if needed while emergency responders are on the way
- Emergency responders can assess ABCs and give oxygen as needed.
- Emergency responders can initiate stroke protocol, perform a physical exam, establish time of symptom onset (last known normal), and check glucose and treat if indicated.
- Emergency responders 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.
- Emergency responders should provide prehospital notification, enabling the hospital to prepare to evaluate and manage the patient more efficiently, and upon arrival, transport to the brain imaging suite.

Provide Critical Emergency Responder Assessments and Actions

Emergency responders must minimize the interval between the onset of symptoms and patient arrival in the ED or brain imaging suite. Specific stroke therapy can be provided only in the appropriate receiving hospital, 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 or brain imaging suite.

Critical Emergency Responder Assessments and Actions

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

- Assess ABCs and give oxygen if needed to patients with hypoxemic stroke (ie, whose saturation is 94% or less) or to those patients with unknown oxygen saturation.
- Initiate stroke protocol.
- Perform physical exam.
- Perform a neurologic assessment using a validated tool. Perform a rapid prehospital stroke screen (eg, Cincinnati Prehospital Stroke Scale [CPSS], Los Angeles Prehospital Stroke Screen) and, if needed, a stroke severity assessment for possible LVO (eg, Los Angeles Motor Scale, 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 the 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 upon arrival, transport to the brain imaging suite.
- 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 in- and out-of-hospital health care professionals should provide supplemental oxygen to patients with hypoxemic stroke (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 emergency responders be trained to recognize stroke by using a validated, abbreviated out-of-hospital neurologic evaluation tool such as the CPSS (Table 4) or the Los Angeles Prehospital Stroke Screen.

Table 4. The CPSS

Test	Findings
Facial droop: Have the patient show teeth or smile (Figure 28).	**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: Have the patient close their eyes and extend both arms straight out, with palms up, for 10 seconds (Figure 29).	**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:** The patient uses correct words with no slurring. **Abnormal:** The 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%.	

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. doi:10.1016/s0196-0644(99)70299-4. Copyright 1999, with permission from Elsevier.

Figure 28. Facial droop.

Figure 29. One-sided motor weakness (left 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, emergency responders 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 emergency responders.

If emergency responders determine that a stroke is likely based on a stroke screen, an additional stroke severity assessment should also be obtained, such as FAST-ED or Los Angeles Motor Scale. The results of the stroke severity assessment will help direct emergency responders to an appropriate ED that can treat an LVO stroke.

Field Emergency Stroke for Emergency Destination

The FAST-ED identifies a possible LVO on the basis of 5 physical findings:

- Facial palsy
- Arm drift
- Speech changes
- Eye deviation
- Denial/neglect

Possible scores of the FAST-ED range from 0 to 9. A score of 4 or greater is the suggested cutoff for suspected LVO stroke triage. This score should be communicated by emergency responders to the ED as a part of prearrival notification.

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
- 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 Stroke Scale (NIHSS)[14*]
- 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
- Rapid Arterial Occlusion Evaluation Score (RACE)
- Three-Item Stroke Scale (3ISS)

*Recommended tool for in-hospital use

Stroke Centers and Stroke Units

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

As stated in the "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."[14] This recommendation is supported by data that demonstrate that the development of stroke centers improves patient care and clinical outcomes. Table 5 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 5. Levels and Capabilities of Hospital Stroke Designation

Hospital attributes	ASRH	PSC	TSC	CSC
Location	**Likely rural**	**Likely urban or suburban**	**Likely urban**	**Likely urban**
Stroke team accessible/available 24 hours/day 7 days/week	Yes	Yes	Yes	Yes
NCCT available 24 hours/day 7 days/week	Yes	Yes	Yes	Yes
Advanced imaging (CT angiography, CT perfusion, MRI, MRA, MRP) available 24 hours/day 7 days/week	No	Yes	Yes	Yes
IV thrombolytic capable	Yes	Yes	Yes	Yes
Thrombectomy capable	No	Possibly	Yes	Yes
Diagnoses stroke pathogenesis and manages poststroke complications	Unlikely	Yes	Yes	Yes
Admits hemorrhagic stroke	No	Possibly	Possibly	Yes
Clips or coils ruptured aneurysms	No	Possibly	Possibly	Yes
Dedicated stroke unit	No	Yes	Yes	Yes

Dedicated neurocritical care unit or ICU	No	Possibly	Possibly	Yes

Acute Stroke Ready Hospital

Acute Stroke Ready Hospitals typically serve rural and under-resourced areas. Emergency 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 patients with stroke, provide alteplase therapy if indicated, and admit patients to a dedicated stroke unit. Roughly half of all patients with stroke 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. **Emergency responder prenotification:** Emergency responders should provide early prenotification to the receiving ED 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, NIHSS, 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 ED prenotification from emergency responders of a patient with stroke or the patient with stroke 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:** AIS care requires an accurate, timely, coordinated, and systematic evaluation of the patient. A universal clock that is visible to the health care professional is an enabling tool for improving the quality of care.
6. **Ensure transfer directly to CT or MRI scanner:** Guided by prespecified protocols, emergency responders can transport eligible patients with stroke, 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 thrombolytic 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 systemic anticoagulation 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 thrombolytic in advance:** Mix the medication and set up the bolus dose and 1-hour infusion pump as soon as a patient is recognized as a possible thrombolytic candidate, even before brain imaging.
10. **Provide rapid access and administration of an IV thrombolytic:** Once eligibility has been determined and intracranial hemorrhage has been excluded, an IV thrombolytic 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 thrombolytic 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.

Target: Stroke III Best-Practice Strategies (Door-to-Needle and Door-to-Device Times)

Tables 6 through 9 include various door-to-needle and door-to-device time goals.

Table 6. The 30-Minute Door-to-Needle Time-Interval Goals

Action	Time, min
Door to physician	≤2½
Door to stroke team	≤5
Door to CT/MRI initiation	≤15
Door to CT/MRI interpretation	≤25
Door-to-needle time	≤30

Table 7. The 45-Minute Door-to-Needle Goal Time-Interval Goals

Action	Time, min
Door to physician	≤5

Door to stroke team	≤10
Door to CT/MRI initiation	≤20
Door to CT/MRI interpretation	≤35
Door-to-needle time	≤45

Table 8. The 60-Minute Door-to-Needle Goal Time-Interval Goals

Action	Time, min
Door to physician	≤10
Door to stroke team	≤15
Door to CT/MRI initiation	≤25
Door to CT/MRI interpretation	≤45
Door-to-needle time	≤60

Table 9. The 90-Minute Door-to-Device Goal Time-Interval Goals

Action	Time, min
Door to physician	≤5
Door to stroke team	≤10
Door to CT/MRI initiation	≤20
Door to CT/MRI interpretation	≤35
Door to neurointerventional team activation	≤40

Door-to-needle time	≤45
Door to patient arrival in cath lab/imaging suite	≤60
Door to puncture	≤75
Door to device	≤90

Immediate General and Neurologic Assessment

Table 10 shows 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 10. Critical Actions in the Evaluation of Potential Acute Stroke

Step	Action
Activate the stroke team.	Activate stroke team upon notification by emergency responders.
Obtain a CT brain scan (NCCT) or MRI scan.	Prepare for emergency CT scan or MRI scan of brain upon arrival. Upon prehospital notification, order an emergency 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 health care professional.
Meet the stroke team.	Stroke team meets emergency responders upon 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 (if <95%).
Obtain IV access.	Obtain IV access and perform laboratory assessments. Do not let this delay obtaining a CT brain scan or administering alteplase.
Check glucose.	Check glucose and promptly treat hypoglycemia (<60 mg/dL).
Obtain patient 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 NIHSS 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 AIS 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 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 ED 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 NIHSS 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 emergency responders, witnesses, and family members.

Conduct Neurologic Examination

Assess the patient's neurologic deficits by using an established stroke scale, preferably the NIHSS or Canadian Neurological Scale.

The NIHSS uses 15 items to assess and quantify neurologic deficits of a patient with stroke. 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 thrombolytic 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.[12]

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

Hemorrhage is not present (Steps 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 thrombolytic 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.

Figure 30 shows 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.

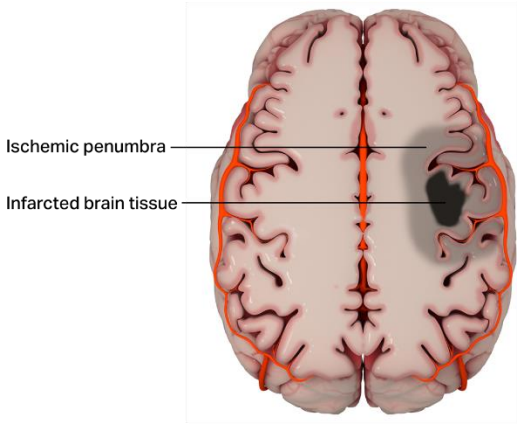

Figure 30. Occlusion in a cerebral artery by a thrombus; area of ischemic penumbra (ischemic, but not yet infarcted [dead] brain tissue) surrounding areas.

Thrombolytic Therapy

Studies have demonstrated that there is a higher likelihood of good to excellent functional outcome when a thrombolytic medication is given to adults with AIS 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.

The AHA and the American Stroke Association recommend giving IV thrombolytics to patients with AIS 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 Thrombolytic Therapy

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

- If the CT/MRI scan shows no hemorrhage, the probability of AIS remains. *Review inclusion and exclusion criteria for IV thrombolytic therapy (Table 11) and repeat the neurologic exam (NIHSS or Canadian Neurological Scale).*
- If the patient's neurologic function is rapidly improving to normal, thrombolytics may be unnecessary.

Table 11. Inclusion and Exclusion Characteristics of Patients With Ischemic Stroke Who Could Be Treated With Thrombolytics Within 3 Hours After Symptom Onset and Extended Window for Select Patients 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) or IV tenecteplase (0.25 mg/kg, maximum dose 25 mg over 5-10 seconds) is recommended for selected patients who may be treated within 3 hours after 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 after 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 after 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)
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) or IV tenecteplase (0.25 mg/kg, maximum dose 25 mg over 5-10 seconds) administered within 4.5 hours after 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 diffusion-weighted MRI lesion smaller than one third of the MCA territory and no visible signal change on fluid-attenuated inversion recovery. (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 to 4.5 hours after 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)¶
Intracranial hemorrhage	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 or intraspinal surgery within 3 months	For patients with AIS and a history of intracranial or 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 a subarachnoid hemorrhage.‡ (COR 3: Harm; LOE C-EO)‖¶
GI malignancy or GI bleed within 21 days	Patients with a structural GI malignancy or recent GI bleeding event within 21 days from 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 anticoagulants 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)§

Table 11A. Thrombolytics 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 minutes with initial 10% of dose given as bolus over 1 minute) or IV tenecteplase (0.25 mg/kg, maximum dose 25 mg over 5-10 seconds) is also recommended for selected patients who can be treated within 3 to 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: aPTT, activated partial thromboplastin time; COR, Class of Recommendation; INR, international normalized ratio; LMWH, low-molecular-weight heparin; LOE, Level of Evidence; MCA, middle cerebral artery; OAC, oral anticoagulant; PT, prothromboplastin time.

*The relative contraindications are abbreviated.

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

‖Level of Evidence amended to conform with the ACC/AHA 2015 recommendation classification system.

¶Class of Recommendation amended to conform with the ACC/AHA 2015 recommendation classification system.

Modified from Table 8 (refer to Table 8 for a full list of specific considerations) in Powers et al.[14]

Unless otherwise specified, these eligibility recommendations apply to patients who can be treated within 0 to 4.5 hours after 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 **https://www.accessdata.fda.gov/drugsatfda_docs/bla/2015/103172Orig1s5203.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[14] and IV tenecteplase in AIS.

Potential Adverse Effects

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

- Confirm that no exclusion criteria are present (Table 11).
- Consider risk and benefits.
- Be prepared to monitor and treat any potential complications.
- The major complication of IV alteplase or tenecteplase or 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 Thrombolytic Therapy

If the patient remains a candidate for thrombolytic 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 thrombolytic therapy, give the patient thrombolytics. Begin your institution's stroke thrombolytic protocol, often called a *postthrombolytic pathway of care*.

Thrombolytics are considered the standard of care for eligible patients with AIS. Because of this treatment's proven benefit and the need to expedite it, health care professionals are justified to proceed with IV thrombolysis in an otherwise eligible adult patient with a disabling AIS 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 thrombolytics, typically until a follow-up CT scan at 24 hours shows no intracranial hemorrhage.

Extended IV Alteplase/Tenecteplase Window: 3 to 4.5 Hours

Treatment of carefully selected patients with AIS with IV alteplase/tenecteplase 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.[14]

The use of IV alteplase/tenecteplase within the 3- to 4.5-hour window has not been approved by the US Food and Drug Administration, although it is recommended by the 2019 AHA AIS guidelines for those who meet the ECASS-3 eligibility criteria (Table 11).

Endovascular Therapy

Substantial high-quality research on the clinical efficacy of endovascular treatments of AIS 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 AIS due to an LVO.[15]

As with thrombolytic 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 AIS.

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
- NIHSS score of 6 or greater
- ASPECTS (Alberta Stroke Program Early CT Score) 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 AIS 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 AIS 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 Thrombolysis

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 thrombolytic medications that are not available. A clinically beneficial dose of intra-arterial alteplase is not established, and alteplase does not have US Food and Drug Administration 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 AIS 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 BP.
- Monitor temperature.
- Perform dysphagia screening.
- Monitor for complications of stroke and thrombolytic therapy.
- Transfer to a higher level of care (EVT, neurologic ICU) if indicated.

Begin Stroke Pathway

Admit patients to a stroke unit (if available) for careful observation, including monitoring of BP and neurologic status. If neurologic status worsens, order an emergency 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 outcomes in patients with AIS. Although there is no direct evidence that active glucose control improves clinical outcome, there 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 AIS when the serum glucose level is greater than 180 mg/dL.

Monitor for Complications of Stroke and Thrombolytic 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 BP with a concurrent decrease in heart rate. Continue to control BP to reduce the potential risk of bleeding.

Hypertension Management in Thrombolysis Candidates

Although management of hypertension in the patient with stroke is controversial, patients who are candidates for thrombolytic therapy should have their BP controlled to lower the risk of intracerebral hemorrhage after administration of alteplase. General guidelines for the management of hypertension are outlined in Table 12.

Table 12. Options to Treat Arterial Hypertension in Patients With AIS Who Are Candidates for Emergency Reperfusion Therapy[14]

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/tenecteplase.	
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 SBP >180-230 mm Hg or DBP >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 DBP >140 mm Hg, consider IV sodium nitroprusside.	

Abbreviation: DBP, diastolic blood pressure.

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.[16]

If a patient is eligible for thrombolytic therapy, BP 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/tenecteplase 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 BP 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 AIS is not well established (Class 2b; Level of Evidence C).[17] Patients who have malignant hypertension or other medical indications for aggressive treatment of BP should be treated accordingly.[17]

In addition to hypertension, hypotension is a particular challenge for patients given sedation for interventional therapy. Treatment is not very different from other hypotension; treat the underlying cause:

- Give fluids if the patient is hypovolemic.
- Give pressors if the patient is vasodilated.
- Give inotropes if the patient has poor cardiac function.
- Give vagolytics or chronotropes if the patient has symptomatic bradycardia. Avoid atropine in patients with acute brain injury.

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. *Patients with symptomatic bradycardia who are considered unstable and need immediate treatment present with hypotension, acute altered mental status, ischemic chest discomfort, or acute heart failure.*

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 AV block
- Using atropine as the medication intervention of first choice

- Deciding when to start dopamine, epinephrine, or other rate-accelerating adrenergic agonists
- Deciding when to initiate transcutaneous pacing (TCP)
- Knowing when to consider expert consultation or transvenous pacing

Rhythms for Bradycardia

- Sinus bradycardia (Figure 31A)
- 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 31B). 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 31C):
        - 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 (Figure 31D)

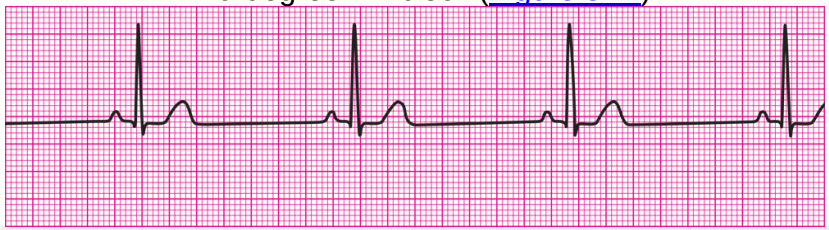

Figure 31A. Examples of AV block. A, Sinus bradycardia.

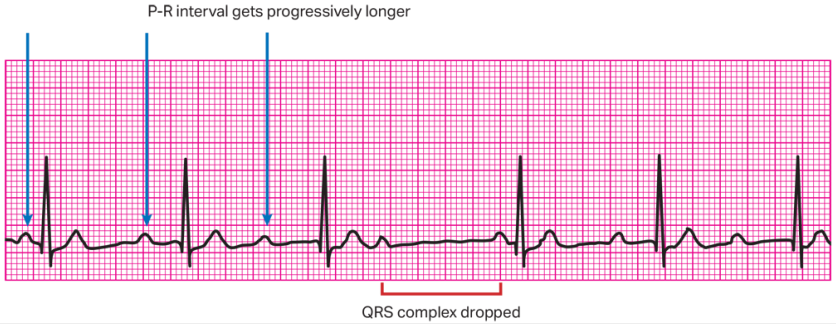

Figure 31B. **Second-degree AV block type I.**

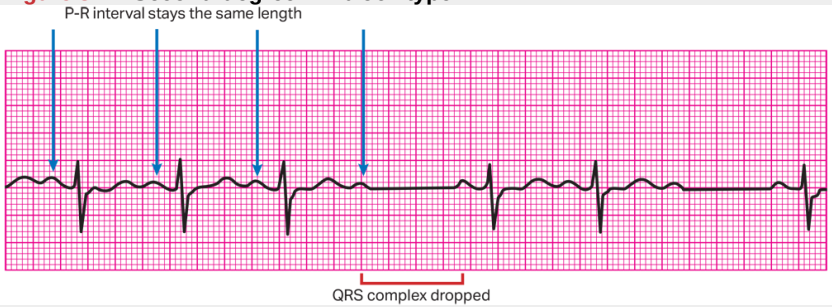

Figure 31C. **Second-degree AV block type II.**

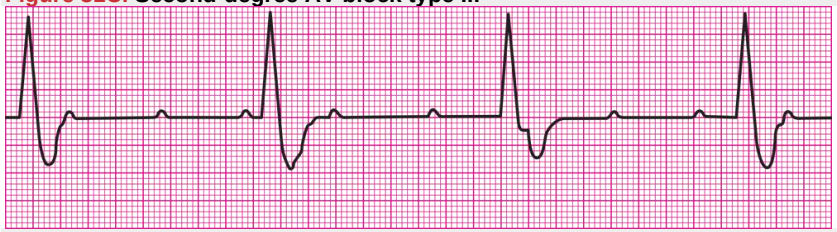

Figure 31D. **Third-degree AV block (complete AV block).**

You should know the major AV blocks because important treatment decisions are based on the type of block (Figure 31). 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 an unstable bradycardia due to AV block is a primary goal, and recognizing the type of AV block is secondary.

Medications and Treatment for Bradycardia

Medications and procedures for bradycardia include

- Atropine
- TCP
- 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.[18]

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 cardiopulmonary compromise with signs and symptoms that include

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

Managing Bradycardia: Adult Bradycardia With a Pulse Algorithm

The Adult Bradycardia With a Pulse Algorithm (Figure 32) 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) and determining if there is cardiopulmonary compromise. First steps include the components of the BLS assessment and the primary assessment.

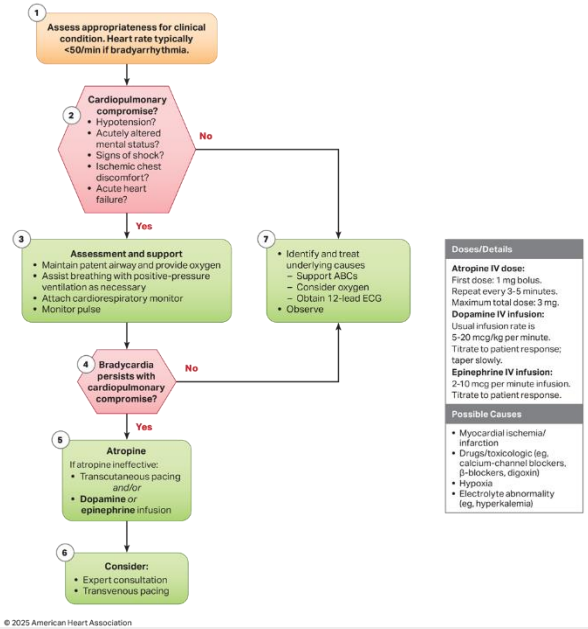

Figure 32. Adult Bradycardia With a Pulse Algorithm.

