Advanced Life Support

Participant's Manual

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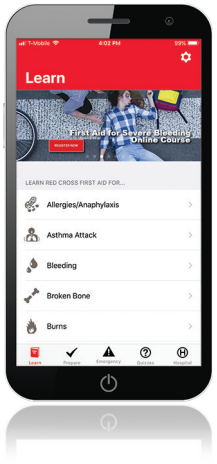

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This Participant's Manual is part of the American Red Cross Advanced Life Support program. The emergency care procedures outlined in the program materials reflect the standard of knowledge and accepted emergency practices in the United States at the time this manual was published. It is the reader's responsibility to stay informed of changes in emergency care procedures.

Printed in the United States of America

ISBN: 978-1-7337007-2-6

Science and Technical Content

The technical content within the American Red Cross Advanced Life Support program is consistent with the most current science and treatment recommendations from:

- The International Liaison Committee on Resuscitation (ILCOR) 2015-2018 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations
- The 2015-2018 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Guidance for this course was provided by the Red Cross Scientific Advisory Council, a panel of nationally recognized experts in various fields, including emergency medicine, emergency medical services, nursing, occupational health, sports medicine, school and public health, aquatics, emergency preparedness and disaster mobilization. More information on the science of the course content can be found at the following websites:

- ilcor.org
- redcross.org/science

Dedication

This program is dedicated to the thousands of employees and volunteers of the American Red Cross who contribute their time and talent to supporting and teaching lifesaving skills worldwide and to the thousands of course participants who have decided to be prepared to take action when an emergency strikes.

Acknowledgments

Many individuals shared in the development of the American Red Cross Advanced Life Support program in various technical, editorial, creative and supportive ways. Their commitment to excellence made this manual possible.

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We would like to extend our gratitude to the following members of the American Red Cross Scientific Advisory Council for their guidance and review of this program:

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Special thanks to the program development team for their expertise and mix of patience and persistence to bring this program through to completion: Melanie Cann, Danielle DiPalma, Sarah Kyle, Maureen Pancza, Maureen Schultz, Laura Scott and Nichole Steffens.

Supporting Organizations

Thank you to the following organizations and their staffs for accommodating the Red Cross and coordinating resources for this program's video production.

- Chester County Intermediate Unit Practical Nursing Program (CCIU PNP) Learning Lab—The Technical College High School Brandywine Campus
- Fairfax County Fire and Rescue Department
- Independence Blue Cross Medical Simulation Center at Drexel University College of Medicine
- Jefferson Stratford Hospital
- St. Christopher's Hospital for Children

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CHAPTER 1

Advanced Life Support Course Introduction

Introduction

This chapter provides an overview of American Red Cross advanced life support (ALS) training.

Course Purpose

When a patient experiences a life-threatening cardiovascular, cerebrovascular or respiratory emergency, you need to act swiftly to assess the situation and the patient and provide lifesaving care.

The purpose of the American Red Cross Advanced Life Support course is to ensure that healthcare providers have the requisite knowledge and skills to assess, recognize and care for patients who are experiencing a cardiovascular, cerebrovascular or respiratory emergency (Figure 1-1). The course emphasizes providing high-quality patient care by integrating psychomotor skills, rhythm interpretation, electrical interventions and pharmacologic knowledge with critical thinking and problem solving to achieve the best possible patient outcomes.

The American Red Cross Advanced Life Support course covers the skills required for certification as an ALS provider. In addition, the key concepts that support proficient performance of these skills are reviewed.

Course Preparation

The American Red Cross Advanced Life Support course is designed for professional healthcare providers who directly care for patients in a variety of settings and who could be called on to care for a critically ill patient. This could include, but is not limited to, nurses, nurse practitioners, physicians, physician assistants, respiratory therapists, dentists, emergency medical services personnel, public safety personnel and other professional responders.

Figure 1-1 | The goal of advanced life support training is to ensure that healthcare providers working in healthcare settings have the skills to provide care to patients who are experiencing a cardiovascular, cerebrovascular or respiratory emergency.

High-quality CPR skills (including high-quality chest compressions and basic airway management) and AED skills are an essential part of ALS (Figure 1-2). In addition, strong patient assessment skills; knowledge and understanding of the medications used in cardiovascular, cerebrovascular and respiratory emergencies; expert medication administration skills; and an ability to read and interpret ECG rhythms are valuable before taking this course.

Course Objectives

Upon successfully completing the American Red Cross Advanced Life Support course, you will be able to:

- Demonstrate high-quality basic life support skills, including high-quality chest compressions, effective ventilations and use of an AED.
- Apply concepts of effective teamwork when caring for a patient experiencing a cardiovascular, cerebrovascular or respiratory emergency.
- Integrate advanced communication, critical thinking and problem-solving skills when responding as part of a team to a cardiovascular, cerebrovascular or respiratory emergency.
- Effectively assess a cardiovascular, cerebrovascular or respiratory emergency situation using a systematic approach.
- Quickly recognize the nature of a cardiovascular, cerebrovascular or respiratory emergency.
- Provide effective and appropriate advanced life support care to address a cardiovascular, cerebrovascular or respiratory emergency.
- Provide effective and appropriate care after a return of spontaneous circulation (ROSC) during a resuscitation effort.

Figure 1-2 | High-quality CPR is the foundation of every successful cardiac arrest resuscitation effort.

Your Role as an Advanced Life Support Provider

ALS refers to the healthcare that providers deliver to adults who are experiencing a cardiovascular, cerebrovascular or respiratory emergency. The psychomotor skills needed to perform high-quality CPR; use an AED, a defibrillator, or both; and relieve an obstructed airway are the foundation of ALS. As an ALS provider, you must also be able to use advanced assessment skills to recognize problems and prioritize interventions. Finally, you must be able to provide effective care for cardiovascular, cerebrovascular and respiratory emergencies based on specific advanced life support treatment guidelines. ALS integrates the following key concepts to help providers achieve optimal patient outcomes (Figure 1-3):

- **Teamwork:** Integration and coordination of all team members working together toward a common goal
- **Communication:** A closed-loop process involving a sender, message and receiver
- **Critical thinking:** Clear and rational thinking used to identify the connection between information and actions
- **Problem solving:** Using readily available resources to identify solutions to issues that arise while providing care

Course Completion Requirements

Many agencies, organizations and individuals look to the Red Cross for formal training that results in certification. *Red Cross certification* means that on a particular date an instructor verified that a course participant could demonstrate competency in all required skills taught in the course. *Competency* is defined as being able to demonstrate correct decision-making processes, sequence care steps properly and demonstrate proficiency in completing all required skills without any coaching or assistance.

Figure 1-3 | Teamwork, communication, critical thinking and problem solving are essential for achieving optimal patient outcomes.

There are two ways to complete American Red Cross ALS training. You may take a traditional instructor-led course, or you may take a blended learning course that consists of an online session and an in-person skills session conducted by a Red Cross–certified instructor.

To successfully complete the Advanced Life Support Instructor-Led Training course, you must:

- Attend the entire course and participate in all class lessons.
- Actively participate in all course activities, including assuming various roles during skill practice and practice scenarios.
- Demonstrate competency in all required skills.
- Demonstrate competency in leading a team response during the team response testing scenarios.
- Successfully pass the final exam with a minimum grade of 84 percent.

To successfully complete the Advanced Life Support Blended Learning course, you must:

- Complete the online session, which includes:
  - Successfully completing each lesson, including the post-assessment.
  - Successfully passing the online final exam with a minimum grade of 84 percent.
- Attend and actively participate in the in-person skills session, during which you must:
  - Participate in all skill stations.
  - Demonstrate competency in all required skills.
  - Demonstrate competency in leading a team response during the team response testing scenarios.

Upon successful completion of the course and after the training has been reported, you will receive a course completion certificate from the American Red Cross that includes your name, the course name, the completion date and the certification validity period. The course completion certificate can be downloaded, printed or shared, as needed. Each American Red Cross certification contains a QR code that can be used by participants, instructors, employers or the American Red Cross to validate certificate authenticity.

CHAPTER 2

Basic Life Support Review

Introduction

Expert basic life support (BLS) skills (performing high-quality CPR, using an AED and relieving an obstructed airway) are essential skills for all healthcare providers to possess. Mastery of these foundational skills is vital in order to achieve the best possible outcomes for patients in cardiac or respiratory arrest and for patients who have an obstructed airway.

Cardiac Chain of Survival

The Cardiac Chain of Survival describes five actions that, when performed in rapid succession, increase the patient's likelihood of surviving sudden cardiac arrest. The five links in the Cardiac Chain of Survival vary slightly, depending on where the cardiac arrest occurs.

In-Hospital Cardiac Chain of Survival

Only one of every five cardiac arrests in the United States occurs inside of a hospital. The Adult In-Hospital Cardiac Chain of Survival (Figure 2-1) includes five links:

- **Surveillance and prevention.** Hospitalized patients often show changes in vital signs and other clinical parameters in the minutes and hours leading up to cardiac arrest. Closely monitoring for changes in the patient's condition that could be warning signs of impending arrest and activating the rapid response team as appropriate may allow providers to intervene and prevent the arrest from occurring.
- **Recognition of a cardiac emergency and activation of the emergency response system.** Recognizing cardiac arrest and summoning advanced help in the form of the resuscitation team or emergency medical services (EMS) provides the patient with access to necessary personnel, equipment and interventions as soon after arrest as possible.
- **Early CPR to keep oxygen-rich blood flowing and to help delay brain damage and death.** CPR, starting with compressions, should be initiated immediately once cardiac arrest is recognized.
- **Early defibrillation to help restore an effective heart rhythm and significantly increase the patient's chance for survival.** Defibrillation may restore an effective heart rhythm, increasing the patient's chance for survival.
- **Integrated post–cardiac arrest care to optimize ventilation and oxygenation and treat hypotension immediately after the return of spontaneous circulation.** After return of spontaneous circulation (ROSC), survival outcomes are improved when providers work to stabilize the patient, minimize complications, and diagnose and treat the underlying cause.

Out-of-Hospital Cardiac Chain of Survival

Most sudden cardiac arrests occur outside of the hospital. When this is the case, the patient relies on members of the community, EMS and healthcare providers to implement the Cardiac Chain of Survival. The Adult Out-of-Hospital Cardiac Chain of Survival (Figure 2-2) includes five links:

- **Recognition of a cardiac emergency and activation of the emergency response system.** Immediate recognition of cardiac arrest and activation of the EMS system provides the patient with access to necessary personnel, equipment and interventions as soon after arrest as possible.
- **Early CPR to keep oxygen-rich blood flowing and to help delay brain damage and death.** CPR, starting with compressions, should be initiated immediately once cardiac arrest is recognized.
- **Early defibrillation to help restore an effective heart rhythm and significantly increase the patient's chance for survival.** Use of an AED may restore an effective heart rhythm, increasing the patient's chance for survival.

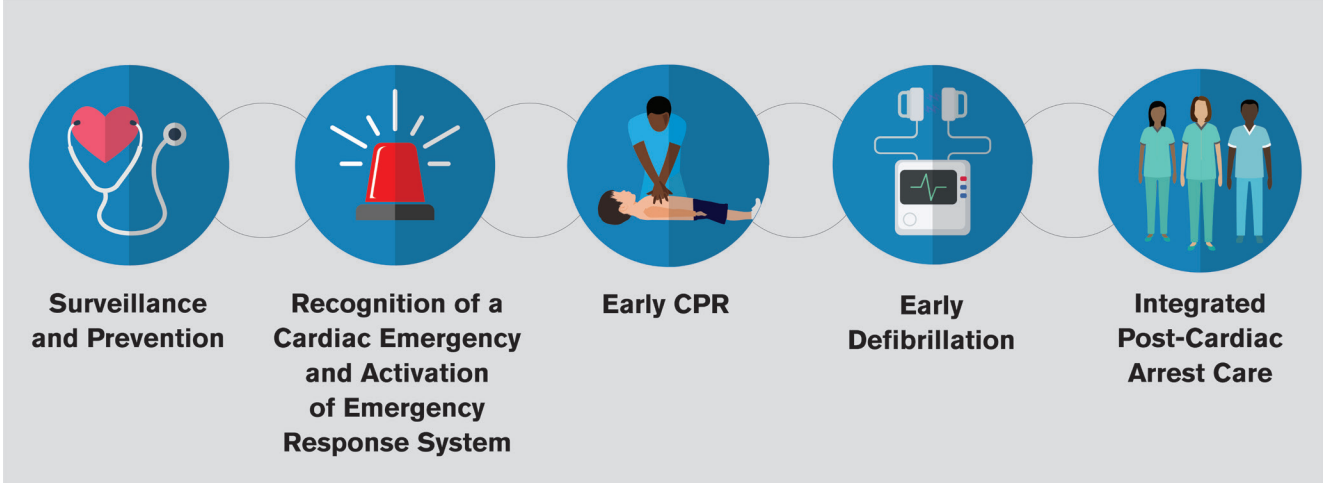


Figure 2-1 | The In-Hospital Cardiac Chain of Survival for adults

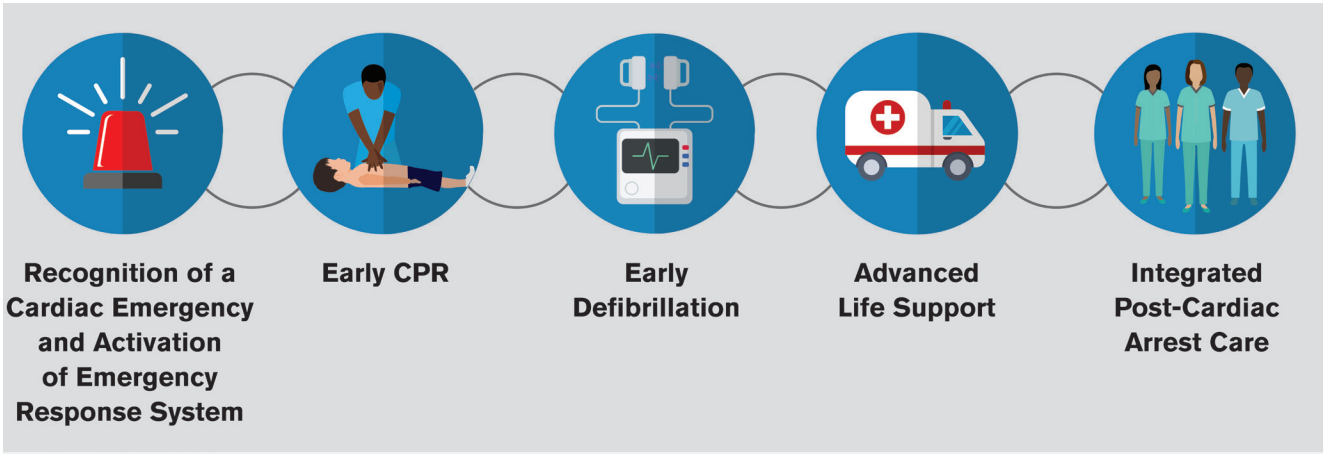


Figure 2-2 | The Out-of-Hospital Cardiac Chain of Survival for adults

- **Advanced life support using advanced medical personnel who can provide the proper tools and medication needed to continue the lifesaving care.** Early advanced life support provided by EMS personnel at the scene and en route to the hospital provides the patient with access to emergency medical care delivered by trained professionals.
- **Integrated post–cardiac arrest care to optimize ventilation and oxygenation and treat hypotension immediately after the return of spontaneous circulation.** After ROSC, survival outcomes are improved when providers work to stabilize the patient, minimize complications, and diagnose and treat the underlying cause.

High-Quality CPR

The point of CPR is to circulate oxygenated blood to vital organs when the heart and normal breathing have stopped. However, even at its best, CPR provides only a fraction of the normal blood flow to the brain and heart. To optimize patient outcomes and increase the likelihood of ROSC, providers must strive to provide the highest quality CPR at all times.

Principles of High-Quality CPR

To provide high-quality CPR:

- Position the patient supine on a firm, flat surface and expose the patient's chest, then immediately begin chest compressions.
- Provide compressions at the correct rate (100 to 120 per minute) and at the proper depth (at least 2 inches [5 cm] but no more than 2.4 inches [6 cm] for an adult). When given at the proper rate, it should take 15 to 18 seconds to perform 30 compressions.
- Allow the chest to recoil fully after each compression.
- Minimize interruptions in chest compressions. When compressions stop, blood flow to vital organs stops. In addition, after stopping compressions, some time is required to regain the minimum **coronary perfusion pressure (CPP)** necessary to achieve ROSC (Figure 2-3). The CPP is the difference between the pressure in the aorta and the pressure in the right atrium during diastole and is a reflection of myocardial blood flow. Maintaining adequate CPP (greater than 20 mmHg) during CPR has been shown to increase the likelihood of ROSC and survival.

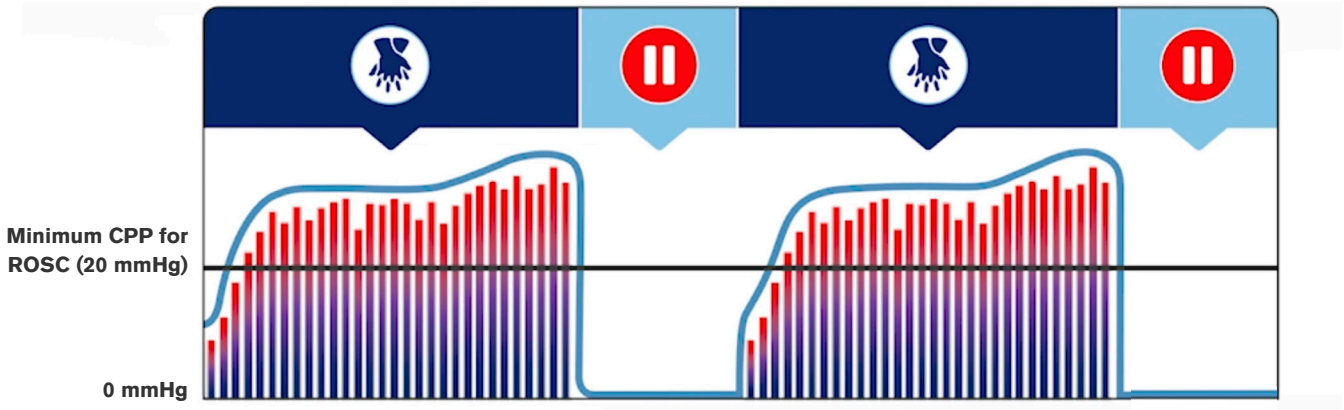


Figure 2-3 | When compressions stop, the coronary perfusion pressure (CPP) drops below 20 mmHg, the minimum level needed to increase the likelihood of return of spontaneous circulation (ROSC) and survival.

- Avoid excessive ventilations. Each ventilation should last about 1 second and deliver just enough volume to make the chest begin to rise.

Proper compression technique is important. Position the heel of one hand in the center of the chest, on the lower half of the sternum, with your other hand on top. Interlace your fingers and lift your fingers off the chest. Position yourself so that your shoulders are directly over your hands, and keep your arms as straight as possible (Figure 2-4). Compress the chest using a straight up-and-down motion. Avoid leaning on the patient's chest.

High-performance CPR refers to providing high-quality CPR as part of a well-organized team response to a cardiac arrest. Coordinated, efficient teamwork helps to minimize interruptions to compressions (Figure 2-5). In addition, a team approach to CPR helps to maintain the quality of compressions by minimizing provider fatigue. Providers should switch off giving compressions every 2 minutes—or sooner if the provider giving compressions is fatigued or the AED begins analyzing. Finally, working as a team helps to ensure that high-quality CPR is provided because the team leader is responsible for monitoring the delivery of CPR and making adjustments in real time, enabling the team to achieve quality goals.

Continuous Quality Improvement

Healthcare providers and their employers have a responsibility to ensure that they provide the highest quality CPR throughout every resuscitation event. To achieve this goal, it is necessary to gather data and use that data to inform improvements in individual and team performance. Data collected about the effectiveness of CPR allows for continuous improvement, both "in the moment" and during future resuscitation events. After every resuscitation event, a debriefing session should be held, during which the team analyzes their performance using both qualitative and quantitative data. The purpose of this analysis is to make changes as necessary to positively affect the outcome of future resuscitation events.

Figure 2-4 | Providing high-quality CPR relies on using proper technique.

Figure 2-5 | Efficient teamwork contributes to the provision of high-quality CPR.

Methods of evaluating CPR quality include visual observation, the use of feedback devices, calculations such as the chest compression fraction (CCF) and physiologic data obtained through capnography or hemodynamic monitoring.

Visual Observation

Visual observation is an important qualitative measure of high-quality CPR. Visual observation allows for in-the-moment adjustments to technique based on feedback from the team leader or another team member. For example, the team leader may observe that the provider giving compressions is tiring or that compressions are not being delivered at the correct rate or depth. These observations allow the team leader to redirect the team as necessary to get back on track.

Feedback Devices

Feedback devices use technology to gather data about CPR performance and provide real-time feedback. These devices collect objective data, such as the rate at which compressions and ventilations are being delivered, the depth of compressions and the amount of chest recoil. Many different types of feedback devices are available, ranging from apps on smart watches to self-contained systems, some with attachments to place on the patient. All feedback devices are designed to act as "virtual coaches," guiding providers to adjust technique in order to perform effective, high-quality CPR. In addition, most feedback devices record data that can be analyzed

after the resuscitation event, enabling improvements to be implemented for future resuscitation events.

Chest Compression Fraction

The **chest compression fraction (CCF)** represents the amount of time spent performing compressions and is another way to gain objective feedback about the quality of CPR. It is calculated by dividing the time that providers are in contact with the patient's chest by the total duration of the resuscitation event, beginning with the arrival of the resuscitation team and ending with the achievement of ROSC or the cessation of CPR. According to expert consensus, a CCF of at least 60% is needed to promote optimal outcomes, and the goal should be 80%. Many feedback devices are able to calculate the CCF based on the data they collect. When a feedback device is not in use, a team member may be assigned to record data that can be used to calculate the CCF, such as the duration of the resuscitation event and the duration of periods when compressions were paused.

Capnography

Capnography is a noninvasive technique that uses sensors to detect end-tidal carbon dioxide ($ETCO_2$) levels, which are displayed as waveforms on a monitor (Figure 2-6). Carbon dioxide delivery to the lungs depends on cardiac output. When circulation is adequate, a predictable amount of carbon dioxide should be exhaled. So, the $ETCO_2$ level is a quantitative measure of cardiac output—and by extension, the effectiveness of compressions. An $ETCO_2$ level in the expected range also suggests that ventilations are effective.

Normal $ETCO_2$ levels are in the range of 35 to 45 mmHg. In low-perfusion states such as cardiac arrest, the $ETCO_2$ levels are much lower (15 to 20 mmHg when high-quality CPR is provided). If $ETCO_2$ levels fall below 10 mmHg, there could be a problem with the rate or quality of compressions. Information obtained by monitoring capnography values allows the team to make the necessary adjustments to achieve higher-quality CPR.

Capnography is also an effective tool for determining ROSC and for helping the resuscitation team to decide when to discontinue CPR. A spike in $ETCO_2$ levels (to 40 mmHg or more) is a strong indicator that the patient has achieved ROSC. Conversely, if $ETCO_2$ levels remain less than 10 mmHg in an intubated patient who has been receiving high-quality CPR for at least 20 minutes, the likelihood that the patient will achieve ROSC is decreased, and the decision may be made to terminate the resuscitation effort. If, however, $ETCO_2$ levels remain greater than 15 mmHg, the patient has an increased chance to achieve ROSC and resuscitation efforts should continue.

Invasive Hemodynamic Monitoring

In-hospital cardiac arrest often occurs in settings in which invasive hemodynamic monitoring is in progress or can be quickly established. In such cases, if the patient already has an indwelling arterial catheter, the arterial pressure waveform can be used to evaluate the adequacy of chest compressions. ROSC is more likely when the arterial diastolic pressure is greater than 25 mmHg.

Approach to the Patient: Basic Life Support

The *BLS: Adults* Treatment Guideline summarizes the approach to providing basic life support care for an adult.

Rapid Assessment

The rapid assessment is used to gather information about the patient and the emergency. First, conduct a quick visual survey: assess for safety, form an initial impression about the patient's condition and determine the need for additional resources. Next, if the patient appears to be unresponsive, quickly check for responsiveness, breathing and a pulse (Figure 2-7).

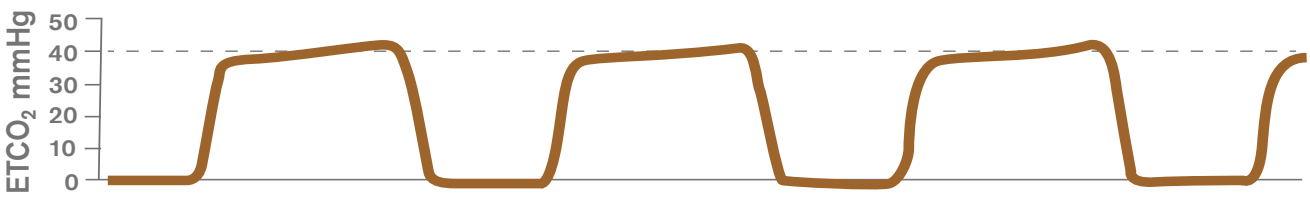


Figure 2-6 | The normal capnography waveform is square with a flat plateau. In patients with normal perfusion, capnography values are between 35 and 45 mmHg.

Figure 2-7 | When a patient appears to be unresponsive, check for responsiveness, breathing and a pulse.

- To check for responsiveness, use a "shout-tap-shout" sequence. Shout "Are you OK?" using the patient's name if you know it. Tap the patient on the shoulders, then shout again. If the patient does not respond, initiate the emergency response by calling for EMS, the rapid response team or the resuscitation team as appropriate and send someone to get an AED.
- To check for breathing and a pulse, open the airway by using the head-tilt/chin-lift technique to tilt the head to a past-neutral position. Use the modified jaw-thrust maneuver if you suspect a head, neck or spinal injury. Once the airway is open, simultaneously check for breathing and a carotid pulse for at least 5 but no more than 10 seconds.

If the patient is unresponsive or has an altered level of consciousness but is breathing normally, monitor the patient until EMS, the rapid response team or the resuscitation team arrives. Use a recovery position to help maintain a clear airway in an unresponsive patient who is uninjured and breathing normally (Box 2-1).

If the patient is unresponsive, is not breathing normally (or is only gasping) but has a pulse, the patient is in respiratory arrest. Provide 1 ventilation every 5 to 6 seconds. Each ventilation should last about 1 second and make the chest begin to rise. Continue giving ventilations until the patient begins to breathe normally on their own, another trained provider takes over, you are presented with a valid do not resuscitate (DNR) order, the patient has no pulse (in which case you should begin CPR or use an AED if one is available and ready to use) or the situation becomes unsafe.

Box 2-1 | Recovery Positions

To place an adult in a recovery position:

- Kneel at the patient's side.
- Lift the patient's arm closest to you up next to their head.
- Place the patient's arm farthest from you next to their side.
- Grasp their leg closest to you, flex it at the hip and bend the knee toward their head.
- Place one of your hands on the patient's shoulder and your other hand on their hip farthest from you.
- Using a smooth motion, roll the patient toward you by pulling their shoulder and hip with your hands. Make sure the patient's head remains in contact with their extended arm.
- Stop all movement when the patient is on their side.
- Place their knee on top of the other knee so that both knees are in a bent position.
- Place the patient's free hand under their chin to help support their head and airway.

Always follow your facility's protocols.

If the patient is unresponsive, is not breathing normally (or is only gasping) and has no pulse, the patient is in cardiac arrest. Begin CPR immediately, starting with compressions, and use an AED when it is available.

The *Rapid Assessment for Adults* Skill Sheet provides step-by-step guidance for conducting a rapid assessment.

CPR and AED

CPR

In most cases, when caring for an adult in cardiac arrest (regardless of whether care is being provided by a single provider or multiple providers), the hand position, compression rate, compression depth and compression-to-ventilation ratio of 30:2 remain the same. The exception to this is when a patient has an advanced airway in place. At minimum, two providers must be present. One provider delivers 1 ventilation every 6 seconds. At the same time, the second provider performs compressions at a rate of 100 to 120 compressions per minute. In this case, the compression-to-ventilation ratio of 30:2 does not apply because compressions and ventilations are delivered continuously with no interruptions.

When multiple providers are available, the first provider performs the rapid assessment and begins providing CPR, starting with chest compressions. Meanwhile, another provider calls for additional resources and gets and prepares the AED, if available. The first provider continues to provide high-quality CPR with 30 compressions to 2 ventilations until another provider is ready to assist or the AED is ready to analyze.

The *CPR for Adults* Skill Sheet provides step-by-step guidance for performing CPR on an adult.

AED

When a patient experiences cardiac arrest, an AED should be applied as soon as one is available (Figure 2-8). AEDs are programmed to deliver a shock to a patient in cardiac arrest when they detect ventricular fibrillation or ventricular tachycardia. Early use of an AED greatly increases the patient's chance for survival.

If CPR is in progress and more than one provider is present, do not stop CPR to apply the AED. If you are alone and an AED is available, you should use it as soon as you have determined that the patient is in cardiac arrest.

Use adult AED pads. Place one pad to the right of the sternum and below the right clavicle, and the other pad on the left side of the chest on the midaxillary line a few inches below the left armpit. Remember to stay clear of the patient while the AED is analyzing the rhythm or delivering a shock, and resume compressions immediately after a shock is a delivered or the AED advises that a shock is not indicated. Perform about 2 minutes of CPR (about 5 cycles of 30 compressions to 2 ventilations) until the AED prompts that it is reanalyzing, the patient shows signs of ROSC or you are instructed by the team leader or more advanced personnel to stop.

Figure 2-8 | Use an AED as soon as one is available.

The *AED Use for Adults* Skill Sheet provides step-by-step guidance for using an AED.

Approach to the Patient: Obstructed Airway

A patient who cannot cough, speak, cry or breathe requires immediate care. If the patient does not receive quick and effective care, an airway obstruction can lead to respiratory arrest, which in turn can lead to cardiac arrest.

Responsive Adult

A patient who is choking typically has a panicked, confused or surprised facial expression. The patient may place one or both hands on their throat (the "universal sign of choking"). Other behaviors may include running about, flailing the arms or trying to get another's attention. You may hear stridor (high-pitched squeaking noises) as the patient tries to breathe, or you may hear nothing at all. The patient's skin may initially appear flushed, but it will become pale or cyanotic as the body is deprived of oxygen.

Encourage the patient who is coughing forcefully to continue coughing until they are able to breathe normally. If the patient cannot breathe or has a weak or ineffective cough, summon additional resources and obtain consent. Then, perform abdominal thrusts or alternate techniques to clear the obstruction.

Abdominal Thrusts

To perform abdominal thrusts, stand behind the patient, with one foot in front of the other for balance and stability. If possible, place your front foot between the patient's feet. If the patient is in a wheelchair, you may need to kneel behind them.

Wrap your arms around the patient's waist. Use one or two fingers to find the navel. Make a fist with one hand and place the thumb side of your fist against the middle of the abdomen, just above the navel. Grab your fist with your other hand and give quick inward and upward thrusts (Figure 2-9). Make sure each thrust is a distinct attempt to dislodge the object. Continue delivering abdominal thrusts until the object is forced out; the patient can cough, speak or breathe; or the patient becomes unresponsive.

Alternate Techniques

Evidence suggests that it may take more than one technique to relieve an airway obstruction. For example, you might not be able to reach far enough around the patient or they might be pregnant, in bed

Figure 2-9 | To perform abdominal thrusts, place the thumb side of your fist just above the navel, cover the fist with your other hand and give quick inward and upward thrusts.

Figure 2-10 | For effective back blows, bend the patient forward at the waist and use the heel of your hand to give back blows between the scapulae.

or in a wheelchair with features that make abdominal thrusts difficult to do. Or, abdominal thrusts just might not be effective.

If this is the case, use chest thrusts, back blows or airway management techniques to dislodge the object from the airway. Follow your facility's protocols when implementing alternate techniques.

Back Blows

In some instances, back blows may be needed to relieve the obstruction. To perform back blows, position yourself to the side and slightly behind the patient. For a patient in a wheelchair, you may need to kneel. Provide support by placing one arm diagonally across the patient's chest. Then bend the patient forward at the waist so that the patient's upper body is parallel to the ground (or as close as it can be). Using the heel of your other hand, give firm back blows between the scapulae (Figure 2-10). Make each blow a separate and distinct attempt to dislodge the object.

If back blows alone do not dislodge the object, use a series of 5 back blows and 5 abdominal (or chest) thrusts. Always follow your facility's protocols.

Chest Thrusts

To perform chest thrusts, position yourself behind the patient as you would for abdominal thrusts. Place the thumb side of your fist against the center of the patient's chest on the lower half of the sternum. Then cover your fist with your other hand and pull straight back, providing a quick inward thrust into the patient's chest. Make sure each thrust is a distinct attempt to dislodge the object.

Airway Management Techniques

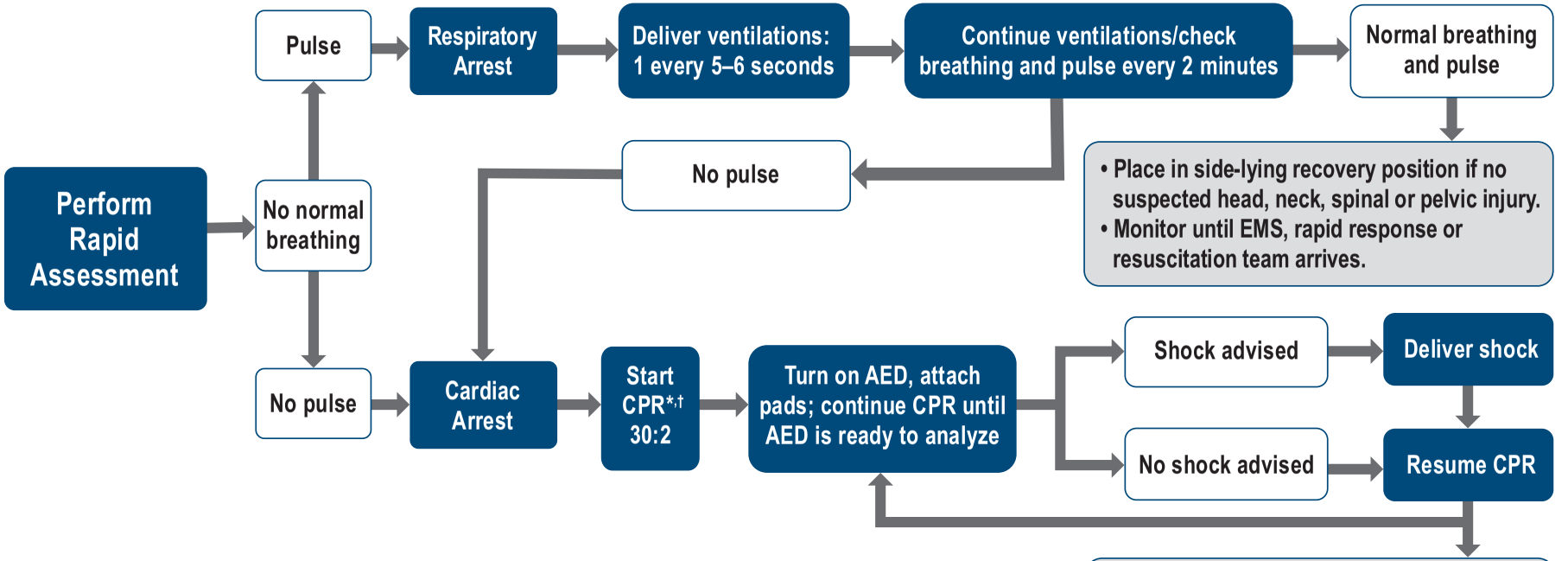
If you are in a healthcare facility and abdominal thrusts are not effective or possible, use a combination of basic or advanced airway management techniques based on your level of training and experience.

Unresponsive Adult

If a patient who is choking becomes unresponsive, carefully lower them to a firm, flat surface, while protecting their head. Then, send someone to get an AED and summon additional resources (if appropriate and you have not already done so). Immediately begin CPR, starting with chest compressions. Compressions may help clear the airway by moving the blockage into the upper airway and the oropharynx, where it can be seen and removed.

After each set of chest compressions and before attempting ventilations, open the patient's mouth and look for the object. If you see the object in the patient's mouth, remove it using a finger sweep. If you do not see the object, do not perform a blind finger sweep. Next, attempt 2 ventilations. Never try more than 2 ventilations during one cycle of CPR, even if the chest does not rise. Continue CPR, checking for an object before each set of ventilations.

BLS: ADULTS



CPR Technique

Switch CPR compressors
- Every 2 minutes
- During AED analysis
- If provider is fatigued

- **Hand position:** Centered on the lower half of the sternum
- **Depth:** At least 2 inches
- **Number:** 30 compressions
- **Rate:** 100 to 120 per minute (15 to 18 seconds)
- **Full chest recoil**

- Each ventilation should last about 1 second and make the chest begin to rise

Suspected or known opioid overdose → See *Opioid Overdose Treatment Guideline*

*If an advanced airway is in place, one provider delivers 1 ventilation every 6 seconds. At the same time, a second provider performs compressions at a rate of 100 to 120 per minute. In this case, the compression to ventilation ratio of 30:2 does not apply because compressions and ventilations are delivered continuously with no interruptions.

†If drowning or other hypoxic event is the suspected cause of cardiac arrest, deliver 2 initial ventilations before starting CPR.

American Red Cross
Training Services

SKILL SHEET

Rapid Assessment for Adults

Step 1 Perform a visual survey

- Make sure the environment is safe—for you, your team and any individuals present during the emergency.
- Gather an initial impression of the patient, which includes looking for severe, life-threatening bleeding.
- Quickly determine the need for additional resources.

⚠ Alert

If you see severe, life-threatening bleeding, immediately use any available resources to control the hemorrhage, including a tourniquet or hemostatic dressing if one is available.

Step 2 Check for responsiveness

- Shout, "Are you OK?" Use the patient's name if you know it.
- Tap the patient's shoulder and shout again (shout-tap-shout).
- If the patient is unresponsive and you are alone, call for help to activate EMS, the rapid response team or the resuscitation team, as appropriate, and call for an AED.
- If the patient is unresponsive and you are with another provider, the first provider stays with the patient. Other providers activate EMS, the rapid response team or the resuscitation team, as appropriate, and retrieve the AED, BVM and other emergency equipment.

Step 3 Simultaneously check for breathing and pulse

- Make sure the patient is in a supine (face-up) position. If they are face-down, you must roll them onto their back, taking care not to create or worsen a suspected injury.
- Open the airway to a past-neutral position using the head-tilt/chin-lift technique; or, use the modified jaw-thrust maneuver if you suspect a head, neck or spinal injury.
- Simultaneously check for breathing and a carotid pulse for at least 5 seconds but no more than 10.

Step 4 Provide care based on the conditions found

SKILL SHEET

CPR for Adults

Step 1 Conduct a rapid assessment

- Perform a quick visual survey, check for responsiveness, open the airway, and simultaneously check for breathing and a carotid pulse for at least 5 seconds but no more than 10.
- If the patient is unresponsive, isn't breathing normally and doesn't have a pulse, begin CPR.

Step 2 Place the patient on a firm, flat surface

- In a healthcare setting, use a bed with a CPR feature, or place a CPR board under the patient.
- Adjust the bed to an appropriate working height or use a step stool. Lower the bed side rail closest to you.
- In other settings, move the patient to the floor or ground and kneel beside them.

Step 3 Position your hands correctly

- Expose the patient's chest to ensure proper hand placement and visualize chest recoil.
- Place the heel of one hand in the center of the patient's chest on the lower half of the sternum.
- Place your other hand on top of the first and interlace your fingers or hold them up so that they are not resting on the patient's chest.

SKILL SHEET

CPR for Adults *(continued)*

Step 4 Position your body effectively

- Position yourself so your shoulders are directly over your hands. This position lets you compress the chest using a straight up-and-down motion.
- To help keep your arms straight, lock your elbows.

> **Practice Note**
>
> If drowning or another hypoxic event is the suspected cause of cardiac arrest, deliver 2 initial ventilations before starting CPR.

Step 5 Perform 30 chest compressions

- For an adult, compress the chest to a depth of at least 2 inches (5 cm). If you are using a feedback device, make sure the compressions are no more than 2.4 inches (6 cm) deep.
- Provide smooth compressions at a rate of 100 to 120 per minute.
- Allow the chest to fully recoil after each compression. Avoid leaning on the patient's chest at the top of the compression.

Step 6 Seal the mask and open the airway

- Use an adult pocket mask for single-provider CPR or a BVM for multiple-provider CPR.
- Seal the mask and simultaneously open the airway to a past-neutral position using the head-tilt/chin-lift technique.
- Or, use the modified jaw-thrust maneuver if you suspect a head, neck or spinal injury.

Step 7 Provide 2 ventilations

- While maintaining the mask seal and open airway, provide smooth, effortless ventilations. Each ventilation should last about 1 second and make the chest begin to rise. Avoid excessive ventilation.
- If you do not have a pocket mask or BVM, provide mouth-to-mouth or mouth-to-nose ventilations.

SKILL SHEET

CPR for Adults *(continued)*

Practice Note

If an advanced airway is in place, one provider delivers 1 ventilation every 6 seconds. At the same time, a second provider performs compressions at a rate of 100 to 120 per minute. In this case, the compression-to-ventilation ratio of 30:2 does not apply because compressions and ventilations are delivered continuously with no interruptions.

Step 8 Switch positions every 2 minutes

- When providing CPR with multiple providers, smoothly switch positions about every 2 minutes. This should take less than 10 seconds.
- The compressor calls for a position change by saying "switch" in place of the number 1 in the compression cycle.

Step 9 Continue CPR

Continue providing CPR until:

- You see signs of ROSC, such as patient movement or normal breathing.
- Other trained providers take over and relieve you from compression or ventilation responsibilities.
- You are presented with a valid do not resuscitate (DNR) order.
- You are alone and too exhausted to continue.
- The situation becomes unsafe.

Practice Note

Upon achieving ROSC, supplemental oxygen should be used based on your facility's protocols to maintain a normal oxygen saturation level while avoiding hyperoxygenation. Providers should use a pulse oximeter to monitor oxygen saturation.

SKILL SHEET

AED Use for Adults

Step 1 Turn on the AED and follow the prompts

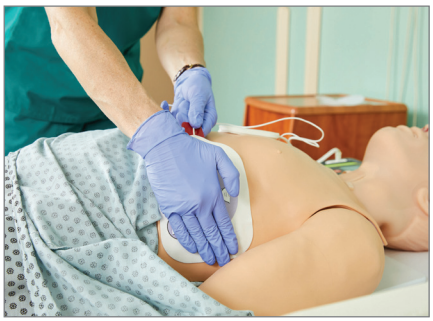
- Because AED models function differently, follow your facility's protocols and the manufacturer's instructions for the AED you have.

Step 2 Expose the chest

- Expose the chest and wipe it dry, if necessary.

Step 3 Attach the pads

- Use an anterior/lateral pad placement, according to the manufacturer instructions:
  - Place one pad on the upper right chest, below the right clavicle to the right of the sternum.
  - Place the other pad on the left side of the chest along the midaxillary line a few inches below the armpit.
- Or, use an anterior/posterior placement, according to the manufacturer instructions:
  - Place one pad to the center of the patient's chest—on the sternum.
  - Place one pad to the patient's back between the scapulae.



⚠ Alert

Do not use pediatric AED pads or pediatric levels of energy on an adult or on a child older than 8 years or weighing more than 55 pounds.

SKILL SHEET

AED Use for Adults *(continued)*

Step 4 Prepare to let the AED analyze the heart's rhythm

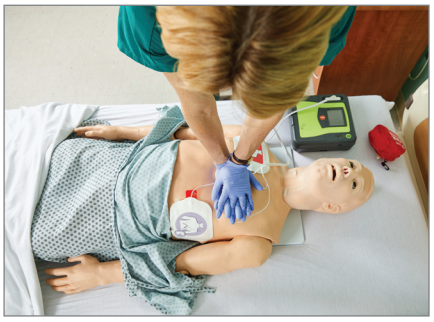
- If necessary, plug in the connector and push the analyze button.
- Instruct everyone to stand clear while the AED analyzes. No one, including you, should be touching the patient.
- As the AED analyzes, switch positions if you are working with a team. The provider giving compressions should hover their hands above the patient's chest.

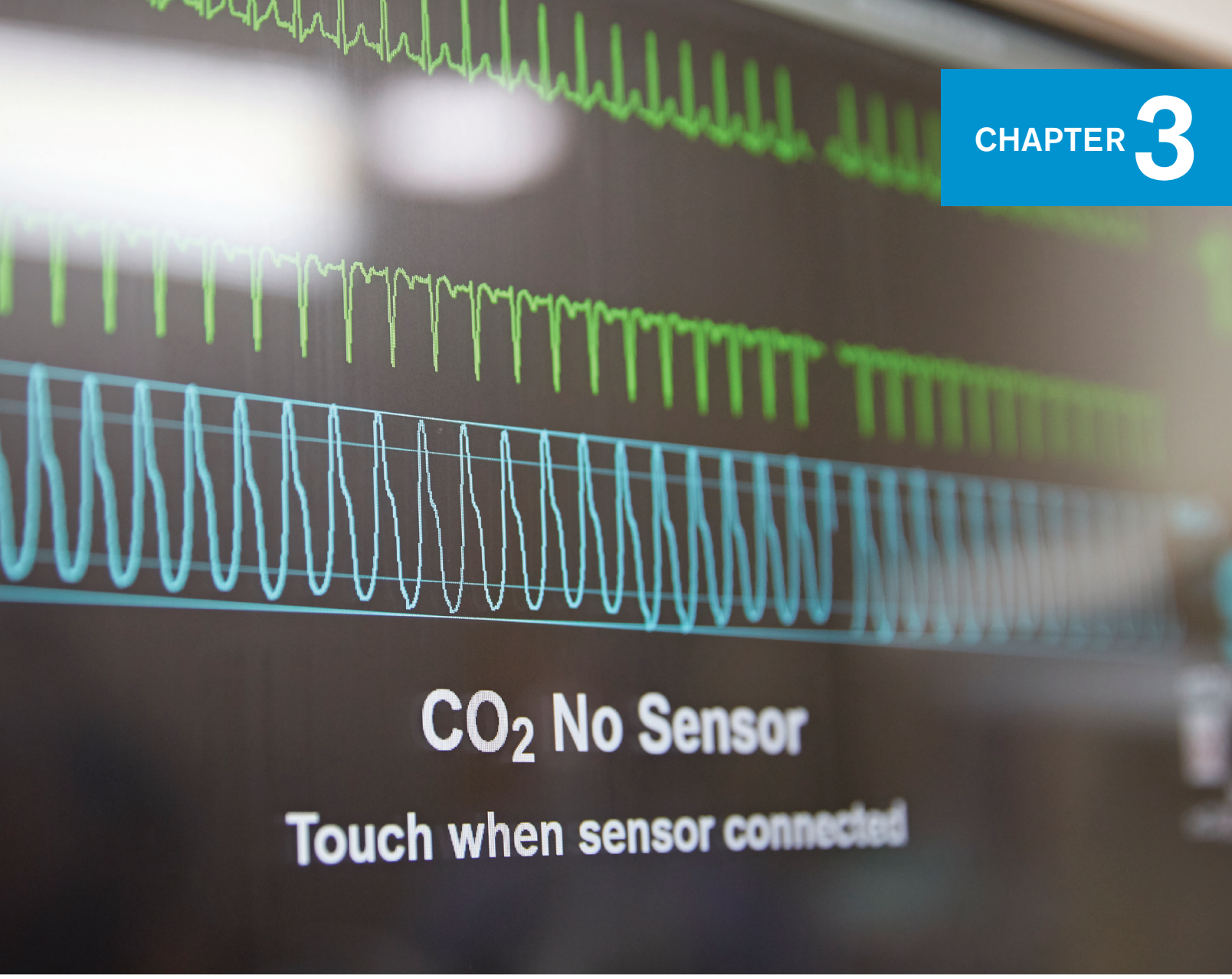
Step 5 Deliver a shock, if the AED determines one is needed

- If the AED advises a shock, again instruct everyone to stand clear. The compressor should continue to hover their hands over the patient's chest in preparation for CPR.
- Press the shock button to deliver the shock.

Step 6 After the AED delivers the shock, or if no shock is needed

- Immediately begin CPR. You do not need to wait for the AED prompt.
- Continue for about 2 minutes until:
  - The AED prompts that it is reanalyzing.
  - The patient shows signs of return of spontaneous circulation.
  - The team leader or other trained providers instruct you to stop.
- If you are working with a team, rotate roles during the analysis to prevent fatigue as needed.





Tools and Therapies

Introduction

As a member of a high-performance resuscitation team, you must be familiar with the equipment and interventions that are used most frequently when caring for a patient who is experiencing a cardiovascular, cerebrovascular or respiratory emergency. This chapter reviews equipment and interventions commonly used to assess and stabilize an acutely ill patient.

Airway Management

Suctioning

Suctioning is used to clear the airway of excessive secretions, vomitus or blood. Suctioning can be performed through the nose or mouth and when a basic or advanced airway is in place.

Suctioning is performed using a flexible or rigid catheter that is attached via tubing to a suction unit, which may be wall-mounted or portable. The suction unit has a pressure gauge to indicate the amount of negative pressure (suction force) and a collection canister.

- Flexible catheters are inserted through the mouth or nose and are best suited for removing thin, fluid secretions from the oropharynx or nasopharynx. A sterile flexible catheter is used to suction an endotracheal tube.
- Rigid (Yankauer) catheters are inserted through the mouth and are best suited for removing thick or particulate matter from the oropharynx.

Practice Note

Use appropriate personal protective equipment (e.g., face shield, gown, gloves) when suctioning a patient.

Practice Note

Suctioning can induce hypoxia and bradycardia (as a result of vagal stimulation). Always monitor the patient's oxygen saturation, heart rate and rhythm and appearance while suctioning. If the patient shows signs of compromise (e.g., decreased oxygen saturation, bradycardia, arrhythmia, cyanosis), stop suctioning immediately, administer high-flow oxygen and provide ventilation assistance as needed.

Basic Airways

An oropharyngeal airway (OPA) or nasopharyngeal airway (NPA) can be used to maintain an open airway.

Practice Note

When providing ventilations to a patient with an OPA or NPA in place, maintain an open airway using the head-tilt/chin-lift technique or the jaw-thrust maneuver.

Oropharyngeal Airway

An OPA provides a channel for air movement and suctioning. The curved body of the airway fits over the tongue and holds it up and away from the posterior wall of the pharynx (Figure 3-1).

An OPA is only indicated for use in an unconscious patient. Do not use an OPA in a conscious or semiconscious patient with intact cough or gag reflexes. In addition, avoid using an OPA in patients with oral trauma or who have recently undergone oral surgery.

The *Inserting an Oropharyngeal Airway (OPA)* Skill Sheet provides step-by-step guidance for inserting an OPA.

Nasopharyngeal Airway

An NPA is a soft rubber tube with a flange on one end and a beveled tip on the other. NPAs are available in a range of diameters. The NPA is inserted through the nose and extends to the posterior pharynx to provide a channel for air movement and suctioning (Figure 3-2).

An NPA may be used in a conscious, semiconscious or unconscious patient. Do not use an NPA in a patient with a possible skull or facial fracture. Exercise caution if considering use of an NPA in a patient with suspected head trauma.

