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PALS

PEDIATRIC ADVANCED LIFE SUPPORT

PROVIDER MANUAL

Pediatric Advanced Life Support

PROVIDER MANUAL

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

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

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

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Part 1
Course Overview

Course Objectives

The Pediatric Advanced Life Support (PALS) Provider Course is designed for health care professionals who manage respiratory or cardiovascular emergencies and cardiopulmonary arrest in pediatric patients. By preparing for the course and participating in the skills stations and simulated cases, you will enhance the recognition of and intervention for respiratory emergencies, shock, and cardiopulmonary arrest.

During the course, you will participate in a series of case scenario practices with simulations that reinforce important concepts, including

- Applying a systematic approach to the pediatric assessment
- Identifying and treating problems that place the child at risk for cardiac arrest
- Using the evaluate-identify-intervene sequence
- Using the PALS algorithms
- Demonstrating effective resuscitation team dynamics

Goal of the PALS Provider Course

The PALS Provider Course aims to improve outcomes for pediatric patients by preparing health care professionals to effectively recognize and intervene in patients with respiratory emergencies, shock, and cardiopulmonary arrest by using high-performance team dynamics and high-quality individual skills.

Learning Objectives

After successfully completing this course, you should be able to

- Perform high-quality cardiopulmonary resuscitation (CPR) per American Heart Association (AHA) basic life support (BLS) recommendations
- Differentiate between patients who do and do not require immediate intervention
- Perform your role as a high-performance team member
- Differentiate between respiratory distress and failure

- Perform early interventions for respiratory distress and failure
- Differentiate between compensated and hypotensive (decompensated) shock
- Perform early interventions for the treatment of shock
- Differentiate between unstable and stable patients with arrhythmias
- Demonstrate treatment of arrhythmias
- Implement postarrest management

Course Description

To help you achieve these objectives, the PALS Provider Course includes

- BLS competency testing
- Skills stations
- Case scenario discussions and simulations
- Case scenario testing stations
- Multiple choice exam

BLS Competency Testing

To receive an AHA PALS Provider Course completion card, you must be proficient in BLS and pass the BLS skills test during the PALS course assessment: the Child CPR and AED Skills Test and the Infant CPR Skills Test.

The PALS Provider Course does not include detailed instructions on how to perform basic CPR or how to use an automated external defibrillator (AED), so you must know this in advance. Consider taking a BLS course to prepare, if necessary.

Before taking the PALS Provider Course, read the *PALS Provider Manual* to prepare for taking the BLS tests.

Skills Stations

The course includes the following skills stations:

- Airway Management
- Vascular Access
- Rhythm Disturbances/Electrical Therapy

In the skills stations, you will use the skills station competency checklists as you practice specific skills and then demonstrate competency. Your instructor will evaluate your skills by using the criteria in these checklists.

To prepare, see the Appendix for the skills station competency checklists, which list detailed steps for performing each skill.

Airway Management Skills Station

In the Airway Management Skills Station, you will show your understanding of oxygen (O_2) delivery systems and airway adjuncts. You will practice and demonstrate competency in airway and breathing supports, including

- Inserting an oropharyngeal (oral) airway (OPA)
- Providing effective bag-mask ventilation
- Confirming advanced airway device placement by physical examination and an exhaled carbon dioxide (CO_2) detector device
- Securing the endotracheal (ET) tube
- Suctioning with an OPA and ET tube if it is within your scope of practice; you may be asked to demonstrate advanced airway skills, including correctly inserting an ET tube or other subglottic airway

Review the Bag-Mask Ventilation section in Part 2 and ET Intubation section in Part 6 of this manual to learn more about airway management skills.

Vascular Access Skills Station

In the Vascular Access Skills Station, you will practice intraosseous (IO) access and other related skills, including

- Inserting an IO needle
- Summarizing how to confirm that the needle has entered the marrow cavity
- Summarizing/demonstrating the method of giving an intravenous (IV)/IO bolus
- Using color-coded length-based resuscitation tape to calculate correct medication doses

Review the IO Access and Color-Coded Length-Based Resuscitation Tape sections in Resources for Managing Circulatory Emergencies in Part 8 of this manual to learn more about vascular access skills.

Rhythm Disturbances/Electrical Therapy Skills Station

In the Rhythm Disturbances/Electrical Therapy Skills Station, you will practice and demonstrate competency in operating a cardiac monitor, identifying different cardiac rhythms, and operating a manual defibrillator. Skills include

- Operating a cardiac monitor
- Correctly placing electrocardiographic (ECG) leads
- Correctly selecting and placing or positioning the paddles or pads
- Identifying rhythms that require defibrillation
- Identifying rhythms that require synchronized cardioversion
- Safely performing manual defibrillation and synchronized cardioversion

Review the following resources in the *PALS Provider Manual* before the course to help you with rhythm identification, cardiac monitoring, and manual defibrillation:

- Pediatric Cardiac Arrest Algorithm in Part 11: Recognizing and Managing Cardiac Arrest
- Rhythm Recognition Review in the Appendix
- Management: Pediatric Bradycardia With a Pulse in Part 10: Managing Arrhythmias
- Managing Tachyarrhythmias in Part 10: Managing Arrhythmias

PALS Case Scenario Discussions and Case Scenario Simulation-Based Practice

In the learning stations, you will participate in a variety of learning activities, including

- Case scenario discussions, using a systematic approach for evaluation and decision-making
- Case scenario simulation-based practice

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

The focus of the PALS Provider Course is to teach you to use a systematic approach when caring for a critically ill or injured child. Read and study the entire *PALS Provider Manual* to understand all the necessary concepts.

PALS Case Scenario Testing Stations

At the end of the course, your team must pass 2 case scenario testing stations to demonstrate the course objectives. You may use the PALS Reference Card and the *2025 Handbook of Emergency Cardiovascular Care for Healthcare Providers* (Handbook of ECC). These simulated clinical scenarios will test the following:

- Ability to evaluate and identify specific clinical conditions covered in the course
- Recognition and management of respiratory and shock emergencies
- Interpretation of core arrhythmias and management by using appropriate medications and electrical therapy
- Performance as an effective team member or Team Leader

This evaluation emphasizes your ability to integrate BLS and PALS skills according to your individual scope of practice.

Review the Roles in a High-Performance Team section in Part 3: High-Performance Teams in this manual to prepare yourself to participate as a team member or Team Leader in the case scenario testing stations.

Exam

The exam measures your mastery of cognitive skills. You must score at least 84% on the exam in the classroom-based course to meet course completion requirements. The AHA has an open-resource policy for exams in eLearning and classroom-based courses.

Open resource means that students may refer to other resources while completing the exam. These resources could include the provider manual, either in printed form or as an eBook on a personal device; any notes the student took during the provider course; the Handbook of ECC; the *2025 AHA Guidelines for CPR and ECC*; posters; etc. *Open resource* does not mean open discussion with other health care professionals or the instructor.

Precourse Preparation

To pass the PALS Provider Course, you must prepare before the course. Do the following:

- Take the precourse self-assessment and, if requested by your instructor, complete the online video prework
- Make sure you are proficient in BLS skills
- Practice identifying and interpreting core ECG rhythms
- Study basic pharmacology and know when to use each medication
- Practice applying your knowledge to clinical scenarios

Precourse Self-Assessment and Video Prework

Complete the precourse self-assessment at **eLearning.heart.org/courses** before you take the PALS Provider Course. (Refer to the Student Resources section for details about how to access this resource.) Print your certificate of completion and score report and bring them with you to class.

Because the PALS Provider Course does not teach algorithms, ECG recognition, pharmacology, or BLS skills in detail, use the precourse self-assessment to identify any deficiencies in your knowledge of these topics. The precourse self-assessment will summarize your strengths and weaknesses. Study the applicable content in the *PALS Provider Manual* or other supplementary resources.

If requested by your instructor, also complete the online video prework at **eLearning.heart.org/courses**. You must pass the precourse self-assessment before gaining access to video lessons. The video prework will ensure that you are prepared for the course.

BLS Skills

Strong BLS skills are the foundation of advanced life support (ALS). Everyone who cares for pediatric patients must be able to perform high-quality CPR, because without it, PALS interventions will fail. For this reason, you must pass the Child CPR and AED and Infant CPR Skills Tests in the PALS Provider Course. Make sure that you are proficient in BLS skills before attending the course.

Refer to the BLS Competency Testing section in the Appendix for testing requirements and resources.

ECG Rhythm Identification

You must be able to identify and interpret the following core rhythms during case scenario practice with simulations and case scenario tests:

- Normal sinus rhythm
- Sinus bradycardia
- Sinus tachycardia (ST)
- Supraventricular tachycardia (SVT)
- Ventricular tachycardia (VT)
- Ventricular fibrillation (VF)
- Asystole
- Pulseless electrical activity (PEA)

The ECG rhythm identification section of the precourse self-assessment will help you evaluate your ability to identify these core rhythms. If you have difficulty with pediatric rhythm identification, study the Rhythm Recognition Review section in the Appendix.

Basic Pharmacology

You must know basic information about medications used in the PALS algorithms, including the indications, contraindications, and methods of administration. You will need to know which medication(s) should be used on the basis of the clinical situation.

The pharmacology section of the precourse self-assessment will help you evaluate and refresh your knowledge of medications used in the course. If you have difficulty with this section of the precourse self-assessment, study the *PALS Provider Manual* and PALS Reference Card. This information can also be found in the Handbook of ECC (sold separately).

Practical Application of Knowledge to Clinical Scenarios

The practical application section of the precourse self-assessment will help you evaluate your ability to apply your knowledge to clinical scenarios. You will need to make decisions based on

- The PALS Systematic Approach Algorithm and the evaluate-identify-intervene sequence
- Identification of core rhythms
- Knowledge of core medications
- Knowledge of PALS algorithms

You should understand the PALS Systematic Approach Algorithm and the evaluate-identify-intervene sequence. Review the core rhythms and medications. Be familiar with the PALS algorithms so that you can apply them to clinical scenarios but note that the PALS Provider Course does not teach the details of each algorithm. You can find more information in the *PALS Provider Manual*, PALS student resources at **eLearning.heart.org/courses**, and the Handbook of ECC.

Course Materials

The PALS Provider Course materials consist of the *PALS Provider Manual*, the PALS Reference Card, and the student resources at **eLearning.heart.org/courses**.

Provider Manual

The *PALS Provider Manual* contains important information that you will use before, during, and after the course. This material includes concepts of pediatric evaluation and the recognition and management of respiratory, shock, and cardiac emergencies. Remember to take this manual with you to class. Throughout the *PALS Provider Manual*, you will find specific information in the following callout box:

Critical Concepts

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

Reference Card

The PALS Reference Card is a valuable learning aid that contains the following resources:

- Vital Signs in Children
- PALS Systematic Approach Algorithm
- Pediatric Septic Shock Algorithm
- Medications used in PALS
- Pediatric Color-Coded Length-Based Resuscitation Tape Chart
- Pediatric Cardiac Arrest Algorithm
- Pediatric Bradycardia With a Pulse Algorithm
- Pediatric Tachyarrhythmia With a Pulse Algorithm
- Cardiovascular Management After ROSC Algorithm
- Components of Post–Cardiac Arrest Care

Take your PALS Reference Card with you to the course to use as a reference during the case scenario discussions and the exam.

Critical Concepts

Meaning of Shapes and Colors in AHA Algorithms

- Red hexagon: decision or question
- Green box: action/steps to consider
- Orange box: assessment
- Gray box: assessment/action
- Blue box: CPR
- White box with gray header: notes
- Red, bold text: possible answers or decisions

Student Resources

Go to **eLearning.heart.org/courses** to access the precourse self-assessment as well as additional information about basic PALS concepts. If you haven't already logged in, the system will ask you to do so. If you haven't visited the site before, you'll be prompted to set up an account. Once logged in, you should find your course name and select Launch to begin.

The precourse self-assessment is a vital part of your course preparation. Feedback from this assessment will help you identify gaps in your knowledge so that you can target specific material to study.

The precourse self-assessment has 3 parts:

- ECG rhythm identification
- Pharmacology
- Practical application

Complete these assessments before the course to identify gaps in and improve your knowledge. Print out your certificate of completion and take it with you to the course.

Student Resources also include PALS videos, video prework (if assigned), and supplementary information.

Course Completion Requirements

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

- Successfully complete the PALS Precourse Self-Assessment (classroom-based course only)
- Actively participate in, practice, and complete all skills stations and learning stations
- Pass the Child CPR and AED and Infant CPR Skills Tests
- Pass an exam with a minimum score of 84% (classroom-based course only)
- Pass 2 PALS case scenario tests

Science Update

We have updated the PALS Provider Course to incorporate the recommendations of the *2025 AHA Guidelines for CPR and ECC*. On an ongoing basis, hundreds of international resuscitation scientists and experts evaluate, discuss, and debate thousands of scientific publications. Then, they reach consensus on the best treatments based on the evidence, which forms the basis for developing the guidelines. Some recommendations are new, whereas others modify previous recommendations. The following list highlights many of the key recommendations for pediatric BLS and ALS.

Major Science Changes in 2025

Some of the major science changes in 2025 include the following:

Pediatric BLS

1. Immediate recognition of cardiac arrest is vital to improving outcomes. For infants and children who are unresponsive with abnormal breathing (including gasping), rescuers should activate emergency medical services and initiate high-quality CPR, beginning with chest compressions.

2. High-quality CPR is the foundation of resuscitation. The key components of high-quality CPR include providing adequate chest compression rate and depth, minimizing interruptions in CPR, allowing complete chest recoil between compressions, and avoiding excessive ventilation.
3. A respiratory rate of 20 to 30 breaths per minute is recommended for infants and children who are (a) receiving CPR with an advanced airway in place or (b) receiving breathing and have a pulse.
4. For infants, the recommended compression techniques include the heel-of-1-hand technique or the 2 thumb–encircling hands technique. The use of 2 fingers along the sternum was eliminated because of ineffectiveness in achieving proper depth in most recent available data.
5. For out-of-hospital cardiac arrest, ventilation in addition to chest compressions improves survival.
6. For children in cardiac arrest, an AED should be attached as soon as possible by using pediatric pads if available.
7. For children with severe foreign-body airway obstruction (FBAO), guidance is the same as for infants, with repeated cycles of 5 back blows alternating with 5 abdominal thrusts, as opposed to solely abdominal thrusts.
8. For infants and children with signs of narcotic or opioid overdose, rescuers should administer naloxone if available in addition to providing breaths or high-quality CPR as needed.
9. Resuscitation does not end with return of spontaneous circulation (ROSC). Excellent post–cardiac arrest care is critically important for achieving the best patient outcomes. For children who do not regain consciousness after ROSC, this care includes targeted temperature management and continuous electroencephalography monitoring. For all children, the prevention and treatment of hypotension, hyperoxia or hypoxia, and hypercapnia or hypocapnia are important.
10. After discharge from the hospital, cardiac arrest survivors can have physical, cognitive, and emotional challenges and may need ongoing therapies and interventions.

PALS

1. High-quality CPR is the foundation of ALS resuscitation for health care professionals. We reaffirm the key components of high-quality CPR: providing adequate chest compression rate and depth, minimizing interruptions in CPR, allowing complete chest recoil between compressions, and providing sufficient ventilation for the pediatric patient population while avoiding excessive ventilation.
2. For initial nonshockable rhythms, administering epinephrine as soon as possible is associated with more favorable outcomes for infants and children in cardiac arrest.
3. Rapid defibrillation remains the priority for cardiac arrest with initial shockable rhythms, although epinephrine administration should not be delayed if defibrillation is not immediately possible.

4. For infants and children with continuous invasive arterial blood pressure monitoring in place during CPR, diastolic blood pressure targets of 25 mm Hg or greater in infants and 30 mm Hg or greater in older children are now included as hemodynamic goals of high-quality CPR.
5. End-tidal CO_2 can be an indicator of CPR quality, although specific end-tidal CO_2 cutoff values alone are not a good measure of when to end resuscitation efforts in infants and children.
6. Preventing hyperthermia is a critical component of post–cardiac arrest care, and avoiding central temperatures greater than 37.5 °C can improve neurological outcomes in infants and children who remain comatose after cardiac arrest.
7. For infants and children, new postarrest systolic and mean arterial blood pressure thresholds greater than the 10th percentile for age and sex are provided.
8. Neuroprognostication after cardiac arrest in infants and children requires multiple modalities at different time points throughout the postarrest period; single tests should not be used because of concerns for inaccurate prediction of neurologic outcomes.
9. After discharge from the hospital, cardiac arrest survivors can have ongoing physical, cognitive, and emotional challenges and need to be evaluated for therapies and interventions.

The Chain of Survival

For many years, the AHA has adopted, supported, and helped develop the concept of emergency cardiovascular care. The term *Chain of Survival* provides a useful metaphor for the elements of the emergency cardiovascular care systems-of-care concept. The Chain of Survival shows the actions that must take place to give the person in cardiac arrest the best chance of survival. Each link is independent, yet connected, to the links before and after. If any link is broken, the chance for a good outcome decreases.

Overview

Cardiac arrest can happen anywhere—on the street, at home, or in a hospital emergency department (ED), in-patient bed, or intensive care unit. A single Chain of Survival that supports the paradigm of prevention and early recognition through recovery after cardiac arrest has now been standardized across infants, children, adults (Figure 1).

Figure 1. **2025 AHA cardiac arrest Chain of Survival.**

Chain of Survival Elements

The Chain of Survival includes the following elements:

- Recognition and emergency activation
- High-quality CPR
- Defibrillation
- Advanced resuscitation interventions
- Post–cardiac arrest care
- Recovery

There is a separate newborn Chain of Care that provides a framework for considering essential elements of the health care system relating to neonatal health. A strong newborn Chain of Care has the potential to improve health in the neonatal period as well as long-term outcomes. The links in the chain include prevention, recognition and activation, initial steps, ventilation, advanced resuscitation, postnatal care, and recovery (Figure 2).

Figure 2. **2025 AHA newborn Chain of Care.**

Part 2

Review of BLS and AED for Infants and Children

This Part describes BLS for infants and children and discusses using an AED in infants and children younger than 8 years.

The following age definitions are used in BLS:

- Infants are younger than 1 year (excluding the newly born).
- Children range from 1 year of age to puberty. Signs of puberty include chest or underarm hair on boys and any breast development in girls.
- When performing BLS for pediatric patients, follow pediatric guidance for infants and children 1 year of age to puberty. For children with signs of puberty and older, follow BLS guidance.
- When performing ALS for pediatric patients, follow pediatric guidance for infants and children up to 18 years of age, excluding newborns.
- Resuscitation of the newborn infant typically applies to the newborn infant who is transitioning from a fluid-filled to air-filled environment. Although pediatric BLS and ALS guidelines may be applied to newborn infants younger than 28 days on the basis of pathophysiology and institutional practice, neonatal guidelines should be followed at birth to address unique aspects of transitional physiology.

Learning Objectives

After completing this Part, you should be able to

- Perform high-quality CPR for infants and pre- and postpubescent children.
- Describe and demonstrate the importance of early use of an AED for infants and children younger than 8 years.

BLS for Infants and Children

Pediatric BLS Algorithm for Health Care Professionals—Single Rescuer

The Pediatric BLS Algorithm (1 Year of Age to Puberty) for Health Care Professionals—Single Rescuer outlines steps for a single rescuer of an unresponsive infant or child (Figure 3). Refer to this algorithm as you read the following steps.

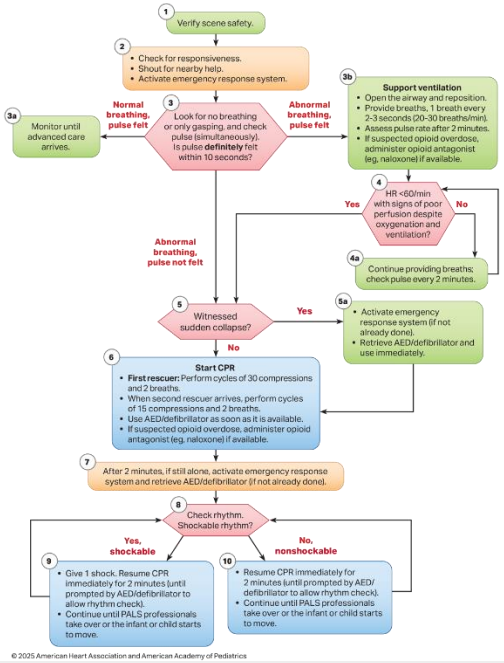


Figure 3. Pediatric BLS Algorithm (Infants to Puberty) for Health Care Professionals—Single Rescuer.

Abbreviation: HR, heart rate.

Algorithm legend: Red hexagon = decision or question; green box = action/steps to consider; orange box = assessment; gray box = assessment/action; blue box = CPR; white box with gray header = notes; and red, bold text = possible answers or decisions.

Infant and Child 1-Rescuer BLS Sequence

If you are alone and encounter an unresponsive infant or child, follow the steps outlined in the Pediatric BLS Algorithm (1 Year of Age to Puberty) for Health Care Professionals—Single Rescuer.

If a child is in cardiac arrest, verify scene safety, check for responsiveness, shout for nearby help, and activate the emergency response system.

Assess breathing and check for a pulse simultaneously. Determine if there is abnormal breathing, which may be gasping or agonal. Agonal breathing may sound like a snort, groan, or snore.

Check Pulse

Infant: To perform a pulse check in an infant, palpate, or feel for, a brachial pulse (Figure 4A).

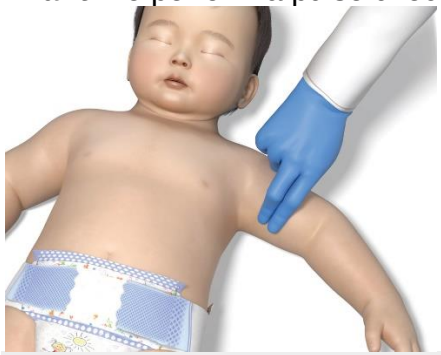


Figure 4A. Pulse check. A, In an infant, feel for a brachial pulse.

Child: To perform a pulse check in a child, palpate a carotid (Figure 4B) or femoral pulse (Figure 4C).

Figure 4B. In a child, feel for a carotid pulse.

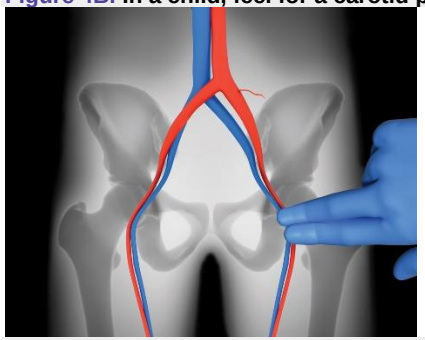


Figure 4C. Or, a femoral pulse.

It can be difficult to determine the presence or absence of a pulse. If you do not definitely feel a pulse within 10 seconds in a child who is unresponsive and not breathing normally, start CPR, beginning with chest compressions. If you are alone, perform cycles of 30 compressions and 2 breaths.

If the infant or child does not have normal breathing and a pulse is felt, give 1 breath every 2 to 3 seconds, or about 20 to 30 breaths per minute. After 2 minutes, assess the pulse for no more than 10 seconds. If the heart rate is less than 60/min, start CPR. If the heart rate is 60/min or greater, continue giving breaths and check the pulse every 2 minutes. If no definite pulse is felt, start CPR.

Infant: Locating the Brachial Artery Pulse

To perform a pulse check in an infant, palpate a brachial pulse (Figure 4A). If you do not definitely feel a pulse in a child who is unresponsive and not breathing normally within 10 seconds, begin high-quality CPR, starting with chest compressions.

Follow these steps to locate the brachial artery pulse:

- Place 2 or 3 fingers on the inside of the upper arm, midway between the infant's elbow and shoulder.
- Press the fingers to attempt to feel for the pulse for at least 5 but no more than 10 seconds (Figure 4A).

Child: Locating the Carotid Artery Pulse

To perform a pulse check in a child, feel for a carotid or femoral pulse (Figures 4B and 4C). For a carotid pulse, use 2 or 3 fingers to locate the child's trachea, and then slide your fingers into the groove just to the side of the trachea. Feeling for the carotid pulse is usually easier to perform on the child's side that is closest to you.

Child: Locating the Femoral Artery Pulse

To perform a pulse check in a child, palpate a carotid or femoral pulse. If you do not definitely feel a pulse within 10 seconds, begin high-quality CPR, starting with chest compressions.

Follow these steps to locate the femoral artery pulse:

1. Place 2 fingers in the inner thigh, midway between the hipbone and the pubic bone and just below the crease where the leg meets the torso (Figure 4C).
2. Feel for a pulse for at least 5 but no more than 10 seconds.

Next Actions

Determine your next actions according to your assessment of breathing and pulse check.

- If the infant or child has normal breathing and a pulse is felt, monitor them until emergency responders arrive.

- If the infant or child has abnormal breathing with a pulse, provide breaths.
- If heart rate is less than 60/min with poor perfusion despite oxygenation and ventilation, start CPR.
    - –If you are alone and witness the sudden collapse of an infant or child, activate the emergency response system (if not already done), and retrieve an AED/defibrillator and use it immediately. For example, call 911 from your phone, mobilize the code team, or notify ALS.
    - –If you are alone and did not witness the sudden collapse of an infant or child, start high-quality CPR for 2 minutes. After about 2 minutes of CPR, if you are still alone and are unable to activate the emergency response system (have no mobile phone), leave the infant or child and activate the emergency response system and get the AED.

Seizure-like activity can also occur with cardiac arrest. If the infant or child is not responsive and not breathing normally, you should start CPR.

Critical Concepts

Signs of Poor Perfusion

- Temperature: Cool extremities
- Mental status: Continued decline in consciousness/responsiveness
- Pulses: Weak pulses
- Skin: Paleness, mottling (patchy appearance), and later cyanosis (turning blue)

If the collapse was witnessed, leave the infant or child and activate the emergency response system. Retrieve an AED and use it immediately.

If there is more than one rescuer, have one person activate the emergency response system and find an AED while the other begins CPR.

If the infant or child is not breathing normally or is only gasping and no pulse is felt, begin chest compressions.

Infant and Child Chest Compressions

Use the following compression-to-ventilation ratios for each population: for prepuberty, use 15:2 with 2 rescuers; for puberty and older, use 30:2 with 2 rescuers; and for any age, use 30:2 with 1 rescuer.

Chest Compression Technique

For infants, rescuers should compress the sternum with the heel of 1 hand or by using the 2 thumb–encircling hands technique. If the rescuer cannot physically encircle the chest, it is recommended to compress the chest with the heel of 1 hand.

For children, use either 1 or 2 hands to compress the chest, whichever technique allows the rescuer to achieve the desired compression depth. For the 2-hand technique, place the heel of one hand in the center of the chest and place the heel of other hand on top of the first hand. For 1-handed compressions, place the heel of one hand in the center of the chest. Compress the chest at least one third the anteroposterior (AP) diameter of the chest (approximately 2 inches, or 5 cm) with each compression.

These techniques are described in the next sections.

Infant and Child: Heel-of-1-Hand Technique

Follow these steps to use the heel of 1 hand on an infant or child:

1. Place the infant or child on a firm, flat surface.
2. Place the heel of one hand, just below the nipple line, on the lower half of the sternum. Do not press the tip of the sternum (Figure 5).
3. Compress at least one third the AP diameter of the chest, approximately 1½ inches (4 cm) for infants and 2 inches (5 cm) for children, at a rate of 100 to 120/min.

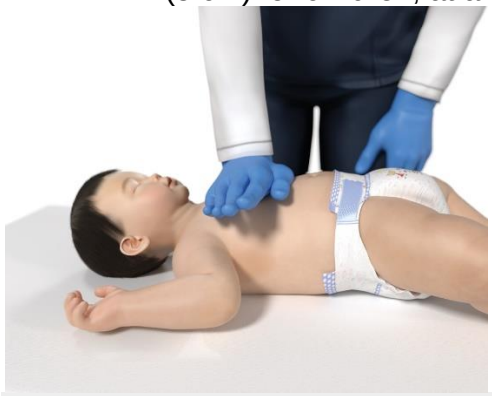


Figure 5. Heel-of-1-hand chest compression technique for an infant.

Infant: 2 Thumb–Encircling Hands Technique

Follow these steps to use the 2 thumb–encircling hands technique on an infant:

- Place the infant on a firm, flat surface.
- Place both thumbs side by side in the center of the infant's chest, on the lower half of the sternum. The thumbs may overlap in very small infants. Encircle the infant's chest and support the infant's back with the fingers of both hands (Figure 6).
- With your hands encircling the chest, use both thumbs to depress the sternum at a rate of 100 to 120/min.
- Compress at least one third the AP diameter of the infant's chest (approximately 1½ inches [4 cm]).

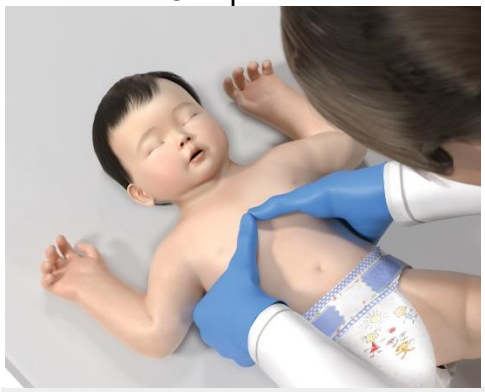


Figure 6. Two thumb–encircling hands technique for an infant.

Critical Concepts

Compression Depth in Adults vs Children and Infants

- Adults and adolescents: At least 2 inches (5 cm)
- Children: At least one third the AP diameter of the chest or approximately 2 inches (5 cm)
- Infants: At least one third the AP diameter of the chest or approximately 1½ inches (4 cm)

Infant and Child Breaths

Opening the Airway

Open the airway to make breaths effective. Two methods for opening the airway are the head tilt–chin lift and jaw-thrust maneuvers.

Important: If you suspect a head or neck injury, use the jaw-thrust maneuver to reduce neck and spine movement. If the jaw thrust does not open the airway, use the head tilt–chin lift maneuver.

To perform the head tilt–chin lift maneuver, place one hand on the infant or child's forehead and push with your palm to tilt the head back. Place the fingers of your other hand under the bony part of the lower jaw, near the chin. Lift the jaw to bring the chin forward. Avoid pressing the soft tissue under the chin. If you tilt (extend) an infant's head beyond the neutral (sniffing) position, the infant's airway may become blocked. Maximize airway patency by positioning the infant with the neck in a neutral position so that the external ear canal is level with the top of the infant's shoulder.

To perform a jaw thrust, position yourself at the infant or child's head. Place one hand on each side of the infant or child's head. Then, place your fingers under the angle of the lower jaw and lift with both hands, displacing the jaw forward. You may rest your elbows on the surface where they are lying. If the infant or child's lips close, open them by using your thumbs to push the lower lip down.

If you suspect a head or spinal injury, use a jaw-thrust maneuver to reduce movement of the spine. If a jaw thrust does not open the airway, use the head tilt–chin lift maneuver.

Critical Concepts

Jaw Thrust

A properly performed jaw thrust is the most effective means to open the pediatric airway. Health care professionals, however, are often inexperienced with this technique because it is difficult to practice the skill with some CPR manikins.

Why Breaths Are Important for Infants and Children in Cardiac Arrest

Infants and children who develop cardiac arrest often have respiratory failure or shock that reduces the oxygen content in the blood before the onset of arrest.

For infants and children in cardiac arrest, the oxygen in the blood is low. Adequate compressions alone will not provide the necessary oxygen to the heart and brain.

For this reason, it is very important to give both compressions and breaths to infants and children during high-quality CPR.

Ventilation for an Infant or Child With a Barrier Device

Use a barrier device (eg, pocket mask) or a bag-mask device to deliver breaths to an infant or child.

When providing bag-mask ventilation for an infant or child, do the following:

- Select a mask that covers the mouth and nose completely without covering the eyes or overlapping the chin.
- Perform a head tilt–chin lift to open the airway. Press the mask to the face as you lift the jaw, making a seal between the child's face and the mask.

- Connect supplemental oxygen when available.

Attempt Defibrillation With the AED

Use the AED as soon as it is available and follow the prompts. Apply pads to bare skin; AED pads must not be placed over any clothing.

After shock delivery or if no shock is advised, immediately resume high-quality CPR for 2 minutes (until prompted by the AED to allow for a rhythm check).

Pediatric BLS Algorithm for Health Care Professionals—2 or More Rescuers

Refer to the Pediatric BLS Algorithm (1 Year of Age to Puberty) for Health Care Professionals—2 or More Rescuers as you read the following steps (Figure 7).

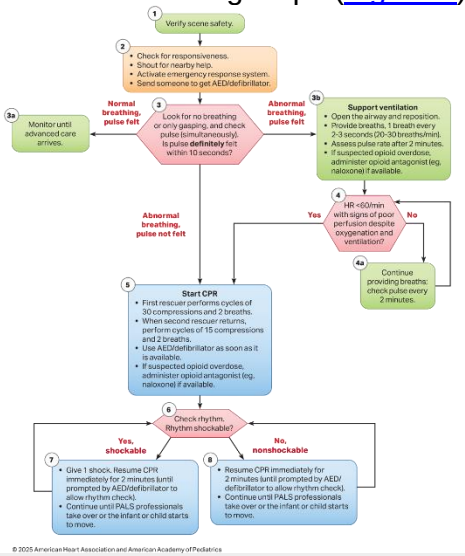


Figure 7. Pediatric BLS Algorithm (Infants to Puberty) for Health Care Professionals—2 or More Rescuers.

Abbreviation: HR, heart rate.

Algorithm legend: Red hexagon = decision or question; green box = action/steps to consider; orange box = assessment; gray box = assessment/action; blue box = CPR; white box with gray header = notes; and red, bold text = possible answers or decisions.

Infant and Child 2-Rescuer BLS Sequence

If the rescuer encounters an unresponsive infant or child and other rescuers are available, follow the steps outlined in the Pediatric BLS Algorithm (1 Year of Age to Puberty) for Health Care Professionals—2 or More Rescuers (Figure 7). The first components of the algorithm remain the same whether there are 1 or 2 rescuers. If 2 rescuers are available, send the second rescuer to activate the emergency response system and obtain an AED (Figure 8). Once the second rescuer arrives, they take over breaths, and the compression-to-ventilation ratio changes to 15:2 for prepubescent children. Switch compressors about every 5 cycles or 2 minutes (or earlier if needed) to avoid reducing CPR quality because of fatigue.

Figure 8A. If the arrest of an infant or child was sudden and witnessed, activate the emergency response system in your setting. A, In-facility setting.

Figure 8B. Prehospital setting.

Table 1 summarizes the components of high-quality CPR for all ages.

Table 1.Summary of High-Quality CPR Components for BLS Providers

Component	Adults and adolescents (postpuberty onset)	Children (age 1 year to puberty)	Infants (younger than 1 year, excluding newborns)
Verifying scene safety	Make sure the environment is safe for rescuers and the person		
Recognizing cardiac arrest	Check for responsiveness No breathing or only gasping (ie, no normal breathing) No definite pulse felt within 10 seconds (Breathing and pulse check can be performed simultaneously in less than 10 seconds)		
Activating emergency response system	**If a mobile device is available, call emergency services (911)**		
	If you are alone with no mobile phone, leave the person to activate the emergency response system and get the AED/defibrillator before beginning CPR Otherwise, send someone and begin CPR immediately; use the AED/defibrillator as soon as it is available	**Witnessed collapse** Follow the same steps as for adults and adolescents **Unwitnessed collapse** Give 2 minutes of CPR Leave the person to activate the emergency response system and get the AED Return to the child or infant and resume CPR; use the AED/defibrillator as soon as it is available	
Compression-to-ventilation ratio *without advanced airway*	**1 or 2 rescuers** 30:2	**1 rescuer** 30:2 **2 or more rescuers** 15:2	
Compression-to-ventilation ratio *with advanced airway*	Continuous compressions at a rate of 100-120/min Give 1 breath every 6 seconds (10 breaths/min)	Continuous compressions at a rate of 100-120/min Give 1 breath every 2-3 seconds (20-30 breaths/min)	

Pulse check	Carotid or femoral	Carotid or femoral	Brachial
Compression rate	100-120/min		
Compression depth	At least 2 inches (5 cm)*	At least one third the AP diameter of the chest Approximately 2 inches (5 cm)	At least one third the AP diameter of the chest Approximately 1½ inches (4 cm)
Hand placement	2 hands on the lower half of the sternum (sternum)	1 hand for a very small child or 2 hands for a large child, on the lower half of the sternum	**1 rescuer** Heel of 1 hand or 2 thumbs in the center of the chest, just below the nipple line **2 or more rescuers** 2 Thumb–encircling hands in the center of the chest, just below the nipple line If the rescuer is unable to achieve the recommended depth, it may be reasonable to use the heel of 1 hand
Chest recoil	Allow complete recoil of the chest after each compression; do not lean on the chest after each compression		
Minimizing interruptions	Limit interruptions in chest compressions to less than 10 seconds with a CCF goal of 80%		

Abbreviations: AP, anteroposterior; CCF, chest compression fraction.

*Compression depth should be no more than 2.4 inches (6 cm).

How to Position the Child

Properly position the child to maintain an open airway. During bag-mask ventilation, you may need to move the child's head and neck gently through a range of positions to optimize ventilation. A sniffing position without hyperextending the neck is usually best for infants and toddlers.

To achieve a sniffing position, place the child supine and flex the neck forward at the shoulder level while extending the head. Position the opening of the external ear canal at the level of or in front of the anterior aspect of the shoulder while the head is extended. Avoid hyperextending the neck because this may obstruct the airway.

Children older than 2 years may require padding under the occiput, while younger children and infants may need padding under the shoulders or upper torso to prevent excessive flexion of the neck that can occur when the prominent occiput rests on a flat surface.

Figure 9 shows these positions. In Figure 9A, the child is on a flat surface (eg, bed, table), and the oral (O), pharyngeal (P), and tracheal (T) axes pass through 3 divergent planes. In Figure 9B, a folded sheet or towel placed under the occiput aligns the pharyngeal and tracheal axes. In Figure 9C, extension of the atlanto-occipital joint results in the alignment of the oral, pharyngeal, and tracheal axes when the head is extended and the chin is lifted.

Note that the external ear canal is anterior to the shoulder. Figure 9D shows the incorrect position with neck flexion, and Figure 9E shows the correct position for ventilation and ET intubation for an infant. Note that the external ear canal is anterior to the shoulder.

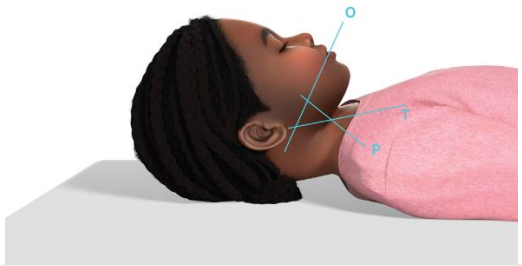


Figure 9A. Correct positioning of the child older than 2 years for ventilation and ET intubation. A, Child on a flat surface (eg, bed, table).

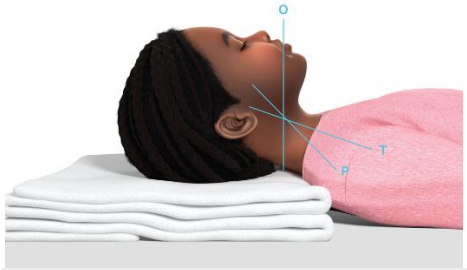


Figure 9B. **A folded sheet or towel under the occiput.**

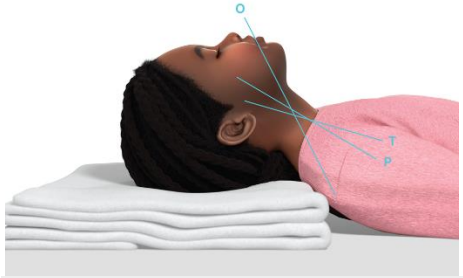


Figure 9C. **Extension of the atlanto-occipital joint.**

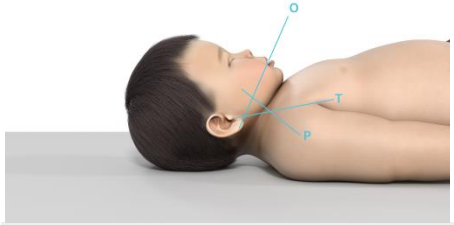


Figure 9D. **Incorrect position with neck flexion.**

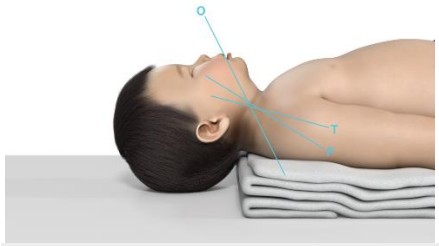


Figure 9E. Correct position for ventilation and ET intubation for an infant.

Modified from Coté CJ, Todres ID. The pediatric airway. In: Coté CJ, Ryan JF, Todres ID, Goudsouzian NG, eds. *A Practice of Anesthesia for Infants and Children*. 2nd ed. WB Saunders Co; 1993:55-83, copyright Elsevier.

Bag-Mask Ventilation

Bag-mask ventilation can adequately oxygenate and ventilate for a child with no or inadequate breathing or inadequate breathing despite an open/patent airway. Signs of inadequate breathing are apnea, abnormal respiratory rate, inadequate breath sounds, and hypoxemia despite supplemental O_2. When properly performed, bag-mask ventilation is as effective as ventilation through an ET tube for short periods and may be safer. In the out-of-hospital setting, bag-mask ventilation is especially useful if the transport time is short or health care professionals are inexperienced in inserting advanced airways or have insufficient opportunities to maintain competence in this skill.

How to Select and Prepare the Equipment

For ventilation to be effective with a bag-mask device, you must know how to select the face mask, prepare the ventilation bag (such as the self-inflating or flow-inflating bag), and provide supplemental O_2 if needed.

Face Mask

Select a transparent face mask, if available, that extends from the bridge of the child's nose to the cleft of the chin, covering the nose and mouth but not compressing the eyes because this may produce a vagal response and decrease the heart rate (Figure 10). A transparent mask allows you to see the color of the child's lips and condensation on the mask (which indicates exhalation) as well as regurgitation. The mask should have a soft rim (eg, flexible cuff) that molds easily to create a tight seal against the face. If the face-mask seal is not tight, O_2 intended for ventilation will escape under the mask and ventilation will not be effective.

Figure 10. Proper area of the face for face-mask application. Note that no pressure is applied to the eyes.

Bag Size

Use a self-inflating bag with a volume of at least 450 to 500 mL or larger for infants and young children because smaller bags may not deliver an effective tidal volume over the longer inspiratory times required by full-term neonates and infants. In older children or adolescents, you may need to use an adult self-inflating bag (1000 mL or larger) to achieve chest rise and minute ventilation.

How to Test the Bag-Mask Device

To ensure proper function of any bag and mask system, test all components before using by checking

- The bag for leaks by occluding the patient outlet valve with your hand and squeezing the bag
- Gas flow control valves (including any continuous positive airway pressure [CPAP] valve) to verify proper function
- The pop-off valve (if present) to ensure that it can be closed
- That O_2 tubing is securely connected to the device and the O_2 source
- The sound of O_2 flowing into the bag
- That the cuff of the mask (if present) is adequately inflated

How to Perform Bag-Mask Ventilation

One or two health care professionals can operate a bag-mask device while another rescuer provides compressions, but because effective bag-mask ventilation requires complex steps, it is not recommended for a lone rescuer to perform during CPR. Instead, the lone rescuer should use the mouth–to–barrier device technique for ventilation during CPR. Bag-mask ventilation can be provided effectively during 2-rescuer CPR.

1-Person Bag-Mask Ventilation Technique

If a single health care professional is performing bag-mask ventilation, they must open the airway and keep the mask sealed to the child's face with one hand (Figure 11) and squeeze the bag with the other hand. Form a tight seal between the mask and the child's face and open the airway by using the E-C clamp technique, described in the following steps.

1. If you do not suspect cervical spine injury, tilt the head back and lift the jaw against the mask, pressing and sealing the mask on the face. This technique moves the tongue away from the posterior pharynx, moves the jaw forward, and opens the mouth. If possible, the mouth should be open under the mask as a result of either lifting the jaw or inserting an OPA.
2. With the other hand, squeeze the ventilation bag until the chest rises. Deliver each breath over 1 second. Avoid excessive ventilation (refer to How to Deliver Effective Ventilation in this Part).

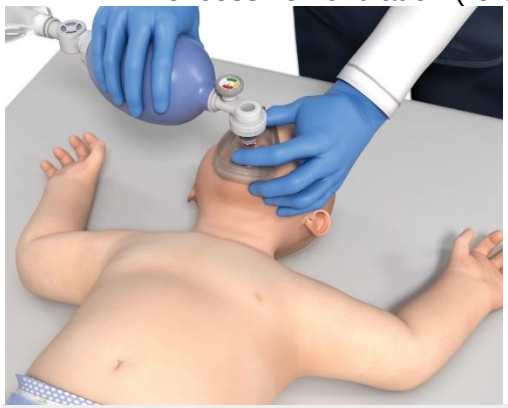


Figure 11. One-handed E-C clamp face-mask application technique. Three fingers of one hand lift the jaw (forming the "E") while the thumb and index finger hold the mask to the face (making a "C").

Critical Concepts
E-C Clamp Technique

The technique of opening the airway and making a seal between the mask and the face is called the *E-C clamp technique*. The third, fourth, and fifth fingers of one hand (forming an "E") are positioned along the jaw to lift it forward; then the thumb and index finger of the same hand (forming a "C") make a seal to hold the mask to the face. Avoid pressing on the soft tissues underneath the chin (the submental area) because this can push the tongue into the posterior pharynx, resulting in airway compression and obstruction.

2-Person Bag-Mask Ventilation Technique

If 2 health care professionals are available to perform bag-mask ventilation, one professional uses both hands to open the airway and keep the mask sealed to the child's face and the other squeezes the bag (Figure 12). Both should ensure that chest rise is visible. Be careful to avoid delivering too large a tidal volume, which may result in excessive ventilation.

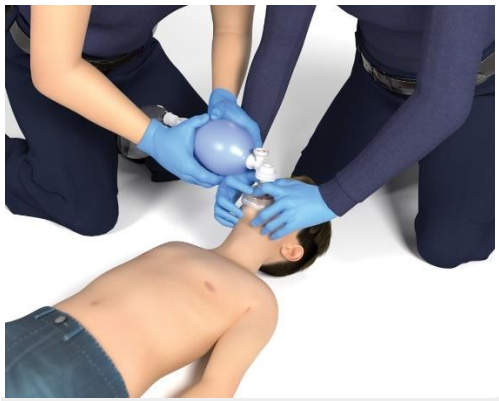


Figure 12. The 2-person bag-mask ventilation technique.