Assess and support (Step 3):

- Maintain patent airway and provide oxygen.
- Assist breathing with positive-pressure ventilation as necessary.
- Attach cardiorespiratory monitor.
- Monitor pulse.

If there is no cardiopulmonary compromise

- Identify and treat underlying causes:
    - –Support ABCs.
    - –Consider oxygen.
    - –Consider 12-lead ECG.
- Observe.

In the differential diagnosis, the primary decision point in the algorithm is to determine if the bradycardia persists with cardiopulmonary compromise, indicating poor perfusion (Step 4). If there are no signs of cardiopulmonary compromise, identify and treat underlying causes and observe (Step 7). If there are persistent signs of cardiopulmonary compromise, administer

atropine (Step 5). If atropine is ineffective, prepare for TCP and/or consider dopamine or epinephrine (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, follow the Adult Cardiac Arrest Algorithm in Part 3.

Applying the Adult Bradycardia Algorithm

Conduct appropriate assessment and interventions as outlined in the Adult Bradycardia Algorithm while searching for and treating possible contributing factors.

Identify Bradycardia

Assess the heart rate:

- Identify if the patient is bradycardic with a pulse, typically a heart rate less than 50/min.
- Evaluate for cardiopulmonary comprise (eg, hypotension, acutely altered mental status, signs of shock, ischemic chest discomfort, acute heart failure).

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 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 there is cardiopulmonary compromise.

- If the patient has **no cardiopulmonary compromise**, identify and treat underlying causes and observe (Step 2).
- If the patient has **persistent** cardiopulmonary compromise, proceed to Step 5.

Treatment Sequence Summary

If bradycardia persists with cardiopulmonary compromise, treat as follows:

- Give atropine as first-line treatment (except for heart transplant patients): 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 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 intranodal 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 medication for acute unstable 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 (except for heart transplant patients) every 3 to 5 minutes (maximum total dose of 3 mg IV).

Caution

Low-Dose Atropine

- 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 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.

Transcutaneous Pacing

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.

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)
    - –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.
- Assess a central pulse (eg, carotid, femoral) to confirm mechanical capture; be aware that electrical stimulation can cause muscular jerking that may mimic the carotid pulse.

Treatment Sequence: TCP and Adrenergic Agonists

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 or 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 33). 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 medications may further decrease BP and affect the patient's mental status. Try to identify and correct the cause of the bradycardia.

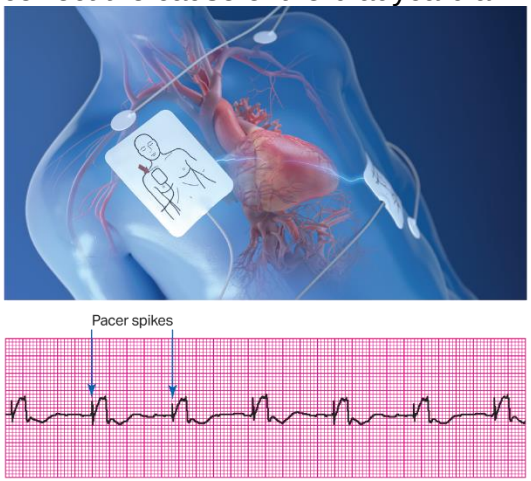

Figure 33. Transcutaneous pacing.

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 emergency 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, prepare for possible transvenous pacing, and request expert consultation.

Assessment of a pulse to confirm mechanical capture can be challenging during TCP because electrical stimulation causes generalized muscular contraction (jerking) that may mimic the carotid pulse. A femoral pulse is occasionally more reliably palpated than a carotid pulse, given the femoral artery's greater distance from the site of pacing and less surrounding muscle.

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.

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 medications are not immediately available. Evaluate the need for sedation in light of the patient's condition and need for immediate pacing. Obtain expert consultation and consider transvenous pacing.

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.

Treatment Sequence: Dopamine, Epinephrine

Although β-adrenergic agonists with rate-accelerating effects are not first-line agents for treating unstable 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, health care professionals must assess the patient's intravascular volume status and avoid hypovolemia when using these medications. Dobutamine and isoproterenol (a β-adrenergic agonist) can improve heart rate; however, these medications might induce coronary ischemia and should be used with caution in those with coronary disease and avoided in those with AMI or unstable angina.

Either epinephrine infusions or dopamine infusions may be used for patients with unstable 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; or 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.

Assess Response to Treatment

Signs of hemodynamic impairment related to bradycardia 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.

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 medications. 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

A β-adrenergic infusion (eg, 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.

Alternative medications 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 atropine is ineffective.

Caution

Atropine Use in Patients With a Heart Transplant

The "2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay" states that unstable bradycardia patients who have undergone a heart transplant should not receive atropine for treatment because of the increased likelihood of paradoxical heart block or, less commonly, sinus arrest.[16] These guidelines classify atropine for unstable heart transplant patients as a Class III: Harm.

- Avoid using atropine for unstable bradycardia in heart transplant patients.
- Treat with pacing and/or dopamine or epinephrine.

Tachycardia: Unstable and Stable

Overview

Tachycardia is generally defined as any rhythm disorder with a heart rate greater than 100/min. **But for assessment and management of a patient with unstable tachycardia**, it is typically defined as having **a heart rate greater than 150/min** or having signs of cardiopulmonary compromise (eg, hypotension, altered mental status, signs of shock, ischemic chest discomfort, acute heart failure).

Managing tachycardia requires

- Differentiating between signs and symptoms caused by the fast rate vs those that are unrelated
- Correctly diagnosing the presence and type of narrow-complex tachycardia or VT to guide appropriate therapies
- Using synchronized cardioversion for unstable tachycardia
- Knowing when to seek expert consultation about complicated rhythm interpretation, medications, or management decisions

Rhythms for Unstable Tachycardia

- Sinus tachycardia (Figure 34A)
- Atrial fibrillation (Figure 34B)
- Atrial flutter (Figure 34C)
- Narrow-complex tachycardia (Figure 34D)
- Monomorphic VT (Figure 34E)
- Polymorphic VT (Figure 34F)
- Wide-complex tachycardia of uncertain type

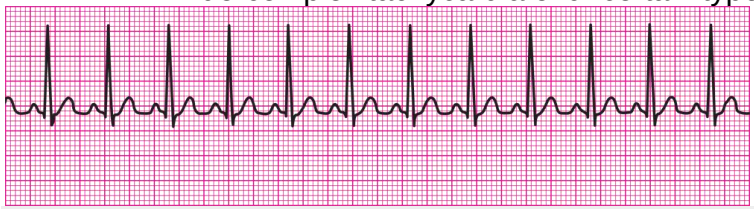

Figure 34A. Examples of tachycardias. A, Sinus tachycardia.

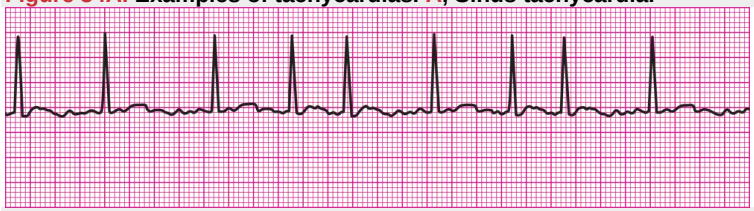

Figure 34B. Atrial fibrillation.

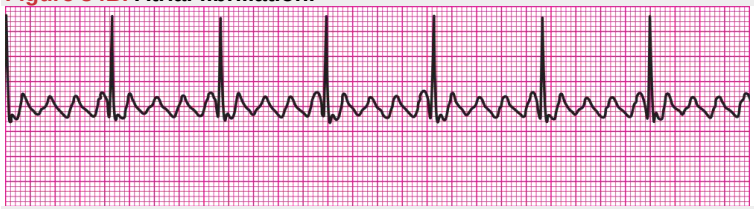

Figure 34C. Atrial flutter.

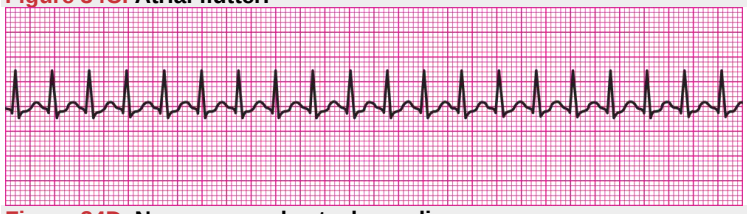

Figure 34D. Narrow-complex tachycardia.

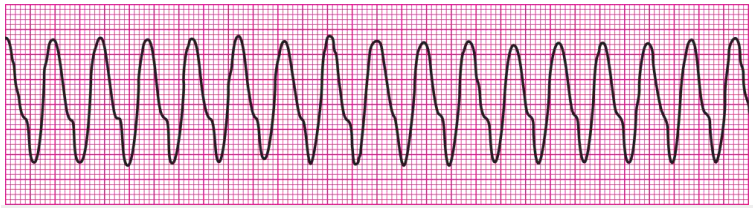

Figure 34E. **Monomorphic VT.**

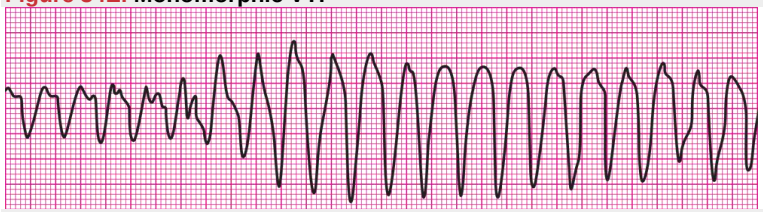

Figure 34F. **Polymorphic VT.**

Medications for Unstable Tachycardia

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

Unstable Tachycardia

Tachycardia 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 never indicated for sinus tachycardia.

Definitions

Definitions used in this case are as follows:

- *Tachycardia*: defined as an arrhythmia with a heart rate typically 100/min or greater
- *Unstable tachycardia*: signs and symptoms of cardiopulmonary compromise
    - –Hypotension

    - –Altered mental status
    - –Signs of shock
    - –Ischemic chest discomfort
    - –Acute heart failure
- 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

Rapid Recognition of Unstable Tachycardia

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 (eg, the pain and distress of an AMI) or vice versa.

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, pain, 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 Tachyarrhythmia With a Pulse Algorithm

The Adult Tachyarrhythmia With a Pulse Algorithm (Figure 35) simplifies initial management of tachycardia. The presence or absence of pulses is considered the key to managing patients with any tachycardia. If a pVT is present, manage the patient according to the Adult Cardiac Arrest Algorithm (Figure 46). 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.

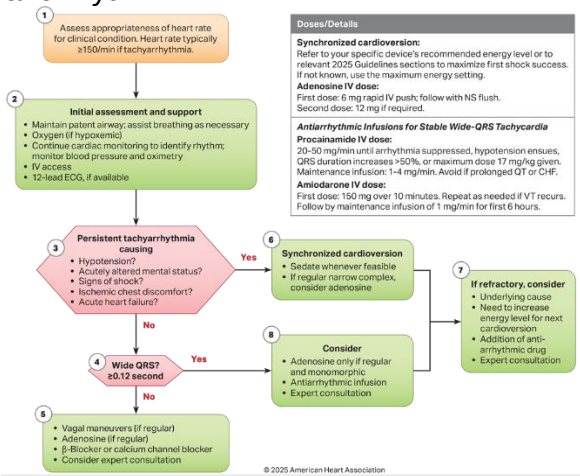

Figure 35. Adult Tachyarrhythmia With a Pulse Algorithm.

Abbreviations: CHF, congestive heart failure; NS, normal saline.

To manage unstable tachycardia, ACLS professionals should perform synchronized cardioversion and sedate whenever feasible and, if regular narrow complex, administer adenosine 6 mg IV (follow with saline flush). If these interventions are not successful and if the tachycardia is refractory, clinicians should look for any underlying causes and consider the need to increase the energy level for the next cardioversion and add antiarrhythmic medications. Health care professionals should also obtain expert consultation. Actions in the steps require advanced knowledge of ECG rhythm interpretation and antiarrhythmic therapy.

These key questions in the Adult Tachyarrhythmia 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 Tachyarrhythmia 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 Tachyarrhythmia With a Pulse Algorithm (Figure 35) to evaluate and manage the patient.

Assess Clinical Condition

Use the BLS, primary, and secondary assessments to guide your approach:

- Assess appropriateness of heart rate for clinical condition (Step 1).
    - –Determine if the patient is having an appropriate response to physiological stress (fever, dehydration).

Initial Assessment and Support

Initial assessment and support (Step 2):

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

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

Determine if the tachyarrhythmia is causing (Step 3)

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

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, so do not delay immediate cardioversion to acquire the 12-lead ECG if the patient is unstable.

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

Assess the patient and determine if instability is related to the tachycardia (Step 3).

Unstable

If the tachycardia is causing the patient to demonstrate rate-related cardiovascular compromise with serious signs and symptoms, proceed to immediate synchronized cardioversion and sedate whenever feasible (Step 6). If there is a regular narrow complex, consider adenosine.

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, which may include 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.

Treatment Based on Tachycardic Rhythm

You may not always be able to distinguish between narrow-complex 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, follow the Adult Cardiac Arrest Algorithm (Figure 46).

If the patient has a wide-complex tachycardia (0.12 second or greater) and is unstable, assume it is VT until proven otherwise. The amount of energy required for cardioversion of VT will depend on the specific device's recommended energy level or to relevant guideline sections to maximize first shock success. If not known, use the maximum energy setting.

- If the patient is unstable but has a pulse with regular uniform wide-complex VT (monomorphic VT), treat with synchronized cardioversion (Step 6). Refer to your specific device's recommended energy level or, if unknown, use the maximum energy setting for your device. 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

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

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

If cardiac arrest develops and the patient is pulseless, follow the Adult Cardiac Arrest Algorithm (Figure 46).

Cardioversion

You must know when cardioversion is indicated and what type of shock to administer (Figure 36). Before cardioversion, establish IV access and sedate the responsive patient, if possible, but do not delay cardioversion in unstable or deteriorating patients.

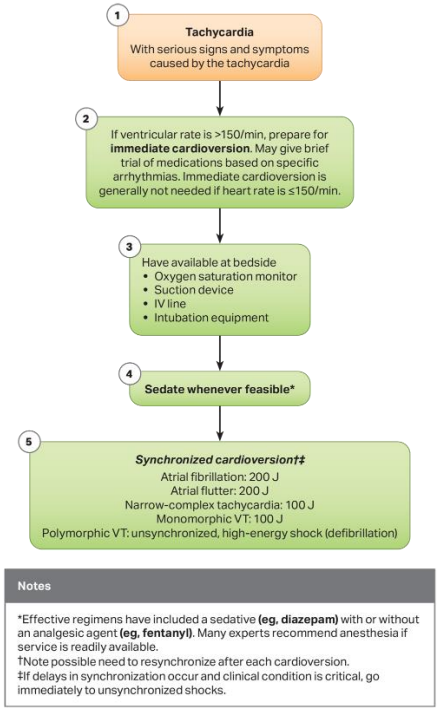

Figure 36. Electrical Cardioversion Algorithm.

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. The term *unsynchronized shock* refers to an electrical shock that 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, when 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.
- 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 narrow-complex tachycardia
- Unstable atrial fibrillation
- Unstable atrial flutter
- Unstable regular monomorphic VT

Unsynchronized high-energy shocks are recommended

- For a patient with no pulse and VF or 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, refer to your specific device's recommended energy level or to relevant Guideline sections to maximize the first shock success. If not known, use the maximum energy setting.

Synchronized Cardioversion

Synchronized cardioversion is the treatment of choice when a patient has a symptomatic (unstable) reentry narrow-complex tachycardia 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 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.
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 specific device's recommended energy level or, if unknown, use the maximum energy setting for your device.
8. Announce to team members: "Charging defibrillator!"
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 36 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 health care professional 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) with or without an analgesic agent (eg, fentanyl). 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 cardiopulmonary compromise, proceed to Step 6. You'll have time to obtain a 12-lead ECG, evaluate the rhythm, and determine if the width of the QRS is 0.12 second or greater. In this case, consider adenosine only if the rhythm is regular and monomorphic, consider antiarrhythmic infusion, and seek expert consultation. If the rhythm is refractory, consider the underlying cause, the need to increase the energy level for the next cardioversion, addition of antiarrhythmic medications, and expert consultation.

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 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
    - –Narrow-complex tachycardia with aberrancy

- Regular or irregular tachycardias

Irregular narrow-complex tachycardias are probably atrial fibrillation.

Medications for Stable Tachycardia

Medications for tachycardia include

- Adenosine 6 mg IV (follow with a rapid saline flush); second dose (if required) 12 mg IV (follow with a rapid saline flush)
- Several analgesic and sedative agents (also used during electrical cardioversion, but those agents are not covered in this course)
- Antiarrhythmic infusion
- β-blocker or calcium channel blocker

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 narrow-complex tachycardia has an abrupt onset and termination.

Note that sinus tachycardia is excluded from the Adult Tachyarrhythmia With a Pulse Algorithm (Figure 35). Sinus tachycardia is caused by external influences on the heart, such as fever, anemia, hypotension, blood loss, or exercise, which are 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.*

Caution

β-Blocker Use in Sinus Tachycardia

- β-blockers may cause clinical deterioration if the cardiac output falls when a sinus 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 = Stroke volume × 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.

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.

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 Tachyarrhythmia With a Pulse Algorithm (Figure 35). 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-blockers, or calcium channel blockers.

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

Applying the Adult Tachyarrhythmia With a Pulse Algorithm to Stable Patients

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 greater. 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 narrow-complex tachycardia with aberrancy.
- An irregular wide-complex tachycardia may be atrial fibrillation with aberrancy, preexcited atrial fibrillation (atrial fibrillation using an accessory pathway for antegrade conduction), polymorphic VT or torsades de pointes. Additional expertise or expert consultation should be considered for these advanced rhythms. In addition, consider adenosine (only if regular and monomorphic) and antiarrhythmic infusion.

If the rhythm is likely VT or narrow-complex tachycardia 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. Additionally, IV antiarrhythmic medications 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.

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. Consider expert consultation.

Treating Tachycardia

You may not always be able to distinguish between narrow-complex (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 (Figure 46).

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

Critical Concepts

Medications 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 medications may cause a paradoxical increase in the ventricular response.

Narrow QRS, Regular Rhythm

The management 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 and adenosine are the preferred initial interventions for terminating narrow-complex tachycardias that are symptomatic (but stable) and narrow complex in origin. These can be followed by β-blockers or calcium channel blockers for acute treatment. Valsalva maneuvers or carotid sinus massage alone will terminate about 25% of narrow-complex tachycardia, and adenosine is required for the remainder.

- If narrow-complex tachycardia 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 narrow-complex tachycardia 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 medication interactions. Adenosine is less effective in patients taking theophylline or caffeine, so larger doses may be required. Reduce the initial dose to 3 mg in patients receiving dipyridamole or carbamazepine. An initial dose of 3 mg is also recommended when adenosine is given to patients with a heart transplant or if the medication is administered by central venous access.

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.[19]

If the rhythm converts with adenosine, it is probably reentry narrow-complex tachycardia. Observe patients for recurrence and treat any recurrence with adenosine or longer-acting AV nodal blocking agents, such as the nondihydropyridine 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 medications for pre-excited atrial fibrillation or flutter because these medications 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.

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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 responder 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 and proficient in their roles
- **Administration:** leadership, measurement, CQI, and number of participating code team members

*High-performing systems target at least 60% but ideally have greater than 80% as a frequent goal.

High-performance teams (Figure 37) 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.

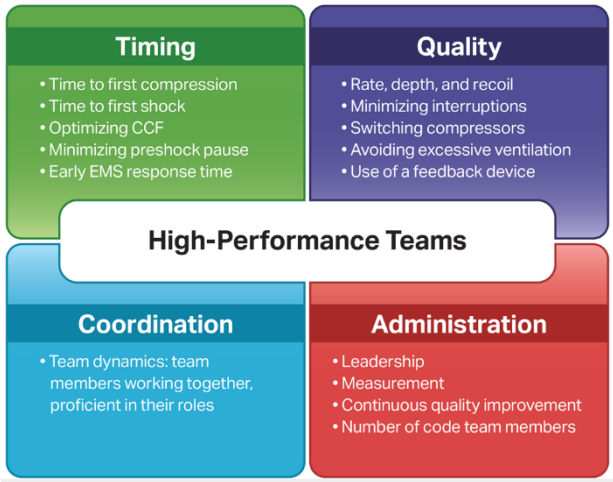


Figure 37. Key areas of focus for high-performance teams to increase survival rates.