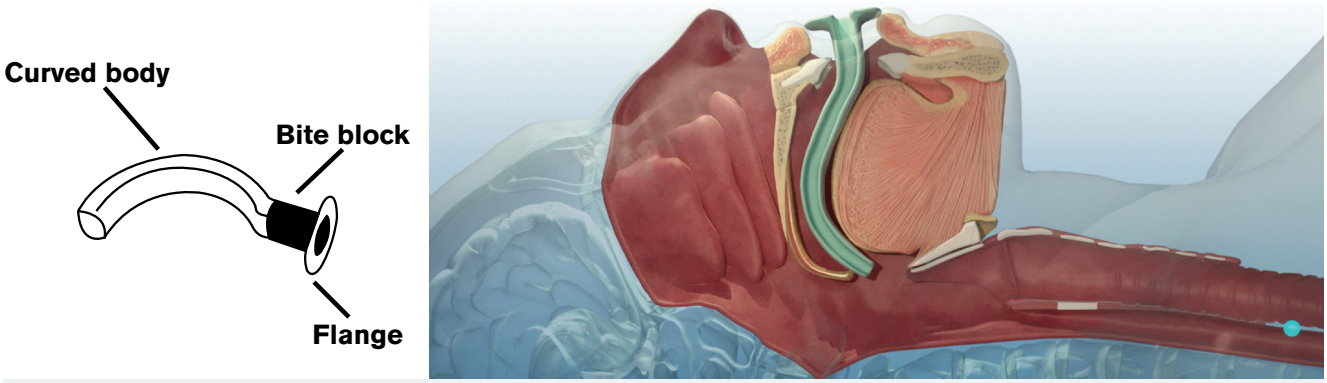


Figure 3-1 | Oropharyngeal airway

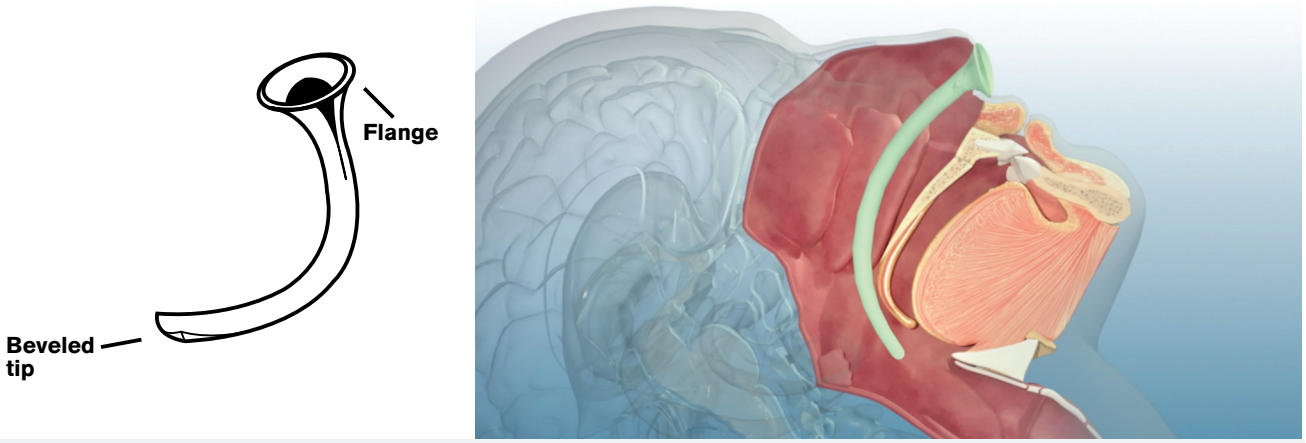


Figure 3-2 | Nasopharyngeal airway

The *Inserting a Nasopharyngeal Airway (NPA)* Skill Sheet provides step-by-step guidance for inserting an NPA.

Advanced Airways

Placement of an advanced airway is indicated when:

- An open airway cannot be maintained using manual techniques or a basic airway.
- Airway protection is necessary because of impaired airway reflexes (e.g., as a result of impaired level of consciousness).
- Continuous ventilation management is required.

The risks of placing an advanced airway must be weighed against the potential benefits. Improper placement of an advanced airway can lead to complications that further destabilize the patient. Additionally, although it is possible to place supraglottic airways while CPR is in progress without pausing compressions, endotracheal intubation may require pausing compressions briefly. If it is possible to maintain adequate ventilation with a bag-valve-mask (BVM) resuscitator, consider delaying placement of an advanced airway.

Types of Advanced Airways

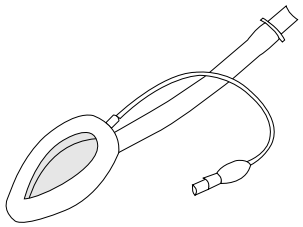
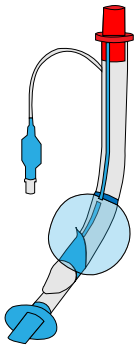
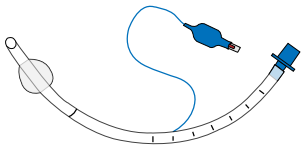
Advanced airway options include supraglottic airways and transglottic airways (Table 3-1). **Supraglottic airways**, such as laryngeal mask airways and laryngeal tubes, do not pass through the vocal cords, whereas **transglottic airways**, such as endotracheal tubes, do. The choice of advanced airway depends on the patient's condition, the available resources and the provider's capabilities and scope of practice.

Laryngeal Mask Airway

The laryngeal mask airway consists of an airway tube and a mask with an inflatable cuff at the distal end. The mask is advanced along the contour of the pharynx with the aperture of the mask facing the tongue until resistance is met. Properly positioned, the mask opening overlies the glottis, while the bottom rim wedges up against the upper esophageal sphincter, creating a seal. Once the cuff of the mask is inflated, the glottis is isolated, permitting air from the tube to enter the trachea (Figure 3-3).

Figure 3-3 | Laryngeal mask airway

Table 3-1 | Advanced Airways

Advanced Airway	Advantages	Limitations	Precautions
■ Laryngeal mask airway 	■ Trained providers more readily available; easier to learn how to insert (as compared with an endotracheal tube) ■ Alternative to endotracheal intubation when patient access is limited or positioning of the patient for endotracheal intubation is impossible ■ Lower risk for regurgitation (as compared with BVM ventilation) ■ Can be inserted during CPR without interrupting chest compressions	■ Does not offer complete protection of the airway from aspiration as an endotracheal tube does ■ May not be effective for patients requiring higher ventilation pressures (e.g., those with lung disease)	■ Improper placement may cause suboptimal ventilation, gastric distension or laryngospasm/ obstruction of the airway ■ Head movement and suctioning of the pharynx can cause displacement of the device ■ Tissue damage may occur with prolonged use
■ Laryngeal tube 	■ Trained providers more readily available; easier to learn how to insert (as compared with an endotracheal tube) ■ Isolates the airway and reduces the risk for aspiration (as compared with BVM ventilation) ■ Can be inserted during CPR without interrupting chest compressions	...	...
■ Endotracheal tube 	■ Offers complete protection of the airway from aspiration ■ Facilitates tracheal suctioning ■ Provides an alternate administration route for some medications ■ Best choice when the need for long-term assisted ventilation is anticipated	■ Training is more complex than for supraglottic airways ■ Skill is outside scope of practice for many providers ■ Providers require a great deal of practice and experience to become proficient ■ Requires laryngoscopy ■ High incidence of complications when placement is attempted by inexperienced providers	■ Improper placement can result in severe complications, including brain damage and death

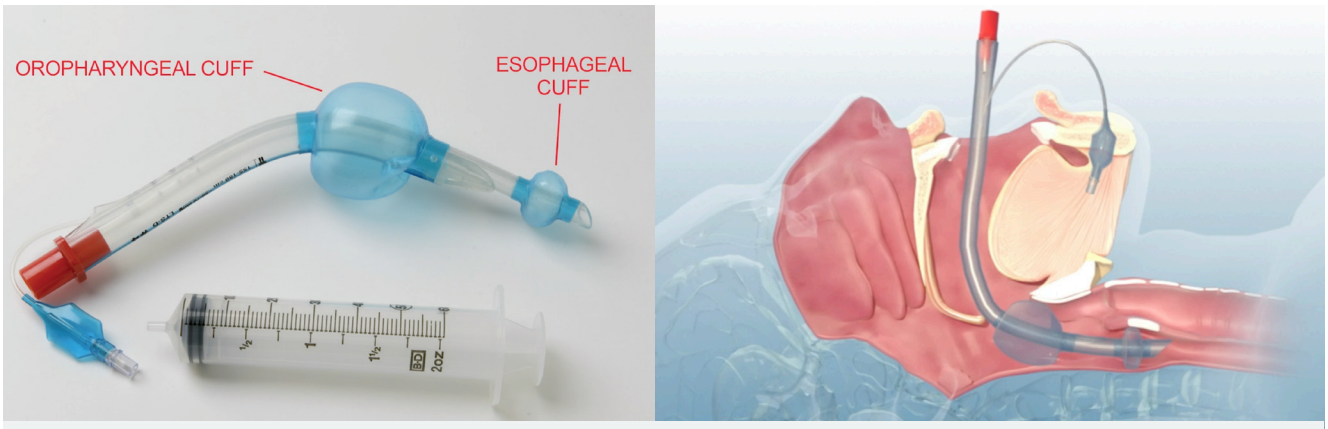


Figure 3-4 | Laryngeal tube

Laryngeal Tube

The laryngeal tube consists of an airway tube with a larger, proximal oropharyngeal cuff and a smaller, distal esophageal cuff. The laryngeal tube is inserted against the hard palate and advanced down the midline until resistance is met. When properly positioned and the cuffs are inflated, the proximal cuff isolates the laryngopharynx from the oropharynx and nasopharynx, and the distal cuff isolates the laryngopharynx from the esophagus (Figure 3-4).

Endotracheal Tube

Use of an endotracheal tube may be preferred over a supraglottic airway when there is disease at or below the level of the glottis, when prolonged assisted ventilation is needed or when higher ventilatory pressures are required. Endotracheal intubation also protects the airway from aspiration of gastric contents, a protection that cannot be ensured with supraglottic airways.

The endotracheal tube may be inserted through the mouth (i.e., orotracheal intubation) or the nose (i.e., nasotracheal intubation). The orotracheal route is used most often because it is faster and associated with fewer complications. Endotracheal intubation is facilitated by direct visualization of the airway using a laryngoscope equipped with a light source.

The endotracheal tube consists of an airway tube with an inflatable cuff and beveled tip at the distal end and a connector at the proximal end. A stylet may be used to stiffen and shape the endotracheal tube to facilitate insertion. The laryngoscope blade is used to displace the epiglottis and the endotracheal tube is passed through the vocal cords and into the trachea. When the endotracheal tube is properly inserted and the cuff is inflated, the cuff forms a seal against the walls of the trachea (Figure 3-5).

Laryngoscope blades (Figure 3-6) may be straight (e.g., Miller blade) or curved (e.g., Macintosh blade) and are available in various sizes. Ensure that backup blades a few sizes smaller and a few sizes larger than the selected blade are available before beginning the procedure. Test the lightbulbs on the selected and backup blades by attaching the blade to the

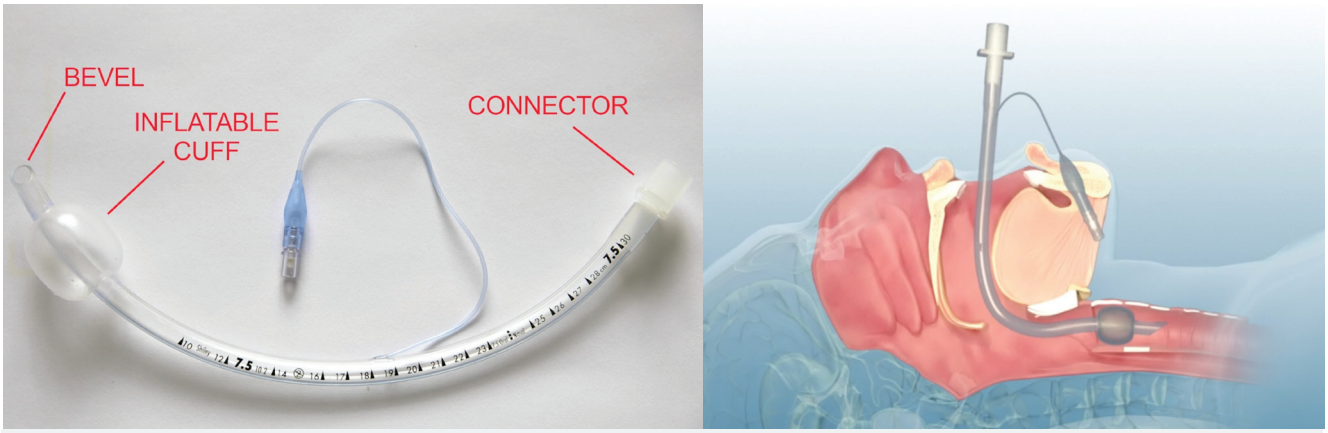


Figure 3-5 | Endotracheal tube

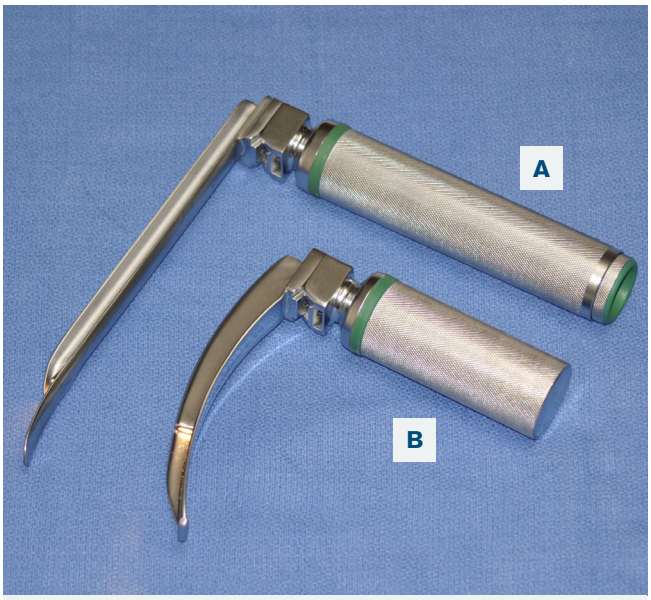


Figure 3-6 | Laryngoscope blades. (A) Straight (Miller) blade. (B) Curved (Macintosh) blade.

laryngoscope handle. Tighten each lightbulb to prevent dislodgement during intubation.

Practice Note

To minimize interruptions to chest compressions during endotracheal intubation, be prepared to insert the laryngoscope blade and advance the endotracheal tube as soon as compressions are paused. Pause compressions only long enough to pass the endotracheal tube through the vocal cords.

Practice Note

Intubation of the right (most common) or left mainstem bronchus is a possibility if the endotracheal tube is advanced too far. In this case, only one lung will be ventilated, which can lead to hypoxemia and overinflation of the ventilated lung. Clinical signs of right or left mainstem bronchus intubation include unilateral chest expansion and breath sounds.

Confirming Placement of an Advanced Airway

When an advanced airway is in use, confirming its correct placement initially is essential. In addition, airway placement should be confirmed whenever the patient's position changes and on an ongoing basis. Correct placement of an advanced airway is verified using both physical assessment techniques and confirmation devices, such as capnography, a colorimetric or other nonwaveform exhaled carbon dioxide detector or an esophageal detector device.

Practice Note

Nonwaveform exhaled carbon dioxide detectors are not effective for confirming correct airway placement in low-perfusion states (e.g., cardiac arrest, shock).

Physical Assessment

Always assess the patient after placing an advanced airway for clinical indications of correct placement. These include bilateral chest rise with ventilations, bilateral breath sounds and an absence of gurgling sounds on auscultation of the epigastric region.

Capnography

Capnography is considered the most reliable means of quickly confirming and monitoring endotracheal tube placement. Although studies have not yet validated capnography's effectiveness for confirming and monitoring placement of other types of advanced airways, it stands to reason that effective ventilations when a supraglottic airway is in use would also produce the expected end-tidal carbon dioxide ($ETCO_2$) values and capnography waveforms.

To confirm endotracheal tube placement using capnography:

- Attach the capnography sampling device directly to the endotracheal tube and attach the ventilation bag to the adapter.
- Provide 1 full ventilation and wait for a waveform to appear on the monitor. With some devices, this may take up to 3 seconds.
  - A four-point square waveform indicates tracheal intubation. Note that in cardiac arrest, capnography will only record a waveform when compressions and ventilations are being given.
- Equal bilateral breath sounds and three square waveforms in a row on the monitor confirm correct placement of the endotracheal tube.
  - Esophageal intubation will not produce a waveform, or it will produce a waveform that is not well defined.
  - If the waveform is square but breath sounds are absent on the left, right mainstem bronchus intubation is likely.

- After confirming correct placement, secure the endotracheal tube and control ventilations, keeping the $ETCO_2$ level between 35 and 45 mmHg in normal perfusion states.

Practice Note

When an advanced airway is in place, always monitor the patient for signs of compromise. Clinical signs of compromise, a change in capnography waveform appearance or a sudden decrease in the $ETCO_2$ level may be signs of airway displacement or other complications.

Bag-Valve-Mask Ventilation

Ventilation is the mechanical process of moving air into and out of the body. When spontaneous breathing is absent or is insufficient to support adequate ventilation and gas exchange, assisted ventilation is indicated. A BVM resuscitator is used to ventilate the patient while awaiting placement of an advanced airway or when the need for assisted ventilation is expected to be short-term. Using a BVM resuscitator correctly requires ample training and practice.

The BVM resuscitator consists of a cushioned mask that fits over the patient's mouth and nose and is connected via a one-way valve to a self-inflating compressible chamber, or bag (Figure 3-7). Squeezing the bag with the mask properly sealed over the patient's mouth and nose forces air into the lungs (positive pressure ventilation).

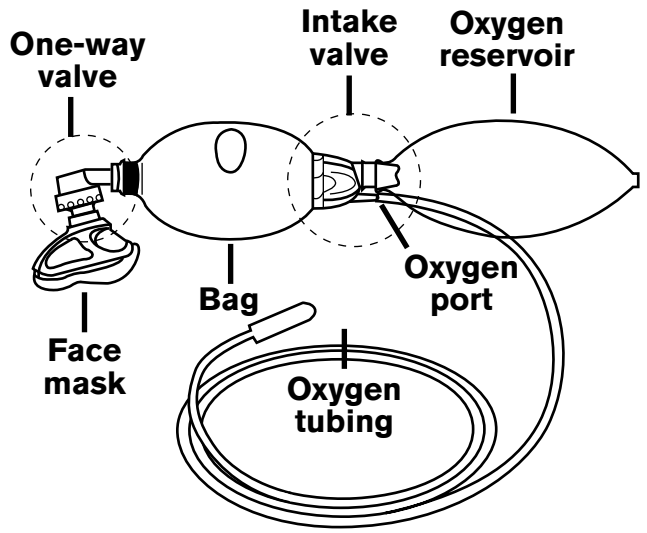


Figure 3-7 | Parts of a bag-valve-mask (BVM) resuscitator

Releasing the bag causes it to self-inflate by drawing air in from the other end. The one-way valve between the bag and the mask prevents exhaled air from re-entering the bag. When an advanced airway is in place, the bag is attached to the advanced airway, rather than to a mask.

The BVM resuscitator can be used with ambient air or attached to supplemental oxygen. Most BVM resuscitators come with the oxygen reservoir already attached. When the BVM resuscitator is connected to a high-flow (15 L/min) supplemental oxygen source, the reservoir fills with oxygen. The reservoir, which fills when the patient exhales, allows for the maximum concentration of oxygen to be delivered to the patient with each ventilation.

A BVM resuscitator can be used by one or two providers. Because ensuring a proper mask seal and an open airway with one hand while delivering ventilations with the other can be difficult, evidence supports having two providers operate the BVM resuscitator during CPR when an advanced airway is not being used.

To minimize complications when using a BVM resuscitator, depress the bag slowly (over 1 second) and only about halfway to deliver the minimal tidal volume. Complications that can result from improper technique include:

- **Decreased cardiac output**. Positive pressure ventilation increases the intrathoracic pressure, which in turn decreases venous return.
- **Barotrauma**. Delivering too much pressure can damage the airways, lungs and other organs.
- **Volutrauma.** Excessive volume can lead to tension pneumothorax (especially in patients with lung disease).
- **Gastric insufflation**. Increased airway pressure can cause air to enter the stomach, leading to gastric distension and increasing the risk for regurgitation and aspiration.

Pay close attention to any increasing difficulty when providing ventilations using a BVM resuscitator. This difficulty may indicate an increase in intrathoracic pressure, inadequate airway opening or other complications. Be sure to share this information with the team for corrective actions.

The *Using a Bag-Valve-Mask (BVM) Resuscitator—One Provider* Skill Sheet and the *Using a Bag-Valve-Mask Resuscitator—Two Provider* Skill Sheet provide step-by-step guidance for using a BVM resuscitator.

Supplemental Oxygen

The administration of supplemental oxygen is often indicated for patients experiencing a cardiovascular, cerebrovascular or respiratory emergency. Oxygen delivery systems consist of an oxygen source (wall-mounted or an oxygen cylinder), a flowmeter and a delivery device. Oxygen cylinders also have a pressure regulator, which reduces the pressure of the oxygen to a safe level.

Oxygen Delivery Devices

The maximum flow rate and concentration of oxygen that can be delivered vary according to the delivery device (Table 3-2). Factors that influence the choice of delivery device include whether the patient is spontaneously breathing or requires assisted ventilation, the degree of oxygen desaturation, equipment availability and patient comfort.

Nasal Cannula

A nasal cannula is only suitable for use on a spontaneously breathing patient. The nasal cannula is commonly used for patients with only minor breathing difficulty or for those who have a history of respiratory disease and is useful for patients who cannot tolerate a face mask. Patients experiencing a serious breathing emergency generally breathe through the mouth and need a device that can supply a greater concentration of oxygen.

Simple Oxygen Face Mask

Simple oxygen masks can deliver high-flow oxygen. However, because room air is entrained through the side ports and, potentially, around the mask, delivered oxygen concentrations are in the range of 35% to 55%.

Non-Rebreather Mask

A non-rebreather mask is used to deliver high concentrations of oxygen to a breathing patient. The one-way valve prevents exhaled air from mixing with the oxygen in the reservoir bag. The patient inhales oxygen from the bag and exhaled air escapes through flutter valves on the side of the mask. To inflate the reservoir bag, occlude the one-way valve with your gloved thumb before placing the mask on the patient's face. The oxygen reservoir bag should be sufficiently inflated (about two-thirds full) so it does not deflate when the patient inhales. If the bag deflates, increase the flow rate of the oxygen to refill the reservoir bag.

Bag-Valve-Mask Resuscitator

A BVM resuscitator can be used on a breathing or nonbreathing patient. A BVM resuscitator with an oxygen reservoir bag is capable of supplying an oxygen concentration of 90% or more when used at a flow rate of 15 L/min or more. As when using a non-rebreather mask, occlude the one-way valve before applying the mask to allow the reservoir to fill with oxygen.

Pulse Oximetry

Oxygen therapy is typically titrated to achieve an oxygen saturation of at least 94% on pulse oximetry.

An oximeter consists of a clip-on probe with light-emitting diodes on one side and a light detector on the other. When the probe is placed on a finger, toe or earlobe, beams of red and infrared light are passed through the tissues to the light detector on the other side. Oxygenated hemoglobin absorbs more infrared light, allowing more red light to pass through, and deoxygenated hemoglobin absorbs more red light, allowing more infrared light to pass through. The ratio of red to infrared light that reaches the light detector is translated into a measurement of how much oxygen the blood is carrying, referred to as the peripheral capillary hemoglobin oxygen saturation, or SpO_2.

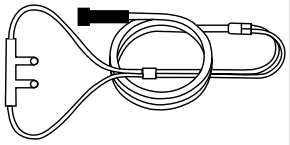
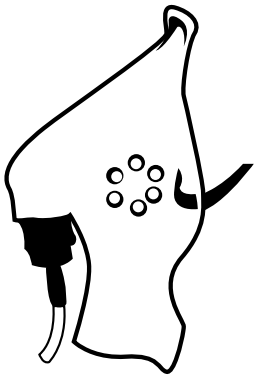
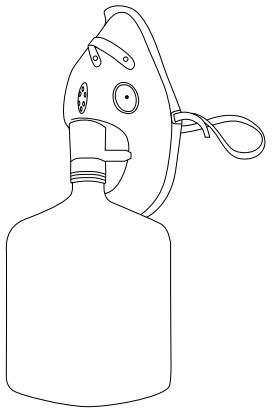
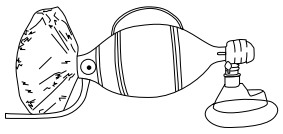
To establish pulse oximetry monitoring:

- Position an appropriately sized probe on a finger, toe or earlobe. If using a finger or toe, remove any nail polish from the nail. Avoid placing the probe on an extremity being used for blood pressure monitoring because cuff inflation will interfere with pulse oximetry readings.
- Connect the probe to the pulse oximeter and ensure that the probe is working by confirming that it is emitting a red light.
- After a few seconds, look for a pulse indicator or waveform on the monitor indicating that the probe has detected a pulse; otherwise, the reading will be inaccurate.

Practice Note

Some factors may reduce the reliability of the pulse oximetry reading, including hypoperfusion (shock), cardiac arrest, excessive patient motion, carbon monoxide poisoning, hypothermia, sickle cell disease or anemia, a history of smoking and edema.

Table 3-2 | Oxygen Delivery Devices

Delivery Device	Description	Oxygen Flow Rate, L/min	Oxygen Concentration, %	Appropriate for
Nasal cannula Low flow High flow 	Held in place over the patient's ears; oxygen is delivered through two small prongs inserted into the nostrils	1–6	24–44	Breathing patients only
Simple oxygen face mask 	Pliable, dome-shaped mask that fits over the mouth and nose with an oxygen inlet and side ports to permit egress of exhaled gas	6–15	35–55	Breathing patients only
Non-rebreather mask 	Face mask with an attached oxygen reservoir bag and one-way valve between the mask and bag; patient inhales oxygen from the bag, and exhaled air escapes through flutter valves on the side of the mask	10–15	Up to 90	Breathing patients only
BVM resuscitator 	Handheld breathing device consisting of a self-inflating bag, a one-way valve, a face mask and an oxygen reservoir bag	≥ 15	≥ 90	Breathing and nonbreathing patients

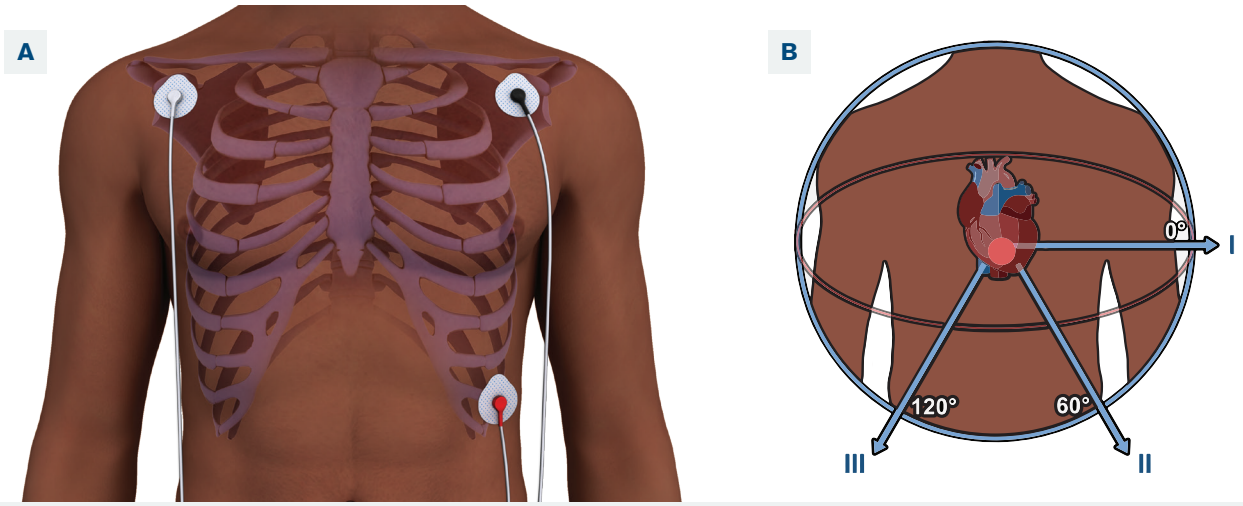


Figure 3-8 | Three-electrode system. (A) The white electrode is placed under the right clavicle at the midclavicular line, the black electrode is placed under the left clavicle at the midclavicular line, and the red electrode is placed on the lower left abdomen. (B) The three-electrode system allows monitoring of leads I, II and III.

Cardiac Monitoring

For basic monitoring of heart rate and rhythm, most cardiac monitors/defibrillators use a three- or five-electrode system. A three-electrode system (Figure 3-8) permits monitoring of the bipolar limb leads (i.e., leads I, II and III). Usually only one lead can be viewed on the monitor at a time. A five-electrode system (Figure 3-9) uses four limb electrodes and one chest electrode to provide seven views of the electrical activity of the heart. The four limb electrodes produce six leads in the frontal plane: I, II, III, augmented voltage of the right arm (aVR), augmented voltage of the left arm (aVL) and augmented voltage of the left foot (aVF). The chest electrode produces one lead in the horizontal plane: V_1. Two or more leads can usually be viewed on the monitor at once.

Practice Note

Most cardiac monitors/defibrillators have a wide range of functions, including continuous monitoring capabilities (e.g., heart rate and rhythm, blood pressure, pulse oximetry and capnography), diagnostic capabilities (i.e., 12-lead ECG) and therapeutic capabilities (i.e., modes for defibrillation, synchronized cardioversion and transcutaneous pacing). Make sure you are familiar with the features and functions of the equipment in use at your facility.

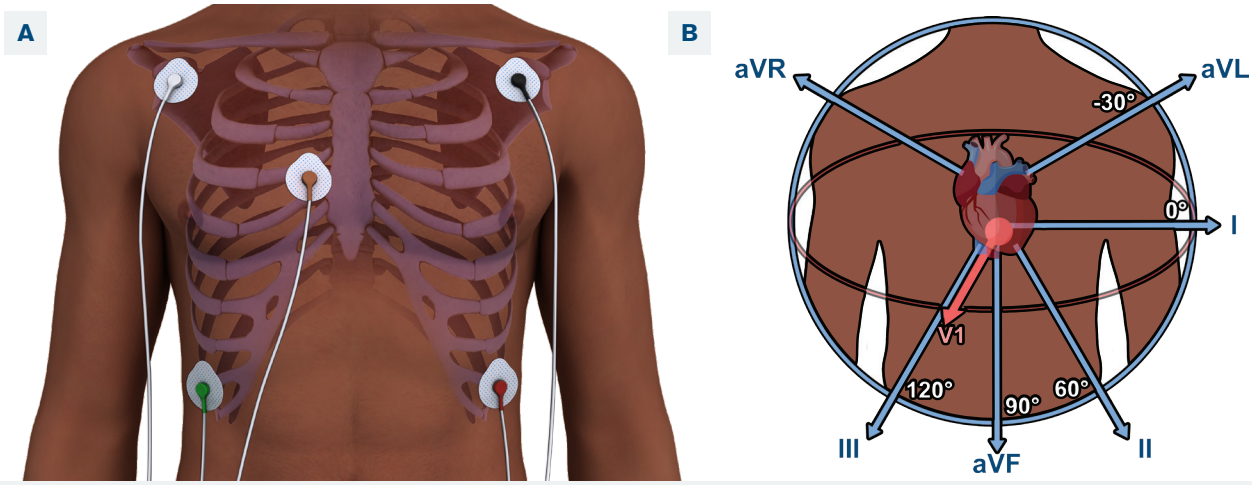


Figure 3-9 | Five-electrode system. (A) In addition to the electrodes placed for a three-electrode system, the brown electrode is placed in the fourth intercostal space along the right sternal border and the green electrode is placed on the lower right abdomen. (B) The five-electrode system allows monitoring of leads V_1, I, II, III, aVR, aVL and aVF.

Electrocardiography

Most cardiac monitors/defibrillators can also be used to obtain a 12-lead ECG, which is necessary for accurately diagnosing arrhythmias, acute coronary syndromes (ACS) and other conditions affecting the electrical activity of the heart. The 12-lead ECG uses four limb electrodes and six chest electrodes to provide 12 views of the heart (Figure 3-10). The four limb electrodes produce six leads in the frontal plane: I, II, III, aVR, aVL and aVF. The six chest electrodes produce six leads in the horizontal plane: V_1, V_2, V_3, V_4, V_5 and V_6. In some clinical situations, obtaining a 15-lead ECG may be necessary, which is done by obtaining a 12-lead ECG and then repositioning some of the electrodes to obtain the additional three views (Figure 3-11).

Preparing the skin where the electrodes will be placed is important to minimize artifact. The skin should be clean, dry and free of excess hair. In addition, proper electrode placement is essential for accurate rhythm identification. Misplacement of an electrode by as little as one intercostal space can cause waveform morphology to change, potentially leading to misdiagnosis.

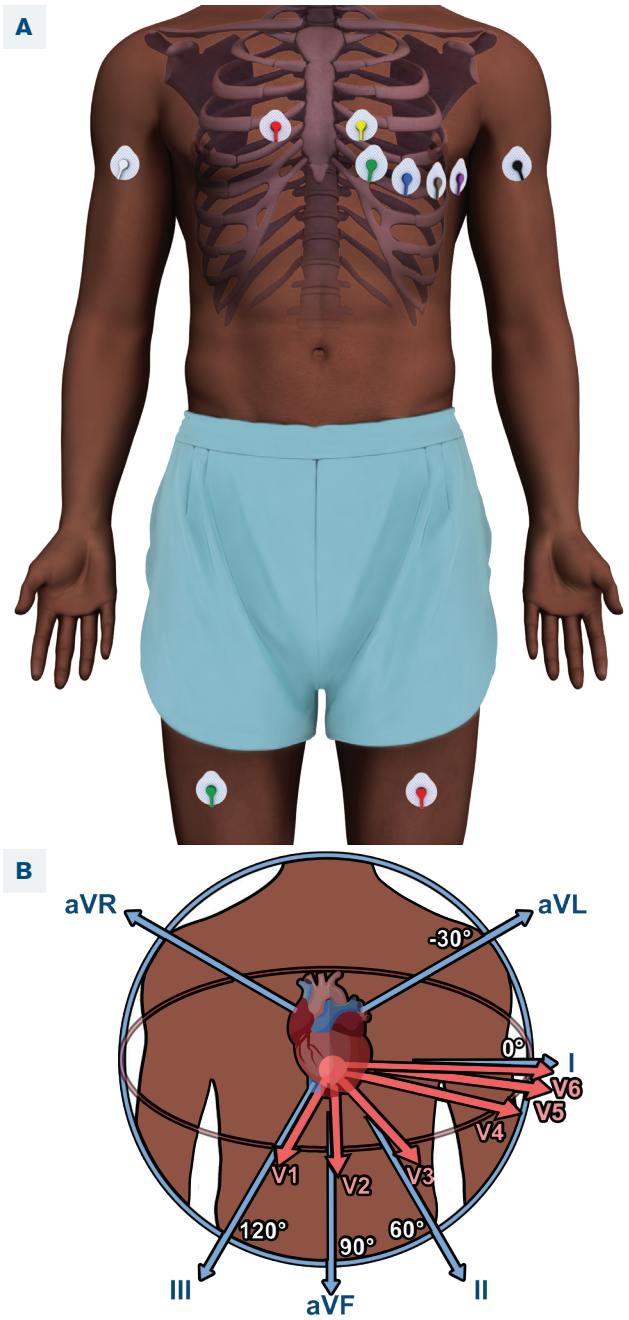


Figure 3-10 | The 12-lead ECG uses (A) four limb electrodes and six chest electrodes to provide (B) 12 views of the electrical activity of the heart.

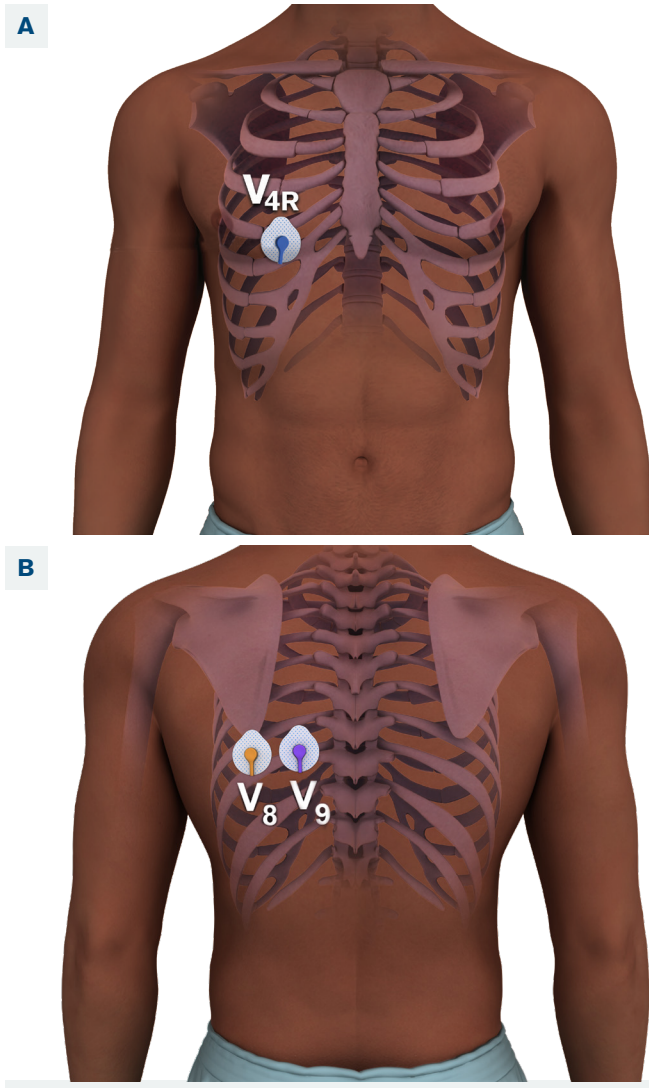


Figure 3-11 | The 15-lead ECG uses three additional electrodes to obtain three additional views of the heart. (A) Lead V_{4R} is placed over the fifth intercostal space at the midclavicular line on the right side. (B) Leads V_8 and V_9 are placed posteriorly. V_8 is placed over the fifth intercostal space at the midscapular line on the left side and V_9 is placed over the fifth intercostal space between V_8 and the spine.

The *Placing Electrodes for Electrocardiography* Skill Sheet provides step-by-step guidance for placing electrodes for a 12-lead and 15-lead ECG.

Electrical Therapies

Commonly used electrical therapies include manual defibrillation, synchronized cardioversion and transcutaneous pacing.

Manual Defibrillation

Defibrillation is indicated for shockable cardiac arrest rhythms (i.e., ventricular fibrillation and pulseless ventricular tachycardia). Defibrillation involves the administration of direct-current electricity. The delivery of electricity is not synchronized in any way with the cardiac cycle. The electricity depolarizes the myocardial cells, making them unresponsive to abnormal pacemakers in the heart and ideally allowing the sinoatrial (SA) node to resume its normal pacemaker function, terminating the arrhythmia.

Electricity is delivered to the patient via two adhesive pads that contain a conductive gel layer. The conductive gel layer helps to overcome **transthoracic impedance** (the body's resistance to current flow that is caused by the thoracic structures, including soft tissue and bone, between the defibrillation pads and the heart) and minimizes the risk for burns.

The pads may be placed using anterolateral placement or anterior-posterior placement:

- **Anterolateral placement** (Figure 3-12A): Place the sternal pad on the patient's right side adjacent to the upper sternum, below the clavicle. Place the apical pad on the patient's left side over the fourth and fifth intercostal spaces, with the center of the pad at the midaxillary line.
- **Anterior-posterior placement** (Figure 3-12B): Place the anterior pad on the patient's left side over the fourth and fifth intercostal spaces, with the center of the pad at the midaxillary line. Place the posterior pad in the left infrascapular region.

The energy dose depends on the type of defibrillator.

- **If using a biphasic defibrillator**, follow the manufacturer's recommendations for the initial dose (usually between 120 and 200 joules). Subsequent doses should be the same as or higher than the initial dose. If the manufacturer's recommendations for the initial dose are not known, use the highest energy dose available for the first and all subsequent shocks.
- **If using a monophasic defibrillator**, set the energy dose at 360 joules. Use this energy dose for each subsequent shock.

Practice Note

To minimize interruptions to chest compressions, continue providing compressions while placing the pads on the patient's chest and charging the cardiac monitor/defibrillator.

Practice Note

Always precede the delivery of a shock by announcing the intention to shock in a clear, succinct manner. Before delivering a shock, perform a visual scan to ensure that no one is touching the patient, the bed or the stretcher and that oxygen delivery devices have been removed and set aside, away from the patient. When delivering the shock, continue to face the team, rather than the defibrillator.

The *Manual Defibrillation* Skill Sheet provides step-by-step guidance for manual defibrillation.

Synchronized Cardioversion

Synchronized cardioversion is indicated for the treatment of unstable patients with arrhythmias that have preserved QRS complexes on ECG, such as supraventricular tachycardia, atrial fibrillation and atrial flutter. Synchronized cardioversion is also used as a first-line therapy in patients with monomorphic ventricular tachycardia who are unstable but have a pulse and after unsuccessful pharmacologic therapy in patients with stable monomorphic ventricular tachycardia.

Synchronized cardioversion involves the delivery of a low dose of direct-current electricity that is timed to correlate with the peak of the R wave. This avoids delivery of electricity during the refractory phase of the cardiac cycle (represented by the T wave on the ECG), which could precipitate ventricular fibrillation or torsades de pointes.

As in defibrillation, a cardiac monitor/defibrillator is used for synchronized cardioversion, and shocks are delivered through adhesive pads placed on the patient's chest. However, the cardiac monitor/defibrillator must be set to synchronous mode (indicated by the appearance of sync markers at the

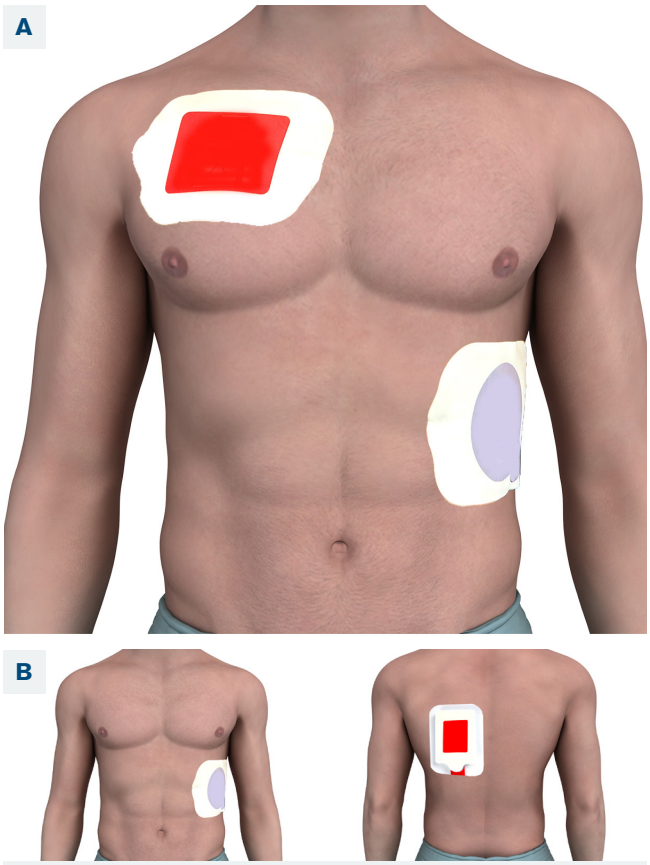


Figure 3-12 | Defibrillation pad placement. (A) Anterolateral placement. (B) Anterior-posterior placement.

top of each R wave on the monitor). Additionally, it is necessary to press and *hold* the shock button until the shock is delivered. Table 3-3 summarizes the key differences between synchronized cardioversion and defibrillation.

Practice Note

If the sync markers are hard to see, try selecting another lead on the monitor.

Because synchronized cardioversion can be uncomfortable for the patient, administer sedation or analgesia unless the patient's condition is deteriorating rapidly. Energy doses depend on the arrhythmia and the type of defibrillator (Table 3-4). After delivering the shock, reassess the rhythm and the patient:

- If the rhythm did not convert, reset the cardiac monitor/defibrillator to synchronous mode, increase the energy level in a stepwise fashion, charge the pads and deliver a shock.
- If the rhythm did convert, check the patient's vital signs and ensure adequate airway, breathing and circulation.

Table 3-3 | Key Differences Between Synchronized Cardioversion and Manual Defibrillation

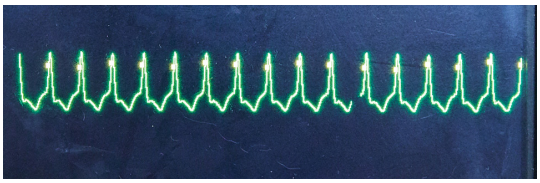
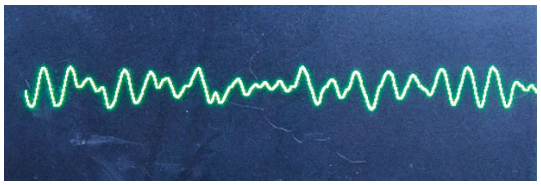
Feature	Synchronized Cardioversion	Manual Defibrillation
Mechanism	Delivery of electricity timed with QRS complex on ECG 	Delivery of electricity not timed with any part of the cardiac cycle 
Indications	Unstable: ■ Supraventricular tachycardia ■ Atrial flutter ■ Atrial fibrillation ■ Monomorphic ventricular tachycardia with pulse Stable monomorphic ventricular tachycardia that is refractory to pharmacologic therapy	Ventricular fibrillation Pulseless ventricular tachycardia
Use	■ Must select synchronous mode prior to delivering shock ■ Must press and hold the shock button to deliver the shock	■ No need to select mode prior to delivering shock ■ No need to hold the shock button to deliver the shock

Table 3-4 | Energy Doses for Synchronized Cardioversion

Arrhythmia	Biphasic Defibrillator (J)	Monophasic Defibrillator (J)
Narrow-complex regular rhythms	50–100	50–100
Narrow-complex irregular rhythms	120–200	200
Wide-complex regular rhythms	100	100
Wide-complex irregular rhythms	Defibrillation dose; not synchronized	Defibrillation dose; not synchronized

Practice Note

Most cardiac monitor/defibrillators revert to defibrillation mode after delivering a shock. This is because cardioversion may induce ventricular fibrillation, in which case immediate defibrillation is necessary. Always ensure that the cardiac monitor/defibrillator is returned to synchronous mode before each subsequent attempt at synchronized cardioversion.

The *Synchronized Cardioversion* Skill Sheet provides step-by-step guidance for synchronized cardioversion.

Transcutaneous Pacing

Transcutaneous pacing involves delivering an electrical current through the skin to stimulate the heart to contract. Transcutaneous pacing is indicated for patients with symptomatic bradycardia that is not responsive to pharmacologic therapy.

Like synchronized cardioversion, transcutaneous pacing can be uncomfortable for the patient, so administer sedation or analgesia if the patient's condition permits. Set the cardiac monitor/defibrillator to pacing mode, and then set the demand rate and the current milliamperes output. Gradually increase the current milliamperes output until electrical capture (wide QRS complexes and tall, broad T waves following each pacing spike) is observed on the monitor. Confirm mechanical capture by assessing the patient for clinical signs such as a palpable pulse, an increase in blood pressure and an increase in $ETCO_2$ level on capnography.

The *Transcutaneous Pacing* Skill Sheet provides step-by-step guidance for transcutaneous pacing.

Practice Note

Check for a pulse using the right radial artery or the right or left femoral artery. During transcutaneous pacing, skeletal muscle contractions can mimic a pulse in the left radial artery or in the carotid arteries.

Vascular Access

Quick access to the circulation is often needed for delivery of medications and other therapies and for sampling of blood. The best access technique is one that is rapid and does not interfere with CPR.

Intravenous Access

Peripheral veins are commonly used for vascular access during emergencies. The best site for peripheral access is that which permits placement of the largest-diameter catheter possible without interfering with other resuscitative efforts, such as airway maneuvers or chest compressions. Common peripheral sites for intravenous (IV) catheter insertion include the large surface veins in the antecubital fossa and the dorsum of the hands and wrists.

Factors to consider when selecting the site for peripheral IV access include the duration of, and indication for, treatment, the types of solutions to be infused and vein availability.

Practice Note

If upper extremity peripheral venous access cannot be achieved, consider central venous access (e.g., the femoral vein) or lower extremity peripheral venous access (e.g., the saphenous vein or the veins of the dorsum of the foot).

When administering IV therapies in an emergency, keep the following points in mind:

- Use the largest-diameter catheter possible (at least 18 gauge in an adult).
- Because strict aseptic technique may be breached during an emergency, replace the original catheter using strict aseptic technique after the patient is stabilized.
- Ensure that the extremity with IV access is at or above the level of the heart.
- When providing care for a patient in cardiac arrest, follow each peripherally administered drug dose with a 10- to 20-mL normal saline flush to ensure that the medication reaches the central circulation.

- Monitor the patient for local and systemic complications of IV therapy.

Intraosseous Access

Intraosseous (IO) access is often used as an alternative to IV access in emergency situations. IO access uses the bone marrow as the vascular space and typically involves a needle rather than a catheter. As with IV access, IO access can be used to administer medications, fluids and blood products and to collect blood for laboratory analysis. Medication dosing is the same for IO and IV routes, and all medications administered to a patient in cardiac arrest should be followed by a 10- to 20-mL normal saline flush. Because the IO needle should not be left in place for more than 24 hours, IV access should be established as soon as possible, ideally within a few hours.

Sites for IO needle insertion vary according to the device used to achieve access (Table 3-5).

The most commonly used sites are the medial aspect of the anterior proximal tibia and the proximal humerus. These sites provide a flat surface with a relatively thin outer layer of bone, a large marrow cavity and easily identifiable landmarks to facilitate placement. Other sites for IO access include the distal femur, distal radius, anterior-superior iliac spine, medial malleolus and sternum.

IO needle placement should not be attempted in patients with bone fractures at the site or disorders that predispose to fracture (e.g., osteoporosis). Relative contraindications to IO needle placement include infection or burns of the overlying skin and previous attempts to establish access at the same IO access site; however, IO placement may still be considered in these instances when there is no other vascular access during an emergency.

Table 3-5 | Sites for Intraosseous (IO) Access

	IO Drill	Bone Injection Device
Anterior proximal tibia (medial aspect)	X	X
Proximal humerus	X	X
Distal tibia	X	

IO needles can be inserted using a device (such as a drill or injection device) or manually (Figure 3-13). After insertion, monitor the insertion site and the extremity for swelling, which may indicate that the needle is out of place. Dislodging of the needle can lead to complications, including infection, medication or fluid extravasation and compartment syndrome. Remove the IO needle as soon as IV access can be established, ideally within a few hours.

The *Intraosseous Access (Drill)* Skill Sheet and the *Intraosseous Access (Manual Insertion)* Skill Sheet provide step-by-step guidance for achieving IO access using an IO drill and manually, respectively. Always follow the manufacturer's instructions when using a device to insert an IO needle.