The 2-person technique may provide more effective bag-mask ventilation than a 1-person technique. Also, 2-person bag-mask ventilation may be necessary when

- Making a seal between the face and the mask is difficult
- The health care professional's hands are too small to reach from the front of the mask to behind the jaw or to open the airway and create a seal between the face and the mask
- Significant airway resistance (ie, asthma) or poor lung compliance (ie, pneumonia or pulmonary edema) occur
- Restricting spinal motion is necessary

How to Deliver Effective Ventilation

Avoid excessive ventilation; use only the force and tidal volume necessary to just make the chest rise.

Critical Concepts

Effective Ventilation With a Bag-Mask Device

Give each breath slowly over about 1 second. Watch for chest rise. If the chest does not rise, reopen the airway. Verify that there is a tight seal between the mask and the face. Reattempt ventilation.

Providing Positive End-Expiratory Pressure During Bag-Mask Ventilation

Using positive end-expiratory pressure (PEEP) may improve oxygenation in children with lung tissue disease or low lung volumes. Provide PEEP during ventilation with a self-inflating bag by adding a compatible spring-loaded ball or disk or a magnetic-disc PEEP valve to the bag-mask or bag-tube system. Do not use self-inflating bag-mask devices equipped with PEEP valves to provide CPAP during spontaneous breathing because the outlet valve in the bag will not open (and provide gas flow) unless the child generates significant negative inspiratory pressure. PEEP can be provided using a flow-inflating bag by adjusting the outflow of air during ventilation, though this can be difficult to perform without training and experience.

Clinical Parameters of Oxygenation and Ventilation

Frequently monitor the following parameters to assess the effectiveness of oxygenation and ventilation:

- Visible chest rise with each breath
- O_2 saturation
- Exhaled CO_2
- Heart rate
- Blood pressure
- Distal air entry
- Signs of improvement or deterioration (eg, appearance, color, agitation)

Precaution

Delivering excessive ventilation during CPR is harmful because it

- Increases intrathoracic pressure and impedes venous return, thus decreasing filling of the heart between compressions, reducing blood flow generated by the next compression, and reducing coronary perfusion and cerebral blood flow
- Causes air trapping and barotrauma in children with small airway obstruction
- Increases the risk of regurgitation and aspiration in children without an advanced airway

Troubleshooting Ineffective Ventilation

If you cannot achieve effective ventilation (ie, the chest does not rise), do the following:

- Reposition or reopen the airway; attempt to further lift the jaw and ensure that the child is placed in a sniffing position
- Verify mask size and ensure a tight face-mask seal
- Suction the airway if needed

- Check the O_2 source
- Check the ventilation bag and mask
- Treat gastric inflation
- Consider using 2-person bag-mask ventilation and inserting an OPA

Detecting for Changes in Lung Compliance

When performing bag-mask ventilation, be aware of the child's lung compliance. A poorly compliant lung is "stiff" or difficult to inflate. A sudden increase in lung stiffness during ventilation with a bag may indicate airway obstruction, decreased lung compliance, or development of a pneumothorax. Lung distention from excessive inflating pressures, PEEP, or rapid assisted respiratory rates with short exhalation time may also cause the feel of "stiff lungs" during ventilation.

Causes and Prevention of Gastric Inflation

Stomach inflation or distention frequently develops during bag-mask ventilation. Gastric inflation is more likely to develop during assisted ventilation if

- A partial airway obstruction is present
- High airway pressures are needed, such as in a child with poor lung compliance
- The bag-mask ventilation rate is too fast
- The tidal volume delivered is excessive
- The peak inspiratory pressure created is excessive (eg, more than 30 cm H_2O)
- The child is unconscious or is in cardiac arrest (because the gastroesophageal sphincter opens at a lower-than-normal pressure)

Gastric inflation can impair a child's ventilation by limiting lung volumes, interfering with effective ventilation, and causing regurgitation. To minimize gastric inflation

- Ventilate at a rate of 1 breath every 2 to 3 seconds (about 20-30 breaths/min)
- Use a manometer (if available) and, if at all possible, avoid generating excessive peak inspiratory pressures (eg, more than 30 cm H_2O) by delivering each breath over about 1 second
- Deliver enough volume and pressure to produce visible chest rise
- Consider administering cricoid pressure; although cricoid pressure is not routinely recommended during ET intubation of pediatric patients, it can be considered to reduce the risk of gastric inflation
    - –If cricoid pressure is applied, it must be discontinued if
        - A tracheal obstruction compromises bag-mask ventilation *or*
        - The speed or ease of intubation is compromised

Advanced professionals may perform gastric decompression by inserting a nasogastric or orogastric tube.

Spontaneously Breathing Child

In a spontaneously breathing child who requires bag-mask ventilation, it may be difficult to coordinate the bagged breaths with the child's inspirations when using a self-inflating bag. Not only may breaths provided with a self-inflating bag be ineffective, but poorly timed breaths may stimulate coughing, vomiting, laryngospasm, and gastric inflation, which prevent effective ventilation. In these situations, a flow-inflating bag (eg, anesthesia bag) may be more beneficial; these bags provide CPAP and flows that can be manipulated to the needs of the child. If necessary, the health care professional can also augment the child's spontaneous inspirations by squeezing the self-inflating bag.

Self-Inflating Bag

A self-inflating bag, typically used for initial resuscitation, consists of a bag with an intake valve and a nonrebreathing outlet valve. The intake valve allows the bag to fill with either O_2 or room air. When you compress the bag, the intake valve closes and the nonrebreathing outlet valve opens, allowing either room air or an air–O_2 gas mixture to flow to the child. When the child exhales, the nonrebreathing outlet valve closes to prevent the child from rebreathing CO_2 and exhaled gases are vented.

Figure 13 demonstrates how these bags work. Figures 13A and 13B show bags with O_2 reservoirs. When you release these bags, O_2 flows into the bag from the O_2 source and from the reservoir, so the concentration of O_2 in the bag remains 100%, and O_2 continuously flows into the reservoir. Figures 13C and 13D show bags without O_2 reservoirs. When you release these bags, O_2 flows into the bag from the O_2 source, but ambient air is also entrained into the bag. So, the bag becomes filled with a mixture of O_2 and ambient air. With both setups, exhaled patient air flows into the atmosphere near the mask and bag connection (see the gray arrows coming from the mask in Figures 13A and 13C).

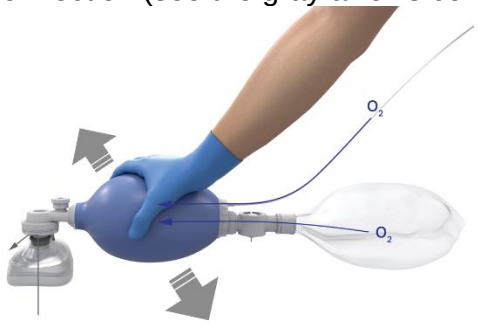


Figure 13A. Self-inflating ventilation bag with face mask, with (A and B) and without (C and D) O_2 reservoir. A, Re-expansion of the bag with O_2 reservoir.

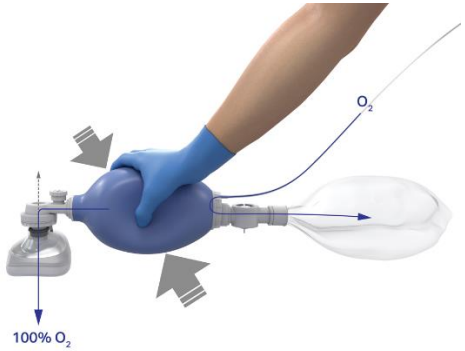


Figure 13B. **Compressing the bag with O_2 reservoir delivers 100% O_2 to the patient (purple arrow).**

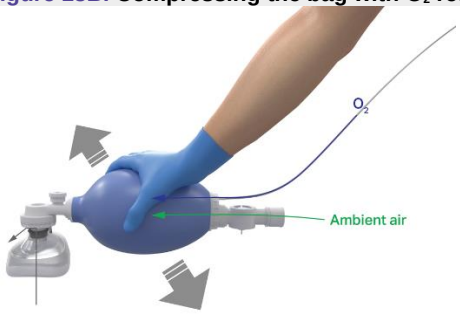


Figure 13C. **Re-expansion of the bag without an O_2 reservoir.**

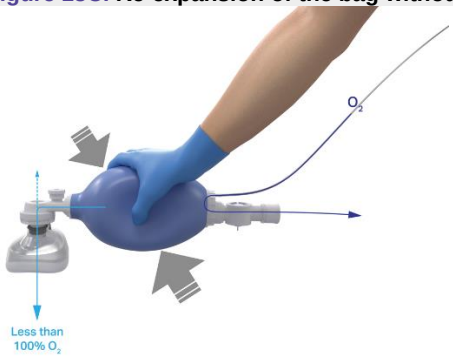


Figure 13D. **Compressing the bag without O_2 reservoir delivers O_2 mixed with room air (aqua arrow).**

The amount of delivered O_2 is affected by tidal volume and peak inspiratory flow rate. Even with supplemental O_2 attached, the delivered O_2 concentration varies from 30% to 80%. To deliver a high O_2 concentration (60%-95%), attach an O_2 reservoir to the intake valve. Maintain an O_2 flow of 10 to 15 L/min into a reservoir attached to a pediatric bag and a flow of at least 15 L/min into an adult bag.

Critical Concepts

Use O_2 During Resuscitation

Attach an O_2 reservoir to the self-inflating bag as soon as possible during a resuscitation attempt. Frequently verify that O_2 is attached and flowing to the bag. Remember to listen for O_2 flow and check O_2 tank pressure or verify connection to a wall O_2 source.

Check whether the bag has a pop-off valve. Many self-inflating bags have a pressure-limited pop-off valve set at 35 to 45 cm H_2O to prevent excessive airway pressures from developing. However, if the child has poor lung compliance, has high airway resistance, or needs CPR, an automatic pop-off valve may prevent delivery of sufficient tidal volume, resulting in inadequate ventilation and chest expansion. Ventilation bags used during CPR should have no pop-off valve, or the valve should be twisted into the closed position.

Critical Concepts

Continuous O_2 Flow Not Possible With Some Self-Inflating Bags

Self-inflating bag-mask devices with a fish-mouth or leaf-flap–operated nonrebreathing outlet valve do not provide a continuous flow of O_2 to the mask. Such valves open only if the bag is squeezed or the mask is sealed tightly to the face and the child generates significant inspiratory force to open the valve. Many infants cannot generate the inspiratory pressure required to open the outlet valve. Do not use this type of bag to provide supplemental O_2 or CPAP to a spontaneously breathing infant or child.

Flow-Inflating Bag

You may use the second kind of basic ventilation bag, a flow-inflating bag (Figure 14) (also called an *anesthesia bag*), in the intensive care unit, delivery room, and operating room.

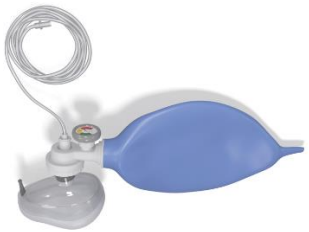


Figure 14A. **Flow-inflating bag with a pressure manometer. A, With a face mask.**

Figure 14B. **Without a mask.**

A flow-inflating bag may require more operator experience to provide safe and effective ventilation than is needed with a self-inflating bag. To ventilate effectively with a flow-inflating bag, the professional must be able to adjust the flow of O_2, adjust the outlet control valve, ensure a proper seal with the face mask, and deliver the appropriate tidal volume at the correct rate. For these reasons, only trained and expert professionals should use flow-inflating bags.

AED for Infants and Children Younger Than 8 Years

Be Familiar With the AED Equipment in Your Setting

Although all AEDs operate in basically the same way, AED equipment varies according to model and manufacturer. For instance, some AED models called *pediatric-capable AEDS*, are designed for both pediatric and adult use. These AEDs deliver a reduced shock dose when pediatric pads are used. Be familiar with the AED in your particular setting.

Delivering a Pediatric Shock Dose

You may reduce the AED shock dose by using pediatric cables or an attenuator. One commonly used method for reducing a shock dose is a pediatric dose attenuator (Figure 15). When attached to an AED, it reduces the shock dose by about two thirds. Typically, you can use child pads to deliver the reduced shock dose.

Figure 15. Example of a pediatric dose attenuator, which reduces the shock dose delivered by an AED. Child pads are also used with this attenuator.

Choosing and Placing the AED Pads

Use child pads (Figure 16), if available, for infants and for prepubescent children. If child pads are not available, use adult pads. Make sure the pads do not touch each other or overlap. Adult pads deliver a higher shock dose but are preferable to no shock at all. If the AED has a key or switch to deliver a child shock dose, turn the key or switch. For children who have entered puberty, use adult pads (Figure 17)—child pads will likely give a shock dose that is too low.

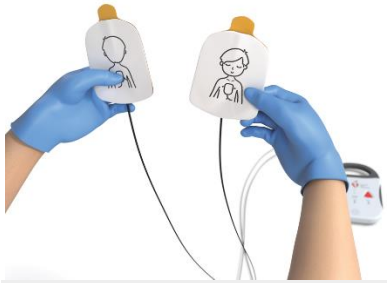


Figure 16. **Child AED pads.**

Figure 17. **Adult AED pads.**

Follow the AED manufacturer's instructions for pad placement and weight recommendations. Illustrations are located on the pads. Weight recommendations will be located on the packaging. Some AEDs require that child pads be placed in a front and back (AP) position (Figure 18), while others require right-left (anterolateral) placement (Figure 19). AP pad placement is commonly used for infants.

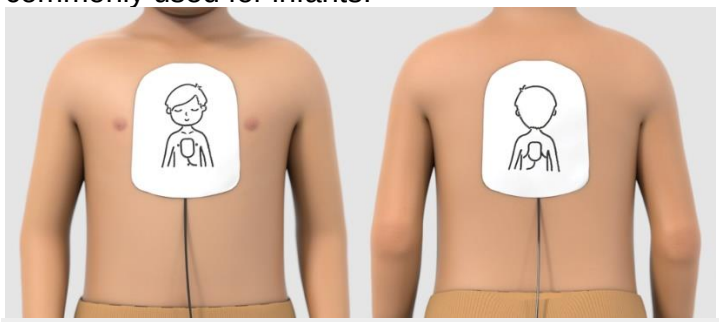


Figure 18. **AP AED pad placement on a child.**

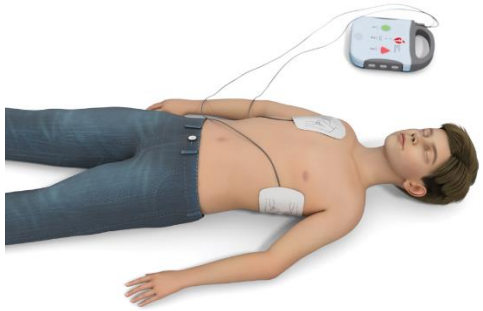


Figure 19. Anterolateral AED pad placement on a child. Right anterior pad: Should be placed vertically on the child's right upper chest, with the top of the pad just below the clavicle. Lateral pad: Should be placed horizontally, centered at the mid-axillary line. The top of the pad should be on the lower ribcage, in line with the sternal border (inferior portion of the xyphoid process). Do not place the pad on breast tissue or on the abdomen.

Using an AED for Infants

For infants, a manual defibrillator is preferred to an AED for defibrillation because it has more capabilities than an AED and can provide lower energy doses needed in infants. Operating a manual defibrillator requires advanced training that will be covered in Part 11 of this course.

- If a manual defibrillator is not available, an AED equipped with a pediatric dose attenuator is preferred.
- If neither is available, you may use an AED without a pediatric dose attenuator.

Part 3
High-Performance Teams

Learning Objective

After completing this Part, you should be able to apply high-performance team dynamics to increase your patients' chance of survival.

During the course, you will participate as both the Team Leader and a team member during case simulations, modeling the behaviors discussed in this Part.

High-performance teams are essential to successful resuscitation attempts, carrying out their roles effectively. This results in superior performance and timing, translating to improved survival for patients in cardiac arrest. These teams incorporate timing, quality, coordination, and administration of appropriate procedures during a cardiac arrest. Each team member should be committed to ensuring high quality performance, not simply following orders. Each member needs to focus on

- **Timing** (immediate CPR and defibrillation, limiting pre- and postshock pause, emergency response): minimize interruptions and pauses in chest compressions and increase chest compression fraction (CCF)
- **CPR quality**: achieve correct rate and depth, allow complete recoil, minimize pause times, avoid hyperventilation
- **Coordination** (team dynamics): team members working together seamlessly toward a common goal, proficient in their roles including closed-loop communication and team coordination
- **Administration**: leadership, planning ahead, obtaining additional resources, tailoring the interventions, continuous quality improvement measurement, assigned number of members working an arrest

High-performing systems target a CCF of at least 60%, with 80% or higher being a frequent goal. High-performance teams (Figure 20) will need to incorporate timing, quality, coordination, and administration of the appropriate procedures during a cardiac arrest. The team will need to consider their overall purpose and goals, skills each team member possesses, appropriate motivation, and efficacy as well as appropriate conflict resolution and the communication needs of the team. In addition, high-performance teams measure their performance, evaluate the data, and look for ways to improve performance and implement the revised strategy.

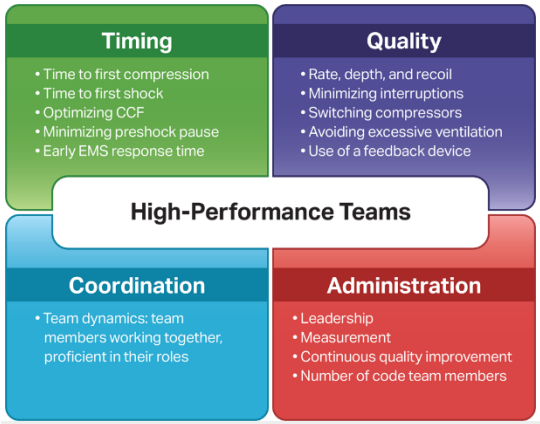


Figure 20. Key areas of focus for high-performance teams to increase survival rates.

Critical Concepts
Ways to Decrease Pause Times (Increase CCF)

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

- Precharge the defibrillator 15 seconds before a 2-minute rhythm analysis (deliver shock immediately if VF or pulseless ventricular tachycardia [pVT] on the monitor). This makes it possible to conduct a rhythm analysis and give a shock (if needed) within 10 seconds or less. If the rhythm analysis shows that a shockable rhythm is not present, dump the charge. This process will vary by manufacturer.
- Palpate a **pulse during the precharge phase** in anticipation of an organized rhythm during analysis (a pulse check during compressions is not a reliable indicator of CPR quality).
- The compressor hovers over the chest (not touching it), ready to start chest compressions immediately after a shock, a rhythm analysis, or other necessary pauses in compressions.
- Have the next compressor ready to take over immediately.
- If placing an advanced airway, do so without pausing compressions.
- Deliver medications during compressions.

High-Performance Team Roles and Dynamics

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

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

Critical Concepts

Understanding Team Roles

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

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

Roles in a High-Performance Team

Team Leader Role

Every high-performance team needs a leader who directs the resuscitation, focusing on the comprehensive patient care and organizing the efforts of the team. Members of a high-performance team should focus on their individual tasks. The Team Leader

- Organizes the group by delegating roles and tasks
- Monitors individual performance of team members
- Backs up team members
- Models excellent team behavior
- Trains and coaches
- Communicates treatment plan and patient status
- Focuses on comprehensive patient care
- Temporarily designates another team member to take over as Team Leader if their attention is occupied by another task such as advanced airway placement or speaking to a family

The Team Leader ensures everything is done correctly and timely by monitoring and integrating individual performances. They help team members understand why certain tasks must be performed in specific ways, such as pushing hard and fast in the center of the chest, ensuring complete chest recoil, minimizing interruptions in chest compressions, and avoiding excessive ventilation.

The Team Leader also helps team members understand why they must perform certain tasks in a specific way. While any team member can perform these skills, the Team Leader is ultimately responsible to explain why it is essential to

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

Monitoring for CPR Quality

During the resuscitation attempt, the Team Leader as well as team members should monitor CPR quality. Use of a CPR coach along with good team communication will ensure that chest compressions are the appropriate depth and rate, that the chest is allowed to completely recoil after each compression, and that ventilation is not excessive. Elements of high-quality CPR include the following:

- Push fast: Push at a rate of 100 to 120/min for infants, children, and adults. Feedback devices may be helpful in assisting team members with correct rate.
- Push hard: Push with enough force to depress the chest at least one third the AP diameter of the chest in pediatric patients (infants [younger than 1 year] to children up to the onset of puberty). This equates to approximately 1½ inches (4 cm) in infants and 2 inches (5 cm) in children. Once children have reached puberty (ie, adolescents), use the recommended adult compression depth of at least 2 inches (5 cm), but no greater than 2.4 inches (6 cm).
- Chest compression recoil: Allow rapid and complete chest recoil after each compression; this allows coronary perfusion and allows the heart to refill with blood.
- Minimize interruptions: Limit interruptions in chest compressions to 10 seconds or less or as needed for interventions (eg, defibrillation). Ideally, compressions are interrupted only for ventilation (until an advanced airway is placed), rhythm check, and shock delivery. Once an advanced airway is in place, provide continuous chest compressions and asynchronous ventilation (ie, without pausing compressions for ventilation).
- Avoid excessive ventilation: Each rescue breath should be given over about 1 second and should result in visible chest rise. After an advanced airway is in place, deliver 1 breath every 2 to 3 seconds or about 20 to 30 breaths per minute (in an infant or child), being careful to avoid excessive ventilation.

Team Member Roles

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

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

Team Member Role: CPR Coach

Many resuscitation teams now include the role of CPR Coach. The CPR Coach supports performance of high-quality BLS skills, allowing the Team Leader to focus on other aspects of clinical care. Studies have shown that resuscitation teams with a CPR Coach perform higher-quality CPR with higher CCF and shorter pause durations compared with teams that do not use a CPR Coach. It is important to train specific team members for the role of CPR Coach. The CPR Coach does not need to be a separate role; they can be most effectively blended into the current responsibilities of the Monitor/Defibrillator. Teams with a trained CPR Coach tend to perform better CPR and have reduced no-flow time during simulated cardiac arrest.

The CPR Coach's main responsibilities are to help team members provide high-quality CPR and minimize pauses in compressions. The CPR Coach needs a direct line of sight to the Compressor, so they should stand directly across from the Compressor, next to the defibrillator. Below is a description of the CPR Coach's actions:

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

Coach to improve the quality of chest compressions: The CPR Coach gives feedback about performance of compression depth, rate, and chest recoil. They state the CPR feedback device's data to help the Compressor improve performance. This is useful because visual assessment of CPR quality is often inaccurate. When chest compressions are being delivered appropriately, positive feedback should be provided to promote excellent performance.

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

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

Help minimize the length of pauses in compressions: The CPR Coach communicates with the team to help minimize the length of pauses in compressions. Pauses happen when the team defibrillates, switches Compressors, and places an advanced airway. The CPR Coach should verbally preview when pauses are upcoming and encourage compressors to resume chest compressions once the task is complete.

Critical Concepts

CPR Coach Role

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

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

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

The remaining roles of an effective team and their duties and responsibilities are outlined in Figure 21. Positioning of personnel is important and highlighted for clinical events.

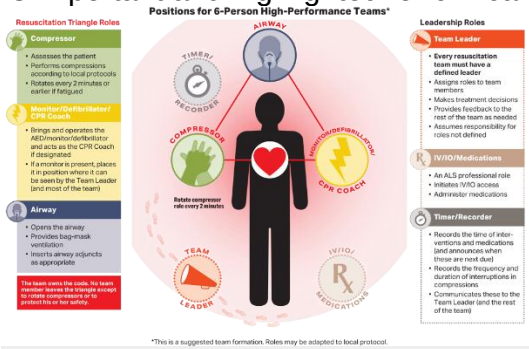


Figure 21. Suggested locations for the Team Leader and team members during case simulations and clinical events.

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

Roles

Clear Roles and Responsibilities

When fewer than 6 people are present, Team Leaders must prioritize these tasks and assign them to the health care professionals present. Figure 21 identifies 6 team roles for resuscitation.

In resource-rich hospitals, the option of having a pharmacist attending a code to assist with medication administration may be helpful. Table 2 lists some additional information about roles.

Table 2 Clear Roles and Responsibilities

Team member	Task
Team Leader	• Clearly define all team member roles in the clinical setting • Distribute tasks evenly to all available team members who are sure of their responsibilities • Encourage team members to participate actively
Team members	• Seek out and perform clearly defined tasks appropriate to their abilities • Accept only assignments that are within their level of expertise or ask for a new assignment • Participate actively, not just follow directions • Communicate ability to handle additional responsibilities • Notify Team Leader if more help is needed

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

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

Knowing Your Limitations

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

During the stress of an attempted resuscitation, do not practice or explore a new skill, especially without seeking advice from more experienced personnel. If you need extra help, request it early rather than waiting until the patient deteriorates further. Asking for help is not a sign of weakness or incompetence; it is better to have more help than needed rather than not enough help, which might negatively affect patient outcome. Table 3 lists some additional information about knowing your limitations.

Table 3.Knowing Your Limitations

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

Constructive Interventions

During a resuscitation attempt, anyone on a high-performance team may need to intervene tactfully if a team member is about to take an inappropriate action. Team Leaders should avoid confrontation with team members and instead debrief afterward if needed. Table 4 lists some additional information about constructive interventions.

Table 4.Constructive Interventions

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

What to Communicate

Knowledge Sharing

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

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

When resuscitation efforts are ineffective, go back to the basics and talk as a team. Discuss the findings from the primary assessment and resuscitation and ask, "Is there something we need to reevalute? Is there anything else we need to consider?" High-performance team members should provide all available information about changes in the patient's condition to ensure that the Team Leader makes appropriate decisions. Table 5 lists some additional information about knowledge sharing.

Table 5.Knowledge Sharing

Team member	Task
Team Leader	• Encourage information sharing; share mental model • Ask for suggestions about interventions, differential diagnoses, and possible overlooked treatments (eg, IV access, medication treatments) • Look for clinical signs that are relevant to the treatment
Team members	• Share information with each other • Accept information that will improve their roles

Summarizing and Reevaluating

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

Team Leaders should periodically state this information to the team and announce the plan for the next few steps. Another way to describe this process is having and sharing a mental model, or the leader's internal view of what is going on. This allows team members the opportunity to agree/disagree, ask clarifying questions, and anticipate next steps in care. Remember that

the patient's condition can change. Be flexible to changing treatment plans and ask for information and summaries from the Timer/Recorder as well. Table 6 lists some additional information about summarizing and reevaluating.

Table 6.Summarizing and Reevaluating

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

How to Communicate

Closed-Loop Communications

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

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

Table 7 lists some additional information about closed-loop communications.

Table 7.Closed-Loop Communications

Team member	Task

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

Clear Messages

Clear messages include concise, calm, and direct instructions. Unclear communication can cause treatment delays or medication errors, so health care professionals should avoid yelling or shouting, which can impair team interaction. Only one person should speak at a time. Table 8 lists some additional information about clear messages.

Table 8.Clear Messages

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

Mutual Respect

High-performance team members must respect each other and work together in a collegial, supportive manner. Ego must be abandoned, and respect must be shown during the resuscitation attempt, regardless of any additional training or experience specific team members may have. Table 9 lists some additional information about mutual respect.

Table 9.Mutual Respect

Team member	Task
Team Leader	• Acknowledge correctly completed assignments by providing positive feedback

Team Leader and team members	• Show interest and listen to what others say • Speak in a friendly, controlled tone of voice • Positively acknowledge team members • Avoid displaying aggression if teammates do not initially understand each other • Understand that when one person raises their voice, others will respond similarly • Try not to confuse directive behavior with aggression

Part 4

Systematic Approach to the Seriously Ill or Injured Child

You should use a systematic approach when caring for a seriously ill or injured child so that you can quickly recognize signs of respiratory distress, respiratory failure, and shock and immediately provide lifesaving interventions.

Learning Objective

After completing this Part, you should be able to differentiate between patients who do and do not require immediate intervention.

You should know all the concepts presented in this Part to identify the child's clinical condition and target appropriate management in case simulations. The ongoing process of evaluate-identify-intervene is a core component of systematic evaluation and care of a seriously ill or injured child.

The PALS Systematic Approach Algorithm

The PALS Systematic Approach Algorithm (Figure 22) outlines the approach to caring for a critically ill or injured child.

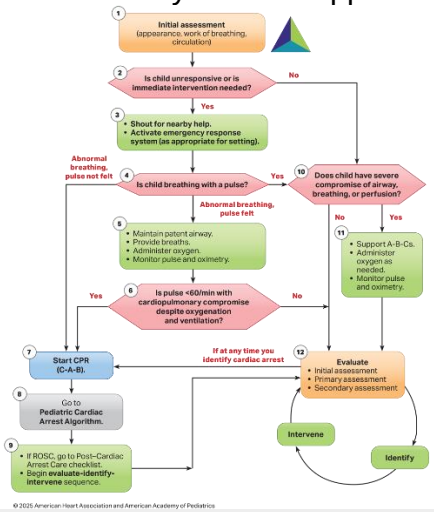


Figure 22. PALS Systematic Approach Algorithm.

Abbreviation: C-A-B, compressions, airway, breathing.

Algorithm legend: Red hexagon = decision or question; green box = action/steps to consider; orange box = assessment; gray box = assessment/action; blue box = CPR; white box with gray header = notes; and red, bold text = possible answers or decisions.

Identify a Life-Threatening Condition and Act

The initial assessment helps you to quickly identify a life-threatening condition. If you identify a life-threatening problem, immediately begin appropriate interventions and activate the emergency response system as indicated in your practice setting. If the child's condition is not life threatening, continue with the systematic approach.

Critical Concepts

Life-Threatening Problems

Signs of a life-threatening condition include

- Airway: complete or severe airway obstruction
- Breathing: apnea, significant increased work of breathing, bradypnea
- Circulation: weak or absent pulses, poor perfusion, hypotension, bradycardia
- Disability: unresponsiveness, decreased level of consciousness
- Exposure: significant hypothermia or hyperthermia, significant bleeding, petechiae, or purpura consistent with septic shock or coagulation problem

Child Who Is Unresponsive and Not Breathing or Is Only Gasping

If the child is unresponsive, shout for help.

If the child is unresponsive with abnormal breathing and a pulse is not felt, activate the emergency response system and begin CPR. If ROSC, implement the Post–Cardiac Arrest Care checklist.

Check Breathing and Pulse

If Abnormal Breathing, but a Pulse Is Felt, Provide Rescue Breaths

Administer oxygen as soon as it is available. (Refer to Rescue Breathing in Part 6 for more information.) For infants and children, give 1 breath every 2 to 3 seconds (about 20-30 breaths/min), and give each breath over 1 second.

Check the heart rate. If the heart rate is less than 60/min with signs of poor perfusion despite adequate oxygenation and ventilation, provide chest compressions and breaths.

If the heart rate is 60/min or greater, continue rescue breathing as needed.

If Normal Breathing and a Pulse Is Felt

If unresponsiveness is a new finding and breathing and pulse are adequate, activate the emergency response system as appropriate for your setting. Continue your systematic assessment.

Initial Assessment to Identify a Life-Threatening Condition

Use the Pediatric Assessment Triangle (PAT) to make your initial assessment during your first quick hands off “from the doorway” observation of the child (Figure 23). Upon entering the room, with the first step, begin with observing the appearance of the child. With the second step, observe the work of breathing; and with the third step as you get closer to the child, observe the skin color. You can use the PAT immediately on entering the scene to help form your general impression. The PAT will help identify the general type of physiologic problem (ie, respiratory, circulatory, or neurologic) and urgency for treatment. It will also help prehospital health care professionals in determining transport priority as well as transport logistics.

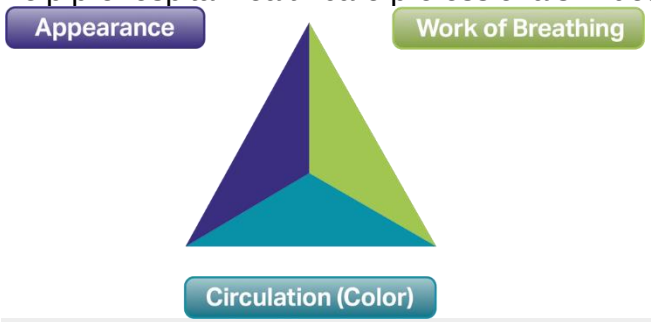


Figure 23. Pediatric Assessment Triangle.

After listening and observing the child and noting the abnormal components of the PAT, categorize the child as to whether they are sick, sicker, or sickest. A child who presents and nothing is noted to be abnormal in the triangle is considered to be sick because the parents have noted a change in the child. If 1 component of the PAT is abnormal, the child is considered to be sicker. If 2 or more areas of the triangle are abnormal, categorize the child as sickest.

Initial Assessment

Use the PAT to form your initial impression. If you identify a life-threatening situation from the PAT, you will begin appropriate interventions.

The PAT uses A-B-C, which stands for appearance, work of breathing, and circulatory status (Figure 24). It begins with evaluating appearance (A) as an indicator of overall physiologic status, including degree of interactivity, muscle tone, and verbal response or cry. Use the TICLS (tone, interactivity, consolability, look/gaze, speech/cry) mnemonic as an adjunct. The second component of the PAT is breathing (B), which determines whether a child has increased work of breathing by assessing the patients' position (ie, tripod or sniffing position), work of breathing (ie, retractions), and adventitial breath sounds (eg, stridor, sonorous respirations). The final component of the PAT evaluates the child's overall circulatory status (C) based on general color (eg, pale, mottled, cyanotic). A child with abnormal PAT findings requires prompt evaluation and management. The findings of the PAT may indicate need for immediate intervention (eg, CPR for a patient who is apneic and pulseless, tourniquet use for exsanguinating hemorrhage of an extremity).

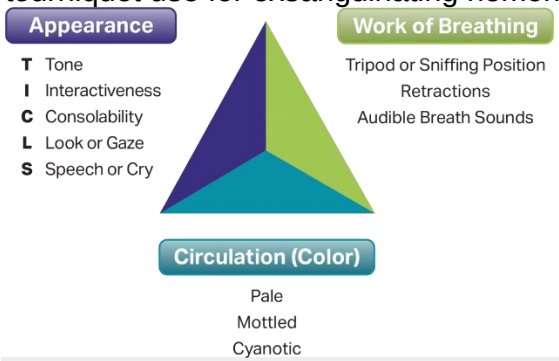


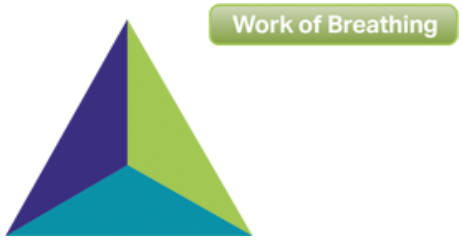
Figure 24. Initial assessment of the PAT.

Appearance

The first part of the PAT is the child's appearance, including level of consciousness and ability to interact. Carefully, but quickly, observe the child's appearance to evaluate the level of consciousness by observing the child's TICLS. If the child is unresponsive, shout for nearby help, assess breathing and pulse, and then activate the rapid response or the emergency response system as appropriate for your clinical setting.

If the child is crying or upset, it can be difficult to know if they are responding appropriately. Try to keep the child as calm as possible by letting them remain with their parent or caregiver if practical and use distractions such as toys.

Breathing



The next part of the PAT is evaluating the child's work of breathing, position, and any audible breath sounds (ie, sounds that can be heard without a stethoscope) (Table 10). Look for signs of absent or increased respiratory effort. Listen for obvious sounds of abnormal breathing, such as grunting, stridor, or wheezing. Note whether the patient's position suggests respiratory distress, such as the tripod position.

Table 10.Evaluating Work of Breathing

Evaluate	Normal	Abnormal
Respiratory effort*	• Regular breathing, no increased effort • Passive expiration	• Nasal flaring • Retractions or use of accessory muscles • Increased, inadequate, or absent respiratory effort • Seesaw (abdominal expansion with chest collapse) • Tripod positioning
Lung and airway sounds*	No abnormal respiratory sounds audible	Noisy breathing (eg, wheezing, grunting, stridor)

*Refer to detailed discussion of respiratory effort and lung and airway sounds in the Primary Assessment section in this Part.

Circulation (Color)

Circulation (Color)

To complete the third part of the PAT, assess the child's overall circulatory status. Assess the child's color, including skin color and pattern or obvious significant bleeding, which may help you assess how well the child is perfusing. You can often identify important information about circulatory status just by looking at a child.

Pallor (paleness), mottling (a patchy or lacy discoloration of the skin, often purple or blue appearing), or cyanosis (bluish/gray skin color) suggests poor perfusion, poor oxygenation, or both. Cyanosis of the lips and fingernails may appear if the child cannot adequately oxygenate their blood.

Observe the exposed parts of the child, such as the face, arms, and legs. A flushed appearance may suggest fever or distributive shock such as from sepsis, toxins, or anaphylaxis. A skin inspection may reveal bruising that suggests injury. You may also see evidence of bleeding within the skin, called *petechiae* or *purpura*. This purplish discoloration of the skin may be a sign of a life-threatening infection.

Evaluate the skin (Table 11). Is it normal or abnormal?

Table 11 Initial Evaluation of the Skin

Evaluate	Normal	Abnormal

Skin color*	Appears normal	• Pallor • Mottling • Cyanosis
Visible bleeding or wounds	Not present	• Obvious significant bleeding • Bleeding within the skin (eg, purpura) • Nonblanching pink dots on the skin (petechiae)

*Refer to the discussion of skin color in the Primary Assessment section in this Part.

Evaluate-Identify-Intervene

Use the evaluate-identify-intervene sequence (Figure 25) when caring for a seriously ill or injured child to help you determine the best treatment or intervention at any point. From the information gathered during your evaluation, identify the child's clinical condition by type and severity. Intervene with appropriate actions, and then repeat the sequence. This process is ongoing.

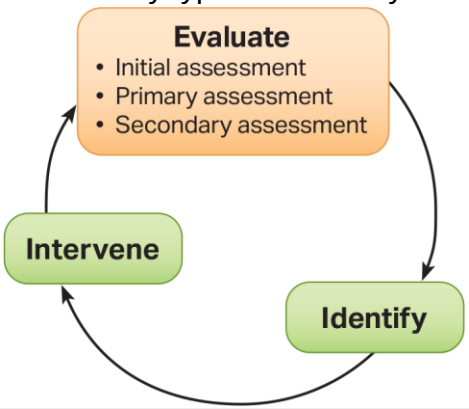


Figure 25. Evaluate-identify-intervene sequence.

If at any point you identify a life-threatening problem, immediately activate the emergency response system (or send someone to do so) while you begin lifesaving interventions.

Sometimes a child's condition may seem stable despite the presence of a life-threatening problem. Examples include a child who has ingested a toxin but is not yet showing effects or a trauma with internal bleeding who may initially maintain blood pressure by increasing heart rate and systemic vascular resistance (SVR). Reassess frequently.

Evaluate

Always verify that the scene is safe and assess for potential environmental dangers before you evaluate the child, especially in out-of-hospital settings.

If no life-threatening condition is present, evaluate the child's condition by using these clinical assessment tools:

- Initial assessment: a first, quick, "from the doorway" observation of the child's appearance, breathing, and color, performed within the first few seconds after encountering the child
- Primary assessment: a rapid, hands-on ABCDE approach to evaluate respiratory, cardiac, and neurologic function; includes assessment of vital signs and pulse oximetry
- Secondary assessment: a focused medical history and a focused physical exam

Identify

Respiratory illness can present as respiratory distress that can progress to respiratory failure:

- Upper airway obstruction
- Lower airway obstruction
- Lung tissue disease, disordered control of breathing

Compensatory shock can progress to hypotensive shock. Shock can be categorized into

- Hypovolemic shock
- Distributive shock
- Cardiogenic shock
- Obstructive shock

Respiratory failure and hypotensive shock can further progress to cardiopulmonary failure and cardiac arrest.

The child's clinical condition can result from a combination of respiratory and circulatory problems. As a seriously ill or injured child deteriorates, one problem may lead to others, such as cardiopulmonary failure and cardiac arrest. Note that in the initial phase of your identification, you may be uncertain about the type or severity of problems.

Identifying the problem will help you determine the best initial interventions. Recognition and management are discussed in detail later in this manual.

Intervene

Once you identify the child's clinical condition, intervene with appropriate actions within your scope of practice. Interventions may include

- Positioning the child to maintain an open/patent airway
- Activating the emergency response system
- Starting CPR
- Obtaining the code cart and monitor
- Placing the child on a cardiac monitor and pulse oximeter
- Administering O_2
- Supporting ventilation
- Starting medications and fluids (eg, nebulizer treatment, IV/IO fluid bolus)

Continuous Sequence

Continue the evaluate-identify-intervene sequence until the child is stable. Use this sequence before and after each intervention to look for trends in the child's condition. For example, after you give O_2, reevaluate the child. Is the child breathing a little easier? Are color and mental status improving? After you give a fluid bolus, reassess the child, looking for improvement in capillary refill, central and peripheral pulses, skin color, and temperature. For example, has capillary refill improved from 5 seconds to 4 seconds? Have peripheral pulses improved in strength, going from weak to strong? Has the skin color improved from pale to pink? Use the evaluate-identify-intervene sequence to evaluate the need for a second fluid bolus. If capillary refill is prolonged and the skin perfusion is poor, identify the child remains in shock and intervene by administering another fluid bolus. After the second fluid bolus, evaluate the same signs to determine improvement. Use the evaluate-identify-intervene sequence when the child's condition changes.

Critical Concepts

The Evaluate-Identify-Intervene Sequence Is Continuous

Remember to repeat the evaluate-identify-intervene sequence until the child is stable:

- After each intervention
- When the child's condition changes or deteriorates

Primary Assessment

The primary assessment uses a hands-on ABCDE approach and includes assessment of the patient's vital signs, including oxygen saturation by pulse oximetry (Figure 26).

- Airway
- Breathing
- Circulation
- Disability
- Exposure

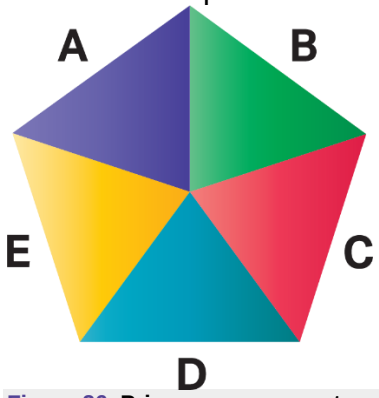
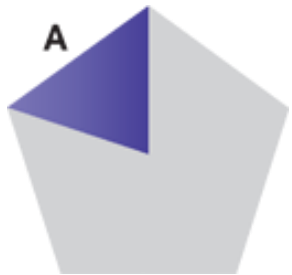


Figure 26. Primary assessment.

As you proceed through each component of the primary assessment, treat life-threatening abnormalities in real time, before you complete the remainder of the primary assessment. In patients with life-threatening conditions evident, finish the primary assessment because correcting those conditions takes precedence over establishing baseline vital sign measures such as blood pressure or pulse oximetry. When you complete the primary assessment and after you address any life-threatening problems, proceed to the secondary assessment.

Airway



When you assess the airway, you determine if it is open/patent. To assess airway openness/patency:

- Look for chest or abdomen movement
- Listen for air movement and breath sounds
- Feel for air movement at the nose and mouth

To check for breathing, scan the child's chest for rise and fall for no less than 5 seconds and no greater than 10 seconds.

- If the child is breathing, monitor until additional help arrives.
- If the child is not breathing or is only gasping, this is abnormal breathing and may be a sign of cardiac arrest.

To minimize delay in starting CPR, you may assess breathing at the same time as you check the pulse. This should take no less than 5 seconds and no greater than 10 seconds.

The following signs suggest that the **upper** airway may be obstructed:

- Increased inspiratory effort with retractions
- Abnormal inspiratory sounds (snoring or high-pitched stridor)
- Episodes where no airway or breath sounds are present despite respiratory effort (ie, complete upper airway obstruction)

If the upper airway is obstructed, determine if you can open and maintain the airway with simple measures or if you need advanced interventions.

Measures to Maintain the Airway

Measures to open and maintain an open/patent upper airway may include one or more of the following:

Positioning

For a responsive child, allow the child to assume a position of comfort or elevate the head of the bed. For an unresponsive child, position supine, if you do not suspect cervical injury or use a head tilt–chin lift or jaw thrust to improve airway patency.

Opening the Airway

Open the airway by using the head tilt–chin lift maneuver or jaw thrust without neck extension (see Opening the Airway in Part 2). Avoid overextending the head and neck in infants because this may occlude, or block, the airway. Opening the airway is a priority. During CPR, stabilize the head and neck manually rather than with immobilization devices.

Suctioning

Suction the nose and oropharynx. Avoid overextending the head and neck in infants because this may occlude the airway.

Managing FBAO

If you suspect an FBAO that is mild (the child is able to make sounds and cough forcefully), do not intervene. Call for help and allow the child to try to clear the obstruction by coughing. If you suspect that the FBAO is severe (the child or infant makes no sound, is unable to speak or cough, has poor or no air exchange, makes a high-pitched noise while inhaling or no noise at all, has increased respiratory difficulty), perform the maneuvers in Table 12.

Table 12.Managing FBAO

Interventions for a responsive infant or child with FBAO	
Infant (younger than 1 year)	**Child (1 year to adolescent [puberty])**
• Confirm severe airway obstruction • Activate the emergency response system in case the infant becomes unresponsive • Alternate giving 5 back blows followed by 5 chest thrusts with the heel of 1 hand until the object is expelled or the infant becomes unresponsive	• Ask, “Are you choking?” If the child nods or otherwise indicates “yes,” say you’re going to help • Activate the emergency response system in case the child becomes unresponsive • Perform 5 back blows followed by 5 abdominal thrusts • Continue alternating 5 back blows followed by 5 abdominal thrusts until the object is dislodged or the person becomes unresponsive
• To perform back blows, use the heel of your hand and forcefully strike the infant or child’s back in between their shoulder blades. You’ll do this 5 times. If back blows do not relieve choking, stand or kneel behind the child and perform 5 abdominal thrusts	
Child or infant becomes unresponsive	
• Shout for help. Send someone to activate the emergency response system • Place the infant on a firm surface or lower the child to the floor. If the infant or child is unresponsive with no breathing or only gasping, begin CPR (no pulse check) • Each time you open the airway to deliver breaths, look into the mouth. If you see an object that can be easily removed, remove it. If you do not see an object, continue CPR. *Note:* Do not perform a blind finger sweep to dislodge a foreign body. This may push the foreign body farther into the airway. It also may cause trauma and bleeding	

- Continue CPR for 5 cycles or about 2 minutes.* If you are alone, leave the child and activate the emergency response system. Continue CPR until more skilled health care professionals arrive

*Providing effective ventilation by using a bag-mask device on a child with an FBAO may be difficult. Consider using the 2-person bag-mask ventilation technique.

Effectiveness and safety of suction-based airway clearance devices have not been established in infants and children. There is insufficient evidence to make a recommendation for infants and children.

