Pediatric Advanced Life Support

Participant's Manual

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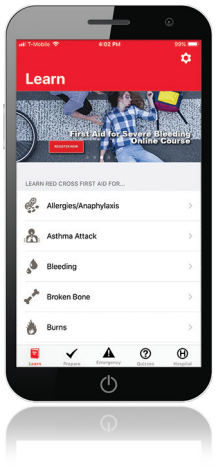

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This Participant's Manual is part of the American Red Cross Pediatric Advanced Life Support program. The emergency care procedures outlined in the program materials reflect the standard of knowledge and accepted emergency practices in the United States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

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Science and Technical Content

The scientific content and approach to care within the American Red Cross Pediatric Advanced Life Support program are based on the 2015, 2017 and 2018 International Liaison Committee on Resuscitation (ILCOR) Consensus on Science and Treatment Recommendations (CoSTR) and are consistent with the 2015–2018 American Heart Association Guidelines for CPR and ECC as well as subsequent Focused Updates. The Red Cross curriculum also incorporates science, policies and guidelines from the Surviving Sepsis campaign, the American Academy of Pediatrics, the American College of Critical Care Medicine, American College of Emergency Physicians, the American College of Surgeons and the U.S. Department of Defense Committee on TCCC.

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- 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.

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CHAPTER 1

Pediatric Advanced Life Support Course Introduction

Introduction

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

Course Purpose

When a patient experiences a life-threatening emergency (such as a respiratory emergency, shock or a cardiac emergency), you need to act swiftly to assess the situation and the patient and provide lifesaving care.

The purpose of the American Red Cross Pediatric Advanced Life Support course is to ensure that healthcare providers who work with pediatric patients have the requisite knowledge and skills to assess, recognize and care for patients who are experiencing a respiratory emergency, shock or a cardiac 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 Pediatric Advanced Life Support course covers the skills required for certification as a PALS provider. In addition, the key concepts that support proficient performance of these skills are reviewed.

Course Preparation

The American Red Cross Pediatric Advanced Life Support course is designed for professional healthcare providers who directly care for pediatric patients in a variety of settings and who could be called on to care for a critically ill pediatric 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 pediatric 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 respiratory emergency, shock or a cardiac emergency.

The only requirement for taking the American Red Cross Pediatric Advanced Life Support course is a current Basic Life Support (BLS) certification. High-quality CPR skills (including high-quality chest compressions and basic airway management) and AED skills are an essential part of PALS (Figure 1-2). In addition, strong patient assessment skills; knowledge and understanding of pharmacology used in respiratory emergencies, shock and cardiac 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 Pediatric Advanced Life Support course, you will be able to:

- Demonstrate high-quality pediatric basic life support skills, including age-appropriate high-quality chest compressions, effective ventilations and age-appropriate use of an AED.
- Apply concepts of effective teamwork when caring for a patient experiencing a respiratory emergency, shock or a cardiac emergency.
- Integrate advanced communication, critical thinking and problem-solving skills when responding as part of a team to a respiratory emergency, shock or a cardiac emergency (Figure 1-3).
- Effectively assess a respiratory emergency, shock or cardiac emergency situation using a systematic approach.
- Quickly recognize the nature of a respiratory emergency, shock or a cardiac emergency.
- Provide effective and appropriate pediatric advanced life support care to address a respiratory emergency, shock or a cardiac 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.

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

Your Role as a Pediatric Advanced Life Support Provider

PALS refers to the care that healthcare providers deliver to children and infants who are experiencing a respiratory emergency, shock or a cardiac emergency. The psychomotor skills needed to perform high-quality CPR; use an AED, a defibrillator, or both; and relieve an obstructed airway for pediatric patients of all ages are the foundation of PALS. As a PALS 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 respiratory emergencies, shock or cardiac emergencies based on specific pediatric advanced life support treatment guidelines. PALS integrates the following key concepts to help providers achieve optimal patient outcomes:

- **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, to sequence care steps properly and to demonstrate proficiency in completing all required skills without any coaching or assistance.

There are two ways to complete American Red Cross PALS 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 Pediatric 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 Pediatric 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 (high-quality CPR, AED and relieving an obstructed airway) are an essential component of pediatric advanced life support (PALS). Understanding how to perform these skills and demonstrating BLS skills mastery are vital in order to achieve the best possible outcomes for children and infants in respiratory or cardiac arrest, as well as for those who have an obstructed airway.

Defining Adolescents, Children and Infants

Children are not small adults. Therefore, they need to be cared for differently in a life-threatening emergency. It is essential to identify which age-dependent CPR and AED guidelines to follow (Figure 2-1).

When determining which CPR/AED protocol to follow, use these guidelines:

- An infant is defined as someone under the age of 1. When providing BLS care, follow infant guidelines and use appropriately sized equipment.
- A child is defined as someone between the age of 1 to the onset of puberty as evidenced by breast development in girls and underarm hair development in boys (usually around the age of 12). When providing BLS care, follow child guidelines and use appropriately sized equipment. The use of pediatric versus adult AED pads or settings for children varies by age and weight.
- An adolescent is defined as someone from the onset of puberty (adolescent) through adulthood. When providing BLS care, follow adult guidelines and use appropriately sized equipment.

Figure 2-1 | CPR/AED guidelines are age dependent. An infant is someone under age 1. A child is someone from age 1 to the onset of puberty. An adolescent is someone from the onset of puberty through adulthood.

Pediatric Cardiac Chain of Survival

The Pediatric Cardiac Chain of Survival (Figure 2-2) is similar to the Adult Cardiac Chain of Survival, but it focuses on prevention. The most common causes of cardiac arrest in children include respiratory emergencies, shock, congenital heart disorders and trauma.

The five links in the Pediatric Cardiac Chain of Survival are:

- **Prevention of arrest**. Prevention is key because cardiac arrest in children often occurs due to a respiratory emergency or shock or as the result of a preventable injury (such as trauma, drowning, choking or electrocution).
- **Early, high-quality CPR**. CPR, starting with compressions, should be initiated within 10 seconds of recognizing cardiac arrest.
- **Rapid activation of the emergency medical services (EMS) system or response team**. Immediate recognition of cardiac arrest and activation of the EMS system or response team gives the patient access to necessary personnel, equipment and interventions as soon as possible after arrest.
- **Effective, pediatric advanced life support**. Effective, pediatric advanced life support gives the patient access to emergency medical care delivered by specially trained professionals.
- **Integrated post–cardiac arrest care**. 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.

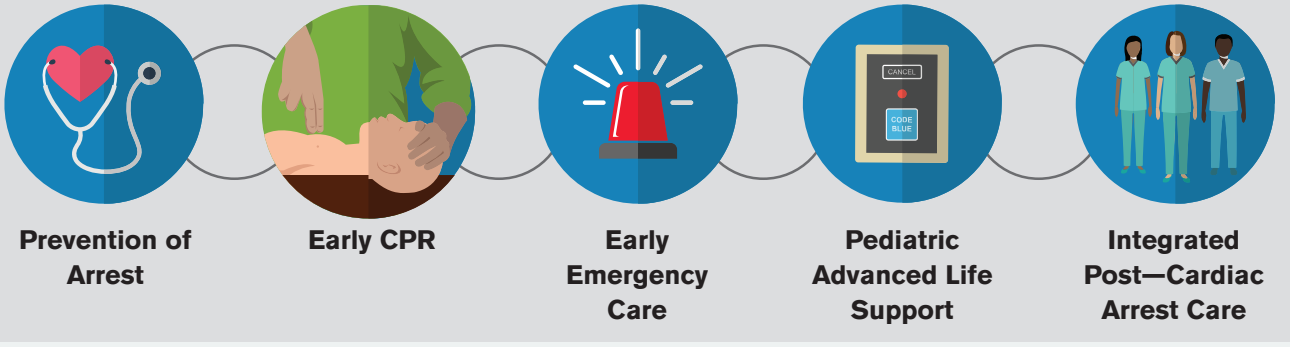


Figure 2-2 | The Pediatric Cardiac Chain of Survival

The Pediatric Cardiac Chain of Survival focuses on prevention because cardiac arrest in children often occurs due to a respiratory emergency or shock or as the result of a preventable injury.

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. To give your patients their best chance for neurologically intact survival following cardiac arrest, practice these critical components of high-quality CPR: provide compressions at the proper rate and at the proper depth, allow full chest recoil, minimize interruptions to chest compressions, and avoid excessive ventilations. These are the keys to every successful resuscitation.

Principals of High-Quality CPR

To provide high-quality CPR in children and infants, first 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** (about 2 inches [5 cm] for children and about 1½ inches [3.8 cm] for infants). When given at the proper rate, it should take 15 to 18 seconds to perform 30 compressions or 7 to 9 seconds to perform 15 compressions if two providers are performing CPR.
- **Allow the chest to recoil fully after each compression**. Don't lean on the patient's chest at the top of the compression. This impedes venous return and prevents the heart from filling completely, which decreases cardiac output.
- **Minimize interruptions in chest compressions**. Limit interruptions in chest compressions to <10 seconds. 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.
- **Avoid excessive ventilations**. Each ventilation should last about 1 second and deliver just enough volume to make the chest begin to rise.

High-performance CPR refers to providing high-quality CPR as part of a well-organized team response to a cardiac arrest. Coordinated, efficient effective teamwork helps to minimize interruptions to compressions (Figure 2-4). 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

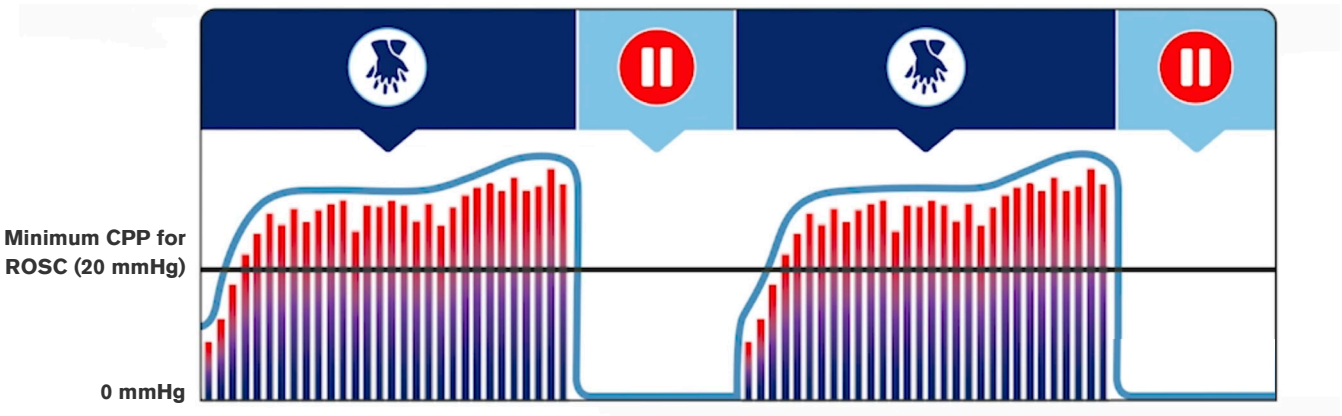


Figure 2-3 | When compressions stop, the coronary perfusion pressure (CPP) drops below 20 mmHg, the minimum level needed to increase the likelihood of return of spontaneous circulation (ROSC) and survival.

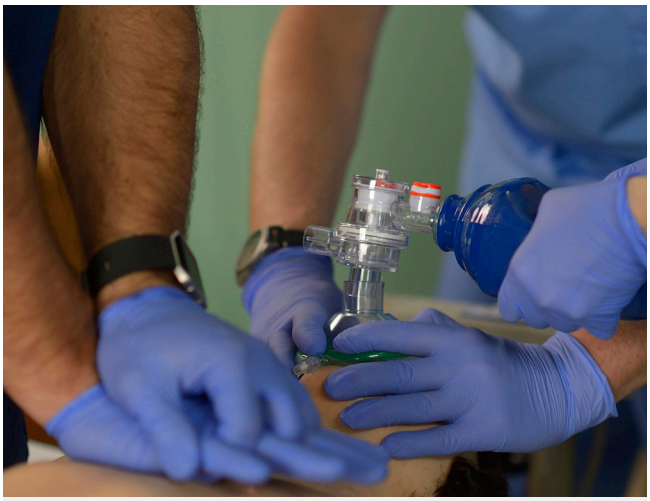


Figure 2-4 | Efficient teamwork contributes to the provision of high-quality CPR.

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 period should occur, 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.

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, which measures the level of carbon dioxide (CO_2) in exhaled breath (the end-tidal CO_2, or $ETCO_2$), provides an objective measure of compression quality with every ventilation. Capnography is a noninvasive technique that uses sensors to detect $ETCO_2$ levels, which are displayed as waveforms on a monitor (Figure 2-5).

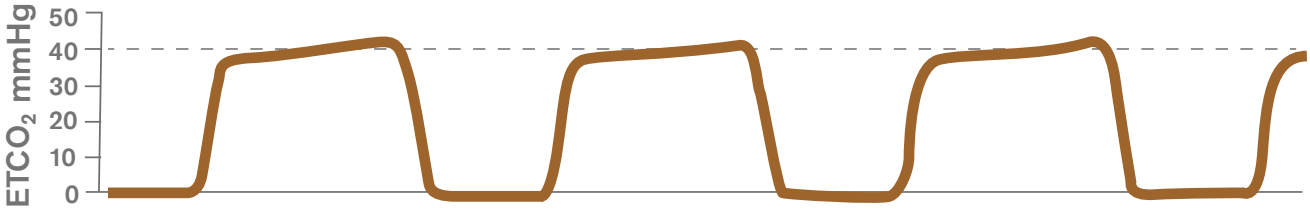


Figure 2-5 | The normal capnography waveform is square with a flat plateau. In patients with normal perfusion, capnography values are between 35 and 45 mmHg.

The sensors can be used with a bag-valve-mask (BVM) resuscitator or an advanced airway. Because blood containing CO_2 is returned to the heart and then pumped through the pulmonary circulation so that the CO_2 can diffuse into the alveoli and be removed from the body, $ETCO_2$ is a quantitative measure of cardiac output (perfusion). 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 than normal. When high-quality CPR is provided, however, the $ETCO_2$ levels are expected to be in the range of 15 to 20 mmHg. If $ETCO_2$ levels fall below 10 mmHg, a problem likely exists either with the rate of ventilations or 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 sudden spike in $ETCO_2$ levels (up 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.

Practice Note

Pediatric 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. Providers should use the systolic and diastolic blood pressure readings to guide the quality of CPR. However, specific target values for blood pressure during CPR have not yet been established for the pediatric population.

BLS Differences Among Adults, Children and Infants

Key differences in technique exist when providing BLS care to adults, children and infants. These differences include rapid assessment, compression technique, compression depth, ventilations, use of appropriately sized breathing barriers, CPR with an advanced airway in place, and use of an AED.

Rapid Assessment

Rapid assessment for a child or infant is similar to rapid assessment for an adult with a few key differences. See *Rapid Assessment for Children and Infants* Skill Sheet.

Shout-Tap-Shout Sequence

If the child or infant appears unresponsive, follow the shout-tap-shout sequence. Shout, “Are you OK?” or use the child or infant’s name if known, as you do with an adult. Tap the child on the shoulder similar to an adult (Figure 2-6, A); however, tap the infant on the *bottom of the foot*. Then shout again. (Figure 2-6, B).

Additional Resources

As with an adult, if a child or infant is unresponsive follow these steps:

- Call for someone to activate EMS, the rapid response team or the resuscitation team, as appropriate and retrieve the AED, bag-valve-mask (BVM) resuscitator and other emergency equipment.
- If you are alone with a child or infant and do not have a mobile phone or other form of communication, you must decide to call first or care first. See *Learn More: Call First or Care First?*

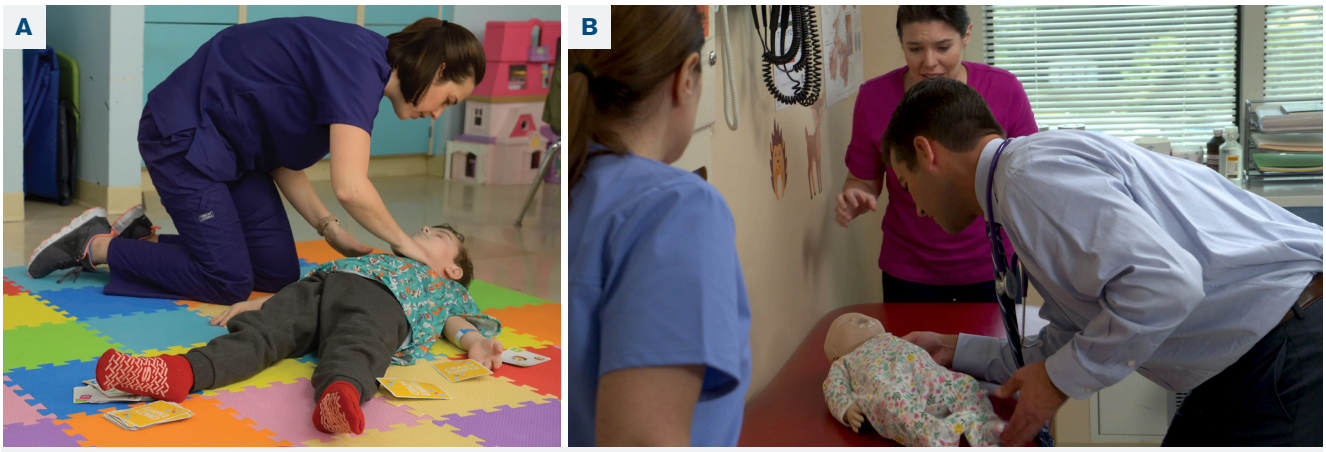


Figure 2-6 | If a child or infant appears unresponsive, follow the shout-tap-shout sequence. (A) For a child, tap the shoulder as you do for an adult. (B) For an infant, tap the bottom of the foot instead.

LEARN MORE

Call First or Care First?

Although it is rare in the professional healthcare setting to be alone with a child or infant or to be unable to shout for help, you should follow certain steps if this is ever the case.

If you are alone and do not have a mobile phone or other method of communication, you must decide to call first or care first.

Call First

For a child or an infant whom you witnessed suddenly collapse, or for an unresponsive child or infant with a known cardiac condition:

- Call for help to activate EMS, the rapid response team or the resuscitation team, as appropriate, and call for an AED.
- Then, provide care based on the conditions found.

Care First

For an unresponsive child or infant whom you did not see collapse:

- Provide 2 minutes of care based on the conditions found.
- Then, call for help to activate EMS, the rapid response team or the resuscitation team, as appropriate, and call for an AED.

Practice Note

Because most child or infant cardiac arrests occur as a result of a hypoxic event (e.g., an asthma exacerbation, an airway obstruction or a drowning), ventilations and appropriate oxygenation are important for successful resuscitation. Laryngeal spasm may occur in these situations, making passive ventilation during chest compressions minimal or nonexistent. Therefore, it is critical to make sure the child or infant's brain is oxygenated before leaving them to get additional resources.

ALERT

If you are caring for an infant, bring them with you to get additional resources.

Figure 2-7 | Use the head-tilt/chin-lift technique to open the airway. (A) For a child, tilt the head to a slightly past-neutral position. (B) For an infant, only tilt the head to a neutral position.

Open the Airway

Subtle differences in positioning are applied when opening the airway of a child or infant compared with an adult. To open the airway of a child or infant, use the same head-tilt/chin-lift technique or jaw-thrust maneuver as for an adult. However, when using the head-tilt/chin-lift technique, only tilt the head to a slightly past-neutral position for a child (Figure 2-7, A) or a neutral position for an infant (Figure 2-7, B).

Take care to avoid any hyperextension or flexion in the neck. Be careful not to place your fingers on the soft tissues under the chin or neck to open the airway.

Check for Breathing and a Pulse

Simultaneously check for breathing a pulse for at least 5 seconds but no more than 10. Check the carotid pulse for a child (Figure 2-8, A). However, for an infant, check the *brachial pulse* with two fingers on the inside of the upper arm. Do not use your thumb because it has its own detectable pulse. You will need to expose the arm to accurately feel a brachial pulse (Figure 2-8, B).

Recognize and Care

After completing your rapid assessment, obtain **consent** (Box 2-1) and provide care based on conditions found.

Box 2-1 | Obtaining Consent for Children and Infants

Legally, adults who are awake and alert can consent to treatment; if they are not alert, consent is implied. However, for most infants and children up to the age of 17 years, you must obtain consent from the child's parent or legal guardian if he or she is present regardless of the child's level of consciousness.

To gain consent, state who you are, what you observe and what you plan to do when asking a parent or legal guardian permission to care for his or her child. If no parent or legal guardian is present, consent is implied in life-threatening situations. Always follow your local laws and regulations as they relate to the care of minors.

Always follow your facility's protocols.

- If the child or infant is unresponsive or experiencing an altered level of consciousness, is breathing normally and has a pulse: Place them in recovery position to help maintain a clear airway and monitor them until EMS, the rapid response team or the resuscitation team arrives. See *Learn More: Recovery Positions for Children and Infants*.
- If the child or infant is in respiratory arrest: Deliver 1 ventilation every 3 to 5 seconds. See Basic Life Support Sequence for Children and Infants later in the chapter.

Figure 2-8 | As part of rapid assessment, simultaneously check for breathing and a pulse. (A) For a child, check at the carotid artery, as you do for an adult. (B) For an infant, check at the brachial artery.

- If the child or infant is in cardiac arrest: Begin CPR within 10 seconds and initiate AED use as soon as one is available. See Basic Life Support Sequence for Children and Infants later in the chapter.

The *CPR for Children* Skill Sheet and *CPR for Infants* Skill Sheet provide step-by-step guidance for performing CPR on a child or an infant.

ALERT

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

Compression Technique for Children

The technique for providing chest compressions are similar for an adult and child: Position one hand on top of the other with your fingers interlaced and off the chest, centered on the lower half of the sternum (Figure 2-9, A). However, for a smaller child, you may opt to use the one-hand technique to deliver compressions (Figure 2-9, B).

Compression Technique for Infants

The technique of providing compressions to an infant is different from an adult and child because of the infant's smaller size. Technique also differs based on the number of providers involved.

When initiating CPR for an infant, use a firm, flat surface. Obtain a CPR board or use a CPR-ready crib or bed. Make sure the crib is at an appropriate working height or use a step stool. Lower the crib side rail closest to you. If you are not at the infant's bedside, use a stable surface such as a table or countertop because it is usually easier to perform compressions from a standing position rather than kneeling at the infant's side.

Two-Finger Technique

To perform compressions for an infant when you are the only provider, use the two-finger technique. Using your hand closet to the infant's feet, place two fingers in the center of the exposed chest, just below the nipple line on the sternum. The fingers should be oriented so that they

LEARN MORE

Recovery Positions for Children and Infants

For children, use the same approach and technique for recovery positions as you would for an adult if you do not suspect a head, neck, spinal or pelvic injury.

Technique may differ for infants. Infants with a suspected head, neck, spinal or pelvic injury should not be placed in a recovery position unless you are unable to manage the airway effectively or you are alone and do not have a mobile phone or other form of communication.

To place an infant in a recovery position, use the same technique you would apply for an older child. You also can hold an infant in a recovery position by:

- Carefully positioning the infant face-down along your forearm.
- Then supporting the infant's head and neck with your other hand while keeping the infant's mouth and nose clear.

Figure 2-9 | (A) Compression technique for a child is the same as for an adult. (B) The one-hand technique may be a better method for smaller children.

are parallel, not perpendicular, to the sternum (Figure 2-10, A). You can use your index and middle fingers or your middle and fourth fingers to provide compressions. Fingers that are more similar in length tend to make compressions easier to deliver.

Encircling Thumbs Technique

When multiple providers are caring for an infant in cardiac arrest, the positioning and chest technique for providing compressions differs from those used for an adult or child. The provider performing chest compressions will be positioned at the infant's feet, while the provider providing ventilations will be at the infant's head. Compressions are delivered using the encircling thumbs technique (Figure 2-10, B).

- Place both thumbs (side-by-side) on the center of the infant's chest, just below the nipple line.
- Use the other fingers to encircle the infant's chest toward the back, providing support.
- Using both thumbs at the same time, compress the chest about 1½ inches at a rate of at least 100 but no more than 120 compressions per minute. Let the chest return to its normal position after each compression.

Compression Rate and Depth for Children

The compression rate of 100 to 120 per minute is the same for a child as for an adult. The depth of compression, however, is different. For an adult, compress the chest *at least* 2 inches but no more than 2.4 inches; however, for a child, compress the chest *about* 2 inches (or one-third the anterior-posterior diameter of the chest). Make certain you compress the chest with sufficient depth. Use a feedback device during CPR to objectively measure your compression rate and depth.

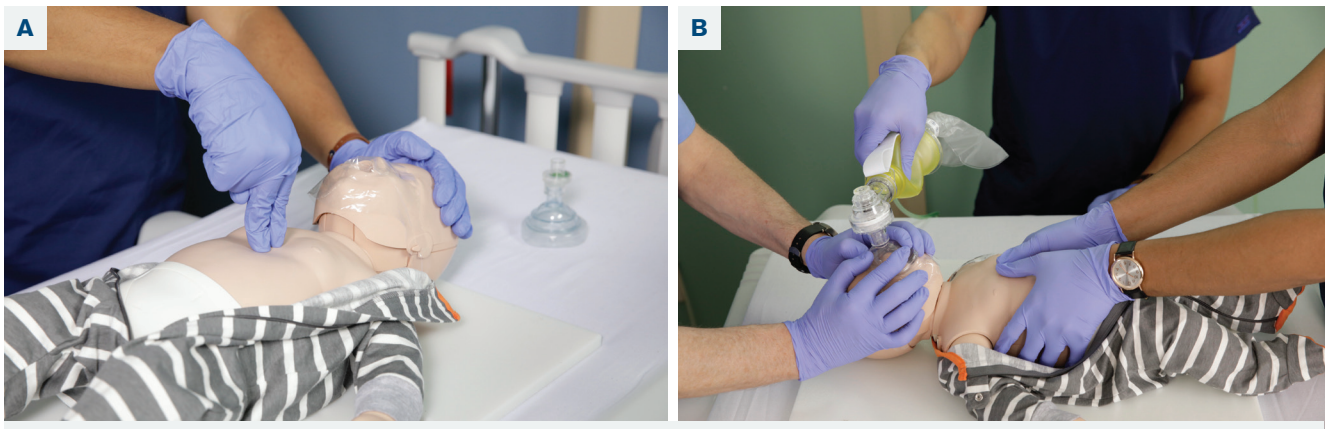


Figure 2-10 | The chest compression technique for an infant differs between single and multiple providers. In both situations, the compression depth for an infant is about 1½ inches. (A) Single providers should use the two-finger technique. (B) However, multiple providers should use the encircling thumbs technique.

Compression Rate and Depth for Infants

Compressions are delivered at the same rate used for adults and children—that is, between 100 and 120 compressions per minute. However, for an infant, only compress the chest *about* 1½ inches (or one-third the anterior-posterior diameter of the chest).

Ventilation Technique for Children and Infants

The technique for providing ventilations to a patient in cardiac arrest is the same for adults, children and infants. Provide smooth, effortless ventilations that last about 1 second and make the chest begin to rise. The rate for providing ventilations to a patient in respiratory arrest is different in children and infants compared with adults. For both children and infants in respiratory arrest, deliver 1 ventilation every 3 to 5 seconds.

Appropriately Sized Equipment

When providing BLS care to a child or infant, it is essential that you use appropriately sized equipment. Always follow the manufacturer's guidelines and your facility's protocols.

Pocket Masks

If you are using a pocket mask, make sure it is sized appropriately. Some pocket masks are one-size-fits-all for adults and children. In this case, the mask can be rotated so that the narrow end fits over the child's chin. In other instances, separate pocket masks are available for use with children. For infants, you must use a specifically sized infant pocket mask. However, do not delay care while searching for a barrier device.

Bag-Valve-Mask Resuscitators

One of the most difficult aspects of bag-valve-mask (BVM) ventilation can be ensuring an effective seal. Therefore, you must use an appropriately sized BVM (Figure 2-11). You may select from sizes for a newborn, infant and child, or options may include small, medium and large. A circular mask may be more appropriate for children and infants.

Verify that you have selected the correct size by checking that the mask does not cover the patient's eyes and that it does not extend beyond the chin. Like the pocket mask, the narrow end of the BVM device fits over the nose. The bags on the device also deliver smaller volumes for infants and young children.

Some BVMs for children and infants also include a pressure relief or "pop-off" valve that helps to prevent excessive pressure during ventilations. In a resuscitation situation it is essential to deactivate the pop-off valve to assure adequate ventilation.

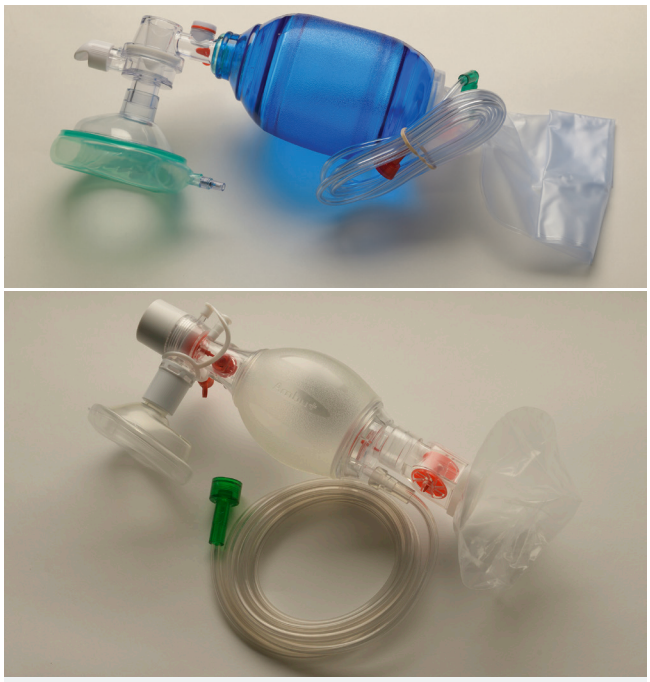


Figure 2-11 | To ensure an effective seal, choose an appropriately sized BVM.

When delivering ventilations with a BVM, maintain the mask seal and open airway in a slightly past-neutral position for children or a neutral position for infants. Avoid any hyperextension or flexion in the neck. Do not place your fingers on the soft tissues under the chin or neck to open the airway.

Practice Note

In children and infants, additional factors must be considered when opening the airway and using the device. The back, lower portion of the head is larger in children than it is in adults, which means there is more flexion of the neck. Other considerations include larger tongues and possible lack of teeth, which may impede ventilation.

Compression-to-Ventilation Ratio for Children

When you are the only provider, the ratio of compressions to ventilations for a child is the same as for an adult—that is, 30 compressions to 2 ventilations (30:2). However, in multiple-provider CPR, this ratio changes to 15 compressions to 2 ventilations (15:2).

Compression-to-Ventilation Ratio for Infants

When you are the only provider, the ratio of compressions to ventilations for an infant is the same as for an adult and child—that is, 30:2. In multiple-provider CPR, however, this ratio changes to 15:2, which is the same as for a child.

Advanced Airways

When an advanced airway is in place, one provider gives 1 ventilation every 6 to 8 seconds. If the child is in cardiac arrest, another provider performs compressions at a rate of 100 to 120 compressions per minute. In this case, the compression-to-ventilation ratio does not apply because compressions and ventilations are delivered continuously with no interruptions.

AED Use for Children and Infants

Although defibrillation is needed less often for a child or an infant than for an adult, the use of an AED remains a critical component of child and infant cardiac arrest care. AEDs work the same way regardless of the patient's age, but the pads or setting used for children and infants differ, as does pad placement, based on the size of the child and infant (Figure 2-12).

- **Children 8 years of age or younger (including infants) or weighing 55 pounds (25 kg) or less**: Use pediatric AED pads or electrical settings, if available. Pediatric AED pads are smaller and designed specifically to deliver a lower level of energy. If pediatric AED pads are not available or the AED does not have a pediatric setting, it is safe to use adult AED pads or adult levels of energy on a child or infant.
- **Children over the age of 8 years or weighing more than 55 pounds (25 kg)**: Use adult AED pads. You should not use pediatric AED pads or the pediatric setting on an adult or child over the age of 8 years or weighing more than 55 pounds (25 kg) because the shock delivered will not be sufficient. In these cases, always use adult AED pads and energy level.

Figure 2-12 | While AEDs work the same regardless of the patient's age, the pads or setting used for children and infants differ.

ALERT

Never use pediatric AED pads or a pediatric electrical setting on a child older than 8 or weighing more than 55 pounds (25 kilograms).

For children, position the pads in the same way you would for an adult. (Use the anterior/lateral or anterior/posterior position according to the manufacturer's instructions.) The AED pads should never touch each other when applied (Figure 2-13, A).

If it appears that the AED pads would touch each other based on the size of the child's chest, use an anterior/posterior pad placement as an alternative. Apply one pad to the center of the child's chest on the sternum and one pad to the child's back between the scapulae. When using an AED on an infant, always use the anterior/posterior pad placement (Figure 2-13, B).

Figure 2-13 | (A) When using the anterior/lateral pad placement on a child, make sure the AED pads do not touch. (B) For an infant, always use the anterior/posterior AED pad placement.

Practice Note

Various models of AEDs and manual defibrillators function differently. Be sure to follow the manufacturer's recommendations and your facility's protocols.

The *AED Use for Children and Infants* Skill Sheet provides step-by-step guidance for using an AED.

Table 2-1 provides a review of the basic life support differences between an adolescent/adult, child and infant.

Table 2-1 | Basic Life Support Differences: Adolescent/Adult, Child and Infant

	Adolescent/Adult	Child (age 1 through onset of puberty)	Infant (birth to age 1)
Shout-tap-shout sequence	Shout "Are you OK?" **Tap the shoulder.** Then shout again.	Shout "Are you OK?" **Tap the shoulder.** Then shout again.	Shout "Are you OK?" **Tap the bottom of the foot.** Then shout again.
Calling for additional resources: If alone and no form of communication	**Leave to call for additional resources.** Then begin CPR.	Witnessed sudden collapse or known cardiac condition: **Leave to call for additional resources.** Then begin CPR. Unwitnessed sudden collapse: **Perform 2 minutes of CPR.** Then leave to call for additional resources.	Witnessed sudden collapse or known cardiac condition: **Leave to call for additional resources.** Then begin CPR. Unwitnessed sudden collapse: **Perform 2 minutes of CPR.** Then leave to call for additional resources.
Airway: Head-tilt/chin-lift	**Past-neutral** position	**Slightly past-neutral** position	**Neutral** position
Ventilations: Respiratory arrest	**1** ventilation every **5** to **6** seconds	**1** ventilation every **3** to **5** seconds	**1** ventilation every **3** to **5** seconds
Compression technique	**Two hands interlaced** on the chest centered on lower half of the sternum	**Two hands interlaced** on the chest centered on lower half of the sternum For smaller children, use the **one-hand technique.**	Single-provider CPR: **Two-finger technique**—two fingers centered on the sternum just below the nipple line Multiple-provider CPR: **Encircling thumbs technique**—two thumbs centered on the sternum just below the nipple line with fingers encircling chest
Compression rate	100 to 120 per minute	100 to 120 per minute	100 to 120 per minute

	Adolescent/Adult	Child (age 1 through onset of puberty)	Infant (birth to age 1)
Compression depth	**At least 2 inches** (5 cm) but no more than 2.4 inches (6 cm)	**About 2 inches** (5 cm) or one-third the anterior-posterior diameter of the chest	**About 1½ inches** (3.8 cm) or one-third the anterior-posterior diameter of the chest
Compression-to-ventilation ratio	Single-provider CPR: **30:2** Multiple-provider CPR: **30:2**	Single-provider CPR: **30:2** Multiple-provider CPR: **15:2**	Single-provider CPR: **30:2** Multiple-provider CPR: **15:2**
CPR with an advanced airway in place	**1** ventilation every **6** seconds; compressions and ventilations are delivered continuously with no interruptions	**1** ventilation every **6** to **8** seconds; compressions and ventilations are delivered continuously with no interruptions	**1** ventilation every **6** to **8** seconds; compressions and ventilations are delivered continuously with no interruptions
AED pads	Use **adult pads. Do not use pediatric pads or setting;** the shock delivered will not be sufficient.	Age >8 years, weight >55 pounds (25 kg): Use **adult pads. Do not use pediatric pads or setting;** the shock delivered will not be sufficient. Age ≤8 years, weight ≤55 pounds (25 kg): Use **pediatric pads or setting.** Use **adult pads** if pediatric pads or setting are not available.	Use **pediatric pads or setting.** Use **adult pads** if pediatric pads or setting are not available.
AED pad placement	**Anterior/lateral** placement Upper right chest below right clavicle to right of sternum Left side of chest several inches below left armpit on midaxillary line **Anterior/posterior** placement, if recommended by manufacturer	**Anterior/lateral** placement Upper right chest below right clavicle to right of sternum Left side of chest several inches below left armpit on midaxillary line **Anterior/posterior** placement, if pads risk touching each other or recommended by manufacturer	**Anterior/posterior** placement Middle of chest Back between scapulae

Basic Life Support Sequence for Children and Infants

When responding to a pediatric respiratory or cardiac event, you must complete a rapid assessment, recognize the problem and provide quality care. *BLS: Children and Infants* Treatment Guideline summarizes the approach to providing basic life support care for a child or infant.

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.

Respiratory Arrest

Follow these steps for a child or infant who is unresponsive, not breathing normally (or only gasping) but has a pulse >60 bpm:

- If you have not already done so, call for help to activate EMS, the rapid response team or the resuscitation team, as appropriate, and call for an AED.
- If you are alone and do not have a mobile phone or other form of communication, you must decide to call first or care first.
- Deliver 1 ventilation every 3 to 5 seconds; each ventilation should last about 1 second and make the chest begin to rise (Figure 2-14).
- Continue ventilations. Check the pulse and breathing about every 2 minutes. If the pulse decreases to 60 bpm or less with signs of poor perfusion, begin CPR and reassess about every 2 minutes.

Follow these steps for a child or infant who is unresponsive, showing signs of poor perfusion and not breathing normally (or only gasping) but has a pulse ≤60 bpm:

- If you have not already done so, call for help to activate EMS, the rapid response team or the resuscitation team, as appropriate, and call for an AED.

Figure 2-14 | For both children and infants in respiratory arrest, deliver 1 ventilation every 3 to 5 seconds.

- If you are alone and do not have a mobile phone or other form of communication, you must decide to call first or care first.
- Begin CPR.
- Continue ventilations and chest compressions.
- Check the pulse and breathing about every 2 minutes.
  - If the child is not breathing normally and the pulse increases to greater than 60 bpm, stop chest compressions but continue providing 1 ventilation every 3 to 5 seconds.
  - If the child is not breathing normally but has a pulse less than 60 bpm, continue CPR.

Practice Note

Signs of inadequate perfusion in a child or infant include cool, moist skin; pallor, mottling or cyanosis; a weak or thready pulse; decreased capillary refill; and hypotension.

Cardiac Arrest

When you are the only provider present, you must complete the rapid assessment, perform CPR and use an AED. Performing CPR can be exhausting, so you should seek additional resources as early as possible during the visual survey. When you are the only provider present, the ratio of compressions to ventilations for a child or infant is the same as for an adult, that is, 30 compressions to 2 ventilations (30:2). Remember for single-provider infant CPR, use the two-finger technique to provide compressions.

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. When two providers are performing CPR for children and infants, the ratio changes to 15 compressions to 2 ventilations (15:2). If an advanced airway is in place, the compression-to-ventilation ratio of 15:2 does not apply because compressions and ventilations are delivered continuously with no interruptions. Remember for multiple-provider infant CPR, use the encircling thumbs technique to provide compressions.

It is the responsibility of the team leader to orchestrate movements between providers to ensure no one

provider becomes fatigued and that all critical areas are addressed when caring for children and infants. For example, additional providers may assimilate into roles of compressor or airway manager/ventilator to ensure that high-quality CPR is maintained (Figure 2-15).

> For single providers, the compression-to-ventilation ratio is 30:2. For multiple providers, the ratio changes to 15:2.

Caring for a Pediatric Patient with an Obstructed Airway

Airway obstructions are a common emergency. You need to assess the situation and recognize that 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.

Caring for a Responsive Child

A child who is choking typically has a panicked, confused or surprised facial expression. They may run about, flail their arms or try to get another's attention. The child may place one or both hands on their throat. This act of clutching the throat is commonly referred to as the universal sign of choking (Figure 2-16).

Figure 2-15 | High-performance resuscitation teams work together in a well-organized effort to provide high-quality CPR for children and infants.

Figure 2-16 | Clutching the throat with one or both hands is commonly referred to as the universal sign of choking.

You may hear stridor as the child tries to breathe, or you may hear nothing at all. Stridor is a high-pitched squeaking noise during inspiration. It is caused by narrowing or obstruction of the upper airway. Stridor is not exclusive to choking and may be a sign of another respiratory disorder (e.g., anaphylaxis, croup).

The child's skin may initially appear flushed, but it will become pale or cyanotic as the body is deprived of oxygen.

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

Performing Abdominal Thrusts

To perform abdominal thrusts:

- First, stand behind the child, with one foot in front of the other for balance and stability. If possible, place your front foot between the child's feet (Figure 2-17, A). If the child is in a wheelchair or is young, you may need to kneel behind them.
- Wrap your arms around the child'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 2-17, B).
- Make sure each thrust is a distinct attempt to dislodge the object.
- Continue delivering abdominal thrusts until the object is forced out and the child can cough, speak or breathe, or until the child becomes unresponsive.

Figure 2-17 | (A) Stand behind the patient, with one foot in front of the other and if possible, between the patient's feet. If the child is young, you may need to kneel behind them. (B) Place the thumb side of your fist against the middle of the abdomen, just above the navel, and grab the fist with your other hand.

Alternate Techniques

Evidence shows 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 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 back blows, chest thrusts or airway management techniques to dislodge the object from the airway. Remember, follow your facility's protocols when implementing alternate techniques.

Performing Back Blows

In some instances, back blows may be needed to relieve the obstruction. To use this option:

- Position yourself to the side and slightly behind the child. For a child in a wheelchair or a young child, you may need to kneel.
- Provide support by placing one arm diagonally across the child's chest.
- Then bend the child forward at the waist so that the child s upper body is parallel to the ground (or as close as it can be).
- Using the heel of your other hand, firmly strike the child between the scapulae (Figure 2-18).
- Make each blow a separate and distinct attempt to dislodge the object.

Practice Note

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.

Performing Chest Thrusts

To perform chest thrusts:

- Position yourself behind the child as you would for abdominal thrusts. If the patient is a young child or is in a wheelchair, you may need to kneel.
- Place the thumb side of your fist against the center of the child's chest on the lower half of the sternum.

Figure 2-18 | For effective back blows, bend the patient forward at the waist so that the patient's upper body is parallel to the ground. Give 5 firm back blows between the patient's scapulae.

Figure 2-19 | For effective chest thrusts, pull straight back, providing a quick inward thrust into the patient's chest.

- Then cover your fist with your other hand and pull straight back, providing a quick inward thrust into the child's chest (Figure 2-19).
- 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.

Caring for an Unresponsive Child

If a child 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, open the child's mouth and look for the object before attempting ventilations.

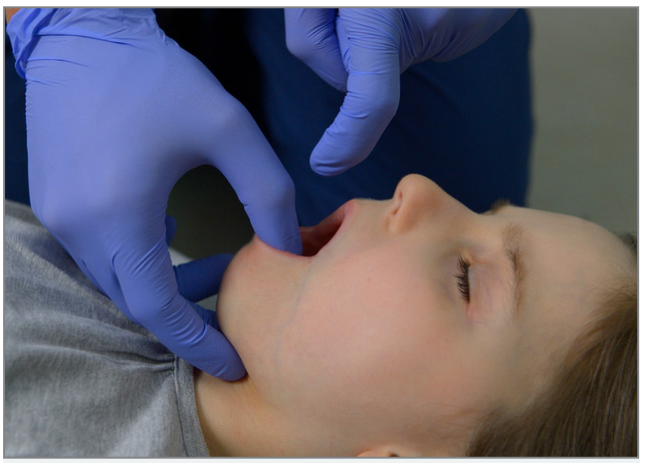


Figure 2-20 | After each set of compressions and before ventilations look for the object. Use a finger sweep to remove it, if seen.

- If you see the object in the child's mouth, remove it using a finger sweep (Figure 2-20). 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.

Caring for a Responsive Infant

If the infant is crying or coughing forcefully, allow the infant to keep coughing but be prepared to clear the infant's airway if the infant's condition changes.

If the infant is unable to cry or is coughing weakly, call for additional resources and to obtain an AED. Obtain consent from the parent or guardian. If the parent or guardian is not available, consent is implied.

Then, perform a series of 5 back blows and 5 chest thrusts.

Performing Back Blows

To deliver back blows:

- Place your forearm along the infant's back, cradling the back of the infant's head with your hand.
- Place your other forearm along the infant's front, supporting the infant's jaw with your thumb and fingers.
- Hold the infant in a face-down position along your forearm using your thigh for support and keeping the infant's head lower than their body (Figure 2-21).
- Use the heel of your hand to deliver a back blow between the infant's scapulae. Keep your fingers up to avoid hitting the infant's head or neck.
- Provide 5 firm back blows, with each one separate from the others

Figure 2-21 | For obstructive airway care, hold the infant in a face-down position along your forearm, using your thigh for support. Keep the infant's head lower than their body.

Performing Chest Thrusts

If back blows do not dislodge the object, try chest thrusts:

- Position the infant between your forearms, support the head and neck, and turn the infant face-up. Then lower the infant onto your thigh with their head lower than their chest.
- Place two fingers in the center of the infant's chest, just below the nipple line.
- Then give 5 quick chest thrusts about 1½ inches deep. Let the chest return to its normal position, keeping your fingers in contact with the breastbone. Each chest thrust should be separate from the others (Figure 2-22).
- Continue to provide sets of 5 back blows and 5 chest thrusts until the object is forced out and the infant can cough, cry or breathe or, the infant becomes unresponsive.

Figure 2-22 | Give 5 quick chest thrusts, about 1½ inches deep. Each should be separate from the others.

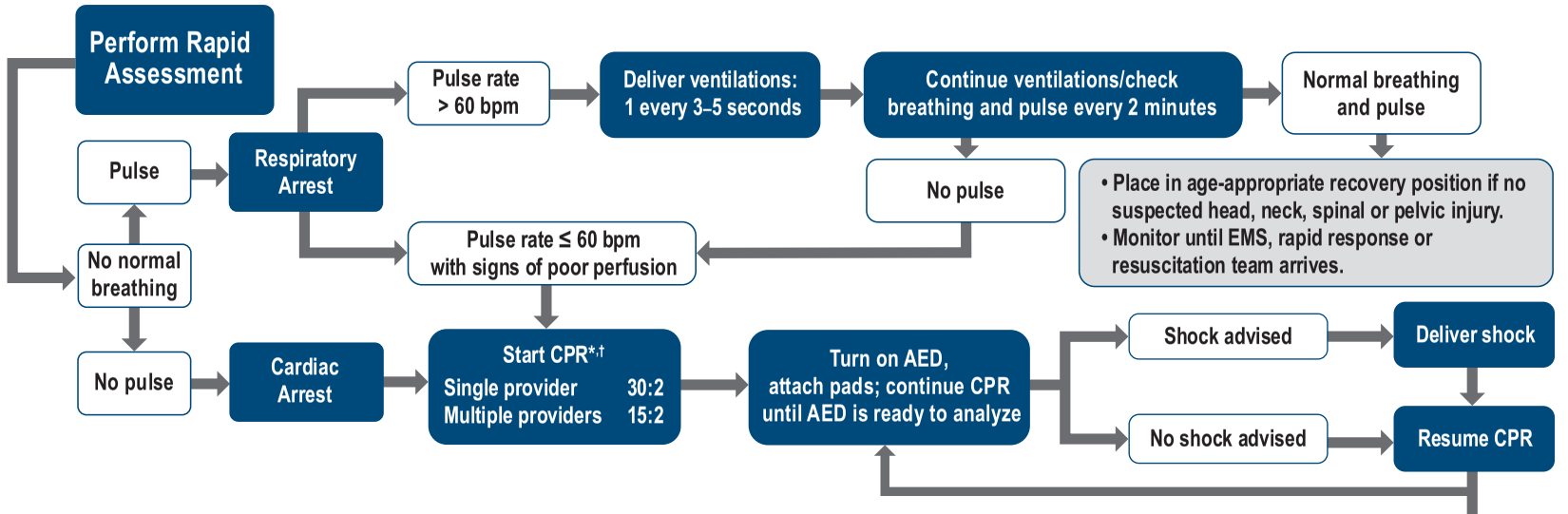
Caring for an Unresponsive Infant

If an infant becomes unresponsive while choking, provide care as you would for an unresponsive child who is choking. However, use your pinky to remove an object, if you can see it (Figure 2-23).

The *Obstructed Airway Care for Adults* and *Children and Obstructed Airway Care for Infants* Skill Sheets provide step-by-step guidance for performing care for pediatric patients with obstructed airways.

Figure 2-23 | For an infant, use your pinky to remove an object, if seen.

BLS: CHILDREN AND INFANTS



Indications of poor perfusion in a child or infant	• Cool moist skin • Mottling or cyanosis • Decreased capillary refill • Pallor • Weak or thready pulse • Hypotension	
CPR Technique	**Infant (< 1 year old)**	**Child (age 1 year to onset of puberty)**
Switch CPR compressors • Every 2 minutes • During AED analysis • If provider is fatigued	• **Hand position:** Fingers/thumbs centered on the lower half of the sternum just below the nipple line. Two-finger for single provider; encircling-thumbs for multiple providers • **Depth:** About 1½ inches • **Rate:** 100 to 120 per minute (single provider: 15 to 18 seconds for 30 compressions; multiple providers: 7 to 9 seconds for 15 compressions) • **Full chest recoil**	• **Hand position:** Centered on the lower half of the sternum • **Depth:** About 2 inches • **Rate:** 100 to 120 per minute (single provider: 15 to 18 seconds for 30 compressions; multiple providers: 7 to 9 seconds for 15 compressions) • **Full chest recoil**
	• Open airway to neutral position (avoid hyperextension)[a]. • Each ventilation should last about 1 second and make the chest begin to rise.	• Open airway to slightly past-neutral position (avoid hyperextension)[a]. • Each ventilation should last about 1 second and make the chest begin to rise.

[a]Use modified jaw-thrust technique instead if you suspect head, neck or spinal injury.

*If an advanced airway is in place, one provider delivers 1 ventilation every 6–8 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 15:2 for multiple-provider CPR 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 Children and Infants

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 child or infant, 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 child's or infant's name if you know it.
- Tap the *child's shoulder* or the *bottom of the infant's foot* and shout again (shout-tap-shout).
- If the child or infant 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 child or infant is unresponsive and you are with another provider, the first provider stays with the child or infant. Other providers activate EMS, the rapid response team or the resuscitation team, as appropriate, and retrieve the AED, BVM and other emergency equipment.

SKILL SHEET

Rapid Assessment for Children and Infants *(continued)*

Step 3 Simultaneously check for breathing and pulse

- Make sure the child or infant 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 *slightly past-neutral* position for a child or to a *neutral* position for an infant 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 a child or a *brachial* pulse for an infant for at least 5 seconds but no more than 10.

Step 4 Provide care based on the conditions found

- Obtain consent from a parent or legal guardian, if present.

SKILL SHEET

CPR for Children

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 child is unresponsive, isn't breathing normally and doesn't have a pulse, begin CPR.

Step 2 Place the child on a firm, flat surface

- In a healthcare setting, use a bed with a CPR feature, or place a CPR board under the child.
- 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 child to the floor or ground and kneel beside them.

Practice Note

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

SKILL SHEET

CPR for Children *(continued)*

Step 3 Position your hands correctly

- Expose the child's chest to ensure proper hand placement and visualize chest recoil.
- Place the heel of one hand in the center of the child'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 child's chest.
- Alternatively, for a small child, you may only need to use one hand, instead of two. Place the heel of one hand in the center of the child's chest.

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.

Step 5 Perform chest compressions

- For a child, compress the chest to a depth of about 2 inches (5 cm).
- 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.
- If you are a single provider, perform 30 chest compressions.
- If you are working with a team of providers, perform 15 chest compressions for a child.

SKILL SHEET

CPR for Children *(continued)*

Step 6 Seal the mask and open the airway

- Use an appropriately sized pocket mask for single-provider CPR or a BVM for multiple-provider CPR.
- Seal the mask and simultaneously open the airway to a slightly past-neutral position using the head-tilt/chin-lift technique. Avoid any hyperextension of flexion of the neck.
- Or, use the modified jaw-thrust maneuver if you suspect 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.

Practice Note

If an advanced airway is in place, one provider delivers 1 ventilation every 6 to 8 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 15:2 for multiple-provider CPR 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.

SKILL SHEET

CPR for Children *(continued)*

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

CPR for Infants

Step 1 Conduct a rapid assessment

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

Step 2 Place the infant on a firm, flat surface

- In a healthcare setting, use a crib with a CPR feature, or place a CPR board under the infant.
- Adjust the crib to an appropriate working height or use a step stool. Lower the crib side rail closest to you.
- In other settings, move the infant to a stable surface above the ground, such as a table or countertop.

Practice Note

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

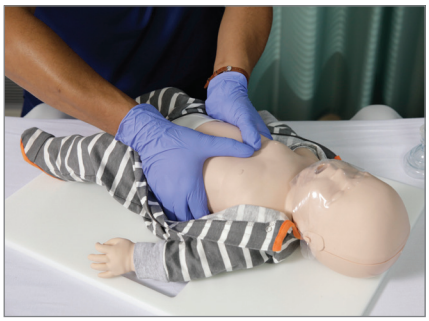
Step 3 Position your hands correctly

- Expose the infant's chest to ensure proper hand placement and visualize chest recoil.
- **If you are a single provider, use the two-finger technique:**
  - Stand to the side of the infant.
  - Place the two fingers of your hand closest to the infant's feet in the center of the exposed chest just below the nipple line on the sternum.
  - Use your index and middle fingers or your middle and fourth fingers to provide compressions. Fingers that are more similar in length tend to make compressions easier to deliver.

SKILL SHEET

CPR for Infants *(continued)*

- **If you are working with a team of multiple providers, use the encircling thumbs technique:**
  - Stand at the infant's feet.
  - Place both thumbs (side-by-side) on the center of the infant's chest just below the nipple line.
  - Then use the other fingers to encircle the infant's chest toward the back, providing support.



Step 4 Perform chest compressions

- For an infant, compress the chest to a depth of about 1½ inches (3.8 cm) or one-third the anterior-posterior diameter of the chest.
- 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 infant's chest at the top of the compression.
- If you are a single provider, perform 30 chest compressions. If you are working with a team of providers, perform 15 chest compressions for an infant.

Step 5 Seal the mask and open the airway

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

Step 6 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 Infants *(continued)*

Practice Note

If an advanced airway is in place, one provider delivers 1 ventilation every 6 to 8 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 15:2 for multiple-provider CPR does not apply because compressions and ventilations are delivered continuously with no interruptions.

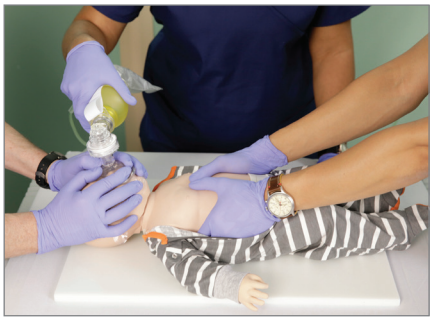
Step 7 Switch positions every 2 minutes

- 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.
- Remember, during multiple-provider CPR, the compressor will stand at the infant's feet, the ventilator will stand at the infant's side, and the provider maintaining the airway will stand at the infant's head.

Step 8 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 Children and Infants

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.

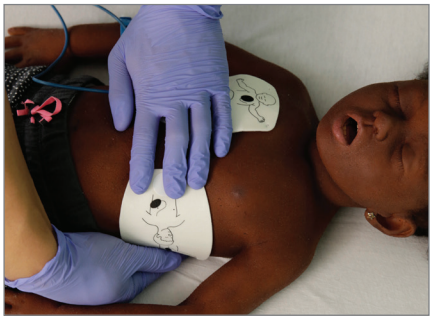
Step 2 Expose the chest

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

Step 3 Attach the pads

- **For infants up to 1 year old:**
  - Use pediatric pads if available. If pediatric pads *aren't* available—or the AED doesn't have a pediatric setting—it's safe to use adult AED pads or adult levels of energy.
  - Always use an anterior/posterior pad placement. To do this, apply one pad to the center of the infant's chest—on the sternum—and one pad to the infant's back between the scapulae.

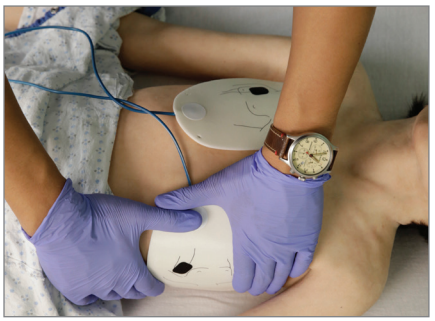
- **For children 8 or younger or weighing 55 pounds (25 kg) or less:**
  - Use pediatric pads if available. If pediatric pads *aren't* available—or the AED doesn't have a pediatric setting—it's safe to use adult AED pads or adult levels of energy.
  - Use an anterior/lateral placement, according to the manufacturer instructions: Place one pad to the right of the sternum and below the right clavicle. Place the other on the left side of the chest on the mid-axillary line, a few inches below the left armpit.
  - Or, use an anterior/posterior pad placement, if the AED pads risk touching each other on the child's chest or the manufacturer recommends.



SKILL SHEET

AED Use for Children and Infants *(continued)*

- **For children older than 8 years or weighing more than 55 pounds (25 kg):**
  - Use adult AED pads.
  - Use an anterior/lateral or anterior/posterior placement, according to manufacturer instructions.



Alert

Never use pediatric AED pads or a pediatric electrical setting on a child older than 8 years or weighing more than 55 pounds (25 kg). That's because the shock delivered will not be sufficient. In these cases, always use adult AED pads and energy levels.

Practice Note

Some AEDs come with pediatric AED pads. These are smaller and designed specifically to deliver a lower level of energy. Also, some AEDs use a switch or key on the device itself instead of changing pads.

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.

SKILL SHEET

AED Use for Children and Infants *(continued)*

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 continues 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.

SKILL SHEET

Obstructed Airway Care for Adults and Children

Step 1 Verify the patient is choking

- If the patient is able to speak to you or is coughing forcefully: Encourage the patient to keep coughing but be prepared to clear the airway if the patient's condition changes.
- If the patient is unable to speak to you or is coughing weakly: Call for additional resources. Continue to Step 2.

Step 2 Obtain consent

- **For the adult**: Obtain consent from the patient.
- **For the child**: Obtain consent from the parent or legal guardian if present. If they're not available, consent is implied.

Step 3A Perform abdominal thrusts

- First, stand behind the patient, with one foot in front of the other for balance and stability. If possible, place your front foot in between the patient's feet.
- If the patient is a young child or is in a wheelchair, you may need to kneel.
- Then, get your hands in place. Using one or two fingers to find the patient's navel, make a fist with your other 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.
- Give quick inward and upward thrusts.
- Be sure to make each thrust a distinct attempt to dislodge the object.
- If abdominal thrusts do not dislodge the object, continue to Step 3B.

SKILL SHEET

Obstructed Airway Care for Adults and Children *(continued)*

Step 3B Perform alternate techniques

Perform alternate techniques—back blows, chest thrusts or airway management techniques if:

- You cannot reach far enough around the patient.
- They might be pregnant.
- They are in a bed or in a wheelchair with features that make abdominal thrusts difficult to do.
- Abdominal thrusts are not effective in dislodging the object.

Note: Remember to always follow your facility's protocol when implementing alternate techniques.

Back Blows

- Position yourself to the side and slightly behind the patient.
- Provide support by placing one arm diagonally across the patient's chest.
- Then bend the patient forward at the waist so the 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 patient's scapulae. Make each blow a separate and distinct attempt to dislodge the object.
- If back blows do not dislodge the object, use a series of 5 back blows and 5 abdominal or chest thrusts.

Chest Thrusts

- Position yourself behind the patient as you would for abdominal thrusts. If the patient is a young child or is in a wheelchair, you may need to kneel.
- 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.
- If chest thrusts do not dislodge the object, use a series of 5 back blows and 5 chest thrusts.

SKILL SHEET

Obstructed Airway Care for Adults and Children *(continued)*

Airway Management Techniques

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

Step 4 Continue to clear the airway

Continue to clear the airway until:

- The object is forced out.
- The patient can cough forcefully, speak, cry or breathe.
- The patient becomes unresponsive.

Practice Note

If the patient becomes unresponsive, carefully lower them to a firm, flat surface, while protecting their head. Then, call for additional resources and to get an AED (if appropriate and you have not already done so). Immediately begin CPR, starting with chest compressions. After each set of compressions and before ventilations, open the patient's mouth and look for the object—if seen, remove it using a finger sweep. 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 performing cycles of 30 compressions and 2 ventilations, checking for an object before each set of ventilations.

SKILL SHEET

Obstructed Airway Care for Infants

Step 1 Verify that the infant is choking

- If the infant is crying or coughing forcefully: Allow the infant to keep coughing but be prepared to clear the infant's airway if the infant's condition changes.
- If the infant is unable to cry or is coughing weakly: Call for additional resources. Continue to Step 2.

Step 2 Obtain consent

- Obtain consent from the parent or legal guardian if present. If they're not available, consent is implied.

Step 3 Position the infant for back blows

- Place your forearm along the infant's back, cradling the back of the infant's head with your hand.
- Place your other forearm along the infant's front, supporting the infant's jaw with your thumb and fingers.
- Hold the infant in a face-down position along your forearm using your thigh for support and keeping the infant's head lower than their body.

Step 4 Deliver 5 back blows

- Use the heel of your hand to deliver back blows between the infant's scapulae. Keep your fingers up to avoid hitting the infant's head or neck.
- Provide 5 firm back blows, with each one separate from the others.

SKILL SHEET

Obstructed Airway Care for Infants
(continued)

Step 5 Deliver 5 chest thrusts

If back blows don't dislodge the object, perform chest thrusts:

- Position the infant between your forearms, support the head and neck, and turn the infant face-up.
- Then lower the infant onto your thigh with their head lower than their chest.
- Now, place two fingers in the center of the infant's chest, just below the nipple line.
- Next deliver 5 quick chest thrusts about 1½ inches deep. Let the chest return to its normal position, keeping your fingers in contact with the breastbone. Each chest thrust should be separate from the others.

Step 6 Continue to clear the airway

Continue to provide sets of 5 back blows and 5 chest thrusts until:

- The object is forced out.
- The infant can cough, cry or breathe.
- The infant becomes unresponsive.

Practice Note

If the infant becomes unresponsive, carefully position them on a firm, flat surface while protecting their head.
Then, call for additional resources and to get an AED (if appropriate and you have not already done so). Immediately begin CPR, starting with chest compressions. After each set of compressions and before ventilations, open the infant's mouth and look for the object—if seen, remove it using a finger sweep. 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 performing cycles of compressions and ventilations (using a ratio of 30:2 if a single provider and 15:2 if working with a team), checking for an object before each set of ventilations.

CHAPTER 3

Tools and Therapies

Introduction

As a member of a high-performance rapid response or 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 respiratory emergency, shock or a cardiac emergency. This chapter reviews equipment and interventions commonly used to assess and stabilize an acutely ill patient.

Airway Management

Airway management encompasses a variety of maneuvers, ranging from noninvasive to increasingly invasive. Often, these interventions occur simultaneously or in rapid succession. Multiple factors will determine which interventions to undertake in the child requiring airway support, the primary determinant being the condition of the patient. Other factors include the expertise of the provider and the availability of necessary resources.

Opening the Airway

To open the airway manually, perform the head-tilt/chin-lift technique or use a modified-jaw thrust maneuver if you suspect head, neck or spinal injury (providing you can effectively maintain an open airway). The *Opening the Airway* Skill Sheet provides step-by-step guidance for opening the airway using the head-tilt/chin-lift technique or the modified jaw-thrust maneuver.

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.

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.

Practice Note

If possible, limit suction intervals to 10 seconds to avoid mucosal damage and prolonged hypoxia.

The *Suctioning the Airway* Skill Sheet provides step-by-step guidance for suctioning the airway.

Basic Airways

When manual maneuvers are unsuccessful, an airway adjunct, namely, an oropharyngeal airway (OPA) or nasopharyngeal airway (NPA), can be used to open the airway as long as the obstruction is above the glottis (i.e., larynx).

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 modified jaw-thrust maneuver.

Oropharyngeal Airway

An OPA is a curved plastic device that 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). OPAs are available in a range of sizes. It is important to determine the correct size to use based on proper measurement.

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 *Basic Airway Insertion* Skill Sheet provides step-by-step guidance for inserting an OPA.

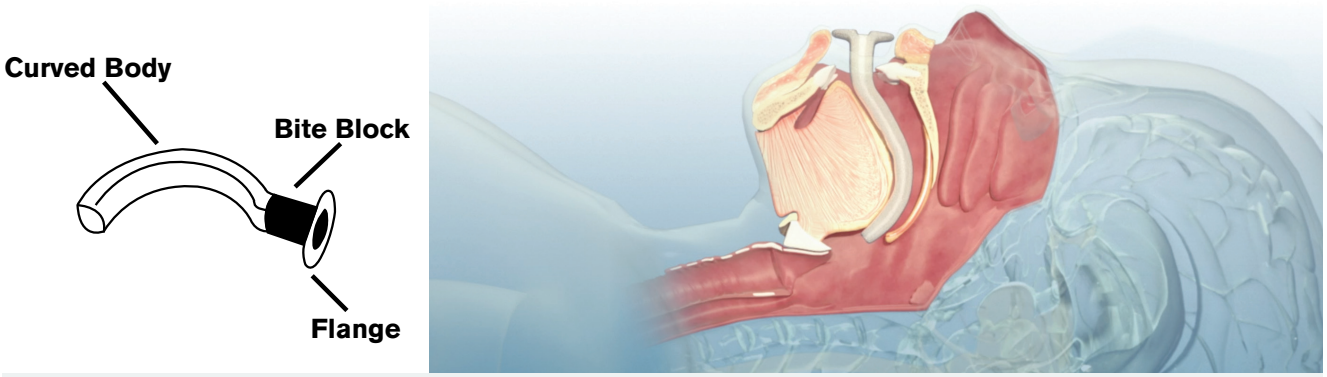


Figure 3-1 | Oropharyngeal airway

Nasopharyngeal Airway

An NPA is a soft rubber tube with a flange on one end and beveled tip on the other. NPAs are available in a range of sizes and diameters. It is important to determine the correct size to use based on proper measurement. 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.

The *Basic Airway Insertion* 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 an 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. Supraglottic airways, such as laryngeal mask airways (LMAs), do not pass through the vocal cords, whereas transglottic airways, such as endotracheal tubes (ETTs), do. The choice of airway depends on the patient's condition, the available

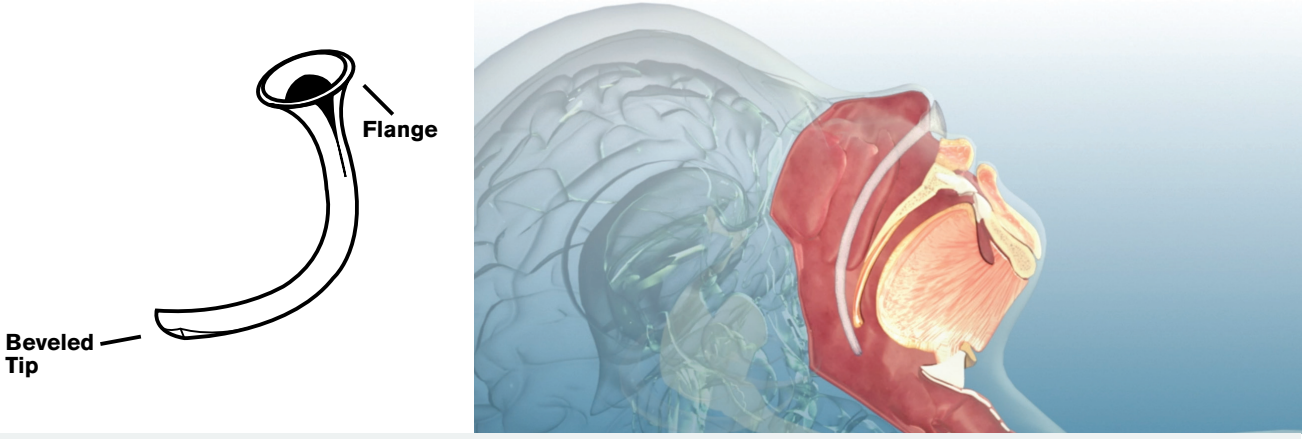


Figure 3-2 | Nasopharyngeal airway

resources, and the provider's capabilities and scope of practice.

Laryngeal Mask Airway

The LMA consists of an airway tube and a mask with an inflatable cuff at the distal end (Figure 3-3). LMAs come in a variety of sizes. It is important to determine the correct size to use based on the patient's age and weight to ensure easy placement and a proper seal.

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.

Endotracheal Intubation

Use of an endotracheal intubation 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 may be required. Endotracheal intubation also protects the airway from aspiration of gastric contents, a protection that cannot be assured with supraglottic airways.

Endotracheal intubation involves the placement of an ETT through the vocal cords and into the trachea. The ETT may be inserted through the mouth (i.e., orotracheal intubation) or the nose (i.e., nasotracheal intubation). The orotracheal route is used more often as it is faster and associated with fewer complications. Endotracheal intubation is usually facilitated by direct visualization of the airway using a laryngoscope equipped with a light source.

The endotracheal tube consists of an airway tube with or without 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 (Figure 3-4).

ETTs come in a variety of sizes. It is important to determine the correct size to use based on the patient's age and weight to ensure easy placement and a proper seal. Using a length-based resuscitation tape provides the most accurate estimate. Consider using a smaller ETT if upper airway obstruction is anticipated. In all cases, you should have tubes that are 0.5 mm larger and smaller than the preselected size on standby.

Some ETTs incorporate a cuff, which is a small inflatable balloon at the end of the tube. The cuff adds to the ETT diameter and should be tested for leaks before insertion. Uncuffed tubes have historically been used in infants and young children with the intent of avoiding excessive pressure in the subglottic area; however, cuffed tubes are increasingly being used in these populations. A cuffed tube should be used instead of an uncuffed tube when the need for higher ventilatory pressures is expected.

Laryngoscope blades (Figure 3-5) 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 light bulbs on the selected and backup blades by attaching the blade to the laryngoscope handle. Tighten each light bulb to avoid dislodgement during intubation.

The laryngoscope blade is used to displace the epiglottis and the endotracheal tube is passed through the vocal

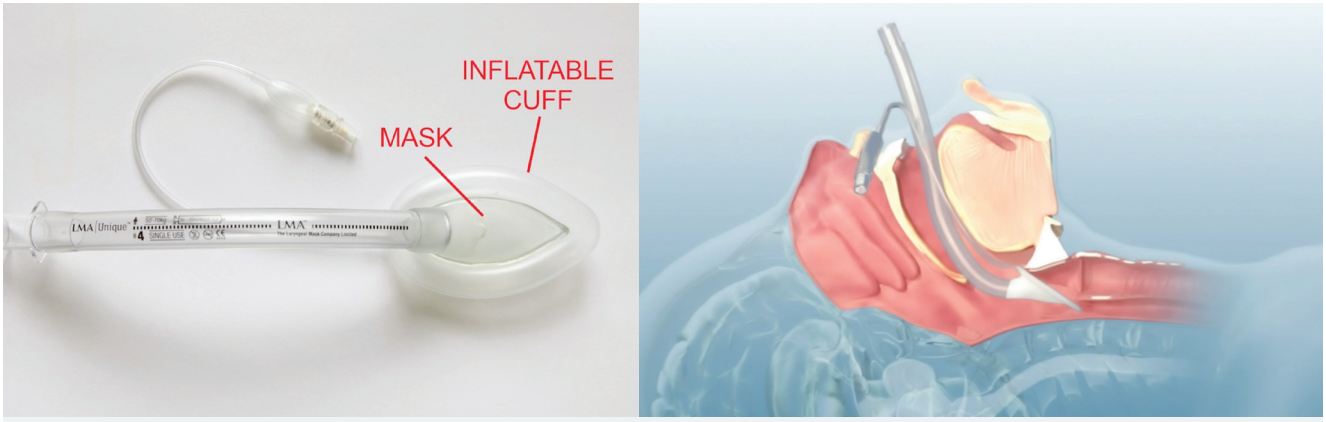


Figure 3-3 | Laryngeal mask airway

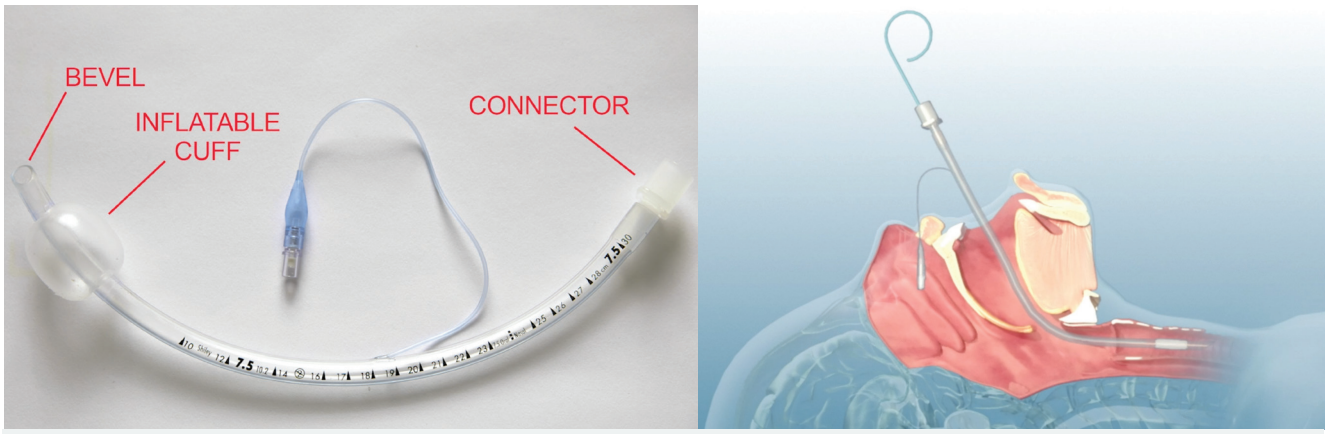


Figure 3-4 | Endotracheal tube

cords and into the trachea. (Note: If using a straight blade, the epiglottis is lifted upward. If using a curved blade, the end of the blade is inserted into the vallecula and the laryngoscope handle is gently "lifted" upward and forward). When the endotracheal tube is properly inserted and the cuff is inflated, the cuff forms a seal against the walls of the trachea.

Confirming Advanced Airway Placement

Whenever an advanced airway is used, you must confirm proper placement immediately after inserting it, 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. In addition, for an endotracheal tube, it is important to verify correct placement with a chest radiograph or ultrasound, once available.

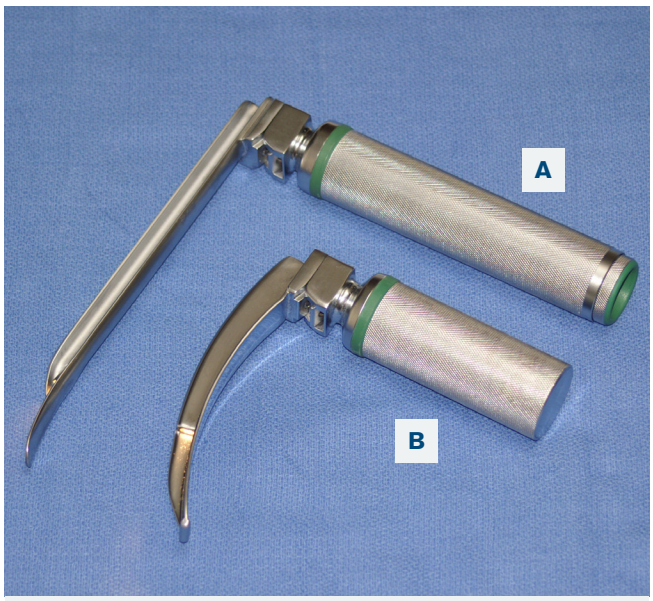


Figure 3-5 | Laryngoscope blades. (A) Straight (Miller) blade. (B) Curved (Macintosh) blade.

Practice Note

Nonwaveform exhaled carbon dioxide detectors may not be 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. First, look for bilateral chest rise with ventilations. The chest should rise evenly on both sides. Auscultate the lungs for bilateral breath sounds, then auscultate over the epigastric region. You should hear bilateral breath sounds, and there should be no gurgling over the epigastrium.

Capnography

Capnography is considered a very 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 one 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.

Monitoring the Patient With an Advanced Airway

Monitor the patient on an ongoing basis. 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. It is also important to suction as necessary.

If a patient with an advanced airway experiences sudden deterioration, use the DOPE mnemonic to troubleshoot potential causes:

- **D**isplacement of the breathing tube (or other advanced airway) from the trachea
- **O**bstruction of the breathing tube or other advanced airway
- **P**neumothorax
- **E**quipment failure

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 oxygenation, 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 3-6). Some BVMs for children and infants also include a pressure relief or "pop-off" valve that helps to prevent excessive pressure during ventilations. In a resuscitation situation it is essential to deactivate the pop-off valve to assure adequate ventilation.

Squeezing the bag with the mask properly sealed over the patient's mouth and nose forces air into the lungs (positive pressure ventilation). 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.

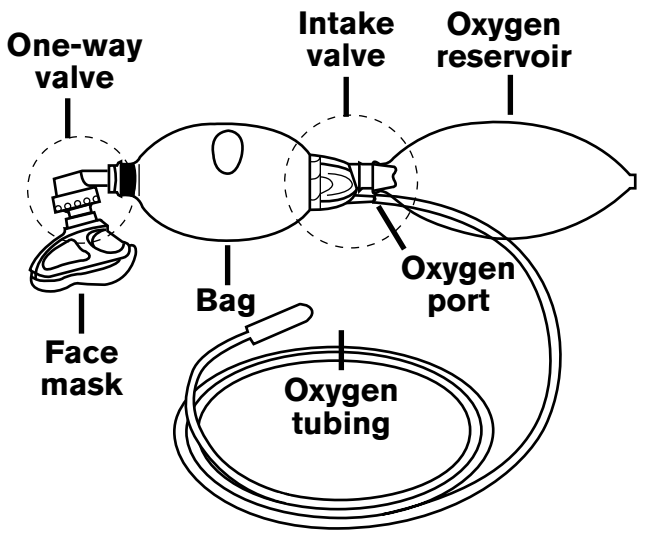


Figure 3-6 | Parts of a bag-valve-mask (BVM) resuscitator

Practice Note

Supplemental oxygen should be attached to the BVM as soon as appropriate. Doing so can increase the oxygen concentration to approximately 90% to 100%.

- BVM with ambient air: 20% to 21%
- BVM with supplemental oxygen: 90% to 100%

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. It is possible to provide BVM ventilations to patients with tracheostomy tubes. See *Learn More: BVM Ventilation and Tracheostomy Tubes*.

The *Using a Bag-Valve-Mask (BVM) Resuscitator* Skill Sheet provides step-by-step guidance for using a BVM resuscitator.

LEARN MORE

BVM Ventilation and Tracheostomy Tubes

If the patient who requires BVM ventilation has a tracheostomy tube, it is possible to provide BVM ventilations via the tracheostomy tube. Attach the BVM directly to the tracheostomy tube (without the mask) and administer ventilations. Watch the patient's chest for expansion to determine effectiveness of ventilations. If ventilations are not effective, try to suction the tracheostomy tube. If you can't suction the tracheostomy tube, it may be necessary to replace it. In patients with tracheostomies who present with respiratory compromise, it is especially important to establish that the tracheostomy tube is not blocked or displaced.

Supplemental Oxygen

The administration of supplemental oxygen is often indicated for patients experiencing a respiratory emergency, shock or a cardiac 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.

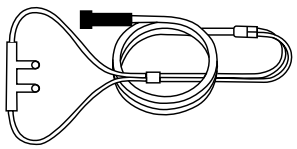
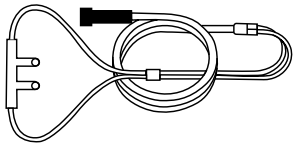
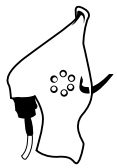
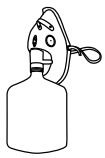
Oxygen Delivery Devices

For patients who are spontaneously breathing, oxygen can be delivered by nasal cannula or by various types of masks. The maximum flow rate and concentration of oxygen that can be achieved varies according to the delivery device (Table 3-1). Therefore, the best method for oxygen delivery for any given patient will depend on the degree of oxygen desaturation. Other factors that may influence the choice of oxygen delivery method include equipment availability and patient comfort.

Nasal Cannula

Traditionally, oxygen flow rates via nasal cannula have been limited to 1 liter per minute (LPM) in newborns and 2 LPM in children, to avoid drying of the nasal mucous membranes and other complications. Flow rates in these ranges generally yield oxygen concentrations of less than 50%, the lowest among all oxygen delivery options

Table 3-1 | Specifications for Oxygen Delivery Device and Type

Delivery Device	Oxygen Flow Rate, LPM	Oxygen Concentration[a], %
Nasal cannula Low-flow oxygen device 	1–4	24–44
High-flow nasal cannula (HFNC) High-flow oxygen device 	Up to 2 L per kg of body weight/min **Note:** Must be used with humidification	>45
Simple mask Low-flow oxygen device 	6–15	>35–55
Non-rebreather mask High-flow oxygen device 	10–15	>60–90

[a]Mean oxygen concentration; varies based on exact LPM, respiratory rate, and whether mouth is open or closed.

Note: It is a good idea to use humidification for all oxygen delivery devices.

for spontaneously breathing patients. Higher oxygen concentrations may be achieved with high-flow nasal cannula (HFNC) oxygen.

High-flow nasal cannula was originally introduced as a method for providing continuous positive airway pressure (CPAP) in preterm infants, but it is increasingly being used in older children and adults with hypoxemic **respiratory failure**. Indeed, HFNC delivery has been shown to avert the need for intubation in infants with bronchiolitis, supporting the utility of this method for generating positive airway pressure as well as improving oxygenation. Oxygen flow rates of up to 2 L per kilogram of body weight per minute are used. Because of the high flow, humidification must be used in concert with HFNC. Though HFNC oxygen is a safe option for children treated in a variety of settings, abdominal distension and anecdotal reports of pneumothorax have been described.

Simple 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%.

Simple oxygen masks can be used to provide oxygen via the “blow by” technique—an effective option for

young children and infants who are fearful of having a mask placed directly on their face. To perform this technique: hold the mask about 2 inches from the child's face and move it slowly from side-to-side (Figure 3-7). This allows the patient to inhale the oxygen as it passes over their face.

Non-Rebreather Mask

A non-rebreather mask is used to deliver high concentrations of oxygen to a breathing patient (more than 60%, and as high as 90%). 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.

Supplemental oxygen can be delivered via a tracheostomy. See *Learn More: Oxygen Delivery When the Child or Infant Has a Tracheostomy.*

Pulse Oximetry

Oxygenation is most readily assessed by using pulse oximetry, which noninvasively measures oxygen saturation (SpO_2) of hemoglobin. Oxygen therapy is typically titrated to achieve an oxygen saturation of >94% on pulse oximetry in the absence of cyanotic heart disease or chronic hypercarbia. Target SpO_2 levels are typically lower for preterm and term neonates. Hyperoxemia should be avoided in all patients.

Figure 3-7 | Simple oxygen masks can be used to provide oxygen via the "blow by" technique.