Critical Concepts
Ways to Increase CCF

Whether you are a team member or the Team Leader during a resuscitation attempt, you should understand how a high-performance team can optimize CPR performance and integrate ALS interventions during a cardiac arrest. One key focus is improvement. 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 approaches 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 health care professionals to simultaneously perform a variety of interventions. Although a CPR-trained bystander working alone can begin the resuscitation of a patient within the first moments after collapse, most cardiac arrest events require multiple health care professionals providing high-quality CPR and ALS interventions. 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
- Assumes responsibility for roles not defined
- Plans ahead for next steps
- Makes sure nothing is missing or skipped
- Provides a team summary and requests input or additional expertise

- Avoids fixation errors
- 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 the 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, debrief, 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. Additionally, Team Leaders should also understand how to integrate ALS interventions, such as administration of medications, advanced airway placement, and postresuscitative care.

Whereas members of a high-performance team should focus on their individual tasks, the Team Leader must focus on comprehensive patient care. The Team Leader should have situational awareness that includes thinking ahead and planning for the next steps (eg, calling for the extracorporeal membrane oxygenation [ECMO] team, heart alert or cath lab activation, PE team for specific arrest situations when indicated).

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 include the role of CPR Coach. The CPR Coach coordinates performance of high-quality BLS skills, allowing the Team Leader to focus on all 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 can be a separate role, or the role can be combined with 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 (Figure 38).

- **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." The CPR Coach should adjust the environment to help ensure high-quality CPR. They can lower the bedrails or the bed, get a step stool, or roll the patient to place a backboard and defibrillator pads.
- **Coach to improve the quality of chest compressions and ventilations:** The CPR Coach gives feedback about performance of compression depth, rate, and chest recoil as well as feedback on timing and volume of ventilations. They state 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 that happen when the team defibrillates, switches Compressors, or places an advanced airway.

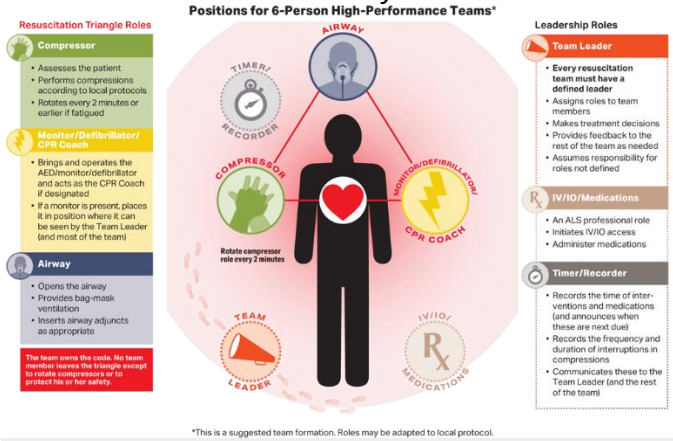


Figure 38. Suggested locations for the Team Leader and team members during case simulations and clinical events.

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 their role and responsibilities because each team member's role is important to the performance of the team. Figure 38 identifies 6 team roles for resuscitation. When fewer than 6 people are present, Team Leaders must prioritize these tasks and assign them to the health care professionals present.

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 health care professionals 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 13 lists some additional information about team roles.

Table 13. Clear Team 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 according to level of expertise and availability
Team members	• Seek out and perform clearly defined tasks appropriate to their abilities • Practice closed-loop communication by telling the Team Leader when an assigned task has been completed • 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 • Quickly assess the situation, and offer to take a needed role

Knowing Your Limitations

Everyone on the team should know their 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 health care professionals. 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 14 lists some additional information about knowing your limitations.

Table 14. 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 health care professionals when the patient's condition worsens despite primary treatment • Allow others to carry out assigned tasks, especially if the task is essential to treatment
Team members	• Seek advice from more experienced health care professional before starting an unfamiliar treatment or therapy • Accept assistance from others when it is readily available

Constructive Interventions

During a resuscitation attempt, anyone on a high-performance team may need to tactfully intervene 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 15 lists some additional information about constructive interventions.

Table 15. Constructive Interventions

Team member	Task
Team Leader	• Instruct that a different intervention be started if it has a higher priority • Reassign a team member who is trying to function beyond their level of skill
Team members	• Suggest an alternative medication or dose confidently • Question a colleague who is about to make a mistake • Intervene if a team member is about to take an inappropriate action (eg, administer a medication 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.”

When resuscitative efforts are ineffective, go back to the basics and talk as a team. This technique can help to reduce fixation errors. Have conversations like, “We’ve observed the following during 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 16 lists some additional information about knowledge sharing.

Table 16. Knowledge Sharing

Team member	Task
Team Leader	• Encourage information sharing • Ask for suggestions about interventions, differential diagnoses, and possible overlooked treatments (eg, IV access or medication 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

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. This assures that the team has a shared mental model so that their efforts are coordinated and smooth. It also invites team members to speak up with new information, ideas, or questions. Remember that the patient's condition can change. Be flexible about changing treatment plans and ask for information and summaries from the Timer/Recorder as well. Table 17 lists some additional information about summarizing and reevaluating.

Table 17. 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 health care professionals of the current status and plans for further action
Team Leader and team members	• Speak up when there are significant changes in the patient's clinical condition • Increase monitoring if patient's condition deteriorates (eg, frequency of respirations and BP)

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, an order, or an assignment to a team member.
2. Request a clear response and eye contact from the team member to ensure that they understood the message.
3. Confirm that the team member completed the task before you assign them another task.

Table 18 lists some additional information about closed-loop communications.

Table 18. Closed-Loop Communications

Team member	Task
Team Leader	• Always assign tasks by using closed-loop communication, such as, "Give 1 mg 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
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 medications after verbally confirming the order with the Team Leader

Clear Messages

Clear messages means concise communication spoken with distinctive speech in a controlled voice. All health care professionals should deliver clear messages calmly and directly. Distinct, concise messages are crucial for clear communication because unclear communication can delay treatment or cause medication errors. The most effective teams are usually quiet, calm, and focused on a common goal—not noisy or upset. Table 19 lists some additional information about clear messages.

Table 19. 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 coordinated, goal-directed manner. Everyone in a high-performance team must abandon ego and show respect during the resuscitation attempt, combining their skills and helping each other. Table 20 lists some additional information about mutual respect.

Table 20. Mutual Respect[1]

Team member	Task
Team Leader	• Acknowledge correctly completed assignments by giving positive feedback, like, “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 • Understand that when one person raises their 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.

Medications for Respiratory Arrest

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

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 carbon dioxide (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)
- Cyanosis—pale skin and nail beds

- Changes in level of consciousness or 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, they 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. Recent studies have shown a positive bias in oxygen saturation measurements in patients with darkly pigmented skin, particularly under low-saturation conditions (<80%).[2] Overestimation is an issue of concern because patients may appear to be healthier than they are with the corresponding risk of adverse effects to health from diseases such as COVID-19.

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 CO_2 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, opioid overdose, or head injury. For an adult in respiratory arrest, provide a tidal volume 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. In these cases, bypassing the pressure-relief valve may allow higher pressure to be delivered to produce chest risk. Keep in mind, these higher pressures may be harmful and caution should be used.

Caution

Tidal Volume

Most adult bag-mask devices provide a higher tidal volume than is recommended. Caution is advised. Squeeze the bag enough to make the chest rise, about one third to one half of an adult 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 importantly, 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). Spend no more than 10 seconds looking for no breathing or only gasping and checking for a pulse (simultaneously).

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 there is no pulse, start CPR.

If an opioid overdose is suspected, 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. 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 continuous waveform capnography will confirm and monitor placement of the advanced airway.

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 (OPA and 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 90%-98% for post–cardiac arrest care). Use 100% oxygen when treating patients in respiratory or cardiac arrest.

See 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 39 shows the airway anatomy). In this case, the patient's tongue falls back and obstructs the airway at the pharynx (Figure 40A).

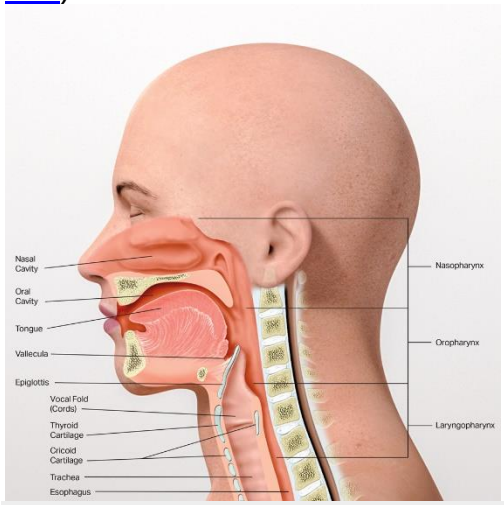


Figure 39. Airway anatomy.

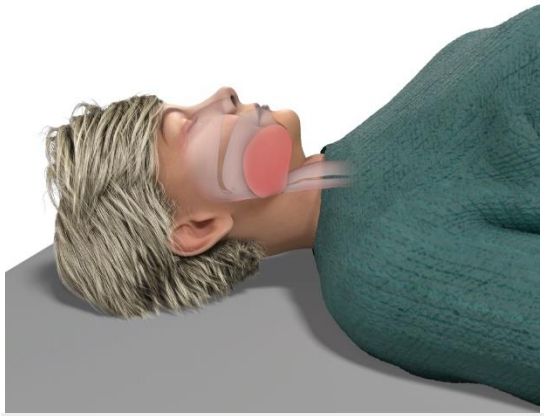


Figure 40A. Obstruction of the airway by the tongue and epiglottis: When a patient is unresponsive, the tongue can obstruct the airway; the head tilt–chin lift relieves obstruction in the unresponsive patient. A, The tongue is obstructing the airway.

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 40B).

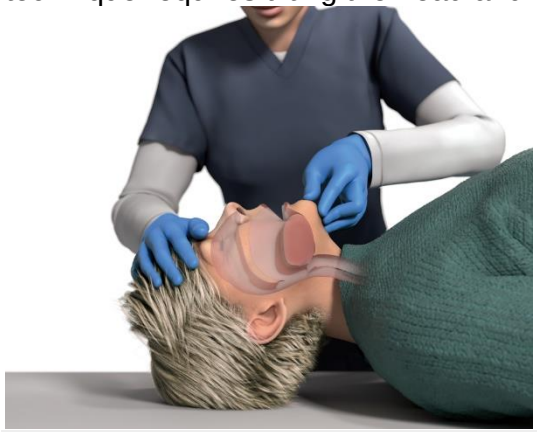


Figure 40B. The head tilt–chin lift lifts the tongue, relieving the obstruction.

In a trauma patient with suspected neck injury, try using a jaw-thrust technique that doesn't extend the head (Figure 40C). 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.

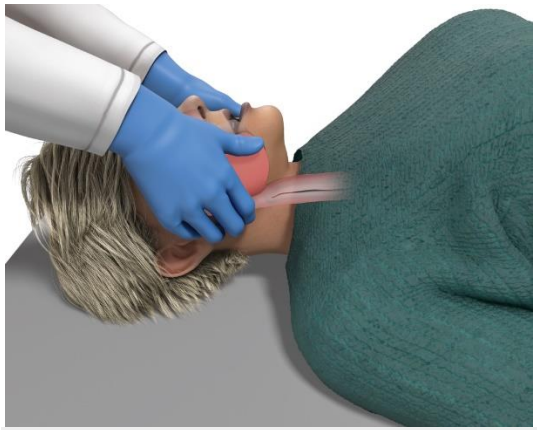


Figure 40C. The jaw thrust without head extension; use if cervical spine trauma is suspected.

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. Do not perform a blind finger sweep. 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

The basic skills used to ventilate a patient after opening the airway (using the head tilt–chin lift or jaw thrust) are

- Mouth-to-mouth ventilation
- Mouth-to-nose ventilation
- Mouth-to–barrier device ventilation (using a pocket mask)
- Bag-mask ventilation

Note: If a jaw thrust is used for suspected trauma and the airway cannot be opened, use the head tilt–chin lift to open the airway. An open airway is a priority.

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. The self-inflating bag may or may not contain a nonrebreathing valve. Masks are made of transparent material to allow detection of regurgitation. They should be capable of creating a tight seal on the face, covering both mouth and nose. These devices are the most common way to provide positive-pressure ventilation. When you use a bag-mask device, deliver enough tidal volume to produce chest rise over 1 second. Consider insertion of an oropharyngeal airway if the patient has no cough or gag reflex to help maintain an open airway. Use a nasopharyngeal airway if there is a gag reflex.

Note: It is recommended that a single emergency responder performing CPR use a pocket mask for ventilation instead of a bag-mask device because studies have shown that this results in the highest CCF. If this is not available, another method should be used.

Health care professionals 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 41): The rescuer gets into position at the patient's head and will use an E-C clamp technique to hold the mask in place. To do this, form a "C" with the thumb and index finger of one hand and press down on the dome of the mask, toward the face, to further seal the rim of the mask. The remaining 3 fingers of that same hand should form an "E" to reach past the edge of the mask, along the bony rim of the jaw, to tilt the head back, lift the jaw toward the mask, and open the airway.
- Use of the bag-mask device by 2 rescuers (Figure 42): 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 to form the "E," lifting both sides of the jaw into the mask. The second rescuer slowly squeezes the bag (over 1 second) until the chest rises. Both rescuers should observe chest rise.

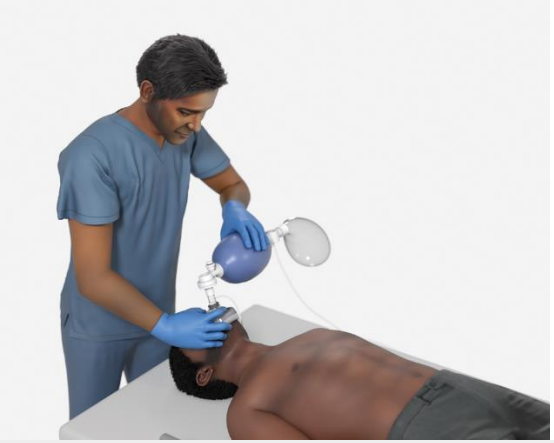


Figure 41. **E-C clamp technique for holding the mask while lifting the jaw.**

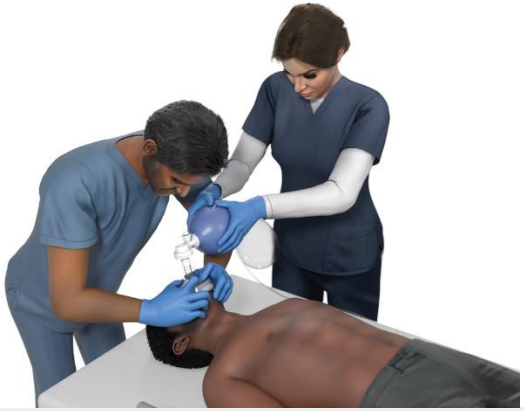


Figure 42. **Use of a bag-mask device by 2 health care professionals.**

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
- Suction ports to clear the airway
- Ports to provide quantitative sampling of end-tidal CO_2 ($ETCO_2$)

You can attach other adjuncts to the patient end of the valve, including a pocket face mask, supraglottic airways, and an ET tube. Ongoing continuous quantitative waveform capnography can also be attached to a bag-mask device to confirm and monitor the effectiveness of the ventilation. An obstructed airway with no air exchange will not produce exhaled CO_2, even if the patient still has a pulse.

Basic Airway Adjuncts: OPA

The OPA is a J-shaped device (Figure 43A) 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 43A. Oropharyngeal airways. A, OPA devices.

You may also use an OPA during bag-mask ventilation when an emergency responder 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, consider 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 43B). 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 43C). 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 43B. OPA device measurement.

Figure 43C. OPA 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. Insertion of an OPA can stimulate the tissues of the upper airway, causing a reflex closure of the vocal cords and laryngospasm.

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, tongue, and laryngeal structures.
- 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 44A) that provides a conduit for airflow between the nostrils and the pharynx.

Figure 44A. Nasopharyngeal airways. A, NPA devices.

Unlike oral airways, NPAs may be used in conscious, semiconscious, or unconscious patients (patients with an intact cough and gag reflex). Using 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.
    - –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.
    - –The NPA should be as long as the distance from the tip of the patient's nose to the earlobe (Figure 44B).
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 44C). If you encounter resistance
    - –Slightly rotate the NPA to insert at the angle of the nasal passage and nasopharynx
    - –Attempt to place through the other nostril (the size of a patient's nasal passages varies)
4. Reevaluate often and maintain an open airway using a head tilt–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 44B. NPA device measurement.

Figure 44C. **NPA 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 NPA.
- If respirations are absent or inadequate, start positive-pressure ventilation at once with an appropriate device.
- If an OPA, an NPA, or other adjuncts are unavailable, provide mouth-to-barrier-device ventilations.

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, an 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

Health care professionals can use continuous 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%. Oxygen saturation may be inaccurate (artificially high) in individuals with darkly pigmented skin, particularly under low saturation conditions.[2] Give supplemental oxygen when indicated. For patients with cardiac arrest, give 100% oxygen. For other clinical conditions, adjust the oxygen administration to achieve an oxygen saturation as follows:

- ACS: 90% or greater
- Stroke: 95% or greater
- Post–cardiac arrest care: 90% 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 tubes
- Supraglottic airways

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 rigorous initial training and ongoing experience. Health care professionals 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 health care professionals should insert these advanced airways.

Refer to ACLS Student Resources for more information about advanced airways.

ET 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 (refer to 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 CO_2 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

A *laryngeal tube* is a single-lumen tube that is more compact and less complicated and easier to insert than the ET tube. 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 comes in a variety of similar shapes and sizes, is an advanced airway alternative to ET intubation, and provides comparable ventilation for airway management in cardiac arrest.

Note: If a patient cannot breathe and oxygenation is not possible with bag-mask ventilation, supraglottic devices, or ET intubation, emergency front-of-neck access or surgical airway may be required. Because this requires specialized training, this option may not be available. Failure to oxygenate quickly leads to death in minutes, so immediately request this assistance if available if a difficult airway is expected or discovered.

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 traumatic injuries, 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 motion restriction is helpful during transport.

Cardiac Arrest: VF or pVT (Shockable)

Overview

To be successful, high-performance teams need a strong base of high-quality CPR and early defibrillation. Timing is critical, so frequent training to improve logistics and reduce time to critical interventions affects survival rates. Administrators must also assess the performance of each system component, ensuring that system participants can effectively intervene to improve care. This process of quality improvement comprises 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 health care professionals interrupt compressions far too often and for too long, in some cases spending 25% to 50% of a resuscitation attempt without delivering chest compressions.

CCF is the proportion of time during a cardiac arrest resuscitation when the health care professional is performing chest compressions. CCF should be as high as possible—at least 60% but ideally greater than 80%. Data suggest lower CCF is associated with decreased ROSC and survival to hospital discharge, while trials have shown that a 10% increase in CCF is roughly equal to an 11% increase in survival.

Rhythms for VF and pVT (Shockable)

- VF (example in Figure 45)
- VT

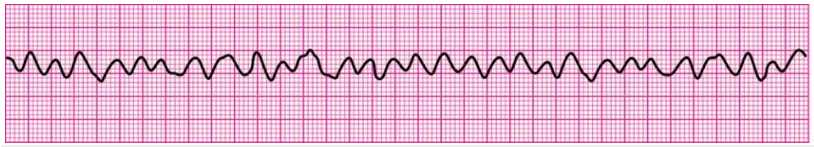


Figure 45. Example of VF.

Medications for VF and pVT

Medications for VF and pVT include

- Epinephrine
- Amiodarone
- Lidocaine
- Magnesium sulfate

- Oxygen
- Other medications, depending on the cause of the VT or pVT arrest

Managing VF and pVT: The Adult Cardiac Arrest Algorithm

The initial step in the Adult Cardiac Arrest Algorithm (Figure 46) 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 (carotid or femoral), and start CPR, beginning with chest compressions. Attach the ECG monitor or AED pads as soon as they are available. Throughout the resuscitation attempt, a high-performance team will provide high-quality CPR, defibrillating as needed. Timing is critical.

- Give oxygen.
- Attach the monitor or defibrillator.