Critical Concepts

Infant Chest Thrusts

The heel-of-1-hand technique for chest thrusts is now recommended for infants with severe FBAO because current CPR literature suggests that it generates greater compression depth than the previously recommended 2-finger technique. While the heel-of-1-hand technique for chest thrusts resembles chest compressions that are used as part of CPR, there is no focus on the other components of high-quality CPR chest compressions (eg, rate, recoil).

Choking Relief for Severe FBAO in an Infant

To relieve choking in an infant, follow the steps as outlined in the Infant FBAO Algorithm (Figure 27).

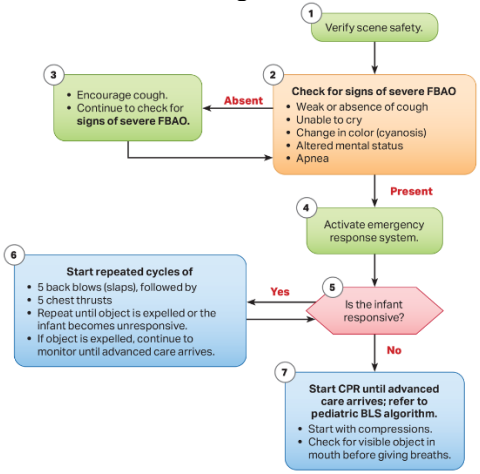


Figure 27. Infant FBAO Algorithm.

Algorithm legend: Red hexagon = decision or question; green box = action/steps to consider; orange box = assessment; gray box = assessment/action; blue box = CPR; white box with gray header = notes; and red, bold text = possible answers or decisions.

The steps to relieve choking in an infant are similar to those for children, with a few key differences. First, don't use abdominal thrusts on infants.

If you find an infant who's choking but is still responsive, activate the emergency response system. Kneel or sit with the infant in your lap. Hold the infant facedown with the head slightly lower than the chest, resting on your forearm (Figure 28A). Support the infant's head and jaw with your hand. Avoid compressing the soft tissue of the infant's throat. Rest your forearm on your lap or thigh to support the infant.

Figure 28A. Relief of choking in an infant. A, Back blows.

With the heel of your other hand, deliver 5 forceful back blows between the infant's shoulder blades. Deliver each back blow with sufficient force to attempt to dislodge the object.

After delivering 5 back blows, place your free hand on the infant's back, supporting their head with the palm of your hand. This will cradle the infant between your 2 forearms, with the palm of one hand supporting the face and jaw while the palm of the other hand supports the back of the infant's head.

Now, turn the infant over while carefully supporting the head and neck. Hold the infant faceup, with your forearm resting on your thigh. Keep the infant's head lower than their chest (Figure 28B).

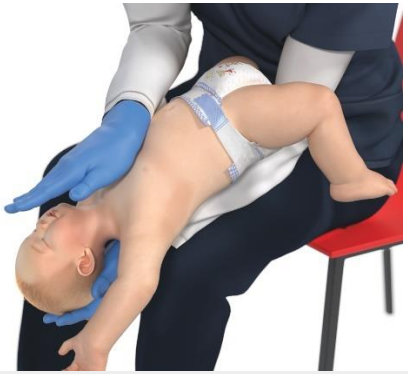


Figure 28B. Chest thrusts.

Provide 5 quick downward chest thrusts with the heel of 1 hand in the middle of the chest, over the lower half of the breastbone.

Deliver chest thrusts at a rate of about 1 per second, each with enough force to dislodge the object.

Repeat the sequence of up to 5 back blows followed by 5 chest thrusts until the object is dislodged or the infant becomes unresponsive.

Never perform a blind finger sweep. Only try to remove the object if you can see it. If you can see the object, carefully try to remove it. If you can't remove it, continue the sequence of 5 back blows and 5 chest thrusts until the object is dislodged or the infant becomes unresponsive. If the object comes out, continue to monitor the infant until advanced care arrives.

If a choking infant becomes unresponsive, shout for help and activate the emergency response system if no one has done so already. Place the infant on a firm, flat surface and begin CPR starting with compressions. Do not perform a pulse check.

To give breaths to an infant who's choking, the mouth-to-mouth-and-nose technique is preferred. If you can't cover the infant's mouth and nose with your mouth, use the mouth-to-mouth technique instead. Start by opening the infant's airway with the head tilt–chin lift maneuver. Each time you open the airway to give breaths, open the infant's mouth and look for the object.

If you see an object that looks easy to remove, remove it with your fingers. Never perform a blind finger sweep because this may cause the object to become lodged farther back in the airway.

Next, place your mouth over the infant's mouth and nose to create an airtight seal. Give 1 breath, blowing for about 1 second. Watch for the chest to rise as you give the breath.

If the chest doesn't rise, repeat the head tilt–chin lift maneuver to reopen the airway, and try to give a breath that makes the chest rise.

If you're alone and have no access to a phone, perform 2 minutes or 5 cycles of CPR before you leave. Or carry the infant with you to activate the emergency response system.

Critical Concepts

No Blind Finger Sweeps

Do not perform a blind finger sweep because it may push the foreign body back into the airway, causing further obstruction or injury.

Choking Relief For Severe FBAO in a Child

To relieve choking in a child, follow the steps as outlined in the Child FBAO Algorithm (Figure 29).

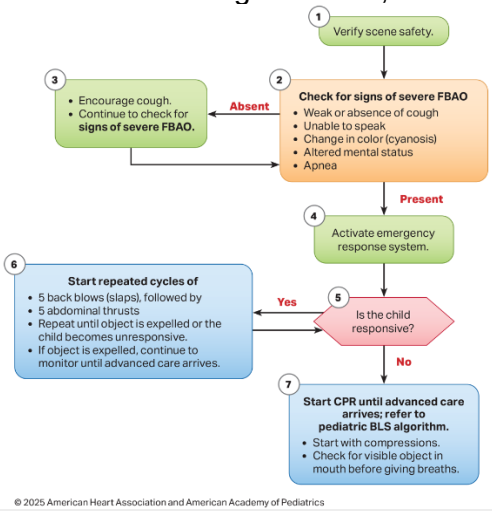


Figure 29. Child FBAO Algorithm.

Algorithm legend: Red hexagon = decision or question; green box = action/steps to consider; orange box = assessment; gray box = assessment/action; blue box = CPR; white box with gray header = notes; and red, bold text = possible answers or decisions.

Giving Effective Relief When There Is an Airway Obstruction

If a child indicates that they're choking and can't talk, you must take action. Older children who are choking may clutch their neck with both hands, making the universal choking sign. For a child who has signs of severe choking, ask them, "Are you

choking?" If they nod "yes" and cannot talk, a severe airway obstruction is present, and you need to take steps immediately to relieve the obstruction.

To relieve choking in a responsive child, perform 5 forceful back blows followed by 5 abdominal thrusts. You should also activate the emergency response system in case the child becomes unresponsive.

Perform 5 back blows, using the heel of your hand and forcefully strike the child's back in between their shoulder blades. If back blows do not relieve choking, perform 5 abdominal thrusts.

Continue alternating 5 back blows followed by 5 abdominal thrusts until the object is dislodged or the child becomes unresponsive.

If the child becomes unresponsive, gently lower them to the ground, shout for help, and activate the emergency response system if no one has already done so. Begin CPR with chest compressions. Do not check for a pulse.

Perform CPR as usual, but with one difference. Each time you open the airway to give breaths, open the child's mouth and look for the object. If you see an object that looks easy to remove, remove it with your fingers. Don't perform a blind finger sweep because this may cause the object to become lodged farther back in the airway. If you don't see an object, continue CPR.

If the choking child is already unresponsive when you arrive, you probably will not know if an FBAO exists. In this situation, you should activate the emergency response system and start high-quality CPR.

When a choking child loses consciousness, the muscles in the throat may relax. This could convert a complete or severe airway obstruction to a partial obstruction. In addition, chest compressions may create at least as much force as abdominal thrusts, so they may help expel the object. Giving 30 compressions and then removing any object that's visible in the mouth may allow you to eventually give effective breaths.

Abdominal Thrusts With the Child Standing or Sitting

Follow these steps to perform abdominal thrusts on a responsive child who is standing or sitting:

1. Stand or kneel behind the child and wrap your arms around the child's waist (Figure 30). Make a fist with one hand.
2. Place the thumb side of your fist against the child's abdomen, in the midline, slightly above the navel and well below the sternum.
3. Grasp your fist with your other hand and press your fist into the child's abdomen with a quick, forceful upward thrust.
4. Give each new thrust with a separate, distinct movement to relieve the obstruction.

5. Continue alternating 5 back blows followed by 5 abdominal thrusts until the object is dislodged or the child becomes unresponsive.

Figure 30. Abdominal thrusts with the child standing.

Actions After Choking Relief

You will know you successfully removed an airway obstruction in an unresponsive person if you saw and removed a foreign body from the person's mouth and the person starts to breathe. However, you don't always have to remove the foreign body to successfully relieve the obstruction. If you can feel air movement and see the chest rise when you give breaths, the airway is no longer obstructed.

After you relieve choking in a person who is unresponsive, proceed as you would with any unresponsive person. Check again for responsiveness, check for breathing and a pulse, confirm that someone has activated the emergency response system, and provide high-quality CPR or breaths as needed.

Encourage a *responsive* person to seek immediate medical attention. A health care professional should evaluate the person for potential complications from abdominal thrusts.

Airway Adjuncts

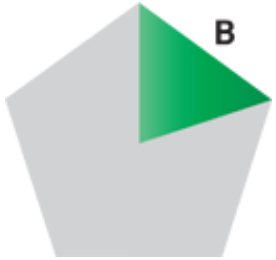
Use airway adjuncts (ie, oral or nasopharyngeal airways) to keep the tongue from falling back and obstructing the airway, but don't rely on an adjunct alone to maintain an open airway. You may still need to use a head tilt–chin lift.

Advanced Interventions to Maintain Airway

Openness/patency may include one or more of the following:

- Removing a foreign body; this intervention may require direct laryngoscopy (ie, visualizing the larynx with a laryngoscope)
- Applying continous positive airway pressure
- ET intubation or placing a supraglottic airway (SGA)
- Cricothyrotomy (a needle puncture or surgical opening through the skin and cricothyroid membrane and into the trachea below the vocal cords)

Breathing



Assessing breathing includes evaluating

- Respiratory rate and pattern
- Respiratory effort (work of breathing)
- Chest expansion and air movement
- Lung and airway sounds
- O_2 saturation by pulse oximetry

Oxygenation and Ventilation

Breathing serves the function of oxygenation and ventilation. A child can be ventilating but not oxygenating and vice versa. If the child is not oxygenating, placing oxygen will improve their oxygen saturation. If the child is not ventilating, improve ventilation by bag-mask device. For example, if the pulse ox is 95% but the rate is 6/min, the child is going to need to be bagged in a very short period of time.

Normal Respiratory Rate

Normal spontaneous breathing is accomplished with minimal work, resulting in quiet breathing with unlabored inspiration and passive expiration. The normal respiratory rate is inversely related to age (Table 13): rapid in the neonate and decreasing as the child gets older.

Table 13.Normal Respiratory Rates by Age*	
Age	**Rate (breaths/min)**
Infant	30-53
Toddler	22-37
Preschooler	20-28
School-age child	18-25
Adolescent	12-20

*Consider the patient's normal range. The child's respiratory rate is expected to increase in the presence of fever or stress

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Evaluate respiratory rate before your hands-on assessment because anxiety and agitation commonly alter the baseline rate. If the child has any condition that causes increased metabolic demand (eg, excitement, anxiety, exercise, pain, fever), it may be appropriate for the respiratory rate to be higher than normal.

Determine the respiratory rate by counting the number of times the chest rises in 30 seconds and multiplying by 2. Normal sleeping infants may have irregular (periodic) breathing with pauses lasting up to 10 or even 15 seconds. If you count the number of times the chest rises for less than 30 seconds, you may estimate the respiratory rate inaccurately. Count the respiratory rate several times as you assess and reassess the child to detect changes. Alternatively, the respiratory rate may be displayed continuously on a monitor.

A decreasing respiratory rate from a rapid to a more "normal" rate may indicate overall improvement if it is associated with an improved level of consciousness and reduced signs of dyspnea and work of breathing. A decreasing or irregular respiratory rate in a child with a deteriorating level of consciousness, however, often indicates a worsening of the child's clinical condition.

Critical Concepts

Very Slow or Very Fast Respiratory Rate Is a Warning Sign

A consistent respiratory rate of less than 10 or more than 60 breaths per minute in a child of any age is always abnormal and warrants further assessment for the presence of a potentially serious condition.

Abnormal Respiratory Rate and Pattern

Abnormal respirations include

- Fast respiratory rate (tachypnea)
- Irregular respiratory pattern
- Slow respiratory rate (bradypnea)
- Apnea

All abnormal respiratory rates have their own set of signs and symptoms and underlying causes. Each type and pattern of respiratory effort can potentially indicate a specific diagnosis. See Table 14 for a summation of abnormal respiratory rates and what may be associated with each.

Table 14.Types of Abnormal Respirations

Type	Signs and symptoms	Underlying causes
Fast respirations (tachypnea)	• Increased respiratory effort—first sign of respiratory distress • Increased rate without increased effort (quiet tachypnea)	• Stress • High fever • Dehydration • Pain • Anemia • Cyanotic congenital heart disease • Metabolic acidosis • Sepsis (serious infection)
Irregular	• Deep gasping breath followed by periods of apnea (no breathing/gasping) • Rapid rate followed by very shallow breathing or apnea	• Neurological
Slow (bradypnea)	• Decreased respiratory rate • Deteriorating level of consciousness	• Respiratory muscle fatigue • Severe hypoxia • Severe shock • Hypothermia • Central nervous system injury or problems that affect the respiratory control center

		• Medications that depress the respiratory control center • Muscle diseases that cause muscle weakness • Medications that depress the respiratory drive
Apnea	• Low oxygen saturation despite high flow supplemental oxygen • Inadequate respiratory rate or no rate • Absent respiratory effort • Absent distal air movement • No air movement (silent chest) • Cyanosis	• Upper airway obstruction • Lower airway obstruction • Lung tissue disease • Disordered control of breathing

Critical Concepts

Bradypnea or Irregular Respiratory Rate Often Signal Impending Arrest

Bradypnea or an irregular respiratory rate in an acutely ill infant or child is an ominous clinical sign and often signals impending arrest.

Increased Respiratory Effort

Increased respiratory effort results from conditions that increase resistance to airflow (eg, asthma, bronchiolitis) or that cause the lungs to be stiffer and difficult to inflate (eg, pneumonia, pulmonary edema, pleural effusion). Nonpulmonary conditions that result in severe metabolic acidosis (eg, shock, diabetic ketoacidosis [DKA], salicylate ingestion, inborn errors of metabolism) can also cause increased respiratory rate and effort. Signs of increased respiratory effort reflect the child's attempt to improve oxygenation, ventilation, or both, so use these signs to assess the severity of the condition and determine the urgency of intervention. Signs of increased respiratory effort include

- Nasal flaring
- Retractions
- Head bobbing
- Grunting
- Seesaw
- Gasping

Nasal Flaring

Nasal flaring is dilation of the nostrils with each inhalation to maximize airflow. It is most observed in infants and younger children and is a sign of respiratory distress.

Retractions

Retractions are inward movements of the chest wall or tissues or sternum during inspiration.

Chest retractions signify that the child is trying to move air into the lungs by using the chest muscles, but air movement is impaired by increased airway resistance or stiff lungs.

Retractions may occur in several areas of the chest. The severity of the retractions generally corresponds with the severity of the child's breathing difficulty.

Table 15 describes the location of retractions commonly associated with each level of breathing difficulty.

Table 15 Location of Retractions Commonly Associated With Each Level of Breathing Difficulty

Breathing difficulty	Location of retraction	Description
Mild to moderate	Subcostal	Retraction of the abdomen, just below the rib cage
Mild to moderate	Substernal	Retraction of the abdomen at the bottom of the sternum
Mild to moderate	Intercostal	Retraction between the ribs
Severe (may include the same retractions as seen with mild to moderate breathing difficulty)	Supraclavicular	Retraction in the tissues just above the collarbone
Severe	Suprasternal	Retraction in the chest, just above the sternum
Severe	Sternal	Retraction of the sternum toward the spine

Retractions accompanied by stridor or an inspiratory snoring sound suggest upper airway obstruction, while retractions accompanied by expiratory wheezing suggest marked lower airway obstruction (asthma or bronchiolitis), causing obstruction

during both inspiration and expiration. Retractions accompanied by grunting or labored respirations suggest lung tissue disease. Severe retraction also may be accompanied by head bobbing or seesaw respirations.

Head Bobbing or Seesaw Respirations

Head bobbing and seesaw respirations often indicate that the child has an increased risk for deterioration.

- Head bobbing is caused by using neck muscles to assist breathing. The child lifts the chin and extends the neck during inspiration and allows the chin to fall forward during expiration. It is most frequently seen in infants and can be a sign of respiratory failure.
- Seesaw respirations are present when the chest retracts and the abdomen expands during inspiration, and then the chest expands and the abdomen moves inward during expiration. In most children with neuromuscular disease, seesaw breathing is caused by weakness of the abdominal and chest wall muscles and by strong contraction of the diaphragm that dominates the weaker abdominal and chest wall muscles. Seesaw respirations usually indicate upper airway obstruction, but they also may be observed in severe lower airway obstruction, lung tissue disease, and states of disordered control of breathing. Seesaw respirations are characteristic of infants and children with neuromuscular weakness. This inefficient form of ventilation can quickly lead to fatigue.

Inadequate Respiratory Effort

When evaluating respiratory effort, look for signs that respiratory effort is inadequate and be prepared to support airway, oxygenation, and ventilation. These signs include

- Apnea
- Weak cry or cough
- Bradypnea
- Agonal gasps

Apnea

Apnea is when breathing stops, typically defined as longer than 20 seconds. Apnea may be further classified as central or obstructive, depending on whether inspiratory muscle activity is present. Central apnea indicates that the child is making no respiratory effort because of an abnormality or suppression of the brain or spinal cord, whereas obstructive apnea occurs when the passage of air is impeded. Either can result in hypoxemia, hypercapnia, or both. Furthermore, in mixed apnea, periods of obstructive apnea and periods of central apnea occur.

Agonal gasps commonly happen in adults after sudden cardiac arrest and may be confused with normal breathing. They also appear late in the deterioration of a very sick child. Agonal gasps will not produce effective oxygenation and ventilation.

Chest Expansion and Air Movement

Evaluate magnitude of chest wall expansion and air movement to assess adequacy of the child's tidal volume, the volume of air inspired with each breath. Normal tidal volume is approximately 6 to 8 mL/kg of ideal body weight throughout life. It is difficult to measure unless a child is mechanically ventilated, so your clinical assessment is very important.

Auscultation With a Stethoscope

Use a stethoscope to auscultate the following points during a respiratory physical exam:

- Anterior (on either side of the sternum)
- Posterior
- Lateral (under the axillae)

Chest Wall Expansion

Chest expansion (chest rise) during inspiration should be symmetrical. It may be subtle during spontaneous quiet breathing, especially when clothing covers the chest, but chest expansion should be readily visible when the chest is uncovered. In normal infants, the abdomen may move more than the chest. Decreased or asymmetrical chest expansion may result from inadequate effort, airway obstruction, atelectasis, pneumothorax, hemothorax, pleural effusion, mucous plug, or foreign-body aspiration.

Air Movement

Auscultation for air movement is critical. Listen for the intensity of breath sounds and quality of air movement in the following areas:

- Anterior, located at mid-chest (just to the left and right of the sternum)
- Lateral, located under the armpits (the best location for evaluating air movement into the lower parts of the lungs)
- Posterior, located on both sides of the back

Critical Concepts

Auscultating Breath Sounds in Infants and Children

Because the chest is small and the chest wall is thin in infants and children, breath sounds readily transmit from one side of the chest to the other and from the upper airway. To evaluate distal air entry, listen below both axillae. Because these areas are farther from the larger conducting airways, upper airway sounds are less likely to be transmitted.

Listen to the loudness of the air movement:

- Typical inspiratory sounds can be heard distally as soft, quiet noises occurring simultaneously with observed inspiratory effort.
- Normal expiratory breath sounds are often short and quieter; sometimes you may not even hear them.

Decreased chest excursion or decreased air movement during auscultation often accompanies poor respiratory effort. In the child with apparently normal or increased respiratory effort, diminished distal air entry suggests airflow obstruction or lung tissue disease. If the child's work of breathing and coughing suggest lower airway obstruction, but you cannot hear wheezes, the amount and rate of airflow may be insufficient to cause wheezing.

Distal air entry may be difficult to hear in the obese child, so it may be difficult to identify significant airway abnormalities in this population.

Minute Ventilation

Minute ventilation is the volume of air that moves into or out of the lungs each minute. It is the product of the number of breaths per minute (respiratory rate) and the volume of each breath (tidal volume).

Minute Ventilation = Respiratory Rate × Tidal Volume

Low minute ventilation (hypoventilation) may result from

- Slow respiratory rate
- Small tidal volume (ie, shallow breathing, high airway resistance, stiff lungs)
- Extremely rapid respiratory rate (resulting in very small tidal volumes)

Lung and Airway Sounds

During the primary assessment, listen for lung and airway sounds. Abnormal sounds include stridor, snoring, grunting, gurgling, wheezing, crackles, and change in cry/phonation/cough (including barking cough). See Summary: Recognizing Respiratory Problems in Part 5.

Stridor

Stridor is a coarse, usually higher-pitched breathing sound typically heard on inspiration, though you may hear it during both inspiration and expiration. Stridor is a sign of upper airway (extra-thoracic) obstruction and may indicate that the obstruction is critical and requires immediate intervention.

Stridor has many causes including

- A foreign body in the airway

- Infection (eg, croup)
- Congenital airway abnormalities (eg, laryngomalacia)
- Acquired airway abnormalities (eg, tumor, cyst)
- Upper airway edema (eg, allergic reaction, swelling after a medical procedure)

Snoring

Although snoring may be common in children during sleep, it also can be a sign of airway obstruction. Soft tissue swelling or decreased level of consciousness may cause airway obstruction and snoring.

Grunting

Grunting is typically a short, low-pitched sound heard during expiration that can sometimes be misinterpreted as a soft cry. It occurs as the child exhales against a partially closed glottis. Although grunting may accompany the response to pain or fever, infants and young children often grunt to help keep the small airways and alveolar sacs in the lungs open to optimize oxygenation and ventilation.

Grunting often signifies lung tissue disease resulting from small airway collapse, alveolar collapse, or both. It may indicate progression of respiratory distress to respiratory failure. Pulmonary conditions that cause grunting include

- Pneumonia
- Pulmonary contusion
- Acute respiratory distress syndrome (ARDS)
- Pulmonary edema (may be caused by cardiac conditions such congestive heart failure [CHF])
- Pain

Identify and treat the cause as quickly as possible, and be prepared to quickly intervene if the child’s condition worsens.

Gurgling

Gurgling is a bubbling sound heard during inspiration or expiration that results from upper airway obstruction due to airway secretions, vomit, or blood.

Wheezing

Wheezing is a high-pitched or low-pitched whistling or sighing sound heard most often during expiration and less frequently during inspiration. This sound typically indicates lower (intrathoracic) airway obstruction, especially of the smaller airways.

Common causes of wheezing are bronchiolitis and asthma. Isolated inspiratory wheezing suggests a foreign body or other cause of partial obstruction of the trachea or upper airway.

Crackles

Crackles, also known as *rales*, are sharp, crackling inspiratory sounds. Dry crackles sound similar to the sound made when you rub several hairs together close to your ear. Dry crackles are more often heard with atelectasis (small airway collapse) and interstitial lung disease. Moist crackles indicate accumulation of alveolar fluid and are typically associated with lung tissue disease (eg, pneumonia, pulmonary edema) or interstitial lung disease. Note that you may not hear crackles despite the presence of pulmonary edema.

Change in Cry/Phonation

If an infant's cry becomes very soft with only short sounds during expiration (ie, so the cry sounds more like the soft "mewing" of a cat) or an older child begins to talk in short phrases or single words instead of sentences, this may indicate severe respiratory distress and shortness of breath.

Identifying Respiratory Distress vs Respiratory Failure

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

- Respiratory distress
- Respiratory failure

Respiratory Distress

Respiratory distress is a clinical state characterized by increased respiratory rate and effort, spanning a spectrum from mild tachypnea with increased effort to severe distress with impending respiratory failure. A description of respiratory distress severity typically includes a description of the respiratory rate and effort, quality of breath sounds, and mental status. These indicators may vary in severity. Note that signs of severe respiratory distress can indicate respiratory failure.

- Mild respiratory distress
    - –Mild tachypnea
    - –Mild increase in respiratory effort (eg, nasal flaring, retractions)
    - –Abnormal airway sounds (eg, stridor, wheezing)
- Severe respiratory distress
    - –Marked tachypnea
    - –Marked increase in respiratory effort (eg, nasal flaring, retractions)
    - –Paradoxical thoracoabdominal breathing (eg, seesaw breathing)
    - –Accessory muscle use (eg, head bobbing)
    - –Abnormal airway sounds (eg, grunting)
    - –Decreased level of consciousness (eg, less responsive)
- Impending respiratory arrest
    - –Bradypnea, apnea, or respiratory pauses
    - –Low oxygen saturation (hypoxemia) despite high-flow supplemental oxygen
    - –Inadequate respiratory effort (eg, shallow respirations)
    - –Decreased level of consciousness (eg, unresponsive)
    - –Bradycardia

Respiratory distress is apparent when a child tries to maintain adequate gas exchange despite airway obstruction, reduced lung compliance, or lung tissue disease. As the child tires or as respiratory function or effort or both deteriorate, adequate gas exchange cannot be maintained. When this happens, clinical signs of respiratory failure develop.

Respiratory Failure

Respiratory failure is a clinical state of inadequate oxygenation, ventilation, or both. It is recognized typically by abnormal appearance (particularly an altered level of consciousness, which may be characterized by agitation or a depressed level of consciousness), poor color, and reduced responsiveness. Although respiratory failure often results from respiratory distress progression, it may occur with little or no respiratory effort. At times, recognizing respiratory failure requires laboratory data (eg, blood gas) to confirm the diagnosis. In other patients, the clinical examination suffices to identify respiratory failure.

Suspect probable respiratory failure if some of the signs in the following list are present.

- Signs of severe respiratory distress
    - –Marked tachypnea
    - –Increased or decreased respiratory effort

- –Poor distal air movement
- –Tachycardia
- –Low oxygen saturation (hypoxemia) despite high-flow oxygen administration
- –Cyanosis

- Signs of probable respiratory failure
  - –Very rapid or inadequate respiratory rate or possible apnea
  - –Significant, inadequate, or absent respiratory effort
  - –Absent distal air movement
  - –Lack of airflow (silent chest)
  - –Extreme tachycardia; bradycardia often indicates life-threatening deterioration
  - –Low oxygen saturation (hypoxemia) despite high-flow supplemental oxygen
  - –Decreased level of consciousness
  - –Cyanosis

Respiratory failure can result from upper or lower airway obstruction, lung tissue disease, and disordered control of breathing (eg, apnea or shallow, slow respirations). When respiratory effort is inadequate, respiratory failure can occur without typical signs of respiratory distress. Respiratory failure is a clinical state that requires intervention to prevent deterioration to cardiac arrest.

Strict criteria for respiratory failure is difficult to define because the baseline respiratory function of an infant or child may be abnormal. For example, an infant with cyanotic congenital heart disease and a baseline arterial O_2 saturation (SaO_2) of 75% is not in respiratory failure on the basis of low O_2 saturation. But the same degree of hypoxemia would be one sign of respiratory failure in a child with normal baseline cardiopulmonary physiology.

The difference between respiratory distress and failure may be determined by abnormal components of the PAT. If only the work of breathing is abnormal, the child is likely in respiratory distress. If both the appearance and work of breathing are abnormal, the child is likely in respiratory failure (Table 16).

Table 16.Progression of Respiratory Distress to Respiratory Failure

Progression of respiratory distress to respiratory failure*		
	Respiratory distress	**Respiratory failure**
Airway	Open and maintainable	Not maintainable
Breathing	Tachypnea	Bradypnea to apnea
	Work of breathing (nasal flaring/retractions)	Increased effort progresses to decreased effort and then to apnea
	Good air movement	Poor to absent air movement
Circulation	Tachycardia	Bradycardia
	Pallor	Cyanosis
Disability	Anxiety, agitation	Lethargy to unresponsiveness
Exposure	Variable temperature	Variable temperature

*Respiratory failure requires **immediate** intervention.

Pulse Oximetry

When caring for a seriously ill or injured child, use pulse oximetry to monitor oxygen saturation and trends in oxygen saturation.

Oxygen Saturation by Pulse Oximetry

The O_2 saturation is the percent of total hemoglobin that is fully saturated with oxygen (ie, the oxyhemoglobin saturation). This oxyhemoglobin saturation does not indicate the amount of O_2 delivered to the tissues. O_2 delivery is the product of arterial O_2 content (oxygen bound to hemoglobin plus dissolved O_2) and cardiac output. O_2 saturation does not provide information about ventilation effectiveness (CO_2 elimination).

An O_2 saturation (SpO_2) of 94% or more while a child is breathing room air usually indicates that oxygenation is adequate; conversely, an SpO_2 less than 94% when the child is breathing room air indicates hypoxemia. Consider administering supplemental O_2 if the O_2 saturation is below this value in a critically ill or injured child. An SpO_2 of less than 90% in a child receiving 100% O_2 is an indication for additional intervention.

When to Use Pulse Oximetry

Use pulse oximetry to monitor oxygen saturation and trends in oxygen saturation. The pulse oximeter measures the percent of hemoglobin that is fully saturated or bound with oxygen. Because pulse oximetry can indicate low O_2 saturation (hypoxemia) before it causes cyanosis or bradycardia, health care professionals can use pulse oximetry to monitor trends in O_2 saturation in response to treatment. Continuously monitor pulse oximetry for a seriously ill or injured child during stabilization, transportation, and post–cardiac arrest care.

The pulse oximeter consists of a probe that is linked to a monitor and attached to the child's finger, toe, or earlobe. The pulse oximeter has 2 parts that must be placed opposite each other, so they are located on either side of a pulsatile tissue bed. It must detect a consistent pulsatile signal. Lights of different wave lengths originate from one side of the probe, and the light is captured on the other side of the tissue by the other side of the probe. A processor in the oximeter calculates the percent of each light that has been absorbed by the tissues. Hemoglobin that is fully saturated with oxygen absorbs light differently than hemoglobin that is not fully saturated with oxygen. By determining the absorption of the different wavelengths of light, the pulse oximeter can estimate the percent of hemoglobin saturated with blood. The unit displays the calculated percentage of saturated hemoglobin. Most units make an audible sound for each heartbeat at and display the heart rate, while some models display the quality of the pulse signal as a waveform or with bar.

Correct Use of Oximetry Equipment

Correct probe positioning, typically on a finger or toe, is critical for accurate oxygen saturation values. The probe is to be placed across an area of pulsatile blood flow. Falsely low values can occur when the probe is not positioned correctly. When this occurs, repositioning the probe can result in immediate improvement in detecting oxygen saturation by the device. Bright light in the room can interfere with accurate light absorption detection by the pulse oximeter.

In addition to applying the probe to a finger or toe, the following locations can be used to solve placement problems: If infant probes are unavailable, use an adult probe around the hand or foot of an infant. If blood flow is significantly reduced and no signal is detected in the extremities, assess and support systemic perfusion and apply an infant probe to the earlobe.

Confirm the Validity of Oximeter Data

Different brands of pulse oximeters vary in how quickly they reflect hypoxemia development and in their accuracy when the child has decreased blood flow; all pulse oximetry devices are inaccurate unless the pulse rate displayed by the oximeter is consistent with the heart rate displayed by the cardiac monitor. Although published studies have resulted in mixed conclusions, pulse oximetry may have different accuracy/validity across races and ethnicities due to varying levels of skin pigmentation. Further reviews of the available evidence and recommendations by the AHA will be forthcoming.

Critical Concepts

Validity of Oximeter Data

- Confirm validity of oximeter data by evaluating the child's appearance. Compare the heart rate displayed by the pulse oximeter with the heart rate displayed by the bedside cardiorespiratory monitor or counted during physical examination.
- Immediately evaluate the child if the oximeter fails to detect a signal, displays an inaccurate pulse rate, indicates a weak signal, or indicates a fall in oxygen saturation.
- If the oximeter fails to detect a signal or indicates a fall in oxygen saturation, immediately evaluate the child. Do not assume that the pulse oximeter is malfunctioning.

Caution in Interpreting Pulse Oximetry Readings

Be careful to interpret pulse oximetry readings in conjunction with your clinical assessment, including considering signs such as respiratory rate, respiratory effort, and level of consciousness. A child may be in respiratory distress yet maintain normal O_2 saturation by increasing respiratory rate and effort, especially if receiving supplemental O_2.

If the pulse oximeter displays a different heart rate or inaccurate waveform tracing from the one determined by ECG monitoring, the displayed O_2 saturation is not reliable. When the pulse oximeter does not detect a consistent pulse or there is an irregular or poor waveform, the child may have poor distal perfusion and the pulse oximeter may not be accurate—check the child and intervene as needed. The pulse oximeter may not be accurate if the child develops severe shock and won't be accurate during cardiac arrest.

Pulse oximetry indicates only O_2 saturation and not O_2 delivery. For example, if the child is profoundly anemic (hemoglobin is very low), the saturation may be 100%, but O_2 content in the blood and O_2 delivery may be low.

The pulse oximeter does not accurately recognize methemoglobin or carboxyhemoglobin (hemoglobin bound to carbon monoxide). If carboxyhemoglobin (from carbon monoxide poisoning) is present, the pulse oximeter will reflect a falsely high O_2 saturation because it counts the carboxyhemoglobin as fully saturated hemoglobin. If methemoglobin concentrations are

above 5%, the pulse oximeter will read approximately 85% regardless of the degree of methemoglobinemia. If you suspect either of these conditions, obtain a blood gas and send it for laboratory analysis of O_2 saturation measurement by co-oximeter.

Errors in pulse oximetry can occur if

- The probe is not placed across an area of pulsatile blood flow
- The pulses are very weak or the extremity is poorly perfused
- The extremity is cold
- The probe is placed so the side emitting light is not directly across from the side that captures the light
- Bright light in the room interferes with accurate light absorption detection
- Carbon monoxide poisoning is present
- The skin has varying levels of pigmentation

Accuracy of Values in Clinical Settings

Pulse oximetry can accurately estimate oxygen saturation but does not provide evidence of oxygen delivery and does not directly evaluate the effectiveness of ventilation (carbon dioxide concentration).

Pulse oximetry may be inaccurate in some settings (Table 17).

Table 17.Settings Where a Pulse Oximeter May Be Inaccurate

Setting	Cause/solution
Cardiac arrest	Cause: Absence of blood flow Solution: None; many devices will not be useful during cardiac arrest
Shock or hypothermia	Cause: Decreased blood flow Solution: Improve blood flow (treat the shock); you may be able to find an alternative site (particularly one that is closer to the heart) where the device can detect pulsatile blood flow
Motion, shivering, or bright overhead lighting	Cause: False signals or inaccurate oxygen saturation values Solution: Move the sensor unit closer to the heart; lightly cover the device in position (ie, if it is placed on a finger, lightly cover the finger to reduce ambient light)
Problem with the skin probe interface	Cause: Low or absent pulse signals Solution: Try an alternate site or alternate skin probe

Misaligning sensor with light source	Cause: Low or absent pulse signals Solution: Reposition the device so that the light source is located directly across the tissue bed from the sensor
Cardiac arrhythmias with low cardiac output	Cause: Arrhythmias that interfere with detection of a pulse and calculation of pulse rate Solution: Assess pulse rate manually; obtain expert consultation for arrhythmia management

Circulation

Circulation is assessed by evaluating

- Heart rate and rhythm
- Pulses (both central and peripheral)
- Capillary refill time
- Skin color and temperature
- Blood pressure
- Urine output (end organ perfusion)

For more information on assessing the level of consciousness, refer to the Disability section later in this Part.

Heart Rate and Rhythm

To determine heart rate, check the pulse rate, listen to the heart, or view a monitor display of the ECG or pulse oximeter waveform. The heart rate should be appropriate for the child's age, level of activity, and clinical condition (Table 18). Note the wide range of normal heart rates. For example, a child who is sleeping or is athletic may have a heart rate lower than the normal range for age, and a child who is crying or upset may have a higher heart rate than the normal range for age.

Table 18.Normal Heart Rates*		
Age	**Awake rate (rate/min)**	**Sleeping rate (rate/min)**
Neonate	100-205	90-160
Infant	100-180	90-160
Toddler	98-140	80-120
Preschooler	80-120	65-100
School-age child	75-118	58-90
Adolescent	60-100	50-90

*Always consider the patient's normal range and clinical condition. Heart rate will normally increase with fever or stress.

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Consider the following when evaluating the heart rate and rhythm in any seriously ill or injured child:

- The child's typical heart rate and baseline rhythm
- The child's level of activity and clinical condition
- The child's cardiac function and perfusion

Children with congenital heart disease may have conduction abnormalities. Consider the child's baseline ECG when interpreting heart rate and rhythm. Children with poor cardiac function are more likely to be symptomatic from cardiac rhythm disturbances (arrhythmias) than are children with normal cardiac function.

The heart rhythm is typically regular with only small fluctuations in rate. When checking the heart rate, assess for abnormalities in the monitored ECG. Arrhythmias result from abnormalities in, or insults to, the cardiac conduction system or heart tissue. Arrhythmias also can result from shock or hypoxia. In the ALS setting, an arrhythmia in a child can be broadly classified according to the observed heart rate or effect on perfusion, with slow heart rate classified as bradycardia, fast heart rate classified as tachycardia, and absent heart rate classified as asystole (cardiac arrest).

Bradycardia is a heart rate slower than normal for a child's age and clinical condition. Slight bradycardia may be normal in athletic children, but a very slow rate in a child with other symptoms is worrisome and may indicate that cardiac arrest is imminent. Hypoxia is the most common cause of bradycardia in children. If a child with bradycardia has signs of cardiopulmonary compromise, immediately support oxygenation and ventilation with a bag and mask and administer supplemental O_2. Start chest compressions if the heart rate remains less than 60/min with signs of cardiopulmonary compromise despite adequate oxygenation and ventilation. If the child with bradycardia is alert and has no signs of cardiopulmonary compromise, consider other causes of a slow heart rate, such as heart block or drug overdose.

Tachycardia is a resting heart rate that is faster than the normal range for a child's age and clinical condition. ST, a common, nonspecific response to a variety of conditions, is often appropriate when the child is anxious, crying, febrile, or seriously ill or injured. To determine if the tachycardia is a ST or represents a cardiac rhythm disturbance, evaluate the child's history, clinical condition, and ECG. Any tachycardia associated with signs of circulatory compromise, including hypotension, altered mental status, or signs of shock, requires immediate evaluation and intervention. It is important to track heart rate trends to determine early signs of life-threatening illness such as shock.

Critical Concepts

Tachycardia Can Be a Sign of a Serious Condition

A heart rate that is greater than 180/min in an infant or toddler and greater than 160/min in a child older than 2 years warrants further assessment and may be a serious condition.

For more information, refer to Part 9: Recognizing Arrhythmias.

Relationship of Breathing to Heart Rhythm

In healthy children, the heart rate may fluctuate with the respiratory cycle, increasing with inspiration and slowing down with expiration. This condition is called *sinus arrhythmia*.

Determine if the child has an irregular rhythm that is not related to breathing. An irregular rhythm may indicate an underlying rhythm disturbance, such as premature ventricular or atrial contractions or an atrioventricular (AV) block.

Pulses

Evaluating central and peripheral pulses is critical to the assessment of systemic perfusion in an ill or injured child. Central pulses are ordinarily stronger than peripheral pulses because they are present in vessels of larger size that are located closer to the heart. An exaggerated difference in quality between central and peripheral pulses occurs when peripheral vasoconstriction is associated with shock. Central pulses include femoral, brachial (in infants), carotid (in older children), and

axillary. Peripheral pulses include radial, dorsalis pedis, and posterior tibial. These pulses are easily palpable in healthy infants and children (unless the child is obese or the ambient temperature is cold).

Beat-to-beat fluctuation in pulse volume may occur in children with arrhythmias (eg, premature atrial or ventricular contractions). Fluctuation in pulse volume with the respiratory cycle (pulsus paradoxus) can occur in children with severe asthma or pericardial tamponade. In an intubated child receiving positive-pressure ventilatory support, a reduction in pulse volume with each positive-pressure breath may indicate hypovolemia.

Critical Concepts

Weakening of Pulses as Perfusion Decreases

Weak central pulses are worrisome and indicate the need for rapid intervention to prevent cardiac arrest. When cardiac output decreases in shock, systemic perfusion decreases incrementally. The decrease in perfusion starts in the extremities with a decrease in intensity of pulses and then an absence of peripheral pulses. As cardiac output and perfusion decrease further, central pulses eventually weaken.

Capillary Refill Time

Capillary refill time is the time it takes for blood to return to tissue blanched by pressure. It increases as skin perfusion decreases. Note that normal capillary refill time is 2 seconds or less, and a prolonged capillary refill time may indicate low cardiac output.

Evaluate capillary refill in a neutral thermal environment (ie, room temperature) by lifting the extremity slightly above the level of the heart, pressing on the skin or nailbed, and rapidly releasing the pressure. Note how many seconds it takes for the area to return to its baseline color.

Common causes of sluggish, delayed, or prolonged capillary refill (a refill time of greater than 2 seconds) are dehydration, shock, and hypothermia. Note that shock can be present despite a normal (or even brisk) capillary refill time. Children may have warm skin and extremities with very rapid (less than 2 seconds) capillary refill (often called *flash capillary refill*). These children are in shock and need immediate treatment (refer to Part 7: Recognizing Shock).

Skin Color and Temperature

Monitor changes in skin color, temperature, and capillary refill time to assess a child's perfusion and response to therapy. Normal skin color and temperature should be consistent over the trunk and extremities. The mucous membranes, nail beds, palms of the hands, and soles of the feet should be pink. When perfusion deteriorates and O_2 delivery to the tissues becomes

inadequate, the hands and feet are typically affected first and become cool, pale, dusky, or mottled. If perfusion becomes worse, the skin over the trunk and extremities may undergo similar changes.

Consider the temperature of the child's surroundings (ie, ambient temperature) when evaluating skin color and temperature. If the ambient temperature is cool, peripheral vasoconstriction may produce mottling or pallor with cool skin and delayed capillary refill, particularly in the extremities. These changes develop despite normal cardiovascular function. A cold environment can cause vasoconstriction and a discrepancy between peripheral and central pulses. However, if cardiac output remains adequate, central pulses should remain strong.

To assess skin temperature, use the back of your hand because it is more sensitive to temperature changes than the palm, which has thicker skin. Slide the back of your hand up the extremity to determine a line where the skin changes from cool to warm and monitor this line of demarcation between warm and cool skin over time to determine the child's response to therapy. The line should move distally as the child improves.

Carefully monitor for the following skin findings (Table 19), which may indicate inadequate O_2 delivery to the tissues:

- Pallor
- Mottling
- Cyanosis

Table 19.Skin Findings, Location, and Causes

Skin color	Location	Causes
Pallor (paleness; lack of normal color)	Skin or mucous membranes	• Decreased blood supply to the skin (cold; stress; shock, especially hypovolemic and cardiogenic) • Decreased number of red blood cells (anemia) • Decreased skin pigmentation • Poor perfusion
Mottling (irregular or patchy discoloration)	Skin	• Normal distribution of skin melanin • Intense vasoconstriction from irregular supply of oxygenated blood to the skin due to hypoxemia, hypovolemia, or shock
Cyanosis (blue discoloration)	Skin or mucous membranes	

Acrocyanosis (bluish discoloration)	Hands and feet and around the mouth (ie, the skin around the lips)	Normal in the newly born
Peripheral cyanosis (bluish discoloration)	Hands and feet (beyond newborn period)	• Shock • CHF • Peripheral vascular disease • Conditions causing venous stasis
Central cyanosis (blue color)	Lips and other mucous membranes	• Ventilation/perfusion imbalance (eg, asthma, bronchiolitis, acute respiratory distress syndromes) • Intracardiac shunt (eg, cyanotic congenital heart disease) • Low ambient O_2 tension (eg, high altitude) • Alveolar hypoventilation (eg, traumatic brain injury, drug overdose) • Diffusion defect (eg, pulmonary fibrosis)

You must interpret pallor within the context of other signs and symptoms because it is not necessarily abnormal and can result from lack of exposure to sunlight or inherited paleness.

Pallor is often difficult to detect in a child with dark skin and in those with thick skin and variation in the vascularity of subcutaneous tissue. However, family members often can tell you if a child's color is abnormal. Central pallor (ie, pale color of the lips and mucous membranes) strongly suggests anemia or poor perfusion. Pallor of the mucous membranes (the lips, lining of the mouth, tongue, lining of the eyes) and pale palms and soles are more likely to be clinically significant.

- Areas that are mottled may appear as an uneven combination of pink, bluish gray, or pale skin tones.
- Blood saturated with O_2 is bright red, whereas unoxygenated blood is dark bluish-red. The location of cyanosis is important.
- Acrocyanosis is a normal neonatal finding in the first 24 to 48 hours after delivery.
- Peripheral cyanosis can be caused by diminished O_2 delivery to the tissues.
- Central cyanosis is a blue color of the lips and other mucous membranes.

Critical Concepts

Variability in Appearance of Central Cyanosis

Cyanosis is not apparent until at least 5 g/dL of hemoglobin are desaturated (not bound to O_2). The O_2 saturation at which a child will appear cyanotic depends on the child's hemoglobin concentration. For example, in a child with a hemoglobin concentration of 16 g/dL, cyanosis will appear at an O_2 saturation of approximately 70% (ie, 30% of the hemoglobin, or 5 g/dL,

is desaturated). If the hemoglobin concentration is low (eg, 8 g/dL), a very low O_2 saturation (eg, less than 40%) is required to produce cyanosis. Thus, cyanosis may be apparent with a milder degree of hypoxemia in a child with polycythemia (increased amount of hemoglobin and red blood cells, eg, in cyanotic heart disease) but may not be apparent despite significant hypoxemia if the child is anemic.

Cyanosis may be more obvious in the mucous membranes and nail beds than in the skin, particularly if the skin is dark. It also can be seen on the soles of the feet, tip of the nose, and earlobes. Children with different hemoglobin levels will be cyanotic at different levels of O_2 saturation; cyanosis is more readily detected at higher O_2 saturations if the hemoglobin level is high. The development of central cyanosis typically indicates the need for emergency intervention, such as O_2 administration and ventilatory support.

Blood Pressure

Accurate blood pressure measurement requires a cuff that extends at least 50% to 75% of the length of the upper arm (from the axilla to the antecubital fossa) and a cuff bladder that covers about 40% of the mid–upper arm circumference. Use caution with automatic blood pressure cuffs as they may not accurately measure blood pressure.