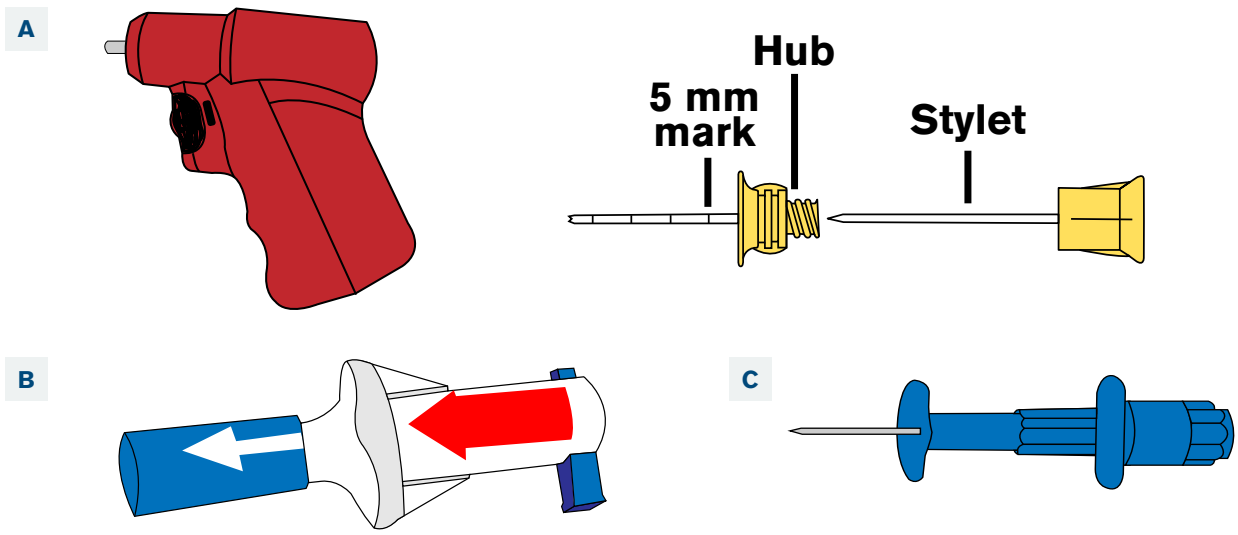


Figure 3-13 | Equipment for intraosseous (IO) access. (A) IO drill and needle. (B) Bone injection device. (C) Jamshidi needle for manual insertion.

Fluid Therapy

Fluid resuscitation is often indicated for patients experiencing a cardiovascular emergency and as part of post–cardiac arrest care. Along with the use of vasoconstrictors or inotropic agents, the administration of fluids can help to maintain cardiac output, blood pressure and perfusion.

Isotonic crystalloid solutions, such as 0.9% normal saline or lactated Ringer's solution, are the primary solutions used for fluid resuscitation. Isotonic fluids are used instead of hypotonic fluids (e.g., 0.45% normal saline) because isotonic solutions allow a greater proportion of the administered volume to remain in the intravascular space. In most situations, normal saline and lactated Ringer's solution are equally effective options for fluid resuscitation. Because of the association between repeated normal saline boluses and hyperchloremic metabolic acidosis, which may obscure an ongoing or developing acidosis secondary to impaired tissue perfusion, use of a "balanced" solution such as lactated Ringer's solution may be preferred in some situations.

Colloid solutions, such as albumin, dextran and hydroxyethyl starch, may also be used for fluid resuscitation in shock. Historically, colloids were proposed to be a more effective option for fluid resuscitation than crystalloid solutions, based on the assumption that a greater proportion of colloid solution remains in the vascular space. However, this property appears to be lost when capillary membranes are "leaky," as they are in various shock states.

Large peripheral IV catheters are adequate for most types of fluid resuscitation, as are central and IO catheters. Infusion pumps typically allow infusion of 1 liter of crystalloid solution in approximately 10 to 15 minutes.

Drug Therapy

A good working knowledge of the drugs most commonly used in the management of cardiovascular, cerebrovascular and respiratory emergencies is essential. Table 3-6 summarizes the action, indications, administration and precautions for drugs that are commonly administered via the IV or IO route in resuscitation situations. Table 3-7 summarizes other drugs that are referenced throughout the course materials and their uses.

Table 3-6 | Commonly Used IV/IO Drugs in Resuscitation Situations

Drug	Action	Indications	Administration	Precautions
Adenosine	Slows conduction of impulses through the AV node	■ Stable regular narrow-complex tachycardias ■ Unstable regular narrow-complex tachycardias if does not delay cardioversion ■ Stable regular monomorphic wide-complex tachycardias	■ Has an extremely short half-life; administer over 1–2 s, at a site as close to the heart as possible ■ 6 mg by rapid IV/IO push follow by 10- to 20-mL NS flush ■ If not effective after 1–2 min, 12 mg by rapid IV/IO push followed by 10- to 20-mL NS flush; may repeat up to once more	■ Therapeutic effects can be blocked by the presence of caffeine or theophylline ■ VF possible if adenosine is administered for unstable, irregular or polymorphic wide-complex tachycardias ■ Can temporarily evoke a transiently slow ventricular rate or complete cessation of electrical activity; as drug is eliminated, electrical activity resumes

Table 3-6 | Commonly Used IV/IO Drugs in Resuscitation Situations *(continued)*

Drug	Action	Indications	Administration	Precautions
Amiodarone	Class III antiarrhythmic; delays repolarization and prolongs the QT interval	■ Shock-refractory VF/pVT ■ Hemodynamically stable VT ■ Wide-complex VT whose origin or cause is unknown	For VF/pVT: ■ 300 mg by rapid IV/IO push ■ If not effective after 3–5 min, 150 mg by rapid IV/IO push For stable VT: Maximum dose 2.2 g IV/IO over 24 h; may be administered as: ■ Rapid infusion: 150 mg IV/IO over 10 min; may repeat every 10 min as needed ■ Maintenance infusion: 1 mg/min for first 6 hours	■ Do not use with other drugs that prolong QT interval
Atropine	Blocks the effect of acetylcholine released by the vagus nerve at muscarinic receptors, thereby increasing the rate of firing of the SA node and conduction through the AV node	■ Symptomatic bradycardia	■ 0.5 mg IV/IO every 3–5 min, not to exceed a total dose of 3 mg or 0.04 mg/kg	■ Use in patients with acute coronary ischemia or myocardial infarction may have negative outcomes because of increased heart rate and myocardial oxygen demand ■ IV/IO doses of less than 0.1 mg may cause paradoxical bradycardia
Dopamine	Has positive chronotropic and inotropic effects, resulting in an increase in heart rate and contractility	■ Symptomatic bradycardia (second-line agent) ■ After cardiac arrest	For bradycardia: ■ 2–20 mcg/kg/min IV/IO titrated slowly to patient response; not to exceed a total dose of 50 mcg/kg/min For post–cardiac arrest care: ■ 5–10 mcg/kg/min IV/IO	■ Can increase myocardial oxygen demand ■ Can cause ventricular arrhythmias

Table 3-6 | Commonly Used IV/IO Drugs in Resuscitation Situations *(continued)*

Drug	Action	Indications	Administration	Precautions
Epinephrine	Acts on both α- and ß-adrenergic receptors; induces systemic vasoconstriction and increases heart rate and contractility	■ Cardiac arrest (VF, pVT, pulseless electrical activity, asystole) ■ After cardiac arrest ■ Symptomatic bradycardia (second-line agent)	For VF/pVT/pulseless electrical activity/asystole: ■ 1 mg IV/IO followed by 10- to 20-mL NS flush every 3–5 min For post–cardiac arrest care: ■ 0.1–0.5 mcg/kg/min IV/IO For bradycardia: ■ 2–10 mcg/min IV/IO titrated to patient response **Note:** 1:1000 concentration = 1.0 mg/mL; 1:10,000 concentration = 0.1 mg/mL	■ Increased blood pressure, heart rate and myocardial oxygen demand may cause myocardial ischemia
Lidocaine	Class Ib antiarrhythmic (sodium channel blocker); delays repolarization and slightly increases the QT interval	■ VF/pVT	For VF/pVT: ■ 1–1.5 mg/kg IV/IO followed by 10- to 20-mL NS flush ■ If not effective, give 0.5–0.75 mg/kg IV/IO followed by 10- to 20-mL NS flush every 5–10 min, up to a maximum dose of 3 mg/kg ■ Doses range from 0.5–0.75 mg/kg and up to 1–1.5 mg/kg ■ Repeat 0.5–0.75 mg/kg every 5–10 min, up to a maximum dose of 3 mg/kg Maintenance: ■ 1–4 mg/min (30–50 mcg/kg/min)	■ Do not use prophylactically in patients with acute myocardial infarction ■ Monitor patient for lidocaine toxicity ■ Reduce maintenance dose in patients with hepatic disease or left ventricular dysfunction ■ Do not alternate between amiodarone and lidocaine

Table 3-6 | Commonly Used IV/IO Drugs in Resuscitation Situations *(continued)*

Drug	Action	Indications	Administration	Precautions
Magnesium sulfate	Decreases acetylcholine in nerve terminals and acts on myocardial tissue to decrease rate of SA node impulse formation, prolonging conduction time	■ Torsades de pointes ■ Digitalis toxicity ■ Hypomagnesemia	■ 1–2 g diluted in 10 mL of 5% dextrose in water or NS administered as an IV/IO bolus over 5–20 min	■ Use with caution in patients with renal failure ■ Rapid administration may induce hypotension ■ May precipitate opioid withdrawal syndrome (rarely life threatening)
Naloxone	Competitively binds to μ-opioid receptors	■ Opioid overdose	■ 0.4 mg IV/IO/IM or 2 mg IN repeated every 4 minutes	■ May precipitate opioid withdrawal syndrome (rarely life threatening)
Norepinephrine	Acts on both α- and ß-adrenergic receptors to increase heart rate, contractility and vasoconstriction; increases systemic blood pressure and coronary blood flow	■ After cardiac arrest	■ 0.1–0.5 mcg/kg/min IV/IO	■ Potent vasoconstrictor; extravasation can lead to necrosis
Procainamide	Class Ia antiarrhythmic (sodium channel blocker); delays repolarization and prolongs the QT interval	Stable wide-complex VT	■ 20–50 mg/min until arrhythmia is suppressed or maximum dose of 17 mg/kg is given ■ Maintenance: 1–4 mg/min	■ May induce torsades de pointes ■ Avoid in prolonged QT or congestive heart failure
Sotalol	Prolongs repolarization; also acts as a ß-blocker	Stable wide-complex VT	■ 100 mg (1.5 mg/kg) over 5 min	■ Avoid in prolonged QT

Table 3-7 | Other Drugs Used in the Treatment of Cardiovascular and Cerebrovascular Disorders

Drug	Used for
Aspirin	Acute coronary syndromes, stroke
β-Blockers (e.g., metoprolol, atenolol, propranolol, esmolol, labetalol)	Acute coronary syndromes, tachyarrhythmias, stroke
Bivalirudin	Acute coronary syndromes
Calcium channel blockers	
■ Diltiazem	Tachyarrhythmias
■ Verapamil	Tachyarrhythmias
■ Nicardipine	Stroke
Glycoprotein IIb/IIIa inhibitors	Acute coronary syndromes
Heparin	Acute coronary syndromes
Morphine	Acute coronary syndromes
Nitroglycerin	Acute coronary syndromes
Nitroprusside	Stroke
$P2Y_{12}$ inhibitors	Acute coronary syndromes
Tissue plasminogen activators	
■ Recombinant tissue plasminogen activator (alteplase)	Acute coronary syndromes, stroke
■ Reteplase	Acute coronary syndromes
■ Tenecteplase	Acute coronary syndromes

SKILL SHEET

Inserting an Oropharyngeal Airway (OPA)

Step 1 Select the proper size

Measure the OPA from the corner of the patient's mouth to the angle of the jaw.

Step 2 Open the patient's airway

Open the airway to a past-neutral position using the head-tilt/chin-lift technique, or use the modified jaw-thrust maneuver.

Step 3 Open the patient's mouth

Open the patient's mouth using the cross-finger technique.

SKILL SHEET

Inserting an Oropharyngeal Airway (OPA) *(continued)*

Step 4 Insert the OPA

- Insert the OPA upside down, with the tip pointing up.
- As the tip approaches the posterior pharynx, rotate the OPA 180 degrees into the proper position.

Step 5 Ensure correct placement

The flange should rest on the patient's lips.

SKILL SHEET

Inserting a Nasopharyngeal Airway (NPA)

Step 1 Select the proper size

- Select an NPA that is smaller in diameter than the inner aperture of the patient's nostril.
- Measure the NPA from the nostril to the angle of the jaw.

Step 2 Apply a water-soluble lubricant

Lubricate the NPA and the opening of the nostril.

SKILL SHEET

Inserting a Nasopharyngeal Airway (NPA) *(continued)*

Step 3 Insert the NPA

- **Right nostril:** Insert the NPA with the bevel toward the septum. Gently advance the NPA straight in, following the floor of the nose and avoiding excessive force.
- **Left nostril:** Insert the NPA with the bevel toward the septum. Gently advance the NPA, rotating it as you advance it past the nasal cavity.

Step 4 Ensure correct placement

The flange should rest on the nostril.

SKILL SHEET

Using a Bag-Valve-Mask (BVM) Resuscitator—One Provider

Step 1 Select an appropriately sized resuscitator

The mask should not cover the patient's eyes or extend below the patient's chin. Assemble the equipment as needed.

Step 2 Place the mask

Position yourself behind the patient's head (cephalic position). Place the mask at the bridge of the nose and then lower it over the nose, mouth and chin.

Step 3 Seal the mask and open the airway

- Place one hand around the mask, forming a C with your thumb and index finger around the side of the mask and an E with the last three fingers under the patient's jaw.
- Simultaneously seal the mask and open the airway to the past-neutral position by lifting the patient's jaw up into the mask.

Step 4 Provide ventilations

- While maintaining the mask seal and an open airway with one hand, use the other hand to depress the bag about halfway to deliver a tidal volume of 400 to 700 mL.
- Watch for chest rise.
- Continue providing smooth and effortless ventilations that last about 1 second and cause the chest to just begin to rise.

SKILL SHEET

Using a Bag-Valve-Mask (BVM) Resuscitator—Two Providers

Step 1 Select an appropriately sized resuscitator

The mask should not cover the patient's eyes or extend below the patient's chin. Assemble the equipment as needed.

Step 2 Place the mask

Provider 1 gets into position behind the patient's head (cephalic position). Provider 1 places the mask at the bridge of the nose and then lowers it over the nose, mouth and chin.

Step 3 Seal the mask and open the airway

Provider 1:

- Places both hands around the mask, forming a C with the thumb and index finger around the sides of the mask and an E with the last three fingers of each hand under the patient's jaw.
- Simultaneously seals the mask and opens the airway to the past-neutral position by lifting the patient's jaw up into the mask.

Step 4 Provide ventilations

- Provider 1 maintains the mask seal and an open airway.
- Provider 2 depresses the bag about halfway to deliver a tidal volume of 400 to 700 mL. Provider 2 provides smooth and effortless ventilations that last about 1 second and cause the chest to just begin to rise.
- Both providers watch for chest rise.

SKILL SHEET

Placing Electrodes for Electrocardiography

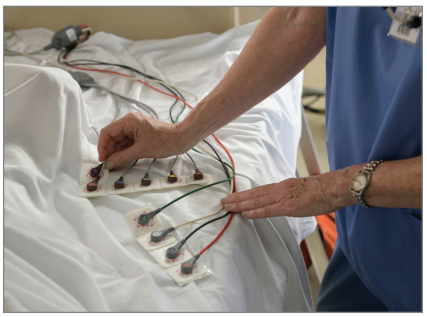
12-Lead ECG

Step 1 Prepare the skin where the electrodes will be placed

- Make sure the skin is clean, dry and free of excess hair.
- Using a skin prep pad, gently abrade the skin to remove dead skin cells.

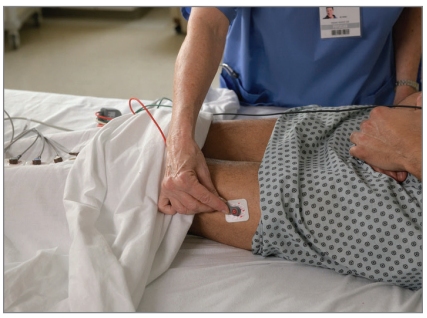
Step 2 Prepare equipment

Attach electrodes to the cables.



Step 3 Apply the limb electrodes

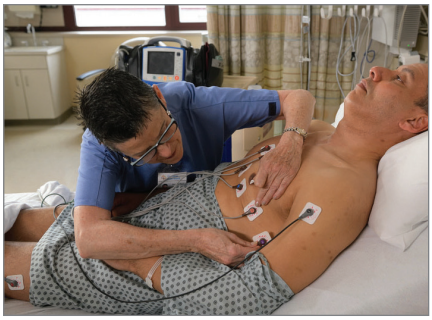
- On the arms, place the electrodes between the shoulders and the elbows.
- On the legs, place the electrodes on the thighs or calves, avoiding bony areas.



SKILL SHEET

Placing Electrodes for Electrocardiography *(continued)*

Step 4 Apply the chest electrodes



- **V_1**: Palpate the jugular notch, then palpate down to identify the sternal angle (angle of Louis), which is adjacent to the second rib. Palpate along the right sternal border to identify the second, third and fourth intercostal spaces. Place the electrode for V_1 over the fourth intercostal space at the right sternal border.
- **V_2**: Place the electrode for V_2 over the fourth intercostal space at the left sternal border.
- **V_4**: Place the electrode for V_4 over the fifth intercostal space at the midclavicular line on the patient's left side.
- **V_3**: Place the electrode for V_3 halfway between electrodes V_2 and V_4.
- **V_5**: Place the electrode for V_5 at the anterior axillary line on the patient's left side, even with electrode V_4.
- **V_6**: Place the electrode for V_6 at the midaxillary line on the patient's left side, even with electrodes V_4 and V_5.

Note: If necessary, lift breast tissue to place electrodes as close to the chest wall as possible.

15-Lead ECG

Step 1 Obtain a 12-lead ECG

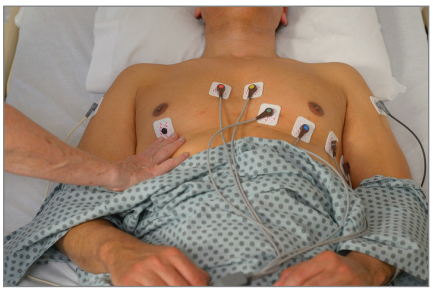
Run a standard 12-lead ECG.

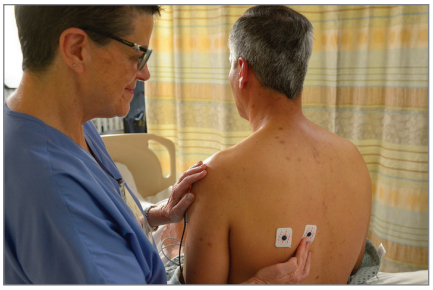
SKILL SHEET

Placing Electrodes for Electrocardiography *(continued)*

Step 2 Place additional electrodes

- **V_{4R}**: Place the electrode for V_{4R} over the fifth intercostal space at the midclavicular line on the patient's right side.
- **V_8**: Place the electrode for V_8 over the fifth intercostal space at the midscapular line on the patient's left side.
- **V_9**: Place the electrode for V_9 over the fifth intercostal space between V_8 and the spine.

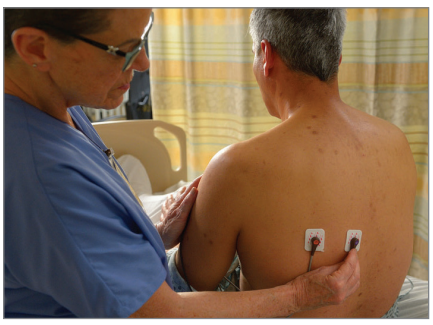




Step 3 Move cables to obtain additional views

Detach the cables for leads V_4, V_5 and V_6.

- Place the cable from V_4 on V_{4R}.
- Place the cable from V_5 on V_8.
- Place the cable from V_6 on V_9.



Step 4 Obtain a second 12-lead ECG

- Run a second 12-lead ECG to capture the three additional leads.
- Re-label the three additional leads on the rhythm strip.

SKILL SHEET

Manual Defibrillation

Step 1 Select appropriately sized pads

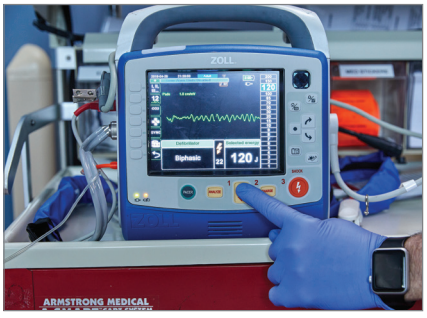
Choose the largest adult pad available.

Step 2 Apply the pads to the patient's chest

Apply the pads to the patient's chest, pressing firmly and making good skin contact.

- **Anterolateral placement:** Place the sternal pad on the patient's right side adjacent to the upper sternum, below the clavicle. Place the apical pad on the patient's left side over the fourth and fifth intercostal spaces, with the center of the pad at the midaxillary line.
- **Anterior-posterior placement:** Place the anterior pad on the patient's left side over the fourth and fifth intercostal spaces, with the center of the pad at the midaxillary line. Place the posterior pad in the left infrascapular region.

Step 3 Set the energy dose



- **Biphasic defibrillator:** Follow the manufacturer's recommendations for the initial dose (usually between 120 and 200 joules). Subsequent doses should be the same as or higher than the initial dose. If the manufacturer's recommendations for the initial dose are not known, use the highest energy dose available for the first and all subsequent shocks.
- **Monophasic defibrillator:** Set the energy dose at 360 joules. Use this energy dose for each subsequent shock.

Step 4 Charge the pads

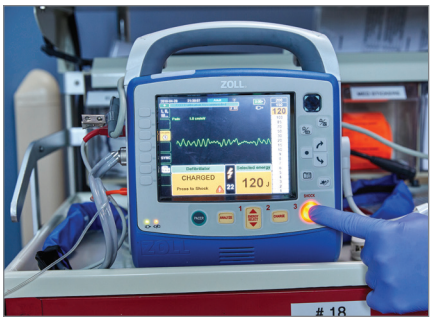
Press the "charge" button on the cardiac monitor/defibrillator.

SKILL SHEET

Manual Defibrillation *(continued)*

Step 5 Deliver the shock

- Instruct the team to "clear."
- Conduct a visual check to ensure that no one is touching the patient or the bed/stretcher and that oxygen delivery devices have been removed and set aside, away from the patient.
- Press the "shock" button on the cardiac monitor/defibrillator to deliver the shock.



Step 6 Resume CPR

Immediately resume CPR and then reassess the rhythm in 2 minutes. If a shockable rhythm persists, deliver another shock, using an energy dose that is the same as or higher than the initial dose.

SKILL SHEET

Synchronized Cardioversion

Step 1 Identify the rhythm

Attach the cardiac monitoring leads and ensure that the patient's cardiac rhythm is shown on the monitor.

Step 2 Select appropriately sized pads

Choose the largest adult pad available.

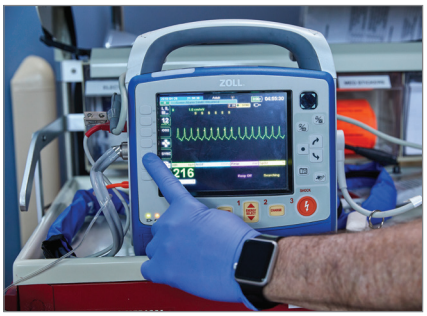
Step 3 Apply the pads to the patient's chest

Apply the pads to the patient's chest, pressing firmly and making good skin contact.

- **Anterolateral placement:** Place the sternal pad on the patient's right side adjacent to the upper sternum, below the clavicle. Place the apical pad on the patient's left side over the fourth and fifth intercostal spaces, with the center of the pad at the midaxillary line.
- **Anterior-posterior placement:** Place the anterior pad on the patient's left side over the fourth and fifth intercostal spaces, with the center of the pad at the midaxillary line. Place the posterior pad in the left infrascapular region.

Step 4 Select synchronous mode

Verify that sync markers are visible at the top of each R wave.

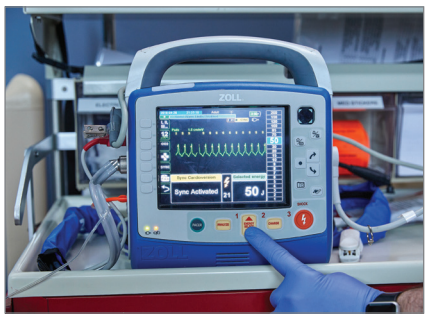


SKILL SHEET

Synchronized Cardioversion *(continued)*

Step 5 Set the energy dose

- **Narrow-complex regular rhythm:** 50 to 100 joules (biphasic or monophasic)
- **Narrow-complex irregular rhythm:** 120 to 200 joules (biphasic), 200 joules (monophasic)
- **Wide-complex regular rhythm:** 100 joules (biphasic or monophasic)



Step 6 Charge the pads

Press the "charge" button on the cardiac monitor/defibrillator.

Step 7 Deliver the shock

- Instruct the team to "clear."
- Conduct a visual check to ensure that no one is touching the patient or the bed/stretcher and that oxygen delivery devices have been removed and set aside, away from the patient.
- Press and hold the "shock" button on the cardiac monitor/defibrillator to deliver the shock.

Step 8 Reassess

Reassess the rhythm and the patient.

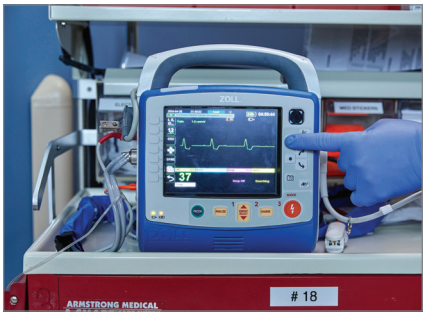
- If the rhythm did not convert, reset the cardiac monitor/defibrillator to synchronous mode, increase the energy level in a stepwise fashion, charge the pads and deliver a shock.
- If the rhythm did convert, check the patient's vital signs and ensure adequate airway, breathing and circulation.

SKILL SHEET

Transcutaneous Pacing

Step 1 Identify the rhythm

Attach the cardiac monitoring leads and ensure that the patient's cardiac rhythm is shown on the monitor.



Step 2 Apply the pacing pads to the patient's chest

Apply the pacing pads to the patient's chest, pressing firmly and making good skin contact.

- **Anterolateral placement:** Place the sternal pad on the patient's right side adjacent to the upper sternum, below the clavicle. Place the apical pad on the patient's left side over the fourth and fifth intercostal spaces, with the center of the pad at the midaxillary line.
- **Anterior-posterior placement:** Place the anterior pad on the patient's left side over the fourth and fifth intercostal spaces, with the center of the pad at the midaxillary line. Place the posterior pad in the left infrascapular region.

Step 3 Select pacing mode

Set the cardiac monitor/defibrillator to pacing mode.

SKILL SHEET

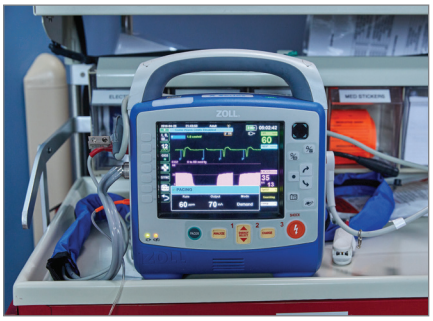
Transcutaneous Pacing *(continued)*

Step 4 Set the demand rate

Set the demand rate to approximately 60 beats per minute. Once pacing is established, the demand rate can be adjusted according to the patient's clinical response.

Step 5 Set the current milliamperes output

Set the current milliamperes output by starting low and gradually increasing it until consistent electrical capture is observed on the monitor (wide QRS complexes with tall, broad T waves).



Step 6 Check for mechanical capture

Check for mechanical capture (evidenced by clinical signs such as a palpable pulse, an increase in blood pressure and an increase in end-tidal carbon dioxide ($ETCO_2$) level on capnography.

SKILL SHEET

Intraosseous Access (Drill)

Step 1 Stabilize the target site

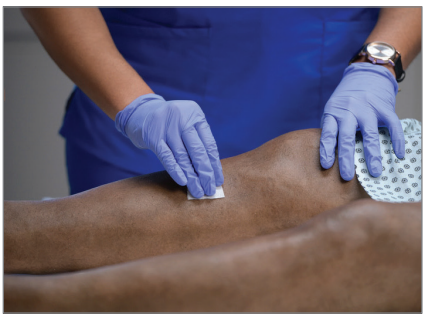
Stabilize the target site on a firm surface.

Step 2 Select the correct needle size

Palpate the site to determine tissue depth.

Step 3 Disinfect the skin

Disinfect the skin overlying the insertion site.



Step 4 Consider pain control

If the patient is awake, consider infiltration of the skin and periosteum with 1% lidocaine.

Step 5 Attach the needle

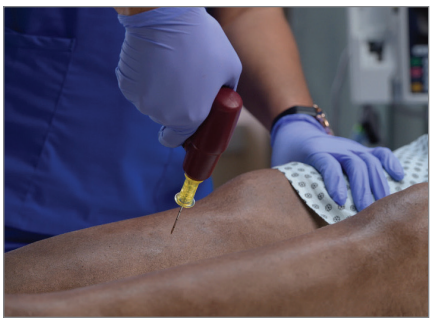
Attach the needle to the IO drill.

SKILL SHEET

Intraosseous Access (Drill) *(continued)*

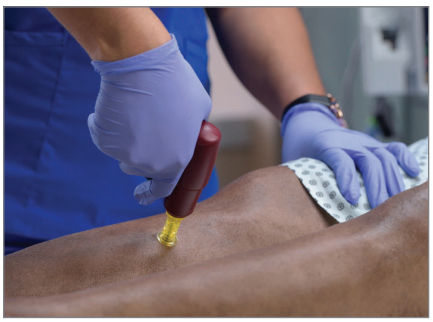
Step 6 Position the needle

Aim the IO drill with the needle at a 90-degree angle to the insertion site. Push the needle through the skin until the tip is against the bone. The 5-mm mark on the needle must be visible above the skin when the needle is resting on the bone.



Step 7 Drill the needle into the bone

Pressing the trigger on the device, lightly drill the needle into the bone until you feel a decrease in resistance.



Step 8 Remove the drill

Hold the needle securely in place as you pull the drill straight off. When inserted correctly, the needle should feel like it is firmly in the bone and should remain upright without support.

SKILL SHEET

Intraosseous Access (Drill) *(continued)*

Step 9 Remove the stylet

Remove the stylet by twisting it counterclockwise, and dispose of it properly.

Step 10 Position the stabilizer dressing

Place the stabilizer dressing over the needle.

Step 11 Attach the primed tubing

Hold the needle securely and attach the primed tubing to the needle.

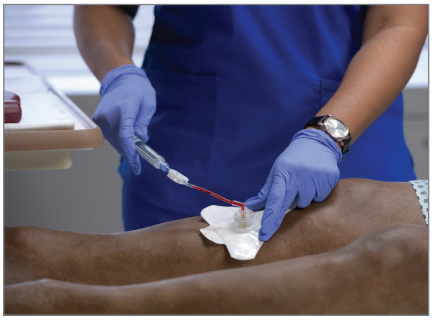
SKILL SHEET

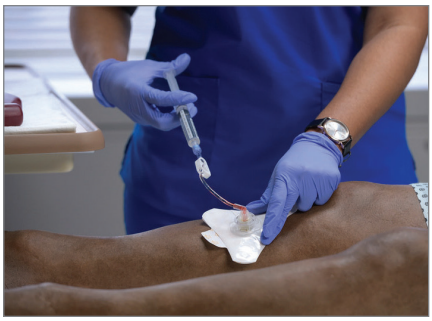
Intraosseous Access (Drill) *(continued)*

Step 12 Confirm placement

Confirm correct placement of the needle by using one of the following methods:

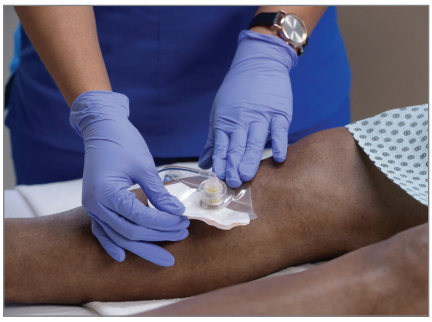
- Aspirate bone marrow or blood through the needle.
- Flush the needle with a small amount of saline and check for extravasation.
- Administer fluids by free flow through the needle.





Step 13 Secure the dressing

Peel the adhesive tabs off the stabilizer dressing and press the dressing firmly onto the skin.



Step 14 Monitor the insertion site

Monitor the insertion site and the extremity for swelling.

SKILL SHEET

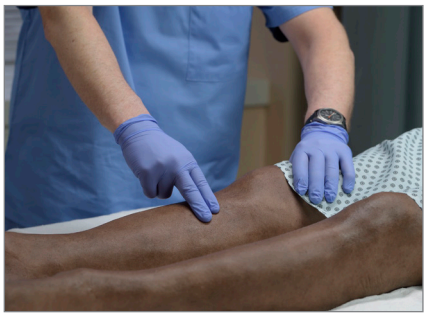
Intraosseous Access (Manual Insertion)

Step 1 Stabilize the target site

Stabilize the target site on a firm surface.

Step 2 Select the correct needle size

Palpate the site to determine tissue depth.



Step 3 Disinfect the skin

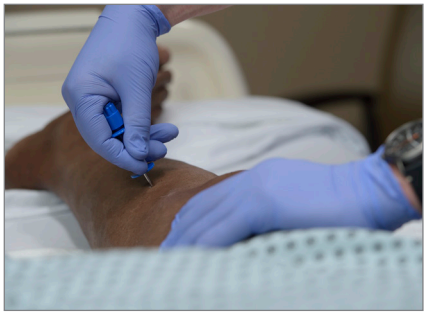
Disinfect the skin overlying the insertion site.

Step 4 Consider pain control

If the patient is awake, consider infiltration of the skin and periosteum with 1% lidocaine.

Step 5 Position the needle

Position the needle at a 90-degree angle to the insertion site.
Push the needle through the skin until the tip is against the bone.

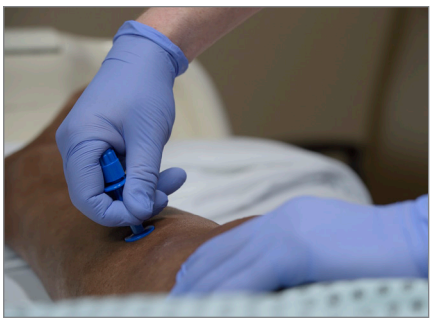


SKILL SHEET

Intraosseous Access (Manual Insertion) *(continued)*

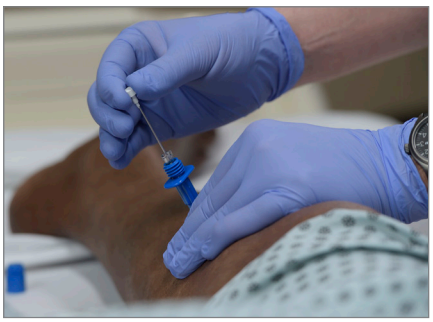
Step 6 Insert the needle into the bone

Once the needle reaches the periosteum, apply pressure in a back-and-forth twisting motion until you feel a decrease in resistance. When inserted correctly, the needle should feel like it is firmly in the bone and should remain upright without support.



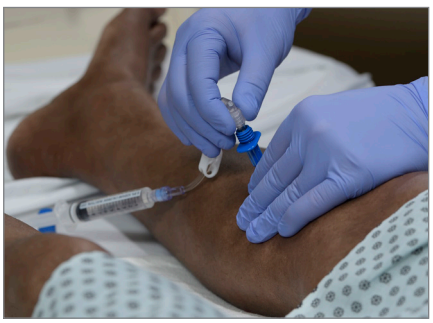
Step 7 Remove the stylet

Remove the stylet by twisting it counterclockwise, and dispose of it properly.



Step 8 Attach the primed tubing

Hold the needle securely and attach the primed tubing to the needle.



Step 9 Confirm placement

Confirm correct placement of the needle by using one of the following methods:

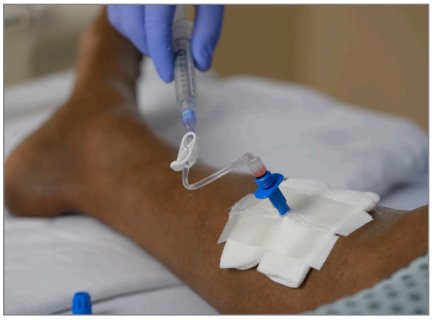
- Aspirate bone marrow or blood through the needle.
- Flush the needle with a small amount of saline and check for extravasation.
- Administer fluids by free flow through the needle.

SKILL SHEET

Intraosseous Access (Manual Insertion) *(continued)*

Step 10 Secure the needle

Tape the flange to the skin and add a gauze dressing for support.



Step 11 Monitor the insertion site

Monitor the insertion site and the extremity for swelling.

CHAPTER 4

Working Well Together in an Emergency

Introduction

Working well together as a team when caring for a patient experiencing a cardiovascular, cerebrovascular or respiratory emergency is vital. Poor teamwork can lead to poor outcomes in emergency situations. Conversely, effective teamwork can be the difference between life and death for the patient who requires emergency care.

Rapid Response and Resuscitation Teams

Many healthcare facilities have implemented systems designed to improve patient outcomes by decreasing the likelihood of cardiopulmonary arrest in unstable patients and reducing mortality when cardiopulmonary arrest does occur. These systems rely on teams of highly trained and skilled personnel, known as rapid response teams and resuscitation teams. The goal of the **rapid response team** is to intervene quickly and effectively to address the warning signs of impending cardiopulmonary arrest so that the arrest can be prevented. The goal of the **resuscitation team** is to respond quickly and effectively to provide advanced life support care to a patient in respiratory or cardiac arrest.

Patients in respiratory distress or shock tend to decompensate quickly, and respiratory and cardiac arrest may result. In critical care settings such as a critical care unit or emergency department, highly trained teams are already in place and advanced life support care will be implemented quickly. However, patients outside of these settings are also at risk for experiencing respiratory or cardiac arrest. Thus all healthcare providers should be trained to recognize early signs of clinical deterioration in patients and should know how to initiate the emergency response as quickly as possible.

Rapid Response Systems

Rapid response systems became commonly integrated in hospitals in the mid-2000s as a response to the 100,000 Lives Campaign initiated by the Institute for Healthcare Improvement, which aimed to reduce the percentage of preventable in-hospital deaths. Today, all hospitals in the United States are required by the Joint Commission National Patient Safety Goals to have a system in place to better recognize and respond to significant changes in a patient's status. Thus, all hospitals, and some other types of healthcare facilities, have some form of a rapid response system in place. Rapid response teams (also called multidisciplinary medical emergency teams) work together to care for the patient when signs and symptoms of cardiopulmonary compromise or shock are noted (Figure 4-1). The specific composition of the team may vary, but generally rapid response teams include critical care nurses, respiratory therapists, and a critical care physician or hospitalist, nurse practitioner or physician assistant. No matter the composition of the team, the goal of every rapid response team is to quickly address the symptoms and underlying cause when a patient is in distress to *prevent* the occurrence of respiratory or cardiac arrest.

Figure 4-1 | Rapid response teams intervene early to prevent a patient's condition from worsening.

Any healthcare provider can call for a rapid response team; often the nurse providing direct patient care makes the call. Regardless, guidelines are always in place for calling a team. Each facility uses its own, often very specific, criteria to activate the rapid response system. These criteria may be called "triggers," or an early warning score system may be used. In general, the rapid response system would be activated for the following reasons:

- An acute change in respiratory rate, pattern or status from baseline (especially acute onset or worsening respiratory distress)
- Airway compromise
- A respiratory rate greater than 28 breaths/min or less than 8 breaths/min
- An oxygen saturation less than 90% despite supplementation
- A heart rate greater than 140 bpm or less than 40 bpm
- A systolic blood pressure greater than 180 mmHg or less than 90 mmHg
- An acute change in urine output or a urine output of less than 50 mL over 4 hours
- An acute change in mental status
- An acute change in pain status (or uncontrolled pain)
- Seizure activity (new onset or prolonged)
- Staff or family concern

Resuscitation Teams

Resuscitation teams, like rapid response teams, are multidisciplinary teams of highly trained and skilled healthcare professionals. Team members may vary, but teams often include a critical care physician, a critical care nurse, a respiratory therapist, a hospitalist, a pharmacist and a chaplain. The goal of the resuscitation team is singular and focused: to provide resuscitative care when a patient experiences respiratory or cardiac arrest (Figure 4-2).

Functioning as a Team

Healthcare providers on rapid response and resuscitation teams must work together in a coordinated effort to achieve the best possible outcomes for each patient. **Teamwork** refers to a group of people with well-defined roles and responsibilities working toward a common goal. Teamwork is crucial during rapid response and resuscitation because the ultimate goal is to save a life, and effective team care requires a coordinated effort by the team leader and the team members.

To achieve the best possible outcomes, every team leader and team member must exhibit key skills including communication, critical thinking and problem solving. Implementing effective and efficient teamwork and exhibiting these critical key skills allows the team to respond efficiently and effectively, and it improves patient outcomes.

Figure 4-2 | The resuscitation team provides resuscitative care to patients in cardiac or respiratory arrest.

Communication

Communication is essential when caring for a patient who is experiencing a cardiovascular, cerebrovascular or respiratory emergency. You need to communicate with your colleagues, the patient and the patient's family.

Communication involves four essential components (Figure 4-3):

- **Sender**: The person initiating the communication
- **Message**: The content of the communication; this must be expressed clearly so that everyone involved knows exactly what the message is
- **Receiver**: The person for whom the message is intended
- **Feedback**: The confirmation by the receiver that the message is received and understood; an essential element of **closed-loop communication**

Communication includes spoken words (verbal messages) and nonverbal messages conveyed through body language, such as gestures and facial expressions.

Communicating with the Team

The foundation of effective teamwork is clear and effective communication among team members. When a team is working to provide care, a designated team leader directs the efforts of the other team members. When communicating information or assigning tasks to team members, the team leader must be sure to speak clearly and deliberately, to convey information in an organized fashion and to "close the loop" by waiting for feedback from the team member responsible for carrying out the action. If feedback is not provided, the team leader should seek it before continuing. Similarly, team members must provide confirmation that they have received the message and that they understand it by repeating the task back to the team leader and acknowledging initiation and completion of the

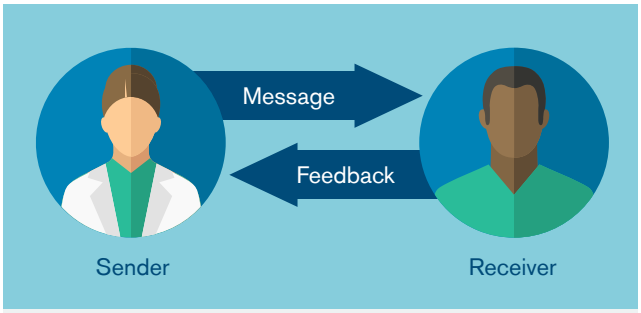


Figure 4-3 | Clear communication among team members is essential. The sender initiates the communication, sending a clear message. The receiver provides feedback, confirming that the message was understood and the task has been completed.

task. No matter what your role is on the team, speak clearly in a calm tone of voice, and take care not to speak over others.

Communicating with the Family

Patients who require resuscitation are unresponsive, making communication with the family very important. Remember, during emergencies, families are stressed and may not always hear what you are saying. Speak slowly and in terms the family can understand. Build rapport and establish trust. Be prepared to repeat information, if necessary. Be open and honest, especially about the patient's condition. Minimize family members' fears, as necessary, but avoid giving misleading information or false hope. Reassure the family that everything that can be done is being done. In doing so, you need to demonstrate credibility and trustworthiness, confidence and empathy.

In advanced life support situations, patients may not survive, despite the team's best resuscitation efforts. As a healthcare provider, you may be involved in communicating with the family about a patient's death. In this situation:

- Provide information honestly and with compassion, in a straightforward manner, and include information about events that may follow.
- Allow the family to begin processing the information.
- Allow time for the family to begin the grief process. Ask whether they would like to contact or have you contact anyone, such as other family members or clergy.
- Anticipate emotional reactions, which may include crying, sobbing, shouting, anger, screaming or physically lashing out.
- Wait and answer any questions that the family may have.

Critical Thinking

Critical thinking refers to thinking clearly and rationally to identify the connection between information and actions. When you use critical thinking, you are constantly identifying new information, adapting to the information logically in order to determine your best next actions and anticipating how those actions will affect the patient.

Critical thinking is an essential skill in healthcare and especially in advanced life support situations. You use critical thinking when you:

- Obtain an initial impression.
- Determine a course of action.
- Anticipate roles and functions as part of a team based on the patient's presentation and condition.
- Consistently re-evaluate the situation for changes, interpret these changes and apply them to the patient's care and treatment.
- Modify your actions based on the changes you observe.

Problem Solving

Problem solving refers to the ability to use readily available resources to find solutions to challenging or complex situations or issues that arise. In emergency situations, problems or issues can occur at any point. For example, a team member may be unable to find a vein adequate for achieving vascular access. Another emergency might occur, leaving the team short-staffed. An upset family member may interfere with care.

Problem solving also requires creativity in finding solutions. Use whatever resources are at hand, including equipment, other team members or other healthcare facility staff.

Working as a High-Performance Team

Your role on an advanced life support team may vary according to your training and areas of expertise. In addition to understanding your own role on the team, it is important to understand the roles of other team members as well. This knowledge will help you function effectively in an advanced life support situation.

Members of effective teams keep their skills and knowledge current, and they practice together regularly (Figure 4-4A). In addition, effective teams hold debriefing sessions after each resuscitation event (Figure 4-4B). The debriefing session is an opportunity to review successes, as well as areas where improvement is needed.

Team Leader Responsibilities

The team leader oversees the entire emergency situation and organizes and runs the response. The team leader does not perform a particular task but is responsible for making sure all team members perform necessary tasks according to their roles. The coordination of all involved is necessary to:

- Ensure that everyone works as a team to help promote the best possible outcome for the patient.
- Promote effective perfusion to vital organs.
- Minimize interruptions of chest compressions, which improves patient survival.

Figure 4-4 | Effective teams (A) practice together regularly and (B) hold debriefing sessions after every resuscitation event.

The team leader:

- Assigns and understands team roles.
- Sets clear expectations.
- Prioritizes, directs and acts decisively.
- Encourages and allows team input and interaction.
- Focuses on the big picture.
- Monitors performance while providing support.
- Acts as a role model.
- Coaches the team.
- Re-evaluates and summarizes progress.
- Leads a debriefing session.

Team Member Responsibilities

Team members provide care with skill and expertise. Team members:

- Have the necessary knowledge and skills to perform their assigned role.
- Stay in their assigned role but assist others as needed, as long as they are able to maintain their own assigned responsibilities.
- Communicate effectively with the team leader if they:
  - Feel they are lacking any knowledge or skills.
  - Identify something that the team leader may have overlooked.
  - Recognize a dangerous situation or need for urgent action.
- Share information with other team members.
- Focus on achieving the goals.
- Ask pertinent questions and share pertinent observations.
- Participate in the debriefing session.

Coordinated Team Response

When the team leader and all of the team members, in their assigned roles, work together as a high-performance team, expert care is delivered and outcomes are improved. In a highly effective team working together in an emergency situation, the team leader will assign the team roles. These roles may differ according to healthcare facility policy. A typical six-person high-performance resuscitation team includes team members who perform CPR/defibrillator roles, as well as team members who perform leadership and supportive roles (Figure 4-5). All team members should be trained and able to perform multiple roles on the team, within their scope of practice.

CPR/Defibrillator Roles

The CPR/defibrillator roles include the following:

- **Compressor.** One team member is responsible for chest compressions.
- **AED/defibrillator operator.** One team member is responsible for managing the AED or defibrillator and for establishing any other monitoring. This team member also relieves the team member who is giving compressions.
- **Airway manager/ventilator.** One team member is responsible for managing the airway and providing ventilations. A trained respiratory therapist, if available, would fill this role.

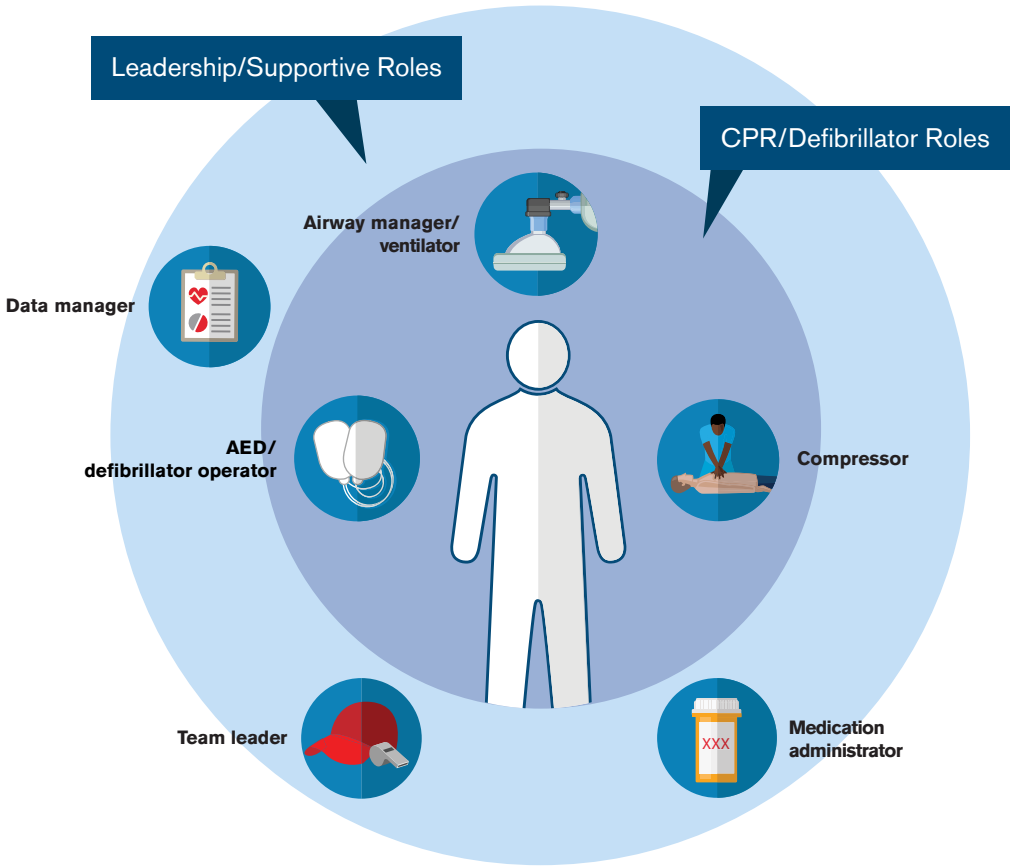


Figure 4-5 | A resuscitation team usually includes six people.

Leadership and Supportive Roles

The leadership and supportive roles include the following:

- **Team leader.** One team member functions as the team leader.
- **Medication administrator.** One team member is responsible for establishing vascular access and administering medications.
- **Data manager.** One team member is responsible for keeping track of elapsed time and communicating and recording key data during the resuscitation effort, such as the nature and timing of interventions.

Crew Resource Management

Crew resource management is a concept that helps to promote effective and efficient teamwork and reduce the likelihood of errors. Originally developed by the aviation industry in the 1970s in response to several airline disasters where human error and poor communication were found to be contributing factors, crew resource management has been adapted for use as a tool in the healthcare setting as well. Crew resource management emphasizes using all available resources (including people, equipment and procedures) to reduce the likelihood of human error and promote effective and efficient teamwork. When following the principles of crew resource management, all members of the team demonstrate respect for one another and use clear, closed-loop communication.