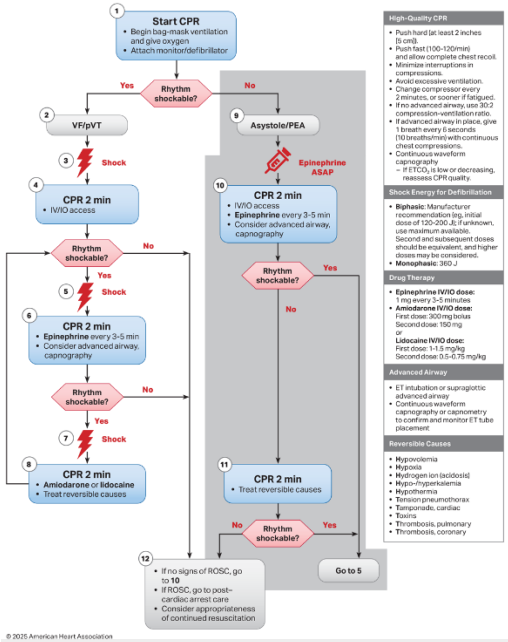


Figure 46. Adult Cardiac Arrest Algorithm (VF/pVT).

Once the monitor or defibrillator is attached, check the rhythm to determine whether it is shockable (VF or pVT) or nonshockable (asystole or PEA) and follow the appropriate cardiac arrest pathway. The Adult Cardiac Arrest 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 comprises the 2 pathways for cardiac arrest:

- A shockable rhythm, displayed on the VF/pVT pathway of the algorithm (Figure 46)
- A nonshockable rhythm, displayed on the asystole/PEA pathway of the algorithm (Figure 54)

VF/pVT Pathway

Because many patients with sudden cardiac arrest may 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 46). 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 and early defibrillation. As soon as a defibrillator is available and ready, deliver high-energy unsynchronized shock.

Applying the Adult Cardiac Arrest Algorithm: VF/pVT Pathway

For this algorithm, emergency responders 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 (Figure 46).

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

Start CPR

Start CPR (Step 1).

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 and 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 continuous quantitative waveform capnography. Timing and quality can be achieved by high-performance teams that are evaluating and achieving the following:

- Rate: 100 to 120/min
- Depth: at least 2 inches (5 cm)
- Complete chest recoil
- CCF: ideally greater than 80%*
- Time to first defibrillation
- Time to first compression
- P_{ETCO_2} at 10 mm Hg or higher to achieve ROSC
- Switching compressors every 2 minutes or earlier if fatigued
- Avoiding excessive ventilation

*High-performing systems target at least 60% but ideally have greater than 80% as a frequent goal.

Obtaining CCF

Health care professionals can obtain CCF using a feedback device or an ACLS app, 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

For purposes of minimizing interruption in compressions (timing), the AHA does not recommend continued use of an AED (or the automatic mode) when a manual defibrillator is available and health care professionals can adequately interpret rhythms. Rhythm analysis and shock administration with an AED may prolong the interruptions in chest compressions.

You should deliver the shock as soon as the compressor removes their hands from the patient's chest and all emergency responders are clear of contact with the patient. The same or next compressor should hover over the chest during the shock, and then resume compressions immediately.

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

Figure 47 illustrates the need to minimize interruptions in compressions. 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. Once compressions are stopped or interrupted, CPP becomes very low and requires several compressions after starting again to achieve a level adequate to potentially achieve ROSC.

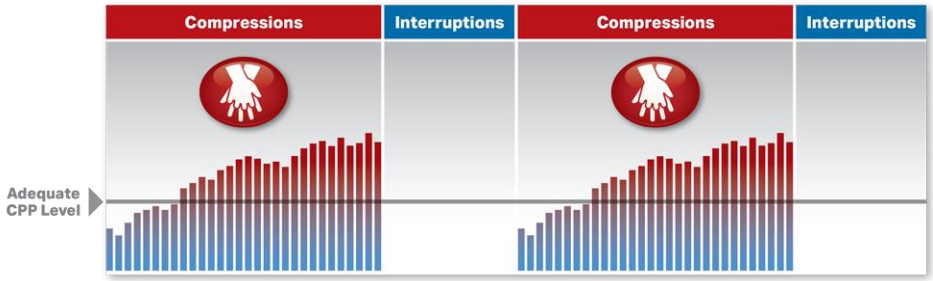


Figure 47. Relationship of quality CPR to CPP demonstrating the need to minimize interruptions in compressions. Once compressions are stopped, it takes several compressions after starting again to achieve a CPP adequate to potentially achieve ROSC.

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 manufacturer of the biphasic defibrillator device.

Biphasic defibrillators use various waveforms that effectively terminate VF over a specific dose range. When using biphasic defibrillators, emergency responders should use the manufacturer's recommended energy dose (eg, initial dose of 120-200 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.

If the only available defibrillator is an AED, follow the device's prompts. Emergency responders should know how their defibrillator operates and limit pauses in chest compressions for rhythm analysis and shock delivery.

More recently, a randomized control trial has discussed the possible benefits of vector change and double sequential defibrillation. However, the usefulness of these approaches has not been established.[3]

Immediately after the shock, resume CPR, beginning with chest compressions. Give 2 minutes of CPR. If there are available health care professionals, IV or intraosseous (IO) access should be established.

In adults experiencing sudden cardiac arrest due to VF, 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 shocks 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).

It is critical to restore circulation to the heart and brain as quickly as possible to minimize the damage caused by loss of perfusion. CPR provides blood flow and oxygen throughout the system but, critically, also to the heart and brain. Successful defibrillation leads to a perfusing rhythm that is typically more effective than chest compressions, so a shock should be delivered as soon as the defibrillator is available and ready. 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 by restoring blood flow. Again, time is critical; if there are 2 or more health care professionals present, CPR should be performed while the defibrillator pads are being attached to the patient's chest.

After defibrillation, the patient needs immediate CPR. 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 because of the following:

- 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 48).

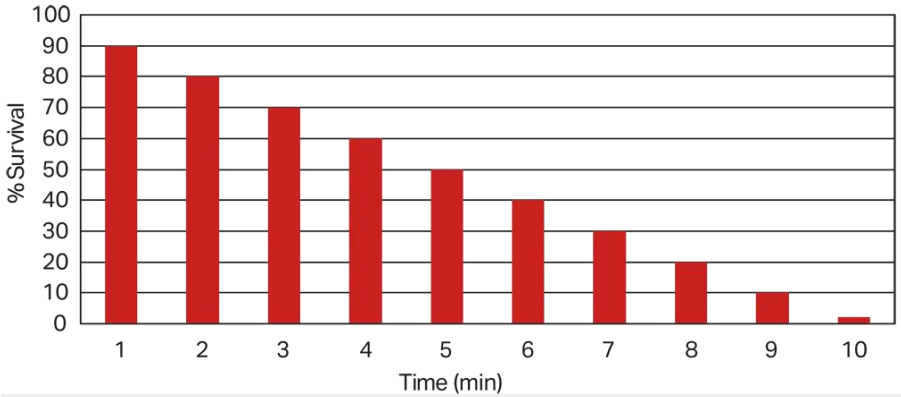


Figure 48. Relationship between survival from VF sudden cardiac arrest and time from collapse to defibrillation.

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 lay rescuer CPR.[4] When lay rescuers perform CPR, the decline is more gradual and averages 3% to 4% per minute.[5-8] Early CPR can double[5,9] or triple[10] 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.

Performing Defibrillation

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, and 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

- Immediately after defibrillation, perform CPR for 2 minutes, starting with compressions.
    - –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 medication delivery.
- The guidelines recommend that health care professionals 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.

Use a default compression-to-ventilation ratio of 30:2 for less-trained health care professionals or if 30:2 is the established protocol. Figure 49 shows the progression from lay rescuers to highly trained and proficient health care professionals.

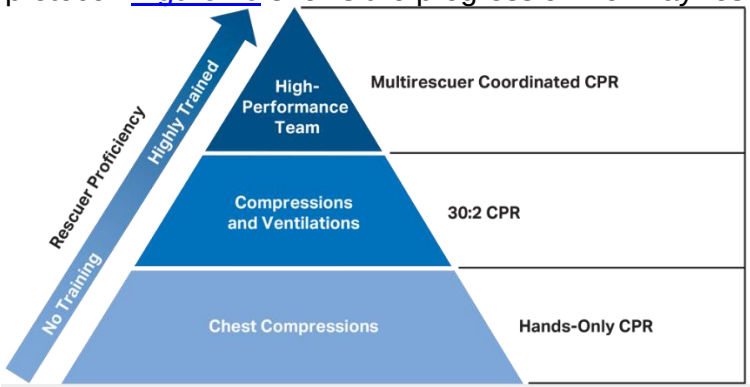


Figure 49. Progression from lay rescuers to highly trained health care professionals for CPR delivery.

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 (carotid or femoral). 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, immediately resume CPR and proceed along the asystole/PEA pathway of the Adult Cardiac Arrest Algorithm (Figure 54) (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 reduce transthoracic impedance—the resistance that the chest has on electrical current.

Vasopressors

Vasopressors optimize cardiac output and BP, and evidence shows that using vasopressors favors initial resuscitation with ROSC. Recent evidence has demonstrated that epinephrine increases ROSC, survival to hospital admission, and survival to hospital discharge, making it a critical intervention during cardiac arrest.[11]

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 (MAP) 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 or 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 medications and prepare them in advance.

Vasopressor medication (epinephrine) can improve aortic diastolic BP, coronary artery perfusion pressure, and the rate of ROSC. The AHA continues to recommend their use.

Consider Placement of an Advanced Airway

In addition to the common reasons for placing an advanced airway (eg, difficulty ventilating, airway compromise, long transport times), the ability to move to continuous compressions early (after the second shock for VF or pVT) allows health care professionals to increase CCF for those who use a 30:2 CPR protocol. Ideally, advanced airway placement should be done without interrupting chest compressions.

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

Health care professionals may consider giving antiarrhythmic medications after the third shock, with repeat dosing as appropriate. Alternating administration of epinephrine with an antiarrhythmic until maximum dose reached is recommended. The focus should be on administering the medications during CPR so that it does not interrupt compressions or delay defibrillation. These medications may be particularly useful for patients with witnessed arrest, for whom time to medication administration may be shorter.[12]

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 or 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.[13] 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 lay rescuer–witnessed cardiac arrest, survival to hospital discharge was higher for patients given amiodarone or lidocaine compared with those given placebo.[12]

Amiodarone or lidocaine may be considered for VF or pVT that is unresponsive to defibrillation. These medications may be particularly useful for patients with witnessed arrest, for whom time to medication administration may be shorter.[12]

- **Amiodarone:** 300 mg IV/IO bolus, then consider 1 additional 150 mg IV/IO
    - –Amiodarone is considered a class III antiarrhythmic medication, but it possesses electrophysiologic characteristics of the other classes. Amiodarone blocks sodium channels at rapid pacing frequencies (a class I effect) and exerts a noncompetitive antisympathetic action (a class II effect). One of the main effects of prolonged amiodarone administration is lengthening of the cardiac action potential (a 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.

Health care professionals can consider magnesium sulfate for torsades de pointes associated with a long QT interval. Magnesium sulfate is not recommended for routine use during cardiac arrest.

- **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 50) summarizes the recommended sequence of CPR, rhythm checks, shocks, and delivery of medications. Continue CPR while preparing and administering medications and charging the defibrillator.

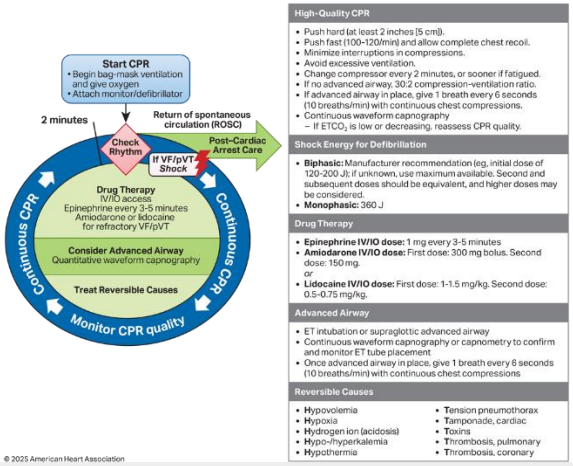


Figure 50. Adult Cardiac Arrest Circular Algorithm.

Physiologic Monitoring During CPR

For intubated patients, the AHA recommends using continuous waveform capnography to monitor CPR quality (Figure 51), optimize chest compressions, and detect ROSC during chest compressions (Figure 52). The capnography tracing in Figure 52 displays P_{ETCO_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 P_{ETCO_2} is less than 12.5 mm Hg during the first minute, indicating very low blood flow. P_{ETCO_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 P_{ETCO_2} (visible just after the fourth vertical line) to greater than 50 mm Hg, which is consistent with a substantial improvement in blood flow.

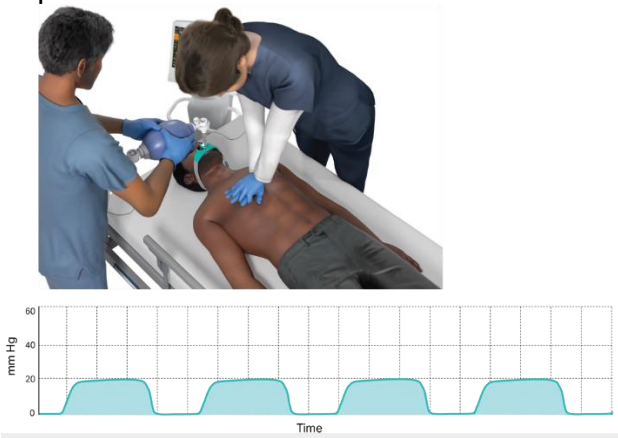


Figure 51A. Physiologic monitoring during CPR. A, High-quality compressions are shown through waveform capnography.

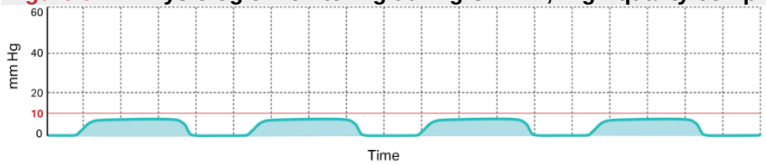


Figure 51B. Ineffective CPR compressions are shown through waveform capnography.

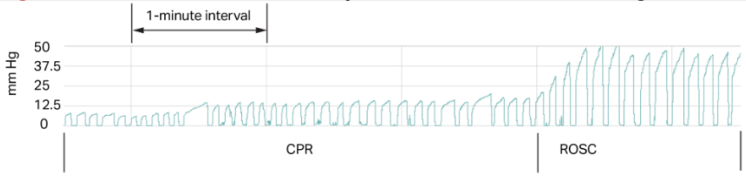


Figure 52. Waveform capnography during CPR with ROSC.

Although invasive monitors are usually not needed during CPR, physiologic parameters, such as intra-arterial relaxation pressures (Figures 51A and 51B) and central venous oxygen saturation ($ScvO_2$), may help optimize CPR and detect ROSC.

Animal and human studies indicate that monitoring P_{ETCO_2}, CPP, and central venous oxygen saturation provides valuable information on the patient's condition and response to therapy.[14-20] 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 P_{ETCO_2} during CPR is blood delivery (cardiac output) to the lungs. Persistently low P_{ETCO_2} values less than 10 mm Hg during CPR in intubated patients (Figure 51B) suggest that ROSC is unlikely, and it is reasonable to try to improve chest compressions and vasopressor therapy. If P_{ETCO_2} abruptly increases to a normal value or higher (Figure 52), it is reasonable to consider this an indicator of ROSC.

Coronary Perfusion Pressure

Increased CPP correlates with both myocardial blood flow and ROSC. A reasonable substitute for CPP during CPR is continuous waveform capnography (Figure 51A).

Routes of Access for Medications

The first priority during cardiac arrest is providing high-quality CPR and early defibrillation; secondary priorities are administering medications and inserting an advanced airway.

Health care professionals usually administer medications via IV or IO. The IV route is preferred over IO. When IV access is unsuccessful or not feasible, IO access may be considered. Medications administered via ET tube is not recommended.

IV Route

Use a peripheral IV for medication and fluid administration unless central venous access is already available.

You do not need to interrupt CPR to establish a peripheral IV line, but medications typically take 1 to 2 minutes to reach the central circulation via the peripheral IV route. If you give a medication by the peripheral IV route, administer it as follows:

- Give the medication 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 medication to the central circulation.

Central venous access is not necessary during most resuscitation attempts, and it may cause interruptions in CPR and complications during insertion. These complications include vascular laceration, hematoma, bleeding, thrombosis, and infection. Inserting a central line in a noncompressible vessel is a relative (not absolute) contraindication to thrombolytic therapy in patients with ACS.

IO Route

If IV access is not successful or feasible, you can safely and effectively deliver medications 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
- Any ACLS medication 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 medications, 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, refer to the Access for Medications section in the ACLS Student Resources.

Fluid Administration

In hypovolemic patients, the extracellular fluid volume is typically restored with normal saline or lactated Ringer's solution, but avoid D_5W because it will reduce serum sodium too rapidly. Monitor serum electrolytes as appropriate.

Ultrasound for Cardiac Arrest

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 ROSC, such as pulse and BP, an abrupt and sustained increase in P_{ETCO_2} (typically, 40 mm Hg or greater), or spontaneous arterial pressure waves with intra-arterial monitoring, follow the Adult Post–Cardiac Arrest Care Algorithm (Figure 64).

If there are no signs of ROSC, resume CPR, administer epinephrine, and treat reversible causes. Consider the appropriateness of continued resuscitation.

Cardiac Arrest: PEA and Asystole (Nonshockable)

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 Asystole (Lack of Rhythm)

You will need to recognize asystole (Figure 53) and all rhythms that do not have a pulse (PEA).

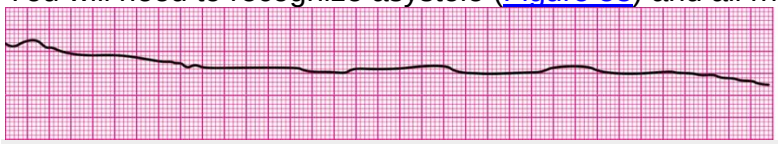


Figure 53. Example of asystole.

Medications for PEA and Asystole

Medications 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 comprises 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, occur at rapid or slow rates, and 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. The heart does not pump 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 or relatively high P_{ETCO_2}, it is more likely that the left ventricle is full and that the cause of PEA is a poorly contractile left ventricle; conversely, if good CPR still does not produce evidence of good cardiac output, consider evaluating for other reversible causes (ie, H’s and T’s).

Approach to Asystole

Asystole occurs when the heart stops beating and the ECG shows no discernible electrical activity (also called *flatline*). You should confirm that the flatline on the monitor is indeed true asystole by validating that the flatline is not

- Some other rhythm (eg, fine VF) masquerading as a flatline
- 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 *flatline* is nonspecific and can refer to a result of several possible factors, 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 others 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 medication overdose. Consider stopping after 20 minutes of CPR, if all reversible causes of cardiac arrest have been addressed, and if P_{ETCO_2} is less than 10 mm Hg.

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 and PEA: The Adult Cardiac Arrest Algorithm

The Adult Cardiac Arrest Algorithm comprises 2 cardiac arrest pathways—the treatment for a shockable rhythm (VF or pVT; Figure 46) and the treatment for a nonshockable rhythm (asystole or PEA; Figure 54). 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.

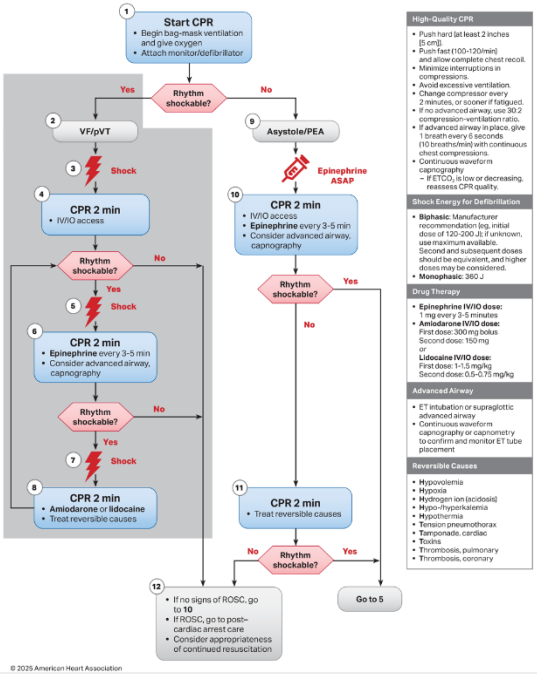


Figure 54. Adult Cardiac Arrest Algorithm (Asystole/PEA).