Normal Blood Pressures

Table 20 summarizes the normal range of systolic and diastolic blood pressures according to age from 1 standard deviation below to 1 standard deviation above the mean in the first year of life and from the 50th to 95th percentile (assuming the 50th percentile for height) for children 1 year or older. As with heart rates, there is a wide range of values within the normal range, and normal blood pressures may fall outside the ranges listed here.

Table 20.Normal Blood Pressures

Age	Systolic pressure (mm Hg)*	Diastolic pressure (mm Hg)*	Mean arterial pressure (mm Hg)†
Birth (12 hours, <1000 g)	39-59	16-36	28-42†
Birth (12 hours, 3 kg)	60-76	31-45	48-57
Neonate (96 hours)	67-84	35-53	45-60
Infant (1-12 months)	72-104	37-56	50-62

Toddler (1-2 years)	86-106	42-63	49-62
Preschooler (3-5 years)	89-112	46-72	58-69
School-age child (6-9 years)	97-115	57-76	66-72
Preadolescent (10-12 years)	102-120	61-80	71-79
Adolescent (12-15 years)	110-131	64-83	73-84

*Systolic and diastolic blood pressure ranges assume 50th percentile for height for children 1 year and older.

†Mean arterial pressures (diastolic pressure + [difference between systolic and diastolic pressure/3]) for 1 year and older, assuming 50th percentile for height.

‡Approximately equal to postconception age in weeks (may add 5 mm Hg).

Modified from Hazinski MF. Children are different. In: Hazinski MF, ed. *Nursing Care of the Critically Ill Child*. 3rd ed. Mosby; 2013:1-18, copyright Elsevier. Data from Gemelli M, Manganaro R, Mamì C, De Luca F. Longitudinal study of blood pressure during the 1st year of life. *Eur J Pediatr*. 1990;149(5):318-320; Versmold HT, Kitterman JA, Phibbs RH, Gregory GA, Tooley WH. Aortic blood pressure during the first 12 hours of life in infants with birth weight 610 to 4,220 grams. Pediatrics. 1981;67(5):607-613; Haque IU, Zaritsky AL. Analysis of the evidence for the lower limit of systolic and mean arterial pressure in children. *Pediatr Crit Care Med*. 2007;8(2):138-144; and National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. *The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents. National Heart, Lung, and Blood Institute; 2005. NIH publication 05-5267.*

Hypotension

Hypotension is defined by the thresholds of systolic blood pressure shown in Table 21.

Table 21.Definition of Hypotension by Systolic Blood Pressure and Age

Age	Systolic blood pressure (mm Hg)
Term neonates (0-28 days)	<60
Infants (1-12 months)	<70

Children 1-10 years	<70+ (age in years × 2) (this estimates systolic blood pressure that is less than the fifth blood pressure percentile for age)*
Children >10 years	<90

*This fifth percentile is a systolic blood pressure that is lower than all but 5% of normal children (ie, it will be hypotensive for 95% of most children).

Note that these blood pressure thresholds approximate just above the fifth percentile systolic blood pressures for age, so they will overlap with normal blood pressure values for 5% of healthy children. An observed decrease in systolic blood pressure of 10 mm Hg from baseline should prompt serial evaluations for additional signs of shock. In addition, remember that these threshold values were established in normal, resting children. Children with injury and stress typically have increased blood pressure, and a blood pressure in the low-normal range may be abnormal in a seriously ill child.

When hypotension develops in a child with shock, physiologic compensatory mechanisms (eg, tachycardia, vasoconstriction) have failed. A child can lose up to 20% of circulating blood volume without hypotension. Hypotension is a late sign of blood loss. When blood loss exceeds 30%, the blood pressure will be low and the child hypotensive. Hypotension in septic shock can occur from loss of intravascular volume and inappropriate vasodilation or severe vasoconstriction and inadequate cardiac output/cardiac index.

The development of bradycardia in a child with hypotension and poor perfusion is an ominous sign. To prevent cardiac arrest, manage airway and breathing and support adequate intravascular volume, cardiac function, and perfusion.

Adequate circulation is also reflected by urine output and level of consciousness. Urine output can indirectly indicate kidney perfusion. Normal urine output requires adequate blood flow and hydration. Normal values for urine output are age dependent:

- Infants and young children: 1.5 to 2 mL/kg per hour normal urine output
- Older children and adolescents: 0.5 to 1 mL/kg per hour normal urine output; children with shock may have decreased urine output

Accurately measuring urine output in all critically ill or injured children requires an indwelling catheter. Initial urine output is not a reliable indicator of the child’s clinical condition because much of the urine may have been produced before the onset of symptoms. An increase in urine output is a good indicator of positive responses to therapy.

Disability

Disability is assessed by evaluating

- AVPU (Alert, Responsive to Voice, Responsive to Pain, Unresponsive) Pediatric Response Scale
- Glasgow Coma Scale (GCS)
- Pupil response to light
- Blood glucose test

Perform the disability assessment to quickly evaluate neurologic function, using one of several tools to evaluate responsiveness and level of consciousness. Perform this evaluation at the end of the primary assessment and repeat it during the secondary assessment to monitor for changes in the child's neurologic status.

Clinical factors that reflect brain perfusion can provide indirect evidence of circulatory function in the ill or injured pediatric patient.

AVPU Pediatric Response Scale

To rapidly evaluate cerebral cortex function, use the AVPU Pediatric Response Scale. This scale is a system for rating a child's level of consciousness, an indicator of cerebral cortex function. The scale consists of 4 ratings:

- Alert: The child is awake, active, and appropriately responsive to caregivers and external stimuli. Assess "appropriate response" in terms of the anticipated response based on the child's age and/or developmental level and the setting or situation.
- Voice: The child responds only to voice (eg, calling the child's name, speaking loudly).
- Pain: The child responds only to a painful stimulus, such as a sternal rub or pinching the trapezius.
- Unresponsive: The child does not respond to any stimulus.

Decreased level of consciousness in children can be caused by

- Hypoglycemia
- Poor cerebral perfusion
- Severe shock
- Traumatic brain injury
- Seizure activity
- Encephalitis, meningitis
- Medications
- Hypoxemia
- Hypercarbia

A common mnemonic that can be used is AEIOU-TIPS

- A: alcohol
- E: epilepsy
- I: insulin
- O: opiate
- U: uremia/uremic poisoning
- T: trauma
- I: infection
- P: poisons/psychogenic
- S: stoke/subdural (in the head) hematoma/epidural hematoma

Altered mental status refers to the range of mental states from agitation to coma. For a pediatric patient with altered mental status, consider hypoglycemia and evaluate blood glucose as soon as possible.

If an ill or injured child has decreased responsiveness, immediately assess oxygenation, ventilation, perfusion, and blood glucose.

Signs of inadequate O_2 delivery to the brain correlate with the severity and duration of cerebral hypoxia.

Sudden and severe cerebral hypoxia may cause the following neurologic signs:

- Decreased level of consciousness
- Loss of muscular tone
- Generalized seizures
- Pupil dilation

You may observe other neurologic signs when cerebral hypoxia develops gradually. Because these signs can be subtle, repeat measurements over time to best detect them:

- Decreased level of consciousness with or without confusion
- Irritability
- Lethargy
- Agitation alternating with lethargy

GCS Overview

The GCS is the most widely used method of evaluating a child's level of consciousness and neurologic status. The best eye opening (E), verbal (V), and motor (M) responses are individually scored (Table 22) and then added together to produce the GCS score.

Table 22.Glasgow Coma Scale

Eye opening	Best motor response	Best verbal response
4 Spontaneous	6 Obeys commands	5 Oriented
3 To speech	5 Localizes pain	4 Confused
2 To pain	4 Withdraws from pain	3 Inappropriate words
1 No response	3 Abnormal flexion	2 Incomprehensible words/sounds
	2 Abnormal extension	1 No response
	1 No response	

Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. *Lancet*. 1974;2(7872):81-84.

For example, a person who has spontaneous eye opening (E = 4), is fully oriented (V = 5), and is able to follow commands (M = 6) is assigned a GCS score of 15, the highest possible score. A person with no eye opening (E = 1), no verbal response (V = 1), and no motor response (M = 1) to a painful stimulus is assigned a GCS score of 3, the lowest possible score.

Severity of head injury is categorized into 3 levels based on GCS score:

- Mild head injury: GCS score 13 to 15

- Moderate head injury: GCS score 9 to 12
- Severe head injury: GCS score 3 to 8

GCS Scoring

The GCS has been modified for preverbal or nonverbal children (Table 23). While scores for eye opening are essentially the same as in the standard GCS, the best motor and verbal response scores were adapted to accommodate age-appropriate responses.

Table 23.Pediatric GCS*

Score	Child	Infant
Eye opening		
4	Spontaneously	Spontaneously
3	To verbal command	To shout, speech
2	To pain	To pain
1	No response	No response
Best motor response		
6	Obeys commands	Spontaneous movements
5	Localizes pain	Withdraws to touch
4	Flexion-appropriate withdraw to pain	Flexion-appropriate withdraw to pain
3	Flexion-abnormal (decorticate rigidity)	Flexion-abnormal (decorticate rigidity)
2	Extension (decerebrate rigidity)	Extension (decerebrate rigidity)
1	No response	No response
Best verbal response		
5	Oriented and converses	Smiles, coos, and babbles

4	Disoriented, confused	Cries but is consolable
3	Inappropriate words	Persistent, inappropriate crying and/or screaming
2	Incomprehensible sounds	Moans, grunts to pain
1	No response	No response
Total = 3 to 15		

*Score is the sum of the individual scores from eye opening, best motor response, and best verbal response, using age-specific criteria. GCS score of 13 to 15 indicates mild head injury; GCS score of 9 to 12 indicates moderate head injury; and GCS score of 8 indicates severe head injury.

Modified from James HE, Trauner DA. The Glasgow Coma Score and Modified Coma Score for Infants. In: James HE, Anas NG, Perkin RM, eds. *Brain Insults in Infants and Children: Pathophysiology and Management*. Grune & Stratton Inc; 1985:179-182, copyright Elsevier.

Important: When using the GCS or its pediatric modification, record the individual components of the score. If the patient is intubated, unconscious, or preverbal, the most important part of this scale is motor response. Health care professionals should carefully evaluate this component.

The best disability scale for an individual child may be site specific. For example, the AVPU scale may be appropriate in the prehospital setting, whereas the GCS (particularly the motor component) or pediatric GCS may be better in the ED and hospital. The differences between the AVPU scale and GCS or pediatric GCS do not appear significant when associated with neurologic outcome. Each component of the AVPU scale generally correlates with the GCS scores shown in Table 24.

Table 24.AVPU Scale and GCS Equivalents

Response	GCS score
Alert	15
Verbal	13
Painful stimulation	8
Unresponsive to noxious stimulation	6

Pupil Response to Light

Health care professionals also should assess and record the pupillary size and response to light for each eye in any patient with altered level of consciousness. Pupil response to light is a useful indicator of brainstem function. Normally, pupils constrict in response to light and dilate in a dark environment, so if the pupils don't constrict in response to direct light (eg, flashlight directed at the eyes), suspect a brainstem injury. The pupils are typically equal in size, although slight variations are normal. Irregularities in pupil size or response to light may occur due to ocular trauma or other conditions, such as increased intracranial pressure (ICP). Refer to Table 25 for examples of abnormal pupil responses and their possible causes.

Table 25.Abnormal Pupil Responses and Possible Causes

Abnormal pupil response	Possible cause
Pinpoint pupils	• Narcotic ingestion (eg, opioid)
Dilated pupils	• Predominant sympathetic autonomic activity • Sympathomimetic ingestion (eg, cocaine) • Anticholinergic ingestion (eg, local or systemic atropine) • Increased ICP
Unilaterally dilated pupils	• Inadvertent topical absorption of a breathing treatment (eg, ipratropium) • Dilating eye drops
Unilaterally dilated pupils with altered mental status	• Ipsilateral (same side) uncal herniation (lateral herniation of the temporal lobe, caused by increased ICP)

During the disability assessment, assess and record the following for each eye:

- Size of pupils (in millimeters)
- Equality of pupil size
- Constriction of pupils to light (ie, the magnitude and rapidity of the response to light)

The acronym PERRL (Pupils Equal, Round, Reactive to Light) describes the normal pupil responses to light.

Blood Glucose Test

Hypoglycemia refers to blood glucose <45 mg/dL or less in the newly born and 60 mg/dL or less in a child. It may result in brain injury if not recognized and effectively treated. Base treatment decisions on patient symptoms, and potentially include oral

glucose. Monitor the blood glucose concentration of any seriously ill infant or child. A low blood glucose concentration may cause altered level of consciousness or even brain injury if it is not quickly identified and adequately treated. Measure the blood glucose concentration with a point-of-care glucose test.

For more information about recognizing and treating hypoglycemia, refer to the Glucose section in Part 8.

Exposure

Exposure, or undressing the seriously ill or injured child to perform a focused physical examination, is the final component of the primary assessment. Remove clothing one area at a time to carefully observe the child's face and head, trunk (front and back), extremities, and skin. Maintain cervical spine precautions when turning any child with a suspected neck or spine injury. If necessary, use blankets to keep the child comfortable and warm and, if available, heating lamps to prevent cold stress or hypothermia. Assess core temperature, noting any difference in warmth between trunk and extremities. Identify the presence of fever, which may indicate infection and early need for antibiotics (eg, sepsis).

During this part of the examination, look for evidence of trauma, such as bleeding, burns, or unusual markings that suggest nonaccidental trauma. Such signs include bruises in different stages of healing, injuries that don't correlate with the child's history, and delay from time of injury until the child receives medical attention.

Look for the presence and progression of petechiae and purpura (nonblanching purple discolorations in the skin caused by bleeding from capillaries and small vessels). Petechiae appear as tiny red dots and suggest a low platelet count, whereas purpura appears as larger areas. Both petechiae and purpura may signify septic shock. Also look for other rashes that may suggest shock (eg, hives in anaphylactic shock).

Look for signs of injury to the extremities, including deformities or bruising. Palpate the extremities and note the child's response. If tender, suspect injury; if necessary, immobilize the extremity.

Secondary Assessment

Secondary assessment consists of a focused history and detailed physical examination with ongoing reassessment of physiologic status and response to treatment.

Focused History

Obtain a focused history to gather information about the patient and the incident, particularly information that might help explain impaired respiratory or cardiovascular function. You can use the SAMPLE mnemonic, a systematic method for gathering information on a sick child, as a memory aid for obtaining a focused history. Obtain an accurate timeline for all signs, symptoms, and events leading up to the current presentation and previous visits to a health care professional for the child with the same symptoms. Gather the following information for each category:

- Signs and symptoms at onset of illness, such as
    - –Breathing difficulty (eg, cough, rapid breathing, increased respiratory effort, breathlessness, abnormal breathing pattern, chest pain on deep inhalation), wheezing
    - –Tachypnea
    - –Tachycardia
    - –Diaphoresis
    - –Decreased level of consciousness, fatigue
    - –Agitation, anxiety
    - –Fever, headache
    - –Decreased oral intake
    - –Diarrhea, vomiting, decreased urine output, number of wet diapers
    - –Abdominal pain
    - –Bleeding
    - –Duration of symptoms
    - Allergies
    - –Medications, foods, latex, etc
    - –Associated reactions
- Medications
    - –Patient medications (eg, over-the-counter, vitamins, inhalers, herbal supplements), medications that can be found in the child’s environment
    - –Last dose and time of recent medications

- Past medical history
    - –Health history (eg, premature birth, previous illnesses, hospitalizations)
    - –Significant underlying medical problems (eg, asthma, chronic lung disease, congenital heart disease, arrhythmia, congenital airway abnormality, seizures, head injury, brain tumor, diabetes, hydrocephalus, neuromuscular disease)
    - –Past surgeries
    - –Immunization status
- Last meal/last menstrual period
    - –Time and nature of last intake of liquid or food (including breast or bottle feeding in infants)
    - –Elapsed time between last meal and presentation of current illness can affect treatment and management of the condition (eg, possible anesthesia, possible intubation)
    - –As appropriate, date of last menstrual period
- Events
    - –Events leading to current illness or injury (eg, onset sudden or gradual, type of injury)
    - –Hazards at scene
    - –Treatment during interval from onset of disease or injury until evaluation, estimated time of onset (if out-of-hospital onset)

Focused Physical Examination

Next, perform a focused physical examination based on the severity of the child's illness or injury. Carefully assess the primary area of concern of the illness or injury (ie, respiratory assessment with respiratory distress) and perform a brief head-to-toe evaluation. Some examples of areas to assess for certain illnesses include

- The nose/mouth (signs of obstruction, nasal congestion, stridor, mucosal edema), chest/lungs, heart (tachycardia, gallop, or murmur), and level of consciousness (somnolence secondary to hypercarbia, anxiety secondary to hypoxia) for respiratory distress
- The heart (gallop or murmur), lungs (crackles, difficulty breathing, intolerance of supine position), abdomen (evidence of hepatomegaly consistent with right heart failure), and extremities (peripheral edema) for suspected heart failure or arrhythmias
- The abdomen and back for trauma

Ongoing Reassessment

Ongoing reassessment of all patients is essential to evaluate the response to treatment and to track the progression of identified physiologic and anatomic problems. Apply this reassessment in real time as needed based on the child's clinical condition through all phases of assessment. Do not limit it to the last part of the assessment sequence. You may also identify new problems on reassessment. Data from the reassessment will guide ongoing treatment. The elements of ongoing reassessment involve continuous application of the initial, primary, and secondary assessments to determine the effectiveness of interventions.

Diagnostic Assessments

Diagnostic assessments help detect and identify the presence and severity of respiratory and circulatory problems. You may use some of these assessments (such as rapid bedside glucose or point-of-care laboratory testing) early in your evaluation.

The clinical situation will determine which diagnostics will be needed for the child. Diagnostics may include

- Blood gas (arterial blood gas [ABG], venous blood gas [VBG], capillary blood gas)
- Hemoglobin concentration
- Central venous O_2 saturation
- Arterial lactate
- Central venous pressure monitoring
- Invasive arterial pressure monitoring
- Chest x-ray
- ECG
- Echocardiogram (if within scope of practice)
- Point-of-care ultrasound sonography (if within scope of practice)
- Peak expiratory flow rate

Arterial Blood Gas

An ABG analysis measures the partial pressure of arterial O_2 (PaO_2) and CO_2 ($PaCO_2$) dissolved in the blood plasma (ie, the liquid component of blood). An additional tool to assess the adequacy of arterial oxygenation is the pulse oximeter, a device that estimates hemoglobin saturation with O_2. It may also be calculated from the PaO_2 and pH (by using an oxyhemoglobin dissociation curve) or directly measured by using a co-oximeter. Obtain co-oximeter measurement if there is uncertainty about the calculated O_2 saturation and to rule out the presence of carbon monoxide intoxication or methemoglobinemia.

Critical Concepts

Normal PaO_2 Does Not Confirm Adequate Blood O_2 Content

PaO_2 reflects only the O_2 dissolved in the arterial blood plasma. If the child's hemoglobin is only 3 g/dL, the PaO_2 may be normal or high, but O_2 delivery to the tissues will be inadequate. Because most O_2 is carried by hemoglobin in the red blood cells rather than the plasma, a hemoglobin of 3 g/dL is inadequate to carry sufficient O_2. In this case, the pulse oximeter may reflect 100% saturation despite inadequate O_2 content and delivery.

Identify respiratory failure on the basis of inadequate oxygenation (hypoxemia) or inadequate ventilation (hypercarbia). Use an ABG analysis to confirm your clinical impression and evaluate the child's response to therapy. An ABG analysis, however, is not required to initiate therapy or to make the diagnosis of respiratory failure. The following list will help you interpret an ABG:

- Hypoxemia: low PaO_2
- Hypercarbia: High $PaCO_2$
- Acidosis: pH less than 7.35
- Alkalosis: pH greater than 7.45

Critical Concepts

ABG and Treatment Decisions in Seriously Ill or Injured Children

Don't wait for an ABG so you can start treatment or determine therapy. Limitations of ABG analysis in pediatric critical care include the following:

- An ABG analysis may not be available (eg, during transport); do not delay therapy initiation.
- A single ABG analysis provides only information at the time the sample was obtained. It does not provide information about trends in the child's condition. Monitoring clinical response to therapy by using serial ABG analyses is often more valuable than any single ABG analysis.

To interpret ABG results, consider the child's clinical appearance and condition. For example, an infant with bronchopulmonary dysplasia (a form of chronic lung disease) may have chronic hypercarbia. Diagnosing acute respiratory failure in this infant requires clinical examination and evaluating the arterial pH. The infant will compensate for chronic hypercarbia by renal retention of bicarbonate, and, as a result, the child's arterial pH is likely to be normal or nearly normal at baseline. Deterioration will be apparent if the child's respiratory status (ie, hypercarbia) is significantly worse than the baseline status and acidosis develops.

Hyperoxia is increased arterial partial pressure detected by ABG sample. This has been associated with worse outcomes, such as after ROSC, in the newly born and in patients with some forms of cyanotic heart disease. Because of the uncertainty of

measuring hyperoxia without ABGs, administering oxygen to achieve a displayed pulse oximetry of 100% is not recommended in these conditions.

The arterial pH and bicarbonate (HCO_3) concentrations obtained with ABG analysis may be useful in diagnosing acid-base imbalances. Note that ABG values do not reliably reflect O_2, CO_2, or acid-base status in the tissues. However, it is useful to monitor these values over time as an index of improving or worsening tissue oxygenation, as reflected by an increasing base deficit (accumulation of acid in the blood).

Venous Blood Gas

A venous blood pH, measured by VBG analysis, typically correlates well with the pH on ABG analysis. A VBG analysis is not as useful for monitoring ABG status (PaO_2 and $PaCO_2$) in acutely ill children. If the child is well perfused, the venous PCO_2 is usually within 4 to 6 mm Hg of the arterial PCO_2. If the child is poorly perfused, however, the gradient between arterial and venous PCO_2 increases. In general, venous PO_2 is not useful in assessing arterial oxygenation.

When interpreting a VBG, also consider the source of the venous specimen. A peripheral specimen that is free flowing from a well-perfused extremity may give results similar to the ABG, but if a tourniquet is used and the specimen is from a poorly perfused extremity, it often shows a much higher PCO_2 and lower pH than an arterial specimen. For this reason, a central venous specimen, if available, is preferred to a peripheral venous specimen. A VBG may be used if an arterial sample is unavailable. There is generally adequate correlation with ABG samples to make venous pH useful in detecting acid-base imbalance.

Capillary Blood Gas

If arterial collection is not practical, CBG analysis can be used. Arterialization of the capillary bed yields pH and $PaCO_2$ comparable to arterial blood. A CBG analysis is not as useful for estimating arterial oxygenation (PaO_2).

Hemoglobin Concentration

Hemoglobin concentration determines the O_2-carrying capacity of the blood. O_2 content is the total amount of O_2 bound to hemoglobin plus the unbound (dissolved) O_2 in arterial blood and is determined chiefly by the hemoglobin concentration (in grams per deciliter) and its saturation with O_2 (SaO_2). The amount of O_2 dissolved in the blood is determined by the PaO_2, also referred to as the *arterial oxygen tension*, and represents a very small part of total O_2 content at normal hemoglobin concentrations. For more information on determining the arterial oxygen content, refer to Impaired Oxygenation and Ventilation in Respiratory Problems in Part 5.

Central Venous Oxygen Saturation

VBGs may indicate changes in the balance between O_2 delivery to the tissues and tissue O_2 consumption. Trends in central venous O_2 saturation (SvO_2) may be used as a surrogate for monitoring trends in O_2 delivery (ie, the product of cardiac output and arterial O_2 content). Such trending assumes that O_2 consumption remains stable (an assumption that is not always correct, so remain diligent).

Normal SvO_2 is about 70% to 75%, assuming arterial O_2 saturation is 100%. If the arterial O_2 saturation is not normal, the SvO_2 should be about 25% to 30% below the arterial O_2 saturation. For example, if the child has cyanotic heart disease and the arterial O_2 saturation is 80%, the SvO_2 should be about 55%.

When O_2 delivery to the tissues is low, the tissues consume proportionately more O_2, so the difference between arterial O_2 saturation and SvO_2 is more substantial when shock is present. For more information about SvO_2, refer to Part 8: Managing Shock.

Arterial Lactate

The arterial concentration of lactate reflects the balance between lactate production and metabolism or breakdown. In a seriously ill or injured child, the arterial lactate can rise as a result of increased production of lactate (associated with tissue hypoxia and resultant anaerobic metabolism). Arterial lactate is easy to measure, and it's a good prognostic indicator that you can follow sequentially to assess the child's response to therapy. However, arterial lactate can be inaccurate if not drawn with a free-flowing sample or if testing is delayed. In the ED setting, the initial lactate may be venous.

An elevated lactate concentration does not always represent tissue ischemia, especially without accompanying metabolic acidosis. For example, lactate concentration also can be elevated in conditions associated with increased glucose production, such as stress hyperglycemia. In general, it is more helpful to monitor trends in lactate concentration over time than any single measurement. If treatment of shock is effective, the lactate concentration should decrease, but the trend in concentration over time is more predictive than the initial concentration. Lack of response to therapy (ie, the lactate concentration does not decrease) is more predictive of poor outcome than the initial elevated lactate concentration. Monitor central venous lactate concentration if arterial blood samples are not readily available.

Invasive Arterial Pressure Monitoring

Invasive arterial pressure monitoring enables you to continually evaluate and view the systolic and diastolic blood pressures. The arterial waveform pattern may provide information about SVR and compromised cardiac output (eg, pulsus paradoxus, an exaggerated decrease in the systolic blood pressure during inspiration). This type of monitoring requires an arterial catheter,

monitoring (noncompliant) tubing, a transducer, and a monitoring system. Accurate measurements require you to appropriately zero, level, and calibrate the transducer.

Near-Infrared Spectroscopy

Near-infrared spectroscopy is a noninvasive optical technique to monitor tissue oxygenation in the brain and other tissues. The monitors measure the concentrations of oxyhemoglobin and desaturated hemoglobin to determine regional oxygen saturation, often placed to follow cerebral oxygen saturation, which you can follow to assess central venous oxygenation trends. With the cerebral oximeter electrode on the forehead below the hairline, its 2 light-emitting diodes and receivers detect light from shallow and deep tissue, and a computer analyzes the data to provide continuous measurements. Near-infrared spectroscopy monitoring has wide interpatient variability but is used to trend cerebral and other tissue oxygenation in the critical care settings.

Chest X-Ray

A chest x-ray is useful in respiratory illness to aid in diagnosing the following conditions:

- Airway obstruction (upper airway or lower airway)
- Lung tissue disease
- Air leak (pneumothorax, pneumomediastinum)
- Pleural disease (pleural effusion/pneumothorax)

A chest x-ray will show the depth of ET tube placement but should not be used determine tracheal vs esophageal placement.

Use the chest x-ray and clinical assessment to evaluate circulatory abnormalities. The chest x-ray may be helpful to assess heart size and presence or absence of CHF (pulmonary edema).

- A small heart is often present with reduced cardiac preload or severe lung hyperinflation.
- A large heart may be associated with normal or increased cardiac preload, pericardial effusion, CHF, or when the patient is unable to take a deep breath (eg, with severe abdominal distension).
- An x-ray taken from the front (AP view) or during exhalation, the heart will appear larger as compared with the heart size when taken from a posterior-anterior view.

Electrocardiogram

Obtain a 12-lead ECG to assess for cardiac arrhythmias. For more information, refer to Part 10: Managing Arrhythmias.

Echocardiogram

Echocardiography is a valuable noninvasive tool for determining

- Cardiac chamber size
- Ventricular wall thickness
- Ventricular wall motion (contractility)
- Valve configuration and motion
- Pericardial space
- Estimated ventricular pressures
- Interventricular septal position
- Congenital anomalies

It can be useful in diagnosing and evaluating cardiac disease. Technical expertise in performing and interpreting the echocardiogram is essential.

Part 5

Recognizing Respiratory Problems

Respiratory distress is defined as abnormal respiratory rate or effort, encompassing a spectrum of signs from tachypnea with retractions to agonal gasps. It includes

- Increased work of breathing
- Inadequate respiratory effort (eg, shallow respirations) or rate (bradypnea)
- Irregular breathing

PALS providers must identify respiratory conditions that are treatable with simple measures, such as by clearing airway secretions or administering O_2. Yet it may be even more important to identify those respiratory conditions that are subtly but rapidly progressing toward respiratory failure. These conditions require timely interventions with more advanced airway techniques (eg, assisted bag-mask ventilation).

In infants and children, respiratory distress can quickly progress to respiratory failure and finally to cardiac arrest. Good outcome (ie, neurologically intact survival to hospital discharge) is more likely after respiratory arrest than after cardiac arrest. Once the child with a respiratory problem develops cardiac arrest, the outcome is often poor, so you can greatly improve outcome by identifying and managing respiratory distress and failure before the child deteriorates to cardiac arrest.

Learning Objective

After completing this Part, you should be able to differentiate between respiratory distress and failure.

You need to understand the concepts in this Part to be able to quickly identify signs of respiratory distress and respiratory failure as well as be able to recognize respiratory problems by type so that you can choose appropriate interventions.

Fundamental Issues Associated With Respiratory Problems

Children with respiratory problems have impaired oxygenation, ventilation, or both. This section discusses

- Impaired oxygenation and ventilation
- Physiology of respiratory disease

Impaired Oxygenation and Ventilation in Respiratory Problems

Physiology of the Respiratory System

The main function of the respiratory system is gas exchange, in which air is taken into the lungs with inspiration. From there, O_2 diffuses from the alveoli into the blood, where some O_2 dissolves in plasma. Most O_2 that enters the blood is bound to hemoglobin. The percent of hemoglobin that is bound to O_2 is called *oxygen saturation*. When blood passes through the lungs, CO_2 diffuses from the blood into the alveoli and is exhaled. Acute respiratory problems can result from alterations in any part of this system, from the airway to the alveoli (lung parenchyma). Central nervous system disease, such as seizures or head trauma, can impair respiration control, leading to decreased respiratory rate or an irregular respiratory pattern and/or ineffective respirations. Muscle weakness, either primary (eg, muscular dystrophy) or secondary (eg, fatigue), may also impair oxygenation or ventilation.

Children have a high metabolic rate, so O_2 demand per kilogram of body weight is high. Therefore, hypoxemia and tissue hypoxia can develop more rapidly in a child than in an adult if apnea or inadequate alveolar ventilation occurs.

Respiratory problems can result in

- Hypoxemia
- Hypercarbia
- A combination of both hypoxemia and hypercarbia

Hypoxemia (Low Oxygen Saturation)

Hypoxemia is defined as a decreased arterial oxygen saturation detected by pulse oximetry or directly measuring oxygen saturation in an ABG sample. It is generally defined as arterial oxygen saturation below 94% in a normal child breathing room air. Various conditions may lower the threshold, such as altitude or cyanotic heart disease. Permissive hypoxemia is a pulse oximetry percentage of less than 94%, which may be appropriate in certain circumstances (eg, some cases of congenital heart disease).

It is important to distinguish between *hypoxemia* and *tissue hypoxia*. Hypoxemia is low arterial O_2 saturation (SaO_2 less than 94%), while hypoxia is a pathologic condition in which the body as a whole (*generalized hypoxia*) or a region of the body (*tissue hypoxia*) is deprived of an adequate oxygen supply. Note that hypoxemia does not necessarily lead to tissue hypoxia and that tissue hypoxia may occur when arterial oxygen saturation is normal. For example, when hypoxemia is chronic (eg, unrepaired cyanotic heart disease), compensatory increases in blood flow (ie, increased cardiac output) or hemoglobin concentration (polycythemia) increase the O_2-carrying capacity of the blood and helps maintain arterial O_2 content at near-normal

concentrations despite hemoglobin saturation being low. This can help maintain oxygen delivery and tissue oxygenation even when hypoxemia is present. Conversely, if tissue perfusion is poor or the patient has severe anemia, tissue hypoxia may occur with normal arterial oxygen saturation.

In response to tissue hypoxia, the child may initially compensate by increasing respiratory rate and depth, or hyperventilate. *Hyperventilation* refers to increased alveolar ventilation resulting in a decrease in $PaCO_2$ to less than 35 mm Hg. This may be caused by an increased respiratory rate, increased tidal volume, or combination of both. Initially use capnography to ensure that $PaCO_2$ does not decrease below 30 mm Hg. Confirm with blood gas when able. Tachycardia may also develop in response to hypoxemia to increase cardiac output. As tissue hypoxia worsens, these signs of cardiopulmonary distress become more severe (Table 26).

Table 26.Signs of Tissue Hypoxia

Early signs of tissue hypoxia	Late signs of tissue hypoxia
Fast respiratory rate (tachypnea)	Slow respiratory rate (bradypnea), inadequate respiratory effort, apnea
Increased respiratory effort: nasal flaring, retractions	Increased respiratory effort: head bobbing, seesaw respirations, grunting
Tachycardia	Bradycardia
Pallor, mottling, cyanosis*	Pallor, mottling, cyanosis*
Agitation, anxiety, irritability	Decreased level of consciousness

*Pallor, mottling, and cyanosis can appear as both early and late signs.

Critical Concepts

Arterial O_2 Content

Arterial O_2 content is the total amount of O_2 carried in the blood (in milliliters O_2 per deciliter of blood). It is the sum of the quantity of O_2 bound to hemoglobin plus the O_2 dissolved in arterial blood. O_2 content is determined largely by the hemoglobin concentration (grams per deciliter) and its saturation with O_2 (SaO_2). Use the following equation to calculate arterial O_2 content:

Arterial O_2 content = [1.36 × hemoglobin concentration × SaO_2] + (0.003 × PaO_2)

Under normal conditions, dissolved O_2 (0.003 × PaO_2) is an inconsequential portion of the total arterial O_2 content. An increase in dissolved O_2 can produce a relatively significant increase in arterial O_2 content for a child with severe anemia.

Hypoxemia can be caused by several different mechanisms leading to respiratory distress and failure (Table 27).

Table 27.Mechanisms of Hypoxemia

Factor	Causes	Mechanism	Treatment
Ventilation/perfusion (V/Q) mismatch	• Pneumonia • Pulmonary edema • Atelectasis • Acute respiratory distress syndrome • Asthma • Bronchiolitis • Foreign body	Mismatch of ventilation and perfusion: blood flow through areas of the lung that are inadequately ventilated results in incomplete oxygenation of the blood returning to the left side of the heart. The result is a decreased arterial O_2 saturation and PaO_2 and, to a lesser extent, increased $PaCO_2$	Use PEEP to increase mean airway pressure*; supplemental O_2; ventilatory support
Low atmospheric PO_2	High altitude (decreased barometric pressure)	Decreased PaO_2	Give supplemental O_2
Alveolar hypoventilation	• Central nervous system infection • Traumatic brain injury • Drug overdose • Neuromuscular weakness • Apnea	Increased alveolar CO_2 tension (hypercarbia) displaces alveolar O_2, resulting in decreased alveolar O_2 tension (PaO_2)	Restore normal ventilation; supplemental O_2
Diffusion defect	Interstitial lung disease	Impaired movement of O_2 and CO_2 between the alveoli and blood results in decreased PaO_2 (hypoxemia) and, if severe, an increased $PaCO_2$ (hypercarbia)	Apply noninvasive ventilation (either CPAP or biphasic/bilevel CPAP) with supplemental O_2
Right-to-left shunt	• Cyanotic congenital heart disease	Shunting of unoxygenated blood from the right side of the heart to the left (or from the pulmonary artery into the aorta) results in a low PaO_2. Effects are similar to right-to-left shunt in the lungs	Obtain expert consultation

	• Extracardiac (anatomical) vascular shunt • Same causes listed for V/Q imbalance†		

*Positive end-expiratory pressure should be used carefully in children with asthma; expert consultation is advised.

†With pneumonia, acute respiratory distress syndrome, and other lung tissue diseases, the pathophysiology is often characterized by a mix of hypoxemia mechanisms. The most extreme form of V/Q mismatch would be a segment of lung with blood flow (Q) but no ventilation (V), a situation in which the blood would not become oxygenated. When it returns to the left side of the heart, it mixes with the oxygenated blood, resulting in a lower O_2 saturation. The degree of desaturation depends on the size of the unventilated lung segment.

Hypercarbia (Inadequate Ventilation)

Hypercarbia is an increased CO_2 tension in the arterial blood ($PaCO_2$). When hypercarbia is present, ventilation is inadequate.

CO_2 is a by-product of tissue metabolism. Normally, the lungs eliminate it to maintain acid-base homeostasis because, when ventilation is inadequate, CO_2 elimination is inadequate. The resulting increase in $PaCO_2$ causes the blood to become acidic (respiratory acidosis). Causes of hypercarbia are

- Airway obstruction (upper or lower)
- Lung tissue disease
- Decreased or inadequate respiratory effort (central hypoventilation)

Most children with hypercarbia present with respiratory distress and tachypnea. Children may become tachypneic in an attempt to eliminate excess CO_2. However, the child with hypercarbia may also present with poor respiratory effort, including decreased respiratory rate. In this case, hypercarbia results from inadequate ventilation secondary to impaired respiratory drive. This inadequate ventilation may result from drugs, such as a narcotic overdose. It may also result from a central nervous system disorder with respiratory muscle weakness preventing the development of compensatory tachypnea. Detecting an inadequate respiratory drive requires careful observation and assessment. Consequences of inadequate ventilation become more severe as the $PaCO_2$ increases and respiratory acidosis worsens.

Detecting Hypercarbia

Hypercarbia is more difficult to detect than hypoxemia because it produces no obvious clinical sign, such as cyanosis. Precisely measuring PCO_2 requires a blood sample (arterial, capillary, or venous). Exhaled CO_2 detectors are now available for use in

children with or without advanced airways. The end-tidal CO_2 (or exhaled CO_2 measured at the end of exhalation) measured by capnography may not be identical to the arterial CO_2. However, if the airway is open/patent, there is no increased dead space from air trapping (eg, asthma), and cardiac output is adequate, the end-tidal CO_2 will increase in the presence of hypercarbia. End-tidal CO_2 reflects both the cardiac output produced and ventilation efficacy and may provide feedback on the quality of CPR. A sudden rise in the end-tidal CO_2 may be an early sign of ROSC.

When to Suspect Hypercarbia

Decreased level of consciousness is a critical symptom of both inadequate ventilation and hypoxia. If a child's clinical condition deteriorates from agitation and anxiety to decreased responsiveness despite supplemental O_2, this may indicate that the $PaCO_2$ is rising. Note that even if the pulse oximeter indicates adequate O_2 saturation, ventilation may be impaired and hypercarbia may be present. If a child with respiratory distress has a decreased level of consciousness despite adequate oxygenation, ventilation may be inadequate and hypercarbia and respiratory acidosis may be present.

Signs of inadequate ventilation are nonspecific and include one or more of the following:

- Tachypnea or inadequate respiratory rate for age and clinical condition
- Nasal flaring, retractions, accessory muscle use
- Change in level of consciousness: initial anxiety, agitation, and then decreased level of consciousness

Physiology of Respiratory Disease

One may accomplish normal spontaneous breathing with minimal work. Breathing is quiet with unlabored, smooth inspiration and passive expiration. In children with respiratory disease, "work of breathing" becomes more apparent. Important factors associated with increased work of breathing include

- Increased airway resistance (upper and lower)
- Decreased lung compliance
- Use of accessory muscles of respiration
- Disordered central nervous system control of breathing

Airway Resistance

Airway resistance, or the impedance to airflow within the airways, is primarily increased by reducing the size of the conducting airways (either by airway constriction or inflammation). Turbulent airflow also causes increased airway resistance. Airflow may become turbulent when the flow rate increases, even if the airway size remains unchanged. When airway resistance increases, work of breathing increases in an attempt to maintain airflow.

Larger airways provide lower resistance to airflow than smaller airways. Airway resistance decreases as lung volume increases (inflation) because airway dilation accompanies lung inflation. Conditions such as edema, bronchoconstriction, secretions, mucus, or a mediastinal mass impinging on large or small airways can decrease airway size, thereby increasing airway resistance. Resistance in the upper airway, particularly in the nasal or nasopharyngeal passages, can represent a significant portion of total airway resistance, especially in infants.

Airway Resistance in Laminar Airflow

During normal breathing, because airflow is laminar (ie, quiet, smooth, and orderly) and airway resistance is relatively low, only a small driving pressure (ie, difference in pressure between the atmosphere and the pleural space) is needed to produce adequate airflow. When airflow is laminar (quiet), resistance to airflow is inversely proportional to the fourth power of the airway radius, so even a small reduction in airway diameter results in a significant increase in airway resistance and work of breathing.

Figure 31 shows normal airways on the left and edematous airways (with 1 mm of circumferential edema) on the right. Resistance to flow is inversely proportional to the fourth power of the radius of the airway lumen for laminar flow and to the fifth power of the radius when air flow is turbulent. The net result is a 75% decrease in cross-sectional area and a 16-fold increase in airway resistance in the infant vs a 44% decrease in cross-sectional area and a 3-fold increase in airway resistance in the adult during quiet breathing. Turbulent flow in an infant (eg, crying) increases airway resistance and thus the work of breathing from 16- to 32-fold.

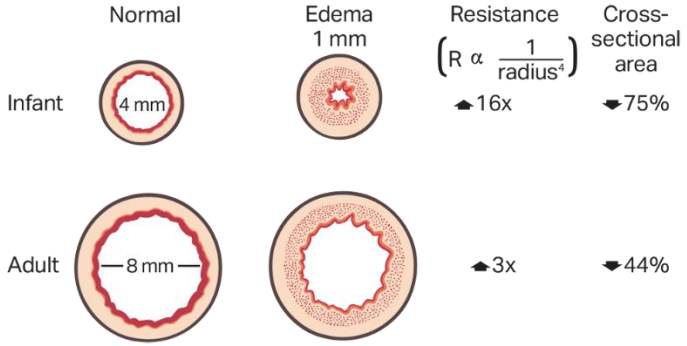


Figure 31. Effects of edema on airway resistance in the infant vs the young adult.

Modified from Coté CJ, Todres ID. The pediatric airway. In: Coté CJ, Ryan JF, Todres ID, Goudsouzian NG, eds. *A Practice of Anesthesia for Infants and Children*. 2nd ed. WB Saunders Co; 1993:55-83, copyright Elsevier.

Airway Resistance in Turbulent Airflow

When airflow is turbulent (ie, irregular), resistance is inversely proportional to the fifth power of the radius of the airway lumen, a 10-fold increase over the resistance to airflow that occurs during reduction in the airway radius when airflow is normal and laminar. In this state, a much larger driving pressure is required to produce the same rate of airflow. A patient who is agitated (causing rapid, turbulent airflow) has a significant increase in airway resistance and thus work of breathing compared to a patient who is calm (allowing for laminar airflow). To prevent turbulent airflow from generating (eg, during crying), try to keep a child with airway obstruction as calm as possible.

Lung Compliance

Compliance refers to the distensibility of the lung, chest wall, or both. *Lung compliance*, specifically, is defined as the change in lung volume produced by a change in driving pressure across the lung. When lung compliance is high, the lungs easily inflate (a large change in volume produced by a slight change in driving pressure). The lungs are stiffer in a child with low lung compliance, so it takes more effort to inflate them. To create a significant pressure gradient to produce air flow into the stiff lung, the diaphragm contracts more forcefully, increasing intrathoracic volume and reducing intrathoracic pressure. Poorly compliant lungs will also lead to increased work of breathing. During mechanical ventilation, increased positive airway pressure is needed to achieve adequate ventilation when lung compliance decreases.

Causes of decreased compliance include extrapulmonary conditions like pneumothorax and pleural effusion as well as intrapulmonary conditions such as pneumonia and inflammatory lung tissue diseases, such as ARDS, pulmonary fibrosis, and pulmonary edema. These conditions are associated with an increase in the water content in the interstitial space and alveoli. The impact of this on lung compliance is similar to what happens when a sponge becomes saturated with water. A normal sponge re-expands quickly when compressed, while a wet sponge is harder to compress and re-expands more slowly because its normal elasticity is opposed by the extra weight of the fluid in the sponge.

The chest wall in infants and young children is much more compliant than in older children and adults. Therefore, relatively small pressure changes can move the chest wall. During normal breathing, diaphragm contraction in infants pulls the lower ribs slightly inward but does not cause significant chest retraction. However, forcefully contracting the diaphragm results in a large drop in pressure within the chest, pulling the child’s compliant chest inward (ie, retracting it) and pushing the abdomen out during inspiration, which can actually limit lung expansion during inspiration. These muscular contractions are useful in older children and adolescents when the chest wall is more rigid, but in infants and young children, the compliant chest wall (which is not fully calcified) actually can be counterproductive.

Children with neuromuscular disorders have a weak chest wall and weak respiratory muscles that can make breathing and coughing ineffective. Muscle weakness can result in characteristic seesaw breathing (the chest wall retracts and the abdomen expands simultaneously).

Inspiratory and Expiratory Flow

The inspiratory muscles of respiration include the diaphragm, intercostal muscles, and accessory muscles (primarily of the abdomen and neck). During spontaneous breathing, inspiratory muscles (chiefly the diaphragm) increase intrathoracic volume, resulting in decreased intrathoracic pressure. When intrathoracic pressure is less than atmospheric pressure, air flows into the lungs (inspiration); the intercostal muscles stabilize the chest wall to optimize intrathoracic inspiratory air flow. Accessory muscles of respiration are not typically needed during normal ventilation; however, in respiratory disorders that increase airway resistance or reduce lung compliance, activation of these accessory muscles helps to improve the child's ability to augment inspiratory air flow.

Expiratory flow results from the inspiratory muscles relaxing and the lung and chest wall elastically recoiling, decreasing intrathoracic volume and, thus, increasing intrathoracic pressure to a higher level than that of the atmospheric pressure. During spontaneous breathing, expiratory flow is primarily a passive process. However, expiration may become an active process in the presence of increased lower airway resistance and may require the abdominal wall muscles and the intercostal muscles.

Critical Concepts

The Diaphragm's Role in Breathing

The normal diaphragm is dome-shaped and contracts most forcefully in this shape. When the diaphragm flattens, as occurs with lung hyperinflation (eg, acute asthma), contraction is less forceful and ventilation is less efficient. Respiration is compromised if diaphragm movement is impeded by abdominal distention and high intra-abdominal pressure (eg, gastric inflation) or by air trapping caused by airway obstruction. During infancy and early childhood, the intercostal muscles serve primarily to stabilize the chest wall. They cannot effectively lift the chest wall to increase intrathoracic volume and compensate for loss of diaphragm motion.

Central Nervous System Control of Breathing

Breathing is controlled by complex mechanisms involving

- Brainstem respiratory centers
- Central and peripheral chemoreceptors
- Voluntary control

A group of respiratory centers located in the brainstem control spontaneous breathing. Voluntary control from the cerebral cortex, such as breath holding, panting, and sighing, can also override breathing. Conditions like infection of the central nervous system, traumatic brain injury, and drug overdose can impair respiratory drive, resulting in hypoventilation or even apnea.