Crew resource management centers around the team leader, who coordinates the actions and activities of team members so that the team functions effectively and efficiently. For example, when team members switch roles during an emergency, the team leader is responsible for coordinating these activities. Crew resource management also guides team members to communicate directly and effectively with the team leader about dangerous or time-critical decisions. Being a member of the team is just as important as being a team leader. Everyone on the team needs to have a voice and be encouraged to speak up if a problem arises. When a problem arises, team members must get the attention of the team leader, state their concern, describe the problem as they see it and suggest a solution. The team leader then provides direction, enabling the team to work together to resolve the issue.

Phased Response Approach

The **phased response approach**, first introduced in 1987 by Burkle and Rice, can be used to describe the process of a resuscitation team in action during a resuscitation event. The phased response approach refers to how a group of people, working as a team, reacts to and handles an emergency using crew resource management and the key skills of communication, critical thinking and problem solving. This response approach consists of seven phases.

Anticipation

In the anticipation phase, the team leader gathers any preliminary information about the emergency (e.g., the patient's age and present illness, significant medical history, and events leading up to the emergency) and plans for the resuscitation or emergency response by assigning team roles and gathering the equipment needed to handle the situation. If the emergency care will take place in a location other than where the team is located, the leader and team members go the site of the emergency as quickly as possible.

Entry

In this phase, the patient is brought to the emergency department or the team arrives at the patient's location in the healthcare facility. The team members assess the patient and collect vital signs, transfer the patient as needed (e.g., from bed to bed) and ensure that the patient is positioned properly for the emergency interventions taking place. One team member quickly and efficiently obtains information from the person who identified the emergency and called for "the code."

Resuscitation

Resuscitation is the most critical phase of the approach. During this phase, the team leader directs the team members to perform actions per treatment guidelines; communication is essential in this phase. The team members must also report any changes in the patient's clinical condition, such as changes in heart rhythm, decreasing oxygen saturation or changes in blood pressure. If resuscitation efforts are successful, a patient in cardiac arrest will achieve return of spontaneous circulation (ROSC).

Maintenance

In the maintenance phase, the team focuses on stabilizing the patient's vital signs and using intelligent intuition to predict any potential problems that may occur. The primary assessment is repeated during this phase, any required laboratory samples are collected and the care provided is documented. If the patient must be transferred, preparation for the transfer takes place in this phase.

Family Notification

Although listed as a phase, family notification is actually an ongoing process throughout the resuscitation or emergency care event. Ideally, one member of the team is dedicated to this role so that family members are kept informed throughout the process.

Transfer

After the rapid response team, the resuscitation team or both provide care, the patient is likely to be transferred to a critical care unit or other unit capable of providing specialized care. The team leader, with input from the team members, determines the appropriate level of care required for each situation.

Debrief

As soon as possible after the resuscitation is over, the team should review their performance. The debriefing session is an opportunity to reflect on and analyze the care that was provided and to learn from mistakes, as well as successes. The purpose of the debriefing session is not to place blame; rather, it is to take a closer look at the decisions that were made and the actions that were taken with the goal of identifying opportunities for improvement at the system, team and individual level. The debriefing phase is also a time for team members to decompress. Some resuscitations can be very traumatic or emotionally laborious; this phase can be a time for the team to grieve.

The team leader leads the debriefing session, which typically follows a consistent format. For example:

- **Review:** The team leader provides a brief recap of the emergency and the interventions that were used.
- **Analyze:** The team reviews and evaluates the objective data obtained during the resuscitation effort.
- **Reflect:** The team reflects on the actions they took and why, discusses the pros and cons of those actions and identifies changes that could be made to improve future outcomes.
- **Summarize:** The team recaps the main takeaway points and develops a list of action items.

CHAPTER 5

Assessment

Introduction

The data you gather through ongoing assessment of the patient informs the decisions that you make in order to provide appropriate care. Taking a systematic approach to assessment allows you and the team to focus on identifying and addressing the most critical problems first, and helps to ensure that important details about the patient's condition and underlying causes are not overlooked.

Assess, Recognize and Care

Assess, Recognize and Care (ARC) is a concept that describes the ongoing process of gathering data about the patient's condition, using that data to identify a problem and then intervening to address the problem (Figure 5-1). Because an acutely ill patient's condition can change rapidly (for better or for worse), you must continuously assess the patient, recognize what is happening with the patient and provide care accordingly.

Assess

Assessment is the process of gathering the data that helps you to determine what is happening with the patient. In order to ensure that the most pressing problems are addressed first, take a phased, systematic approach to assessment: perform a rapid assessment, a primary assessment and (when the patient's condition allows) a secondary assessment. In an emergency situation, assessment is ongoing.

Recognize

After you gather assessment data, use critical thinking, your past clinical experience and your general knowledge to correctly interpret the meaning of the data and gain an understanding of the patient's clinical situation and care needs. This understanding enables you to determine your next steps.

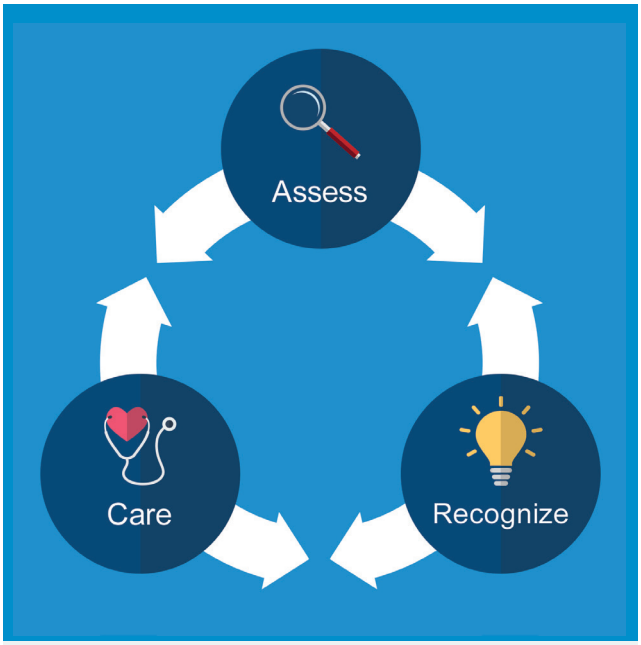


Figure 5-1 | Assess, Recognize and Care (ARC) is a concept that describes the ongoing process of gathering data about the patient's condition, using that data to identify a problem, and then intervening to address the problem.

Care

Based on your understanding of the patient's condition, implement appropriate care. Without effective assessment and accurate recognition of the patient's condition, proper care cannot be provided.

Rapid Assessment

The rapid assessment is a quick survey to ensure safety, form an initial impression about the patient's condition and, if the patient appears to be unresponsive, check for responsiveness, breathing and a pulse. The *Rapid Assessment: Adult* Treatment Guideline summarizes the approach to rapid assessment of an adult. The *Rapid Assessment for Adults* Skill Sheet in Chapter 2 provides step-by-step guidance for conducting a rapid assessment.

You can gain a great deal of preliminary information very quickly just by looking at the patient. For example:

- Does the patient appear to be unresponsive?
- Is the patient speaking?
- Does the patient appear to be working to breathe?
- Does the patient's skin appear to be its normal color, or does it seem pale, ashen, cyanotic or flushed?
- Does the patient appear to be diaphoretic?
- Is there any fluid or blood coming from, on or near the patient?
- Is there severe, life-threatening bleeding?

Practice Note

If you see severe, life-threatening bleeding immediately use any available resources to control the hemorrhage, including a tourniquet or hemostatic dressing if one is available.

If the patient appears to be unresponsive, perform a quick assessment to check for responsiveness, breathing and a pulse, and provide care based on your findings. Then complete the primary assessment (Figure 5-2).

If the patient appears to be responsive, complete the primary assessment first.

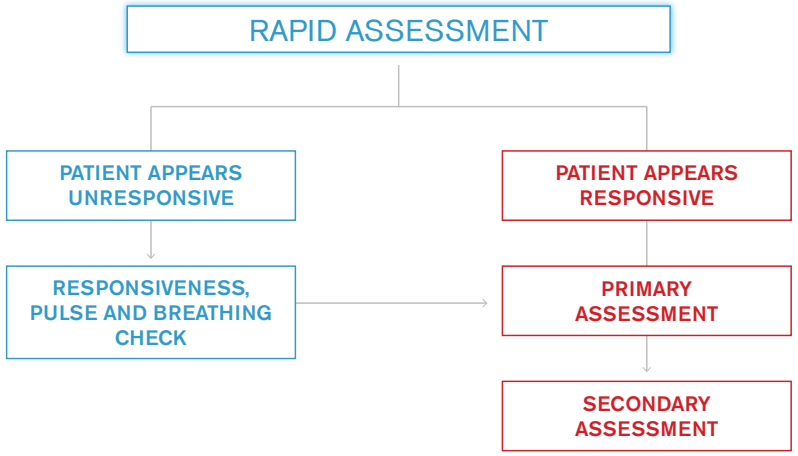


Figure 5-2 | A systematic approach to assessment includes a rapid assessment, a primary assessment and a secondary assessment. If the patient appears to be unresponsive on rapid assessment, check for responsiveness, breathing and a pulse before completing the primary assessment.

Primary Assessment

The primary assessment structures the approach to patient assessment around the mnemonic ABCDE (Airway, Breathing, Circulation, Disability, Exposure). The goal of the primary assessment is to identify potentially life-threatening conditions and correct them immediately, in order to prevent the patient's condition from deteriorating further (Figure 5-3). Provide interventions as needed and as resources permit, delegating care to appropriate team members as needed. The steps of the primary assessment should be repeated until the patient is stabilized, transferred to a higher level of care for further management, or both. The *Primary Assessment: Adult* Treatment Guideline summarizes the approach to primary assessment of an adult.

Figure 5-3 | During the primary assessment, assessment and intervention take place concurrently to identify and address potentially life-threatening problems related to airway, breathing, circulation, disability (neurologic status) and exposure.

Airway

Assess airway patency and maintainability. If the patient is able to speak to you in a normal voice, then the airway is patent. Signs that the upper airway may not be patent include:

- Abnormal sounds (e.g., stridor, wheezing, gurgling or snoring).
- An obstruction or foreign body (including secretions, blood or vomit) in the upper airway.
- Increased work of breathing or respiratory distress (e.g., nasal flaring, retractions).
- Altered level of consciousness.
- Cyanosis.

If the patient's airway is not open and clear, patency must be restored immediately and maintained. Methods of ensuring and maintaining a patent airway include:

- Patient positioning.
- Manual maneuvers, such as the head-tilt/chin-lift technique and the jaw-thrust maneuver.
- Techniques for clearing an airway obstruction (e.g., abdominal thrusts or foreign body removal using forceps with direct laryngoscopy).
- Suctioning.
- Placement of an oropharyngeal airway (OPA) or nasopharyngeal airway (NPA).
- Placement of an advanced airway.

If placement of an advanced airway is indicated, confirm proper placement using capnography and secure the device to prevent it from becoming dislodged.

Breathing

Assess breathing rate, depth and rhythm and observe for chest rise. Auscultate for the presence or absence of breath sounds, the presence of abnormal breath sounds (e.g., wheezes, rales, rhonchi or stridor) and for bilateral equality. Establish pulse oximetry to monitor oxygen saturation and if necessary, provide the minimal level of supplemental oxygen needed to maintain an oxygen saturation of at least 94%. Establish capnography to monitor the adequacy of ventilation. End-tidal carbon dioxide ($ETCO_2$) values in the range of 35 to 45 mmHg confirm adequacy of ventilation. If necessary, support breathing by giving ventilations with a bag-valve-mask (BVM) resuscitator.

Circulation

Assess the pulse rate, quality and rhythm, and establish cardiac monitoring. Assess the adequacy of perfusion by assessing blood pressure and capillary refill time, assessing for signs of shock and noting skin color, appearance and temperature. Signs of compromised perfusion include a prolonged capillary refill time (greater than 2 seconds); cool, pale or cyanotic skin in the extremities; an altered level of consciousness; and abnormally low $ETCO_2$ readings (i.e., less than 30 mmHg). If time and resources permit, obtain a 12-lead ECG.

Determine whether the patient needs defibrillation, cardioversion or pacing. Establish vascular access for the administration of fluids, medications or both.

Practice Note

Capnography allows rapid, objective and reliable assessment of airway, breathing and circulation. An $ETCO_2$ value in the normal range and a square waveform indicates that the patient's airway, breathing and circulation are intact:

- There is no obstruction to carbon dioxide emptying.
- Ventilation is adequate. An $ETCO_2$ value in the normal range correlates closely with the arterial pressure of carbon dioxide ($PaCO_2$), which means that ventilation is adequate.
- Perfusion is adequate. In low-perfusion states, it is impossible to achieve a normal $ETCO_2$ value.

Disability

Perform quick assessments to gain information about the patient's neurologic status:

- Assess the patient's level of consciousness using the AVPU (Alert, Verbal responsive, Pain responsive, Unresponsive) model:
  - **A**wake: The patient is fully awake (but may still be confused).
  - **V**erbal responsive: The patient responds to verbal stimuli.
  - **P**ain responsive: The patient responds to painful stimuli (e.g., a tap on the shoulder).
  - **U**nresponsive: The patient does not respond to any stimuli.
- Assess orientation to person, place and time.
- Check the pupils for size, equality and reactivity to light.
- Measure the patient's blood glucose level. Hypoglycemia can cause changes in level of consciousness and is easily corrected.
- If stroke is suspected, conduct a neurologic examination using the National Institutes of Health Stroke Scale (NIHSS) or similar assessment tool.

Exposure

As the last step of the primary assessment, check the patient's body for obvious signs of injury or illness, such as bleeding, bruises, rashes and deformities. Note skin color, appearance and temperature. Remove clothing as needed to inspect the head, ears, face, and neck; the anterior and posterior trunk; and the upper and lower extremities. If the patient has a suspected head, neck, spinal or pelvic injury, remember to consider spinal motion restriction while turning the patient over.

Practice Note

Older adults do not thermoregulate as well as younger adults; they can lose core body temperature much faster when their skin is exposed. Be sure to keep the areas not being actively assessed covered.

Practice Note

Bruises at various stages of healing or an inconsistent patient history or a history that does not align with the presenting injuries may indicate nonaccidental trauma. Follow your local and institutional policies for reporting suspected abuse.

Secondary Assessment

When the patient's condition has been stabilized, perform a secondary assessment. The secondary assessment includes a focused history, a focused physical examination and diagnostic tests. The goal of the secondary assessment is to gather more detailed information that will allow you to narrow the list of differential diagnoses and discover underlying, treatable causes (Figure 5-4).

Focused History

The focused history can be obtained by collecting data following the SAMPLE mnemonic:

S: Signs and symptoms
A: Allergies
M: Medications
P: Past medical history
L: Last intake and output
E: Events

Signs and Symptoms

Interview the patient (and, if necessary, family members or other healthcare providers) to identify signs and symptoms that occurred at the onset of the illness or injury. Ask follow-up questions as needed. For example, if the patient reports pain, ask the patient where the pain is located, when the pain started, and what the pain feels like.

Allergies

Determine whether the patient has any known allergies to medications, foods, latex, or environmental items. If the patient does report an allergy, ask what type of reaction the patient had in the past when exposed to the allergen, and what care the patient received. Even if the patient does not report an allergy, inquire about possible exposures to substances that are known to be allergens or toxins.

Figure 5-4 | During the secondary assessment, conducted after the patient has been stabilized, information is gathered with the aim of narrowing the differential diagnosis list, identifying underlying causes and determining candidacy for planned interventions.

Medications

Check what medications the patient is taking. This includes prescription medications, over-the-counter medications, vitamins, herbal supplements and any "home remedies." Also explore the possibility of unintentional poisoning in a confused older adult or when polypharmacy may be a concern.

Past Medical History

Ask about hospitalizations, surgeries, previous illnesses and significant chronic diseases that may be pertinent to the patient's current illness. Also inquire about nutritional status and immunization status.

Last Intake and Output

Establish when the patient last had something to eat or drink, either by mouth or via enteral feeding. Accurately establishing the time of the last intake is important because the risk for aspiration may be increased with some advanced treatment interventions, such as intubation or general anesthesia. Note other pertinent details about the patient's last intake, such as the amount, the patient's willingness to eat or drink, and whether or not the patient was having difficulty with eating or drinking. Finally, ask about any changes that could cause fluid imbalance, such as changes in urination or bowel elimination, vomiting, significant bleeding or fever.

Events

Gather details about the patient's activities prior to the onset of signs and symptoms. Note the time between the events leading up to the patient's illness or injury and the onset of signs and symptoms. Also note any treatments the patient may have received prior to arriving at the healthcare facility.

Focused Physical Examination

The information gathered from the rapid and primary assessments, as well as the focused history, will assist you in determining the primary area of concern and the extent of the focused physical assessment. For example, if the primary area of concern is respiratory, then the areas of focus may include the head, neck

and chest. In addition to completing a focused physical exam, a complete head-to-toe assessment should also be performed.

Diagnostic Tests

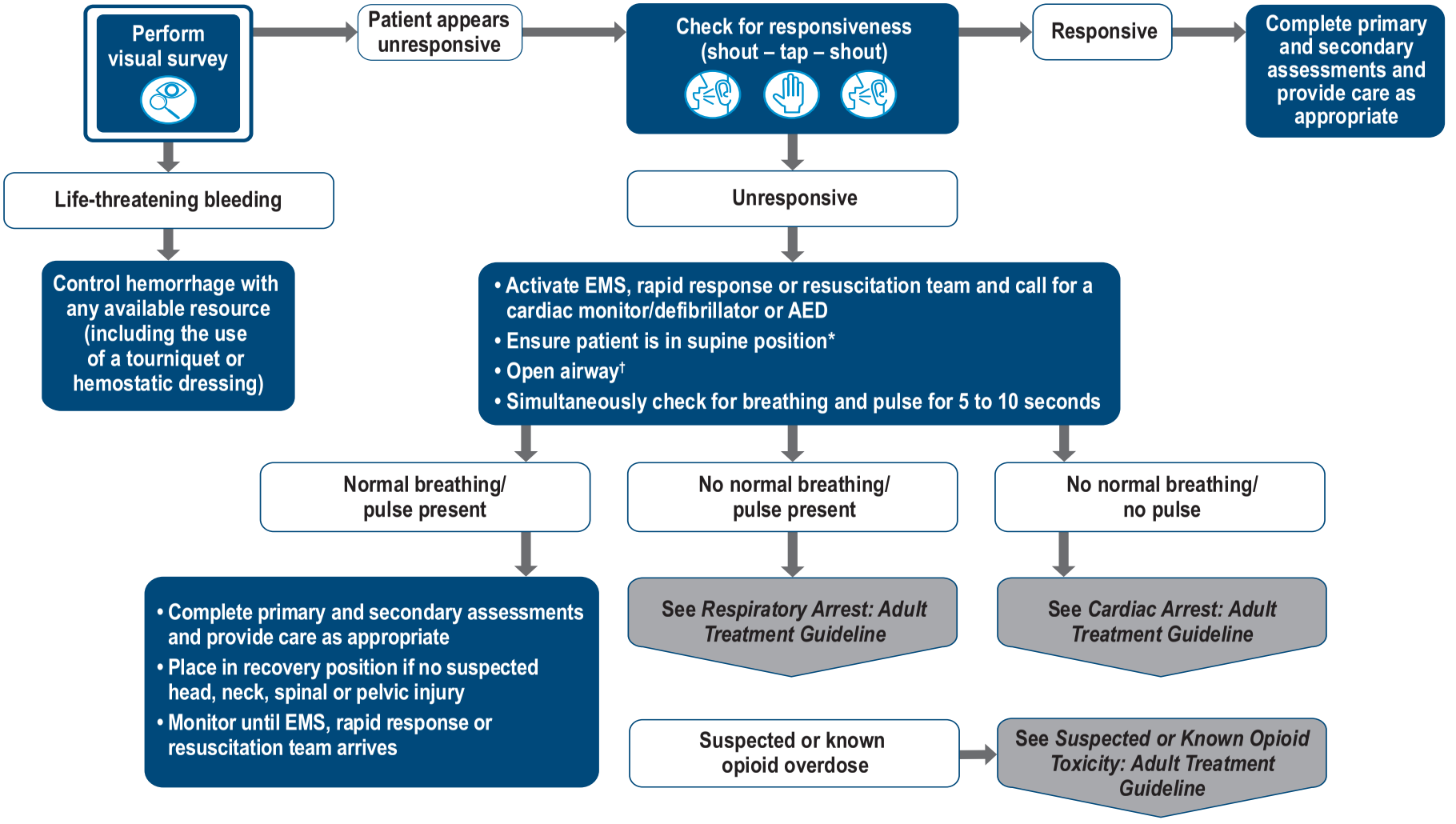
Diagnostic tests may be ordered to:

- Assist in identifying underlying causes, including potentially reversible causes.
- Narrow the list of differential diagnoses.
- Aid in determining candidacy for, or contraindications to, planned therapeutic interventions.

The following diagnostic tests are often indicated in the evaluation of patients experiencing a cardiovascular, cerebrovascular or respiratory emergency:

- **Blood tests**, including arterial blood gases (ABGs), a complete blood count (CBC), an electrolyte panel, a blood glucose level, coagulation studies, a lipid profile, serum cardiac markers and toxicology screens.
- **Imaging studies**, including chest radiography, computed tomography (CT) of the chest or brain, magnetic resonance imaging (MRI) of the brain, vascular imaging of the brain and ultrasound of the chest.
- **Electrocardiography** (12- or 15-lead).

RAPID ASSESSMENT: ADULT



*If patient is face-down, roll them on their back, taking care not to create or worsen an injury.

†Use head-tilt/chin-lift technique or modified jaw-thrust maneuver to open airway.

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PRIMARY ASSESSMENT: ADULT

Perform rapid assessment

Airway

- Assess airway patency and maintainability

Breathing

- Assess breathing rate, depth and rhythm
- Auscultate for the:
  - Presence or absence of breath sounds
  - Presence of abnormal breath sounds (e.g., wheezes, rales, rhonchi or stridor)
  - Bilateral equality
- Determine adequacy of oxygenation and ventilation
  - Capnography: $ETCO_2$ 35 to 45 mmHg*
  - Pulse oximetry: $SpO_2 \geq 94\%$

Circulation

- Assess pulse rate, quality and rhythm (carotid, radial or femoral as appropriate)
  - Establish cardiac monitoring
- Assess adequacy of perfusion
  - Assess blood pressure
  - Assess for signs of shock
  - Assess capillary refill time
  - Note skin color, appearance and temperature
- Obtain a 12- or 15-lead ECG if time and resources permit

Disability

- Assess level of consciousness (e.g., AVPU)
- Assess orientation to person, place and time
- Check the pupils for size, equality and reactivity to light
- Measure the blood glucose level
- Administer NIHSS or a similar assessment scale for suspected stroke patients

Exposure

- Check for obvious signs of injury or illness
  - Note skin color and temperature
  - Remove clothing as needed to inspect the head, ears, face and neck; the anterior and posterior trunk; and the upper and lower extremities

Initial Interventions

As needed and as resources permit, delegate these care steps to the appropriate team member.

- Ensure appropriate patient positioning
- Use head-tilt/chin-lift technique or jaw-thrust maneuver
- Clear foreign body airway obstructions
- Provide suctioning
- Consider OPA or NPA
- Consider advanced airway

- Administer supplemental oxygen
  - High-flow and/or high-concentration
- Consider assisted ventilation (e.g., BVM)
- Perform needle chest decompression or chest tube for tension pneumothorax
- Consider invasive and noninvasive ventilation (CPAP, BiPAP, ventilator)

See *Suspected or Known Opioid Toxicity: Adult Treatment Guideline* as appropriate

- Determine need for defibrillation, cardioversion and/or pacing
- Establish IV/IO for fluids/medication

See *Acute Coronary Syndromes: Adult, Bradyarrhythmia: Adult, Tachyarrhythmia: Adult* and *Post–Cardiac Arrest Care: Adult Treatment Guidelines* as appropriate

- Correct hypoglycemia if needed

See *Acute Stroke: Adult* and *Post–Cardiac Arrest Care: Adult Treatment Guidelines* as appropriate

- If suspected head, neck, spinal or pelvic injury, consider spinal motion restriction
- After examining, keep areas not being assessed covered

Refer to appropriate Advanced Life Support Treatment Guideline based on assessment findings

*Unless clinical condition warrants alternative goals.

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CHAPTER 6

Respiratory Emergencies

Introduction

A patient who is having difficulty breathing requires immediate care. Respiratory distress can quickly progress to respiratory failure, respiratory arrest and cardiac arrest.

Respiratory Anatomy and Physiology

The overall function of the respiratory system is to provide the body's cells with oxygen and to remove the byproduct of cellular metabolism, carbon dioxide. **Respiration** (the process of moving oxygen and carbon dioxide between the atmosphere and the body's cells) includes **ventilation** (the mechanical process of moving air into and out of the body) and **gas exchange** (the molecular process of adding oxygen to, and removing carbon dioxide from, the blood). Effective respiration relies on effective functioning of the respiratory system, the cardiovascular system and the nervous system.

Respiratory System Anatomy

The respiratory system includes the upper and lower airways, the lungs and the muscles of respiration, including the diaphragm and intercostal muscles (Figure 6-1).

Upper Airway

The upper airway consists of the nasal passages, pharynx, larynx and upper portion of the trachea, and ends at the level of the thoracic inlet.

The pharynx has three regions (see Figure 6-1), the:

- **Nasopharynx**, which extends from the base of the skull to the soft palate and is located posterior to the nasal cavities.
- **Oropharynx**, which extends from the hard palate to the level of the hyoid bone and is located posterior to the oral cavity.
- **Laryngopharynx (hypopharynx)**, which extends from the oropharynx to the level of the cricoid cartilage.

The larynx contains the vocal cords, is covered by the **epiglottis** (a leaf-shaped cartilaginous structure that closes over the opening of the larynx during the act of swallowing) and is supported by the thyroid and cricoid cartilages. The larynx serves as the "gatekeeper" between the upper and lower airways. The structures of the upper airway can be categorized according to where they lie in relation to the larynx (see Figure 6-1):

- **Supraglottic:** used to describe structures above the larynx (e.g., the nasal and oral cavities, the nasopharynx and oropharynx)
- **Glottic:** variably used to describe the space between the vocal cords or the structures surrounding the larynx
- **Subglottic:** used to describe structures below the larynx (i.e., the upper portion of the trachea)

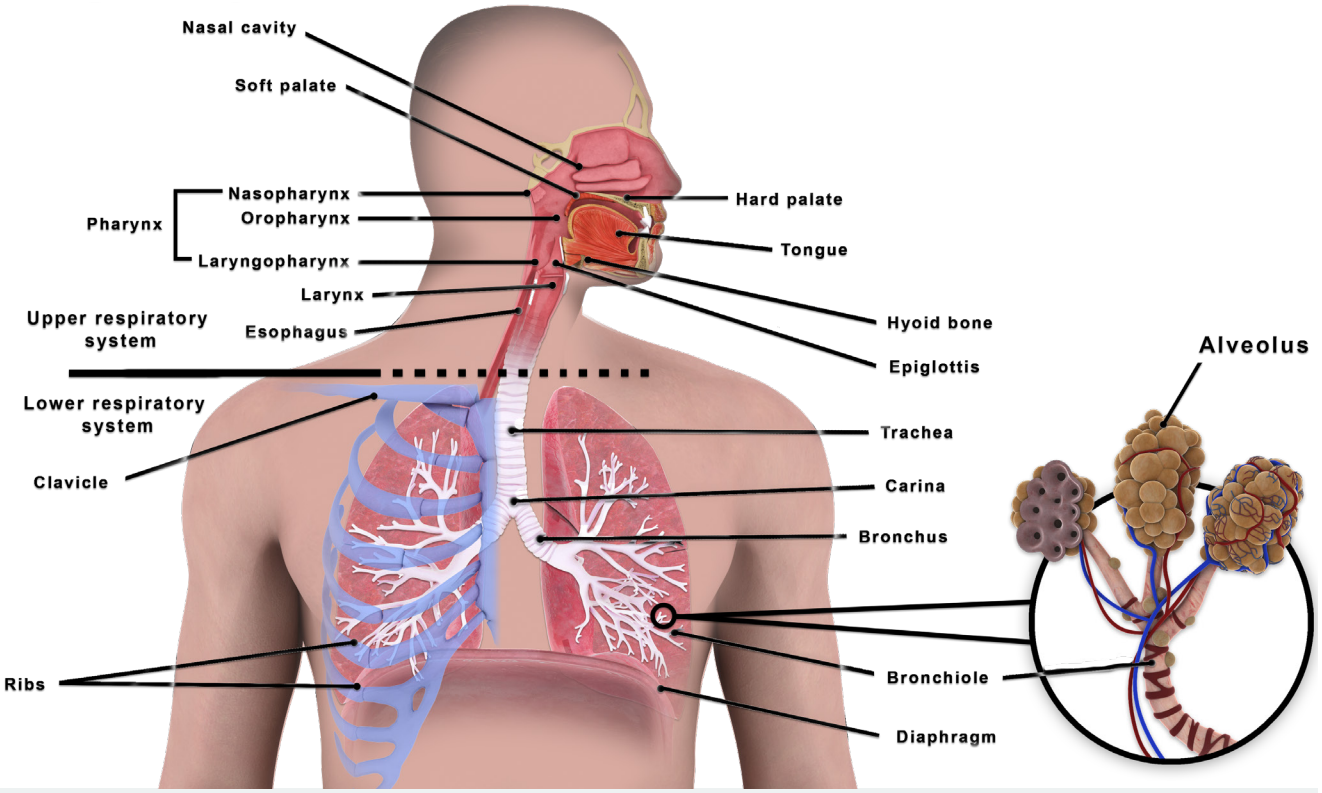


Figure 6-1 | The respiratory system

Lower Airway

The lower airway encompasses the lower trachea, bronchi, bronchioles and alveoli (see Figure 6-1). The trachea, bronchi and bronchioles serve as conduits for the air entering and leaving the lungs. The trachea, which is supported by C-shaped rings of cartilage, extends from the larynx to the **carina** (i.e., the point where the trachea bifurcates into the right and left main bronchi). The right mainstem bronchus has three intrapulmonary branches, whereas the left mainstem bronchus has two, corresponding with the number of lobes on each side. Once inside the lobes of the lungs, the bronchi divide into progressively narrower branches, called bronchioles.

Practice Note

The right mainstem bronchus is more vertically oriented relative to the trachea, whereas the left mainstem bronchus is more horizontally oriented. Additionally, the left mainstem bronchus is short and narrow, and the right mainstem bronchus is comparatively longer and wider. These anatomical features make the right mainstem bronchus easier to access and increase the risk for its accidental intubation.

Alveoli, located at the very end of the smallest bronchioles, are arranged into clusters and surrounded by a rich network of capillaries. The alveoli provide a thin surface for gas exchange between the lungs and the blood (Figure 6-2). The walls of the alveoli contain elastin fibers that allow them to stretch and then return to their normal shape. The inside of the alveolar membrane is coated with surfactant, which allows reinflation and prevents **atelectasis** (alveolar collapse).

Respiratory Physiology

Respiration involves the processes of ventilation and gas exchange.

Ventilation

The diaphragm is the primary muscle responsible for ventilation. On inspiration, the diaphragm contracts downward, increasing space for lung expansion. The external intercostal muscles, located between the ribs, synergistically act with the diaphragm during inspiration to expand the rib cage. When ventilation demands increase, the body recruits accessory muscles for assistance. The sternocleidomastoid, scalene and upper trapezius muscles are the body's accessory muscles of inspiration.

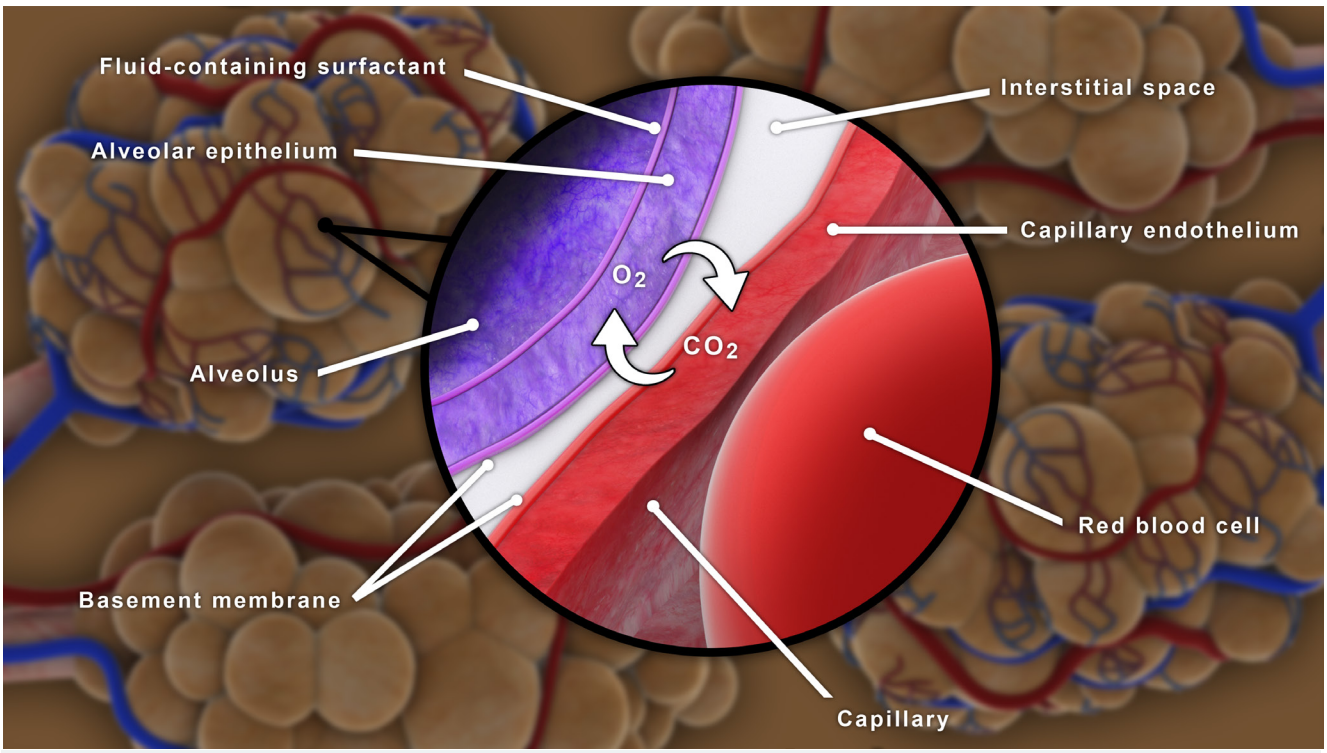


Figure 6-2 | Gas exchange occurs between the air in the alveolus and the red blood cells in the pulmonary capillary.

Expiration is a passive action that occurs when the diaphragm and external intercostal muscles relax. During active (forced) expiration, the internal intercostal muscles, the rectus abdominis and the external and internal oblique muscles are recruited as accessory muscles of ventilation.

The impulse to breathe is controlled by respiratory centers in the brain stem that regulate nerve impulses to the diaphragm and intercostal muscles. The respiratory centers receive input from chemoreceptors located throughout the body. These chemoreceptors detect changes in arterial oxygen and carbon dioxide content and in arterial pH, all of which affect the rate and depth of breathing. Other physiologic parameters that affect ventilation include core body temperature, muscle activity (e.g., during exercise) and activity of the sympathetic nervous system, which increases during times of stress.

Gas Exchange

The **oxygen-hemoglobin dissociation curve**, shown in Figure 6-3, depicts the relationship between the partial pressure of oxygen (PaO_2) and the arterial oxygen saturation (SaO_2). As more oxygen molecules bind with the hemoglobin molecule, hemoglobin's affinity for oxygen increases until the hemoglobin molecule reaches its maximum oxygen-carrying capacity. Many factors can affect hemoglobin's affinity for binding with oxygen and the strength of the bond. These factors can cause the curve to shift to the right or to the left.

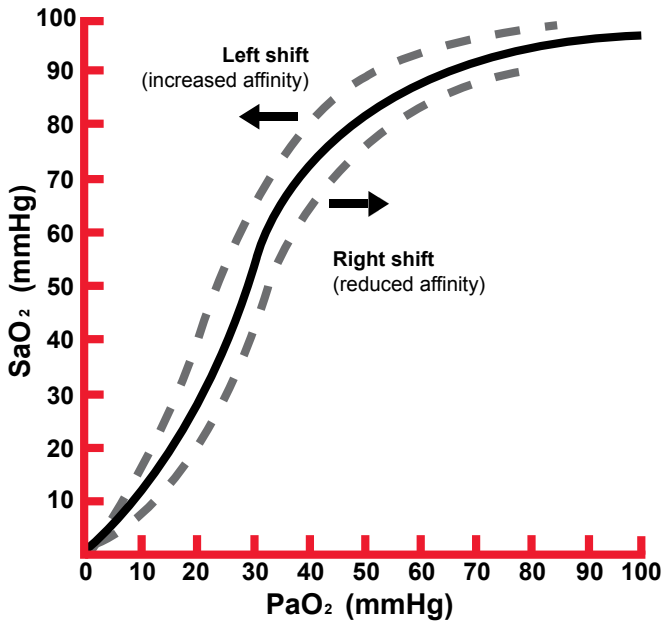


Figure 6-3 | The oxygen-hemoglobin dissociation curve

- **Right shift.** When hemoglobin's affinity for oxygen is decreased, it is harder for oxygen to bind with the hemoglobin, but it is easier for the hemoglobin to offload the oxygen that it is carrying. This causes the curve to shift to the right. **Hypoventilation**, or producing more carbon dioxide than can be exhaled, leads to acidosis and a right shift of the oxygen-hemoglobin dissociation curve. As a result, it is more difficult for oxygen to bind to hemoglobin and to be carried to the cells, and the patient becomes hypoxic.
- **Left shift.** When hemoglobin's affinity for oxygen is increased, oxygen binds to the hemoglobin easily, but offloading is difficult. **Hyperventilation**, or exhaling carbon dioxide at a faster rate than the body can produce it, leads to alkalosis and a left shift of the oxygen-hemoglobin dissociation curve. As a result, it is more difficult for oxygen to be released at the cellular level, and the patient becomes hypoxic.

Practice Note

Measuring exhaled carbon dioxide levels provides an objective measurement of the adequacy of oxygenation. Because oxygenation depends on hemoglobin's ability to load and offload oxygen, carbon dioxide output must be normal to achieve adequate oxygenation.

Gas exchange relies on pulmonary circulation. The main function of the pulmonary circulation is to bring deoxygenated blood to the lungs, perfuse the alveolar-capillary membrane and deliver oxygenated blood to the left side of the heart for systemic circulation (Figure 6-4).

Pulmonary circulation begins with the main pulmonary artery, which receives oxygen-depleted blood from the right ventricle. The main pulmonary artery divides into the right and left pulmonary arteries. Once in the lungs, the pulmonary arteries branch into progressively smaller arteries and arterioles to carry the oxygen-depleted blood to the capillary beds surrounding the alveoli. Because the concentration of oxygen is greater in the alveoli (as compared with the concentration of oxygen in the blood in the capillaries), oxygen diffuses across the alveolar-capillary membrane into the blood. Similarly, because the concentration of carbon dioxide is greater in the blood, carbon dioxide diffuses across the alveolar-capillary membrane into the alveoli, to be removed from the body on exhalation.

The oxygenated blood is carried through a network of pulmonary venules and veins until it reaches the right and left pulmonary veins, which empty into the left atrium. From the left atrium, the oxygenated blood is pumped into the left ventricle and out to the rest of the body.

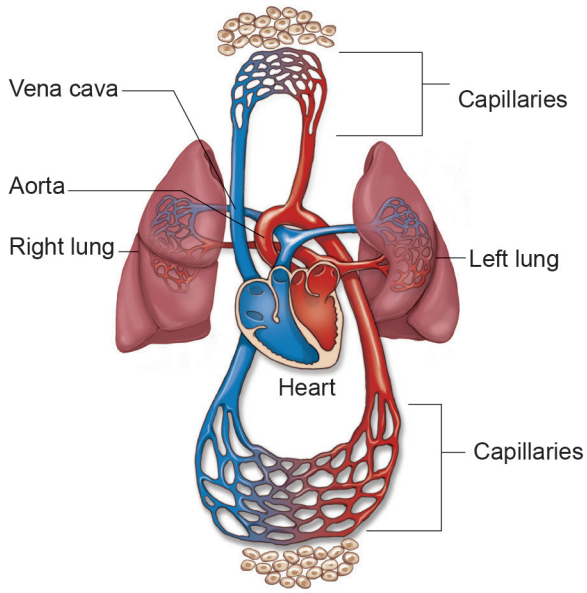


Figure 6-4 | The pulmonary circulation transports deoxygenated blood to the lungs and oxygenated blood back to the heart for distribution to the rest of the body.

Pathophysiologic Mechanisms Contributing to Respiratory Emergencies

Respiratory emergencies arise due to problems with ventilation, diffusion (gas exchange) or perfusion. Problems with these processes may have many different sources. Remember, for effective respiration, the following must be present:

- Intact neuromuscular control of breathing
- A patent airway
- An intact alveolar-capillary membrane
- Oxygen-rich blood
- Functioning pulmonary and systemic circulatory systems
- Adequate blood pressure to promote diffusion and perfusion

Inadequate Ventilation

Problems with ventilation occur in the presence of conditions that affect the body's ability to move air in and out of the lungs. Conditions that can lead to impaired ventilation include:

- **Airway obstruction.** Obstruction of the upper airway can result from a foreign body, fluids or secretions. Lower airway obstruction may result from pathologic processes involving the intrathoracic airways (i.e., the distal trachea, bronchi and bronchioles). Lower airway obstruction may be due to inflammation, constriction or mucus plugging of the airways themselves. It may also be due to external compression, for example, by interstitial fluid in pulmonary edema. Obstruction at the level of the upper airway makes it difficult for air to get in, potentially impeding alveolar ventilation. Obstruction of the lower airway makes it difficult for air to get out, potentially leading to a greater volume of air, or "air trapping," in the lungs at the end of expiration. Air trapping ultimately increases the inspiratory work of breathing.
- **Neuromuscular conditions.** Conditions such as amyotrophic lateral sclerosis (ALS), myasthenia gravis, Guillain-Barré syndrome and muscular dystrophy impair neural signaling to the respiratory muscles, and over time, respiratory weakness and fatigue develop, impairing ventilation.
- **Central nervous system conditions.** Overdose of certain substances (such as opioids and alcohol) can suppress the respiratory drive, leading to impaired ventilation. Similarly, certain medications may suppress the respiratory drive.
- **Chronic lung disease.** In restrictive lung disease, such as pulmonary fibrosis and interstitial lung disease, **lung compliance** (i.e., the ability of the lung to stretch and expand) is reduced. In obstructive lung disease, such as chronic obstructive pulmonary disease (COPD), obstruction of the airways leads to increased airway resistance. Both types of lung disease are associated with impaired ventilation and increased work of breathing.
- **Trauma.** Blunt or penetrating trauma to the chest wall and traumatic brain injury (TBI) can lead to impaired ventilation.

Inadequate Diffusion and Perfusion

When diffusion is impaired, the exchange of oxygen and carbon dioxide between the alveoli and the pulmonary capillaries is affected. Diffusion defects may result from obstruction of the smaller airways in the lungs or from conditions that create an increased barrier to diffusion (e.g., interstitial lung disease). Diffusion defects may also occur when alveoli are filled with fluid or collapsed (e.g., due to pneumonia, pulmonary edema or COPD). **Ventilation-perfusion mismatch** refers to an imbalance between the air that reaches the alveoli (i.e., ventilation) and blood flow to the alveoli (i.e., perfusion) in a portion of the lung (Figure 6-5). Cardiac disorders, pulmonary embolism and tension pneumothorax can affect blood flow to the pulmonary capillary beds, resulting in "dead space ventilation" (i.e., ventilation without perfusion). Disorders that result in pulmonary edema or atelectasis,

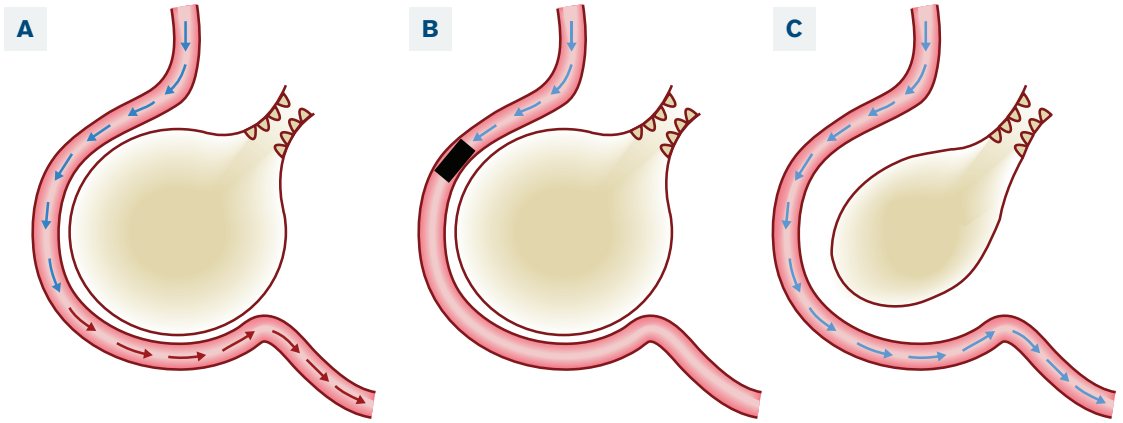


Figure 6-5 | Ventilation-perfusion mismatch refers to an imbalance between the air that reaches the alveoli and blood flow to the alveoli in a portion of the lung. (A) In the normal state, ventilation matches perfusion. (B) When blood flow to the alveolus is blocked, ventilation is adequate, but perfusion is not. (C) Shunting occurs when blood flow to the alveolus is adequate, but ventilation is not.

such as pneumonia, can cause perfusion to exceed ventilation. The result is a physiologic shunt, in which poorly oxygenated blood coming back from these areas mixes with fully oxygenated blood returning from the rest of the lungs, ultimately resulting in the delivery of desaturated blood to the body.

Respiratory Distress, Respiratory Failure and Respiratory Arrest

Respiratory compromise manifests along a continuum (Figure 6-6).

Respiratory Distress

Respiratory distress represents the earliest stage on the continuum. A patient in respiratory distress is using compensatory mechanisms to maintain adequate oxygenation and ventilation. The patient's work of breathing is increased, but physiologically, oxygenation and ventilation are adequate to meet metabolic demands. However, if the patient's respiratory distress is not relieved, these compensatory mechanisms will soon become inadequate, at which point the patient may develop respiratory failure.

Signs and symptoms of respiratory distress may include:

- Dyspnea.
- Speech dyspnea (i.e., the need to pause between words to take a breath), speaking in short one- or two-word sentences, or an inability to speak.
- Changes in breathing rate or depth.
- Tachycardia or bradycardia.
- Decreasing SaO_2 levels (however, SaO_2 levels may be unaffected in some patients).
- End-tidal carbon dioxide ($ETCO_2$) levels that are initially low (less than 35 mmHg) but with increasing

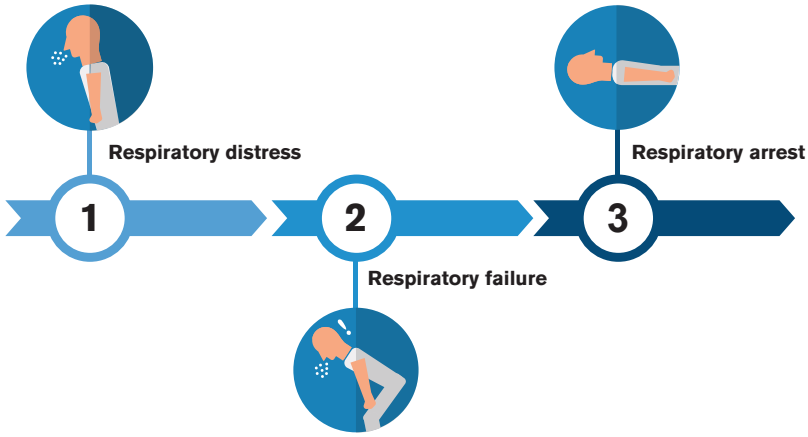


Figure 6-6 | Respiratory compromise occurs along a continuum.

distress move into the normal range (35 to 45 mmHg) and then become elevated (greater than 45 mmHg).
- Decreased, absent or abnormal breath sounds (e.g., wheezes, crackles, rhonchi).
- Use of accessory muscles to assist in breathing, evidenced by supraclavicular, suprasternal, intercostal or substernal retractions.
- Tripod positioning (leaning forward with the hands supported on the thighs or other surface).
- Diaphoresis (the skin is often cool and clammy).
- Irritability, restlessness or anxiety.
- Changes in level of consciousness.
- Cyanosis.

Practice Note

Capnography can provide an objective assessment of the severity of the patient's respiratory distress. Early on, the patient will tend to hyperventilate, which leads to hypocapnia and is reflected by a low $ETCO_2$ value (i.e., less than 35 mmHg). As the patient's respiratory distress increases and the patient begins to tire, the $ETCO_2$ value may return to the normal range (35 to 45 mmHg). But with the onset of respiratory failure, the $ETCO_2$ level will increase to greater than 45 mmHg, indicating hypoventilation.

Respiratory Failure

Respiratory failure occurs when the respiratory system can no longer meet metabolic demands. Respiratory failure usually represents a progression from respiratory distress, but patients may also initially present in respiratory failure. Respiratory failure must be addressed quickly to prevent respiratory arrest.

There are two types of respiratory failure: hypoxic respiratory failure (characterized by a PaO_2 less than 60 mmHg) and hypercapnic respiratory failure (characterized by a $PaCO_2$ greater than 50 mmHg). Patients may also have a combined form. Hypoxic failure is most often associated with ventilation-perfusion mismatch, whereas hypercapnic failure is most often associated with decreased tidal volume or increased dead space. Signs of respiratory failure could include:

- Changes in level of consciousness.
- Cyanosis.
- SaO_2 values less than 90%.
- $ETCO_2$ values greater than 50 mmHg.
- Tachycardia.
- A decreased or irregular respiratory rate.

Practice Note

An SaO_2 of less than 90% (PaO_2 of less than 50 mmHg) accompanied by $ETCO_2$ values greater than 50 mmHg is indicative of respiratory failure.

Respiratory Arrest

Respiratory arrest is complete cessation of the breathing effort. The body can tolerate respiratory arrest for only a very short time before the heart stops functioning as well, leading to cardiac arrest. Signs of respiratory arrest include:

- Loss of consciousness.
- Absent breath sounds.
- A lack of chest movement.
- Cyanosis or pallor.
- Tachycardia (which can quickly progress to bradycardia).
- Hypotension.

Capnography in Respiratory Emergencies

Quantitative capnometry is the measurement of $ETCO_2$ expressed as a value, and **quantitative capnography** is the measurement of $ETCO_2$ expressed as a value and as a waveform. Capnography is useful for assessing the severity of the patient's clinical condition, discerning the underlying pathophysiology and evaluating the patient's response to interventions.

Normal Waveform

The capnography waveform is a graphical representation of the movement of carbon dioxide through the respiratory system. The normal waveform has four phases (Figure 6-7).