LEARN MORE

Oxygen Delivery When the Child or Infant Has a Tracheostomy

When working with pediatric patients who have tracheostomies, supplemental oxygen can be directly delivered via the tracheostomy. Oxygen is delivered via a tracheostomy mask (low- and high-flow) or a heat and moisture exchanger (low-flow); the latter attaches to both the tracheostomy tube and an oxygen source and humidifies the oxygen being delivered to the airway. Suctioning is critical in patients with tracheostomies to remove any secretions and ensure patency and proper positioning of the tracheostomy tube.

To establish that the tracheostomy tube is not blocked or displaced, a soft suction catheter should be passed through the tracheostomy tube to assess patency and placement. If the suction catheter passes easily through the tip of the tracheostomy tube into the trachea, patency and proper placement are confirmed. If the suction catheter does not pass, the tracheostomy tube should be removed and replaced immediately. If the tracheostomy tube cannot immediately be replaced, oxygen can be applied to the face and stoma.

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 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.

The *Measuring SpO_2 Levels by Pulse Oximetry* Skill Sheet provides step-by-step guidance for measuring SpO_2 levels using pulse oximetry.

Capnography

Capnography, which measures end-tidal carbon dioxide ($ETCO_2$) levels and is expressed as a value and as a

waveform, is useful for assessing the severity of the patient's clinical condition, discerning the underlying pathophysiology and evaluating the patient's response to interventions.

There are a couple different ways to measure the end-tidal carbon dioxide level. An adapter can be attached to a BVM or advanced airway or a nasal cannula can be applied under a mask.

In pediatric emergencies, capnography is used to help confirm proper placement of advanced airways, namely transglottic airways that pass through the vocal cords. In addition, it is used to monitor effectiveness of compressions during cardiac arrest ($ETCO_2$ levels between 15 and 20 mmHg indicate effective compressions) and, a sudden spike in end-tidal carbon dioxide level is often the first sign of ROSC (>45 mmHg).

In addition, capnography can be used to avoid hyper- and hypoventilation in the peri- and post-arrest patient. It is important to monitor $ETCO_2$ values with each breath. Values in the range of 35 to 45 mmHg indicate proper ventilation and perfusion.

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 3-8).

- **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.

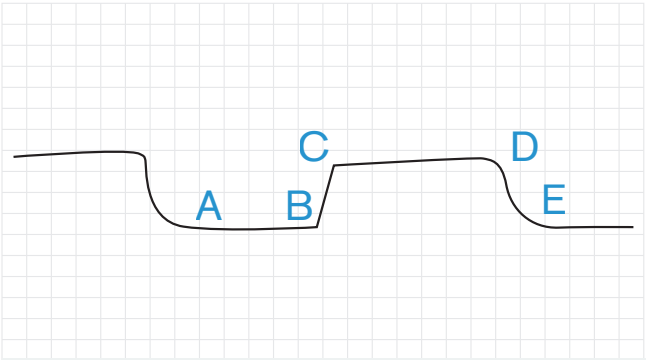


Figure 3-8 | Phases of the normal capnography waveform. **A–B:** respiratory baseline; **B–C:** respiratory upstroke; **C–D:** expiratory plateau; **D–E:** inspiratory downslope.

- **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.
- **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 3-9, A).
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 (Figure 3-9, B).

4. **Look at the expiratory plateau.** Is the expiratory plateau flat? Loss of plateau (Figure 3-9, C) 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, 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.

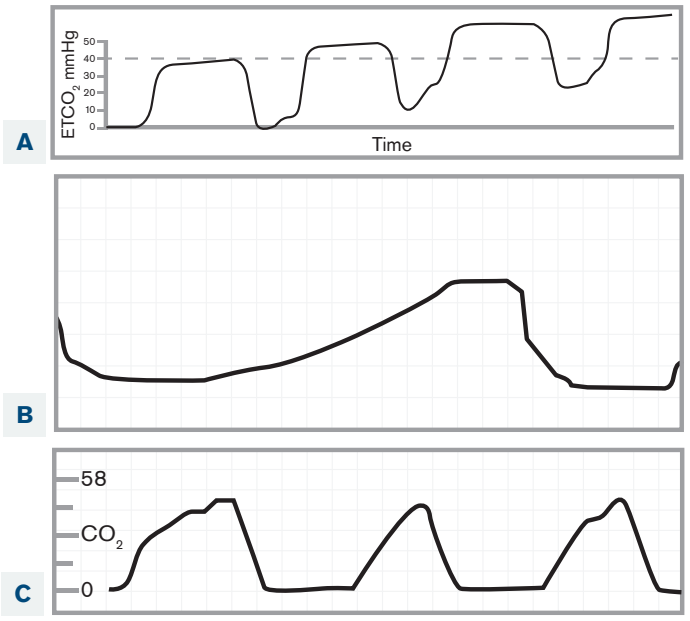


Figure 3-9 | Abnormal capnography waveform morphology. (A) Upward sloping respiratory baselines indicate that the patient is rebreathing carbon dioxide. (B) A sloping, prolonged respiratory upstroke indicates uneven alveolar emptying. (C) A loss of expiratory plateau indicates bronchospasm and air trapping.

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).

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 3-10) 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 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.

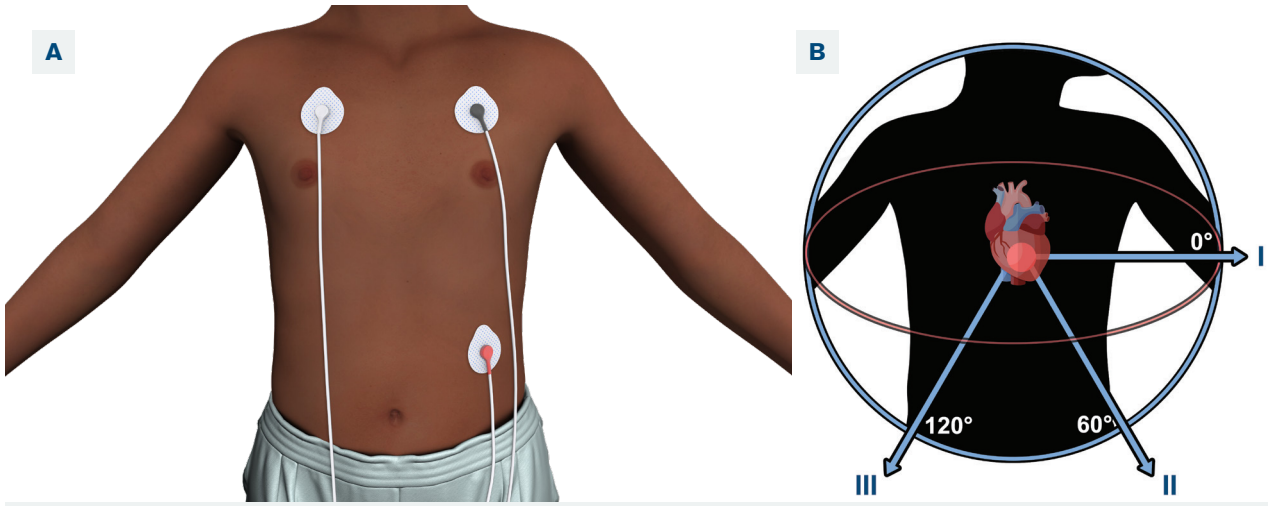


Figure 3-10 | Three-electrode system. (A) The white electrode is placed under the right clavicle at the midclavicular line, the black electrode is placed under the left clavicle at the midclavicular line, and the red electrode is placed on the lower left abdomen. (B) The three-electrode system allows monitoring of leads I, II and III.

12-Lead Electrocardiogram

The heart is a three-dimensional organ with surfaces on multiple planes, and its center axis is tilted at an angle within the chest cavity. Therefore, for a more comprehensive and detailed view of the whole heart, and to identify potentially serious and life-threatening damage or conditions that may not present in a single lead, multiple angles and perspectives are needed. The 12-lead ECG provides a full picture of the rate and rhythm of the heart.

Most cardiac monitors/defibrillators can also be used to obtain a 12-lead ECG, which is necessary for accurately diagnosing arrhythmias 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 electrical activity of the heart (Figure 3-11). The four limb electrodes produce six views in the frontal plane: I, II, III, aVR, aVL and aVF. The six chest electrodes produce six views in the horizontal plane: V_1, V_2, V_3, V_4, V_5 and V_6. When analyzed in totality, these 12 unique views of the heart give the provider a substantial

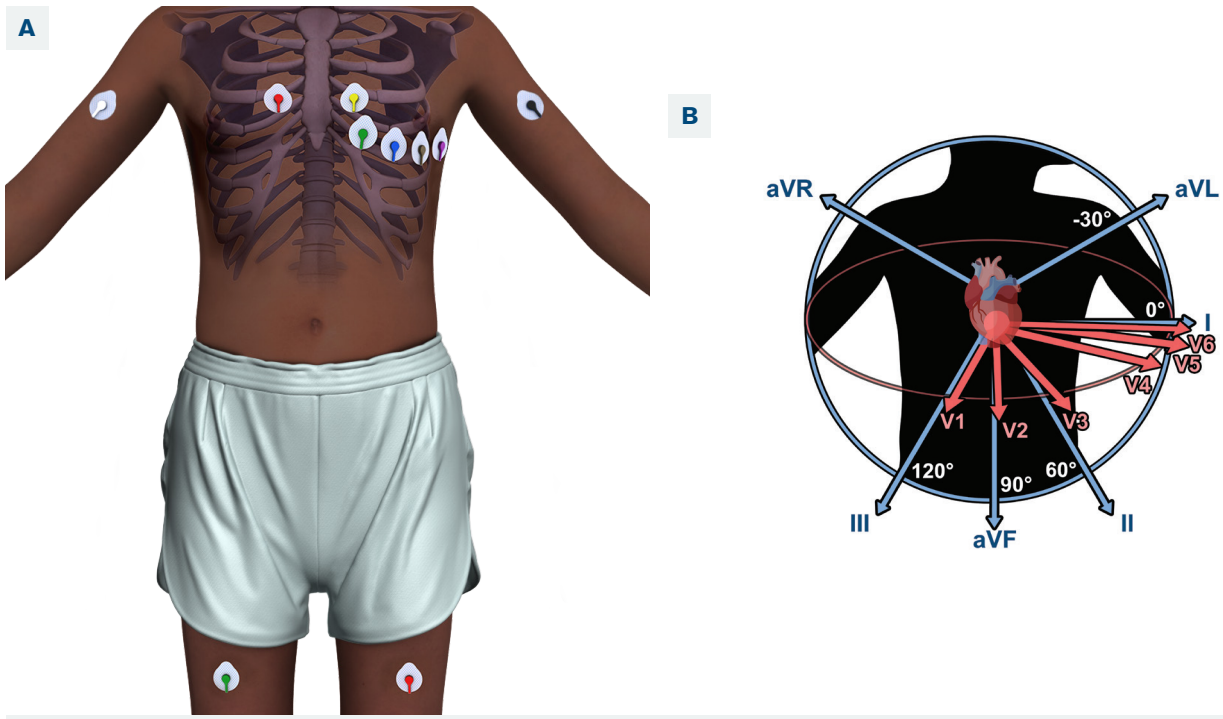


Figure 3-11 | The 12-lead ECG uses (A) four limb electrodes and six chest electrodes to provide (B) 12 views of the electrical activity of the heart.

amount of information regarding both electrical conduction and possible abnormalities or damage within the myocardium.

Practice Note

In some clinical situations, it may be necessary to obtain a 15-lead ECG, which is done by obtaining a 12-lead ECG and then repositioning some of the electrodes to obtain the additional three views.

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 inaccurate rhythm identification and misdiagnosis.

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

Reading the ECG Rhythm Strip

Rhythm Strip Components

Each heart beat consists of several electrical events within a single cardiac cycle. Most of these events are represented on the rhythm strip (Figure 3-12).

- **P wave**: The P wave represents depolarization of the atria. This event leads to contraction of the atria, though the actual contraction is not seen on the ECG.
- **PR interval**: The PR interval measures 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. It reflects the delay in conduction as the electrical impulse traverses the AV node, which allows the atria to finish contracting before the start of ventricular contraction.
- **QRS complex**: The QRS complex represents depolarization of the ventricles. This event leads to contraction of the ventricles, though the actual contraction is not seen on the ECG.

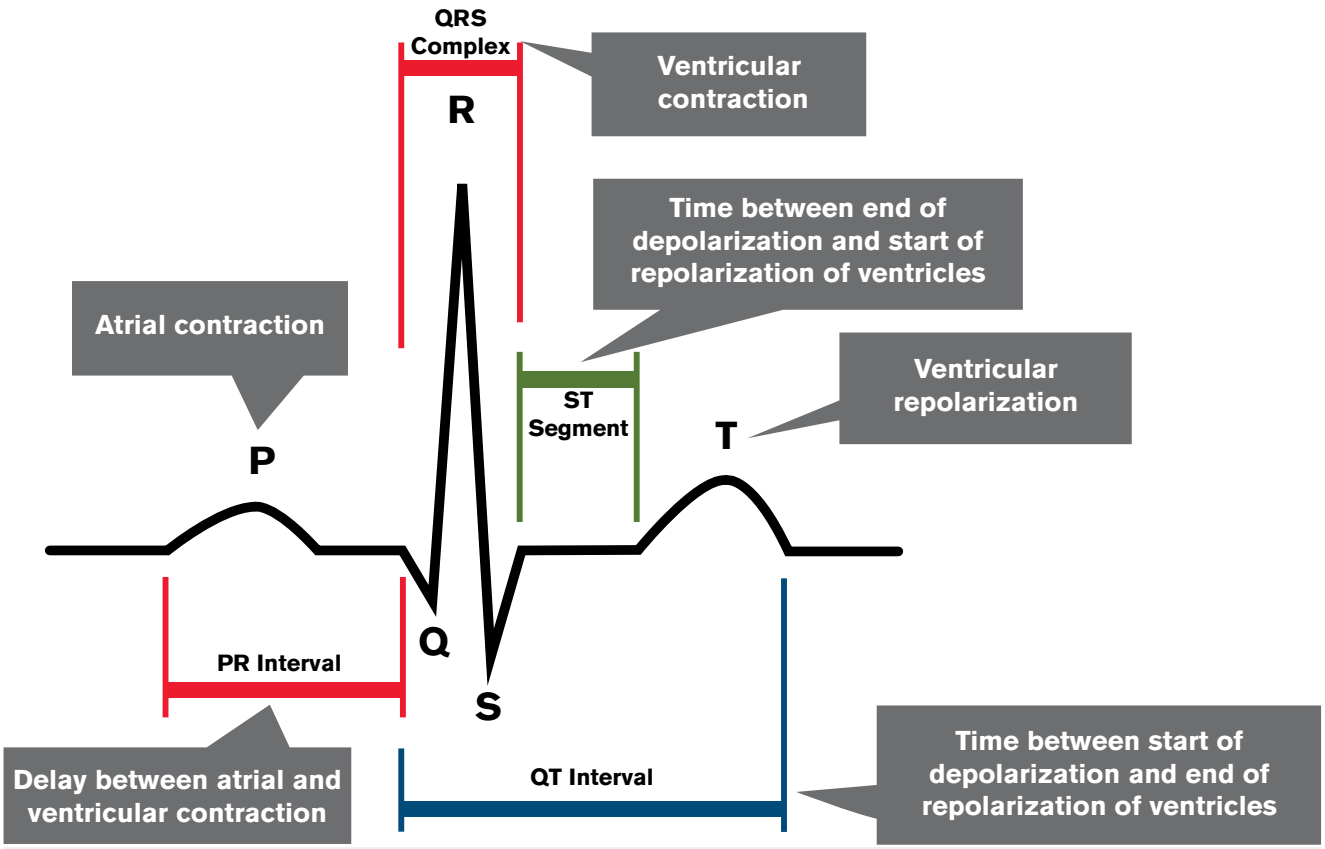


Figure 3-12 | ECG components and their correlation with electrical conduction

- **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.
- **T wave**: The T wave is a recording of the rest and repolarization of the ventricles. (Note: Atrial repolarization occurs during ventricular depolarization and is not represented on the ECG.)
- **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 ventricular depolarization to the end of repolarization.

Interpreting the Rhythm Strip

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

1. **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?
2. **Rhythm:** Look at the rhythm to see if 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 R-R interval)?
3. **P waves:** Are P waves present? Do they all have the same morphology? Is there a 1:1 ratio between the number of P waves and QRS complexes, or are there more P waves than QRS complexes?
4. **PR interval:** Measure the PR interval (Figure 3-13 A). Is it within the normal range for the patient's age? (See Table 3-2.) Is it consistent throughout the strip? If it varies, does the variation occur in a certain pattern?
5. **QRS complex:** Does the QRS complex appear narrow or wide? Measure the duration of the QRS complex (Figure 3-13 B). Count the number of small squares and multiply by 0.04 to get the total duration in seconds. Is the duration within the normal range for age? (See Table 3-2.) Do all the QRS complexes have the same morphology?

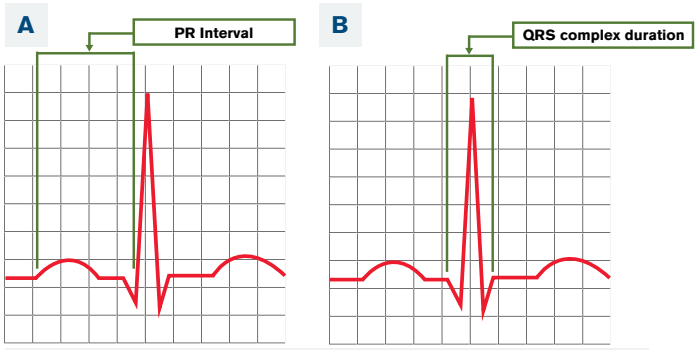


Figure 3-13 | (A) Measuring the PR interval. (B) Measuring the QRS complex duration.

6. **QT interval:** Measure the QT interval (Figure 3-14) and calculate the corrected QT interval (QTc) using a consistent lead. Because the QT interval varies normally with the heart rate, the QTc is used to assess absolute QT prolongation. The QTc adjusts for heart rate differences by dividing the QT interval (in seconds) by the square root of the R-R interval (i.e., one cardiac cycle).

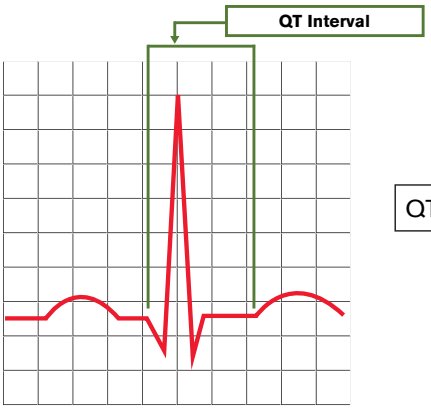
   Normal values for QTc for children are as follows:

   - 0.44 seconds = 97th percentile for infants 3 to 4 days old
   - ≤0.45 seconds for all boys >1 week of age and for all prepubescent girls
   - ≤0.46 seconds for postpubescent girls

   If the heart rate is greater than 120 beats per minute or less than 50 beats per minute, the formula for calculating QTc has limited usefulness.
7. **Clinical significance:** Determine the rhythm and its clinical significance. Is the patient showing signs or symptoms of arrhythmia? Is the rhythm potentially life threatening?

Table 3-2 | Normal Age-Based ECG Parameters for Children

Age	PR Interval, Mean (Range), seconds	QRS Duration, Mean (98th Percentile), seconds
0–7 d	0.10 (0.08–0.12)	0.05 (0.07)
1–3 wk	0.10 (0.08–0.12)	0.05 (0.07)
1–6 mo	0.11 (0.08–0.13)	0.05 (0.07)
6–12 mo	0.12 (0.10–0.14)	0.05 (0.07)
1–3 y	0.12 (0.10–0.14)	0.06 (0.07)
4–5 y	0.13 (0.11–0.15)	0.07 (0.08)
6–8 y	0.14 (0.12–0.16)	0.07 (0.08)
9–11 y	0.14 (0.12–0.17)	0.07 (0.09)
12–16 y	0.15 (0.12–0.17)	0.07 (0.10)
>16 y	0.15 (0.12–0.20)	0.08 (0.10)



$$QTc = QT \div \sqrt{R\text{-}R}$$

Figure 3-14 | Measuring the QT interval and calculating the QTc

Vagal Maneuvers

Vagal maneuvers are an initial treatment option in children with hemodynamically stable supraventricular tachycardia (SVT). Studies in children and adults have shown an approximately 25% success rate for vagal maneuvers, though reported rates vary widely (from 6% to 54%). These maneuvers are intended to increase parasympathetic, or vagal, tone in an effort to slow down conduction through the atrioventricular (AV) node of the heart. This action interrupts the "circuit" that precipitates SVT in most cases.

Various vagal maneuvers are appropriate for use in children. There are no guidelines that specify how many vagal maneuver attempts to make before moving on to other therapies; however, most providers will make a maximum of two attempts.

Vagal maneuvers include various Valsalva maneuvers. Because Valsalva maneuvers require patient cooperation, they are best used with older children. There are several ways to perform Valsalva maneuvers. A few common ways include:

- Instructing the child to close the mouth and bear down as if they are having a bowel movement.
- Instructing the child to pretend that they are blowing up an imaginary balloon (Figure 3-15).
- Instructing the child to blow through an obstructed straw.

Each of these maneuvers should be performed for 10 to 20 seconds.

Another vagal maneuver is the ice bag method. Because the ice bag method does not require patient cooperation, it's good for patients of any age. But it is commonly used for young children and infants. To be effective, apply the ice bag over the bridge of the nose, the eyes, and the forehead for 10 to 20 seconds. This elicits the dive reflex, which can result in bradycardia. Be careful not to cover the child's nose or mouth with the ice bag.

Electrical Therapies

Commonly used electrical therapies include synchronized cardioversion and manual defibrillation.

Synchronized Cardioversion

Synchronized cardioversion is used when arrhythmias with preserved QRS complexes on ECG, such as SVT, atrial fibrillation, and atrial flutter, become unstable or fail to respond to pharmacologic therapy. Synchronized cardioversion is also used for ventricular tachycardia (VT) when a pulse is present. It is used as a first-line therapy in patients with VT who are unstable but have a pulse and after unsuccessful pharmacologic therapy in patients with stable VT.

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.

A cardiac monitor/defibrillator is used for synchronized cardioversion, and 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,

Figure 3-15 | Vagal maneuvers are an initial treatment option in children with hemodynamically stable supraventricular tachycardia (SVT).

including soft tissue and bone, between the defibrillation pads and the heart) and minimizes the risk for burns.

Pediatric pads are used for children and infants weighing less than 10 kilograms and adult pads are used for children weighing 10 kilograms or more. Always use the largest size pad that will fit on the patient's chest without touching or overlapping.

The pads may be placed using anterolateral placement or anterior-posterior placement. Use an anterior-posterior position if the pads risk touching or overlapping on the patient's chest or if the patient has a pacemaker.

- **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.

The cardiac monitor/defibrillator must be set to synchronous mode (indicated by the appearance of sync markers at the 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. The energy dose depends on the child's or infant's weight and whether it is a first shock or subsequent shocks (Table 3-3). 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.

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 *Electrical Therapy* Skill Sheet provides step-by-step guidance for synchronized cardioversion.

Manual Defibrillation

Manual defibrillation is indicated for shockable cardiac arrest rhythms (i.e., ventricular fibrillation and pulseless ventricular tachycardia).

Defibrillation involves the administration of a high initial dose 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 node to resume its normal pacemaker function, terminating the arrythmia.

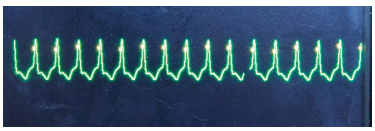
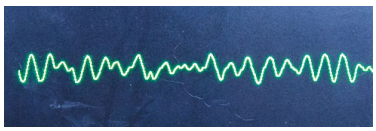
As in synchronized cardioversion, a cardiac monitor/defibrillator is used for manual defibrillation, and shocks are delivered through adhesive pads placed on the patient's chest. The energy dose depends on the child's or infant's weight and whether it is a first shock or subsequent shocks (see Table 3-3).

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.

The *Electrical Therapy* Skill Sheet provides step-by-step guidance for manual defibrillation.

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

Feature	Synchronized Cardioversion	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 or medication-refractory supraventricular tachycardia (SVT) ■ Atrial flutter ■ Atrial fibrillation ■ Monomorphic ventricular tachycardia with a pulse	■ Ventricular fibrillation (VF) ■ Pulseless ventricular tachycardia (pVT)
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
Energy dosage, J/kg		
Initial dose	■ 0.5 to 1	■ 2
Subsequent doses	■ 2 (If starting with 0.5, may increase dose more gradually to 1)	■ 4 ■ ≥4 to maximum 10, or the adult dose

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.

Vascular Access

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

Nonvascular routes of access are also an option during pediatric emergencies. See *Learn More: Nonvascular Routes of Access*.

LEARN MORE

Nonvascular Routes of Access

Nonvascular routes of access are also an option during pediatric emergencies. Options include the intramuscular, subcutaneous, sublingual, intranasal, and rectal routes, though most are appropriate only for certain medications and all have limitations. In the child requiring concomitant intubation, an endotracheal tube (ETT) can be a route for administering specific drugs, namely, lidocaine, epinephrine, atropine, or naloxone (i.e., the LEAN drugs). However, the IV and IO routes are preferred to the ETT route because of erratic drug absorption, higher risk for adverse effects, and in the case of naloxone, limited evidence for efficacy with ETT administration.

Intravenous Access

A common method for establishing vascular access in emergencies is peripheral venous access. This form of access can be challenging to establish in children and infants because of their small size. 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 antecubital vein of the arm, the saphenous vein of the leg, dorsal veins of the hands and feet, and the superficial veins of the scalp (Figure 3-16). The external jugular vein in the neck is also a possibility if no risk of cervical spine injury exists.

Factors to consider when selecting the type and site of peripheral IV access include the duration of, and indication for, treatment, the potential types of solutions to be infused, and vein availability. When administering IV therapies in an emergency, keep the following points in mind:

- Use the largest-diameter catheter possible (e.g., as a guide, use a 24-gauge catheter in an infant and a 22-gauge catheter in an older child).
- Take special care when administering certain agents through a peripheral IV catheter, including vasopressors, hyperosmolar solutions, and calcium chloride, which may cause damage if they infiltrate into local tissues.
- When providing care for a patient in cardiac arrest, follow each peripherally administered drug dose with a 5- to 10-mL normal saline flush to ensure that the medication reaches the central circulation.
- Monitor the patient for local and systemic complications of IV therapy.

Central Venous Access

Vascular access may also be obtained using larger central veins, such as the femoral, subclavian, and internal jugular veins (see Figure 3-16). Although central venous catheters offer certain advantages, such as longer-term access and the capacity for administering medications that are potentially harmful to peripheral tissues, their placement requires specialized skill. Central venous catheter placement is also associated with multiple potential risks, including bleeding due to unintended arterial puncture or vein perforation, pneumothorax when using the internal jugular or subclavian sites, and infection. Because of the relative ease of establishing intraosseous access, the role of central venous catheters as a form of access in pediatric emergencies has been deemphasized.

Intraosseous Access

Intraosseous (IO) access is often used as an alternative to IV access in pediatric emergency situations. IO uses the bone marrow as the vascular space and typically involves

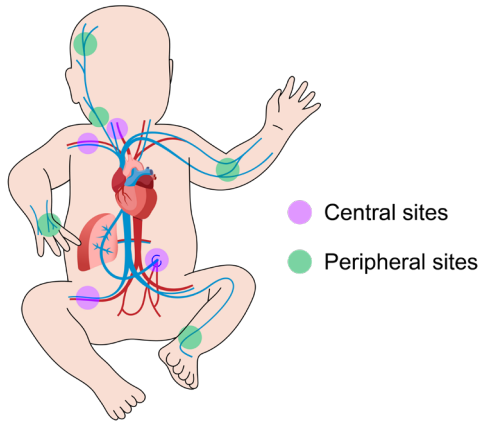


Figure 3-16 | Common sites for peripheral and central venous access in children and infants

a needle rather than a catheter. This procedure can be safely and efficiently performed in children and infants of all ages, including in newborns as a faster alternative to umbilical catheter placement. In pediatric emergencies, IO access is preferred over central venous access when attempts at peripheral IV access have failed.

As with IV access, IO access can be used for administering medications, fluids, and blood products and to collect blood for laboratory analyses. 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 5- to 10-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.

Contraindications to IO needle placement include bone fractures or disorders that predispose to fracture (e.g., osteogenesis imperfecta), infection or burns of the overlying skin, and previous IO site in the target bone. However, IO placement may still be considered in these instances when there is no other vascular access during an emergency. In preterm newborns weighing less than 1000 grams, use IO access with caution and only in true emergencies.

Depending on the type, designated IO needles can be inserted manually or with the use of an semiautomatic device such as a “drill” (Figure 3-17, A, B, C).

The preferred site for IO needle insertion is the anterior proximal tibia, 1 to 2 cm below the tibial tuberosity to avoid the growth plate found at the end of long bones. This site is preferred because it provides a flat surface with a relatively thin outer layer of bone, a large marrow cavity, and easily identifiable landmarks to facilitate placement. Other potential sites for IO placement

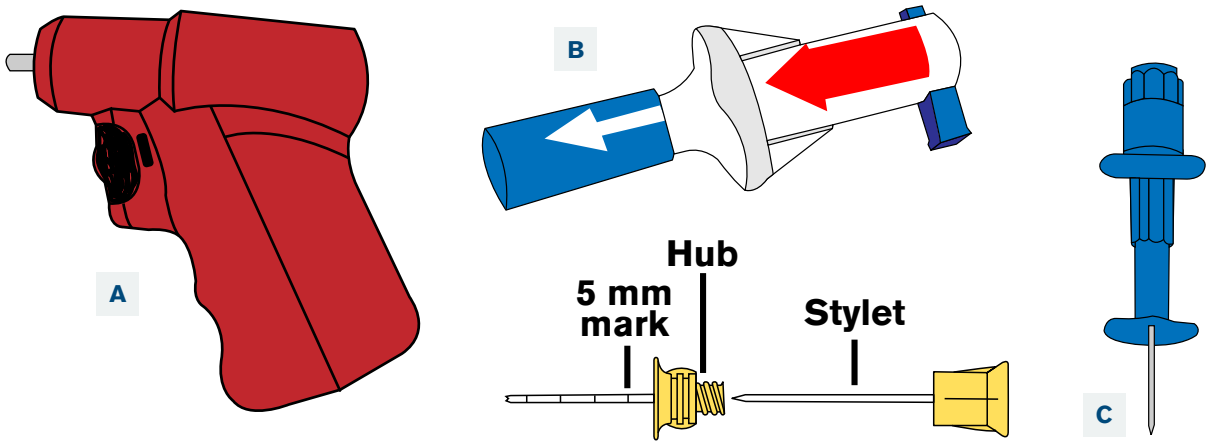


Figure 3-17 | Equipment for intraosseous (IO) access. (A) IO drill and needle. (B) Bone injection device. (C) Jamshidi needle for manual insertion.

include the distal tibia, the distal femur of the upper leg, the distal radius of the forearm, and the proximal humerus of the upper arm. Note that the distal radius of the forearm is not a recommended site of administration when using a semi-automatic device (e.g., drill).

There are different needle sizes available for different sized pediatric patients and they are often color coded for easy identification. It is important to decide the best needle size to use for your patient based on tissue depth at the insertion site. In order to determine the best needle size to use at the chosen site, assess the tissue depth at the insertion site. To do this, palpate the insertion site with your finger to see how much tissue there is before the bone. Choosing the appropriately sized needle is important. If the needle is too short, the needle may become displaced, leading to tissue infiltration or extravasation. If the needle is too long, it is possible to pass the needle through the bone to the other side of the extremity (especially in infants and smaller children). Use a needle with a preset depth gauge, when available.

Practice Note

After insertion, using an IV pump or pressure bag to administer fluids through an IO site may be needed if free flow doesn't seem to be infusing well.

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 *Achieving Intraosseous Access (Drill)* Skill Sheet provides step-by-step guidance for achieving IO access using an IO drill. Always follow the manufacturer's instructions when using a device to insert an IO needle.

To achieve IO access using manual insertion, follow these steps:

1. Stabilize the target site on a firm surface.
2. Palpate the site to determine tissue depth.
3. Disinfect the skin overlying the insertion site.
4. If the patient is awake, consider infiltration of the skin and periosteum with 1% lidocaine.
5. 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.
6. 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.
7. Remove the stylet by twisting it counterclockwise, and dispose of it properly.
8. Hold the needle securely and attach the primed tubing to the needle.

9. 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.
10. Tape the flange to the skin and add a gauze dressing for support.
11. Monitor the insertion site and the extremity for swelling.

Fluid and Blood Therapy

Fluid resuscitation is often indicated for patients experiencing shock and as part of post–cardiac arrest care (in the case of myocardial dysfunction and/or an ischemia/reperfusion response).

In addition, packed red blood cells (PRBCs) or whole blood may be indicated in combination with isotonic crystalloids for volume resuscitation in hemorrhagic shock. In addition to being used to immediately expand volume in patients with hemorrhagic shock, administration of PRBCs may be considered, as clinically indicated, to optimize hemoglobin concentration and hence oxygen delivery in shock.

The most common indication for fluid resuscitation in children is hypovolemic shock resulting from actual fluid or blood losses. However, most patients in shock have some degree of diminished intravascular volume, whether actual or relative (e.g., due to capillary leak). The main goal of fluid resuscitation is to restore intravascular volume to reverse cellular hypoxia and ischemia before irreversible end-organ damage occurs.

Isotonic crystalloid solutions, such as 0.9% normal saline (NS) or lactated Ringer's solution (LR), are the primary solutions used for fluid resuscitation. Isotonic fluids are used instead of hypotonic fluids (e.g., 0.45% NS) because isotonic solutions allow a greater proportion of the administered volume to remain in the intravascular space. In most situations, NS and LR are equally effective options for fluid resuscitation. Because of the association between repeated NS 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 LR may be preferred in some settings.

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 L of crystalloid solution in approximately 10 to 15 minutes.

Optimizing tissue perfusion is the ultimate goal of fluid therapy. Optimal perfusion is best indicated by normal urine output (infants and young children: 1.5 to 2 mL/kg/hr; adolescents: 1 mL/kg/hr). Heart rate, mental status and capillary refill are not always reliable measures of optimal perfusion because they may be altered by other underlying diseases. Blood pressure should be interpreted with caution as it may be normal in the setting of shock. Central venous pressure (CVP) provides a continuous, real-time assessment of preload, but measurement of CVP requires placement of a central venous catheter.

Monitoring of patient response to fluid resuscitation should be ongoing. Always assess the child or infant after each fluid bolus and discontinue fluid administration if clinical signs or symptoms of hypervolemia develop (e.g., hepatomegaly, crackles on chest auscultation).

Once sufficient volume has been restored or euvolemia has been achieved, stop resuscitative fluid administration. Patients who remain in shock despite adequate fluid resuscitation are candidates for other supportive, namely vasoactive, therapies.

Maintenance fluids are administered in the appropriate calculated amount once the patient is stabilized.

Table 3-4 summarizes fluid and blood therapies commonly used in pediatric emergencies.

Medication Therapy

A good working knowledge of the medications most commonly used in the management of respiratory emergencies, shock and cardiac emergencies is essential. Table 3-5 summarizes the action, indications, administration and precautions for medications that are commonly used in pediatric emergencies.

Table 3-4 | Fluid and Blood Therapies Commonly Used in Pediatric Emergencies

Fluid Resuscitation

Fluid Type	Subtypes	Indication(s)	Dosing and Administration	Special Considerations
Crystalloid solutions Isotonic, electrolyte-containing	■ 0.9% normal saline (NS) ■ Lactated Ringer's (LR)	Primary solutions used for fluid resuscitation (e.g.,in shock)	■ Initial bolus: 20 mL/kg IV/IO rapidly; give smaller volume (5–10 mL/kg) more slowly (over 10–20 min) in patients with cardiogenic shock and in neonates ■ Repeat as needed until perfusion improves (e.g., reversal of hypotension, normal urine output for age, normalized capillary refill and pulses) ■ Children in shock may require up to 60 mL/kg of fluid, as needed in the first 30 to 60 minutes of resuscitation See Chapter 7 and Chapter 9 for specifics on fluid resuscitation in various types of shock and in post-cardiac arrest myocardial dysfunction	■ In most situations, NS and LR are equally effective options for fluid resuscitation ■ Decrease fluid bolus volume or discontinue fluid bolus administration if signs of hypervolemia (e.g., palpable liver edge, crackles in lungs) develop
Colloid solutions Contain natural or synthetic high-molecular-weight substances (e.g., albumin, starches)	■ Albumin ■ Dextran ■ Hydroxyethyl starch	Fluid resuscitation (e.g., in shock)	■ As for crystalloid solutions	■ Secondary option for fluid resuscitation; offer no clinical benefit over crystalloid solutions in critically ill children and are more costly

Maintenance Fluid Therapy

Fluid Type	Subtypes	Indication(s)	Dosing and Administration	Special Considerations
Isotonic maintenance fluid	■ NS or LR ■ With or without glucose ■ With or without potassium	Maintenance of hydration after stabilization (including fluid resuscitation) following a pediatric emergency or after ROSC following cardiac arrest	■ Calculate maintenance rate based on patient weight as follows (4-2-1 method): ○ <10 kg: 4 mL/kg/hr ○ 10 to 20 kg: 40 mL/hr + 2 mL/kg/hr for every kilogram above 10 kg ○ ≥20 kg: 60 mL/hr + 1 mL/kg for every kilogram above 20 kg ■ Add dextrose for infants and for children who are hypoglycemic or at risk for becoming hypoglycemic ■ Add 10 to 20 mEq/L potassium if renal function and urine output are adequate, able to monitor serum potassium levels and no contraindications (e.g., hyperkalemia, muscle injury)	■ Maintenance fluid rates do not account for ongoing fluid losses (e.g., due to GI losses, third-spacing, fever); be sure to replace any such losses ■ Fluid balance, patient weight, electrolytes and glucose should be monitored closely during maintenance fluid therapy, adjusting fluid composition and rate as indicated

Hyperosmolar Fluid				
Fluid Type	**Subtypes**	**Indication(s)**	**Dosing and Administration**	**Special Considerations**
Hypertonic saline	N/A	Treatment of increased ICP	■ Bolus dosing for acute use: 6.5 to 10 mL/kg IV/IO ■ Continuous infusion for ongoing control of ICP: 0.1 to 1 mL/kg/hour IV/IO titrated to minimal rate required to maintain ICP <20 mm Hg	■ Maintain serum osmolality <360 mOsm/L and monitor sodium levels ■ Monitor for signs of osmotic demyelination syndrome (demyelinating condition of the brain caused by rapid increase in serum sodium concentration)

Blood Products				
Type	**Subtypes**	**Indication(s)**	**Dosing and Administration**	**Special Considerations**
Whole blood Plasma, red blood cells and platelets	N/A	Volume resuscitation in hemorrhagic shock	10 to 20 mL/kg IV/IO if shock persists after initial fluid resuscitation	■ Not available in all centers (requires specific handling and storage) ■ Risk for hemolytic transfusion reaction when uncrossmatched (i.e., type O whole blood is used in non-group O recipients (unless donor has low anti-A and -B antibody titers)

Blood Products				
Type	**Subtypes**	**Indication(s)**	**Dosing and Administration**	**Special Considerations**
PRBCs Derived from whole blood after centrifugation and removal of most of plasma component	N/A	■ Volume resuscitation in hemorrhagic shock ■ As clinically indicated (to optimize hemoglobin concentration and, hence, oxygen delivery)	For hemorrhagic shock: ■ 10 mL/kg if shock persists after two or three initial crystalloid boluses. ■ Use type O-negative PRBCs if immediate transfusion is warranted (e.g., for hypotension) and there is insufficient time for type- and cross-matching For increasing hemoglobin concentration in shock: ■ 5 mL/kg of PRBCs increases hemoglobin concentration by ~1 g/dL	■ Infusion of large volumes may induce hypothermia, which may cause arrhythmias or even cardiac arrest. To prevent this complication, blood may be warmed as it is infused using an IV set with a heat-exchange device ■ Because the anticoagulant in PRBCs (citrate) can bind calcium, hypocalcemia may occur with massive PRBC transfusion ■ Hyperkalemia may also occur with transfusion of PRBCs stored for >1 week or if mechanical hemolysis occurs during transfusion ■ The presence of hypothermia may amplify the risk for hypocalcemia and hyperkalemia ■ Replacement of clotting factors and platelets should be considered with massive (75–80 mL/kg) PRBC transfusion

Blood Products				
Type	**Subtypes**	**Indication(s)**	**Dosing and Administration**	**Special Considerations**
Plasma therapies Plasma components of blood containing coagulation factors	■ FFP ■ Cryoprecipitate	■ Correction of coagulopathy in thrombotic purpura disorders (e.g., DIC) ■ Replacement of clotting factors following massive (75–80 mL/kg) PRBC transfusion	■ Use FFP in patients with multiple coagulation factor deficiencies (e.g., due to DIC) who are bleeding ■ Typical FFP dose is 10 to 15 mL/kg, though higher doses may be required based on clinical situation and laboratory parameters ■ For replacement of clotting factors during massive PRBC transfusion: 1 U FFP for every 1 to 2 U PRBCs	■ Plasma therapies should not be used as volume expanders for fluid resuscitation ■ Monitor for fluid overload, particularly in patients with cardiovascular or renal compromise
Platelets Platelet concentrate obtained by centrifugation or separation from (apheresis) whole blood	■ Pooled platelets (multiple donors) ■ Single-donor platelets from aphersis	■ Thrombocytopenia in the setting of severe shock, defined as follows: ○ Platelet count ≤10,000/mm^3 if no apparent bleeding ○ Platelet count ≤20,000/mm^3 if significant risk for bleeding ○ Platelet count <50,000/mm^3 if actively bleeding or before surgery or other invasive procedure ■ Replacement of platelets following massive PRBC transfusion	■ 0.1 to 0.2 U/kg of platelets will increase platelet count by ~30,000 to 50,000/mm^3 ■ Increase in platelet count may be less than expected in patients with sepsis ■ For replacement of platelets following massive PRBC transfusion: 1 U platelets for every 1 to 2 U PRBCs	■ Platelet transfusion may also be required despite the presence of a normal platelet count when known platelet dysfunction or microvascular bleeding is present

Practice Note

Time is of the essence in a pediatric emergency. A patient's weight is critical information to have, especially for drug dosing or energy dosing for synchronized cardioversion or manual defibrillation. However, an exact weight may not be known or easily obtainable. A length-based resuscitation tool (e.g., Broselow tape) can be used to provide a quick estimate of a child's weight based on his or her length. Many of these tools are color-coded to provide a quick means of determining appropriate medication and energy doses and resuscitation equipment sizes for any given pediatric patient in an emergency setting.

ECC Updates in Pharmacology

Antiarrhythmic Medications for Shock-Refractory VF or Pulseless VT

In the past, amiodarone was recommended as the first-line antiarrhythmic agent over lidocaine for shock- and vasopressor-refractory VF and pulseless VT (pVT). However, recent observational data has shown improved rates of return of spontaneous circulation and 24-hour survival among children who received lidocaine versus amiodarone for in-hospital arrest due to VF or pVT. Accordingly, it is now recommended that either amiodarone or lidocaine may be used first line for shock- and vasopressor-refractory VF and pVT.

Vasopressors for Resuscitation

Data regarding the effectiveness of vasopressors, including epinephrine, in pediatric cardiac arrest

are inconclusive. A randomized, prospective study of out-of-hospital cardiac arrest in adults found that epinephrine was associated with increased rates of return of spontaneous circulation compared with placebo. Epinephrine continues to be recommended as a reasonable treatment for pediatric cardiac arrest.

Table 3-5 | Commonly Used Medications in Pediatric Emergencies

Drug	Action	Indications	Administration	Precautions
Adenosine	Slows conduction of impulses through the AV node	■ Stable SVT ■ Unstable SVT if cardioversion is being prepared or delayed ■ Stable regular monomorphic wide-complex tachycardias	■ 0.1 mg/kg (6-mg maximum dose) by rapid IV/IO push followed by 5- to 10-mL NS flush ■ If not effective, 0.2 mg/kg (12-mg maximum dose) by rapid IV/IO push followed by 5- to 10-mL NS flush ■ Has an extremely short half-life; administer over 1–3 s, at a site as close to the heart as possible using rapid bolus technique ■ Use rapid bolus technique ■ Patient should be on a monitor and have a rhythm strip run during adenosine administration	■ 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
Albuterol	Selective, short-acting, inhaled β_2-adrenergic agonist used primarily as a bronchodilator	■ Asthma exacerbations ■ Bronchospasm due to anaphylaxis or viral respiratory tract infections ■ Hyperkalemia	By metered-dose inhaler (MDI; 90 mcg/actuation): ■ 4 to 8 inhalations every 20 minutes as needed for 3 doses, then every 1 to 4 hours or as clinically indicated By nebulizer (intermittent): ■ Children <12 years: ○ Initial dosing: 0.15 mg/kg (minimum, 2.5 mg) every 20 minutes as needed for 3 doses, then 0.15 to 0.3 mg/kg (maximum, 10 mg) every 1 to 4 hours or as clinically indicated ■ Children ≥12 years: ○ Initial dosing: 2.5 to 5 mg every 20 minutes as needed for 3 doses, then 2.5 to 10 mg every 1 to 4 hours or as clinically indicated By nebulizer (continuous): ■ Children <20 kg: 7.5 mg/hour ■ Children ≥20 kg: 10 mg/hour ■ Children ≥12 years: 10 to 15 mg/hour	■ May cause cardiac arrhythmias, increase or decrease in blood pressure; may also cause transient hypokalemia, which may exacerbate the occurrence of arrhythmias ■ Use with caution in patients with cardiac arrhythmias or hypertension
Amiodarone	Class III antiarrhythmic; delays repolarization and prolongs the QT interval	■ Shock-refractory VF/pVT ■ Supraventricular tachycardia (SVT) ■ Stable VT with pulse ■ Unstable cardioversion-refractory VT with a pulse	Shock-refractory VF/pVT: ■ 5 mg/kg IV/IO bolus (maximum single dose, 300 mg) ■ If not effective, 2 additional repeat doses may be given, for a maximum cumulative dose of 15 mg/kg (or 2.2 g in adolescents) over a 24-hour period All other indications: ■ 5 mg/kg (maximum, 300 mg) IV/IO over 20 to 60 minutes ■ If not effective, 2 additional repeat doses may be given, for a maximum cumulative dose of 15 mg/kg (or 2.2 g in adolescents) over a 24-hour period	■ Do not use with other drugs that prolong QT interval (e.g., procainamide) ■ May cause hypotension; infuse slowly and monitor for hypotension during infusion ■ May cause bradycardia or AV block; slow or discontinue infusion if bradycardia occurs; ensure availability of temporary pacing if using in children who are predisposed to bradycardia or AV block ■ Consultation with cardiologist strongly recommended when using this agent

Drug	Action	Indications	Administration	Precautions
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	■ Unstable sinus bradycardia (first-line if due to increased vagal tone or cholinergic drug [e.g., organophosphate] poisoning) ■ Symptomatic, primary second- or third-degree AV block ■ Known or suspected organophosphate poisoning	For unstable sinus bradycardia or symptomatic AV block: ■ 0.02 mg/kg (minimum, 0.1 mg; maximum, 0.5 mg) via IV/IO ○ May repeat dose once after 3 to 5 minutes ○ Maximum total dose is 1 mg in children and 3 mg in adolescents ■ 0.04 to 0.06 mg/kg via ETT For known or suspected organophosphate poisoning: ■ Children <12 years: ○ Initial dose: 0.05 mg/kg IV/IO ○ Repeat every 5 minutes as needed, doubling the previous dose, until symptoms resolve ■ Children ≥12 years: ○ Initial dose: 1 mg/kg IV/IO ○ Repeat every 5 minutes as needed, doubling the previous dose, until symptoms resolve	Higher doses may be required for organophosphate poisoning
Calcium chloride	Parenteral calcium solution (**Note:** Preferred calcium formulation in critically ill children because it raises ionized calcium levels more quickly than calcium gluconate)	■ Hypocalcemia ■ Hyperkalemia with widened QRS interval on ECG to prevent onset of VF ■ Calcium channel blocker toxicity	■ Initial dose: 20 mg/kg (0.2 mL/kg of the 10% solution) IV/IO slow push ■ Repeat dose as needed to achieve desired clinical effect	■ Should only be used for cardiac resuscitation in the presence of documented hypocalcemia, hyperkalemia, hypermagnesemia or calcium channel blocker toxicity ■ Monitor for symptomatic bradycardia; discontinue infusion if occurs ■ Administration through a central venous catheter preferred, as extravasation through a peripheral IV catheter may lead to severe skin or soft tissue injury ■ Administer with caution in patients receiving digitalis
Calcium gluconate	Parenteral calcium solution; alternative to calcium chloride	As above	■ 60 mg/kg (0.6 mL/kg of the 10% solution) IV/IO slow push ■ Repeat dose as needed to achieve desired clinical effect	■ Use only if calcium chloride unavailable ■ **CONTRAINDICATED** in neonates receiving ceftriaxone; may cause intravascular ceftriaxone-calcium precipitates with resulting end-organ damage. If using in children >28 days who are receiving concomitant ceftriaxone, do not administer simultaneously through the same IV line or connector; may administer sequentially through the same line with thorough flushing between medications ■ Rapid administration may lead to hypotension, bradycardia, arrhythmias, syncope or cardiac arrest ■ Administration through a central venous catheter preferred, as extravasation through a peripheral IV catheter may lead to severe skin or soft tissue injury ■ Avoid in patients receiving cardiac glycosides; may cause cardiac arrhythmias ■ Extravasation may lead to local skin and soft tissue injury

Drug	Action	Indications	Administration	Precautions
Dexamethasone	Synthetic glucocorticoid (steroid) with anti-inflammatory activity	■ Upper airway edema in croup ■ Acute asthma exacerbation	0.6 mg/kg (maximum, 16 mg) PO/IV/IM, (as one dose for croup and every 24 hours for asthma exacerbation)	May cause hyperglycemia/reduced glucose tolerance, especially in patients predisposed to diabetes mellitus
Diphenhydramine	First-generation (sedating) antihistamine; competes with binding of histamine to specific histamine (H1) receptors	Anaphylaxis	1 to 2 mg/kg (maximum, 50 mg) IM/IV/PO	■ May cause sedation and respiratory depression, especially if administered with other sedating medications ■ May cause hypotension ■ Rapid IV infusion may precipitate seizures ■ May cause paradoxical agitation
Dobutamine	Synthetic adrenergic agent with activity at $ß_1$- and $ß_2$-adrenergic receptors; used primarily as an inotropic agent in conditions in which cardiac function is impaired	■ Certain forms of shock requiring inotropic support ■ Conditions affecting the heart muscle (i.e., myocarditis or cardiomyopathy)	2 to 20 mcg/kg/min IV/IO infusion, titrated to effect	■ May decrease blood pressure via interaction with $ß_2$-receptors; dopamine or other agents with α-adrenergic activity may be concurrently administered when hypotension occurs ■ May cause tachyarrhythmias
Dopamine	Natural precursor of norepinephrine and epinephrine with chronotropic, inotropic or vasopressor effects, depending on dose	Fluid-refractory shock	2 to 20 mcg/kg/min IV/IO infusion titrated to clinical effect	■ Doses greater than 20 mcg/kg/min may cause tachyarrhythmias; consider other agent when doses above this range are required ■ Can cause end-organ infarction in hypovolemic states; correct hypovolemia before initiating therapy ■ Do not use with sodium bicarbonate ■ Effects of dopamine may be reduced in infants and patients with chronic congestive heart failure

Drug	Action	Indications	Administration	Precautions
Epinephrine	Acts on both α- and ß-adrenergic receptors; increases SVR, contractility and heart rate	■ Cardiac arrest (VF, pVT, PEA, asystole) ■ Unstable bradycardia ■ Certain forms of fluid-refractory shock requiring vasopressor and/or inotropic support ■ Anaphylaxis-related airway edema and angioedema. ■ Acute severe asthma exacerbation ■ As an alternative to racemic epinephrine ○ Upper airway edema in croup ○ Bronchospasm due to bronchiolitis	For VF/pVT/PEA/asystole and unstable bradycardia: ■ IV/IO: 0.01 mg/kg (ie, 0.1 mL/kg of the 0.1-mg/mL concentration), repeated every 3 to 5 minutes ■ ETT: 0.1 mg/kg (or 0.1 mL/kg of the 1-mg/mL concentration), diluted in 5 to 10 mL of sterile water or NS directly into the ETT at the same times or sequence position as indicated for the IV/IO route For shock: ■ 0.1 to 1 mcg/kg/min IV/IO infusion, titrating to effect For anaphylaxis-related airway edema/angioedema: ■ 0.01 mg/kg (maximum, 0.3 mg) (0.01 mL/kg of the 1-mg/mL concentration) IM every 10 to 15 minutes as needed to control symptoms. For acute severe asthma exacerbation: ■ Epinephrine 0.01 mg/kg [diluted 1-mg/mL concentration; maximum dose, 0.3–0.5 mg] subcutaneously every 20 minutes for three doses, then as clinically indicated For croup or bronchiolitis: ■ 3 mg (3 mL of the 1-mg/mL concentration) mixed with 3 mL NS via nebulizer	■ May increase blood pressure, heart rate and myocardial oxygen demand ■ No high-quality pediatric studies have demonstrated the effectiveness of epinephrine in cardiac arrest. ■ IV infiltration may cause severe localized skin injury; phentolamine may be injected intradermally to counteract this effect
Epinephrine, racemic	As above	■ Upper airway edema in croup ■ Bronchospasm due to bronchiolitis	■ 0.25 to 0.5 mL of the 2.25% solution diluted in 3 mL of NS via nebulizer ■ Note: When racemic epinephrine is not available, may administer 3 to 5 mg of standard epinephrine (1-mg/mL concentration) mixed with 3 mL NS via nebulizer	May increase blood pressure, heart rate and myocardial oxygen demand
Flumazenil	Benzodiazepine antagonist/reversal agent	Benzodiazepine reversal	■ Initial dose: 0.01 mg/kg (maximum, 0.2 mg) IV given over 15 seconds ■ Repeat doses: 0.01 mg/kg (maximum, 0.2 mg) IV given at 1-minute intervals for a maximum of 4 doses; limit total cumulative dose to 0.05 mg/kg or 1 mg, whichever is lower	■ Because its duration of action is shorter than for most benzodiazopenes, repeat dosing of flumazenil may be required ■ It is very important to provide supportive care, including management and preservation of the child's airway throughout therapy with this drug ■ May precipitate seizures in children being treated with benzodiazepines for a seizure disorder. May provoke acute withdrawal in benzodiazepine-dependent patients

Drug	Action	Indications	Administration	Precautions
Furosemide	Loop diuretic	■ Fluid overload ■ Congestive heart failure/pulmonary edema	■ 1 mg/kg initial IV/IM dose ■ If necessary, can increase dose by up to 1 mg/kg and give ≥2 hours after the previous dose, repeating until desired response is achieved, to a maximum dose of 6 mg/kg	■ May cause severe hypokalemia ■ Excessive diuresis can cause intravascular volume depletion and circulatory collapse
Glucose	Glucose replacement solution	Low blood sugar level (ie, hypoglycemia)	Glucose is administered as dextrose and dosed at approximately 0.5 to 1 g/kg IV/IO in pediatric patients This translates to: ■ Newborns, infants or children: 5 to 10 mL/kg 10% dextrose in water ■ Infants and children: 2 to 4 mL/kg 25% dextrose in water	■ Newborns generally have higher metabolic energy requirements than humans at other stages of development. Newborns therefore may develop hypoglycemia in situations that place them at higher levels of energy expenditure. ■ Hyperglycemia is potentially detrimental in critical illness and should be avoided when treating hypoglycemia, when possible.
Heparin (unfractionated; low–molecular weight)	Inhibits reactions involved in the clotting of blood	Pulmonary embolism	■ Unfractionated heparin: IV loading dose of 75 U/kg over 10 minutes followed by 20 U/kg/hr for children and adolescents or 28 U/kg/hr for infants ■ Low–molecular weight heparin: 1 mg/kg subcutaneously every 12 hours in children >2 months old to 18 years and 1.5 mg/kg every 12 hours in infants <2 months old	■ Use unfractionated heparin in patients who are not clinically stable or who are at high risk for bleeding (more easily reversed and has a shorter half-life than low–molecular weight heparin) ■ Coagulation laboratory values should be monitored frequently during unfractionated heparin administration ■ Both unfractionated and low–molecular weight heparin may cause bleeding or thrombocytopenia ■ Difficult to achieve therapeutic levels with use of low–molecular weight heparin in infants
Hydrocortisone	Corticosteroid used to replace steroids that are normally produced by the body	Fluid-refractory, catecholamine-resistant shock with confirmed or at risk for adrenal insufficiency (e.g., purpura fulminans, prior steroid therapy, adrenal or pituitary abnormality)	50 mg/m^2/24 h IV infusion	■ Obtain pretreatment blood sample for determination of baseline cortisol level, if possible ■ May cause fluid retention or hyperglycemia/glucose intolerance
Insulin, regular	Short-acting recombinant version of insulin, a naturally occurring hormone that regulates transfer of glucose into cells and glucose levels in the blood; also promotes movement of potassium into cells	■ Diabetic ketoacidosis (DKA) ■ Persistent post–cardiac arrest hyperglycemia (glucose level >144–180 mg/dL) ■ Hyperkalemia (administered in combination with glucose)	DKA: ■ 0.05 to 0.10 U/kg/hr IV infusion; continue at this rate, as tolerated, until ketoacidosis resolves Post–cardiac arrest hyperglycemia: ■ 0.025 to 0.05 U/kg/hr IV infusion to start; titrate to maintain glucose level ≤144 to 180 mg/dL Hyperkalemia: ■ 0.1 U/kg IV with 400 mg/kg glucose IV (1 U insulin for every 4 g of glucose)	■ Monitor glucose and potassium levels every hour or more frequently as needed to avoid hypoglycemia and hypokalemia ■ Gradually reduce glucose levels (by 50 to 100 mg/dL per hour) when treating DKA or hyperglycemia ■ Ensure appropriate concomitant fluid and electrolyte replacement when treating DKA

Drug	Action	Indications	Administration	Precautions
Ipratropium Bromide	Quaternary ammonium salt that is a derivative of atropine	Often used in concert with albuterol for treatment of acute bronchospasm due to asthma and other conditions	■ 0.5 mg via nebulizer every 20 minutes for up to 3 doses ■ Can alternatively be administered as a combined ipratropium bromide/albuterol formulation (3mL=0.5 mg ipratropium bromide/2.5 mg albuterol [base]) as follows: ○ 3 mL via nebulizer every 20 minutes for up to three doses	■ Should not be used as first-line therapy ■ Not significantly absorbed systemically ■ May cause dry mouth and gastrointestinal upset ■ Accidental eye exposure may cause pupillary dilation and an increase in ocular pressure
Ketamine	Dissociative anesthetic agent with bronchodilator activity	■ Sedation for endotracheal intubation (preferred agent in status asthmaticus) ■ Refractory bronchospasm (status asthmaticus)	■ Preintubation: 1 to 2 mg/kg IV ■ Continuous infusion (infants ≥5 months, children, and adolescents): 0.5 to 2 mg/kg IV, then 5 to 20 mcg/kg/min IV infusion, starting at lowest dosage and titrating upward, as needed, to effect	■ Optimal dose for treatment of refractory bronchospasm has not been established ■ May cause hypersalivation; drying agents (e.g., atropine, scopolamine) may be co-administered to prevent or reduce this effect ■ May cause laryngospasm; therefore, prior airway instability and tracheal surgery or stenosis are relative contraindications for ketamine use ■ Increases intracranial and intraocular pressure
Levalbuterol	Selective, short-acting, inhaled ß2-adrenergic agonist; one of (and more pharmacologically active of) two "mirror-image" molecules making up albuterol	■ Asthma exacerbation ■ Bronchospasm from other causes	By MDI: ■ 4 to 8 inhalations every 20 minutes as needed for 3 doses, then every 1 to 4 hours or as clinically indicated By nebulizer (intermittent): ■ Children <12 years: ○ Initial dosing: 0.075 mg/kg (minimum, 1.25 mg) every 20 minutes as needed for 3 doses, then 0.075 to 0.15 mg/kg (maximum, 5 mg) every 1 to 4 hours or as clinically indicated ■ Children ≥12 years: ○ Initial dosing: 1.25 to 2.5 mg every 20 minutes as needed for 3 doses, then 1.25 to 5 mg every 1 to 4 hours or as clinically indicated	■ May cause cardiac arrhythmias, increase or decrease in blood pressure; may also cause transient hypokalemia, which may exacerbate the occurrence of arrhythmias ■ Use with caution in patients with cardiac arrhythmias or hypertension
Lidocaine	Class Ib antiarrhythmic (sodium channel blocker); delays repolarization and slightly increases the QT interval	Shock-refractory VF/pVT	■ Initial dose: ○ IV/IO: 1 mg/kg ○ ETT: 2 to 3 mg/kg ■ Maintenance infusion: 20 to 50 mcg/kg/min; repeat initial dose if infusion is initiated more than 15 minutes after initial dose	■ Amiodarone or lidocaine is equally acceptable for the treatment of shock-refractory VF/pVT in pediatric patients ■ Excess lidocaine levels may cause circulatory depression, hypotension and seizures ■ Contraindicated in patients with complete heart block and wide-complex tachycardia attributed to accessory conduction pathways ■ Do not alternate between amiodarone and lidocaine

Drug	Action	Indications	Administration	Precautions
Magnesium sulfate	Cofactor in the transport of sodium, calcium and potassium across the cell membrane	■ Torsades de pointes ■ Severe asthma attacks that do not respond to first-line measures (i.e., status asthmaticus) ■ Hypomagnesemia	Pulseless torsades de pointes: ■ 25 to 50 mg/kg, IV/IO bolus (maximum dose, 2 g) Status asthmaticus: ■ 25 to 50 mg (maximum, 2 g) IV/IO over 15 to 30 minutes Hypomagnesemia: ■ 25 to 50 mg/kg IV/IO over 10 to 20 minutes	Rapid infusion may cause hypotension or bradycardia. Have calcium chloride available to reverse magnesium toxicity if it occurs.
Mannitol	Osmotic diuretic; induces the movement of intracellular water into the extracellular and vascular spaces, thereby reducing intracranial edema (i.e., swelling) and pressure	Increased intracranial pressure	■ 0.25 to 1 g/kg IV/IO over 20 to 30 minutes ■ Repeat as needed to maintain serum osmolality <320 mOsm/kg	■ Use in conjunction with other measures aimed at reducing intracranial pressure (e.g., elevating head of bed, sedation) ■ Monitor blood pressure and avoid hypotension ■ Insert urinary catheter when using mannitol ■ Monitor for serum hyperosmolality and intravascular volume depletion
Methylprednisolone	Synthetic glucocorticoid (steroid) with anti-inflammatory activity	■ Anaphylaxis ■ Upper airway edema due to other conditions (e.g., croup, as an alternative to dexamethasone) ■ Acute asthma exacerbation	■ 2 mg/kg IV/IO/IM as an initial dose ■ Maintenance dosing: 0.5 mg/kg IV every 6 hours or 1 mg/kg IV every 12 hours (maximum daily dose, 120 mg)	■ Monitor for hyperglycemia/glucose intolerance and hypokalemia ■ May cause hypertension and fluid retention
Milrinone	Phosphodiesterase 3 inhibitor with inotropic, lusitropic and peripheral vasodilatory properties	■ Certain forms of shock requiring inotropic support and/or afterload reduction ■ Conditions affecting the heart muscle (i.e., myocarditis or cardiomyopathy)	■ Loading dose: 50 mcg/kg IV/IO over 10 to 60 minutes ■ Infusion: 0.25 to 0.75 mcg/kg/min	■ May cause hypotension and ventricular arrhythmias. Monitor blood pressure and ECG continuously during use and ensure adequate intravascular volume ■ May omit loading dose in patients who are bordering hypotension
Naloxone	Competitively binds to μ-opioid receptors	Apnea or respiratory depression due to opioid overdose	If complete reversal of opioid effects is warranted: ■ 0.1 mg/kg (maximum, 2 mg) IV/IO/IM/SC every 2 minutes as needed If complete reversal of opioid effects is not warranted: ■ 1 to 5 mcg/kg IV/IO/IM/SC, titrated to effect Maintenance dosing: ■ 0.002 to 0.16 mg/kg/hr continuous IV/IO infusion	■ May precipitate acute withdrawal in opioid-dependent patients ■ In neonates, opioid withdrawal may be life threatening if not recognized and appropriately treated ■ To avoid complete reversal of analgesia, use lower doses of naloxone (1 to 15 mcg/kg) to reverse respiratory depression associated with therapeutic opioid use
Nitroglycerin	Vasodilator (venous > arterial)	■ Acute heart failure/cardiogenic shock ■ Hypertensive crisis ■ Myocardial ischemia	■ Infants and children: ○ Initial dosing: 0.25 to 0.5 mcg/kg/min IV/IO infusion ○ Increase by 1 mcg/kg/min every 15 to 20 minutes, as tolerated, to desired effect ■ Adolescents: ○ Initial dosing: 5 to 10 mcg/min IV/IO infusion ○ Increase to maximum of 200 mcg/min, as tolerated, to desired effect	■ May cause severe hypotension and paradoxical bradycardia ■ Use with caution in patients who have pre-existing hypotension or are volume depleted

Drug	Action	Indications	Administration	Precautions
Nitroprusside	Nitric oxide donor; induces vasodilation via smooth muscle relaxation	Low–cardiac output, high-SVR shock	■ Starting dose: 0.3 to 1 mcg/kg/min IV/IO; start at lowest possible dose and titrate to effect (maximum dose, 8 mcg/kg/min) ■ Mix in 5% dextrose in water and protect from light	■ May induce profound hypotension. Should be monitored continuously with an indwelling arterial line. Avoid accidental flushing or bolus injection of the IV line ■ Cover bottle, burette or syringe pump with foil to protect against breakdown by light exposure ■ May cause cyanide/thiocyanate toxicity, particularly with prolonged use or higher doses and in patients with hepatic or renal insufficiency; monitor for metabolic acidosis and monitor daily thiocyanate levels if rate of 3 mcg/kg/min exceeded
Norepinephrine	Acts on both α- and ß-adrenergic receptors to increase heart rate, contractility and vasoconstriction; increases systemic blood pressure and coronary blood flow	■ Certain types of fluid-refractory shock requiring vasopressor support	0.1 to 2 mcg/kg/min IV/IO infusion, titrated upward, as needed, to desired clinical effect	■ May cause tachycardia, reflexive bradycardia, arrhythmias and hypertension ■ Potent vasoconstrictor; extravasation can lead to necrosis. Phentolamine may be injected intradermally to counteract this effect
Phenylephrine	Pure α-adrenergic receptor agonist; increases blood pressure via vasoconstriction	Shock characterized by isolated peripheral vasodilation, with normal or increased cardiac output (e.g., neurogenic shock, anaphylactic shock that is refractory to epinephrine)	0.1 to 0.5 mcg/kg/min IV/IO infusion, titrated to desired clinical effect	■ May cause hypertension or reduce cardiac output ■ May induce reflex bradycardia ■ May cause severe peripheral vasoconstriction, resulting in tissue necrosis if extravasation occurs with IV use ■ May cause severe visceral vasoconstriction; monitor kidney function during use
Potassium chloride	Potassium supplement	■ Symptomatic hypokalemia (serum potassium <3.5 mEq/L) ■ Hypokalemia-associated arrhythmias (e.g., torsades de pointes, ventricular fibrillation, pulseless ventricular tachycardia, asystole)	■ 0.5 to 1 mEq/kg (maximum 40 mEq) IV infused at a rate of ≤0.5 mEq/kg/hr ■ Re-evaluate serum potassium concentration 1 to 2 hours after infusion completed and repeat dose as needed based on result	■ Potassium replacement should be accompanied by more immediate recommended pharmacologic and electrical therapies for arrhythmias/cardiac arrest ■ Ensure cardiac monitoring during IV replacement ■ Treat concurrent hypomagnesemia to maximize potassium reabsorption in the kidneys
Prednisone/ prednisolone	Synthetic glucocorticoid (steroid) with anti-inflammatory activity	■ Acute mild, moderate or severe asthma exacerbation ■ Upper airway edema in croup	■ 1 to 2 mg/kg/day PO/IV divided in 1 to 2 doses for 3 to 10 days total (maximum, 60 mg/day) ■ Taper dose if given for >10 days	■ No advantage of IV route over oral route if gastrointestinal absorption is not impaired ■ Monitor for hyperglycemia/glucose intolerance and hypokalemia ■ May cause hypertension and fluid retention ■ May cause adrenal suppression if given in supraphysiologic doses for prolonged periods

Drug	Action	Indications	Administration	Precautions
Procainamide	Class Ia antiarrhythmic (sodium channel blocker); delays repolarization and prolongs the QT interval	■ VT with a pulse (second line after cardioversion if unstable) ■ Supraventricular tachycardia (SVT)	15 mg/kg IV/IO over 30 to 60 minutes	■ Should not be used with amiodarone or other drugs that prolong the QT interval ■ May cause hypotension, decreased cardiac function, prolonged QT interval, torsades de pointes, heart block or cardiac arrest ○ If a ≥50% widening of the QRS interval or hypotension occurs during administration, withhold the remainder of the dose ■ Consultation with cardiologist strongly recommended when using this agent
Prostaglandin E_1	Naturally occurring vasodilator normally involved in maintaining patency of ductus arteriosus during fetal and early neonatal development	Suspected or confirmed ductal-dependent cardiac lesions (used as a bridging therapy to temporarily maintain or restore ductal dependency until corrective or palliative surgery can be done)	■ Initial: 0.05 to 0.1 mcg/kg/min IV/IO infusion, titrating up to 0.1 mcg/kg/min as needed ■ Maintenance: 0.01 to 0.05 mcg/kg/min IV/IO infusion	■ May cause apnea, hyperthermia or seizures; infusion should not be discontinued for any of these complications. Prepare to provide respiratory support, if needed ■ May cause hypotension. Ensure adequate intravascular volume replacement
Sodium bicarbonate	Alkalinizing agent	■ Hyperkalemia with widened QRS interval on ECG to prevent onset of VF ■ Severe metabolic acidosis (pH <7.15) including after prolonged cardiac arrest/resuscitation, that persists despite adequate oxygenation and ventilation ■ Sodium channel blocker (e.g., tricyclic antidepressant) toxicity	■ 1 mEq/kg IV/IO given slowly ■ For sodium channel blocker toxicity, titrate to maintain a serum pH of 7.45 to 7.55; follow with a 150 mEq/L infusion to maintain metabolic alkalosis	■ Routine initial use in cardiac arrest is not recommended ■ Ensure adequate ventilation before administering (to allow for effective elimination of excess CO_2 produced by bicarbonate) ■ Do not administer via ETT ■ Use the 0.5 mEq/mL (4.2%) concentration only for infants <1 month (or dilute available stock to this concentration)
Terbutaline	Systemic (i.e., injected) and short-acting selective ß2-agonist; bronchodilator	Acute, severe asthma exacerbation	■ Subcutaneously: 0.01 mg/kg every 20 minutes for up to 3 doses with a maximum of 0.25 mg ■ IV/IO infusion: 0.4 mg/kg/hr (starting dose), then 0.1 to 10 mcg/kg/min	May cause changes in blood pressure, heart rate and ECG; use with caution in patients with cardiovascular conditions, including arrhythmias and hypertension. May also cause transient hypokalemia, which may exacerbate the occurrence of arrhythmias
Tranexamic acid (TXA)	Antifibrinolytic agent; prevents blood clots from breaking down	Hemorrhage due to trauma	■ Loading dose: 15mg/kg (maximum, 1g) IV over 10 minutes (dilute in a convenient volume of NS) ■ Maintenance infusion: 2mg/kg/hr IV (suggested dilution: 500 mg TXA in 500 mL NS given at a rate of 2mL/kg/hr); give for at least 8 hours or until bleeding stops	■ Potential risk for thrombosis; use with caution in patients with a history of thromboembolic disease ■ Ureteral obstruction may occur in patients with upper urinary tract bleeding due to clot formation ■ Contraindicated in patients with subarachnoid hemorrhage due to anecdotal reports of cerebral edema and infarction ■ May cause defects in color vision; therefore, use is contraindicated in patients with defective color vision (as effect on color vision can be used as a determinant of toxicity)

Drug	Action	Indications	Administration	Precautions
Vasopressin	Pituitary hormone analog; promotes vasoconstriction via arterial V_1 receptors	Certain types of shock requiring vasopressor support (catecholamine resistant)	0.0002 to 0.002 u/kg/min IV/IO infusion	May induce water intoxication/ hyponatremia; use with caution in patients with renal dysfunction or pre-existing hyponatremia

SKILL SHEET

Opening the Airway

Head-Tilt/Chin-Lift Technique

To perform the head-tilt/chin lift technique:

1. Press down on the forehead while pulling up on the bony part of the chin with 2 to 3 fingers of your other hand.
2. Tilt the head to a slightly past-neutral position for children and to a neutral position for infants. Avoid hyperextension of the neck.
3. In infants, be careful not to place your fingers on the soft tissues under the chin or neck to open the airway.

Modified Jaw-Thrust Maneuver

If you suspect head, neck and spinal injury, use the modified jaw-thrust maneuver providing you can effectively maintain an open airway. To perform the modified jaw-thrust maneuver:

1. Position yourself above the patient's head.
2. Put one hand on each side of the patient's head with your thumbs near the corners of the mouth and pointed toward the chin. Use your elbows for support.
3. Slide your fingers under the angles of the jawbone without moving the patient's head or neck.
4. Thrust the jaw up (again without moving the head or neck) to lift the jaw and open the airway.

SKILL SHEET

Suctioning the Airway

Suctioning is used to clear the airway of excessive secretions. It is particularly important in patients with advanced airways, especially if mechanically ventilated, as normal airway clearance mechanisms are impaired and/or bypassed in these patients.

Step 1 Obtain equipment

When preparing to suction a patient, obtain all necessary equipment, including:

- Appropriately sized suction catheter (smaller and flexible or larger and rigid)
- Vacuum (e.g., wall or portable unit) and oxygen sources
- Gloves
- Personal protective equipment (e.g., gown and mask), as appropriate

Practice Note

Suctioning can be performed through the nose, the mouth, or basic and advanced airways such as OPAs, NPAs, endotracheal and tracheostomy tubes. To suction the mouth and pharynx, a larger, rigid (e.g., Yankauer) catheter may be used in lieu of smaller flexible catheters.

Step 2 Preoxygenate the patient

Preoxygenate the patient with 100% oxygen, as necessary.

Step 3 Insert the selected catheter

Insert the selected catheter to the desired depth.

Practice Note

Consider lubricating the end of the catheter if suctioning through the nose.

Alert

Stop advancing the catheter if you encounter resistance or if the patient exhibits signs of clinical deterioration.

SKILL SHEET

Suctioning the Airway *(continued)*

Step 4 Apply suction

- Cover the control port on the catheter.
- Maintain intermittent suctioning during withdrawal.
- Use slow, spiral motions to minimize injury to the mucous membranes.
- Limit suction intervals to 10 seconds to avoid mucosal damage and prolonged hypoxia.
- Monitor the patient for bradycardia, which can occur due to vagal stimulation. If this occurs stop suctioning.

SKILL SHEET

Basic Airway Insertion

Inserting an Oropharyngeal Airway

Step 1 Confirm the patient is unconscious and is without an intact gag reflex

An oropharyngeal airway, or OPA, can only be used on an unconscious patient who does not have an intact gag reflex.

Step 2 Measure for correct size

Measure the OPA to ensure it's the correct size by placing the flange against the corner of the patient's mouth. A properly sized OPA will extend from the corner of the mouth to the angle of the jaw.

Practice Note

If the OPA is too large, it may obstruct the airway. If the OPA is too small, it may push the tongue back against the posterior pharynx.

Step 3 Open the airway and mouth

- Make sure the airway is open to a slightly past-neutral position for children and to a neutral position for infants.
- Next, use the cross-finger technique to open the patient's mouth.

SKILL SHEET

Basic Airway Insertion *(continued)*

Step 4 Insert the OPA

- Use a tongue depressor to move the tongue out of the way and gently guide the OPA into the correct placement within the oropharynx.
- Or, insert the airway into the mouth sideways, rotating it 90 degrees as the tip approaches the back of the mouth.

Step 5 Check for proper insertion

- When properly inserted, the OPA fits over the tongue and holds it up and away from the posterior pharynx.
- The flange should rest on the patient's lips.

Step 6 Monitor the patient

- Once the airway OPA is inserted, monitor the patient and maintain a patent airway.
- Suction as needed.

Practice Note

Preoxygenate and limit suction intervals to 10 seconds to avoid mucosal damage and prolonged hypoxia.

SKILL SHEET

Basic Airway Insertion *(continued)*

Inserting a Nasopharyngeal Airway

Step 1 Determine that the patient has an intact gag reflex

- Unlike an OPA, a nasopharyngeal airway, or NPA, is typically used for a patient who has an intact gag reflex.
- The patient may be conscious or unconscious.

Step 2 Measure for correct size

Select an NPA that is smaller in diameter than the inner aperture of the patient's nostril and runs in length from the tip of the nose to the angle of the jaw.

Practice Note

An NPA that is too short won't separate the soft palate from the pharynx, which is necessary to open the airway. An NPA that is too long may cause bradycardia secondary to vagal stimulation or injure the epiglottis or vocal cords. It can also cause the patient to gag or vomit, increasing their risk of aspiration.

Step 3 Insert the NPA into the right nostril

- Apply a water-soluble lubricant to the opening of the right nostril, to the bevel and along the length of the tube.
- With the bevel of the NPA facing the nasal septum, insert the NPA gently into the right nostril, following the floor of the nose and avoiding excessive force.

SKILL SHEET

Basic Airway Insertion *(continued)*

Step 4 Alternative: Insert the NPA into the left nostril

- If you encounter problems inserting the airway, try the left nostril.
- When using the left nostril, insert the NPA with the bevel facing the nasal septum and then rotate it 180 degrees as you advance it beyond the nasal cavity.

Step 5 Check for proper insertion

- When properly inserted, the NPA extends to the posterior pharynx to provide a channel for air movement and suctioning.
- The flange should rest on the nostril.

Practice Note

Your ability to rotate the NPA in the nostril is a good indication of a proper fit.

Step 6 Monitor the patient

- Once the NPA is inserted, monitor the patient and maintain a patent airway.
- Suction as needed.

Practice Note

Preoxygenate and limit suction intervals to 10 seconds to avoid mucosal damage and prolonged hypoxia.

SKILL SHEET

Using a Bag-Valve-Mask (BVM) Resuscitator

Indications for BVM

When spontaneous breathing is absent or is insufficient to support adequate ventilation, assisted ventilation with a BVM is indicated.

Practice Note

Supplemental oxygen should be attached to the BVM as soon as appropriate. Doing so can increase the oxygen concentration to approximately 90% to 100%.

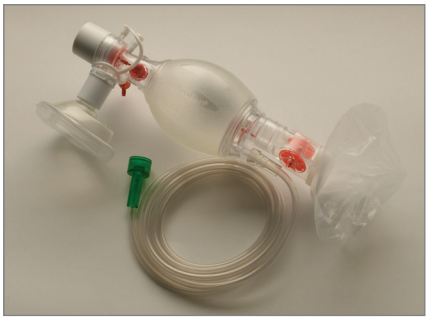
Oxygen Concentration

- BVM: 20% to 21%
- BVM with supplemental oxygen: 90% to 100%

Technique for One Provider

Step 1 Select a BVM resuscitator

Select an appropriately sized BVM device and BVM mask for your patient. Then, if necessary, assemble the BVM by connecting the BVM mask to the BVM device (or "bag"), most of which are self-inflating.



Practice Note

Some BVMs for children and infants also include a pressure relief or "pop-off" valve that helps to prevent excessive pressure during ventilations. In a resuscitation situation it is essential to deactivate the pop-off valve to assure adequate ventilation.

SKILL SHEET

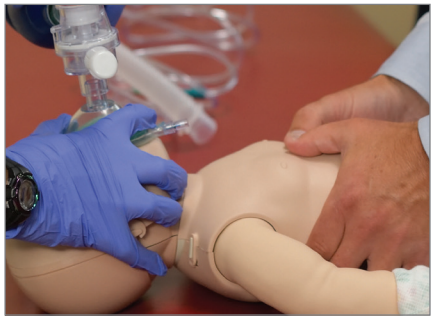
Using a Bag-Valve-Mask (BVM) Resuscitator *(continued)*

Step 2 Place the mask

- Position yourself behind the patient's head in a cephalic position.
- Place the mask at the bridge of the nose and then lower it over the patient's nose, mouth and chin. The mask should not extend past the patient's chin.

Step 3 Seal the mask and open the airway

- To hold the mask in place, position one hand around the mask, forming a "C" with your thumb and index finger and an "E" with the last three fingers. This is the E-C hand position.
- Simultaneously open the airway to a slightly past-neutral position for children and a neutral position for infants by lifting the jaw into the mask.



Step 4 Provide ventilations

- Deliver a ventilation by depressing the bag about halfway to deliver an appropriate volume.
- Deliver smooth, effortless ventilations that last about 1 second and make the chest begin to rise. Ventilations that are too fast or have too much volume can be dangerous. If there is insufficient time for exhalation then there can be over distension of the lungs which will impede further ventilation.

SKILL SHEET

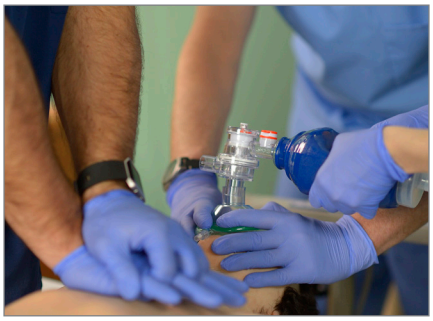
Using a Bag-Valve-Mask (BVM) Resuscitator *(continued)*

Technique for Two Providers

Although a single provider often uses a BVM, evidence shows that two providers are needed to most effectively operate the equipment.

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 places the mask at the bridge of the nose and then lowers it over the patient's nose, mouth and chin. The mask should not extend past the patient's chin.

Step 3 Seal the mask and open the airway

- Provider 1 holds the mask in place by positioning one hand around the mask, forming an "E" with the last three fingers and a "C" with the thumb and index finger—the E-C hand position.
- Provider 1 seals the mask completely around the patient's mouth and nose and simultaneously opens the airway to a slightly past-neutral position for children and a neutral position for infants by lifting the jaw into the mask.

Step 4 Provide ventilations

- Provider 1 maintains the mask seal and open airway.
- Provider 2 depresses the bag about halfway to deliver an appropriate volume.
- Provider 2 delivers smooth, effortless ventilations that last about 1 second and make the chest begin to rise. Remember, ventilations that are too fast or have too much volume can be dangerous.

SKILL SHEET

Measuring SpO_2 Levels by Pulse Oximetry

Oxygenation is most readily assessed using pulse oximetry, which noninvasively measures SpO_2 of hemoglobin.

Step 1 Select an appropriately sized probe

Step 2 Position the probe

- Position an appropriately sized probe on a finger, toe, earlobe or foot (for small infants). If using a finger, remove any nail polish from the nail.
- Avoid the arm limb being used for blood pressure monitoring, as cuff inflation will interfere with pulse oximetry readings.

Step 3 Check for an indication

Check for an indication that the probe has detected a pulse:

- If the probe has not detected the correct pulse, the reading will be inaccurate.
- Depending on the monitor, the indicator may be a pulse oximeter waveform or some other signal. A clear wave with dichrotic notch is evidence of accurate detection.

Practice Note

Hemodynamic factors such as poor perfusion or a weakened pulse may interfere with a pulse oximeter reading. Conditions affecting binding of oxygen to hemoglobin may also affect SpO_2 readings. Identification of these conditions is of paramount importance because they interfere with the binding of oxygen to hemoglobin, potentially threatening oxygen delivery to the tissues.