Note that central chemoreceptors respond to changes in the hydrogen ion concentration of cerebrospinal fluid, which is largely determined by the arterial CO_2 tension ($PaCO_2$). Peripheral chemoreceptors (eg, in the carotid body) respond primarily to a decrease in arterial O_2 (PaO_2); some receptors also respond to an increase in $PaCO_2$.

Identifying Respiratory Problems by Type

Respiratory distress or failure can be classified into one or more of the following types:

- Upper airway obstruction
- Lower airway obstruction
- Lung tissue disease
- Disordered control of breathing

Respiratory problems do not always occur one at a time. A child may have more than 1 cause of respiratory distress or failure, such as disordered control of breathing caused by a head injury and then pneumonia (lung tissue disease). A patient may also exhibit symptoms consistent with more than 1 class of respiratory abnormality.

Upper Airway Obstruction

Obstruction of the upper airways (ie, the airways outside the thorax) can occur in the nose, pharynx, or larynx and can range from mild to severe.

Causes of Upper Airway Obstruction

Common causes of upper airway obstruction are foreign-body aspiration (eg, food, a small object), and airway swelling (eg, anaphylaxis, tonsillar hypertrophy, croup, epiglottitis). Other causes include a mass that compromises the airway lumen (eg, pharyngeal or peritonsillar abscess, retropharyngeal abscess, tumor), thick secretions obstructing the nasal passages, or any congenital airway abnormality (eg, congenital subglottic stenosis) resulting in narrowing of the airway, or poor control of the upper airway due to a decreased level of consciousness. It also may be hospital acquired. For example, subglottic stenosis may develop secondary to trauma induced by ET intubation.

Due to their small airway, infants and small children may easily develop upper airway obstruction. Because an infant's tongue is large in proportion to the oropharyngeal cavity, if the infant has a decreased level of consciousness, the muscles may relax and allow the tongue to fall back and obstruct the oropharynx. Infants also have a prominent occiput. If the infant with a decreased level of consciousness is supine, resting on the large occiput can cause flexion of the neck, resulting in upper airway obstruction.

Signs of Upper Airway Obstruction

The major clinical signs, such as stridor, hoarseness, or a change in voice or cry, typically occur during the inspiratory phase of the respiratory cycle. Inspiratory retractions, use of accessory muscles, and nasal flaring are often present. The respiratory rate is often only mildly elevated because upper airway obstruction is worse with faster breathing. Examples include foreign body obstruction, croup, and epiglottitis. Other signs of upper airway obstruction include

- Increased respiratory rate and effort
- Drooling, snoring, or gurgling sounds
- Poor chest rise
- Poor air entry on auscultation

Lower Airway Obstruction

Obstruction of the lower airways (ie, the airways within the thorax) can occur in the lower trachea, the bronchi, or the bronchioles.

Causes of Lower Airway Obstruction

Common causes of lower airway obstruction are asthma and bronchiolitis.

Signs of Lower Airway Obstruction

The major clinical signs typically occur during the expiratory phase of the respiratory cycle, wherein the child often has wheezing and a prolonged expiratory phase that requires increased expiratory effort. The respiratory rate is usually elevated, particularly in infants. Inspiratory retractions become prominent when the lower airway obstruction impairs inspiration and exhalation, requiring increased respiratory effort. Examples include asthma and bronchiolitis. Signs of lower airway obstruction include

- Cough
- Prolonged expiratory phase
- Wheezing

- Retractions
- Decreased air movement
- Poor air entry on auscultation or silent chest

Lower Airway Obstruction and Respiratory Rate

Small airways are obstructed through a variety of mechanisms in acute lower airway obstruction. In asthma, small airways are obstructed via smooth muscle bronchial constriction, mucus plugging, inflammation, and external collapse due to high intrathoracic pressures. In bronchiolitis, small airway obstruction occurs predominantly because of mucus plugging and bronchial edema. If these obstructions are severe, air trapping and lung hyperinflation occur, further exacerbating the obstruction of the smaller airways. An infant with lower airway obstruction, by comparison, typically has a rapid respiratory rate. The infant has a compliant chest wall. If the infant attempts to breathe more deeply, the resulting decrease in intrapleural pressure may result in greater chest wall retractions. When lower airway obstruction is significant, it is more efficient for the infant to breathe at a fast rate with small tidal volumes to maintain minute ventilation, keeping a relatively larger volume of gas in the lungs.

Lung Tissue Disease

This condition describes diseases involving the substance (ie, parenchyma or tissue) of the lung. In this state, the child's lungs become stiff because of fluid accumulation in the alveoli, interstitium, or both, requiring increased respiratory effort during inspiration and exhalation. Therefore, retractions and accessory muscle use are common. Hypoxemia is often marked due to alveolar collapse or reduced oxygen diffusion caused by pulmonary edema fluid and inflammatory debris in alveoli. Tachypnea is common and often quite marked. Patients frequently attempt to counteract alveolar and small airway collapse by increasing efforts to maintain an elevated end-expiratory pressure, often manifested by grunting respirations. Signs of lung tissue disease include

- Crackles on auscultation
- Grunting
- Decreased breath sounds
- Tachypnea
- Hypoxemia

Causes of Lung Tissue Disease

Causes of lung tissue disease vary and can include pneumonia or pneumonitis from any cause (eg, bacterial, viral, chemical, aspiration) and cardiogenic (eg, CHF) and noncardiogenic pulmonary edema (eg, ARDS). Other potential causes include

- Pulmonary contusion (trauma)
- Allergic reaction
- Toxins
- Vasculitis
- Infiltrative disease
- Allergic, vascular, widespread inflammatory, environmental, and other factors

Signs of Lung Tissue Disease

Children with lung tissue disease can often maintain ventilation (ie, CO_2 elimination) with a relatively small number of functional alveoli, but they cannot maintain oxygenation as effectively. Tachycardia and hypoxemia are earlier signs of lung tissue disease than hypercarbia. Compromised ventilation, indicated by hypercarbia, is typically a late manifestation of the disease process.

Grunting produces early glottic closure during expiration. Grunting is a compensatory mechanism to maintain positive airway pressure and prevent collapse of the alveoli and small airways. Grunting is an ominous sign of impending respiratory failure.

Disordered Control of Breathing

In this state, there is inadequate respiratory effort, and the parent may say that the child is “breathing funny.” There may be periods of increased respiratory rate, effort, or both followed by decreased rate, effort, or both, or the child’s respiratory rate or effort may be continuously inadequate. Often the net effect is hypoventilation leading to hypoxemia and hypercarbia.

Causes of Disordered Control of Breathing

Disordered control of breathing may result from a host of conditions, including neurologic disorders (eg, seizures, central nervous system infections, head injury, brain tumor, hydrocephalus, neuromuscular disease), metabolic abnormalities, and drug overdose. Because disordered control of breathing is typically associated with conditions that impair neurologic function, these children often have a decreased level of consciousness.

Signs of Disordered Control of Breathing

Signs of disordered control of breathing include

- Variable or irregular respiratory rate and pattern (tachypnea alternating with bradypnea)
- Variable respiratory effort
- Shallow breathing with inadequate effort (frequently resulting in hypoxemia and hypercarbia)

- Central apnea (ie, apnea without any respiratory effort)
- Normal or decreased air movement

Summary: Recognizing Respiratory Problems

This section summarizes the recognition and identification of respiratory problems. This summary does not include all respiratory emergencies but instead provides key characteristics for a limited number of diseases.

Upper Airway Obstruction

- Airway: patent airway open and maintainable/not maintainable
- Breathing:
    - –Respiratory rate/effort increased
    - –Breath sounds:
        - Stridor (typically inspiratory)
        - Barking cough
        - Hoarseness
        - Change in pitch or cry
    - –Decreased air movement
- Circulation:
    - –Heart rate: tachycardia (early); bradycardia (late)
    - –Skin: pallor, cool skin (early); cyanosis (late)
- Disability: level of consciousness: anxiety, agitation (early); lethargy, unresponsiveness (late)
- Exposure: variable temperature

Lower Airway Obstruction

- Airway: patent airway open and maintainable/not maintainable
- Breathing:
    - –Respiratory rate/effort increased
    - –Breath sounds:
        - Wheezing (typically expiratory)
        - Prolonged expiratory phase
    - –Decreased air movement

- Circulation:
    - –Heart rate: tachycardia (early); bradycardia (late)
    - –Skin: pallor, cool skin (early); cyanosis (late)
- Disability: level of consciousness: anxiety, agitation (early); lethargy, unresponsiveness (late)
- Exposure: variable temperature

Lung Tissue Disease

- Airway: patent airway open and maintainable/not maintainable
- Breathing:
    - –Respiratory rate/effort increased
    - –Breath sounds:
        - Grunting
        - Crackles
        - Decreased breath sounds
    - –Decreased air movement
- Circulation:
    - –Heart rate: tachycardia (early); bradycardia (late)
    - –Skin: pallor, cool skin (early); cyanosis (late)
- Disability: level of consciousness: anxiety, agitation (early); lethargy, unresponsiveness (late)
- Exposure: variable temperature

Disordered Control of Breathing

- Airway: patent airway open and maintainable/not maintainable
- Breathing:
    - –Variable respiratory rate/effort
    - –Normal breath sounds
    - –Variable air movement
- Circulation:
    - –Heart rate: tachycardia (early); bradycardia (late)
    - –Skin: pallor, cool skin (early); cyanosis (late)
- Disability: level of consciousness: anxiety, agitation (early); lethargy, unresponsiveness (late)
- Exposure: variable temperature

Part 6

Managing Respiratory Problems

Respiratory problems are a major cause of cardiac arrest in children. In fact, many infants and children who require CPR (both in and out of hospital) have respiratory problems that progress to cardiopulmonary failure. It may not be possible to differentiate between respiratory distress and respiratory failure on the basis of clinical examination alone because respiratory failure can develop even without significant signs of distress. In children, clinical deterioration in respiratory function may progress rapidly, so there is little time to waste. Promptly recognizing and effectively managing respiratory problems are fundamental to PALS.

Critical Concepts

Intervene Quickly to Restore Respiratory Function

PALS providers must intervene quickly to restore adequate respiratory function. Outcomes may be greatly improved by early identification and prompt management of respiratory distress and failure. Once respiratory failure progresses to cardiac arrest, outcomes are often poor.

Learning Objective

After completing this Part, you should be able to perform early interventions for respiratory distress and failure.

During the course, you will participate in the Airway Management Skills Station. You will practice and demonstrate your proficiency in performing basic airway management skills, such as inserting airway adjuncts, effective bag-mask ventilation, and suctioning. Refer to the Appendix for a checklist of required competencies. Refer to Part 2 for details on bag-mask ventilation and to Part 4 for details on respiratory function monitoring devices (eg, monitoring of O_2 saturation by pulse oximetry, monitoring of exhaled CO_2). O_2 delivery systems are discussed later in this Part.

Rescue Breathing

Respiratory Arrest

Respiratory arrest is the absence of respirations (ie, apnea) with detectable cardiac activity. The health care professional must provide rescue breathing to prevent cardiac arrest.

Rescue Breathing

Guidelines for rescue breathing for infants and children are as follows:

- Give 20 to 30 breaths per minute (about 1 breath every 2-3 seconds).
- Give each breath in 1 second.
- Each breath should result in visible chest rise.
- Check the pulse every 2 minutes; if the child becomes pulseless, shout for help and provide compressions as well as ventilation (CPR).
- Use oxygen as soon as it is available.

Initial Management of Respiratory Problems

The first priority in managing a seriously ill or injured child who is not in cardiac arrest is evaluating the airway and breathing. If you identify signs of respiratory distress or failure, perform initial interventions that support or restore adequate oxygenation and ventilation. Because respiratory conditions are a major cause of cardiac arrest in infants and children, when respiratory distress or failure is detected, it is important to begin appropriate interventions quickly. Consider expert consultation based on the presentation of the child.

Initial interventions include rapidly evaluating respiratory function to identify the type and severity, rather than the precise etiology, of the respiratory problem. Once both oxygenation and ventilation are stabilized, identify the cause of the problem to facilitate targeted interventions. Use the evaluate-identify-intervene sequence to monitor symptom progression or response to therapy and to prioritize further interventions.

Initially stabilizing and managing a child in respiratory distress or respiratory failure may include the interventions in Table 28.

Table 28 Initial Management of Respiratory Problems

Evaluate	Interventions (as indicated)

Airway	• Support an open airway (allow child to assume position of comfort) or, if necessary, open the airway with
	  ○ –Head tilt–chin lift
	  ○ –Jaw thrust without head tilt if you suspect cervical spine injury. If this maneuver does not open the airway, use the head tilt–chin lift or jaw thrust with gentle head extension
	• Clear the airway if indicated (eg, suction nose and mouth, remove visualized foreign body, do not perform a blind finger sweep)
	• Consider an OPA or nasopharyngeal airway to improve airway openness/patency

Breathing	• Monitor O_2 saturation by pulse oximetry • Provide O_2 (humidified if available). Use a high-concentration delivery device such as a nonrebreathing mask or heated high flow O_2 therapy for treating severe respiratory distress or possible respiratory failure • Administer inhaled medication (eg, albuterol, epinephrine) as needed • Assist ventilation with bag-mask device and supplemental O_2 if needed • Prepare to insert an advanced airway if indicated
Circulation	• Monitor heart rate, heart rhythm, and blood pressure • Establish vascular access (for fluid therapy and medications) as indicated

Principles of Targeted Management

Once you stabilize both oxygenation and ventilation, identify the type of respiratory problem to help prioritize the next interventions. This Part reviews principles of targeted management for the following 4 types of respiratory problems:

- Upper airway obstruction
- Lower airway obstruction
- Lung tissue disease
- Disordered control of breathing

Managing Upper Airway Obstruction

Upper airway obstruction is a mild to severe obstruction of the large airways outside the thorax (ie, in the nose, pharynx, or larynx). Causes of upper airway obstruction are airway swelling, foreign body, or infection. Other causes include edema or

swelling of the soft tissue of the upper airway (large tonsils or adenoids), a mass in the airway, thick secretions, congenital narrowing of the upper airway, or poor control of the upper airway due to a decreased level of consciousness.

Infants and small children are especially prone to upper airway obstruction. If the infant has a decreased level of consciousness, the muscles may relax and allow the tongue to fall back and obstruct the oropharynx. Also, if the infant is supine, resting on the large occiput can cause flexion of the neck, resulting in upper airway obstruction. Positioning of the infant is important to maintain the neutral sniffing position. In young infants, nasal obstruction can impair ventilation. Secretions, blood, and debris in the nose, pharynx, and larynx from infection, inflammation, or trauma also can obstruct the airway. Remember that the smaller the airway is, the more easily it can become obstructed.

General Management of Upper Airway Obstruction

General management of upper airway obstruction includes the initial interventions in Table 28. Additional measures focus on relieving the obstruction and may include opening the airway by

- Allowing the child to assume a position of comfort/maintain infants in a neutral sniffing position
- Performing manual airway maneuvers, such as a jaw thrust or head tilt–chin lift
- Removing a foreign body
- Suctioning the nose or mouth
- Reducing airway swelling with medications
- Minimizing agitation (agitation often worsens upper airway obstruction)
- Deciding whether an airway adjunct or advanced airway is needed
- Deciding early if a surgical airway (tracheostomy or needle cricothyroidotomy) is needed

You may use suctioning to help remove secretions, blood, or debris; however, if the upper airway obstruction is caused by edema from infection (eg, croup) or allergic reaction, carefully weigh potential benefits vs risks of suctioning. Suctioning may further agitate the child and increase respiratory distress, so consider allowing the child to assume a position of comfort. Give nebulized epinephrine, particularly if the swelling is beyond the tongue. Corticosteroids (inhaled, IV, oral, or intramuscular [IM]) also may help in this situation.

With severe upper airway obstruction, call early for advanced help because the health care professional with the greatest skill and experience in airway management is the person most likely to establish an airway safely. Failure to aggressively treat an acute partial upper airway obstruction may lead to complete airway obstruction and, ultimately, to cardiac arrest.

In less severe cases of upper airway obstruction, infants and children may benefit from specific airway adjuncts. For example, in a child with a decreased level of consciousness, an OPA or nasopharyngeal airway may help to relieve obstruction caused

by the tongue. Use an OPA only if the child is deeply unconscious with no gag reflex. A child with an intact gag reflex is more likely to tolerate a nasopharyngeal airway, which should be inserted carefully to avoid nasopharyngeal trauma and bleeding. Avoid using a nasopharyngeal airway in children with increased bleeding risk or head or face trauma.

An infant or child with upper airway obstruction from redundant tissues or tissue edema may benefit from noninvasive ventilation with positive airway pressure.

Specific Management of Upper Airway Obstruction by Etiology

Specific causes of upper airway obstruction require specific interventions. This section reviews management of upper airway obstruction due to

- Croup
- Anaphylaxis
- FBAO

Manage Croup Based on Severity

Manage croup according to your assessment of clinical severity. The characteristics and interventions for croup are listed by degree of severity in Table 29.

Table 29.Managing Croup

Severity of croup	Signs and symptoms	Interventions
Mild	• Occasional barking cough • Little or no stridor at rest • Absent or mild retractions	Consider dexamethasone
Moderate to severe	**Moderate** • Frequent barking cough • Easily audible stridor at rest • Retractions at rest • Little or no agitation • Good air entry by auscultating the peripheral lung fields **Severe**	• Administer humidified O_2/heated high flow nasal oxygen therapy • Give nothing by mouth other than necessary medications • Administer nebulized epinephrine • Administer dexamethasone

	• Frequent barking cough • Prominent inspiratory and occasional expiratory stridor • Marked retractions • Significant agitation • Decreased air entry by auscultating the lungs	• After giving nebulized epinephrine, observe for at least 2 hours to ensure continued improvement (no recurrence of stridor) • Consider using heliox (helium-oxygen mixture) for severe disease if the child requires no higher than 40% inspired oxygen concentration. If the obstruction is severe the oxygen concentration will need to be less
Respiratory distress or impending respiratory failure	Barking cough (may not be prominent if the child's respiratory effort is growing weaker with the development of severe hypoxemia and hypercarbia), audible stridor at rest (can be difficult to hear with failing respiratory effort), retractions (may not be severe if respiratory effort is failing)	• Administer a high concentration of O_2; use a nonrebreathing mask or heated high-flow nasal oxygen therapy, CPAP, or bilevel positive airway pressure (BiPAP) if available • Administer dexamethasone IV/IM • Provide assisted ventilation (ie, bag-mask ventilation timed to support the child's own inspiration) for persistent, severe hypoxemia (<90% O_2 saturation) despite O_2 administration, inadequate ventilation, or changes in level of consciousness • Perform ET intubation if indicated; to avoid injury to the subglottic area, use a smaller ET tube size (a half size smaller than predicted for the child's age • Prepare for surgical airway if needed
Respiratory failure/impending respiratory arrest	Poor air movement on auscultation, lethargy or decreased level of consciousness, and, sometimes, pallor or cyanosis despite administering supplemental O_2	• Administer a high concentration of O_2; use a nonrebreathing mask or heated high-flow nasal oxygen therapy, CPAP, or BiPAP if available • Administer dexamethasone IV/IM • Provide assisted ventilation (ie, bag-mask ventilation timed to support the child's own inspiration) for persistent, severe hypoxemia (<90% O_2 saturation) despite O_2 administration, inadequate ventilation, or changes in level of consciousness

		• Perform ET intubation if indicated; to avoid injury to the subglottic area, use a smaller ET tube size (a half size smaller than predicted for the child's age) • Prepare for surgical airway if needed

General management for upper airway obstruction includes the initial interventions in Table 28 and may include the specific interventions for managing croup listed in Table 29.

O_2 saturation may be slightly low in mild and moderate croup and is commonly well below normal in severe croup.

Critical Concepts

ET Intubation

ET intubation of the child with upper airway obstruction is a high-risk procedure and should be performed by a team with significant pediatric airway expertise. Use neuromuscular blockade only if you are confident the child's oxygenation and ventilation can be supported with bag-mask ventilation.

Managing Anaphylaxis

In addition to the initial interventions in Table 28, specific interventions that may be used for managing anaphylaxis are listed in Table 30.

Table 30.Managing Anaphylaxis

Severity of allergic reaction	Interventions

Mild (no respiratory distress, no hemodynamic instability, patent airway)	• Remove the offending agent (eg, stop the IV antibiotic, removing bee stinger) • Get help • Ask the child or caregiver about any history of allergy or anaphylaxis; look for a medical alert bracelet or necklace • Consider an oral dose of antihistamine

Moderate to severe	• Administer IM epinephrine by autoinjector or regular syringe every 10 to 15 minutes as needed; repeat doses as needed
	• Treat bronchospasm (wheezing) with albuterol administered by metered-dose inhaler or nebulizer solution
	• Give continuous nebulization if indicated (ie, severe bronchospasm)
	• For severe respiratory distress, anticipate further airway swelling and prepare for ET tube intubation
	• To treat hypotension:
	  ○ –Place the child in the supine position as tolerated
	  ○ –Use pharmacological adjuncts once stabilized:
	  ▪ Methylprednisolone or equivalent corticosteroid IV
	  ▪ Diphenhydramine IV
	  ○ –Administer isotonic crystalloid (eg, normal saline, lactated Ringer's) 20 mL/kg bolus IV (repeat as needed)
	  ○ –For hypotension unresponsive to fluids and IM epinephrine, administer an epinephrine infusion titrated to achieve adequate blood pressure for age

Managing Lower Airway Obstruction

Lower airway obstruction, commonly caused by bronchiolitis and asthma, involves the smaller bronchi and bronchioles inside the thorax.

General Management of Lower Airway Obstruction

General management of lower airway obstruction includes the initial interventions in Table 28.

In a child with severe respiratory distress or respiratory failure, first restore adequate oxygenation; immediately correcting hypercarbia to the child's baseline level is not required because most children can tolerate hypercarbia without adverse effects.

If a child with lower airway obstruction needs bag-mask ventilation, provide effective ventilation at a relatively slow rate, allowing more time for expiration. This reduces the risk that air will remain inside the chest at the end of expiration. Providing too many breaths or breaths with too much volume may result in complications, such as

- Air entering the stomach (gastric distention), resulting in increased risk of vomiting and aspiration and possible prevention of adequate diaphragm movement, limiting effective ventilation
- Risk of pneumothorax (air leak into space surrounding the lungs), resulting in decreased blood return to the heart and risk of lung collapse and resultant complications (severe hypoxemia, obstructive shock)
- Severe air trapping, resulting in severe decrease in oxygenation and decreased venous return to the heart and cardiac output

Specific Management of Lower Airway Obstruction by Etiology

Specific causes of lower airway obstruction require specific interventions. This section reviews management of lower airway obstruction due to the following:

- Bronchiolitis
- Acute asthma

Distinguishing between bronchiolitis and asthma in a wheezing infant can be difficult. However, a history of wheezing episodes suggests that the infant has reversible bronchospasm (ie, asthma). Consider a trial of bronchodilators if the diagnosis is unclear.

Managing Bronchiolitis

In addition to the initial interventions in Table 28, specific measures that may be used for managing bronchiolitis include performing oral or nasal suctioning as needed and considering laboratory and other tests, which may include viral studies, chest x-ray, and blood gas.

Randomized controlled trials of bronchodilator or corticosteroid therapy for bronchiolitis have shown mixed results. Some infants improve when treated with nebulized epinephrine or albuterol, whereas nebulizer therapy aggravates respiratory symptoms in other infants. Consider a trial of nebulized epinephrine or albuterol treatment, and discontinue it if you observe no improvement. Administer supplemental O_2 if O_2 saturation is less than 94%.

Managing Acute Asthma

Manage asthma according to your assessment of clinical severity (Table 31). In addition to the initial interventions in Table 28, use specific interventions for managing acute asthma (Table 32).

Table 31.Classifying Mild, Moderate, and Severe Asthma

Parameter*	Mild	Moderate	Severe	Respiratory arrest imminent
Breathlessness and positioning	• While walking • Can lie down	• While talking (infant will have softer, shorter cry; difficulty feeding) • Prefers sitting	• At rest (infant will stop feeding) • Hunched forward	
Talks in	Sentences	Phrases	Words	
Alertness	May be agitated	Usually agitated	Usually agitated	Drowsy or confused
Respiratory rate†	Increased	Increased	Often >30/min	
Accessory muscles and suprasternal retractions	Usually not	Usually	Usually	Paradoxical thoraco-abdominal movement
Wheeze	Moderate, often only end-expiration	Loud	Usually loud, often inspiratory and expiratory	Absence of wheeze
Pulse/min‡	<100	100-120	>120	Bradycardia
Pulsus paradoxus	Absent <10 mm Hg	May be present 10-25 mm Hg	Often present >25 mm Hg (adult); 25-40 mm Hg (child)	Absence suggests respiratory muscle fatigue
Peak Expiratory Flow (PEF) (if used in clinical practice) after	>80%	Approximately 60%-80%	<60% predicted or personal best (<100 L/min adults) or response lasts <2 hours	

initial bronchodilator % predicted or % personal best				
$PaCO_2$	Low to normal[§]	Normal to high	High	High
SpO_2 (room air)	>95%	91%-95%	<90%	

[*]The presence of several parameters, but not necessarily all, indicates the general classification of the severity.

[†]Guide to limits of normal respiratory rate in infants and children: age <2 months, respiratory rate <60/min; age 2-12 months, respiratory rate <50/min; age 1-5 years, respiratory rate <40/min; age 6-8 years, respiratory rate <30/min.

[‡]Guide to limits of normal pulse rate in infants and children: infant (2-12 months), pulse rate <160/min; toddler (1-2 years), pulse rate <120/min; preschool/school age (2-8 years), pulse rate <110/min.

[§]Normal $PaCO_2$ is 35-45 mm Hg.

Adapted from National Heart, Lung, and Blood Institute and World Health Organization. *Global Strategy for Asthma Management and Prevention NHLBI/WHO Workshop Report*. US Department of Health and Human Services; 1997. Publication 97-4051.

Table 32.Managing Acute Asthma

Asthma severity	Severity interventions
Mild to moderate	• Administer humidified O_2 in high concentration via nasal cannula or O_2 mask; titrate according to pulse oximetry. Keep O_2 saturation >94% • Administer albuterol by metered-dose inhaler or nebulizer solution • Administer oral corticosteroids
Moderate to severe	• Administer humidified O_2 in high concentrations to keep O_2 saturation >94%; use a nonrebreathing mask if needed. If this is unsuccessful, further support such as noninvasive positive-pressure ventilation or heated high-flow nasal cannula may be indicated • Administer albuterol by metered-dose inhaler (with spacer) or nebulizer solution. If wheezing and aeration are not alleviated, continuous albuterol administration may be required • Administer ipratropium bromide by nebulizer solution. Albuterol and ipratropium may be mixed for nebulization • Consider establishing vascular access for administering fluids and medications • Administer corticosteroids PO/IV • Consider administering magnesium sulfate by slow (15-30 min) IV bolus while monitoring heart rate and blood pressure

	• Perform diagnostic assessments (eg, arterial blood gas, chest x-ray) as indicated
Severe to impending respiratory failure	All of the previous therapies are indicated in addition to the following: • Administer O_2 in high concentrations; use a nonrebreathing mask if available • Administer albuterol by continuous nebulizer • Administer corticosteroid IV if not already given • Consider administration of IM epinephrine • Terbutaline remains an alternative, subcutaneously or by continuous IV infusion. Practically, however, terbutaline can often be difficult to obtain and administer in a timely manner; IM epinephrine is preferred in an emergent situation • Consider noninvasive ventilation such as heated high-flow nasal canula, CPAP, or bilevel positive airway pressure, especially in alert, cooperative children • Consider ET intubation or SGA for children with refractory hypoxemia (low O_2 saturation), worsening clinical condition (eg, decreasing level of consciousness, irregular breathing), or both despite the aggressive medical management described already. Intubation and SGA in an asthmatic child carries significant risk for respiratory and circulatory complications. Because of the high intrathoracic pressures in patients with asthma, we highly recommend inserting a cuffed ET tube

Managing Lung Tissue Disease

Lung tissue disease (also called *parenchymal lung disease*) refers to various clinical conditions. Common causes of lung tissue disease are pneumonia (eg, infectious, chemical, aspiration) and cardiogenic pulmonary edema, but ARDS and traumatic pulmonary contusion are other causes. Lung tissue disease can also result from allergic, vascular, widespread inflammatory, environmental, and other factors.

General Management of Lung Tissue Disease

General management of lung tissue disease includes the initial interventions in Table 28. In children with hypoxemia refractory to high inspired O_2 concentrations, other modalities that offer varying degrees of inspiratory and expiratory pressure support are

usually helpful; these include heated humidified high-flow nasal cannula, CPAP, bilevel positive airway pressure (BiPAP), mechanical ventilation, and positive expiratory pressure (CPAP, noninvasive ventilation, or mechanical ventilation with PEEP).

Specific Management of Lung Tissue Disease by Etiology

Specific causes of lung tissue disease require specific interventions. This section reviews the management of lung tissue disease from the following causes:

- Infectious pneumonia
- Chemical pneumonitis
- Aspiration pneumonitis
- Cardiogenic pulmonary edema
- Noncardiogenic pulmonary edema (ARDS)

Managing Infectious Pneumonia

Infectious pneumonia results from viral, bacterial, or fungal inflammation of the alveoli. Viruses, bacteria (eg, *Streptococcus pneumoniae*), and atypical bacteria (eg, *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*) commonly cause acute community-acquired pneumonia in children. Methicillin-resistant *Staphylococcus aureus* is increasingly common and may cause empyema (ie, a collection of pus and fluid in the pleural cavity).

In addition to the initial interventions in Table 28, specific interventions for managing acute infectious pneumonia include

- Performing diagnostic assessments (eg, ABG, chest x-ray, viral studies, complete blood count, blood culture, sputum gram stain and culture) as indicated
- Administering antibiotic therapy (the goal is to administer within the first hour after medical contact); it may not be necessary to draw blood cultures before giving antibiotics, so follow facility protocol
- Treating wheezing with albuterol by metered-dose inhaler (MDI) or nebulizer solution
- Considering the use of noninvasive positive-pressure ventilation (eg, heated humidified high-flow nasal cannula, BiPAP, CPAP); in severe cases, ET intubation and mechanical ventilation may be required
- Reducing metabolic demand by normalizing temperature (ie, treating fever)

Managing Chemical Pneumonitis

Chemical pneumonitis is an inflammation of the lung tissue caused by inhaling or aspirating toxic liquids, gases, or particulate matter such as dust or fumes. Aspirating hydrocarbons or inhaling irritant gases (eg, chlorine) can result in noncardiogenic pulmonary edema with increased capillary permeability.

In addition to the initial interventions in Table 28, managing chemical pneumonitis may include specific interventions, such as

- Treating wheezing with nebulized bronchodilator
- Considering the use of noninvasive ventilation such as heated humidified high-flow nasal cannula, CPAP, or BiPAP. Intubation and mechanical ventilation may be required. Consider early intubation particularly if the child requires transport to a tertiary care facility, is not tolerating secretions, or demonstrates evidence of upper airway edema and obstruction.

Obtain early consultation in a child with rapidly progressive symptoms. Consider referral to a tertiary center for advanced technologies (eg, high-frequency oscillation, pediatric extracorporeal membrane oxygenation [ECMO]).

Managing Aspiration Pneumonitis

Aspiration pneumonitis is a form of chemical pneumonitis resulting from the toxic effects of aspirated oral secretions or stomach acid and enzymes and the subsequent inflammatory response. Obtain early consultation from specialists and referral to a tertiary center.

In addition to the initial interventions for the general management of lung tissue disease in Table 28, managing aspiration pneumonitis includes specific interventions, such as

- Considering using high-flow nasal cannula, CPAP, BiPAP, or noninvasive ventilation. Intubation and mechanical ventilation may be required in severe cases.
- Considering administering antibiotics if the child has a fever and an infiltrate is present on a chest x-ray. Prophylactic antimicrobial therapy is not indicated.

Managing Cardiogenic Pulmonary Edema

In cardiogenic pulmonary edema, high pressure in the pulmonary capillaries causes fluid to leak into the lung interstitium and alveoli. The most common cause of acute cardiogenic pulmonary edema in children is left ventricular myocardial dysfunction, which can result from congenital heart disease, myocarditis, cardiomyopathy, inflammatory processes, hypoxia, and cardiac-depressant medications (eg, β-adrenergic blockers, calcium channel blockers). It is important to obtain expert consultation early in the presentation.

In addition to the initial interventions in Table 28, specific interventions for managing cardiogenic pulmonary edema include

- Providing respiratory support (ie, noninvasive ventilation or mechanical ventilation with PEEP) as needed
- Considering diuretics to reduce left atrial pressure, inotropic infusions, and afterload-reducing agents to improve ventricular function.

- Reducing metabolic demand by normalizing temperature (treat fever)

Indications for respiratory support (noninvasive ventilation or ET tube intubation with mechanical ventilation) in children with cardiogenic pulmonary edema include

- Persistent hypoxemia despite oxygen administration and noninvasive ventilation
- Impending respiratory failure
- Hemodynamic compromise (eg, hypotension, severe tachycardia, signs of shock)

Add PEEP during mechanical ventilation to help reduce the need for high O_2 concentrations. You may usually start it at about 5 cm H_2O and adjust upward until O_2 saturation and oxygen delivery improve. Too much PEEP may create pulmonary hyperinflation that impedes systemic venous return to the heart, thus reducing cardiac output and O_2 delivery.

Managing Noncardiogenic Pulmonary Edema

ARDS usually follows a pulmonary (eg, pneumonia, aspiration) or systemic (eg, sepsis, pancreatitis, trauma) disease process that injures the interface between the alveoli and pulmonary capillaries and triggers release of inflammatory mediators. As a result, oxygen diffusion into the blood and, to a lesser degree, CO_2 diffusion from the blood to the alveoli is compromised. Recognizing and treating bacteremia, shock, and respiratory failure early may help prevent the progression to ARDS.

Characteristics of ARDS include

- Acute onset (within 7 days after insult)
- Oxygenation index* 4 or greater or oxygen saturation index† 5 or greater in patients who are mechanically ventilated
- PaO_2/FIO_2 300 mm Hg or less or SpO_2/FIO_2 250 or less in patients who are noninvasively ventilated‡
- New infiltrate consistent with acute pulmonary parenchymal disease not primarily atelectasis or pleural effusion
- Not fully explained by cardiac failure or fluid overload

*Oxygenation index is (FIO_2 × mean airway pressure × 100)/PaO_2

†Oxygen saturation index is mean airway pressure × FIO_2/SpO_2

‡Requires full facemask interface with CPAP (or expiratory positive airway pressure in BiPAP) 5 cm H_20 or greater

In addition to the initial interventions in Table 28, specific interventions for managing ARDS may include

- Monitoring heart rate and rhythm, blood pressure, respiratory rate, pulse oximetry, and end-tidal CO_2
- Obtaining laboratory studies, including ABG, and complete blood count
- Providing ventilatory support (ie, noninvasive ventilation or mechanical ventilation with PEEP) as needed

Indications for ventilatory support (noninvasive ventilation or ET intubation with mechanical ventilation) in children with ARDS are

- Worsening clinical and radiographic lung disease
- Hypoxemia refractory to high concentrations of inspired O_2

Correcting hypoxemia is the most important intervention, accomplished by mechanical ventilation strategies including high PEEP, low tidal volume. “Permissive” hypercarbia is a treatment approach that recognizes that correcting increased $PaCO_2$ is less important than correcting hypoxemia. Maintaining low tidal volumes (5-8 mL/kg; lower for children with decreased lung compliance) and keeping peak inspiratory pressure (less than 30 cm H_2O) to maintain easy chest rise is more important than correcting the $PaCO_2$. It is important to prevent barotrauma while optimizing ventilatory needs.

When intubating use a cuffed ET tube to prevent glottic air leak. When using a cuffed tube, carefully monitor cuff inflation pressure and maintain it according to manufacturer’s recommendations (typically less than 20 to 25 cm H_2O).

Expert consultation is important.

Managing Disordered Control of Breathing in Mechanically Ventilated Patients

Disordered control of breathing results in an abnormal respiratory pattern that produces inadequate minute ventilation. Common causes are neurologic disorders, including increased ICP, neuromuscular disease (weakness), central nervous system infections, head injury, brain tumor, and hydrocephalus, as well as conditions that depress the level of consciousness, such as deep sedation, seizures, metabolic disorders such as hyperammonemia, poisoning, or drug overdose.

General Management of Disordered Control of Breathing

General management to treat disordered control of breathing includes the initial interventions in Table 28.

Specific Management of Disordered Control of Breathing by Etiology

Specific causes of disordered control of breathing require specific interventions. This section reviews managing disordered control of breathing caused by the following:

- Increased ICP
- Neuromuscular disease
- Poisoning or drug overdose

Managing Respiratory Distress/Failure Due to Increased ICP

Increased ICP can be a complication of meningitis, encephalitis, intracranial abscess, subarachnoid hemorrhage, subdural or epidural hematoma, traumatic brain injury, hypoxic/ischemic insult, hydrocephalus, and central nervous system tumor. An irregular respiratory pattern is one sign of increased ICP, and a combination of irregular breathing or apnea, increased mean arterial pressure, and bradycardia is called *Cushing's triad*. This triad suggests a marked increase in ICP and impending brain herniation. However, children with increased ICP also can present with

- Irregular breathing with hypertension and tachycardia (as opposed to bradycardia)
- Isolated hypertension without irregular breathing or bradycardia
- Isolated bradycardia without irregular breathing or hypertension
- Isolated irregular breathing without hypertension or bradycardia

If you suspect increased ICP, obtain intensive care and neurosurgical consult. In addition to the initial interventions in Table 28, specific interventions for disordered control of breathing due to increased ICP may include

- Ensuring the head is midline, manually stabilizing the cervical spine, and using a jaw-thrust maneuver if you suspect trauma and need to open the airway
- Verifying open/patent airway, adequate oxygenation, and adequate ventilation. You may occasionally use a brief period of mild hyperventilation as temporizing rescue therapy in response to signs of impending brain herniation (eg, irregular respirations or apnea, bradycardia, hypertension, unequal or dilated pupil[s] not responsive to light, decerebrate or decorticate posturing) in the initial 48 hours after injury. If you use hyperventilation, you may consider advanced neuromonitoring for evaluating cerebral ischemia.
    - –Hyperventilation works by decreasing blood flow to the brain. It can only be used for short periods of time given the risk of secondary hypoxic injury.
- Ensuring airway protection and adequate oxygenation and ventilation with ET tube intubation
- Maintaining normocapnia with a target P_{CO_2} of 35 to 40 mm Hg
    - –If there are signs of impending brain herniation, a brief period of hyperventilation, along with the use of other therapies to decrease ICP, may be employed until more definitive therapy can be administered.
- Elevating the head of the bed. If there is concern for spine injury, use reverse Trendelenburg in order to elevate the head without flexing the spine.
- Decreasing environmental stimuli such as turning down the lights or turning down the monitor alarms in the room to ring only at the central station.
- Administering 20 mL/kg IV isotonic crystalloid (eg normal saline or lactated Ringer's) if the child has poor perfusion or other evidence of poor end-organ function

- Administering pharmacologic therapy for managing increased ICP (eg, osmotic agents, hypertonic saline)
- Treating agitation and pain aggressively once the airway is established and ventilation is adequate
- Avoiding hypotension because it can compromise cerebral blood flow in the setting of elevated ICP
    - –Hypertension, as part of Cushing's triad, is often present when ICP is increased. Therapeutic measures in the acute phase should be aimed at reducing the ICP, not administering antihypertensive medications.
- Avoiding and aggressively treating fever

Avoid severe prophylactic hyperventilation (to $PaCO_2$ less than 30 mm Hg) because hyperventilation can depress cardiac output by impairing venous return. Hyperventilation must be used cautiously—if at all—in the child with traumatic brain injury because excessive hyperventilation may cause cerebral vasoconstriction, leading to brain ischemia and a worse outcome. In patients with severe traumatic brain injury, consider hyperventilation in the initial 48 hours after injury only if there are acute signs of cerebral herniation. It must be guided by neuromonitoring for evaluating cerebral ischemia.

Respiratory Distress/Failure Due to Neuromuscular Disease

Chronic progressive neuromuscular diseases can affect the muscles of respiration. Affected children can develop an ineffective cough and difficulty managing secretions. Complications include atelectasis, restrictive lung disease, pneumonia (including aspiration pneumonitis and pneumonia), chronic respiratory insufficiency, and respiratory failure. Consider the initial interventions in Table 28 for disordered control of breathing due to neuromuscular disease. For children with advanced restrictive lung disease, you may use long-term noninvasive ventilation.

Critical Concepts

Medications to Avoid in Children With Neuromuscular Disease

Recall that the use of succinylcholine for intubating children with neuromuscular diseases may trigger life-threatening conditions, such as hyperkalemia or malignant hyperthermia. Several commonly used pediatric acute care medications, such as aminoglycosides, have intrinsic neuromuscular blocking activity that can worsen respiratory muscle weakness.

Managing Respiratory Distress/Failure Due to Poisoning or Drug Overdose

One of the most common causes of respiratory distress or failure after a poisoning or drug overdose is depression of central respiratory drive; less common causes are respiratory muscle weakness or paralysis, loss of consciousness, and upper airway obstruction by the tongue.

Complications of disordered breathing in this setting include upper airway obstruction and poor respiratory effort and rate, leading to hypoxemia and/or hypercarbia and respiratory failure. Complications of a decreased level of consciousness such as

aspiration leading to an aspiration pneumonitis may result. Finally, some ingestions may lead directly to non-cardiogenic pulmonary edema. If you suspect poisoning, contact your local poison control center.

Supporting the airway and ventilation is the main therapeutic intervention for managing respiratory distress or failure caused by poisoning or drug overdose. In addition to the initial interventions in Table 28, specific interventions for disordered control of breathing due to poisoning or drug overdose may include

- Contacting a poison control center (In the United States 1-800-222-1222)
- Suctioning the airway in case of vomiting
- Administering an antidote as indicated. Administer naloxone for opioid overdose (IM and intranasal preparations available) in addition to standard respiratory support for patients with known or suspected opioid overdose who have respiratory arrest but with a definite pulse.
- Performing diagnostic assessments as indicated (eg, ABG, ECG, chest x-ray, electrolytes, glucose, drug screen)
- Potentially preparing for transfer when in a rural or nonchildren's hospital

Suspected Opioid Poisoning

For suspected opioid poisoning, the initial actions remain the same as for any unresponsive infant or child. The important difference is patients with suspected opioid overdose should also be treated with an opioid antagonist (eg, naloxone) along with the AED (if available). Determine if the child is stable or unstable. Looking for cardiac compromise is an important step in determining treatment recommendations.

Breathing

Breathing continues to be a top priority for the unresponsive infant or child. Preventing deterioration with normal breathing and supporting breathing for abnormal breathing are imperative. In either instance, an opioid antagonist (eg, naloxone) needs to be administered. Children with opioid-associated emergencies may not be able to maintain an open airway or breathe normally. Even those who receive an opioid antagonist such as naloxone may develop respiratory problems that can lead to cardiac arrest. If at any time the child's breathing deteriorates, check for a pulse and begin chest compressions if indicated.

- If an opioid antagonist such as naloxone is available and you suspect an opioid overdose, it is reasonable to give it according to package directions and per local protocol. High-quality CPR should take priority over giving naloxone. Repeat doses of naloxone may be needed.

Summary: Managing Respiratory Emergencies

This section summarizes general management of respiratory emergencies and specific management by etiology. This summary does not include all respiratory emergencies but provides key management strategies for a limited number of diseases.

General Management for Respiratory Emergencies

- Airway positioning
- Suction as needed
- Oxygen
- Pulse oximetry
- ECG monitor as indicated
- BLS as indicated

Specific Management for Selected Conditions

Upper Airway Obstruction

- Croup
    - –Nebulized epinephrine
    - –Corticosteroids
- Anaphylaxis
    - –IM epinephrine (or autoinjector)
    - –Albuterol
    - –Antihistamines
    - –Corticosteroids
- Aspiration of foreign body
    - –Allow position of comfort
    - –Specialty consultation

Lower Airway Obstruction

- Bronchiolitis
    - –Nasal suctioning
    - –Consider bronchodilator trial
- Asthma
    - –Albuterol ± ipratropium

- –Corticosteroids
- –Magnesium sulfate
- –IM epinephrine (if severe)
- –Terbutaline

Lung Tissue Disease

- Pneumonia/pneumonitis (infectious, chemical, aspiration)
  - –Albuterol
  - –Antibiotics (as indicated)
  - –Consider noninvasive or invasive ventilatory support with PEEP
- Pulmonary edema (cardiogenic or noncardiogenic [ARDS])
  - –Consider noninvasive or invasive ventilatory support with PEEP
  - –Consider vasoactive support
  - –Consider diuretic

Disordered Control of Breathing

- Increased ICP
  - –Avoid hypoxemia
  - –Avoid hypercarbia
  - –Avoid hyperthermia
  - –Avoid hypotension

Poisoning/Overdose

- Noninvasive or invasive ventilatory support as needed
- Antidote (if available)
- Contact poison control

Neuromuscular Disease

Consider noninvasive or invasive ventilatory support.

Resources for Managing Respiratory Emergencies

Suction Devices

Suction devices can be either portable or wall-mounted units.

- Although portable suction devices are easy to transport, they may not have adequate suction power. A suction force of −80 to −120 mm Hg is generally needed to remove airway secretions.
- Bulb or syringe suction devices are simple to use and require no outside vacuum source. These devices may be inadequate in larger patients or when secretions are thick or copious.
- Wall-mounted suction units can provide a high suction force of more than −300 mm Hg.

Suction devices used in children should have adjustable suction regulators so that you can use sufficient suction force and minimize tissue trauma. Large-bore, noncollapsible suction tubing should always be joined to the suction unit. Semirigid pharyngeal tips (tonsil suction tips) and appropriate sizes of catheters should be available.

Indications

You may need to suction secretions, blood, or vomit from the oropharynx, nasopharynx, or trachea to achieve or maintain an open/patent airway.

Complications

Complications of suctioning can include

- Hypoxia
- Vagal stimulation resulting in bradycardia
- Gagging and vomiting
- Soft tissue injury
- Agitation

Soft vs Rigid Catheters

Both soft, flexible and rigid suctioning catheters are available. Use a soft, flexible suction catheter for aspirating thin secretions from the oropharynx and nasopharynx and for suctioning an advanced airway (eg, ET tube). Use a rigid wide-bore suction cannula (“tonsil tip”) for suctioning the oropharynx, particularly if thick secretions, vomit, or blood are present.

Catheter Sizes

For a guide to selecting the appropriate-sized suction catheter, use a color-coded length-based resuscitation tape or other reference.

Oropharyngeal Suctioning Procedure

Follow these steps to suction the oropharynx:

1. Gently insert the distal end of the suction catheter or device into the oropharynx over the tongue. Guide it into the posterior pharynx (back of the throat).
2. Apply suction by covering the catheter side opening. At the same time, withdraw the catheter with a rotating or twisting motion.
3. Try to limit suction attempts to 10 seconds or less to help reduce the risk of hypoxemia (low oxygen saturation). You may give short periods of 100% oxygen immediately before and after each suctioning attempt.