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 asystole or 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

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 or defibrillator.

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

Managing Asystole and PEA

If the patient has no discernible electrical activity and no pulse, it is asystole (Step 9). If the patient has an organized rhythm on the monitor but no pulse, it 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 54).

Critical Concepts

Administer Epinephrine

Give epinephrine as soon as IV or IO access becomes available.

- All patients with cardiac arrest should be given epinephrine. 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.[11]
- Repeat epinephrine 1 mg IV/IO every 3 to 5 minutes or every 4 minutes as a midrange (ie, every other rhythm check).
- Administer medications during CPR. Do not stop CPR to administer medications.
- Prioritize establishing IV or 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 asystole or PEA while they perform their assigned roles.

Perform a Rhythm Check

Check the rhythm and give 2 minutes of CPR after administering epinephrine, 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 continuous waveform capnography.

Nonshockable Rhythm

- If *no electrical activity is present* (asystole) during the rhythm check, continue 2 minutes of CPR, starting with compressions, and then repeat the sequence.
- If *organized electrical activity is present*, try to feel for a pulse for at least 5 seconds but no more than 10 seconds.
- 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 54 summarizes experts' recommended sequence of CPR, rhythm checks, and delivery of medications for PEA and asystole.

Identifying and Correcting Underlying Causes

Treating asystole or 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 or PEA for resuscitative efforts to be potentially successful. Use the H's and T's to recall conditions that could have contributed to asystole or PEA, and remember that hypovolemia and hypoxia are the 2 most common underlying and potentially reversible causes of asystole and PEA.

When in Doubt

If it is unclear whether the rhythm is fine VF or asystole or PEA, an initial attempt at defibrillation may be warranted. Fine VF may result from a prolonged arrest.

Patients With Do-Not-Attempt-Resuscitation 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
- Decomposition
- Hemisection
- Decapitation
- Clear indicators of do-not-attempt-resuscitation status (eg, bracelet, anklet, written documentation)
- Threat to the safety of the health care professionals

Out-of-hospital emergency responders need to know EMS-specific policies and protocols applicable to these situations. In-hospital health care professionals 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 do-not-attempt-resuscitation order, which may also be known as physician orders for life-sustaining treatment or medical orders for life-sustaining treatment, is unclear or uncertain, resuscitation should be initiated and continued until it can be clarified.

Terminating Resuscitative Efforts

In-Hospital

If health care professionals 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 ED, and the decision should be made considering multiple 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 predict 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 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 that ECPR is not indicated or not available.

Out-of-Hospital

Continue out-of-hospital resuscitative efforts until 1 of the following occurs:

- There is restoration of effective, spontaneous circulation and ventilation.
- Transfer of care to a senior emergency responder occurs.
- Reliable criteria indicate irreversible death.
- Exhaustion or dangerous environmental hazards prevent the emergency responders from continuing.
- Continued resuscitation places the lives of others in jeopardy.
- A valid do-not-attempt-resuscitation 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 (Figure 55). If ROSC of any duration occurs, it may be appropriate to consider extending the resuscitative effort.

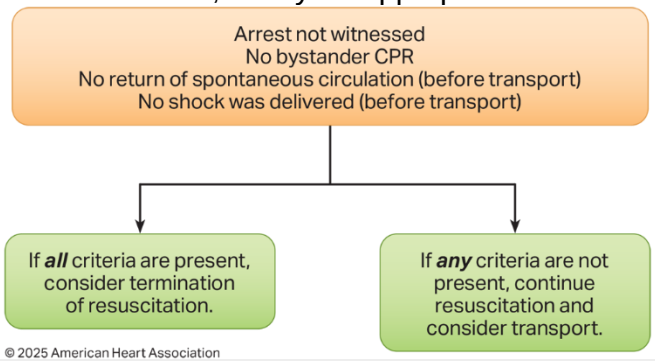


Figure 55. ALS termination of resuscitation information rule.

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 medication 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, medication overdose, or other potentially reversible causes of arrest.

Organ Donation and Cardiac Arrest

Organ transplantation wait times in the United States are increasing as patients in need of transplants outpace available organs, leaving thousands who die annually waiting for transplantation. Patients with cardiac arrest may make up an important pool of potential donors because cardiac arrest is common and a substantial proportion of those who cannot recover may still be able to donate.

After sudden cardiac arrest, donation after circulatory death can be pursued in patients

- With ROSC after planned withdrawal of life-sustaining therapies and the transition to comfort-oriented care
- Without ROSC after unsuccessful resuscitation

Key Considerations for Organ Donation After Cardiac Arrest

- Organ donation should be considered in
    - –All patients resuscitated from cardiac arrest who meet neurologic criteria for death
    - –All patients resuscitated from cardiac arrest before planned withdrawal of life-sustaining therapies
- Local legal and regulatory requirements should be followed in all organ donation decisions.
- Organ donation is also reasonable in patients resuscitated with ECPR who remain supported on ECMO and meet neurologic criteria for death or for whom withdrawal of life-sustaining therapies is planned.

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 deceased (eg, rigor mortis, decomposition, hemisection, decapitation) (refer to the discussion on do-not-attempt-resuscitation status 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

Due to the dangers of performing CPR during transport, decisions to transport with CPR in progress should be made by leveraging predefined medical protocols that consider the risks and benefits to the patient and emergency responders.

After OHCA with ROSC, transport the patient to the nearest, most-appropriate hospital, ideally one with a comprehensive post–cardiac arrest treatment system of care that includes acute coronary interventions, neurologic care, critical care, and hypothermia. In-hospital post–cardiac arrest patients with ROSC should be transported to an appropriate critical care unit that can provide comprehensive post–cardiac arrest care.

Cardiac Arrest: Selected Special Situations

Treating VF and pVT in Life-Threatening Environmental Hypothermia

Defibrillation is appropriate for patients with cardiac arrest in VF or pVT with severe accidental hypothermia (a body temperature less than 30 °C [86 °F]). If a patient does not respond to the initial shock, it is reasonable to defer additional defibrillation attempts until the core temperature is 30 °C (86 °F) or greater. Hypothermic patients may have a reduced rate of drug metabolism, and medications may accumulate to toxic levels with standard dosing regimens. It is reasonable to defer administering a vasopressor according to the standard ACLS algorithm until the core temperature is 30 °C (86 °F) or greater.

For in-hospital patients in cardiac arrest with severe accidental hypothermia, aim ACLS treatment at rapid core rewarming.

For patients in cardiac arrest with moderate hypothermia (30-34 °C [86-93.2 °F]), start CPR, attempt defibrillation, give medications according to local protocols, and, if in-hospital, provide active core rewarming.

Treating Respiratory or Cardiac Arrest Associated With Opioid Overdose

- The rate of opioid-related deaths appears to be decreasing, but this issue still remains a significant public health emergency in the United States.[11]
- Isolated opioid toxicity is associated with central nervous system and respiratory depression that can progress to respiratory and cardiac arrest. Most opioid-related deaths involve ingesting multiple medications or having medical and mental health comorbidities.[21-23] In addition, methadone can cause torsades de pointes and cardiotoxicity has been reported as being associated with other opioids.[24-30] Except in specific clinical settings (eg, unintended opioid overdose during a medical procedure), emergency responders cannot be certain that the patient's clinical condition is due to opioid-induced central nervous system and respiratory depression toxicity alone.

- Opioid antagonists (eg, naloxone) are potent opioid receptor antagonists in the brain, spinal cord, and GI system. Naloxone has an excellent safety profile and can rapidly reverse central nervous system and respiratory depression in patients with an opioid-associated resuscitative emergency. Depending on their training and clinical circumstance, emergency responders 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 Emergencies

Complete the following steps to manage an opioid-associated life-threatening emergency. More information on the management of opioid-associated life-threatening emergencies is given in Figure 56, including information for treating respiratory and cardiac arrest related to suspected opioid overdose.

- For respiratory arrest
    - –If an opioid overdose is suspected, administer an opioid antagonist (eg, naloxone) if available
    - –Continue providing breaths once every 6 seconds
- For cardiac arrest
    - –Start high-quality CPR
    - –Use an AED or defibrillator as soon as it is available
    - –If an opioid overdose is suspected, consider an opioid antagonist (eg, naloxone) if available

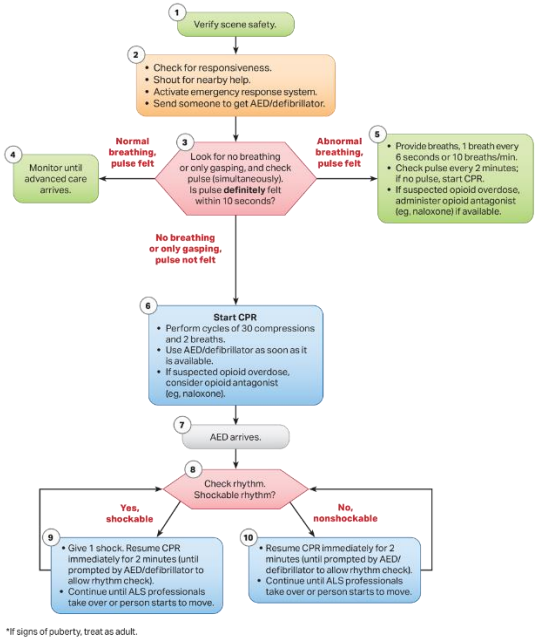


Figure 56. Adult* BLS Algorithm for Health Care Professionals.

Treating Cardiac Arrest With ECPR

ECPR is a means of using a machine to circulate blood around the body of a patient in cardiac arrest and refers to venoarterial ECMO. ECPR techniques require adequate vascular access and specialized equipment (Figure 57). By using ECPR, health care professionals 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, medication intoxication) are treated. ECPR can also serve as a bridge for left ventricular assist device (LVAD) implantation or cardiac transplantation.

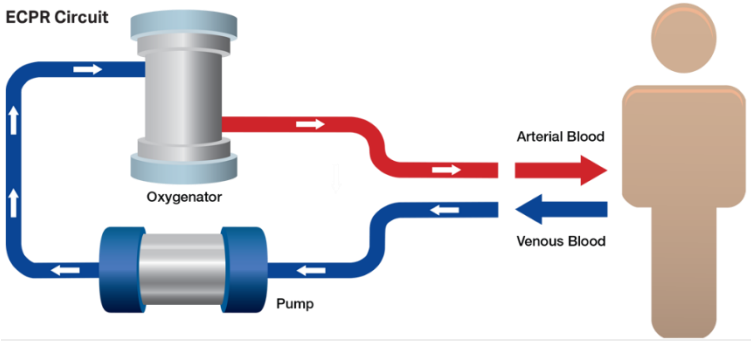


Figure 57. Schematic depiction of components of an ECMO circuit as used for ECPR, including a venous cannula, a pump, an oxygenator, and an arterial cannula.

Currently, ECPR requires vascular access with large-bore cannulas inserted into the central vasculature, specialized equipment, and expertise in using ECMO. ECPR should be considered for select patients with cardiac arrest.

Consider ECPR in settings where the necessary equipment and trained health care professionals can be deployed rapidly for select patients with cardiac arrest 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 an LVAD
- A right ventricle with a right ventricular assist device (RVAD)
- Both ventricles with a biventricular assist device

Figure 58 shows the support intended with an LVAD, an RVAD, and a biventricular assist device. Most VADs are implanted inside the thoracic or abdominal cavity (intracorporeal) (Figure 59). 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]

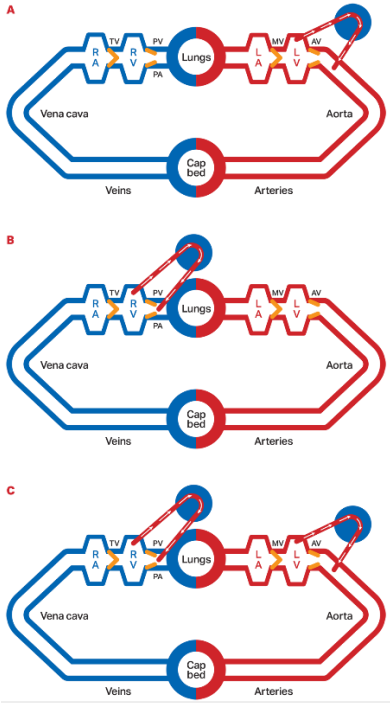


Figure 58. VAD configurations. A, LVAD. B, RVAD. C, Biventricular assist device.

Abbreviations: LA, left atrium; MV, mitral valve; PA, pulmonary artery; PV, pulmonary valve; RA, right atrium; TV, tricuspid valve.

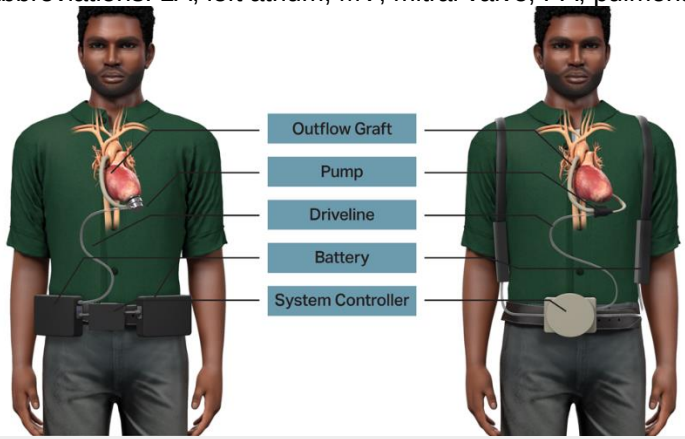


Figure 59. Intracorporeal pumps.

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 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 emergency responders determine if an unresponsive or mentally altered patient is, in fact, in cardiac arrest or circulatory collapse.

The 2 most common causes of VAD 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. Emergency responders must keep patients and their backup equipment together at all times because replacement equipment may be limited or nonexistent at receiving EDs, particularly at non-VAD centers. To reiterate, when a mechanical circulatory support patient is transported by emergency responders, all of the patient's VAD equipment must accompany them to the ED 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 crushing 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 a Patient With an LVAD

To manage a patient with an LVAD who is unresponsive, refer to the following steps. More information is given in the Adult and Pediatric Durable LVAD Algorithm (Figure 60).

- Assist ventilation if necessary and assess perfusion.

- Determine if there is adequate perfusion. Because patients may not have a palpable pulse, adequate perfusion is defined as normal skin color and temperature or normal capillary refill being present.
- Determine if the LVAD is working. If so, is the MAP greater than 50 mm Hg? (If a noninvasive BP cuff is nonfunctional, use a doppler or arterial line, if available.)
- Determine if the P_{ETCO_2} is greater than 20 mm Hg (only when an ET tube or tracheostomy is used to ventilate the patient because use of a supraglottic airway results in a falsely elevated P_{ETCO_2} value).
    - –If yes
        - Do not perform external chest compressions.
        - Assess and treat non-LVAD causes of unresponsiveness.
        - Continue to monitor perfusion.
        - Transport and contact a VAD center.
    - –If no
        - Perform external chest compressions.
        - Assess LVAD function if a second emergency responder is available.
        - Assess and treat non-LVAD causes for unresponsiveness.
        - Continue BLS/ALS.
        - Transport and contact a VAD center.

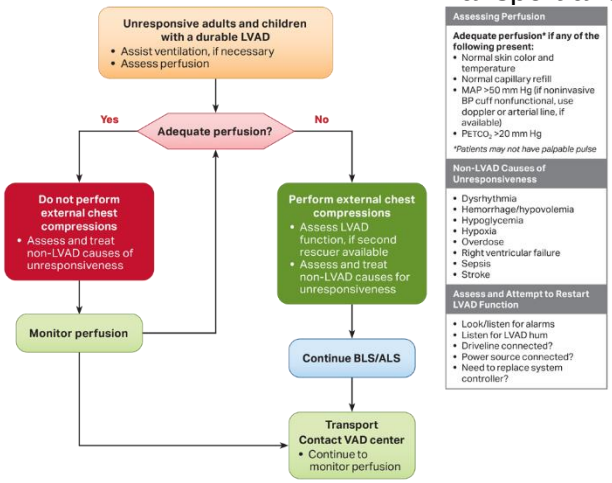


Figure 60. Adult and Pediatric Durable LVAD Algorithm.

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 do-not-attempt-resuscitation 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 jewelry 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 ED.

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, emergency responders must recognize that their patient may be in a state of pseudo-PEA and not have a palpable pulse or measurable BP yet have poor perfusion due to hypotension. If there is adequate mental status, an emergency responder 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 health care professionals (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.

Cardiac Arrest Associated With Pregnancy

Background

During attempted resuscitation of a pregnant patient, the emergency responders have 2 potential patients—the pregnant patient and the fetus. The best hope for fetal survival is maternal survival. For the critically ill pregnant patient, emergency responders must provide appropriate resuscitation with consideration of the physiologic changes due to pregnancy.

The Second Patient

A cardiovascular emergency in a pregnant patient creates a special situation for the ACLS provider. You must always consider the fetus when an adverse cardiovascular event occurs in a pregnant patient. 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 a perimortem cesarean delivery must be made quickly when the pregnant patient is in cardiac arrest. Emergency cesarean delivery—also known as *resuscitative hysterotomy or delivery*—may improve the outcome for both the pregnant patient and child.

Key Interventions: Helping To Prevent Cardiac Arrest in Pregnancy

To treat the critically ill pregnant patient

- Place the patient in the left lateral decubitus position (see Figure 61 for an example) 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]

Figure 61. Example of supporting a patient in the left lateral decubitus position.

If cardiac arrest occurs, refer to the following steps from the Cardiac Arrest in Pregnancy Algorithm (Figure 62):

- Start BLS or ACLS:
    - –Provide high-quality CPR.
    - –Provide continuous left lateral uterine displacement when the fundal height is at or above the umbilicus.
    - –Use an AED or a defibrillator when indicated.
- Activate the cardiac arrest in pregnancy team, and consider the etiology of the arrest.
- Optimize resuscitation in pregnancy:
    - –Prioritize early airway management.

- –Place IV access above the diaphragm.
- –If the patient is receiving IV magnesium, stop the magnesium and give calcium.
- –Detach fetal monitors.
- –Activate massive blood transfusion protocol if amniotic fluid embolism is suspected.

- Prepare for resuscitative delivery when fundal height is at or above the umbilicus.
- Perform resuscitative delivery.
  - –Deliver by 5 minutes if without ROSC.
- Continue ALS care.
- Resuscitate neonate using the neonatal resuscitation algorithm.

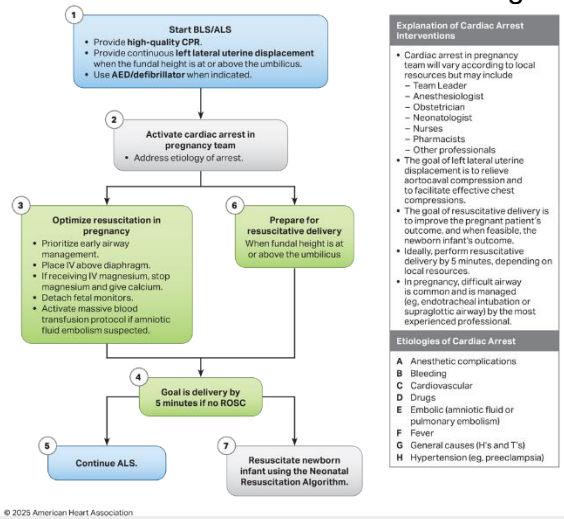


Figure 62. Cardiac Arrest in Pregnancy Algorithm.

Consider the potential etiology and reversible causes of cardiac arrest, and identify any preexisting medical conditions that may be complicating the resuscitation, such as

- Anesthetic complications
- Bleeding
- Cardiovascular complications
- Drugs
- Embolic complications (amniotic fluid or PE)
- Fever

- General causes of cardiac arrest (H's and T's)
- Hypertension (eg, preeclampsia)

Advanced Airway

In pregnancy, a difficult airway is common and should be managed with an advanced airway device by the most experienced health care professional. Provide ET intubation or supraglottic advanced airway. Perform continuous 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 pregnant patient 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.[47]

Pregnant 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,[48] 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,[49] they result in decreased CPR quality (less forceful chest compressions) than is possible in the supine position.[50] Manual left lateral uterine displacement effectively relieves aortocaval pressure in patients with hypotension (Figure 63).[51]

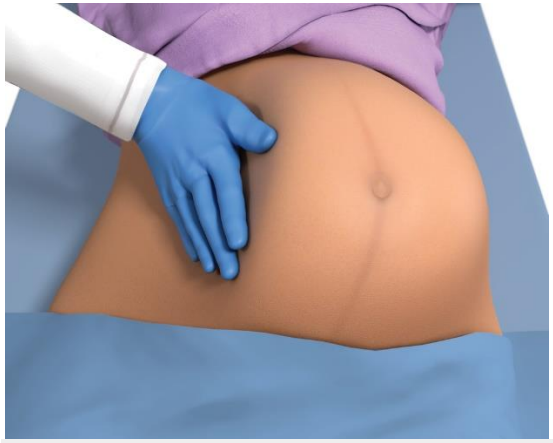


Figure 63A. Manual left uterine displacement. A, Performed with the 2-handed technique.