SKILL SHEET

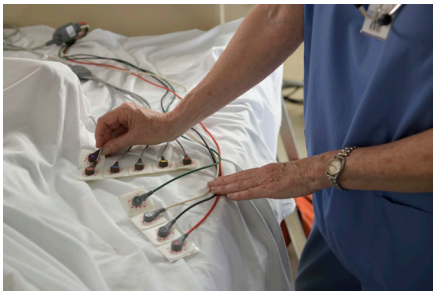
Placing Electrodes for Electrocardiography

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 Prepare to start treatment

Remove any oxygen delivery devices and metal objects from the patient's chest.

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.

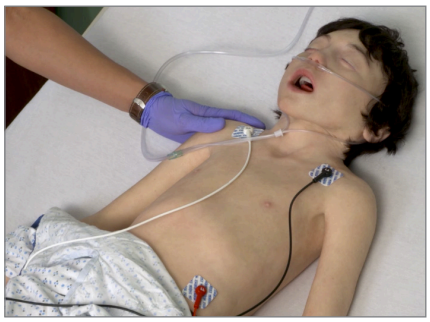
Note: If necessary, lift breast tissue to place electrodes as close to the chest wall as possible.

SKILL SHEET

Electrical Therapy

Performing Synchronized Cardioversion

Step 1 Apply the cardiac monitor leads



- Apply the defibrillator-compatible ECG leads to the patient. Normally, three color-coded leads are used:
  - White is the negative lead. It's placed under the right clavicle along the midclavicular line.
  - Red is the positive lead. It's placed on the left lower ribs with the electrode on the lower left abdomen.
  - The last lead is the ground. It's usually black, green or brown. It's placed with the electrode under the left clavicle along the midclavicular line.
- Select the lead with the tallest R waves for display (to optimize sensing of the machine).

Step 2 Confirm the cardiac rhythm

- Confirm there is a cardiac rhythm that warrants synchronized cardioversion.
- Cardiac rhythms that warrant synchronized cardioversion include:
  - Unstable or medication-refractory supraventricular tachycardia
  - Atrial fibrillation
  - Atrial flutter
  - Ventricular tachycardia with pulse

SKILL SHEET

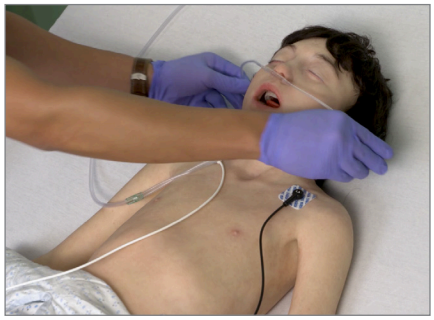
Electrical Therapy *(continued)*

Step 3 Prepare for cardioversion

- If the patient is unstable, prepare for immediate cardioversion. In such cases, electrical therapy takes priority over all other resuscitative procedures.
- When performing elective cardioversion for a stable patient, sedation or analgesia may be used.

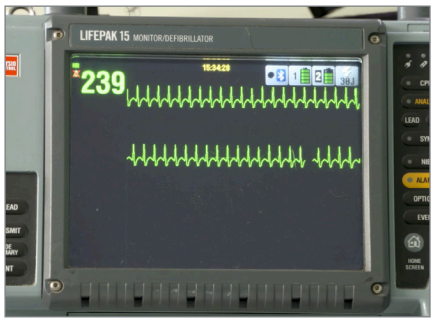
Step 4 Remove oxygen and metal objects

Remove any oxygen delivery devices and metal objects from the patient's chest.



Step 5 Check the monitor

Make sure the cardiac rhythm is displayed on the cardiac monitor/defibrillator.



Step 6 Select the appropriate pad size

Use pediatric pads for children and infants weighing less than 10 kilograms and adult pads for children weighing 10 kilograms or more.

Practice Note

Always use the largest size pad that will fit on the patient's chest without touching or overlapping.

SKILL SHEET

Electrical Therapy *(continued)*

Step 7 Position the pads

Position the pads on the front of the patient's chest:

- Place the sternal pad just below the right clavicle, lateral to the sternum.
- Place the apical pad between the fourth and fifth rib spaces, just lateral to the left nipple.
- Ensure at least 3 centimeters of space between the pads.
- Apply firm pressure when placing the self-adhesive pads.

Practice Note

Always follow the manufacturer's direction for pad placement.

Practice Note

Some electrode pads provide a green light signal when they are placed correctly.

Step 8 Alternate pad position

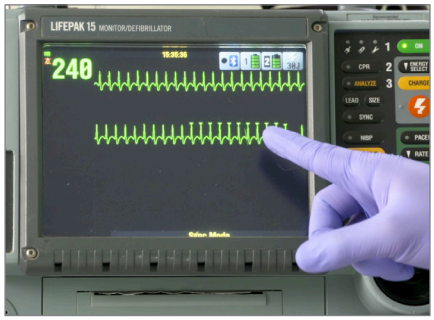
Use an anterior-posterior position if the pads risk touching or overlapping on the patient's chest or if the patient has a pacemaker.

SKILL SHEET

Electrical Therapy *(continued)*

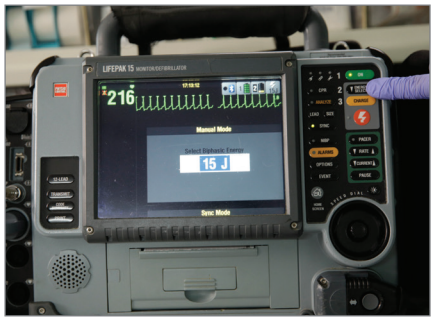
Step 9 Set to synchronized mode

- Set the defibrillator to the cardioversion synchronized mode.
- Sync markers should appear at the top of each R wave on the monitor, indicating that the defibrillator is in synchronized mode.
- If the defibrillator is not syncing, try selecting another lead on the monitor.



Step 10 Choose the initial energy dose

For synchronized cardioversion, select 0.5 to 1 J/kg.



Step 11 Charge the pads

Charge the pads by pressing the “Charge” button on the defibrillator.

SKILL SHEET

Electrical Therapy *(continued)*

Step 12 Deliver an "all clear" verbal warning

- Immediately before cardioverting, deliver a verbal warning for all providers to stand clear of the patient and the bed or stretcher.
- Look to make sure all providers are clear of the patient.

Step 13 Deliver the shock

- Press and hold the button on the defibrillator until the shock is delivered (this may take more than 1 second).
- For the patient who is spontaneously breathing, time the shock with the end of an expiration.

Step 14 Reassess the patient and the rhythm

- If the arrhythmia persists and the indication for cardioversion remains, return the machine to synchronized mode and deliver an additional shock.
- Set the energy dose to 2 J/kg on subsequent attempts of synchronized cardioversion and recharge the defibrillator each time. If starting with 0.5 J/kg, may increase dose more gradually to 1 J/kg.

SKILL SHEET

Electrical Therapy *(continued)*

Performing Manual Defibrillation

Step 1 Recognize the shockable rhythm

Shockable rhythms include:

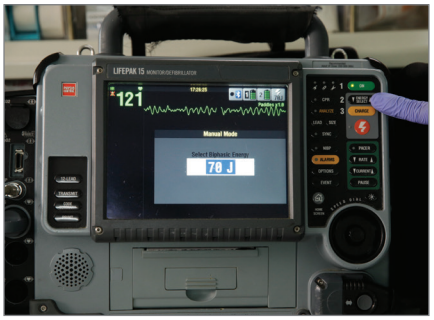
- Ventricular fibrillation
- Pulseless ventricular tachycardia

Practice Note

Any time a manual defibrillator is being used, CPR would be in progress. The team should continue to provide compressions until the pads are in place and you are ready to do a rhythm check or deliver a shock.

Step 2 Position the pads and select the initial energy dose

- First attach the pads to the patient's chest or chest and back.
- Then, set the initial energy dose to 2 J/kg.



Step 3 Charge the pads

Charge the pads by pressing the "Charge" button on the defibrillator.

SKILL SHEET

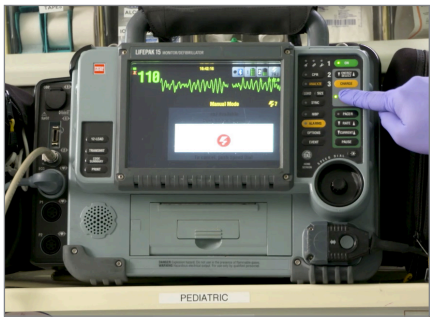
Electrical Therapy *(continued)*

Step 4 Deliver an "all clear" verbal warning

- Immediately before discharging the shock, deliver a verbal warning for all providers to stand clear of the patient and the bed or stretcher.
- Look to make sure all providers are clear of the patient.

Step 5 Deliver the shock

Deliver the shock by pressing the "Shock" button on the defibrillator.



Step 6 Reassess

- Immediately resume CPR, and then reassess the rhythm after 2 minutes.
- If a shockable rhythm persists, deliver another shock, using 4 J/kg. Remember to continue CPR between shocks.

Practice Note

If additional shocks are necessary, deliver shocks using ≥4 to maximum 10 J/kg, or the adult dose.

SKILL SHEET

Achieving Intraosseous Access (Drill)

Indications and Contraindications for Intraosseous Use

Intraosseous (IO) access is a reliable and effective alternative during cardiac arrest or other emergency if an intravenous (IV) catheter cannot be placed. This procedure can be safely and efficiently performed on children of all ages, including in newborns. An IO needle can be used for administering resuscitation drugs, fluids, and blood products and for collecting blood for laboratory analyses. Medication dosing is the same for IO and IV routes.

⚠ Alert

Contraindications to IO needle placement include bone fractures or disorders that predispose to fracture (e.g., osteogenesis imperfecta), infection or burns of the overlying skin, and previous IO site in the target bone.

Step 1 Choose a device and an insertion site

IO access can be achieved using an IO drill or other device or manually. This skill sheet illustrates how to achieve IO access using an IO drill. This skill illustrates how to achieve IO access at the medial aspect of the anterior proximal tibia on a child. The steps are the same when achieving access on an infant or in other locations such as the femur or humerus.

Practice Note

The medial aspect of the anterior proximal tibia is the preferred IO access site for children and infants, but other IO access sites used in pediatrics include the distal tibia, the distal femur, the distal radius and the proximal humerus. Note that the distal radius is not a recommended site of administration when using an IO drill.

Step 2 Position the patient

Extend the extremity out to get easy access to the medial aspect of the anterior proximal tibia.

Practice Note

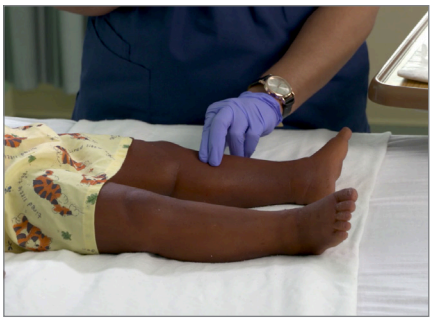
If you need to, you can place a rolled towel under the knee to better access the site, which is 1 to 2 centimeters below the tibial tuberosity.

SKILL SHEET

Achieving Intraosseous Access (Drill) *(continued)*

Step 3 Choose the correct needle length

- To determine the needle length that you'll use, check the tissue depth at the insertion site.
- Palpate the site with your fingers to see how much tissue there is before you reach bone.

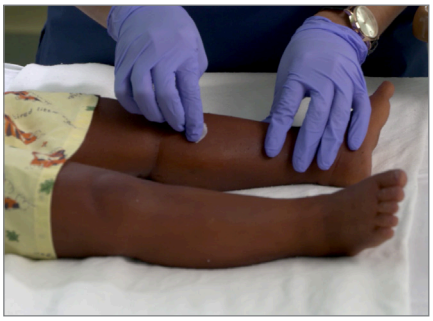


Practice Note

There are different-length needles available for different-sized pediatric patients. It's important to check the tissue depth at the insertion site to make sure you have the right needle size. If it's too short, the needle can become displaced and can cause tissue infiltration or extravasation of the site. In infants or smaller children, it could pass through the bone to the other side if it's too long.

Step 4 Disinfect and anesthetize the site

- Disinfect the skin overlying the insertion site using an antiseptic wipe or approved alternative.
- If the patient is awake, consider local infiltration of the skin and periosteum with 1% lidocaine to prevent pain at the site.

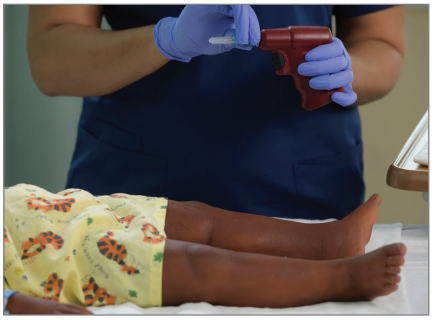


SKILL SHEET

Achieving Intraosseous Access (Drill) *(continued)*

Step 5 Prepare the intraosseous drill

- Attach the needle to the IO drill.
- Aim the IO drill with the needle at a 90-degree angle to the insertion site.



Practice Note

Make sure to insert the needle away from the growth plate found at the ends of long bones.

Step 6 Insert the needle

Push the needle through the skin until the tip is against the bone.

Practice Note

The 5-mm mark on the needle must be showing above the skin while the needle is resting on the bone for confirmation of adequate needle set length.

Step 7 Drill the needle into the bone

Press the trigger of the device and lightly drill the needle into the bone until you feel a decrease in resistance. This often feels like a "pop" when the needle enters the marrow. Immediately release the trigger.

SKILL SHEET

Achieving Intraosseous Access (Drill) *(continued)*

Step 8 Hold the needle securely in place as you pull the IO drill straight off

Practice Note

When inserted correctly, the needle should feel like it is firmly in the bone and should remain upright without support.

Step 9 Twist off the stylet

Twist off the sylet using a counter-clockwise motion and dispose of it properly.

Step 10 Apply a stabilizer dressing

Place a stabilizer dressing over the needle.

Step 11 Attach the primed tubing

While you're holding the needle securely, attach the primed tubing to the needle.

SKILL SHEET

Achieving Intraosseous Access (Drill) *(continued)*

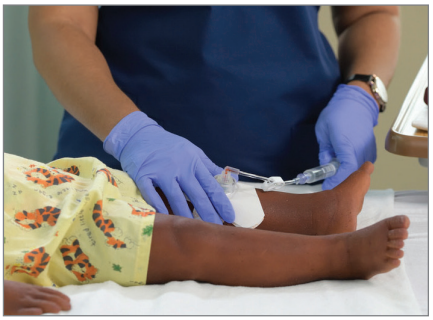
Step 12 Confirm correct needle placement

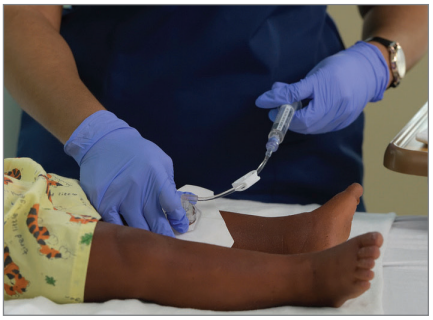
Confirm correct placement of the needle by:

- Aspirating bone marrow or blood through the needle.
- Flushing the needle with a small amount of saline and checking for infiltration.
- Administering fluids by free flow through the needle.

Practice Note

If fluids do not infuse via free flow in a properly placed IO site, use an IV pump or pressure bag.





Step 13 Secure the needle and tape the IV tubing

- Secure the needle by peeling the adhesive tabs off the stabilizer dressing and pressing it firmly onto the skin to secure it.
- Tape the IV tubing to the child's or infant's skin. Taping can prevent unwanted pulling on the inserted needle, which could lead to the needle becoming displaced.

Step 14 Start your treatment and monitor the area

- Once the IO needle has been successfully inserted, you are now ready to start your treatment.
- Monitor the area around the IO needle and the overall extremity for swelling, which may indicate that the needle is out of place.

Alert

If the needle becomes displaced, it can lead to complications, including infection, medication or fluid extravasation, and compartment syndrome.

CHAPTER 4

Working Well Together in an Emergency

Introduction

Working well together as a team when caring for a child or infant experiencing a respiratory emergency, shock or a cardiac emergency is vital. It has been well established in the literature that poor teamwork can lead to poor outcomes in emergency situations. Effective teamwork really can be the difference between life and death for the pediatric patient who requires emergency response or resuscitation.

Pediatric 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. 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 events because the ultimate goal is to save a life. Effective team care requires a coordinated effort by the team leaders and the team members.

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 (Figure 4-1). The goal of the **resuscitation team** is to respond quickly and effectively to provide pediatric advanced life support care to a patient in respiratory or cardiac arrest (Figure 4-2).

Pediatric patients in respiratory distress or shock tend to decompensate quickly, and respiratory and cardiac arrest may result. In critical care settings such as a pediatric intensive care unit (PICU) or emergency department (ED), highly trained teams are already in place and pediatric 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.

Skills and Best Practices

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 improves patient outcomes.

Communication

Communication is essential when caring for a child who is experiencing signs and symptoms of shock or a pediatric respiratory or cardiac emergency. You need to communicate with your team, the patient and the patient's family. Communication includes spoken words

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

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

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

Communication involves four essential components:

- **Sender**: The person initiating the communication
- **Message**: The content of the communication 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**

Communicating with the Team

The foundation of effective teamwork is clear and effective communication among team members (Figure 4-3). When a team is working to provide care, a designated team leader directs the efforts of the other team members.

When communicating (sending) information:

- Speak clearly and deliberately.
- Convey information in an organized fashion.
- "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.

When receiving information:

- Provide confirmation that you have received the message and that you understand it by repeating the task back to the sender.
- Acknowledge initiation and completion of the task.
- Speak clearly in a calm tone of voice; avoid speaking over others.

Communicating with the Family

Children 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 child's or infant's condition. Minimize family members' fears, as necessary, but avoid giving any 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 (Figure 4-4).

Communicating with the Family After a Patient's Death

In pediatric advanced life support situations, patients may not survive despite the team's best resuscitation attempts. As a healthcare provider, you may be involved in communicating with the family about a patient's death. Dealing with death is difficult, even for healthcare professionals. In this situation:

- Provide the 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 a myriad of reactions by family members, such as crying, sobbing, shouting, anger, screaming or physically lashing out.
- Wait and answer any questions that the family may have.

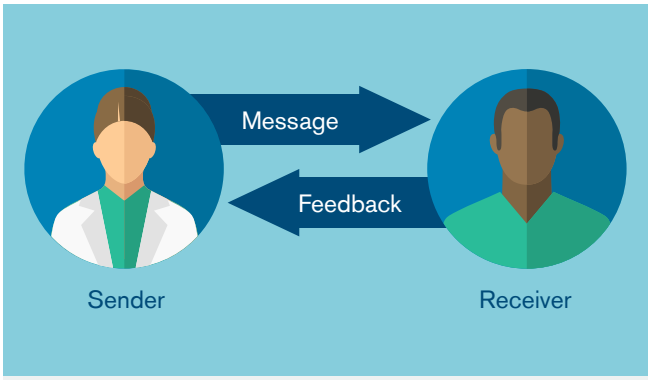


Figure 4-3 | Clear communication among team members is essential. The sender initiates the communication, sending a clear message. The receiver provides feedback, confirming that the message was understood and the task has been completed.

Figure 4-4 | Communication with the family is very important.

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 and situations, 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 pediatric advanced life support situations. You use critical thinking when you:

- Perform a rapid assessment and determine a course of action.
- Anticipate roles and functions as part of a team based on the patient's presentation and condition.
- Re-evaluate the situation for changes, interpret these changes and modify patient care accordingly.

Problem Solving

Problem solving refers to the ability to use readily available resources to find solutions to challenging or complex situations. In emergency situations, problems or issues can happen at any point. For example, the automated external defibrillator (AED) may be delayed in arriving or have a low battery. A parent may be upset and interfere with care.

> Problem solving often requires creativity and adaptability.

Use whatever resources are at hand, including equipment, other team members or other healthcare facility staff.

Practicing and Debriefing

Members of effective high-performance teams keep their skills and knowledge current, and they practice together regularly (Figure 4-5, A). In addition, effective high-performance teams hold debriefing sessions after each resuscitation event (Figure 4-5, B).

The purpose of the debriefing session 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 team leader leads the debriefing session, which typically follows a consistent format:

- **Review**: The team leader provides a brief recap of the emergency and the interventions that were used.
- **Analyze**: The team reviews and evaluates the qualitative and quantitative 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.

The debriefing phase is also a time for the team members to decompress. Some resuscitations can be very traumatic or emotionally laborious; this phase can be a time for the team to grieve.

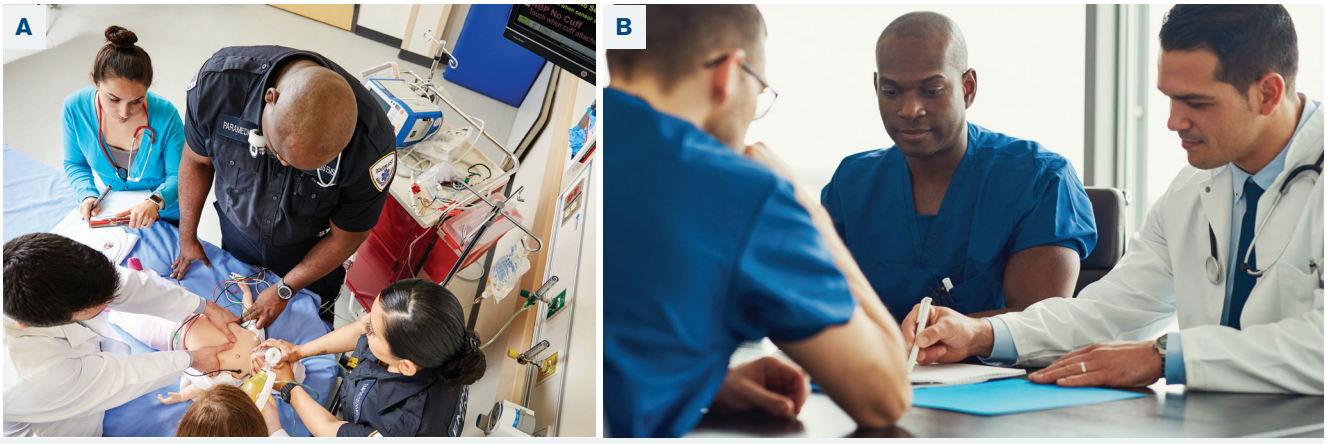


Figure 4-5 | Effective teams (A) practice together regularly and (B) hold debriefing sessions after every resuscitation event.

Roles and Responsibilities

Your role on a pediatric advanced life support team may vary according to your training and areas of expertise and scope of practice. In addition to understanding your own role on the team, it is important to understand the roles of other team members as well.

Team Leader

The team leader oversees the entire emergency situation and organizes and runs the response or code. 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 to ensure that everyone works as a team to help promote the best possible outcome for the patient.

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 Members

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 that they are lacking any knowledge or skills to perform assigned roles.
  - 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 debriefing sessions.

Crew Resource Management

Crew resource management is a concept that helps to promote effective and efficient teamwork and reduce the likelihood of errors. 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.

Crew resource management also guides team members to communicate directly and effectively with the team leader about dangerous or time-critical decisions. 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.

> 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.

Six-Person High Performance Resuscitation Team

A typical six-person high-performance resuscitation team includes team members who perform the three CPR/AED roles, as well as team members who perform the three leadership and supportive roles. 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 (Figure 4-6).

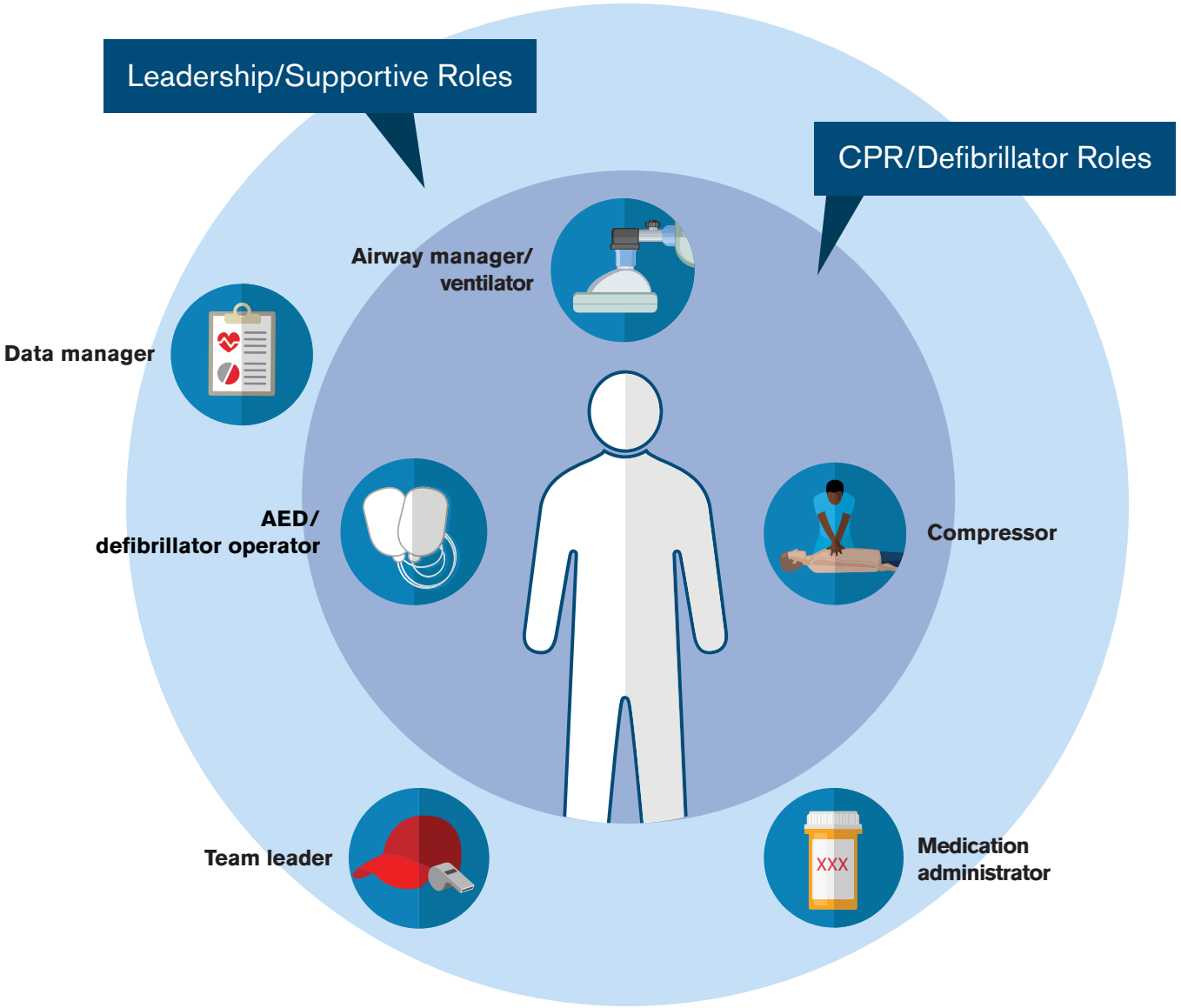


Figure 4-6 | A resuscitation team usually includes six people.

- **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 may be the team member who also relieves the team member providing compressions, depending on facility policy.
- **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.
- **Team leader**. One team member is responsible for prioritizing and directing the other team members' actions.
- **Medication administrator**. One team member is responsible for establishing intravenous or intraosseous access and administering medications.
- **Data manager**. One team member is responsible for communicating and recording key data during the resuscitation effort (for example, data related to medication administration and interruptions to chest compressions).

Note: This is a suggested team formation. Team members and roles may differ according to facility policy.

CHAPTER 5

Systematic Assessment

Introduction

A systematic approach to assessing a respiratory emergency, shock or a cardiac emergency is critical to quick and accurate assessment of the seriousness of an illness or injury in a pediatric patient. Rapid recognition of the signs and symptoms of a respiratory emergency, shock or a cardiac emergency allows for immediate care. Immediate care is especially critical in a respiratory emergency, shock or a cardiac emergency because pediatric patients, especially young children and infants, may quickly progress from a serious illness or injury to respiratory failure, respiratory arrest and cardiac arrest.

Assess, Recognize and Care

As a healthcare provider who finds a child or infant experiencing signs and symptoms of a respiratory emergency, shock or a cardiac emergency, you must act quickly and precisely to assess the patient, recognize the condition and implement the appropriate care based on your findings. This systematic approach is the assess, recognize and care (ARC) concept. This concept is critical for quick and accurate assessment, rapid recognition and immediate care in emergency situations. The ARC concept is a continuous cycle (Figure 5-1).

> A pediatric patient's condition can change rapidly and deterioration can follow; therefore, frequent reassessment, recognition and care are critical.

You should repeat these steps until the patient is stabilized and/or transferred to a higher level of care for further management. For example, once you've performed a care intervention, you need to reassess to identify whether that measure has been effective, then determine through recognition what the patient's condition is. This would then be followed by applying another appropriate care measure; and the cycle continues.

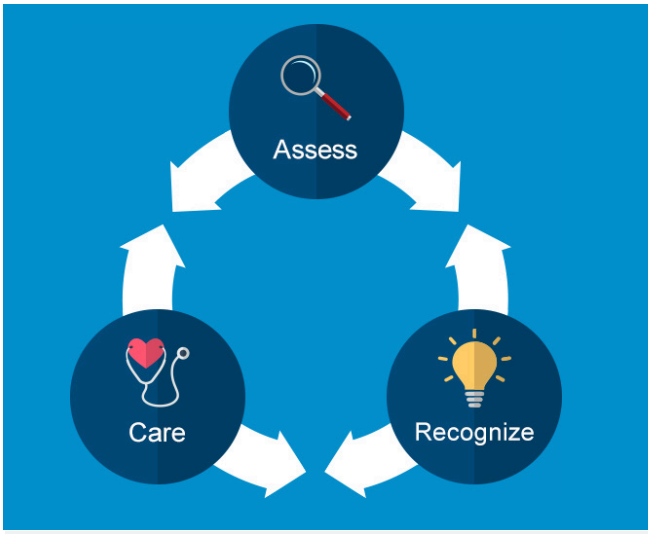


Figure 5-1 | Assess, recognize and care (ARC) is a concept that describes the ongoing process of gathering data about the patient's condition, using that data to identify a problem, and then intervening to address the problem.

Assess

Assessment is the process of gathering the data that helps you to determine what is happening with the patient. 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. These assessment techniques furnish critical physical, physiological and, in some cases, psychosocial data. The findings gathered from the assessment will help you recognize the emergency condition and then provide effective care. In an emergency situation, assessment is ongoing.

Recognize

After you gather assessment data, use critical thinking, your 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.

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. Care may include initiating CPR measures, repositioning the patient, administering intravenous fluids or oxygen, or simply collecting vital signs and continuing to monitor the child or infant for changes. After providing care, it is important to reassess to identify whether the measures have been effective.

Systematic Approach to Assessment

A systematic approach to assessment includes a rapid assessment, a primary assessment and a secondary assessment (Figure 5-2).

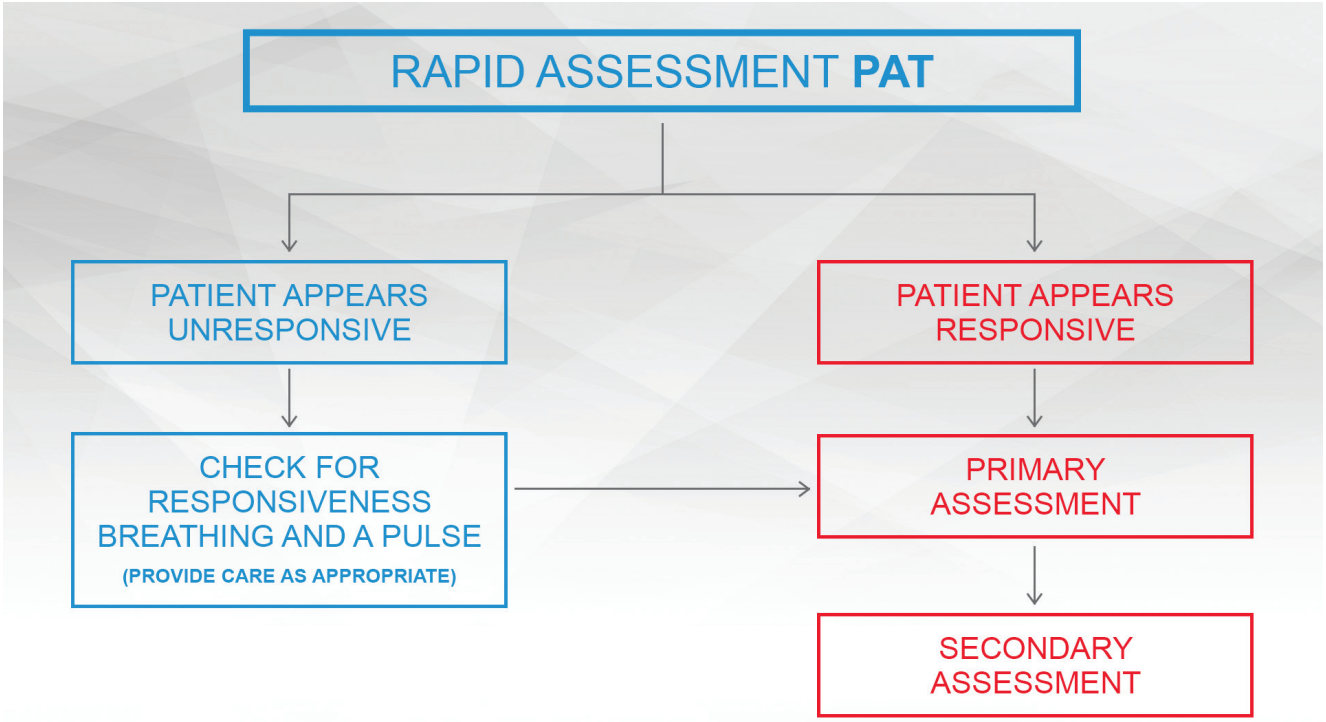


Figure 5-2 | A systematic approach to assessment includes a rapid assessment, a primary assessment and a secondary assessment.

Rapid Assessment: Pediatric Assessment Triangle

It is important to perform a quick visual survey of the emergency situation before collecting data or providing care. This quick survey allows you to make sure that the environment is safe for you and any individuals present during the event and to formulate an initial impression of the child or infant experiencing an emergency. An initial impression allows you to quickly recognize whether the patient is experiencing a life-threatening or a non-life-threatening condition, including life-threatening bleeding.

To form an initial impression of the patient, follow the Pediatric Assessment Triangle (PAT), which uses an A-B-C approach (Appearance, work of Breathing, Circulation).

During the visual survey, it also is important to quickly determine what additional resources you may need in the emergency situation. Ask yourself questions such as:

- Do I need a team member's help? Who is available to help?
- Are there additional resources, such as a rapid response team or resuscitation team or an advanced life support unit, available to respond?
- Do I need any additional equipment such as a defibrillator?

Appearance

The first step of the PAT is to promptly and carefully assess the patient's appearance and level of responsiveness. Ask yourself the following questions:

- Is the patient interacting? Moving? Gesturing?
- Is the patient speaking or crying?
- What does the patient's muscle tone look like?

In this step, you may follow the TICLS mnemonic (muscle **T**one, **I**nteractivity, **C**onsolability, **L**ook/gaze, **S**peech/cry) to assist with collecting this data.

If the patient appears unresponsive during the appearance step of the PAT, check for responsiveness using the shout-tap-shout sequence. Tap the shoulder of a child and the bottom of the foot for an infant.

If the patient is *unresponsive*:

- Call for help to activate EMS or the rapid response or resuscitation team and call for a cardiac monitor/defibrillator or AED.
- Ensure that the patient is in a supine position. (If the patient is face down, roll them on their back, taking care not to create or worsen any injury.)
- Open the airway to a slightly past-neutral position for children or a neutral position for infants. (Use the head-tilt/chin-lift technique or modified jaw-thrust maneuver to open the airway.)

- Simultaneously check for breathing and a central pulse (carotid for children; brachial for infants) for at least 5 seconds, but no more than 10 (Figure 5-3).
- If the patient is breathing and has a pulse, proceed to the primary and secondary assessments and provide care as appropriate and monitor until EMS, rapid response or resuscitation team arrives.
  - Note: It is a good idea to place the patient in a recovery position if you do not suspect a head, neck, spinal or pelvic injury after completing assessments and providing care, if indicated.
- If the patient is not breathing, but has a pulse, provide care for respiratory arrest.
- If the patient is not breathing and does not have a pulse, provide immediate basic life support care as appropriate.

If the patient appears or is responsive, but appears to have life-threatening airway, breathing or circulation compromise:

- Provide immediate care as appropriate before proceeding to the primary and secondary assessments.

If the patient appears or is responsive and does not appear to have life-threatening airway, breathing or circulation compromise:

- Complete the primary and secondary assessments and provide care as appropriate.

Work of Breathing

During this step, evaluate the patient's work of breathing, using visual and auditory assessments and the patient's body positioning. Ask yourself the following questions:

- Is the patient positioning (e.g., tripod position)?

Figure 5-3 | During the rapid assessment, the patient's appearance and level of responsiveness is assessed. If the patient is unresponsive, the healthcare provider checks for breathing and a pulse immediately.

- Can you hear the breath sounds without using a stethoscope? Are they abnormal (e.g., stridor, grunting, wheezing)?
- Are there signs of increased work of breathing or respiratory distress (e.g., nasal flaring, use of accessory muscles, intercostal or suprasternal retractions)?
- Does it appear that the patient is breathing too fast or too slow?
- Is there inadequate or absent respiratory effort?

In a normal state, a child or infant will exhibit regular breath sounds that should not be audible at a distance or without the use of a stethoscope. In addition, there will be no signs indicating increased effort to breathe.

Circulation

The final piece of the PAT is to assess the adequacy of the patient's circulation. To do this, inspect and observe the child's or infant's skin and mucous membranes (inside of the mouth and hard palate). Skin changes that indicate inadequate perfusion include:

- Pallor (paleness; however, gray, dusky skin color may be noted in patients with darker skin tones).
- Cyanosis (bluish skin color).
- Mottling (irregular blotchy color).
- Flushing (blushed appearance).

See *Rapid Assessment* Treatment Guideline for a summary of the rapid assessment.

ALERT

Because you are assessing the skin for signs of inadequate perfusion, you may also notice signs of bleeding, bruising or other skin injuries.

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.

Petechiae or purpura may indicate a serious life-threatening systemic infection. Follow your institutional protocols regarding PPE and appropriately isolating the patient.

Primary Assessment

Once the rapid assessment has been completed and you have established that the patient is not experiencing a life-threatening emergency, complete a

primary assessment, which includes gathering physical and physiological data to facilitate recognition of the underlying cause(s) of the patient's emergency condition (Figure 5-4).

The goal of the primary assessment is to identify potentially life-threatening conditions and correct them immediately to prevent the patient's condition from deteriorating further. 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.

ALERT

As you perform the primary assessment, be alert for signs that the patient's condition has worsened or for any change in areas already assessed. Delegate necessary initial interventions to the appropriate team members so that immediate care can be implemented as you continue the primary assessment.

How to Gather Physiological Data

You will begin to gather physiological measurements (vital signs), including temperature, pulse, respirations and noninvasive blood pressure, during the primary assessment. Collecting vital signs is a crucial part of the process of gathering data in order to recognize and care for a child or infant with signs and symptoms of shock or a respiratory or cardiac emergency condition. Therefore, expert skills in this area are important. Keep in mind that it may be necessary to modify the order in which you collect certain vital signs to ensure accurate measurements. For example, measuring blood pressure first, using an inflatable cuff, may cause the child or infant to become anxious or uncomfortable, thus causing a falsely high heart rate and respiratory rate.

Figure 5-4 | Primary assessment includes gathering physical and physiological data.

Temperature

Infants and children up to 2 years of age should have temperature measured in the following locations:

- Axillary (under the arm; armpit)
- Rectal (if a definitive core temperature reading is needed for infants older than 1 month of age; rectal temperatures should be avoided when contraindicated)

Children 2 to 5 years of age should have the temperature measured in the following locations:

- Axillary
- Tympanic (in the ear)
- Oral (when child can hold thermometer under the tongue)
- Rectal (if a definitive core temperature reading is needed; rectal temperatures should be avoided when contraindicated)

Any pediatric patient over the age of 5 years can have their temperature taken via the oral, axillary or tympanic route.

ALERT

Determine the most appropriate route based on time and the patient's condition. For example, taking an oral temperature would not be appropriate in a patient who is experiencing respiratory distress.

Pay attention to any external factors that may affect the patient's temperature reading:

- Is the patient unclothed in a cold room?
- Is the infant under a radiant warmer?
- Did the child drink a cold/hot beverage before taking an oral temperature?

Pulse

To determine heart rate, palpate the pulses, auscultate the heart sounds or view the cardiac monitor to observe the ECG waveform or pulse oximeter waveform.

Pulses can be palpated centrally or peripherally as follows:

- Central-brachial (in infants), femoral or carotid (in older children; carotid pulses should not be palpated bilaterally at the same time)
- Peripheral-radial, dorsalis pedis, posterior tibial

Measure the pulse for no more than 10 seconds (then multiply by 6 for a complete rate). Be prepared to initiate chest compressions if the heart rate is less than 60 beats per minute. In a routine assessment, count the pulse for one full minute. The normal heart rates for the pediatric patient can be found in Table 5-1.

When measuring the heart rate, take note of the patient's presentation and any influence it may have on the rate. For example, if the child or infant is screaming in pain, crying or anxious, it is reasonable for the heart rate to be elevated (tachycardia). In addition to using the normal ranges as a reference point, it is also important to consider the patient's normal baseline heart rate and any underlying chronic or acute clinical conditions.

Respirations

Normal breathing (eupnea) takes little effort. It should be passive, unlabored and relatively quiet. The normal respiration rates for the pediatric patient can be found in Table 5-1.

To measure the respiration rate, it's generally more effective to observe abdominal movements (stomach rise and fall) rather than the rise and fall of the chest, because respirations in small children and infants are primarily diaphragmatic. However, the chest wall can be closely observed (with the patient's shirt removed) for expansion symmetry. Count the respirations for 30 seconds and then multiply by 2. Continuously re-evaluate respiration rates during assessments.

Respirations that are irregular, increased or decreased can indicate a deteriorating clinical status. In addition, visible signs such as head bobbing and seesaw respirations are critical signs that the patient's respiratory status is worsening.

A sleeping infant may normally *not* take a breath for 10 to 15 seconds. However, anything *over* 15 seconds is considered apnea and indicates that breathing and/or ventilation is seriously impaired.

When auscultating the breath sounds, it's important to listen at the following areas:

- Anterior (mid chest, just to the left and right of the sternum)
- Lateral (under the armpits)
- Posterior (both sides of the back)

Listen for normal and abnormal breath sounds such as stridor, grunting, snoring, wheezing or crackles.

Oxygen Saturation

O_2 saturation over 94% is a normal finding. Consider supplemental oxygen for an O_2 saturation under 94%. If the patient is receiving 100% O_2 and has an O_2 saturation rate below 90%, advanced interventions may be needed.

Table 5-1 | Normal Pediatric Vital Signs

Age Group	Respiratory Rate	Awake Heart Rate	Systolic Blood Pressure	Diastolic Blood Pressure
Newborn	30–60	100–200	60–85	35–55
Infant (1–12 mo)	30–50	100–180	70–100	35–60
Toddler (1–2 yrs)	24–40	90–140	85–105	40–65
Preschooler (3–5 yrs)	20–30	80–130	89–115	45–70
School Age (6–12 yrs)	16–26	70–120	94–120	55–80
Adolescent (13–17 yrs)	12–20	60–100	110–135	60–85

QUICK ASSESSMENT FOR HYPOTENSION	
Age Group	**Systolic Blood Pressure**
Neonate	<60
Infant	<70
Toddler–School Age	<70 + (age in years x 2)
Adolescent	<90

Noninvasive Blood Pressure

No matter what type of noninvasive technique is used, the most important factor in accurately measuring blood pressure (BP) is the use of an appropriately sized cuff. The bladder of the blood pressure cuff should have a width about 40% of the mid-to-upper arm circumference. The cuff bladder usually covers 80% to 100% of the circumference of the arm.

Cuffs that do not fit properly *will* affect the accuracy of BP measurements. If the cuff size is too small, the reading will be falsely high. If the cuff size is too large, the reading will be falsely low.

Noninvasive blood pressure is normally measured in the upper arm, but if contraindicated based on the patient's condition, it can be measured in the lower arm/forearm, thigh or ankle. The normal blood pressure findings for the pediatric patient and a quick assessment for hypotension can be found in Table 5-1.

Carefully observe for hypotension and follow up by assessing for signs of shock and poor perfusion.

Sequence for the Primary Assessment

The primary assessment uses an A-B-C-D-E mnemonic to highlight the approach of evaluating the child or infant.

Airway

Assess the patient's upper airway to determine patency. Auscultate the movement of air by placing your ear close to the patient's nose and mouth. If the airway is patent, you will observe normal breathing and breath sounds in the patient. If the airway is not patent, the following signs may be noted:

- Increased work of breathing or respiratory distress (i.e., nasal flaring, use of accessory muscles, intercostal or suprasternal retractions)
- Abnormal breath sounds (i.e., stridor, grunting, wheezing)
- Absent breath sounds

If the patient's airway is not open and clear, patency must be restored and maintained immediately.

An obstructed but maintainable airway can be kept open manually using simple interventions like positioning the patient or applying manual maneuvers to open the airway (e.g., head-tilt/chin-lift technique or modified jaw-thrust maneuver). In addition, it is important to clear the airway by suctioning or removing any visible foreign bodies (do *not* do a blind finger sweep). Inserting a basic airway (oropharyngeal or nasopharyngeal airway) may be necessary to maintain the airway (see Chapter 3).

An airway that is obstructed and not maintainable with manual maneuvers, suctioning or a basic airway, necessitates the use of CPAP, noninvasive ventilation or an advanced airway (see Chapter 3).

> Consider immediate consultation with an advanced airway specialist for a difficult airway.

If the child's or infant's airway is obstructed and you suspect that the cause is a foreign body, perform the following care:

- If the child or infant is choking, but is able to cough, speak, cry or breathe, encourage coughing to clear the airway.
- If the child or infant cannot cough, speak, cry or breathe, perform immediate measures to clear the airway, including abdominal thrusts (children only), back blows and chest thrusts, as appropriate.
- If the child or infant becomes unresponsive, immediately begin CPR, starting with chest compressions.

Breathing

Assess the patient's breathing to determine the adequacy of ventilation and oxygenation. To assess the child's or infant's breathing, observe for:

- **Rate, depth and rhythm of the breathing**: Is the patient breathing very slow or very fast? Are the chest and abdominal movements normal? Is the rhythm normal or abnormal? Note air movement and chest expansion.
- **Effort of breathing**: Is there increased work of breathing, such as nasal flaring, use of accessory muscles, and intercostal or suprasternal retractions?
- **Auscultate breath sounds**: Are sounds such as wheezing, grunting, stridor, gurgling or crackling heard?
- **Voice or cry changes**: Are voice or cry changes (e.g., hoarseness, hot potato voice) noted?
- **Oxygen saturation**: Based on a pulse oximeter reading, what is the patient's oxygen saturation?
- **End-tidal carbon dioxide ($ETCO_2$)**: Establish capnography to monitor the adequacy of ventilation. $ETCO_2$ values in the range of 35 to 45 mmHg confirm adequacy of ventilation.

Administer supplemental oxygen as needed to maintain an oxygen saturation greater than 94%. If necessary, support breathing by delivering ventilations with a bag-valve-mask (BVM) resuscitator. Implement noninvasive or invasive ventilation as necessary. In the case of tension pneumothorax, perform immediate needle thoracentesis.

See Chapter 3, Tools and Therapies, for content related to oxygen delivery devices, using a pulse oximeter, capnography and using a BVM resuscitator.

Circulation

Assess the adequacy of perfusion. Decreased perfusion can lead to decreased oxygenation (hypoxia) of the tissues and vital organs and decreased supply of nutrients. Assessing the circulation in the patient can be accomplished by checking:

- **Central and peripheral pulses**: Are the pulses easily palpable? Are they bounding or weak and thready? Is the quality of the central and peripheral pulses much different?
- **Blood pressure**: Is the blood pressure decreased (hypotension)?
- **Heart rate and rhythm**: Is the heart rate increased (tachycardia)? Decreased (bradycardia)? Is an irregular ECG rhythm noted on cardiac monitoring?
- **Skin and mucous membrane color**: Is the patient pale (or gray/dusky), mottled, cyanotic (centrally and/or peripherally)? Are the mucous membranes pink or pale?
- **Skin temperature**: Does the skin feel cool to touch in a normothermia environment?
- **Capillary refill time**: Is the capillary refill time prolonged (greater than 2 seconds)?

Provide supportive care as needed. Establish intravenous access, intraosseous access or both for the administration of fluids, medications or both. Prepare for electrical therapy if indicated.

Other considerations to assess circulation include placing an indwelling catheter to monitor for normal urine output for age (1.5 to 2 mL/kg/hr for infants and young children and 1 mL/kg/hr for adolescents). In addition, central venous pressure (CVP) monitoring and invasive arterial pressure monitoring may be used to assess and guide care in critically ill patients.

See Chapter 3 for content related to cardiac monitoring, vascular access and electrical therapies.

Disability

During the disability assessment, quickly assess the child's or infant's neurological status. Assess the patient's level of consciousness, pupillary response, and blood glucose level and use clinical evaluation tools such as AVPU, the Glasgow Coma Scale (GCS) and TICLS.

- **Level of consciousness**: Is level of consciousness decreased? Is the patient confused? Suddenly agitated or difficult to arouse? Is seizure activity occurring?
- **Pupillary response**: PERRL (Pupils Equal, Round, Reactive to Light). Are the pupils pinpoint? Dilated?
- **Blood glucose**: Is the patient hypoglycemic, causing altered level of consciousness?

The AVPU scale uses four ratings to determine the pediatric patient's level of consciousness:

- **A**wake: Patient is alert and interactive and responds appropriately based on their developmental level.
- Responds to **V**erbal stimulation or voice: Patient responds only to verbal commands (e.g., shouting their name, talking or yelling).
- Responds to **P**ain: Patient can be aroused only by painful stimuli (e.g., pinching the trapezius area or performing a sternal rub).
- **U**nresponsive: Patient does not respond to any stimuli.

The GCS is a similar neurological assessment tool and is most commonly used for pediatrics. The GCS has three categories: eye opening, motor response and verbal response (Figure 5-5). Each is scored based on the patient's best response. The highest score that a patient can attain on the GCS is 15, the lowest score they can attain is a 3. The higher the score, the less the severity of neurological insult (i.e., a score of 15 means mild or no brain injury). The lower the score, the greater the insult or injury (i.e., a score of 3 indicates severe neurological insult/brain injury).

Exposure

As the last step of the primary assessment, check the patient's body for obvious signs of injury or illness and 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. Obtain the patient's weight and body temperature if not already done/available.

Practice Note

In children, medications are typically dosed based on weight. If a child's weight is not known or cannot be quickly and easily measured, use a length-based resuscitation tool (e.g., Broselow tape) tape to estimate the child's weight. The tool also provides predetermined medication doses correlating with the color block corresponding to the patient's length.

PEDIATRIC GLASGOW COMA SCALE (PGCS)				
	>1 year		**<1 year**	
EYE OPENING	Spontaneously		Spontaneously	4
	To verbal command		To shout	3
	To pain		To pain	2
	No response		No response	1
MOTOR RESPONSE	Obeys		Spontaneous	6
	Localizes pain		Localizes pain	5
	Flexion-withdrawal		Flexion-withdrawal	4
	Flexion-abnormal (decorticate rigidity)		Flexion-abnormal (decorticate rigidity)	3
	Extension (decerebrate rigidity)		Extension (decerebrate rigidity)	2
	No response		No response	1
	>5 Years	**2–5 Years**	**0–23 months**	
VERBAL RESPONSE	Oriented	Appropriate words/ phrases	Smiles/coos appropriately	5
	Disoriented/confused	Inappropriate words	Cries and is consolable	4
	Inappropriate words	Persistent cries and screams	Persistent inappropriate crying and/or screaming	3
	Incomprehensible sounds	Grunts	Grunts, agitated and restless	2
	No response	No response	No response	1
			TOTAL PEDIATRIC GLASGOW COMA SCORE (3–15):	

Figure 5-5 | The Pediatric Glasgow Coma Scale

If a head, neck, spinal or pelvic injury is suspected in the patient, consider spinal motion restriction.

While observing the patient's body, look for any unusual markings, injuries or signs of trauma, such as:

- Burns.
- Bleeding.
- Bruising.
- Petechiae and/or purpura.
- Rashes.
- Deformities.
- Fractures.
- Tenderness.

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

Skin temperature and color can also be noted during this assessment, providing valuable information about circulation and perfusion. However, it is important to keep in mind that small children and infants do not thermoregulate as well as adults; they can lose core body temperature must faster when their skin is exposed. Be sure to keep the areas not being actively assessed covered and warmed, or use equipment such as a radiant warmer for infants.

See *Primary Assessment* Treatment Guideline for a summary of the primary assessment.

Secondary Assessment

When the primary assessment is completed and the patient's condition remains stable, the secondary assessment takes place (Figure 5-6). This assessment is much more focused and detail oriented than the rapid assessment and the primary assessment.

The secondary assessment includes a focused history, focused physical assessment (and brief head-to-toe assessment) and diagnostic tests.

Figure 5-6 | During the secondary assessment, conducted when the patient's condition allows and time and resources permit, information is gathered with the aim of narrowing the differential diagnosis list, identifying underlying causes and determining candidacy for planned interventions.

ALERT

Throughout the secondary assessment, it is important to keep the ongoing ARC concept in mind. That is, the patient should be continuously reassessed to identify any changes in condition and the child's or infant's response to the clinical interventions.

Focused History

The focused history can be obtained through collecting data following the SAMPLE mnemonic, which consists of **S**igns and symptoms, **A**llergies, **M**edications, **P**ast medical history, **L**ast intake and output and **E**vents.

Signs and Symptoms

Identify any signs and symptoms that occurred at the onset of the illness or injury. Collect these data by interviewing the patient (if appropriate), the parent/legal guardian or any witnesses to the event. Signs and symptoms may include, but are not limited to:

- Fever.
- Change in appetite.
- Nausea or vomiting.
- Diarrhea.
- Difficulty breathing.
- Agitation or anxiety.
- Headache.
- Bleeding.
- Fatigue.

Allergies

Determine whether the patient has any known allergies to things such as drugs, foods, environmental items and latex. If allergies are noted, be sure to record what type of reaction that child or infant experiences when they encounter this allergen.

If the patient has no known allergies, it's still important to inquire whether the child or infant has been exposed to any substance that is known to be an allergen or toxin.

Medications

Check what medications (oral, inhalation, IV, injection, suppository) the patient is taking. This includes prescription medications, over-the-counter medications, vitamins, herbal supplements and any "home remedies."

Also explore the possibility that the patient ingested an inappropriate medication or substance. For example, does the child or infant have access to the medications or drugs that belong to someone else in their surroundings?

Past Medical History

Learning about the patient's past medical history is an important piece of the secondary assessment. Verify the child's or infant's past medical history by asking about:

- Complicated birth history.
- Hospitalizations.
- Surgeries.
- Previous illnesses.
- Significant chronic diseases.
- Dietary history.
- Immunization status (up-to-date on immunizations).

Last Intake and Output

Establish when the patient's last intake (meal) was. This may include liquid or food intake, by mouth or by enteral feeding. Along with asking what time/how long ago the intake occurred, note any details about the intake. For example, was the child or infant refusing food or having difficulty eating?

In addition, ask about the patient's recent voiding and bowel elimination habits (output) to evaluate where any disproportion exists between intake and output. Note: Output can also include vomiting, diarrhea and significant bleeding.

It's important to get a time of the last intake that is as accurate as possible, as it may affect advanced treatment interventions. For example, if the patient needs to be intubated or placed under general anesthesia, the risk for aspiration is increased if they have recently eaten or drank.

Events

Events include any occurrence that took place leading up to the patient's illness or injury. Note any treatment and the time between the onset of the event and the presentation of the illness or injury (including if this occurred outside of the healthcare facility).

Focused Physical Assessment

The information gathered from the rapid and primary assessments, as well as the focused history, assists in determining the primary area of concern and the extent of the focused physical assessment. In addition to a focused physical assessment, complete a head-to-toe assessment.

If the primary area of concern is respiratory, then a focused respiratory assessment should be done to include assessing the airways, inspecting the chest and auscultating breath sounds, checking pulse oximetry, and so on. If the concern in cardiac, then the assessment should include listening to heart sounds and lungs sounds, inspecting for edema, and so on.

Though a complete head-to-toe assessment should be performed, a focused assessment should be completed carefully.

Keep in mind that even though a systematic head-to-toe approach is recommended when performing a physical assessment, this may not always be the best method when assessing a child or an infant. Instead, it may be necessary to modify the order in which you assess certain systems.

In addition, anticipating normal developmental reactions in the patient during the secondary assessment is a key component. For example, the child may become fearful during the physical assessment due to an unfamiliar situation or fear of the equipment being used. Finally, as you perform the focused physical assessment, it is important to observe for physical cues or verbalizations of pain. Physical cues may include loud crying, grimacing or thrashing in an infant, or guarding, increased muscle tension and anxiety in an older child. Many institutionally available pediatric pain assessment tools can be used to help the healthcare provider determine the severity (0 to 10) of the pain.

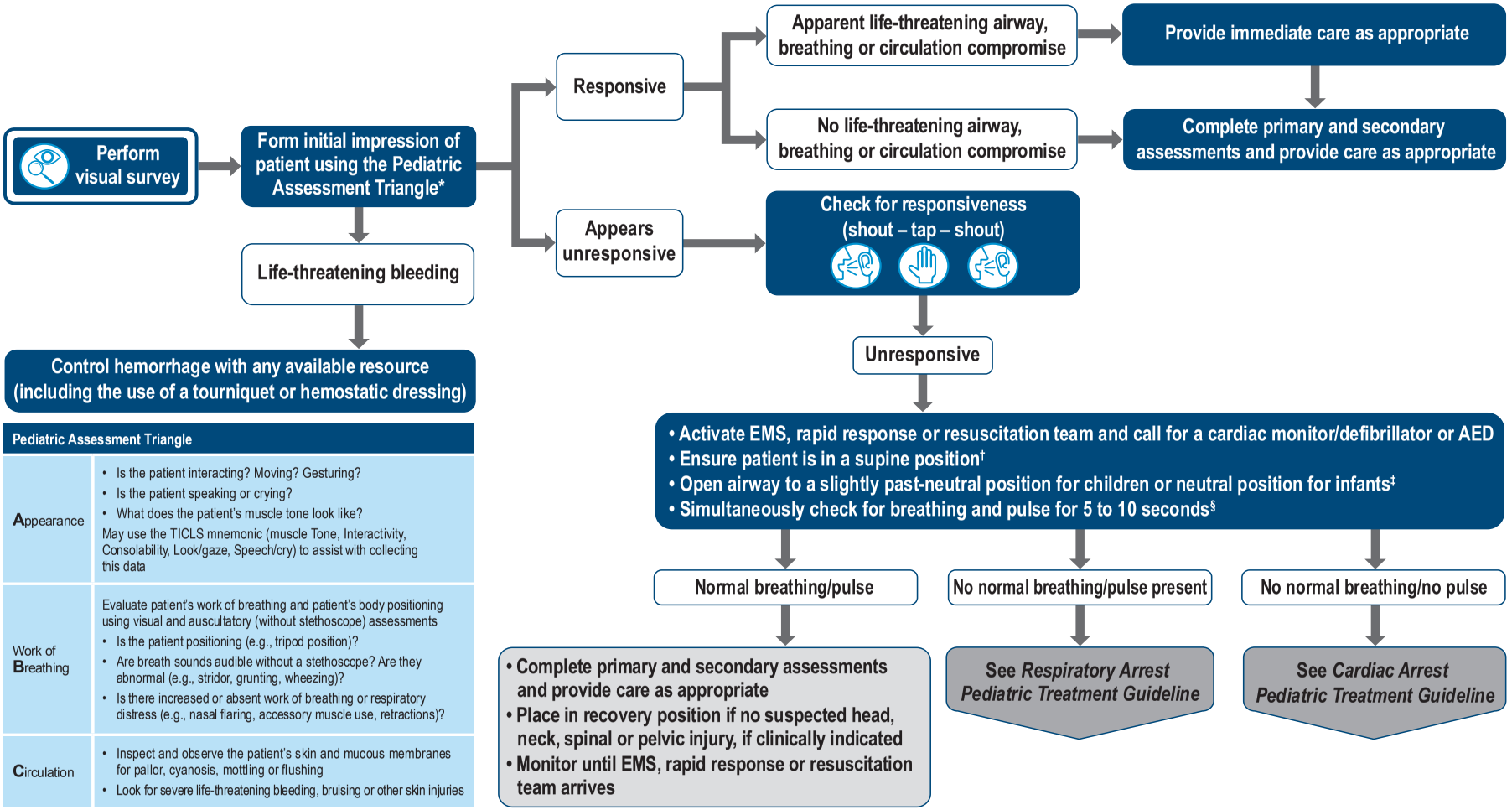
Laboratory and Diagnostic Tests

Laboratory and diagnostic tests may be ordered to assist in identifying underlying causes, narrow the list of differential diagnoses and aid in determining candidacy for (or contraindications to) planned therapeutic interventions. The following laboratory and diagnostic tests are often indicated in the evaluation of patients experiencing a respiratory emergency, shock or a cardiac emergency:

- **Blood tests**, including blood gas (arterial, venous or capillary), a complete blood count (CBC), an electrolyte panel, a blood glucose level, coagulation studies, hemoglobin levels, arterial lactate levels, coagulation panels, SvO_2 levels (central venous O_2 saturation), blood cultures, toxicology screens, renal and liver function tests
- **Diagnostic testing**, including sputum cultures, respiratory viral testing and peak expiratory flow rate (PEFR)
- **Imaging studies**, including radiography, computed tomography (CT), **echocardiogram**, magnetic resonance imaging (MRI), vascular imaging and ultrasound
- **Electrocardiography (12-lead)** (Figure 5-7)

Figure 5-7 | 12-Lead ECG

RAPID ASSESSMENT



Pediatric Assessment Triangle	
Appearance	• Is the patient interacting? Moving? Gesturing? • Is the patient speaking or crying? • What does the patient's muscle tone look like? May use the TICLS mnemonic (muscle Tone, Interactivity, Consolability, Look/gaze, Speech/cry) to assist with collecting this data
Work of **B**reathing	Evaluate patient's work of breathing and patient's body positioning using visual and auscultatory (without stethoscope) assessments • Is the patient positioning (e.g., tripod position)? • Are breath sounds audible without a stethoscope? Are they abnormal (e.g., stridor, grunting, wheezing)? • Is there increased or absent work of breathing or respiratory distress (e.g., nasal flaring, accessory muscle use, retractions)?
Circulation	• Inspect and observe the patient's skin and mucous membranes for pallor, cyanosis, mottling or flushing • Look for severe life-threatening bleeding, bruising or other skin injuries

*If discovered on circulation assessment, petechiae or purpura may indicate a serious life-threatening systemic infection. Follow your institutional protocols regarding personal protective equipment and appropriately isolating the patient.

†If patient is face down, roll them on their back, taking care not to create or worsen a suspected injury.

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

§Check carotid pulse in children and brachial pulse in infants.

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PRIMARY ASSESSMENT

Perform rapid assessment (PAT)

Airway

- Assess airway patency and maintainability

Breathing

- Assess:
  - Breathing rate, depth and rhythm
  - Breathing effort (e.g., nasal flaring, use of accessory muscles, presence of retractions)
  - Air movement and chest expansion
  - For changes in voice or cry
- Auscultate for the presence of abnormal breath sounds (e.g., wheezing, grunting, stridor, gurgling, crackles)
- Determine adequacy of oxygenation and ventilation:
  - Pulse oximetry: SpO_2 > 94%
  - Capnography: $ETCO_2$ 35 to 45 mmHg

Circulation

- Assess for presence and strength of central and peripheral pulses
- Measure blood pressure
- Assess heart rate and rhythm
  - Establish cardiac monitoring
- Assess adequacy of perfusion by checking:
  - Skin and mucous membrane color
  - Skin temperature
  - Capillary refill time
  - Urine output

Disability

- Assess level of consciousness and responsiveness using tools such as AVPU (alert, verbal responsive, pain responsive, unresponsive), Glasgow Coma Scale or TICLS
- Check pupils for size, equality and reactivity to light
- Measure the blood glucose level

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
- Obtain the patient's weight and body temperature, if not already done/available

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 modified jaw-thrust maneuver
- Clear foreign body airway obstructions
- Provide suctioning
- As needed, consider:
  - OPA or NPA placement
  - CPAP or noninvasive ventilation
  - Advanced airway
- Immediately consult an advanced airway specialist when a difficult airway is anticipated*

See *Partial Upper Airway Obstruction Pediatric Treatment Guideline* as appropriate

- Administer supplemental oxygen as needed to maintain an oxygen saturation > 94%
- Assist ventilation as needed using any of the following:
  - BVM ventilation
  - Noninvasive positive airway pressure
  - Invasive (i.e., mechanical) ventilation
- Perform needle thoracentesis for tension pneumothorax

See *Respiratory Arrest, Respiratory Distress or Failure, Lower Airway Obstruction, Partial Upper Airway Obstruction, Lung Tissue Disease, and Neurological and Metabolic Disorders of Ventilation Pediatric Treatment Guidelines* as appropriate

- Determine need for electrical therapy
- Establish IV/IO for administration of fluids or medications

See *Bradycardia With a Pulse, Stable Tachyarrhythmia, Unstable Tachyarrhythmia, General Shock, Hypovolemic Shock, Septic Shock, Anaphylactic Shock, Neurogenic Shock, Cardiogenic Shock, Obstructive Shock and Post–Cardiac Arrest Care Pediatric Treatment Guidelines* as appropriate

- Correct hypoglycemia if needed

See *Neurological and Metabolic Disorders of Ventilation and Post–Cardiac Arrest Care Pediatric Treatment Guidelines* as appropriate

- If suspected head, neck, spinal or pelvic injury, consider spinal motion restriction
- Manage body temperature

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

Please see reverse side for additional information

*Such as an anesthesiologist or otolaryngologist.

PRIMARY ASSESSMENT CONTINUED

Normal Pediatric Vital Signs

Age Group	Respiratory Rate (Breaths per Minute)	Awake Heart Rate (Beats per Minute)	Systolic Blood Pressure, mmHg	Diastolic Blood Pressure, mmHg
Newborn	30–60	100–200	60–85	35–55
Infant (1 to 12 months)	30–50	100–180	70–100	35–60
Toddler (1 to 2 years)	24–40	90–140	85–105	40–65
Preschooler (3 to 5 years)	20–30	80–130	89–115	45–70
School Age (6 to 12 years)	16–26	70–120	94–120	55–80
Adolescent (13 to 17 years)	12–20	60–100	110–135	60–85

Quick Assessment for Hypotension

Age Group	Systolic Blood Pressure, mmHg
Neonate	< 60
Infant	< 70
Toddler to School Age	< 70 + (age in years x 2)
Adolescent	< 90

Identifying Hypoglycemia

Plasma glucose threshold:

- Neonates: < 45 mg/dL
- Infant, children and adolescents: < 60 mg/dL

CHAPTER 6

Respiratory Emergencies

Introduction

Pediatric respiratory emergencies are a leading cause of emergency department visits and the main cause of cardiac arrest in children and infants. Children and infants are more vulnerable to airway obstruction and respiratory failure than adults because of differences in their anatomy and physiology. Respiratory emergencies can also occur as a result of nonpulmonary conditions, such as those affecting the heart, nervous system, muscles or metabolism. Assessing and recognizing a respiratory emergency in a child or infant is critical so that immediate care can be given to avoid respiratory failure, respiratory arrest and potential cardiac arrest.

Respiratory System Overview

Anatomy and Physiology of the Respiratory System

The anatomy of the respiratory system consists of an upper airway and a lower airway, each comprising multiple structures. These structures work together with various muscles to move air into and out of the body. Respiratory physiology encompasses the processes of oxygenation (i.e., delivering oxygen [O_2] to the blood) and ventilation (i.e., moving gases into and out of the lungs).

Understanding the normal anatomy and physiology of the respiratory system is essential to providing effective care, as is understanding the key anatomic and physiologic differences between adults, children and infants. These differences make children and infants more susceptible to acute airway obstruction, impaired gas exchange and respiratory compromise. Understanding these key differences can help the healthcare provider to recognize potential or actual problems quickly and provide effective care in a pediatric respiratory emergency.

Normal Respiratory Anatomy

In normal respiratory anatomy, the upper airway extends from the nostrils to the upper part of the trachea, ending at the level of the thoracic inlet (Figure 6-1). The lower airway encompasses the lower trachea, the bronchi, and the smaller airways and structures of the lungs (i.e., bronchioles and alveoli). The respiratory anatomy also includes various muscles involved in respiration, including the intercostal muscles of the ribs and the diaphragm.

Upper Airway Anatomy

The larynx is a central landmark of the airway. It serves as the "gatekeeper" between the oropharynx and the lower respiratory tract. The larynx consists of the vocal cords and several supportive cartilages, including the cricoid cartilage and the epiglottis. The structures of the upper airway can be categorized according to where they lie in relation to the larynx:

- **Supraglottis**: structures above the larynx (e.g., nasal and oral cavities, pharynx)
- **Glottis:** variably defined as the space between the vocal cords or as the structures surrounding the larynx
- **Subglottis:** structures below the larynx (upper portion of the trachea)

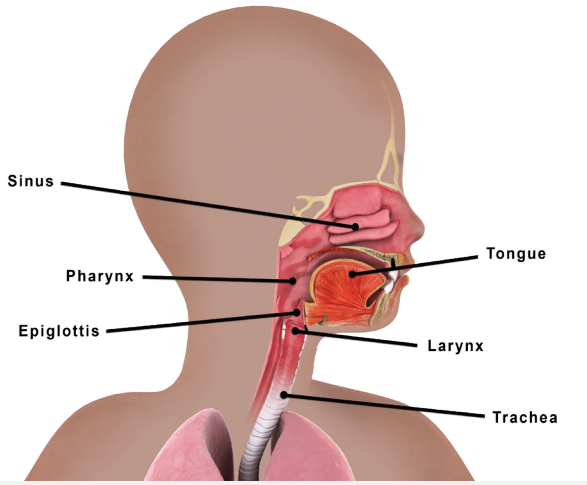


Figure 6-1 | Basic anatomy of the upper airway

The narrowest portion of the airway sits below the vocal chords in children. This anatomical consideration is important in pediatric intubation and airway management. The cricoid cartilage forms the only complete cartilage ring within the respiratory tract, making this portion of the pediatric airway especially susceptible to narrowing in the presence of mucosal edema (i.e., swelling). The epiglottis is a leaf-shaped cartilaginous structure that covers the glottis during the act of swallowing. It is an important landmark when performing endotracheal intubation.

Lower Airway Anatomy

The lower airway structures lie below the thoracic inlet and facilitate gas exchange in the lungs (Figure 6-2). Lower airway structures include:

- **Bronchi and bronchioles:** These branching airways act as conduits for the air entering and leaving the lungs. Three bronchi serve the right lung and two serve the left, corresponding with the number of lobes on each side.
- **Alveoli:** Air sacs known as alveoli at the very end of the smallest airways provide a thin surface for gas exchange between the lungs and the blood. The alveoli are "arranged" into clusters, known as alveolar sacs, each surrounded by a rich network of capillaries.

Muscles of Ventilation

The processes of inspiration and expiration are mediated by various muscles within the respiratory system. These include the diaphragm, a thick dome-shaped muscular sheet that separates the thoracic and abdominal cavities, and a group of muscles between the ribs called the intercostal muscles. The intercostal muscles connect neighboring ribs and consist of three layers.

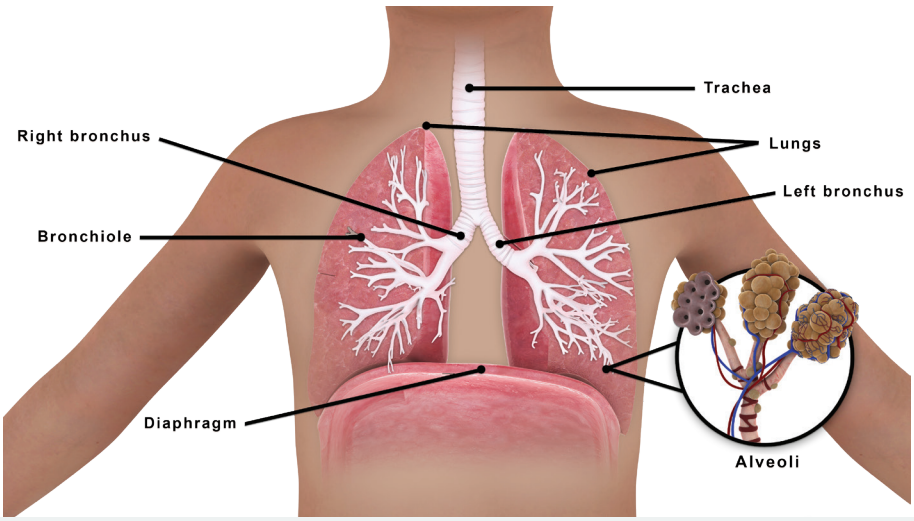


Figure 6-2 | Lower respiratory airway, diaphragm and (inset) gas exchange unit

Key Differences Between Pediatric and Adult Airway Anatomy

The airways of adults, children and infants have several key anatomical differences. Anatomical differences are most pronounced in infants younger than 1 year. By the age of 8 years, a child's airway is similar to that of an adult. These differences put children at greater risk for acute airway compromise. It's important to keep the clinical significance of these anatomic differences in mind as you care for children and infants experiencing respiratory emergencies (Table 6-1).

Normal Respiratory Physiology

The main functions of the respiratory system are to deliver O_2 to the blood (i.e., oxygenation) while removing carbon dioxide (CO_2) from the body.

Ventilation is the process by which air is delivered to the alveoli, allowing for the exchange of O_2 for CO_2 in the blood contained in the pulmonary capillaries. In addition to enabling gas exchange, ventilation plays a role in the regulation of acid-base balance in the body.

Control of Ventilation

Ventilation begins with the impulse to breathe. This is controlled by respiratory centers in the brain stem, which regulate nerve impulses to the diaphragm and intercostal muscles. The respiratory centers receive input from many sensors located throughout the body. These sensors detect changes in arterial O_2 and CO_2 content and in arterial pH, all of which can affect the rate or depth of respirations (Table 6-2). Other physiologic parameters that affect ventilation include core body temperature, muscle activity (e.g., during exercise and activity of the sympathetic

Table 6-1 | Anatomical Differences in the Pediatric Airway

Distinguishing Anatomical Feature	Clinical Significance
Large occiput (i.e., back of the head)	May cause neck flexion in the supine position; accordingly: ■ The sniffing position is preferable. ■ In small infants, a towel roll or folded diaper may be placed under the shoulders to raise the chest relative to the head.
Tongue occupies a greater proportion of the oral cavity	May require an oral or nasal airway to alleviate obstruction
More prominent tonsils and adenoids	May contribute to airway obstruction during spontaneous or mask ventilation
Larynx is higher and more anterior, large floppy epiglottis	May make visualization of the vocal cords more difficult during direct laryngoscopy
Smaller airway diameter	Makes airways more susceptible to obstruction by foreign bodies, inflammation and secretions
Functional narrowing of the airway at the cricoid cartilage (complete cartilage ring)	Makes this part of the airway more likely to further narrow from edema and more susceptible to injury from prolonged or repeated intubation

Table 6-2 | Physiologic and Pathologic Factors That Affect Ventilatory Drive

Stimulate Ventilation	Suppress Ventilation
■ Low O_2 ■ High CO_2 ■ Low pH (acidosis) ■ Increased muscle activity (e.g., during exercise) ■ Increased body temperature ■ Increased stress hormones (i.e., catecholamines) ■ Increased intracranial pressure (ICP) ■ Brain lesions affecting the cerebral hemispheres or midbrain ■ Certain drugs (e.g., stimulants, salicylates)	■ Low CO_2 ■ High pH (alkalosis) ■ Brain stem conditions or injury ■ CNS-depressant medications

nervous system, which increases during times of stress). Pathologic factors that can affect ventilatory drive include central nervous system disease or injury and certain drug or toxin exposures (see Table 6-2).

Inspiration occurs as an active process resulting from the contraction of these muscles: the outermost (external) layer of intercostal muscles lifts the ribs and expands the rib cage, while the diaphragm flattens, allowing more room for the lungs to expand. Expiration occurs when these muscles passively relax. Additional respiratory muscles are used for active, or forced, expiration (e.g., the innermost layers of intercostal muscles) or as accessory muscles during times of respiratory distress.

Lung Volumes

The volume of air entering and leaving the lungs (specifically the aveoli) is an important determinant of adequate ventilation. The tidal volume refers to the amount of air taken in with each breath. Normal tidal volume varies based on age, body size and sex. When tidal volume is reduced, respiratory rate must increase to maintain adequate ventilation. As such, children and infants normally have higher respiratory rates than adults because of their naturally smaller tidal volumes. Pathologic conditions affecting the mechanics of the airways and lungs can also affect tidal volume.

Another clinically important measure of lung volume is the functional residual capacity (FRC), the volume of gas present at the end of a normal breath. This volume is important in preventing complete collapse of the alveoli after expiration. Because infants have an overly compliant chest wall that provides little opposition to the natural deflating tendency (or recoil) of their lungs, they have a reduced FRC that approaches the volume required to keep alveoli open. Accordingly, little reserve is available to prevent alveolar collapse in infants who develop lung disease or incur lung damage.

Compliance and Resistance

The volume of air that enters the lungs and, hence, the adequacy of ventilation, are determined by the compliance of the lungs and chest wall, among other factors. Compliance reflects the amount of pressure (or work) required to generate a given volume. Lung compliance is a measure of the lung's ability to stretch and expand (i.e., the distensibility of the lungs). Compliance relates inversely to elasticity. Because the elastic properties of the lungs and chest wall normally work in opposition to inflate the lungs, changes in chest wall compliance may have different effects on ventilation and work of breathing than directionally similar changes in lung compliance.

Airway resistance is another mechanical factor that must be overcome in the process of ventilation. Resistance is dependent on the diameter of the airway; as the airway diameter decreases, resistance increases. As such, airway resistance is normally higher in infants and young children compared with adults because of anatomically smaller airways (Figure 6-3).

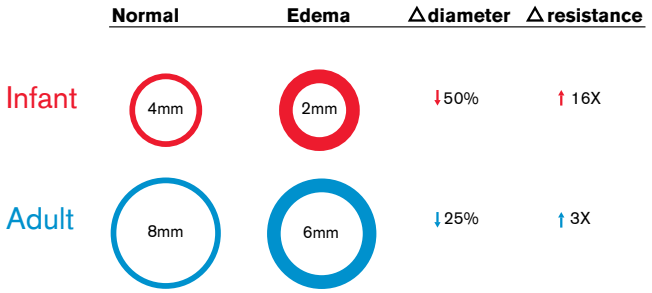


Figure 6-3 | Comparative effects of airway narrowing on airway resistance in infants versus adults

Table 6-3 | Physiologic Differences Among Adults, Children and Infants

Distinguishing Physiologic Feature(s) of Children and Infants	Impact
■ Fewer and smaller alveoli ■ Reduced capacity for collateral ventilation ■ Reduced surfactant production in preterm infants	Prone to atelectasis
■ Smaller diameter of airways	More susceptible to airway obstruction and increased airway resistance
■ Smaller lung volumes relative to metabolic demand ■ Greater percentage of fatigue-prone muscle fibers in the intercostal muscles and diaphragm; reduced glycogen and fat storage in vital respiratory muscles ■ Highly compliant chest wall (in infants)	More susceptible to respiratory muscle fatigue and respiratory failure
■ Higher metabolic rate ■ Reduced FRC due to smaller tidal volumes ■ Highly compliant chest wall (in infants)	More likely to become hypoxemic, especially during periods of apnea (i.e., temporary pause in breathing)

Key Physiologic Differences Among Adults, Children and Infants

Several normal physiologic differences exist among adults, children and infants that make children and infants more susceptible to impaired gas exchange and respiratory compromise. It's important to keep the clinical significance of these physiologic differences in mind as you care for children and infants experiencing respiratory emergencies (Table 6-3).

Pathophysiology of Respiratory Problems in Children and Infants

Respiratory problems in children and infants reflect abnormalities in any of the following components of normal respiration:

- Compliance of the lungs or chest wall or resistance through the airways
- Integrity of the alveolar blood-gas interface
- Neuromuscular control of breathing

These abnormalities may clinically manifest with impairments in oxygenation or carbon dioxide exchange.

Impaired Oxygenation

When the process of oxygenation is hindered in any way, hypoxemia may result. Hypoxemia refers to a decreased amount of O_2 in the blood. Clinically, hypoxemia is typically defined as an O_2 saturation less than 94%. However, a lower threshold for O_2 saturation may be appropriate in neonates and in children with cyanotic heart disease and chronic hypercarbia.

Hypoxemia may be caused by any of the following mechanisms.

- **Low inspired oxygen level:** A low inspired concentration of oxygen may lead to hypoxemia. This is most commonly encountered at high altitude and is thus an uncommon cause of hypoxemia.
- **Hypoventilation:** Hypoventilation refers to insufficient ventilation of the lungs. Hypoventilation results in reduced delivery of O_2 to the alveolus and in low alveolar O_2 content (PaO_2). Causes of hypoventilation include upper or lower airway obstruction, neuromuscular disease, and other conditions or medications that suppress respiratory drive. Hypoventilation does not typically lead to significant hypoxemia in patients with healthy lungs. It is more likely to cause low O_2 levels in the presence of underlying lung disease.
- **Diffusion impairment:** Diffusion impairment affects the exchange of O_2 between the alveoli and the pulmonary capillaries. Diffusion defects may result from obstruction of the smaller airways in the lungs or an increased barrier to diffusion (e.g., due to fibrosis of interstitial lung tissue). Diffusion defects may also occur when alveoli are filled with fluid or collapsed (e.g., due to pneumonia or pulmonary edema).
- **Ventilation-perfusion mismatch:** Ventilation-perfusion mismatch refers to an imbalance between blood flow (i.e., perfusion) and ventilation in a portion of the lung. Oxygenation is specifically affected when perfusion exceeds ventilation, as may occur when areas of the lung cannot be ventilated because of inflammation or alveolar collapse (i.e., atelectasis). The result is a phenomenon called venous admixture or physiological 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.

- **Right-to-left shunt:** Hypoxemia may also occur in the setting of congenital heart defects that allow deoxygenated ("blue") blood from the right side of the heart to pass into the left side of the heart without first going through the lung. This is referred to as a right-to-left shunt.

Impaired Carbon Dioxide Exchange

When normal ventilation is impaired, the normal exchange of O_2 for CO_2 is impeded. This may lead to hypercarbia, an increase in the amount of CO_2 in the blood. Hypercarbia is a hallmark finding of hypoventilation, regardless of cause. Hypercarbia may also occur in conditions that affect lung tissue.

On occasion, children presenting with respiratory distress will have an abnormally low CO_2 level, or hypocarbia. This typically occurs with certain toxins or nonrespiratory conditions that increase respiratory drive and, hence, respiratory rate—for example, metabolic conditions such as diabetic ketoacidosis that increase the acidity of the blood. Hypocarbia may also be seen in the course of conditions affecting the airways or lungs, again because of increased respiratory rate.

Normal CO_2 levels range from 35 to 45 mmHg in arterial blood. Hypercarbia in particular is difficult to detect clinically. The methods for measuring CO_2 levels are described in Table 6-4.

Mechanisms of Respiratory Compromise in Children

Potential mechanisms of respiratory compromise in children include altered mechanics of the lungs, chest wall and airways. Diseases of the lung can also compromise gas exchange across the alveolar-capillary unit. Finally, abnormalities affecting any of the "components" involved in the control and execution of breathing can cause respiratory compromise; these include the respiratory centers in the brain, any inputs affecting the respiratory centers (e.g., changes in pH), and the muscles of respiration.