Note: Suction attempts may need to be longer than 10 seconds if the airway is obstructed (eg, by blood). You cannot adequately oxygenate or ventilate unless the airway is open and clear.

Critical Concepts

Monitoring During Suctioning

Monitor the child's heart rate, oxygen saturation, and clinical appearance during suctioning. In general, if bradycardia develops or clinical appearance deteriorates, interrupt suctioning. Give high-flow oxygen and bag-mask ventilation, if needed, until the heart rate and clinical appearance return to normal.

Oropharyngeal Airway

Description

Usually made of plastic, the OPA consists of a flange, a short bite-block segment, and a curved body. It is shaped to provide an air channel and a passage for a suction catheter to the pharynx. The OPA fits over the tongue to prevent it and other soft structures of the throat from obstructing the airway. It is not intended to be used long-term as a bite-block device in agitated patients.

Indications

An OPA may relieve upper airway obstruction caused by the tongue. Using a correct-sized OPA will not damage laryngeal structures. It may be used in the unconscious child with no gag reflex if procedures to open the airway (eg, head tilt–chin lift, jaw thrust) fail to provide and maintain a clear, unobstructed airway. However, an OPA should not be used in a conscious or semiconscious child because it may stimulate gagging and vomiting. Before using an OPA, check if the child has a gag reflex. If so, do not use an OPA.

Complications

Choose the correct-sized OPA. A properly sized OPA relieves airway obstruction caused by the tongue without damaging the larynx. The tip of the OPA should end just at the angle of the jaw, so that once inserted, it will align with the glottis opening (Figure 32A). If the OPA is too large, it can block the airway or cause trauma to the laryngeal structures (Figure 32B). If the OPA is too small or is inserted improperly, it can push the tongue into the back of the throat and contribute to airway obstruction (Figure 32C).

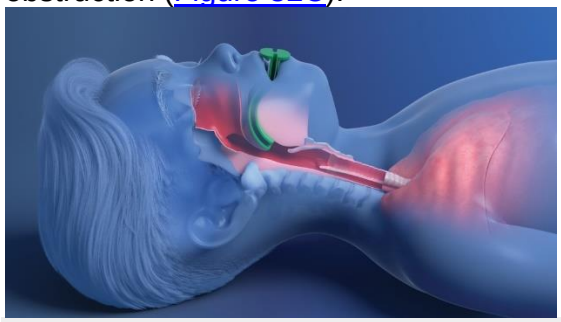


Figure 32A. Selecting an OPA. A, Properly sized OPA.

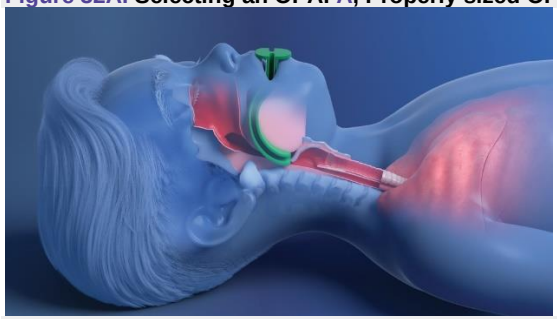


Figure 32B. If the OPA is too large, it will obstruct the airway by pushing the epiglottis down.

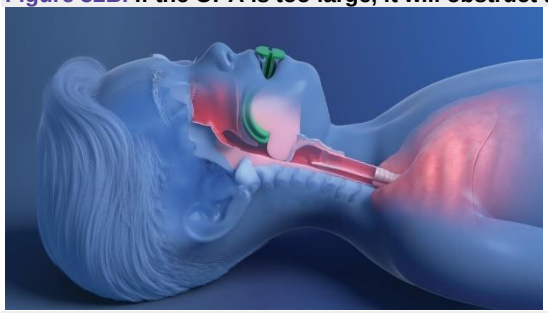


Figure 32C. If the OPA is too small, it will worsen airway obstruction by pushing the tongue into the back of the throat.

Modified from Coté CJ, Todres ID. The pediatric airway. In: Coté CJ, Ryan JF, Todres ID, Goudsouzian NG, eds. *A Practice of Anesthesia for Infants and Children*. 2nd ed. WB Saunders Co; 1993:55-83, copyright Elsevier.

Airway Selection and Insertion Procedure

OPA sizes range from 4 to 10 cm in length (Guedel sizes 000-4). Follow these steps to choose the correct-sized OPA and insert it into the airway:

1. Place the OPA against the side of the child's face. The tip of the OPA should extend from the corner of the mouth to the angle of the jaw (Figure 32A).
2. Gently insert the OPA directly into the oropharynx. The use of a tongue blade to depress the tongue may be helpful.
3. After inserting an OPA, monitor the child. Keep the head and jaw positioned properly to maintain an open/patent airway. Suction the airway as needed.

ET Intubation

Potential Indications

Consider ET intubation if the child is unable to maintain effective airway, oxygenation, or ventilation despite initial intervention.

Preparing for ET Intubation

In the Airway Management Skills Station, you will need to know the equipment needed for ET intubation (Table 33).

Table 33 Pre-event Equipment Checklist for Endotracheal Intubation

Check	Equipment
	Universal precautions (gloves, masks, eye protection)
	Cardiac monitor, pulse oximeter, and blood pressure monitoring device
	End-tidal CO_2 detector or exhaled CO_2 capnography (or esophageal detector device, if appropriate)
	Intravenous and intraosseous infusion equipment
	Oxygen supply, bag mask (appropriate size)

	Oral/tracheal suction equipment (appropriate size); confirm that it is working
	Oral and nasopharyngeal airways (appropriate size)
	Endotracheal tubes with stylets (all sizes) and sizes 0.5 mm (internal diameter) above and below anticipated size for patient; it is reasonable to choose cuffed ET tubes over uncuffed ET tubes
	Laryngoscope (curved and straight blades) and/or video laryngoscope; backup laryngoscope available
	3-, 5-, and 10-mL syringes to test inflate endotracheal tube balloon
	Adhesive/cloth tape or commercial endotracheal tube holder to secure tube
	Towel or pad to align airway by placing under head or torso
	Specialty equipment as needed for difficult airway management or anticipated complications (supraglottic, transtracheal, and/or cricothyrotomy)

Sudden Deterioration in an Intubated Patient (DOPE Mnemonic)

Sudden deterioration in an intubated patient may be caused by one of several complications. Use the mnemonic DOPE to help remember these:

- Displacement of the tube/distention of the abdomen: The tube may be displaced out of the trachea or advanced into the right or left main bronchus. If the abdomen becomes sufficiently distended, it can impair the ability of the lungs to fill and may require decompression with a nasogastric/orogastric tube or opening up a G-tube to allow for decompression.
- Obstruction of the tube: Obstruction may be caused by secretions, blood, pus, a foreign body, or kinking of the tube.
- Pneumothorax/PEEP requirement: Simple pneumothorax usually results in a sudden deterioration in oxygenation (reflected by a sudden decrease in SpO_2) and decreased chest expansion and breath sounds on the involved side. Tension pneumothorax may result in the above plus evidence of hypotension and decreased cardiac output. The trachea is usually shifted away from the involved side (this is a late sign).
- Equipment failure: Equipment may fail for several reasons, such as disconnection of the O_2 supply from the ventilation system, leak in the ventilator circuit, failure of power supply to the ventilator, and malfunction of valves in the bag or circuit.

Evaluating the Patient's Status

If an intubated patient's condition deteriorates, first support oxygenation and ventilation. While attempting this, rapidly assess the child and attempt to determine and correct the cause of deterioration. If the child is being mechanically ventilated, hand ventilate with a bag while you assess the patient's airway, ventilation, and oxygenation as follows:

- Observe for chest rise and symmetry of chest movement.
- Auscultate over both sides of the anterior chest and at the midaxillary line and over the stomach. Listen carefully over the lateral lung fields for asymmetry in breath sounds or abnormal sounds such as wheezing.
- Check monitors (eg, pulse oximetry; if available, capnography).
- Check heart rate.
- Suction the ET tube or SGA if you suspect obstruction with secretions.
- Use sedatives or analgesics, with or without neuromuscular blockers, if needed to reduce the child's agitation and control ventilation. Administer these agents only after you rule out a correctable cause of the acute distress and are sure that you can provide positive-pressure ventilation. Caution should be used with neuromuscular blockades as there is significant risk of losing an effective airway if the advanced airway is not properly placed and secured.

Your initial assessment will determine the urgency of the required response. If you cannot verify that the ET tube is in the airway, direct visualization of the tube passing through the glottis is advised. If the child's condition is deteriorating and you strongly suspect that the tube is no longer in the trachea, you may need to remove it and ventilate with a bag-mask device.

Patient Agitation

Once you confirm the ET tube position and openness/patency and rule out failure of ventilation equipment and pneumothorax, evaluate oxygenation and perfusion. If oxygenation and perfusion are adequate or unchanged, it is possible that agitation, pain, or excessive movement is interfering with adequate ventilation.

If so, try one or more of the following:

- Analgesia (eg, fentanyl, morphine) to control pain
- Sedation (eg, lorazepam, midazolam) for anxiety or agitation
- Neuromuscular blocking agents and analgesia or sedation to optimize ventilation and minimize the risk of barotrauma and unintentional tube displacement

Continuous capnography is the gold standard during mechanical ventilation as an adjunct to clinical assessment. A sudden decrease in exhaled CO_2 can indicate ET or SGA tube displacement or cardiac arrest, while a gradual decrease in exhaled CO_2 may indicate development of ET tube obstruction or decreasing cardiac output. In addition, capnography may help to

detect hypoventilation or hyperventilation, and is particularly useful during transport and diagnostic procedures. Use a colorimetric detector or capnography during intrahospital and interhospital transport.

Oxygen Delivery Systems

Indications for Oxygen

For children with respiratory distress or shock, oxygen uptake by the lungs and oxygen delivery to the tissues typically reduce. At the same time, tissue demand for oxygen may increase. Give high-flow oxygen to all seriously ill or injured children with severe respiratory distress, shock, or changes in mental status. As soon as possible, add humidification to the oxygen delivery system to help prevent airway dryness.

Giving Oxygen to a Conscious Child

Because agitation can increase oxygen demand and respiratory distress, balance the need to improve oxygen delivery against possible agitation that may result from applying an oxygen delivery device to an alert child in respiratory distress. If a child is agitated by one method of oxygen delivery, try an alternative technique. For example, if the child is upset by an oxygen mask, try directing a “blow-by” stream of humidified oxygen toward the child’s mouth and nose. It may be helpful to have a person familiar to the child, such as a parent, introduce the oxygen delivery equipment.

When giving oxygen to an alert child in respiratory distress, allow the child to remain in a position of comfort to minimize respiratory effort and help keep the airway as open as possible. For infants and young children, the best position might be in the arms of the parent or caregiver.

Giving Oxygen to a Child With a Decreased Level of Consciousness

If a child has a decreased level of consciousness, the airway may become obstructed by a combination of the following:

- Flexing the neck
- Relaxing the jaw
- Displacing the tongue against the back of the throat
- Accumulating secretions

If the child is unconscious with no cough or gag reflex, open the airway and insert an OPA. Use the head tilt–chin lift maneuver or a jaw thrust to open the airway.

If you don’t suspect trauma and the child is breathing normally, roll the child onto their side in a neutral position. Place the child on their side only if you do not need to perform any other interventions.

Suction the oropharynx and nasopharynx to clear secretions, mucus, or blood if needed. Once the airway is open and clear, you can give oxygen by a variety of oxygen delivery systems.

Types of Oxygen Delivery Systems

For a spontaneously breathing child who needs supplemental oxygen, you need to know which oxygen delivery system to use—either low flow or high flow. Consider the child's clinical status and the desired concentration of inspired oxygen when choosing the appropriate system. Low-flow oxygen delivery systems include a nasal cannula and simple oxygen mask, while high-flow oxygen delivery systems include a nonrebreathing mask with a reservoir and a high-flow nasal cannula.

Several factors determine the concentration of inspired oxygen. These include oxygen flow into the device, the child's inspiratory flow, and how tightly the device fits against the child's face.

Low-Flow Oxygen Delivery Systems

A low-flow oxygen delivery system delivers air through a nasal cannula or a simple mask that does not fit tightly against the child's face. The oxygen flow into the delivery device is less than the child's inspiratory flow rate. When the child inhales, the child inspires some room air along with the oxygen provided by the device. As a result, the oxygen from the device mixes with room air, so the child receives a variable oxygen concentration. The higher the oxygen flow provided, the higher the inspired oxygen concentration.

Low-flow systems generally provide an inspired oxygen concentration of about 22% to 60% and are used for relatively stable children who require a relatively low inspired oxygen concentration, such as when a child is not in severe respiratory distress or shock. The nasal cannula and simple oxygen mask are examples of low-flow oxygen delivery systems.

Nasal Cannula

The nasal cannula is typically a low-flow oxygen delivery device that delivers an inspired oxygen concentration of 22% to 60%. The appropriate oxygen flow rate for the nasal cannula is 0.25 to 4 L/min.

The nasal cannula is suitable for infants and children who require only low concentrations of supplemental oxygen. Note that in small infants, a nasal cannula may deliver a high inspired oxygen concentration (refer, also, to High-Flow Nasal Cannula later in this Part). The oxygen flow rate alone cannot reliably determine the inspired oxygen concentration delivered via nasal cannula. Other factors have influence, such as

- The child's size
- Inspiratory flow rate
- Volume of inspired air

- Nasopharyngeal and oropharyngeal volume
- Nasal resistance (eg, oxygen delivery is compromised if nares are obstructed)
- Oropharyngeal resistance

Be sure that the cannula is big enough to comfortably fit inside the nare.

Simple Oxygen Mask

The simple oxygen mask is a low-flow device that delivers an inspired oxygen concentration of 35% to 60%. The appropriate flow rate for the simple oxygen mask is 6 to 10 L/min.

The simple oxygen mask cannot deliver an inspired oxygen concentration greater than 60% because room air enters between the mask and the face and through ports in the side of the mask during inspiration. The oxygen concentration delivered to the child is reduced if

- The child's inspiratory flow is high
- The mask does not fit tightly against the face
- The oxygen flow into the mask is low

A minimum oxygen flow rate of 6 L/min is needed to maintain an increased inspired oxygen concentration and prevent rebreathing of exhaled CO_2.

Several types of oxygen masks can deliver humidified oxygen in a wide range of concentrations. The soft vinyl pediatric mask may agitate and upset infants and toddlers, thus increasing oxygen demand and potentially resulting in increased respiratory distress. This mask may be used effectively in older children.

High-Flow Oxygen Delivery Systems

High-flow oxygen systems reliably deliver an oxygen concentration of greater than 60%. In a high-flow oxygen delivery system, the oxygen flow rate is high, at least 10 L/min. A nonrebreathing mask is the most common example of a high-flow system; high-flow nasal cannula is another example.

High-flow systems should be used in emergency settings whenever the child has respiratory distress or shock.

Nonrebreathing Mask

The nonrebreathing mask (Figure 33) is a high-flow delivery device. An inspired oxygen concentration of 95% can be achieved with an oxygen flow rate of 10 to 15 L/min and the use of a well-sealed face mask.

Figure 33. Nonrebreathing mask with reservoir.

A nonrebreathing mask consists of a face mask and reservoir bag with the addition of 2 one-way valves:

- A valve in 1 or both exhalation port(s) to prevent room air from entering the mask during inspiration
- A valve placed between the reservoir bag and the mask to prevent the flow of exhaled gas into the reservoir

Adjust the oxygen flow rate into the mask to prevent bag collapse (usually greater than 10 L/min). The bag is filled with oxygen to meet the child's total maximum inspired flow requirements. During inspiration, the child draws 100% oxygen from the reservoir bag and the oxygen inflow. Room air does not enter the mask if the mask is tight-fitting and the delivery system is closed.

High-Flow Nasal Cannula

The nasal cannula can also be used as a high-flow delivery device by adjusting the oxygen flow rate and adding heat and humidity for comfort. Flow rates vary from 4 L in infants to up to 40 L or more in adolescents. The flow can be titrated to adjust FIO_2, provide a low level of pressure, and improve dead space washout. Together, these improve respiratory efficiency and can improve work of breathing. The cannulas come in multiple sizes. Choose a size with a goal of not occluding more than 50% of the nare.

High-flow nasal cannula systems deliver a combination of both room air and oxygen which can be titrated from 21% to 100%. With these systems, the oxygen concentration is titrated based on the patient's needs and oxyhemoglobin saturations.

Nebulizer

Components

There are a variety of nebulizers. Most nebulizers have the following components:

- Nebulizer reservoir
- Nebulizer cap
- T-piece
- Spacer
- Handheld mouthpiece or face mask
- Plastic oxygen tubing
- Oxygen source or compressed air

Older children may use the handheld mouthpiece instead of the face mask.

Steps for Using a Nebulizer With a Handheld Mouthpiece

General steps for using a nebulizer with a handheld mouthpiece (Figure 34) include the following:

1. Unscrew the cap of the nebulizer reservoir, add medication (eg, albuterol) to the nebulizer reservoir, and reattach the cap.
2. Connect the bottom of the T-piece to the top of the nebulizer reservoir, and then connect the spacer to one end of the T-piece and the mouthpiece to the other end.
3. Connect plastic tubing between the bottom of the nebulizer bottle and the pressurized oxygen/gas source.
4. Set the gas flow at 6 to 8 L/min for jet nebulizers to create an aerosol using medical gases. Use manufacturer recommended flow rates for nebulizers.
5. Hold the nebulizer reservoir upright during delivery of the medication through the mouthpiece. Place the mouthpiece in the child's mouth and show them how to hold it. Tell the child, "Take long, slow, deep breaths through your mouth." Continue the treatment (about 8-10 minutes) until the nebulizer reservoir is empty and no mist flows from the T-piece.

Figure 34. Child receiving a treatment by nebulizer and handheld mouthpiece.

Steps for Using a Nebulizer With a Face Mask

Follow these steps to use a nebulizer with a face mask:

1. Unscrew the cap of the nebulizer reservoir, add medication (eg, albuterol) to the nebulizer reservoir, and reattach the cap.
2. Attach the face mask to the top of the nebulizer reservoir.
3. Connect plastic tubing between the bottom of the nebulizer reservoir and the pressurized oxygen/gas source.
4. Set the gas flow at 6 to 8 L/min for jet nebulizers to create an aerosol using medical gases. Use manufacturer recommended flow rates for nebulizers.
5. Hold the nebulizer reservoir upright during delivery of the medication through a face mask. Place the face mask over the child's face so that it covers both the nose and mouth; press the mask to the face to ensure a light seal. Tell the child, "Take long, slow, deep breaths through your mouth." Continue the treatment (about 8-10 minutes) until the nebulizer bottle is empty and no mist flows from the mask.

Metered-Dose Inhaler

Using an MDI With a Spacer Device

Follow these steps to use an MDI with a spacer device (with and without a face mask):

1. Remove the cap from the spacer, and insert the mouthpiece of the MDI into the rubber-sealed end of the spacer (Figure 35). Once assembled, shake the MDI and the spacer vigorously. Place the mouthpiece of the spacer device into the child's mouth.

or

If you are using a spacer device with a face mask, place the mask over the child's face so that it covers both the nose and the mouth (Figure 36). Press the mask to the face to ensure a tight seal.

2. While the child is exhaling, activate the MDI by pressing down on the inhaler to release the medication into the spacer device (or have the child do so). Tell the child to take 3 to 5 slow, deep breaths through the mouthpiece and hold the last breath for 10 seconds.

   or

   If you are using a spacer device with a face mask, depress the inhaler and allow the child to breathe normally through the mask for 3 to 5 breaths after release of medication.

Figure 35. Child self-administering a treatment by MDI with a spacer device.

Figure 36. Child receiving a treatment by MDI with a mask spacer device.

Part 7
Recognizing Shock

When shock is present, rapidly identifying and promptly intervening are critical to improving outcomes. If left untreated, shock can quickly progress to cardiopulmonary failure followed by cardiac arrest. Once an infant or child develops cardiac arrest secondary to inadequately treated shock, the outcome is poor.

This Part discusses the following:

- Pathophysiology of shock
- Effect of different types of shock on blood pressure
- Systolic blood pressure as a method of categorizing the severity of shock (ie, compensated or hypotensive)
- Etiology and signs of the 4 most common types of shock
- Systematic approach to evaluating the cardiovascular system

Your evaluation will help you identify the child's shock based on type and severity. These clinical findings will direct your interventions, as discussed in Part 8: Managing Shock. The earlier you recognize shock, establish priorities, and start therapy, the better the child's chance for a good outcome.

Learning Objective

After completing this Part, you should be able to differentiate between compensated and hypotensive shock.

During the course, you will need to identify different types and severities of shock. Evaluating patient information will help you determine effective interventions.

Defining Shock

Shock is defined as a physiologic state characterized by inadequate tissue perfusion to meet metabolic demand and tissue oxygenation. It is often, but not always, characterized by inadequate peripheral and end-organ perfusion. In children, most shock is characterized by low cardiac output; however, in some types of shock (eg, that caused by sepsis or anaphylaxis), cardiac output may be high. All types of shock can result in impaired function of vital organs such as the brain (decreased level of consciousness) and kidneys (low urine output, ineffective filtering).

Critical Concepts
Shock and Blood Pressure

The definition of shock does not require the presence of hypotension. Shock can be present with a normal, increased, or decreased systolic blood pressure.

Shock can result from

- Inadequate blood volume or oxygen-carrying capacity (hypovolemic shock, including hemorrhagic shock)
- Inappropriate distribution of blood volume and flow (distributive shock)
- Impaired cardiac contractility (cardiogenic shock)
- Obstructed blood flow (obstructive shock)

Conditions such as fever, infection, injury, respiratory distress, and pain may contribute to shock by increasing tissue demand for O_2 and nutrients. Whether due to inadequate supply, increased demand, or a combination of both, O_2 and nutrient delivery to the tissues is inadequate relative to metabolic needs. Inadequate tissue perfusion can lead to tissue hypoxia, anaerobic metabolism, accumulating lactic acid and CO_2, irreversible cell damage, and, ultimately, organ damage. Death may then result rapidly from cardiovascular collapse or more slowly from multiorgan failure.

Critical Concepts
Goal for Treating Shock

When treating shock, the goal is to improve systemic perfusion and O_2 delivery to help prevent end-organ injury and stop the progression to cardiopulmonary failure and cardiac arrest.

Pathophysiology of Shock

The major function of the cardiopulmonary system is to deliver O_2 to body tissues and remove metabolic by-products of cellular metabolism (primarily CO_2). When O_2 delivery cannot adequately meet tissue demand, cells use anaerobic metabolism to produce energy, but this generates lactic acid as a by-product. Anaerobic metabolism can only maintain limited cell function. Unless O_2 delivery is restored, organ dysfunction or failure will result.

Critical Concepts
Central Venous O_2 Saturation ($ScvO_2$) and Cardiac Output

In healthy children with normal metabolic demand, the arterial blood contains more O_2 than the tissues need. If demand increases and/or O_2 delivery decreases, the tissues will extract a greater percent of the O_2 delivered. This results in reduced O_2 saturation in the venous blood returning to the heart. The $ScvO_2$, therefore, can be used to assess the balance between O_2 delivery and demand. If metabolic demand and O_2 content are unchanged, a decreased $ScvO_2$ indicates a fall in cardiac output and, therefore, a fall in O_2 delivered to the tissues. In addition, greater extraction of O_2 occurs as the result of reduced delivery.

Components of Tissue Oxygen Delivery

Adequate tissue O_2 delivery (Figure 37) depends on

- Sufficient O_2 content in the blood
- Sufficient oxygen carrying capacity (ie, hemoglobin in the blood)
- Adequate blood flow to the tissues (cardiac output)
- Appropriate distribution of blood flow to the tissues

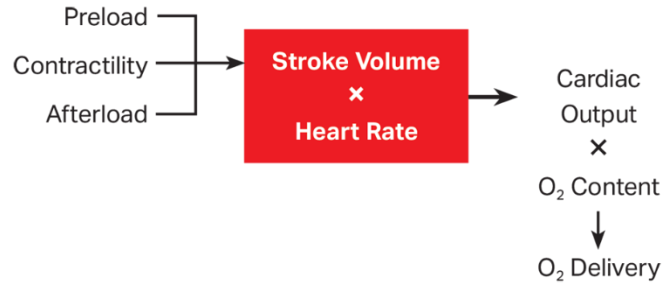


Figure 37. Factors influencing O_2 delivery.

The hemoglobin concentration and percent of the hemoglobin that is saturated with O_2 (ie, the arterial oxygen saturation, or SaO_2) primarily determines blood O_2 content. Plasma carries a small amount of dissolved oxygen.

Critical Concepts

Compensatory Mechanisms for Hypoxemia

Tissue hypoxia is present when a region of the body or an organ is deprived of adequate O_2 supply. Low O_2 saturation (hypoxemia) alone does not necessarily result in tissue hypoxia. Oxygen delivery to the tissues is the product of the arterial O_2 content (determined by the oxygen bound to hemoglobin plus dissolved O_2) and the volume of blood pumped per minute (cardiac output). O_2 delivery may be normal despite hypoxemia if cardiac output increases commensurate with the decrease in O_2 content.

When hypoxemia is chronic (eg, unrepaired cyanotic heart disease), hemoglobin concentration increases (polycythemia). The increased hemoglobin concentration will increase the O_2-carrying capacity of the blood and help maintain arterial O_2 content at near-normal concentrations, despite hemoglobin oxygen saturation being low.

If cardiac output decreases or hypoxemia worsens, these compensatory mechanisms may not be sufficient to maintain tissue O_2 delivery, and tissue hypoxia will likely develop.

Cardiac output and vascular resistance determine adequate blood flow to the tissues. Cardiac output (the volume of blood pumped by the heart per minute) is the product of stroke volume (the volume of blood pumped by the ventricles with each contraction) and heart rate (number of times the ventricles contract per minute):

Cardiac Output = Stroke Volume × Heart Rate

According to this formula, if the heart rate decreases, stroke volume must increase commensurately to maintain the cardiac output. Cardiac output can increase either by an increase in heart rate, in stroke volume, or both. However, the increased cardiac output produced by increasing heart rate does have its limit. If the heart rate is too fast, as can happen with extreme tachyarrhythmias (eg, SVT), stroke volume can fall because there is inadequate time to fill the heart (ie, the diastolic phase is too short). Some arrhythmias, such as complete heart block or junctional tachycardia, can be associated with a decrease in ventricular filling because atrial contraction (which normally contributes about 25% of ventricular filling) does not precede ventricular contraction.

Critical Concepts

Infant Cardiac Output Dependent on Heart Rate

Infants have a very small stroke volume with limited ability to increase it, therefore making them dependent on an adequate heart rate to maintain or increase cardiac output. Adolescents and adults are better able to increase stroke volume, and cardiac output is less dependent on the heart rate. Tachycardia can be an early sign of shock, particularly in infants.

Appropriate blood flow distribution is determined by the size of the blood vessels supplying a specific organ. This property is known as *vascular resistance*. If the vessel is large, vascular resistance is low; if the vessel is small, vascular resistance is high. Vascular resistance is adjusted by tissues to locally regulate blood flow to meet metabolic demands. Abnormally increased resistance (vasoconstriction) or decreased resistance (vasodilation) can interfere with blood flow distribution, even if cardiac output is adequate.

Stroke Volume

Stroke volume, the amount of blood ejected by the ventricles with each contraction, is determined by the following 3 factors:

- Preload: volume of blood present in the ventricle before contraction
- Contractility: strength of contraction
- Afterload: resistance against which the ventricle is ejecting

Inadequate preload is the most common cause of low stroke volume and, therefore, low cardiac output. Several conditions (eg, hemorrhage, severe dehydration, vasodilation) can cause inadequate preload, which results in hypovolemic shock.

You may assess preload indirectly by measuring the central venous pressure, but the relationship between central venous pressure and preload is complex. Preload to the ventricles is the volume of blood in the ventricles that stretches the ventricular fibers before a contraction (ie, the end-diastolic volume). However, the measurements used clinically measure pressure, not volume. The relationship between changes in ventricular volume and changes in ventricular end-diastolic pressure is affected by ventricular compliance (or, conversely, ventricular stiffness).

In general, assess the right ventricle preload by measuring the central venous pressure, measured in the superior vena cava or right atrium. Generally, increased central venous pressure corresponds to increased right ventricular end-diastolic volume and preload. However, if there is increased pressure around the right atrium and ventricle from a tension pneumothorax or pericardial tamponade, or if the right ventricle is stiff (ie, not compliant) because of a congenital heart defect or pulmonary hypertension, ventricular end-diastolic pressure increases despite no increase (or even a decrease) in right ventricular end-diastolic volume and preload.

Preload is not the same as total blood volume. At steady state, most of the blood (about 70%) is in the veins. If the veins dilate while the total blood volume is maintained, an inadequate amount of blood may return to the heart (reduced preload) because of a drop in the driving pressure. This is part of the problem with sepsis: there is often severe venodilation, so preload to the heart may be inadequate. In addition, the maldistribution of blood flow results in some tissue hypoxia.

Poor contractility (also referred to as *myocardial dysfunction*) impairs stroke volume and cardiac output, potentially leading to cardiogenic shock. Poor contractility can be due to an intrinsic problem with pump function or an acquired abnormality, such as an inflamed heart muscle (ie, myocarditis). Poor contractility also can occur from metabolic problems, such as hypoglycemia, or from toxic ingestions (eg, calcium channel blockers).

Increased afterload is an uncommon primary cause of low stroke volume and impaired cardiac output in children. When cardiac output reduces, the body responds with vasoconstriction to maintain blood pressure and blood flow to vital organs. Paradoxically, vasoconstriction increases impedance to ventricular ejection and further decreases stroke volume and cardiac output. Certain conditions, such as severe pulmonary or systemic hypertension or congenital abnormalities of the aorta, can increase afterload so significantly that cardiogenic shock results.

Compensatory Mechanisms

As shock develops, compensatory mechanisms attempt to maintain O_2 delivery to vital organs. These mechanisms include

- Tachycardia
- Increased SVR (vasoconstriction)
- Increased strength of cardiac contraction (contractility)
- Increase in venous smooth muscle tone

The body's first action to maintain cardiac output is to increase heart rate (tachycardia), which can increase cardiac output to a limited degree.

When O_2 delivery to the tissues is compromised, blood flow is redirected or shunted from nonvital organs and tissues (eg, skin, skeletal muscles, gut, kidneys) to vital organs (eg, brain, heart). This redirection occurs by a selective increase in SVR (vasoconstriction).

Clinically, this results in reduced peripheral perfusion (ie, delayed capillary refill, cool extremities, weaker peripheral pulses) and reduced perfusion to the gut (vomiting, feeding intolerance) and kidneys (decreased urine volume).

Another compensatory mechanism to maintain stroke volume and cardiac output is increased strength of cardiac contractions (contractility) with more complete emptying of the ventricles. Stroke volume may also be supported by increased venous smooth muscle tone, improving venous return to the heart and preload.

Effect on Blood Pressure

Blood pressure is determined by cardiac output and SVR. As cardiac output decreases, blood pressure can be maintained by an increase in SVR. In children with shock, this compensatory mechanism can be so effective that systolic blood pressure may initially remain normal or even slightly elevated. Pulse pressure, the difference between the systolic and diastolic blood pressure, often narrows because increased SVR raises the diastolic pressure. In contrast, if SVR is low (as in sepsis), diastolic blood pressure decreases and pulse pressure widens.

If cardiac output is inadequate, tissue perfusion is compromised, even if blood pressure is normal. Signs of poor tissue perfusion—including cool, pale skin, delayed capillary refill, lactic acidosis, end-organ dysfunction, and weak peripheral or central pulses (late finding)—will be present even if blood pressure is normal.

When SVR cannot increase further, blood pressure begins to decline. Then, O_2 delivery to vital organs is severely compromised. Clinical signs include metabolic acidosis and evidence of end-organ dysfunction (eg, impaired mental status,

decreased urine output). Ultimately O_2 delivery to the myocardium becomes inadequate, causing myocardial dysfunction, decreased stroke volume, and hypotension. These may rapidly lead to cardiovascular collapse, cardiac arrest, and irreversible end-organ injury.

Identifying Shock by Severity (Compensated vs Hypotensive)

Shock severity is frequently characterized by its effect on systolic blood pressure. Shock is described as compensated if compensatory mechanisms can maintain a systolic blood pressure within a normal range (ie, above the fifth percentile systolic blood pressure for age). When compensatory mechanisms fail and systolic blood pressure declines, shock is classified as hypotensive (previously referred to as *decompensated*). It's important to remember that shock may be present even if the child's blood pressure is normal.

You can easily identify hypotensive shock by measuring blood pressure, while you may find it more difficult to diagnose compensated shock. Shock can range from compensated to hypotensive, with its manifestations affected by the type of shock and the child's compensatory responses. Use blood pressure to determine shock severity; however, children with both compensated and hypotensive shock are at high risk for deterioration. The child with low cardiac output (ie, hypovolemic shock) but normal mean blood pressure associated with severe vasoconstriction may have more end-organ compromise than the child with normal or increased cardiac output (ie, septic shock) and low diastolic blood pressure.

Because blood pressure is one method of categorizing shock severity, it is important to recognize that automated blood pressure devices are accurate only when there is adequate distal perfusion. If you cannot feel peripheral pulses, and the extremities are cool and poorly perfused, automated blood pressure values may not be reliable. Treat the child based on your entire clinical evaluation. If measuring blood pressure is not feasible, clinically evaluate tissue perfusion to guide treatment.

Compensated Shock

Compensated shock refers to a clinical state in which clinical signs of inadequate tissue perfusion appear, but the patient's blood pressure is in the normal range. In this stage of shock, the body can maintain blood pressure despite impaired delivery of O_2 and nutrients to the vital organs. Clinical findings include tachycardia, delayed capillary refill, mental status changes, and decreased urine output.

Critical Concepts

Systolic Blood Pressure in Identifying Shock

Compensated shock refers to the child with signs of poor perfusion but a normal systolic blood pressure (ie, with blood pressure compensation). Systolic blood pressure is used by convention and consensus to determine the presence or absence of hypotension with shock. Infants and children with compensated shock may be critically ill despite an adequate systolic blood pressure.

When O_2 delivery is limited, compensatory mechanisms try to maintain normal blood flow to the brain and heart and act as clues to the presence of shock, varying according to the type of shock. Table 34 lists common compensatory mechanisms in shock and the cardiovascular signs associated with these mechanisms.

Common Signs of Shock Resulting From Cardiovascular Compensatory Mechanisms

Compensatory mechanism	Area	Sign
Increased heart rate	Heart	Tachycardia
Increased SVR	Skin	Cold, pale, mottled, diaphoretic
	Peripheral circulation	Delayed capillary refill
	Pulses	Weak peripheral pulses; narrow pulse pressure (increased diastolic blood pressure)
Increased renal and splanchnic vascular resistance (redistribution of blood flow away from these areas)	Kidney	Oliguria (decreased urine output)
	Intestine	Vomiting, ileus
Cerebral autoregulation	Brain	Altered mental status, anxiety/restlessness, disorientation or decreased level of consciousness, or even coma

Signs specific to shock type are discussed in Identifying Shock by Type later in this Part.

Hypotensive Shock

Hypotensive (decompensated) shock can result from many causes and is characterized by evidence of impaired perfusion that will rapidly progress to cardiac arrest if not corrected. Signs include abnormal clinical appearance and evidence of severely impaired perfusion (ie, weak or absent peripheral pulses and weak central pulses, cool extremities, mottled skin, or altered

level of consciousness). Shock represents a continuum of severity, and the presence of signs and symptoms of shock should prompt immediate action rather than waiting for direct measurement of blood pressure to document hypotension.

Hypotension is a late finding in most types of shock and may signal impending cardiac arrest. It can occur early in septic shock because mediators of sepsis produce vasodilation and reduce SVR. In this setting, the child may initially appear to have warm extremities, brisk capillary refill, and full peripheral pulses despite hypotension.

Critical Concepts
Hypotension in Septic Shock

Hypotension formula: In children 1 to 10 years of age, hypotension is present if the systolic blood pressure is less than 70 mm Hg + [child's age in years × 2] mm Hg.

For more information, refer to Table 21. Definition of Hypotension by Systolic Blood Pressure and Age in Part 4.

Systolic blood pressure is a complex combination of the patient's cardiac output, intravascular volume, and SVR. Septic shock is a dynamic process and does not always follow a specific clinical presentation; serial assessments are mandatory because the clinical presentation is likely to evolve over time. Hypotension can be an early or late sign in septic shock and must be immediately recognized and treated aggressively.

Accelerating Process

Shock progression is unpredictable. It may take hours for compensated shock to progress to hypotensive shock but only minutes for hypotensive shock to progress to cardiopulmonary failure and cardiac arrest. This progression is typically called an *accelerating process* (Figure 38).

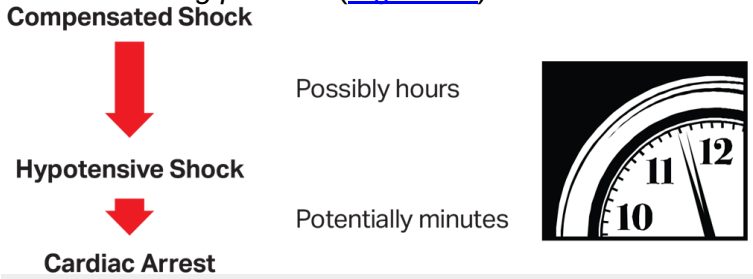


Figure 38. Accelerating process of shock.

Critical Concepts
Halting the Progression

Early recognition and rapid intervention are critical to halting the progression from compensated shock to hypotensive shock to cardiopulmonary failure and cardiac arrest.

These and other clinical manifestations are discussed in greater detail later in this Part.

Identifying Shock by Type

Shock can be categorized into 4 basic types:

- Hypovolemic: resulting from gastroenteritis, burns, hemorrhage, inadequate fluid intake, increased body fluid losses, or osmotic diuresis
- Cardiogenic: resulting from congenital heart disease, myocarditis, cardiomyopathy, or arrhythmia
- Distributive: resulting from sepsis, anaphylaxis, or spinal cord injury (neurogenic)
- Obstructive: resulting from tension pneumothorax, cardiac tamponade, pulmonary embolism, or constriction of the ductus arteriosus in infants with ductal-dependent congenital heart lesions (eg, coarctation, hypoplastic left ventricle)

Although some signs and symptoms of shock fit just one category, there may be instances when the type of shock is not as clear, presenting a mixed picture.

Hypovolemic Shock

Hypovolemic shock refers to a clinical state of reduced intravascular volume. The most common type of shock in pediatric patients, it can be caused by extravascular fluid loss (eg, diarrhea, dehydration) or intravascular volume loss (eg, hemorrhage) and results in decreased preload and cardiac output. Volume loss leading to hypovolemic shock can result from

- Diarrhea
- Vomiting
- Hemorrhage (internal and external)
- Inadequate fluid intake
- Osmotic diuresis (eg, DKA)
- Third-space losses (fluid leak into tissues, eg, in sepsis)
- Large burns

Hypovolemic shock is the result of an absolute deficiency of intravascular blood volume, but, in fact, it typically represents depletion of both intravascular and extravascular fluid volume. As a result, adequate fluid resuscitation often requires administering fluid boluses that exceed the volume of the estimated intravascular deficit.

Tachypnea, a respiratory compensation to maintain acid-base balance, is often present in hypovolemic shock. The respiratory alkalosis that results from hyperventilation partially compensates for the metabolic acidosis that accompanies shock. In a highly monitored setting, SvO_2 lactic acid can be monitored.

Physiology of Hypovolemic Shock

Hypovolemic shock is characterized by decreased preload leading to reduced stroke volume and low cardiac output. Tachycardia, increased SVR, and increased cardiac contractility are the main compensatory mechanisms.

- Preload: decreased
- Contractility: initially normal or increased
- Afterload: increased

Signs of Hypovolemic Shock

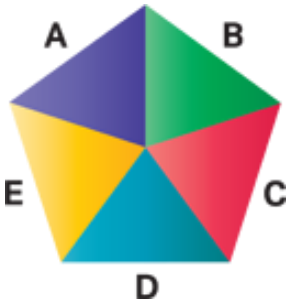


Table 35 outlines typical signs of hypovolemic shock found during the initial assessment and primary assessment.

Table 35.Findings Consistent With Hypovolemic Shock

Primary assessment	Finding
Airway	Typically open/patent unless level of consciousness is significantly impaired

Breathing	Tachypnea without increased effort (quiet tachypnea)
Circulation	• Tachycardia • Adequate systolic blood pressure, narrow pulse pressure, or systolic hypotension with a narrow pulse pressure* • Weak or absent peripheral pulses • Normal or weak central pulses • Delayed capillary refill • Cool to cold, pale, mottled, diaphoretic skin • Dusky/pale distal extremities • Changes in level of consciousness • Oliguria
Disability	Decreasing level of consciousness as shock progresses
Exposure	Extremities often cooler than trunk

*Sign that distinguishes hypovolemic shock from distributive shock.

Although septic, anaphylactic, neurogenic, and other distributive forms of shock are not classified as hypovolemic, they are characterized by relative hypovolemia. The relative hypovolemia results from arterial and venous vasodilation, increased capillary permeability, and plasma loss into the interstitium ("third spacing" or capillary leak).

Distributive Shock

Distributive shock refers to a clinical state characterized by reduced SVR leading to maldistribution of blood volume and blood flow. This group includes

- Septic shock
- Anaphylactic shock
- Neurogenic shock (eg, spinal injury)

In septic and anaphylactic shock, there may also be increased capillary permeability, leading to volume loss from the intravascular space (ie, decreased preload). In neurogenic shock, there is loss of sympathetic tone leading to vasodilation and lack of compensatory mechanisms (ie, tachycardia and peripheral vasoconstriction).

Distributive shock caused by sepsis is typically characterized by reduced SVR resulting in maldistribution of blood flow. The vasodilation and venodilation cause blood pooling in the venous system and a relative hypovolemia. Septic shock also causes

increased capillary permeability, so the vascular space loses plasma. This increases hypovolemia severity. Myocardial contractility may also be depressed in septic shock.

In anaphylaxis, shock is caused by arterial and venous dilation and increased capillary permeability leading to reduced blood pressure. A compensatory increase in cardiac index may be seen, but ultimately, relative hypovolemia will impair blood pressure.

Neurogenic shock is characterized by generalized loss of vascular tone, most often after a high cervical spine injury, leading to severe vasodilation and hypotension. Normally, the sympathetic nervous system increases heart rate in response to hypotension. Children with neurogenic shock may be unable to generate a faster heart rate in response to hypotension and typically have a normal or low heart rate for age. As a result, cardiac output and blood flow to tissues decrease dramatically.

Pathophysiology of Distributive Shock

In distributive shock, cardiac output may be increased, normal, or decreased. Although myocardial dysfunction may be present, stroke volume can be adequate, particularly if there is aggressive volume resuscitation and decreased SVR. Tachycardia and increased ventricular end-diastolic volume (from volume resuscitation) help maintain cardiac output. Tissue perfusion is compromised by maldistribution of blood flow. Some tissue beds (eg, splanchnic circulation, renal circulation) may be inadequately perfused; in other tissues (eg, some skeletal muscle and skin) perfusion may exceed metabolic needs. Hypoxic tissues generate lactic acid, leading to metabolic acidosis. Early in the clinical course, a child with distributive shock may present with decreased SVR and increased blood flow to the skin, producing warm extremities and bounding peripheral pulses, and flash capillary refill (“warm shock”).

The typically high cardiac output and low SVR often observed in distributive shock differ from the low cardiac output and high SVR seen in cardiogenic and obstructive shock. As distributive shock progresses, concomitant hypovolemia and/or myocardial dysfunction produce a decrease in cardiac output. SVR can then increase, resulting in inadequate blood flow to the skin, cold extremities, and weak pulses (“cold shock”). The late phase of distributive shock, therefore, can be similar to the clinical picture of cardiogenic shock.

Distributive shock is most often characterized by many changes in cardiovascular function, including

- Low SVR, which typically leads to a lower diastolic blood pressure, is the etiology of the wide pulse pressure that is commonly, but not always, present in the early phases of distributive shock
- Increased blood flow to some peripheral tissue beds
- Inadequate perfusion of the splanchnic and kidney vascular beds
- Release of inflammatory and other mediators and vasoactive substances

- Volume depletion caused by capillary leak
- Accumulating lactic acid in poorly perfused tissue beds

Critical Concepts

Relative Hypovolemia

Although most types of distributive shock are not typically classified as hypovolemic shock, all are characterized by relative hypovolemia unless adequate fluid resuscitation is provided.

Signs of Distributive Shock

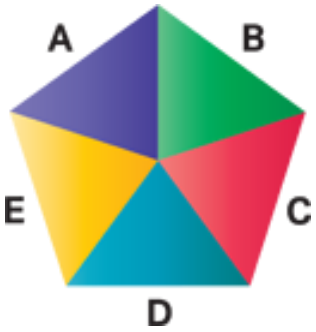


Table 36 outlines typical signs of distributive shock seen during the initial assessment and primary assessment.

Table 36.Findings Consistent With Distributive Shock

Primary assessment	Finding
Airway	Usually open/patent unless level of consciousness is significantly impaired
Breathing	Tachypnea, usually without increased work of breathing (“quiet tachypnea”) unless the child has additional compromise as seen in pneumonia, ARDS, pulmonary edema, or bronchospasm
Circulation	• Tachycardia (predominantly); bradycardia (very rarely) (eg, neurogenic shock typically due to catastrophic high cervical spinal cord injury); bounding (early)* or decreased peripheral pulses (late) • Flash (early)* or delayed capillary refill (late)

	• Warm, flushed skin peripherally (warm extremities, early)* or pale, mottled skin with vasoconstriction (cold extremities, late) • Hypotension with a wide pulse pressure (when warm extremities are present, early)* or hypotension with a narrow pulse pressure (when cold extremities are present, late) or normotension may be present • Changes in level of consciousness • Oliguria
Disability	Changes in level of consciousness
Exposure	• Fever or hypothermia • Extremities may be warm (early) or cool (late) • Petechial or purpuric rash (may be seen in septic shock) or urticarial rash (anaphylaxis)*

*Sign that distinguishes distributive shock from other forms of shock.

Septic Shock

Sepsis represents an important cause of shock in infants and children. *Sepsis* and *septic shock* are terms used to characterize shock caused by an infectious agent or inflammatory stimulus.

Although its presentation can be mixed, septic shock is the most common form of distributive shock and in later stages presents with cooler extremities and poor cardiac output. It is caused by an abnormal host immune response to infectious organisms or their by-products (eg, endotoxin) that lead the small blood vessels to dilate and to leak fluid into the tissues.