- **Phase I (A–B):** This is the **respiratory baseline**, representing the beginning of exhalation. During this phase, "dead space" air is exhaled from the body. This is the air in the airways from the bronchioles to the nasal cavity that does not contain carbon dioxide.
- **Phase II (B–C):** This is the **respiratory upstroke**, representing air from the alveoli that contains carbon dioxide being exhaled from the body. For most patients, the respiratory upstroke should be nearly vertical.

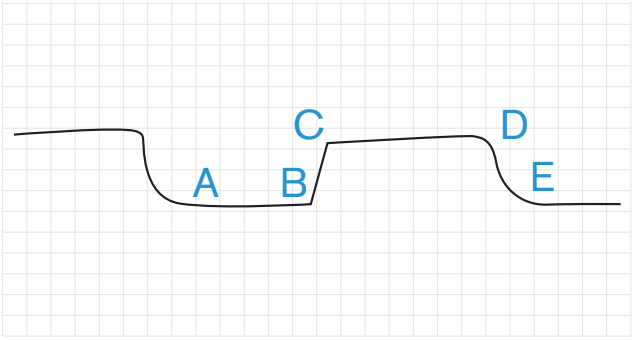


Figure 6-7 | Phases of the normal capnography waveform. **A–B:** respiratory baseline; **B–C:** respiratory upstroke; **C–D:** expiratory plateau; **D–E:** inspiratory downslope.

- **Phase III (C–D):** This is the **expiratory plateau**. During this phase, the last of the carbon dioxide–laden air from the most distal alveoli is exhaled from the body. The $ETCO_2$ value is measured at the end of exhalation (point D), which represents the peak level.
- **Phase IV (D–E):** This is the **inspiratory downslope**. During this phase, inhalation occurs and the carbon dioxide is rapidly purged from the airways and alveoli.

A normal capnogram is square with a flat respiratory baseline, a flat expiratory plateau and an $ETCO_2$ value between 35 and 45 mmHg. The square waveform indicates that carbon dioxide flow is not obstructed. The flat respiratory baseline means that the patient is not rebreathing carbon dioxide. The flat expiratory plateau means that the patient is exhaling carbon dioxide to the peak level.

Practice Note

In inflammatory conditions, the waveform may still be square, despite narrowing of the airway, because the alveoli still empty at the same rate. However, in conditions that cause bronchospasm, alveolar emptying is uneven, producing abnormal respiratory upstroke and expiratory plateau morphology.

Waveform Interpretation

To evaluate a capnography waveform, take a five-step approach:

1. **Look at the waveform.** Is there a waveform? Even an abnormal waveform is an indication that carbon dioxide is present.
2. **Look at the respiratory baseline.** Is the respiratory baseline flat and consistent from breath to breath? A respiratory baseline that slopes upward and increases with each breath suggests that the patient is rebreathing carbon dioxide (Figure 6-8A).
3. **Look at the respiratory upstroke.** Is the respiratory upstroke nearly vertical? A sloping, prolonged respiratory upstroke that is not vertical represents uneven alveolar emptying as a result of bronchospasm (see Figure 6-8B).
4. **Look at the expiratory plateau.** Is the expiratory plateau flat? Loss of plateau (see Figure 6-8C) is produced by uneven alveolar emptying secondary to severe bronchospasm that leads to air trapping. An absent plateau suggests **dynamic hyperinflation**, also called auto–positive end-expiratory pressure (auto–PEEP). Auto–PEEP occurs when exhalation time is insufficient and the lungs do not completely empty before the next breath, preventing the respiratory system from returning to its resting end-expiratory equilibrium volume between breath cycles. This is a serious condition because the patient's $ETCO_2$ level is much higher than the value recorded by capnography.
5. **Read the $ETCO_2$ value.** Finally, evaluate the $ETCO_2$ measurement. An $ETCO_2$ measurement greater than 45 mmHg suggests hypercapnia,

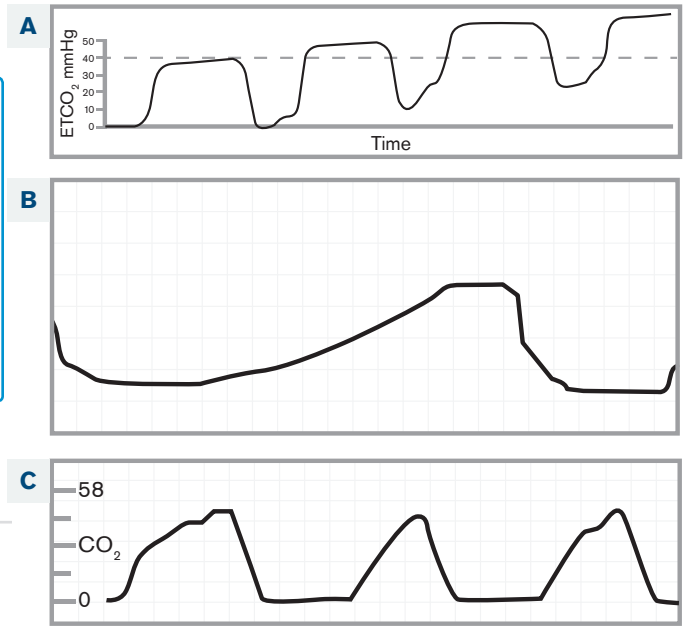


Figure 6-8 | Abnormal capnography waveform morphology. (A) Upward sloping respiratory baselines indicate that the patient is rebreathing carbon dioxide. (B) A sloping, prolonged respiratory upstroke indicates uneven alveolar emptying. (C) A loss of expiratory plateau indicates bronchospasm and air trapping.

which may be caused by respiratory failure. An $ETCO_2$ measurement less than 35 mmHg suggests hypocapnia, which may be caused by hyperventilation or hypoperfusion.

Practice Note

To interpret a waveform accurately, print it out in real time on paper. The waveform displayed on the monitor is compressed and cannot be used for diagnostic purposes, other than for noting the presence of a square waveform or a flat line (apnea).

Patient Assessment

Data gathered during the rapid, primary and secondary assessments can help you to determine where the patient is on the continuum of respiratory compromise and may offer clues as to the underlying cause.

Rapid Assessment

Signs of respiratory compromise that you may observe during the rapid assessment include retractions and the use of accessory muscles to breathe; tripod positioning; an inability to speak in complete sentences (or at all); pale, ashen or cyanotic skin; diaphoresis; and restlessness, agitation or an altered level of consciousness.

A patient in respiratory arrest will be unresponsive. The pulse and breathing check will reveal a pulse, but no breathing (or only gasping).

Primary Assessment

Conduct a primary assessment following the ABCDE approach and provide initial interventions as needed:

- Ensure a patent airway.
- Assist with ventilation as necessary. Patients who cannot ventilate adequately despite an open airway or who have insufficient respiratory effort require assisted ventilation, which is initially provided using a bag-valve-mask (BVM) resuscitator.
- Establish pulse oximetry and provide the minimal level of supplemental oxygen needed to maintain an oxygen saturation of at least 94% (88% to 92% in patients who rely on hypoxic drive).
- Establish capnography and adjust ventilations as needed to maintain $ETCO_2$ values in the range of 35 to 45 mmHg.
- Establish vascular access.

Practice Note

Some patients with end-stage COPD may have chronically elevated arterial carbon dioxide levels and chronically low arterial oxygen levels. In these patients, the stimulus to breathe becomes dependent on the peripheral chemoreceptors, which respond to arterial oxygen levels rather than arterial carbon dioxide levels. To avoid a reduction in the respiratory rate and depth and subsequent hypercapnia in patients who rely on hypoxic drive, titrate supplemental oxygen administration to achieve an arterial oxygen saturation of 88% to 92%.

Secondary Assessment

The goals of the secondary assessment are to discern the underlying cause of the respiratory compromise and to evaluate the severity of the patient's condition. Although there are many potential differential diagnoses for respiratory compromise, the acute onset of respiratory distress is frequently pulmonary or cardiac in origin (Box 6-1).

History

Conduct a focused history. If the patient is having difficulty speaking or has an altered level of consciousness, you may need to obtain the history from other healthcare providers, family members or bystanders. Ask about the patient's medical history and whether they have experienced similar episodes in the past. Information about the onset of the respiratory distress (was it acute, or did it develop over time?), associated signs and symptoms (such as chest pain, nausea or fever) and medications can also provide valuable insight into the underlying cause.

Physical Examination

Evaluate the patient's general appearance and vital signs and conduct a focused examination of the chest, including auscultation of the lungs and heart.

Diagnostic Tests

Diagnostic tests that may be ordered in the initial evaluation of a patient with respiratory compromise include blood gases (arterial or venous); serum cardiac markers; a basic metabolic panel; a toxicology screen; chest radiography, chest computed tomography (CT), or both; a 12-lead ECG; and bedside echocardiography or ultrasonography.

Box 6-1 | Pulmonary and Cardiac Differential Diagnoses for Acute-Onset Respiratory Distress

Pulmonary

- Pulmonary embolism
- Chronic obstructive pulmonary disease (COPD) exacerbation
- Asthma exacerbation
- Pneumonia
- Pneumothorax
- Noncardiogenic pulmonary edema/acute respiratory distress syndrome (ARDS)

Cardiac

- Cardiogenic pulmonary edema/congestive heart failure (CHF)
- Acute coronary syndromes (ACS)
- Cardiac tamponade
- Acute valvular insufficiency

Approach to the Patient

For a patient with respiratory compromise, interventions depend on the underlying cause. Common interventions include supplemental oxygen, medications specific to the underlying cause (e.g., bronchodilators, diuretics) and assisted ventilation (noninvasive or invasive).

A continuous positive airway pressure (CPAP) device "splints" the alveoli open and allows for better gas exchange, which improves oxygenation and may reduce the work of breathing. A bilevel positive airway pressure (BiPAP) device provides two levels of pressure—one for the inspiratory positive airway pressure (IPAP) and one for the expiratory positive airway pressure (EPAP), as well as CPAP when the IPAP and EPAP are equal. This method of noninvasive ventilation provides support to ventilation and stenting of the airways and is preferred for patients with ventilatory failure in addition to oxygenation issues.

After any intervention, reassess the patient to determine response and adjust the treatment plan as necessary. Monitor the patient for signs of worsening respiratory function, such as increased work of breathing, an increase or decrease in respiratory rate, hypoxemia or mental status changes. Pulse oximetry and capnography provide ongoing data about the patient's respiratory function and should be monitored closely.

Respiratory Arrest

The *Respiratory Arrest: Adult* Treatment Guideline summarizes the approach to a patient in respiratory arrest.

Assess and Recognize

A patient in respiratory arrest is unresponsive and not breathing normally (or only gasping) but has a pulse.

Care

Ensure a patent airway and deliver 1 ventilation every 5 to 6 seconds. Each ventilation should last about 1 second and make the chest begin to rise. Provide high-flow supplemental oxygen, titrating to achieve and maintain an oxygen saturation of 94% to 99%. Adjust ventilations as needed to maintain $ETCO_2$ values in the range of 35 to 45 mmHg. While supporting ventilations, consider and address potential underlying causes. About every 2 minutes, check for breathing and a pulse. If there is a pulse but no normal breathing, continue ventilations. If there is no pulse, begin CPR immediately.

Opioid Overdose

Opioid overdose suppresses the central respiratory drive and can quickly induce respiratory arrest or cardiac arrest. The *Suspected or Known Opioid Toxicity: Adult* Treatment Guideline summarizes the approach to a patient with known or suspected opioid toxicity.

Assess and Recognize

Respiratory depression is the hallmark of opioid overdose, especially when accompanied by unresponsiveness and miosis.

Care

For a patient in respiratory arrest as a result of opioid toxicity, ensure a patent airway and assist ventilation at a rate of 1 ventilation every 5 to 6 seconds. Administer naloxone as soon as it is available. Naloxone can completely reverse the effects of opioid toxicity if administered in time during respiratory arrest. Naloxone may be administered via the intravenous (IV), intraosseous (IO), intramuscular (IM) or intranasal (IN) route. The initial dose of naloxone is 0.4 mg IV/IO/IM or 2 mg IN. The dose may be repeated after 4 minutes. Reassess the patient every 4 minutes and administer additional doses of naloxone as needed.

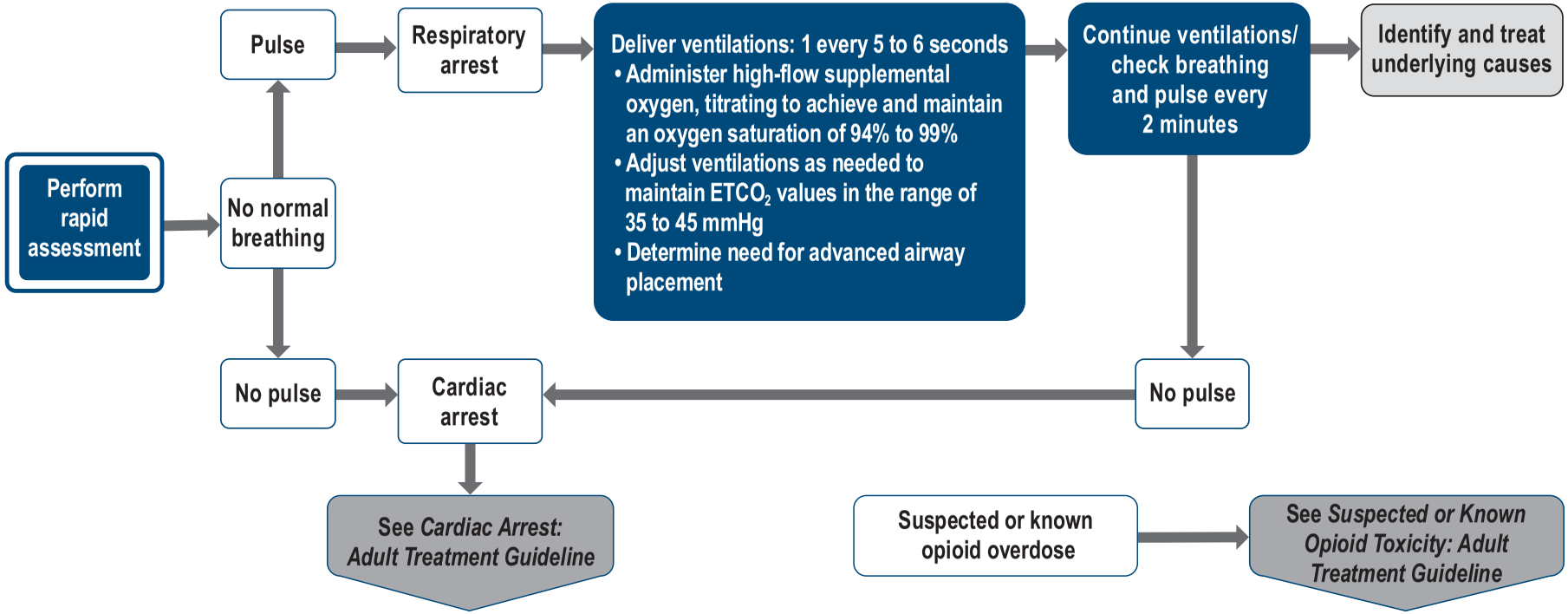
Practice Note

If the maximum dose of naloxone (15 mg) does not reverse the respiratory depression, it is unlikely that opioid overdose is the underlying cause.

Continue treatment until respiration is adequate, as evidenced by a normal respiratory rate, a PaO_2 greater than 90% and a $PaCO_2$ less than 45 mmHg. Monitor the patient for at least 4 to 6 hours after the last dose of naloxone is given. Longer observation times may be indicated if the cause of the overdose was an extended-release or long-acting opioid. Because respiratory depression may recur with an extended-release or long-acting opioid, consider admission to the critical care unit and initiation of a continuous naloxone infusion at two-thirds of the effective dose per hour, titrated to patient response.

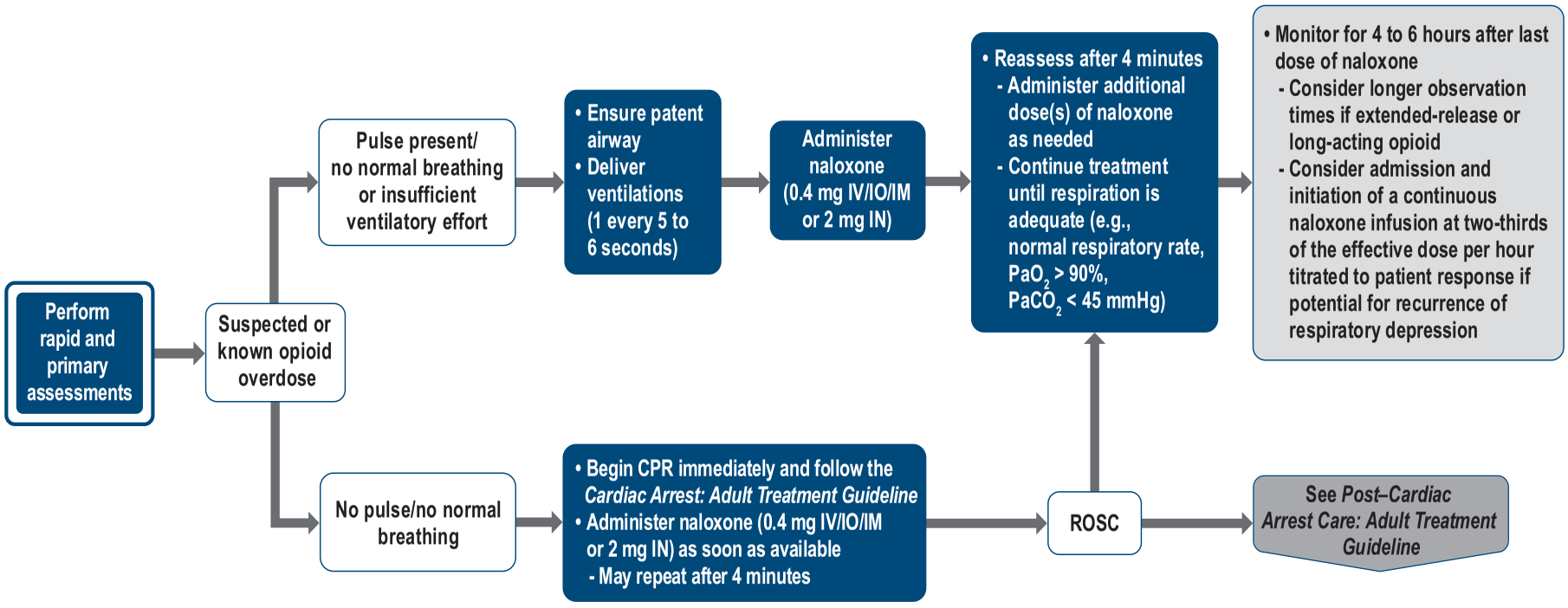
Although no evidence supports any benefit to naloxone administration during cardiac arrest, administration of naloxone during cardiac arrest is recommended when opioid overdose is suspected. For a patient in cardiac arrest, high-quality CPR is prioritized over the administration of naloxone.

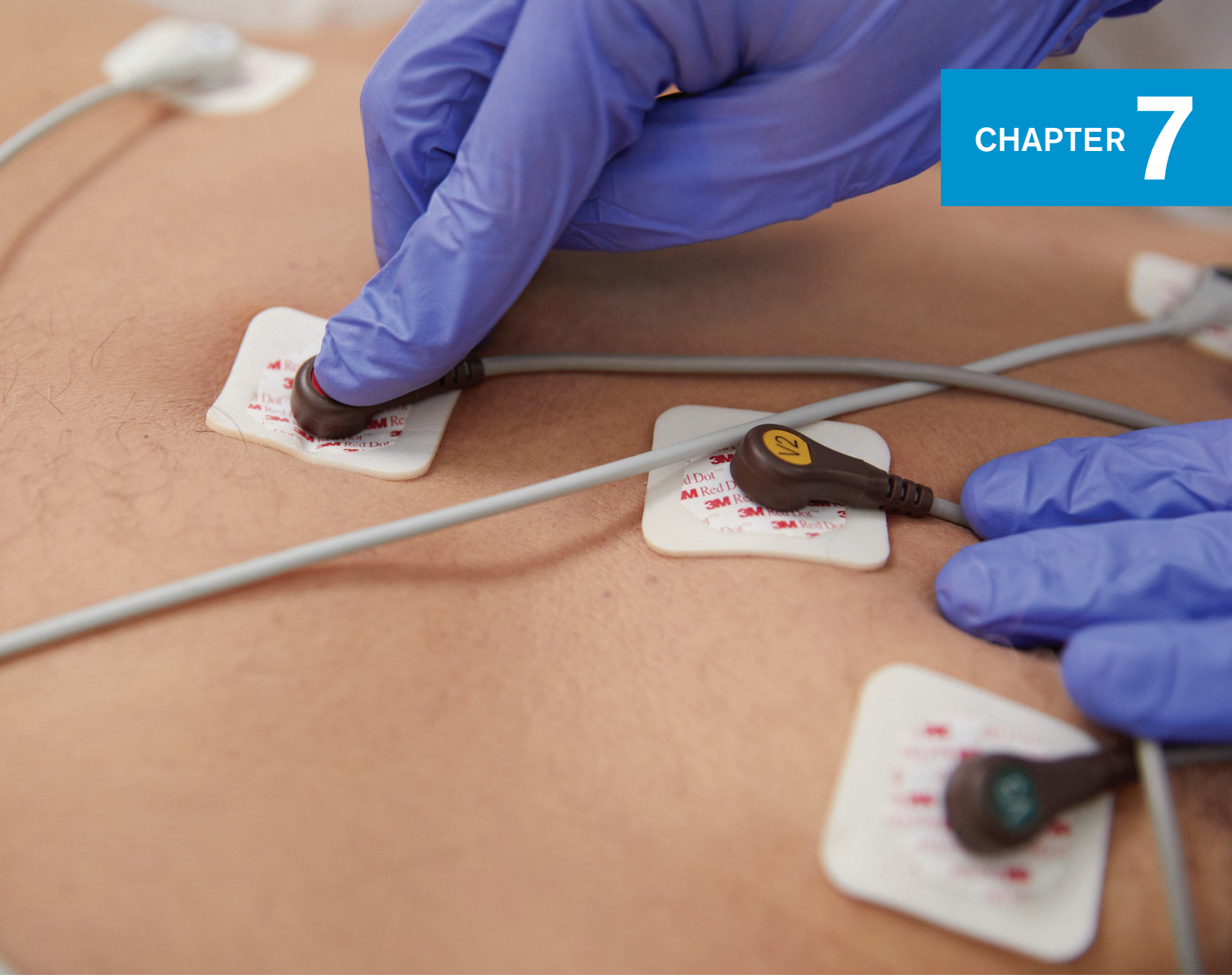
RESPIRATORY ARREST: ADULT



Ventilation Technique
To minimize complications when using a BVM resuscitator, depress the bag slowly (over 1 second) and only about halfway to deliver the minimal tidal volume (400 to 700 mL for an adult) and make the chest begin to rise.
Avoid overinflation and hyperventilation.
Increasing difficulty when providing ventilations using a BVM resuscitator may indicate an increase in intrathoracic pressure, inadequate airway opening or other complications.

SUSPECTED OR KNOWN OPIOID TOXICITY: ADULT





CHAPTER 7

Arrhythmias

Introduction

A cardiac arrhythmia is a deviation from the normal heart rate, electrical activity pattern or rhythm that varies from normal sinus rhythm. Although not every arrhythmia is dangerous to the patient, many can be serious, and some require immediate treatment to prevent sudden death. For this reason, ALS providers must be able to identify abnormal rhythms accurately and interpret them within the overall clinical context.

Electrical Conduction

Cardiac Conduction System

The cardiac conduction system is a group of specialized myocardial cells that generate and transmit the electrical signals that cause the heart muscle to contract. A well-functioning conduction system is essential for ensuring the rhythmic, coordinated contraction of the heart that is necessary to maintain cardiac output.

The cardiac conduction system consists of the sinoatrial (SA) node, the atrioventricular (AV) node, the bundle of His, the left and right bundle branches and the Purkinje fibers (Figure 7-1). Normally, the SA node, located in the upper right atrium, generates the electrical impulses that initiate the rhythm and rate of the heart. The impulse generated by the SA node travels via specialized pathways across the walls of the atria, causing the atria to contract, until it reaches the AV node at the base of the right atrium. The AV node, which briefly slows the transmission of the impulse, plays an important role in coordinating and maintaining appropriate AV conduction. From the AV node, the impulse travels through the bundle of His, which descends the membranous aspect of the interventricular septum before dividing into the right and left bundle branches. The impulse continues down the bundle branches and through the Purkinje fibers, causing the ventricles to contract.

The SA node is the primary pacemaker of the heart. However, in the event of SA node dysfunction or failure, the AV node can function as a backup pacemaker. Similarly, the cells of the **atrioventricular (AV) junction** (the zone of tissue surrounding the AV node), the bundle branches and the ventricles can generate impulses to maintain some level of contraction and cardiac output.

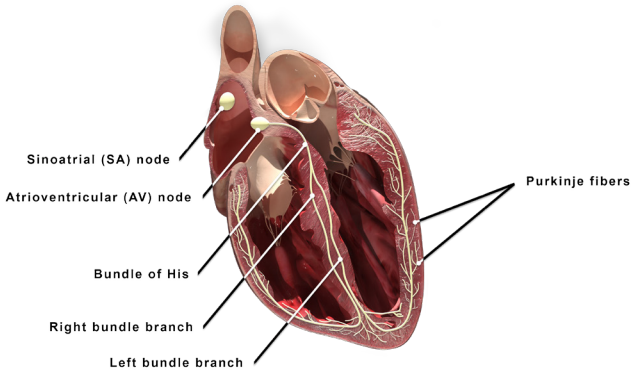


Figure 7-1 | The cardiac conduction system

Features of an ECG Tracing

An ECG tracing is a graphical representation of the electrical impulse as it travels through the conduction system, causing depolarization and then repolarization of the myocardium. The waveforms and intervals seen on an ECG tracing correspond to events in the cardiac cycle (Figure 7-2):

- **P wave**. The P wave represents depolarization of the atrial myocardial cells.
- **QRS complex**. The QRS complex represents depolarization of the ventricular myocardial cells. The normal duration of the QRS complex is less than 120 milliseconds (0.12 second).
- **T wave**. The T wave represents repolarization of the ventricular myocardial cells. (Atrial repolarization occurs during ventricular depolarization and is not seen on the ECG; it is overshadowed by the depolarization of the larger ventricles.)
- **PR interval**. The PR interval represents the time from the beginning of atrial depolarization to the beginning of ventricular depolarization. It is measured from the beginning of the P wave to the beginning of the QRS complex. The normal duration of the PR interval is 120 to 200 milliseconds (0.12 to 0.2 second).
- **QT interval**. The QT interval is measured from the beginning of the QRS complex to the end of the T wave. This encompasses the time from the beginning of ventricular depolarization to the end of ventricular repolarization. Because the QT interval varies normally with the heart rate, the **corrected QT interval (QTc)** is used to give a value that is theoretically independent of rate. The QTc adjusts for heart rate differences by dividing the QT interval by the square root of the RR interval (i.e., one cardiac cycle). In general, a QTc greater than 460 milliseconds (0.46 second) is considered to be prolonged.

Practice Note

If the heart rate is faster than 120 bpm or slower than 50 bpm, the formula for calculating the QTc is not considered valid and should not be used.

- **ST segment**. The ST segment represents the time between the end of ventricular depolarization and the beginning of ventricular repolarization. It is measured from the end of the QRS complex to the beginning of the T wave.
- **J point**. The J point is the point where the QRS complex ends and the ST segment begins.

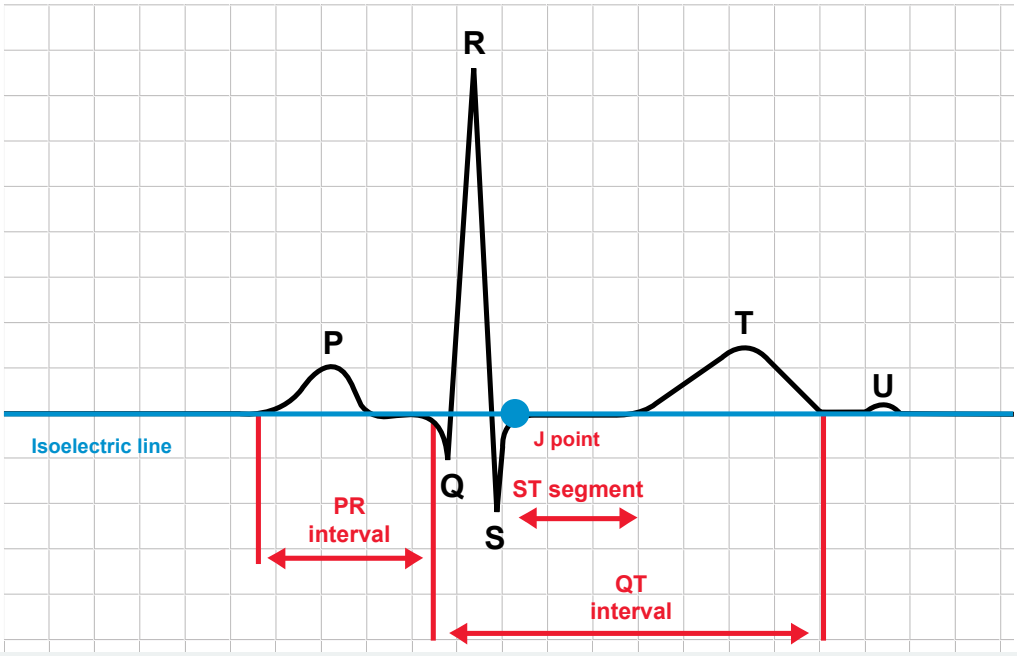


Figure 7-2 | Features of an ECG tracing

Approach to Evaluating an ECG Tracing

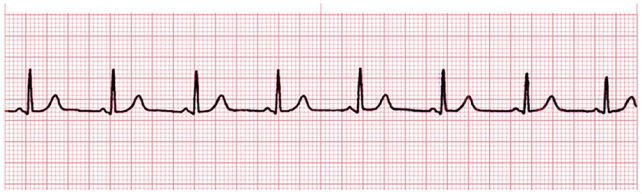
Taking a methodical approach to evaluating the rhythm strip ensures that you gather relevant details that can help you to identify the rhythm accurately.

1. **Regularity**. Look at the rhythm to see whether it is regular. Is the amount of time between each P wave the same? What about the amount of time between each QRS complex (i.e., the RR interval)?
2. **Rate**. To estimate the atrial rate, count the number of P waves over a 6-second period and multiply by 10. To estimate the ventricular rate, do the same with the QRS complexes. Alternatively, if the rhythm is regular, divide 300 by the number of large squares between two P waves (to get the atrial rate) and between two R waves (to get the ventricular rate). If the heart rate is very fast, divide 1500 by the number of small squares between two P waves (to get the atrial rate) and between two R waves (to get the ventricular rate). Are the atrial and ventricular rates the same or different? Are they within normal limits?
3. **P waves**. Look at the P wave morphology. Is it consistent? Is there one—and only one—P wave associated with each QRS complex? Note that in lead II, the P waves are usually upright but in lead V_1, the P waves may be inverted or biphasic.
4. **QRS complex**. Measure the QRS complex. Is it within the normal range? (QRS complexes that exceed 0.12 second in duration are abnormal.) Do all the QRS complexes have the same morphology?
5. **PR interval**. Measure the PR interval. Is it within the normal range (0.12 to 0.2 second)? Is it consistent throughout the tracing? If it varies, is the variation predictable?
6. **Clinical significance**. Determine the rhythm and its clinical significance. Is the patient showing signs or symptoms? Is the rhythm potentially life-threatening?

Normal Sinus Rhythm

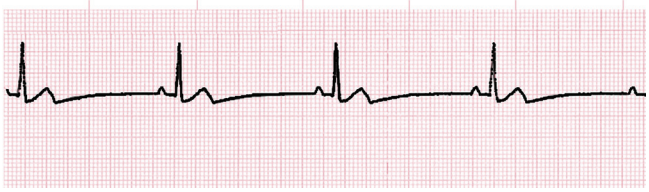
In normal sinus rhythm (Figure 7-3):

- The rhythm is regular (but may vary slightly during respirations).
- The rate ranges between 60 and 100 bpm.
- The P waves are uniform in shape, indicating that the SA node is the only pacemaker driving atrial depolarization.
- Each P wave is linked in a 1:1 fashion to each QRS complex (i.e., atrial depolarization is always linked to ventricular depolarization).



Regularity: regular
Rate: 60–100 bpm
P wave: upright and uniform; one for every QRS complex
QRS complex: < 0.12 second
PR interval: 0.12–0.20 second

Figure 7-3 | Sinus rhythm



Regularity: regular
Rate: < 60 bpm
P wave: upright and uniform; one for every QRS complex
QRS complex: < 0.12 second
PR interval: 0.12–0.20 second

Figure 7-4 | Sinus bradycardia

Recognizing Bradyarrhythmias

Bradyarrhythmias described in this chapter include sinus bradycardia, first-degree AV block, second-degree AV block (types I and II) and third-degree (complete) AV block.

Practice Note

Bradycardia is defined as a heart rate less than 60 bpm. In most cases, patients present with symptoms when the heart rate is less than 50 bpm.

Sinus Bradycardia

Sinus bradycardia is identical to normal sinus rhythm, except the rate is less than 60 bpm. Cardiac activation starts at the SA node but is slower than normal. Sinus bradycardia may be a normal finding in some patients, particularly healthy young adults and trained athletes. In other patients, however, sinus bradycardia is a pathologic finding.

Causes

Causes of sinus bradycardia include:

- Vagal stimulation.
- Myocardial infarction.
- Hypoxia.
- Medications (e.g., β-blockers, calcium channel blockers, digoxin).
- Coronary artery disease.
- Hypothyroidism.
- Iatrogenic illness.
- Inflammatory conditions.

Signs and Symptoms

Sinus bradycardia may not cause signs or symptoms. However, when sinus bradycardia significantly affects cardiac output, signs and symptoms may include:

- Dizziness or light-headedness.
- Syncope.
- Fatigue.
- Shortness of breath.
- Confusion or memory problems.

ECG Findings

On ECG, sinus bradycardia appears the same as sinus rhythm, except the heart rate is less than 60 bpm (Figure 7-4).

Atrioventricular Block

AV block is partial or complete interruption of impulse transmission from the atria to the ventricles. The block may occur at the AV node, the bundle of His, the bundle branches or the Purkinje fibers. Common causes of AV block include fibrosis and scarring of the conduction system and myocardial infarction. Other causes include medications (such as β-blockers, calcium channel blockers, digoxin and amiodarone), electrolyte abnormalities, myocardial ischemia, infectious or inflammatory disorders and congenital heart conditions. Depending on the cause, AV block can be transient or persistent. AV blocks are classified as first, second or third degree. Figure 7-5 summarizes how to differentiate AV blocks according to ECG findings.

First-Degree Atrioventricular Block

First-degree AV block is characterized by a prolonged delay in conduction at the AV node or bundle of His. The impulse is conducted normally from the sinus node through the atria, but upon reaching the AV node, it is delayed for

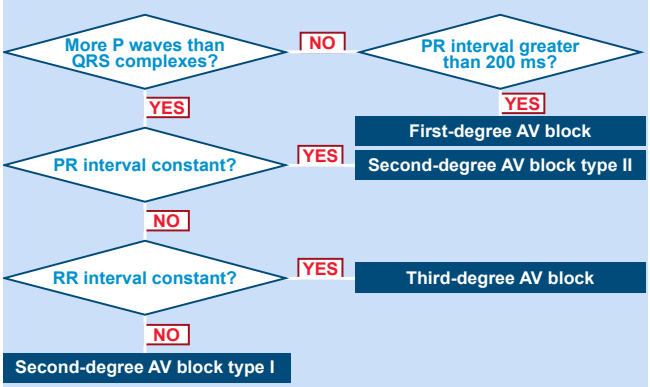


Figure 7-5 | Approach to differentiating atrioventricular (AV) blocks according to ECG findings

longer than the usual 100 milliseconds (0.1 second). In first-degree AV block, although the impulses are delayed, each atrial impulse is eventually conducted through the AV node to cause ventricular depolarization.

Causes

First-degree AV block may be a normal finding in athletes and young patients with high vagal tone. It can also be an early sign of degenerative disease of the conduction system or a transient manifestation of myocarditis or drug toxicity.

Signs and Symptoms

First-degree AV block rarely produces symptoms.

ECG Findings

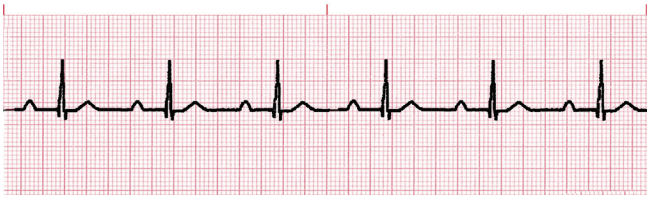
First-degree AV block (Figure 7-6) is characterized by normal P waves that are each followed by QRS complexes, but because the impulse is delayed at the AV node or bundle of His, the PR interval is longer than normal (i.e., it exceeds 0.2 second). QRS complexes of normal duration suggest that the delay is occurring at the level of the AV node, whereas wide QRS complexes suggest that the delay is **infranodal** (distal to the node).

Second-Degree Atrioventricular Block Type I

In second-degree AV block type I (also called Mobitz type I or Wenckebach block), impulses are delayed and some are not conducted through to the ventricles. After three or four successive impulse delays, the next impulse is blocked. After the blocked impulse, the AV node resets, and the pattern repeats. Second-degree AV block type I usually occurs at the AV node but may be infranodal.

Causes

Because the block usually occurs above the bundle of His, conditions or medications that affect the AV node (such as myocarditis, electrolyte abnormalities,



Regularity: regular
Rate: variable; can occur with normal rate, bradycardia or tachycardia
P wave: upright and uniform: one for every QRS complex
QRS complex: < 0.12 second
PR interval: > 0.20 second

Figure 7-6 | First-degree atrioventricular (AV) block

inferior wall myocardial infarction or medications such as digoxin) can cause second-degree AV block type I. This type of arrhythmia can also be physiologic.

Signs and Symptoms

Second-degree AV block type I rarely produces symptoms. Some patients may have signs and symptoms of bradycardia.

ECG Findings

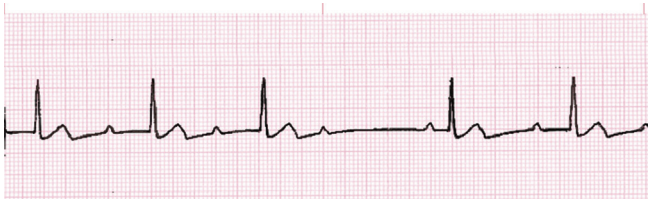
Because some impulses are not conducted through to the ventricles, the ratio of P waves to QRS complexes is greater than 1:1 (Figure 7-7). Because each impulse is delayed a little more than the last until eventually one impulse is completely blocked, the ECG shows progressive lengthening of the PR interval with each beat, then a P wave that is not followed by a QRS complex (a "dropped beat"). In most cases, the RR interval decreases before each dropped beat. After the dropped beat, impulse conduction through the AV node resumes and the sequence repeats.

Second-Degree Atrioventricular Block Type II

In second-degree AV block type II (Mobitz type II), the block occurs below the AV node, in the bundle of His. As with second-degree AV block type I, some atrial impulses are conducted through to the ventricles, and others are not. However, there are no progressive delays. The blocked impulses may be chaotic or occur in a pattern (e.g., 2:1, 3:1 or 4:1). In high-grade second-degree AV block type II, the ratio is greater than 2:1 (i.e., 3:1, 4:1, or variable).

Causes

Second-degree AV block type II is always pathologic. It is usually caused by fibrotic disease of the conduction system or anterior wall myocardial infarction.



Regularity: irregular in a pattern
Rate: variable; usually < 100 bpm
P wave: upright and uniform; more P waves than QRS complexes
QRS complex: < 0.12 second
PR interval: becomes progressively longer until a P wave is not conducted, then cycle repeats

Figure 7-7 | Second-degree atrioventricular (AV) block type I

Signs and Symptoms

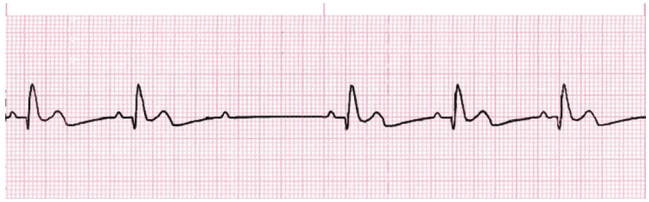
Patients may present with light-headedness or syncope, or they may be asymptomatic. The clinical presentation varies, depending on the ratio of conducted to blocked impulses.

ECG Findings

Second-degree AV block type II is characterized by a constant PR interval (Figure 7-8). Because impulses are intermittently blocked, there are more P waves than QRS complexes.

Third-Degree Atrioventricular Block

In third-degree (complete) AV block, no impulses are conducted through to the ventricles. The block can occur at the level of the AV node but is usually infranodal. Pacemaker cells in the AV junction, bundle of His or the ventricles stimulate the ventricles to contract, usually at a rate of 30 to 45 bpm. This means that the atria and ventricles are being driven by independent pacemakers and are contracting at their own intrinsic rates, a situation known as **atrioventricular (AV) dissociation**.



Regularity: regular (2:1), unless conduction ratio varies
Rate: usually < 100 bpm, tendency for bradycardia
P wave: upright and uniform; more P waves than QRS complexes (2:1, 3:1, 4:1 or variable)
QRS complex: < 0.12 second
PR interval: < 0.20 second or prolonged; constant for every QRS complex

Figure 7-8 | Second-degree atrioventricular (AV) block type II

Causes

Degenerative disease of the conduction system is the leading cause of third-degree AV block. This arrhythmia may also result from damage caused by myocardial infarction, Lyme disease or antiarrhythmic medications.

Signs and Symptoms

If ventricular contraction is stimulated by pacemaker cells above the bifurcation of the bundle of His, the ventricular rate is relatively fast (40 to 60 bpm) and reliable, and symptoms may be mild (such as fatigue, orthostatic hypotension and effort intolerance). However, if ventricular contraction is stimulated by pacemaker cells in the ventricles, the ventricular rate will be slower (20 to 40 bpm) and less reliable, and symptoms of decreased cardiac output may be more severe (such as syncope).

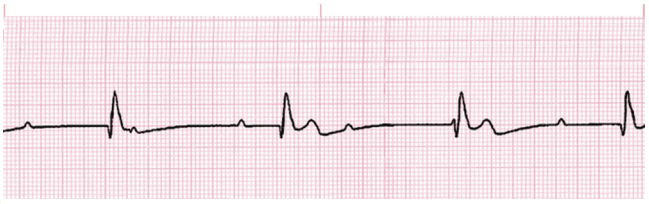
ECG Findings

In third-degree AV block, there is no electrical communication between the atria and ventricles, so there is no relationship between P waves and QRS complexes (Figure 7-9). The RR interval is usually constant. The PP interval is constant or slightly irregular. If pacemaker cells in the AV junction stimulate ventricular contraction, the QRS complexes will be narrow (less than 0.12 second in duration). Impulses that originate in the ventricles produce wide QRS complexes.

Recognizing Tachyarrhythmias

Tachyarrhythmias can be categorized as narrow complex (supraventricular) or wide complex.

- Narrow-complex (supraventricular) tachyarrhythmias include atrial flutter and atrial fibrillation. **Supraventricular tachycardia** is a general term for tachyarrhythmias that originate above the ventricles in the atria or AV node and run normally through the bundle branches, producing a normal QRS complex.
- Wide-complex tachyarrhythmias originate in the ventricles and include monomorphic and polymorphic ventricular tachycardia, torsades de pointes (a form of polymorphic ventricular tachycardia) and ventricular fibrillation. Supraventricular tachycardia with aberrant conduction can also produce a wide-complex tachyarrhythmia.



Regularity: usually regular RR interval, regular PP interval
Rate: varies depending on escape focus; junctional (40–60 bpm) and ventricular (< 40 bpm)
P wave: upright and uniform, more P waves than QRS complexes
QRS complex: < 0.12 second if junctional escape, ≥ 0.12 second if ventricular escape
PR interval: total dissociation from QRS complexes

Figure 7-9 | Third-degree atrioventricular (AV) block

Sinus Tachycardia

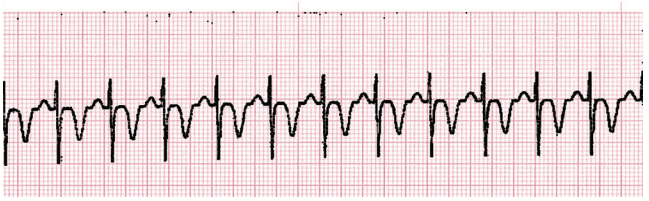
Sinus tachycardia, the most common tachyarrhythmia, is identical to normal sinus rhythm, except the rate is between 100 and 150 bpm (Figure 7-10). Sinus tachycardia is a normal physiologic response when the body is under stress (such as that caused by exercise, illness or pain). It may also be seen in patients with heart failure, lung disease, shock or hyperthyroidism.

Atrial Flutter

Atrial flutter is caused by an ectopic focus in the atria that causes the atria to contract at a rate of 250 to 350 bpm. The underlying mechanism of atrial flutter is most often a reentrant circuit that encircles the tricuspid valve annulus.

Causes

Atrial flutter is often seen in patients with heart disease (such as heart failure, rheumatic heart disease or coronary artery disease) or as a postoperative complication.



Regularity: regular
Rate: 100–150 bpm
P wave: upright and uniform; one for every QRS complex
QRS complex: < 0.12 second, regular
PR interval: 0.12–0.20 second

Figure 7-10 | Sinus tachycardia

Signs and Symptoms

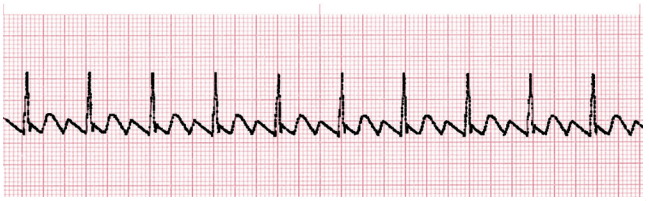
Patients may be asymptomatic or present with shortness of breath, palpitations, effort intolerance, chest constriction, weakness or syncope.

ECG Findings

In atrial flutter, atrial contraction occurs at such a rapid rate that discrete P waves separated by a flat baseline cannot be seen (Figure 7-11). Instead, the baseline continually rises and falls, producing the "flutter" waves. In leads II and III, the flutter waves may be quite prominent, creating a "sawtooth" pattern. Because of the volume of atrial impulses, the AV node allows only some of the impulses to pass through to the ventricles. In atrial flutter, a 2:1 ratio is the most common (i.e., for every two flutter waves, only one impulse passes through the AV node to generate a QRS complex). Ratios of 3:1 and 4:1 are also frequently seen.

Atrial Fibrillation

Atrial fibrillation is caused by multiple ectopic foci in the atria that cause the atria to contract at a rate of 350 to 600 bpm. Rarely, the atrial rate may be as high as 700 bpm. The AV node only allows some of the impulses to pass through to the ventricles, generating an irregularly irregular rhythm that is completely chaotic and unpredictable.



Regularity: usually regular (could be irregular with variable conduction)
Rate: varies with conduction; < 100 bpm is controlled; > 100 bpm is uncontrolled (rapid ventricular response); usually has ventricular rates of 75 bpm (4:1), 100 bpm (3:1) or 150 bpm (2:1), depending on conduction ratio
P wave: none; flutter (F) waves; characteristic "sawtooth" baseline
QRS complex: < 0.12 second
PR interval: not discernible

Figure 7-11 | Atrial flutter

Causes

Atrial fibrillation can occur in young patients with no history of cardiac disease. Acute alcohol toxicity can precipitate an episode of atrial fibrillation in otherwise healthy patients ("holiday heart syndrome"). However, atrial fibrillation commonly occurs in the presence of underlying heart disease, lung disease, hyperthyroidism or myocardial infarction.

Signs and Symptoms

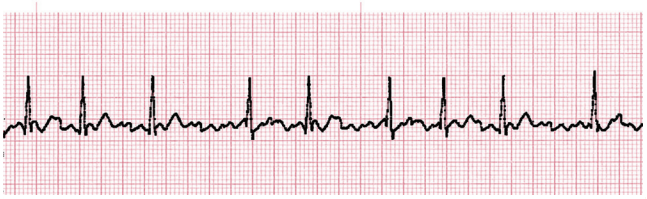
Patients with atrial fibrillation may be asymptomatic. However, ventricular rates greater than 100 bpm are usually not tolerated well because the filling time for the ventricles is significantly reduced. Symptoms may include shortness of breath, palpitations, chest pain, light-headedness, dizziness and fatigue. In extreme cases, hypotension, syncope and heart failure can occur.

ECG Findings

The two key features of atrial fibrillation on ECG are the absence of discrete P waves and the presence of irregularly irregular QRS complexes (Figure 7-12). The baseline appears flat or undulates slightly, producing **fibrillatory waves**.

Ventricular Tachycardia

Ventricular tachycardia occurs when a ventricular focus below the bundle of His becomes the new pacemaker. The ventricles contract rapidly (usually at a rate faster than 100 bpm) and usually with a regular rhythm. The rapid ventricular rate significantly diminishes cardiac output and can only be sustained for a short period before the patient becomes hemodynamically compromised. Ventricular tachycardia can quickly turn into ventricular fibrillation, leading to cardiac arrest. Torsades de pointes is a highly unstable form of polymorphic ventricular tachycardia that may revert to sinus rhythm or degenerate into pulseless ventricular tachycardia or ventricular fibrillation.



Regularity: irregularly irregular
Rate: varies with conduction; < 100 bpm is controlled; > 100 bpm is uncontrolled (rapid ventricular response)
P wave: none; fibrillation (f) waves; chaotic baseline
QRS complex: < 0.12 second
PR interval: not discernible

Figure 7-12 | Atrial fibrillation

Causes

Ventricular tachycardia usually occurs in the presence of heart disease or damage, such as that caused by acute or remote myocardial infarction or cardiomyopathy. There is a significant risk for ventricular tachycardia after myocardial infarction, and this risk can last for weeks, months or years. Ventricular tachycardia may also be precipitated by medications that prolong the QT interval, including amiodarone or other antiarrhythmics and certain antibiotics and antidepressants. Electrolyte derangements (including hypocalcemia, hypomagnesemia and hypokalemia) can also be involved.

Torsades de pointes most often accompanies prolonged QT intervals, which may be the result of a congenital condition, acute myocardial infarction, medications or drug–drug interactions. The risk for torsades de pointes is increased when the QTc is greater than 500 milliseconds (0.5 second).

Signs and Symptoms

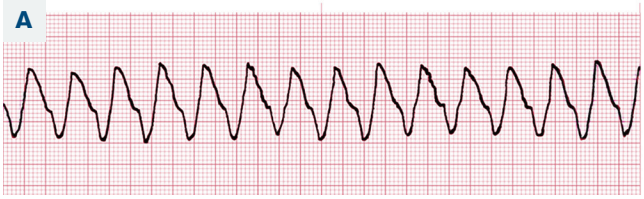
With sustained ventricular tachycardia, signs and symptoms of reduced cardiac output and hemodynamic compromise develop, including chest pain, hypotension and loss of consciousness.

ECG Findings

In ventricular tachycardia, the QRS complexes are wide (lasting longer than 0.12 second). When there is only one ectopic focus in the ventricles, **monomorphic ventricular tachycardia** is seen on the ECG (i.e., the QRS complexes are generally the same shape; Figure 7-13A). When there are two or more ectopic foci, **polymorphic ventricular tachycardia** is seen. In polymorphic ventricular tachycardia, the QRS complexes vary in shape and rate (see Figure 7-13B).