Reduced Lung Compliance and Increased Airway Resistance

Respiratory compromise may ensue when either lung compliance or airway resistance is abnormal.

Table 6-4 | Methods for Measuring Carbon Dioxide Levels

Method	Description	Comments
Capnometry	Measures exhaled (i.e., end-tidal) CO_2 level	■ Noninvasive ■ Allows for continuous assessment of CO_2 levels (as opposed to intermittent assessments with blood sampling methods) ■ Requires an artificial airway ■ Not as accurate as arterial blood sampling (difference of approximately 2 to 5 mmHg in CO_2 levels) ■ Less accurate in the presence of certain conditions (e.g., decreased cardiac output, pulmonary embolism)
Arterial blood gas	Measures CO_2 level in arterial blood	■ Gold standard ■ Requires operator expertise and skill ■ Relatively contraindicated in patients with bleeding disorders or clotting problems
Capillary blood gas	Measures CO_2 level in a cutaneous blood sample	■ Obtained from a highly vascular (i.e., arterialized) area (e.g., heel, finger, earlobe) ■ High correlation with arterial CO_2 ■ Less invasive and painful than arterial sampling ■ Easier to obtain than arterial sample ■ Limited value for assessing oxygenation
Venous blood gas	Measures CO_2 level in venous blood	■ Does not require specialized skill ■ Does not correlate with arterial CO_2 as well as capillary sample

Reduced lung compliance correlates with a "stiffer" lung. When lung compliance is reduced owing to, for example, pulmonary edema or pneumonia, the capacity of the lung to expand is restricted, thereby requiring more work to generate a normal tidal volume. Conversely, because the chest wall normally provides opposition to the lungs' natural tendency to recoil and collapse inward, an overly compliant chest wall (such as that encountered in infants and in children with neuromuscular disease) may increase the work of breathing. When such work can no longer be maintained, gas exchange is compromised, and respiratory failure may set in.

When airways are obstructed by edema, inflammation or mucus, airway resistance is increased. In children younger than 5 years, peripheral airway resistance is normally 4 times higher than that in adults (see Figure 6-3). Any further decrease in the diameter of the already small airways of infants and children may increase airway resistance exponentially. Obstruction at the level of the upper airway makes it difficult for air to get in, potentially impeding alveolar ventilation. Obstruction of the lower airways 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.

Pulmonary edema resulting from conditions such as heart disease in children and infants affects both lung compliance and airway resistance. Pulmonary edema results from fluid from the pulmonary capillaries moving into the alveoli and interstitium, which increases lung compliance. Fluid in the interstitium may further compress small airways, leading to obstruction of those airways.

Assessing the Pediatric Patient with a Respiratory Emergency

A systematic approach and adherence to the assessment, recognition and care is required when assessing a respiratory emergency.

Prompt assessment, recognition and care of a pediatric patient with impaired oxygenation, ventilation or perfusion may prevent deterioration to respiratory arrest, which if untreated can result in cardiac arrest.

As you perform your systematic assessment, it is important to keep in mind the anatomical and physiological differences among adults, children and infants.

Rapid Assessment

Begin your systematic assessment by performing a rapid assessment. Start with a quick visual survey of the emergency situation before collecting data or providing care. This allows you to make sure that the environment is safe and to formulate an initial impression of the child or infant experiencing an emergency. An initial impression allows you to quickly recognize whether the patient is experiencing a life-threatening or a non-life-threatening condition, including life-threatening bleeding. During the visual survey, it also is important to quickly determine what additional resources you may need in the emergency situation.

Formulate an Initial Impression (PAT)

To form an initial impression of the patient, follow the Pediatric Assessment Triangle (PAT), which uses an A-B-C approach.

- **A**ppearance (TICLS): Assess appearance and responsiveness; observe muscle tone, interactivity (e.g., lethargic, fatigued), movement/gesturing, speaking or crying and demeanor (e.g., calm, anxious or irritable).
- Work of **B**reathing: Note work of breathing; check for patient positioning, audible breath sounds (normal or abnormal—e.g., stridor, wheezing or grunting) and signs of increased work of breathing or respiratory distress (e.g., nasal flaring, using accessory muscles to breathe, intercostal, substernal or suprasternal retractions and/or managing secretions), appears to be breathing too fast or too slow and signs of inadequate or absent respiratory effort.
- **C**irculation: Assess adequacy of circulation by assessing skin color and visible mucous membranes; check for pallor (or gray/dusky color), cyanosis, mottling or flushing and life-threatening bleeding.

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.

ALERT

If you observe that the child or infant is unresponsive during the appearance step of the PAT, check for responsiveness, breathing and pulse and provide immediate care as necessary. If you observe that the child or infant is responsive during the appearance step of the PAT, but you observe potential life-threatening airway, breathing or circulation compromise, provide immediate care as necessary before proceeding to the primary assessment. If the child or infant is responsive and is not experiencing life-threatening airway, breathing or circulation compromise, proceed directly to the primary assessment.

Primary Assessment

After completing a rapid assessment, conduct a primary assessment of the patient. This primary assessment enables you to collect physical and physiologic data to facilitate recognition of the underlying cause(s) of the patient's respiratory emergency. To perform a primary assessment use the ABCDE approach.

Airway

- Assess airway to determine patency. Listen and feel for movement of air by placing your ear close to the patient's nose and mouth. Observe for the rise and fall of the chest and/or abdomen with each breath.
- Determine the following: Is the airway clear and open? Is the airway obstructed but can be kept open with simple manual interventions? Is the airway not maintainable and is the use of CPAP, non-invasive ventilation or an advanced airway required?
- Maintain a patent airway as appropriate based on assessment findings.

Breathing

Assess the child's or infant's breathing to determine adequacy of ventilation and oxygenation:

- Look for increased work of breathing (e.g. nasal flaring, using accessory muscles to breathe, intercostal, substernal or suprasternal retractions) or respiratory distress, abnormal breath sounds (e.g., stridor, grunting, wheezing, gurgling), absent breath sounds, management of secretions.
- Note respiratory rate, depth and rhythm (Table 6-5).
- Auscultate breath sounds (e.g., stridor, grunting, wheezing, crackles).
- Note voice or cry changes (e.g., hoarseness, hot potato voice).
- Measure oxygen saturation with pulse oximetry: Is the oxygen saturation reading normal or abnormal?
- Measure $ETCO_2$: For intubated patients, and when available in non-intubated patients, measure $ETCO_2$. Is the reading normal or abnormal?
- Prepare supplemental O_2 and provide supplemental O_2 as appropriate based on assessment findings.
- If necessary, support breathing by delivering ventilations with a BVM resuscitator.
- Implement noninvasive or invasive ventilation as necessary.
- In the case of tension pneumothorax, perform immediate needle thoracentesis.

Circulation

- Assess the child's or infant's circulation to determine adequate perfusion of tissues.
- Palpate central and peripheral pulses.
- Measure the child's or infant's blood pressure (see Table 6-5).
- Connect the child or infant to a cardiac monitor to assess heart rate and rhythm (see Table 6-5).
- Note skin and mucous membrane color, skin temperature and capillary refill time.
- Prepare to achieve vascular access based on assessment findings.

Table 6-5 | Normal Pediatric Vital Signs

Age Group	Respiratory Rate	Awake Heart Rate	Systolic Blood Pressure*	Diastolic Blood Pressure
Newborns	30 to 60	100 to 120	60 to 85	35 to 55
Infant (1 to 12 mo)	30 to 50	100 to 180	70 to 100	35 to 60
Toddler (1 to 2 yrs)	24 to 40	90 to 140	85 to 105	40 to 65
Preschooler (3 to 5 yrs)	20 to 30	80 to 130	89 to 115	45 to 70
School Age (6 to 12 yrs)	16 to 26	70 to 120	94 to 120	55 to 80
Adolescent (13 to 17 yrs)	12 to 20	60 to 100	110 to 135	60 to 85

- Prepare for fluid or medication therapy, if indicated.
- Prepare for electrical therapy, if indicated.
- Monitor urine output (as appropriate).

Disability

- Assess neurological status to determine adequate brain perfusion.
- Check level of consciousness, pupillary response (PERRL) and blood glucose level.
- Use the following tools: AVPU, GCS (eye opening, motor response, verbal response; low score represents more severe neurological insult) and TICLS.

Exposure

- Assess the body overall, focusing on one area at a time.
- Look for abrasions, burns, bleeding, contusions (bruising), crepitus, deformities, fractures, instability, lacerations, penetrations, petechiae and/or purpura, rashes, tenderness, abnormal skin temperature and color (to assess circulation and perfusion).
- Obtain the patient's weight and body temperature if not already done/available.
- If a head, neck, spinal or pelvic injury is suspected in the patient, consider spinal motion restriction.

ALERT

As you perform the primary assessment, be alert for signs that the patient's condition has worsened or for any change in areas already assessed. Delegate necessary initial interventions to the appropriate team members so that immediate care can be implemented as you continue the primary assessment.

Secondary Assessment

After the primary assessment is completed, if the patient's condition remains stable and resources permit, perform a secondary assessment. This focused and detailed assessment will likely center around the respiratory system and its effects on the patient's vital functions.

The secondary assessment of a child presenting with respiratory compromise will also include a focused patient history and several common laboratory and diagnostic studies (Table 6-6). The findings of these studies may be used to further discern the severity and underlying cause of respiratory compromise and to guide continued management.

ALERT

Throughout the secondary assessment, it is important to keep the concept of assess, recognize and care in mind. Reassess the patient continuously for any changes in condition and assess the patient's response to any clinical interventions.

Focused History

- Record signs and symptoms, allergies, medications (including recreational drug use as applicable), past medical history, last intake/output and events (SAMPLE).

Focused Physical Assessment

- Complete a focused physical assessment of the area of concern as determined by information gathered from the initial impression, primary assessment and focused history.
- Observe for signs and symptoms of pain.

Laboratory and Diagnostic Tests

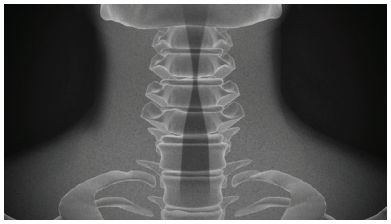
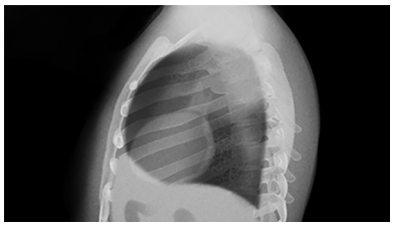
- Perform laboratory and diagnostic tests to determine the presence, type and severity of a respiratory emergency (see Table 6-6 and *Learn More: Peak Expiratory Flow*).
- Determine specific tests and timing based on the patient's situation and status.

Recognizing Respiratory Emergencies

Respiratory compromise manifests along a continuum. **Respiratory distress** represents the earliest stage on the continuum, encompassing a range of progressive signs and symptoms of respiratory compromise.

Respiratory failure occurs when oxygenation and/or ventilation is no longer adequate to meet metabolic demands. This is usually a progression from respiratory distress, but respiratory failure can occur as the initial presentation. While the definition is based on blood levels of oxygen and carbon dioxide, in clinical care initial recognition of respiratory failure is based on clinical signs. Most patients with respiratory failure will require ventilatory assistance in addition to supplemental oxygen. There are two types of respiratory failure—hypoxic respiratory failure ($PaO_2 < 60$) and hypercapnic respiratory failure ($PCO_2 > 50$), but patients can also have a combined form. Hypoxic failure is most often due to V/Q (ventilation/perfusion) mismatch while hypercapnic failure is due to decreased tidal volume or increased dead space.

Table 6-6 | Common Initial Laboratory and Radiographic Studies Used in the Evaluation of Respiratory Problems in Children

Study	Purpose
Radiographs	
Lateral or anterior-posterior neck views 	Evaluate subglottic region The image here shows an example of the radiographic features of croup.
Lateral or anterior-posterior chest views 	Confirm (or exclude) underlying respiratory tract pathology The image here shows an example of the radiographic features of air trapping in asthma.
Laboratory Studies	
Blood gas (arterial, venous, or capillary)	Assess gas exchange Identify primary acid-base abnormalities (e.g., metabolic acidosis)
Complete blood count	Evaluate for evidence of infection (low or high WBC count) or anemia
Electrolytes	Identify electrolyte imbalances
Respiratory viral testing	Confirm or exclude suspected viral respiratory pathogens (e.g., RSV, influenza; sampling and type of testing will vary by pathogen)
Blood and sputum cultures	Identify potential bacterial pathogens

Respiratory arrest, complete cessation of breathing effort, occurs when respiratory failure is not addressed promptly. The body can tolerate respiratory arrest for only a very short time before the heart stops functioning as well, leading to cardiac arrest.

> Respiratory failure is the most common cause of cardiac arrest in children and infants.

Accordingly, prompt recognition and treatment of children and infants who present with respiratory distress is critical to avoid progression to respiratory failure, respiratory arrest and, ultimately, cardiac arrest in these patients.

Primary Assessment Findings

The predominant clinical picture in respiratory distress (e.g., tachypnea, increased work of breathing) reflects the child's or infant's effort to overcome a condition or injury that compromises normal ventilation and/or oxygenation. Children in respiratory distress may be irritable and anxious and may not be able to find a comfortable position. Alternatively, affected children may assume a position of comfort. For example, children with advanced respiratory compromise may assume an upright posture with their hands placed either on their thighs or on the bed ("tripod position").

LEARN MORE

Peak Expiratory Flow

Peak expiratory flow (PEF) is a bedside test used to measure the severity of airway obstruction in patients with asthma or other diseases characterized by lower airway obstruction. It measures the air flow that a patient can generate during forced exhalation after maximal inspiration (i.e., taking a deep breath in). Peak expiratory flow decreases as airway obstruction increases.

- To perform the procedure, the PEF meter indicator is moved to the bottom of the scale by manually moving it or shaking the meter (Figure 6-4). Ideally, the patient should be standing to perform the procedure but can do so sitting upright if unable to stand.
- Next, the patient is instructed to take a deep breath and put his or her lips around the meter's mouthpiece, being careful not to block the aperture with his or her tongue.
- The patient then breathes out as quickly and forcefully as possible. The number at the location of the indicator corresponds with the patient's PEF on that attempt. Typically, the patient is asked to repeat the procedure an additional two times, and the highest number of the three attempts is used for assessment.

The severity of airway obstruction is assessed by how the PEF compares with a normal reference (i.e., predicted) PEF or with the patient's best PEF. Normal reference values for PEF are based on the patient's height and can be obtained from a chart or various online calculators. A PEF <50% of a normal or personal best PEF is indicative of severe obstruction.

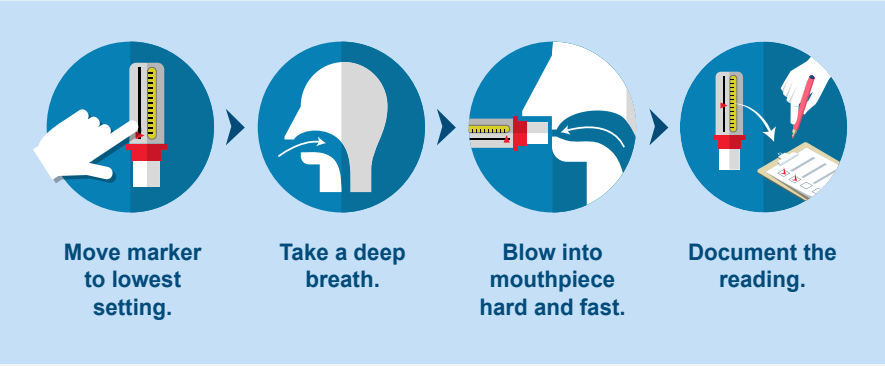


Figure 6-4 | Steps for performing the PEF procedure

Cyanosis may be a sign of respiratory distress, but should resolve with administration of supplemental oxygen.

Respiratory failure ensues when the child or infant is no longer able to maintain that effort. Key signs of respiratory failure include:

- Decreased breath sounds or air movement upon auscultation of the chest.
- Bradycardia.
- Slowed respiratory rate or apnea.

A low O_2 saturation is another potential indicator of severe respiratory failure.

> Recognizing the signs and symptoms of respiratory failure is key in preventing progression to respiratory arrest.

Signs and symptoms of respiratory arrest include:

- Loss of consciousness.
- Absent breath sounds.
- Lack of chest movement.
- Cyanosis or pallor.
- Bradycardia (may initially see tachycardia).
- Hypotension.

Primary assessment findings in respiratory distress and respiratory failure are summarized in Table 6-7.

Certain respiratory signs and symptoms are common across multiple types of respiratory problems, including changes in respiratory rate (most often tachypnea) and increased work of breathing. However, differences in these changes may help to localize the primary underlying *type* of respiratory compromise (Table 6-8).

Important Assessment Findings in Various Respiratory Disorders

Important assessment findings seen in various respiratory disorders include respiratory rate, depth of respiration, increased respiratory effort and presence of certain breath sounds.

Respiratory Rate

Rate of breathing may be increased in patients who have conditions that directly or indirectly affect lung tissue or who have metabolic acidosis. Conversely, rate of breathing may be decreased in patients who have conditions affecting respiratory drive or who are in respiratory failure, regardless of cause. Consider the patient's respiratory rate in the context of a normal respiratory rate, which varies widely in the pediatric population based on age. Normal respiratory rates (breaths/min) based on age are:

- Newborn: 30 to 60
- Infant (1 to 12 months): 30 to 50
- Toddler (1 to 2 years): 24 to 40
- Preschooler (3 to 5 years): 20 to 30
- School Age (6 to 12 years): 16 to 26
- Adolescent: 12 to 20

Depth of Respiration

Also consider the depth of the patient's breathing, as it too may be affected in certain conditions and may hint at an underlying cause. For example, conditions that decrease lung compliance typically cause rapid, shallow breathing, whereas metabolic acidosis typically results in rapid, deep breathing.

Increased Respiratory Effort

Look for signs of increased respiratory effort. First look for signs that the patient is using accessory respiratory muscles. Retractions are especially likely in infants (Figure 6-5), given their relatively compliant chest walls. Nasal flaring may also be seen and is thought to be an attempt at reducing airway resistance at the nostrils, particularly in infants, who typically breathe through the nose. In addition, look for tripod positioning and signs of paradoxical thoracoabdominal movement or "seesaw" breathing. "Seesaw" breathing is characterized by chest contraction during inspiration and chest expansion during expiration; the reverse of normal breathing movements. You may also note unusual abdominal movements. If "seesaw" breathing is accompanied by signs of respiratory distress, it can indicate an impending life-threatening respiratory emergency.

Table 6-7 | Primary Assessment Findings in Respiratory Distress and Respiratory Failure

Primary Assessment Component	Potential Signs and Symptoms	
	Respiratory Distress	Respiratory Failure
Airway	Varying degree of airway obstruction (as evidenced by stridor, drooling, wheezing)	Unmaintainable airway/airway obstruction leading to inability to breathe and potential respiratory arrest.
Breathing	Increased respiratory rate, accessory muscle use, nasal flaring, abnormal breath sounds and grunting	Slowed respiratory rate (may initially be very rapid), poor or absent air movement, low O_2 saturation or low PaO_2, high $ETCO_2$ or high PCO_2
Circulation	Tachycardia, pallor, cyanosis (should resolve with supplemental oxygen)	Bradycardia (may initially see tachycardia), pallor, cyanosis (central; does not resolve with supplemental oxygen)
Disability	Irritability, anxiety, assuming position of comfort (e.g., tripod positioning)	Altered level of consciousness (lethargy, somnolence, or unconsciousness
Exposure	Cyanosis (should resolve with supplemental oxygen)	Pallor, cyanosis (central; does not resolve with supplemental oxygen)

Table 6-8 | General and Local Signs of Respiratory Problems in Pediatric Patients

Sign	Upper Airway Obstruction	Lower Airway Obstruction	Lung Tissue Disease	Neurological and Metabolic Disorders of Ventilation
Respiratory rate	Increased (decreased late)			Increased, decreased or irregular
Work of breathing	Increased			Variable
Breathing noises	Stridor (initially heard on inspiration, but with progression or a fixed obstruction, can be heard on inspiration and expiration)	Wheezing, grunting, rhonchi, crackles	Grunting, decreased breath sounds, localized crackles, generalized crackles and wheezes	Normal
Air movement	Decreased			Variable

Presence of Certain Breath Sounds

Listen to the breath sounds the patient is making, as they may help identify the site of respiratory disease or injury.

- Abnormal breath sounds are usually heard first on inspiration when the problem is above the thoracic inlet (upper airway) and on expiration when occurring below the thoracic inlet (lower airway). But with greater compromise or fixed obstructions, abnormal breath sounds may be heard during both inspiration and expiration.
- High-pitched, noisy breathing, or stridor, typically accompanies airway obstruction above the thoracic inlet, whereas expiratory wheezing generally indicates obstruction below the thoracic inlet.
- Children and infants with conditions affecting the lower airways or lung tissue may present with grunting, a sound produced by forced exhalation against a closed glottis. Grunting is thought to generate positive end-expiratory pressure (PEEP). Grunting is often a sign of impending respiratory failure as it signifies a last-ditch attempt to keep the terminal airways open.

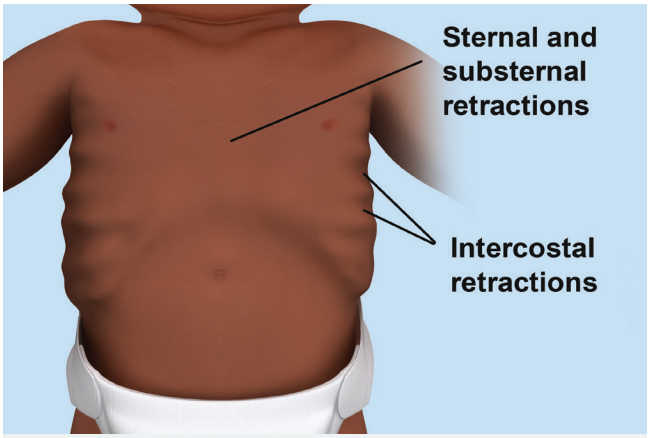


Figure 6-5 | Retractions

Secondary Assessment Findings

Secondary assessment findings in respiratory failure are notable for abnormalities in blood gas analysis, namely PaO_2 <60 mmHg while breathing room air and PCO_2 >50 mgHg with acidosis. However, certain clinical parameters such as the patient's general appearance and respiratory effort and the potential for impending exhaustion are considered more important indicators than blood gas values.

Causes Overview

Both respiratory distress and respiratory failure may arise from primary disease of the upper or lower airway or lung tissue, or from conditions affecting other organ systems (Table 6-9). Failure to recognize nonrespiratory causes of respiratory distress or failure may lead to inappropriate care. The primary cause of respiratory distress or respiratory failure may be clear based on the patient's medical history.

Upper Airway Obstruction

Upper airway obstruction may result from disease or injury involving the nasal or oral cavities, pharynx, larynx or upper trachea. Obstruction may arise from intrinsic airway pathology, such as inflammation, from foreign-body aspiration or from pathology outside the airway, such as a retropharyngeal abscess or tumor. Significant signs of upper airway obstruction include stridor, drooling and increased work of breathing. Upper airway obstruction can be partial or complete, depending on the cause and/or severity of the disease process. Also, partial obstruction can progress to complete without prompt recognition and care.

Table 6-9 | Anatomical Sites of Conditions Causing Respiratory Distress or Respiratory Failure

Anatomical Site or System	Conditions That Cause Respiratory Distress or Failure	Mechanisms of Respiratory Distress or Failure
Upper airway	■ Laryngotracheitis (i.e., croup) ■ Epiglottitis (i.e., inflammation of the epiglottis) ■ Foreign body aspiration ■ Anaphylaxis ■ Subglottic stenosis (i.e., narrowing below the larynx) ■ Retropharyngeal abscess (i.e., infection of lymph nodes in the space behind the pharynx)	Airway obstruction
Lower airway	■ Asthma ■ Bronchiolitis (i.e., inflammation of the bronchioles) ■ Foreign body aspiration ■ Anaphylaxis	Airway obstruction
Lung tissue	■ Pneumonia ■ Pulmonary edema ■ Acute respiratory distress syndrome (ARDS) ■ Respiratory distress syndrome	Decreased lung compliance
Neurological/metabolic system	■ Increased ICP ■ Central nervous system (CNS) trauma ■ CNS infection (e.g., encephalitis) ■ CNS depressants ■ Diabetic ketoacidosis ■ Toxins (e.g., salicylates) ■ Neuromuscular diseases	■ Stimulation or suppression of ventilatory drive ■ Decreased chest wall compliance and inspiratory tone due to respiratory muscle weakness, leading to airway closure and atelectasis; impaired cough reflex and airway clearance

Causes

Potential causes of upper airway obstruction in children and infants are shown in Box 6-1.

Croup

Croup is the most common cause of a partial upper airway obstruction in children. Croup is a general term applied to a symptom complex characterized by hoarseness, a resonant cough described as "barking" or "brassy" (croupy), varying degrees of inspiratory stridor and varying degrees of respiratory distress resulting from swelling or obstruction in the region of the larynx and subglottic airway. Croup is typically viral in origin; parainfluenza is the most common causative agent.

Croup may be categorized by severity:

- Mild croup: Occasional barking cough; no stridor at rest
- Moderate croup: Frequent barking cough; audible stridor and retractions at rest

Box 6-1 | Causes of Upper Airway Obstruction in Children

- **Choanal atresia** or **choanal stenosis**
- Enlarged tonsils or adenoids
- Retropharyngeal or peritonsillar abscess
- **Laryngomalacia**
- Epiglottitis
- **Vocal cord paralysis**
- Croup
- Allergic reaction/anaphylaxis
- **Tracheitis**
- **Subglottic stenosis**
- **Vascular ring**
- **Mediastinal** mass
- Foreign body aspiration
- Burns (inhalational thermal injuries)

- Severe croup: Frequent barking cough; marked stridor and visible retractions at rest; agitation/distress

Epiglottitis

Croup must be distinguished from epiglottitis, a less common but more severe cause of airway obstruction. Epiglottitis is a life-threatening infection involving the epiglottis and other supraglottic structures. The patient with epiglottitis has a toxic appearance and typically presents with a precipitous onset of high fever, drooling, sore throat, a muffled voice and rapid progression of airway obstruction. Epiglottitis has a classic presentation with four Ds:

- Drooling
- Dyspnea (trouble breathing)
- Dysphagia (trouble swallowing)
- Dysphonia (trouble speaking)

The incidence of epiglottitis has decreased significantly since the introduction of the vaccine for *Haemophilus influenzae* type B, which was once the primary causative agent. However, many other potential causative organisms exist. Ideally, epiglottitis is definitively diagnosed in the operating room when a physician with advanced airway experience examines the epiglottis under direct laryngoscopy. If epiglottitis is confirmed, immediate intubation is nearly always the rule.

Allergic Reaction/Anaphylaxis

Allergic reaction/anaphylaxis is a common cause of upper airway obstruction in children and infants. Allergic reactions occur when the child's immune system overreacts to something otherwise harmless in the environment. Common allergies in children include food allergies, seasonal allergies and allergies to dust mites or insect bites. Often, allergic reactions are mild or moderate and cause symptoms such as:

- Runny nose.
- Watery eyes.
- Skin reactions.
- Itching.
- Nausea and vomiting.
- Minor swelling.

However, children and infants can also have severe allergic reactions. The most extreme form of an allergic reaction is anaphylaxis. Anaphylaxis is a life-threatening condition that requires immediate care. Recognizing the signs and symptoms of anaphylaxis is key. These include:

- Cutaneous and mucosal findings (e.g., swelling of the lips, tongue or uvula [angioedema], hives, flushing, itching).
- Respiratory compromise (stridor, hoarseness, wheezing, dyspnea, increased work of breathing).
- Cardiovascular compromise (tachycardia, hypotension).
- Signs of end-organ tissue dysfunction.
- Gastrointestinal symptoms (e.g., nausea, vomiting, abdominal pain).

Practice Note

Diagnostic criteria for anaphylactic shock:

- Cutaneous and mucosal findings and one of the following:
  - Respiratory compromise
  - Hypotension and signs of end-organ dysfunction

OR

- Two or more of the following that occur rapidly post-exposure to an allergen:
  - Cutaneous and mucosal findings
  - Respiratory compromise
  - Hypotension and signs of end-organ dysfunction
  - Gastrointestinal symptoms

OR

- Hypotension post-exposure to an allergen

See Chapter 7, Shock.

Foreign-Body Aspiration

Foreign body aspiration is a common cause of partial or complete upper or lower airway obstruction in children and infants. It is especially prevalent in young children and infants due to aspiration of items such as food, small toys or coins. Because the onset of symptoms (airway obstruction and respiratory compromise) is often very sudden, quick recognition and care is essential.

Other Conditions

Conditions localized to the nose, oropharynx or trachea may also cause upper airway obstruction in children. Obstruction of the nostrils (i.e., choanal atresia or stenosis) may be especially problematic in infants, who typically breathe through the nose.

Lower Airway Obstruction

Lower airway obstruction may result from pathologic processes involving the intrathoracic airways, that is, the distal trachea, bronchi and bronchioles. 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. Lower airway obstruction is the

most common cause of respiratory distress in children. Lower airway obstruction can be partial or complete, depending on the cause and/or severity of the disease process. Also, partial obstruction can progress to complete without prompt recognition and care.

Causes

Common causes of lower airway obstruction in children include:

- Asthma.
- Bronchiolitis.
- Croup (when lower trachea or bronchi are involved).
- Foreign-body aspiration.
- Lower airway lesion (e.g., **papilloma**, **hemangioma**).
- Cystic fibrosis.
- Pulmonary edema ("cardiac asthma"; due to extrinsic compression on small airways).

Asthma

Asthma is a respiratory condition characterized by inflammation and spasm of the lower airways. Lower airway spasm (bronchospasm) may be triggered by such factors as allergens or infection. Airway inflammation and bronchospasm lead to air trapping in the lungs, which impedes exhalation and increases the work required during inspiration.

The severity of asthma is determined based on symptoms, signs and functional assessment findings (Table 6-10).

Bronchiolitis

Bronchiolitis is a lower respiratory tract infection that primarily affects young children and infants. Affected infants are typically between the ages of 2 and 4 months and significant lower airway obstruction is rare in children older than 2 years.

It typically presents in the middle of winter or early spring after a prodrome of upper respiratory tract infection and fever. It is characterized by inflammation, spasm and mucous plugging of the lower airways (namely, bronchioles). It is most often caused by respiratory syncytial virus (RSV) but may be caused by other viruses as well. Children and infants with chronic lung or heart disease or who are immunocompromised are especially at risk for developing severe bronchiolitis.

Foreign-Body Aspiration

Children and infants are also particularly susceptible to lower airway obstruction as a result of foreign-body aspiration.

Cystic Fibrosis

Cystic fibrosis, a genetic disease that often presents in childhood, is another potential cause of lower airway obstruction. It is characterized by production of abnormally thick, sticky mucus that leads to airway obstruction, recurrent lung infections, and end-stage lung disease.

Lung Tissue Disease

Lung tissue disease collectively refers to diseases affecting the alveoli and the interstitium of the lung. Such diseases typically affect gas exchange via their involvement of the alveolar-pulmonary capillary interface. They also reduce lung compliance.

Causes

Common conditions among children and infants in this category include:

- Infectious and aspiration pneumonia.
- Pulmonary edema.
- Acute respiratory distress syndrome (ARDS).
- Respiratory distress syndrome due to surfactant deficiency in preterm infants.

Interstitial lung diseases are another category of lung tissue disease characterized by inflammation and fibrosis of the alveolar walls. They may be caused by environmental or drug exposures or various systemic diseases (e.g., connective tissue disorders) and are on the whole rare in children.

Pneumonia

Pneumonia represents inflammation of the lung parenchyma caused by infection, aspiration or toxic agents. Infectious pneumonia may be caused by bacteria, viruses, mycoplasma or fungus. Pneumonia can be confined to one or more lobes of the lungs (lobar pneumonia) or involve the alveolar walls (interstitial pneumonia). Aspiration pneumonia results from contamination of the lower respiratory tract by foreign, nongaseous material such as saliva, gastric contents, aspirated food or chemicals (e.g., hydrocarbons). Resulting injury to the lung tissue leads to bleeding, necrosis, surfactant impairment and pulmonary edema, all of which may reduce lung compliance or cause ventilation-perfusion mismatch.

Pulmonary Edema

Pulmonary edema may be noncardiogenic or cardiogenic (i.e., arising from heart disease or dysfunction). In cardiogenic edema, excessive blood flow to the lungs or increased pressure in the pulmonary veins leads to transudate of fluid into the lung interstitium and the alveoli.

Table 6-10 | Severity Classification for Asthma Exacerbations

Assessment Component	Asthma Severity Categories			
	Mild	**Moderate**	**Severe**	**Respiratory Arrest Imminent**
Symptoms	■ Breathlessness while walking ■ Can lie down ■ Talks in sentences ■ May be agitated	■ Breathlessness while at rest (softer, shorter cry and difficulty feeding in infants) ■ Prefers sitting ■ Talks in phrases ■ Usually agitated	■ Breathlessness while at rest ■ Sits upright ■ Talks in words ■ Usually agitated	■ Inability to breathe ■ Drowsiness, confusion or loss of consciousness
Signs	■ Respiratory rate increased ■ Moderate wheezing ■ Pulse normal for age (may be elevated if patient receiving bronchodilators)	■ Respiratory rate increased ■ May have decreased air movement ■ Use of accessory muscles and suprasternal retractions commonly present ■ Loud wheezing ■ Pulse elevated for age ■ Pulsus parodoxus may be present (10 to 25 mmHg)	■ Respiratory rate often >30/minute, or increased for age ■ Severely decreased air movement ■ Use of accessory muscles and suprasternal retractions usually present ■ Loud wheezing throughout inhalation and exhalation (may not be heard due to lack of air movement) ■ Significant tachycardia for age ■ Pulsus paradoxus often present (20 to 40 mmHg)	■ Parodoxical thoracoabdominal movement ("seesaw breathing") ■ Absence of air movement ■ Absence of wheeze ■ Bradycardia ■ Absence of pulsus parodoxus (suggests respiratory muscle fatigue)
Functional Assessment	■ Peak expiratory flow (percent predicted or personal best) ≥70% ■ Arterial blood gas findings on room air (test not usually necessary): ○ PaO_2 normal ○ $PaCO_2$ <42 mmHg ○ SaO_2 >95%	■ Peak expiratory flow (percent predicted or personal best) ~40% to 69% or response lasts <2 hours ■ Arterial blood gas findings on room air (test not usually necessary): ○ PaO_2 ≥60 mmHg ○ $PaCO_2$ <42 mmHg ○ SaO_2 90 to 95%	■ Peak expiratory flow (percent predicted or personal best) <40% ■ ABG findings on room air: ○ PaO_2 <60 mmHg ○ $PaCO_2$ ≥42 mmHg ○ SaO_2 <90%	■ Peak expiratory flow (percent predicted or personal best) <25% ■ Arterial blood gas findings on room air: ○ PaO_2 <60 mmHg ○ $PaCO_2$ ≥42 mmHg ○ SaO_2 <90%

Note: The presence of several parameters, but not necessarily all, indicates the general severity classification of the exacerbation.

Noncardiogenic edema is a secondary effect of various lung tissue diseases like aspiration pneumonia and ARDS. It results from diffuse damage of lung capillaries and alveoli, leading to exudate of fluid into the alveoli and interstitial spaces. Regardless of origin, pulmonary edema decreases lung compliance and may also cause obstruction of the smaller airways of the lungs.

Tension Pneumothorax

Tension pneumothorax is a serious extrapulmonary condition that is important to be aware of because of its potentially life-threatening presentation. See Chapter 7, Shock, for assessment, recognition and care of tension pneumothorax.

Neurological and Metabolic Disorders of Ventilation

Children and infants with neurological or metabolic conditions may present with respiratory signs and symptoms, generally abnormalities of breathing rate and pattern. These findings often do not signify actual pulmonary disease or injury. Nevertheless, children with breathing abnormalities due to nonrespiratory conditions may require treatment similar to those with breathing abnormalities due to intrinsic pulmonary disease or injury.

Causes

Neurological causes of disordered ventilation include conditions or agents affecting the central nervous system (CNS)/brain and neuromuscular diseases. Metabolic conditions may also affect ventilation.

CNS/Brain Conditions

In children and infants with brain injury or structural abnormalities of the brain, increased ICP may stimulate respiratory centers, causing increased rate (i.e., tachypnea) and depth (i.e., hyperpnea) of respirations. In such cases, hyperventilation is a compensatory mechanism that reduces the blood content of CO_2, thereby constricting the cerebral arteries and reducing ICP.

In certain CNS conditions, abnormal cardiovascular parameters may be seen. For example, bradycardia and hypertension may accompany abnormal breathing (e.g., irregular, decreased respirations) in cases of increased ICP. This cluster of signs is known as the Cushing's triad.

Disease or injury affecting the brain stem may produce abnormal breathing patterns such as Cheyne-Stokes breathing, which alternates periods of rapid breathing with slow breathing. Drugs that depress the respiratory drive (e.g., opioids) may cause apnea, as may hypoxic injury, trauma or infection affecting the brain. On occasion, neurologic conditions may have effects on pulmonary function via neurogenic pulmonary edema or hypoventilation.

Neuromuscular Diseases

A variety of neuromuscular conditions may cause weakness of the muscles of respiration. These conditions may affect the nerves supplying the muscles or the muscles themselves and may be acute (e.g., spinal cord trauma, Guillain-Barré syndrome, botulism) or chronic (e.g., Duchenne muscular dystrophy, myotonic dystrophy).

Respiratory muscle weakness in these conditions may lead to an ineffective cough reflex, shallow breathing with insufficient effort, impaired airway clearance, an increased risk for aspiration, and development of **atelectasis**.

Some patients with neuromuscular diseases may also have an abnormal curvature of the spine called **kyphoscoliosis**, or chest wall deformity, which may further restrict lung function. The extremities of patients with neuromuscular diseases may also appear thin or atrophied.

While certain chronic neuromuscular conditions may progress more slowly, superimposition of an acute respiratory infection or an aspiration event can be problematic in any patient with neuromuscular disease.

Metabolic Conditions

Metabolic conditions that may affect ventilation include diabetic ketoacidosis and organic acidemias. These conditions typically cause tachypnea and hyperpnea, which are compensatory responses to metabolic acidosis.

Similarly, toxins that cause metabolic acidosis, such as methanol or ethylene glycol, may cause hyperventilation. **Hyperammonemia** related to inherited metabolic disorders or liver disease may cause hyperventilation because ammonia stimulates respiratory centers.

Key Primary and Secondary Assessment Findings

Notable primary and secondary assessment findings for each category of respiratory problem are summarized in Table 6-11.

Caring for the Pediatric Patient with a Respiratory Emergency

Immediate Care for Respiratory Emergencies

Respiratory Arrest

It is important to provide rescue breathing for any child or infant in respiratory arrest to prevent progression to cardiac arrest. Note: While caring for the patient in respiratory arrest, it is important to determine the need for advanced airway placement as appropriate and to identify and treat underlying causes.

If the child is not breathing normally (or only gasping) but has a pulse >60 bpm:

- Deliver 1 ventilation every 3 to 5 seconds; each ventilation should last about 1 second and make the chest begin to rise.
- Continue ventilations. Check the pulse and breathing about every 2 minutes.
- If during pulse checks the pulse decreases to ≤60 with signs of poor perfusion, add chest compressions.
- If you find no pulse, begin CPR.

Practice Note

Signs of inadequate perfusion in a child include cool, moist skin; pallor, mottling or cyanosis; a weak or thready pulse; decreased capillary refill; and hypotension.

If the child is not breathing normally (or only gasping) but has a pulse <60 bpm with signs of poor perfusion:

- Start CPR.
- Continue ventilations and chest compressions. Check the pulse and breathing about every 2 minutes.
- If during pulse checks the pulse increases to greater than 60 bpm, stop chest compressions but continue delivering 1 ventilation every 3 to 5 seconds if the child remains in respiratory arrest.
- If you find no pulse, continue CPR.

Table 6-11 | Key Assessment Findings by Respiratory Problem Type

Assessment Component	Upper Airway Obstruction (Partial)	Lower Airway Obstruction	Lung Tissue Disease	Neurological and Metabolic Disorders of Ventilation
Airway	■ Stridor (inspiratory; may be expiratory) ■ Trouble swallowing, drooling/difficulty managing secretions ■ Voice (or cry) changes (e.g., hoarseness/hot potato voice) ■ Unmaintainable airway (late) ■ Sudden-onset signs of airway obstruction and respiratory compromise (foreign-body aspiration)	Unmaintainable airway (late)	Unmaintainable airway (late)	■ Unmaintainable airway due to altered mental status ■ Impaired swallowing, drooling, ineffective airway clearance (neuromuscular diseases)

Assessment Component	Upper Airway Obstruction (Partial)	Lower Airway Obstruction	Lung Tissue Disease	Neurological and Metabolic Disorders of Ventilation
Breathing				
Respiratory rate	■ Tachypnea ■ Bradypnea or apnea (late)	■ Tachypnea ■ Bradypnea or apnea (late)	■ Tachypnea ■ Bradypnea or apnea (late)	■ Tachypnea, bradypnea, or apnea ■ Irregular breathing pattern (e.g., Cheyne-Stokes breathing)
Work of breathing	■ Increased ■ Retractions ■ Nasal flaring	■ Increased ■ Retractions ■ Nasal flaring	■ Increased ■ Retractions ■ Nasal flaring	Normal, increased or irregular
Air movement	Decreased	■ Decreased ■ Prolonged exhalation	Decreased	Variable
Breathing sounds	Stridor (inspiratory; may be expiratory)	■ Wheezing ■ Grunting ■ Rhonchi (bronchiolitis) ■ Crackles	■ Grunting ■ Decreased breath sounds (pneumonia) ■ Localized crackles on auscultation (pneumonia) ■ Generalized crackles and wheezes on auscultation (pulmonary edema)	Normal
Other	"Barking" or "brassy" cough (croup)	■ Unable to talk in full sentences ■ Wet, "junky" cough (bronchiolitis)	■ Shallow respirations ■ Cough	■ Ineffective cough (neuromuscular diseases) ■ Cushing's triad (abnormal breathing, hypertension and bradycardia; associated with increased ICP)
Circulation	■ Tachycardia ■ Pallor, cyanosis (late)	■ Tachycardia (bradycardia with hypoxia and respiratory failure) ■ Pulsus paradoxus ■ Pallor, cyanosis (late)	■ Tachycardia ■ Pallor, cyanosis (late)	■ Tachycardia ■ Bradycardia ■ Hypertension ■ Cyanosis (apnea)

Assessment Component	Upper Airway Obstruction (Partial)	Lower Airway Obstruction	Lung Tissue Disease	Neurological and Metabolic Disorders of Ventilation
Disability	■ Restless, anxious, irritable, unable to get comfortable ■ Assuming a position of comfort (e.g., tripod positioning) ■ Agitation, somnolence, or unconsciousness (late)	■ Restless, anxious ■ Reluctance to lie flat ■ Agitation, somnolence, or unconsciousness (late)	■ Restless, anxious ■ Agitation, somnolence, or unconsciousness (late)	■ Altered mental status (CNS conditions, toxins, metabolic conditions) ■ Pupillary changes (CNS conditions, toxins) ■ Global muscle weakness, hypotonia in infants (neuromuscular diseases) ■ Posturing (CNS conditions)
Exposure	Skin reactions (rashes), increased or decreased temperature, toxic appearance, swelling (anaphylaxis, infection or abscess)	Increased or decreased temperature	Increased or decreased temperature	Signs of trauma, bleeding, skin reactions (rashes), needle marks (injection), increased or decreased temperature, chest wall deformity, kyphoscoliosis, thin or atrophied extremities, contractures (neuromuscular diseases)
Key Secondary Assessment Findings	■ Hypoxemia in severe obstruction (croup) ■ Radiograph may show steeple sign (croup) or "thumb sign" (epiglottitis)	■ Hypoxemia ■ Chest radiograph may show hyperinflation or air trapping (both sides in asthma/bronchiolitis; one side foreign-body aspiration) ■ Chest radiograph may show object (foreign-body aspiration) ■ Rapid RSV testing positive (bronchiolitis) ■ Rapid flu testing, respiratory viral studies positive	■ Hypoxemia ■ Chest radiography may show airspace opacity, lobar consolidation, or interstitial opacities	Acute or chronic metabolic alkalosis or acidosis

Practice Note

If the first ventilation does not cause the chest to rise, re-tilt the head to ensure that the airway is properly opened and make sure the patient's nose and mouth are properly sealed before delivering a second ventilation. If the second ventilation does not cause the chest to rise, an object may be blocking the patient's airway. At this point, perform CPR with modifications. After each set of compressions and before delivering ventilations, open the mouth, look for an object in the patient's mouth, and if you see it, remove it using a finger sweep (do not perform a blind finger sweep). Attempt 2 ventilations. Never try more than 2 ventilations during 1 cycle of CPR, even if the chest does not rise. Continue performing CPR cycles, checking for an object before each set of ventilations.

In addition, consider using basic and advanced airway techniques based on level of training and expertise to clear the airway.

See *Respiratory Arrest* Treatment Guideline for a summary of care.

Respiratory Distress or Respiratory Failure

Regardless of underlying cause, it is important to take immediate action for any child who presents with apparent respiratory compromise. Ensuring an open airway takes priority.

When the patient is unable to maintain an open airway owing to anatomical or functional obstruction, take immediate measures to open the airway.

Possible interventions, from least to most invasive, include:

- Positioning the patient to alleviate any anatomical obstruction (or allow the patient to assume a position of comfort).
- Applying manual maneuvers to open the airway (head-tilt/chin-lift technique or modified jaw-thrust maneuver).
- Clearing the airway by suctioning or removing any visibile foreign bodies (do *not* do a blind finger sweep).
- Inserting a basic airway (oral and nasopharyngeal airways).
- Inserting an advanced airway such as an endotracheal tube or laryngeal mask airway.

In addition, it is important to consult with an advanced airway specialist with pediatric experience when a difficult airway is anticipated.

Allow the child who is oxygenating adequately, with no signs of current or impending respiratory failure, to assume a position of comfort. Measure pulse oximetry to determine O_2 saturation in all patients and $ETCO_2$ in intubated patients, and if available, in noninutbated patients.

With few exceptions provide humidified supplemental O_2 to the child who is adequately ventilating on their own to keep oxygen saturation above 94%. Depending on the degree of desaturation, provide supplemental O_2 via a nasal cannula, simple or non-rebreather face mask or, for the child who is particularly anxious, "blow-by" through a mask (Figure 6-6). Non-rebreather masks provide the highest concentration of O_2. Nasal cannula is typically the preferred method of O_2 delivery in children younger than 5 years who only need low concentration to maintain O_2 saturation >94% (Figure 6-7).

Patients who cannot oxygenate or ventilate adequately (or have insufficient respiratory effort) despite an open airway and administration of supplemental oxygen may require administration of inhaled medications, such as albuterol or racemic epinephrine, and/or assisted ventilation, which is initially provided via bag-valve-mask ventilation. Noninvasive positive airway pressure or, in intubated patients, invasive (i.e., mechanical) ventilation may be necessary. Be prepared to establish an advanced airway in case of progressive obstruction, respiratory failure or respiratory arrest. Perform immediate needle thoracentesis for suspected or known tension pneumothorax.

Beyond the initial steps of ensuring an open airway providing supplemental O_2, administering medications or assisting ventilation as needed, other measures include:

- Connecting the child to a cardiac monitor to assess heart rate and rhythm and blood pressure.
- Establishing vascular access:
  - To avoid agitating the patient, you may opt to defer vascular access in spontaneously ventilating patients who do not require IV therapy or who are not exhibiting signs of deterioration.

Figure 6-6 | Providing supplemental oxygen via "blow-by" through a mask

Figure 6-7 | Providing supplemental oxygen via nasal cannula

- Assessing the patient's general appearance and level of consciousness.
- Monitoring for signs of worsening respiratory function, including increasing work of breathing, increasing/decreasing respiratory rate, hypoxemia, change in general appearance, change in mental status and change in muscle tone.

In addition, it is important to identify the underlying type of respiratory problem and provide care as appropriate.

See *Respiratory Distress or Failure* Treatment Guideline for a summary of care.

Care for Respiratory Emergencies Due to Partial Upper Airway Obstruction

Keep the child or infant with evidence of acute partial upper airway obstruction who is spontaneously ventilating and adequately oxygenated as calm as possible to minimize turbulent air flow, which may worsen obstruction. In particular, do not attempt to visualize the posterior pharynx in children with epiglottitis; doing so can precipitate complete airway obstruction.

Only establish vascular access if absolutely required. If clinically indicated, delay establishing IV access until a provider experienced in pediatric airway management evaluates the patient.

Deliver supplemental O_2 in the least invasive and least threatening manner possible to keep oxygen saturation above 94%.

Depending on the underlying cause, suctioning may be appropriate and, along with repositioning, may be sufficient to resolve airway obstruction. Provide ventilatory support as needed. Be prepared for the possibility of progressive airway obstruction, respiratory failure or respiratory arrest and have on hand any equipment necessary to establish an advanced airway, including ETTs that are smaller in diameter than normally recommended for the patient's age and size. Endotracheal intubation is a high-risk procedure in children with upper airway obstruction. When a difficult airway is anticipated, immediately consult a specialist with advanced airway experience (e.g., anesthesiologist, **otolaryngologist**).

In some cases, noninvasive ventilatory methods avoid the need for an artificial airway.

Anaphylaxis

Airway edema in anaphylaxis is managed with epinephrine, but in IM form. Albuterol, methylprednisolone and diphenhydramine are given as additional therapies for airway obstruction due to anaphylaxis.

Foreign-Body Aspiration

Keep the patient calm and monitor for worsening airway obstruction. Arrange for timely removal (using Magill forceps if foreign body is visible above the vocal cords or via rigid bronchoscopy; do *not* do a blind finger sweep). If the patient is oxygenating and ventilating adequately and obstruction is partial, arrange for transport to the operating room for removal as appropriate. If there is progression to complete airway obstruction, perform care to clear the obstruction. (See Chapter 2, Basic Life Support Review, for skills sheets on Obstructed Airway Care for Adults and Children and Obstructed Airway Care for Infants.)

Abscess/Infection

Administer antibiotics for retropharyngeal abscess or suspected bacterial infection of the oropharynx or neck. Arrange for imaging to confirm diagnosis and consider surgical consultation for drainage, if appropriate.

Croup

Patients with partial upper airway obstruction due to croup may benefit from humidified supplemental oxygen as needed and administration of the following as clinically indicated.

Nebulized racemic epinephrine, which constricts the blood vessels of the airway, reducing inflammation and edema is administered as needed for moderate and severe croup.

Corticosteroids reduce the likelihood of hospitalization and progression to respiratory failure in croup and are thus an essential component of croup management.

Dexamethasone is typically used for this purpose and is considered or administered for all croup severities. Consider heliox in cases of moderate-to-severe croup as long as long as SpO_2 can be maintained (see *Learn More: Heliox*).

In cases of severe croup with no improvement after administration of oxygen and medications, consider noninvasive or invasive ventilation. If intubating, use a smaller-diameter ETT than normally recommended for age/size. In addition, consider alternative diagnoses.

Epiglottitis

Immediately consult an advanced airway specialist with pediatric airway experience for definitive diagnosis (ideally in operating room) and management. In addition, give nothing by mouth and assemble equipment to manage progressive airway obstruction. It is important to minimize stimulation by keeping the patient calm and avoiding all potentially anxiety-provoking procedures, *including intraoral examination*. Allow the patient to assume a position of comfort. Finally, monitor the patient for progressive airway obstruction or respiratory arrest.

See *Partial Upper Airway Obstruction* Treatment Guideline for a summary of care.

Care for Respiratory Emergency Due to Lower Airway Obstruction

Treatment for a respiratory emergency due to lower airway obstruction will vary based on what is causing the issue. Review care for each of the following conditions.

Asthma (Bronchospasm)

Care for asthma is based on severity of the exacerbation (see Table 6-10). In addition to benefiting from basic airway management and supplemental oxygen to keep oxygen saturation >94% and support of ventilations as needed, children with asthma may benefit from bronchodilator medications. A short-acting nebulized beta-agonist, usually albuterol, is used as first-line bronchodilator therapy. Three doses are administered, as needed, every 20 minutes.

A nebulized anticholinergic medication, such as ipratroprium bromide, may be considered in combination with albuterol (up to three doses). The albuterol and ipratropium bromide combination is always used when the patient is experiencing a severe asthma exacerbation or when respiratory arrest is imminent. However, some studies have shown the benefit of adding ipratropium bromide to the initial three doses of albuterol inhalation treatments for any acute exacerbation of asthma.

Corticosteroids are recommended to reduce airway inflammation in children with asthma.

For asthma that does not improve with initial treatment or worsens, albuterol is repeated or may be administered continuously and adjunctive therapies (e.g., magnesium sulfate, IV/IO, epinephrine, SC or terbutaline, SC) may be considered. Fluid resuscitation may be considered if the patient is hypovolemic.

When respiratory arrest is imminent, support ventilation. Choices include noninvasive or invasive ventilation. For noninvasive ventilation, CPAP may be used to help stent the airway and facilitate exhalation and BiPAP may be used to provide the same benefits as CPAP and assist with ventilation.

If intubation and mechanical ventilation is needed, intubate with a cuffed endotracheal tube, if possible, in anticipation of the potential need for higher ventilating pressures. Ketamine is the preferred agent for sedation in this setting. Minimize ventilating pressures to minimize risk for further air trapping and barotrauma. Optimize PEEP to assist with stenting alveoli to facilitate exhalation. Prolonged expiratory times should be used.

In addition, when respiratory arrest is imminent, albuterol and ipratropium bromide should be administered, and an IV corticosteroid should be administered within 1 hour of presentation. Adjunctive therapies and parenteral bronchodilators should also be considered.

Bronchiolitis

Provide supplemental oxygen as required to keep oxygen saturation >94%. Provide nasal and oral suctioning as needed.

Bronchodilators (e.g., albuterol, nebulized epinephrine or racemic epinephrine) may be of benefit in some children and infants with bronchiolitis if nonpharmacologic

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Heliox

Heliox, a mixture of helium and oxygen, may be beneficial as an alternative to O_2 in some children or infants with airway obstruction. Helium has a lower density than O_2, so the addition of helium improves laminar flow through areas of obstruction, reducing airway resistance.

supportive measures are ineffective. Continue bronchodilator therapy only if patient improves.

- Note: While current evidence suggests no benefit for routine bronchodilator therapy in bronchiolitis, studies to date have generally excluded children with severe disease or respiratory failure; therefore, clinical judgment is warranted.

Corticosteroids may also be considered in children and infants with bronchiolitis if they have a personal or family history of asthma or if they are experiencing progressive respiratory distress, respiratory failure or respiratory arrest.

- Note: While a systematic review suggested no benefit for routine corticosteroid use in bronchiolitis, this review excluded studies incorporating children with a history of recurrent wheezing or formal asthma diagnosis, children in intensive care settings and intubated or ventilated children; therefore, clinical judgment is warranted.

Monitor for progressive respiratory distress, respiratory failure or apnea. Support ventilations as needed with noninvasive (CPAP, BiPAP) or invasive ventilation. Maintain hydration with IV fluid as needed.

Foreign-Body Aspiration

Children and infants with a foreign body aspiration typically require bronchoscopy, ideally after confirming the diagnosis by radiograph. Support ventilation as needed. Foreign body aspiration may lead to bronchospasm or pneumonia. It is important to monitor for these complications and provide care as needed.

See *Lower Airway Obstruction* Treatment Guideline for a summary of care.

Care for Respiratory Emergency Due to Lung Tissue Disease

Treatment beyond standard airway and ventilatory management in lung tissue disease is typically disease specific. Children and infants with cardiogenic pulmonary edema may benefit from the administration of diuretics, which aim to reduce lung "water" and improve pulmonary compliance. In addition, vasoactive, inotropic or inodilator therapy and noninvasive or invasive ventilation may be needed.

Treatment for ARDS consists of using a multi-focal approach based on latest evidence, including use of low tidal volumes (as clinically indicated), titration of positive end-expiratory pressure (PEEP) and avoidance of fluid overload. Treating the underlying etiology is also important.

If patients with pneumonia have evidence of bronchospasm, consider a trial of inhaled bronchodilators. In addition, promptly administer appropriate antibiotics in children and infants with suspected bacterial pneumonia. Select the antibiotic treatment based on latest evidence (i.e., likely pathogens, antibiotic resistance patterns). Consider broad-spectrum antibiotics if systemic involvement or sepsis is suspected. Obtain blood cultures if systemic involvement/sepsis is suspected. For patients with pneumonia who are experiencing worsening respiratory distress or respiratory failure or arrest, assisted (noninvasive or invasive) ventilation may be necessary.

See *Lung Tissue Disease* Treatment Guideline for a summary of care.

Care for Respiratory Emergency Due to Neurological and Metabolic Disorders of Ventilation

Treatment for a respiratory emergency due to a neurological or metabolic disorder of ventilation will vary based on what is causing the issue. Review care for each of the following conditions.

Brain Injury or Other CNS Disease/Injury

Patients with respiratory depression due to brain injury or other CNS disease or injury may require assisted ventilation. Such patients may also require placement of an artificial airway when normal airway reflexes are compromised.

Increased ICP

In patients with underlying increased ICP:

- Maintain airway with spinal precautions, if indicated.
- Elevate the head of the bed 30 degrees and keep head midline.
- Ensure adequate oxygenation and ventilation. Short-term hyperventilation may be warranted in patients who are showing signs of imminent herniation.
- As clinically indicated, treat increased ICP with hypertonic 3% saline or mannitol.
- Manage cerebral perfusion pressure:
  - Maintain adequate arterial blood pressure.
  - Administer isotonic crystalloid fluid boluses or, if indicated, blood products for volume expansion as needed.
  - Administer vasopressors if needed to maintain cerebral perfusion pressure.
  - Avoid hypotonic fluids even for maintenance fluid therapy.

- Avoid or treat any factors that might further increase ICP:
  - Consider administering lidocaine prior to endotracheal intubation.
  - Minimize noxious stimuli (e.g., tracheal suctioning).
  - Maintain minimal stimulation setting for the patient.
  - Treat seizures.
  - Treat hyperthermia.
  - Treat pain.
  - Correct hyponatremia.

Consider pursuing neurosurgical consultation when surgical intervention may be needed (e.g., epidural or subdural hematoma, tumor), when invasive intracranial monitor placement is indicated, or for refractory intracranial hypertension.

Neuromuscular Diseases

Children with neuromuscular diseases presenting with respiratory failure require assisted ventilation. Initiate or adjust non-invasive and invasive ventilation as indicated. Intubate to protect the airway, as clinically indicated. A trial of non-invasive ventilation may be considered as an alternative to endotracheal intubation and invasive ventilation in children with neuromuscular diseases who do not have tracheostomies. Airway clearance (e.g., suctioning) is a critical component of airway management in patients with neuromuscular disorders.

Suspected Drug Intoxication

Immediately contact the American Association of Poison Control Centers (800-222-1222) for assistance with patient management. Be prepared to suction the airway in the event of emesis. In addition, be prepared to protect the airway with an advanced airway.

> In patients with ineffective ventilation or apnea caused by suspected drug intoxication, take immediate measures to reverse the drug's effect, when possible (e.g., with naloxone in the case of opioid overdose).

Likewise, administer an antidote, if available, and as recommended by the American Association of Poison Control Centers. Other measures may be taken to limit absorption (e.g., gastric lavage) or enhance excretion (e.g., urinary alkalization) when intoxicants are suspected in the patient presenting with abnormal breathing, as recommended by the American Association of Poison Control Centers.

- Note: Gastric lavage is contraindicated in patients with an unprotected airway and for certain toxins (e.g., hydrocarbons, caustic agents).

Order laboratory and diagnostic studies as needed.

See *Learn More: Opioid Intoxication* for how to care for the patient with suspected opioid intoxication.

Metabolic Conditions

Treatment of metabolic conditions varies based on the specific condition. Treatment of diabetic ketoacidosis consists of administering insulin, after restoring intravascular volume and total body normovolemia. Caution in speed of fluid replacement is warranted.

Acute management of organic acidemias and other inherited disorders of metabolism varies based on the specific condition and may involve measures to correct biochemical abnormalities such as acidosis, hypoglycemia or **hyperammonemia** (including hemodialysis), or administration of specific pharmacologic agents or nutrients as indicated based on etiology.

See *Neurological and Metabolic Disorders of Ventilation* Treatment Guideline for a summary of care.

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Opioid Intoxication

Respiratory depression is a hallmark feature of opioid intoxication. Opioids are natural or synthetic agents that are used to treat pain but may also be used illicitly for their euphoric effects. Opioids interact with receptors in the brain that control respiratory drive in addition to mediating analgesia. Excessive doses may cause a slowed respiratory rate or apnea. Other signs of opioid intoxication include small, constricted pupils (miosis) and stupor.

Hospitalizations for opioid intoxication among children and adolescents increased two-fold between 1997 and 2012. Prescription opioids accounted for most hospitalizations; however, hospitalizations for illicit opioid intoxication also significantly increased among adolescents aged 15 to 19 years during this time period. The greatest increase in hospitalizations occurred among children aged 1 to 4 years.

In addition to assisting ventilation as needed, immediate management of opioid intoxication includes the administration of the reversal agent naloxone. It can be administered by the IV, IO, IM, or SC route. Naloxone should be dosed as follows, repeating the dose every 2 minutes as needed to attain or maintain opioid reversal:

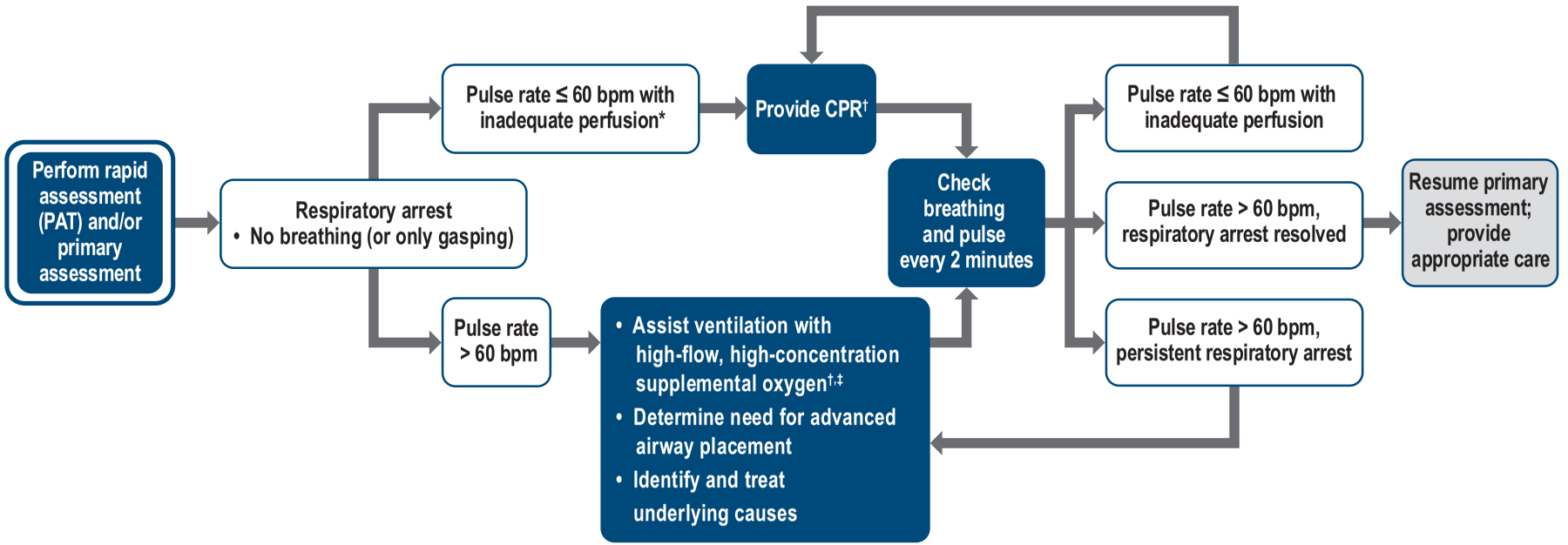
- 0.1 mg/kg (maximum, 2 mg), IV/IO/IM/SC

Patients should be monitored for recurrent respiratory depression after initial reversal with naloxone.

ALERT

Administration of naloxone may precipitate acute withdrawal in opiate-dependent patients or severe symptoms in patients with a source of pain.

RESPIRATORY ARREST



Obstructed Airway Management

- If the first ventilation does not cause the chest to rise, re-tilt the head to ensure that the airway is properly opened and ensure that the patient's nose and mouth are properly sealed before delivering the second ventilation
- If the second ventilation does not make the chest rise, an object may be blocking the patient's airway
- Perform CPR with modifications:
  - After each set of compressions and before ventilations, open the patient's mouth and look for the object – if seen, remove it using a finger sweep. Do not perform a blind finger sweep
  - Attempt 2 ventilations. Never try more than 2 ventilations during 1 cycle of CPR, even if the chest does not rise
  - Continue performing CPR cycles, checking for an object before each set of ventilations
- Consider using basic and advanced airway techniques based on level of training and expertise to clear the airway

Principles of Bag-Valve-Mask Ventilation

- Each ventilation should last about 1 second and make the chest begin to rise
- Adjust ventilations as needed to maintain $ETCO_2$ values in the range of 35 to 45 mmHg
- Avoid hyperventilation (i.e., ventilations that are too fast or have too much volume)
- Increasing difficulty when providing ventilations using a BVM resuscitator may indicate an increase in intrathoracic pressure, inadequate airway opening, gastric distension, pneumothorax or other complications

*Signs of inadequate perfusion include cool moist skin, pallor, mottling or cyanosis, weak or thready pulse, decreased capillary refill and hypotension.

†If unable to ventilate, re-tilt and if still unable to ventilate go to FBAO clearing procedure.

‡See *Principles of Bag-Valve-Mask Ventilation* table.

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RESPIRATORY DISTRESS OR FAILURE

Perform rapid assessment (PAT), primary assessment and initial interventions

Airway
- Open the airway using head-tilt/chin-lift technique or modified jaw-thrust maneuver
- Clear the airway (e.g., suctioning, removal of visible foreign body [do not do a blind finger sweep])*
- Consider a basic (oro- or nasopharyngeal) airway if clinically indicated
- Consider advanced airway if clinically indicated
- Consult with advanced airway specialist for difficult airway†

Breathing
- Establish pulse oximetry
- Establish capnography in intubated patients and, if available, in nonintubated patients
- Provide humidified supplemental oxygen as required to keep O_2 saturation > 94%
- Assist ventilation as needed using any of the following:
  - BVM ventilation
  - Noninvasive positive airway pressure
  - Invasive (i.e., mechanical) ventilation
- Perform immediate needle thoracentesis for suspected or known tension pneumothorax

Other Measures
- If not already done:
  - Establish cardiac monitoring and assess perfusion
  - Establish vascular access
    - Note: to avoid agitating the patient, may opt to defer vascular access in spontaneously ventilating patients who do not require IV therapy or who are not exhibiting signs of deterioration
- Monitor for signs of worsening respiratory function (e.g., increasing work of breathing, increasing/decreasing respiratory rate, hypoxemia, change in general appearance, change in mental status or change in muscle tone)

Identify type of respiratory problem (see *"Key" Potential Assessment Findings by Respiratory Problem Type* table, on reverse side)

Respiratory Compromise Continuum

Respiratory distress → Respiratory failure → Respiratory arrest → Cardiac arrest

Differentiating Severity of Respiratory Compromise

Stage	Description	Signs and Symptoms	
Respiratory distress	Earliest stage of respiratory compromise; patient maintains adequate oxygenation and ventilation via compensatory mechanisms. Can progress into respiratory failure	• Tachypnea • Increased work of breathing (accessory muscle use, nasal flaring) • Varying degrees of airway obstruction (as evidenced by stridor, drooling, wheezing) • Abnormal breath sounds • Grunting	• Tachycardia • Irritability or anxiety • Assuming position of comfort (e.g., tripod positioning) • Pallor • Cyanosis which resolves with supplemental oxygen
Respiratory failure	Patient unable to maintain adequate oxygenation (hypoxic) or ventilation (hypercapneic) to meet metabolic demands; usually requires ventilatory support; will lead to respiratory arrest if not quickly addressed	• Slowed respiratory rate (may initially be very rapid) • Poor or absent air movement • Low oxygen saturation or low PaO_2 • High $ETCO_2$ or high PCO_2	• Bradycardia (may initially see tachycardia) • Altered level of consciousness (e.g., lethargy, somnolence or unconsciousness) • Central cyanosis (may not resolve with supplemental oxygen) • Pallor
Respiratory arrest	Complete cessation of breathing effort; leads to cardiac arrest after a very short time	• Absent breath sounds • Lack of chest movement • Bradycardia (may initially see tachycardia)	• Hypotension • Loss of consciousness • Cyanosis or pallor

Normal Pediatric Vital Signs

Age Group	Respiratory Rate, Breaths per Minute	Awake Heart Rate, Beats per Minute	Systolic Blood Pressure, mmHg	Diastolic Blood Pressure, mmHg
Newborn	30–60	100–200	60–85	35–55
Infant (1 to 12 months)	30–50	100–180	70–100	35–60
Toddler (1 to 2 years)	24–40	90–140	85–105	40–65
Preschooler (3 to 5 years)	20–30	80–130	89–115	45–70
School Age (6 to 12 years)	16–26	70–120	94–120	55–80
Adolescent (13 to 17 years)	12–20	60–100	110–135	60–85

Please see reverse side for additional information

*For a complete airway obstruction see *BLS Obstructed Airway: Adults and Children* or *BLS Obstructed Airway: Infants (< 1 Year Old) Treatment Guidelines.*

†Such as an anesthesiologist or otolaryngologist.

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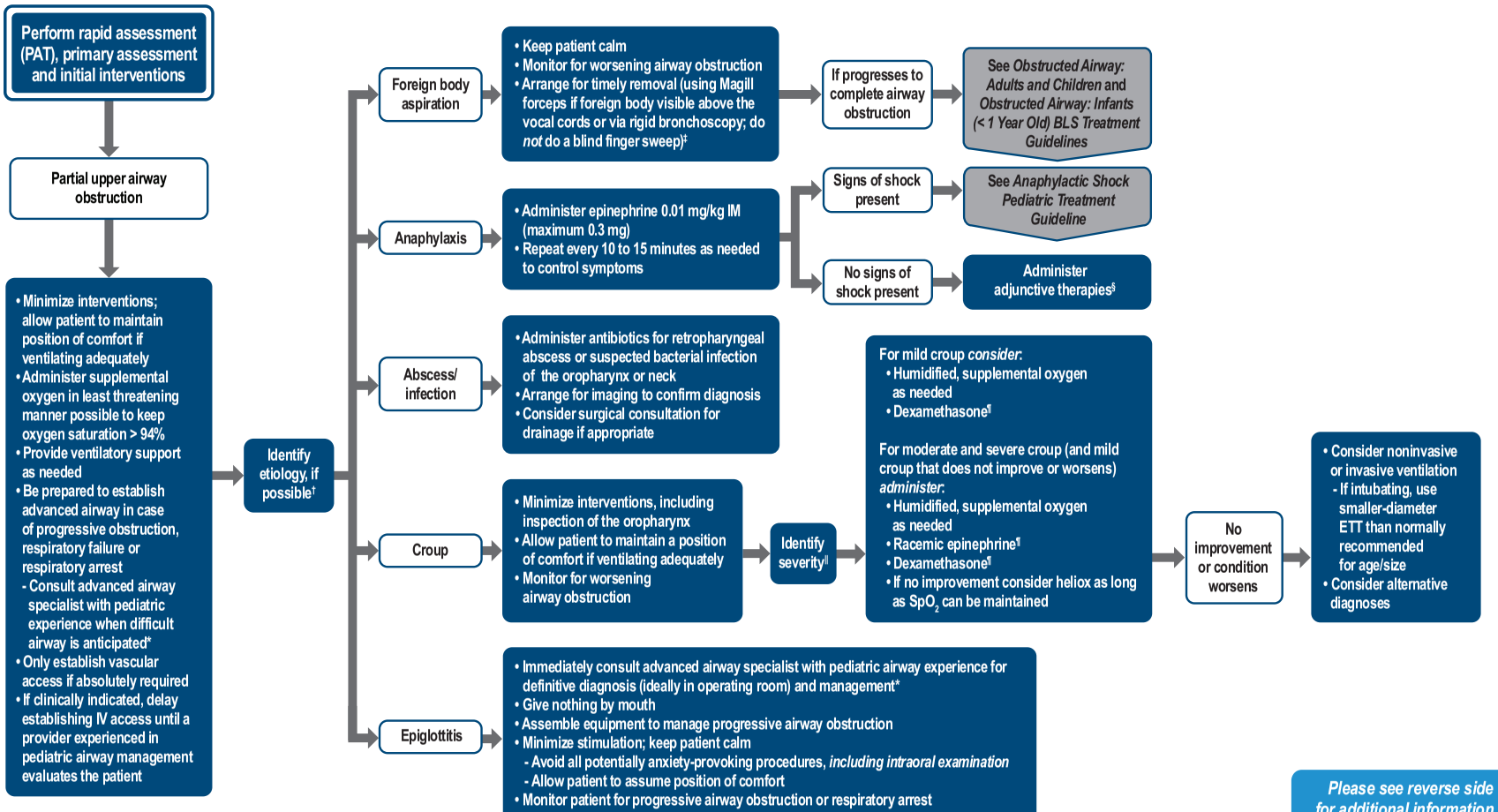
RESPIRATORY DISTRESS OR FAILURE CONTINUED

"Key" Potential Assessment Findings by Respiratory Problem Type

Assessment	Partial Upper Airway Obstruction	Lower Airway Obstruction	Lung Tissue Disease	Neurologic and Metabolic Disorders of Ventilation
Airway	• Stridor (inspiratory; may be expiratory) • Trouble swallowing, drooling/difficulty managing secretions • Voice (or cry) changes (e.g., hoarseness/"hot potato" voice) • Unmaintainable airway (late) • Sudden-onset signs of airway obstruction and respiratory compromise (foreign body aspiration)	• Unmaintainable airway (late)	• Unmaintainable airway (late)	• Unmaintainable airway due to altered mental status • Impaired swallowing, drooling (neuromuscular diseases) • Ineffective airway clearance
Breathing: respiratory rate	• Tachypnea • Bradypnea or apnea (late)	• Tachypnea • Bradypnea or apnea (late)	• Tachypnea • Bradypnea or apnea (late)	• Tachypnea, bradypnea or apnea • Irregular breathing pattern (e.g., Cheyne-Stokes breathing)
Breathing: work of breathing	• Retractions • Nasal flaring	• Retractions • Nasal flaring	• Retractions • Nasal flaring	• Normal, increased or irregular
Breathing: air movement	• Decreased	• Decreased • Prolonged exhalation	• Decreased	• Variable
Breathing: abnormal sounds	• Stridor (inspiratory; may be expiratory)	• Wheezing • Grunting • Rhonchi (bronchiolitis) • Crackles	• Grunting • Decreased breath sounds (pneumonia) • Localized crackles (pneumonia) • Generalized crackles and wheezes (pulmonary edema)	• None
Other	• Barking or brassy cough (croup)	• Unable to talk in full sentences • Wet, "junky" cough (bronchiolitis)	• Shallow respirations • Cough	• Ineffective cough (neuromuscular diseases) • Cushing's triad (abnormal breathing, hypertension and bradycardia; associated with increased ICP)
Circulation	• Tachycardia • Pallor, cyanosis	• Tachycardia (bradycardia with hypoxia and respiratory failure) • Pulsus paradoxus • Pallor, cyanosis	• Tachycardia • Pallor, cyanosis (late)	• Tachycardia • Hypertension • Bradycardia • Cyanosis (apnea)
Disability	• Restless, anxious, irritable, unable to get comfortable • Assuming a position of comfort (e.g., tripod positioning) • Agitation, somnolence or unconsciousness (late)	• Restless, anxious • Reluctance to lie flat • Agitation, somnolence or unconsciousness (late)	• Restless, anxious • Agitation, somnolence or unconsciousness (late)	• Altered mental status (CNS conditions, toxins, metabolic conditions) • Pupillary changes (CNS conditions, toxins) • Global muscle weakness, hypotonia in infants (neuromuscular diseases) • Posturing (CNS conditions)
Exposure *(possible findings)*	• Skin reactions (rashes) • Increased or decreased temperature • Toxic appearance • Swelling (anaphylaxis, infection or abscess)	• Increased or decreased temperature	• Increased or decreased skin temperature	• Signs of trauma, bleeding, needle marks (injection), increased or decreased temperature • Chest wall deformity, kyphoscoliosis, thin or atrophied extremities, contractures (neuromuscular diseases)
Secondary assessment	• Hypoxemia in severe obstruction (croup) • Radiograph may show steeple sign (croup) or "thumb sign" (epiglottitis)	• Hypoxemia • Chest radiograph may show hyperinflation or air trapping (both sides in asthma/bronchiolitis; one side with foreign body aspiration) • Chest radiograph may show object in foreign body aspiration • Rapid RSV testing positive (bronchiolitis) • Rapid flu testing, respiratory viral studies positive	• Hypoxemia • Chest radiography: airspace opacity, lobar consolidation or interstitial opacities	• Acute or chronic metabolic alkalosis or acidosis
	See *Partial Upper Airway Obstruction Pediatric Treatment Guideline*	See *Lower Airway Obstruction Pediatric Treatment Guideline*	See *Lung Tissue Disease Pediatric Treatment Guideline*	See *Neurologic and Metabolic Disorders of Ventilation Pediatric Treatment Guideline*

PARTIAL UPPER AIRWAY OBSTRUCTION



Please see reverse side for additional information

*Such as an anesthesiologist or otolaryngologist.

†See *Identifying Underlying Etiologies* table, on reverse side.

‡If the patient has a partial obstruction and is oxygenating and ventilating adequately arrange for transport to the operating room for foreign body removal as appropriate.

§See *Adjunctive Therapies for Anaphylaxis* table, on reverse side.

‖See *Croup Severity Categories* table, on reverse side.

¶See *First-Line Therapies for Croup* table, on reverse side.

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PARTIAL UPPER AIRWAY OBSTRUCTION CONTINUED

Identifying Underlying Etiologies

"Key" Condition-Specific Findings:

Croup
- "Barking" or "brassy" cough

Epiglottitis
- Four Ds
  - Drooling
  - Dyspnea
  - Dysphagia (trouble swallowing)
  - Dysphonia (trouble speaking)
- High fever
- Toxic appearance

Anaphylaxis
- Cutaneous and mucosal finding (e.g., swollen lips, tongue or uvula [angioedema], hives, flushing, itching) and one of the following:
  - Respiratory compromise (stridor, hoarseness, wheezing, dyspnea, increased work of breathing)
  - Hypotension and signs of end-organ dysfunction

OR
- Two or more of the following post exposure to an allergen:
  - Cutaneous and mucosal finding (e.g., swollen lips, tongue or uvula [angioedema], hives, flushing, itching)
  - Respiratory compromise (stridor, hoarseness, wheezing, dyspnea, increased work of breathing)
  - Hypotension and signs of end-organ dysfunction
  - Gastrointestinal symptoms (e.g., nausea, vomiting, abdominal pain)

OR
- Hypotension post exposure to a known allergen

Foreign Body Aspiration
- Sudden-onset signs of airway obstruction and respiratory compromise

Abscess/Infection
- Swelling
- Fever
- Trouble swallowing
- Voice or cry changes

Croup Severity Categories

Mild	• Occasional barking cough • No stridor at rest
Moderate	• Frequent barking cough • Audible stridor and retractions at rest
Severe	• Frequent barking cough • Marked stridor and visible retractions at rest • Agitation/distress

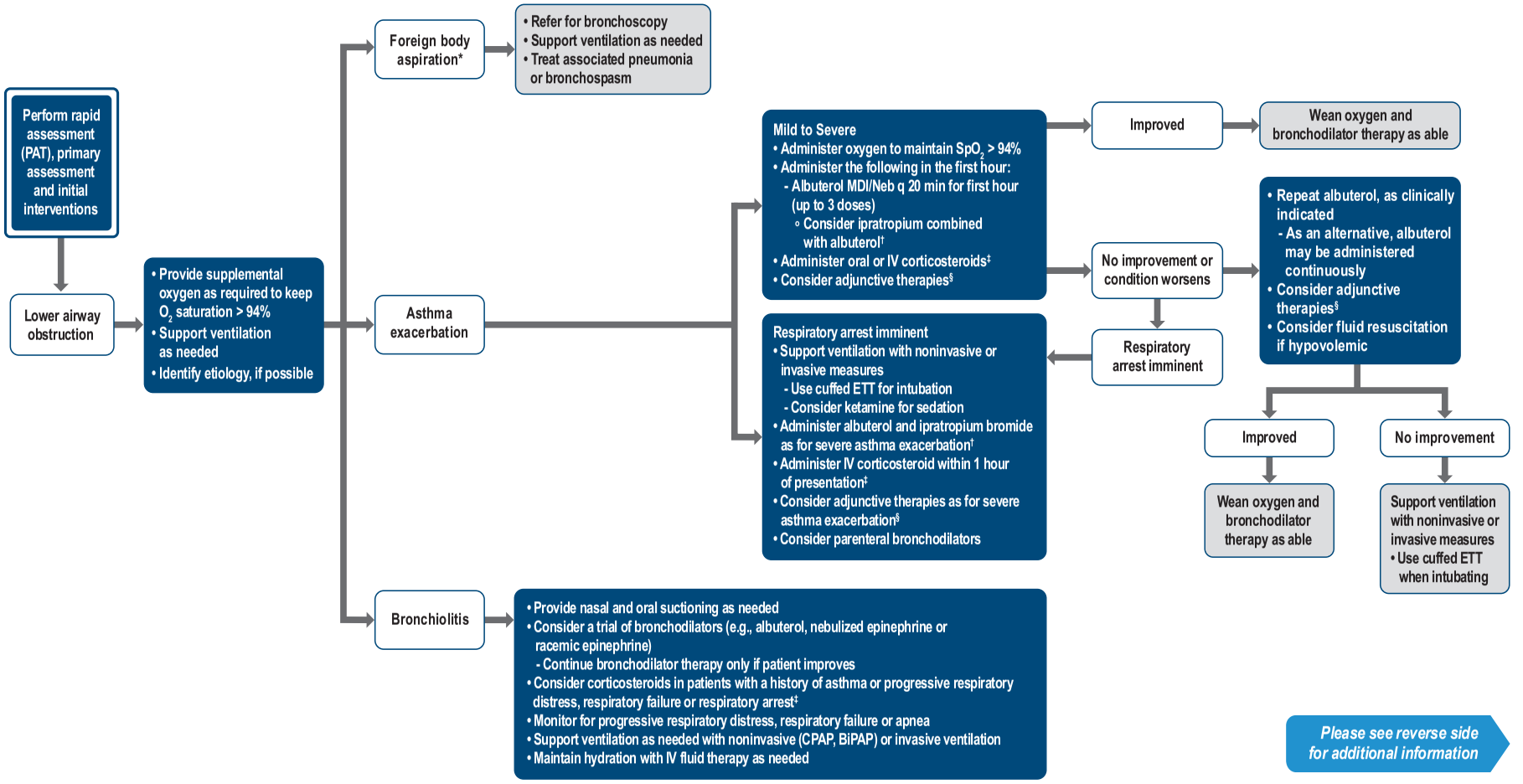
First-Line Therapies for Croup

Medication	Dosage
Racemic epinephrine	0.25 to 0.5 mL of the 2.25% solution diluted in 3 mL of NS via nebulizer Note: when racemic epinephrine is not available, may administer 3 to 5 mg of standard epinephrine (1-mg/mL concentration) mixed with 3-mL NS via nebulizer
Dexamethasone	0.6 mg/kg (maximum, 16 mg) PO/IV/IM every 24 hours Note: may use other steroid (e.g., prednisone)

Adjunctive Therapies for Anaphylaxis

Medication	Dosage
Albuterol	By metered-dose inhaler (MDI; 90 mcg/actuation): • 4 to 8 inhalations every 20 minutes as needed for 3 doses, then every 1 to 4 hours, or as clinically indicated By nebulizer (intermittent): • Children < 12 years: - Initial dosing: 0.15 mg/kg (minimum, 2.5 mg) every 20 minutes as needed for 3 doses, then 0.15 to 0.3 mg/kg (maximum, 10 mg) every 1 to 4 hours, or as clinically indicated • Children ≥ 12 years: - Initial dosing: 2.5 to 5 mg every 20 minutes as needed for 3 doses, then 2.5 to 10 mg every 1 to 4 hours, or as clinically indicated By nebulizer (continuous): • Children < 20 kg: 7.5 mg/hour • Children ≥ 20 kg: 10 mg/hour • Children ≥ 12 years: 10 to 15 mg/hour
Methylprednisolone	2 mg/kg IV/IO/IM as an initial dose Maintenance dosing: 0.5 mg/kg IV every 6 hours or 1 mg/kg IV every 12 hours (maximum daily dose, 120 mg)
Diphenhydramine	1 to 2 mg/kg (maximum, 50 mg), IM/IV/PO

LOWER AIRWAY OBSTRUCTION



*If complete foreign body airway obstruction see *BLS Treatment Guidelines BLS Obstructed Airway: Adult* and *Child and BLS Obstructed Airway: Infant*.