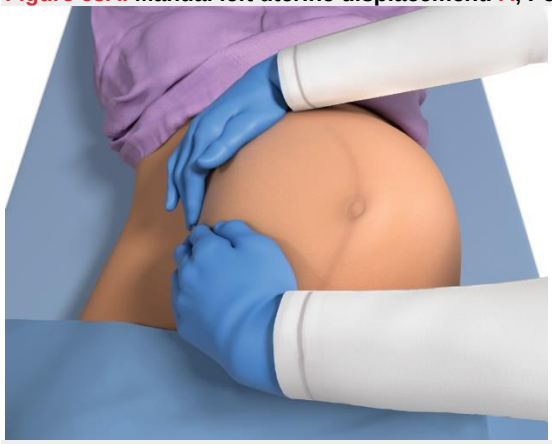


Figure 63B. Performed with the 1-handed technique during resuscitation.

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 63A), and pull the gravid uterus leftward and upward toward your abdomen.

- If it is not possible to stand on the left side of the patient, use one hand to push the gravid uterus (Figure 63B) 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 pregnant patient 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,[48] chest compressions performed in the tilted position are less forceful when compared with chest compressions performed in the supine position.[50] 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 Patients

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 pregnant patient in the second half of pregnancy.[52] Systematic preparation and training are the keys to a successful response to such infrequent 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.[47]

ACLS for Drowning

Drowning is the third leading cause of death from unintentional injury worldwide, accounting for 7% of all injury-related deaths.[53] Physical trauma, such as head injuries, spinal injuries, and fractures from accidents (eg, diving and boating accidents), can impair a person's ability to remain conscious, swim, or stay afloat, increasing the risk of drowning. Drowning generally progresses from initial respiratory arrest due to submersion-related hypoxia to cardiac arrest; thus, it can be challenging to distinguish respiratory arrest from cardiac arrest because pulses are difficult to accurately palpate within the recommended 10-second window. Therefore, resuscitation from cardiac arrest attributable to this specific circumstance must focus on restoring breathing as much as it does circulation.[54]

Drowning is a process resulting in primary respiratory impairment from submersion or immersion in liquid. Implicit in this definition is that a liquid interface is present at the entrance of the patient's airway, preventing the patient from breathing air. The drowning process is a continuum that begins when a patient's airway lies below the surface of liquid, usually water, at which time the person voluntarily holds their breath. Breath-holding is usually followed by an involuntary period of laryngospasm secondary to the presence of liquid in the oropharynx or larynx.[55] During this period of breath-holding and laryngospasm, the patient is unable to breathe air. This results in oxygen being depleted and CO_2 not being eliminated. The patient then becomes hypercarbic, hypoxemic, and acidotic.[53] During this time, the patient will frequently swallow large amounts of water.[54] The patient's respiratory movements may become very active, but there is no exchange of air because of the obstruction at the level of the larynx. As the patient's arterial oxygen tension drops further, laryngospasm abates, and the patient actively breathes liquid.[56]

The most important and detrimental consequence of drowning is hypoxia; therefore, oxygenation, ventilation, and perfusion should be restored as quickly as possible. This will require immediate lay rescuer CPR and activation of the EMS system.

Emergency responders should check to make sure the scene is safe before assessing and treating the patient. After removing the patient from the water (if not already done), perform the BLS assessment to determine the next appropriate steps depending on the patient condition (ie, breathing, not breathing but has a pulse, cardiac arrest). If the patient is in cardiac arrest, start CPR with breaths first due to the hypoxic nature of the incident. Use of an AED is reasonable but should not delay the start of CPR. Also, the initiation of CPR should not be delayed to retrieve or apply an AED in cardiac arrest after drowning. Emergency responders should provide supplemental oxygen if it is available to patients with cardiac arrest after drowning.

Post–Cardiac Arrest Care

Overview

Health care professionals increasingly recognize that systematic post–cardiac arrest care after ROSC, including a bundle of care, 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.[57,58] Studies also show that most deaths occur during the first 24 hours after resuscitation from cardiac arrest,[4,59] 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.[60,61]

The immediate goal after ROSC is to prevent rearrest by identifying and treating life-threatening problems. A growing body of research focuses on identifying and optimizing practices that improve the outcomes of patients who achieve ROSC after

cardiac arrest.[62] Merely restoring BP 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 medications, and invasive devices. In addition, temperature control 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.[63-65] 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.

Rhythms for Post–Cardiac Arrest Care

You will need to recognize the following rhythms with regard to

- Rate: too fast or too slow
- Width of QRS complexes: wide vs narrow

Medications for Post–Cardiac Arrest Care

Medications for post–cardiac arrest care include

- Epinephrine
- Dopamine
- Norepinephrine infusions
- Other medications as needed depending on presentation

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, temperature control, 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.

Post–cardiac arrest patients may be awake and able to follow commands or be unconscious and unable to follow commands. For unconscious patients, ensure an adequate airway and support breathing immediately after ROSC because these patients usually require an advanced airway for mechanical support of breathing. Also, elevate the head of their 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 continuous waveform capnography, and continuously monitor the patient's oxygenation with pulse oximetry.

Brain injury and cardiovascular instability are the major factors that determine survival after cardiac arrest.[66] Because temperature control is currently the only intervention demonstrated to improve neurologic recovery, consider temperature control for any patient who is comatose and unresponsive to verbal commands after ROSC. Obtain a CT brain scan, have electroencephalogram 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,[67,68] 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 emergency 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 temperature control 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 temperature control, 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 temperature control. For those treated with temperature control, 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,[63,69,70] 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

Post–cardiac arrest care focuses on managing and optimizing cardiopulmonary function and perfusing vital organs after ROSC. The Adult Post–Cardiac Arrest Care Algorithm (Figure 64) outlines the steps to immediately assess and manage post–cardiac arrest patients with ROSC. Health care professionals should, during initial patient stabilization after ROSC, manage the airway, continue to maintain good oxygen and ventilation, and manage hemodynamics. Additionally, early diagnostic testing starting with a 12-lead ECG and other diagnostic testing (eg, CT or ultrasound) should be considered. As management continues, health care professionals will need to consider the possible arrest etiologies and complications that could have contributed to the cardiac arrest and consider emergency coronary angiography or mechanical circulatory support. From there, teams will need to decide if the patient is following commands or not and tailor treatments and interventions accordingly.

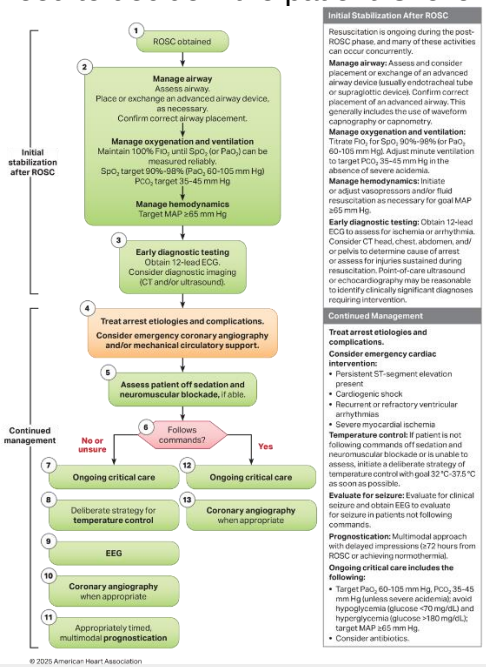


Figure 64. Adult Post–Cardiac Arrest Care Algorithm.

Application of the Adult Post–Cardiac Arrest Care Algorithm

Health care professionals 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 64). 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.

Managing the Airway

Post–cardiac arrest patients may be awake and able to follow commands or unconscious and unable to follow commands. Step 2 of the algorithm directs you to assess and consider placement or exchange of an advanced airway device (usually an ET tube or a supraglottic device). Confirm correct placement of an advanced airway. This generally includes the use of continuous waveform capnography or capnometry (Figures 65 and 66). Continue to monitor the placement of the advanced airway (particularly during patient transport) by continuous waveform capnography, and continuously monitor the patient's oxygenation with pulse oximetry.

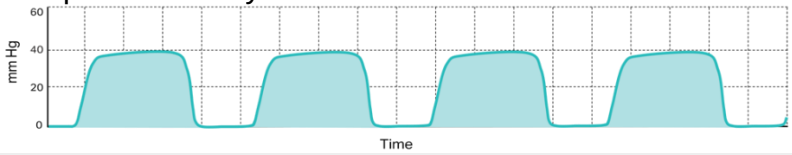


Figure 65A. Waveform capnography. A, Normal range of 35 to 45 mm Hg.

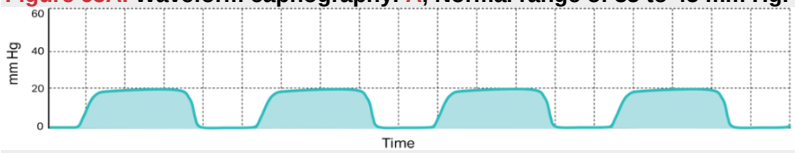


Figure 65B. 20 mm Hg.

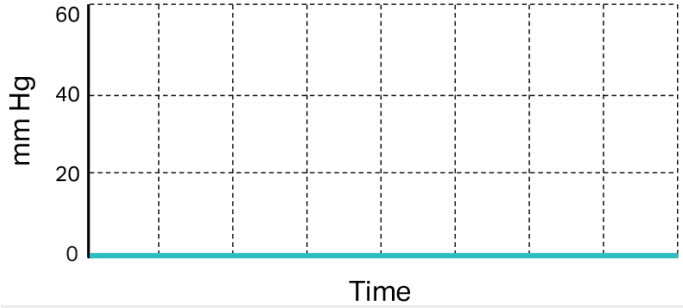


Figure 65C. 0 mm Hg.

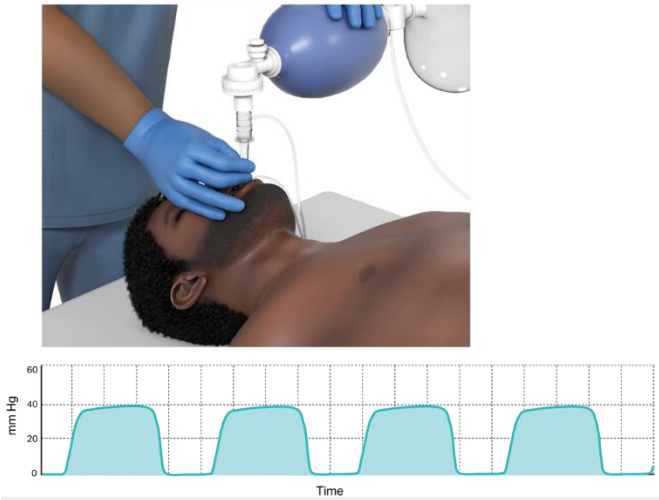


Figure 66. Waveform capnography with an ET showing a normal (adequate) ventilation pattern of $PETCO_2$ 35 to 45 mm Hg.

Manage Oxygenation and Ventilation

Step 2 directs you to ensure an adequate airway and support breathing immediately after ROSC. An unconscious or unresponsive patient requires an advanced airway to mechanically support breathing.

To manage oxygenation and ventilation, health care professionals should titrate fraction of inspired oxygen (FIO_2) for peripheral oxygen saturation (SpO_2) 90% to 98% (or PaO_2 60-105 mm Hg). Adjust minute ventilation to target PCO_2 35 to 45 mm Hg in the absence of severe acidemia.

To avoid hypoxia in adults with ROSC after cardiac arrest, you may use the highest available oxygen concentration until you can reliably measure the arterial oxyhemoglobin saturation or the partial pressure of arterial oxygen, if the appropriate equipment is available. Once reliable measurement is available, titrate the fraction of inspired oxygen to maintain the oxyhemoglobin saturation of 90% to 98%.

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.

Continuous Waveform Capnography

$ETCO_2$ is the concentration of CO_2 in exhaled air at the end of expiration, typically expressed as a partial pressure (P_{ETCO_2}) in millimeters of mercury. There are 2 types of capnography devices: mainstream and sidestream. Mainstream devices measure the CO_2 directly on the airway and send the signal back to the device to display. Sidestream devices sample the gas from the airway and measure 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, P_{ETCO_2} correlates well with cardiac output during CPR.

Observe a continuous capnographic waveform with ventilation to confirm and monitor ET tube placement in the field, in the transport vehicle, upon arrival at the ED, 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.

Manage Hemodynamics

Hypotension should be avoided in adults after cardiac arrest by maintaining a target MAP of 65 mm Hg or greater. Obtain IV access if not already established, and verify that any IV lines are open. Initiate or adjust vasopressors or fluid resuscitation as necessary for goal MAP 65 mm Hg or greater.

Hypotension should be treated with the following steps:

- **IV bolus:** 1 to 2 L normal saline or lactated Ringer's solution
    - –While considering other post–cardiac arrest factors, use IV fluids to the point that adequate volume status is achieved.
- **Norepinephrine:** 0.1 to 0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg/min) IV infusion adjusted to achieve a minimum SBP of greater than 90 mm Hg or a MAP 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 MAP 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 MAP 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.

Early Diagnostic Testing

After initial stabilization of airway, oxygenation or ventilation, and hemodynamics, obtain 12-lead ECG to assess for ischemia or arrythmia. Both in-hospital and out-of-hospital health care professionals should obtain a 12-lead ECG as soon as possible after ROSC to identify those patients with STEMI or patients with a high suspicion of AMI (Step 3).

Emergency responders should transport these patients to a facility that reliably provides emergency coronary angiography (Step 4).

Health care professionals should also consider CT scans of the head, chest, abdomen, and pelvis to determine the cause of arrest or assess for injuries sustained during resuscitation. Point-of-care ultrasound or echocardiography may be reasonable to identify clinically significant diagnoses requiring intervention.

Treat Arrest Etiologies and Complications

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.

Health care professionals should also consider emergency cardiac interventions for patients with the presence of persistent ST-segment elevation, cardiogenic shock, recurrent or refractory ventricular arrythmias, or severe myocardial ischemia.

In cases of out-of-hospital STEMI, provide advance notification to receiving facilities.

Following Commands

Step 6 directs you to examine the patient's ability to follow verbal commands. If possible, this is best done with the patient off sedation and neuromuscular blockade. If the patient is following commands, continue ongoing critical care and consider delayed coronary angiography as appropriate. Key aspects of ongoing critical care include titrating oxygenation and ventilation to a target Pa_{O_2} 60 to 105 mm Hg and P_{CO_2} 35 to 45 mm Hg (unless severe acidemia), avoiding hypoglycemia (glucose less than 70 mg/dL) and hyperglycemia (glucose greater than 180 mg/dL), maintaining hemodynamics at a target MAP of 65 mm Hg or greater, and considering administration of prophylactic antibiotics in selected cases.

For those patients who do not follow commands, the high-performance team should continue ongoing critical care as above but also begin a deliberate strategy of temperature control, perform electroencephalogram monitoring, and consider delayed coronary angiography as appropriate (Steps 7, 8, 9, and 10).

Temperature Control

The AHA has redefined the concept of targeted temperature management to *temperature control*, thereby including strategies of hypothermic temperature control, normothermic temperature control, and temperature control with fever prevention. Temperature control includes choosing one temperature between **32 °C** (89.6 °F) and **37.5 °C** (99.5 °F) and then holding that temperature for **at least 36 hours**. This recommendation applies to all adults after ROSC, regardless of arrest location (in-hospital or out-of-hospital) or presenting rhythm, who do not follow verbal commands. Patients with spontaneous hypothermia after ROSC who are unresponsive to verbal commands should not routinely be actively or passively rewarmed faster than 0.5 °C per hour. It is also recommended that hospitals develop protocols for postarrest temperature control.

Emergency responders should not use rapid infusion of cold IV fluids for prehospital cooling of patients after ROSC. IV fluid administration in the prehospital setting may increase pulmonary edema and rearrest. Other alternative ways of performing temperature control, such as applying ice packs, can be used. Once a temperature within the recommended range (32-37.5 °C [89.6-99.5 °F]) is achieved, it should be maintained for at least 36 hours (through delivery into the ED).

Temperature control is the only intervention demonstrated to improve neurologic recovery after cardiac arrest. The optimal duration of temperature control is at least 36 hours; however, this is an important area of ongoing research.

During temperature control, 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.

For temperature control, health care professionals should select and maintain a constant target temperature between 32 °C (89.6 °F) and 37.5 °C (99.5 °F) for at least 36 hours. Although the optimal method of achieving the target temperature is unknown, use of endovascular catheters, surface cooling devices, or simple surface interventions appears to be safe and effective.

Specific patient features may necessitate selecting one temperature over another for temperature control. 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 (89.6 °F) and 37.5 °C (99.5 °F) is not contraindicated in any patients, so all patients who require intensive care are eligible for this treatment.

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

Continued management of patients during postresuscitation care:

- **Evaluate for seizure:** Monitor for clinical seizures and obtain electroencephalogram to evaluate for seizures in patients not following commands. Treat all clinically apparent seizures after arrest and, in nonconvulsive seizures diagnosed by electroencephalogram only, it is reasonable to treat these seizures. Seizures after arrest can be treated with the same antiseizure medications used to treat seizures caused by other etiologies. Importantly, routine seizure prophylaxis in adult survivors of cardiac arrest is not recommended.
- **Glucose management:** In adult patients with cardiac arrest who have achieved ROSC, it may be reasonable to avoid hypoglycemia (glucose less than 70 mg/dL) as well as hyperglycemia (glucose greater than 180 mg/dL).
- **Prophylactic antibiotics:** The routine use of prophylactic antibiotics in postarrest patients is of uncertain benefit. However, in specific situations with clinical suspicion of infection, consider antibiotics.
- **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 currently being 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.

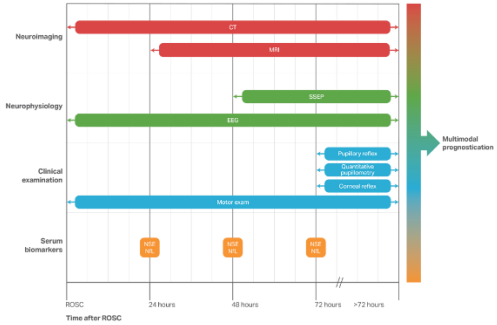
Neuroprognostication

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.[66,71] Most deaths attributable to postarrest brain injury are due to active withdrawal of life-sustaining treatment on the basis of a predicted poor neurologic outcome. Prognostication is in the core of goals of care decisions because surrogates commonly make informed decisions pertaining to care paths relying on the perceived estimated likelihood of recovery that is shared by the treatment team. 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.[72]

Neuroprognostication is a process through which care teams gauge the extent of brain injury burden and estimate the likelihood for recovery to a favorable functional state using tools that assess for evidence of structural injury (eg, neuroimaging, biomarkers) or evaluate neurologic function (eg, neurophysiologic tests, clinical examination). The process of postarrest neuroprognostication begins as soon as ROSC is achieved and continues throughout the hospitalization. The ideal timing for neuroprognostic tests is test-specific and avoids the effects of confounding (eg, from temperature, medications, systemic organ dysfunction). The interpretation of the results of these tests are then consolidated to convey the estimated likelihood for recovery to a favorable or unfavorable state. Most neuroprognostic studies utilize outcome scales in a dichotomized manner for good and poor outcome, with a good functional outcome defined by the ability to achieve independence (ie, Glasgow-Pittsburgh Cerebral Performance Category scores 1-2 or modified Rankin Scale scores 0-3), although some studies have thresholds characterized by recovery of consciousness (ie, Glasgow-Pittsburgh Cerebral Performance Category scores 1-3 or modified Rankin Scale scores 0-4). These studies neither capture granular details on the functional state of patients, nor do they consider the patients' individual preferences or values; therefore, these inherent limitations of the literature are to be considered when applying the recommendations of these guidelines in clinical practice. 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*.