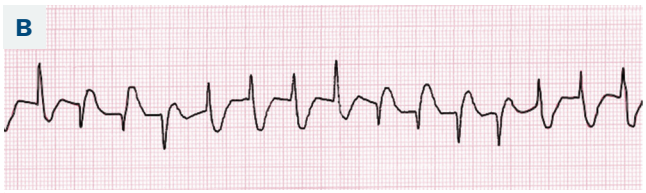
When the QRS complexes in ventricular tachycardia are polymorphic and wide, the rhythm may be torsades de pointes. In torsades de pointes ("twisting of the points"), the QRS complexes appear to pivot around the isoelectric line, deflecting upward and then downward, with their amplitude becoming smaller and larger, then smaller again (see Figure 7-13C). The rhythm may be regular or irregular. The rate is faster than 200 bpm. P waves are not visible, and the QRS complexes are wide (lasting longer than 0.12 second) and difficult to measure.

A



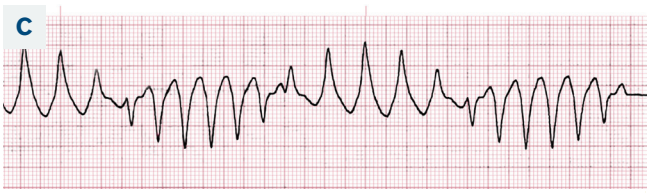
Regularity: regular
Rate: > 100 bpm
P wave: not discernible
QRS complex: ≥ 0.12 second, uniform in shape
PR interval: not discernible

B



Regularity: irregular (can appear regular due to fast rate)
Rate: > 100 bpm
P wave: not discernible
QRS complex: ≥ 0.12 second, variable in shape
PR interval: not discernible

C



Regularity: regular or irregular
Rate: > 200 bpm
P wave: not discernible
QRS complex: ≥ 0.12 second, variable in shape
PR interval: not discernible

Figure 7-13 | Ventricular tachycardia. (A) Monomorphic ventricular tachycardia. (B) Polymorphic ventricular tachycardia. (C) Torsades de pointes.

Practice Note

Monomorphic ventricular tachycardia is more likely caused by a chronic condition, such as scarring from a healed infarction. It may also be seen with reentrant rhythms. Polymorphic ventricular tachycardia is more likely caused by an acute condition, such as ischemia, current infarction or profound electrolyte disturbance.

Patient Assessment

Rapid Assessment

The patient's appearance on rapid assessment can vary widely, depending on the arrhythmia. The patient may appear to be in minimal distress, show signs of hemodynamic compromise (e.g., pale, mottled or cyanotic skin) or show signs related to the underlying cause of the arrhythmia (e.g., pain, diaphoresis).

Primary Assessment

Conduct a primary assessment following the ABCDE approach and provide care as needed. Obtain and monitor the patient's vital signs. Establish cardiac monitoring, pulse oximetry and vascular access, and be prepared to provide CPR and defibrillation if the patient's condition deteriorates. Provide the minimal level of supplemental oxygen needed to maintain an oxygen saturation of at least 94% and provide assistance with ventilation if needed. Assess level of consciousness in patients who appear to have changes in mental status. Obtain a 12- or 15-lead ECG if time and resources permit.

Secondary Assessment

The goals of the secondary assessment are to:

- Identify signs and symptoms and determine whether they are being caused by the arrhythmia or another condition.
- Determine the severity of the signs and symptoms.
- Identify the arrhythmia, if not done as part of the primary assessment.
- Identify potentially reversible causes of the arrhythmia.

History

Key information to elicit during the history includes:

- A description of the patient's symptoms, including a full description of symptoms such as chest pain or syncope.
- A medical history of arrhythmias, cardiac disorders or cardiac surgery.
- The presence of comorbid conditions, including cardiovascular disease, pulmonary disease and thyroid disease.
- Current medications, including antiarrhythmic agents.

Physical Examination

Perform a focused examination of the chest.

Diagnostic Tests

A 12-lead ECG aids in identifying underlying causes of the arrhythmia (e.g., myocardial ischemia or infarction, electrolyte disturbance) and is also helpful for distinguishing a wide-complex ventricular tachycardia from supraventricular tachycardia with aberrant conduction (Box 7-1).

Approach to the Patient

Bradyarrhythmia

The *Bradyarrhythmia: Adult* Treatment Guideline summarizes the approach to a patient with a bradyarrhythmia.

Assess and Recognize

When bradyarrhythmia is suspected, the team takes actions to ensure adequate airway, breathing and circulation, including obtaining and monitoring vital signs, establishing cardiac monitoring and pulse oximetry, administering supplemental oxygen if needed and ensuring vascular access. If time and resources permit, a 12-lead ECG should be obtained. Because the rhythm may change to a cardiac arrest rhythm, the team should be prepared to initiate CPR and defibrillation.

Because care depends on whether the patient is clinically stable or unstable, the key determination to make when assessing and caring for a patient with bradycardia is whether the bradyarrhythmia is causing hemodynamic compromise. Findings that may suggest that the patient is experiencing hemodynamic compromise (and is therefore unstable) include changes in mental status, ischemic chest discomfort, hypotension, signs of shock or acute heart failure.

Care

Throughout treatment, work to determine the underlying cause of the bradycardia and continually reassess the patient's condition. Clinical signs of improved cardiac output include a palpable pulse, an increase in blood pressure, an improved level of consciousness, and improved skin color and temperature.

Treatment pathways depend on whether the patient is stable or unstable.

Stable Bradyarrhythmia

If the patient is not showing signs and symptoms of hemodynamic compromise, the patient's condition should be monitored (cardiac monitoring, blood pressure monitoring, vital signs). A 12-lead ECG may be obtained and an effort should be made to identify and address the underlying cause of the bradyarrhythmia.

Unstable Bradyarrhythmia

If the patient is hemodynamically unstable, initiate treatment immediately (Figure 7-14). It may be necessary to implement multiple treatment measures simultaneously. First-line therapy is with atropine. Second-line therapies include transcutaneous pacing and β-adrenergic agonists. Consider implementing one of the second-line therapies immediately if the patient has second-degree AV block type II or third-degree AV block. Seek expert consult and consider transvenous pacing if first- and second-line therapies are not effective.

Atropine

Atropine is an anticholinergic drug that increases SA node firing by counteracting vagus nerve action to increase the heart rate. It is the first-line therapy for symptomatic bradycardia. Administer a 0.5-mg bolus intravenously every 3 to 5 minutes, up to a maximum dose of 3 mg. Administering the maximum dose of atropine can take up to 30 minutes. If the atropine is

Figure 7-14 | Interventions for unstable bradyarrhythmia. (A) Atropine is considered first-line therapy for symptomatic bradycardia. (B) Transcutaneous pacing is a second-line therapy for symptomatic bradycardia.

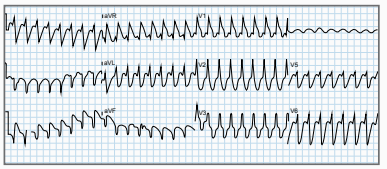
Box 7-1 | 12-Lead ECG Criteria Suggestive of Ventricular Tachycardia

A wide-complex tachyarrhythmia is most often ventricular tachycardia. However, a supraventricular tachycardia with aberrant conduction can also produce wide QRS complexes on ECG. When providing care in emergent and urgent situations, the patient's overall condition and whether the QRS complex is narrow or wide should guide therapy. Determining the specific type of tachyarrhythmia is less important. However, a 12-lead ECG or lead V_1 on a 5-lead ECG may be helpful for distinguishing between supraventricular tachycardia with aberrant conduction and ventricular tachycardia.

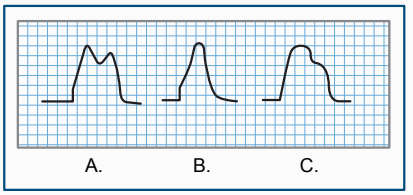
The following are validated criteria suggestive of ventricular tachycardia with a high degree of specificity (greater than 90%). If any one of these criteria is met, treat the arrhythmia as stable ventricular tachycardia. Note that although these criteria can assist with diagnosing ventricular tachycardia, they are of little use in ruling it out. In other words, even if none of these criteria are met, the patient may still have ventricular tachycardia.

Differentiating supraventricular tachycardia with aberrant conduction from ventricular tachycardia can be complex. If in doubt and the patient's condition permits, seek expert consultation. If the patient is unstable, the treatment is synchronized cardioversion.

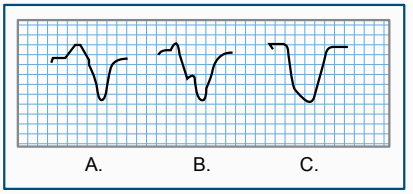
1. QRS axis > 180 (extreme right axis deviation) with a positive deflection in lead V_1



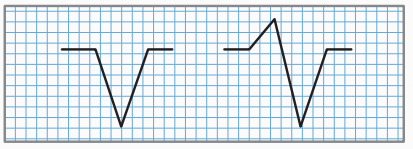
2. Positive deflection of the QRS complex in lead V_1 with characteristic morphology:
   - **a.** Taller left peak than right peak
   - **b.** Single upright "steeple" sign
   - **c.** "Mountain" with slope



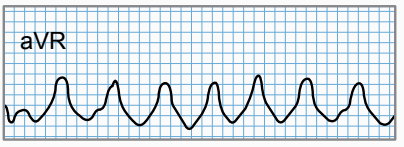
3. Negative deflection of the QRS complex in lead V_1 with characteristic morphology:
   - **a.** Initial wide (> 0.4 second) r wave
   - **b.** Notch on the initial downward deflection
   - **c.** Slur on the initial downward deflection



4. Negative deflection of the QRS complex in lead V_6



5. Initial r wave in lead aVR



having no effect, do not wait to reach the maximum dose of atropine before initiating second-line therapies.

Practice Note

Use atropine with caution in patients with acute coronary ischemia or myocardial infarction because in these patients, atropine can cause adverse effects, including ventricular tachycardia or ventricular fibrillation.

Transcutaneous Pacing

If at any point atropine is not effective, consider initiating transcutaneous pacing as a second-line therapy. Also consider implementing transcutaneous pacing immediately if vascular access is difficult to achieve or the patient has second-degree AV block type II or third-degree AV block. Transcutaneous pacing can be painful. If the patient's clinical condition permits, administer sedation or analgesia before pacing. After initiating pacing, verify electrical capture (evidenced by wide QRS complexes and tall, broad T waves following each pacing spike on the monitor). Confirm mechanical capture by assessing the patient for clinical signs of improved cardiac output.

β-Adrenergic Agonists

Epinephrine or dopamine may be administered to patients with symptomatic bradycardia as an alternative second-line therapy. These medications may also be considered when the cause of the bradyarrhythmia is an overdose with a β-blocker or calcium channel blocker. The initial dose is 2 to 10 mcg/min by IV infusion for epinephrine or 2 to 20 mcg/kg/min by IV infusion for dopamine, and then the dose is titrated to the patient's response.

Tachyarrhythmia

The *Tachyarrhythmia: Adult* Treatment Guideline summarizes the approach to a patient with a tachyarrhythmia.

Assess and Recognize

When tachyarrhythmia is suspected, the team takes actions to ensure adequate airway, breathing and circulation, including obtaining and monitoring vital signs, establishing cardiac monitoring and pulse oximetry, administering supplemental oxygen if needed and ensuring vascular access. If time and resources permit, a 12-lead ECG should be obtained. Because the rhythm may change to a cardiac arrest rhythm, the team should be prepared to initiate CPR and defibrillation.

When a patient is found to have tachyarrhythmia with a pulse, take a three-step approach to determining appropriate care.

- First, look at the heart rate. Tachycardia caused by a systemic condition is usually associated with a heart rate between 100 and 150 bpm, whereas tachyarrhythmias are usually associated with heart rates greater than 150 bpm. If the heart rate is between 100 and 150 bpm, the tachycardia is most likely sinus tachycardia. Search for an underlying systemic cause (such as dehydration, blood loss, fever, infection or anxiety) and treat that first. If the heart rate is 150 bpm or more, the tachycardia is likely caused by an arrhythmia.

Practice Note

Even when a patient's heart rate is between 100 and 150 bpm, signs of instability should be sought and, if found, treatment for unstable tachyarrhythmia should be initiated.

- Next, if the patient's heart rate is greater than 150 bpm and the tachycardia is not presumed to be sinus tachycardia, determine whether the patient is stable or unstable. A patient who is showing signs and symptoms of hemodynamic compromise despite initial management of airway and breathing is unstable and requires immediate therapy with synchronized cardioversion.
- Finally, if the patient's heart rate is greater than 150 bpm, the tachycardia is not presumed to be sinus tachycardia and the patient is stable (i.e., not showing signs of hemodynamic compromise), determine whether the QRS complexes are narrow (< 0.12 second) or wide (≥ 0.12 second).

Care

Throughout treatment, work to determine the underlying cause of the tachycardia and continually reassess the patient's clinical condition.

Sinus Tachycardia

If the patient's heart rate is between 100 and 150 bpm and the patient is not showing signs of instability, the patient's condition should be monitored (e.g., cardiac monitoring, noninvasive blood pressure monitoring, vital signs). A 12-lead ECG may be obtained and an effort should be made to identify and address the underlying cause of the tachycardia.

Unstable Tachyarrhythmia with a Pulse

Synchronized cardioversion is indicated for the treatment of unstable supraventricular tachyarrhythmias (including

atrial flutter and atrial fibrillation) and monomorphic ventricular tachycardia with a regular rhythm (Figure 7-15). If the patient's condition allows, administer sedation before initiating cardioversion. Dosages are as follows:

- Narrow-complex regular rhythm: 50 to 100 joules
- Narrow-complex irregular rhythm: 120 to 200 joules (biphasic defibrillator) or 200 joules (monophasic defibrillator)
- Wide-complex regular rhythm: 100 joules
- Wide-complex irregular rhythm: defibrillation dose; not synchronized

Consider adenosine (6 mg via rapid IV push followed by a rapid 10- to 20-mL normal saline flush) before synchronized cardioversion if vascular access is readily available.

Stable Narrow-Complex Supraventricular Tachyarrhythmia

If the patient is stable, the QRS complex is narrow and the rhythm is regular, attempt vagal maneuvers (Table 7-1) to break the tachyarrhythmia. If vagal maneuvers are ineffective, administer adenosine (6 mg via rapid IV push followed by a 10- to 20-mL normal saline flush). Up to two more 12-mg doses of adenosine (each followed by a normal saline flush) may be given if the rhythm does not convert. If vagal maneuvers or adenosine are effective and the patient's rhythm converts, monitor the patient for recurrence of the tachyarrhythmia. If the tachyarrhythmia converts but then returns or does not convert, consider a calcium channel blocker or β-blocker and seek expert consultation.

Figure 7-15 | If a patient with tachyarrhythmia and a pulse is unstable, the treatment is synchronized cardioversion.

Table 7-1 | Vagal Maneuvers

Maneuver	Method
Valsalva maneuver	Instruct the patient to exhale forcefully against a closed airway or blow through an occluded straw.
Cold stimulus	Apply a cold stimulus (e.g., an ice pack or a washcloth soaked in cold water) to the patient's nose and mouth for 10 seconds.
Gagging	Touch a tongue depressor to the back of the patient's throat to stimulate the gag reflex.
Carotid massage	With the patient's neck extended and the head turned away, apply pressure underneath the angle of the jaw in a circular motion for 10 seconds (not recommended for patients with carotid artery stenosis or a history of smoking).

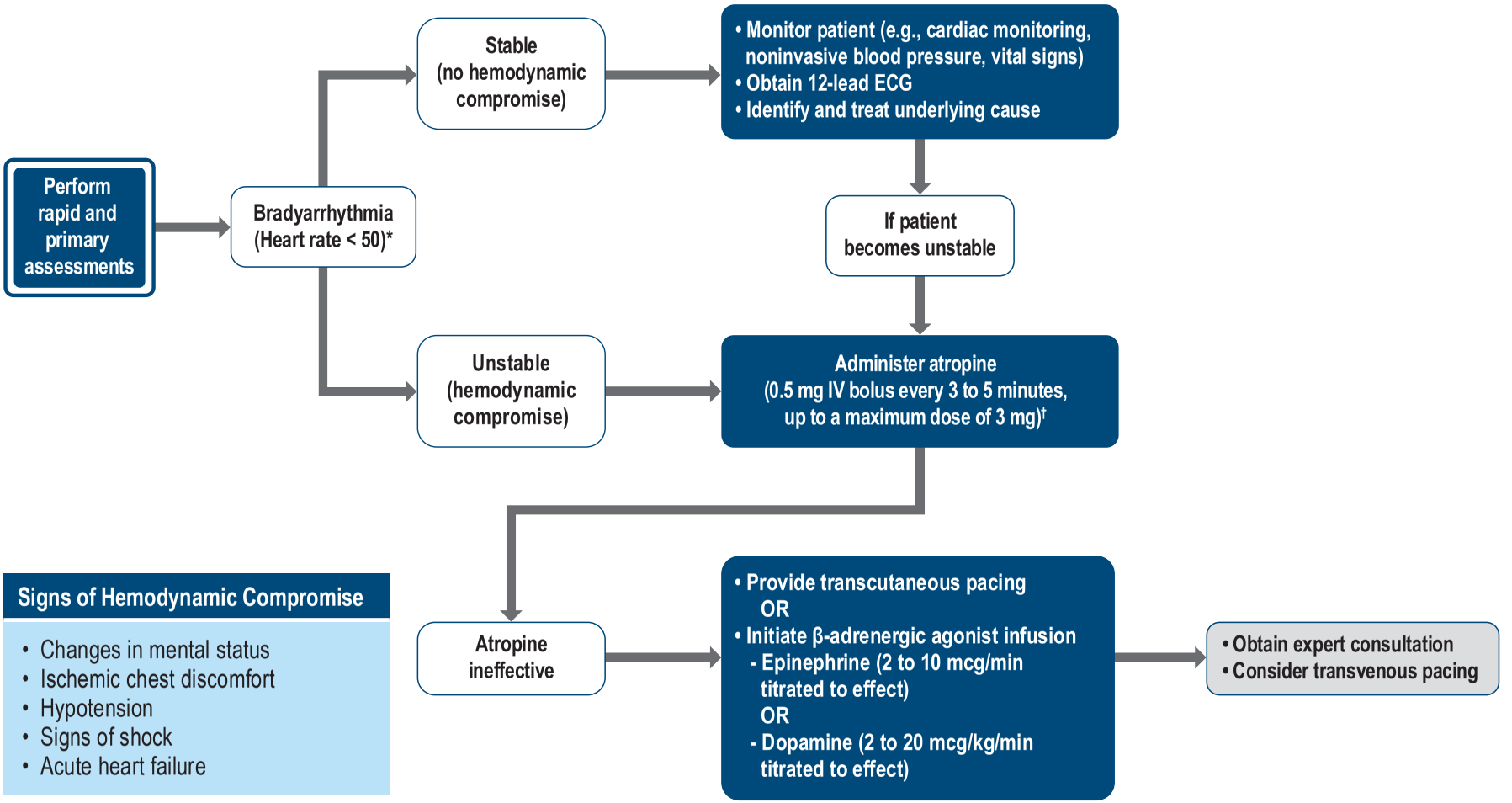
Stable Wide-Complex Ventricular Tachycardia

If the rhythm is wide (QRS complex ≥ 0.12 second), regular and monomorphic and the patient is stable, the rhythm may be supraventricular tachycardia with aberrant conduction. In this case, consider adenosine (6 mg via rapid IV push followed by a 10- to 20-mL normal saline flush). For stable patients with wide rhythms, consider an antiarrhythmic infusion of procainamide, amiodarone or sotalol.

- **Procainamide**: Avoid in patients with known prolonged QT intervals or congestive heart failure. Administer 20 to 50 mg/min until the arrhythmia is suppressed or a maximum dose of 17 mg/kg is given. Stop administration if hypotension develops or the QRS duration increases by more than 50%. The maintenance infusion is 1 to 4 mg/min.
- **Amiodarone**: Administer 150 mg over 10 minutes. Repeat as needed if the tachyarrhythmia recurs. The maintenance infusion is 1 mg/min for the first 6 hours.
- **Sotalol**: Avoid in patients with known prolonged QT intervals. Administer 100 mg (1.5 mg/kg) over 5 minutes.

Consultation with an expert regarding management of the tachyarrhythmia and the choice of antiarrhythmic may be necessary.

BRADYARRHYTHMIA: ADULT



*Bradycardia is defined as less than 60 bpm. In most instances, symptoms present when the heart rate is less than 50 bpm.

†Consider implementing transcutaneous pacing or β-adrenergic agonist therapy immediately for patients with second-degree atrioventricular (AV) block type II or third-degree AV block while preparing for transvenous pacing. Consider implementing transcutaneous pacing immediately if vascular access is difficult to achieve.

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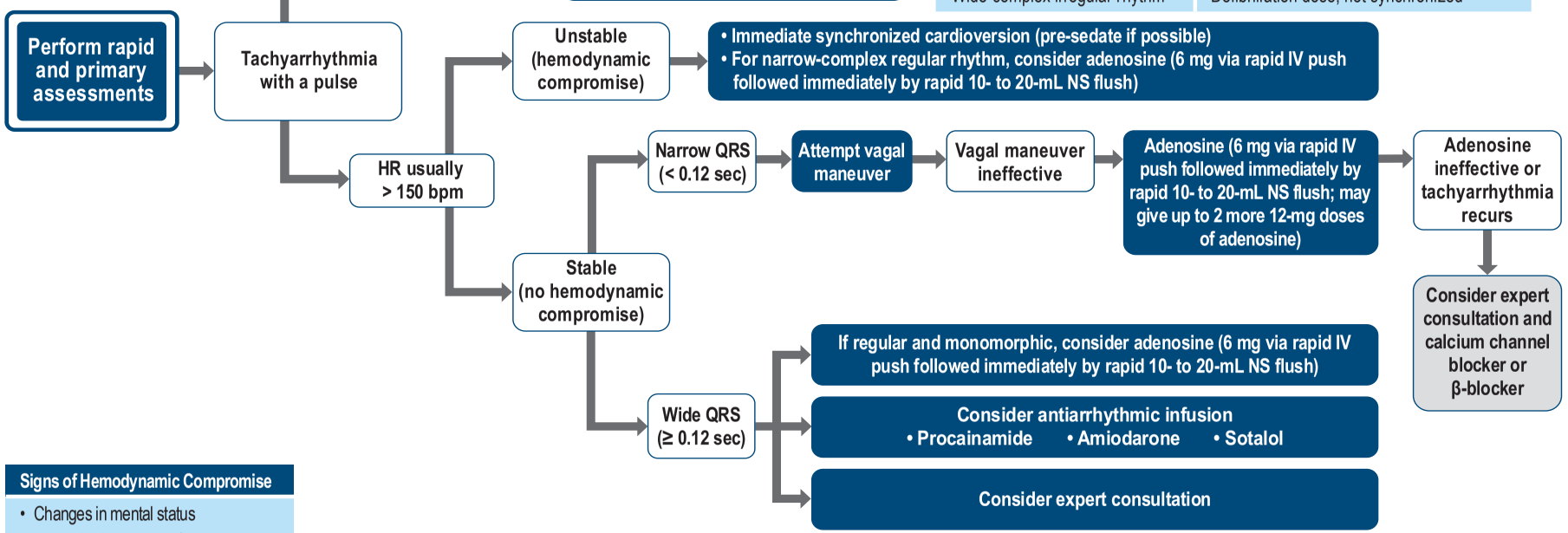
TACHYARRHYTHMIA: ADULT



Energy Doses for Synchronized Cardioversion	
Narrow-complex regular rhythm	50 to 100 J
Narrow-complex irregular rhythm	Biphasic: 120 to 200 J Monophasic: 200 J
Wide-complex regular rhythm	100 J
Wide-complex irregular rhythm	Defibrillation dose; not synchronized

Signs of Hemodynamic Compromise

- Changes in mental status
- Ischemic chest discomfort
- Hypotension
- Signs of shock
- Acute heart failure

Vagal Maneuvers

- Valsalva maneuver
- Cold stimulus
- Gagging
- Carotid massage

Antiarrhythmic Infusion	
Procainamide	• Avoid in prolonged QT or congestive heart failure • 20 to 50 mg/min until arrhythmia is suppressed or maximum dose of 17 mg/kg is given; stop if hypotension develops or QRS duration increases by more than 50% • Maintenance infusion: 1 to 4 mg/min
Amiodarone	• 150 mg IV over 10 minutes; repeat as needed if arrhythmia recurs • Maintenance infusion: 1 mg/min for first 6 hours
Sotalol	• Avoid in prolonged QT • 100 mg (1.5 mg/kg) over 5 minutes

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CHAPTER 8

Cardiac Arrest

Introduction

In the United States, approximately 200,000 cases of in-hospital cardiac arrest and approximately 395,000 cases of out-of-hospital cardiac arrest occur annually. No matter where cardiac arrest occurs, patient outcomes are improved when each link in the Cardiac Chain of Survival is implemented swiftly and properly.

Recognizing Cardiac Arrest Rhythms

Rhythms associated with cardiac arrest include ventricular fibrillation, pulseless ventricular tachycardia, pulseless electrical activity (PEA) and asystole. The first step in recognizing a cardiac arrest rhythm is establishing that the patient does not have a pulse by checking for a carotid pulse.

Ventricular Fibrillation

Ventricular fibrillation is characterized by erratic, rapid and completely ineffective depolarization of the ventricles. Rather than contracting, the ventricles quiver. This rhythm is fatal if not corrected quickly.

Causes

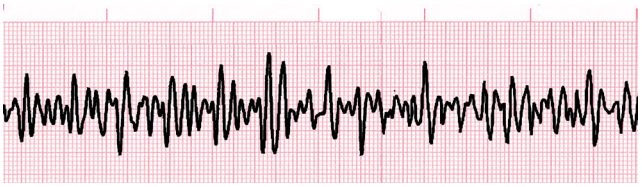
Precipitating causes of ventricular fibrillation include myocardial ischemia or infarction, shock, electrocution, stimulant overdose and ventricular tachycardia (including torsades de pointes).

ECG Findings

The rhythm is irregular and there are no discernible P waves, QRS complexes or T waves (Figure 8-1). The waveforms that are seen may vary in amplitude, from coarse to fine. As ventricular fibrillation progresses, the waveforms may change from coarse to fine and eventually disappear (asystole).

Pulseless Ventricular Tachycardia

Patients in ventricular tachycardia may or may not have a pulse. Pulseless ventricular tachycardia occurs when the ventricles are not contracting effectively enough to sustain sufficient cardiac output.



Regularity: irregular
Rate: not measurable
P wave: not discernible
QRS complex: chaotic, not discernible
PR interval: not discernible

Figure 8-1 | Ventricular fibrillation

Causes

The underlying causes of pulseless ventricular tachycardia are the same as for ventricular tachycardia (see Chapter 7) and include heart disease or damage (as from myocardial infarction), certain medications and electrolyte imbalances.

ECG Findings

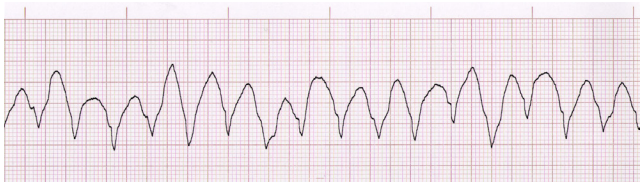
In pulseless ventricular tachycardia, the ventricular rate is usually greater than 180 bpm, and the QRS complexes are very wide (Figure 8-2).

Pulseless Electrical Activity

Pulseless electrical activity (PEA) is a term used to describe several rhythms that are organized on ECG (i.e., the QRS complexes are similar in appearance) but the patient has no palpable pulse. The heart's conduction system is functioning, but the myocardium is not contracting (or contracting too weakly) to produce cardiac output, or volume is not sufficient to maintain cardiac output.

Causes

PEA may be seen immediately after successful defibrillation of a patient with ventricular fibrillation or pulseless ventricular tachycardia. But when PEA is the presenting rhythm ("primary PEA"), the underlying cause is usually a condition that either affects contractility or ejection (e.g., hypoxia, acidosis, anterior wall myocardial infarction) or leads to inadequate preload (e.g., severe hypovolemia, pulmonary embolism, tension pneumothorax, cardiac tamponade, right ventricular infarction).



Regularity: regular or irregular
Rate: usually > 180 bpm
P wave: not discernible
QRS complex: ≥ 0.12 second
PR interval: not discernible

Figure 8-2 | Pulseless ventricular tachycardia

ECG Findings

In PEA, the monitor shows an identifiable rhythm but no pulse can be palpated. The rhythm may be sinus, atrial, junctional or ventricular in origin. The rate may be fast or slow. The QRS complexes are similar in appearance and may be narrow or wide. When the cause of the PEA is cardiac in origin, it is most common to see a slow rate and wide QRS complexes. Noncardiac causes of PEA are often associated with a rapid rate and narrow QRS complexes.

Asystole

In asystole, there is no electrical activity and therefore no contraction.

Causes

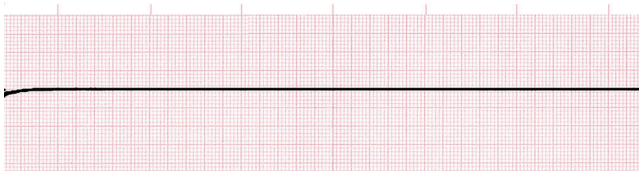
Asystole is often the terminal rhythm in untreated pulseless ventricular tachycardia or ventricular fibrillation or when resuscitation efforts are unsuccessful. Other causes include narcotic drug overdose, hypothermia, myocardial infarction, pulmonary embolism, hyperkalemia, hypoxia (drowning, suffocation) and indirect lightning strike.

ECG Findings

Asystole is characterized by a lack of discernible electrical activity on ECG, which results in a "flatline" appearance (Figure 8-3). Very rarely, the sinoatrial (SA) node may generate impulses that cause atrial depolarization but are completely blocked at the atrioventricular (AV) node, resulting in "P-wave asystole."

Practice Note

Care must be taken to differentiate asystole from fine ventricular fibrillation. To do this, quickly look at another lead to evaluate the electrical activity in a different plane.



Regularity: not discernible
Rate: not discernible
P wave: usually absent
QRS complex: not discernible
PR interval: not discernible

Figure 8-3 | Asystole

Practice Note

Although asystole is said to have a "flatline" appearance on ECG, there is some fluctuation from the baseline in asystole. A completely flat baseline is likely the result of disconnected leads or equipment problems.

Reversible Causes of Cardiac Arrest (Hs and Ts)

When caring for a patient in cardiac arrest, it is important to recognize reversible causes for the arrest and address them (Figure 8-4). This is especially important with PEA and asystole, which often have reversible underlying causes. The mnemonic *Hs and Ts* can help you to remember the reversible causes of cardiac arrest (Box 8-1).

While resuscitation is underway, review the patient's medical history with the providers who were caring for the patient at the time of the arrest or family members to identify details that could point to one of the Hs or Ts as an underlying cause (Table 8-1). Of particular importance are changes in the patient's clinical condition leading up to the arrest, disorders or situations that could predispose a patient to developing one of the Hs or Ts, risk factors for cardiac and pulmonary conditions and medication use.

Figure 8-4 | While resuscitation is underway, consider possible underlying causes for the cardiac arrest (Hs and Ts) with the team.

Box 8-1 | Reversible Causes of Cardiac Arrest: Hs and Ts

Hs

- Hypovolemia
- Hypoxia
- Hydrogen ion excess (acidosis)
- Hyper- or hypokalemia
- Hypothermia

Ts

- Toxins
- Tamponade
- Tension pneumothorax
- Thrombosis (pulmonary embolism)
- Thrombosis (myocardial infarction)

Diagnostic studies that may prove useful for identifying underlying causes of the cardiac arrest include a 12- or 15-lead ECG, arterial blood gases, a serum electrolyte panel, chest radiography and bedside ultrasonography. Although some diagnostic studies can be done while resuscitation efforts are underway, others (such as a 12-lead ECG) should be delayed until after return of spontaneous circulation (ROSC) is achieved.

Practice Note

Ultrasonography can be a useful tool for recognizing several underlying causes of cardiac arrest, including pulmonary embolism, tension pneumothorax, cardiac tamponade and hypovolemia. Consider use of ultrasonography if the equipment and a skilled technician are readily available, and if doing so will not impede or delay resuscitation efforts.

Hypovolemia

Hypovolemia (a reduction of fluid volume in the circulatory system) develops because of excessive blood or fluid loss (for example, because of trauma, internal bleeding or severe dehydration). A fluid challenge can aid in determining whether hypovolemia is contributing to the cardiac arrest.

Hypoxia

Suspect hypoxia in any patient who had respiratory difficulties preceding cardiac arrest. To address hypoxia as a cause of cardiac arrest, ensure that the patient's airway is patent and provide adequate ventilation and supplemental oxygen.

Table 8-1 | Clinical Clues to Underlying Causes of Cardiac Arrest (Hs and Ts)

Reversible Cause	History May Be Significant for:	Monitoring and Diagnostic Tests
Hypovolemia	Internal or external bleeding, dehydration, diabetes insipidus, diarrhea/vomiting, peritonitis, profound preload compromise (e.g., right ventricular infarction) Peri-arrest signs and symptoms: hypotension; oliguria; cyanosis; rapid, shallow breathing; confusion	12-lead ECG: narrow-complex ventricular tachycardia 15-lead ECG: ST-segment elevation in lead V_{4R}
Hypoxia	Trauma, chronic or acute respiratory disorder Peri-arrest signs and symptoms: respiratory distress, respiratory failure, respiratory arrest	Peri-arrest $ETCO_2$: > 50 mmHg Peri-arrest arterial oxygen saturation: < 90%
Hydrogen ion excess	**Metabolic acidosis** Diabetes, sepsis, renal disease, alcoholism Peri-arrest signs and symptoms: lethargy, drowsiness, hyperventilation, headache **Respiratory acidosis** Chronic or acute respiratory disorder Peri-arrest signs and symptoms: tachycardia, tachypnea, hypercapnia, hypertension, hypoxemia, hypercarbia, confusion, headache	Arterial blood gases: pH < 7.35 $ETCO_2$: < 29 mmHg in metabolic acidosis, > 50 mmHg in respiratory acidosis

Table 8-1 | Clinical Clues to Underlying Causes of Cardiac Arrest (Hs and Ts) *(continued)*

Reversible Cause	History May Be Significant for:	Monitoring and Diagnostic Tests
Hyper- or hypokalemia	**Hyperkalemia** Renal disease, trauma, burns, potassium supplementation Peri-arrest signs and symptoms: cramps, muscle twitching, hypotension **Hypokalemia** Renal disease, diuretics, eating disorder, diarrhea/vomiting Peri-arrest signs and symptoms: muscle weakness or spasms, confusion, drowsiness	**Hyperkalemia** 12-lead ECG: wide QRS complexes and tall, pointed T waves **Hypokalemia** 12-lead ECG: flat T waves, prominent U waves and possibly prolonged QT intervals
Hypothermia	Exposure, drowning, fluid resuscitation	...
Toxins	Drug abuse, medications	...
Tamponade	Chest trauma, pericarditis (lung/breast cancer, radiation therapy, post–myocardial infarction, infection, connective tissue disorders, chronic renal disease, postoperative complication) Peri-arrest signs and symptoms: dyspnea, tachypnea, tachycardia, anxiety, changes in mental status, hypotension, muffled heart sounds, jugular venous distension, pericardial friction rub, pulsus paradoxus	PEA rhythm with narrow QRS complexes, electrical alternans Bedside ultrasonography can aid in diagnosis
Tension pneumothorax	Chest trauma, thoracentesis, central venous catheter insertion Peri-arrest signs and symptoms: tachypnea, diminished or absent breath sounds, unequal chest expansion, tracheal deviation (late sign), jugular venous distension, hypotension, anxiety, diaphoresis, cyanosis, high peak inspiratory pressures (in patients receiving mechanical ventilation)	PEA rhythm with narrow QRS complexes Bedside ultrasonography can aid in diagnosis
Thrombosis (pulmonary embolism)	Abnormal clotting (cancer/cancer treatment, inherited disorder, cardiovascular disease, stroke, hormone therapy, history of deep venous thrombosis, anticoagulant therapy); stasis (bed rest, travel); vessel injury (post-surgical, central venous catheter, trauma) Peri-arrest signs and symptoms: dyspnea, tachycardia, pleuritic chest pain, hemoptysis, anxiety, diaphoresis, syncope	Bedside ultrasonography can aid in diagnosis
Thrombosis (myocardial infarction)	Coronary artery disease Peri-arrest signs and symptoms: chest pain/discomfort; dizziness, light-headedness or syncope; nausea or vomiting; dyspnea; diaphoresis; cyanosis	12- or 15-lead ECG: ST-segment changes, T-wave inversion

Hydrogen Ion Excess (Acidosis)

Suspect acidosis in patients with diabetes or probable acute or chronic renal failure. Arterial blood gases are used to confirm the diagnosis of acidosis. In patients with metabolic acidosis, the administration of an initial dose of sodium bicarbonate (1 mEq/kg) may be indicated. Sodium bicarbonate, if used, should be administered early in conjunction with standard cardiac arrest care.

Hyperkalemia or Hypokalemia

Potassium imbalances can precipitate cardiac arrest.

Suspect hyperkalemia in patients with acute or chronic renal failure and in those who had wide QRS complexes and tall, peaked T waves on ECG prior to the arrest. Several measures may be taken to reduce potassium levels, including administering sodium bicarbonate, glucose and insulin, or nebulized albuterol. Sodium bicarbonate is the preferred method of addressing hyperkalemia in patients in cardiac arrest because it causes a rapid shift in serum potassium level. Other therapies take much longer to work.

Suspect hypokalemia in patients with dehydration or overuse of diuretics. In hypokalemia, flat T waves, prominent U waves and possibly prolonged QT intervals may be seen on ECG before arrest. Treatment is intravenous administration of a dilute solution of potassium chloride.

Hypothermia

For patients with severe hypothermia (body temperature less than 86° F [30° C]) and cardiac arrest, core rewarming (with cardiopulmonary bypass, extracorporeal blood warming with partial bypass or thoracic lavage with warmed fluids) is indicated. Warmed fluids and warmed humidified oxygen may be administered as adjunctive therapies.

Toxins

Overdoses (of both illicit and therapeutic drugs) and poisoning can induce cardiac arrest. Drugs that are frequently implicated in cardiac arrest include cocaine, methamphetamines, opioids (heroin, fentanyl), β-blockers, calcium channel blockers, digoxin and tricyclic antidepressants. Reversal agents are specific to the toxin (e.g., glucagon for β-blocker overdose, sodium bicarbonate for tricyclic antidepressant overdose, naloxone for opioid overdose).

Tamponade

Cardiac tamponade occurs when fluid accumulates in the pericardial sac, compressing the heart and preventing it from pumping effectively. Causes of cardiac tamponade include trauma, cancer, renal failure, infections and idiopathic pericarditis. Cardiac tamponade may also be seen postoperatively.

Pre-arrest physical examination findings may include the three cardinal signs of cardiac tamponade (Beck's triad): hypotension (weak pulse or narrow pulse pressure), muffled heart sounds and jugular venous distension. In a patient with PEA, narrow QRS complexes and **electrical alternans** (i.e., beat-to-beat variation in the amplitude of the QRS complexes) may be seen.

Treatment is pericardiocentesis (needle aspiration of fluid from the pericardial sac).

Tension Pneumothorax

Tension pneumothorax occurs when air accumulates in the pleural space, causing the lung on the affected side to collapse and the mediastinum to shift, compressing the heart and vena cava. Compression of the vena cava leads to impaired venous return and decreased cardiac output. Penetrating chest trauma is a common cause of tension pneumothorax, but the condition can also develop in older patients with underlying lung disease and in patients who smoke.

Pre-arrest physical examination findings may include hypotension, tachycardia, absent breath sounds on the affected side, jugular venous distension, hyperresonance on percussion and tracheal deviation away from the affected side (a late sign). Difficulty ventilating the patient may also be a sign of tension pneumothorax. In a patient with PEA, narrow QRS complexes should raise suspicion for tension pneumothorax as a possible underlying cause.

Initial treatment is with needle chest decompression or thoracostomy.

Thrombosis (Pulmonary Embolism)

In massive pulmonary embolism, obstruction of the pulmonary artery and the release of vasoconstrictive

mediators from the thrombus lead to cardiogenic shock, which can quickly lead to cardiac arrest. Conditions in the patient history associated with prolonged immobilization or venous stasis, hypercoagulability or damage to the veins should increase suspicion for pulmonary embolism as a potential cause of cardiac arrest. Witnessed cardiac arrest and respiratory distress before arrest also may point to pulmonary embolism as the cause. PEA is the initial rhythm in one-third to one-half of patients with cardiac arrest caused by pulmonary embolism. Fibrinolytic therapy may be initiated for patients with known or suspected pulmonary embolism.

Thrombosis (Myocardial Infarction)

Myocardial infarction can lead to cardiac arrest. If a pre-arrest 15-lead ECG shows an inferior wall myocardial infarction with right ventricular involvement (evidenced by ST-segment elevation in lead V_{4R}), then impaired preload is likely the cause of cardiac arrest and fluid bolus therapy should be provided during resuscitation. If a pre-arrest 12-lead ECG shows anterior wall myocardial infarction, then impaired contractility is the likely cause of cardiac arrest. When myocardial infarction is the suspected cause of cardiac arrest, a 12-lead ECG should be obtained after ROSC is achieved to guide therapy and inotropic support may be needed to address cardiogenic shock.

Approach to the Patient

The *Cardiac Arrest: Adult* Treatment Guideline summarizes the approach to a patient in cardiac arrest.

Assess and Recognize

After determining that the patient is not responsive, not breathing and has no pulse, call for assistance and a defibrillator and initiate CPR immediately. Without interrupting high-quality CPR, attach the cardiac monitor/defibrillator and identify the arrest rhythm.

Care

When a patient is in cardiac arrest, treatment focuses on providing high-quality CPR (see Chapter 2), providing shocks (if the rhythm is shockable) and, if possible, determining and addressing the underlying cause. Ventilations should be provided with high-concentration, high-flow supplemental oxygen. Medications, although potentially helpful, are secondary interventions and have not been proven to lead to improved survival rates or neurologic function in patients who experience cardiac arrest. Obtaining vascular access should be delayed until after the first shock has been administered (in the case of a shockable rhythm) or CPR has been provided for at least 2 minutes (in the case of a nonshockable rhythm). Similarly, placement of an advanced airway and establishment of capnography should be delayed until after at least 2 shocks have been administered (in the case of a shockable rhythm) or CPR has been provided for at least 2 minutes (in the case of a nonshockable rhythm).

Practice Note

When a patient is receiving CPR, use capnography to monitor the effectiveness of compressions and for ROSC.

Practice Note

If the airway and adequate ventilations can be maintained without placing an advanced airway, consider delaying placement until after ROSC is achieved. After placing an advanced airway, verify correct placement using clinical parameters and capnography, and secure the device.

Practice Note

Remember that when an advanced airway is in place, chest compressions are performed continuously without pausing to deliver ventilations. Ventilations are delivered at a rate of 1 ventilation every 6 seconds. Each ventilation should last approximately 1 second and cause the chest to just begin to rise.

Shockable Rhythms

Ventricular fibrillation and pulseless ventricular tachycardia require defibrillation as soon as possible. Because shocks may not be successful or, in the case of successful defibrillation, the resultant rhythm may not be adequate to sustain perfusion or a pulse, resume CPR immediately after each shock. After every 2 minutes of CPR, reassess the rhythm (while minimizing interruptions

to chest compressions; Figure 8-5) to determine next actions:

- If the rhythm is shockable, resume CPR immediately and administer 1 shock as soon as the defibrillator is charged.
- If the rhythm is nonshockable and organized, attempt to palpate a pulse. If a definitive pulse is palpated, the patient has achieved ROSC and post–cardiac arrest care should be initiated. If a definitive pulse cannot be palpated, resume CPR immediately and follow the treatment guidelines for a nonshockable rhythm.

Practice Note

Check the pulse only if an organized rhythm is present.

Practice Note

During rhythm and pulse checks, pause compressions for no more than 10 seconds.

Repeat cycles of 2 minutes of CPR, rhythm check, shock and medication as appropriate until the rhythm check reveals a nonshockable rhythm, ROSC is achieved or the resuscitation effort is terminated. Clinical indications of ROSC include a palpable pulse, a measurable blood pressure or an $ETCO_2$ value greater than 40 mmHg.

Defibrillation

Administering shocks establishes a temporary "blank slate" by eliminating all electrical activity in the heart (in other words, it temporarily induces asystole). Ideally, the temporary pause will give the heart's normal pacemaker a chance to reestablish a regular rhythm that will ultimately lead to ROSC.

Figure 8-5 | Minimize pauses in compressions to less than 10 seconds during rhythm and pulse checks.

The energy dose depends on whether the defibrillator is biphasic or monophasic. Providers should familiarize themselves with the equipment used in their facility.

- If using a biphasic defibrillator, follow the manufacturer's recommendations for the initial dose (usually between 120 and 200 joules). Subsequent doses should be the same as or higher than the initial dose. If the manufacturer's recommendations for the initial dose are not known, use the highest energy dose available for the first and all subsequent shocks.
- If using a monophasic defibrillator, set the energy dose at 360 joules. Use this energy dose for each subsequent shock.

If defibrillation is initially successful in terminating the cardiac arrest rhythm but ventricular fibrillation or pulseless ventricular tachycardia resumes, use the energy dose that successfully terminated the rhythm for subsequent shocks.

Always precede the delivery of a shock by announcing the intention to shock in a clear, succinct manner. Before delivering a shock, perform a visual scan to ensure that no one is touching the patient or the bed and that oxygen delivery devices have been removed and set aside, away from the patient.

When delivering the shock, face the team, rather than the defibrillator (Figure 8-6).

When administering shocks, minimize interruptions to CPR.

- Continue providing compressions while placing the defibrillator pads on the patient's chest.
- If the rhythm check reveals a shockable rhythm, resume compressions as soon as the charging sequence begins and continue until immediately before the shock button is pushed and the shock is delivered.
- Immediately after the shock is delivered, resume CPR for 2 minutes before pausing compressions to conduct a rhythm check.

Medications

Various medications may be used in the treatment of ventricular fibrillation or pulseless ventricular tachycardia. Remember that in cardiac arrest, all medications administered through the IV or IO route should be followed by a 10- to 20-mL fluid bolus.

Vasoconstrictors

After 2 shocks have been delivered, epinephrine (1 mg IV/IO every 3 to 5 minutes) may be administered. The vasoconstrictive and positive ionotropic effects of epinephrine help to increase cerebral and coronary

Figure 8-6 | Ensure that all providers are clear of the patient and the bed before delivering a shock. When delivering the shock, face the team, rather than the defibrillator.

perfusion. Evidence suggests that epinephrine is most effective when administered early.

Antiarrhythmics

After 3 shocks have been delivered, consider administering an antiarrhythmic agent.

- Amiodarone: The initial dose of amiodarone is 300 mg administered as an IV/IO bolus. If the arrest rhythm persists, consider giving a second dose of 150 mg as an IV/IO bolus 3 to 5 minutes later.
- Lidocaine: Alternatively, lidocaine may be used. The initial dose is 1 to 1.5 mg/kg IV/IO, followed by 0.5 to 0.75 mg/kg IV/IO every 5 to 10 minutes, up to a maximum dose of 3 mg/kg.

> **Practice Note**
>
> Choose one antiarrhythmic agent (amiodarone or lidocaine) and use it for the duration of the resuscitation effort. Do not alternate between the two.

- Magnesium sulfate: If the rhythm is torsades de pointes, consider magnesium sulfate (1 to 2 g diluted in 10 mL of 5% dextrose in water or normal saline administered as an IV/IO bolus over 5 to 20 minutes). Magnesium sulfate should not be administered too quickly because it may induce hypotension.

Nonshockable Rhythms

Defibrillation is not indicated when the rhythm is PEA or asystole. Management of these rhythms involves providing continuous high-quality CPR, administering epinephrine (1 mg IV/IO repeated every 3 to 5 minutes) as soon as vascular access is established and performing rhythm checks after every 2 minutes of CPR. In addition, it is extremely important to look for and address potential underlying causes of the cardiac arrest. PEA and asystole often have an underlying cause, and unless that cause is addressed, the resuscitative effort will not be successful.

Repeat cycles of 2 minutes of CPR, rhythm check and medication as appropriate until the rhythm check reveals a shockable rhythm, ROSC is achieved or the resuscitation effort is terminated.

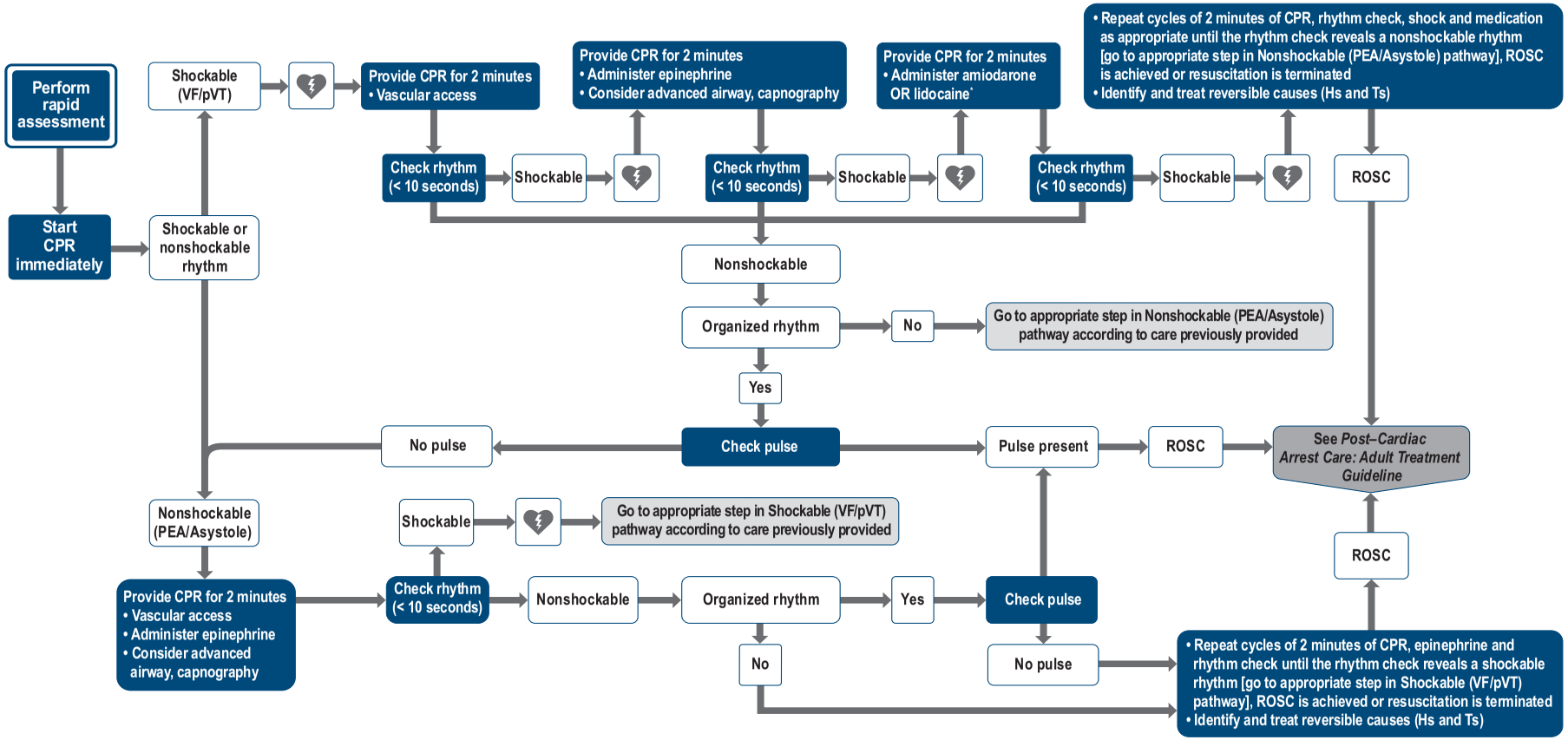
Terminating the Resuscitation Effort

If it seems unlikely that ROSC will be achieved, the team leader may decide to terminate the resuscitation effort. Many factors are considered when deciding to terminate the resuscitation effort, including:

- How much time elapsed before CPR was initiated and the first shock was provided.
- The patient's health status before the cardiac arrest and the presence of comorbidities.
- The initial cardiac arrest rhythm.
- The duration of the resuscitation effort.
- Physiologic data, such as an $ETCO_2$ level less than 10 mmHg after 20 minutes of high-quality CPR.