†The albuterol and ipratropium bromide combination is always used when the patient is experiencing a severe asthma exacerbation or when respiratory arrest is imminent.

‡For full dosing and adminstration information, see *Medications for Treatment of Bronchospasm* table, on reverse side.

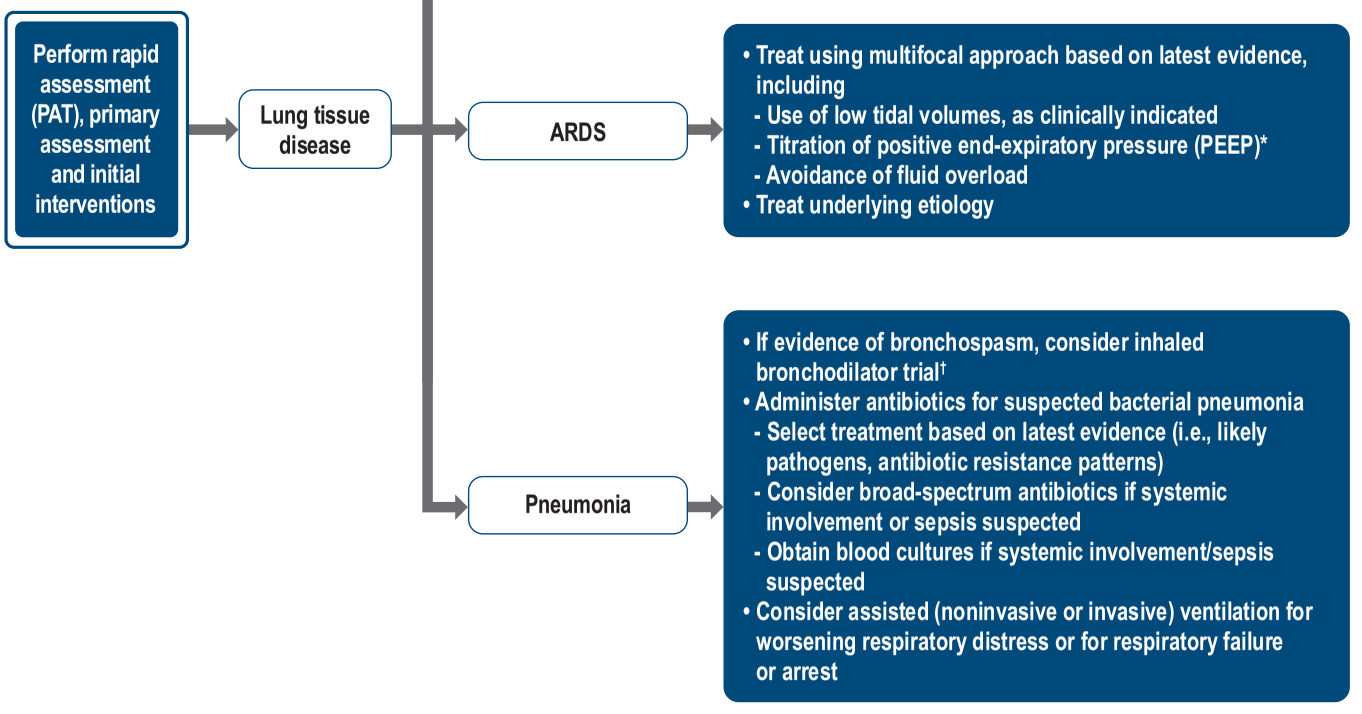
§See *Adjunctive Therapies for Severe Asthma Exacerbation* table, on reverse side.

LOWER AIRWAY OBSTRUCTION CONTINUED

Medications for Treatment of Bronchospasm	
Inhaled Bronchodilators	
Albuterol	By metered-dose inhaler (MDI; 90 mcg/actuation): • 4 to 8 inhalations every 20 minutes as needed for 3 doses, then every 1 to 4 hours, or as clinically indicated By nebulizer (intermittent): • Children < 12 years: - Initial dosing: 0.15 mg/kg (minimum, 2.5 mg) every 20 minutes as needed for 3 doses, then 0.15 to 0.3 mg/kg (maximum, 10 mg) every 1 to 4 hours, or as clinically indicated • Children ≥ 12 years: - Initial dosing: 2.5 to 5 mg every 20 minutes as needed for 3 doses, then 2.5 to 10 mg every 1 to 4 hours, or as clinically indicated By nebulizer (continuous): • Children < 20 kg: 7.5 mg/hour • Children ≥ 20 kg: 10 mg/hour • Children ≥ 12 years: 10 to 15 mg/hour
Levalbuterol	By MDI: • 4 to 8 inhalations every 20 minutes as needed for 3 doses, then every 1 to 4 hours, or as clinically indicated By nebulizer (intermittent): • Children < 12 years: - Initial dosing: 0.075 mg/kg (minimum, 1.25 mg) every 20 minutes as needed for 3 doses, then 0.075 to 0.15 mg/kg (maximum, 5 mg) every 1 to 4 hours, or as clinically indicated • Children ≥ 12 years: - Initial dosing: 1.25 to 2.5 mg every 20 minutes as needed for 3 doses, then 1.25 to 5 mg every 1 to 4 hours, or as clinically indicated
Ipratropium bromide	• 0.5 mg via nebulizer every 20 minutes for up to 3 doses
Combined albuterol/ipratropium bromide formulation (3 mL = 0.5-mg ipratropium bromide/2.5-mg albuterol base)	• 3 mL via nebulizer every 20 minutes for up to 3 doses
Racemic epinephrine	• 0.25 to 0.5 mL of the 2.25% solution diluted in 3 mL of NS via nebulizer • Note: when racemic epinephrine is not available, may administer 3 to 5 mg of standard epinephrine (1-mg/mL concentration) mixed with 3-mL NS via nebulizer
Corticosteroids	
Dexamethasone	• 0.6 mg/kg (maximum, 16 mg) PO/IV/IM every 24 hours
Methylprednisolone	• 2 mg/kg IV/IO/IM as an initial dose • Maintenance dosing: 0.5 mg/kg IV every 6 hours or 1 mg/kg IV every 12 hours (maximum daily dose, 120 mg)
Prednisone/prednisolone	• 1 to 2 mg/kg/day divided in 1 to 2 doses for 3 to 10 days total (maximum, 60 mg/day) • Taper dose if given for > 10 days
Adjunctive Therapies for Severe Asthma Exacerbation	
Magnesium sulfate	• 25 to 50 mg IV/IO (maximum, 2 g) over 15 to 30 minutes
Epinephrine	• 0.01 mg/kg (maximum dose, 0.3 to 0.5 mg) subcutaneously every 20 minutes for 3 doses, then as clinically indicated
Terbutaline	• Subcutaneously: 0.01 mg/kg every 20 minutes for up to 3 doses with a maximum of 0.25 mg • IV/IO infusion: 0.4 mg/kg/hr (starting dose), then 0.1 to 10 mcg/kg/min

Assessment Component	Asthma Severity Categories			
	Mild	Moderate	Severe	Respiratory Arrest Imminent
Symptoms	• Breathlessness while walking • Can lie down • Talks in sentences • May be agitated	• Breathlessness while at rest (softer, shorter cry and difficulty feeding in infants) • Prefers sitting • Talks in phrases • Usually agitated	• Breathlessness while at rest • Sits upright • Talks in words • Usually agitated	• Inability to breathe • Drowsiness, confusion or loss of consciousness
Signs	• Respiratory rate increased • Moderate wheezing • Pulse normal for age (may be elevated if receiving bronchodilators)	• Respiratory rate increased • May have decreased air movement • Use of accessory muscles and suprasternal retractions commonly present • Loud wheezing • Pulse elevated for age • Pulsus paradoxus may be present (10 to 25 mmHg)	• Respiratory rate often > 30/minute (or increased for age) • Severely decreased air movement • Use of accessory muscles and suprasternal retractions usually present • Loud wheezing throughout inhalation and exhalation (may not be heard due to lack of air movement) • Significant tachycardia for age • Pulsus paradoxus often present (20 to 40 mmHg)	• Paradoxical thoracoabdominal movement ("seesaw breathing") • Absence of air movement • Absence of wheeze • Bradycardia • Absence of pulsus paradoxus (suggests respiratory muscle fatigue)
Functional assessment	• PEF (percent predicted or personal best) ≥ 70 • ABG findings on room air (test not usually necessary): - PaO_2 normal - $PaCO_2$ < 42 mmHg - SaO_2 > 95%	• PEF (percent predicted or personal best) ~ 40 to 69% or response lasts < 2 hours • ABG findings on room air (test not usually necessary): - PaO_2 ≥ 60 mmHg - $PaCO_2$ < 42 mmHg - SaO_2 90 to 95%	• PEF (percent predicted or personal best) < 40 • ABG findings on room air: - PaO_2 < 60 mmHg - $PaCO_2$ ≥ 42 mmHg - SaO_2 < 90%	• PEF (percent predicted or personal best) < 25% • ABG findings on room air: - PaO_2 < 60 mmHg - $PaCO_2$ ≥ 42 mmHg - SaO_2 < 90%

LUNG TISSUE DISEASE



Assessment Findings: Lung Tissue Diseases

Primary Assessment Findings:

- Unmaintainable airway (late)
- Tachypnea
- Bradypnea or apnea (late)
- Increased work of breathing (retractions, nasal flaring)
- Cough
- Grunting
- On lung auscultation:
  - Decreased breath sounds (pneumonia)
  - Decreased air movement
  - Localized crackles (pneumonia)
  - Generalized crackles and wheezes (pulmonary edema)
- Tachycardia
- Altered mental status
- Pallor, cyanosis (late)
- Fever (pneumonia)
- Restless, anxious
- Agitation, somnolence or unconsciousness (late)

Please see reverse side for additional information

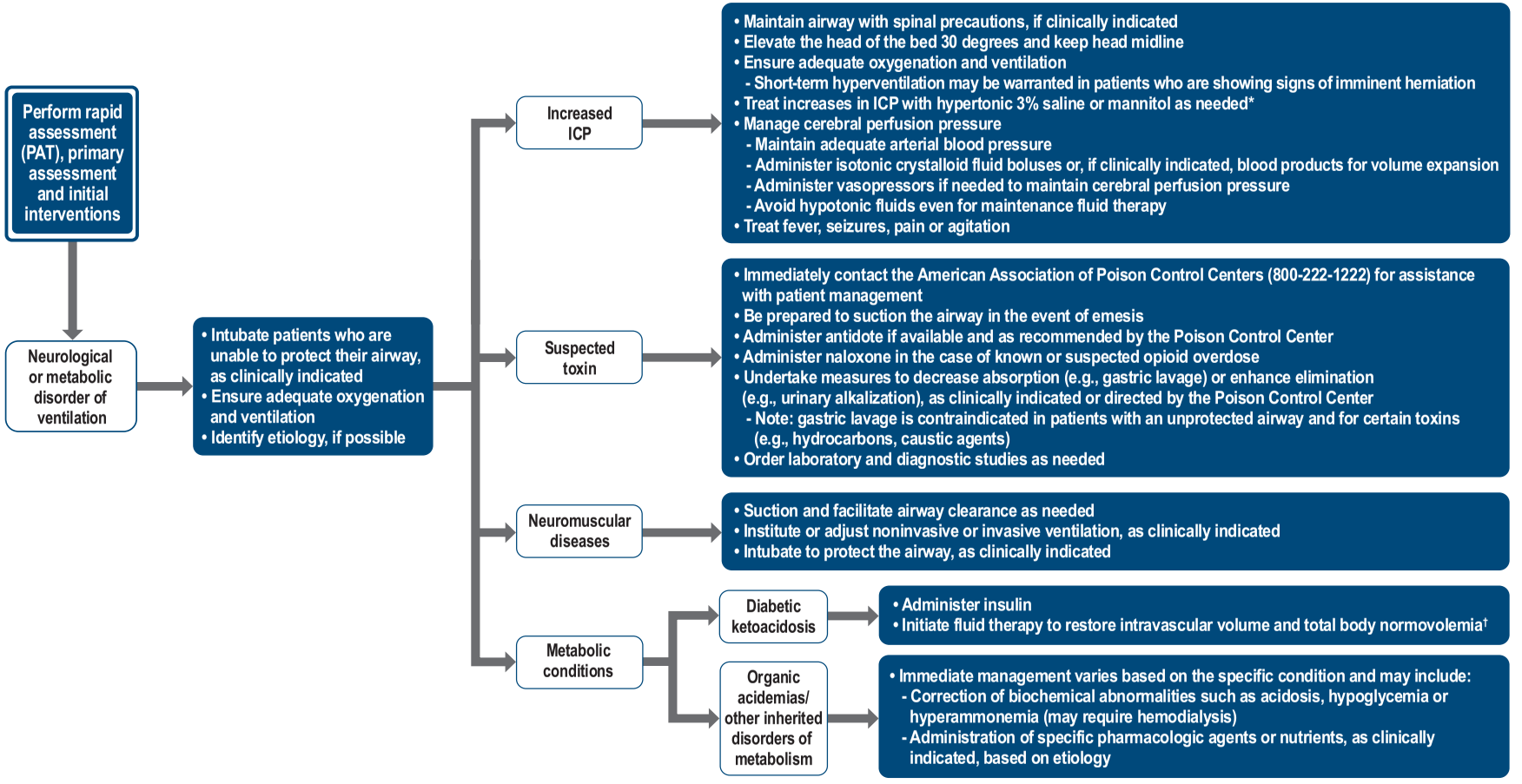
*PEEP should be titrated based on patient's observed oxygenation and hemodynamic response.

†See *Initial Bronchodilator Therapy* table, on reverse side.

LUNG TISSUE DISEASE CONTINUED

Initial Bronchodilator Therapy	
Medication	**Dosage**
Albuterol	By metered-dose inhaler (MDI; 90 mcg/actuation): • 4 to 8 inhalations every 20 minutes as needed for 3 doses, then every 1 to 4 hours, or as clinically indicated By nebulizer (intermittent): • Children < 12 years: - Initial dosing: 0.15 mg/kg (minimum, 2.5 mg) every 20 minutes as needed for 3 doses, then 0.15 to 0.3 mg/kg (maximum, 10 mg) every 1 to 4 hours, or as clinically indicated • Children ≥ 12 years: - Initial dosing: 2.5 to 5 mg every 20 minutes as needed for 3 doses, then 2.5 to 10 mg every 1 to 4 hours, or as clinically indicated By nebulizer (continuous): • Children < 20 kg: 7.5 mg/hour • Children ≥ 20 kg: 10 mg/hour • Children ≥ 12 years: 10 to 15 mg/hour
Ipratropium bromide	• 0.5 mg via nebulizer every 20 minutes for up to 3 doses
Combined albuterol/ipratropium bromide formulation (3 mL = 0.5-mg ipratropium bromide/2.5-mg albuterol base)	• 3 mL via nebulizer every 20 minutes for up to 3 doses

NEUROLOGICAL AND METABOLIC DISORDERS OF VENTILATION



Please see reverse side for additional information

*In addition, in patients with underlying increased ICP due to trauma, consider consulting a neurosurgeon.

†Caution in the speed of fluid replacement is warranted.

NEUROLOGICAL AND METABOLIC DISORDERS OF VENTILATION CONTINUED

Identifying Underlying Etiologies

"Key" Condition-Specific Findings:

Increased ICP
- Variable respiratory rate and pattern, including:
  - Compensatory hyperventilation (i.e., increased rate and depth of respiration)
  - Irregular, decreased respirations
  - Cheyne-Stokes breathing (i.e., alternating periods of rapid and slow breathing)
- Cushing triad (abnormal breathing, hypertension, bradycardia)
- Altered mental status
- Pupillary changes
- Posturing
- Signs or history of trauma

Neuromuscular Diseases
- Ineffective cough or airway clearance
- Chest wall deformity
- Kyphoscoliosis
- Hypotonia
- Thin or atrophied extremities; contractures
- History of neuromuscular disease

Metabolic Conditions
- Tachypnea (and associated hyperpnea in many conditions due to acidosis)
- Altered mental status

Suspected Toxins
- Unmaintainable airway
- Tachypnea, bradypnea or apnea
- Altered mental status
- Pupillary changes
- Needle marks

Medications for Increased ICP

Hypertonic Saline
- Bolus dosing for acute use: 6.5 to 10 mL/kg IV/IO
- Continuous infusion for ongoing control of ICP: 0.1 to 1 mL/kg/hour IV/IO titrated to minimal rate required to maintain ICP < 20 mmHg

Special Considerations:
- Maintain serum osmolality < 360 mOsm/L and monitor sodium levels

Mannitol
- 0.25 to 1 g/kg IV/IO over 20 to 30 minutes; may repeat as needed to maintain serum osmolality < 320 mOsm/kg

CHAPTER 7

Shock

Introduction

Shock is an acute state in which the circulatory system is unable to provide adequate oxygen and nutrients to meet the metabolic demands of organs and tissues. In compensated shock, blood pressure is maintained by compensatory peripheral vasoconstriction. Decompensated shock occurs when compensatory mechanisms have been exceeded. It is characterized by hypotension, acidosis and evidence of impaired organ perfusion. There are many potential causes of shock in children and infants, including trauma, gastrointestinal losses (e.g., vomiting, diarrhea), cardiac disease, infection and anaphylaxis.

Shock is a leading cause of morbidity and mortality in children and infants and can take various forms. Hypovolemic shock, which is caused by fluid or blood loss, is the most common form seen in children and infants. Other types of shock include distributive shock (including septic, anaphylactic and neurogenic shock), cardiogenic shock and obstructive shock. Recognizing the signs and symptoms of shock is key because early and aggressive care is of absolute importance when dealing with this pediatric emergency.

Circulatory System Overview

Delivery of oxygen and nutrients to the tissues is critical to support normal cell function. The circulatory system is the "chief executor" of this process. Impaired oxygen and nutrient delivery is central to the pathogenesis of shock. Therefore, in addition to understanding these concepts of pathogenesis, an understanding of the normal functioning of the circulatory system and how it facilitates oxygen delivery to the tissues is important.

Anatomy and Physiology of the Circulatory System

The circulatory system is a closed circuit consisting of a pump (the heart) and conduits (the blood vessels) that deliver oxygen and nutrients to the tissues while removing carbon dioxide and other waste products. Blood is the vehicle by which oxygen and nutrient delivery and waste product removal occur.

The Heart

The heart is a muscular pump that provides the necessary force to circulate blood throughout the entire body. It consists of four chambers: the right atrium, right ventricle, left atrium and left ventricle (Figure 7-1). The atria are thin-walled chambers that receive blood as it comes into the heart, whereas the thick-walled ventricles pump blood out of the heart. One-way valves separate the atrium and ventricle on each side, and a septum separates the atria and ventricles on both sides.

With regard to function, the heart can be thought of as containing two sides. The right side of the heart contains poorly oxygenated ("blue") blood that has returned from the body, and the left side of the heart contains oxygen-rich ("red") blood that will be pumped back out through the body. Blood passes through the lungs to be replenished with oxygen on its way from the right side of the heart to the left.

Blood

Blood carries oxygen and nutrients to the tissues and removes carbon dioxide and other waste products. The vast majority (98%) of oxygen that is carried in the blood is bound to hemoglobin, which is contained within red blood cells that together with plasma make up blood. Blood also transports nutrients derived from digestion, hormones, and disease-fighting proteins and cells to various tissues.

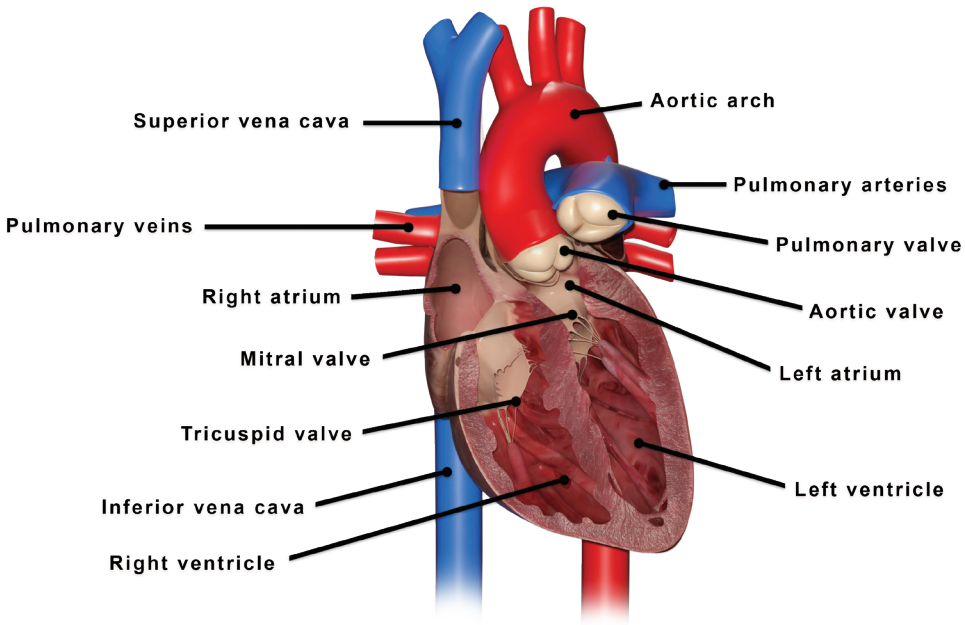


Figure 7-1 | Anatomy of the heart

Blood Vessels

Blood vessels are the conduits through which blood travels to the body's tissues. Three types of blood vessels exist: arteries, capillaries and veins.

Arteries carry blood away from the heart. Blood is pumped from the ventricles into large arteries that branch into progressively smaller arteries and, finally, arterioles that supply the capillaries. The arterial wall contains a muscular layer that constricts or dilates an artery to regulate blood flow and pressure.

Capillaries are the smallest and most numerous type of blood vessel. They provide a connection between the arterial and venous systems and are the site where materials are exchanged between the blood and tissues (Figure 7-2). Capillaries are more abundant in metabolically active tissues (e.g., skeletal muscle, liver, kidney) and are less abundant or even absent in less metabolically active tissues. In the lung, capillaries interface with alveoli, facilitating gas exchange.

Veins are the blood vessels that carry blood from both the body and the lungs back to the heart. Blood that has passed through the capillaries enters the smallest veins, or venules, traveling through progressively larger veins until it enters the heart. The walls of veins are much thinner and less muscular than those of arteries, which allows them to hold a greater volume of blood: at any given time, nearly 70 percent of total blood volume is contained within the veins.

Pulmonary and Systemic Circuits

Blood vessels are functionally divided into two distinct circuits: a pulmonary circuit and a systemic circuit (Figure 7-3).

Within the pulmonary circuit, blood flows between the heart and the lungs. In this circuit, deoxygenated blood coming back from the body is pumped out of the right ventricle through the pulmonary artery and into the lungs, where it is reoxygenated. The circuit is completed with the return of the reoxygenated blood to the left atrium via the four pulmonary veins.

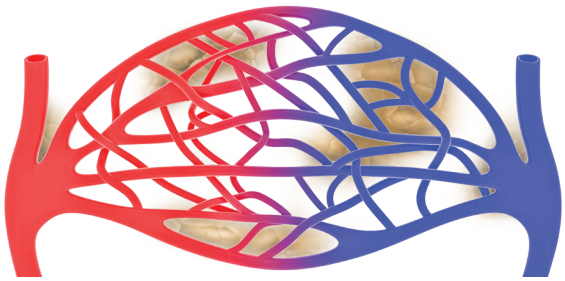


Figure 7-2 | Capillary anatomy

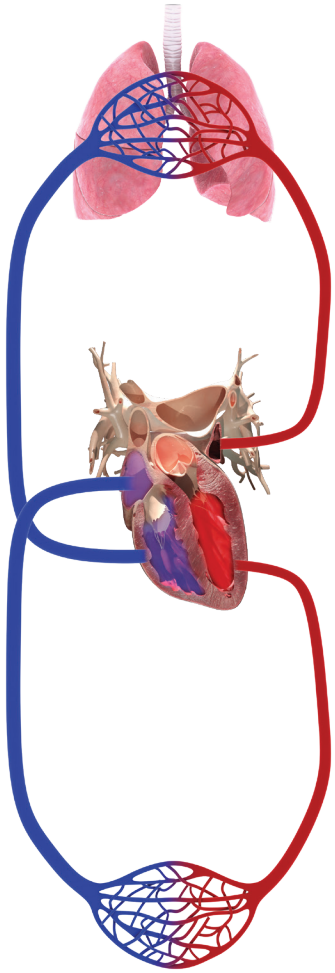


Figure 7-3 | Pulmonary and systemic circuits

In the systemic circuit, blood flows between the heart and the body. Specifically, newly oxygenated blood is pumped from the left ventricle into the aorta, from which it is delivered to the rest of the body. After traversing the capillaries at the tissue level, the now-deoxygenated blood returns to the right atrium via the superior vena cava and inferior vena cava.

Although the pulmonary and systemic circulations are normally separate in children and adults, the two circulations are connected in the developing fetus. See *Learn More: Anatomy and Physiology of Fetal Circulation*.

LEARN MORE

Anatomy and Physiology of Fetal Circulation

The pulmonary and systemic circulations are connected in the developing fetus. A blood vessel called the **ductus arteriosus**, which connects the pulmonary artery and aorta, and the **foramen ovale**, an opening between the right and left atria, allow blood coming back from the body to bypass the lungs, which are underdeveloped and nonfunctional in the fetus. Oxygen and other nutrients are instead provided to the fetus from the maternal circulation via the placenta.

Both the ductus arteriosus and foramen ovale normally close shortly after birth, thus dividing the pulmonary and systemic circulations. Normal closure of the ductus arteriosus may be problematic in children with congenital problems affecting outflow from the left side of the heart, because this connection provides an alternative route through which oxygenated blood can enter the systemic circulation. Conversely, persistence of the ductus arteriosus in infants with otherwise normal hearts may be problematic because it leads to an imbalance between the pulmonary and systemic circulations.

Cardiac Output

As previously stated, the main function of the circulatory system is to deliver oxygen and nutrients to the tissues, with blood as the transport medium. Cardiac output determines how much blood is delivered to the tissues: it is the volume of blood (liters) that leaves the heart each minute and is a product of heart rate (number of times the heart beats per minute) and stroke volume (volume of blood leaving the left ventricle with each heartbeat).

Cardiac output is the product of two variables, heart rate and stroke volume. If one of these variables decreases, the other must increase in order to maintain sufficient cardiac output. Cardiac output can be increased either by an increase in heart rate or by an increase in stroke volume. However, there are some exceptions to this. In infants, maintaining and increasing cardiac output greatly depends on heart rate, not stroke volume. This is because infants have a very small stroke volume with limited ability to increase it. However, increasing cardiac output via an increased heart rate is limited in that if the heart rate is too fast, stroke volume can decrease as a result of a lack of time to fill the ventricles between heartbeats.

Cardiac Output Equation

Cardiac output (L) = Heart rate (beats/min) × Stroke volume (mL/beat)

Stroke Volume

Stroke volume, as stated above, represents the volume of blood leaving the left ventricle with each heartbeat. With each heartbeat, the ventricles undergo a period of relaxation (diastole) and contraction (systole). Each phase is important in the generation of stroke volume. Determinants of stroke volume include ventricular preload, compliance, contractility and afterload.

Preload and Compliance

Preload describes the stretch of muscle fibers in the ventricle before contraction (systole). The amount of stretch determines the amount of tension the muscle fibers can generate. As the length (or stretch) of the muscle fibers increases, the tension generated by the fibers increases, thereby increasing stroke volume.

Factors that affect preload include the amount of blood returning to the heart from the body, which is a reflection of overall intravascular volume, and the compliance of the ventricle. A more compliant ventricle is capable of greater expansion—and hence filling—at a given pressure than a stiff ventricle. Preload may also be augmented by a reduced heart rate, which increases the time spent in diastole, thereby allowing a longer period for ventricular filling.

Contractility and Afterload

Ventricular contractility is a measure of the speed and force with which the ventricle contracts. As ventricular contractility increases, the speed of ejection of blood from the ventricle increases. This allows more time for ventricular filling, thereby increasing preload.

Afterload can be thought of as the force against which the ventricle must contract. As afterload increases, stroke volume decreases. A key determinant of left ventricular afterload is the resistance to blood flow in the systemic arteries, or the systemic vascular resistance (SVR), which is primarily determined by changes in arterial diameters. A decrease in arterial diameter (i.e., vasoconstriction) is associated with an increase in SVR, whereas an increase in arterial diameter (i.e., vasodilation) has the opposite effect. The

viscosity, or thickness, of the blood also affects SVR. Increased viscosity of the blood increases the resistance within systemic arteries by impeding blood flow. This mechanism may be particularly relevant during shock, in which the coagulation system may be activated and lead to the development of clots (i.e., thrombosis), thereby dramatically increasing blood viscosity.

Figure 7-4 summarizes the determinants of cardiac output and stroke volume.

Arterial Blood Pressure

Reciprocal changes in cardiac output and SVR maintain normal arterial blood pressure. Therefore, if cardiac output falls, the arteries constrict, thereby increasing SVR and preserving blood pressure.

Corresponding with the phases of the cardiac cycle, the arterial pressure generated during ventricular contraction is referred to as the systolic blood pressure, and the arterial pressure generated during ventricular relaxation is referred to as the diastolic blood pressure.

Normal blood pressure in children varies based on age, sex and height. Median blood pressure ranges for each age group are shown in Table 7-1. In addition, Table 7-2 provides a quick way to assess for hypotension in children and infants. If the child's systolic blood pressure is less than the number listed, then the child is hypotensive.

The difference between systolic and diastolic blood pressures is referred to as the pulse pressure (Figure 7-5). A normal pulse pressure is approximately

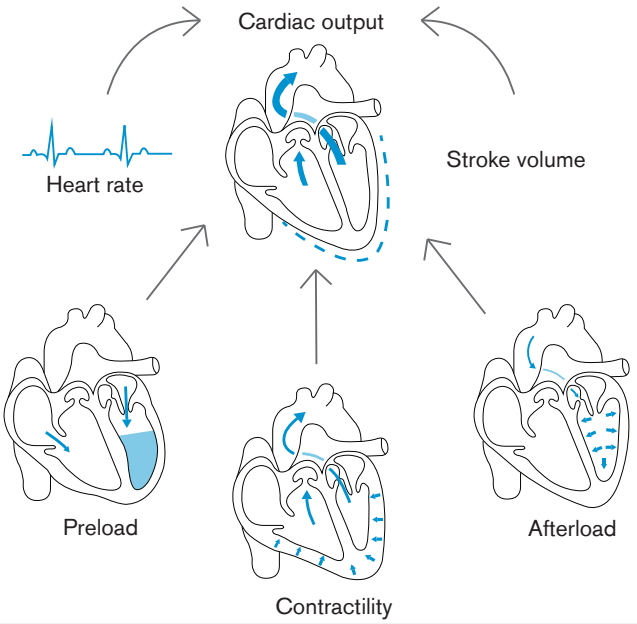


Figure 7-4 | Determinants of cardiac output

Table 7-1 | Normal Pediatric Blood Pressure

Age Group	Systolic Blood Pressure	Diastolic Blood Pressure
Newborn	60–85	35–55
Infant (1–12 mo)	70–100	35–60
Toddler (1–2 yrs)	85–105	40–65
Preschooler (3–5 yrs)	89–115	45–70
School Age (6–12 yrs)	94–120	55–80
Adolescent (13–17 yrs)	110–135	60–85

Table 7-2 | Quick Assessment for Hypotension

Age Group	Systolic Blood Pressure
Neonate	<60
Infant	<70
Toddler–School Age	<70 + (age in years x 2)
Adolescent	<90

40 mmHg. The pulse pressure may provide additional information beyond what systolic and diastolic blood pressure readings provide.

Oxygen Delivery and Oxygen Demand

An optimal balance normally exists between the amount of oxygen delivered to the tissues and the amount needed by the tissues.

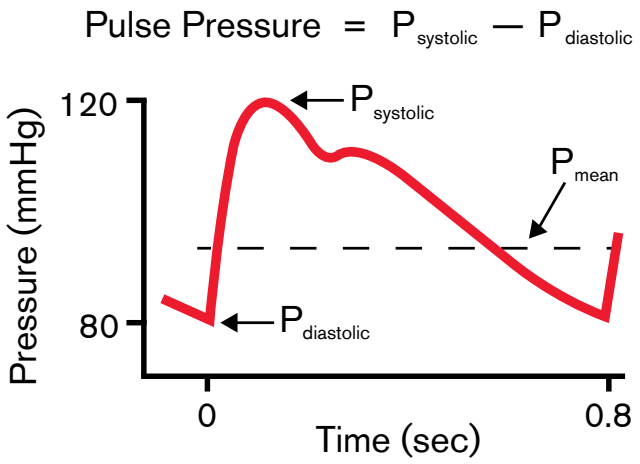


Figure 7-5 | Pulse pressure formula

Oxygen delivery is primarily determined by two factors: how much oxygen is contained in the blood (i.e., oxygen content) and how much of the blood is carried to the tissues, which is determined by cardiac output (Figure 7-6). The main determinants of oxygen content are the hemoglobin concentration and the percentage of hemoglobin that is saturated with oxygen (i.e., O_2 saturation). Regional oxygen delivery is further dependent on appropriate distribution of blood flow among the organs and tissues.

Physiology of Shock

Reduced Stroke Volume

Stroke volume influences the increase or decrease of cardiac output and thus the delivery of oxygen to vital organs and tissues. As previously noted, three variables determine stroke volume: preload, contractility and afterload. A number of medical conditions can affect stroke volume, impairing cardiac output and oxygen delivery and ultimately leading to shock.

Reduced Preload

An inadequate preload often underlies a low stroke volume and cardiac output in children in shock. Preload reflects the stretch of the muscle fibers of the ventricle before contraction, which is determined in part by the volume of blood at the end of diastole. Therefore, conditions that reduce intravascular volume, such as traumatic blood loss, gastrointestinal losses from severe vomiting and diarrhea, or accumulation of fluids in the interstitial space from vasodilation (i.e., third-spacing), can all affect preload. Because methods for assessing preload measure the pressure and not volume in the ventricles, accurate assessments of preload may be difficult to obtain in certain conditions that cause shock. These include conditions that affect the compliance of the right ventricle, such as pericardial tamponade, or conditions that increase intrathoracic pressure, such as tension pneumothorax.

As previously indicated, preload is clinically assessed by indirectly measuring the pressure in the ventricles. Common bedside methods measure either the central venous pressure (CVP), which measures the pressure in the right atrium and vena cava and can be used to estimate the right ventricular preload, or the pulmonary capillary wedge pressure, which normally reflects the pressure in the left ventricle.

Poor Contractility

Poor contractility may also contribute to decreased stroke volume and hence decreased cardiac output and oxygen delivery in shock. Myocardial dysfunction can result from conditions directly affecting the heart or metabolic imbalances, such as acidosis or electrolyte abnormalities.

As previously noted, children have a reduced ability to augment stroke volume as a means of increasing cardiac output. Specifically, the ability to increase preload and contractility is limited in children.

Increased Afterload

Increased afterload may be a cause or consequence of shock in children and infants. Congenital abnormalities such as coarctation of the aorta may increase afterload,

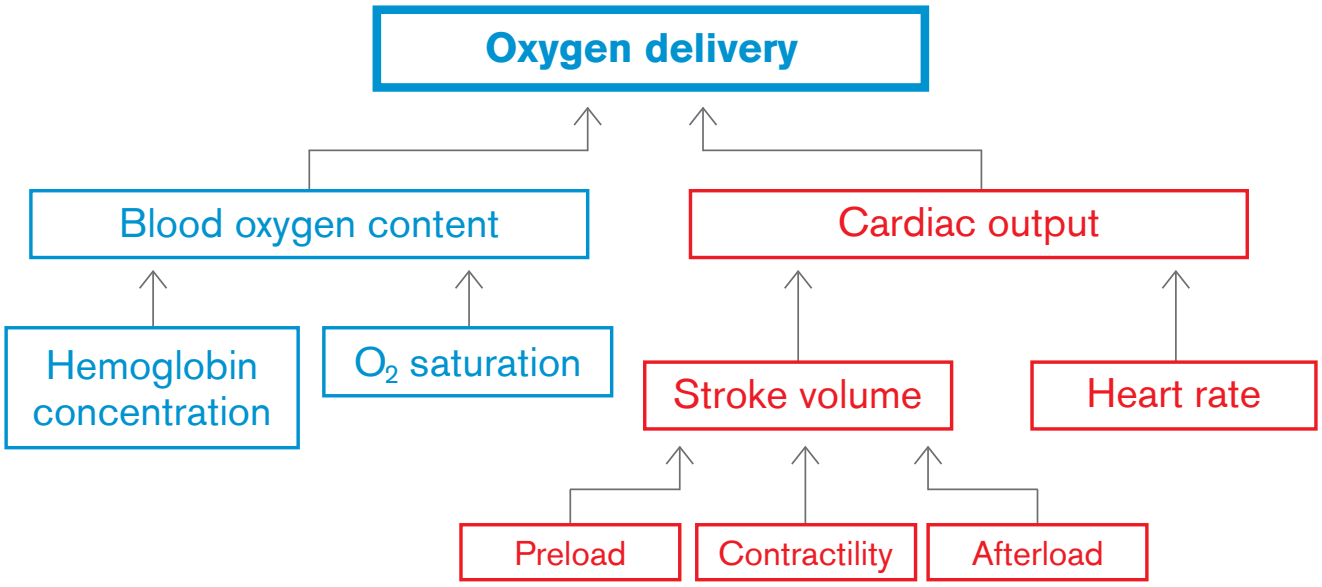


Figure 7-6 | Components of oxygen delivery

ultimately reducing stroke volume and cardiac output and causing shock. Conversely, increased afterload may be a consequence of shock, resulting from peripheral vasoconstriction that occurs to preserve blood pressure and blood flow to vital organs. Even in this compensatory state, therapeutically reducing afterload may be beneficial to reduce the workload of the heart in shock.

Table 7-3 summarizes the typical changes in stroke volume variables observed in the four types of shock.

Other Compensatory Changes in Shock

In children, tachycardia is the body's first response to maintain oxygen delivery to vital organs and tissues. Peripheral vasoconstriction is another early response to decreasing cardiac output during shock. Peripheral vasoconstriction results in redirection of blood flow to vital organs, such as the brain and the heart, through constriction of select vascular beds; it also results in an increase in SVR. As in the cardiac output equation, blood pressure and SVR also share a relation in peripheral vasoconstriction. An increase in SVR via peripheral vasoconstriction can maintain blood pressure as cardiac output decreases.

Blood Pressure in Shock

As previously noted, pulse pressure may provide additional information—beyond the systolic and diastolic blood pressure readings—regarding the physiology underlying shock, thereby helping to guide therapy. A decreased or "narrow" pulse pressure indicates decreased intravascular volume, decreased stroke volume or increased SVR. Upon clinical exam, patients with a narrow pulse pressure tend to have cooler extremities with weak, "thready" pulses. An increased or "wide" pulse pressure is seen with increased stroke volume, as occurs when the heart is in a hyperdynamic state (e.g., due to fever) or with decreased SVR. Patients with a widened pulse pressure generally have warm extremities with bounding peripheral pulses on clinical exam.

Table 7-4 summarizes the compensatory changes that are characteristic of shock. These potential changes provide insight into what you may encounter in your clinical assessment of a child or infant in shock.

Table 7-4 | Compensatory Changes in Shock

Change	Affected Anatomic Location	Clinical Effect
Increased heart rate	Heart	Tachycardia
Increased systemic vascular resistance	Circulation	Delayed capillary refill <2 seconds
	Extremities	Weak pulses; cool to touch; pale; narrow pulse pressure
Shunting or redistribution of blood away from less vital organs	Gastrointestinal tract	Nausea and vomiting; ileus
	Kidney	Decreased urine output

Abnormal Balance Between Oxygen Delivery and Oxygen Demand

Shock is characterized by an imbalance between oxygen delivery and oxygen demand that results in tissue hypoxia. When oxygen delivery is insufficient to meet the oxygen demand of the tissues (a state known as *oxygen debt*), the tissues become hypoxic. Tissue hypoxia adversely affects cellular metabolism. Cells resort to **anaerobic metabolism**, which results in the accumulation of lactic acid. This form of metabolism is much less efficient than normal **aerobic** cellular **metabolism** and does not support normal cellular functions for very long.

This imbalance most often results from diminished cardiac output. Both oxygen delivery and oxygen demand can be manipulated in an effort to "move the balance" to favor oxygen delivery (Table 7-5). Oxygen delivery can be augmented by optimizing its major components, the oxygen content of blood and cardiac output. On the other side of the balance, oxygen demand can be minimized by measures to reduce oxygen consumption in the setting of shock.

Table 7-3 | Application of Stroke Volume Variables to Shock Types

	Hypovolemia	Distributive	Cardiogenic	Obstructive
Preload	Decreased	Normal or decreased	Variable	Decreased
Contractility	Normal or increased	Variable	Decreased	Decreased
Afterload	Increased	Increased	Increased	Normal or increased

Table 7-5 | Strategies for Restoring Oxygen Delivery and Oxygen Consumption Balance

Increase O_2 Delivery	Decrease O_2 Demand
■ Increase cardiac output ■ Increase O_2 in blood ○ Correct hypoxemia ○ Correct anemia	■ Reduce work of breathing ■ Reduce body temperature ■ Provide sedation

Assessing the Pediatric Patient in Shock

A systematic approach and adherence to assessment, recognition and care is required when assessing a pediatric patient with shock.

Prompt assessment, recognition and care of a pediatric patient with impaired oxygenation and perfusion may prevent deterioration to decompensated, or late, shock and ultimately, cardiac arrest.

Rapid Assessment

First, perform a rapid assessment. This assessment starts with a quick visual survey of the emergency situation. Make sure that the environment is safe and formulate an initial impression of the child or infant experiencing an emergency. An initial impression allows you to quickly recognize whether the patient is experiencing a life-threatening condition, including bleeding, or a non-life-threatening one. During the visual survey, also quickly determine what additional resources you may need in the emergency situation.

Formulate an Initial Impression (PAT)

To form an initial impression of the patient, follow the Pediatric Assessment Triangle (PAT), which uses an A-B-C approach.

- **A**ppearance (TICLS): Assess appearance and responsiveness; observe muscle tone, interactivity (e.g., lethargic, fatigued), movement/gesturing, speaking or crying and demeanor (e.g., calm, anxious or irritable).
- Work of **B**reathing: Note work of breathing; check for patient positioning, audible breath sounds (normal or abnormal—e.g., stridor, wheezing or grunting) and signs of increased work of breathing or respiratory distress (e.g., nasal flaring, using accessory muscles to breathe, intercostal, substernal or suprasternal retractions and/or managing secretions), appears to be breathing too fast or too slow and signs of inadequate or absent respiratory effort.
- **C**irculation: Assess adequacy of circulation by assessing skin color and visible mucous membranes; check for pallor (or gray/dusky color), cyanosis, mottling or flushing and life-threatening bleeding.

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.

ALERT

If you observe that the child or infant is unresponsive during the appearance step of the PAT, check for responsiveness, breathing and pulse and provide immediate care as necessary. If you observe that the child or infant is responsive during the appearance step of the PAT, but you observe potential life-threatening airway, breathing or circulation compromise, provide immediate care as necessary before proceeding to the primary assessment. If the child or infant is responsive and is not experiencing life-threatening airway, breathing or circulation compromise, proceed directly to the primary assessment.

Primary Assessment

After completing a rapid assessment, conduct a primary assessment of the patient. This "hands-on" assessment enables you to collect physical and physiologic data to facilitate recognition of the underlying cause(s) of the patient's shock.

ALERT

As you perform the primary assessment, be alert for signs that the patient's condition has worsened or for any change in areas already assessed. Delegate necessary initial interventions to the appropriate team members so that immediate care can be implemented as you continue the primary assessment.

Practice Note

If during your assessment you note a petechial rash and/or purpura, take special precautions to isolate the patient and wear appropriate PPE. These findings may indicate sepsis.

Airway

- Assess airway to determine patency. Listen and feel for movement of air by placing your ear close to the patient's nose and mouth. Observe for the rise and fall of the chest and/or abdomen with each breath.
- Determine the following: Is the airway clear and open? Is the airway obstructed but can be kept open with simple manual interventions? Is the airway not maintainable and is the use of CPAP, non-invasive ventilation or an advanced airway required?
- Maintain a patent airway as appropriate based on assessment findings.

Breathing

Assess the child's or infant's breathing to determine adequacy of ventilation and oxygenation:

- Look for: Increased work of breathing (e.g. nasal flaring, using accessory muscles to breathe, intercostal, substernal or suprasternal retractions), respiratory distress, abnormal breath sounds (e.g., stridor, grunting, wheezing, gurgling), absent breath sounds and management of secretions.
- Note respiratory rate, depth and rhythm (Table 7-6).
- Auscultate breath sounds (e.g., stridor, grunting, wheezing, crackles).
- Note voice or cry changes (e.g., hoarseness, hot potato voice).
- Measure oxygen saturation with pulse oximetry: Is the oxygen saturation reading normal or abnormal?
- Measure $ETCO_2$: For intubated patients, and when available in non-intubated patients, measure $ETCO_2$. Is the reading normal or abnormal?
- Prepare supplemental O_2 and provide supplemental O_2 as appropriate based on assessment findings.
- If necessary, support breathing by delivering ventilations with a BVM resuscitator.
- Implement noninvasive or invasive ventilation as necessary.
- In the case of tension pneumothorax, perform immediate needle thoracentesis.

Circulation

- Assess the child's or infant's circulation to determine adequate perfusion of tissues.
- Palpate central and peripheral pulses.
- Measure the child's or infant's blood pressure (see Table 7-6).
- Connect the child or infant to a cardiac monitor to assess heart rate and rhythm.
- Note skin and mucous membrane color, skin temperature, capillary refill time.
- Prepare to achieve vascular access based on assessment findings.
- Prepare for fluid or medication therapy, if indicated.
- Prepare for electrical therapy if indicated.
- Monitor urine output (as appropriate).

Disability

- Assess neurological status to determine adequate brain perfusion.
- Check level of consciousness, pupillary response (PERRL) and blood glucose level.
- Use the following tools: AVPU, GCS and TICLS.

Exposure

- Assess the body overall, focusing on one area at a time.
- Look for: Abrasions, burns, bleeding, contusions (bruising), crepitus deformities, fractures, instability, lacerations, penetrations, petechiae and/or purpura, rashes, tenderness and abnormal skin temperature and color (to assess circulation and perfusion).
- Obtain the patient's weight and body temperature if not already done/available.
- If a head, neck, spinal or pelvic injury is suspected in the patient, consider spinal motion restriction.

Secondary Assessment

After completing the primary assessment, if the patient's condition remains stable, perform a secondary assessment. This focused and detailed assessment will likely center around the circulatory system and its effects on the patient's vital functions. The secondary assessment also inlucdes a focused patient history and laboratory and diagnostic tests.

Table 7-6 | Normal Pediatric Vital Signs

Age Group	Respiratory Rate	Awake Heart Rate	Systolic Blood Pressure	Diastolic Blood Pressure
Newborn	30 to 60	100 to 200	60 to 85	35 to 55
Infant (1 to 12 mo)	30 to 50	100 to 180	70 to 100	35 to 60
Toddler (1 to 2 yrs)	24 to 40	90 to 140	85 to 105	40 to 65
Preschooler (3 to 5 yrs)	20 to 30	80 to 130	89 to 115	45 to 70
School Age (6 to 12 yrs)	16 to 26	70 to 120	94 to 120	55 to 80
Adolescent (13 to 17 yrs)	12 to 20	60 to 100	110 to 135	60 to 85

The secondary assessment can be used to help further define the type of shock and etiology, to help evaluate response to treatment and to further guide management.

ALERT

Throughout the secondary assessment, it is important to keep the concept of assess, recognize and care in mind. Reassess the patient continuously for any changes in condition and assess the patient's response to any clinical interventions.

Focused History

Assess signs and symptoms, allergies, medications, past medical history, last intake/output and events (SAMPLE).

Focused Physical Assessment

- Complete a physical assessment of area of the concern as determined by information gathered from the initial impression, primary assessment and focused history.
- Observe for signs and symptoms of pain.

Laboratory and Diagnostic Tests

Determine specific tests and timing based on individual patient situation and status. Certain laboratory tests can be used to evaluate the balance between oxygen delivery and consumption.

Lactate levels can be used to assess the adequacy of oxygen delivery. Lactate accumulates in the blood when cells must resort to anaerobic metabolism because of insufficient oxygen delivery. Therefore, an elevated lactate level is a marker of insufficient oxygen delivery. Other laboratory and diagnostic tests can be used to evaluate individual components of oxygen delivery, including hemoglobin levels and blood oxygen content (PaO_2), and to evaluate the underlying causes of shock.

Common laboratory and diagnostic tests include:

- Blood gas (arterial and venous).
- CBC.
- Coagulation panel.
- Blood/sputum cultures.
- Metabolic panel.
- Liver and renal function.
- Chest radiograph.
- Echocardiogram.
- ECG.
- Possible other diagnostic tests to evaluate the underlying cause of shock (e.g., ultrasound and CT scan).

Recognizing Shock

Shock is defined as an acute state in which the circulatory system is unable to provide adequate oxygen and nutrients to meet the metabolic demands of the organs and tissues.

Hypotension ensues only when normal compensatory mechanisms have failed to maintain blood pressure and tissue perfusion.

It is important to note that the progression of shock accelerates as it goes from a compensated to a decompensated (hypotensive) state. Because children are able to maintain vascular tone and normal blood pressure until shock is quite advanced, the progression to cardiac arrest may occur quickly once decompensated shock develops (Figure 7-7).

> Hypotension is *not* a consistent feature of shock; in fact, it is often a late and ominous sign of shock in children.

Therefore, recognizing and caring for shock in its earliest, compensated form will prevent inevitable progression to decompensated shock (and cardiopulmonary failure and cardiac arrest) if left untreated.

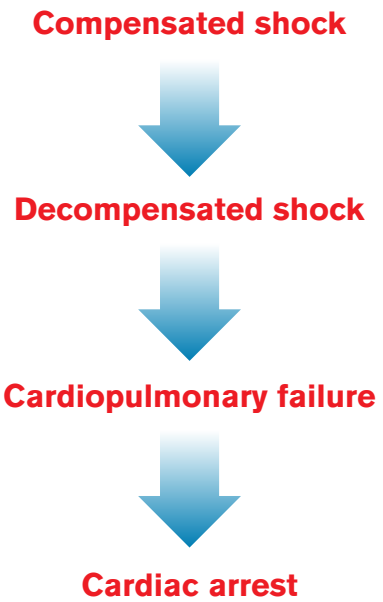


Figure 7-7 | The progression of shock accelerates as it goes from a compensated to a decompensated state.

Practice Note

Although hypotension is often regarded as a late and ominous finding in shock, in some instances hypotension may be an early finding in shock. This occurs in shock states that are initially characterized by decreased SVR (i.e., vasodilation), such as anaphylactic shock and neurogenic shock.

Compensated Shock

Compensated shock is typically identified by the lack of hypotension. In this earliest stage of shock, the sympathetic nervous system maintains blood pressure by releasing catecholamines such as epinephrine that increase the heart rate and constrict peripheral arteries. Peripheral vasoconstriction diverts blood from nonessential tissues to more vital organs, including the heart, brain and kidneys. It also allows children to maintain a normal blood pressure until their shock has reached an advanced state.

Signs of compensated shock may be subtle. Compensatory peripheral vasoconstriction typically results in such signs as delayed capillary refill and cool, mottled extremities. In early septic shock, however, the extremities may instead be warm and peripheral pulses may be bounding (so-called warm shock). Tachypnea may also be present; this represents a compensatory response to metabolic acidosis resulting from by-products of abnormal cellular metabolism. Urine output may be decreased as the body strives to conserve fluid. Children in compensated shock may also exhibit mild mental status changes, such as irritability or lethargy.

Decompensated Shock

In decompensated shock, the body exceeds its compensatory mechanisms. As a result, oxygen delivery to the tissues is no longer sufficient. Hypotension is a hallmark of decompensated shock. Other signs of impaired perfusion are present as well, including decreased or absent urine output, acidosis, and lethargy or coma. By this point, organ damage is evident. Shock becomes irreversible when damage to key organs cannot be undone even if adequate cardiovascular function is restored. Potential signs forewarning imminent cardiac arrest in a child in shock include hypotension, bradycardia and diminished central pulses.

Primary Assessment Findings Indicating the Presence and Severity of Shock

The presence and severity of shock are established during the rapid and primary assessment, as shown in Table 7-7. These findings apply to shock of any cause; although in some cases additional signs are used to identify the underlying cause/type of shock.

Recognizing the Types of Shock

Shock can occur through various physiologic mechanisms that form the basis for the classification of shock into four types: hypovolemic, distributive, cardiogenic and obstructive.

Hypovolemic Shock

Hypovolemic shock is the most common form of shock in children (Figure 7-8). The central physiologic abnormality in hypovolemic shock is a reduction of intravascular blood volume. This most commonly occurs as a result of actual fluid or blood loss from the body. Alternatively, when the integrity of capillary walls is disrupted in such conditions as burns or sepsis, fluid may "leak" from the intravascular space into surrounding tissues, resulting in relative hypovolemia. The primary mechanism of impaired oxygen delivery in hypovolemic shock is reduced cardiac output resulting from decreased preload and stroke volume.

Causes

The causes of hypovolemic shock can be broadly categorized based on the underlying mechanism leading to intravascular volume depletion: decreased intake of fluid, excessive loss of fluid or translocation of fluid from the intravascular space (Table 7-8). In children and infants, hypovolemic shock most commonly arises from gastrointestinal fluid loss due to diarrhea or vomiting. Diabetes mellitus is another common cause, resulting in excessive fluid losses through the kidneys.

Figure 7-8 | Hypovolemic shock is the most common form of shock in children.

Table 7-7 | Primary Assessment Findings in Shock

Assessment Component	Compensated Shock	Decompensated Shock
Airway	Typically unaffected unless by underlying cause (e.g., anaphylaxis causing airway edema)	Possible impairment of airway reflexes due to reduced level of consciousness
Breathing	Tachypnea	Rapid, shallow breathing, hypoxemia, respiratory failure
Circulation	■ Normal blood pressure for age, tachycardia, signs of cold shock (e.g., mildly delayed capillary refill, diminished peripheral pulses, narrow pulse pressure, cool, pale, mottled skin) or warm shock (e.g., flash capillary refill, bounding pulses, widened pulse pressure, warm, flushed skin), decreased urine output for age ■ Note: Key findings are tachycardia and signs of peripheral vasoconstriction (diminished peripheral pulses, skin changes, delayed capillary refill) but signs of peripheral vasoconstriction may be masked or altered in warm shock.	Hypotension, increasing tachycardia; delayed capillary refill; absent peripheral pulses, diminished central pulses; cool to cold, pale, mottled skin; decreased urine output for age or absent urine output; bradycardia (late)
Disability	Mild mental status changes (e.g., irritability)	Progressive mental status changes ranging from agitation and confusion to lethargy and coma
Exposure	Cool, mottled extremities (cold shock); warm, flushed extremities (warm shock)	Cold, mottled extremities; possible signs of impaired coagulation (e.g., bleeding from catheter sites, purpura, petechiae)

Children and infants are particularly vulnerable to the development of hypovolemia for several reasons. Fever and tachypnea, which often accompany pediatric illnesses, can contribute to hypovolemia by causing evaporative fluid losses (otherwise known as insensible losses). Infants are especially susceptible to hypovolemic shock because they have a relatively large amount of body water, a high metabolic rate and immature kidneys that are less able to retain fluid, and because they rely on others to meet their fluid intake needs.

Signs and Symptoms

Early signs of hypovolemic shock include those attributable to peripheral vasoconstriction (e.g., diminished peripheral pulses, delayed capillary refill, cool extremities). Other signs may include tachypnea and irritability. Pulse pressure may be decreased (narrow) owing to decreased stroke volume. Urine output is typically decreased; however, urine output may be falsely reassuring in the setting of conditions characterized by excess urine production, such

Table 7-8 | Causes of Hypovolemic Shock in Children

Underlying Mechanism	Examples
Decreased fluid intake	■ Conditions causing oropharyngeal pain (e.g., stomatitis, pharyngitis) ■ Anorexia or fluid deprivation
Fluid losses	■ Gastrointestinal losses: diarrhea, vomiting ■ Renal losses: diabetes mellitus, diabetes insipidus, adrenal insufficiency, diuretic usage ■ Hemorrhage
Translocation of intravascular fluid	■ Burns ■ Peritonitis ■ Sepsis ■ Anaphylaxis

as diabetes mellitus or diabetes insipidus. Other signs of hypovolemia include decreased tear production, dry mucous membranes, sunken eyes and a sunken **fontanelle** in infants. In hypovolemic shock due to hemorrhage, bleeding will be seen. If a patient has internal bleeding, pain and signs of trauma may be present.

Primary assessment findings in hypovolemic shock can vary significantly based on the extent of fluid loss. Children with acute fluid losses equaling up to 10 percent of their blood volume may have normal heart rates, distal pulses and capillary refill.

> Because compensatory mechanisms allow children to maintain a normal blood pressure until a substantial amount of fluid has been lost, hypotension should be viewed as a late and ominous sign in hypovolemic shock.

While the primary focus is recognizing shock as early as possible and intervening to prevent the patient from progressing to decompensated shock, it is also helpful to recognize when the patient may be progressing to decompensated shock. Decompensated shock occurs with progression from compensated but can also occur initially in severe hemorrhage and is a state of impaired perfusion that compensation methods cannot address, which can quickly progress to cardiac arrest. This is indicated by hypotension and associated symptoms of the impaired perfusion. Explore the following potential primary assessment findings that may indicate when a patient's compensatory mechanisms are no longer sufficient and the patient has progressed from compensated to decompensated hypovolemic shock.

Airway

Patent (compensated) to possible airway compromise due to altered level of consciousness (decompensated)

Breathing

Normal respiratory rate for age or tachypnea (compensated) to marked tachypnea and possible bradypnea to respiratory arrest (late) (decompensated)

Circulation

Normal systolic blood pressure for age (compensated) to hypotension (decompensated); normal heart rate for age or tachycardia (compensated) to marked tachycardia (and possible bradycardia [late] (decompensated); diminished peripheral pulses and delayed capillary refill (compensated) to absent peripheral pulses, weak central pulses, and very prolonged to absent capillary refill (decompensated); No changes or pale, cool skin (compensated) to cool or cold, pale, and mottled skin (decompensated) normal or decreased urine output for age (compensated) to decreased urine output for age or negligible urine output (decompensated)

Disability

No change in mental status or anxiety (compensated) to altered mental status or decreased level of consciousness (e.g., coma) (decompensated)

Exposure

No changes or pale, cool extremities (compensated) to cool or cold, pale, mottled extremities (decompensated)

Some have described hypovolemic shock as going through four stages. The stages of hypovolemic shock and corresponding primary assessment findings are shown in Table 7-9.

Distributive Shock

In distributive shock, there is an abnormal distribution of the intravascular volume resulting from inappropriate vasodilation. This vasodilation effectively enlarges the entire vascular space. As a result, venous return to the heart is diminished, thereby decreasing preload and, ultimately, cardiac output. In some types of distributive shock, there may be associated capillary leak may further exacerbate the decrease in preload by causing a relative hypovolemia.

Distributive shock may be caused by drugs or conditions that reduce vasomotor tone. Septic shock, anaphylactic shock and neurogenic shock are all types of distributive shock.

Septic Shock

Septic shock is the final stage of a clinical continuum that begins with sepsis (Figure 7-9). Sepsis is a systemic response to a known or suspected infection. The systemic response in sepsis is clinically characterized by tachycardia, tachypnea, a high or low body temperature and a high white blood cell count. Recognition and aggressive treatment of sepsis in this earliest stage may prevent progression to septic shock.

Table 7-9 | Stages of Hypovolemic Shock and Corresponding Primary Assessment Findings

Stage	Blood Volume Lost (%)	Blood Pressure	Capillary Refill	Primary Assessment Findings	
1	≤15%	Maintained	Normal	**A:** airway patent **B:** normal respiratory rate **C:** normal heart rate, blood pressure and urine output for age **D:** no changes **E:** no changes	Compensated
2	15%–25%	Systolic maintained, diastolic decreased	Delayed	**A:** airway patent **B:** tachypnea **C:** tachycardia; decreased urine output for age, cool, pale skin, diaphoresis **D:** anxiety **E:** cool, pale skin; diaphoresis	
3	25%–40%	Systolic decreased	Delayed	**A:** possible airway compromise due to altered level of consciousness **B:** tachypnea **C:** tachycardia, decreased urine output for age, cool, pale mottled skin **D:** altered mental state **E:** cool, pale, mottled skin	Decompensated (early)
4	>40%	Systolic significantly decreased	Absent	**A:** possible airway compromise due to altered level of consciousness **B:** marked tachypnea **C:** marked tachycardia, weak pulse, negligible urine output **D:** decreased level of consciousness (e.g., coma) **E:** cool, pale, mottled skin	Decompensated (late—may become irreversible if sustained)

Sepsis is considered severe when evidence indicates organ dysfunction or inadequate tissue perfusion.

Septic shock is defined as severe shock with hypotension that cannot be reversed with fluid resuscitation only. Although considered a form of distributive shock, septic shock often incorporates physiologic elements that are characteristic of other types of shock as well—namely, absolute or relative hypovolemia, as in hypovolemic shock, and impaired cardiac function, as in cardiogenic shock.

Figure 7-9 | Septic shock is the final stage of a continuum that begins with sepsis.

Most children in septic shock present with cold shock (i.e., peripheral vasoconstriction; narrow pulse pressure; delayed capillary refill; cool, mottled extremities) instead of warm shock, which is the predominant presentation in adults. Most often, cold shock predictably correlates with vasoconstriction, but some children presenting with cold septic shock are actually vasodilated. Children in septic shock may also transition from warm shock to cold shock as their condition worsens.

Multiple organ systems may be affected in septic shock. Common manifestations of organ dysfunction in septic shock include an altered mental status, acute lung injury, kidney or liver dysfunction and impaired **coagulation**.

> Although it may occur with any type of shock, multiple organs are typically affected in septic shock.

Coagulation abnormalities may clinically manifest with a petechial or purpuric rash, particularly in septic shock caused by certain organisms (e.g., Neisseria meningitidis). The adrenal glands are also susceptible to injury caused by impaired perfusion or hemorrhage, especially in the setting of severe sepsis with coagulation abnormalities. Such injury may impair the ability of the adrenal glands to secrete cortisol as they normally would in response to stress, further contributing to hemodynamic dysfunction in septic shock.

ALERT

Petechiae or purpura may indicate a serious life-threatening systemic infection. Follow your institutional protocols regarding PPE and appropriately isolating the patient.

Potential primary assessment findings in septic shock are summarized in Table 7-10.

The diagnostic criteria for sepsis include a number of specific hematologic and biochemical abnormalities. The secondary assessment of a child with suspected septic shock should include laboratory testing to evaluate for these abnormalities (Table 7-11).

Anaphylactic Shock

Anaphylactic shock represents a systemic reaction to an allergic stimulus such as a food, medication or bee sting. This reaction is mediated by the massive release of **cytokines**, which causes a rapid loss of vascular tone and third-spacing of intravascular volume. Correlating cardiovascular compromise clinical signs may include tachycardia and hypotension. Anaphylactic shock is often complicated by respiratory distress due to **angioedema** of the airway, face, lips and tongue.

Angioedema involving the upper airway may manifest with stridor or hoarseness and may progress to the point that an advanced airway is needed. Angioedema of the lower airways may manifest with wheezing and, if progressive, may lead to respiratory arrest. Skin manifestations such as hives or flushing are seen in most children with anaphylaxis, but their absence does not exclude a diagnosis of anaphylaxis. Gastrointestinal manifestations such as nausea, vomiting, diarrhea and abdominal pain may also be present. Mental status changes may be evident as well, ranging from fussiness and irritability to somnolence.

Potential primary assessment findings in anaphylactic shock are summarized in Table 7-12.

Diagnostic criteria for anaphylactic shock:

- Cutaneous and mucosal findings and one of the following:
  - Respiratory compromise
  - Hypotension and signs of end-organ dysfunction

OR

- Two or more of the following that occur rapidly post-exposure to an allergen:
  - Cutaneous and mucosal findings
  - Respiratory compromise
  - Hypotension and signs of end-organ dysfunction
  - Gastrointestinal symptoms

OR

- Hypotension post-exposure to an allergen

Table 7-10 | Primary Assessment Findings in Septic Shock

Assessment Component	Possible Findings
Airway	Patent unless level of consciousness impaired
Breathing	Tachypnea, **hyperpnea**, increased work of breathing, hypoxemia
Circulation	Tachycardia or bradycardia; diminished (cold shock) or bounding (warm shock) pulses; delayed (cold shock) or brisk (warm shock) capillary refill; normal or reduced blood pressure; narrow pulse pressure and pale, cool, mottled skin (cold shock); wide pulse pressure and warm, flushed skin (warm shock); decreased urine output for age
Disability	Mental status is normal or altered (e.g., restlessness, agitation, anxiety, somnolence, coma)
Exposure	Body temperature is increased (>38° C for infants <3 months or >38.5° C for infants/children ≥3 months) or decreased (<36° C for all ages); cool, mottled extremities or warm, flushed extremities; petechiae and/or purpura

Table 7-11 | Potential Laboratory Findings in Septic Shock

Laboratory Test	Possible Findings
Complete blood count	Increased or decreased white blood cell count; increased immature white blood cell forms ("left shift"); low platelet count
Blood cultures	Presence of pathogens, such as bacteria, in the blood
Blood gas	Metabolic acidosis; respiratory alkalosis; hypoxemia
Coagulation panel	Prolonged **prothrombin time (PT)** or **activated partial thromboplastin time (aPTT)**
Renal function tests	Increased creatinine level
Liver function panel	Increased total bilirubin level
Lactate level	Increased lactate level

Neurogenic Shock

Neurogenic shock is a rare and usually transient condition that follows an acute spinal cord injury. Vasodilation results from disruption of the sympathetic pathways within the spinal cord, which normally maintain vascular tone. Cardiac output may be preserved early on, leading to isolated hypotension *with a wide pulse pressure* in the absence of tachycardia. The hypotension that accompanies neurogenic shock may further exacerbate the underlying spinal cord injury by reducing blood flow to the spinal cord. Relative bradycardia is another classic sign of neurogenic shock. It may be exacerbated by hypoxemia or such interventions as suctioning or turning the patient. Additional signs of neurogenic shock may include hypothermia and warm, flushed skin. Depending on the level of the spine at which the injury occurred, patients may also present with signs of respiratory muscle compromise, including apnea, rapid and shallow breathing, a weak cough and diaphragmatic breathing with paradoxical chest wall movement.

Table 7-12 | Primary Assessment Findings in Anaphylactic Shock

Assessment Component	Possible Findings
Airway	Signs of angioedema (e.g., swollen lips, tongue or uvula), airway obstruction
Breathing	Dyspnea, increased work of breathing, stridor, hoarseness, wheezing
Circulation	Tachycardia, hypotension, signs of end-organ dysfuntion, flushing, pallor
Disability	Fussiness, irritability, pain/discomfort,* lethargy, somnolence
Exposure	Hives, flushing, itching, pallor, diaphoresis

*Potential sources of pain or discomfort include gastrointestinal signs and symptoms of anaphylaxis (e.g., nausea, vomiting, abdominal pain).

Potential primary assessment findings in neurogenic shock are summarized in Table 7-13.

Cardiogenic Shock

The central physiologic abnormality in cardiogenic shock is decreased cardiac output. In most cases, this decrease is due to impaired contractility of the heart, or "pump" failure. Certain types of congenital heart defects and heart rate or rhythm abnormalities may also underlie decreased cardiac output in cardiogenic shock.

Causes

Conditions that affect the heart muscle, such as **myocarditis** and **cardiomyopathy**, may cause cardiogenic shock via direct impairment of myocardial contractility. Congenital heart disease is the most common cause of cardiogenic shock in children—specifically, diseases that affect outflow from the left ventricle or aorta (e.g., critical coarctation or stenosis of the aorta) or the normal balance

Table 7-13 | Primary Assessment Findings in Neurogenic Shock

Assessment Component	Possible Findings
Airway	Patent unless level of consciousness is impaired (e.g., due to concurrent head injury or shock) or protective reflexes impaired
Breathing	Apnea, fast and shallow breathing, paradoxical chest wall movement, weak cough, increased airway secretions (all depend on the level of spinal cord injury)
Circulation	Hypotension in the absence of tachycardia (relative bradycardia); wide pulse pressure; warm, flushed skin
Disability	Altered level of consciousness if concurrent head injury or advanced shock, neurologic deficits concordant with level of spinal cord injury
Exposure	Hypothermia, warm and flushed skin

between the systemic and pulmonary circulations. See *Learn More: Congenital Heart Defects and Shock*.

In the case of arrhythmias, the underlying mechanism of cardiogenic shock differs depending on the arrhythmia type. In bradycardia, a low heart rate leads to low cardiac output, whereas in other arrhythmias, cardiac output is decreased because the time for cardiac filling is reduced. **Tachyarrhythmias** may further impair myocardial contractility by increasing the work of, and hence oxygen consumption by, the heart. Finally, cardiogenic shock is often a late manifestation of shock of any cause and may present in association with septic shock and suboptimally managed hypovolemic shock.

Signs and Symptoms

Many potential clinical manifestations of cardiogenic shock overlap with those of other types of shock, including the following:

- Altered mental status
- Tachycardia
- Tachypnea
- Cold extremities
- Weak pulses
- Decreased (narrow) pulse pressure
- Decreased urine output and hypotension

However, cardiogenic shock may be differentiated by such physical signs as liver enlargement (hepatomegaly), neck vein distension and swelling (edema) of the extremities.

Auscultation of the chest during a focused physical assessment may reveal a heart murmur or gallop rhythm. Pulmonary edema may also be present, as evidenced by tachypnea, retractions, nasal flaring, grunting or decreased O_2 saturation. Wheezing or "crackles" (rales) may be heard upon chest auscultation, although the latter are uncommon in infants and may suggest an associated pneumonia.

Table 7-14 | Primary Assessment Findings in Cardiogenic Shock

Assessment Component	Possible Findings
Airway	Patent unless impaired level of consciousness
Breathing	Tachypnea, retractions, nasal flaring, grunting, wheezing, crackles on lung auscultation, cyanosis
Circulation	Tachycardia or bradycardia; narrow pulse pressure; hypotension; diminished peripheral pulses; cold extremities; neck vein distention; arrhythmia; pale, cool, mottled skin; diaphoresis
Disability	Altered level of consciousness ranging from anxiety and restlessness to coma
Exposure	Peripheral edema; pale, mottled skin; cold extremities; diaphoresis

Potential primary assessment findings in cardiogenic shock are summarized in Table 7-14.

During the secondary assessment, focused history may reveal poor feeding or poor weight gain in the infant or a history of being less active or difficulty keeping up with peers in older children. A focused physical assessment

LEARN MORE

Congenital Heart Defects and Shock

Infants with congenital heart defects that affect outflow from the left ventricle and aorta may initially present with obstructive shock. In infants with such defects, a patent ductus arteriosus—a fetal remnant that connects the pulmonary artery and aorta—maintains the systemic circulation after birth. This connection allows some of the blood coming from the right ventricle to enter the aorta and thereby the systemic circulation, bypassing the obstruction in the left ventricle or aorta.

When the ductus arteriosus closes, outflow of blood to the systemic circulation is abruptly jeopardized, initially predisposing to obstructive shock. The ventricles may eventually begin to fail, potentially contributing to cardiogenic shock as well. As such, congenital defects affecting left ventricular or aortic outflow may underlie either obstructive or cardiogenic shock and should be considered in infants presenting with either type of shock in the first month of life.

findings may include a heart murmur or gallop rhythm on heart auscultation, and hepatomegaly. Chest radiography may be helpful in distinguishing cardiogenic shock from other types of shock by demonstrating the presence of an enlarged heart and pulmonary edema.

Obstructive Shock

Obstructive shock rarely occurs in children. Conditions that cause obstructive shock generally impede either outflow of blood from the heart or aorta or return of blood to the heart despite normal intravascular volume.

Causes

Obstructive shock may be caused by conditions affecting both the heart and lungs. Causes of obstructive shock in children include cardiac tamponade, tension pneumothorax, pulmonary embolism and congenital heart defects. See *Learn More: Congenital Heart Defects and Shock*.

Signs and Symptoms

The distinguishing signs and symptoms of each of the major causes of obstructive shock in children and are outlined in Table 7-15. Prompt recognition of the underlying cause of obstructive shock is critical, as most of the causative conditions require specific medical or surgical treatment beyond normal shock management.

Caring for the Pediatric Patient in Shock

Irrespective of the cause, the primary goal in circulatory shock is to stop its progression and restore metabolic homeostasis to adequately meet the body's oxygen needs. Strategies for accomplishing this include fluid resuscitation, administration of medication therapies and correction of metabolic abnormalities, all with the intent of augmenting oxygen delivery to the tissues (Figure 7-10).

Monitoring the Child or Infant in Shock

As with all critically ill children and infants, immediately place all children or infants in shock on a cardiac monitor to continuously assess heart rate and rhythm, assess their central and peripheral pulses and capillary refill and place on a pulse oximeter to continuously measure O_2 saturation. Measure blood pressure immediately and noninvasively, and remeasure at frequent, regular

Figure 7-10 | Strategies for stopping the progression of shock include fluid resuscitation, administration of medication therapies and correction of metabolic abnormalities.

intervals. Assess perfusion, mental status and body temperature as part of the primary assessment and continue throughout the secondary assessment.

While not initial steps in the emergent management of shock, consider placing an indwelling urinary catheter to facilitate ongoing assessment of urine output. In addition, placing a central venous catheter to continuously measure CVP and oxygen saturation in the superior vena cava ($ScvO_2$) can help with management in shock and is used to help guide medication choices. Consider ketamine for sedation when placing a central venous catheter.

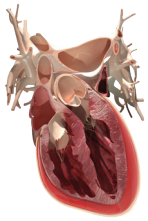
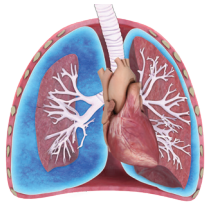
Ideally, place an indwelling arterial catheter to allow for continuous blood pressure measurement.

Initial parameters for assessing progression of shock and response to therapy include heart rate, blood pressure, capillary refill, peripheral pulses, skin temperature, urine output and mental status (Table 7-16). A normalizing heart rate is the earliest indicator of shock reversal. In addition, biochemical analyses such as serum pH, lactate and $ScvO_2$ may be used as indicators of tissue perfusion. Continually monitor and reassess children and infants in shock given the potential for rapid deterioration.

Balancing Oxygen Delivery and Demand

The primary therapeutic goal in shock, regardless of cause, is to restore a favorable balance between tissue perfusion

Table 7-15 | Major Causes of Obstructive Shock in Children: Underlying Pathophysiology and Primary and Secondary Assessment Findings

Causative Condition	Description	Physiologic Factors Underlying Shock	Signs and Symptoms
Cardiac tamponade 	Accumulation of fluid in the space between the heart and the sac that surrounds it (pericardium) as a result of infection, cancer, trauma or heart surgery	Accumulating pericardial fluid constricts the heart, resulting in reduced preload and ventricular filling	**Primary assessment:** **A:** patent unless impaired level of consciousness **B:** tachypnea, dyspnea **C:** Beck's triad (hypotension, muffled heart sounds, neck vein distention), tachycardia, diminished pulses, narrowed pulse pressure, pulsus paradoxus, pericardial rub, cold extremities **D:** altered level of consciousness **E:** cold extremities, peripheral edema **Secondary assessment:** ■ Possible electrical alternans on electrocardiogram (alternating, varying amplitude of QRS complexes) ■ Cardiomegaly, "water bottle"–shaped cardiac silhouette or pleural effusions on chest radiograph ■ Pericardial effusion and collapse of the right ventricle during diastole on echocardiogram
Tension pneumothorax 	Accumulation of air in the space between the lung and the outer lining of the lung (pleura) as a result of trauma or certain lung diseases	Increasing pleural air and pressure lead to mechanical compromise of central venous structures and reduced venous return to the heart	**Primary assessment:** **A:** tracheal deviation to unaffected side **B:** **dyspnea**, chest pain upon inspiration, decreased breath sounds and hyperresonance to percussion on affected side **C:** tachycardia, hypotension, neck vein distention, pulsus paradoxus, cold extremities **D:** altered level of consciousness **E:** cold extremities **Secondary assessment:** ■ Chest radiography to confirm and localize tension pnuemothorax. Will reveal air in the pleural cavity, with contralateral deviation of mediastinal structures.

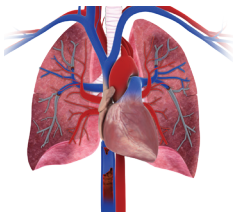
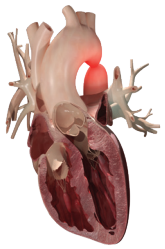
Causative Condition	Description	Physiologic Factors Underlying Shock	Signs and Symptoms
Pulmonary embolism 	Occlusion of one or more pulmonary arteries by clots **embolus** that originate elsewhere, usually in the large veins of the legs or pelvis, in the presence of certain risk factors (e.g., indwelling central venous catheter, recent surgery or trauma, clotting disorder)	Clot(s) in the pulmonary circulation increase afterload on the right ventricle, resulting in right ventricular dilation, bowing of the intraventricular septum into the left ventricle and reduced left ventricular filling	**Primary assessment:** **A:** patent unless impaired level of consciousness **B:** tachypnea, dyspnea, pleuritic chest pain, coughing up blood (hemoptysis) **C:** tachycardia, hypotension, signs of acute right heart failure (e.g., distended neck veins, peripheral edema, ascites, hepatomegaly), arrhythmias (most often sinus tachycardia), cold extremities **D:** altered level of consciousness, anxiety **E:** cold extremities, signs of deep vein thrombosis (e.g., swelling, pain or erythema) most often in lower extremities **Secondary assessment:** ■ Focused assessment findings: hepatomegaly ■ Laboratory test findings: elevated **D-dimer**, increased arterial-alveolar oxygen gradient on blood gas ■ Electrocardiogram findings: ST segment changes, right **bundle branch block** or right axis deviation
Obstructive cardiac and aortic lesions 	Lesions obstructing outflow from the left side of the heart or aorta	Obstructed outflow from left ventricle or aorta compromises systemic circulation; may occur suddenly with closure of the ductus arteriosus in infants	**Primary assessment:** **A:** patent unless impaired level of consciousness **B:** tachypnea, irregular breathing, respiratory distress, cyanosis (may or may not be present, depending on the lesion) **C:** signs of cold shock; hypotension; diminished or absent femoral pulses (if aortic lesion); differential blood pressure, O_2 saturation, or pulse between upper and lower extremities or between the right upper extremity and all other extremities (i.e., left upper extremity and lower extremities), cold extremities **D:** irritability, change in level of consciousness (may occur rapidly) **E:** cold extremities, diaphoresis, low weight for age **Secondary assessment:** ■ Focused physical assessment: heart murmur (though lack of heart murmur does not exclude congenital heart defect), hepatomegaly ■ Electrocardiogram: obstructive cardiac or aortic lesion

Table 7-16 | Initial Parameters and Therapeutic End Points for Monitoring Shock

Parameter	Target End Point
Heart rate	Normal heart rate for age
Blood pressure	Normal blood pressure for age
Capillary refill	Normal (≤2 seconds)
Peripheral pulses	Normal, with no differential between peripheral and central pulses
Peripheral skin temperature	Warm extremities
Urine output	1.5–2 mL/kg/hr for infants and young children and 1 mL/kg/hr for adolescents
Mental status	Normal

and metabolic demand with a focus on oxygen delivery and oxygen demand. Several common therapies and strategies may be used for this purpose (Table 7-17).

Optimizing Oxygen Delivery

Optimization of blood oxygen content is a key component of improving oxygen delivery. This is best accomplished by optimizing both the hemoglobin concentration and the percentage of hemoglobin saturated with oxygen (i.e., O_2 saturation). Normalization or optimization of cardiac output is another critical component of improving oxygen delivery.

Minimizing Oxygen Demand

Another strategy for improving the balance between oxygen delivery and demand in shock is to minimize oxygen demand, when possible. Potential measures for achieving this objective include fever and pain control, sedation, noninvasive positive airway pressure and mechanical ventilation after intubation. These and others are summarized in Table 7-18.

Of note, evidence from animals and humans suggests that controlling the work of breathing through the use of mechanical ventilation improves both blood flow to vital organs and tissue hypoxia in the setting of shock.

Initiating Fluid Therapy

Early fluid resuscitation is the cornerstone of therapy in shock. Regardless of the underlying cause, most children in shock have absolute or relative hypovolemia, which decreases preload, stroke volume and cardiac output. Accordingly, establishing rapid intravascular access to administer fluid is a priority in the child in shock. If an intravenous (IV) catheter cannot be placed establish intraosseous (IO) access immediately.

Managing Fluid Resuscitation

Once access has been obtained, fluid resuscitation can begin. An isotonic crystalloid solution such as normal saline (NS) or lactated Ringer's (LR) is typically used for fluid resuscitation in shock. In general, a volume of 20 mL/kg should be administered as quickly as possible. However, there are exceptions to this based on the type of shock that the child or infant is experiencing.

Children and infants in septic shock may require up to 60 mL/kg or more of fluid in the first 30 to 60 minutes of resuscitation. Children and infants in cardiogenic shock should receive smaller fluid boluses (5 to 10 mL/kg), and boluses should be given more slowly (over 10 to 20 minutes).

Table 7-17 | Goals and Strategies for Optimizing Oxygen Delivery and Demand

Goal	Potential Strategies	When to Use
Increase blood oxygen content	Optimize oxygenation	Always
	Hemoglobin level	As clinically indicated. Recent literature has suggested a more conservative strategy for transfusion, even in the setting of shock
Increase cardiac output	Increase heart rate	Bradycardia
	Increase stroke volume ■ Increase preload ■ Increase contractility ■ Decrease afterload	■ Absolute or relative hypovolemia ■ Cardiac dysfunction ■ Increased systemic vascular resistance
Decrease oxygen consumption	Initiate noninvasive positive airway pressure or mechanical ventilation to reduce work of breathing; control fever	As indicated based on work of breathing and severity of shock

Table 7-18 | Methods for Improving the Balance Between Oxygen Delivery and Demand in Shock

Increase Blood Oxygen Content	Decrease Oxygen Demand
■ Provide 100% oxygen by a non-rebreather face mask if child is spontaneously breathing ■ Initiate assisted (invasive or noninvasive) ventilation, as needed, for persistent hypoxemia ■ Consider packed red blood cell (PRBC) transfusion as clinically indicated	■ Consider mechanical ventilation ■ Treat fever, when present ■ Provide analgesics for pain ■ Treat or prevent agitation using sedation ■ Consider sedation and neuromuscular blockade, as clinically indicated ■ Treat seizures, when present

Fluid boluses should be repeated as needed until hypotension is reversed and perfusion improves, as evidenced by increasing urine output and normalizing capillary refill, peripheral pulses and mental status.