Key Considerations for Neuroprognostication

- In patients who remain comatose after cardiac arrest, neuroprognostication should involve a multimodal approach (Figure 67) and not be based on any single finding.
- In patients who remain comatose after cardiac arrest, neuroprognostic impressions should be delayed. Adequate time should be allowed 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 early, regular, and transparent multidisciplinary discussions with families or surrogates about the anticipated time course for uncertainties around neuroprognostication. Because early neuroprognostic impressions need to be delayed, regular multidisciplinary meetings may be needed to improve communication.
- In patients who remain comatose after cardiac arrest, it is reasonable to consolidate the interpretation of multimodal prognostic assessments at a minimum of 72 hours after normothermia and discontinuation of sedatives.



Neuroprognostic modality	Unfavorable outcome more likely	Favorable outcome more likely
Neuroimaging	• Reduced GWR on CT • Extensive areas of reduced ADC on MRI at 2-7 days after cardiac arrest • Extensive areas of restricted diffusion on MRI at 2-7 days after cardiac arrest	• Absence of restricted diffusion on MRI at 2-7 days after cardiac arrest
Neurophysiology	• Bilaterally absent N20 peaks on SSEP at 48 hours or more after cardiac arrest • Burst suppression at ≥72 hours after cardiac arrest in the absence of sedation • Status epilepticus ≥72 hours after cardiac arrest	• Continuous EEG background without discharges within 72 hours after cardiac arrest
Clinical examination	• Bilaterally absent pupillary light reflex at ≥72 hours after cardiac arrest • Bilaterally absent corneal reflexes at ≥72 hours after cardiac arrest • Decreased response on quantitative pupillometry at ≥72 hours after cardiac arrest	• Withdrawal or better motor response
Serum biomarkers	• High NSE within 72 hours after cardiac arrest • High NfL within 72 hours after cardiac arrest	• Normal NSE within 72 hours after cardiac arrest

Figure 67. Schematic overview of a multimodal approach to neuroprognostication.

Abbreviations: ADC, apparent diffusion coefficient; CT, computed tomography; EEG, electrocardiogram; GWR, gray-white matter ratio; MRI, magnetic resonance imaging; NSE, neuron specific enolase; NfL, neurofilament light; ROSC, return of spontaneous circulation; SSEP, somatosensory evoked potentials.

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Appendix

Testing Checklists, Megacode 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 1 minute (100-120/min) _____ (number of compressions)
- ☐ Compresses at least 2 inches (5 cm)
- ☐ Complete chest recoil. (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) _____ Ventilating once every 4 seconds or less or 8 seconds or more does not pass • Gives proper ventilation speed (over 1 second) • Gives proper ventilation volume (about half a bag)	

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 _____

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/Team Members	
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 ☐ Ventilation rate _____	
Team Leader ensures that team members communicate well	
Bradycardia Management	
Starts oxygen if needed, places monitor, starts IV	
Places monitor leads in proper position	
Recognizes symptomatic/unstable bradycardia	
Administers correct treatment	
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	
Verbalizes need for endotracheal intubation and continuous waveform capnography, ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, and orders laboratory test	
Considers temperature control	

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/Team Members	
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 ☐ Ventilation rate _____	
Team Leader ensures that team members communicate well	
Bradycardia Management	
Starts oxygen if needed, places monitor, starts IV	
Places monitor leads in proper position	
Recognizes symptomatic/unstable 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	
Verbalizes need for endotracheal intubation and continuous waveform capnography, ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, and orders laboratory test	
Considers temperature control	

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/Team Members	
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 ☐ · Ventilation rate _____	
Team Leader 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	
Verbalizes need for endotracheal intubation and continuous waveform capnography, ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, and orders laboratory test	
Considers temperature control	

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/Team Members		
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 ☐ Ventilation rate _____	
Team Leader ensures that team members communicate well		
Bradycardia Management		
Starts oxygen if needed, places monitor, starts IV		
Places monitor leads in proper position		
Recognizes symptomatic/unstable 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		
Verbalizes need for endotracheal intubation and continuous waveform capnography, ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, and orders laboratory test		
Considers temperature control		

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/Team Members						
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 ☐	Ventilation rate _____	
Team Leader 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						
Verbalizes need for endotracheal intubation and continuous waveform capnography, ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, and orders laboratory test						
Considers temperature control						

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 _____

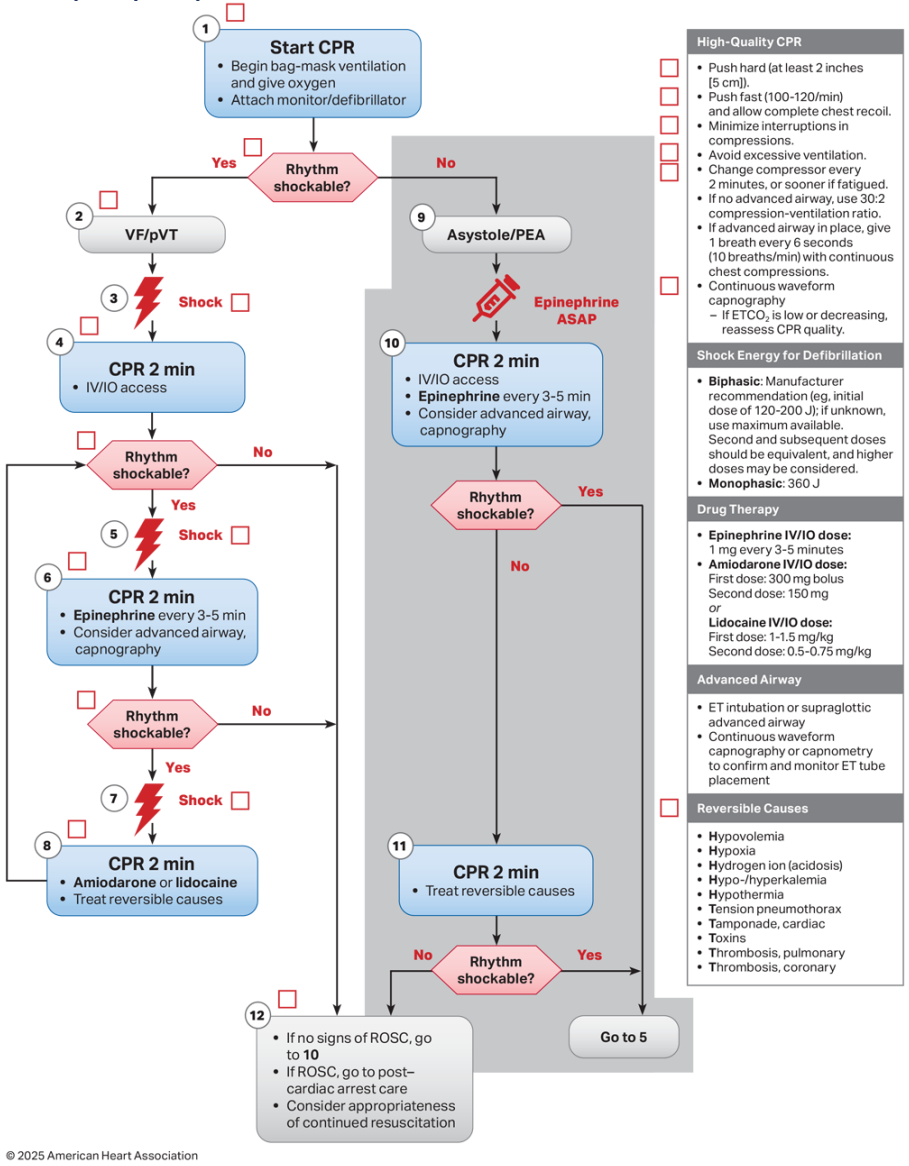
Critical Performance Steps						Check if done correctly
Team Leader/Team Members						
Team Leader assigns team member roles						
Ensures high-quality CPR at all times	Compression rate 100-120/min ☐	Compression depth of ≥2 inches ☐	Chest compression fraction >80% _____%	Chest recoil ☐	Ventilation rate _____	
Team Leader ensures that team members communicate well						
Bradycardia Management						
Starts oxygen if needed, places monitor, starts IV						
Places monitor leads in proper position						
Recognizes symptomatic/unstable 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						
Verbalizes need for endotracheal intubation and continuous waveform capnography, ensures BP and 12-lead ECG are performed and O_2 saturation is monitored, and orders laboratory test						
Considers temperature control						

STOP TEST

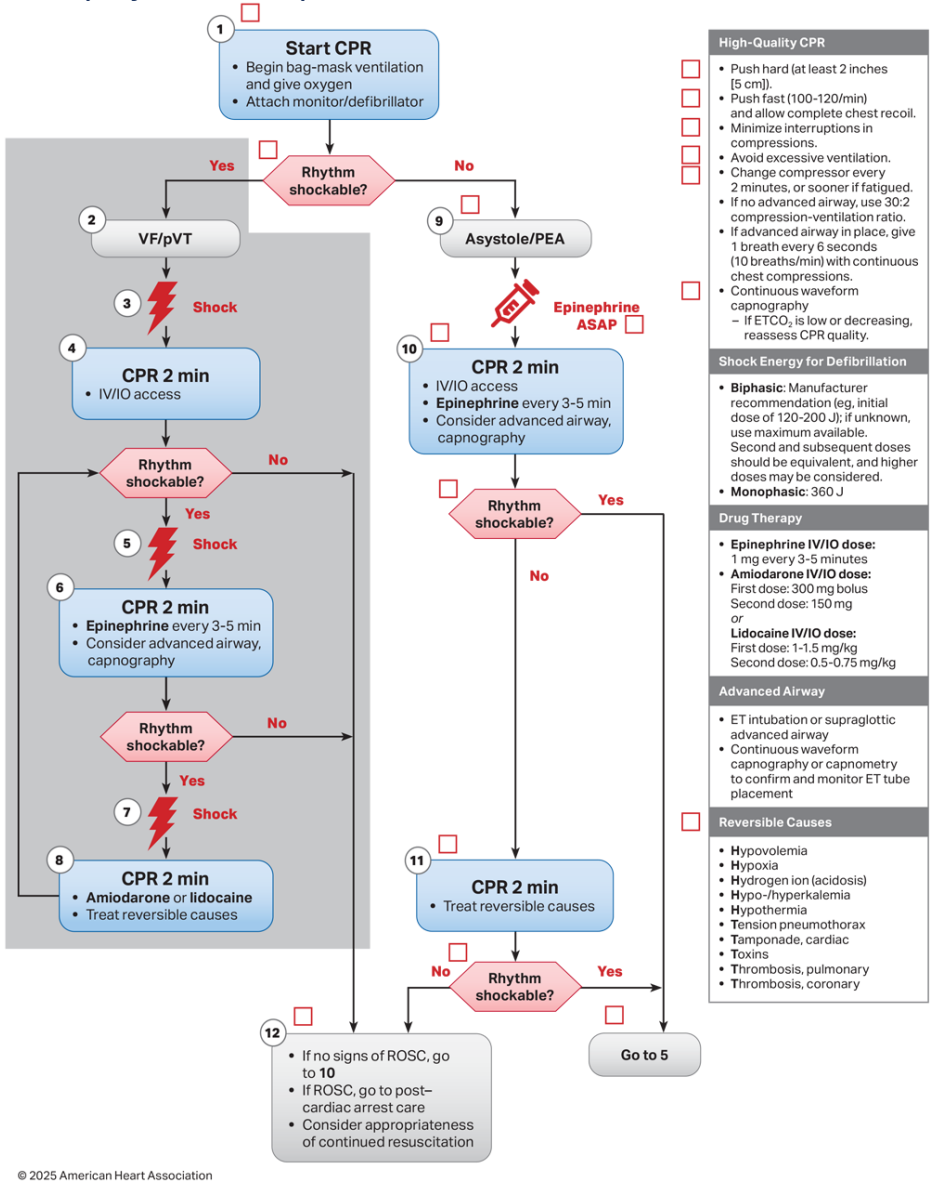
Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**
Instructor Initials _____ Instructor Number _____ Date _____		

Learning Station Competency
☐ Bradycardia ☐ Tachycardia ☐ Cardiac Arrest/Post–Cardiac Arrest Care ☐ Megacode Practice

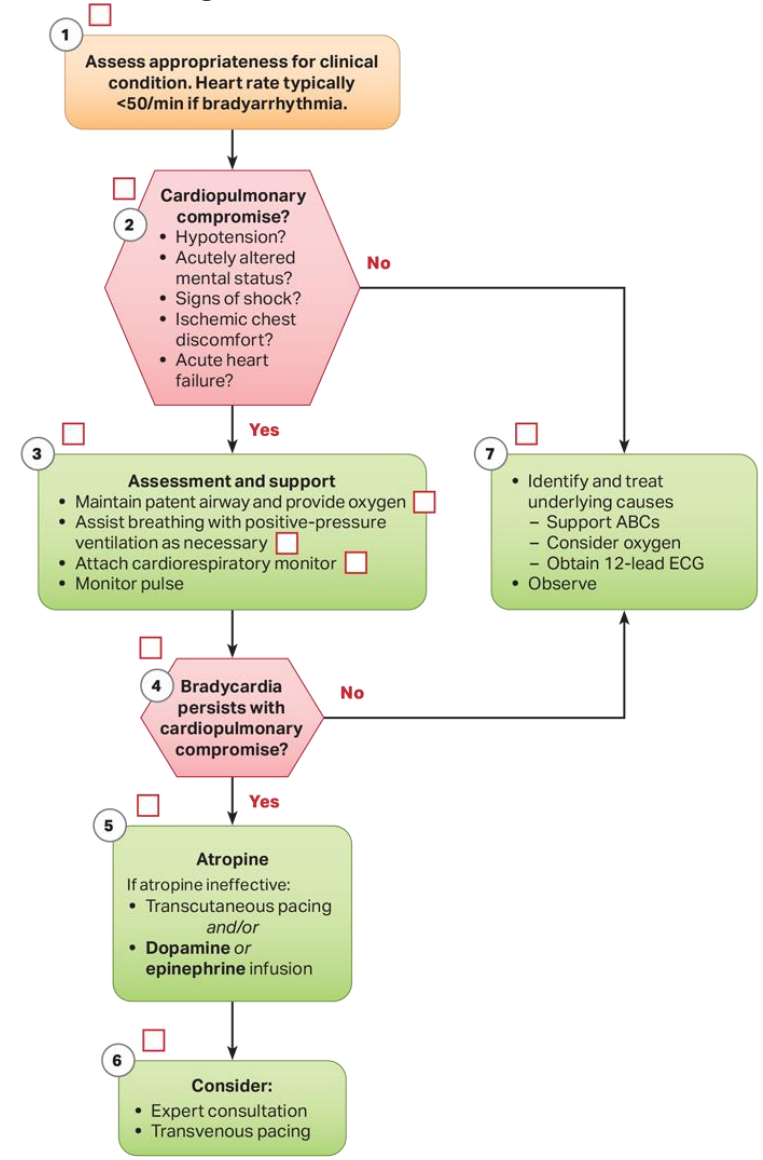
Adult Cardiac Arrest Algorithm (VF/pVT)



Adult Cardiac Arrest Algorithm (Asystole/PEA)



Adult Bradycardia With a Pulse Algorithm



Doses/Details

Atropine IV dose:
First dose: 1 mg bolus.
Repeat every 3-5 minutes.
Maximum total dose: 3 mg.

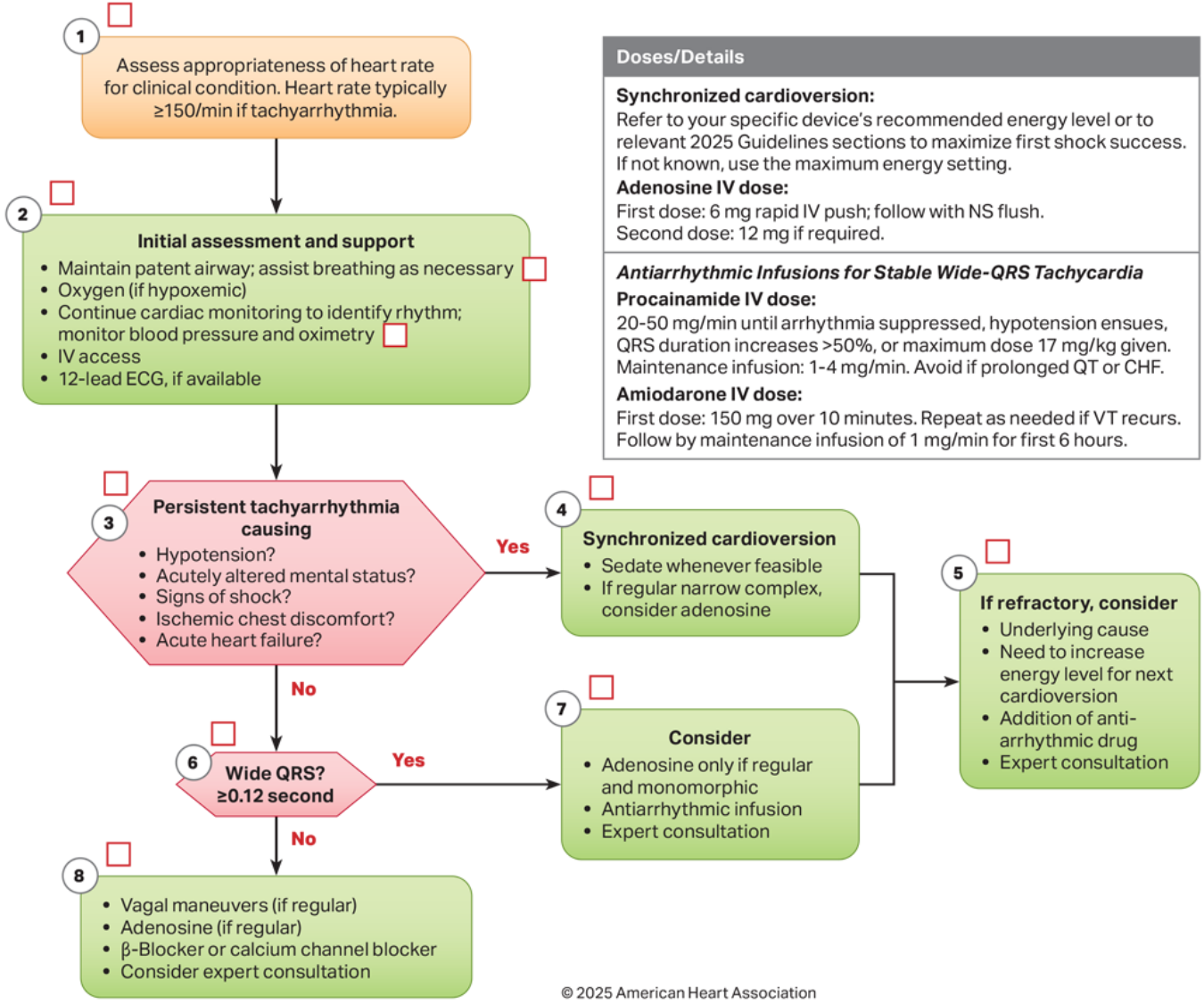
Dopamine IV infusion:
Usual infusion rate is 5-20 mcg/kg per minute.
Titrate to patient response; taper slowly.

Epinephrine IV infusion:
2-10 mcg per minute infusion.
Titrate to patient response.

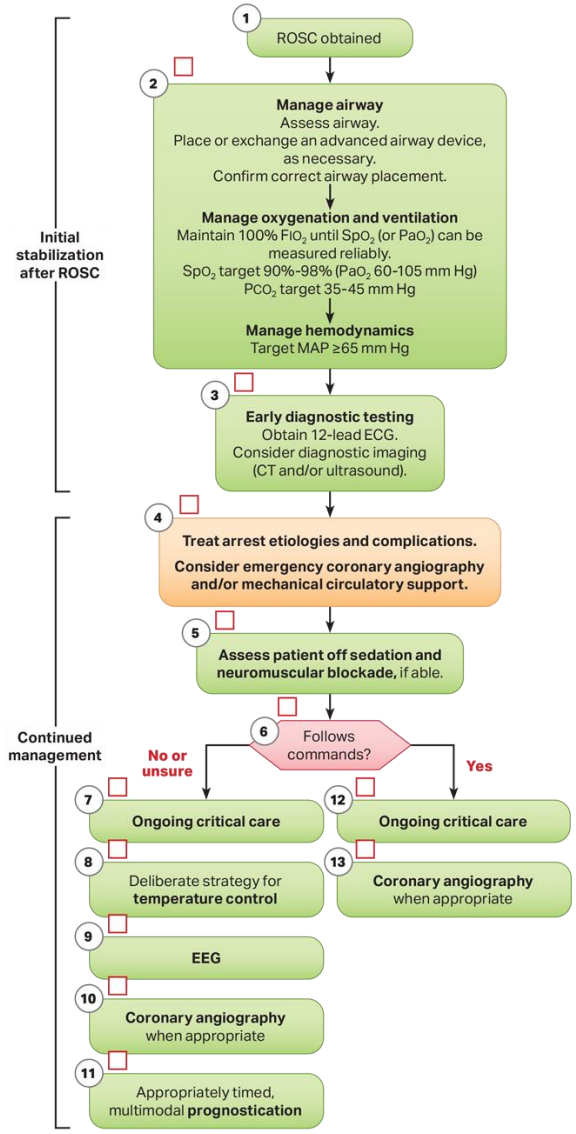
Possible Causes

- Myocardial ischemia/infarction
- Drugs/toxicologic (eg, calcium-channel blockers, β-blockers, digoxin)
- Hypoxia
- Electrolyte abnormality (eg, hyperkalemia)

Adult Tachyarrhythmia With a Pulse Algorithm



Adult Post–Cardiac Arrest Care Algorithm



Initial Stabilization After ROSC

Resuscitation is ongoing during the post-ROSC phase, and many of these activities can occur concurrently.