In general, the longer a patient is in cardiac arrest, the less likely the patient is to survive (or to survive with neurological function intact). However, in some situations it may be appropriate to consider prolonging the resuscitation effort, using specialized interventions or both. For example, it may be appropriate to prolong the resuscitation effort when more time is needed to address the underlying cause of the cardiac arrest (for example, drug overdose, hypothermia, pulmonary embolism). Specialized interventions, such as **extracorporeal cardiopulmonary resuscitation (ECPR)** may also be considered. In ECPR, large-bore cannulas are inserted into a major artery (e.g., the femoral artery) and the corresponding vein. Deoxygenated blood is removed from the body, passed through a membrane that removes the carbon dioxide and adds oxygen, warmed, filtered, and then pumped back into the body. For patients who are good candidates for the procedure, survival and neurological outcomes may be improved with the use of ECPR (compared with outcomes associated with standard CPR).

CARDIAC ARREST: ADULT



Medications	
Epinephrine	• 1 mg IV/IO bolus • May repeat every 3 to 5 minutes
Amiodarone	• 300 mg IV/IO bolus • May administer 1 additional dose of 150 mg after 3 to 5 minutes
Lidocaine	• 1 to 1.5 mg/kg IV/IO for first dose • 0.5 to 0.75 mg/kg IV/IO every 5 to 10 minutes, up to a maximum dose of 3 mg/kg

Defibrillation Energy Doses	
Biphasic	• Per manufacturer's recommendation (typically 120 to 200 J) or, if unknown, maximum available • Subsequent doses equal to or greater than first dose
Monophasic	• 360 J for all doses

*For torsades de pointes, consider magnesium sulfate (1 to 2 g diluted in 10 mL of D5W or NS administered as an IV bolus over 5 to 20 minutes).

Please see reverse side for additional information

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CARDIAC ARREST: ADULT CONTINUED

CPR Technique

Switch CPR compressors
- Every 2 minutes
- During rhythm check
- If provider is fatigued

- **Hand position:** Centered on the lower half of the sternum
- **Depth:** At least 2 inches
- **Number:** 30 compressions
- **Rate:** 100 to 120 per minute (15 to 18 seconds)
- **Full chest recoil**

- Each ventilation should last about 1 second and make the chest begin to rise
- Advanced airway: provide 1 ventilation every 6 seconds without pausing chest compressions

Discontinue CPR If:

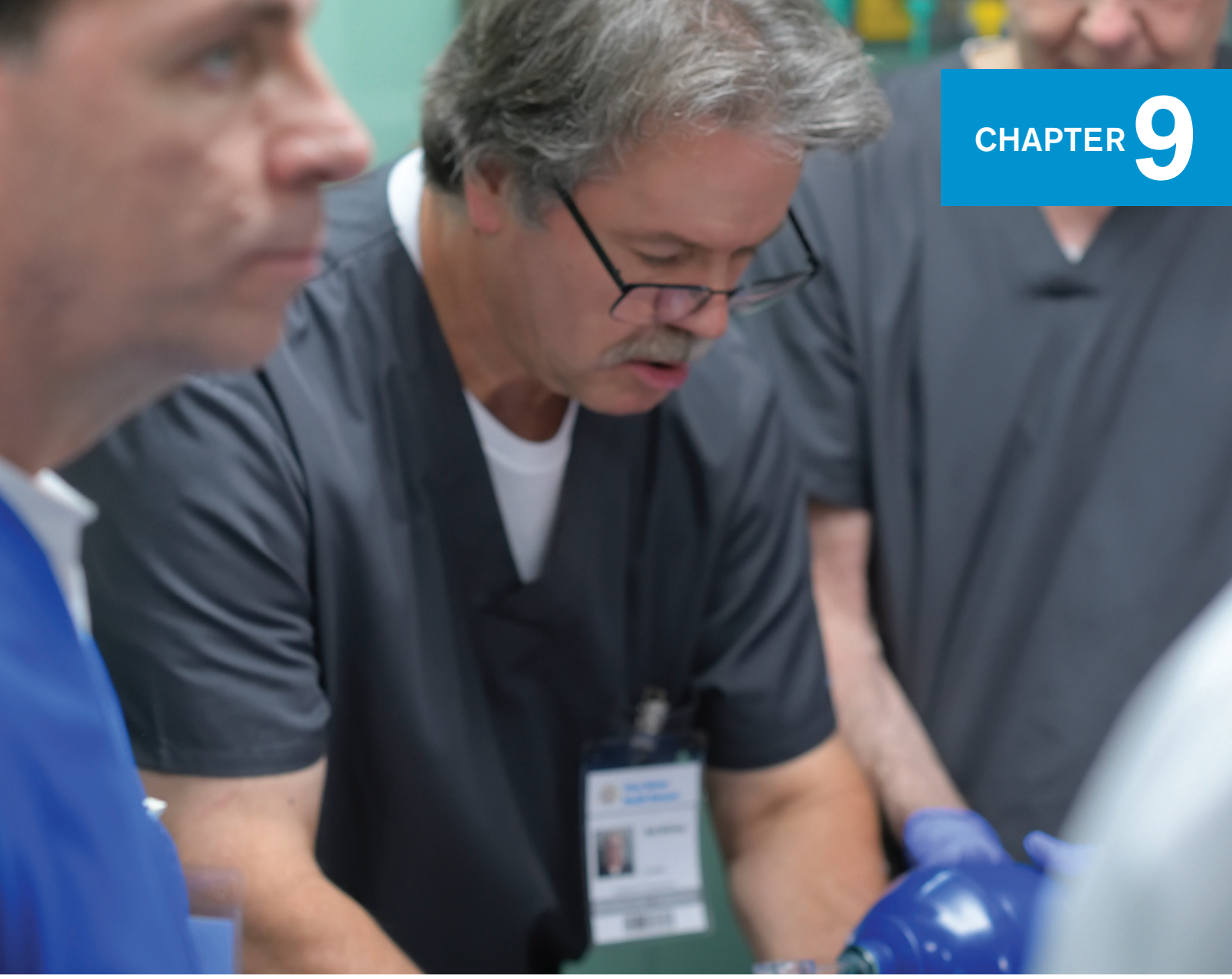
- Other trained providers arrive to relieve you
- You see signs of ROSC
- You are presented with a valid DNR order
- You are too exhausted to continue
- The situation becomes unsafe
- Care team leader terminates resuscitation

Hs

- Hypovolemia
- Hypoxemia
- Hydrogen ion excess (acidosis)
- Hyper-/hypokalemia
- Hypothermia
- Hyper-/hypoglycemia

Ts

- Tamponade
- Tension pneumothorax
- Thrombosis (pulmonary embolism)
- Thrombosis (myocardial infarction)
- Toxins



CHAPTER 9

Post–Cardiac Arrest Care

Introduction

Expert post–cardiac arrest care delivered by a multidisciplinary team can reduce both short- and long-term morbidity and mortality following the return of spontaneous circulation (ROSC). Post–cardiac arrest care includes interventions aimed at optimizing oxygenation, ventilation and perfusion; minimizing the systemic consequences of cardiac arrest; preventing future cardiac arrest; and assessing the patient's prognosis for recovery.

Pathophysiologic Consequences of Cardiac Arrest

Among patients who achieve ROSC, the short-term mortality rate (i.e., within the first 24 hours of arrest) is high. This is largely a result of the global consequences of hypoxemia and the **ischemia/reperfusion response**, in addition to the precipitating cause of the cardiac arrest itself. Blood flow must be restored to tissues that have been deprived of oxygen in order to prevent tissue death; however, reperfusion of previously ischemic tissues can induce an inflammatory response that causes cellular injury in addition to that caused by the ischemia itself. This is known as the ischemia/reperfusion response.

Sometimes referred to as **post–cardiac arrest syndrome**, the pathophysiologic consequences of cardiac arrest comprise four key areas:

- **Brain injury**. Brain injury, caused by ischemia and cerebral edema, is a significant cause of morbidity and mortality in patients who achieve ROSC.
- **Myocardial dysfunction**. Myocardial stunning secondary to the ischemia/reperfusion response causes systolic and diastolic dysfunction, leading to hemodynamic instability in the immediate post-arrest period.
- **Systemic dysfunction**. The ischemia/reperfusion response can trigger a systemic inflammatory response, which can lead to multiple organ dysfunction. In addition, organs that are sensitive to changes in perfusion pressure, such as the kidneys, are at increased risk for reperfusion injury.
- **Persistent precipitating conditions.** The underlying cause of the cardiac arrest may continue to have pathophysiologic consequences during the post-arrest period.

The duration of the cardiac arrest (i.e., from collapse through ROSC) directly affects the severity of the post–cardiac arrest syndrome.

After ROSC, survival outcomes are improved when a multidisciplinary team of providers works to stabilize the patient, minimize complications, and diagnose and treat the underlying cause (Figure 9-1).

Patient Assessment

Primary Assessment

As soon as ROSC is achieved, conduct a primary assessment to ensure adequate airway, breathing and circulation. Establish cardiac monitoring, pulse oximetry, capnography and noninvasive blood pressure monitoring or arterial pressure monitoring as needed. Quickly assess the patient's level of consciousness (using AVPU) and neurologic status (by checking the pupils for size, equality and reactivity to light).

Figure 9-1 | Expert care during the immediate post–cardiac arrest period can improve outcomes for the post–cardiac arrest patient.

Secondary Assessment

History

Review the patient's medical history for information that could help identify the underlying cause of the arrest (see Chapter 8).

Physical Examination

Conduct a focused cardiopulmonary physical examination to identify signs of underlying causes of the arrest (e.g., pneumothorax, cardiac tamponade) and check for CPR-related injuries or complications. A thorough neurologic examination is also necessary to establish a baseline for subsequent evaluations and to identify focal neurologic deficits.

Diagnostic Tests

Diagnostic studies aid in determining the pathophysiologic consequences of cardiac arrest, identifying the underlying cause and monitoring the patient's response to interventions (Table 9-1).

Table 9-1 | Diagnostic Tests That May Be Ordered in the Evaluation of the Post–Cardiac Arrest Patient

Diagnostic Test	Purpose
Comprehensive metabolic panel	Detecting metabolic and electrolyte derangements and assessing acute renal injury
Serial lactate	Monitoring perfusion status (decreasing lactate levels suggest improved perfusion)
Serum glucose	Detecting hyperglycemia (associated with poor neurological outcomes) and hypoglycemia
Arterial blood gases	Monitoring for acidosis, hypoxia and hypercapnia; tailoring ventilation and oxygenation
Serum cardiac markers	Detecting myocardial infarction
12- or 15-lead ECG	Detecting myocardial infarction, arrhythmias
Chest radiograph	Verifying placement of endotracheal tube; detecting underlying causes of cardiac arrest
Ultrasonography	Detecting underlying causes of cardiac arrest
Echocardiography	Evaluating myocardial stunning and cardiac functioning; detecting ventricular wall abnormalities (suggestive of focal ischemia) and structural abnormalities
Coronary angiography	Evaluating coronary artery disease; diagnosing coronary thrombosis; performing percutaneous coronary intervention (PCI)

Approach to the Patient

The *Post–Cardiac Arrest Care: Adult* Treatment Guideline summarizes the approach to caring for a patient during the immediate post–cardiac arrest period.

Assess and Recognize

Clinical indications of ROSC include a palpable pulse, a measurable blood pressure or an end-tidal carbon dioxide ($ETCO_2$) value greater than 40 mmHg. Upon recognizing ROSC, the team conducts a primary assessment and performs initial interventions, including:

- Establishing cardiac monitoring as needed.
- Establishing pulse oximetry as needed.
- Establishing capnography as needed. Capnography and arterial blood gases are used to guide ventilatory management.
- Establishing noninvasive blood pressure monitoring or arterial pressure monitoring to assist in managing hemodynamics.
- Obtaining a 12- or 15-lead ECG and blood samples for laboratory testing expediently.

Care

Interventions during the immediate post–cardiac arrest period focus on ensuring optimal ventilation and oxygenation, managing hemodynamics, addressing underlying causes, identifying and treating myocardial infarction and promoting neurologic recovery (Figure 9-2).

Optimizing Ventilation and Oxygenation

Provide high-flow supplemental oxygen until the oxygen saturation can be measured, and then provide the minimal level of supplemental oxygen needed to maintain an oxygen saturation of at least 94%. Consider placement of an advanced airway. If the need for long-term ventilatory support is anticipated, mechanical ventilation may be initiated at this time. ROSC is evidenced by a spiked and elevated $ETCO_2$ reading on capnography, indicating that the patient is acidotic. Support ventilations, starting at a rate of 10 breaths per minute and adjusting as needed to keep the $ETCO_2$ between 35 and 40 mmHg and the $PaCO_2$ between 40 and 45 mmHg. Avoid hyperventilation. Hypocapnia can decrease cerebral blood flow and worsen neurological outcomes. Continuously monitor the patient using capnography and pulse oximetry to ensure optimal ventilation and oxygenation.

Managing Hemodynamics

Blood pressure can be extremely labile during the post–cardiac arrest period. Current recommendations suggest that hypotension should be treated when the systolic blood pressure is less than 90 mmHg or the mean arterial pressure (MAP) is less than 65 mmHg. Treat hypotension by administering an IV fluid bolus of 1 to 2 liters of normal saline or lactated Ringer's solution

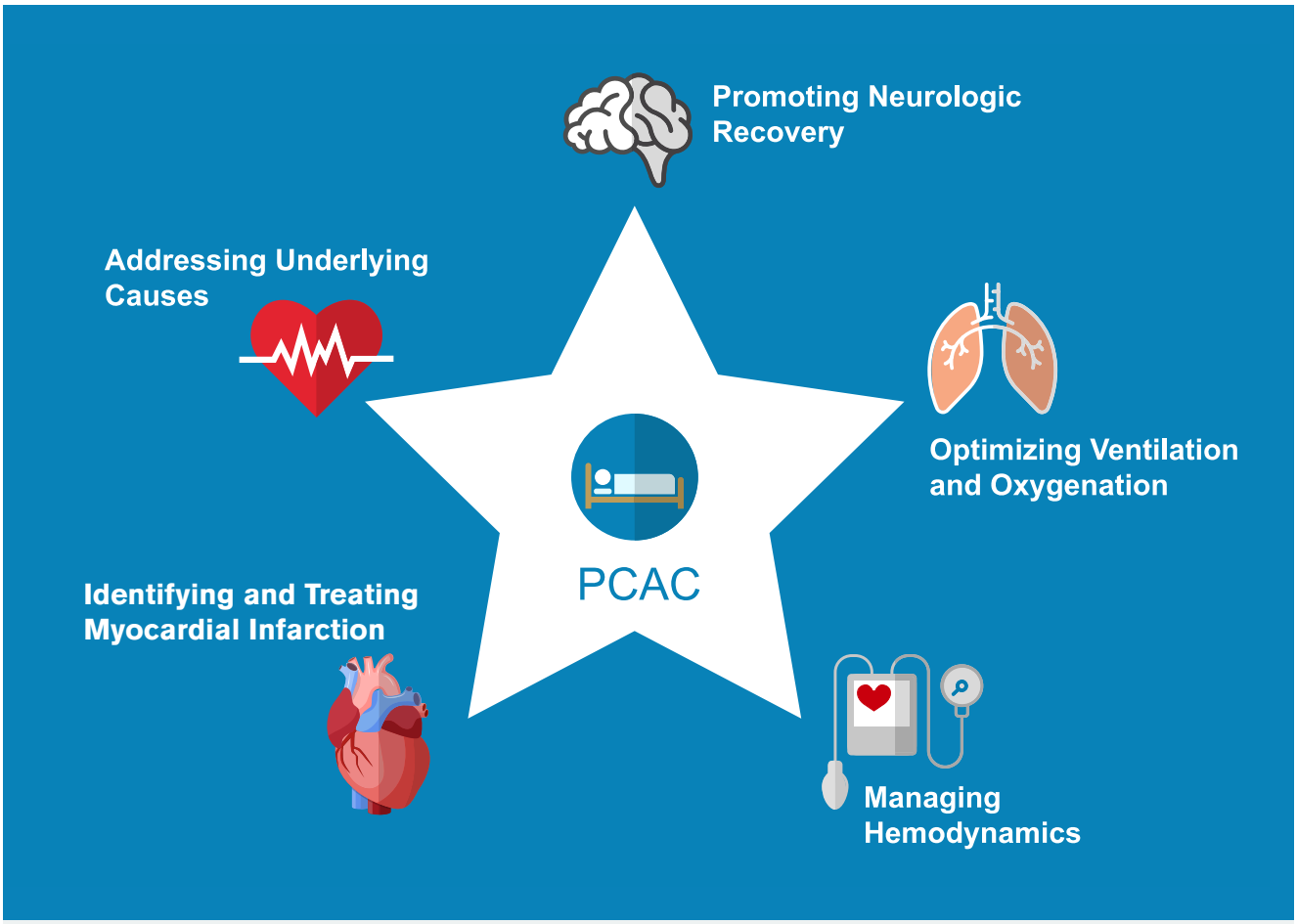


Figure 9-2 | During the immediate post–cardiac arrest period, interventions are focused on ensuring optimal ventilation and oxygenation, managing hemodynamics, addressing underlying causes, identifying and treating myocardial infarction and promoting neurologic recovery.

and initiating a vasopressor infusion (Table 9-2). Once vasopressor therapy is initiated, titrate the drug infusion rate according to hemodynamic parameters and physical examination findings. Identify and treat underlying causes (Hs and Ts; see Chapter 8).

Identifying and Treating Myocardial Infarction

Correction of cardiac instability may improve neurological outcomes. Emergency coronary angiography is recommended for all patients, awake or comatose, who have ECG and laboratory findings suggestive of acute myocardial infarction (i.e., ST-segment elevation myocardial infarction [STEMI] or non–ST-segment myocardial infarction [NSTEMI]). Patients with STEMI require immediate reperfusion therapy with percutaneous coronary intervention (PCI), fibrinolytic therapy or both (see Chapter 10). Reperfusion therapy can be initiated in patients who are comatose and concurrently with targeted temperature management (TTM).

Table 9-2 | Vasopressor Infusion for the Post–Cardiac Arrest Patient

Vasopressor	Dosage
Epinephrine	0.1 to 0.5 mcg/kg/min IV/IO
Norepinephrine	0.1 to 0.5 mcg/kg/min IV/IO
Dopamine	5 to 10 mcg/kg/min IV/IO

Promoting Neurologic Recovery

Neurological Thermoprotection

Targeted temperature management (TTM) should be considered for all patients who remain comatose after ROSC, as indicated by an inability to follow verbal commands. TTM may reduce global oxygen demand and improve overall outcomes after cardiac arrest.

In TTM, a target temperature between 32° C (89.6° F) and 36° C (96.8° F) is established, the patient's body

temperature is maintained at the targeted temperature for at least 24 hours, and then the patient's body temperature is slowly brought back up at a rate of 0.25° C per hour. Patient-specific factors should be considered when determining the target temperature; for example, a temperature at the lower end of the range may be preferred for patients with seizures or cerebral edema. Various methods of inducing hypothermia may be used, including administering an ice-cold IV fluid bolus (30 mL/kg), using endovascular catheters or employing surface cooling strategies (e.g., cooling blankets, ice packs). The patient's core body temperature should be continuously monitored throughout therapy using an esophageal thermometer, a bladder catheter (in anuric patients) or a pulmonary artery catheter (if one is in place).

Practice Note

Providers should not initiate TTM in the prehospital setting.

Moderate Glycemic Control

Glycemic control is important, although optimal strategies for managing glucose concentrations are unknown. Tight blood glucose control has been associated with hypoglycemic episodes that may be harmful. Follow your facility's protocol with regard to glucose targets and insulin infusions in the post–cardiac arrest patient. In general, hypoglycemia (blood glucose < 80 mg/dL) should be treated with dextrose, and hyperglycemia should be treated with insulin to target a blood glucose level in the range of 144 to 180 mg/dL.

POST–CARDIAC ARREST CARE: ADULT

Post–cardiac arrest (ROSC)

Conduct primary assessment and perform initial interventions*
- Establish (if not already done) cardiac monitoring, pulse oximetry, capnography and noninvasive blood pressure monitoring or arterial pressure monitoring
- Obtain a 12- or 15-lead ECG and blood samples for laboratory testing expediently

Optimize ventilation and oxygenation
- Provide minimal level of supplemental oxygen needed to maintain saturation of ≥ 94%
- Consider advanced airway
- Support ventilations starting at a rate of 10 per minute and adjusting as necessary to keep carbon dioxide levels in physiologic range ($ETCO_2$ between 35 and 40 mmHg and $PaCO_2$ between 40 and 45 mmHg)
- Continuously monitor with capnography and pulse oximetry

Manage hemodynamics
- Monitor with noninvasive blood pressure monitoring or arterial pressure monitoring
  - Maintain SBP > 90 mmHg
- If SBP < 90 mmHg or MAP < 65 mmHg:
  - Administer an IV fluid bolus (1 to 2 L NS or LR solution)
  - Initiate a vasopressor infusion (titrate to effect)
- Identify and treat underlying causes (Hs and Ts)

Clinical suspicion of STEMI or suspected acute myocardial infarction

Yes → **Initiate coronary reperfusion therapy**

No → **Able to follow verbal commands**

Yes → **Admit to critical care unit for ongoing care and monitoring**

No → **Initiate TTM†**

Targeted Temperature Management†

Maintain core body temperature 32° C to 36° C for at least 24 hours
Methods include: • Ice-cold IV fluid bolus (30 mL/kg) • Endovascular catheters • Surface-cooling strategies (e.g., cooling blankets, ice packs)
Continuously monitor core temperature via esophageal thermometer, bladder catheter (in anuric patients) or pulmonary artery catheter (if one is in place)

Hs	Ts
• Hypovolemia • Hypoxemia • Hydrogen ion excess (acidosis) • Hyper-/hypokalemia • Hypothermia • Hyper-/hypoglycemia	• Tamponade • Tension pneumothorax • Thrombosis (pulmonary embolism) • Thrombosis (myocardial infarction) • Toxins

Vasopressor Infusion

Epinephrine	0.1 to 0.5 mcg/kg/min IV/IO
Dopamine	5 to 10 mcg/kg/min IV/IO
Norepinephrine	0.1 to 0.5 mcg/kg/min IV/IO

*As time and resources permit, use standard glucose management protocols, obtain a medical history and perform focused physical and neurologic examinations.

†Providers should not initiate targeted temperature management in the prehospital setting.

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CHAPTER 10

Acute Coronary Syndromes

Introduction

Acute coronary syndromes (ACS) is a general term for a group of life-threatening conditions that occur because of a sudden reduction in blood flow to the heart. Each year in the United States, an estimated 660,000 people require hospitalization for myocardial infarction related to coronary artery disease. Quickly identifying and triaging patients with ACS is critical because ACS carries a significantly increased risk of death and complications associated with myocardial ischemia.

Classification of Acute Coronary Syndromes

Based on 12-lead ECG findings, the ischemic conditions that comprise ACS can be subdivided into two categories (Figure 10-1):

- STEMI, or ST-segment elevation myocardial infarction, which is characterized by new ST-segment elevation that suggests myocardial infarction.
- NSTE-ACS, or non–ST-segment elevation ACS, which is characterized by ST-segment depression, T-wave inversion or transient T-wave elevation that suggests non–ST-segment elevation myocardial infarction (NSTEMI) or unstable angina (UA). NSTEMI and UA are similar conditions that differ in severity. Patients with ECG findings consistent with NSTE-ACS who have elevated serum cardiac markers are considered to have NSTEMI. Those who do not have elevated serum cardiac markers and who have low risk-stratification scores may be considered to have UA.

Goals for Management

When ACS is determined to be the cause of a patient's chest pain or discomfort, the goals for management are to:

- Identify those patients with STEMI and determine candidacy for early reperfusion therapy with either **percutaneous coronary intervention (PCI)** or **fibrinolytic therapy**.
- Relieve ischemic chest pain and discomfort.
- Manage complications, such as ischemia-induced arrhythmias that can lead to cardiac arrest and death.
- Determine and manage risk for major adverse cardiac events, including death, new or recurrent myocardial infarction or severe recurrent ischemia requiring urgent revascularization.

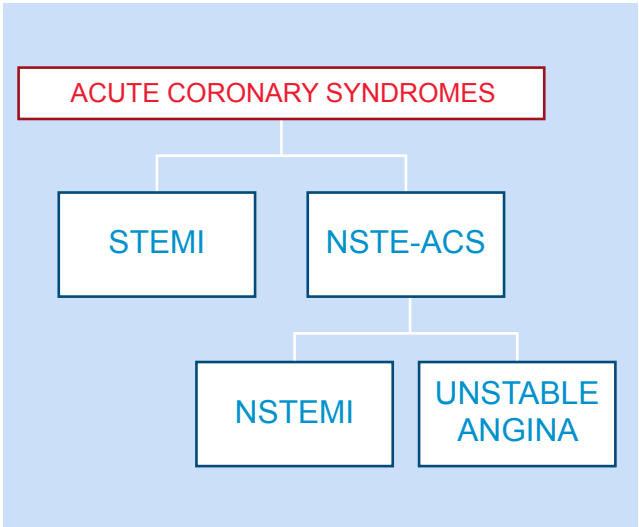


Figure 10-1 | Acute coronary syndromes (ACS) are subdivided into two main categories, ST-segment elevation myocardial infarction (STEMI) and non–ST-segment elevation ACS (NSTE-ACS). NSTE-ACS can be further divided into non–ST-segment elevation myocardial infarction (NSTEMI) and unstable angina (UA).

Pathophysiology of Acute Coronary Syndromes

The underlying cause of ACS is a sudden reduction in the blood supply to the myocardium. In most cases, the precipitating cause of the reduced blood supply is rupture of a vulnerable atherosclerotic plaque. Disruption of the lipid-laden plaque results in transient platelet aggregation and the release of vasoactive substances from the diseased endothelium, causing vasospasm, plaque erosion and eventually the development of an occlusive intracoronary thrombus. The thrombus and associated inflammatory changes lead to partial or complete occlusion of the coronary artery, producing a spectrum of ACS (Figure 10-2). In about 70% of patients who present with ACS each year, arterial occlusion results in myocardial ischemia without persistent ST-segment elevation (UA or NSTEMI). In the other approximately 30% of patients who present with ACS, arterial occlusion results in STEMI.

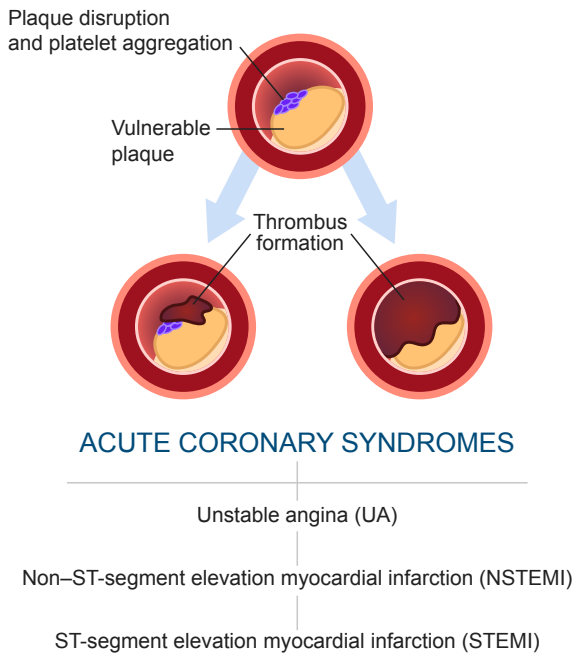


Figure 10-2 | Pathogenesis of acute coronary syndromes (ACS)

STEMI Chain of Survival

The STEMI Chain of Survival illustrates how a coordinated effort among members of the community, prehospital providers and in-hospital providers can increase the patient's likelihood of surviving STEMI (Figure 10-3).

Early Recognition and Activation of the Emergency Response System

Early recognition of signs and symptoms along with activation of emergency medical services (EMS) is the first link in the STEMI Chain of Survival.

Rapid Dispatch, Rapid Transport and Prearrival Notification

The second link in the STEMI Chain of Survival entails rapid dispatch of EMS personnel to the patient's location, rapid transport of the patient to a facility capable of administering reperfusion therapies and notification of the receiving facility in advance of the patient's arrival. During this stage, prehospital providers initiate diagnostic and therapeutic measures, such as obtaining a 12- or 15-lead ECG, administering oxygen and medications and completing a fibrinolytic checklist.

Rapid In-Hospital Assessment and Diagnosis

After the patient arrives at the receiving facility, providers work to obtain a definitive diagnosis and initiate appropriate care.

Rapid Treatment

Reperfusion therapy in the form of PCI or fibrinolytic therapy is recommended for all patients with STEMI who present within 12 hours of symptom onset.

- **PCI**. When the receiving facility is equipped to perform PCI, the procedure should be performed within 90 minutes of the patient's first medical contact. If transfer to a facility that is capable of performing PCI is necessary, PCI should be performed within 120 minutes of the patient's first medical contact. As the time from first medical contact to PCI increases, so does the mortality rate.
- **Fibrinolytic therapy**. When reperfusion will be achieved through fibrinolysis, infusion of fibrinolytic agents should be initiated within 30 minutes of the patient's first medical contact.

Recognizing Acute Coronary Syndromes

Signs and symptoms of ischemia, especially in a patient with risk factors for coronary artery disease, should raise suspicion for ACS. The patient's clinical presentation and results from diagnostic tests (including the 12- or 15-lead ECG and serum cardiac marker testing) aid in

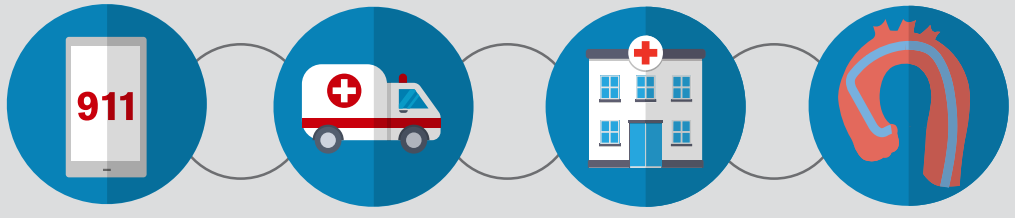


Figure 10-3 | STEMI Chain of Survival

diagnosing ACS, assessing the patient's risk for major adverse cardiac events and determining a management strategy.

Clinical Presentation

ACS should initially be considered in all patients who present with chest pain or discomfort or other signs or symptoms of ischemia or infarction (Figure 10-4). Patients may report retrosternal pressure, squeezing, tightness, aching or heaviness that may radiate to one or both arms or shoulders, the back, the neck, the jaw or the epigastric region. The pain or discomfort is persistent, lasting longer than 3 to 5 minutes, and may be intermittent.

The pain or discomfort may or may not be accompanied by other symptoms, including:

- Dizziness, light-headedness or syncope.
- Sudden, unexplained dyspnea, which may occur in the absence of chest pain or discomfort.
- Nausea or vomiting.
- Pale, ashen or slightly cyanotic skin, especially on the face and fingers.
- Diaphoresis.
- Anxiety or a feeling of impending doom.
- Extreme fatigue.
- Loss of consciousness.

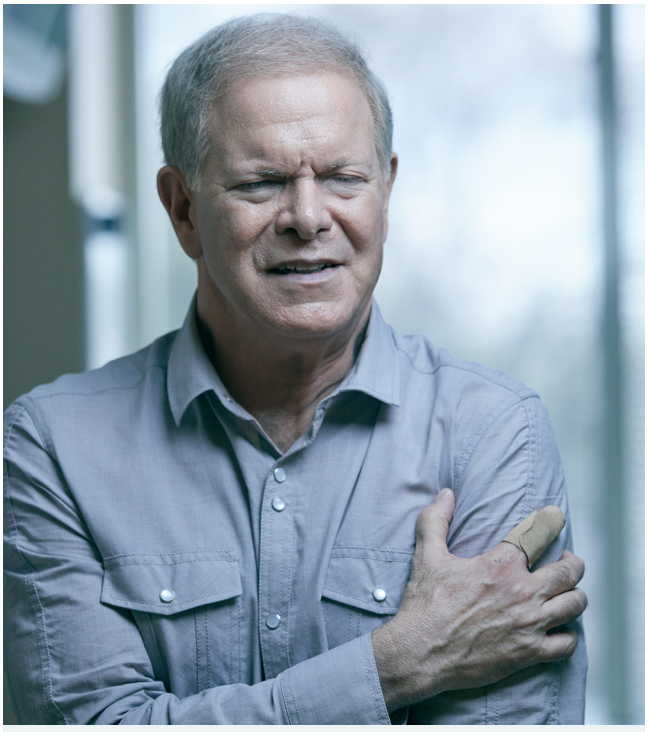


Figure 10-4 | Pain or discomfort is a common symptom associated with ACS.

Women, patients younger than 40 years or older than 75 years, and those with medical conditions such as diabetes may present with atypical symptoms of ischemia or infarction. For example, patients with diabetes may experience ischemia without pain ("silent ischemia").

ECG Findings

A 12- or 15-lead ECG is an essential component of the evaluation of a patient presenting with chest pain (Figure 10-5). ECG findings can support a diagnosis of ACS. In addition, in patients thought to have ACS, ECG findings can be used to assign patients to one of three clinical categories, which in turn helps to determine risk and guide treatment decisions. For the purposes of recognizing ACS, ECG changes must be noted in two or more contiguous leads. Normal or nonspecific ECG findings do not rule out the possibility of ACS. For this reason, ECG findings must always be evaluated in the context of the patient's overall clinical presentation.

- **STEMI**. New ST-segment elevation at the J point in leads V_2 and V_3 of at least 0.2 mV (≥ 2 mm) in men older than 40 years, 0.25 mV (≥ 2.5 mm) in men 40 years or younger or 0.15 mV (≥ 1.5 mm) in women is considered diagnostic of STEMI. Alternatively, new ST-segment elevation of at least 0.1 mV (≥ 1 mm) in two or more contiguous leads other than V_2 and V_3 is diagnostic for STEMI, as is new or presumed new left bundle branch block (LBBB). ST-segment depression in reciprocal leads is a common feature of STEMI and in some studies has been associated with a worse prognosis.

Figure 10-5 | ECG findings can support a diagnosis of ACS and assist in guiding treatment decisions.

- **High-risk NSTE-ACS.** Patients with high-risk NSTE-ACS show changes suggestive of ischemia, such as ST-segment depression or T-wave inversion, in two or more contiguous leads. Transient ST-segment elevation, defined as ST-segment elevation of at least 0.05 mV (≥ 0.5 mm) that lasts for less than 20 minutes, may also be seen in patients with high-risk NSTE-ACS.
- **Intermediate- or low-risk NSTE-ACS**. Patients with intermediate- or low-risk NSTE-ACS show nondiagnostic ST-segment or T-wave changes on ECG, or no changes at all. ST-segment deviation less than 0.05 mV (0.5 mm) in either direction or T-wave inversion of 0.2 mV (2 mm) or less may be seen on ECG. These patients require additional risk stratification supported by the results of other diagnostic tests, such as serum cardiac marker testing and functional testing.

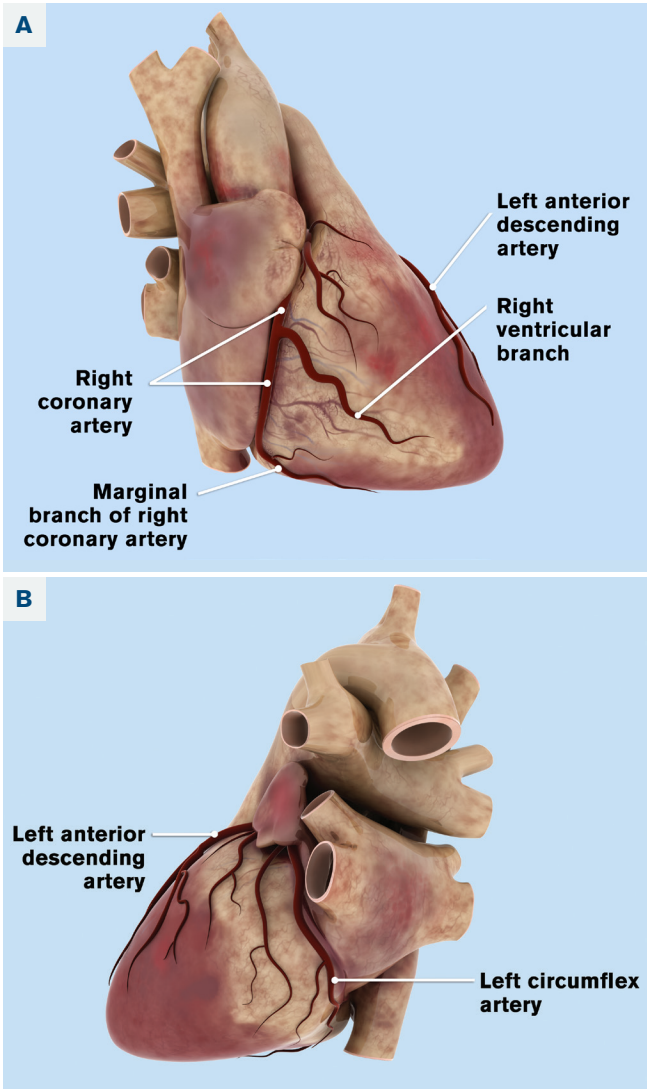


Figure 10-6 | (A) Anterior view of the heart (right ventricle). (B) Posterior view of the heart (left ventricle).

A 15-lead ECG can be used to identify right ventricular infarction or posterior wall myocardial infarction (Figure 10-6). Lead V_{4R} records electrical activity in the right ventricle and leads V_8 and V_9 record left ventricular posterior wall activity.

A 15-lead ECG should be obtained:

- To screen for posterior wall myocardial infarction when ACS is suspected but the 12-lead ECG does not show ST-segment elevation.
- To screen for right ventricular infarction when the 12-lead ECG reveals evidence of inferior wall myocardial infarction (i.e., ST-segment elevation in leads II, III and aVF).
- Whenever there is strong suspicion for posterior wall or right ventricular involvement.

The *Placing Electrodes for Electrocardiography* Skill Sheet (in Chapter 3) provides step-by-step guidance for placing electrodes for a 12-lead and 15-lead ECG.

Serum Cardiac Markers

Levels of serum cardiac markers should be assessed in all cases of suspected ACS. Cardiac troponin T (cTnT) and cardiac troponin I (cTnI) biomarkers are preferred for diagnosing myocardial injury. For diagnosis, it is important to assess both the peak troponin level and changes in troponin level over time, so cardiac troponin levels should be measured at initial presentation and then 3 to 6 hours later. For high-sensitivity markers, some protocols suggest measurement at initial presentation and then 2 hours later. In patients who present with normal troponin levels initially but who show ECG changes suggestive of intermediate- or high-risk NSTE-ACS, an additional cardiac troponin measurement beyond 6 hours may be indicated. In addition to being prognostic (for both short- and long-term outcomes), cardiac troponin levels in combination with risk stratification are useful to guide treatment decisions.

Patient Assessment

Chest pain or discomfort results in approximately 7 million emergency department visits each year. Among these patients, approximately 1.6 million are admitted with a diagnosis of ACS. To expedite appropriate therapy for all patients presenting with chest pain or discomfort, providers must be able to rapidly discern the underlying cause.

Rapid Assessment

On initial impression, some patients with ACS appear acutely ill (e.g., anxious, pale, diaphoretic, dyspneic, in pain). However, others may appear to be in minimal distress or show only subtle signs of discomfort, such as rubbing the upper arm or chest.

Primary Assessment

Conduct a primary assessment following the ABCDE approach and provide initial interventions as needed. Assess vital signs. Establish cardiac monitoring and pulse oximetry. Patients with dyspnea may require supplemental oxygen. Provide the minimal level needed to maintain an oxygen saturation of at least 94%. Obtain vascular access. Obtain a 12-lead ECG within 10 minutes. Be prepared to provide CPR and defibrillation if the patient's condition deteriorates.

Secondary Assessment

The goals of the secondary assessment are to differentiate ACS from other causes of acute chest pain (Box 10-1) and to gather data for risk stratification. Risk-stratification tools such as the HEART (History, ECG, Age, Risk factors, initial Troponin) score (Figure 10-7), Thrombolysis in Myocardial Infarction (TIMI) score and Global Registry of Acute Coronary Events (GRACE) score assign weights to various predictive factors to calculate the patient's risk for experiencing a major adverse cardiac event in the next 1 to 6 months.

History

Key information to elicit during the history includes:

- A full description of the chest pain or discomfort, including provoking and alleviating factors, qualities, where it is located, its onset and duration, its severity and any associated symptoms.
- Symptoms suggestive of heart failure or other complications of ischemia.
- The presence of risk factors for cardiovascular disease.
- Relevant events from the medical history, including a history of cardiovascular or cerebrovascular disease.
- Contraindications to fibrinolysis.

Box 10-1 | Life-Threatening Causes of Acute Chest Pain

- Aortic dissection
- Pulmonary embolism
- Pneumothorax
- Ruptured esophagus
- Perforating peptic ulcer disease

Physical Examination

Physical examination may reveal signs that aid in the differential diagnosis or that assist in identifying the hemodynamic consequences of ischemia. Findings may include signs of left ventricular dysfunction (e.g., hypotension, crackles, weak peripheral pulses) or cardiogenic shock (e.g., cool, clammy skin). Other significant findings may include jugular venous distension, a third or fourth heart sound or murmurs. In some patients with ACS, the physical examination does not reveal anything of significance.

Diagnostic Tests

ACS cannot be diagnosed on the basis of history and physical examination findings alone. Tests that are routinely ordered in the assessment of a patient with possible ACS include a 12- or 15-lead ECG, serum cardiac markers, a serum electrolyte panel and coagulation studies. A chest radiograph is typically obtained at presentation to rule out other causes of acute chest pain and to identify pulmonary congestion (a negative prognostic factor in patients with ACS).

Approach to the Patient

Prompt evaluation and intervention for all patients with suspected ACS is critical to limit ischemic damage to the myocardium and prevent death. The *Acute Coronary Syndromes: Adult* Treatment Guideline summarizes the approach to a patient with ACS.

Assess and Recognize

When ACS is suspected, the team takes actions to ensure adequate airway, breathing and circulation, including establishing cardiac monitoring and pulse oximetry, administering supplemental oxygen if needed and ensuring vascular access.

Additionally, the team works to gather information that will aid in definitive diagnosis, risk stratification and treatment decisions. Actions include:

- Obtaining a 12-lead ECG (and 15-lead ECG, if necessary) within 10 minutes of the patient's arrival at the facility.
- Ordering serum cardiac marker, electrolyte and coagulation studies within 10 minutes of the patient's arrival at the facility.

HEART SCORE FOR CHEST PAIN			
History	Highly suspicious	2	
	Moderately suspicious	1	
	Slightly or nonsuspicious	0	
ECG	Significant ST-segment depression	2	
	Nonspecific repolarization	1	
	Normal	0	
Age	65 years or older	2	
	45 to 64 years	1	
	44 years or younger	0	
Risk Factors ■ Diabetes mellitus ■ Hypercholesterolemia ■ Hypertension ■ Smoking ■ Family history of coronary artery disease ■ Obesity	3 or more risk factors OR a history of coronary artery disease	2	
	1 or 2 risk factors	1	
	0 known risk factors	0	
Troponin	> 3x normal limit	2	
	1–3x normal limit	1	
	≤ normal limit	0	
		Total:	

Figure 10-7 | Risk stratification tools, such as the HEART score, assign weights to various predictive factors to calculate the patient's short-term risk for experiencing a major adverse cardiac event. Adapted with permission from Backus BE, Six AJ, Kelder JC, et al. A prospective validation of the HEART score for chest pain patients at the emergency department. Int J Cardiol. 2013;168(3):2154.

- Obtaining a brief medical history and conducting a focused physical examination.
- Obtaining a chest radiograph within 30 minutes of the patient's arrival at the facility.
- Assessing the patient's risk using a risk-stratification tool.

Practice Note

For patients with suspected STEMI, do not delay activation of the PCI team or initiation of fibrinolytic therapy while awaiting the results of serum cardiac marker or radiographic studies.

Initiate general drug therapy for patients with signs and symptoms of ischemia or infarction as soon as possible after screening for contraindications.

- **Aspirin**. If not already administered by prehospital providers, administer antiplatelet therapy in the form of aspirin (162 to 325 mg) to patients without contraindications (such as aspirin allergy or a history of gastrointestinal bleeding). The patient should be instructed to chew the aspirin. If the oral route is not feasible (e.g., owing to nausea, vomiting, peptic ulcer disease or another upper gastrointestinal tract disorder), consider administering aspirin as a rectal suppository (300 mg).

Practice Note

Except for aspirin, use of nonsteroidal anti-inflammatory agents (NSAIDs) is contraindicated for patients with STEMI.

- **Nitroglycerin**. Administer nitroglycerin (0.4 mg every 5 minutes by sublingual tablet or spray, up to three doses) to relieve ischemic chest pain, unless contraindicated. If the pain persists, consider intravenous nitroglycerin.

Practice Note

Nitroglycerin must be used with caution or not at all in certain situations. Conditions that may preclude the use of nitroglycerin include inferior wall myocardial infarction with right ventricular involvement, hypotension (systolic blood pressure < 90 mmHg), significant bradycardia (heart rate < 50 bpm), tachycardia and use of phosphodiesterase inhibitors within the previous 24 to 48 hours. Nitroglycerin is a vasodilator and its administration under these conditions may prevent the patient from maintaining adequate cardiac output and blood pressure.

- **Morphine**. Consider morphine (1 to 5 mg) for patients who continue to experience chest pain despite antianginal therapy.

Practice Note

Morphine should not be administered to hypotensive patients or those with right ventricular infarction. Additionally, morphine should be used with caution in patients with NSTE-ACS because its use in these patients has been associated with increased mortality.

Care

Assigning the patient to a clinical category (STEMI, high-risk NSTE-ACS or intermediate- or low-risk NSTE-ACS) helps to guide management decisions.

STEMI

Treatment for STEMI depends on how much time has passed since symptom onset. Patients who present within 12 hours of symptom onset should receive early reperfusion therapy (PCI or fibrinolysis) and adjuvant drug therapy. Those who present more than 12 hours after symptom onset and are found to be at high risk for experiencing a major adverse cardiac event in the next 1 to 6 months may benefit from an early invasive strategy (e.g., coronary angiography with or without revascularization within 24 hours of admission).

Reperfusion Therapy

Reperfusion therapy is the standard of care for all patients with STEMI who are diagnosed within 12 hours of symptom onset and who have no contraindications.

PCI is performed in a cardiac catheterization suite and entails balloon angioplasty, with or without stent placement. When performed at a PCI-capable facility, **primary PCI** (i.e., PCI performed as first-line therapy for STEMI) is associated with better patient outcomes, compared with outcomes following fibrinolytic therapy. If primary PCI cannot be performed within 90 minutes of the patient's first medical contact, fibrinolysis should be provided within 30 minutes of the patient's first medical contact. Fibrin-specific agents (such as recombinant tissue plasminogen activator [rtPA], reteplase and tenecteplase) are preferred over non–fibrin-specific agents (such as streptokinase). Following fibrinolysis, angiography and PCI may be performed within 3 to 24 hours, if indicated.

Adjuvant Drug Therapy

In addition to reperfusion therapy, anticoagulant and antiplatelet therapies are initiated to inhibit the formation or recurrence of intracoronary thrombi, and medications may be administered to support cardiac function (Box 10-2).

High-Risk NSTE-ACS

An early invasive strategy should be considered for patients with NSTE-ACS who have elevated troponin levels or high risk-stratification scores. Indications for an early invasive strategy include:

- Refractory ischemic chest discomfort.
- Recurrent or persistent ST-segment deviation.
- Ventricular tachycardia.
- Hemodynamic instability.
- Signs or symptoms of heart failure.
- A score indicating high risk on a risk-stratification tool.

Provide adjuvant drug therapies as indicated and consider expert consultation.

Intermediate- or Low-Risk NSTE-ACS

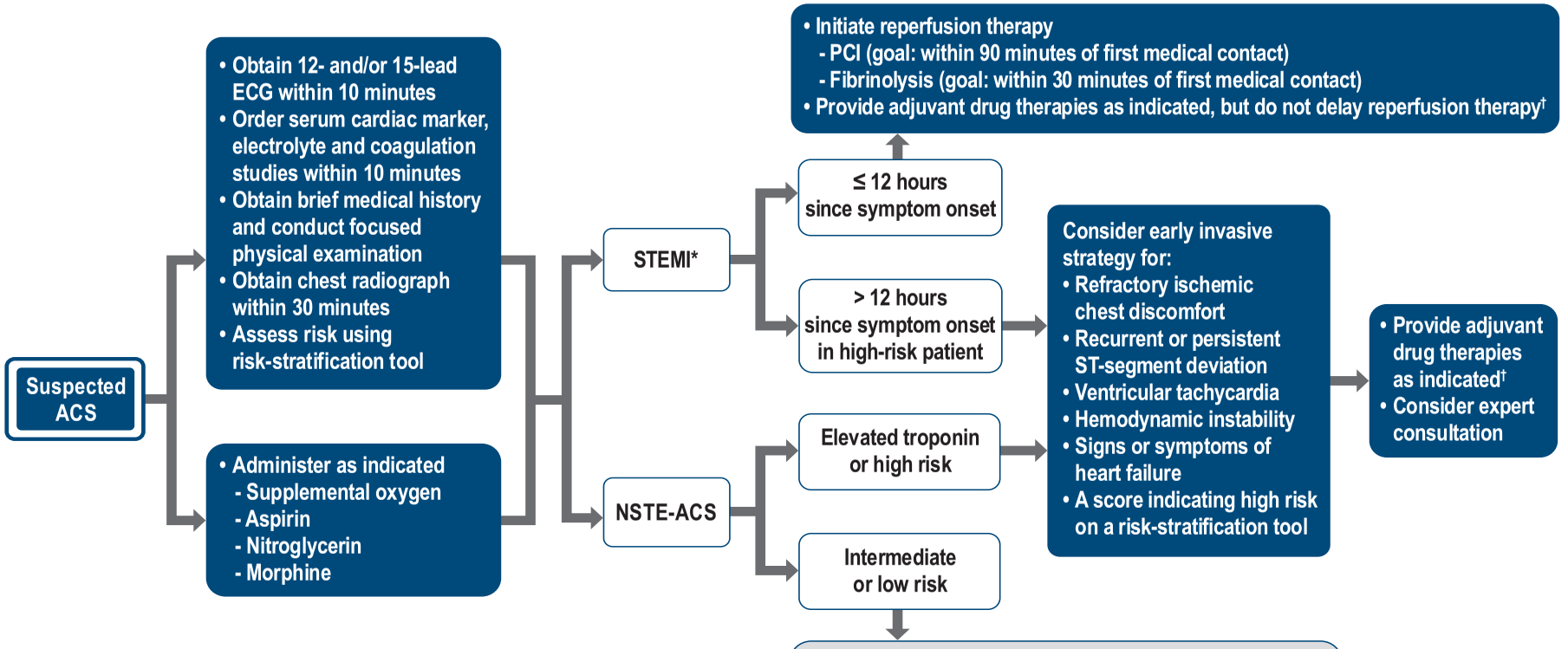
For patients with intermediate- or low-risk NSTE-ACS, consider admission to a chest pain unit or other monitored bed. The HEART score can be a useful adjunct to clinical judgment when deciding whether to admit or discharge a patient with suspected

intermediate- or low-risk NSTE-ACS. For those patients who are admitted, monitor for the development of ischemia through the use of serial serum cardiac markers and repeat ECG or continuous ST-segment monitoring. Noninvasive imaging and functional testing may also be indicated.