ALERT

Always assess the child or infant after each fluid bolus and discontinue fluid administration if clinical signs or symptoms of hypervolemia develop (e.g., hepatomegaly, crackles on chest auscultation).

Administering Blood Products

Packed red blood cells (PRBCs) or whole blood may be indicated in combination with isotonic crystalloids for volume resuscitation in hemorrhagic shock.

Children with hemorrhagic shock should receive PRBCs (10 mL/kg) or whole blood (10 to 20 mL/kg) if shock persists after two or three initial crystalloid boluses. Type O-negative PRBCs or whole blood should be used if immediate transfusion is warranted (e.g., for hypotension) and there is insufficient time for type- and cross-matching.

If large volumes of PRBCs or whole blood are infused, care must be taken to avoid hypothermia, which may cause arrhythmias or even cardiac arrest. To prevent this complication, blood may be gently warmed as it is infused using an IV set with a heat-exchange device.

Because the anticoagulant used in PRBCs (citrate) can bind calcium, hypocalcemia may occur with massive PRBC transfusion. Hyperkalemia may also occur with the transfusion of PRBCs stored for more than 1 week or if mechanical **hemolysis** occurs during transfusion. The presence of hypothermia may amplify the risk for hypocalcemia and hyperkalemia.

In addition to being used to immediately expand volume in patients with hemorrhagic shock, administration of PRBCs may be considered, as clinically indicated, to optimize hemoglobin concentration and hence oxygen delivery in shock.

In some cases, other blood products in addition to PRBCs may be needed in children in shock (Table 7-19).

See Table 3-4 in Chapter 3, Tools and Therapies, for more information regarding fluid and blood therapies used in pediatric emergencies.

Initiating Medication Therapy

Children who remain in shock despite adequate fluid resuscitation or who cannot tolerate ongoing aggressive fluid therapy (e.g., those in cardiogenic shock) are candidates for other supportive (namely, vasoactive) therapies.

Vasoactive therapies may not be needed in cases of shock in which the underlying cause is promptly reversed (e.g., restoration of intravascular volume in hypovolemic shock, decompression of a tension pneumothorax in obstructive shock). When needed, vasoactive therapies should be tailored to treat the physiologic abnormalities underlying shock in any given patient.

Table 7-19 | Other Blood Products Used in Shock

Blood Product	Indications
Plasma therapies	■ Correction of coagulopathy in thrombotic purpura disorders (e.g., disseminated intravascular coagulation) ■ Replacement of clotting factors following massive (75–80 mL/kg) PRBC volume replacement
Platelets	■ Platelet count in children with severe shock: ○ ≤10,000/mm^3 if no apparent bleeding ○ ≤20,000/mm^3 if significant risk for bleeding ○ <50,000/mm^3 if actively bleeding or before surgery or other invasive procedure ■ Replacement of clotting factors following massive (75–80 mL/kg) PRBC volume replacement

Practice Note

Alternative diagnoses (e.g., septic or distributive shock) should be considered when shock persists despite fluid resuscitation in a patient with presumed hypovolemic shock.

Depending on the patient, treatment may aim to increase SVR, increase cardiac contractility, reduce afterload or any combination thereof. Individual vasoactive medications have variable effects on these parameters. Depending on the agent, these effects are mediated by alpha, $beta_2$ and dopaminergic receptors in the vasculature; $beta_1$ receptors in the heart; and other molecules that affect cardiovascular function (i.e., phosphodiesterase and nitric oxide).

Drugs that act on alpha receptors can be used to affect vasoconstriction. Drugs that act on $beta_1$ receptors or inhibit the enzyme phosphodiesterase increase cardiac contractility (inotropy). Drugs that act on $beta_2$ receptors inhibit phosphodiesterase or release nitric oxide, promoting vasodilation. Table 7-20 shows the desired clinical effects of medications commonly used in pediatric shock and the molecular targets mediating those effects.

Options for treating shock that persists after fluid resuscitation include dopamine, epinephrine and norepinephrine. The hemodynamic effects of dopamine and epinephrine are dose dependent. At lower doses, beta-adrenergic effects such as increased heart rate and increased contractility predominate. As the dose increases, the inotropic effects increase, but so do the alpha-adrenergic effects, resulting in vasoconstriction.

Norepinephrine has potent alpha-adrenergic effects and less pronounced beta-adrenergic effects, making it especially suitable when low SVR is the predominant or only hemodynamic abnormality underlying shock (i.e., warm shock). The pure alpha-agonist phenylephrine may also be used in the setting of isolated peripheral vasodilation with normal or increased cardiac output.

Inotropic agents such as dobutamine or milrinone may be considered for use in patients with cardiogenic shock, or with low cardiac output and high SVR as part of other types of shock. These agents do not increase ventricular afterload, which may be an added benefit in patients with poorly functioning hearts. Pure vasodilators such as nitroprusside may also be used to reduce afterload in patients with persistently elevated SVR and shock despite fluid resuscitation and inotropes.

Table 7-20 | Desired Cllinical Effects of Medications Used in Pediatric Shock

Desired Clinical Effect	Physiologic Effect	Target and Effect	Drugs
Increased blood pressure	■ Vasoconstriction	■ Alpha receptor agonist	■ Phenylephrine ■ Norepinephrine ■ Epinephrine ■ Vasopressin ■ Dopamine (high dose)
Increased cardiac output	■ Increased heart rate (chronotropy) ■ Increased cardiac contractility (inotropy)	■ Direct dilator ■ Phosphodiesterase inhibitors	■ Epinephrine ■ Milrinone ■ Dobutamine ■ Norepinephrine ■ Dopamine
Decreased afterload	■ Vasodilation	■ $Beta_2$ receptor agonist ■ Stimulate CGMP	■ Nitroglycerine ■ Nitroprusside ■ Milrinone
Increased urine output	■ Increased renal blood flow	■ Dopamine receptor agonist	■ Dopamine (low dose)
Increased cardiac output Decreased afterload	■ Increased contractility (inotropy) ■ Increased myocardial relaxation during diastole (lusitropy) ■ Peripheral vasodilation	■ Phosphodiesterase 3 inhibitor	■ Milrinone

Practice Note

Ideally, vasoactive agents should be administered through a central venous catheter. If a patient's clinical condition warrants more immediate treatment, however, these agents may be administered through an IO or even peripheral line. Consider ketamine for sedation when placing a central venous catheter.

A vasodilator medication, Prostaglandin E_1, is administered if an infant is suspected of having, or confirmed to have, a ductal-dependent cardiac lesion until ductal-dependent cardiac lesion is ruled out by echocardiogram (Table 7-21).

Practice Note

Prostaglandin E_1 is administered to prevent the ductus arteriosus from closing or to help restore ductal patency if it has already closed. Prostaglandin E_1 increases systemic blood flow and reduces the risk for onset of metabolic acidosis. Administration of prostaglandin E_1 is a temporary measure to manage the infant until they can be evaluated and undergo surgery.

See Table 3-5 in Chapter 3, Tools and Therapies, for a complete list of commonly used medications used in pediatric emergencies.

Table 7-21 | Medications Used in Shock

Class	Physiologic Effect	Applicable Agents and Dosing
Vasopressors	■ Increase SVR ■ Increase contractility (except vasopressin)	■ Epinephrine: 0.1 to1 mcg/kg/min IV/IO infusion, titrated to desired clinical effect ■ Norepinephrine: 0.1 to 2 mcg/kg/min IV/IO infusion, titrated upward as needed to desired clinical effect ■ Dopamine: 2 to 20 mcg/kg/min IV/IO infusion, titrated to desired clinical effect ■ Vasopressin: 0.0002 to 0.002 u/kg/min IV/IO infusion ■ Phenylephrine: 0.1 to 0.5 mcg/kg/min IV/IO infusion, titrated to desired clinical effect
Inotropes	■ Increase HR and cardiac contractility ■ SVR effect is dependent on agent and dosage ■ Has agents with both alpha- and beta-receptor binding	■ Epinephrine: 0.1 to 1 mcg/kg/min IV/IO infusion, titrated to desired clinical effect ■ Dobutamine: 2 to 20 mcg/kg/min IV/IO infusion, titrated to desired clinical effect ■ Dopamine: 2 to 20 mcg/kg/min IV/IO infusion, titrated to desired clinical effect
Inodilators	■ Decrease SVR ■ Improve coronary blood flow ■ Improve contractility	■ Milrinone: loading dose of 50 mcg/kg IV/IO over 10 to 60 minutes, followed by infusion of 0.25 to 0.75 mcg/kg/min
Vasodilators	■ Decrease SVR ■ Decrease venous tone	■ Nitroprusside: starting dose of 0.3 to 1 mcg/kg/min IV/IO; start at lowest possible dose and titrate to effect (maximum dose, 8 mcg/kg/min) ■ Nitroglycerin: ○ Infants and children: Initial dosing: 0.25 to 0.5 mcg/kg/min IV/IO infusion. Increase by 1 mcg/kg/min every 15 to 20 minutes, as tolerated, to desired effect. ○ Adolescents: Initial dosing: 5 to 10 mcg/min IV/IO infusion. Increase to maximum of 200 mcg/min, as tolerated, to desired effect.
Vasodilator	■ Maintain ductal patency	■ Prostaglandin E_1: Initial dose: 0.05 to 0.1 mcg/kg/min IV/IO infusion, titrating up to 0.1 mcg/kg/min as needed. Maintenance dose: 0.01 to 0.05 mcg/kg/min IV/IO infusion.

Managing Metabolic Abnormalities

Metabolic abnormalities, including hypoglycemia, hypocalcemia, hypokalemia, hyperkalemia and metabolic acidosis, are common in shock.

Managing Hypoglycemia

Hypoglycemia can be the result of inadequate glycogen stores, increased glucose requirements or other metabolic imbalances. Children and infants have limited glycogen stores, which may rapidly deplete during times of physiologic stress such as shock. Hypoglycemia is relative to age:

- Neonates: plasma glucose <45 mg/dL
- Infants, children and adolescents: plasma glucose <60 mg/dL

When caring for a child or infant with shock, providers should perform a rapid bedside glucose test (Figure 7-11). Replacement with IV dextrose may be necessary if the glucose level is low and if the patient is symptomatic—particularly if they are unable to take anything by mouth.

> Immediate treatment consists of a bolus of 10% or 25% dextrose in water, given slowly to avoid hyperglycemia and subsequent rebound hypoglycemia.

The bolus should be followed by a continuous dextrose infusion with frequent glucose checks to confirm that a sufficient glucose level is being maintained.

Figure 7-11 | Infants and children have limited glycogen stores, which may rapidly deplete during shock. Healthcare providers should perform a rapid bedside glucose test to assess for hypoglycemia.

Managing Hypocalcemia, Hypokalemia, Hyperkalemia and Metabolic Acidosis

Because calcium and potassium abnormalities can impair cardiac contractility, patients should be monitored and treated for significant abnormalities in these levels. Lactate levels may be elevated in shock and are often used to evaluate the adequacy of treatment.

Acidosis may also occur as a result of accumulation of other acids (e.g., sulfates and other acids in renal failure, ketone bodies in diabetic ketoacidosis [DKA]) or bicarbonate losses (e.g., due to diarrhea or renal tubular acidosis). Identification and treatment of acidosis are important to avoid the negative impact it may have on cardiac contractility. An elevated lactate level in the presence of acidosis indicates possible tissue hypoxia and warrants further investigation.

Metabolic abnormalities that are common in shock and their causes and treatments are shown in Table 7-22.

General Care for a Child or Infant in Compensated or Decompensated Shock

For a child who presents with shock, initial care includes completing the rapid, primary and secondary assessments as the patient's condition allows (Figure 7-12). As with all pediatric emergencies, immediate actions include supporting airway, breathing and circulation.

Start by positioning the patient. If the child or infant is stable, proper positioning is whatever is comfortable for them; by contrast, an unstable child or infant should be positioned supine. At this point, initiate basic life support measures:

- Assess the airway and assist ventilation as necessary (e.g., BVM ventilations).
- Provide supplemental oxygen.

Figure 7-12 | Immediate actions for the pediatric patient who presents in shock include supporting the airway, breathing and circulation.

Table 7-22 | Common Metabolic Abnormalities in Shock and Corresponding Treatments

Metabolic Abnormality	Possible Causes	Treatment
Hypocalcemia	■ PRBC transfusion (due to citrate) ■ Septic shock ■ Sodium bicarbonate	■ Administer calcium
Hypokalemia	■ Diuretics ■ Gastrointestinal losses (diarrhea or vomiting)	■ Administer potassium ■ Reassess fluid balance
Hyperkalemia	■ Renal insufficiency ■ Acute renal failure ■ Adrenal insufficiency ■ Diabetic ketoacidosis ■ PRBC transfusion	■ Correct acidosis ■ Administer calcium chloride or gluconate to stabilize the myocardium ■ Administer insulin and glucose or initiate dialysis to reduce blood potassium levels
Metabolic acidosis	■ Lactic acidosis ■ Diabetic ketoacidosis ■ Renal failure ■ Gastrointestinal or renal bicarbonate losses	■ Treat underlying cause ■ Reserve bicarbonate for severe acidosis (i.e., pH <7.15) if ventilation is sufficient or supported

- Obtain IV or IO access. If unable to obtain intravenous access, immediately establish intraosseous access, if clinically warranted. If possible, two large-bore IV (or IO) access points are best for hypovolemic shock.
- Establish cardiac monitoring, blood pressure and oxygen saturation, assess peripheral pulses and capillary refill, assess skin temperature and color, urine output, and monitor the patient's level of consciousness.
- Consider placing the following:
  - An indwelling arterial catheter to allow for continuous blood pressure measurement
  - An indwelling urinary catheter to facilitate ongoing assessment of urine output
  - A central venous catheter to continuously measure CVP and oxygen saturation in the superior vena cava ($ScvO_2$)

Assisted ventilation with BVM and ultimately an advanced airway may be required in children or infants with profoundly altered mental status, acute lung injury or respiratory failure. Intubation may also be undertaken to facilitate mechanical ventilation as a means to reduce both the work of breathing and oxygen consumption.

> Begin treatment to reverse the circulatory abnormalities underlying shock as soon as possible.

Most often this involves fluid resuscitation, because preload becomes the main determinant of cardiac output once compensatory mechanisms have been activated in shock.

Begin with crystalloid fluid (0.9% NS or LR) bolus at 20 mL/kg IV/IO. Repeat as needed until normovolemia is achieved or signs of hypervolemia develop (palpable liver edge or crackles in the lungs). Administer smaller (10-mL/kg) fluid bolus volumes in neonates. Also consider smaller (5 to 10-mL/kg) fluid bolus volumes in children with cardiogenic shock if hypovolemic or if you are unsure of their volume status. In most shock states after administration of 60 mL/kg of fluid, you should re-evaluate to determine if more fluid is needed, if medications should be added or if other etiologies (such as hemorrhage) need to be addressed.

For patients in hypovolemic shock due to hemorrhage, control the bleeding, administer fluids and replace blood loss with PRBCs or whole blood as indicated (see *Hypovolemic Shock* Treatment Guideline).

Medication therapy may be implemented early in certain subsets of patients, such as children presenting with decompensated or cardiogenic shock, and infants with suspected or confirmed ductal-dependent cardiac lesions.

Consider vasoactive therapy for fluid-refractory (or fluid-intolerant) shock. Choice of vasoactive therapy will depend on the desired physiologic effect(s). Note: Placement of a central venous catheter should be undertaken for administration of vasoactive drugs,

if possible, but doing so should not delay therapy. Consider ketamine for sedation when placing a central venous catheter.

> Begin prostaglandin E_1 infusion in any infant with a known or suspected ductal-dependent cardiac lesion (pending confirmation or exclusion by echocardiogram).

See Table 7-23 for unique but emergent interventions for specific circumstances that may occur in the patient with shock.

See *General Shock* Treatment Guideline for a summary of care.

Additional care for the child or infant in compensated or uncompensated shock depends on the underlying type and cause of shock. Consult a critical care specialist (or other subspecialist for specific causes). See specific care for hypovolemic, distributive, cardiogenic and obstructive shock in the following sections.

Care for a Child or Infant With Hypovolemic Shock

Fluid resuscitation is the cornerstone of therapy in children with hypovolemic shock. An initial 20 mL/kg crystalloid fluid bolus should be administered rapidly and be repeated as needed. Two IVs are preferred.

ALERT

It is important to keep reassessing the child or infant after each fluid bolus to make sure therapy is effective, to assess the need for additional fluid boluses and to assess for signs of hypervolemia.

Normalized heart rate for age, normal pulses and capillary refill, normal blood pressure for age, urine output normal for age (1.5 to 2 mL/kg/h for infants and young children and 1 mL/kg/h for adolescents) and normal mental status suggest restored intravascular volume.

Ongoing requirements for >60 mL/kg fluid should raise concern for ongoing losses or an alternative diagnosis, such as septic shock with capillary leak. Attention should also be paid to the treatment of hypoglycemia in children presenting with hypovolemic shock; if present, the patient should receive an IV bolus of dextrose in addition to crystalloid fluids, which do not contain dextrose. In addition, give attention to children or infants with DKA. Fluid resuscitation is warranted in a child in DKA and shock, but as a smaller

Table 7-23 | Unique Interventions for Specific Circumstances in Shock

Circumstance in Shock	Unique Intervention
Tension pneumothorax	Perform immediate needle thoracentesis
Cardiac tamponade	Perform immediate pericardiocentesis (under echocardiographic guidance, if possible)
Minimize oxygen demand and other metabolic demands	Control ventilation; treat fever, pain or seizures when present; consider sedation and neuromuscular blockade as clinically indicated
Metabolic abnormalities	Obtain laboratory samples (e.g., CBC, CMP, lactate, ABG). Identify and manage metabolic abnormalities (e.g., hypoglycemia, hypocalcemia, acidosis and adrenal crisis)
Patients with refractory shock	Consider blood transfusion as clinically indicated in order to increase the oxygen-carrying capacity of the blood
Patients refractory to fluid or vasoactive therapy or who have a history of recent or chronic steroid use or evidence of adrenal insufficiency	Consider administering corticosteroids
Distributive shock due to anaphylaxis	Administer epinephrine IM

aliquot (10 mL/kg) and at a much slower rate to avoid causing cerebral edema. However, follow facility protocols for fluid resuscitation in children and infants with DKA.

The immediate care and management for hemorrhagic hypovolemic shock is slightly different from the care and management for hypovolemic shock unrelated to traumatic blood loss. Consider two goals when treating an acutely bleeding patient: control the bleeding and replace the blood lost.

Practice Note

Providers should consider the administration of tranexamic acid (TXA) preferably within the first three hours for pediatric patients who are actively bleeding as a result of trauma. TXA is an antifibrinolytic agent and is used to reduce blood loss.

In patients who do not respond to initial fluid resuscitation, PRBC or whole blood transfusion is recommended for repletion of intravascular volume.

See *Hypovolemic Shock* Treatment Guideline for a summary of care.

Care for a Child or Infant with Distributive Shock

General management of distributive shock consists of therapies that aim to restore normal vascular tone and treat the relative hypovolemia that results from vasodilation and capillary leak. Early vasopressor therapy may be indicated to treat vasodilation. Additional therapies are tailored to the specific subtype of distributive shock—namely, septic shock, anaphylactic shock and neurogenic shock.

Septic Shock

Within the First Hour

Evidence-based guidelines exist for the management of septic shock in children and have been shown to improve outcomes. These guidelines emphasize aggressive fluid resuscitation, administration of antimicrobial therapy within the first hour of presentation and administration of medications for fluid refractory shock (Figure 7-13).

Monitor and support airway, breathing and circulation. Monitor heart rate, blood pressure and oxygen saturation. Assure vascular access (IV/IO), if not already obtained.

Fluid therapy should be initiated within 30 minutes. Fluid boluses of 20 mL/kg should be administered rapidly to a total volume of up to 60 mL/kg as needed. Administer smaller fluid bolus volumes in children with pre-existing cardiovascular compromise.

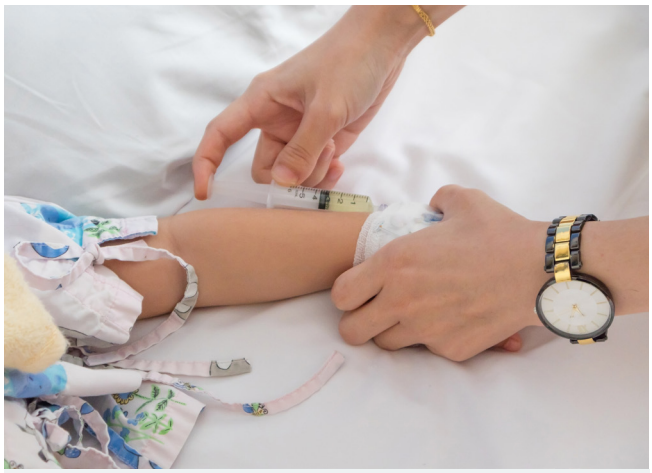


Figure 7-13 | Management of septic shock in children includes aggressive fluid resuscitation, administration of antimicrobial therapy within the first hour, and administration of medications for fluid refractory shock.

For neonates, administer boluses of 10 mL/kg of crystalloid or colloids until 40 mL/kg. Closely monitor before and after each fluid bolus. Fluid administration should be continued as long as static (e.g., blood pressure, heart rate) or dynamic (e.g., pulse pressure, urine output) variables improve or until signs of intolerance (e.g., rales, hepatomegaly) develop.

In addition, it is also important to identify and treat hypoglycemia and hypocalcemia.

Draw blood for cultures (including from any indwelling vascular access device, unless it was placed in the past 48 hours) and laboratory studies (e.g., glucose, calcium, lactate). Other appropriate cultures (e.g., urine, cerebrospinal fluid, wound, respiratory) should also be obtained before administering antimicrobial therapy. *Note: Do not delay fluid or antimicrobial therapy to wait for blood cultures or to perform any other diagnostic studies or assessments.*

> Antimicrobial therapy should be administered as soon as possible (i.e., no later than 1 hour) after identifying the presence of septic shock.

Studies show that with each hour of delay in administering effective antimicrobial therapy in septic shock, mortality measurably increases.

Empiric antimicrobial therapy should consist of one or more drugs that have activity against any likely pathogens (e.g., bacteria, viruses, fungi) and are likely to penetrate infected tissues in sufficient concentrations. Early implementation of measures to control the source of infection, such as surgical debridement or drainage or removal of any infected devices, is also recommended, when warranted.

Ready access to critical care resources such as mechanical ventilation and inotropic support should be ensured for children and infants undergoing fluid resuscitation for septic shock.

Recommended initial therapeutic end points for resuscitation include normal peripheral pulses, a capillary refill time ≤2 seconds, normal heart rate for age, normal blood pressure for age, normal urine output for age (1.5-2 mL/kg/h for infants and young children and 1 mL/kg/h for adolescents) normal mental status, correction of acidosis and metabolic abnormalities (e.g., glucose and calcium), normal lactate levels and $ScvO_2$ >70%.

If shock persists despite aggressive fluid resuscitation, vasoactive therapy (catecholamines) should be initiated within 60 minutes of presentation using peripheral access, if necessary, until central venous access is established. Epinephrine is recommended for cold shock; dopamine is a secondary option. Norepinephrine is recommended for warm shock; dopamine is a secondary option. For neonates in cold or warm shock initiate a dopamine infusion and add epinephrine for dopamine refractory shock. A pediatric critical care specialist should be consulted.

Critical Care Beyond the First Hour

For catecholamine-resistant shock, additional pharmacologic therapies are added as appropriate based on ongoing hemodynamic abnormalities.

Consider administering hydrocortisone in patients with persistent shock despite vasoactive therapy and who are at risk for absolute adrenal insufficiency.

Place a central venous catheter and invasive blood pressure monitoring, if not already done. Invasive parameters are used to direct care for shock that persists beyond the first hour. These include the CVP, which is measured in the vena cava and is a surrogate for preload, and $ScvO_2$, which is thought to be an indirect measure of tissue oxygenation.

Fluid and medications are titrated to maintain a CVP of 8 to 12 mmHg and $ScvO_2$ >70%; recommendations suggest that medication therapy depends on the clinical presentation of shock (cold vs. warm) and blood pressure.

Goals of management are $ScvO_2$ >70%, normal perfusion pressure (i.e., MAP-CVP) and blood pressure for age, normalized heart rate, adequate cardiac output and end organ perfusion.

PRBC transfusion may be considered if clinically indicated in the pediatric patient in catecholamine-resistant, cold shock.

If the patient meets the therapeutic endpoints and $ScvO_2$ is >70%, continue monitoring and supporting hemodynamic status. Titrate medications as appropriate, continue interventions as appropriate and continue monitoring. Continue assessment for etiology of septic shock and direct therapy accordingly. Continue to asses for morbidities and complications.

If catecholamine-resistant shock persists, assess for perfusion, evaluate for untreated or unrecognized morbidities and consider ECMO for continued refractory shock. Unrecognized morbidities may include:

- Unrecognized or uncontrolled source infection.
- Pericardial effusion.
- Pneumothorax.
- Hypoadrenal.
- Hypothyroid.
- Hemorrhage or ongoing blood loss.
- Increased intra-abdominal pressure.
- Excessive immunosuppression and/or immunocompromise.

See *Septic Shock* Treatment Guideline for a summary of care.

Anaphylactic Shock

Immediate management of anaphylactic shock consists of intramuscular (IM) administration of epinephrine (every 10 to 15 minutes, as needed) to counteract the effects of the allergic mediators released including vasodilation that is characteristic of this type of shock.

ALERT

Be prepared to intubate the patient, if necessary, owing to airway edema.

If respiratory compromise persists after administration of epinephrine IM, provide supplemental oxygen as needed, assist ventilation as needed and administer albuterol by MDI or nebulizer as needed. If hypotension or signs of shock persist after administration of epinephrine IM, initiate fluid resuscitation (20 mL/kg crystalloid fluid bolus; repeat as needed). Fluid refractory hypotension may be treated with vasopressor therapy (e.g., epinephrine).

Antihistamines and steroids may impede the release of mediators that contribute to the pathogenesis of anaphylaxis and may thus help to reverse symptoms. Prevent any ongoing exposure to the agent that precipitated anaphylaxis and monitor the patient for recurrence of anaphylactic symptoms.

See *Anaphylactic Shock* Treatment Guideline for a summary of care.

Neurogenic Shock

In neurogenic shock due to acute spinal cord injury, vasodilation results in relative hypovolemia as a result of increased venous capacity; therefore, fluid resuscitation is often necessary in this setting. Administer 20 mL/kg crystalloid fluid bolus rapidly and assess response.

> It is important to manage impaired perfusion and hypotension immediately and aggressively to avoid secondary spinal cord injury.

In patients who have spinal cord injury from a traumatic event, hemorrhage must be excluded as an underlying cause of shock. If present, bleeding should be controlled, IV fluids given and blood loss replaced with PRBC's or whole blood as clinically indicated.

If hypotension persists despite adequate fluid resuscitation, it should be managed immediately. In patients with normal cardiac output, agents with primary or exclusive alpha-adrenergic effects, such as phenylephrine or norepinephrine, are reasonable first-line treatments. If bradycardia is present, treat with atropine or vasoactive medications with chronotropic activity.

See *Neurogenic Shock* Treatment Guideline for a summary of care.

Care for a Child or Infant with Cardiogenic Shock

For patients with cardiogenic shock, it is important to obtain and evaluate a 12-lead ECG, treat any arrhythmias and obtain laboratory and diagnostic tests, including an echocardiogram in order to identify and treat underlying causes (e.g., pericardial effusion). In addition, early expert cardiac/critical care consultation is important.

When providing care to a patient with cardiogenic shock, consider supporting or providing ventilation (noninvasive and noninvasive) to reduce cardiac workload to help improve perfusion. In addition, consider sedation and manage temperature to further reduce metabolic demands and reduce cardiac workload.

Children in cardiogenic shock may be hypo-, normo- or hypervolemic. Therefore, fluid resuscitation should be carried out with caution, starting with smaller-than-usual boluses of 5 to 10 mL/kg and closely monitoring response and for signs of pulmonary edema and signs of worsening perfusion. Diuretics may be indicated in children who present with hypervolemia.

Pharmacologic therapy is the mainstay of treatment in children and infants presenting with cardiogenic shock. Inotropes, inodilators and/or vasoactive agents are often needed to improve cardiac contractility and, with the use of drugs that reduce afterload, to reduce the work that the heart has to do.

Correction of metabolic abnormalities such as acidosis, hypocalcemia, hypoglycemia or hyperkalemia may further enhance cardiac function. If the patient remains in persistent cardiogenic shock after implementation of these care measures, consider mechanical circulatory support.

See *Cardiogenic Shock* Treatment Guideline for a summary of care.

Care for a Child With Obstructive Shock

Fluid therapy for patients with obstructive shock should be administered as appropriate and as needed. In addition to fluid therapy, care and treatment of the child

or infant in obstructive shock largely depends on the underlying cause. Invasive intervention is central to the immediate management of cardiac tamponade and tension pneumothorax.

Likewise, specialized therapies are indicated for pulmonary embolism and obstructive cardiac and aortic lesions.

Cardiac Tamponade

In children with cardiac tamponade as the underlying cause of obstructive shock, fluid in the pericardial sac should be drained immediately, using a procedure called **pericardiocentesis**. This procedure should be done under echocardiographic guidance if possible. Optimization of intravascular volume and preload is crucial while awaiting drainage of pericardial fluid.

Tension Pneumothorax

Treatment of an underlying tension pneumothorax involves immediate evacuation of the air in the pleural space in a procedure known as a **needle thoracentesis**, or needle decompression. (See *Learn More: Needle Thoracentesis*.) Once the patient is stabilized after thoracentesis, a chest tube should be placed in the pleural space to allow ongoing removal of any accumulating air.

Pulmonary Embolism

Anticoagulation is used to treat a pulmonary embolism. For a life-threatening pulmonary embolism, an agent such as tissue plasminogen activator or surgical removal may also be considered. Children with pulmonary embolism, will require anticoagulant therapy beyond the initial treatment.

Obstructive Cardiac and Aortic Lesions

Infants with suspected or documented ductal-dependent cardiac lesions should receive prostaglandin E_1 infusion. Prostaglandin E_1 is a vasodilator that maintains the patency of the ductus arteriosus. In infants with severe shock and a suspected left-sided obstructive lesion, treatment with prostaglandin E_1 should be initiated before a more thorough diagnostic evaluation is undertaken. This intervention may save the life of the infant who is ultimately found to have a ductal-dependent cardiac condition. In fact, treatment with prostaglandin E_1 may be considered in any infant presenting with shock until a ductal-dependent lesion can be excluded.

Additional treatment of a confirmed cardiac or aortic lesion causing obstructive shock consists of inotropes to support ventricular function, as needed, and consideration of ventilatory support to reduce cardiac workload. In addition, it is important to consult a pediatric cardiologist for diagnostic evaluation and definitive management. Immediate surgical or catheter-based intervention may be warranted.

See *Obstructive Shock* Treatment Guideline for a summary of care.

LEARN MORE

Needle Thoracentesis

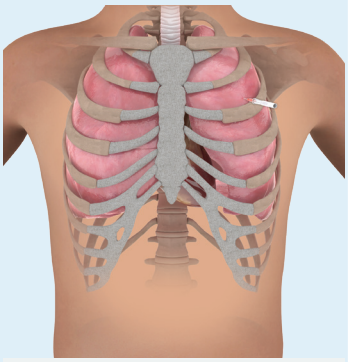
Needle thoracentesis is a procedure used to quickly decompress a life-threatening tension pneumothorax. The procedure is most often carried out using a large-bore catheter. You will require the following equipment to perform needle thoracentesis:

- A 14- or 16-gauge catheter
- A syringe
- Antiseptic solution
- Tape or other adhesive dressing

The patient should be supine for the procedure and connected to a cardiac monitor and pulse oximeter. First, identify the location for catheter insertion. Options include the second intercostal space in the midclavicular line or, alternatively, the fourth or fifth intercostal space just anterior to the midaxillary line on the side of the affected lung. To locate the second rib, find the sternal notch and walk your fingers down the manubrium until you feel a bony ridge. This ridge is known as the *sternal angle or angle of Louis* and aligns with the second rib. The second intercostal space is below this rib.

To perform the procedure:

- Cleanse the skin overlying the site for needle insertion.
- Consider using a local anesthetic in patients who are conscious (time permitting).
- Attach the syringe to the catheter.
- While withdrawing the plunger in the syringe, insert the catheter vertically (at a 90° angle to the chest wall) just above the rib below the chosen intercostal space in the appropriate anatomic (i.e., midclavicular or mid-axillary) line. (Inserting the needle below the rib above the intercostal space risks injury to the blood vessels and nerves that run along the bottom of each rib.)
- Advance the catheter until the syringe fills with air. (You may also feel a "pop" or decrease in resistance, followed by an audible rush of air, when the needle passes through the pleura.)
- Remove the stylet and syringe, leaving the catheter in place. Secure the catheter using tape or an adhesive dressing.
  - Note: A three-way stopcock may be attached to the catheter to allow for ongoing aspiration of air.
- Arrange for definitive thoracostomy tube placement.



Landmarks and site for needle insertion in needle thoracentesis

GENERAL SHOCK

Perform rapid assessment (PAT), primary assessment and initial interventions

↓

Suspected sepsis

↓

See *Septic Shock Pediatric Treatment Guideline*

- Ensure adequate oxygenation and ventilation
- Establish cardiac monitoring and IV access, if not already done*
- Assess perfusion
- Monitor blood pressure (noninvasive or invasive)

Intravascular Volume Restoration

- Begin with crystalloid fluid (0.9% NS or LR) bolus at 20 mL/kg IV/IO rapidly†
- Repeat as needed until normovolemia achieved or signs of hypervolemia develop‡
- For patients in hypovolemic shock due to hemorrhage:
  - Control the bleeding, administer fluids, replace blood loss with PRBCs or whole blood as indicated. See *Hypovolemic Shock Pediatric Treatment Guideline*

Medication Therapy

- Consider vasoactive therapy for fluid-refractory (or -intolerant) shock
- Choice of vasoactive therapy will depend on the desired physiologic effect(s)
- Placement of a central venous catheter should be undertaken for administration of vasoactive drugs, if possible, but *should not delay therapy*
- Begin prostaglandin E_1 infusion in any infant with a known or suspected ductal-dependent cardiac lesion (pending confirmation or exclusion by echocardiogram)

Additional Considerations

- If tension pneumothorax suspected, perform immediate needle thoracentesis
- If cardiac tamponade suspected, perform immediate pericardiocentesis (under echocardiographic guidance, if possible)
- Initiate assisted ventilation as clinically indicated
- Minimize oxygen demand
  - Consider mechanical ventilation
  - Treat fever, pain or seizures, when present
  - Consider sedation and neuromuscular blockade, as clinically indicated
- Obtain laboratory samples (e.g., CBC, CMP, lactate, ABG)
- Identify and manage metabolic abnormalities (e.g., hypoglycemia, hypocalcemia, acidosis, adrenal crisis)
- Consult critical care specialist (or other subspecialist for specific causes)

Therapeutic End Points in Shock

- Normal HR for age
- Normal peripheral pulses
- Normal capillary refill (≤ 2 sec)
- Normal BP for age
- Normal urine output
  - Infants and young children: 1.5 to 2 mL/kg/hr
  - Adolescents: 1 mL/kg/hr
- Normal mental status
- Correction of acidosis
- Normal lactate levels
- $ScvO_2$ > 70%

Identify type of shock (see *Identification of Type of Shock* table, on reverse side)

Please see reverse side for additional information

*If unable to obtain intravenous access, establish intraosseous access, if clinically warranted. If possible, two large-bore IV (or IO) access points are best for hypovolemic shock.

†Administer smaller (10-mL/kg) fluid bolus volumes in neonates. Also consider smaller (10-mL/kg) fluid bolus volumes in children with heart failure who are euvolemic. (Fluid therapy may be contraindicated in children with hypervolemic conditions).

‡Signs of hypervolemia include a palpable liver edge or crackles in the lungs.

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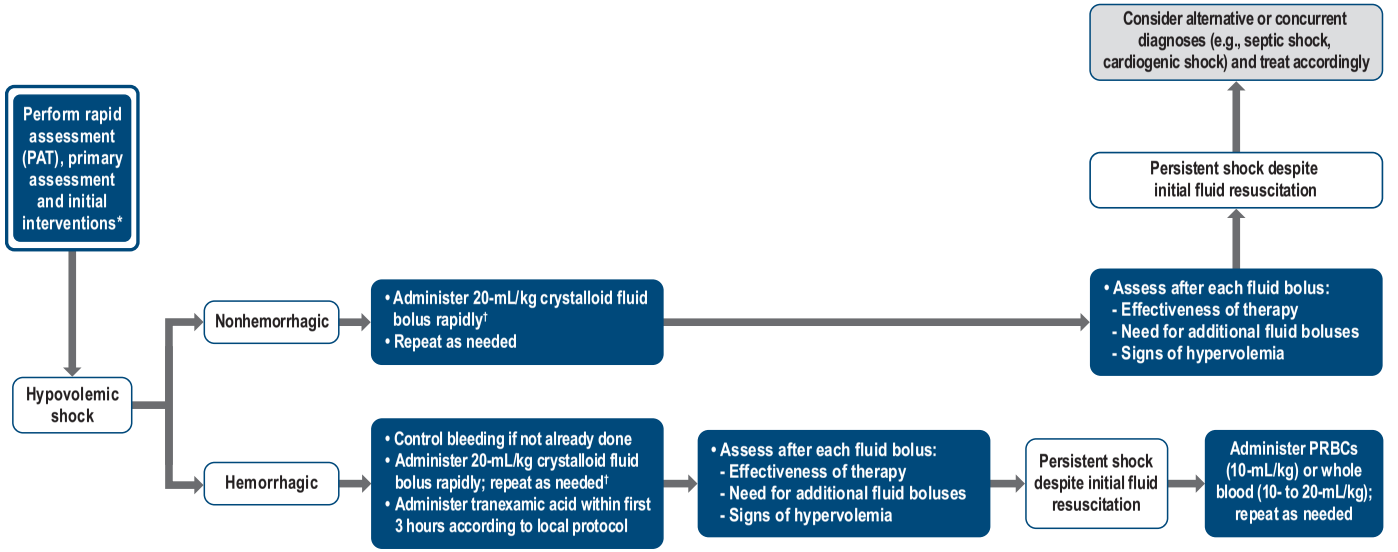
GENERAL SHOCK CONTINUED

Identification of Type of Shock			
Type of Shock	**Primary Assessment Findings**		
Hypovolemic	• From compensated to decompensated shock: - Airway patent to possible airway compromise due to altered level of consciousness - Tachypnea to possible bradypnea (late) - Normal systolic blood pressure for age to hypotension - Tachycardia to possible bradycardia (late) - Diminished peripheral pulses and delayed capillary refill to absent peripheral pulses, weak central pulses and absent capillary refill - Pale, cool skin to cold, pale, mottled skin	- Decreased to negligible urine output - Anxiety to altered mental status or decreased level of consciousness - Pale, cool extremities to cold, pale, mottled extremities • Hemorrhage • Dry mucous membranes • Sunken eyes or fontanelle (infants)	See *Hypovolemic Shock Pediatric Treatment Guideline*
Distributive	• Anaphylactic shock: - Hypotension post exposure to known allergen - See *Anaphylactic Shock Pediatric Treatment Guideline* for other diagnostic criteria • Neurogenic shock: - Airway patent unless level of consciousness impaired - Hypotension in the absence of tachycardia (relative bradycardia) - Wide pulse pressure - Warm, flushed skin (due to vasodilation due to interrupted sympathetics) - Hypothermia - Signs not specifically attributed to shock (e.g., signs of respiratory compromise depending on level of spinal injury, neurologic deficits, altered level of consciousness if concurrent head injury) • Septic shock: - Airway patent unless level of consciousness impaired - Tachypnea and/or hyperpnea	- Increased work of breathing - Hypoxemia - Tachycardia or bradycardia - Diminished (cold shock) or bounding (warm shock) pulses - Delayed (cold shock) or brisk (warm shock) capillary refill - Normal or reduced blood pressure - Narrow pulse pressure and pale, cool, mottled skin (cold shock) - Wide pulse pressure and warm, flushed skin (warm shock) - Decreased urine output - Normal or altered mental status (e.g., restlessness, agitation, anxiety, somnolence, coma) - Increased or decreased body temperature - Cool, mottled extremities (cold shock) or warm flushed extremities (warm shock) - Petechiae and/or purpura	See *Anaphylactic Shock, Neurogenic Shock and Septic Shock Pediatric Treatment Guidelines*
Cardiogenic	• Airway patent unless level of consciousness impaired • Tachypnea • Wheezing or crackle on lung auscultation (latter rare in infants) • Grunting • Retractions or nasal flaring • Pulmonary edema • Cyanosis • Tachycardia or bradycardia • Weak pulses • Hypotension	• Narrowed pulse pressure • Pale, cool, mottled skin • Cold extremities • Decreased urine output • Arrhythmia • Altered level of consciousness • Neck vein distention • Peripheral edema • Diaphoresis	See *Cardiogenic Shock Pediatric Treatment Guideline*
Obstructive	• General - Airway patent unless level of consciousness impaired - Tachypnea and/or dyspnea - Tachycardia - Altered level of consciousness - Cold extremities • Cardiac tamponade: - Beck's triad (hypotension, muffled heart sounds, neck vein distention) - Diminished pulses - Narrowed pulse pressure - Pulsus paradoxus - Pericardial rub - Peripheral edema • Tension pneumothorax: - Tracheal deviation to unaffected side - Chest pain upon inspiration - Decreased breath sounds and hyperresonance to percussion on affected side - Hypotension - Neck vein distention - Pulsus paradoxus	• Pulmonary embolism: - Chest pain - Coughing up blood (hemoptysis) - Signs of acute right heart failure (e.g., distended neck veins, peripheral edema, ascites, hepatomegaly) - Hypotension - Arrhythmias (most often sinus tachycardia) - Signs of deep vein thrombosis (e.g., swelling, pain, erythema), most often in lower extremities • Obstructive cardiac and aortic lesions: - Respiratory distress - Cyanosis (may or may not be present depending on the lesion) - Signs of cold shock - Hypotension - Diminished or absent femoral pulses (if aortic lesion) - Differential blood pressure, O_2 saturation, or pulse between upper and lower extremities or between the right upper extremity and all other extremities (i.e., left upper extremity and lower extremities) - Diaphoresis	See *Obstructive Shock Pediatric Treatment Guideline*

HYPOVOLEMIC SHOCK



Recognize Hypovolemic Shock

Primary Assessment Findings:

- Airway patent unless level of consciousness impaired
- Tachycardia
- Signs attributable to peripheral vasoconstriction
  - Diminished or absent peripheral pulses
  - Delayed capillary refill (> 2 seconds)
  - Cool, pale or mottled extremities
  - Narrowed pulse pressure
- Hemorrhage
- Hypotension (decompensated)
- Tachypnea
- Decreased urine output (unless caused by condition characterized by increased urine output, such as diabetes mellitus)
- Altered mental status
- Dry mucous membranes
- Sunken eyes or fontanelle (infants)

Secondary Assessment Findings:

- Focused physical assessment: decreased tear production
- History of:
  - Trauma (hemorrhagic)
  - Fluid losses from gastrointestinal tract
  - Third-spacing (e.g., burn)

Tranexamic Acid Dosing

Initial dose: 15 mg/kg (maximum dose, 1 g) IV over 10 minutes (dilute in a convenient volume of NS)

Subsequent dosing: 2 mg/kg/hr IV (suggested dilution: 500-mg TXA in 500-mL NS given at a rate of 2 mL/kg/hr); give for at least 8 hours or until bleeding stops

Therapeutic End Points in Shock

Primary Assessment Findings:

- Normal HR for age
- Normal peripheral pulses
- Normal capillary refill (≤ 2 sec)
- Normal BP for age
- Normal urine output
  - Infants and young children: 1.5 to 2 mL/kg/hr
  - Adolescents: 1 mL/kg/hr
- Normal mental status
- Correction of acidosis
- Normal lactate levels
- ScvO2 > 70%

*Check bedside glucose level, as clinically indicated, and provide treatment for hypoglycemia – bolus 10% or 25% dextrose in water IV/IO given slowly. Follow with continuous dextrose infusion as needed.

†Two large-bore IV (or IO) access points are preferred for especially rapid administration.

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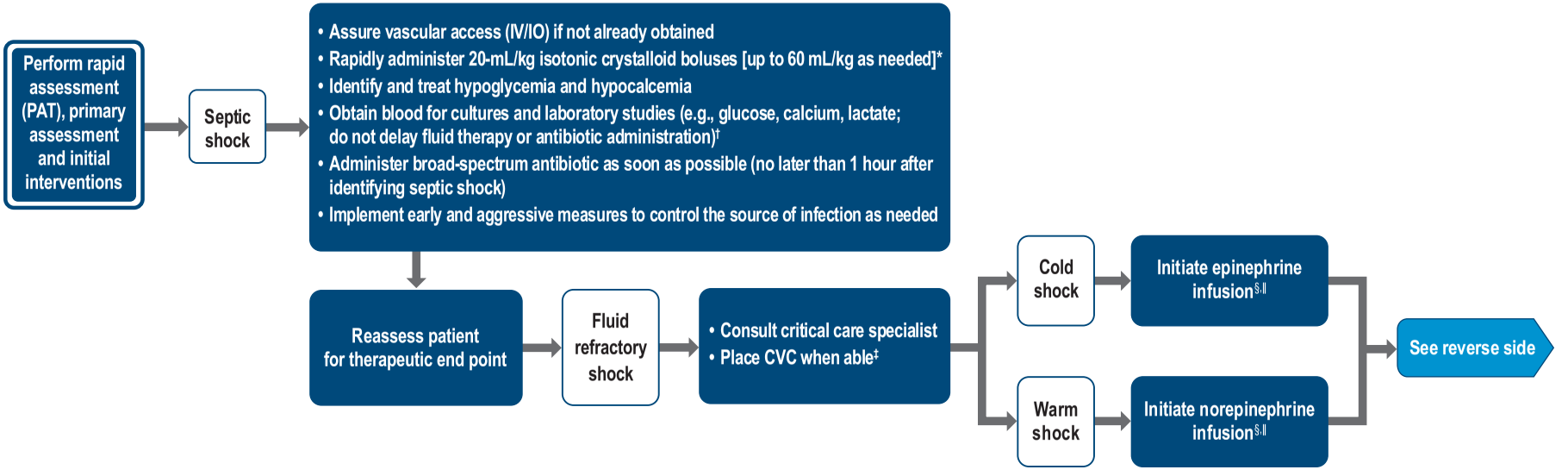
SEPTIC SHOCK



Within First Hour

Hypoglycemia in Shock	
Plasma Glucose Threshold	**Care**
• Neonates: < 45 mg/dL • Adolescents, children and infants: < 60 mg/dL	• Bolus 10% or 25% dextrose in water IV/IO given slowly • Follow with continuous dextrose infusion • Recheck glucose frequently

Therapeutic End Points in Shock

- Normal peripheral pulses and capillary refill (≤ 2 sec)
- Normal HR for age
- Normal BP for age
- Normal urine output
  - Infants and young children: 1.5 to 2 mL/kg/hr
  - Adolescents: 1 mL/kg/hr
- Normal mental status
- Correction of acidosis and metabolic abnormalities (e.g., glucose and calcium)
- Normal lactate levels
- $ScvO_2$ > 70%

*Fluid administration initiated within 30 minutes. For neonates administer boluses of 10 mL/kg of crystalloid or colloids until 40 mL/kg. Reassess after each bolus for signs of hypervolemia and/or congestive heart failure (crackles, hepatomegaly).

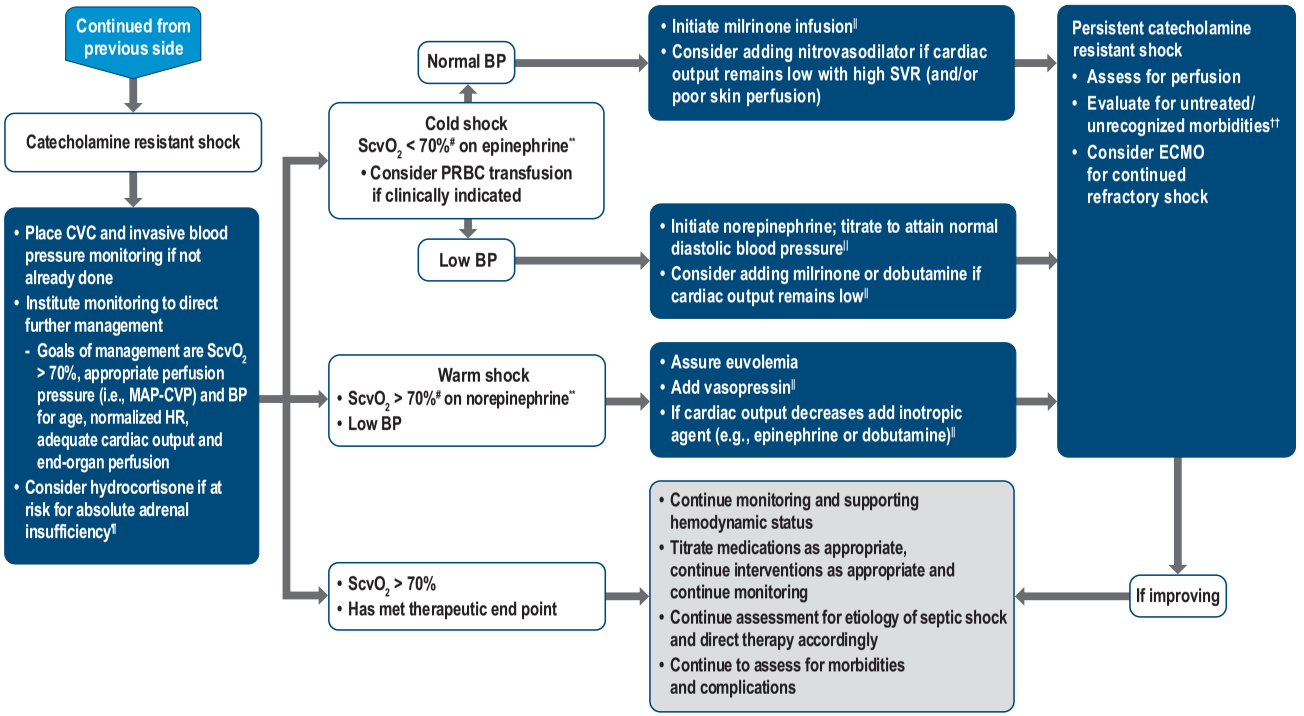
†Obtaining cultures should not delay initiation of antimicrobial therapy within 60 minutes. Cultures should include at least 1 set of peripheral blood cultures and cultures from any indwelling vascular access device, unless it was placed in the past 48 hours.

‡Central venous catheter should be placed only by those with sufficient training and expertise in CVC placement. Central venous catheter placement should not delay initiation of vasoactive agents. Consider ketamine for sedation when placing CVC.

§May alternatively initiate dopamine infusion as initial catecholamine if epinephrine/norepinephrine are not available. For neonates in cold or warm shock initiate a dopamine infusion and add epinephrine for dopamine refractory shock.

‖See *Medication Dosages* table.

SEPTIC SHOCK CONTINUED



Medication	Dosage
Epinephrine	0.1- to 1-mcg/kg/min IV/IO infusion, titrating to effect
Dopamine	2- to 20-mcg/kg/min IV/IO infusion titrated to clinical effect
Hydrocortisone	50-mg/m²/24 h IV infusion
Milrinone	• Loading dose: 50 mcg/kg IV/IO over 10 to 60 minutes • Infusion: 0.25 to 0.75 mcg/kg/min
Norepinephrine	0.1- to 2-mcg/kg/min IV/IO infusion, titrated upward as needed to desired clinical effect
Dobutamine	2- to 20-mcg/kg/min IV/IO infusion, titrated to effect
Vasopressin	0.0002- to 0.002-units/kg/min IV/IO infusion

Unrecognized Morbidities

- Unrecognized/uncontrolled source infection
- Pericardial effusion
- Pneumothorax
- Hypoadrenal
- Hypothyroid
- Hemorrhage or ongoing blood loss
- Increased intra-abdominal pressure
- Excessive immunosuppression and/or immunocompromise

‖See *Medication Dosages* table.

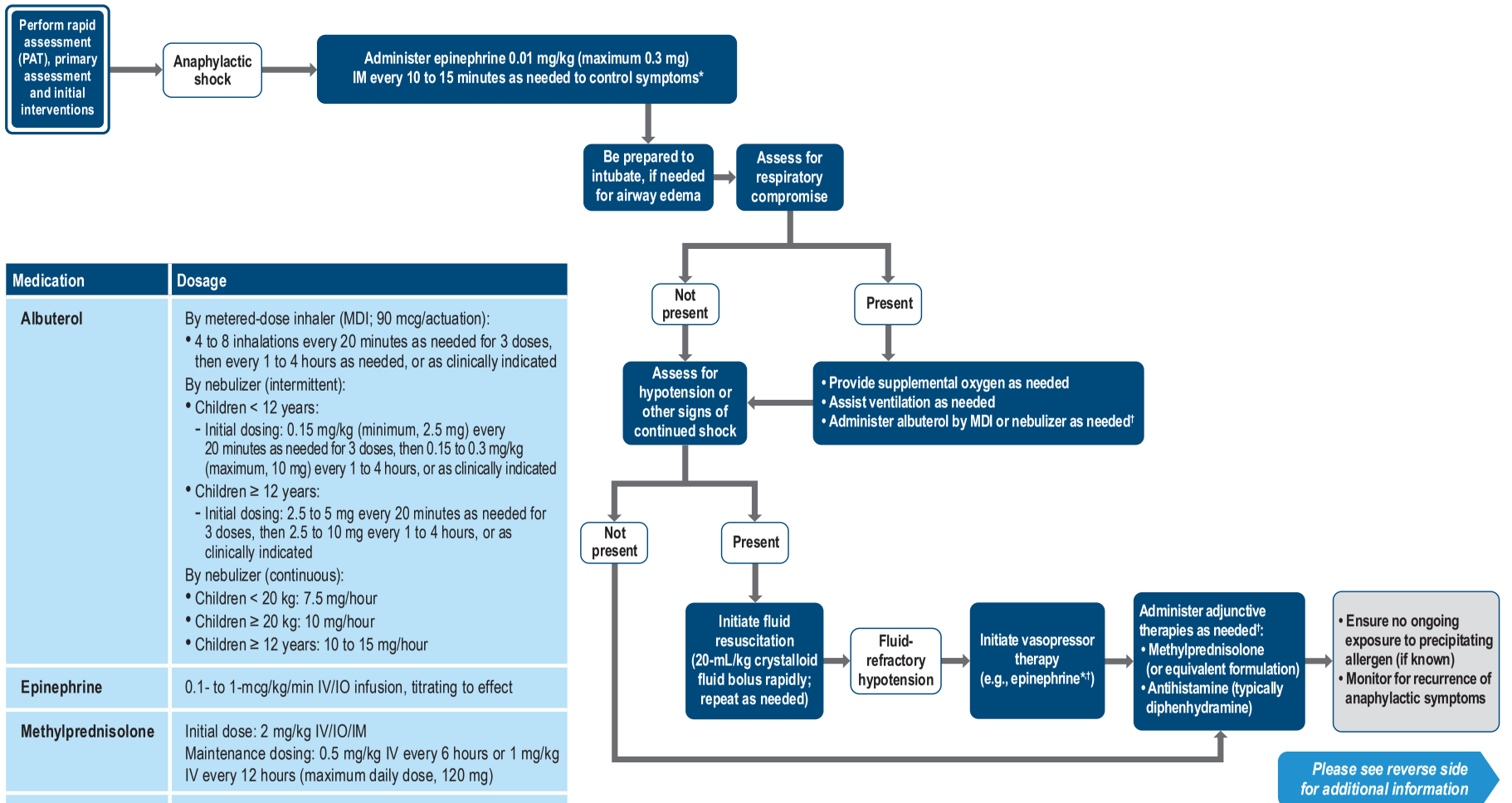
¶Children with purpura fulminans, recent or chronic steroid use or pituitary or adrenal abnormalities are at risk for absolute adrenal insufficiency.

#If shock is not reversed proceed to restore and maintain normal perfusion pressure (MAP – CVP) for age, $ScvO_2$ > 70% (except congenital heart patients with mixing lesions), and cardiac index > 3.3 < 6 L/min/m_2.

**For neonates, modification of this approach may be indicated.

††See *Unrecognized Morbidities* table.

ANAPHYLACTIC SHOCK



Medication	Dosage
Albuterol	By metered-dose inhaler (MDI; 90 mcg/actuation): • 4 to 8 inhalations every 20 minutes as needed for 3 doses, then every 1 to 4 hours as needed, or as clinically indicated By nebulizer (intermittent): • Children < 12 years: - Initial dosing: 0.15 mg/kg (minimum, 2.5 mg) every 20 minutes as needed for 3 doses, then 0.15 to 0.3 mg/kg (maximum, 10 mg) every 1 to 4 hours, or as clinically indicated • Children ≥ 12 years: - Initial dosing: 2.5 to 5 mg every 20 minutes as needed for 3 doses, then 2.5 to 10 mg every 1 to 4 hours, or as clinically indicated By nebulizer (continuous): • Children < 20 kg: 7.5 mg/hour • Children ≥ 20 kg: 10 mg/hour • Children ≥ 12 years: 10 to 15 mg/hour
Epinephrine	0.1- to 1-mcg/kg/min IV/IO infusion, titrating to effect
Methylprednisolone	Initial dose: 2 mg/kg IV/IO/IM Maintenance dosing: 0.5 mg/kg IV every 6 hours or 1 mg/kg IV every 12 hours (maximum daily dose, 120 mg)
Diphenhydramine	1 to 2 mg/kg IV/IM/PO (maximum, 50 mg)

*For severe anaphylaxis, epinephrine may be administered via IV infusion if the child does not respond to IM injections.

†See *Medication Dosages* table.

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ANAPHYLACTIC SHOCK CONTINUED

Recognize Anaphylactic Shock

Diagnostic Criteria:

- Cutaneous and mucosal finding (e.g., swollen lips, tongue or uvula [angioedema], hives, flushing, itching) and one of the following:
  - Respiratory compromise (stridor, hoarseness, wheezing, dyspnea, increased work of breathing)
  - Hypotension and signs of end-organ dysfunction

OR

- Two or more of the following post exposure to an allergen:
  - Cutaneous and mucosal finding (e.g., swollen lips, tongue or uvula [angioedema], hives, flushing, itching)
  - Respiratory compromise (stridor, hoarseness, wheezing, dyspnea, increased work of breathing)
  - Hypotension and signs of end-organ dysfunction
  - Gastrointestinal symptoms (e.g., nausea, vomiting, abdominal pain)

OR

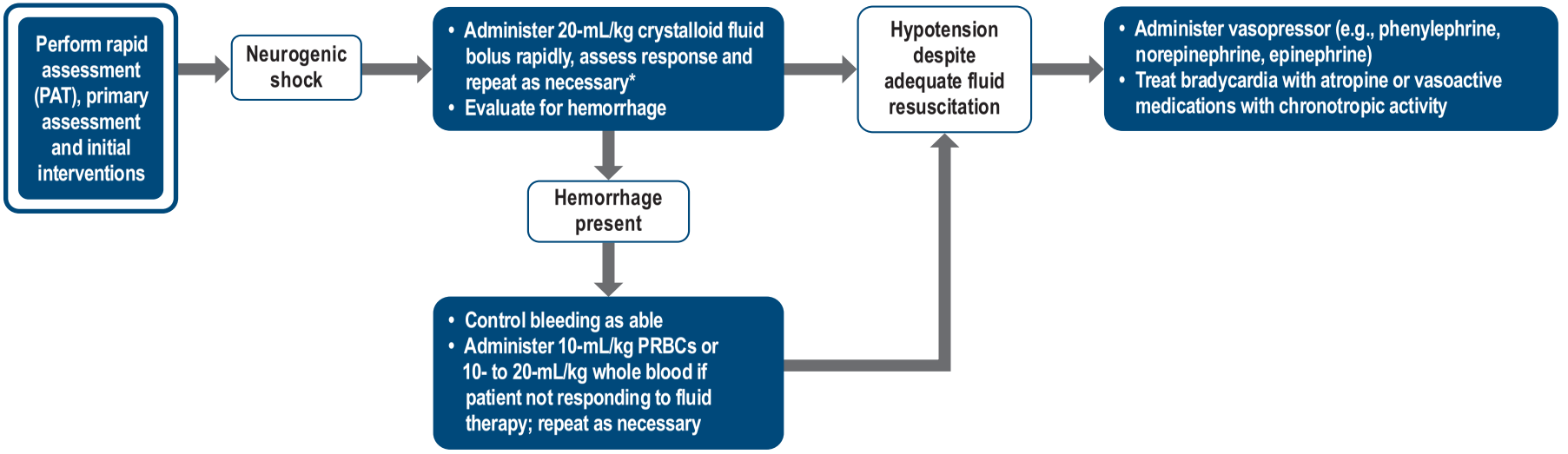
- Hypotension post exposure to a known allergen

Therapeutic End Points in Shock

Primary Assessment Findings:

- Normal HR for age
- Normal peripheral pulses
- Normal capillary refill (≤ 2 sec)
- Normal BP for age
- Normal urine output
  - Infants and young children: 1.5 to 2 mL/kg/hr
  - Adolescents: 1 mL/kg/hr
- Normal mental status
- Correction of acidosis
- Normal lactate levels
- $ScvO_2$ > 70%

NEUROGENIC SHOCK



Recognize Neurogenic Shock

Primary Assessment Findings:

- Hypotension in the absence of tachycardia (relative bradycardia)
- Wide pulse pressure
- Warm, flushed skin (due to vasodilation from disrupted sympathetics)
- Hypothermia
- Possible associated signs not specifically attributable to shock:
  - Signs of respiratory compromise (depending on the level of spinal injury)
    - Apnea
    - Rapid, shallow breathing
    - Weak cough
    - Increased airway secretions
    - Diaphragmatic breathing with paradoxical chest wall movement
  - Altered level of consciousness if concurrent head injury
  - Neurologic deficits concordant with level of spinal cord injury

Secondary Assessment Findings:

- Known or suspected trauma predisposing to possible spinal cord injury

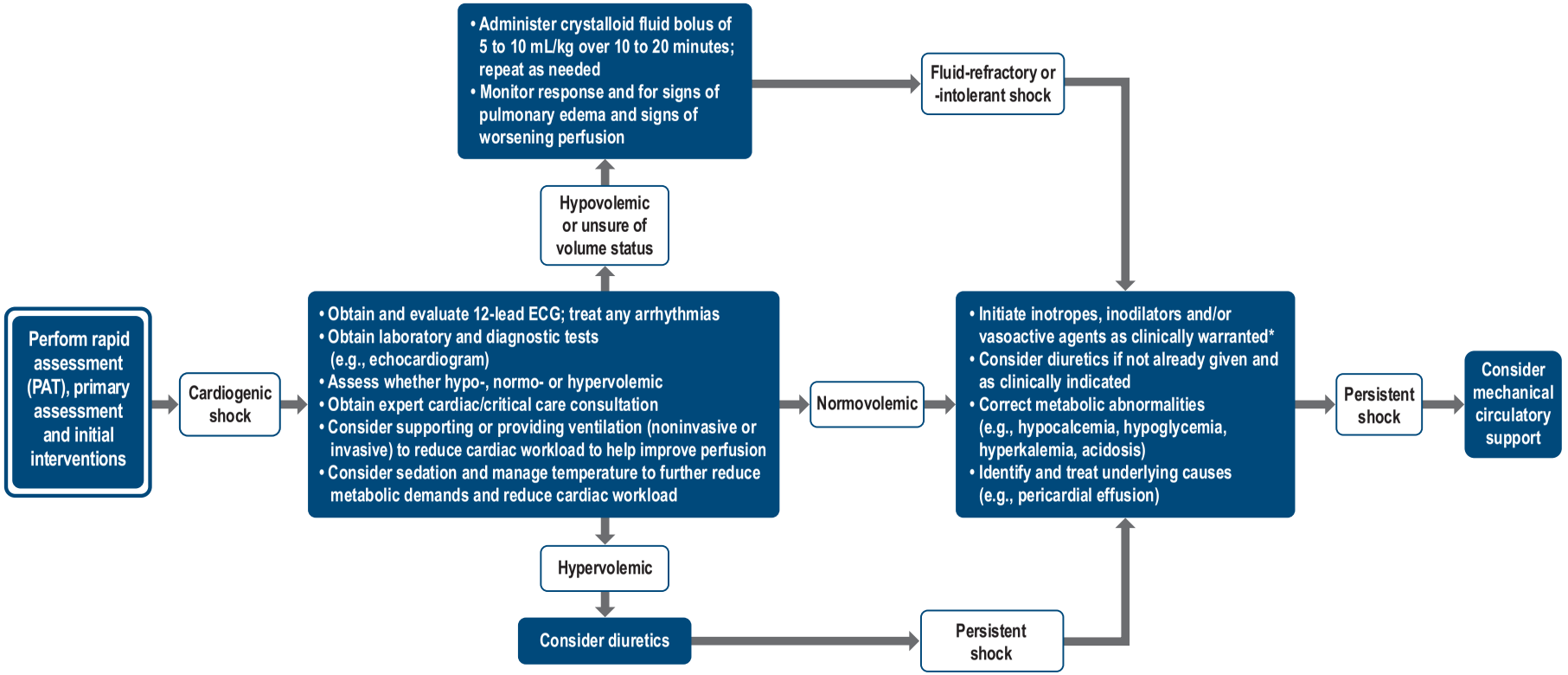
Therapeutic End Points in Shock

Primary Assessment Findings:

- Normal HR for age
- Normal peripheral pulses
- Normal capillary refill (≤ 2 sec)
- Normal BP for age
- Normal urine output
  - Infants and young children: 1.5 to 2 mL/kg/hr
  - Adolescents: 1 mL/kg/hr
- Normal mental status
- Correction of acidosis
- Normal lactate levels
- $ScvO_2$ > 70%

*Treat impaired perfusion and hypotension immediately and aggressively to avoid secondary spinal cord injury.

CARDIOGENIC SHOCK



Please see reverse side for additional information

*Base choice of medication therapy on underlying etiology, vascular status, heart rate and cardiac function.

CARDIOGENIC SHOCK CONTINUED

Recognize Cardiogenic Shock

Primary Assessment Findings:

- Airway patent unless level of consciousness impaired
- Tachypnea
- Wheezing or crackle on lung auscultation (latter rare in infants)
- Grunting
- Retractions or nasal flaring
- Pulmonary edema
- Cyanosis
- Tachycardia or bradycardia
- Weak pulses
- Hypotension
- Narrowed pulse pressure
- Pale, cool, mottled skin
- Cold extremities
- Decreased urine output
- Arrhythmia
- Altered level of consciousness
- Neck vein distension
- Peripheral edema
- Diaphoresis

Secondary Assessment Findings:

- Focused physical assessment: heart murmur or gallop rhythm on cardiac auscultation
- Focused history: poor feeding or poor weight gain in infant; history of being less active or difficulty keeping up with peers in older children
- Diagnostic studies: cardiomegaly or pulmonary edema on chest radiograph
- Hepatomegaly

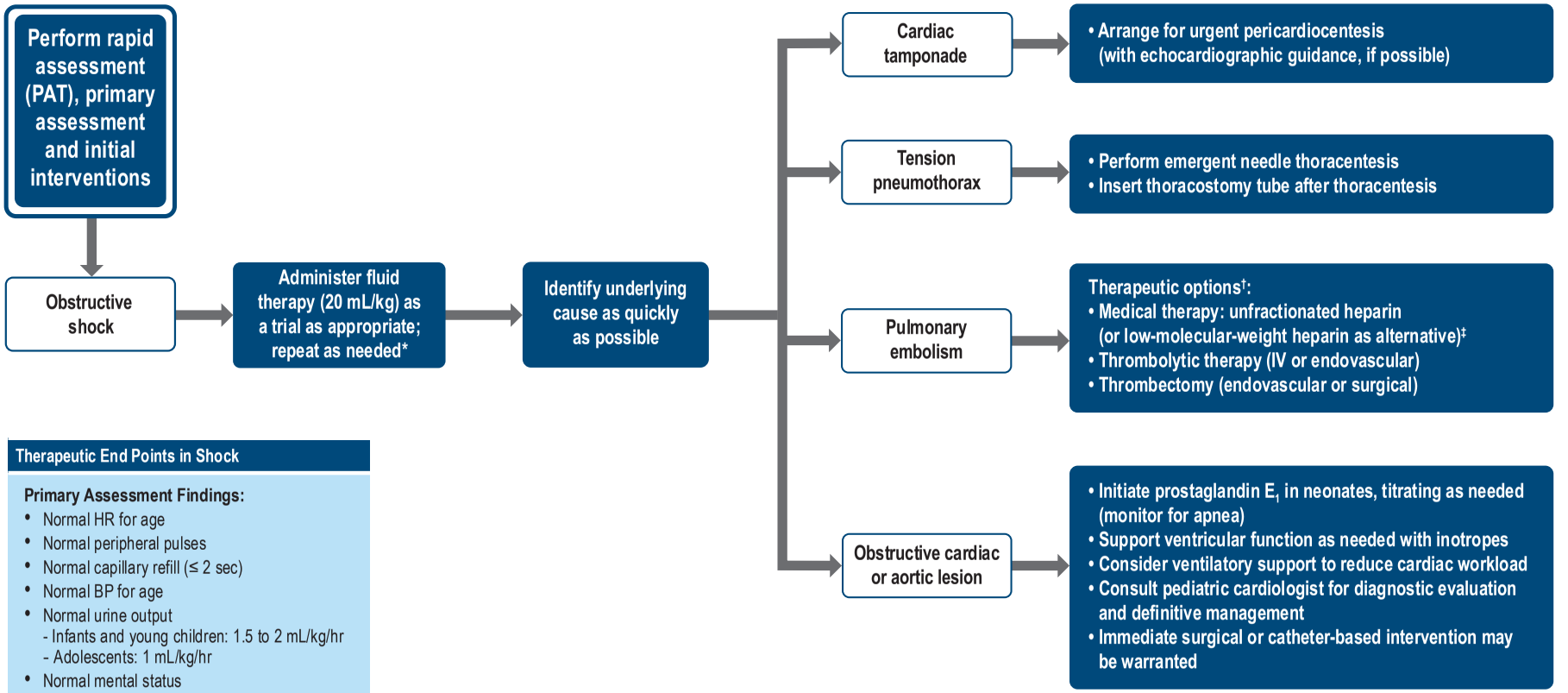
Drug Class Examples

Inotrope	Epinephrine, dopamine, dobutamine
Inodilator	Milrinone
Vasoactive	Vasoconstrictors: norepinephrine, epinephrine, dopamine Vasodilator: nitroprusside, nitroglycerin

Therapeutic End Points in Shock

- Normal HR for age
- Normal peripheral pulses
- Normal capillary refill (≤ 2 sec)
- Normal BP for age
- Normal urine output
  - Infants and young children: 1.5 to 2 mL/kg/hr
  - Adolescents: 1 mL/kg/hr
- Normal mental status
- Correction of acidosis
- Normal lactate levels
- $ScvO_2$ > 70%

OBSTRUCTIVE SHOCK



Therapeutic End Points in Shock

Primary Assessment Findings:

- Normal HR for age
- Normal peripheral pulses
- Normal capillary refill (≤ 2 sec)
- Normal BP for age
- Normal urine output
  - Infants and young children: 1.5 to 2 mL/kg/hr
  - Adolescents: 1 mL/kg/hr
- Normal mental status
- Correction of acidosis
- Normal lactate levels
- $ScvO_2$ > 70%

Please see reverse side for additional information

*Administer smaller (10-mL/kg) fluid bolus volumes in neonates. Also consider smaller (10-mL/kg) fluid bolus volumes in children with heart failure who are euvolemic. (Fluid therapy may be contraindicated in children with hypervolemic conditions.)

†Base choice of therapy on severity of patient's condition, location and type of lesion, and latest evidence. Obtain baseline prothrombin time, partial thromboplastin time and platelet count before thrombolytic therapy.

‡Consider other novel pharmacological agents, as clinically indicated.

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OBSTRUCTIVE SHOCK CONTINUED

Underlying Cause	Key Findings	
	Primary Assessment	Secondary Assessment
General	• Airway patent unless level of consciousness impaired • Tachypnea and/or dyspnea • Tachycardia • Altered level of consciousness • Cold extremities	• N/A
Cardiac tamponade	• Beck's triad (hypotension, muffled heart sounds, neck vein distention) • Diminished pulses • Narrowed pulse pressure • Pulsus paradoxus • Pericardial rub • Peripheral edema	• ECG: electrical alternans (alternating, varying amplitude of QRS complexes) • Chest radiograph: cardiomegaly, "water bottle"–shaped cardiac silhouette, pleural effusions • Echocardiogram: pericardial effusion and collapse of the right ventricle during diastole
Tension pneumothorax	• Tracheal deviation to unaffected side • Chest pain upon inspiration • Decreased breath sounds and hyperresonance to percussion on affected side • Hypotension • Neck vein distention • Pulsus paradoxus	• Chest radiograph: air in the pleural cavity, with contralateral deviation of mediastinal structures
Pulmonary embolism	• Chest pain • Coughing up blood (hemoptysis) • Signs of acute right heart failure (e.g., distended neck veins, peripheral edema, ascites, hepatomegaly) • Hypotension • Arrhythmias (most often sinus tachycardia) • Signs of deep vein thrombosis (e.g., swelling, pain, erythema), most often in lower extremities	• Laboratory test findings: elevated D-dimer, increased arterial-alveolar oxygen gradient on blood gas • ECG findings: ST-segment changes, right bundle branch block or right axis deviation
Obstructive cardiac and aortic lesions	• Respiratory distress • Cyanosis (may or may not be present depending on the lesion) • Signs of cold shock • Hypotension • Diminished or absent femoral pulses (if aortic lesion) • Differential blood pressure, O_2 saturation, or pulse between upper and lower extremities or between the right upper extremity and all other extremities (i.e., left upper extremity and lower extremities) • Diaphoresis	• Focused physical assessment: heart murmur (though lack of heart murmur does not exclude congenital heart disease); hepatomegaly • Echocardiogram: obstructive cardiac or aortic lesion

CHAPTER 8

Arrhythmias

Introduction

Cardiac rhythm disturbances are relatively uncommon in children. When they do occur, they can be either a primary arrhythmia or an arrhythmia resulting from an underlying illness or condition. Some arrhythmias result in the loss of a pulse, whereas with others, a pulse is maintained. The most common arrhythmias in children—sinus tachycardia, supraventricular tachycardia (SVT), and bradycardia—typically fall into the latter category. Although they may adversely affect perfusion, they are rarely life threatening when promptly treated. As is the case with respiratory emergencies and shock, the key factors are to effectively assess the patient for the signs and symptoms of an arrhythmia, to recognize the abnormal rhythm quickly, and to provide immediate and appropriate care to promote hemodynamic stability.

Cardiac Conduction System Overview

The cardiac conduction system comprises 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 8-1). Under normal circumstances, the SA node, located in the upper right atrium, generates the electrical impulses that set the rhythm and rate of the heart (Figure 8-2). 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 and protecting the ventricles from atrial tachyarrhythmias. 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.

In the event of SA node dysfunction or failure, the AV node can function as a backup pacemaker. In a similar way, the cells of the AV junction (the zone of tissue surrounding the AV node), the bundle branches and the ventricles also can generate impulses to maintain some level of contraction and cardiac output.

Refer to Chapter 7, Shock, to learn more about normal cardiac function and circulation.

Overview of Cardiac Arrhythmias

A heart rhythm that originates from the SA node and follows normal conduction pathways is referred to as normal sinus rhythm (NSR). Any heart rhythm or rate that is not NSR is called an arrhythmia. Primary arrhythmias occur in children with anatomically normal hearts and reflect an intrinsic and often inherited abnormality of electrical cardiac conduction. Secondary arrhythmias occur because of diseases affecting the heart or causing metabolic abnormalities, including:

- Congenital heart defects.
- Cardiac surgery or cardiopulmonary bypass.
- Cardiomyopathy.
- Cardiac tumors.
- Inflammatory heart conditions (e.g., Kawasaki disease, myocarditis).
- Pulmonary hypertension.
- Electrolyte imbalances (especially those involving potassium, magnesium, or calcium).
- Acidosis.
- Hypoxia.
- Hypotension.
- Drug effects (e.g., vasoactive agents at therapeutic doses, digoxin or tricyclic antidepressant toxicity).

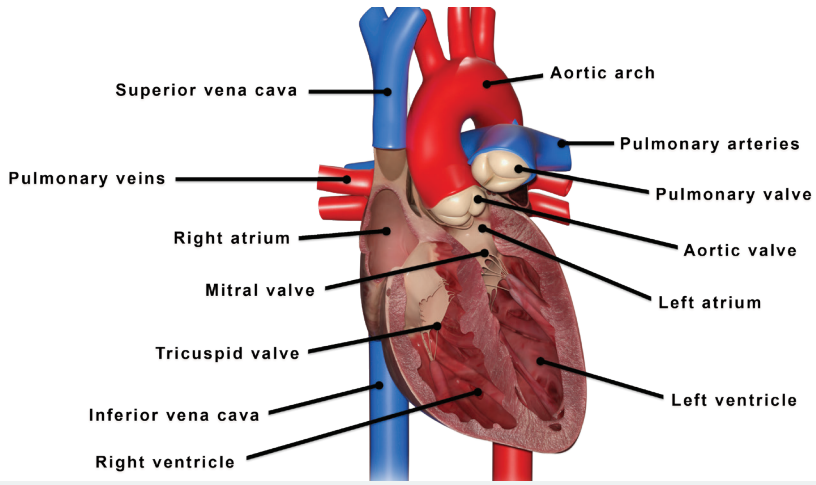


Figure 8-1 | Components of cardiac conduction system

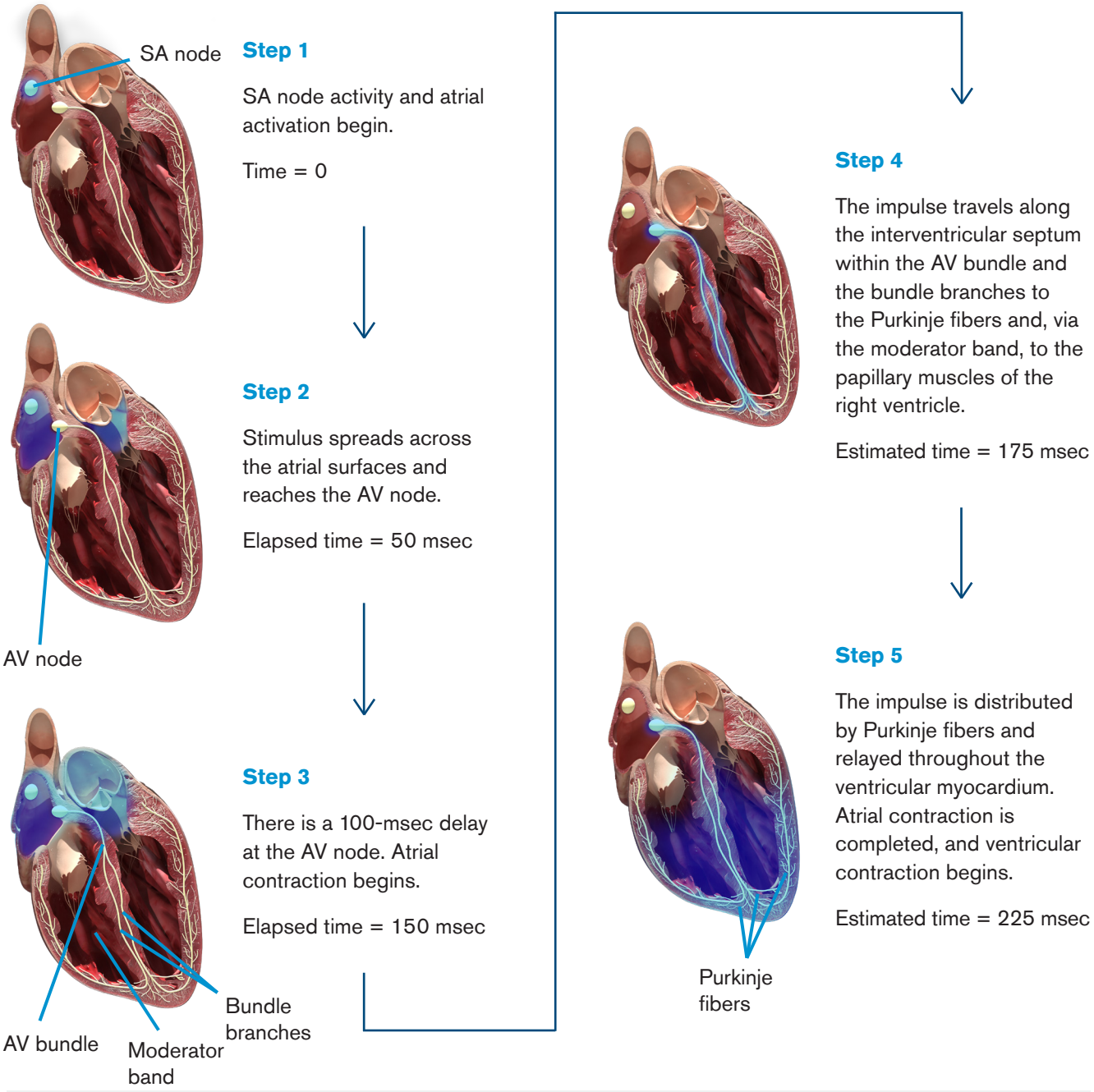


Figure 8-2 | Electrical impulse conduction through the heart

Practice Note

A primary arrhythmia may occasionally "masquerade" as a secondary arrhythmia. For example, a primary arrhythmia may underlie or be triggered by a choking episode or near-drowning event, yet the arrhythmia might be presumed to be secondary (e.g., due to asphyxia). Identification of such cases is important because the treatment may differ substantially based on the type of arrhythmia.

Arrhythmias can be classified in several ways, including based on heart rate (i.e., too slow or too fast). Arrhythmias that are characterized by an abnormally slow heart rate are collectively referred to as bradyarrhythmias. Arrhythmias that cause an abnormally fast heart rate are collectively referred to as tachyarrhythmias.

> Some arrhythmias are well tolerated and self-limiting, whereas others may cause decreased cardiac output with associated symptoms, with the worst cases leading to sudden death.

Thus, pediatric arrhythmias with a pulse can be further characterized by whether perfusion is adversely affected (unstable) or not (stable).

Six pulse-preserving arrhythmias that are seen more commonly in pediatric patients and are discussed in detail in this chapter include the following:

- Bradyarrhythmias
  - Sinus bradycardia
  - Atrioventricular (AV) block
- Tachyarrhythmias
  - Sinus tachycardia
  - Supraventricular tachycardia (SVT)
  - Atrial flutter
  - Ventricular tachycardia (VT)

Assessing the Pediatric Patient with an Arrhythmia

A systematic approach and adherence to the concept of assessment, recognition and care is required when assessing the pediatric patient with an arrhythmia.

> Prompt assessment, recognition and care of a pediatric patient with a cardiac arrhythmia may prevent deterioration to hemodynamic instability, which if untreated can result in shock, heart failure or cardiac arrest.

Practice Note

Children with arrhythmias may not be immediately identifiable. Some common ways children may present with bradyarrhythmias and tachyarrhythmias include difficulty breathing or shortness of breath, lightheadedness, palpitations, fatigue or activity intolerance, syncope and decreased level of consciousness.