Infection is a pathologic process caused by pathogenic or potentially pathogenic microorganisms invading normally sterile tissue, fluid, or a body cavity. You may suspect or prove infection by positive result of a culture, tissue stain, or polymerase chain reaction test. Without these tests, evidence of infection includes positive findings on clinical examination, imaging, or laboratory tests consistent with tissue invasion by a pathogenic organism leading to a host response (eg, white blood cells in normally sterile body fluid, perforated viscus, chest radiograph consistent with pneumonia, petechial or purpuric rash, purpura fulminans).

Pathophysiology of Septic Shock

Septic shock in children typically evolves along a continuum. A systemic inflammatory response may be seen in the early stages and progress to septic shock in the late stages. Because of the wide variability in clinical presentation and progression, this continuum may evolve over days or just a few hours.

- Preload: decreased

- Cardiac output: normal or increased (early); decreased (late)
- Afterload (eg, SVR): variable

Inflammatory Cascade Response to Sepsis

The pathophysiology of the septic cascade includes the following, often referred to as the *systemic inflammatory response*:

- The infectious organism or its by-products (eg, endotoxin) activates the immune system, including neutrophils, monocytes, and macrophages.
- These cells, or their interaction with the infecting organism, stimulate release or activation of inflammatory mediators (cytokines) that perpetuate the inflammatory response.
- Cytokines produce vasodilation and damage to the lining of the blood vessels (endothelium), causing increased capillary permeability.
- Cytokines activate the coagulation cascade and may result in microvascular thrombosis and disseminated intravascular coagulation.
- Specific inflammatory mediators can impair cardiac contractility and cause myocardial dysfunction.

The Challenge of Treating Septic Shock

In septic shock, inadequate perfusion combined with possible microvascular thrombosis leads to ischemia, which is diffuse and patchy so that individual organs have varying levels of hypoxia and ischemia. The variability of perfusion throughout the body is what makes treating sepsis so difficult.

Adrenal Insufficiency in Septic Shock

The adrenal glands are especially prone to microvascular thrombosis and hemorrhage in septic shock. Because adrenal glands produce cortisol, an important hormone in the body's stress response, children with sepsis may develop absolute or relative adrenal insufficiency. Adrenal insufficiency contributes to low SVR and myocardial dysfunction in septic shock.

Signs of Septic Shock

In the early stages, signs of septic shock are often subtle and may be difficult to recognize because peripheral perfusion may initially appear adequate. Because septic shock is triggered by an infection or its by-products, the child may have fever or hypothermia, and the white blood cell (WBC) count may be decreased, normal, or increased.

In addition to the findings listed in the Identifying Shock by Type section, the child with septic shock may have other abnormalities identified by diagnostic assessments. Examples include metabolic acidosis (lactic acidosis), respiratory alkalosis,

leukocytosis (high WBC count), leukopenia (low WBC count), or left shift (increased percent of bands or immature white blood cells). With some types of infections, the child may develop a petechial or purpuric rash.

Central $ScvO_2$ in Septic Shock

In contrast to hypovolemic and cardiogenic shock, $ScvO_2$ may be normal, increased, or decreased in septic shock. There are 2 mechanisms to explain a normal, or even increased, $ScvO_2$, despite the presence of inadequate cardiac output/index. Because of this potential situation in septic shock, lactate is a more useful marker of adequate oxygen delivery than $ScvO_2$.

- Children with low SVR and increased cardiac output will extract less O_2 from the blood because some tissues are receiving more blood flow than they need. Other tissues don't receive enough blood flow, so they don't have the opportunity to extract the O_2.
- Children with sepsis may be unable to use O_2 at the cellular level.

Toxins and inflammatory mediators circulating in sepsis can prevent aerobic metabolism even in the setting of adequate O_2 delivery. As a result, lactic acidosis and end-organ dysfunction can occur even in the setting of normal or increased $ScvO_2$.

Identify and Intervene: Septic Shock

Early recognition and treatment of septic shock are critically important determinants of outcome. Evaluate temperature, heart rate, systemic perfusion, blood pressure, and clinical signs of end-organ function to identify sepsis and septic shock before severe organ dysfunction develops. If you suspect sepsis, and especially if shock develops, provide appropriate volume resuscitation and hemodynamic support (refer to Part 8: Managing Shock for details). Search for and treat the underlying cause. Administer broad spectrum antibiotics early, within one hour of recognition.

Physiology of Anaphylactic Shock

Anaphylactic shock, an acute multisystem response caused by a severe reaction to a medication, vaccine, food, toxin, plant, venom, or other antigen, is characterized by venodilation, arterial vasodilation, and increased capillary permeability. It can occur within seconds to minutes after exposure.

Signs of Anaphylactic Shock

Signs and symptoms may include those listed in Table 37.

Table 37 Findings Consistent With Anaphylactic Shock

Primary assessment	Finding	Resulting from
Airway	Angioedema (swelling of the face, lips, and tongue)*	Swelling of the tongue and tissues related to fluid leak from blood vessels
Breathing	Respiratory distress with stridor, wheezing, or both*	Constriction of airways by inflammatory response
Circulation	Hypotension	Vasodilation, hypovolemia, and diminished cardiac output
Circulation	Tachycardia	Inadequate blood flow to the tissues
Disability	Anxiety and agitation	Low oxygen concentration and reduced brain perfusion
Exposure	Urticaria (hives)*	Histamine release
Other	Nausea and vomiting	Histamine and other mediator release

*Sign that distinguishes anaphylactic shock from other forms of shock.

Angioedema may partially or completely obstruct the upper airway. Hypotension results from vasodilation, hypovolemia, and diminished cardiac output. Relative hypovolemia is caused by the vasodilation, and absolute volume loss is caused by capillary leak.

Neurogenic Shock

Neurogenic shock, also known as *spinal shock*, results from a cervical (neck) or upper thoracic (above T6) injury that disrupts the sympathetic nervous system innervation of blood vessels and of the heart.

Physiology of Neurogenic Shock

The sudden loss of sympathetic nervous system signals to the smooth muscle in the vessel walls results in uncontrolled vasodilation. The same disruption prevents tachycardia developing as a compensatory mechanism.

- Preload: decreased

- Contractility: normal
- Afterload: decreased

Signs of Neurogenic Shock

Primary signs of neurogenic shock are

- Hypotension with a wide pulse pressure
- Normal heart rate or bradycardia
- Hypothermia

Other signs may include increased respiratory rate, diaphragmatic breathing (the use of muscles in the diaphragm rather than the chest wall), and other evidence of a high thoracic or cervical spine injury (ie, motor or sensory deficits). Loss of diaphragmatic innervation may lead to apnea.

You must differentiate neurogenic shock from hypovolemic shock. Hypovolemic shock is typically associated with hypotension, a narrow pulse pressure from compensatory vasoconstriction, and compensatory tachycardia. In neurogenic shock, these compensatory mechanisms are not apparent because sympathetic innervation of the heart and blood vessels is interrupted, and SVR will not increase.

Cardiogenic Shock

Cardiogenic shock refers to reduced cardiac output secondary to abnormal cardiac function or pump failure, resulting in decreased systolic function and cardiac output. Common causes of cardiogenic shock include

- Congenital heart disease
- Myocarditis (inflamed heart muscle)
- Cardiomyopathy (an inherited or acquired abnormality of pumping function)
- Arrhythmias
- Sepsis
- Poisoning or drug toxicity
- Myocardial injury (eg, trauma)

Physiology of Cardiogenic Shock

Marked tachycardia, high SVR, and decreased cardiac output characterize cardiogenic shock. End-diastolic volume within the left and right ventricles increases, resulting in congestion within the pulmonary and systemic venous systems. This pulmonary venous congestion leads to pulmonary edema and increased work of breathing. Typically, intravascular volume is normal or

increased unless a concurrent illness causes hypovolemia (eg, in a child who has viral myocarditis with recent vomiting, fever, poor oral intake).

- Preload: variable
- Contractility: decreased
- Afterload: increased

Cardiogenic shock is often characterized by sequential compensatory and pathologic mechanisms, including

- Increased heart rate and left ventricular afterload, which increases left ventricular work and myocardial O_2 consumption
- Compensatory increase in SVR to redirect blood from peripheral and splanchnic tissues to the heart and brain
- Decreased stroke volume due to decreased myocardial contractility and increased afterload
- Increased venous tone, which increases central venous (right atrial) and pulmonary capillary (left atrial) pressures
- Diminished renal blood flow resulting in fluid retention
- Pulmonary edema resulting from myocardial failure and high left ventricular end-diastolic, left atrial, and pulmonary venous pressures, and from increased venous tone and fluid retention

The same compensatory mechanisms that maintain perfusion to the brain and heart in hypovolemic shock are often detrimental during cardiogenic shock. For example, compensatory peripheral vasoconstriction can maintain blood pressure in hypovolemic shock, but it increases left ventricular afterload (commonly thought of as increased resistance to left ventricular ejection) in cardiogenic shock.

Because the heart muscle also needs O_2, almost all children with severe or sustained shock may eventually have inadequate myocardial O_2 delivery relative to myocardial O_2 demand. Therefore, severe or sustained shock of any type eventually causes impaired myocardial function (ie, these children develop cardiogenic shock in addition to the primary cause of shock). Once myocardial function declines, the child's clinical status usually deteriorates rapidly.

Signs of Cardiogenic Shock

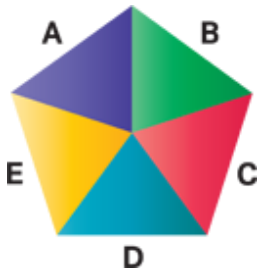


Table 38 outlines signs of cardiogenic shock typically found during the initial assessment and primary assessment of the child.

Table 38.Findings Consistent With Cardiogenic Shock

Primary assessment	Finding
Airway	Usually open/patent unless level of consciousness is significantly impaired
Breathing	• Tachypnea • Increased respiratory effort (retractions, nasal flaring, grunting) resulting from pulmonary edema*
Circulation	• Tachycardia • Normal or low blood pressure with a narrow pulse pressure • Weak or absent peripheral pulses • Normal and then weak central pulses • Delayed capillary refill with cool extremities • Signs of CHF (eg, pulmonary edema, hepatomegaly, jugular venous distention, gallop, murmur)* • Cyanosis (caused by cyanotic congenital heart disease or pulmonary edema)* • Cold, pale, mottled, diaphoretic skin • Changes in level of consciousness • Oliguria
Disability	Changes in level of consciousness
Exposure	Extremities often cooler than trunk

*Sign that distinguishes cardiogenic shock from other forms of shock.

Critical Concepts

Distinguishing Signs of Cardiogenic Shock

Increased respiratory effort often distinguishes cardiogenic shock from hypovolemic shock. Hypovolemic shock is characterized by “quiet tachypnea,” while children with cardiogenic shock may demonstrate retractions, grunting, and use of accessory muscles.

In cardiogenic shock, decreased arterial O_2 saturation secondary to pulmonary edema may present.

Rapidly providing volume resuscitation of cardiogenic shock in the setting of poor myocardial function can aggravate pulmonary edema and further impair myocardial function, further compromising oxygenation, ventilation, and cardiac output. Gradually provide volume resuscitation for cardiogenic shock; give smaller (5-10 mL/kg) boluses of isotonic crystalloid and deliver over a longer period of time (ie, over 10-20 minutes). Carefully monitor hemodynamic parameters during fluid infusion, and repeat infusion as needed.

Infants and children with cardiogenic shock often require medications to increase and redistribute cardiac output, improve myocardial function, and reduce SVR. Additional treatment includes reducing metabolic demand, such as decreasing work of breathing and controlling fever, which allows a limited cardiac output to better meet tissue metabolic demands. For more details, refer to Part 8: Managing Shock.

Obstructive Shock

Obstructive shock refers to conditions that physically impair blood flow by limiting venous return to the heart or limit the pumping of blood from the heart, resulting in decreased cardiac output. Causes of obstructive shock include

- Pericardial tamponade
- Tension pneumothorax
- Ductal-dependent congenital heart defects (eg, critical coarctation of the aorta, hypoplastic left ventricle)
- Massive pulmonary embolism

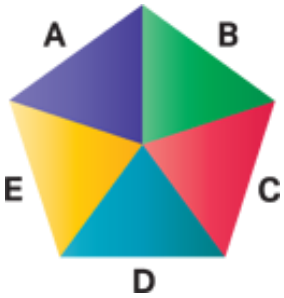
The physical obstruction to blood flow results in low cardiac output, inadequate tissue perfusion, and a compensatory increase in SVR. The early clinical presentation of obstructive shock can be indistinguishable from hypovolemic shock. However, careful clinical examination may reveal signs of systemic or pulmonary venous congestion that are not consistent with hypovolemia. In the case of massive PE, tension pneumothorax, and tamponade, the onset is usually acute, as opposed to hypovolemia, which has a more gradual progression to shock. As the condition progresses, increased respiratory effort, cyanosis, and signs of vascular congestion become more apparent.

- Preload: variable
- Contractility: normal
- Afterload: increased

Pathophysiology and Clinical Signs of Obstructive Shock

Pathophysiology and clinical signs vary according to the cause of the obstructive shock.

Cardiac Tamponade



Cardiac tamponade is caused by accumulating fluid, blood, or air in the pericardial space. Increased intrapericardial pressure and compression of the heart impede systemic venous and pulmonary venous return. This reduces ventricular filling and causes decreased stroke volume and cardiac output. If untreated, cardiac tamponade results in cardiac arrest with PEA.

In children, cardiac tamponade most often occurs after penetrating trauma or cardiac surgery. It also may develop as a result of pericardial effusion complicating an inflammatory disorder, an infection of the pericardium, a tumor, or an extremely high white blood cell count. Table 39 outlines signs of cardiac tamponade typically found during the initial assessment and primary assessment.

Table 39 Findings Consistent With Cardiac Tamponade

Primary assessment	Finding
Airway	Usually open/patent unless level of consciousness is significantly impaired
Breathing	Respiratory distress with increased respiratory rate and effort
Circulation	• Tachycardia • Poor peripheral perfusion (weak peripheral pulses, cool extremities, delayed capillary refill) • Muffled or diminished heart sounds* • Narrowed pulse pressure • Pulsus paradoxus (decrease in systolic blood pressure by >10 mm Hg during spontaneous inspiration) • Distended neck veins (may be difficult to see in infants, especially with severe hypotension)
Disability	Changes in level of consciousness

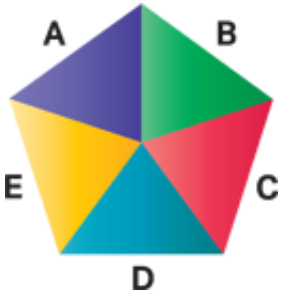
Exposure	Extremities often cooler than trunk

*Sign unique to pericardial tamponade.

After cardiovascular surgery in children, signs of tamponade may be indistinguishable from those of cardiogenic shock. Favorable outcome depends on urgent diagnosis and immediate treatment. In children with a large pericardial effusion, the ECG typically shows small QRS complexes (low voltage), but echocardiography provides a definitive diagnosis. Pulsus paradoxus is an exaggerated manifestation of a normal variation in stroke volume that occurs during the phases of spontaneous respiration. Stroke volume decreases slightly during inspiration and increases slightly during expiration. In pulsus paradoxus, the systolic blood pressure declines by greater than 10 mm Hg on inspiration, compared with expiration. True assessment for pulsus paradoxus requires measuring blood pressure with a manual pressure cuff. Inflate the cuff until no sounds are heard (as usual). Slowly decrease the cuff pressure and note when you initially hear the Korotkoff sounds, which will be when the child is exhaling. Continue to slowly deflate the cuff and note when you consistently hear the Korotkoff sounds throughout the respiratory cycle. If the difference between these 2 points is greater than 10 mm Hg, the child has a clinically significant pulsus paradoxus.

You may be able to detect a significant pulsus paradoxus by palpating the pulse, noting a distinct variation in pulse amplitude as the child inhales and exhales. Pulsus paradoxus may also be apparent on arterial and pulse oximetry waveforms but is not as easily quantified unless the waveform can be saved on the monitor screen or printed for review.

Tension Pneumothorax



Tension pneumothorax is caused by air entering the pleural space and accumulating under pressure. This air can enter from lung tissue injured by an internal tear or from a penetrating chest injury. An air leak that enters the pleural space but then stops spontaneously is called a *simple pneumothorax*. An ongoing leak can result from positive-pressure ventilation or chest trauma that forces air out of the injured lung and into the pleural space. If air continues to leak into the pleural space, it accumulates

under pressure, creating a tension pneumothorax. As this pressure increases, it compresses the underlying lung and pushes the mediastinum to the opposite side of the chest. Compression of the lung rapidly causes respiratory failure. The high intrathoracic pressure and direct pressure on mediastinal structures (heart and great vessels) impede venous return, resulting in a rapid decline in cardiac output and hypotension. Untreated tension pneumothorax leads to cardiac arrest characterized by PEA.

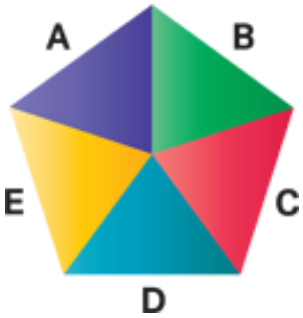
Suspect tension pneumothorax in a chest trauma or in any intubated child who deteriorates suddenly while receiving positive-pressure ventilation (including bag-mask or noninvasive ventilation). Table 40 outlines signs of tension pneumothorax typically found during the initial assessment and primary assessment.

Table 40.Findings Consistent With Tension Pneumothorax

Primary assessment	Finding
Airway	• Variable depending on situation and primary cause of respiratory distress • Advanced airway may already be in place • Tracheal deviation toward contralateral side (the side opposite the side of the pneumothorax); may be difficult to appreciate in infants
Breathing	• Respiratory distress with increased respiratory rate and effort • Hyperresonance of affected side; hyperexpansion of affected side • Diminished or absent breath sounds on affected side
Circulation	• Distended neck veins (may be difficult to appreciate in infants or in children with severe hypotension) • Pulsus paradoxus (decrease in systolic blood pressure by >10 mm Hg during spontaneous inspiration) • Rapid deterioration in perfusion; commonly, rapid evolution from tachycardia to bradycardia and hypotension as cardiac output decreases
Disability	Changes in level of consciousness
Exposure	Extremities often cooler than trunk

Favorable outcome depends on immediate diagnosis and treatment.

Ductal-Dependent Lesions



Ductal-dependent congenital cardiac abnormalities usually present in the first days to weeks of life. Ductal-dependent lesions include

- Cyanotic congenital heart lesions (ductal dependent for pulmonary blood flow)
- Left ventricular outflow obstructive lesions (ductal dependent for systemic blood flow)

The congenital heart lesions that depend on the ductus for pulmonary blood flow present with cyanosis rather than signs of shock. The left ventricular outflow obstructive lesions often present with signs of obstructive shock in the first few days or weeks of life when the ductus arteriosus closes. These left heart and aortic lesions include critical coarctation of the aorta, interrupted aortic arch, critical aortic valve stenosis, and hypoplastic left heart syndrome. Restoring and maintaining openness/patency of the ductus arteriosus is critical for survival until surgical intervention is possible, because the ductus serves as a conduit for systemic or pulmonary blood flow that bypasses the obstruction. If the infant is to survive, you must quickly recognize the presence of a ductal-dependent lesion and promptly provide treatment to open and maintain an open/patent ductus arteriosus.

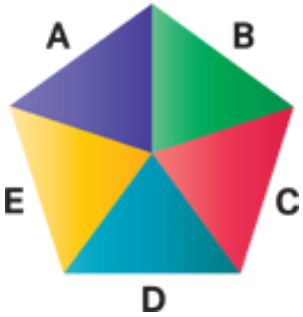
Preductal and postductal oxygen saturations are helpful for diagnosing obstructive shock in neonates with congenital heart disease. Preductal saturation is typically measured on the right hand (reflecting blood from the aorta before the ductus arteriosus), while postductal saturation is measured on a lower extremity (reflecting blood from the aorta after the ductus arteriosus). A health care professional should observe for cyanosis or significant discrepancy between pre and post measurements. If you suspect a ductal-dependent lesion, start prostaglandin infusion and call for a pediatric cardiology consultation.

Table 41 outlines findings consistent with left ventricular outflow obstructive lesions that may be found while evaluating the child.

Table 41.Findings Consistent With Left Ventricular Outflow Obstructive Lesions	
Primary assessment	**Finding**
Airway	Usually open/patent unless level of consciousness is significantly impaired
Breathing	Respiratory failure with signs of pulmonary edema or inadequate respiratory effort
Circulation	• Rapid progressive deterioration in systemic perfusion • CHF (cardiomegaly, hepatomegaly) • Higher preductal vs postductal blood pressure (eg, coarctation or interrupted aortic arch)* • Higher (>5%-10%) preductal vs postductal arterial O_2 saturation (eg, coarctation or interrupted aortic arch)* • Absent or weaker femoral pulses (eg, coarctation or interrupted aortic arch)* • Metabolic acidosis (elevated lactate)
Disability	Rapid deterioration in level of consciousness
Exposure	Cool skin

*Sign unique to left ventricular outflow obstructive lesions.

Massive Pulmonary Embolism



Pulmonary embolism is a total or partial obstruction of the pulmonary artery or its branches by a blood clot, fat, air, amniotic fluid, catheter fragment, or injected matter. Most commonly, a pulmonary embolus is a thrombus that migrates to the pulmonary

circulation. Pulmonary embolism is rare in children but may develop when an underlying condition predisposes the child to intravascular thrombosis. Examples include immobility, central venous catheters, sickle cell disease, malignancy, connective tissue disorders, hormone use, and inherited disorders of coagulation (eg, antithrombin III, protein S, protein C deficiencies).

Pulmonary embolism results in ventilation/perfusion mismatch, hypoxemia, increased pulmonary vascular resistance leading to right heart failure, decreased left ventricular filling, and decreased cardiac output. Pulmonary embolism may be difficult to diagnose because signs may be subtle and nonspecific (cyanosis, tachycardia, and hypotension). Health care professionals may not suspect pulmonary embolus, especially in children. However, signs of systemic venous congestion and right heart failure as well as acute onset do help distinguish it from hypovolemic shock. Some children with pulmonary embolism will say they have chest pain, reflecting lack of oxygenated blood flow to the lung tissue itself.

Table 42 outlines findings consistent with pulmonary embolism that may be found while evaluating the child.

Table 42 Findings Consistent With Pulmonary Embolism

Primary assessment	Finding
Airway	Usually open/patent unless level of consciousness is significantly impaired
Breathing	Respiratory distress with increased respiratory rate and effort
Circulation	• Tachycardia • Cyanosis • Hypotension • Systemic venous congestion and right heart failure • Chest pain
Disability	• Changes in level of consciousness • Anxiety • Agitation
Exposure	Extremities may be cool and mottled

Summary

Treatment of obstructive shock is cause specific; immediately recognizing and correcting the underlying cause of the obstruction can save a life. The most critical tasks for PALS providers are prompt recognition, diagnosis, and treatment of

obstructive shock. Without them, children with obstructive shock often progress rapidly to cardiopulmonary failure and cardiac arrest.

Summary: Recognizing Shock

This section summarizes the 4 basic types of shock.

Clinical Signs of Hypovolemic Shock

- Airway
  - –Open and maintainable/not maintainable
- Breathing
  - –Increased respiratory rate
  - –Normal to increased respiratory effort
  - –Normal breath sounds
- Circulation
  - –Compensated shock can progress to hypotensive shock if left untreated
  - –Narrow pulse pressure
  - –Increased heart rate
  - –Weak peripheral pulses
  - –Pale, cool skin
  - –Delayed capillary refill
  - –Decreased urine output
- Disability
  - –Level of consciousness: irritable early, lethargic late
- Exposure
  - –Variable temperature

Clinical Signs of Distributive Shock

- Airway
  - –Open and maintainable/not maintainable
- Breathing
  - –Increased respiratory rate

    - –Normal to increased respiratory effort
    - –Normal breath sounds (± crackles)
- Circulation
    - –Compensated shock can progress to hypotensive shock if left untreated
    - –Variable pulse pressure
    - –Increased heart rate
    - –Bounding or weak peripheral pulses
    - –Warm or cool skin
    - –Variable capillary refill
    - –Decreased urine output
- Disability
    - –Level of consciousness: irritable early, lethargic late
- Exposure
    - –Variable temperature

Clinical Signs of Cardiogenic Shock

- Airway
    - –Open and maintainable/not maintainable
- Breathing
    - –Increased respiratory rate
    - –Labored respiratory effort
    - –Normal breath sounds, crackles, grunting
- Circulation
    - –Compensated shock can progress to hypotensive shock if left untreated
    - –Narrow pulse pressure
    - –Increased heart rate
    - –Weak peripheral pulses
    - –Pale, cool skin
    - –Delayed capillary refill
    - –Decreased urine output
- Disability
    - –Level of consciousness: irritable early, lethargic late

- Exposure
    - –Variable temperature

Clinical Signs of Obstructive Shock

- Airway
    - –Open and maintainable/not maintainable
- Breathing
    - –Increased respiratory rate
    - –Labored respiratory effort
    - –Normal breath sounds, crackles, grunting
- Circulation
    - –Compensated shock can progress to hypotensive shock if left untreated
    - –Narrow pulse pressure
    - –Increased heart rate
    - –Weak peripheral pulses
    - –Pale, cool skin
    - –Delayed capillary refill
    - –Decreased urine output
- Disability
    - –Level of consciousness: irritable early, lethargic late
- Exposure
    - –Variable temperature

Table 43 summarizes the cardiac changes associated with each type of shock.

Table 43.Types of Shock and Cardiac Changes

Types of shock	Preload	Contractility	Afterload	Cardiac output
Hypovolemic	Decreased	Normal or increased	Increased	Decreased
Distributive	Decreased	Variable	Decreased	Variable
Septic	Decreased	Variable	Variable	Variable

Anaphylactic	Decreased	Decreased	Decreased	Decreased
Neurogenic	Decreased	Normal	Decreased	Decreased
Cardiogenic	Variable	Decreased	Increased	Decreased
Obstructive	Variable	Normal	Increased	Decreased

Part 8
Managing Shock

Once you identify shock in a critically ill or injured child, intervene early to reduce morbidity and mortality. This Part discusses the goals and priorities of shock management, treatment fundamentals, general and advanced management, and specific management according to etiology.

Learning Objective

After completing this Part, you should be able to perform early interventions for treating shock.

During the course, you will be asked to manage children in shock. To do this, you must know general and specific treatment based on the different types of shock.

Goals of Shock Management

The goals in treating shock are to

- Improve O_2 delivery
- Balance tissue perfusion and metabolic demand
- Support organ function
- Prevent progression to cardiac arrest

Immediate intervention for a child in shock may be lifesaving. The more time that passes between the onset of signs of shock and the restoration of adequate O_2 delivery and organ perfusion, the poorer the outcome is. Once a child in shock progresses to cardiac arrest, prognosis is poor.

Warning Signs

Watch for signs that compensatory mechanisms are failing in a seriously ill or injured child. Once you recognize that the child's condition is deteriorating, act decisively with the resuscitation team to provide effective resuscitation therapy. Identifying compensated shock early is critical to effective treatment and good outcome. Warning signs that indicate progression from compensated to hypotensive shock include

- Increasing tachycardia

- Diminishing or absent peripheral pulses
- Weakening central pulses
- Narrowing pulse pressure
- Cold distal extremities with prolonged capillary refill
- Decreasing level of consciousness
- Hypotension (late finding)

Once the child develops hypotensive shock, organ perfusion is typically severely compromised, and organ dysfunction may develop even if the child does not progress to cardiac arrest.

Fundamentals of Shock Management

The acute treatment of shock focuses on restoring O_2 delivery to the tissues and improving the balance between tissue perfusion and metabolic demand. This treatment consists of

- Optimizing O_2 content of the blood
- Improving volume and distribution of cardiac output
- Reducing O_2 demand
- Correcting metabolic derangements

Try to identify and reverse the underlying cause of shock while promptly intervening.

Optimizing Oxygen Content of the Blood

O_2 content of the blood is determined by the hemoglobin concentration and its saturation with oxygen. To optimize O_2 content

- Administer a high concentration of O_2 (use nonrebreathing mask to deliver 100% O_2)
- Use invasive or noninvasive mechanical ventilation to improve oxygenation by correcting a ventilation/blood flow (V/Q) mismatch or other respiratory disorders
- If hemoglobin concentration is low, consider packed red blood cell (PRBC) transfusion

Improving Volume and Distribution of Cardiac Output

For most forms of shock, use bolus fluid administration to improve volume and distribution of cardiac output. You may consider noninvasive or invasive positive-pressure ventilation to reduce the work of breathing and improve oxygenation. Children in shock may also benefit from vasoactive agents such as vasopressors, vasodilators, indicators, and/or inotropes.

Reducing Oxygen Demand

For all forms of shock, try to improve the balance between O_2 delivery and supply by reducing O_2 demand. The most common factors that contribute to increased O_2 demand are

- Increased work of breathing
- Pain and anxiety
- Fever

Support breathing with noninvasive or invasive ventilation and assisted ventilation. To facilitate intubation and mechanical ventilation, you may administer sedatives or analgesics and neuromuscular blockade. You may also need to control pain and anxiety with analgesics and sedatives. Use sedative and analgesic agents with extreme caution; they may suppress the child's endogenous stress response, impair compensatory mechanisms such as tachycardia, reduce blood pressure, and potentially lead to cardiac arrest. The sedative effects of these agents can also make it more difficult to evaluate the child's level of consciousness and response to treatment. Control fever by administering antipyretics and other cooling measures.

Correcting Metabolic Derangements

Many conditions that lead to shock may result in or be complicated by metabolic derangements, such as

- Hypoglycemia
- Hypocalcemia
- Hyperkalemia
- Metabolic (lactic) acidosis

All of these conditions can adversely affect cardiac contractility. Metabolic acidosis is characteristic of all forms of shock.

Hypoglycemia is low serum glucose concentration that, if left untreated, can cause seizures and brain injury. Glucose is vital for proper cardiac and brain function. Glucose stores may be low in infants and chronically ill children.

Hypocalcemia is a low serum ionized calcium concentration. Calcium is essential for effective cardiac function and vasomotor tone. Hypocalcemia can result from administering blood products, colloid, and buffering medications such as sodium bicarbonate.

Hyperkalemia is a high serum potassium concentration, which may result from renal dysfunction, cell death, excess potassium administration, or acidosis. Acidosis causes a shift of potassium from the intracellular to the extracellular—including the intravascular—space. As a result, acidosis, or a fall in serum pH, typically is associated with a rise in serum potassium. The serum potassium will fall when acidosis is corrected or alkalosis develops.

Metabolic acidosis develops from production of acids, such as lactic acid, when tissue perfusion is inadequate. Renal or gastrointestinal dysfunction can also cause metabolic acidosis. Renal dysfunction can cause organic acid retention or bicarbonate ion loss while gastrointestinal dysfunction, such as diarrhea, can result in bicarbonate ion loss. Severe metabolic acidosis may depress myocardial contractility and reduce the effect of vasopressors. Unless metabolic acidosis is due solely to bicarbonate losses, it does not respond well to buffer therapy. Treat the acidosis by attempting to restore tissue perfusion with fluid resuscitation and vasoactive agents. If treatment is effective, the metabolic acidosis will resolve.

On occasion, you may need buffers (eg, sodium bicarbonate) to acutely correct profound metabolic acidosis that is impairing vital organ function. Sodium bicarbonate works by combining with hydrogen ions (acids) to produce carbon dioxide and water; increased alveolar ventilation then eliminates the carbon dioxide. Ventilation support is always important in the critically ill child, but it is especially important for treating metabolic acidosis with sodium bicarbonate. One scenario in which sodium bicarbonate should be avoided is DKA, where the serum bicarbonate can be profoundly low but giving sodium bicarbonate can result in worse outcomes.

Correcting metabolic derangements may be essential to optimizing organ function. Measure glucose concentration, and replenish as indicated. It is important to measure glucose a few times during resuscitation, particularly if it is borderline or the patient has already received dextrose for a previously low glucose. Replacement of calcium and sodium bicarbonate is occasionally used in critically ill children with heart disease.

General Management of Shock

Components of General Management

General management of shock consists of the following:

- Positioning
- Supporting airway, oxygenation, and ventilation
- Establishing vascular access
- Providing fluid resuscitation
- Monitoring
- Performing frequent reassessment
- Obtaining laboratory studies
- Providing medication therapy
- Consulting appropriate subspecialists

Note that several of these interventions may be implemented by the team simultaneously.

Positioning

Initial management of shock includes positioning the critically ill or injured child. If the child is responsive and hemodynamically stable, allow them to remain in the most comfortable position (eg, sitting in the arms of a caregiver) to decrease anxiety and activity as you form your initial assessment and conduct the primary assessment. If the child is hypotensive and breathing is not compromised, place the child in the supine position.

Supporting Airway, Oxygenation, and Ventilation

Maintain an open/patent airway and support oxygenation and ventilation. Give a high concentration of supplemental O_2 to all children with shock, usually by a high-flow O_2 delivery system. Sometimes you must combine O_2 delivery with ventilatory support if respirations are ineffective, mental status is impaired, or work of breathing is significantly increased. Appropriate interventions may include noninvasive positive airway pressure or mechanical ventilation after ET intubation.

Vascular Access

Once the airway is open/patent and oxygenation and ventilation are supported, establish vascular access for fluid resuscitation and administering medications. For compensated shock, initial attempts at peripheral venous cannulation are appropriate. For hypotensive shock, you can best accomplish critical immediate vascular access by the IO route if peripheral IV access is not readily achieved. Depending on the health care professional's experience and expertise and clinical circumstances, central venous access may be useful. However, gaining central venous access takes longer than placing IO access.

Critical Concepts

IO Access

If peripheral vascular access cannot be readily obtained in a child with compensated or hypotensive shock, be prepared to establish IO access.

For more information on establishing IO access, refer to IO Access in Resources for Managing Circulatory Emergencies later in this Part.

Fluid Resuscitation

Once you establish vascular access, start fluid resuscitation immediately.

Critical Concepts
Fluid Resuscitation

In general, isotonic crystalloid should be given as a bolus over 5 to 20 minutes: 10 to 20 mL/kg for children with suspected septic shock, 20 mL/kg for hypovolemic shock, and 5 to 10 mL/kg for cardiogenic shock. In children with severe, hypotensive, hypovolemic shock, fluid should be given over 5 to 10 minutes. Keep a high level of suspicion for cardiogenic shock as you fluid resuscitate. **If after a fluid bolus there is no improvement in vitals or they worsen, stop fluid bolus.** If you suspect cardiogenic shock, use smaller fluid boluses of 5 to 10 mL/kg given over 10 to 20 minutes. Carefully monitor for signs of pulmonary edema or worsening tissue perfusion. Stop the infusion if such signs occur. Be prepared to support oxygenation and ventilation as necessary.

Reassess and repeat boluses to restore blood pressure and tissue perfusion.

Rapid, appropriate fluid bolus administration is a priority. Inadequate intravascular volume leads to low stroke volume and hypotension. A child in septic shock often requires a large volume of fluid to restore perfusion. It may be necessary to rapidly infuse 10 to 20 mL/kg of isotonic crystalloid solution (up to 40-60 mL/kg total). Titrate the volume and rate of fluid administration during and after each bolus by assessing the child's mental status, heart rate, temperature, blood pressure, and organ perfusion (including presence and quality of central and peripheral pulses, capillary refill, skin temperature and color, and urine output).

Specific Treatment Considerations

- Use individualized patient evaluation before administering IV fluid boluses.
- Do not delay vasoactive support if needed. Delay is associated with increased length of stay and mortality in pediatric septic shock. Begin within the first hour if shock persists in spite of fluid boluses.
- Determine diseases such as anemia and malnutrition when providing fluid boluses.
- In limited resource settings, when access to mechanical ventilation and inotropic support may not be available, use caution when treating the febrile child. Fluid boluses may be more harmful than therapeutic.
- When fluid overload is present—such as hepatomegaly or pulmonary edema—use smaller fluid volumes if additional fluid is required.
- If fluid overload develops, be prepared for potential ET intubation and mechanical ventilation with PEEP.
- Pulmonary edema may develop with cardiogenic shock. Use smaller fluid volume if additional fluid is required.
- Correct metabolic derangements immediately. Hypoglycemia and ionized hypocalcemia contribute to myocardial dysfunction.

Monitoring

No single resuscitation end point has been identified as a consistent marker of adequate tissue perfusion and cellular homeostasis. However, you can assess the effectiveness of fluid resuscitation and medication therapy by frequent or continuous monitoring (Table 44). As soon as possible, start noninvasive monitoring (SpO_2, heart rate, blood pressure), assess mental status, measure temperature, and measure urine output with an indwelling bladder catheter.

Table 44.Monitoring in Circulatory Emergencies

Frequently or continuously monitor	Indication of positive response to shock therapy
Oxygen saturation with pulse oximetry	94% or higher when breathing room air
Heart rate	Appropriate for age and clinical condition; typically will fall from a rapid rate toward a normal range (refer to Table 18. Normal Heart Rates in Part 4)
Peripheral pulses	Weak pulses will become stronger, and bounding pulses will be less bounding but still strong
Capillary refill	Will shorten toward 2 seconds or less
Skin color and temperature	Normal skin color and mucous membranes; warm extremities
Blood pressure	Increase to within normal range for age (Table 20. Normal Blood Pressures in Part 4) with normal pulse pressure
Level of consciousness	Child will respond more appropriately (ie, mental status improves)
Ongoing fluid losses	Bleeding and diarrhea controlled
Urine output	• Infants and young children: approximately 1.5 to 2 mL/kg per hour • Older children and adolescents: approximately 1 mL/kg per hour

Also consider invasive monitoring (eg, arterial and central venous catheterization), depending on the health care professionals' experience and available resources. Signs that indicate clinical improvement toward a normal hemodynamic state are decreased serum lactate and smaller base deficit.

Critical Concepts
Accurately Assess Tissue Perfusion

Although measuring blood pressure is easy, it is important to assess other clinical parameters to evaluate tissue perfusion. Remember that blood pressure may be normal in children with severe shock, and noninvasive blood pressure measurement may be inaccurate if perfusion is poor.

Frequent Reassessment

Frequently reassess the child's respiratory, cardiovascular, and neurologic status to

- Evaluate trends in the child's condition
- Determine response to therapy
- Plan the next interventions

A child in shock is in a dynamic clinical condition that can deteriorate at any moment and require lifesaving interventions, such as ET intubation. Frequently reassess until the child's condition becomes stable or the child is transferred to advanced care.

Critical Concepts
Monitor to Evaluate Trends

The condition of a child in shock is dynamic. Continuously monitor and frequently reassess to evaluate trends in the child's condition and determine response to therapy.

Laboratory Studies

Laboratory studies provide important information to help you

- Identify the etiology and severity of shock
- Evaluate organ dysfunction secondary to shock
- Identify metabolic derangements
- Evaluate response to therapy

Refer to Part 12: Post–Cardiac Arrest Care for additional information on evaluating end-organ function. Also consider expert consultation in diagnosis and management of end-organ failure.

Table 45 outlines some laboratory studies that can help identify the etiology and severity of shock and guide therapy.

Table 45.Laboratory Studies to Evaluate Shock and Guide Therapy

Laboratory study	Finding	Possible etiology	Possible interventions
Complete blood cell count	Hemoglobin/hematocrit decreased	• Hemorrhage • Fluid resuscitation (dilution) • Hemolysis	• Administer 100% O_2 • Control bleeding • Transfuse blood • Titrate fluid administration
	White blood cell count increased or decreased	• Sepsis	• Obtain appropriate cultures • Give antibiotics
	Platelets decreased	• Disseminated intravascular coagulation • Decreased platelet production	• Transfuse platelets if child has serious bleeding • Obtain prothrombin time/partial thromboplastin time, fibrinogen, and D-dimers
Glucose	Increased or decreased	• Stress (usually increased but may be decreased in infants) • Sepsis • Decreased production (eg, liver failure) • Adrenal insufficiency	• If hypoglycemia is present, give dextrose bolus and start infusion of dextrose-containing solution if needed • Severe hyperglycemia may require treatment (per institutional protocols or obtain expert consultation)
Potassium	Increased or decreased	• Renal dysfunction • Acidosis (increases serum potassium concentration) • Diuresis (decreased) • Adrenal insufficiency (increased)	• Treat significant or symptomatic hyperkalemia or hypokalemia

Calcium	Decreased (ionized calcium concentration)	• Sepsis • Transfusion of blood preserved with citrate-phosphate-dextran • Colloid administration • Buffering agents (eg, sodium bicarbonate)	• Give calcium
Lactate	Increased as product of anaerobic metabolism from tissue hypoperfusion	• Tissue hypoxia • Increased glucose production (gluconeogenesis) • Decreased metabolism (eg, liver failure, inborn error of metabolism, toxic ingestion)	• Improve tissue perfusion • Consider other unusual etiologies for lactic acidosis if lactate not improving with improved systemic oxygen delivery • Consider buffering acidosis in specific situations
Blood gas*	pH decreased in acidosis; increased with alkalosis	• Lactic acid accumulation caused by tissue hypoperfusion • Renal failure • Inborn error of metabolism • Diabetic ketoacidosis • Poisoning/overdose • Diarrhea or ileostomy losses • Hyper-/hypoventilation (sepsis, poisoning) • Vomiting	• Give fluid • Support ventilation • Correct shock • Consider buffer (in special situations) • Evaluate anion gap† to determine if acidosis is from increased unmeasured ions
$ScvO_2$	Variable	• Low central venous O_2 saturation—inadequate O_2 delivery or increased consumption • High central venous O_2 saturation—maldistribution of blood flow or decreased O_2 utilization	• Attempt to maximize O_2 delivery and minimize O_2 demand

*In the ICU setting, consider arterial blood gas as indicated.

†Anion gap = [serum Na^+] − ([serum Cl^-] + [serum HCO_3^-]); normal = approximately 8 to 12 mEq/L.

Medication Therapy

Use medication therapy when managing shock to affect myocardial contractility, heart rate, and vascular resistance. The choice of agent(s) is determined by the child's physiologic state.

Vasoactive agents are indicated when shock persists despite adequate volume resuscitation to optimize preload. For example, a child with septic shock who remains hypotensive with signs of vasodilation despite fluid bolus administration may benefit from a vasoconstrictor. Administering vasoactive medications is potentially harmful if the child has not been adequately fluid resuscitated first. In children with cardiogenic shock, use vasoactive agents early because fluid resuscitation is not key to improving myocardial function and may be harmful. Most children with cardiogenic shock benefit from an inotrope to increase cardiac output.

Inotropes, vasodilators, and vasopressors are classes of pharmacologic agents commonly used in shock. Table 46 lists vasoactive medications by class and pharmacologic effects.

Table 46.Vasoactive Therapy Used in Treating Shock

Class	Medication	Effect
Inotropes	• Epinephrine • Dobutamine	• Increase cardiac contractility • Increase heart rate • Produce variable effects on SVR *Note:* Includes agents with both α-adrenergic and β-adrenergic effects
Vasodilators	• Nitroglycerin • Nitroprusside	• Decrease SVR and venous tone
Vasopressors (vasoconstrictors)	• Epinephrine (doses >0.1 mcg/kg per minute) • Norepinephrine • Vasopressin	• Increase SVR • Increase myocardial contractility (except vasopressin)

Critical Concepts
Color-Coded Length-Based Tape

Use a color-coded length-based tape to determine the child's weight (if not known) for calculating medication doses and for selecting the correct sizes of resuscitation equipment. Refer to Resources for Managing Circulatory Emergencies in Part 8 of this manual for an example.

Subspecialty Consultation

For specific shock categories, some required lifesaving diagnostic assessments and therapeutic interventions may be beyond many PALS providers' scope of practice. For example, a health care professional may not be trained to interpret an echocardiogram or perform a thoracostomy or pericardiocentesis. Recognize limitations to your own scope of practice and call for help when needed. Early subspecialty consultation (eg, pediatric critical care, pediatric cardiology, pediatric surgery) is essential to shock management and may influence outcome.

Critical Concepts
Expert Consultation

When treating a child in shock, health care professionals must obtain consultation from appropriate experts as soon as possible.

Summary: Initial Management Principles

This summary reviews initial shock management principles discussed in this section:

- Positioning the child
  - –Stable—Allow to remain with caregiver in a position of comfort
  - –Unstable—If hypotensive, place in supine position unless breathing is compromised
- Optimizing arterial O_2 content
  - –Administer a high concentration of O_2 via a nonrebreathing mask; consider blood transfusion in cases of significant blood loss or other causes of severe anemia; and consider using CPAP, noninvasive positive airway pressure, or mechanical ventilation with PEEP
- Supporting ventilation as indicated (invasive or noninvasive)
- Establishing vascular access
  - –Consider IO access early
- Beginning fluid resuscitation

    - –Hypovolemic shock: Give an isotonic crystalloid bolus of 20 mL/kg over 5 to 20 minutes (5-10 minutes with severe, hypotensive, hypovolemic shock); repeat 20 mL/kg boluses as needed to restore blood pressure and tissue/organ perfusion.
    - –Hemorrhagic shock: Give an isotonic crystalloid bolus of 10 to 20 mL/kg. May repeat 1 to 2 times as needed to restore blood pressure and tissue organ perfusion. If the child does not respond, give 10 to 20 mL/kg of PRBCs and 10 to 20 mL/kg of fresh frozen plasma and platelets.
    - –Septic shock: For children with shock due to suspected sepsis, administration of 10 to 20 mL/kg is recommended, with repeat boluses of 10 to 20 mL/kg as well. Reassess the child after each bolus.
    - –Cardiogenic shock: Modify bolus fluid therapy to deliver 5 to 10 mL/kg over 10 to 20 minutes if you suspect cardiogenic shock or severe myocardial dysfunction.
- Monitoring
    - –SpO_2, heart rate, respiratory rate and effort, blood pressure, level of consciousness, temperature, and urine output
- Performing frequent reassessment
    - –Evaluate trends and determine response to therapy
- Conducting laboratory studies
    - –To identify shock etiology and severity, evaluate organ dysfunction secondary to shock, identify metabolic derangements, and evaluate the response to therapy
- Administering pharmacologic support—refer to Table 46. Vasoactive Therapy Used in Treating Shock
    - –To increase heart rate, improve myocardial function or redistribute cardiac output (increase contractility, reduce or increase SVR, improve organ perfusion), correct metabolic derangements, and manage pain and anxiety
    - –Obtain subspecialty consultation

Summary: Managing Shock

Here is a summary of general management of shock and specific management by etiology.