Box 10-2 | Adjuvant Drug Therapy

- **$P2Y_{12}$ platelet receptor inhibitors**. Three $P2Y_{12}$ platelet receptor inhibitors—clopidogrel, ticagrelor and prasugrel—are approved for treatment of patients with ischemic myocardial disorders who are unable to tolerate aspirin because of an allergy or gastrointestinal disease.
- **Glycoprotein IIb/IIIa inhibitors**. Intravenous glycoprotein IIb/IIIa inhibitors are recommended for use in patients with ACS who are treated with an early invasive strategy and dual antiplatelet therapy and who have intermediate- or high-risk features (such as positive serum cardiac markers). Tirofiban and eptifibatide are considered the preferred agents. Abciximab may be considered for patients undergoing primary PCI.
- **Heparin.** Unfractionated or low molecular weight heparin may be used for anticoagulation therapy following fibrinolytic therapy or PCI. Incorrect dosing or monitoring of heparin in patients with STEMI has been associated with intracerebral bleeding and hemorrhage.
- **Bivalirudin**. Bivalirudin is a direct thrombin inhibitor that may be used as an alternative to combination therapy with heparin and a glycoprotein IIb/IIIa inhibitor for anticoagulation following PCI.
- **β-Blockers**. Treatment with β-blockers should be initiated within 24 hours of presentation unless there are contraindications (e.g., acute heart failure, low cardiac output, risk for cardiogenic shock).
- **Intravenous nitroglycerin**. In patients with STEMI, intravenous nitroglycerin may be used when chest pain or discomfort is recurrent or refractory to nitroglycerin administered sublingually or by spray. STEMI that is complicated by pulmonary edema or hypertension may also warrant the use of intravenous nitroglycerin. When used to relieve refractory chest discomfort, titrate to maintain a systolic blood pressure of 90 mmHg or more (or 30 mmHg below baseline in patients with hypertension). When used to improve pulmonary edema or hypertension, titrate to maintain a systolic blood pressure that is 10% less than the baseline pressure in patients with blood pressure in the normal range, and 30 mmHg below baseline in patients with hypertension.

ACUTE CORONARY SYNDROMES: ADULT



Please see reverse side for additional information

Medication Administration	
Aspirin	162 to 325 mg aspirin, chewed (if not previously taken or contraindicated); if PO route not feasible consider rectal suppository (300 mg)
Nitroglycerin	0.4 mg SL every 5 minutes up to 3 times
Morphine	1 to 5 mg IV only if discomfort not relieved by nitroglycerin

*Complete fibrinolytic checklist if necessary.
†See *Adjuvant Drug Therapies* table, on reverse side.

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ACUTE CORONARY SYNDROMES: ADULT CONTINUED

Clinical Presentation of Acute Coronary Syndromes

Consider in all patients presenting with chest pain or discomfort:

- Retrosternal pressure, squeezing, tightness, aching or heaviness
- May radiate to one or both arms or shoulders, the back, neck, jaw or epigastric region
- Persistent (more than 3 to 5 minutes); may be intermittent

Other possible signs and symptoms:

- Dizziness, light-headedness or syncope
- Sudden, unexplained dyspnea, which may occur without chest pain or discomfort
- Nausea or vomiting
- Pale, ashen or slightly cyanotic skin, especially on the face and fingers
- Diaphoresis
- Anxiety or a feeling of impending doom
- Extreme fatigue
- Loss of consciousness

Note: Women, patients < 40 years or > 75 years, and those with medical conditions may present with atypical symptoms of ischemia (e.g., patients with diabetes may experience ischemia without pain, or "silent ischemia").

Adjuvant Drug Therapies

Drug Class	Use
$P2Y_{12}$ platelet receptor inhibitors	For patients who are unable to tolerate aspirin because of an allergy or gastrointestinal disease
Glycoprotein IIb/IIIa inhibitors	For patients who are treated with an early invasive strategy and dual antiplatelet therapy and who have intermediate- or high-risk features (e.g., positive cardiac markers)
Heparin (unfractionated or low molecular weight)	For anticoagulation therapy following fibrinolytic therapy or PCI
Bivalirudin	An alternative to combination therapy with heparin and a glycoprotein IIb/IIIa inhibitor for anticoagulation after PCI
β-Blockers	Initiate within the first 24 hours unless there are contraindications (e.g., acute heart failure, low cardiac output)
Intravenous nitroglycerin	For recurrent or refractory chest pain, pulmonary edema or hypertension accompanying STEMI

ECG Findings in Acute Coronary Syndromes

STEMI	NSTE-ACS	
	High Risk	Intermediate or Low Risk
• New ST-segment elevation at the J point in leads V_2 and V_3 of: - ≥ 0.2 mV (≥ 2 mm) in men > 40 years - ≥ 0.25 mV (≥ 2.5 mm) in men ≤ 40 years - ≥ 0.15 mV (≥ 1.5 mm) in women • New ST-segment elevation ≥ 0.1 mV (≥ 1 mm) in two or more contiguous leads other than V_2 and V_3 • New or presumed new left bundle branch block (LBBB)	• Changes suggestive of ischemia, such as ST-segment depression or T-wave inversion, in two or more contiguous leads • Transient ST-segment elevation ≥ 0.05 mV (≥ 0.5 mm) lasting < 20 minutes	• No ECG changes, or nondiagnostic ST-segment or T-wave changes • ST-segment deviation < 0.05 mV (0.5 mm) in either direction or T-wave inversion ≤ 0.2 mV (2 mm)

Normal or nonspecific ECG findings do not rule out the possibility of acute coronary syndromes. Always evaluate ECG findings in the context of the patient's overall clinical presentation.

CHAPTER 11

Stroke

Introduction

Among American adults, stroke is the fifth-leading cause of death and is a leading cause of long-term disability. Each year approximately 795,000 Americans have a stroke, which equates to about one stroke occurring every 40 seconds. Among people experiencing a stroke, one third will die, one third will have long-term disability and one third will recover with minimal or no disability. Early management of acute stroke is included in ALS training because, just as with cardiac emergencies, time is a critical component of treatment. Timely recognition, assessment and management of acute stroke can minimize brain injury and improve patient outcomes.

Overview of Stroke

A stroke is a sudden neurologic deficit that occurs because of impaired blood flow to part of the brain. Stroke is characterized by:

- A sudden onset of signs and symptoms.
- Primary involvement of the central nervous system.
- A vascular cause.

There are two main types of stroke: ischemic and hemorrhagic.

Ischemic Stroke

Ischemic stroke occurs when a blood vessel carrying blood to the brain becomes obstructed. Ischemic strokes account for about 87% of all strokes. Ischemic stroke is classified as thrombotic or embolic.

- A **thrombotic stroke** is most often caused by rupture of an atherosclerotic plaque in a cerebral artery, resulting in the formation of a thrombus that occludes the artery. Patients frequently have a history of hypercholesterolemia and atherosclerosis. Large vessel thrombosis, which occurs in the larger arteries of the brain, is the most common form of thrombotic stroke and is associated with a poor prognosis. Lacunar infarction occurs when one of the small, deep penetrating arteries of the brain becomes obstructed and is associated with a more favorable prognosis.
- An **embolic stroke** occurs when a plaque fragment or blood clot forms elsewhere within the circulatory system and travels to the cerebral circulation. Often the source of the embolus is a blood clot that forms in the heart or the large arteries in the upper chest or neck. Between 15% and 20% of embolic strokes are associated with atrial fibrillation.

For acute ischemic stroke, the goal of treatment is to relieve the obstruction and restore blood flow to the brain tissue. Acute ischemic stroke can be treated with fibrinolytic therapy, endovascular therapy, or both, but the window for treatment is narrow.

- Fibrinolytic therapy is ideally administered as soon as possible and within 3 hours of symptom onset, although this time frame may be extended to 4.5 hours for some patients.
- Endovascular therapy is ideally administered as soon as possible and within 6 hours of symptom onset, although this time frame may be extended to 24 hours for some patients.

Hemorrhagic Stroke

Hemorrhagic stroke occurs when a weakened blood vessel in the brain ruptures and pressure from the bleeding damages the brain tissue. This type of stroke is frequently caused by hypertension or aneurysms. Hemorrhagic strokes are less common than ischemic strokes, accounting for about 13% of all strokes, but are responsible for about 40% of all stroke-related deaths. Hemorrhagic strokes can be classified as intracerebral or subarachnoid.

- **Intracerebral hemorrhage**, the most common form of hemorrhagic stroke, occurs when an artery located within the brain bursts, causing bleeding into the surrounding brain tissue. Causes of intracerebral hemorrhage include **arteriovenous malformation**, anticoagulant therapy and chronic hypertension.
- **Subarachnoid hemorrhage** occurs when a blood vessel located on the surface of the brain ruptures, causing bleeding into the subarachnoid space. This type of hemorrhage is most often caused by a ruptured aneurysm but can also be caused by an arteriovenous malformation, bleeding disorder, head injury or anticoagulant therapy.

Acute Stroke Chain of Survival

The Acute Stroke Chain of Survival (Figure 11-1) illustrates how a coordinated effort among members of the community, out-of-hospital providers and in-hospital providers can optimize outcomes for patients with stroke.

Early Recognition and Activation of the Emergency Response System

Because treatment of acute ischemic stroke is time dependent, early recognition of signs and symptoms of stroke and activation of emergency medical services (EMS) is key.

Rapid Dispatch and Stroke Protocol Activation

The second link in the Acute Stroke Chain of Survival is rapid dispatch and stroke protocol activation. The EMS dispatcher should handle all potential stroke calls as critical. Assistance should be dispatched to these patients with the highest level of priority.

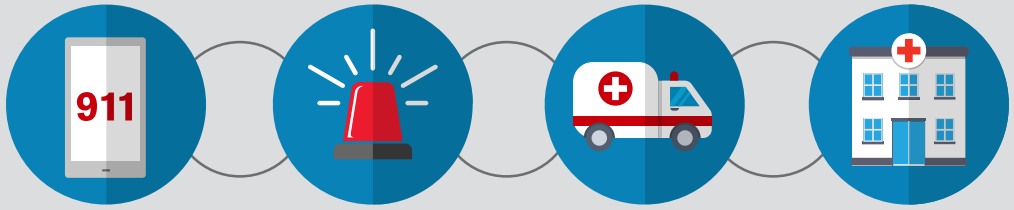


Figure 11-1 | Acute Stroke Chain of Survival

Rapid Transport and Prearrival Notification

Rapid transport and prearrival notification is the third link in the Acute Stroke Chain of Survival. The goal is to efficiently transport the patient to a facility where definitive assessment and treatment can take place, and to ensure that appropriate personnel and resources are assembled in anticipation of the patient's arrival. The receiving facility should be capable of administering reperfusion therapies and providing post-stroke care on a dedicated stroke unit (Box 11-1).

Practice Note

Patients with potential acute ischemic stroke who present to a facility that is not equipped to provide fibrinolytic or endovascular therapy should be treated according to the facility's highest level of care or scope of practice, stabilized as much as possible and then transported to the closest facility with stroke treatment capabilities.

Rapid In-Hospital Assessment, Diagnosis and Treatment

The final link of the Acute Stroke Chain of Survival is rapid in-hospital assessment, diagnosis and treatment. Time frames have been established for these activities to take place for a patient with acute ischemic stroke (Figure 11-2).

- Within 10 minutes of the patient's arrival, the stroke team is activated, blood glucose is measured and treated if necessary, an initial neurologic screening assessment is completed and an urgent brain computed tomography (CT) or magnetic resonance imaging (MRI) scan is ordered. Additionally, laboratory studies and a 12-lead ECG are obtained.
- Within 20 minutes of the patient's arrival, the CT or MRI scan is performed, a focused history is obtained and a more comprehensive neurologic assessment using the National Institutes of Health Stroke Scale (NIHSS) or similar tool is completed.
- Within 45 minutes of the patient's arrival, the CT or MRI scan is interpreted.
- Within 45 to 60 minutes of the patient's arrival, fibrinolytic therapy is initiated for patients with ischemic stroke who have no contraindications.
- Within 3 hours of the patient's arrival, the patient is admitted to a monitored bed on a dedicated stroke unit or critical care unit.

Practice Note

If a patient with suspected stroke presents to a facility that does not have a dedicated stroke unit, protocols should be in place to expedite patient transfer to the nearest hospital capable of providing a higher level of stroke care and post-treatment monitoring. If possible, the receiving facility should initiate fibrinolytic therapy before transferring the patient to a facility capable of providing a higher level of stroke care and post-treatment monitoring. If the receiving facility is not equipped to administer fibrinolytic therapy, the patient should be immediately transfered to a primary or comprehensive stroke center.

The 8 Ds of Stroke Care

The "8 Ds of Stroke Care" highlight critical steps in the management of a patient with stroke (Box 11-2). A delay in carrying out any of these critical actions can be detrimental to the patient.

Box 11-1 | Stroke Care Facilities

Every hospital that provides emergency services should have a written document detailing its capabilities and protocols for caring for patients with acute stroke and should share this information with the public and with the local emergency medical services (EMS) system. This document should identify areas of expertise in stroke care; identify capabilities with regard to neuroimaging, administering reperfusion therapies and providing post-stroke care; and establish criteria and protocols for patient transfer or direct transport to a higher level of care.

For hospitals that hold Joint Commission accreditation, the Joint Commission offers four levels of stroke program certification.

- **Acute stroke–ready hospitals**: These hospitals do not possess dedicated stroke units but have access to providers with expertise in diagnosing and treating acute ischemic stroke with fibrinolytic therapy 24 hours a day, 7 days a week. Additionally, these hospitals have transfer agreements with local primary or comprehensive stroke centers.
- **Primary stroke centers**: These hospitals have staff with neurological expertise dedicated to stroke care and are capable of administering fibrinolytic therapy. These facilities must have a dedicated stroke unit. Additionally, these hospitals have transfer agreements with local comprehensive stroke centers.
- **Thrombectomy-capable stroke center**: Like primary stroke centers, these hospitals have staff with extensive neurological expertise dedicated to stroke care and are capable of administering fibrinolytic therapy, but thrombectomy-capable stroke centers must also offer mechanical thrombectomy. These facilities must have dedicated neurointensive care beds for complex stroke patients on site and available 24 hours a day, 7 days a week. Additionally, these hospitals have transfer agreements with local comprehensive stroke centers.
- **Comprehensive stroke center**: These hospitals have staff with extensive neurological expertise dedicated to stroke care and the ability to administer fibrinolytic therapy and perform mechanical thrombectomy, endovascular coiling and microsurgical clipping procedures for aneurysms.

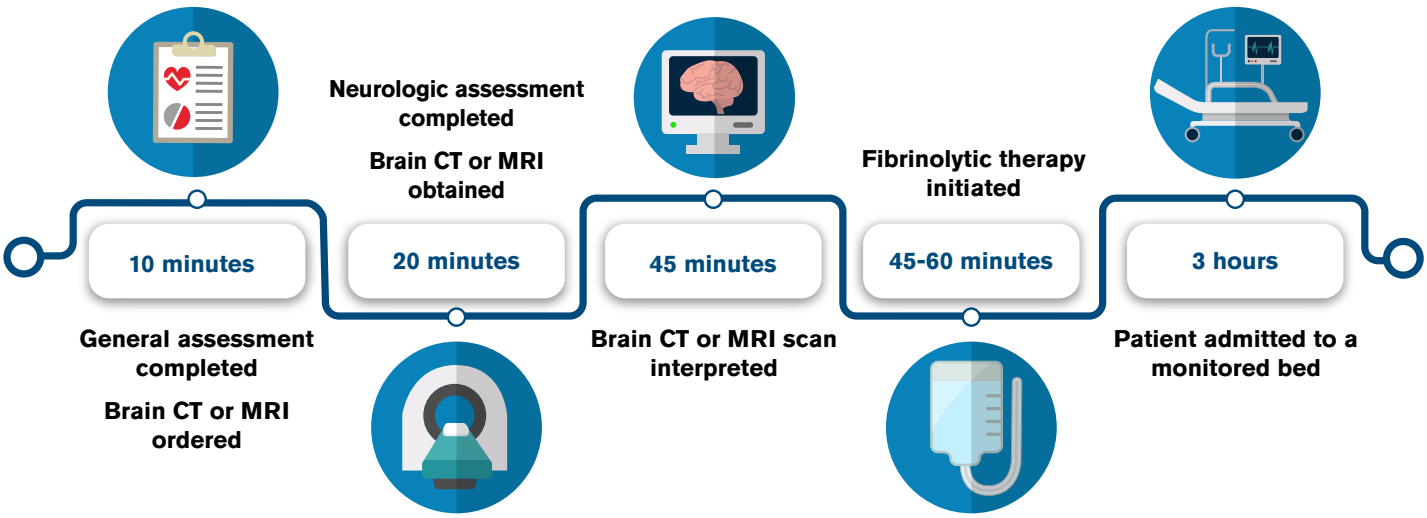


Figure 11-2 | Critical time periods for in-hospital assessment and management of acute ischemic stroke

Box 11-2 | The 8 Ds of Stroke Care

- **Detection:** Early recognition of the signs and symptoms of stroke
- **Dispatch:** Early activation of, and response by, emergency medical services (EMS) personnel
- **Delivery:** Prompt transport of the patient to a facility capable of providing acute stroke care, with advanced notification to the receiving facility
- **Door:** Immediate triage by receiving staff
- **Data:** Prompt collection of the data necessary to inform treatment decisions, including medical history and physical examination data and data obtained through laboratory and imaging studies
- **Decision:** Prompt and expert evaluation of the data to inform treatment decisions
- **Drug/device:** Administration of fibrinolytic therapy, endovascular therapy or both within recommended time frames
- **Disposition:** Admission to a dedicated stroke unit or critical care unit

Box 11-3 | Cincinnati Prehospital Stroke Scale (CPSS)

An abnormal finding in any one of the following three areas is associated with a 72% probability of stroke.

Facial Droop (ask patient to show teeth/smile)

- Normal: both sides of face move equally
- Abnormal: one side of the face does not move as well as the other side

Arm Drift (ask patient to close eyes and extend both arms straight out with the palms up for 10 seconds)

- Normal: both arms move the same, or both arms do not move at all
- Abnormal: one arm does not move, or one arm drifts downward as compared with the other

Abnormal Speech (ask patient to say "You can't teach an old dog new tricks")

- Normal: patient uses correct words without slurring
- Abnormal: patient uses incorrect words, slurs words or is unable to speak

Recognizing Stroke

Clinical Presentation

Signs and symptoms of stroke include:

- Sudden weakness, numbness or tingling on one side of the face or body.
- Sudden onset of confusion.
- Sudden difficulty with language, including difficulty speaking, difficulty understanding or garbled speech.
- Sudden vision difficulties in one or both eyes.
- Difficulty with walking, balance or coordination.
- Sudden severe headache.

Stroke Assessment Tools

Cincinnati Prehospital Stroke Scale

A rapid stroke assessment tool, such as the Cincinnati Prehospital Stroke Scale (CPSS), can be used to screen for signs of stroke (Box 11-3). The CPSS screens for three physical indicators of stroke: facial droop (Figure 11-3), arm drift and abnormal speech. An abnormal finding in any one of these areas is associated with a 72% probability of a stroke and is sufficient reason to activate the EMS system.

Miami Emergency Neurologic Deficit Checklist

The Miami Emergency Neurologic Deficit (MEND) checklist assesses three areas (mental status, cranial nerve function and limb function) and provides more detailed information about the severity and location of the stroke (Figure 11-4). It can also be used to establish a baseline of neurologic function for future comparison. The MEND checklist may be completed by prehospital providers or hospital providers.

Rapid Arterial Occlusion Evaluation Scale

The Rapid Arterial Occlusion Evaluation (RACE) scale scores the patient's condition in five areas (facial palsy, arm motor function, leg motor function, head and gaze deviation, and agnosia/aphasia) and is used by prehospital providers to screen for possible large vessel occlusion (Figure 11-5). A score of 4 or greater indicates a high likelihood of large vessel occlusion.

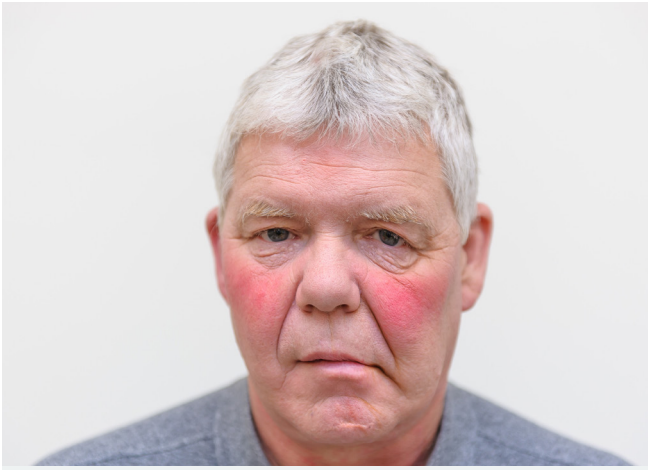


Figure 11-3 | Facial droop

MEND Checklist				
On Scene: *Perform LOC and CPSS (in gray boxes).* ***En route:*** *If time allows, perform the complete MEND exam.*				
Mental Status	**Check if Abnormal**			
	On Scene		**En Route**	
■ Level of Consciousness (AVPU)	☐		☐	
■ Speech "You can't teach an old dog new tricks."	☐		☐	
■ Questions (age, month)			☐	
■ Commands (close, open eyes)	☐			
Cranial Nerves	**R**	**L**	**R**	**L**
■ Facial Droop (show teeth or smile)	☐	☐	☐	☐
■ Visual Fields (four quadrants)			☐	☐
■ Horizontal Gaze (side to side)			☐	☐
Limbs	**R**	**L**	**R**	**L**
■ Motor — Arm and Leg				
○ Arm Drift (close eyes and hold out both arms)	☐	☐	☐	☐
○ Leg Drift (open eyes and lift each leg separately)			☐	☐
■ Sensory — Arm and Leg (close eyes and touch, pinch)			☐	☐
■ Coordination — Arm and Leg (finger to nose, heel to shin)			☐	☐

Figure 11-4 | The Miami Emergency Neurologic Deficit (MEND) checklist. Used with permission from University of Miami, Gordon Center for Research in Medical Education.

National Institutes of Health Stroke Scale

The National Institutes of Health Stroke Scale (NIHSS), which can be used to both assess and quantify deficits, should be administered to all patients with suspected stroke once they arrive at the receiving facility (Figure 11-6). The NIHSS evaluates level of consciousness, visual function, motor function, sensation and neglect, cerebellar function, and language deficits and helps to determine both the location and the severity of the stroke.

Patient Assessment

Rapid Assessment

On initial impression, the patient may appear to be fully alert, have some degree of impaired level of consciousness or appear unresponsive. Other signs that may be immediately apparent include difficulty speaking or facial droop.

Primary Assessment

As with any acutely ill patient, the goal of the primary assessment is to quickly assess the patient's airway, breathing, circulation, disability and exposure and provide care as needed. Establish cardiac monitoring and pulse oximetry. If necessary, provide the minimal level of supplemental oxygen needed to maintain an oxygen saturation of at least 94%. Take care to avoid hyperoxia. Assess perfusion. If the patient is hypotensive, give fluids as needed to maintain adequate perfusion. Obtain vascular access. Expediently perform a neurologic screening assessment using the NIHSS or a similar assessment scale. Place the patient on NPO status until the ability to swallow can be assessed.

Practice Note

Patients with hemorrhagic stroke often require airway and breathing support. Ischemic stroke, unless the area of infarct is large or involves the brain stem, is less often associated with compromise of the airway, breathing or circulation.

Rapid Arterial Occlusion Evaluation (RACE) Scale			
Item	**Instruction**	**Result**	**Score**
Facial Palsy	Ask patient to show their teeth (smile)	**Absent** (symmetrical movement)	0
		Mild (slight asymmetrical)	1
		Moderate to Severe (completely asymmetrical)	2
Arm Motor Function	Extending the arm of the patient 90° (if sitting) or 45° (if supine)	**Normal to Mild** (limb upheld more than 10 seconds)	0
		Moderate (limb upheld less than 10 seconds)	1
		Severe (patient unable to raise arm against gravity)	2
Leg Motor Function	Extending the leg of the patient 30° (in supine)	**Normal to Mild** (limb upheld more than 5 seconds)	0
		Moderate (limb upheld less than 5 seconds)	1
		Severe (patient unable to raise leg against gravity)	2
Head and Gaze Deviation	Observe eyes and head deviation to one side	**Absent** (eye movements to both sides were possible and no head deviation was observed)	0
		Present (eyes and head deviation to one side was observed)	1
Aphasia (right side)	Difficulty understanding spoken or written words. **Ask patient to follow two simple commands.** 1. Close your eyes. 2. Make a fist.	**Normal** (performs both tasks requested correctly)	0
		Moderate (performs only 1 of 2 tasks requested correctly)	1
		Severe (cannot perform either task requested correctly)	2
Agnosia (left side)	Inability to recognize familiar objects. **Ask patient:** 1. "Whose arm is this?" (while showing the affected arm) 2. "Can you move your arm?"	**Normal** (recognizes arm, and attempts to move arm)	0
		Moderate (does not recognize arm or is unaware of arm)	1
		Severe (does not recognize arm and is unaware of arm)	2
*Any score above 4 is a **Stroke Alert** and high likelihood of an LVO*			

Figure 11-5 | The Rapid Arterial Occlusion Evaluation (RACE) scale

Figure 11-6 | A neurologic assessment using the National Institutes of Health Stroke Scale (NIHSS) or a similar assessment tool should be conducted when the patient arrives at the facility.

Secondary Assessment

The goals of the secondary assessment are to establish a diagnosis of ischemic or hemorrhagic stroke, rule out differential diagnoses ("stroke mimics"; Box 11-4), determine the potential underlying cause of the stroke and gather information that will aid in determining candidacy for planned interventions.

Box 11-4 | Common Stroke Mimics

- Seizure
- Migraine
- Toxicity or metabolic disturbance (e.g., drug or alcohol intoxication, hypoglycemia)
- Intracranial tumor or infection
- Somatoform or conversion disorder

History

Key information to elicit during the history includes:

- The time of symptom onset and the events leading up to the onset of signs and symptoms. If the patient was asleep when the stroke occurred, consider the time of onset to be the last time the patient was known to be asymptomatic.
- The presence of risk factors for stroke, arteriosclerosis and cardiac disease.
- Prior occurrences of migraine, seizure, infection, trauma or illicit drug use.
- The presence of comorbid conditions, including hypertension, diabetes and atrial fibrillation.
- The use of medications, including anticoagulants, antiplatelet agents, antihypertensive agents and insulin.
- Relevant events from the past medical history, including recent stroke or transient ischemic attack, myocardial infarction, surgery, trauma or bleeding.

Physical Examination

Conduct a focused physical examination of the head and neck, chest and extremities to identify potential causes of stroke and rule out stroke mimics. The head and neck examination may reveal signs of cardiovascular disease (e.g., bruits, jugular venous distension) or injury as a result of trauma or seizure. The focus of the chest examination is to identify potential cardiac causes of stroke, such as a valve disorder. Examination of the extremities may reveal signs of cardiac disorders or other conditions, such as platelet disorders or coagulopathies.

Additionally, obtain vital signs and repeat the neurologic examination at frequent regular intervals.

Diagnostic Tests

Brain imaging is essential for evaluating patients with suspected acute stroke. Brain imaging enables differentiation of ischemic stroke from hemorrhagic stroke and can reveal structural abnormalities that may be causing the patient's signs or symptoms or that might be contraindications to fibrinolytic therapy. Both noncontrast CT and MRI are acceptable imaging options. Noncontrast CT is the most widely used tool for urgent brain imaging in the setting of acute stroke.

Other diagnostic studies that are routinely ordered for patients with suspected acute ischemic stroke are summarized in Box 11-5. These tests assist in making a definitive diagnosis, determining underlying causes and evaluating candidacy for therapeutic interventions. Additional tests may be ordered depending on history and physical examination findings.

Practice Note

Obtaining laboratory studies and interpreting their results should not delay ordering and initiation of therapy unless there is clinical suspicion for conditions that would increase the patient's risk for bleeding.

Approach to the Patient

The *Acute Stroke: Adult* Treatment Guideline summarizes the approach to a patient with acute stroke.

Assess and Recognize

When stroke is suspected on the basis of the rapid and primary assessments, activate the stroke team immediately per facility protocol. Order a noncontrast CT or MRI scan of the brain within 10 minutes of the patient's arrival. Results should be obtained within 20 minutes and interpreted within 45 minutes. Measure blood glucose levels and provide treatment as necessary. Order laboratory studies (such as a complete blood count and coagulation studies) and obtain a 12-lead ECG, but do not delay brain imaging to do so. Obtain a focused history (including the time of symptom onset) and complete a neurologic screening assessment using the NIHSS or a similar assessment tool. Place the patient on NPO status until the ability to swallow can be assessed.

Care

Hemorrhagic Stroke

If brain imaging reveals hemorrhage, treatment depends on the cause and severity of the bleeding. In addition to basic life support measures, care includes measures to control the internal bleeding, manage increased intracranial pressure, manage blood pressure and treat

Box 11-5 | Recommended Diagnostic Tests for Patients with Suspected Acute Ischemic Stroke

All Patients

- Brain imaging (noncontrast CT or MRI)
- Blood glucose level
- Serum electrolyte panel with renal function tests*
- Complete blood count*
- Cardiac markers*
- Prothrombin time and international normalized ratio*
- Activated partial thromboplastin time*
- 12-Lead ECG

Select Patients

- Thrombin time and/or ecarin clotting time (for patients known or suspected to be on direct thrombin inhibitors or direct factor Xa inhibitors)
- Hepatic function tests
- Toxicology screen
- Blood alcohol level
- Pregnancy test
- Arterial blood gases (in cases of suspected hypoxia)
- Chest radiography (in cases of suspected lung disease)
- Lumbar puncture (in cases where subarachnoid hemorrhage is suspected, but CT scan results are negative for blood)
- Electroencephalography (in cases of suspected seizures)

*Obtaining these tests and interpreting their results should not delay ordering and initiation of therapy unless there is clinical suspicion of a bleeding abnormality or thrombocytopenia, the patient has received heparin or warfarin, the patient has received other anticoagulants or laboratory results have revealed a platelet count of less than 100,000/mm^3, an international normalized ratio (INR) greater than 1.7, an activated partial thromboplastin time (aPTT) greater than 40 seconds or a prothrombin time (PT) greater than 15 seconds.

seizures. For patients receiving anticoagulant therapy, reversal agents, platelet transfusion or both should be considered. A neurology or neurosurgical consult is necessary and the patient may need to be transferred to a comprehensive stroke center or a neurosurgical center for definitive care.

Ischemic Stroke

If brain imaging does not reveal hemorrhage, consider fibrinolytic therapy, endovascular therapy, or both, based on inclusion and exclusion criteria. If serial neurologic examinations show that the patient's neurologic function is improving toward normal, fibrinolytic therapy may not be indicated. In this case, consider adjuvant therapies (e.g., aspirin), seek neurology consult and admit the patient to a stroke unit or critical care unit.

Fibrinolytic Therapy

For patients with ischemic stroke who meet the eligibility criteria (Table 11-1), fibrinolytic therapy is the first-line treatment. Administration of intravenous recombinant tissue plasminogen activator (rtPA) as soon as possible and within 3 hours of the onset of signs and symptoms is optimal (with a goal "door-to-needle" time of less than 60 minutes). For select patients, intravenous rtPA may be administered within 4.5 hours after the onset of signs and symptoms. Following the administration of rtPA, therapy with anticoagulant or antiplatelet agents should be stopped for 24 hours.

Endovascular Therapy

Studies suggest that eligible patients should receive endovascular therapy in addition to fibrinolytic therapy. Endovascular therapy entails the use of catheters to deliver a clot-disrupting or a clot-retrieval device directly to the site of the obstruction. In mechanical thrombectomy, stent retrievers are preferred over coil retrievers. In order to be eligible for endovascular therapy with a stent retriever, patients must meet the following criteria:

- A prestroke modified Rankin Scale (mRS) score of 0 to 1
- A diagnosis of acute ischemic stroke receiving intravenous rtPA within 4.5 hours of the onset of signs and symptoms
- Causative occlusion of the internal carotid artery or proximal middle cerebral artery
- Age 18 years or older
- An NIHSS score of 6 or greater
- Alberta Stroke Programme Early Computed Tomography (ASPECT) score of 6 or greater
- No contraindications to initiation of endovascular therapy (groin puncture) within 6 hours of the onset of signs and symptoms

Table 11-1 | Eligibility Criteria for Intravenous rtPA Administration in Patients with Acute Ischemic Stroke

Treatment Timing	Inclusion Criteria	Absolute Exclusion Criteria	Relative Exclusion Criteria*
Within 3 hours of symptom onset	■ Ischemic stroke diagnosis ■ Measurable neurologic deficit ■ ≥ 18 years of age	■ Significant head trauma or stroke within last 3 months ■ Symptoms suggestive of subarachnoid hemorrhage ■ Arterial puncture at noncompressible site within last 7 days ■ History of intracranial hemorrhage, intracranial tumor, arteriovenous malformation or aneurysm ■ Recent intracranial or intraspinal surgery ■ Hypertension (systolic blood pressure > 185 mmHg or diastolic blood pressure > 110 mmHg) ■ Active internal bleeding ■ Risk factors for acute bleeding, including but not limited to a low platelet count (< 100,000/mm^3); heparin administration within the last 48 hours resulting in an aPTT value greater than the upper limit of normal; current use of an anticoagulant with an INR > 1.7 or a PT > 15 seconds; current use of direct thrombin inhibitors or direct factor Xa inhibitors with elevated results on sensitive laboratory tests (e.g., aPTT; INR; platelet count and ECT; TT; or appropriate factor Xa activity assays) ■ Low blood glucose level (< 50 mg/dL or 2.7 mmol/L) ■ Multilobar infarction on CT	■ Minor or rapidly improving stroke symptoms (clearing spontaneously) ■ Pregnancy ■ Seizure at onset ■ Major surgery or serious trauma within past 14 days ■ Recent gastrointestinal or urinary tract hemorrhage within past 21 days ■ Recent acute myocardial infarction within past 3 months
Within 3 to 4.5 hours of symptom onset	■ Ischemic stroke diagnosis ■ Measurable neurologic deficit	■ Current anticoagulant therapy (INR > 1.7) ■ History of ischemic stroke within previous 3 months	■ Patients ≥ 80 years of age with a history of both diabetes mellitus and prior stroke

*Mounting evidence suggests that under some circumstances, with careful consideration of risk to benefit ratio, patients may receive fibrinolytic therapy despite one or more relative contraindications.

aPTT = activated partial thromboplastin time; ECT = ecarin clotting time; INR = international normalized ratio; NIHSS = National Institutes of Health Stroke Scale; PT = prothrombin time; TT = thrombin time.

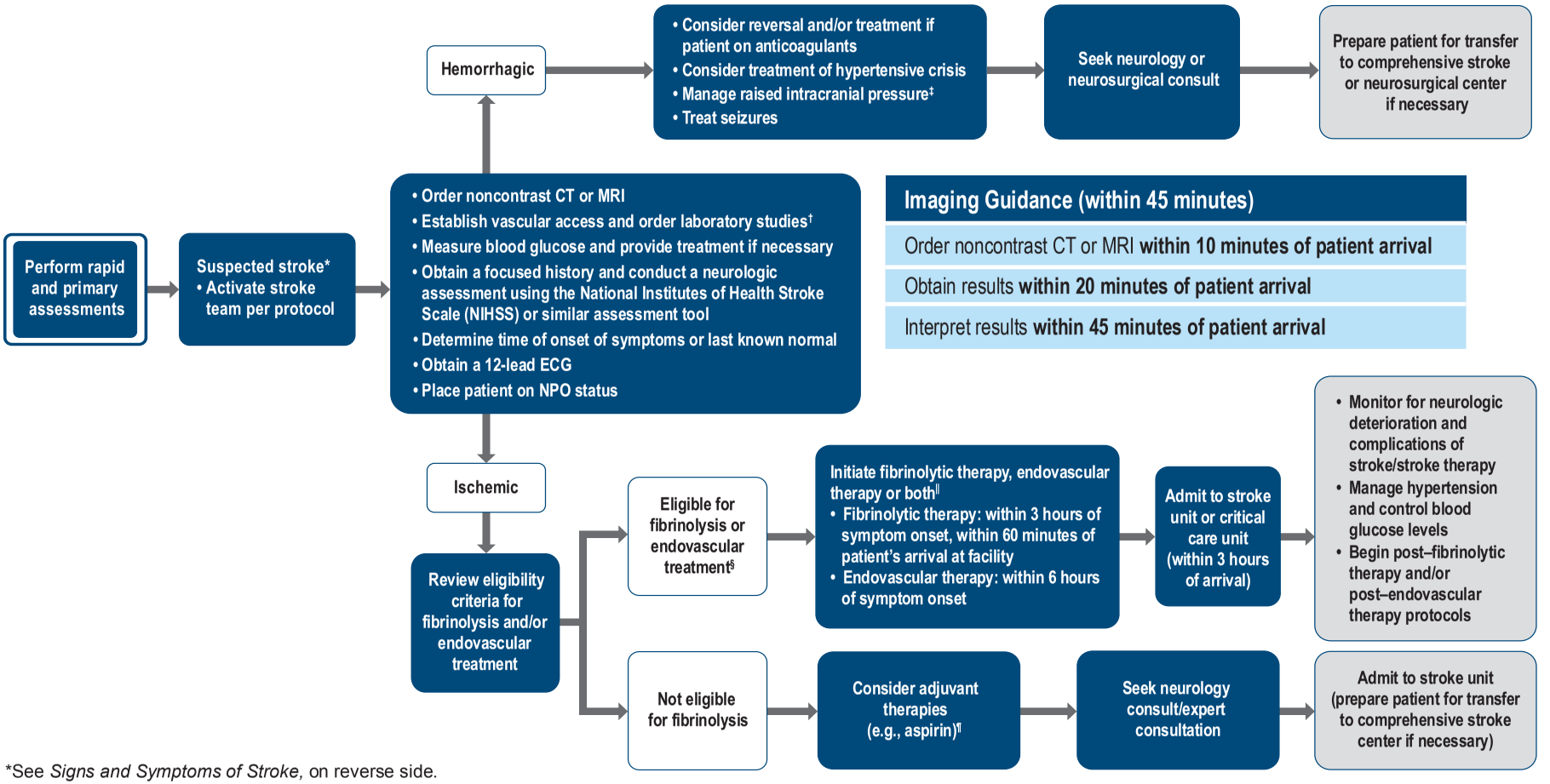
Practice Note

Intra-arterial fibrinolysis with rtPA is considered beneficial for patients with ischemic stroke caused by occlusion of the middle cerebral artery who are not eligible for intravenous rtPA. Intra-arterial fibrinolysis should only be performed at a qualified stroke center, and it should not delay mechanical thrombectomy. When performed in conjunction with mechanical thrombectomy, intra-arterial fibrinolysis is considered to have the best overall success rate of recanalization without having an appreciable effect on the rate of intracranial hemorrhage. Intra-arterial administration of rtPA has not yet been approved by the Food and Drug Administration.

Admission

Within 3 hours of arriving at the facility, the patient should be admitted to a monitored bed on a dedicated stroke unit or critical care unit. Care at this stage includes monitoring for complications of the stroke and its treatment (e.g., intracranial hemorrhage and other bleeding complications); managing hypertension; controlling blood glucose levels; and initiating the post-fibrinolytic protocol, the post-endovascular therapy protocol, or both, as appropriate.

ACUTE STROKE: ADULT



*See *Signs and Symptoms of Stroke,* on reverse side.

†Laboratory studies that are routinely ordered for all patients with suspected acute ischemic stroke include a serum electrolyte panel with renal function tests, complete blood count, cardiac markers, prothrombin time, international normalized ratio and activated partial thromboplastin time.

‡If the patient is compensating for increased intracranial pressure, then management of blood pressure focuses on maintaining cerebral perfusion pressure.

§See *Eligibility Criteria for Intravenous rtPA Administration in Patients with Acute Ischemic Stroke*, on reverse side.

‖Discontinue therapy with anticoagulant or antiplatelet agents for 24 hours after rtPA administration.

¶After completing dysphagia screening.

Please see reverse side for additional information

American Red Cross
Training Services

ACUTE STROKE: ADULT CONTINUED

Signs and Symptoms of Stroke

- Changes in mental status
- Sudden weakness, numbness or tingling on one side of the face or body
- Sudden onset of confusion
- Sudden difficulty with language (e.g., difficulty speaking, difficulty understanding, garbled speech)
- Sudden vision difficulties in one or both eyes
- Difficulty with walking, balance or coordination
- Sudden severe headache

Eligibility Criteria for Intravenous rtPA Administration in Patients with Acute Ischemic Stroke

Treatment Timing	Inclusion Criteria	Absolute Exclusion Criteria	Relative Exclusion Criteria
Within 3 hours of symptom onset	• Ischemic stroke diagnosis • Measurable neurologic deficit • ≥ 18 years of age	• Significant head trauma or stroke within last 3 months • Symptoms suggestive of subarachnoid hemorrhage • Arterial puncture at noncompressible site within last 7 days • History of intracranial hemorrhage, intracranial tumor, AVM or aneurysm • Recent intracranial or intraspinal surgery • Hypertension (systolic blood pressure > 185 mmHg or diastolic blood pressure > 110 mmHg) • Active internal bleeding • Risk factors for acute bleeding, including but not limited to - Low platelet count (< 100,000/mm^3) - Heparin administration within the last 48 hours, resulting in an aPTT value greater than the upper limit of normal - Current use of an anticoagulant with an INR > 1.7 or a PT > 15 seconds - Current use of direct thrombin inhibitors or direct factor Xa inhibitors with elevated results on sensitive laboratory tests (e.g., aPTT, INR, platelet count or ECT; TT or appropriate factor Xa activity assays) • Low blood glucose level (< 50 mg/dL or 2.7 mmol/L) • Multilobar infarction on CT	• Minor or rapidly improving stroke symptoms (clearing spontaneously) • Pregnancy • Seizure at onset • Major surgery or serious trauma within past 14 days • Recent gastrointestinal or urinary tract hemorrhage within past 21 days • Recent acute myocardial infarction within past 3 months
Within 3 to 4.5 hours of symptom onset	• Ischemic stroke diagnosis • Measurable neurologic deficit	In addition to the exclusion criteria for treatment within 3 hours of symptom onset: • Current anticoagulant therapy (INR > 1.7) • History of ischemic stroke within previous 3 months	• Patients ≥ 80 years of age with a history of both diabetes mellitus and prior stroke

Glossary

Acute coronary syndromes
A general term for a group of life-threatening conditions that occur because of a sudden reduction in blood flow to the heart

Arteriovenous malformation
A congenital vascular lesion, often found in the central nervous system, consisting of a mass of entwined arteries and veins connected by fistulae

Atelectasis
Alveolar collapse

Atrioventricular (AV) dissociation
A situation that occurs when the atria and ventricles are being driven by independent pacemakers and are contracting at their own intrinsic rates

Atrioventricular (AV) junction
The zone of tissue surrounding the atrioventricular (AV) node

Capnography
A noninvasive way of measuring the end-tidal carbon dioxide ($ETCO_2$) level

Carina
The point where the trachea bifurcates into the right and left main bronchi

Chest compression fraction (CCF)
An indicator of CPR quality; represents the percentage of time during the resuscitation effort spent performing chest compressions

Closed-loop communication
A communication technique used to prevent misunderstandings; the receiver confirms that the message is received and understood

Coronary perfusion pressure (CPP)
A reflection of myocardial blood flow; the difference between the pressure in the aorta and the pressure in the right atrium during diastole

Corrected QT interval (QTc)
A calculation used to give a QT value that is theoretically independent of rate

Crew resource management
A concept that helps to promote effective and efficient teamwork and reduce the likelihood of errors by encouraging problem solving and communication among team members

Critical thinking
The process of thinking clearly and rationally to identify the connection between information and actions

Dynamic hyperinflation
Auto–positive end-expiratory pressure (auto–PEEP); occurs when exhalation time is insufficient and the lungs do not completely empty before the next breath, preventing the respiratory system from returning to its resting end-expiratory equilibrium volume between breath cycles

Electrical alternans
Beat-to-beat variation in the amplitude of QRS complexes

Embolic stroke
A type of ischemic stroke that occurs when a plaque fragment or blood clot forms elsewhere within the circulatory system and travels to the cerebral circulation

Epiglottis
A leaf-shaped cartilaginous structure that closes over the opening of the larynx during the act of swallowing

Expiratory plateau
The phase of the capnography waveform representing exhalation of the last of the carbon dioxide–laden air from the most distal alveoli

Extracorporeal cardiopulmonary resuscitation (ECPR)
A specialized intervention that uses venoarterial extracorporeal membrane oxygenation (ECMO) in addition to standard CPR

Fibrillatory waves
The slight undulation of the baseline seen on the ECG in atrial fibrillation

Fibrinolytic therapy
Drug therapy to lyse blood clots

Gas exchange
The molecular process of adding oxygen to, and removing carbon dioxide from, the blood

Hemorrhagic stroke
A sudden neurologic deficit caused by rupture of a weakened blood vessel in the brain

Hyperventilation
The state of exhaling carbon dioxide at a faster rate than the body can produce it

Hypoventilation
The state of producing more carbon dioxide than can be exhaled

Infranodal
Distal to the node

Inspiratory downslope
The phase of the capnography waveform representing inhalation

Intracerebral hemorrhage
A type of hemorrhagic stroke that occurs when an artery located within the brain bursts, causing bleeding into the surrounding brain tissue

Ischemia/reperfusion response
An inflammatory response induced by the reperfusion of tissues previously deprived of blood flow, as in cardiac arrest

Ischemic stroke
A sudden neurologic deficit caused by obstruction of a blood vessel supplying the brain

Laryngopharynx
The region of the pharynx that extends from the oropharynx to the level of the cricoid cartilage; also called the hypopharynx

Lung compliance
The ability of the lung to stretch and expand

Monomorphic ventricular tachycardia
A wide-complex ventricular tachycardia characterized by QRS complexes that are generally the same shape; produced by one ectopic focus in the ventricles

Nasopharynx
The region of the pharynx that extends from the base of the skull to the soft palate and is located posterior to the nasal cavities

Oropharynx
The region of the pharynx that extends from the hard palate to the level of the hyoid bone and is located posterior to the oral cavity

Oxygen–hemoglobin dissociation curve
A graphical depiction of the relationship between the partial pressure of oxygen (PaO_2) and the arterial oxygen saturation (SaO_2)

Percutaneous coronary intervention (PCI)
A procedure that uses balloon angioplasty, with or without stent placement, to open occluded coronary arteries

Phased response approach
A model that organizes the team response to an emergency into seven phases and incorporates the concept of crew resource management and the skills of communication, critical thinking and problem solving

Polymorphic ventricular tachycardia
A wide-complex ventricular tachycardia characterized by QRS complexes that vary in shape and rate; produced by two or more ectopic foci in the ventricles

Post–cardiac arrest syndrome
The pathophysiologic consequences of cardiac arrest, comprising four areas (brain injury, myocardial dysfunction, systemic dysfunction and persistent precipitating conditions)

Primary percutaneous coronary intervention (PCI)
Percutaneous coronary intervention performed as the first-line therapy for ST-segment elevation myocardial infarction (STEMI)

Problem solving
The ability to use readily available resources to find solutions to challenging or complex situations or issues that arise

Pulseless electrical activity (PEA)
A term used to describe rhythms that are organized on the monitor (i.e., all of the QRS complexes are similar in appearance) but which fail to produce a palpable pulse

Quantitative capnography
The measurement of end-tidal carbon dioxide ($ETCO_2$) expressed as a value and as a waveform

Quantitative capnometry
The measurement of end-tidal carbon dioxide ($ETCO_2$) expressed as a value

Rapid response team
A team of highly trained and skilled personnel who work together to care for a patient when signs of cardiopulmonary compromise or shock are noted

Respiration
The process of moving oxygen and carbon dioxide between the atmosphere and the body's cells

Respiratory arrest
Complete cessation of the breathing effort

Respiratory baseline
The phase on the capnography waveform representing the beginning of exhalation

Respiratory distress
The earliest stage on the continuum of respiratory compromise; the patient is using compensatory mechanisms to maintain oxygenation and ventilation adequate to meet metabolic demands

Respiratory failure
The intermediate stage on the continuum of respiratory compromise; the respiratory system is no longer able to meet metabolic demands

Respiratory upstroke
The phase on the capnography waveform representing the exhalation of air containing carbon dioxide from the alveoli

Resuscitation team
A team of highly trained and skilled personnel who work together to provide resuscitative care when a patient experiences respiratory or cardiac arrest

Subarachnoid hemorrhage
A type of hemorrhagic stroke that occurs when a blood vessel located on the surface of the brain ruptures, causing bleeding into the subarachnoid space

Supraglottic airway
An advanced airway that is not passed through the vocal cords, such as the laryngeal mask airway and the laryngeal tube

Supraventricular tachycardia
A general term for tachyarrhythmias that originate above the ventricles in the atria or atrioventricular node and run normally through the bundle branches, producing a normal QRS complex

Targeted temperature management (TTM)
A neuroprotective intervention that involves lowering and maintaining the core body temperature in the range of 32° C to 36° C for a period of at least 24 hours

Teamwork
The actions of a group of people with well-defined roles and responsibilities making a coordinated effort to achieve a common goal

Thrombotic stroke
A type of ischemic stroke caused by rupture of an atherosclerotic plaque in a cerebral artery that results in the formation of a thrombus

Transglottic airway
An advanced airway that is passed through the vocal cords, such as an endotracheal tube

Transthoracic impedance
The body's resistance to current flow that is caused by the thoracic structures, including soft tissue and bone, between defibrillation pads or paddles and the heart

Ventilation
The mechanical process of moving air into and out of the body

Ventilation-perfusion mismatch
An imbalance between the air that reaches the alveoli (i.e., ventilation) and blood flow to the alveoli (i.e., perfusion) in a portion of the lung

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Note: *b* indicates box; *t* indicates table

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- The International Liaison Committee on Resuscitation (ILCOR) 2015-2018 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations.
- The 2015-2018 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care.

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