Rapid Assessment

Begin your systematic assessment by performing a rapid assessment. Start with a quick visual survey of the emergency situation before collecting data or providing care. This allows you to make sure that the environment is safe and to formulate an initial impression of the child or infant experiencing an emergency. An initial impression allows you to quickly recognize whether the patient is experiencing a life-threatening or a non-life-threatening condition, including life-threatening bleeding. During the visual survey, it also is important to quickly determine what additional resources you may need in the emergency situation.

Formulate an Initial Impression (PAT)

To form an initial impression of the patient, follow the Pediatric Assessment Triangle (PAT), which uses an A-B-C approach.

- **A**ppearance (TICLS): Assess appearance and responsiveness; observe muscle tone, interactivity (e.g., lethargic/fatigued), movement/gesturing, speaking or crying and demeanor (e.g., calm, anxious or irritable).
- Work of **B**reathing: Note work of breathing; check for patient positioning, audible breath sounds (normal or abnormal—e.g., stridor, wheezing or grunting), signs of increased work of breathing or respiratory distress (e.g., nasal flaring, using accessory muscles to breathe, intercostal, substernal or suprasternal retractions and/or managing secretions), appears to be breathing too vfast or too slow and signs of inadequate or absent respiratory effort.
- **C**irculation: Assess adequacy of circulation by assessing skin color and mucous membranes; check for pallor (or gray/dusky color), cyanosis, mottling, flushing and life-threatening bleeding.

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.

ALERT

If you observe that the child or infant is unresponsive during the appearance step of the PAT, check for responsiveness, breathing and pulse and provide immediate care as necessary. If you observe that the child or infant is responsive during the appearance step of the PAT, but you observe potential life-threatening airway, breathing or circulation compromise, provide immediate care as necessary before proceeding to the primary assessment. If the child or infant is responsive and is not experiencing life-threatening airway, breathing or circulation compromise, proceed directly to the primary assessment.

Primary Assessment

After completing the rapid assessment, conduct a primary assessment of the patient (ABCDE). This primary assessment enables you to collect physical and physiological data to facilitate recognition of an arrhythmia and, in some cases, the underlying cause of the patient's arrhythmia emergency (Figure 8-3).

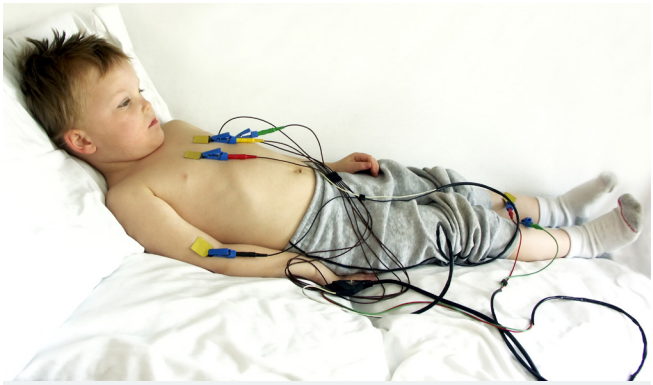


Figure 8-3 | The primary assessment enables you to collect physical and physiological data to facilitate recognition of an arrhythmia.

ALERT

As you perform the primary assessment, be alert for signs that the patient's condition has worsened or for any change in areas already assessed. Delegate necessary initial interventions to the appropriate team members so that immediate care can be implemented as you continue the primary assessment.

Airway

- Assess airway to determine patency. Listen and feel for movement of air by placing your ear close to the patient's nose and mouth. Observe for the rise and fall of the chest and/or abdomen with each breath.
- Determine the following: Is the airway clear and open? Is the airway obstructed but can be kept open with simple manual interventions? Is the airway not maintainable and is the use of CPAP, non-invasive ventilation or an advanced airway required?
- Maintain a patent airway as appropriate based on assessment findings.

Breathing

Assess the child's or infant's breathing to determine adequacy of ventilation and oxygenation:

- Look for increased work of breathing (e.g. nasal flaring, using accessory muscles to breathe, intercostal, substernal or suprasternal retractions) or respiratory distress, abnormal breath sounds (e.g., stridor, grunting, wheezing, gurgling), absent breath sounds, management of secretions.
- Note respiratory rate, depth and rhythm.
- Auscultate breath sounds (e.g., stridor, grunting, wheezing, crackles).
- Note voice or cry changes (e.g., hoarseness, hot potato voice).
- Measure oxygen saturation with pulse oximetry: Is the reading normal or abnormal?
- Measure $ETCO_2$ for intubated patients, and when available in non-intubated patients, measure $ETCO_2$. Is the reading normal or abnormal?
- Prepare supplemental O_2 and provide supplemental O_2 as appropriate based on assessment findings.
- If necessary, support breathing by delivering ventilations with a BVM resuscitator.
- Implement noninvasive or invasive ventilation as necessary.
- In the case of tension pneumothorax, perform immediate needle thoracentesis.

Circulation

- Assess the child's or infant's circulation to determine adequate perfusion of tissues.
- Palpate central and peripheral pulses.
- Measure the child's or infant's blood pressure.
- Connect the child or infant to a cardiac monitor to assess heart rate and rhythm.
- Review normal heart rate ranges by age (Table 8-1).
- When an arrhythmia is suspected (i.e., the cardiac monitor shows an abnormal heart rate or rhythm), the apical heart rate is counted for 1 full minute and compared with the radial rate. A consistently high or low heart rate for the patient's age should be regarded as suspicious.
- Obtain a 12-lead ECG for a full picture of the rate and rhythm of the heart (see Chapter 3, Tools and Therapies).
- Note skin and mucous membrane color, skin temperature and capillary refill time.
- Ideally, you should determine whether any physiological or physical abnormalities are attributable to the suspected arrhythmia or to another condition. If any abnormalities are attributed to the arrhythmia, investigate whether the patient is experiencing hemodynamic instability, as evidenced by hypotension or other signs of shock, syncope, shortness of breath or congestive heart failure.
- Prepare to achieve vascular access based on assessment findings.
- Prepare for fluid or medication therapy, if indicated.
- Prepare for electrical therapy if indicated.
- Monitor urine output (as appropriate).

Disability

- Assess neurological status to determine adequate brain perfusion.
- Check level of consciousness, pupillary response (PERRL) and blood glucose level.
- Use the following tools: AVPU, GCS and TICLS.

Exposure

- Assess the body overall, focusing on one area at a time.
- Look for abrasions, burns, bleeding, contusions (bruising), crepitus deformities, fractures, instability, lacerations, penetrations, petechiae and/or purpura, rashes, tenderness, abnormal skin temperature and color (to assess circulation and perfusion).
- Obtain the patient's weight and body temperature if not already done/available.
- If a head, neck, spinal or pelvic injury is suspected in the patient, consider spinal motion restriction.

Secondary Assessment

After the primary assessment is completed, if the patient's condition remains stable, perform a secondary assessment. This focused and detailed assessment will likely center around the cardiac system and its effects on the patient's vital functions.

The secondary assessment of a child or infant presenting with a possible arrhythmia will also include a focused patient history, physical assessment and laboratory and diagnostic tests.

Table 8-1 | Normal Pediatric Vital Signs

Age Group	Respiratory Rate	Awake Heart Rate	Systolic Blood Pressure	Diastolic Blood Pressure
Newborn	30 to 60	100 to 200	60 to 85	35 to 55
Infant (1 to 12 mo)	30 to 50	100 to 180	70 to 100	35 to 60
Toddler (1 to 2 yrs)	24 to 40	90 to 140	85 to 105	40 to 65
Preschooler (3 to 5 yrs)	20 to 30	80 to 130	89 to 115	45 to 70
School Age (6 to 12 yrs)	16 to 26	70 to 120	94 to 120	55 to 80
Adolescent (13 to 17 yrs)	12 to 20	60 to 100	110 to 135	60 to 85

Quick Assessment for Hypotension	
Age Group	**Systolic Blood Pressure**
Neonate	<60
Infant	<70
Toddler to School Age	<70 + (age in years x 2)
Adolescent	<90

> It is particularly important to gather patient history of underlying heart disease or cardiac surgery.

In addition to the 12-lead ECG that should be performed to investigate suspected arrhythmias, blood tests, such as CBC, complete metabolic panels, cardiac function tests (blood and diagnostic) and toxin screens may be ordered. A chest radiograph and/or an echocardiogram may be necessary to rule out underlying heart disease (congenital or otherwise). The findings of the patient history and laboratory and diagnostic studies may be used to determine the underlying cause of the arrhythmia and to guide care.

ALERT

Throughout the secondary assessment, it is important to keep the concept of assess, recognize and care in mind. Reassess the patient continuously for any changes in condition and assess the patient's response to any clinical interventions.

Focused History

- Assess signs and symptoms, allergies, medications, past medical history, last intake/output and events (SAMPLE).

Focused Physical Assessment

- Complete a physical assessment of the area of concern as determined by information gathered from the initial impression, primary assessment and focused history.
- Observe for signs and symptoms of pain.

Laboratory and Diagnostic Tests

- Perform laboratory and diagnostic tests to determine the presence, type and severity of an arrhythmia emergency.
- Determine specific tests and timing based on individual patient situation and status.

Recognizing Arrhythmias

Your first step is to recognize an abnormal heart beat, based on its rate, and then identify the specific rhythm. Recognizing the abnormality will determine the appropriate measures to correct the problem and maintain the patient's cardiopulmonary stability. In this step, you can identify potentially reversible causes of the arrhythmia to be addressed during care.

> Recognition of an arrhythmia is facilitated by familiarity with normal heart rates (dependent on the child's age) and the normal sinus rhythm of the heart.

To assess heart rate and rhythm, the child or infant is attached to a cardiac monitor. To analyze cases of suspected arrhythmias in-depth, a 12-lead ECG is performed. Knowing how to read and interpret the ECG rhythm strip is vital to accurately recognizing arrhythmias. See Chapter 3, Tools and Therapies, for a complete review on how to read and interpret an ECG strip.

Normal sinus rhythm (NSR) is the normal heart rhythm originating from the SA node (Figure 8-4).

In NSR you will find:

- Normal heart rate based on the child's age.
- Regular atrial and ventricular rhythms.
- P waves before each QRS complex.
- Normal PR interval based on the child's age.
- Normal QRS complex duration based on the child's age.

If the rhythm and/or rate is not normal, identifying the abnormality is important. Arrhythmias commonly seen in pediatrics are discussed in the next section.

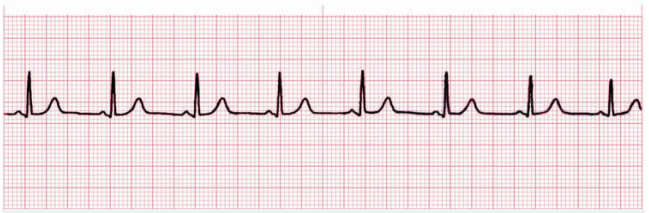


Figure 8-4 | Normal sinus rhythm strip

Practice Note

A slight irregularity in rhythm may occasionally occur that otherwise looks like NSR. This is commonly referred to as sinus arrhythmia, though it is not a true arrhythmia. Sinus arrhythmia reflects variation of the heart rate with breathing; inspiration increases the heart rate, and expiration slows it. This is a normal variant of NSR (Figure 8-5).

Recognition and determination of an arrhythmia begins with identifying the heart rate as being too slow (bradycardia) or too fast (tachycardia). Common bradycardias in children include sinus bradycardia—the third most common arrhythmia in children—and various types of atrioventricular (AV) block. Tachycardias fall into two categories based on the morphology of the QRS complex. Narrow-complex tachycardias are the most common arrhythmias seen in children (i.e., sinus tachycardia and supraventricular tachycardia). Wide-complex tachycardias that may be encountered in children include ventricular tachycardia (VT; in this case, with a pulse) and supraventricular tachycardia (SVT) with aberrant intraventricular conduction.

Bradyarrhythmias

Bradycardia is characterized by a heart rate that is slower than normal for age. Bradycardia can be a normal physiological finding (namely, in the case of sinus bradycardia) or may be pathologic. Pathologic bradycardia may arise from conditions directly affecting the heart's conduction system or from extrinsic factors. The pathology of bradycardia involves the sinus node, AV node, bundle of His or bundle branches.

Sinus Bradycardia

Sinus bradycardia has a rhythm identical to that of NSR, but the rate is slower than normal for the child's age. Sinus bradycardia may be a normal finding (physiological) in some patients, such as in adolescents who are well-conditioned athletes. In other patients, however, sinus bradycardia can be a response to hypoxia (the most common cause in children) or hypovolemia (i.e., secondary bradycardia). In addition, pediatric patients who have undergone cardiac surgery involving the atria may experience bradycardia. However, a primary cardiac cause of pathologic sinus bradycardia is very uncommon in children (Figure 8-6).

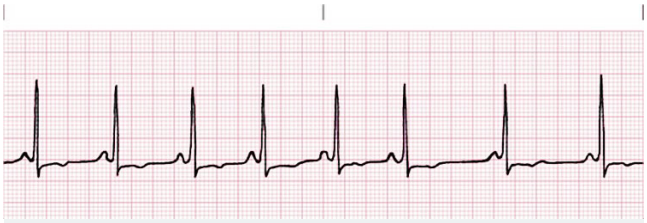


Figure 8-5 | Sinus arrhythmia rhythm strip

Causes

Causes of sinus bradycardia include:

- Vagal stimulation.
- Hypotension.
- Hypoxia.
- Hydrogen ion excess (i.e., acidosis).
- Hypokalemia or hyperkalemia.
- Hypothermia.
- Toxins (including prescription medications [i.e., beta-blockers, calcium channel blockers, opioids, digoxin] or recreational drugs).
- Hypothyroidism.
- Malnutrition (including anorexia nervosa).
- Conditions affecting the heart, including:
  - Congenital heart abnormalities.
  - Cardiac surgery.
  - Myocarditis.
- Increased intracranial pressure.

Electrocardiographic Findings

On an electrocardiogram (ECG), sinus bradycardia rhythm appears the same as sinus rhythm, except the heart rate is slower than normal (Figure 8-7).

Signs and Symptoms

Sinus bradycardia may not cause any signs or symptoms. However, when sinus bradycardia significantly affects cardiac output, leading to

Figure 8-6 | In children, sinus bradycardia can be a response to hypoxia.

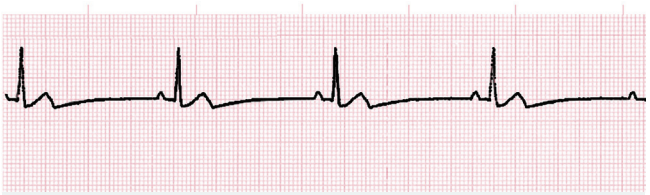


Figure 8-7 | Sinus bradycardia

hemodynamic instability, signs and symptoms of unstable bradycardia are evident, as shown in Table 8-2.

Atrioventricular Block

Atrioventricular 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.

Causes

Atrioventricular block has several pathologic causes, though it may be a completely normal finding in a healthy child. Causes include:

- Hypervagal tone.
- Myocarditis.
- Electrolyte abnormalities.
- Cardiac surgery.
- Medications.
- Inflammatory heart conditions.
- Myocardial infarction.
- Congenital complete heart block.

Classification

AV blocks are classified as first degree, second degree (type I and type II) or third degree. It is important to note that, although they are named in degrees, the block types first to third are not really a linear progression. Also, depending on the cause, AV block can be transient or persistent.

Table 8-2 | Primary Assessment Findings in Unstable Bradycardia

Airway	No changes
Breathing	Difficulty breathing
Circulation	Hypotension, diminished pulses, evidence of shock, cool extremities, mottled skin
Disability	Decreased level of consciousness, sudden collapse
Exposure	Cool extremities

First-Degree AV Block

First-degree AV block is characterized by delayed conduction at the AV node or bundle of His. The impulse is slowed but does reach the ventricles, resulting in a normal heart rate (Figure 8-8).

> **Practice Note**
>
> Although first-degree AV block is categorized within the discussion of bradyarrhythmias, it does not typically cause bradycardia.

First-degree AV block may be physiological in children with high vagal tone and in well-conditioned athletes, but it can also be an early indication of degenerative disease of the conduction system or a transient manifestation of electrolyte abnormalities, myocarditis, myocardial infarction, postsurgical damage to the AV node, rheumatic fever, Lyme disease or drug toxicity (e.g., digoxin, calcium channel or β-blockers).

Normal P waves that are all followed by QRS complexes characterize first-degree AV block, but because the impulse is held up at the AV node or bundle of His, the PR interval is longer than normal. Each P wave is linked to each QRS complex in a 1:1 fashion. A wide QRS complex points to a possible delay distal to the AV node. First-degree AV block rarely produces symptoms.

Second-Degree AV Block, Type I

Delay is a key feature of second-degree AV block type I (also referred to as Mobitz type I or Wenckebach block). Unlike in first-degree AV block, not all the impulses make it through to the ventricles. After three or four progressively longer delays, the next impulse is blocked. After the blocked impulse, the AV node resets and the pattern repeats.

Similar to first-degree AV block, this type of block may be a normal finding in children. Pathologic causes include increased vagal tone, congenital heart disease, cardiac surgery, myocarditis, drug toxicity and Lyme disease.

Because some impulses are not conducted through to the ventricles, the ratio of P waves to QRS complexes

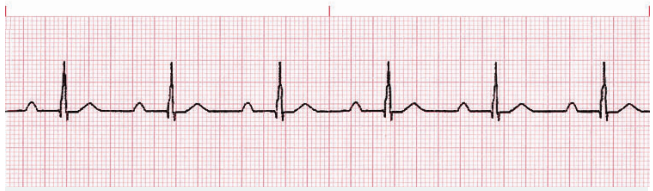


Figure 8-8 | First-degree AV block

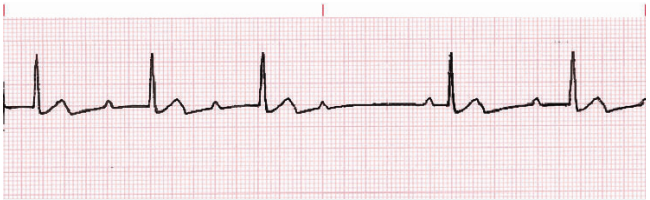


Figure 8-9 | Second-degree AV block type I

is greater than 1:1. Because each impulse is delayed a little longer than the previous one, 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"). After the dropped beat, impulse conduction through the AV node resumes and the sequence repeats itself. This is an example of a "regularly irregular" heart rhythm (Figure 8-9).

Second-degree AV block type I rarely produces symptoms. However, the patient may experience light-headedness.

Second-Degree AV Block, Type II

In second-degree AV block type II (Mobitz type II), the block occurs 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, but in this case the impulses are not progressively delayed (Figure 8-10). 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). This type of bock is characterized by a consistent PR interval. Because impulses are intermittently blocked, more P waves than QRS complexes are seen.

Second-degree AV block type II is usually pathologic and can be caused by fibrotic disease of the conduction system, cardiac surgery, myocardial infarction, increased vagal tone or drug toxicity. Patients may present with findings ranging from palpitations to poor perfusion. Some may be asymptomatic. The clinical presentation varies depending on the ratio of conducted to blocked impulses.

Third-Degree (Complete) AV Block

In third-degree (complete) AV block, no impulses are conducted through to the ventricles. This block can occur at or below the AV node but is usually below it. Pacemaker cells in the AV junction or the ventricles stimulate the ventricles to contract, usually at a rate of 30 to 45 beats/min. This means that the atria and ventricles are being driven by independent pacemakers and are contracting at their own intrinsic rates (i.e., 60 to 100 beats/min for the atria and 30 to 45 beats/min for the ventricles), a situation known as **AV dissociation**.

Third-degree AV block can be present at birth (congenital complete heart block) or occur later in life. Although congenital AV block is very rare, it occurs more often if the mother has systemic lupus erythematosus. It is also more common in infants born with a rare heart defect called *congenitally corrected transposition of the great arteries*.

Third-degree AV block can also occur after cardiac surgery, with a greater risk when the surgery involves areas that are close to the normal conduction system, as in repair of the cardiac valves and septum. Other causes of complete AV block include increased vagal tone, hypoxia, myocarditis and drug toxicities.

In third-degree AV block, no electrical communication occurs between the atria and ventricles, so no relation exists between P waves and QRS complexes. The R-R interval is constant. The QRS complexes will be narrow if ventricular contraction is stimulated by pacemaker cells in the AV junction. Impulses that originate in the ventricles produce wide QRS complexes (Figure 8-11).

If pacemaker cells above the bifurcation of the bundle of His stimulate ventricular contraction, the heart rate is relatively reliable and symptoms may be mild (such as fatigue, orthostatic hypotension and activity intolerance). If, however, pacemaker cells in the ventricles stimulate ventricular contraction, the heart rate will be slower and less reliable, and symptoms of decreased cardiac output may be more severe (such as syncope).

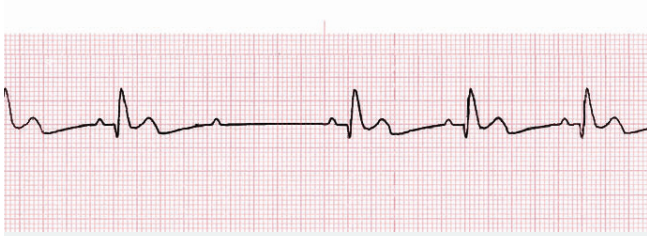


Figure 8-10 | Second-degree AV block type II

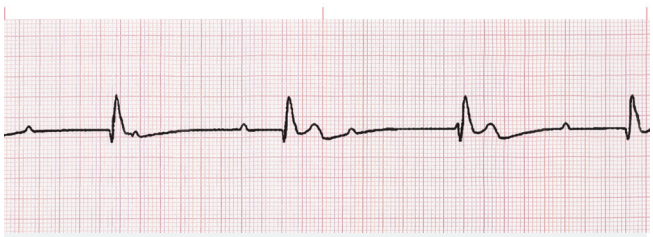


Figure 8-11 | Third-degree (complete) AV block

Tachyarrhythmias

Tachycardia is characterized by a heart rate that is faster than the upper limit of normal for age (Figure 8-12). Tachycardias account for most of the arrhythmias encountered in children, and sinus tachycardia accounts for approximately half of those cases.

> SVT is the most common symptomatic arrhythmia in children and accounts for approximately 10 percent of arrhythmias in children overall.

Tachycardias can be distinguished based on the width of the QRS complex—narrow or wide:

- Narrow complex (≤0.09 second): sinus tachycardia, SVT or atrial flutter
- Wide complex (>0.09 second): VT, SVT with aberrant intraventricular conduction

Narrow-Complex Tachycardias

Sinus Tachycardia

Sinus tachycardia is identical to NSR except the rate is faster than the normal heart rate for the child's age. It is a normal physiological response when the body is under stress, for example, due to fever, anxiety or pain. It may also be a compensatory response to pathologic stressors such as hypoxia, hypovolemia, anemia, shock or diseases of the heart or lungs. Other causes of sinus tachycardia include hyperthyroidism and drugs (both prescribed and illicit).

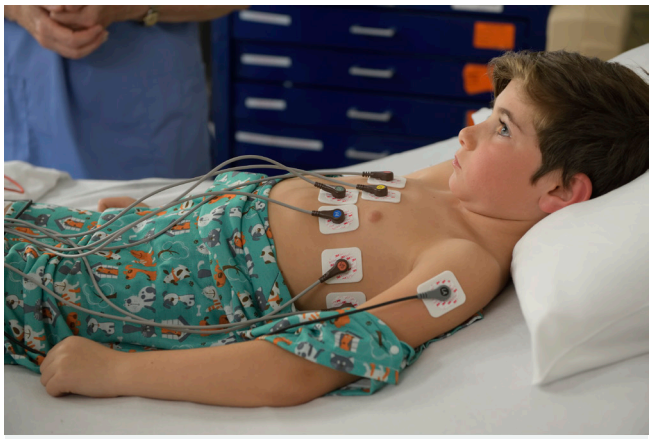


Figure 8-12 | Tachycardias account for most of the arrhythmias encountered in children.

Causes

Sinus tachycardia can have several causes:

- Vigorous physical activity
- Anxiety
- Pain
- Infection and fever
- Tissue hypoxia
- Hypovolemia (caused by fluid or blood loss)
- Anemia
- Shock
- Congestive heart failure/heart disease
- Medications (e.g., catecholamines)
- Illicit drugs (primarily stimulants)
- Pulmonary embolism
- Tension pneumothorax
- Pericardial tamponade

These and other causes (that result in increased cardiac output) should be ruled out before determining that a child's increased heart rate is a pathologic condition.

Electrocardiographic Findings

The ECG in sinus tachycardia (Figure 8-13) is morphologically similar to that for NSR. P waves and QRS complexes appear at a 1:1 ratio, and a normal P wave precedes every QRS complex, which are narrow. Beat-to-beat (R-R interval) variability may occur based on activity level or respiration. Although elevated for age, the heart rate in sinus tachycardia is typically <180 beats/min in children and <220 beats/min in infants.

Signs and Symptoms

Some patients with sinus tachycardia have no symptoms, and the condition is only discovered during a physical assessment or when the rapid heart rate appears on a cardiac monitor. Other patients may report the sensation of a rapid heart rate or palpitations, shortness of breath or light-headedness.

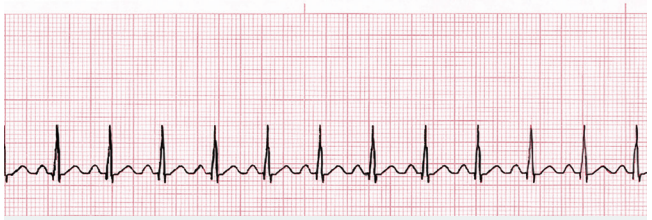


Figure 8-13 | Sinus tachycardia

> Hemodynamic instability may occur if the rhythm becomes very rapid or sustained.

Signs and symptoms of unstable sinus tachycardia are shown in Table 8-3.

Supraventricular Tachycardia

Supraventricular tachycardia (SVT) is a general term for tachycardias that originate above the ventricles (i.e., in the atria or the AV node). The most common forms of SVT in children are so-called re-entrant tachycardias involving an abnormal conduction pathway between the atria and ventricles or within the AV node. SVT is therefore often used synonymously with these re-entrant tachycardias. In these types of SVT, tachycardia occurs because of the presence of an "extra," or accessory, pathway for conduction between the atria and ventricles outside of the normal conduction pathway. This accessory pathway may be outside the AV node (AV re-entry tachycardia) or within the AV node (AV nodal re-entrant tachycardia). SVT is the most common symptomatic tachyarrhythmia found in children and refers to a rapid, regular heart rate usually exceeding 180 beats/min in older children and 220 beats/min in infants.

Causes

In children, about 50 percent of SVT cases are idiopathic, 25 percent are due to congenital heart disease, and 10 to 20 percent are attributable to a type of AV re-entry tachycardia known as Wolff-Parkinson-White (WPW) syndrome. Other causes, which make up the remaining percentage, include:

- Fever.
- Drug toxicity (e.g., stimulants, salicylates, tricyclic antidepressants).
- Anxiety.
- Anemia.
- Dehydration.
- Hypoxia.
- Acidosis.
- Hypoglycemia.
- Pain.

Table 8-3 | Primary Assessment Findings in Unstable Sinus Tachycardia

Airway	May or may not be patent
Breathing	Difficulty breathing
Circulation	Palpitations, hypotension, diminished pulses, delayed capillary refill, cool extremities, mottling
Disability	Light-headedness or syncope, decreased level of consciousness
Exposure	Cool extremities

Electrocardiographic Findings

The most common finding on an ECG is a series of narrow QRS complexes, typically with absent, or not clearly identifiable, P waves (Figure 8-14). Alternatively, the P waves may appear after the QRS complexes or may be retrograde in certain leads. If SVT is due to WPW syndrome, characteristic ECG findings after conversion to NSR include "slurring" of the upstroke of the QRS complex, known as a delta wave, which gives the PR interval a shortened appearance and the QRS complex a widened appearance (Figure 8-15). The rhythm is usually regular, with minimal to no beat-to-beat variability. The ventricular rate is extremely fast, typically exceeding 180 beats/min in older children and 220 beats/min in infants.

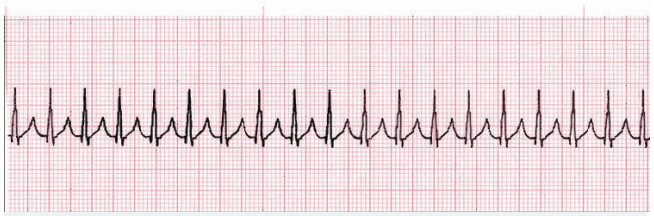


Figure 8-14 | Supraventricular tachycardia

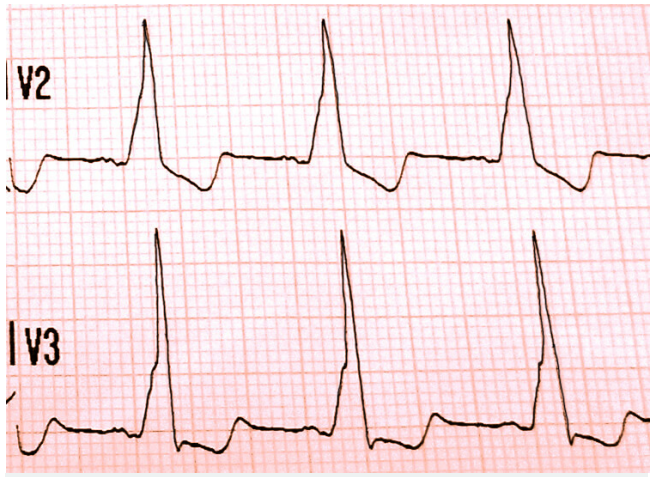


Figure 8-15 | Delta wave seen in WPW syndrome

Signs and Symptoms

SVT often has a sudden onset and a variable duration; the rhythm may end abruptly and convert back to NSR. Infants with SVT often present with nonspecific symptoms such as irritability, lethargy, poor feeding, pallor or sweating during feeding (Figure 8-16). In many cases these symptoms are manifestations of congestive heart failure resulting from a prolonged (>24-hour) period of SVT. By contrast, older children are often able to verbalize early symptoms attributable to SVT, such as palpitations, chest pain and shortness of breath; consequently, congestive heart failure rarely occurs in older children with SVT. Older children may also experience syncope, though it is rare and should raise suspicion for other arrhythmias when it occurs.

In SVT, the ventricles do not have sufficient time to refill before contracting again. Therefore, if SVT is sustained, cardiac output may be compromised and heart failure may develop. Possible signs and symptoms of unstable SVT are shown in Table 8-4.

Differentiating Sinus Tachycardia from SVT

It may be difficult to differentiate between sinus tachycardia and SVT, given their overlapping heart rate ranges and causes. Several features of the rhythm may be used to differentiate sinus tachycardia from SVT.

- Clinical features:
  - SVT may be paroxysmal (i.e., abrupt onset, rapid conversion back to NSR).
  - SVT is often accompanied by heart failure, particularly in infants with SVT for more than 24 hours, whereas sinus tachycardia may or may not be, depending on the underlying cause.

Table 8-4 | Primary Assessment Findings in Unstable Supraventricular Tachycardia

Airway	No changes
Breathing	Tachypnea, increased work of breathing, crackles or grunting if congestive heart failure develops
Circulation	Tachycardia, palpitations, chest pain, diaphoresis, hypotension, diminished pulses, delayed capillary refill, flushing (early), pallor (late), cool extremities, mottling or cyanosis
Disability	Poor feeding, irritability, light-headedness, decreased level of consciousness
Exposure	Cool extremities

Figure 8-16 | Infants with SVT often present with nonspecific symptoms such as irritability.

- Electrocardiographic features:
  - The rate for sinus tachycardia is rarely >180 beats/min for a child or >220 beats/min for an infant.
  - P waves are typically not clearly identifiable in SVT, whereas they are visible in sinus tachycardia.
  - The rate and R-R interval may vary for sinus tachycardia, whereas in SVT they typically do not vary.

Atrial Flutter

Atrial flutter is most often caused by a re-entry circuit within the atrial muscle, usually near the tricuspid valve separating the right atrium and ventricle. In atrial flutter, trial rates range from 240 to >300 beats/min, but the rate of ventricular contraction (and hence heart rate) vary. The AV node is unable to propagate all the atrial impulses coming its way, so typically some are blocked. This block usually occurs at a 2:1 ratio, meaning for every two atrial impulses, one makes it across the AV node and causes ventricular contraction. AV block ratios of 3:1 and 4:1 have been seen.

Causes

Among children, atrial flutter is most common during the fetal and newborn stages. It is rare beyond the newborn period and is almost always seen in children with underlying heart disease (such as congenital heart disease, especially after surgery). However, it may also be seen in Duchenne muscular dystrophy or following central nervous system injury.

Electrocardiographic Findings

In atrial flutter, atrial contraction occurs so quickly that the usual pattern of discrete P waves separated by a flat baseline is not seen on ECG. Instead, the rapid atrial contractions produce “flutter” waves, creating a characteristic “sawtooth” pattern. As previously

described, the ventricular response will vary depending on the AV block ratio (Figure 8-17).

Signs and Symptoms

Infants with atrial flutter may be asymptomatic unless they have been tachycardic for more than 48 hours. Symptoms occurring beyond that duration may include poor feeding, irritability, pallor or sweating. Older children may report palpitations, chest pain or light-headedness. Children with prolonged atrial flutter or with rapid ventricular conduction may present with hemodynamic compromise. Signs and symptoms of unstable atrial flutter are shown in Table 8-5.

Wide-Complex Tachycardias

Ventricular Tachycardia

Ventricular tachycardia (VT) is a wide–QRS complex tachycardia that occurs when a ventricular focus below the bundle of His becomes the new pacemaker. This rhythm is uncommon among the pediatric population. In VT, the ventricles may contract at a near normal rate or very quickly (>200 beats/min) but usually with a regular rhythm. A rapid ventricular rate significantly diminishes ventricular filling, stroke volume and cardiac output and can only be sustained for a short period before the patient becomes hemodynamically unstable.

> VT can quickly turn into ventricular fibrillation (VF), leading to cardiac arrest.

VT can arise from one (monomorphic) or multiple (polymorphic) ectopic foci in the ventricles. This distinction is important because it affects the electrocardiographic appearance and may alter treatment of VT in any given case.

Causes

VT usually occurs in the presence of heart disease or damage, heart surgery, myocarditis or cardiomyopathy.

Table 8-5 | Primary Assessment Findings in Unstable Atrial Flutter

Airway	No changes
Breathing	Shortness of breath
Circulation	Palpitations
Disability	Weakness or syncope, activity intolerance
Exposure	No changes

It may also be precipitated by drugs (such as antidepressants or stimulants), hypoxia or electrolyte imbalances (including hypocalcemia, hypomagnesemia and hypokalemia). A specific type of polymorphic VT called torsades de pointes is the prototypical rhythm accompanying a prolonged QT interval, whether congenital or acquired (i.e., due to drugs or electrolyte abnormalities such as hypomagnesemia or hypokalemia).

When you are collecting health history information, be sure to inquire about any family history of sudden, unexplained death in a child or young adult. Sudden cardiac death may indicate the presence of familial conditions that predispose the patient to ventricular arrhythmias, including certain types of cardiomyopathy or long QT syndrome.

Electrocardiographic Findings

VT usually can be recognized on the monitor. In some cases a 12-lead ECG may be needed to further define the rhythm. Lead V1 on a 5-lead ECG can also be used to differentiate a wide-complex tachycardia from other arrhythmias.

In VT, the QRS complexes are usually wide. The P wave is typically absent or not identifiable because it is buried in the wide QRS complex. There is AV dissociation if P waves are seen. In VT, the ventricular rate is between 120 and >200 beats/min (monomorphic VT) and 150 and 250 beats/min (polymorphic VT). Monomorphic VT is characterized by QRS complexes that have a generally uniform shape (Figure 8-18).

In polymorphic VT, the shape and rate of the QRS complexes vary. Torsades de pointes ("twisting of the

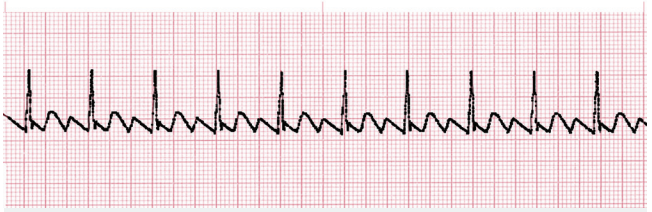


Figure 8-17 | Atrial flutter

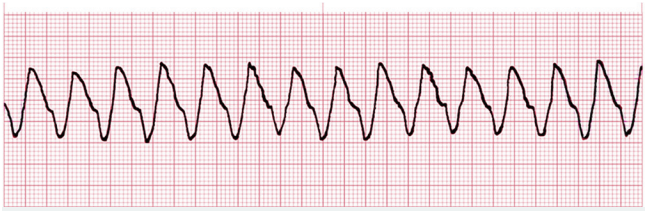


Figure 8-18 | Monomorphic ventricular tachycardia

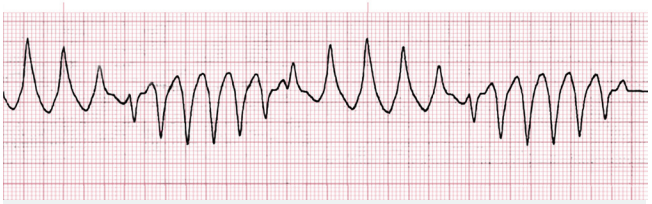


Figure 8-19 | Torsades de pointes

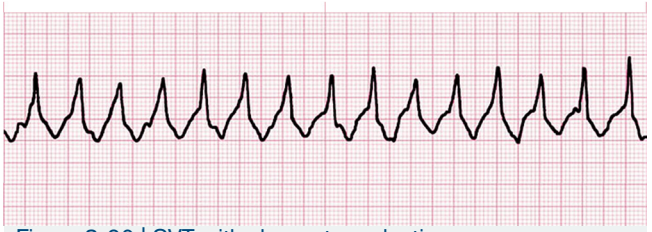


Figure 8-20 | SVT with aberrant conduction

points") is a common type of polymorphic VT. It has a readily identifiable "party streamer" shape (Figure 8-19).

The QRS complexes appear to oscillate around an axis, deflecting upward and then downward, with their amplitudes becoming smaller and larger, then smaller again. P waves are generally absent or not identifiable, and the QRS complexes are wide and difficult to measure. There is AV dissociation if P waves are seen.

Signs and Symptoms

With sustained VT, signs and symptoms of reduced cardiac output and hemodynamic instability may develop even as a pulse is maintained. Signs and symptoms of unstable VT with a pulse are shown in Table 8-6.

SVT with Aberrant Conduction

SVT with aberrant conduction is evidenced by a widening of the QRS complex (>0.09 second) as a result of conduction delays, blocks, or both, along the bundle branches or fascicles. This rhythm is uncommon among the pediatric population. SVT with aberrant conduction can occur secondary to a preexistent bundle branch block. SVT with aberrant conduction is characterized by wide-QRS complexes that are monomorphic with a regular R-R interval (Figure 8-20). P waves are generally absent or not visible. If P waves are visible, there is 1:1 AV conduction.

Table 8-6 | Primary Assessment Findings in Unstable Ventricular Tachycardia with a Pulse

Airway	May or may not be patent
Breathing	Difficulty breathing or no breathing
Circulation	Chest pain, hypotension, diminished pulses, diaphoresis, flushing (early), pallor (late), cool extremities, mottled skin
Disability	Decreased level of consciousness or loss of consciousness
Exposure	Cool extremities

SVT with aberrant conduction and VT share several similarities:

- Symptomatic hemodynamic instability
- Abnormally fast heart rates
- Widened QRS complexes

Because of these similarities, differentiating between these two arrhythmias can be difficult. While differentiating VT from SVT with aberrance is usually not important in emergent care, a 12-lead ECG and evaluation by someone skilled in its interpretation is usually needed to differentiate between the two rhythms. In addition, gathering health history information is important in determining the likelihood of this rhythm disturbance.

Caring for the Pediatric Patient with an Arrhythmia

For a child who presents with an arrhythmia with a pulse, initial care includes completing the rapid, primary and (if the patient's condition allows) secondary assessments (Figure 8-21). As with all pediatric emergencies, immediate actions include:

- Supporting the patient's airway through positioning or manual airway maneuvers.

Figure 8-21 | Caring for the pediatric patient with an arrhythmia begins with the rapid, primary and (if the patient's condition allows) secondary assessments.

- Providing supplemental oxygen and assisting with ventilation, if needed.
- Establishing cardiac monitoring (i.e., connecting the patient to a cardiac monitor).
- Monitoring the patient's blood pressure and oxygen saturation.
- Establishing intravenous (IV) or intraosseous (IO) access.

In addition, a 12-lead ECG should be obtained, and blood should be collected as soon as possible for use in any needed laboratory tests.

Practice Note

If obtaining a 12-lead ECG will delay important necessary care for the patient, it may be deferred. In most cases, you can use the cardiac monitor to observe heart rate and rhythm for a patient with an unstable rhythm.

Continuously reassessing the patient's cardiac rate and rhythm, as well as the patient's physical presentation, is important, especially after interventions are completed. This will help to determine whether treatment has been effective whether any sudden changes occur in the child's condition.

Refer to Chapter 3, Tools and Therapies, for more details on therapeutic interventions for arrhythmias (such as medications, vagal maneuvers and synchronized cardioversion).

Care for a Child or Infant with Bradycardia with a Pulse

Treatment depends on whether the patient is stable or unstable. If the patient does not show signs and symptoms of hemodynamic instability, their condition should be monitored.

Stable Sinus Bradycardia

Stable sinus bradycardia typically requires no immediate intervention beyond supportive care and treatment for potentially reversible causes. In cases of bradycardia caused by AV block, additional measures may be required for short- or long-term management, depending on the degree. Consultation with a cardiologist may be necessary and it is important to monitor the patient for clinical decompensation.

Unstable Bradycardia

Treatment should be initiated if the patient with bradycardia is hemodynamically unstable. It may be necessary to implement multiple treatment measures simultaneously. Assurance of adequate oxygenation and ventilation is the first-line intervention for unstable bradycardia. Additional interventions for unstable bradycardia may include CPR and pharmacologic therapies aimed at maintaining adequate cardiac output. Temporary electrical pacing may ultimately be required, particularly in patients with irreversible forms of AV block, until a permanent pacemaker can be surgically implanted.

> Throughout treatment for unstable bradycardia, work to determine the underlying cause of the bradycardia and continually reassess the patient's clinical condition. It is also important to consult with a cardiologist.

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.

In second-degree AV block type II and third-degree (complete) AV block, a pacemaker is indicated to regulate the heart rate and rhythm. Treatment is not necessary for the patient with second-degree AV block type I, unless the patient is symptomatic, in which case a pacemaker may be indicated. Patients may require and benefit from temporary electrical pacing before surgical implantation of a pacemaker and during symptomatic episodes.

See *Bradycardia with a Pulse* Treatment Guideline for a summary of care.

Care for a Child or Infant with Tachycardia with a Pulse

Important considerations in the care of a child presenting with tachycardia with a preserved pulse include the severity of the patient's signs and symptoms and the QRS complex duration (i.e., narrow versus wide).

If the patient does not show signs of hemodynamic instability because of the tachycardia, the patient is considered stable. In this case, next steps involve evaluating the ECG findings to determine whether the arrhythmia is narrow or wide and providing next steps of care.

A patient who shows signs and symptoms of hemodynamic instability despite initial management of airway, breathing and circulation is unstable and requires immediate lifesaving treatment.

Care for a Child or Infant with Stable Tachycardia with a Pulse

The care of a child with stable tachycardia with a pulse largely depends on the specific rhythm, which may initially be determined based on whether the QRS complex is narrow or wide.

Stable Narrow-Complex Tachycardia

Care of a child with a narrow-complex tachycardia with a pulse and adequate perfusion begins with supporting the airway, breathing and circulation as needed. The next step is to determine the actual rhythm (i.e., sinus tachycardia or SVT), because care will vary substantially on this basis.

Sinus Tachycardia

Specific arrhythmia treatment of sinus tachycardia is rarely indicated and in fact may be harmful, as tachycardia is often a compensatory response aimed at preserving cardiac output and tissue perfusion. Therefore, after confirming sinus tachycardia based on ECG findings, treatment should be aimed at the underlying cause of the arrhythmia.

Stable SVT

First-line treatment of stable SVT typically consists of vagal maneuvers that aim to slow conduction through the AV node (Figure 8-22). No guidelines specify how many vagal maneuvers to attempt before moving on to other therapies; however, most providers make a maximum of two attempts. See Chapter 3, Tools and Therapies, for more information regarding vagal maneuvers.

Figure 8-22 | First-line treatment of stable SVT consists of vagal maneuvers, such as blowing through an obstructed straw.

When vagal maneuvers are ineffective, pharmacologic treatment with adenosine using rapid bolus technique is initiated.

Note: It is important to use continuous ECG monitoring while administering adenosine. In addition, the patient may experience a brief period of "asystole" following administration. This is normal and typically self-limited.

Additional pharmacologic therapies or synchronized electrical cardioversion may be required if initial therapies prove unsuccessful.

Practice Note

If using an IV/IO catheter, attach a three-way stopcock to the hub of the catheter. Attach syringes containing the adenosine dose and a saline flush to the ports. Familiarize yourself with how to operate the stopcock to ensure a patent connection between each port and the patient; this procedure may vary depending on the particular type of stopcock. Turn the bar of the stopcock to first administer the adenosine by rapid push, then immediately turn the bar of the stopcock to administer the saline flush by rapid push.

Atrial Flutter

Treatment of stable atrial flutter consists of pharmacologic and synchronized electrical cardioversion with expert consultation.

Stable Wide-Complex Tachycardia

Care of a child with a wide-complex tachycardia with a pulse and adequate perfusion depends on the underlying rhythm. Pulse-preserving wide-complex tachycardias consist of VT and SVT with aberrant conduction.

Pharmacologic cardioversion and treatment of reversible causes typically compose the first-line intervention for stable VT with a pulse. Synchronized electrical cardioversion is a second-line or alternative therapy for VT with a pulse unless the patient becomes unstable. It is important to consult with a cardiologist for assistance with immediate or long-term treatment.

Pharmacologic treatment with adenosine may be considered in the case of a regular monomorphic wide-complex tachycardia (possible SVT with aberrant conduction).

Note: It is important to use continuous ECG monitoring while administering adenosine. In addition, the patient may experience a brief period of "asystole" following administration. This is normal and typically self-limited.

Treatment of reversible causes and additional pharmacologic or synchronized electrical cardioversion may be required if initial therapies prove unsuccessful.

It is important to consult with a cardiologist to assist with initial care. In addition, cardiologist involvement in long-term treatment is essential.

> **Practice Note**
>
> If unable to prove VT on the basis of criteria, and if the rhythm is wide, regular and monomorphic (all complexes are the same shape), consider administering adenosine.

See *Stable Tachyarrhythmia* Treatment Guideline for a summary of care.

Care for a Child with Unstable Tachycardia with a Pulse

A patient who shows signs and symptoms of hemodynamic instability (e.g., inadequate perfusion, weak pulses, decreasing level of consciousness, falling blood pressure) despite initial management of the airway, breathing and circulation is unstable and requires immediate lifesaving treatment.

Similar to treatment of stable tachycardia, treatment of unstable tachycardia with a pulse may vary based on the QRS complex duration and the specific rhythm. Focus on immediately restoring normal cardiac conduction (i.e., NSR) to prevent further hemodynamic decompensation or cardiac arrest.

Unstable Narrow-Complex Tachycardia with a Pulse

Beyond immediate lifesaving measures to support the airway, breathing and circulation, the treatment of unstable narrow-complex tachycardia varies based on the actual rhythm.

Unstable Sinus Tachycardia

Treatment for unstable sinus tachycardia is aimed at the underlying cause rather than at an attempt to suppress the abnormal rhythm, which is typically a compensatory response intended to preserve or augment cardiac output.

Unstable SVT

For treatment of unstable SVT, administration of adenosine (if IV/IO access is immediately available) and, if ineffective, synchronized electrical cardioversion are typically used early to quickly convert the rhythm and restore or improve cardiac output.

Atrial Flutter

Treatment of unstable atrial flutter consists of pharmacologic and synchronized electrical cardioversion with expert consultation.

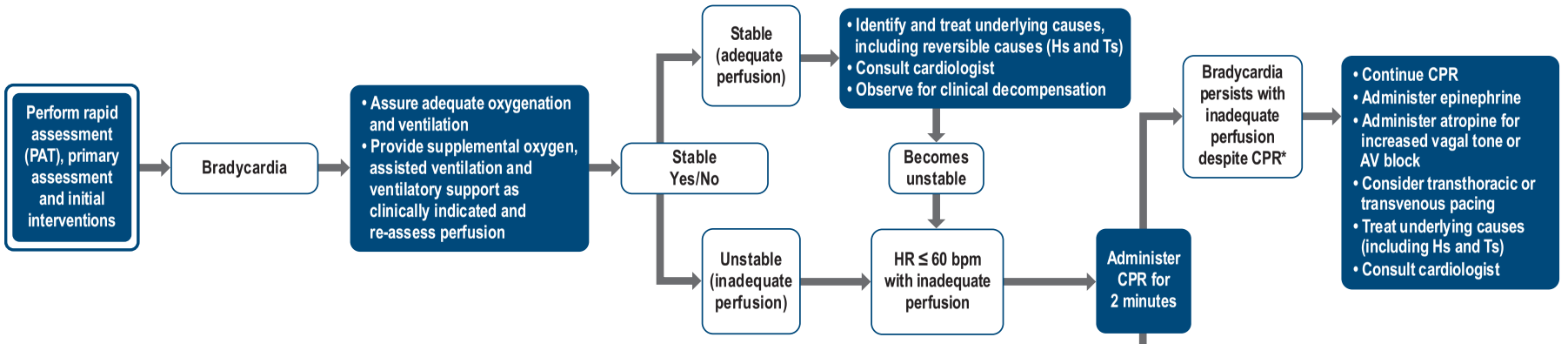
Unstable Wide-Complex Tachycardia with a Pulse

First-line treatment for unstable wide-complex tachycardias, including VT and SVT with aberrant conduction, consists of synchronized electrical cardioversion, particularly when signs of hemodynamic compromise are apparent. If the rhythm is consistent with SVT with aberrant conduction as evidenced by a regular rhythm and monomorphic QRS complexes, a dose of adenosine can be administered if doing so will not delay synchronized electrical cardioversion.

If unstable wide-complex tachycardias do not convert to normal sinus rhythm with synchronized electrical cardioversion, it is important to consult a cardiologist and consider pharmacologic cardioversion with either amiodarone or procainamide.

See *Unstable Tachyarrhythmia* Treatment Guideline for a summary of care.

BRADYCARDIA WITH A PULSE



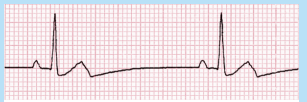
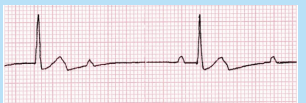
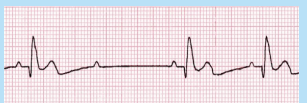
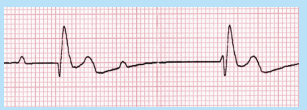
Epinephrine	Atropine
Dosage • 0.01 mg/kg IV/IO • 0.1 mg/kg via ETT, diluted in 5 to 10 mL of sterile water or NS directly into the ETT **Frequency** • May repeat every 3 to 5 minutes	**Dosage** • 0.02 mg/kg IV/IO (minimum, 0.1 mg; maximum, 0.5 mg); may repeat dose once after 3 to 5 minutes • 0.04 to 0.06 mg/kg via ETT

Stable Bradycardia	Unstable Bradycardia	Hs	Ts
Defined by signs of adequate perfusion, including: • Adequate pulses • Normal capillary refill • Normal level of consciousness • Normal BP for age	Defined by signs of inadequate perfusion, including: • Hypotension • Diminished pulses • Delayed capillary refill • Cool or mottled skin • Decreased level of consciousness	• Hypovolemia • Hypoxemia • Hydrogen ion excess (acidosis) • Hyper-/hypokalemia • Hypothermia • Hyper-/hypoglycemia	• Tamponade (cardiac) • Tension pneumothorax • Thrombosis (pulmonary embolism) • Thrombosis (myocardial infarction) • Toxins

Please see reverse side for additional information

*If at any point cardiac arrest develops see *Cardiac Arrest Pediatric Treatment Guideline*.

BRADYCARDIA WITH A PULSE CONTINUED

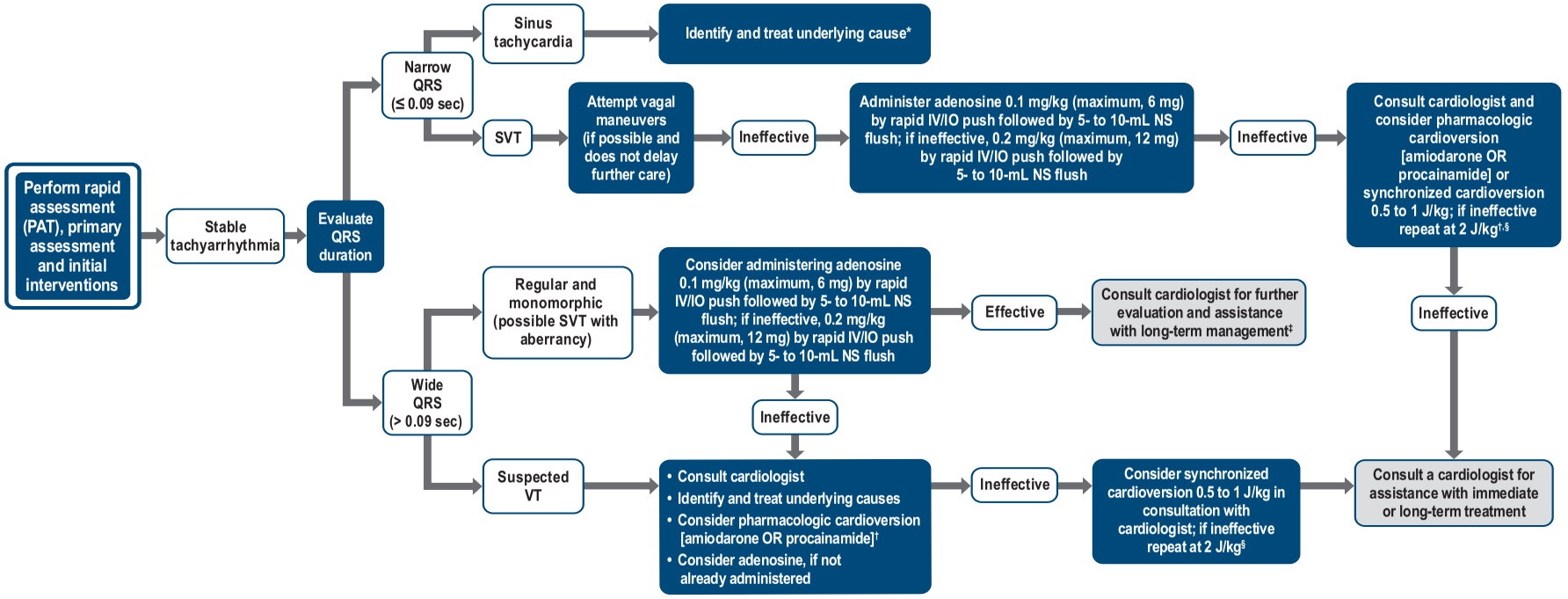
Bradyarrhythmia	ECG Features	
Sinus bradycardia	Identical to normal sinus rhythm, but HR is slower than normal for age	
Second-degree AV block type I	Repeated pattern of progressively delayed atrial conduction (prolonged PR interval) followed by completely blocked conduction (dropped beat)	
Second-degree AV block type II	Some atrial impulses conducted and others not, but no progressive delays; blocked impulses may occur in a pattern (e.g., 2:1; 3:1 or 4:1 in high-grade block)	
Third-degree AV block	No atrial impulses conducted to ventricles (AV dissociation)	

Age Group	Awake Heart Rate (Beats per Minute)
Newborn	100–200
Infant (1 to 12 months)	100–180
Toddler (1 to 2 years)	90–140
Preschooler (3 to 5 years)	80–130
School age (6 to 12 years)	70–120
Adolescent (13 to 17 years)	60–100

CPR Technique		Infant (< 1 Year Old)	Child (Age 1 Year to Onset of Puberty)
Switch CPR compressors • Every 2 minutes • If provider is fatigued		• **Hand position:** Fingers/thumbs centered on the lower half of the sternum just below the nipple line. Two-finger for single provider; encircling-thumbs for multiple providers • **Depth:** About 1½ inches • **Rate:** 100 to 120 per minute (single provider: 15 to 18 seconds for 30 compressions; multiple providers: 7 to 9 seconds for 15 compressions) • **Full chest recoil**	• **Hand position:** Centered on the lower half of the sternum • **Depth:** About 2 inches • **Rate:** 100 to 120 per minute (single provider: 15 to 18 seconds for 30 compressions; multiple providers: 7 to 9 seconds for 15 compressions) • **Full chest recoil**
		• Open airway to neutral position (avoid hyperextension)[a] • Each ventilation should last about 1 second and make the chest begin to rise	• Open airway to slightly past-neutral position (avoid hyperextension)[a] • Each ventilation should last about 1 second and make the chest begin to rise

[a]Use modified jaw-thrust maneuver instead if you suspect head, neck or spinal injury.

STABLE TACHYARRHYTHMIA



- If patient becomes unstable, see *Unstable Tachyarrhythmia Pediatric Treatment Guideline*
- If patient goes into cardiac arrest, see *Cardiac Arrest Pediatric Treatment Guideline*

Stable Tachycardia	Unstable Tachycardia
Defined by signs of adequate perfusion, including: • Adequate pulses • Normal BP for age • Normal capillary refill • Normal level of consciousness	Defined by signs of inadequate perfusion, including: • Diminished pulses • Hypotension • Delayed capillary refill • Cool or mottled skin • Decreased level of consciousness

Amiodarone and Procainamide Dosing and Administration	
Amiodarone	5 mg/kg (maximum, 300 mg) IV/IO over 20 to 60 minutes
Procainamide	15 mg/kg IV/IO over 30 to 60 minutes
Note: Do not administer amiodarone and procainamide together	

QRS Complex	
Narrow QRS	≤ 0.09 sec
Wide QRS	> 0.09 sec

Please see reverse side for additional information

*See *Potential Causes of Sinus Tachycardia*, on reverse side.

†See *Amiodarone and Procainamide Dosing and Administration* for dosing and administration instructions.

‡If monomorphic wide-QRS tachycardia converts with adenosine, do not presume SVT; consult cardiologist to exclude VT.

§If starting with 0.5 J/kg, may increase dose more gradually to 1 J/kg.

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STABLE TACHYARRHYTHMIA CONTINUED

Potential Causes of Sinus Tachycardia

- Vigorous physical activity
- Anxiety
- Pain
- Infection and fever
- Tissue hypoxia
- Hypovolemia (caused by fluid or blood loss)
- Anemia
- Shock
- Congestive heart failure/heart disease
- Medications (e.g., catecholamines)
- Illicit drugs (primarily stimulants)
- Pulmonary embolism
- Tension pneumothorax
- Pericardial tamponade

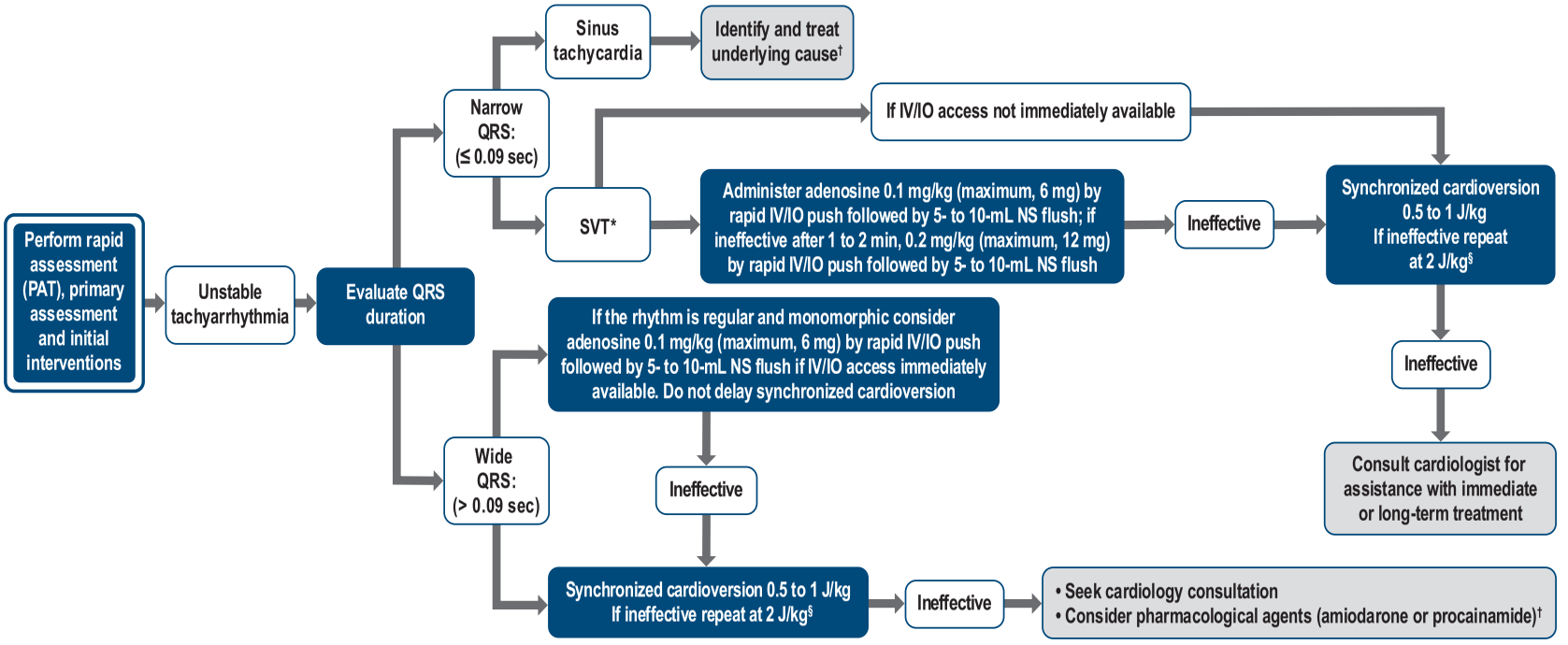
Guidance for Adenosine Administration

Patient should be on a monitor and have a rhythm strip run during adenosine administration	Administer using rapid bolus techniques followed by a 5- to 10-mL NS flush	Patient may experience a brief period of "asystole" following administration. This is normal and typically self-limited

Narrow QRS[a]		Wide QRS[a]	
Sinus Tachycardia	**SVT**	**Suspected VT with a Pulse**	**Possible SVT with Aberrancy**
On ECG: • Normal P-waves present before every QRS complex • Beat-to-beat variability (variable R-R interval) with constant PR interval **Clinical:** • HR usually < 180 bpm for children and < 220 bpm for infants • History or physical exam suggesting underlying cause (e.g., hypovolemia, fever, pain, drug toxicity)	**On ECG:** • P-waves absent or abnormal • Minimal to no beat-to-beat variability **Clinical:** • HR usually > 180 bpm for children and > 220 bpm for infants • Paroxysmal in nature • May have known history of heart disease or SVT	**On ECG:** • Wide QRS complexes (mono- or polymorphic) • Generally absent P-waves - AV dissociation if P-waves are seen **Clinical:** • History or physical exam suggesting underlying cause (e.g., heart disease, drug toxicity, electrolyte abnormalities) • Family history, if available, of sudden death or predisposing condition (e.g., long QT syndrome)	**On ECG:** • Wide QRS complexes (monomorphic) • Regular R-R interval • Generally absent P-waves - 1:1 AV conduction if P-waves are visible **Clinical:** • Possible history of prior events

[a]ECG and clinical presentations may include, but are not limited to, this information.

UNSTABLE TACHYARRHYTHMIA



If patient goes into cardiac arrest, see *Cardiac Arrest Pediatric Treatment Guideline*

Stable Tachycardia	Unstable Tachycardia
Defined by signs of adequate perfusion, including: • Adequate pulses • Normal BP for age • Normal capillary refill • Normal level of consciousness	Defined by signs of inadequate perfusion, including: • Diminished pulses • Low BP for age • Delayed capillary refill • Cool or mottled skin • Decreased level of consciousness

Amiodarone and Procainamide Dosing and Administration	
Amiodarone	5 mg/kg (maximum, 300 mg) IV/IO over 20 to 60 minutes
Procainamide	15 mg/kg IV/IO over 30 to 60 minutes
Note: Do not administer amiodarone and procainamide together	

QRS Complex	
Narrow QRS	≤ 0.09 sec
Wide QRS	> 0.09 sec

Please see reverse side for additional information

*Attempt vagal maneuvers if patient able/will not delay critical treatment.

†See *Potential Causes of Sinus Tachycardia* table, on reverse side.

‡See *Amiodarone and Procainamide Dosing and Administration* table for dosing and administration instructions.

§If starting with 0.5 J/kg, may increase dose more gradually to 1 J/kg.

UNSTABLE TACHYARRHYTHMIA CONTINUED

Potential Causes of Sinus Tachycardia

- Vigorous physical activity
- Anxiety
- Pain
- Infection and fever
- Tissue hypoxia
- Hypovolemia (caused by fluid or blood loss)
- Anemia
- Shock
- Congestive heart failure/heart disease
- Medications (e.g., catecholamines)
- Illicit drugs (primarily stimulants)
- Pulmonary embolism
- Tension pneumothorax
- Pericardial tamponade

Guidance for Adenosine Administration

Patient should be on a monitor and have a rhythm strip run during adenosine administration	Administer using rapid bolus techniques followed by a 5- to 10-mL NS flush	Patient may experience a brief period of "asystole" following administration. This is normal and typically self-limited

Narrow QRS[a]

Sinus Tachycardia

On ECG:

- Normal P-waves present before every QRS complex
- Beat-to-beat variability (variable R-R interval) with constant PR interval

Clinical:

- HR usually < 180 bpm for children and < 220 bpm for infants
- History or physical exam suggesting underlying cause (e.g., hypovolemia, fever, pain, drug toxicity)

SVT

On ECG:

- P-waves absent or abnormal
- Minimal to no beat-to-beat variability

Clinical:

- HR usually > 180 bpm for children and > 220 bpm for infants
- Paroxysmal in nature
- May have known history of heart disease or SVT

Wide QRS[a]

Suspected VT with a Pulse

On ECG:

- Wide QRS complexes (mono- or polymorphic)
- Generally absent P-waves
  - AV dissociation if P-waves are seen

Clinical:

- History or physical exam suggesting underlying cause (e.g., heart disease, drug toxicity, electrolyte abnormalities)
- Family history, if available, of sudden death or predisposing condition (e.g., long QT syndrome)

Possible SVT with Aberrancy

On ECG:

- Wide QRS complexes (monomorphic)
- Regular R-R interval
- Generally absent P-waves
  - 1:1 AV conduction if P-waves are visible

Clinical:

- Possible history of prior events

[a]ECG and clinical presentations may include, but are not limited to, this information.

CHAPTER 9

Cardiac Arrest

Introduction

A child or infant in cardiac arrest is in a life-threatening situation. It is essential to assess for, and recognize, cardiac arrest in children and infants and provide immediate resuscitation, including CPR and oxygen support, while determining the particular rhythm disturbance to guide care. Any delay in implementing CPR decreases the chance of survival for the child or infant in cardiac arrest.

Definition, Prevention and Common Causes of Cardiac Arrest

Definition and Prevention

Cardiac arrest is defined by the cessation of mechanical activity of the heart, as evidenced by the inability to palpate a central pulse. Sudden cardiac arrest from a primary arrhythmia such as ventricular tachycardia or fibrillation is rare in children and infants.

> In most cases, a child or infant experiences a respiratory emergency or shock before cardiac arrest. Recognizing the signs and symptoms of common causes of cardiac arrest may allow for intervention before cardiac arrest occurs.

Preventing cardiac arrest is the goal, when possible, as fewer than half of children who experience in-hospital cardiac arrest survive to discharge, despite improvements in resuscitative care (Figure 9-1).

Figure 9-1 | Outcomes following cardiac arrest in children are generally poor, so preventing cardiac arrest is the goal. When cardiac arrest does occur, providing high-quality CPR improves outcomes.

Practice Note

Outcomes following out-of-hospital cardiac arrest are generally poor, with pediatric survival to discharge at 3% to 10%. Outcomes following in-hospital cardiac arrest are generally better and have shown marked improvement over the last decade owing to emphasis on high-quality CPR and improved postresuscitation care. However, fewer than half of children experiencing in-hospital cardiac arrest survive to hospital discharge, with the highest rates reported in children whose cardiac arrest occurred in an intensive care unit (45% to 47%). Overall, these data highlight the importance of preventing cardiac arrest, when possible, and ensuring high-quality CPR when cardiac arrest does occur. (See *Learn More: Cardiac Arrest Survival Factors.*)

Common Causes of Cardiac Arrest

In children, cardiac arrest most often arises from respiratory failure or shock. Cardiac arrest in infants is nearly always due to respiratory failure or airway obstruction. Primary cardiac causes are rare in children and more commonly contribute to in-hospital cardiac arrest.

Common causes of out-of-hospital pediatric cardiac arrest include trauma, respiratory conditions and drowning. Sudden infant death syndrome is the most common cause of cardiac arrest in infants younger than 6 months of age. See *Learn More: Sudden Unexpected Infant Death*.

Respiratory Failure and Arrest

Any event or condition that interferes with normal breathing may lead to cardiac arrest. Common respiratory causes of cardiac arrest in children include:

- Drowning.
- Respiratory infections.
- Asthma.
- Smoke inhalation.
- Airway obstruction.
- Hanging (asphyxiation).

In each of these events or conditions, a lack of oxygen or excess of carbon dioxide can lead to a progressive decline in cardiac function, bradycardia, acidosis and hypotension. Cardiac arrest may follow if respiratory function cannot be rapidly restored.

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Cardiac Arrest Survival Factors

Location	The rate of survival to discharge is higher after in-hospital cardiac arrest than after out-of-hospital cardiac arrest for all age groups. Children with perioperative cardiac arrests that occur in the operating or recovery room have a significantly higher survival rate than those with arrest that occurs in other locations in and out of the healthcare facility.
Initial documented cardiac rhythm	Ventricular fibrillation or ventricular tachycardia at presentation is associated with better rates of survival to discharge than asystole or pulseless electrical activity.
Duration of compressions	The probability of survival and favorable neurologic recovery decreases exponentially in children who receive more than 15 minutes of compressions for an in-hospital cardiac arrest.
Prediction of neurological outcome	Prediction of neurologic outcome is an important consideration after cardiac arrest. Limited data suggest a potential role for electroencephalography (EEG) in the first 7 days after cardiac arrest. A continuous, reactive tracing correlates with a higher likelihood of favorable neurologic outcome at discharge. Pupil reactivity at 12 to 24 hours has been shown to be predictive of improved short-term survival and neurologic function in observational studies. In a similar way, preliminary data suggest a potential role for serum biomarkers of brain injury in predicting short-term survival and neurologic function.

Immediate identification and treatment of actual or impending respiratory failure and arrest is crucial to avert progression to cardiac arrest.

LEARN MORE

Sudden Unexpected Infant Death (SUID)

About 3,500 babies in the United States die suddenly and unexpectedly each year. The Centers for Disease Control (CDC) suggests using the term Sudden Unexpected Infant Death (SUID) to encompass all sudden infant deaths, many of which occur while the infant is sleeping or in their sleep area. This would include SIDS (Sudden Infant Death Syndrome), accidental suffocation or strangulation in a sleeping environment, and other, unexplained causes.

Visit the American Academy of Pediatrics' (AAP) website to view recommendations to reduce the risk of all sleep-related infant deaths.

Hypotensive Shock

In hypotensive or decompensated shock, cardiac arrest may result from insufficient blood flow to the heart muscle, and the heart may also be affected by acidosis and metabolic derangements. This is known as ischemic cardiac arrest. This type of arrest can also be seen with coronary artery problems, but shock is a much more common cause in children.

Table 9-1 | Conditions Predisposing to Arrhythmogenic Cardiac Arrest in Children

Congenital	■ Congenital heart defects ■ Coronary artery anomalies ■ Prolonged QT syndrome ■ Familial cardiomyopathies ■ Mitochondrial diseases
Acquired	■ Acquired cardiomyopathies (e.g., infectious, toxin related) ■ Drugs (especially stimulants) ■ Abnormalities in electrolyte levels (e.g., hyperkalemia) ■ Commotio cordis ("concussion" of the heart caused by blunt trauma to the chest)

Sudden Ventricular Arrhythmias

Cardiac arrest due to a sudden ventricular arrhythmia (not secondary to another condition such as hypoxia or acidosis)—so-called arrhythmogenic cardiac arrest—is rare in the pediatric population. It accounts for approximately 10 percent of cardiac arrests in children. Arrhythmogenic

cardiac arrest occurs most often in patients with pre-existing heart disease, often following cardiac surgery. Conditions that may predispose to arrhythmogenic cardiac arrest are summarized in Table 9-1.

Unfortunately, in some cases, cardiac arrest may be the first manifestation of a condition predisposing to ventricular arrhythmias. In other cases, warning signs and symptoms are present, including syncope, seizure-like episodes, chest pain, palpitations and difficulty breathing. On occasion, these signs and symptoms are wrongly attributed to other conditions. Syncope and seizure-like episodes may be assumed to be neurologic in origin, and dyspnea with exertion may be attributed to exercise-induced asthma. A family history of premature sudden death should raise suspicion for a structural or electrical heart disorder.

Reversible Causes of Cardiac Arrest

In all cases, reversible conditions that commonly contribute to cardiac arrest should be considered. These reversible causes are often referred to as the Hs and Ts of cardiac arrest. Treatment should be aimed at correcting these conditions. Reversible Hs and Ts of cardiac arrest, their possible causes and peri-arrest signs and symptoms are shown in Table 9-2.

Table 9-2 | Reversible Hs and Ts of Cardiac Arrest

Condition	Possible Causes	Peri-Arrest Signs and Symptoms
Hypovolemia	Bleeding, fluid losses or shifts out of the intravascular space	■ Hypotension, oliguria, cyanosis, tachypnea, confusion, decreased level of consciousness
Hypoxemia	Airway obstruction, lung disease, hypoventilation or apnea	■ Tachypnea, bradypnea or apnea, increased work of breathing (e.g., accessory muscle use, nasal flaring, grunting), stridor or wheezing, altered mental status ranging from irritability to unconsciousness, cyanosis
Hydrogen ion excess (acidosis)	Shock, diabetic ketoacidosis, hypoventilation, renal failure	■ Metabolic acidosis: coma, hyperventilation/hyperpnea, cardiac dysfunction, ventricular arrhythmia ■ Respiratory acidosis: tachycardia, tachypnea, hypertension, headache, confusion, drowsiness, stupor
Hyperkalemia (serum potassium >5.5 mEq/L)	Missed dialysis, renal failure, hypoaldosteronism (as in congenital adrenal hyperplasia)	■ Muscle weakness, flaccid paralysis, nausea, irregular heart rate/pulse; often asymptomatic
Hypokalemia (serum potassium <3.5 mEq/L)	Gastrointestinal or urinary losses, malnutrition, hyperaldosteronism (as in Cushing syndrome)	■ Muscle weakness, cramps or spasms, paralysis, hypoventilation, hypotension, confusion, paresthesia, drowsiness
Hypothermia (body temperature <35° C)	Traumatic injury with large-volume fluid resuscitation, cold-water drowning, exposure	■ Shivering (if temperature >31° C), bradycardia, bradypnea, nonreactive pupils, confusion, lethargy, coma
Hyperglycemia	Diabetic ketoacidosis	■ Nausea; vomiting; increased thirst and urination; lethargy; somnolence; coma; hypotension; tachycardia; rapid, deep respirations (Kussmaul breathing); fruity breath (due to exhaled acetone)
Hypoglycemia	Insufficient intake, too much insulin	■ Tachycardia, tachypnea, altered mental status ranging from irritability to coma, diaphoresis, hypothermia, pallor, seizures

Condition	Possible Causes	Peri-Arrest Signs and Symptoms
Tamponade (cardiac)	Trauma, infection, post heart surgery	■ Dyspnea, tachypnea, tachycardia, changes in mental status, hypotension, muffled heart sounds, jugular venous distension, pericardial friction rub, narrow pulse pressure, pulsus paradoxus
Tension pneumothorax	Trauma, certain lung diseases	■ Dyspnea, diminished or absent breath sounds, unequal chest expansion, hyperresonance to percussion on affected side, tracheal deviation (late sign), jugular venous distension, hypotension, pulsus paradoxus, anxiety, diaphoresis, hypoxemia, cyanosis, high peak inspiratory pressures (in patients receiving mechanical ventilation)
Thrombosis (pulmonary)	Deep vein thrombosis in the presence of certain risk factors (e.g., indwelling central venous catheter, recent surgery or trauma, clotting disorder)	■ Dyspnea, tachycardia, pleuritic chest pain, hemoptysis, signs of deep vein thrombosis (e.g., swelling, pain or erythema of affected extremity)
Thrombosis (coronary)	Kawasaki disease, familial or accelerated hypercholesterolemia, hypercoagulable state (e.g., nephrotic syndrome, inherited thrombophilia), coronary embolism (e.g., in cardiomyopathy, from intracardiac tumor)	■ Chest pain, nausea, vomiting, dyspnea, weakness, syncope, diaphoresis, pallor, cyanosis, thready pulse, hypotension, jugular venous distention in right ventricular infarction
Toxin	Illicit drugs (e.g., heroin, cocaine), medications (e.g., opiates, stimulants, sedatives/hypnotics, tricyclic antidepressants, digoxin, calcium channel blockers, beta-blockers)	■ Opiates: pinpoint pupils, reduced level of consciousness, slowed or absent respirations, cyanosis, hypotension, bradycardia, hypothermia ■ Stimulants: dilated pupils, tachycardia, hypertension, tachypnea, fever, diaphoresis, tremor, restlessness, paranoia, mania, seizures ■ Sedatives/hypnotics: hypotension, bradypnea, bradycardia, confusion, stupor, drowsiness, slurred speech, coma, **ataxia** ■ Tricyclic antidepressants: anticholinergic effects (e.g., dilated pupils, fever, delirium), seizures, vomiting, tachycardia, hypotension ■ Digoxin: nausea, vomiting, confusion, visual changes (e.g., blind spots, altered color perception), reduced level of consciousness, tachycardia ■ Calcium channel blockers: hypotension, dyspnea, bradycardia, confusion, slurred speech, drowsiness ■ Beta-blockers: dyspnea, wheezing (in children with asthma), hypotension, bradycardia, nervousness, diaphoresis, fever, confusion, seizures

ALERT

The number of pediatric opioid ingestions requiring hospitalization nearly doubled between 2004 and 2015 in the United States. Nearly 30 percent of the children hospitalized for opioid ingestions during this period required naloxone.

Methadone and heroin ingestions were present among children between the ages of 1 and 17 years old. Healthcare providers should educate parents and guardians/caregivers of infants and children on the importance of the safekeeping of all medications at home, or anywhere children have access to them, and explain the hazards of accidental medication and drug ingestion.

Assessing the Pediatric Patient in Cardiac Arrest

Recognizing Cardiac Arrest

Prompt recognition of cardiac arrest is paramount so that immediate interventions to support circulation can begin.

> Cardiac arrest is identified by unresponsiveness, apnea (or only agonal breaths) and the absence of a palpable central pulse (e.g., carotid, femoral, or in infants, brachial).

If a pulse cannot be felt within 10 seconds, an unresponsive, apneic patient should be assumed to be in cardiac arrest, and CPR should be immediately initiated after calling for help as needed (Figure 9-2).

Cardiac arrest correlates with one of a few cardiac rhythms that are unable to sustain a sufficient cardiac output and, thus, a pulse. Identification of the causative rhythm is important because specific immediate interventions are needed in addition to CPR for certain cardiac rhythms. Rhythm identification may also provide insight into the underlying cause of cardiac arrest.

Figure 9-2 | Cardiac arrest is identified by unresponsiveness, apnea (or agonal breaths) and the absence of a palpable central pulse.

Rapid Assessment

- Using the Pediatric Assessment Triangle, perform a rapid assessment:
  - **A**ppearance: Assess the patient's appearance and level of responsiveness.
  - Work of **B**reathing: Evaluate the patient's work and quality of breathing using visual and auditory assessments. Evaluate for inadequate or absent respiratory effort.
  - **C**irculation: Assess skin and mucous membranes for pallor (or gray/dusky color), cyanosis, mottling or flushing. Also assess for severe, life-threatening bleeding.
- If a patient appears unresponsive, check for level of consciousness using the shout-tap-shout sequence.
- If there is no response, call for immediate help and alert your facility's resuscitation team. Gather emergency equipment for airway management and cardiopulmonary resuscitation.
- Simultaneously check for breathing and feel for a carotid pulse in children or a brachial pulse in infants. Check for at least 5 seconds, but no more than 10.
- If there is no breathing and no pulse, provide immediate basic life support (BLS) care, including CPR (compressions and ventilations).
  - Ensure that the patient is in a supine position, and, if not on a hard surface, place a CPR board underneath the patient and/or place the bed or crib in the CPR position in CPR-ready beds or cribs.
- Once the resuscitation team arrives, the team leader assigns key roles to team members (i.e., compressor, AED/defibrillator operator, airway manager/ventilator, data manager, medication administrator).

Primary Assessment and Initial Basic Life Support Interventions

Since the child or infant is in cardiac arrest, a typical ABCDE primary assessment is not possible or warranted. Instead, without interrupting CPR, conduct a modified primary assessment and implement immediate basic life support (BLS) interventions. Key actions include the following:

- **Ensure the delivery of high-quality CPR.** Monitor qualitative and quantitative indicators of CPR quality.
- **Attach the cardiac monitor/defibrillator or AED.** Identify the arrest rhythm and provide shocks if indicated.
- **Assist ventilation with high-flow, high-concentration supplemental oxygen.**
- **Establish vascular access.** Prepare to administer fluids, medications, or both, as indicated.
- **Obtain the patient's weight.** In children and infants, defibrillation and medication doses are typically based on weight. If a patient's weight is not known or cannot be quickly and easily measured, use a length-based resuscitation (e.g., Broselow) tape to estimate the patient's weight. The tape also provides predetermined defibrillation and medication doses correlating with the color block corresponding to the patient's length.

Practice Note

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

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 to 8 seconds (8 to 10 breaths/minute). Each ventilation should last approximately 1 second and make the chest begin to rise.

Secondary Assessment

The secondary assessment of a child or infant in cardiac arrest (performed if resources permit) focuses on identifying the cause of the cardiac arrest by performing a focused history and ordering laboratory and diagnostic studies to identify potential underlying causes of cardiac arrest (Hs and Ts).

The focused history can be obtained through collecting data from family members or the providers who were caring for the patient at the time of the arrest. Follow the SAMPLE mnemonic:

- **S**igns and symptoms
- **A**llergies
- **M**edications
- **P**ast medical history
- **L**ast intake and output (I&Os)
- **E**vents

Hs and Ts

Of particular importance is identifying details that could point to one of the Hs or Ts as an underlying cause, including 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.

Laboratory and diagnostic studies that may prove useful for identifying reversible underlying causes of cardiac arrest include arterial blood gases, a serum electrolyte panel, glucose levels, drug screen and bedside echocardiogram. Diagnostic studies should be obtained without interrupting resuscitative efforts. Laboratory and Diagnostic Findings for Reversible Causes of Cardiac Arrest (Hs and Ts) are shown in Table 9-3.

Recognizing Cardiac Arrest Rhythms

The predominant cardiac rhythms underlying pediatric cardiac arrest for both out-of-hospital and in-hospital cardiac arrest include asystole and pulseless electrical activity. These are seen in more than 60% to 80% of cases. Children presenting with ventricular fibrillation (VF) and pulseless ventricular tachycardia (pVT) as the initial rhthym occurs in only approximately 10% to 20% of both out-of-hospital cardiac arrest and in-hospital cardiac arrest.