Manage airway: Assess and consider placement or exchange of an advanced airway device (usually endotracheal tube or supraglottic device). Confirm correct placement of an advanced airway. This generally includes the use of waveform capnography or capnometry.

Manage oxygenation and ventilation: Titrate FIO_2 for SpO_2 90%-98% (or PaO_2 60-105 mm Hg). Adjust minute ventilation to target PCO_2 35-45 mm Hg in the absence of severe acidemia.

Manage hemodynamics: Initiate or adjust vasopressors and/or fluid resuscitation as necessary for goal MAP ≥65 mm Hg.

Early diagnostic testing: Obtain 12-lead ECG to assess for ischemia or arrhythmia. Consider CT head, chest, abdomen, and/or pelvis to determine cause of arrest or assess for injuries sustained during resuscitation. Point-of-care ultrasound or echocardiography may be reasonable to identify clinically significant diagnoses requiring intervention.

Continued Management

Treat arrest etiologies and complications.

Consider emergency cardiac intervention:

- Persistent ST-segment elevation present
- Cardiogenic shock
- Recurrent or refractory ventricular arrhythmias
- Severe myocardial ischemia

Temperature control: If patient is not following commands off sedation and neuromuscular blockade or is unable to assess, initiate a deliberate strategy of temperature control with goal 32 °C-37.5 °C as soon as possible.

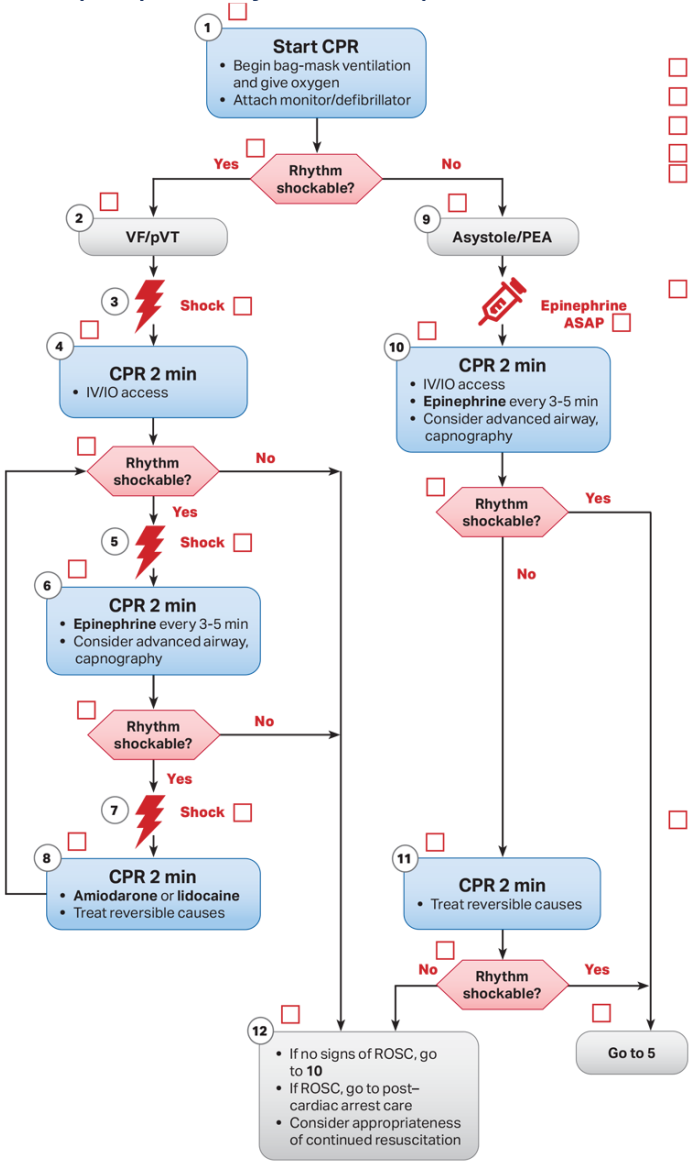
Evaluate for seizure: Evaluate for clinical seizure and obtain EEG to evaluate for seizure in patients not following commands.

Prognostication: Multimodal approach with delayed impressions (≥72 hours from ROSC or achieving normothermia).

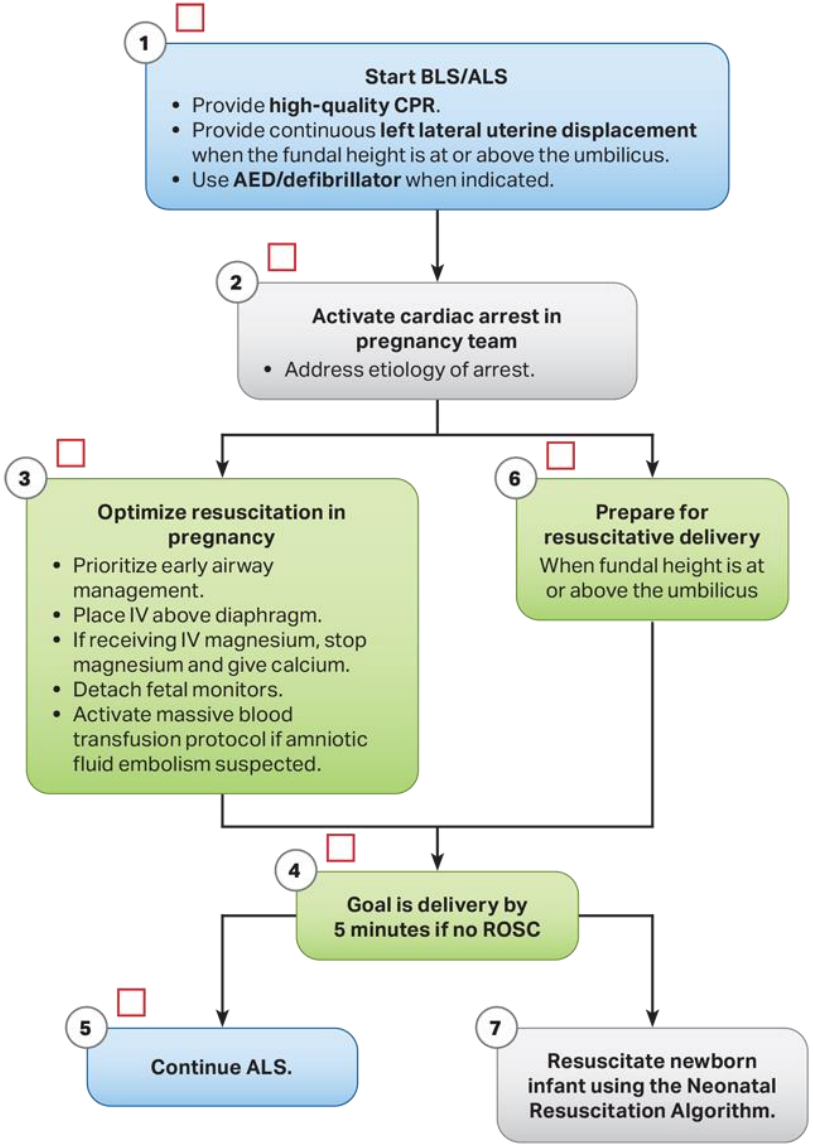
Ongoing critical care includes the following:

- Target PaO_2 60-105 mm Hg, PCO_2 35-45 mm Hg (unless severe acidemia); avoid hypoglycemia (glucose <70 mg/dL) and hyperglycemia (glucose >180 mg/dL); target MAP ≥65 mm Hg.
- Consider antibiotics.

Adult Cardiac Arrest Algorithm (VF/pVT/Asystole/PEA)



Cardiac Arrest in Pregnancy Algorithm



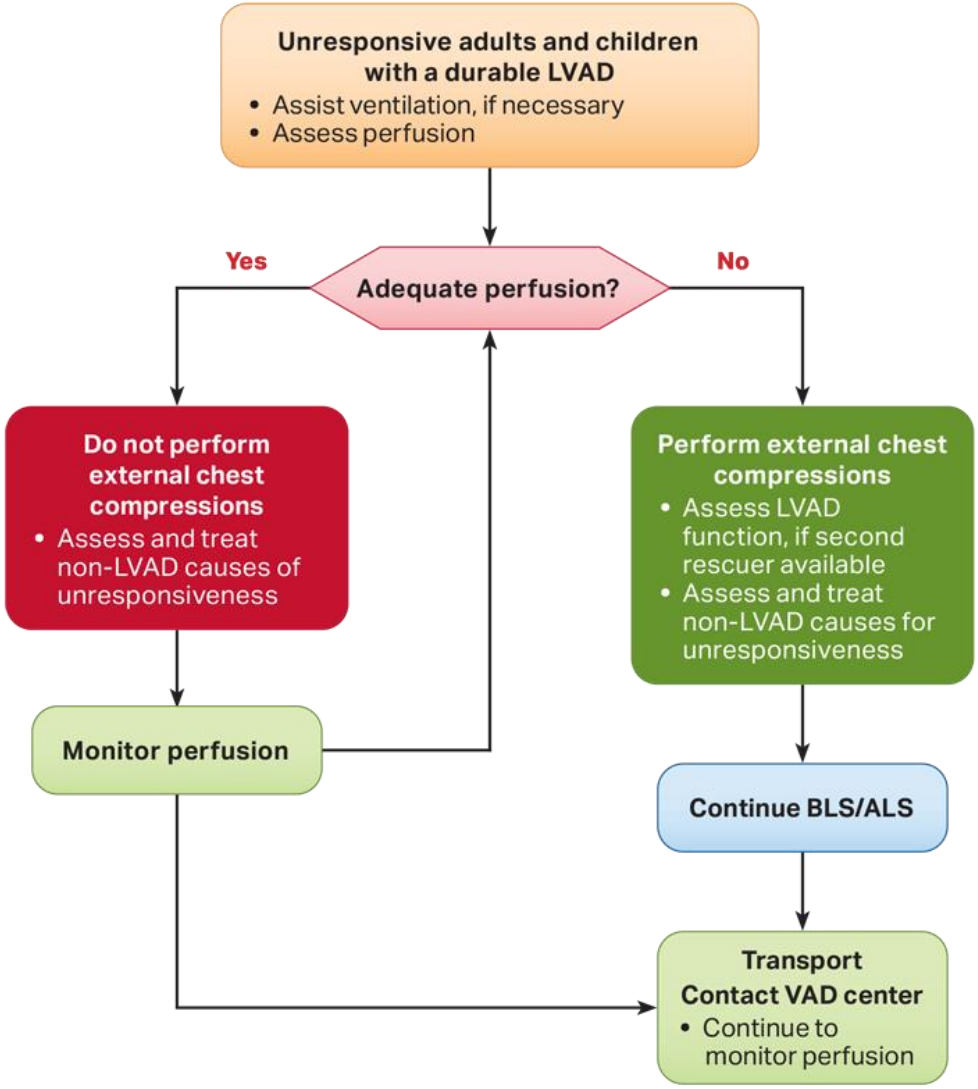
Explanation of Cardiac Arrest Interventions

- Cardiac arrest in pregnancy team will vary according to local resources but may include:
  - Team Leader
  - Anesthesiologist
  - Obstetrician
  - Neonatologist
  - Nurses
  - Pharmacists
  - Other professionals
- The goal of left lateral uterine displacement is to relieve aortocaval compression and to facilitate effective chest compressions.
- The goal of resuscitative delivery is to improve the pregnant patient's outcome, and when feasible, the newborn infant's outcome.
- Ideally, perform resuscitative delivery by 5 minutes, depending on local resources.
- In pregnancy, difficult airway is common and is managed (eg, endotracheal intubation or supraglottic airway) by the most experienced professional.

Etiologies of Cardiac Arrest

A Anesthetic complications
B Bleeding
C Cardiovascular
D Drugs
E Embolic (amniotic fluid or pulmonary embolism)
F Fever
G General causes (H's and T's)
H Hypertension (eg, preeclampsia)

Adult and Pediatric Durable Left Ventricular Assist Device (LVAD) Algorithm



Assessing Perfusion

Adequate perfusion* if any of the following present:

- Normal skin color and temperature
- Normal capillary refill
- MAP >50 mm Hg (if noninvasive BP cuff nonfunctional, use doppler or arterial line, if available)
- $PETCO_2$ >20 mm Hg

**Patients may not have palpable pulse*

Non-LVAD Causes of Unresponsiveness

- Dysrhythmia
- Hemorrhage/hypovolemia
- Hypoglycemia
- Hypoxia
- Overdose
- Right ventricular failure
- Sepsis
- Stroke

Assess and Attempt to Restart LVAD Function

- Look/listen for alarms
- Listen for LVAD hum
- Driveline connected?
- Power source connected?
- Need to replace system controller?

ACLS Pharmacology Summary Table

This table provides information about common medications used in ACLS.

Medication	Indications	Precautions and contraindications	Adult dosage
Adenosine	• First medication for most forms of stable narrow-complex tachycardia; 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 monomorphic wide-complex tachycardia, thought to be or previously defined to be reentry narrow-complex tachycardia • Does not convert atrial fibrillation, atrial flutter, or VT • Diagnostic maneuver: stable narrow-complex tachycardia	• Contraindicated in poison-/medication-induced tachycardia or second- or third-degree heart block • Avoid in patients with known or suspected bronchoconstriction, bronchospasm, or asthma • Transient side effects include flushing, chest pain or tightness, brief periods of asystole or bradycardia, ventricular ectopy • Less effective (larger doses may be 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)	**IV Rapid Push** • Place patient in mild reverse Trendelenburg position before administration of medication • Initial bolus of 6 mg given rapidly over 1-3 seconds followed by NS bolus of 20 mL; then elevate the extremity • A second dose (12 mg) can be given in 1-2 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

		• Transient periods of sinus bradycardia and ventricular ectopy are common after termination of narrow-complex tachycardia • Safe and effective in pregnancy	adenosine closest to patient; clamp IV tubing above injection port • Push IV adenosine as quickly as possible (1-3 seconds) • While maintaining pressure on adenosine plunger, push NS flush as rapidly as possible after adenosine • Unclamp IV tubing
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. ***Caution*: Multiple complex medication interactions**	• 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 medications that prolong QT interval (eg, procainamide) • Terminal elimination is extremely long (half-life)	**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)
Atropine sulfate	• First medication for symptomatic sinus bradycardia • May be beneficial in presence of AV nodal block; **not likely to be effective for type II second- or third-degree AV block or a block in nonnodal tissue** • Routine use during PEA or asystole is unlikely to have a therapeutic benefit • Organophosphate (eg, nerve agent) poisoning: extremely large doses	• Atropine has neurologic effects, including dilated pupils, tremor, and delirium • 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 may cause paradoxical slowing; be prepared to pace or give catecholamines) • Do not give to heart transplant patients	**Bradycardia (With or Without ACS)** 1 mg IV every 3-5 minutes as needed, not to exceed total dose of 0.04 mg/kg (total 3 mg) **Organophosphate Poisoning** Extremely large doses (2-4 mg or higher) may be needed
Dopamine IV infusion	• Second-line medication for symptomatic bradycardia (after atropine) • Use for hypotension (SBP ≤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 congestive heart failure	**IV Administration** • Usual infusion rate is 5-20 mcg/kg per minute • Titrate to patient response; taper slowly

		• May cause tachyarrhythmias, excessive vasoconstriction • Do not mix with sodium bicarbonate	
Epinephrine Available in 0.1 mg/mL and 1 mg/mL concentrations	• **Cardiac arrest:** VF, pVT, 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:** Administer lower dose IM early and monitor. Combine with large fluid volume, corticosteroids, antihistamines	• Raising BP and increasing 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- or medication-induced shock	**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 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 of 0.1-0.5 mcg/kg per minute (for 70-kg patient, 7-35 mcg/min); titrate to response **Profound Bradycardia or Hypotension** 2-10 mcg/min infusion; titrate to patient response **Anaphylaxis**

			Initial dose 0.3-0.5 mg IM in lateral thigh. Repeat if symptoms persist/recur. If continued symptoms, initiate continuous IV infusion.
Lidocaine	• 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 left ventricular 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 left ventricular 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 to 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 of 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	• Occasional fall in BP with rapid administration	**Cardiac Arrest (Due to Hypomagnesemia or 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	• Use with caution if renal failure is present	1-2 g (2-4 mL of a 50% solution diluted in 10 mL [eg, D_5W, 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, D_5W, normal saline) over 5-60 minutes IV • Follow with 0.5-1 g/hour IV (titrate to control torsades)

Science Summary Table

This table compares topics from 2020 with 2025, providing a quick reference to what has changed and what is new in the science of ACLS.

ACLS topic	2020	2025
Tachycardia	• 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)	• Synchronized cardioversion initial recommended doses: ○ –Narrow-complex tachycardia: 100 J ○ –Monomorphic VT: 100 J ○ –Atrial fibrillation: 200 J ○ –Atrial flutter: 200 J ○ –Polymorphic VT: defibrillation dose (not synchronized) • Removed sotalol from the algorithm • Changed supraventricular tachycardia to narrow-complex tachycardia

Post–Cardiac Arrest Care	• Targeted temperature management
	  ○ –32-36 °C
	  ○ –Hold temperature for 24 hours
	  ○ –Do not give OHCA patients with ROSC targeted temperature management
	• Hypotension: <90 mm Hg
	• Oxygen saturation: 92%-98%
		• Temperature control
		  ○ –32-37.5 °C
		  ○ –Hold temperature for at least 36 hours
		  ○ –OK to give OHCA patients with ROSC temperature control as long as it is not cold IV fluids
		• Hypotension: MAP ≥65 mm Hg
		• Oxygen saturation: 90%-98%

Cardiac Arrest, Chain of Survival	• 6 links for both chains (IHCA and OHCA): added a Recovery link to the end of both chains	• 6 links for 1 universal chain
ACLS topic	**2025**	
Stroke	• Adding tenecteplase as a thrombolytic agent	
ACS	• Removed LBBB as a definitive diagnosis for STEMI • Removing clopidogrel as primary anticoagulant • Adding fentanyl (opioids) for secondary pain control (in addition to morphine) • Adding enoxaparin or fondaparinux (anticoagulants) • Adding ACE inhibitors	
Airway	• Removed 600-800 mL for ventilations, adding “one third” squeeze and focusing on chest rise. “Squeeze the bag one third and one half, enough to see visible chest rise.” • Removed delivering medications down an ET tube	

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 cardiopulmonary resuscitation are supplemented with medication administration, intravenous 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 that is used to analyze heart rhythm and, if an appropriate rhythm is detected, will charge and prompt to shock a patient in cardiac arrest
B	
basic life support (BLS)	emergency treatment of a patient with 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 carbon dioxide levels in the airways, which can be performed by infrared spectroscopy
cardiac arrest	temporary or permanent cessation of the heart rate
cardiopulmonary resuscitation (CPR)	a basic emergency procedure for life support, mainly involving manual external compressions 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 or abnormal heart rhythm
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 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	a condition in which a patient's core body temperature is <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	a condition is which a patient's core body temperature is between 93.2 °F (34 °C) and 96.8 °F (36 °C)
moderate hypothermia	a condition in which a patient's core body temperature is between 86 °F (30 °C) and 93.2 °F (34 °C)
N	
nasopharyngeal airway (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 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
S	
severe hypothermia	a condition in which a patient's core body temperature is <86 °F (30 °C)
sinus rhythm	the rhythm of the heart produced by impulses from the sinoatrial node
supraglottic	situated or occurring above the glottis
synchronized cardioversion	a process that 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 from a defibrillator that is 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