Remember to consider the reversible Hs and Ts as potential causes of cardiac arrest rhythms.

Asystole

In asystole, there is no electrical activity and therefore no contraction of the heart. It typically occurs in the setting of severe hypoxemia and metabolic acidosis and as a terminal rhythm when resuscitative efforts have not been successful.

Table 9-3 | Laboratory and Diagnostic Findings for Reversible Causes of Cardiac Arrest (Hs and Ts)

Condition	Studies and Findings
Hypovolemia	■ Laboratory findings: elevated serum blood urea nitrogen-to-creatinine ratio, elevated urine specific gravity, hypoglycemia, hypokalemia with significant gastrointestinal losses
Hypoxemia	■ Arterial blood gas: PaO_2 <80 mmHg, possibly hypercarbia
Hydrogen ion excess (acidosis)	Metabolic acidosis: ■ Arterial blood gas: pH <7.35, decreased $PaCO_2$ (1.2 mmHg decrease for every 1 mEq/L decrease in HCO_3–) ■ Serum electrolyte levels: HCO_3– <24 mEq/L, normal or elevated anion gap ■ Other laboratory findings consistent with underlying cause (e.g., elevated serum or urinary levels of glucose and ketones in diabetic ketoacidosis, elevated lactate level in lactic acidosis) Respiratory acidosis: ■ Arterial blood gas: pH <7.35, $PaCO_2$ >40 mmHg, increased HCO_3– (1 to 2 mEq/L increase for every 10 mmHg increase in $PaCO_2$), possibly hypoxemia
Hyperkalemia	■ Serum potassium level >5.5 mEq/L ■ Other diagnostic findings consistent with underlying cause (e.g., abnormal blood urea nitrogen and creatinine levels or renal sonogram in acute kidney failure, low sodium level in conditions associated with hypoaldosteronism [e.g., congenital adrenal hyperplasia])
Hypokalemia	■ Serum potassium level <3.5 mEq/L ■ Other diagnostic findings consistent with underlying cause (e.g., metabolic alkalosis due to vomiting, hypomagnesemia, high thyroxine level in hyperthyroidism)
Hypothermia	■ Arterial blood gas (do not correct for body temperature): increased pH, decreased PaO_2 and $PaCO_2$ ■ Hyperglycemia or hypoglycemia, hyperkalemia or hypokalemia ■ Increased hematocrit (2% increase for every 1° C decrease in body temperature), unless hemorrhaging ■ Prolonged **prothrombin time** and **activated partial thromboplastin time** ■ Elevated creatine kinase or amylase levels
Hyperglycemia	■ Random plasma glucose level: >150 mg/dL in neonates; >200 mg/dL in infants, children and adolescents ■ Serum and urine ketones in diabetic ketoacidosis
Hypoglycemia	■ Plasma glucose level: <45 mg/dL in neonates; <60 mg/dL in infants, children and adolescents
Tamponade (cardiac)	■ ECG: possible electrical alternans (alternating, varying amplitude of QRS complexes ■ Chest radiograph: cardiomegaly, "water bottle"-shaped cardiac silhouette or pleural effusions ■ Echocardiogram: pericardial effusion and collapse of the right ventricle during diastole
Tension pneumothorax	■ Chest radiography: to confirm and localize tension pnuemothorax. Will reveal air in the pleural cavity, with contralateral deviation of mediastinal structures. ■ Bedside echocardiogram: separation between the visceral and parietal pleural layers
Thrombosis (pulmonary)	■ Bedside echocardiogram: dilated right ventricle; occasionally visible clot in the right ventricle, right ventricular outflow tract or pulmonary artery ■ Laboratory findings: elevated D-dimer, increased arterial-alveolar gradient on blood gas
Thrombosis (coronary)	■ Elevated serum markers of cardiac injury (e.g., creatine kinase-muscle/brain [CK-MB], troponin I and T) ■ Other diagnostic findings consistent with underlying cause (e.g., elevated cholesterol levels in familial hypercholesterolemia; increased white blood cell count, platelet count and other **acute phase reactants** in Kawasaki disease)
Toxin	■ Positive drug screen results (opiates, stimulants, sedative/hypnotics, tricyclic antidepressants) or supratherapeutic drug level (digoxin) ■ Plasma glucose level: increased (calcium channel blockers) or decreased (beta-blockers)

Causes

In children and infants, asystole is a common rhythm resulting from apnea or respiratory failure and may follow a period of bradycardia. Other potential causes include nonfatal drowning, narcotic drug overdose, hypothermia, hyperkalemia and pulmonary embolism. Asystole is often the terminal rhythm in untreated pVT or VF or when resuscitation efforts are unsuccessful.

ECG Findings

Asystole presents as a flat line on ECG (Figure 9-3). There are no discernible P waves or QRS complexes present.

ALERT

Confirm that the appearance of asystole on ECG is not due to a loose or disconnected lead.

Pulseless Electrical Activity

Pulseless electrical activity (PEA) is a term used to describe rhythms that are organized on ECG (e.g., the QRS complexes are similar in appearance) but, as the name implies, are insufficient to produce a pulse. In PEA, the heart's conduction system is functioning, but the myocardium is contracting too weakly, or not at all, to produce cardiac output, or there is insufficient volume to maintain cardiac output.

Causes

The most common causes of PEA in children are hypovolemia and hypoxemia. Other possible reversible Hs and Ts causes of PEA include hypothermia, tension pneumothorax, cardiac tamponade, electrolyte abnormalities and, rarely, pulmonary embolism.

ECG Findings

PEA encompasses any organized rhythm that would normally be associated with a pulse (e.g., normal sinus rhythm); therefore, ECG findings will vary. The rate may be slow, normal or fast, and the QRS complex may be narrow, normal or wide. However, there will be no correspondence between the rhythm on ECG and a palpable pulse. Figure 9-4 is an example of PEA; a sinus rhythm is seen on ECG, but there is no palpable pulse detected.

Figure 9-3 | Asystole rhythm strip

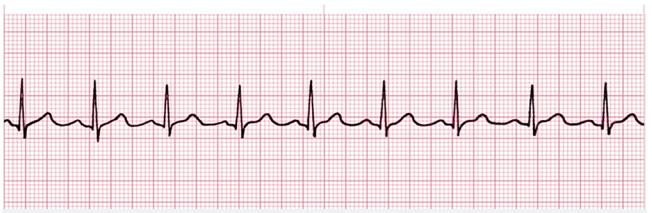


Figure 9-4 | In PEA, an organized rhythm is seen but there is no associated pulse

Ventricular Fibrillation

Ventricular fibrillation (VF) 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. Ventricular fibrillation is relatively rare in children and infants, though it may occur during resuscitation for more common rhythms (i.e., asystole and PEA) as reperfusion of the heart is established, and may occur more frequently but earlier in arrest than is recognized.

Causes

Causes of initial VF in children include pre-existing heart disease, acute respiratory failure, airway obstruction, electrolyte abnormalities, nonfatal drowning, drugs., tricyclic antidepressant overdose, renal failure with hyperkalemia or concussion of the heart (i.e., commotio cordis) caused by a sudden impact to the heart. Initial VF appears to have a better outcome than VF occurring in the course of resuscitation for other rhythms.

ECG Findings

On ECG, VF is irregular, with no discernible P waves, QRS complexes or T waves (Figure 9-5). The waveforms that are seen may vary in amplitude, from coarse to fine. As VF progresses, the waveforms may change from coarse to fine and eventually disappear (asystole).

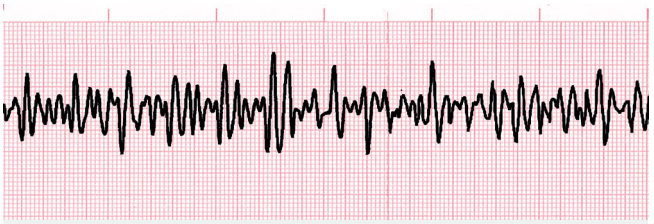


Figure 9-5 | VF rhythm strip

Pulseless Ventricular Tachycardia

In ventricular tachycardia (VT), the ventricles take over the role of the heart's pacemaker. Patients in VT may or may not have a pulse. Pulseless ventricular tachycardia (pVT) occurs when the ventricles do not contract effectively enough to sustain sufficient cardiac output. Sustained pVT may also deteriorate into VF.

Causes

Causes of pVT in children and infants include congenital heart disease, myocarditis, long QT syndrome, drug toxicity, hypoxemia, hypokalemia and hypomagnesemia.

ECG Findings

In pVT, the ventricular rate ranges from just above normal sinus rhythm to more than 200 beats per minute, and the QRS complexes are very wide.

Pulseless ventricular tachycardia can arise from one (monomorphic) or multiple (polymorphic) ectopic foci in the ventricles. Monomorphic pVT is characterized by QRS complexes that are generally uniform in shape, whereas in polymorphic VT, the QRS complexes vary in shape and rate (Figure 9-6).

Torsades de pointes ("twisting of the points") is a common type of polymorphic VT. It is a highly unstable form of VT that may revert to sinus rhythm or degenerate into VF. Torsades de pointes most often accompanies a prolonged QT interval, whether congenital (e.g., long QT syndrome) or acquired (e.g., due to drugs or electrolyte abnormalities such as hypomagnesemia or hypokalemia). Torsades de pointes has a readily identifiable "party streamer" shape on ECG (Figure 9-7).

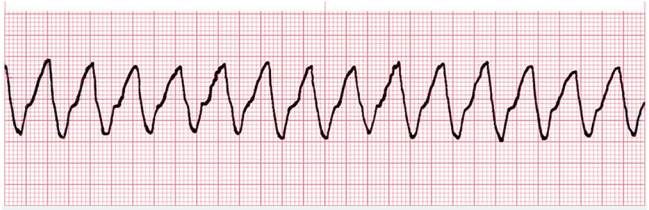


Figure 9-6 | pVT rhythm strip

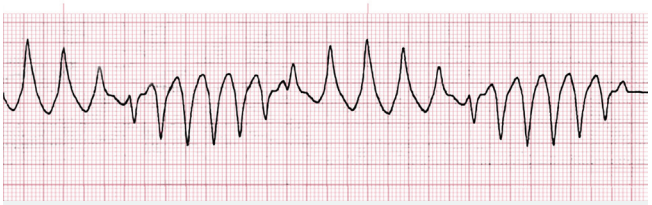


Figure 9-7 | Torsades de pointes rhythm strip

Caring for the Pediatric Patient in Cardiac Arrest

Key Steps in Caring for a Child or Infant in Cardiac Arrest

The initial steps in treating a child or an infant in cardiac arrest are the same regardless of the underlying cardiac rhythm or cause of cardiac arrest.

In cases of in-hospital cardiac arrest, CPR should be in progress when the pediatric advanced life support team arrives (Figure 9-8). Actions taken immediately to care for the pediatric patient in cardiac arrest include the following:

- Ensure the delivery of high-quality CPR.
- Manage the airway and provide effective ventilations.
- If not done so already, attach the cardiac monitor/defibrillator or AED and identify the arrest rhythm.
- Prepare for defibrillation.
- Establish vascular access.
- Prepare to administer medication therapy.
- Identify and treat reversible causes.
- Monitor for signs of return of spontaneous circulation (ROSC)

Figure 9-8 | CPR should be in progress when the pediatric advanced life support team arrives.

Key questions to answer in the assessment and treatment of a patient in cardiac arrest include:

- Is the rhythm shockable (ventricular fibrillation and pulseless ventricular tachycardia) or nonshockable (pulseless electrical activity and asystole)?
- What caused the patient's cardiac arrest?

> The initial steps involved in the care of a patient in cardiac arrest should be carried out simultaneously or in rapid succession when possible to expedite initiation of chest compressions, limit interruptions in CPR and, ultimately, delivery of more definitive therapies.

For example, once a pediatric advanced life support team is assembled, airway management and vascular access may be addressed simultaneously provided there is minimal interruption of chest compressions, defibrillation and ventilations (Figure 9-9).

Figure 9-9 | Immediate care for the pediatric patient in cardiac arrest includes ensuring the delivery of high-quality CPR, managing the airway and providing effective ventilations.

Administration of medication therapy and treatment of reversible causes, while very important, are not the priority. They are implemented after high-quality CPR and defibrillation (if appropriate) have been established.

For a complete review of high-quality CPR, BVM use, advanced airways, cardiac monitoring, vascular access, medication therapy and defibrillation, see Chapter 2, Basic Life Support Review, and Chapter 3, Tools and Therapies.

Ensure the Delivery of High-Quality CPR

Begin high-quality CPR starting with chest compressions immediately after determining that there is no pulse (or if unable to feel one within 10 seconds). Use an age-appropriate compression rate and depth as shown in Table 9-4.

As part of high-quality CPR, ensure the following:

- Allow the chest to recoil fully between compressions.
- Assess the adequacy of chest compressions as they are being administered, using at least one of the following methods:
  - Presence of a palpable pulse with each compression
  - An end-tidal carbon dioxide ($ETCO_2$) level between 15 and 20 mmHg in an intubated patient who can be monitored with capnometry
  - Presence of an arterial waveform in patients with indwelling arterial catheters

Practice Note

Optimal target values for blood pressure during CPR in children have not been ascertained.

- Interrupt chest compressions only for essential interventions (e.g., defibrillation) and to reassess for a perfusing cardiac rhythm.
- The provider performing chest compressions should be rotated *every 2 minutes* to avoid provider fatigue, which may adversely affect the quality of compressions.

Table 9-4 | Age-Based Compression Rate and Depth

	Infant (Birth Through Age 1)	Child (Age 1 Through Onset of Puberty)	Adolescent (Post-Onset of Puberty)
Compression rate	100 to 120 per minute	100 to 120 per minute	100 to 120 per minute
Compression depth	About 1½ inches or one-third the anterior-posterior diameter of the chest	About 2 inches or one-third the anterior-posterior diameter of the chest	At least 2 inches

Manage the Airway and Provide Effective Ventilations

Open the airway using manual maneuvers and airway adjuncts as needed. Administer two breaths using bag-valve-mask (BVM) ventilation with 100-percent oxygen for every 15 compressions (2 breaths for every 30 compressions for one provider) until an advanced airway can be established with minimal interruption of CPR. Provide smooth, effortless ventilations that last about 1 second each and make the chest begin to rise. Take care to avoid overventilating the patient, as this may reduce venous return to the heart and decrease blood flow to the heart muscle.

Practice Note

When a patient is in cardiac arrest, priority treatments are providing high-quality CPR, providing shocks (if the rhythm is shockable) and, if possible, determining and addressing the underlying cause.

If the airway and adequate ventilations can be maintained without placing an advanced airway (i.e., ETT), consider delaying placement. Expert clinical judgment and decision making by the team leader should be utilized when considering placement of an advanced airway. Careful planning and organization of equipment and team members is necessary in order to maintain high quality CPR and, most importantly, minimize chest compression interruptions.

If an advanced airway is established, ventilate at a rate of 1 ventilation every 6 to 8 seconds (8 to 10 breaths/min).

Practice Note

Remember that when an advanced airway is in place, chest compressions are performed continuously without pausing to deliver ventilations.

Attach the Cardiac Monitor/Defibrillator or AED

If not already done, connect the patient to a cardiac monitor/defibrillator or AED as soon as possible, minimizing any interruption in CPR. Identify the cardiac rhythm and determine whether it is a shockable rhythm (VF or pVT) or nonshockable rhythm (asystole or PEA).

Cardiac rhythms consistent with a shockable rhythm based on the following ECG findings:

- VF: irregular, with no discernible P waves, QRS complexes or T waves
- pVT: ventricular rate between just above NSR and >200 beats per minute, with wide QRS complexes that are uniform or variable in shape
  - Torsades de pointes: widened QRS complexes that appear to oscillate around an axis, becoming smaller and larger, and then smaller again (party-streamer shape)

Cardiac rhythms consistent with a nonshockable rhythm based on the following ECG findings:

- Asystole: flat line with no discernible P waves or QRS complexes
- PEA: rhythm that would normally be associated with a pulse (e.g., NSR)

Prepare for Defibrillation

A defibrillator should be immediately available in all cases of cardiac arrest. Operation of the defibrillator should be assigned to a team member who is skilled in its use. Preparation for defibrillation involves selecting the proper pad size for the patient and setting the proper mode and energy dose on the defibrillator.

Defibrillate the patient as soon as possible after a shockable rhythm (VF or pVT) is confirmed. Defibrillation doses for pediatric patients are as follows:

- First shock: 2 J/kg
- Second shock: 4 J/kg
- Subsequent shocks: ≥4 to 10 J/kg max, or the adult dose

Continue chest compressions should up to the moment of defibrillation and between defibrillation attempts.

Note: If a defibrillator is not immediately available or you are not authorized to use one, use an automated external defibrillator (AED). Because AED models function differently, follow your facility's protocols and the manufacturer's instructions for the AED you have.

ALERT

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.

Establish Vascular Access

If not already obtained, establish vascular access (IV or IO) as soon as possible without interrupting CPR. Vascular access is essential for administration of medications, fluids (as indicated) and collection of blood for laboratory studies.

Prepare to Administer Medications

The type and timing of medications administered during cardiac arrest will vary based on the underlying cardiac rhythm and, in some cases, the underlying cause. Medications can be administered via the IV, IO or ET route.

Medications used for the shockable rhythms VF and pVT include:

- Epinephrine.
- Amiodarone or Lidocaine.
- Magnesium sulfate (Torsades de pointes only).

Medication used for nonshockable rhythms asystole and PEA include:

- Epinephrine.

These commonly used medications (e.g., epinephrine, amiodarone, lidocaine) should be immediately available; ensure that they are included in the resuscitations team's crash cart.

> In children, medications are typically dosed based on weight.

If a child's weight is not known or cannot be quickly and easily measured, use a length-based resuscitation (e.g., Broselow) tape to estimate the child's weight. The tape also provides predetermined medication doses correlating with the color block corresponding to the patient's length.

Practice Note

Remember that in cardiac arrest, all IV/IO medications should be administered by rapid IV/IO push during compressions and followed by a 5- to 10-mL NS fluid bolus. This is done in order to ensure the IV/IO line is cleared of all the medication, and to move the medication into the central circulation quickly.

LEARN MORE

International Liaison Committee on Resuscitation Updates in Pharmacology for Cardiac Arrest

Vasopressors for Resuscitation	Data regarding the effectiveness of vasopressors, including epinephrine, in pediatric cardiac arrest are inconclusive. However, a randomized, prospective study of out-of-hospital cardiac arrest in adults found that epinephrine was associated with increased rates of ROSC compared with placebo. Epinephrine continues to be recommended as a reasonable treatment for pediatric cardiac arrest.
Antiarrhythmic Medications for Shock-Refractory Ventricular Fibrillation or Pulseless Ventricular Tachycardia	Amiodarone is recommended for treatment of shock- and vasopressor-refractory VF and pVT in children. However, recent observational data showed improved rates of ROSC and 24-hour survival among children who received lidocaine versus amiodarone for in-hospital arrest due to VF or pulseless VT. Accordingly, either amiodarone or lidocaine may be used for shock- and vasopressor-refractory VF and pVT.

For additional detailed information regarding medications used for shockable rhythms (VF and pVT) and nonshockable rhythms (asystole and PEA), reference Table 3-5 in Chapter 3.

See *Learn More: International Liaison Committee on Resuscitation Updates in Pharmacology for Cardiac Arrest*.

Identify and Treat Reversible Causes

Potentially reversible causes of cardiac arrest (the Hs and Ts) should be identified and treated as outlined in Table 9-5.

Practice Note

If opioid overdose is suspected to be the cause of cardiac arrest, initiate CPR and administer naloxone as soon as it is available, using the following dosing:

- 0.1 mg/kg (maximum, 2 mg), IV/IO/IM/SC

A second dose may be given after 2 minutes as needed.

It is important to note, when possible, if the patient is under any pain management therapy and taking prescribed opioids.

Table 9-5 | Treatment for Reversible Causes (Hs and Ts) of Cardiac Arrest

Condition	Treatment
Hypovolemia	■ Restore intravascular volume with fluids or blood. ■ Control fluid losses.
Hypoxia	■ Provide supplemental oxygen. ■ Ensure airway patency and adequate ventilation.
Hydrogen ion excess (acidosis)	■ Ensure effective CPR and oxygenation. ■ Consider bicarbonate as a buffer for prolonged resuscitations (i.e., >30 min).
Hyperkalemia	■ Administer IV calcium and bicarbonate if there is widened QRS complex on ECG. ■ Administer glucose (in the form of IV dextrose) and IV insulin in all cases to drive potassium into cells.
Hypokalemia	■ Administer potassium.
Hypothermia	■ Increase body temperature using warmed IV fluids, cardiopulmonary bypass or warm-fluid lavage of major body cavities (e.g., thorax, abdomen). ■ Continue CPR until core temperature increases.
Hyperglycemia	■ Restore intravascular volume with fluid resuscitation. ■ Administer IV insulin.
Hypoglycemia	■ Administer glucose in the form of IV dextrose.
Tamponade (cardiac)	■ Perform immediate pericardiocentesis. ■ Optimize intravascular volume while awaiting pericardiocentesis.
Tension pneumothorax	■ Perform immediate needle decompression followed by chest tube placement.
Thrombosis (pulmonary)	■ Consider thrombolytic agent if no contraindications. ■ Consider surgical or mechanical clot removal if pharmacologic thrombolysis is contraindicated. ■ Administer anticoagulant (typically heparin initially) in all cases.
Thrombosis (coronary)	■ Support cardiac function as needed with vasoactive therapy. ■ Consult pediatric cardiologist for possible angiography or other definitive evaluation and management.
Toxin	■ Administer antidote if available. ■ Support circulation with fluid resuscitation, vasopressor therapy and cardiopulmonary bypass if necessary. ■ Optimize electrolyte concentrations and correct abnormalities in glucose level (e.g., hypoglycemia in the case of beta-blockers).

Practice Note

Because shocks may not be successful or, in the case of successful defibrillation, the resultant rhythm may not be adequate to sustain perfusion or even a pulse, resume high-quality CPR immediately after each shock.

Monitor for Signs of Return of Spontaneous Circulation

The goal of care for the pediatric patient in cardiac arrest is return of spontaneous circulation (ROSC). It is important to monitor the patient for signs of ROSC, which include:

- Organized rhythm on the cardiac monitor/defibrillator combined with a palpable central pulse.
- Clinical signs of perfusion, including a sudden increase in the $ETCO_2$ level, measurable blood pressure and improved skin color.
- Additional signs, including patient movement, normal breathing or coughing, arterial pulse waveform on an a-line when no compressions are being delivered.

Specific Care for Shockable and Nonshockable Rhythms

Care for Shockable Rhythms: Ventricular Fibrillation or Pulseless Ventricular Tachycardia

If a shockable rhythm is identified, administer a shock as soon as possible. Provide chest compressions until defibrillation can be carried out and resume CPR immediately following delivery of the shock and between shocks.

Practice Note

Assessing for a pulse is not required when the rhythm is unorganized. However, it is very important to perform a pulse check, for no more than 10 seconds, when the rhythm is organized.

If a patient has an arterial line ("A-line") in place or $ETCO_2$ monitoring ongoing, you can also assess for evidence of perfusion through these physiologic feedback methods. Remember, if the rhythm is not an organized rhythm, resume CPR immediately.

After **every** 2 minutes of CPR, reassess the rhythm (while minimizing interruptions to chest compressions) to determine next actions:

- If the rhythm remains **shockable** (VF, pVT), resume CPR, administer a shock and then resume CPR immediately.
- If the rhythm is **nonshockable and unorganized** (asystole), resume CPR immediately and follow Care for Nonshockable Rhythms: Pulseless Electrical Activity/Asystole.
- If the rhythm is **nonshockable and organized**, attempt to palpate a central pulse:
  - If a definitive central pulse cannot be palpated, resume CPR immediately and follow Care for Nonshockable Rhythms: Pulseless Electrical Activity/Asystole.
  - If a definitive central pulse is palpated, provide post–cardiac arrest care.

Introduce medication therapy if initial defibrillation attempts are unsuccessful. If necessary, administer epinephrine after delivering two shocks, and then every 3 to 5 minutes throughout the resuscitation. Consider insertion of an advanced airway after delivery of 2 shocks. If necessary, administer amiodarone or lidocaine after delivering 3 shocks.

Consider and promptly address reversible causes of cardiac arrest (i.e., the Hs and Ts) throughout the course of resuscitation.

ALERT

Monitor closely for signs of ROSC, especially in a rhythm that goes from pVT to VT with a pulse or from PEA to a perfusing rhythm. It is very important not to make the mistake of failing to recognize ROSC and giving unnecessary interventions, which could be harmful after the patient achieves ROSC.

Care for Nonshockable Rhythms: Pulseless Electrical Activity or Asystole

Management of nonshockable rhythms focuses on:

- Delivering continuous high-quality CPR.
- Administering epinephrine every 3 to 5 minutes.

After **every** 2 minutes of CPR, reassess the rhythm (while minimizing interruptions to chest compressions) to determine next actions:

- If the rhythm is **shockable** (VF, pVT), follow Care for Shockable Rhythms: Ventricular Fibrillation or Pulseless Ventricular Tachycardia.
- If the rhythm is **nonshockable and unorganized** (asystole), resume CPR immediately and follow Care for Nonshockable Rhythms: PEA/Asystole.
- If the rhythm is **nonshockable and organized**, attempt to palpate a central pulse:
  - If a definitive central pulse cannot be palpated, resume CPR immediately and follow Care for Nonshockable Rhythms: PEA/asystole.
  - If a definitive central pulse is palpated, provide post–cardiac arrest care.

Consider insertion of an advanced airway. Consider and promptly address reversible causes of cardiac arrest (i.e., the Hs and Ts) throughout the course of resuscitation.

ALERT

Closely monitor patients for the development of a shockable rhythm, at which point defibrillation should be implemented as soon as possible.

See *Cardiac Arrest* Treatment Guideline for a summary of care.

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 defibrillation was provided.
- The patient's health status before 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). Nevertheless, in some situations (such as drug overdose, hypokalemia, hypothermia or pulmonary embolism) when it is necessary to buy time to address the underlying cause of the cardiac arrest, it may be appropriate to consider prolonging the resuscitation effort, using specialized interventions or both.

One such specialized intervention is the safe and effective use of extracorporeal membrane oxygenation (ECMO) in pediatric resuscitation—also known as **extracorporeal cardiopulmonary resuscitation (ECPR)**. Figure 9-10 shows how ECMO is used.

For patients who are good candidates for the procedure, survival and neurological outcomes may be improved with the use of ECPR (compared with outcomes

LEARN MORE

International Liaison Committee on Resuscitation (ILCOR) Update: Extracorporeal Cardiopulmonary Resuscitation for In-Hospital Cardiac Arrest

The safe and effective use of extracorporeal membrane oxygenation (ECMO) in pediatric resuscitation—also known as extracorporeal cardiopulmonary resuscitation (ECPR)—has been reported in registry and institutional case series, but this therapy carries added risks and healthcare costs.

Studies to date have shown no survival benefit for ECPR over CPR without ECMO for in-hospital cardiac arrest, except in a single study of children with cardiac conditions; however, the quality of this evidence overall is limited. Accordingly, ILCOR has suggested that ECPR may be considered in children and infants with cardiac diagnoses who experience cardiac arrest in a healthcare facility that possesses sufficient expertise and resources for using ECMO.

There is insufficient evidence to suggest for or against the routine use of ECPR in infants and children without cardiac diagnoses who experience in-hospital cardiac arrest.

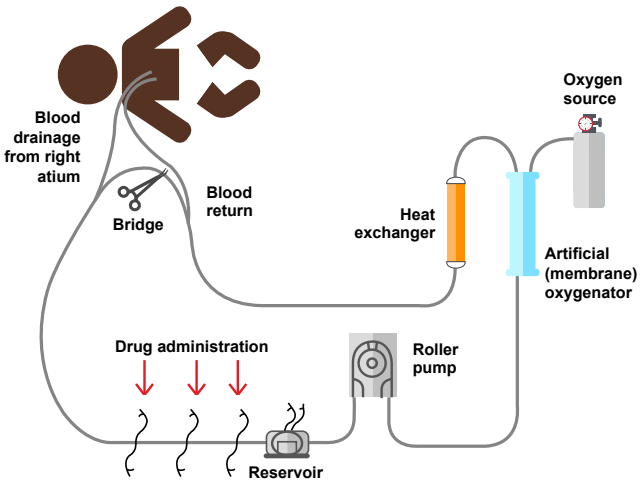


Figure 9-10 | For patients who are good candidates, extracorporeal membrane oxygenation (ECMO) may improve outcomes.

associated with standard CPR) (see *Learn More: International Liaison Committee on Resuscitation (ILCOR) Update: Extracorporeal Cardiopulmonary Resuscitation for In-Hospital Cardiac Arrest*).

Managing Special Cardiac Arrest Situations

Trauma

Trauma in children may result in a number of injuries that may lead to cardiac arrest. These include:

- Injuries to the lungs or tracheobronchial tree resulting in hypoxia.
- Hemorrhage, myocardial contusion or other chest injuries (e.g., cardiac tamponade, tension pneumothorax) resulting in impaired cardiac output.
- Spinal cord injury resulting in distributive shock and severe head injury.

As such, in children presenting with cardiac arrest after trauma, additional measures may need to be undertaken in addition to standard resuscitative measures to address specific injuries.

The airway should be cleared of any secretions, blood, vomitus or foreign bodies. Spinal motion restriction should be maintained until cervical spine injury has been ruled out, particularly during attempts to open the airway, during BMV or when attempting to intubate. But if one cannot manage the airway with spinal motion restriction, airway management is the priority.

Any external hemorrhage should be controlled using direct pressure, topical hemostatic dressings or, in the case of uncontrolled limb hemorrhage, tourniquets. Providers should consider the administration of tranexamic acid (TXA) preferably within the first three hours for pediatric patients who are actively bleeding as a result of trauma. TXA is an antifbrinolytic agent and is used to reduce blood loss. In addition, it is important to replace the blood lost. Administration of fluids and, as necessary, PRBC or whole blood transfusion should be initiated.

Open chest wounds should be covered with an occlusive dressing in the form of a vented chest seal. Immediate needle decompression of tension pneumothoraces should be performed, as should pericardiocentesis in the case of cardiac tamponade.

Fluid resuscitation and vasopressor therapy should be undertaken for shock based on the underlying type (e.g., hemorrhagic, obstructive, neurogenic). Hypothermia is treated or prevented using interventions such as removal of wet clothing, application of warm blankets and warming of administered IV fluids and blood products.

Children with multisystem trauma should ideally be transported to a designated pediatric trauma center.

Drowning

Various medical conditions may underlie a drowning event and should be considered in the drowning victim presenting in cardiac arrest. These include neurological emergencies, arrhythmias, hypoglycemia and drug use. Head or spinal cord injuries may underlie or occur concurrently with drowning and may be the primary cause of cardiac arrest. Such injuries should be considered in drowning events preceded by trauma (e.g., diving into shallow water, being ejected from a water craft) and in unwitnessed drownings.

Hypothermia often complicates drowning. Children are especially vulnerable to hypothermia because of their higher body surface area-to-mass ratio, among other factors. Initial treatment of hypothermia consists of removing wet clothing, applying warm blankets and administering warmed IV fluids and oxygen. In an unstable patient and those with severe hypothermia, more invasive measures, such as gastric, bladder, mediastinal or peritoneal lavage with warm fluids, may be warranted.

However, in hypothermic patients in cardiac arrest, ECMO is the preferred method for rewarming; therefore, prompt transfer of such patients to a center that performs ECMO is recommended, while ensuring continuous manual or mechanical CPR throughout transport. The optimal rate for rewarming is not known; rewarming rates ranging from 1° C per 5 minutes to

1° C per hour are commonly used. Once core temperature exceeds 28° C, defibrillation may be attempted in the patient with a shockable rhythm. Active rewarming should be continued until the core temperature exceeds 32° C. Termination of resuscitative efforts should not be considered in the patient with persistent cardiac arrest until core temperature reaches at least 32° C to 34° C.

Anaphylaxis

In children who experience cardiac arrest due to anaphylaxis, rapid restoration of a patent airway, vascular tone and intravascular volume may suffice to re-establish spontaneous circulation. If an advanced airway is indicated, assume that the procedure will be difficult; call for help as needed, and be prepared to insert a smaller ETT or, less often, a surgical airway if there is significant airway edema. Epinephrine should be administered and dosed as is standard for cardiac arrest rather than by the intramuscular route typically used in anaphylaxis presenting without cardiac arrest. (See *Cardiac Arrest* Treatment Guideline.) Other drugs that are typically administered during an anaphylactic episode, such as antihistamines and steroids, may be given but should not be prioritized over resuscitative measures or epinephrine administration.

Poisoning

Cardiac arrest resulting from poisoning may require additional care beyond standard resuscitative measures. Immediately contact the Poison Control Center (800-222-1222) for assistance with patient management. Antidotes may be available for certain drugs and should be administered as soon as possible. Reversible complications associated with specific drugs, such as hypoglycemia in the case of beta-blockers, should be treated to avoid their contribution to ongoing cardiac arrest.

Depending on the duration of the toxin's effect, prolonged resuscitative efforts may be required. Extracorporeal CPR may be warranted if myocardial function is sufficiently impaired by the offending toxin. A Poison Control Center or toxicologist should be consulted for assistance with management, particularly if termination of resuscitative efforts is being considered.

Single-Ventricle Congenital Heart Defect

Certain congenital heart defects such as hypoplastic left heart syndrome and tricuspid atresia result in a functionally single ventricle that must supply blood to both the body and the lungs. Surgical palliation of such defects occurs in stages and ultimately establishes passive blood flow from the vena cava to the pulmonary circulation so that the single ventricle need only pump blood to the body. Knowing the stage of palliation is important for any given patient with single-ventricle physiology experiencing cardiac arrest to determine appropriate resuscitative procedures and targets, including arterial O_2 saturation.

Standard resuscitation measures should be carried out in children and infants after stage I palliation. In children with suspected or potential occlusion of a systemic-to-pulmonary artery or right ventricle-to-pulmonary artery shunt following stage I repair, emergent surgical intervention including bedside surgery and/or heparin administration may be considered. Extracorporeal CPR may be considered in patients with single-ventricle physiology who have undergone stage I or stage III (Fontan) repair. There is insufficient evidence to support or refute the use of ECPR in patients with stage II physiology. End-tidal CO_2 may not reliably reflect the adequacy of CPR in later stages of surgical palliation because pulmonary blood flow does not reflect cardiac output.

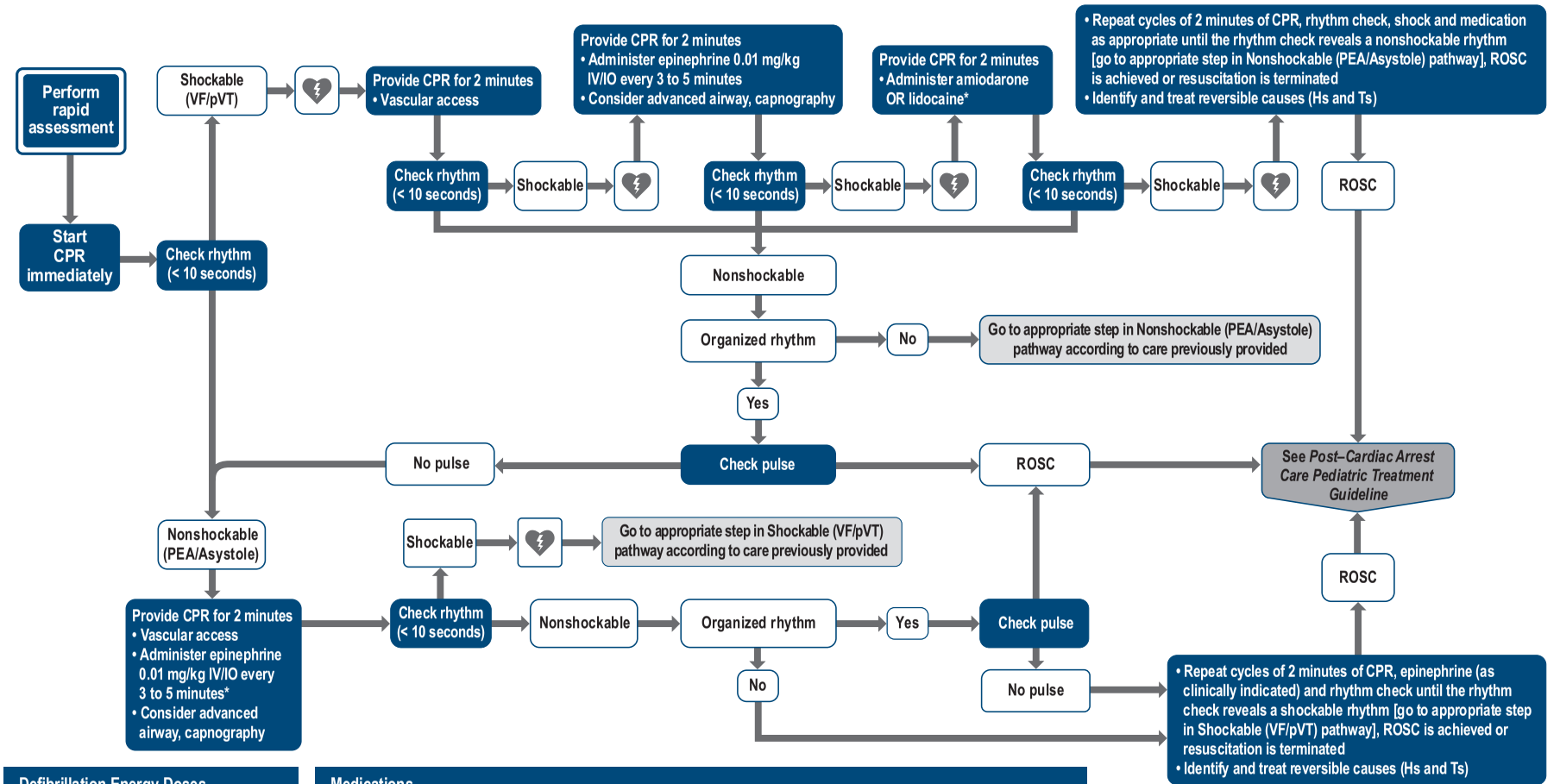
Pulmonary Hypertension

Pulmonary hypertension in children may be idiopathic or associated with congenital heart disease, connective tissue disorders or lung disease. Chronically high pulmonary arterial pressures may lead to right heart failure and death. Medical treatment is typically aimed at dilating the pulmonary vasculature.

Children with pulmonary hypertension are at increased risk for cardiac arrest. Standard resuscitative measures should be employed, attempting to correct or avoid hypercarbia, which causes pulmonary vasoconstriction and may thus exacerbate pulmonary hypertension. Adjustment of ventilation may be used to treat a pulmonary hypertensive crisis.

Any continuous vasodilators being administered before cardiac arrest, such as inhaled nitric oxide or IV prostacyclin and/or sildenafil, should be continued during cardiac arrest. Otherwise, initiation of inhaled nitric oxide or aerosolized prostacyclin should be considered. Treatment can include institution of these agents and sildenafil. Furthermore, mechanical right ventricular support with ECMO may be beneficial in children with pulmonary hypertension in cardiac arrest.

CARDIAC ARREST



Defibrillation Energy Doses
First shock 2 J/kg, second shock 4 J/kg, subsequent shocks ≥ 4 J/kg, maximum 10 J/kg or adult dose

Medications	
Amiodarone	• 5-mg/kg IV/IO bolus (maximum single dose, 300 mg) • May repeat up to 2 doses, for a maximum cumulative dose of 15 mg/kg over a 24-hour period
Epinephrine	• 0.01 mg/kg IV/IO every 3 to 5 minutes • 0.1 mg/kg via ETT, diluted in 5 to 10 mL of sterile water or NS directly into the ETT at the same times or sequence position as clinically indicated for the IV/IO route
Lidocaine	• Initial: 1-mg/kg loading dose IV/IO • Maintenance: 20 to 50 mcg/kg per minute infusion (repeat bolus dose if infusion initiated > 15 minutes after initial bolus therapy)

*For torsades de pointes, administer magnesium sulfate 25- to 50-mg/kg IV/IO bolus (2-g maximum dose).

Please see reverse side for additional information

American Red Cross
Training Services

CARDIAC ARREST CONTINUED

CPR Technique		Infant (< 1 Year Old)	Child (age 1 Year to Onset of Puberty)
Switch CPR compressors • Every 2 minutes • If provider is fatigued		• **Hand position:** Fingers/thumbs centered on the lower half of the sternum just below the nipple line. Two-finger for single provider; encircling-thumbs for multiple providers • **Depth:** About 1½ inches • **Rate:** 100 to 120 per minute (single provider: 15 to 18 seconds for 30 compressions; multiple providers: 7 to 9 seconds for 15 compressions) • **Full chest recoil**	• **Hand position:** Centered on the lower half of the sternum • **Depth:** About 2 inches • **Rate:** 100 to 120 per minute (single provider: 15 to 18 seconds for 30 compressions; multiple providers: 7 to 9 seconds for 15 compressions) • **Full chest recoil**
		• Open airway to neutral position (avoid hyperextension)[a] • Each ventilation should last about 1 second and make the chest begin to rise	• Open airway to slightly past-neutral position (avoid hyperextension)[a] • Each ventilation should last about 1 second and make the chest begin to rise

[a]Use modified jaw-thrust maneuver instead if you suspect head, neck or spinal injury.

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

CHAPTER 10

Post–Cardiac Arrest Care

Introduction

The prognosis for children for whom return of spontaneous circulation (ROSC) has been achieved after cardiac arrest is poor overall, although certain factors such as the location and duration of arrest significantly affect outcome. Healthcare providers can take specific measures to improve prognosis after cardiac arrest. This chapter discusses the goals of post–cardiac arrest care and explains how to care for a child or infant quickly and effectively after cardiac arrest.

Overview of Post–Cardiac Arrest Care

Care of a child who has experienced cardiac arrest extends beyond ROSC. The period after cardiac arrest may be characterized by various pathophysiologic abnormalities resulting from the cessation and restoration of circulation. These abnormalities are collectively known as post–cardiac arrest syndrome (PCAS). Attention to the management of PCAS is a central component of post–cardiac arrest care. The primary goal of such care is to minimize neurologic injury, thereby optimizing neurologic outcome after cardiac arrest.

Post–Cardiac Arrest Syndrome

Post–cardiac arrest syndrome encompasses both the ischemic injury that occurs during cardiac arrest and the reperfusion injury that follows ROSC. Components of PCAS include post–cardiac arrest brain injury, post–cardiac arrest myocardial dysfunction and a systemic response to ischemia/reperfusion (Table 10-1). PCAS is occasionally complicated by persistence of the pathologic process that caused the cardiac arrest in the first place.

Focus of Post–Cardiac Arrest Care

Care after cardiac arrest focuses on mitigating the components of PCAS. Specifically, the preservation of neurologic function is the primary goal and focus of post–cardiac arrest care. Management of other critical functions, including oxygenation, ventilation and circulation, is customized with this goal in mind. Minimizing injury to other organs is important but secondary.

> Optimization of oxygenation, ventilation and circulation is critical to ensure adequate oxygen delivery and blood flow to the brain and to mitigate the effects of the ischemia/reperfusion response.

In a similar way, neuroprotective strategies are a central component of post–cardiac arrest care. They include measures aimed at reducing the metabolic demand of the brain (e.g., seizure control, targeted temperature management, sedation) and controlling glucose. Assessments of prognosis become more reliable and assume a greater focus 72 hours after cardiac arrest.

Assessing the Pediatric Patient Post–Cardiac Arrest

Primary Assessment

Primary assessments applicable to the management of oxygenation and ventilation, circulation and neurologic function after cardiac arrest are summarized below (Figure 10-1).

Table 10-1 | Pathophysiology and Clinical Presentation of PCAS

Component	Underlying Pathophysiology	Clinical Manifestations
Brain injury	Impaired autoregulation of blood flow to the brain; brain swelling (edema); neuron degeneration	Seizures, cognitive dysfunction, coma, persistent vegetative state, brain death
Myocardial dysfunction	Usually reversible; global heart muscle dysfunction with low cardiac output and normal coronary artery blood flow (i.e., myocardial stunning)	Arrhythmia, cardiogenic shock
Systemic response to ischemia/reperfusion	Impaired tissue oxygenation, systemic inflammation, vascular instability and increased coagulation, adrenal suppression, lowered resistance to infection	Fever, hypotension, hyperglycemia, infection, multiorgan failure

Figure 10-1 | Care of the pediatric patient post–cardiac arrest focuses on monitoring and supporting the patient's airway, breathing and circulation.

Airway

- Assess airway to determine patency and reflexes in patients who are spontaneously ventilating and in patients receiving assisted ventilation.
- Is the airway not maintainable and is the use of an advanced airway required?
- If an advanced airway is in place, re-verify patency and proper positioning (look for bilateral chest rise and breath sounds with manual breaths, presence of exhaled CO_2 on capnometry). Suction as needed.
- If an advanced airway is in place, monitor the patient for clinical deterioration. The pneumonic DOPE can help you remember the most common causes of post-intubation hypoxia or deterioration:
  - **D**isplacement: Check the endotracheal tube for displacement (right mainstem) or dislodgement.
  - **O**bstruction: Check the ETT for obstruction (mucous plug, kink in ventilator tubing).
  - **P**neumothorax: Obtain a radiograph to assess for pneumothorax.
  - **E**quipment failure (unusual): Disconnect patient from the ventilator and bag manually.

Breathing

- If patient is breathing spontaneously, assess respiratory rate, effort and pattern.
- If patient is intubated and receiving assisted ventilation, assess chest rise with manual breaths.
- Assess for signs of respiratory compromise (e.g., increased respiratory rate, retractions, grunting).
- Monitor O_2 saturation by pulse oximetry.
  - Is the reading normal?
- Measure $ETCO_2$: For intubated patients, and when available in non-intubated patients, measure $ETCO_2$.
  - Is the reading normal or abnormal?
- Prepare supplemental O_2 and provide the minimal supplemental O_2 to keep O_2 saturation above 94%, as appropriate, based on assessment findings.
- If necessary, support breathing by delivering ventilations with a BVM resuscitator and then with mechanical ventilation for continued ventilatory need (depending on the patient's condition).

Circulation

Assess the child's or infant's circulation to determine adequate perfusion of tissues, assess cardiovascular function, and in some cases, guide therapy.

- If not already done, connect the child or infant to a cardiac monitor to monitor heart rate and rhythm.
- Monitor blood pressure. Because of the importance of optimizing blood pressure, placement of an arterial catheter should be considered for continuous blood pressure monitoring.
- Note skin and mucous membrane color (assess for cyanosis, mottling), skin temperature, and level of consciousness.
- Assess peripheral perfusion (e.g., capillary refill; palpate central and peripheral pulses).
- Monitor urine output in all patients as an indicator of systemic perfusion.
- Obtain vascular access, if not already done (two IV or IO catheters are ideal).
- Placement of a central venous catheter may facilitate CVP and $ScvO_2$ monitoring in the event of shock after cardiac arrest and allows for repeated blood sampling for laboratory analyses. In addition, vasoactive agents should be administered through a central venous catheter. If a patient's clinical condition warrants more immediate treatment, however, these agents may be administered through an IO or even peripheral line.
- Prepare for fluid therapy, if indicated.

Disability

- If patient is breathing spontaneously, assess mental status and level of consciousness.
- Use the following tools:
  - AVPU
  - GCS
  - TICLS
- Check blood glucose level.
- Evaluate for potential or apparent cervical spine injury.
- If patient is receiving assisted ventilation, assess adequacy of sedation.
- Evaluate for focal pupillary response (PERRL) or other neurologic abnormalities or abnormal posturing.
- Monitor for seizure activity.

Exposure

- Assess for cyanosis, mottling.
- Assess for skin and body temperature and color to assess circulation and perfusion.
- Evaluate for signs of coagulopathy (e.g., petechiae, bruising, bleeding from intravascular catheter sites).

Secondary Assessment

Obtain a medical history and perform a focused physical assessment, including a neurologic exam.

Table 10-2 shows laboratory and diagnostic tests that should be considered as part of the secondary assessment of oxygenation, ventilation, circulation and neurologic function after cardiac arrest.

Caring for the Pediatric Patient Post–Cardiac Arrest

Care of the pediatric patient post–cardiac arrest consists of two stages. The first stage consists of addressing primary assessment findings and providing advanced life support care for life-threatening conditions. In doing so, it is important to focus on monitoring and supporting the patient's airway, breathing and circulation (Figure 10-2). In addition, it is important to recognize and care for any persistent or reversible causes of the cardiac arrest (Hs and Ts) or critical illness. Finally, it is important to perform a secondary assessment, as time and resources permit.

The second stage of post–cardiac arrest care focuses on titrating and assuring continued oxygenation, ventilation measures, circulation measures, and instituting neuroprotective measures. Much of this care may be provided in a critical care setting.

Table 10-2 | Laboratory and Diagnostic Tests Post-Cardiac Arrest

Test	Purpose
Blood gases	■ Analyze in patients who are mechanically ventilated, in particular to assess O_2 and CO_2 levels and correlation of O_2 saturation and $ETCO_2$. ■ Ideally, arterial blood gas measurements should initially be obtained to ensure correlation with the method that will be used for ongoing monitoring of CO_2 levels (e.g., venous blood gases, capnography).
Chest radiography	■ Confirm the endotracheal tube position in patients who are intubated and to assess for underlying pulmonary disease or injury in all patients after cardiac arrest. ■ Assess for signs of cardiac dysfunction (e.g., enlarged heart, pulmonary edema).
Electrocardiography	■ Assess heart rate and rhythm. ■ Evaluate for evidence of coronary ischemia.
Serum electrolytes, glucose and calcium	■ Identify treatable abnormalities that may exacerbate cardiac dysfunction or arrhythmias (e.g., hypokalemia, hypocalcemia). ■ Identify abnormalities that can exacerbate brain injury or contribute to seizure activity.
Complete blood count	■ Assess hemoglobin level and white blood cell and platelet counts.
Lactate	■ Establish baseline level.
Computed tomography	■ Consider when the underlying cause of cardiac arrest is unknown or when focal brain lesions (e.g., hematoma, tumor) are suspected.
Electroencephalogram (EEG)	■ Confirm suspected seizure activity or for patients receiving neuromuscular blockade, which may mask seizure activity.

Figure 10-2 | Adequate oxygenation and ventilation is critical after cardiac arrest.

Oxygenation and Ventilation Measures

Inadequate oxygenation and ventilation may adversely affect neurologic outcome after cardiac arrest by contributing to secondary brain injury. Conditions that commonly affect the lungs after cardiac arrest, including acute respiratory distress syndrome and pneumonia, may impair oxygen and CO_2 exchange.

While oxygenation and ventilation abnormalities must be rectified, hyperoxia and hyperventilation resulting from overcorrection of such abnormalities may exacerbate brain injury and may be detrimental after cardiac arrest. In addition the measures themselves that lead to hyperoxia and hyperventilation may cause lung issues due to unnecessary volutrauma and barotrauma.

Goals of care include:

- Ensure airway integrity.
- Optimize oxygenation.
- Optimize ventilation.
- Optimize perfusion and oxygenation of the brain.
- Institute neuroprotective measures.
- Treat any underlying lung disease or injury.

Principles of Managing Oxygenation

Adequate oxygen delivery to the tissues is critical after cardiac arrest. As discussed in Chapter 7, O_2 saturation is a key component of blood oxygen content and hence of oxygen delivery. Therefore, maintaining normoxia is important after cardiac arrest.

On the other hand, excess oxygen delivery (i.e., hyperoxia) may exacerbate some of the processes underlying PCAS, including production of free radicals, which damage tissues. Accordingly, a careful approach to oxygenation is recommended after ROSC, although studies examining the clinical impact of hyperoxia after cardiac arrest have yielded inconsistent results.

To this end, the 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations propose targeting normoxemia (i.e., normal PaO_2) after ROSC unless a patient's specific condition warrants a lower target. In general, oxygen should be weaned to the lowest concentration required to maintain O_2 saturation above 94 percent, but below 100 percent. On the other hand, a lower O_2 saturation may be acceptable (and preferable) in children with certain underlying conditions, such as cyanotic congenital heart disease.

Principles of Managing Ventilation

Monitor CO_2 Levels

Blood CO_2 levels affect blood flow to the brain both under normal conditions and after cardiac arrest. Low levels of CO_2 (i.e., hypocarbia) cause blood vessels to constrict and reduce blood flow to the brain, causing secondary ischemic injury. Hypocarbia is associated with poor neurologic outcome after cardiac arrest in adults and hypoxic-ischemic brain injury in infants. The effect of hypercapnia on neurologic outcome after cardiac arrest is less defined.

A recent analysis found that a normal CO_2 level was associated with a more favorable neurologic outcome than either hypocarbia or hypercarbia in adults after cardiac arrest, whereas the effect of hypercarbia after cardiac arrest in children is not known. Therefore, based on existing data, maintenance of normocarbia (i.e., $PaCO_2$ 35 to 45 mmHg) is generally recommended after cardiac arrest, with some exceptions. (See *Learn More: Exceptions to Maintenance of Normocarbia After Cardiac Arrest.*)

Monitor CO_2 levels using arterial blood gases or, alternatively, venous blood gases or capnography. However, if using alternative methods for monitoring CO_2 levels, ensure that the results correlate with those obtained for arterial blood gases.

Address Lung Injury and Infection After Cardiac Arrest

Several pathologic processes can affect the lungs after cardiac arrest. Lung injury may occur as a result of aspiration, pulmonary contusion from chest compressions or the reperfusion response. Pulmonary infections may also occur from impaired immunity following ROSC. When mechanical ventilation is required, low ventilating pressures are generally recommended given their

LEARN MORE

Exceptions to Maintenance of Normocarbia After Cardiac Arrest

Maintenance of normocarbia may not always be an appropriate goal after cardiac arrest. In certain clinical situations, higher or lower $PaCO_2$ levels may be appropriate. Regardless of whether a patient has lung disease, a strategy of permissive mild hypercarbia (i.e., $PaCO_2$ up to 50 mmHg) may be used in patients who are mechanically ventilated after cardiac arrest to allow for the use of low tidal volumes in patients who have acute or chronic lung problems, which are associated with better outcomes than those achieved with high tidal volumes after cardiac arrest.

Brief periods of therapeutic hyperventilation may be used in children with traumatic brain injury to induce hypocarbia and acutely reduce blood flow to the brain when herniation seems imminent. However, this maneuver is not beneficial and may even be harmful in the kind of hypoxic-ischemic brain injury that typically results from cardiac arrest.

association with better outcomes after cardiac arrest and to minimize any adverse hemodynamic effects.

Circulation Measures

In addition to any conditions that caused pre-arrest hypoperfusion, several pathophysiologic factors may contribute to ongoing hemodynamic compromise after cardiac arrest, including myocardial dysfunction and an ischemia/reperfusion response. The ischemia/reperfusion response consists of a systemic inflammatory response, which can cause inappropriate vasodilation, impaired oxygen utilization at the tissue level, coagulopathy, and adrenal and immune dysfunction. This ischemia/reperfusion response can be reversed with early aggressive therapy.

Goals of care include:

- Manage myocardial dysfunction.
- Maintain hemodynamic stability.

General strategies used to meet these goals include:

- Ensure adequate oxygenation and ventilation.
- Continuously monitor heart rate and rhythm.
- Carefully monitor blood pressure with noninvasive blood pressure monitoring or arterial pressure monitoring (ideal). It is very important to maintain a systolic blood pressure within the normal range for age.
  - Note: Some protocols are directed at mean arterial pressure instead of systolic pressure).
- If not already done, establish vascular access. Consider placement of a central venous catheter (ideal).
- Aggressively manage hemodynamic instability. Initial treatment of hypotension is with fluid therapy to maintain normovolemia. Then, institute pharmacologic therapies as indicated.
- Treat arrhythmias using standard pharmacologic and electrical therapies.
- Monitor urine output; consider CVP monitoring.
- Consider packed red blood cell (PRBC) or whole blood transfusion as clinically indicated.
- Administer maintenance fluids in the appropriate calculated amount once the patient is stabilized.
- Treat metabolic abnormalities (e.g., acidosis, hypocalcemia, electrolyte abnormalities).
- Consider mechanical measures (e.g., ECMO) if refractory to medical management.

For more information and a full review of fluid therapy, see Table 3-4 in Chapter 3.

Practice Note

Avoid hypotonic fluids, which may increase edema (including cerebral edema, leading to increased ICP) and intravascular fluid depletion.

Pharmacologic therapy in the form of inotropic agents or vasopressors may be indicated for persistent hemodynamic instability (normotensive) or persistent hemodynamic instability (hypotensive) in children or infants post cardiac arrest. Agents that focus on contractility and improve coronary perfusion may also be needed.

Medications used to treat normotensive hemodynamic instability (shock) include:

- Milrinone
  - Loading dose: 50 mcg/kg, IV/IO
  - Infusion: 0.25 to 0.75 mcg/kg/min, IV/IO
- Dopamine: 2 to 20 mcg/kg/min, IV/IO infusion
- Epinephrine: 0.1 to 1 mcg/kg/min, IV/IO infusion
- Dobutamine: 2 to 20 mcg/kg/min, IV/IO infusion

Practice Note

Because dobutamine and milrinone have vasodilatory properties, monitor for hypotension when using these agents.

Medications used to treat hypotensive hemodynamic instability (shock) include:

- Epinephrine: 0.1 to 1 mcg/kg/min, IV/IO infusion
- Dopamine: 2 to 20 mcg/kg/min, IV/IO infusion
- Norepinephrine: 0.1 to 2 mcg/kg/min, IV/IO infusion

Practice Note

Follow your facility's protocols regarding initial dosing and infusion rates. Once therapy is initiated, the drug infusion rate can be titrated according to hemodynamic parameters and physical examination findings.

For a full review of medications used for pharmacologic therapy, see Table 3-5 in Chapter 3.

ALERT

Optimization of arterial blood pressure is vitally important after cardiac arrest to ensure adequate brain perfusion. Hypotension is associated with reduced survival rates in both in–hospital and out–of–hospital cardiac arrest in children.

Principles of Managing Circulation

Circulatory management after cardiac arrest aims to treat myocardial dysfunction and any hemodynamic disturbance related to the ischemia/reperfusion response.

> Above all, hypotension must be avoided given its association with poor outcomes after cardiac arrest.

A systolic blood pressure within the normal range for age should be maintained (Table 10-3). When possible, continuous monitoring of arterial blood pressure is recommended to identify hypotension promptly. Some management protocols target mean arterial pressure as opposed to systolic pressure.

Managing Myocardial Dysfunction After Cardiac Arrest

Myocardial dysfunction after cardiac arrest typically is reversible and responds to fluid resuscitation and inotropic therapy. Dysrhythmias may accompany and contribute to myocardial dysfunction and should be treated by correcting any abnormalities in electrolyte levels and using standard medical and electrical therapies. For refractory myocardial dysfunction after ROSC, mechanical measures such as extracorporeal membrane oxygenation (ECMO) may be required.

Table 10-3 | Normal Pediatric Vital Signs

Age Group	Respiratory Rate	Awake Heart Rate	Systolic Blood Pressure	Diastolic Blood Pressure
Newborn	30 to 60	100 to 200	60 to 85	35 to 55
Infant (1 to 12 mo)	30 to 50	100 to 180	70 to 100	35 to 60
Toddler (1 to 2 yrs)	24 to 40	90 to 140	85 to 105	40 to 65
Preschooler (3 to 5 yrs)	20 to 30	80 to 130	89 to 115	45 to 70
School Age (6 to 12 yrs)	16 to 26	70 to 120	94 to 120	55 to 80
Adolescent (13 to 17 yrs)	12 to 20	60 to 100	110 to 135	60 to 85

Quick Assessment for Hypotension	
Age Group	**Systolic Blood Pressure**
Neonate	<60
Infant	<70
Toddler to School Age	<70 + (age in years x 2)
Adolescent	<90

ECMO is a technique used to support a patient's failing heart or lungs. It incorporates a bypass circuit that circulates blood exiting from the body through an artificial lung or membrane oxygenator. 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 pumped back into the body (Figure 10-3).

Anticoagulant medications are required to prevent clotting of the patient's blood within the circuit. Therefore, bleeding is a primary risk with ECMO.

Maintaining Hemodynamic Stability

An early goal-directed hemodynamic optimization strategy has been proposed for shock after cardiac arrest. Although this strategy has not been evaluated in randomized controlled trials, it has been shown to improve survival and neurologic outcomes when used with other neuroprotective therapies in survivors of out–of–hospital cardiac arrest, using historical controls as a comparator. Despite this limited evidence, a hemodynamic optimization strategy has been advocated for managing shock after cardiac arrest.

After initial fluid resuscitation to optimize preload (Figure 10-4), pharmacologic therapy is used to optimize cardiac contractility and afterload, as needed, based on blood pressure (normal vs. low) and other hemodynamic parameters, including central venous pressure (CVP) and $ScvO_2$ level. Blood transfusion may be considered

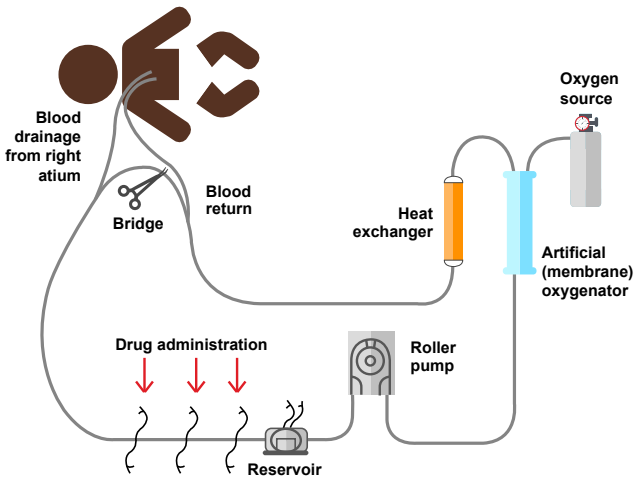


Figure 10-3 | ECMO is used to support a patient's failing heart or lungs. Deoxygenated blood is removed from the body and passed through a membrane that removes the carbon dioxide and adds oxygen. The blood is then warmed, filtered and pumped back into the body.

Figure 10-4 | After initial fluid resuscitation to optimize preload, pharmacologic therapy is used to optimize cardiac contractility and afterload.

in patients with low hemoglobin levels and persistently inadequate oxygen delivery. Target ranges for CVP (8 to 12 mmHg) and $ScvO_2$ (>70 percent) have been proposed, although optimal parameters for shock after cardiac arrest have not been established.

In addition to $ScvO_2$, other indicators of adequate end-organ and tissue perfusion include a urine output normal for age (1.5-2 mL/kg/h for infants and young children and 1 mL/kg/h for adolescents) and normalized or decreasing lactate levels. However, because therapeutic hypothermia may induce diuresis, urine output may be an unreliable indicator of end-organ perfusion in patients who receive this therapy. Likewise, lactate levels may be affected by hypothermia, seizures or liver dysfunction, all of which impair lactate clearance.

Neuroprotective Measures

Optimization of brain function is the primary goal of post–cardiac arrest care. Strategies for neuroprotection after cardiac arrest are summarized in Table 10-4. Treatment primarily aims to ensure adequate perfusion and hence adequate delivery of oxygen and other nutrients to the brain while minimizing oxygen consumption and mitigating further injury to the brain.

Principles of Managing Brain Perfusion

Cerebral blood flow is driven by cerebral perfusion pressure (CPP), which is calculated with the following formula:

CPP = Mean arterial pressure – Intracranial pressure

Table 10-4 | Neuroprotective Strategies After Cardiac Arrest

Goal	Therapeutic Strategies
Optimize cerebral oxygen delivery	■ Optimize cerebral perfusion: ○ Ensure adequate mean arterial pressure (MAP); avoid hypotension. ○ Control increased intracranial pressure and optimize cerebral perfusion pressure. Avoid hyperventilation/hypocarbia unless necessary for the acute, short-term treatment of imminent herniation. ■ Optimize O_2 saturation and hemoglobin level.
Minimize cerebral oxygen consumption	■ Treat seizures if they occur. ■ Initiate targeted temperature management, as clinically indicated. Manage body temperature (i.e., prevent and treat fever in all patients; targeting a hypothermia temperature range may be considered). ■ Control shivering if therapeutic hypothermia is used. ■ Provide sedation as needed.
Minimize further neuron damage	■ Ensure normoglycemia. ■ Avoid hyperoxia.

Ensuring an Adequate MAP

Normally, brain perfusion is maintained despite fluctuations (including decreases) in mean arterial pressure (MAP). This phenomenon is known as autoregulation. After cardiac arrest, cerebral autoregulation is often impaired, meaning cerebral perfusion is less likely to be maintained when MAP decreases.

> Therefore, ensuring an adequate MAP is critical to maintaining sufficient cerebral perfusion after cardiac arrest.

Optimal MAP for CPP is dependent on ICP, and after cardiac arrest, the specific ICP may not be known. Hypotension is clearly detrimental and should be treated promptly.

Treating Increased ICP

When present, increased intracranial pressure (ICP) must be aggressively treated to avoid deleterious effects on cerebral perfusion. However, despite the presence of brain edema following ROSC, clinically relevant increases in ICP are rare after hypoxic-ischemic cardiac arrest events. Therefore, routine ICP-directed treatment is not generally recommended, unless an underlying traumatic brain injury, hemorrhage or mass lesion is suspected. In such cases, standardized protocols for increased ICP are used. In addition, hypertonic saline or mannitol may be used for acutely increased ICP or for impending herniation. Brief periods of hyperventilation may also be used when impending herniation is apparent, though prolonged hypocarbia should be avoided given its adverse effect on cerebral blood flow. Surgical intervention may be required for treatment of any space-occupying lesions (e.g., hematoma, tumor).

Principles of Managing Seizures

The incidence of seizures after cardiac arrest in children is not well defined, but seizures occur in up to 15 percent of adults who achieve ROSC after cardiac arrest. Seizures are especially prevalent in patients who remain comatose after ROSC. Seizure activity substantially increases cerebral metabolic demand and may worsen post–cardiac arrest brain injury. Accordingly, seizures should be promptly detected and treated after ROSC. EEG may be used continuously to detect subclinical seizure activity, especially in patients receiving neuromuscular blockade, or when indicated based on suspected seizure activity. No evidence supports prophylactic treatment of seizures after cardiac arrest.

Principles of Managing Temperature

Aggressive treatment of hyperthermia is universally recommended. As little as a 1° C increase in temperature has been associated with a higher risk of mortality and disability in infants following a hypoxic-ischemic event. Fever should be treated with antipyretics or active cooling.

Therapeutic hypothermia, the practice of purposely reducing body temperature, has been widely investigated as a means to preserve neurologic function after cardiac arrest. This practice is based on the principle that the metabolic rate—and hence the oxygen requirement of the brain—decreases as body temperature decreases. With therapeutic hypothermia, body temperature is typically reduced to the range of 32° C to 34° C (89.6° F to 93.2° F).

Therapeutic hypothermia has been shown to improve neurologic outcomes in adults who remained comatose after ROSC following out-of-hospital cardiac arrest due to ventricular fibrillation or ventricular tachycardia, and in neonates after hypoxic-ischemic arrest. Accordingly, early therapeutic hypothermia is standard practice in these groups. By contrast, therapeutic hypothermia has shown no survival or functional benefit over normothermia in studies of children who were comatose following in- or out-of-hospital cardiac arrest. However, no added adverse reactions were attributed to therapeutic hypothermia in those studies. See *Learn More: Targeted Temperature Management*.

Methods used to induce hypothermia include intravenous infusion of cold fluids and external application of ice packs or other cooling devices. Some external and internal cooling methods offer the added benefit of continuous temperature monitoring, adjusting their output to avoid temperature. Sedation and paralytic medications may be required to manage any shivering during therapeutic hypothermia. Other potential adverse effects include arrhythmias (most often bradycardia), increased SVR, and impaired coagulation and immunity.

Principles of Managing Glucose Level

Post–cardiac arrest care should aim to treat hypoglycemia and, when possible, prevent hyperglycemia.

Hyperglycemia is common after cardiac arrest and is associated with poor outcomes. Accordingly, hyperglycemia should be avoided, although the optimal management of glucose levels after cardiac arrest has not been established.

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Targeted Temperature Management

The 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations state that it is reasonable to consider using a targeted temperature management approach after out-of-hospital arrest.

Either normothermia (36 to 37.5° C) or hypothermia (32 to 34° C) should be maintained for at least 24 hours in children who remain comatose after out-of-hospital cardiac arrest.

Although the optimal target temperature and duration are unknown, a protocol of either 5 days of normothermia or 2 days of hypothermia, followed by 3 days of normothermia, has been proposed.

No similar recommendations were provided for in-hospital cardiac arrest, pending the results of an ongoing study that has since been completed and showed no benefit of therapeutic hypothermia, as previously noted.

Attempts to tightly control glucose levels have not been shown to be of benefit after cardiac arrest and may lead to potentially harmful hypoglycemia. Studies have shown that glucose values as high as 144 mg/dL do not increase the risk for mortality after cardiac arrest.

Glucose levels should be monitored frequently after cardiac arrest, particularly in patients subjected to therapeutic hypothermia, which reduces insulin sensitivity and therefore may contribute to hyperglycemia. Additional therapy (i.e., insulin) may be indicated for severe or persistent hyperglycemia (glucose >144 to 180 mg/dL) but should be carried out with extreme caution to avoid hypoglycemia.

See *Post–Cardiac Arrest Care* Treatment Guideline for a review of care.

POST–CARDIAC ARREST CARE

Post–cardiac arrest (ROSC)

Assessment

- Conduct primary assessment and provide care for life-threatening conditions
  - Establish (if not already done) cardiac monitoring, pulse oximetry, capnography and noninvasive blood pressure monitoring or arterial pressure monitoring
- Identify and provide care for any persistent or reversible causes (Hs and Ts) of the cardiac arrest event
- Perform a secondary assessment (medical history; focused physical assessment, including a neurological exam; laboratory and diagnostic tests) as time and resources permit*

Optimize oxygenation and ventilation

- Provide lowest concentration of supplemental oxygen needed to maintain saturation of > 94% but less than 100%; avoid hyperoxia†
- Consider advanced airway‡
  - If ETT already in place, confirm proper position and patency
- Assist ventilation as needed, maintaining normocarbia ($PaCO_2$ between 35 and 45 mmHg). Unless clinical condition warrants a carbon dioxide level above or below this range.§
- Continuously monitor with capnography and pulse oximetry

Manage circulation

- Monitor with noninvasive blood pressure monitoring or arterial pressure monitoring
  - Maintain a systolic BP within the normal range for age
- Treat hypotension aggressively; initiate fluid therapy (to maintain normovolemia) and pharmacological therapies (e.g., inotropes, vasopressors) as indicated
- Treat arrhythmias using standard pharmacologic and electrical therapies
- Monitor urine output; consider CVP monitoring
- Transfuse PRBCs as clinically indicated
- Correct metabolic abnormalities (e.g., hypocalcemia, hypoglycemia, hyperkalemia, acidosis)
- Consider mechanical measures (e.g., ECMO) if refractory to medical management

Initiate neuroprotective measures

- Optimize cerebral perfusion (ensure adequate MAP, control ICP, avoid hyperventilation unless indicated)§
- Treat seizures, if they occur
  - Continuous EEG monitoring may be used to detect subclinical seizure activity, particularly in patients receiving neuromuscular blockade
- Manage temperature
  - Treat fever aggressively (antipyretics or active cooling)
  - Targeting a hypothermia temperature range may be considered (see *Hypothermia Management* table)‖
- Provide sedation as needed
- Manage glucose
  - Treat hypoglycemia
  - Avoid hyperglycemia; consider insulin for severe or persistent hyperglycemia (but use extreme caution to avoid hypoglycemia)

Please see reverse side for additional information

*Consider the following laboratory and diagnostic tests: blood gases; serum electrolytes, glucose and calcium; complete blood count; lactate level; chest radiography; electrocardiography; computed tomography; and electroencephalogram (EEG).

†Unless clinical condition warrants an oxygen saturation below this range.

‡Use spinal motion restriction techniques during airway interventions in patients with suspected or confirmed cervical spine injury.

§Avoid hyperventilation unless clinically warranted for acute management when herniation seems imminent.

‖Providers should not initiate targeted temperature management in the prehospital setting. See *Hypothermia Management* table, on reverse side.

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POST–CARDIAC ARREST CARE CONTINUED

Hypothermia Management

- In children who remain comatose or are unresponsive after out-of-hospital cardiac arrest, it is reasonable to consider either of the following protocols:
  - Five days of continuous normothermia (36 to 37.5° C)
  - Two days of continuous hypothermia (32 to 34° C) followed by 3 days of continuous normothermia
- In children who remain comatose after in-hospital cardiac arrest, there is insufficient support for recommending hypothermia over normothermia
- Methods for inducing hypothermia include:
  - IV infusion of cold fluids
  - External application of ice packs or other cooling devices
- External application of ice packs or other cooling devices
- Continuously monitor core temperature
- Treat shivering with sedation and neuromuscular blockade as needed
- Monitor urine output and accompanying adverse effects (i.e., volume depletion or abnormalities in electrolyte levels)
- Monitor for other adverse effects (e.g., arrhythmias, increased SVR, impaired coagulation, decreased cardiac output, electrolyte abnormalities, infection)

Determining Hypotension

Age Group	Systolic Blood Pressure
Neonate	< 60 mmHg
Infant	< 70 mmHg
Toddler to School Age	< 70 mmHg + (age in years x 2)
Adolescent	< 90 mmHg

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

Glossary

ABCDE
Acronym that stands for airway, breathing, circulation, disability, exposure.

Activated partial thromboplastin time (aPTT)
A blood test used to measure how quickly (in seconds) blood clots after adding certain reagents. The aPTT assesses the amount and function of specific coagulation factors (i.e., those involved in the intrinsic and common pathways). Abnormalities in any of those coagulation factors prolong the aPTT. This test is also used to ensure adequate anticoagulation in patients receiving heparin (in which case, the aPTT is purposefully prolonged within a certain range).

Acute phase reactant
An increase in the concentration of serum proteins that accompanies inflammation and tissue injury.

Aerobic metabolism
Cellular creation of energy using oxygen.

Anaerobic metabolism
Cellular creation of energy through glycolysis (i.e., glucose breakdown) in the absence of oxygen. This process results in the accumulation of lactic acid as a byproduct.

Angioedema
Rapid swelling of the deeper layers of the skin and tissues beneath the skin or mucous membranes. Often caused by an allergic reaction.

aPTT
Acronym for activated partial thromboplastin time.

Ataxia
Ataxia is a rare neurological disease. It is progressive–affecting a person's ability to walk, talk and use fine motor skills.

Atelectasis
Alveolar collapse.

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.

AVPU
Acronym that means awake, responds to verbal stimulation or voice, responds to pain, unresponsive.

Bundle branch block
A delay or blockage in one of the pathways (i.e., bundle branch) along which the electrical impulses travel through the heart to make it beat. It is most often benign. A bundle branch block may be congenital or the result of damage to the bundle branch (e.g., from heart surgery or myocarditis).

Capnography
A noninvasive way of measuring end-tidal carbon dioxide (CO_2) level.

Cardiomyopathy
Diseases of the heart muscle that may dilate the chambers or thicken the walls of the heart, compromising cardiac function.

Chest compression fraction
The percentage of time spent performing chest compressions during the resuscitation effort; an indicator of CPR quality.

Choanal atresia
A congenital disorder characterized by unilateral or bilateral narrowing of the posterior nasal apertures (i.e., nares), where the nasal passages open into the nasopharynx.

Choanal stenosis
A congenital disorder characterized by unilateral or bilateral narrowing of the passage between the nose and the throat.

Closed-loop communication
A communication technique used to prevent misunderstandings; the receiver confirms that the message has been received and understood.

Coagulation
The process by which a blood clot forms.

Consent
Asking a responsive person (or the parent or guardian of a minor) for permission to help before giving care.

Coronary perfusion pressure (CPP)
The difference between the pressure in the aorta and the pressure in the right atrium during diastole; a reflection of myocardial blood flow.

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.

Cyanosis
A bluish color to the skin or mucous membranes that is usually due to a lack of oxygen in the blood and decrease in perfusion.

Cytokines
Small proteins released by immune and other cells that play a role in cell signaling. They aid cell-to-cell communication within the body's immune responses and facilitate the movement of cells toward sites of injury, infection or inflammation.

D-dimer
Protein fragments produced when a blood clot breaks down. Elevation of D-dimer levels in blood may indicate the presence of a large clot (e.g., deep vein thrombosus) or abnormal coagulation (e.g., disseminated intravascular coagulation).

Ductus arteriosus
A blood vessel in the developing fetus that connects the thoracic aorta and main pulmonary artery, allowing blood to largely bypass the fetus' nonfunctioning lungs. The ductus arteriosus normally closes shortly after birth.

Dyspnea
Shortness of breath.

Echocardiogram
Sonogram of the heart.

EEG
Acronym for electroencephalography.

Embolus
A blockage, such as a blood clot or air, that obstructs a blood vessel.

ETT
Acronym for endotracheal tube.

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 in addition to standard CPR.

Fontanelle
A "soft spot" representing a natural space bordered by the bony plates of an infant's skull. There are two fontanelles in the human infant skull: an anterior one and a posterior one. The posterior fontanelle typically closes within the first several months of life, and the anterior fontanelle typically closes by the age of 2 years. The fontanelles can be used to clinically assess intracranial pressure and overall hydration status in infants.

Foramen ovale
A normal opening in the septum between the top two chambers (atria) of the heart in the developing fetus. It allows blood coming back from the body to pass directly into the left side of the heart, bypassing the fetus' nonfunctioning lungs. The foramen ovale normally closes shortly after birth.

GCS
Acronym for Glasgow Coma Scale.

Hemangioma
A benign tumor composed of excess blood vessels. There are multiple types of hemangiomas. They can occur throughout the body, including in skin, muscle, bone and internal organs.

Hematocrit
The ratio of red blood cells to the total volume of blood (expressed as a percentage).

Hemoglobin
A protein in red blood cells that incorporates an iron-containing molecule called heme. Hemoglobin is the primary transporter of oxygen in the blood. It also binds a smaller percentage of carbon dioxide returning to the lungs for exchange. The concentration of hemoglobin in the blood can be measured as part of a complete blood count.

Hemolysis
Premature destruction of red blood cells, which causes release of cell contents, including hemoglobin, into the blood plasma. Hemolysis may cause anemia if red blood cell production cannot keep up with destruction.

Hyperammonemia
Elevated levels of ammonia in the blood.

Hypercarbia
Abnormally elevated levels of carbon dioxide (CO_2) in the blood.

Hyperpnea
Abnormal increase in the depth of breathing, with or without an increase in rate.

ICP
Acronym for intracranial pressure.

IO
Acronym for intraosseous.

Ischemia/reperfusion response
An inflammatory response induced by the reperfusion of tissues previously deprived of blood flow, as in cardiac arrest.

IV
Acronym for intravenous.

Kyphoscoliosis
Combination of abnormal outward curvature (kyphosis) and lateral curvature (scoliosis) of the spine. Kyphosis creates an abnormally rounded or "humpback" appearance of the upper spine.

Laryngomalacia
Congenital softening of the laryngeal cartilage. Causes collapse of the airway during inspiration and is characterized by stridor.

MAP
Acronym for mean arterial pressure.

Mediastinal
Of or pertaining to the mediastinum, the area in the chest between the two lungs. The mediastinum contains the heart, esophagus and trachea, among other structures.

Myocarditis
Inflammation of the heart muscle.

Needle thoracentesis
A procedure used in the emergency management of tension pneumothorax. The procedure consists of inserting an over-the-needle catheter into the pleural space to release a build-up of air and pressure. This allows the collapsed lung to re-inflate.

Otolaryngologist
A physician trained in the medical and surgical care of diseases and disorders of the head and neck, in particular the ear, nose and throat.

Papilloma
A small, typically benign growth composed of hypertrophied epithelial tissue.

Pericardiocentesis
A procedure used in the emergency management of cardiac tamponade. The procedure consists of inserting a needle and catheter into the pericardial space that surrounds the heart in order to aspirate fluid and release the build-up of pressure on the heart.

PERRL
Acronym that stands for pupils equal, round and reactive to light.

Petechiae
Small (<2-mm) areas of bleeding under the skin or mucous membranes caused by breaking of capillaries. They appear as flat, round spots that are red, purple or brown in color and do not blanch when pressed upon. Petechiae may be attributed to bleeding disorders, infections or other inflammatory conditions, medications or trauma.

Prothrombin time (PT)
A blood test used to measure how quickly (in seconds) blood clots after adding certain reagents. The PT measures the amount and function of specific coagulation factors (i.e., those involved in the extrinsic pathway). Abnormalities in any of those coagulation factors prolong the PT.

Purpura
Red- or purple-colored, nonblanching spots on the skin measuring >2 cm in diameter. Similar to petechiae, they are caused by bleeding underneath the skin and mucous membranes and may be attributed to bleeding disorders, infections or other inflammatory conditions, medications or trauma.

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.

Respiratory arrest
Complete cessation of the breathing effort. A patient who is not breathing normally (or only gasping) but has a pulse is in respiratory arrest.

Respiratory distress
Respiratory distress can include several signs and symptoms (such as increased work of breathing or ineffective breathing) that indicate the patient's ventilatory effort and breathing is abnormal/inadequate.

Respiratory failure
Respiratory failure is a syndrome in which the respiratory system fails in one or both of its gas exchange functions: oxygenation and carbon dioxide elimination. In practice, it may be classified as either hypoxemic or hypercapnic.

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.

SMR
Acronym for spinal motion restriction.

Subglottic stenosis
Narrowing of the subglottic airway (just below the vocal cords). May be congenital or acquired.

SVR
Acronym for systemic vascular resistance.

Tachyarrhythmia
Abnormal heart rhythm that is faster than normal for age.

Teamwork
The actions of a group of people with well-defined roles and responsibilities making a coordinated effort to achieve a common goal.

Thrombi
Plural form of thrombus, which is a blood clot formed within the vascular system. A thrombus remains wherever it originally formed, in contrast to an embolus, which is a blood clot that breaks free from the thrombus site and travels through the circulation to distant locations.

TICLS
Acronym that stands for tone, interactivity, consolability, look and speech/cry.

Tracheitis
Inflammation of the trachea, often caused by viruses or bacterial infection.

Transthoracic pacing
An emergency external-pacing procedure used to treat persistent and life-threatening bradycardia that will not respond to medication and to treat certain forms of bradycardia caused by AV block until a pacemaker can be implanted surgically. The procedure uses pads placed on the patient's chest to deliver pulses of electrical current in an attempt to stimulate the heart.

Transvenous pacing
An emergency external-pacing procedure used to treat persistent and life-threatening bradycardia that will not respond to medication and to treat certain forms of bradycardia caused by AV block until a pacemaker can be implanted surgically. The procedure involves placing a pacer wire through a vein directly into the heart to introduce pulses of electrical current into the heart muscle.

Vascular ring

A congenital disorder characterized by abnormal formation of the aortic arch in which the anomalous arch or some of its associated branching blood vessels encircle and compress the trachea and esophagus. Vascular rings may completely or partially encircle these structures, leading to varying degrees of breathing and feeding difficulty. These symptoms typically present during infancy or early childhood.

Ventilation

The mechanical process of moving air into and out of the body.

Vocal cord paralysis

Paralysis of the vocal cord muscles caused by disruption of nerve impulses to the larynx. It can involve one or both vocal cords and may affect speaking, breathing and swallowing.

WBC

Acronym for white blood cell.

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Note: *b* indicates box; *t* indicates table

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American Red Cross
Training Services

American Red Cross
Training Services

American Red Cross Training Services and the Scientific Advisory Council

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The technical content within this Red Cross 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.

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The American Red Cross prevents and alleviates human suffering in the face of emergencies by mobilizing the power of volunteers and the generosity of donors.