General Management

- Oxygen
- Pulse oximetry
- ECG monitor; frequent blood pressure monitoring

- BLS as indicated
- IV/IO access
- Point-of-care glucose testing

Specific Management of Hypovolemic Shock

- Nonhemorrhagic
    - –20 mL/kg isotonic crystalloid bolus, repeat as needed
    - –Consider colloid
- Hemorrhagic
    - –Control external bleeding
    - –10-20 mL/kg isotonic crystalloid bolus, repeat 1 or 2 × as needed
    - –Transfuse PRBCs as indicated

Specific Management of Distributive Shock

- Septic
    - –Refer to the Pediatric Septic Shock Algorithm
- Anaphylactic
    - –IM epinephrine (or autoinjector)
    - –Fluid boluses (10-20 mL/kg isotonic crystalloid)
    - –Albuterol
    - –Antihistamines, corticosteroids
    - –Epinephrine infusion
- Neurogenic
    - –20 mL/kg isotonic crystalloid bolus, repeat as needed
    - –Vasopressor

Specific Management for Cardiogenic Shock

- Bradyarrhythmia/tachyarrhythmia
    - –Refer to the Pediatric Bradycardia With a Pulse Algorithm
    - –Refer to the Pediatric Tachyarrhythmia With a Pulse Algorithm
- Other (eg, congenital heart disease, myocarditis, cardiomyopathy, poisoning)
    - –5 to 10 mL/kg isotonic crystalloid bolus, repeat as needed
    - –Inotropic and/or vasoactive infusion

    - –Antidote for poisoning
    - –Consider expert consultation

Specific Management for Obstructive Shock

- Ductal-dependent (left ventricular outflow obstruction)
    - –Prostaglandin E_1
    - –Expert consultation
- Tension pneumothorax
    - –Needle decompression
    - –Tube thoracostomy
- Cardiac tamponade
    - –Pericardiocentesis
    - –5-10 mL/kg isotonic crystalloid bolus
    - –Expert consultation
- Pulmonary embolism
    - –5-10 mL/kg isotonic crystalloid bolus, repeat as needed
    - –Consider thrombolytics, anticoagulants
    - –Expert consultation

Fluid Therapy

The primary objective of fluid therapy in shock is to restore intravascular volume and tissue perfusion. Rapid fluid resuscitation is required for hypovolemic and distributive shock, including septic shock. Cardiogenic and obstructive shock, as well as special conditions such as severe poisonings or fluid loss with DKA, may dictate alternative approaches to fluid resuscitation.

In general, boluses of isotonic crystalloid will expand intravascular volume. Blood and blood products are generally not used for volume expansion in children with shock unless shock is due to hemorrhage or existing significant anemia. Blood products may also be indicated for correction of some coagulopathies.

Isotonic Crystalloid Solutions

For most children with shock, isotonic crystalloid solutions, such as normal saline or lactated Ringer’s, are the preferred initial fluids for volume replacement in managing shock. They are inexpensive, readily available, and do not cause sensitivity reactions.

Critical Concepts
Quantity of Crystalloid Solution in Shock Resuscitation

Because isotonic crystalloids are distributed throughout the extracellular space, a large quantity of crystalloid solution may be needed to restore intravascular volume for children in shock. Rapid infusion of a large volume of fluid may be well tolerated by a healthy child but may cause pulmonary and peripheral edema in a critically ill child. Reassess after every fluid bolus.

Colloid Solutions

Colloid solutions (eg, 5% albumin and fresh frozen plasma) may be alternatives to crystalloid solutions. However, they have disadvantages for the acute resuscitation of a child in shock. They are less widely available than crystalloid solutions and may take time to prepare. Blood-derived colloid solutions may cause sensitivity reactions. Synthetic colloids may cause coagulopathies; their use is usually limited to 20 to 40 mL/kg. As with crystalloids, excessive administration of colloids can lead to pulmonary edema, particularly in children with cardiac or renal disease. Despite these limitations, fresh frozen plasma may be used in specific circumstances, such as massive hemorrhage and blood administration (refer to Indications for Administering Blood Products later in this Part).

Rate and Volume of Fluid Administration

Start fluid resuscitation for shock with 20 mL/kg of isotonic crystalloid bolus over 5 to 20 minutes; in children with shock due to suspected sepsis, administer 10 to 20 mL/kg per bolus. In shock consistent with cardiogenic shock, deliver smaller boluses such as 5 to 10 mL/kg. Repeat boluses as needed to restore blood pressure and perfusion. You may often find it difficult to predict the volume of fluid deficit from the child's history, so use clinical examination and supporting laboratory studies to identify the volume needed. You may need to administer more than the estimated volume deficit. Reassess frequently and after administering each bolus.

Give fluid boluses rapidly for hypotensive and septic shock. Children with septic shock may require 60 mL/kg or more of isotonic crystalloid solution during the first hour of therapy; as much as 200 mL/kg or more may be required in the first 8 hours of therapy.

Critical Concepts
Fluid Administration

If you find or suspect myocardial dysfunction or obstructive shock, give smaller volumes of fluid more slowly. Administer boluses of 5 to 10 mL/kg over 10 to 20 minutes and reassess after each bolus; stop bolus administration if vital signs fail to improve or if the child develops signs of worsening respiratory status, rales, or other evidence of pulmonary edema or

hepatomegaly. Obtain further diagnostic assessment and expert consultation (eg, echocardiogram) to confirm suspicions and guide the next interventions. Be prepared to support the airway, oxygenation, and ventilation with PEEP as needed if pulmonary edema develops.

Modifying fluid resuscitation is appropriate for children in shock associated with DKA. Children with DKA may be significantly dehydrated but often have high serum osmolality (caused by hyperglycemia). Rapidly administering crystalloid solution and reducing serum osmolality may contribute to risk of cerebral edema. Therefore, fluid management in DKA is complex. Consider giving an initial bolus of isotonic crystalloid 10 to 20 mL/kg over 1 to 2 hours. This constitutes a fluid bolus, but it is atypical in that it is given over a longer period of time than the usual fluid bolus. However, if the patient with DKA is in hypotensive shock, the treatment approach should default to more aggressive bolus fluids for shock per any other etiology of shock. Many institutions have local protocols about specifically managing DKA and its associated metabolic, electrolyte, and fluid derangements. Consult experts when possible. After giving a fluid bolus, reassess the patient.

Similarly, children who have ingested calcium channel blockers or β-adrenergic blockers may have myocardial dysfunction and are less tolerant of rapid volume expansion. When caring for children with severe febrile illness in settings with limited access to critical care resources (ie, mechanical ventilation and inotropic support), administer bolus IV fluids with extreme caution because it may be harmful. Once again, health care professionals should reassess the patient after every fluid bolus.

Table 47 gives a summary of fluid boluses and rates of delivery based on the underlying cause of shock.

Table 47.Guide to Fluid Boluses and Rates of Delivery Based on Underlying Cause of Shock

Type of shock	Volume of fluid (mL/kg)	Approximate rate of delivery (min)
Hypovolemic	20	5-10 if hypotensive; 5-20 if compensated
Obstructive	5-20	10-20
Cardiogenic (nonpoisoning)	5-10	10-20
Distributive	20	5-20
Septic	10-20	5-20

Rapid Fluid Delivery

IV fluid administration systems generally used for pediatric fluid therapy do not deliver fluid boluses as rapidly as required for managing some forms of shock. To help deliver fluid rapidly

- Place as large an IV catheter as possible, especially if blood or colloid administration is needed; ideally, insert 2 catheters.
- Place an in-line 3-way stopcock in the IV tubing system.
- Deliver fluid by using a 20- to 60-mL syringe to push fluids through the stopcock, or use a pressure bag (beware of risk of air embolism) or a rapid infusion device.
- If IV access cannot be established, establish IO access.

Note: Standard infusion pumps—even if set at the maximum infusion rate—do not provide a sufficiently rapid rate of fluid delivery, especially in larger children. For example, a 50-kg patient with septic shock should ideally receive 1 L of crystalloid in 5 to 10 minutes, but standard infusion pumps may have a maximum rate of 999 mL for the hourly rate.

Frequently Reassess During Fluid Resuscitation

Frequently reassess during fluid resuscitation to manage shock effectively. Monitor to

- Assess the physiologic response to therapy after each fluid bolus
- Determine the need for further fluid boluses
- Assess for signs of detrimental effects (eg, pulmonary edema) during and after fluid resuscitation
- Assess lung sounds and liver position before and after every fluid bolus

Signs of physiologic improvement include improved perfusion, increased blood pressure, decreasing heart rate (toward normal), decreased respiratory rate (toward normal), increased urine output, and improved mental status. If the child's condition does not improve or worsens after fluid boluses, try to identify the cause of the shock to help determine the next interventions. For example, persistently delayed capillary refill despite initial fluid administration may indicate ongoing hemorrhage or other fluid loss or need for vasopressor therapy. Deterioration of the child's condition after fluid therapy may signal cardiogenic or obstructive shock. Increased work of breathing may indicate pulmonary edema.

Indications for Administering Blood Products

Administering PRBCs is recommended for replacing traumatic blood loss if the child's perfusion is inadequate despite receiving 2 to 3 boluses of 20 mL/kg of isotonic crystalloid. Under these circumstances, administer 10 to 20 mL/kg PRBCs as soon as available.

Fully crossmatched blood is generally not available in emergencies because most blood banks require about 1 hour for the crossmatching process. It may become available for children who are stabilized with crystalloid but have ongoing blood losses. Priorities for the type of blood or blood products used in order of preference are

- Crossmatched
- Type specific
- Type O-negative (O[–] preferred for female patients and either O[+] or O[–] for male patients)

Unmatched, type-specific blood may be used if ongoing blood loss results in hypotension despite administration of crystalloid. Most blood banks can supply type-specific blood within 10 minutes. Type-specific blood is ABO and Rh compatible but, unlike fully crossmatched blood, may have other antibodies that are incompatible with the patient's blood.

Use type O blood if blood should be administered immediately to prevent circulatory collapse or cardiopulmonary arrest, because it can be given to children of any blood type. O-negative blood is preferred for female patients of childbearing age to avoid Rh sensitization. Either O-negative or O-positive blood may be administered to male patients.

Complications of Rapid Administration of Blood Products

Rapid infusion of cold blood or blood products, particularly in large volume, may produce several complications, including

- Hypothermia
- Myocardial dysfunction
- Ionized hypocalcemia

Hypothermia may adversely affect cardiovascular function and coagulation as well as compromise several metabolic functions, including metabolism of citrate, which is present in stored blood. Inadequate citrate clearance, in turn, causes ionized hypocalcemia. The combined effects of hypothermia and ionized hypocalcemia can result in significant myocardial dysfunction and hypotension.

To minimize these problems, warm blood and blood products, if possible, with an approved commercial blood-warming device before or during rapid IV administration. Prepare calcium if the child becomes hypotensive during rapid transfusion; in some cases, it may be beneficial to administer calcium empirically to prevent hypocalcemia.

Glucose

Monitor blood glucose concentration as a component of shock management. Hypoglycemia, a common finding in critically ill children, can result in brain injury if not rapidly identified and effectively treated. In one pediatric study, hypoglycemia was

present in 18% of children who received resuscitative care in an ED for decreased level of consciousness, status epilepticus, respiratory failure, cardiopulmonary failure, or cardiac arrest.

Glucose Monitoring

Measure serum glucose concentration as soon as possible in all critically ill infants and children (eg, those with altered mental status, respiratory compromise, shock) from capillary, venous, or arterial blood samples with a point-of-care device. Do not delay treatment waiting on lab analysis. Small infants and chronically ill children have higher glucose utilization rates and limited stores of glycogen. This limited supply may rapidly deplete during episodes of physiologic stress, resulting in hypoglycemia. Infants receiving non–glucose-containing IV fluids are at increased risk for developing hypoglycemia.

Critical Concepts

Identify Hypoglycemia

In all critically ill or injured children, perform a rapid glucose test to rule out hypoglycemia as a cause of or a contributing factor to shock or decreased level of consciousness.

Hyperglycemia, also frequently present in seriously ill or injured children, may result from a relative insulin-resistant state induced by high concentrations of endogenous catecholamines and cortisol. Although controlling serum glucose concentration within a narrow range by using insulin infusion improved survival in critically ill adult and pediatric patients, tight glucose control was also associated with more frequent episodes of hypoglycemia. Insufficient data support routine use of this tight control of glucose concentration in critically ill children. Consider treating hyperglycemia in high-risk groups, such as brain-injured children, while monitoring closely to prevent hypoglycemia.

Diagnosing Hypoglycemia

Hypoglycemia may be difficult to recognize clinically by the child’s appearance. Some children have no outward signs or symptoms (ie, asymptomatic hypoglycemia), while others may show nonspecific clinical signs, such as

- Poor perfusion
- Diaphoresis
- Tachycardia
- Hypothermia
- Irritability or lethargy
- Hypotension

These clinical signs are also common to many other conditions, including hypoxemia, ischemia, or shock.

Although single threshold values are not applicable to every patient, the following lowest acceptable glucose concentrations can be used to define hypoglycemia:

- Preterm and term neonates: less than 45 mg/dL
- Infants, children, and adolescents: less than 60 mg/dL

The reported low range of normal glucose is typically related to sample measurements obtained in nonstressed, fasting infants and children. It is difficult to extrapolate these thresholds to the glucose concentration required by a stressed, critically ill, or injured child.

Managing Hypoglycemia

If the glucose concentration is low and the child has minimal symptoms and normal mental status, you may administer glucose orally (eg, with orange juice or other glucose-containing fluid). If the concentration is very low or the child is symptomatic, give IV glucose at a dose of 0.5 to 1 g/kg. Do not exceed the adult dose of glucose (25 g). IV dextrose is commonly administered as $D_{25}W$ (2-4 mL/kg) or $D_{10}W$ (5-10 mL/kg). Dextrose is the same substance as glucose. Reassess the serum glucose concentration after dextrose administration. Provide a continuous infusion of glucose-containing IV fluid to prevent recurrent hypoglycemia.

Do not routinely infuse dextrose-containing fluids for volume resuscitation of shock. This can cause hyperglycemia, increase the serum osmolality, and produce an osmotic diuresis that will further exacerbate hypovolemia and shock. Electrolyte imbalances (eg, hyponatremia) can also develop.

Management According to Type of Shock

Effective management of shock targets treatment to the etiology of the shock. For the purposes of the PALS Provider Course, shock is categorized into 4 types, based on the underlying cause. However, this classification method oversimplifies the physiologic state seen in individual patients. Some children with shock have elements of hypovolemic, distributive, and cardiogenic shock, with 1 type being dominant. Any child with severe shock may develop characteristics of myocardial dysfunction and maldistribution of blood flow and capillary leak with relative hypovolemia.

For a more comprehensive discussion of shock by etiology, refer to Part 7: Recognizing Shock.

Managing the following types of shock is discussed in this section:

- Hypovolemic
- Distributive

- Cardiogenic
- Obstructive

Managing Hypovolemic Shock

Rapid administration of isotonic crystalloids is the primary therapy for hypovolemic shock. Children with hypovolemic shock who receive an appropriate volume of fluid within the first hour after resuscitation have the best chance for survival and recovery. Timely administration of fluid is key to preventing deterioration from compensated hypovolemic shock to hypotensive and refractory shock.

Critical Concepts

Timely Fluid Resuscitation in Hypovolemic Shock

It is important to provide rapid, adequate fluid resuscitation for hypovolemic shock. Avoid the common errors of inadequate or delayed administration of fluid resuscitation.

Other components in effectively managing hypovolemic shock are

- Identifying the type of volume loss (nonhemorrhagic vs hemorrhagic)
- Replacing volume deficit
- Preventing and replacing ongoing losses (eg, bleeding, GI losses)
- Restoring acid-base balance
- Correcting metabolic derangements

Determining Adequate Fluid Resuscitation

Dehydration is defined as a loss of water with varying loss of electrolytes leading to a hypertonic (hypernatremic), isotonic, or hypotonic (hyponatremic) state. The losses can be from some combination of the interstitial, intracellular, and intravascular compartments; the relative loss from each component helps determine clinical symptoms. Severity of dehydration is generally related to the percentage of total body water loss (ie, percent dehydration), but the percentage is not consistent across all age groups because the relative proportion of fluid loss based on total body weight is size dependent.

Adequate fluid resuscitation in hypovolemic shock is determined by the

- Extent of volume depletion
- Type of volume loss (eg, blood, electrolyte-containing fluid, electrolyte-and-protein–containing fluid)

The extent of volume depletion may be underestimated and undertreated. In many cases, volume loss is compounded by inadequate fluid intake. Use vital signs and physical examination to assess the child's response to each fluid bolus. The clinical parameters used to help determine the percentage of dehydration include

- General appearance
- Presence or absence of tears and appearance of eyes (normal vs sunken)
- Moisture of mucous membranes
- Skin elasticity (ie, skin turgor)
- Respiratory rate and depth
- Heart rate
- Blood pressure
- Capillary refill time
- Urine output
- Mental status

Clinically significant dehydration in children is generally associated with at least 5% volume depletion (ie, 5% or greater loss in body weight) corresponding to a fluid deficit of 50 mL/kg or greater. Therefore, treating a child with clinically evident dehydration with administration of a single 20 mL/kg bolus of isotonic crystalloid may be insufficient. Conversely, it is usually unnecessary to completely correct the estimated deficit within the first hour. After perfusion is restored and the child is no longer in shock, the total fluid deficit may be corrected over the next 24 to 48 hours.

Although all forms of hypovolemic shock are initially treated with rapid infusion of isotonic crystalloid, early identification of the type of volume loss can optimize further treatment. Fluid losses may be classified as nonhemorrhagic and hemorrhagic. Nonhemorrhagic losses include electrolyte-containing fluids (eg, diarrhea, vomiting, osmotic diuresis associated with DKA) and protein-and-electrolyte–containing fluids (eg, losses associated with burns and peritonitis).

Nonhemorrhagic Hypovolemic Shock

Common sources of nonhemorrhagic fluid loss are gastrointestinal (ie, vomiting and diarrhea), urinary (eg, diabetes insipidus), and capillary leak (eg, burns). Hypovolemia caused by nonhemorrhagic fluid loss is generally classified in terms of percent loss of body weight (Table 48). Correlation of blood pressure and fluid deficits is imprecise. As a general rule, however, shock may be observed in children with fluid deficits of 50 to 100 mL/kg (particularly in hyponatremic dehydration), but it is more consistently observed with deficits of 100 mL/kg or greater.

Table 48.Stages and Signs of Dehydration

Severity of dehydration	Infant estimated weight loss, % (mL/kg)*	Adolescent estimated weight loss, % (mL/kg)*	Clinical signs	Pitfalls in assessment
Mild	5 (50)	3 (30)	• Dry mucous membranes • Oliguria	• Oral mucosa may be dry in chronic mouth breathers • Frequency and amount of urine are difficult to assess during diarrhea, especially with infants and children wearing diapers
Moderate	10 (100)	5-6 (50-60)	• Poor skin turgor • Sunken fontanel • Marked oliguria • Tachycardia • Quiet tachypnea	• Affected by sodium concentration; increased sodium concentration better maintains intravascular volume • Fontanel open only in infants • Oliguria is affected by fever, sodium concentration, and underlying disease
Severe	15 (150)	7-9 (70-90)	• Marked tachycardia • Weak to absent peripheral pulses • Narrow pulse pressure • Tachypnea • Anuria • Hypotension and altered mental status (late findings)	• Clinical signs are affected by fever, sodium concentration, and underlying disease; increased sodium concentration better maintains intravascular volume

*mL/kg refers to the estimated corresponding fluid deficit normalized to body weight.

Modified with permission from Roberts KB. Fluid and electrolytes: parenteral fluid therapy. *Pediatr Rev*. 2001;22(11):380-387.

Rapidly infuse 20 mL/kg boluses of isotonic crystalloid to effectively treat children with hypovolemic shock secondary to dehydration. Failure to improve after at least 3 boluses (ie, 60 mL/kg) of isotonic crystalloid indicates that

- The extent of fluid losses may be underestimated
- The type of fluid replacement may need to be altered (eg, need for colloid or blood)
- There are ongoing fluid losses (eg, occult bleeding)
- Your initial assumption about the etiology of the shock may be incorrect (ie, consider alternative or combined types of shock)

Ongoing fluid losses (eg, diarrhea, burns) must be replaced in addition to correcting existing fluid deficits. Colloid is not routinely indicated as the initial treatment of hypovolemic shock. Albumin and other colloids, however, have been used successfully for volume replacement in children with large “third-space” losses or albumin deficits.

Hemorrhagic Hypovolemic Shock

Hemorrhagic hypovolemic shock is classified according to an estimated percent of total blood volume loss (Table 49).

Table 49.Systemic Responses to Blood Loss in Pediatric Patients

System	Mild blood volume loss (<30%)	Moderate blood volume loss (30%-45%)	Severe blood volume loss (>45%)
Cardiovascular	Increased heart rate; weak, thready peripheral pulses; normal systolic blood pressure (80-90 + 2 × age in years); normal pulse pressure	Markedly increased heart rate; weak, thready central pulses; low normal systolic blood pressure (70-80 + 2 × age in years); narrowed pulse pressure	Tachycardia followed by bradycardia; very weak or absent central pulses; absent peripheral pulses; hypotension (<70 + 2 × age in years); narrowed pulse pressure (or undetectable diastolic blood pressure)
Central nervous system	Anxious; irritable; confused	Lethargic; dulled response to pain*	Comatose
Skin	Cool, mottled; prolonged capillary refill	Cyanotic; markedly prolonged capillary refill	Pale and cold
Urine output‡	Low to very low	Minimal	None

*The child’s dulled response to pain with this degree of blood loss (30% to 45%) may be indicated by a decreased response to IV catheter insertion.

[‡]After initial decompression by urinary catheter. Low normal is 2 mL/kg per hour (infant), 1.5 mL/kg per hour (younger child), 1 mL/kg per hour (older child), and 0.5 mL/kg per hour (adolescent).

Reproduced from American College of Surgeons Committee on Trauma. Pediatric trauma. In: *Advanced Trauma Life Support for Doctors: ATLS Student Course Manual*. 10th ed. American College of Surgeons; 2018:249.

In children, the dividing line between mild and compensated vs moderate or severe hypotensive hemorrhagic shock is thought to correlate with an acute loss of about 30% of blood volume. The estimated total blood volume of a child is 75 to 80 mL/kg; a 30% blood volume loss, therefore, represents a blood loss of about 25 mL/kg.

Fluid resuscitation in hemorrhagic shock begins with rapid infusion of isotonic crystalloid in boluses of 20 mL/kg. Because isotonic crystalloids are distributed throughout the extracellular space, it may be necessary to give up to 3 boluses of 20 mL/kg (60 mL/kg) of fluid to replace a 25% loss of blood volume; approximately 3 mL of crystalloid is needed for every 1 mL of blood lost. If the child remains hemodynamically unstable despite 2 to 3 boluses of 20 mL/kg isotonic crystalloid, PRBCs should be administered. If ongoing administration of PRBCs is needed, platelets and fresh frozen plasma should be strongly considered to avoid coagulopathy associated with multiple PRBC infusions.

Critical Concepts

3 mL to 1 mL Rule

For fluid resuscitation in hemorrhagic shock, give about 3 mL of isotonic crystalloid for every 1 mL of blood lost.

For blood replacement, use PRBCs in 10 to 20 mL/kg boluses. Whole blood (20 mL/kg) can be given in place of PRBCs, but it is harder and more time-consuming to obtain. The risk of transfusion reaction is significantly increased if the blood is not crossmatched. Warm the blood if a blood-warming device is available, especially when transfusing rapidly, to minimize adverse effects.

Indications for transfusion in hemorrhagic shock include

- Crystalloid-refractory hypotension or poor perfusion
- Known significant blood loss

Crystalloid-refractory hemorrhagic shock is defined as persistent hypotension despite administration of 40 to 60 mL/kg crystalloid. Children with rapid hemorrhage may demonstrate a normal or low initial hemoglobin concentration. Transfuse blood for a low hemoglobin concentration because anemia increases the risk of tissue hypoxia from inadequate arterial O_2 content and O_2 delivery.

Medication Therapy

Vasoactive agents are not routinely indicated for managing hypovolemic shock. Moribund children with profound hypovolemic shock and hypotension may require short-term administration of vasoactive agents, such as epinephrine, to restore cardiac contractility and vascular tone once adequate fluid resuscitation is provided.

Acid-Base Balance

Early in the progression of hypovolemic shock, the child may develop tachypnea and respiratory alkalosis. However, the alkalosis does not completely correct the metabolic (lactic) acidosis produced by hypovolemic shock. A child with long-standing or severe shock may have severe acidosis because the child eventually develops fatigue or cardiorespiratory failure. Children with head or chest injuries may not demonstrate compensatory tachypnea.

Persistent acidosis and poor perfusion are indications of inadequate resuscitation or, in hemorrhagic shock, of ongoing blood loss. Sodium bicarbonate is not recommended for treating metabolic acidosis secondary to hypovolemic shock. As long as fluid resuscitation improves perfusion and end-organ function, metabolic acidosis is well tolerated and will correct gradually. Sodium bicarbonate administration is indicated if the metabolic acidosis is caused by significant bicarbonate losses from renal or gastrointestinal losses (ie, a non–anion gap metabolic acidosis) because it is difficult to compensate for an ongoing bicarbonate loss.

Specific Treatment Considerations

Follow the initial management principles listed in the Summary: Initial Management Principles section in addition to these considerations specific to hypovolemic shock:

- Initiate fluid resuscitation as quickly as possible.
    - –In all patients, rapidly infuse isotonic crystalloid (normal saline or lactated Ringer's) in 20 mL/kg boluses; repeat as needed.
    - –In patients with crystalloid-refractory hemorrhagic shock, give a transfusion of PRBCs, 10 to 20 mL/kg.
    - –If loss of protein-containing fluids is documented or suspected (low albumin concentration), consider administration of colloid-containing fluids if the child fails to respond to crystalloid resuscitation.
- Correct metabolic derangements.
- Identify the type of volume loss (hemorrhagic or nonhemorrhagic) to determine best treatment.
- Control any external hemorrhage with direct pressure; measure and replace ongoing losses (eg, continued nonhemorrhagic losses with diarrhea).
- Consider other laboratory studies:

- –Complete blood cell count
- –Type and crossmatch
- –ABG with particular attention to the base deficit
- –Electrolyte panel to calculate anion gap, glucose, and ionized calcium
- –Serum or plasma lactate concentration
- –Diagnostic imaging to identify the source of bleeding or volume loss

Managing Distributive Shock

Initial management of distributive shock focuses on expanding intravascular volume to correct hypovolemia and fill the expanded dilated vascular space. Use vasoactive agents if the child remains hypotensive or poorly perfused despite rapid bolus fluid administration or if the diastolic pressure remains low with a wide pulse pressure.

This section discusses managing the following types of distributive shock:

- Septic shock
- Anaphylactic shock
- Neurogenic shock

Managing Septic Shock

The clinical, hemodynamic, and metabolic changes observed in septic shock result from the host's response to an infection, including the release or activation of inflammatory mediators. The primary goals in the initial management of septic shock are

- Restoration of hemodynamic stability
- Support of organ function
- Identification and control of infection

Fundamental principles of management include increasing tissue O_2 delivery by optimizing cardiac output and arterial O_2 content and minimizing O_2 consumption.

Identifying septic shock early is key to initiating resuscitation and preventing multisystem organ failure and cardiac arrest from developing. Hemodynamic support to maintain O_2 delivery can reduce pediatric morbidity and mortality from septic shock.

Overview of Pediatric Septic Shock Algorithm

The recommended treatment approach to restore hemodynamic stability for septic shock in children is presented in the Pediatric Septic Shock Algorithm (Figure 39). It outlines 3 phases requiring rapid recognition, stabilization/resuscitation, and critical care management, as follows:

- Early detection of signs of septic shock, which may include alteration in mental status, heart rate, temperature, and perfusion. Note that systolic or diastolic hypotension may or may not be present.
- Initial stabilization/resuscitation:
    - –Within 10 to 15 minutes after recognizing signs of shock, monitor and support airway, oxygenation, and circulation, monitor heart rate and pulse oximetry, and establish vascular access (IV/O). Initiate fluid resuscitation and give 10 to 20 mL/kg boluses of balanced crystalloid such that 40 to 60 mL/Kg of total fluid is administered over the first hour in settings with intensive care.
    - –Reassess child during and after each fluid bolus. Stop rapid fluid bolus administration if vital signs fail to improve or if rales, respiratory distress, or hepatomegaly develops. If additional boluses are needed to treat shock, it may be necessary to administer smaller boluses over longer intervals.
    - –Within the first hour after onset of shock, administer antibiotics, give fluid boluses (repeating, as needed, to correct shock), and initiate vasoactive medication therapy if shock persists despite administration of fluid boluses.
    - –Critical care: If signs of shock persist, critical care expertise is needed to establish hemodynamic monitoring, administer initial vasoactive medication therapy, and titrate additional medication therapy, as needed, to treat shock.

Once shock has been effectively treated, further management includes continued monitoring and support of organ function, treatment of the source of the infection, and evaluation of the child’s clinical course and therapy.

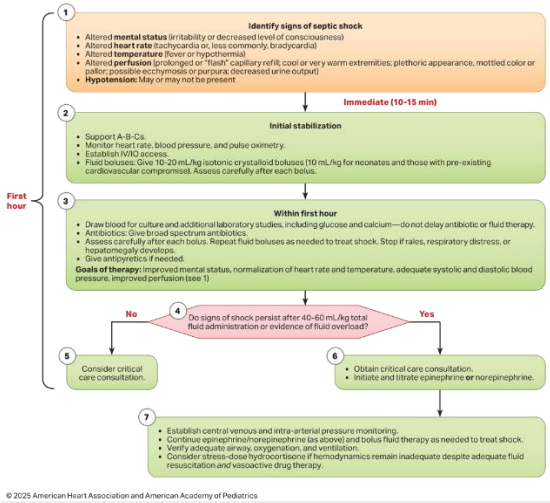


Figure 39. Pediatric Septic Shock Algorithm.

Abbreviation: A-B-C, airway, breathing, and circulation.

Algorithm legend: Red hexagon = decision or question; green box = action/steps to consider; orange box = assessment; gray box = assessment/action; blue box = CPR; white box with gray header = notes; and red, bold text = possible answers or decisions.

Weiss SL, Peters MJ, Alhazzani W, et al. Surviving Sepsis Campaign international guidelines for the management of septic shock and sepsis-associated organ dysfunction in children. *Pediatr Crit Care Med*. 2020;21(2):e52-e106. doi:10.1097/PCC.0000000000002198.

Identify signs of septic shock: Shock in an infant or child can have devastating outcomes. Timely identification of the signs of shock is important to begin treatment.

Initial stabilization: Initial stabilization in the first hour after the onset of symptoms is critical to maximize survival for septic shock. Immediately in the first 10 to 15 minutes:

- Support the ABC's, establish monitoring of the child, and give a fluid bolus of 10 to 20 mL/kg of isotonic crystalloid (10 mL/kg for neonates and those with preexisting heart disease) of isotonic crystalloid.

Within the first hour: Administer antibiotics within 1 hour after first medical contact. Draw labs (if possible, draw a blood sample for culture before antibiotic administration, but do not delay antibiotics). When a child is in shock, considering critical care consultation is prudent. The consultant may suggest initiation of further fluid boluses or pressor therapy such as epinephrine or norepinephrine. They may assist with transfer to a pediatric critical care facility. Once in critical care, other therapies may include intra-arterial monitoring or stress dose hydrocortisone for signs of shock such as poor perfusion or hypotension in spite of fluid resuscitation and vasoactive medication therapy.

Caution With Fluid Resuscitation

When caring for children with severe febrile illness (such as those included in the FEAST trial) in settings with limited access to critical care resources (ie, mechanical ventilation and inotropic support), use extreme caution when administering bolus IV fluids because it may be harmful (refer to Maitland et al. *N Engl J Med*. 2011;364[26]:2483-2495 for details). In the absence of hypotension, start with maintenance IV fluids and with hypotension, bolus with 10 to 20 mL/kg per bolus up to 40 mL/kg in total while reassessing throughout. Consider clinical status thereafter.

The other scenario in which you should exercise caution with fluid boluses is in patients with evidence of fluid overload. Signs and symptoms of fluid overload include hepatomegaly as well as increased respiratory distress and pulmonary edema, such as rales/crackles, which may result from increased vascular permeability. If such signs develop, stop rapid bolus fluid administration. The child requires supplemental O_2 and may require noninvasive positive pressure or even ET intubation and mechanical ventilation with PEEP. If additional fluid boluses are needed, give smaller volumes more slowly (eg, 5-10 mL/kg over 10-20 minutes).

Pulmonary edema may also develop with cardiogenic shock because of poor myocardial contractility and manifest with hepatomegaly and cardiomegaly. In both instances, you may need to reduce the volume and rate of fluid administration.

Anticipate the possible need for vasopressors and stress-dose hydrocortisone. Order these medications from the pharmacy early so that they will be at the bedside. They should be immediately available if the shock is fluid refractory or if adrenal insufficiency is suspected.

Vasopressors can be administered via peripheral venous or IO access, so initiation of vasopressor therapy should not await transfer to a critical care unit. Because delaying initiation of vasoactive support has been associated with increased length of stay and mortality in pediatric septic shock, it is important to begin vasoactive medication therapy within the first hour if shock persists despite bolus fluid administration.

Identify and correct metabolic derangements immediately. Hypoglycemia and ionized hypocalcemia are commonly seen in septic shock and may contribute to myocardial dysfunction.

Managing Fluid-Refractory Septic Shock

Vasoactive medications are appropriate to initiate after 40 to 60 mL/kg of fluid has been administered and the patient continues to have signs/symptoms of compromised perfusion. Vasoactive medications may be appropriate to administer before this fluid threshold if there is evidence of or concern about fluid overload (eg, vital signs fail to improve, rales or hepatomegaly develop, there is a suspicion of cardiogenic shock).

- Administer vasoactive therapy to improve tissue perfusion and blood pressure.
- Either epinephrine or norepinephrine are appropriate as the first vasoactive agent being initiated; there is no evidence to support one over the other based on the patient being in cold shock vs warm shock.
- Despite the lack of evidence, the majority of the expert authors of the 2020 Surviving Sepsis Campaign International Guidelines preferred epinephrine as the initial vasoactive agent in patients with low cardiac output, myocardial dysfunction, or both. For patients in septic shock with low SVR, norepinephrine was the initial vasoactive agent of choice among this panel of experts.
- Both epinephrine and norepinephrine are preferable to dopamine in pediatric septic shock. Administer additional fluid boluses of 10 to 20 mL/kg isotonic crystalloid as deemed clinically appropriate on the basis of the patient's clinical assessment.
- Consider ET intubation and early assisted ventilation with supplemental O_2 and PEEP as needed.
- If the patient continues with hypotension, inadequate perfusion, or both, despite high-dose catecholamine infusion(s), consider initiating a vasopressin infusion.

After initial stabilization/resuscitation, including administration of antibiotics, fluid boluses, and vasoactive therapy within the first hour of care, evaluate heart rate, blood pressure, and peripheral perfusion to determine the next intervention. If heart rate, blood pressure, and perfusion are returning to normal, arrange admission or transfer to an appropriate pediatric facility. Initiate consultation with a pediatric critical care unit or transport team. If the child remains hypotensive or poorly perfused, proceed to the next level in the algorithm. Continue fluid and vasoactive therapy while preparing for admission or transfer.

Critical Care of Septic Shock

Once critical care expertise is available for the child with persistent shock despite initial fluid bolus and vasoactive medication therapy, additional hemodynamic monitoring can be established and additional vasoactive medications and fluid boluses can be provided.

- Establish intra-arterial pressure monitoring.
- Assess the child's heart rate, blood pressure, and perfusion and consider advanced hemodynamic monitoring, including invasive arterial blood pressure monitoring. Analyze laboratory data (eg, serum glucose, electrolytes, end-organ function, lactate).
- Continue epinephrine/norepinephrine and add additional vasoactive medications as needed.
- Provide fluid boluses as needed to treat shock; assess the child frequently and after each fluid bolus.
- Consider stress-dose hydrocortisone (particularly if the child has been on chronic systemic steroids). Cortisol levels are not routinely indicated or recommended.

It is not always clear from the physical examination whether a child has vasodilation or vasoconstriction. For example, some children with cool extremities may have vasodilation but be poorly perfused because of low stroke volume and poor cardiac function. The reasons for specific medication selection according to type of shock are described later in this manual.

While managing septic shock, it is important to monitor and support, as needed, organ function, serum electrolytes, acid-base balance, and lactate, glucose, and calcium concentrations. In titrating medication and fluid therapy, goals of care are to improve the child's lactate, blood pressure, and organ perfusion.

Correction of Adrenal Insufficiency

A child in septic shock with fluid-refractory and epinephrine- or norepinephrine-resistant shock may have adrenal insufficiency. If you suspect adrenal insufficiency, or a patient is at known risk for adrenal insufficiency (ie, history of steroid use), give hydrocortisone 1 to 2 mg/kg IV bolus early. Obtain expert consultation for any additional evaluation and management.

Therapeutic End Points

Titrate vasoactive agents in septic shock to therapeutic end points, including

- Normalized heart rate (declining from very rapid rate toward normal)
- Adequate blood pressure
- Good peripheral pulses and perfusion with capillary refill 2 seconds or less
- Improved level of consciousness/responsiveness
- Appropriate urine output
- Improving lactate concentration

Strict adherence to end points is recommended to avoid excessive vasoconstriction in key organs.

Poststabilization Care

Once signs of shock are resolved, the child requires continued monitoring and support of organ function. The source infection must be treated appropriately. In addition, the health care team should evaluate the cause of the infection to determine if it could have been prevented (and how) and the speed of the recognition of the septic shock (could it have been improved), and identify any potential improvements that could be made to increase the efficiency and effectiveness of shock resuscitation for future patients with septic shock.

Managing Anaphylactic Shock

Managing anaphylactic shock focuses on treating life-threatening cardiopulmonary problems and reversing or blocking the mediators released as part of the uncontrolled allergic response. Primary therapy is administration of epinephrine to reverse the effects of histamine and other allergic mediators. The epinephrine dose may be repeated if needed. Angioedema (tissue swelling resulting from a marked increase in capillary permeability) may result in complete upper airway obstruction, so health care professionals should anticipate the need for very early airway intervention with assisted ventilation and intubation. The epinephrine may prevent or reverse hypotension, and fluid resuscitation may also be effective in restoring blood pressure and supporting effective perfusion.

Specific Treatment Considerations

Consider the initial general management of shock outlined in the Summary: Initial Management Principles section in addition to the following specific treatments for anaphylactic shock:

- Place the patient supine, administer oxygen, and maintain airway.
- Epinephrine: first-line treatment
    - –IM epinephrine 0.01 mg/kg every 5 to 15 minutes for up to 3 injections if patient is not responding (maximum dose: 0.3 mg in a prepubertal child and up to 0.5 mg in a teenager or older patient) or epinephrine by autoinjector (pediatric or adult, depending on the child's size) is the most important agent for the treatment of anaphylaxis.
    - –A second dose of IM epinephrine or an epinephrine infusion may be needed after 10 to 15 minutes in severe anaphylaxis. Frequently, a low-dose infusion (less than 0.05 mcg/kg per minute) is effective.
- Administer isotonic crystalloid fluid boluses as needed to support circulation.
- Albuterol
    - –Administer albuterol as needed for bronchospasm by MDI, intermittent nebulizer, or continuous nebulizer.

Adjunct Treatments

- Antihistamines
    - –H1 blocker (ie, diphenhydramine)
    - –Consider an H2 blocker (eg, famotidine)
- Corticosteroids
    - –Methylprednisolone or equivalent corticosteroid

Observation is indicated for identification and treatment of late-phase symptoms. Late-phase symptoms may occur in 25% to 30% of children several hours after acute-phase symptoms. The likelihood of late-phase symptoms increases in proportion to the severity of acute-phase symptoms.

Managing Neurogenic Shock

Children with neurogenic shock typically present with hypotension, bradycardia, and sometimes hypothermia. Minimal response to fluid resuscitation is commonly observed. Blood pressure is characterized by a low diastolic blood pressure with a wide pulse pressure because of loss of vascular tone. Children with spinal shock may be more sensitive to variations in ambient temperature and may require supplemental warming or cooling.

Specific Treatment Considerations

The initial management principles for shock outlined in the Summary: Initial Management Principles section may be considered in addition to the following specific treatments for neurogenic shock:

- Position the child flat or head down to improve venous return.
- Administer a trial of fluid therapy (isotonic crystalloid) and assess response.
- For fluid-refractory hypotension, use vasopressors (eg, norepinephrine, epinephrine) as indicated.
- Provide supplemental warming or cooling as needed.

Managing Cardiogenic Shock

Cardiogenic shock is a condition of inadequate tissue perfusion that results from myocardial dysfunction. Initially, cardiogenic shock may resemble hypovolemic shock, so identifying a cardiogenic etiology may be difficult. If you suspect cardiogenic shock, consider a slow administration (10-20 minutes) of a relatively small fluid bolus (ie, 5-10 mL/kg) while carefully monitoring the child for response. Cardiogenic shock is probably present if the child does not improve or remains unchanged, the child's respiratory function deteriorates, or the child develops signs of pulmonary edema. Evidence of venous congestion (eg, elevated central venous pressure, distended jugular veins, hepatomegaly) and cardiomegaly (on chest x-ray) are also suggestive of a cardiac etiology of shock.

Main Objectives

A main objective in managing cardiogenic shock is to improve the effectiveness of cardiac function and cardiac output by increasing the efficiency of ventricular ejection. Another main objective is to minimize metabolic demand.

Many children with cardiogenic shock have high preload and do not require additional fluid therapy. Others may require a cautious fluid bolus to increase preload. The most effective way to increase stroke volume in these patients is to reduce afterload (SVR) rather than give an inotropic agent. Inotropes may increase cardiac contractility but will also increase myocardial O_2 demand. However, children who are already hypotensive may require fluid therapy and inotropic support before they will tolerate afterload reduction. Specific management includes

- Early consultation with an expert in pediatric cardiology and/or pediatric critical care
- Cautious fluid administration and monitoring
- Laboratory and other diagnostic studies
- Medications
- Mechanical circulatory support

Consult a pediatric critical care or pediatric cardiology specialist at the earliest opportunity. This will help to establish a diagnosis (eg, echocardiogram), guide ongoing therapy, and facilitate direct transfer to definitive care.

Cautious Fluid Administration and Monitoring

Large heart size on the chest x-ray in a child with evidence of shock and poor cardiac output is the hallmark of cardiogenic shock with adequate intravascular volume. Obtain an echocardiogram for more objective and accurate data about preload and cardiac function.

Do not give large or rapid fluid boluses to a child with cardiogenic shock. The boluses can worsen heart function and increase fluid in the lungs. If objective data or the child's history (eg, vomiting, poor intake) are consistent with inadequate preload, you may give a small fluid bolus cautiously. Remember the following:

- Give small isotonic fluid boluses (5-10 mL/kg)
- Give fluid boluses over relatively longer periods of time (eg, 10-20 minutes)
- Monitor the child closely during fluid infusion
    - –Assess respiratory function frequently
    - –Watch for development of pulmonary edema and deterioration in pulmonary function

Give supplemental O_2. Be prepared to provide assisted ventilation. Noninvasive positive pressure support may reduce the need for mechanical ventilation by decreasing the work of breathing and improving oxygenation.

In the intensive care unit, consider establishing central venous access to facilitate measurement of central venous pressure as an index of preload status and to assess right ventricular end-diastolic pressure and to provide access for multiple infusions.

Central venous access also allows monitoring of $ScvO_2$ as an objective measurement of the adequacy of O_2 delivery relative to metabolic demand.

Laboratory and Other Diagnostic Studies

Obtain laboratory studies to assess the impact of shock on end-organ function. No single laboratory study is completely sensitive or specific for cardiogenic shock. Appropriate studies often include

- A blood gas to determine the magnitude of metabolic acidosis and adequacy of oxygenation and ventilation
- Hemoglobin concentration to ensure that O_2-carrying capacity is adequate
- Lactate concentration and $ScvO_2$ as indicators of the adequacy of O_2 delivery relative to metabolic demand
- Cardiac enzymes (eg, creatine kinase, myocardial, troponin) and thyroid function tests

Other useful studies include the following:

- Chest x-ray: provides information about cardiac size, pulmonary vascular markings, pulmonary edema, and coexistent pulmonary pathology
- ECG: may detect arrhythmia, myocardial injury, ischemic heart disease, or evidence of medication or drug toxicity
- Echocardiogram: may be diagnostic, revealing congenital heart disease, poor cardiac function, or valvular dysfunction; also provides objective measurement of ventricular chamber volume (ie, preload) and function

Medications

If the child is normotensive, medication therapy consists of diuretics and vasodilators. Diuretics are indicated when the child has evidence of pulmonary edema or systemic venous congestion. Vasodilators are typically given by continuous infusion. Discuss with an expert early.

Children with cardiogenic shock may require medications to increase cardiac output by improving contractility. Most also require agents to reduce peripheral vascular resistance. For a detailed discussion of these agents, refer to Medication Therapy in in this Part.

Increased metabolic demand, particularly increased myocardial O_2 demand, plays a role in the vicious cycle of cardiogenic shock. Reducing metabolic demand is a critical component in managing cardiogenic shock. Use ventilatory support and antipyretics to reduce metabolic demand. Analgesics and sedatives reduce O_2 consumption but also reduce the endogenous stress response that is helping redistribute blood flow to compensate for low cardiac output. Give these agents in consultation with pediatric critical care or pediatric cardiology physicians and in small doses. Monitor the child carefully for evidence of potential respiratory depression or hypotension.

Mechanical Circulatory Support

Children with cardiogenic shock who do not respond to medical management may benefit from mechanical circulatory support if the cause of shock is potentially reversible. Extracorporeal life support can provide temporary maintenance of cardiac output, oxygenation, and ventilation while the underlying cause of cardiopulmonary failure is treated. Forms of extracorporeal life support include ECMO and ventricular assist devices. One specific etiology of cardiogenic shock for which ECMO may be considered is acute fulminant myocarditis with high risk of imminent cardiac arrest. Extracorporeal life support is usually available only in tertiary pediatric centers that have the resources and expertise to manage children with acute cardiopulmonary failure.

Specific Treatment Considerations

Follow the initial management principles for shock outlined in the Summary: Initial Management Principles section in addition to the following considerations specific to cardiogenic shock:

- Administer supplemental O_2 and consider need for noninvasive positive pressure.
- Give 5 to 10 mL/kg isotonic crystalloid infusion slowly (over 10-20 minutes); repeat as needed.
- Assess frequently for pulmonary edema.
- Be prepared to assist ventilation.
- Obtain expert consultation early.
- Order laboratory and other studies to determine underlying cause and degree of cardiac and end-organ dysfunction.
- Administer pharmacologic support (eg, vasodilators, phosphodiesterase enzyme inhibitors, inotropes, analgesics, antipyretics).
- Consider mechanical circulatory support.

Patients with cardiogenic shock may need to be intubated and receive mechanical ventilation. Health care professionals should anticipate hemodynamic instability and cardiac arrest with induction. This would ideally take place at a center with mechanical support capabilities. Discuss with an expert early.

Managing Obstructive Shock

Managing obstructive shock is specific to the type of obstruction. This section discusses managing

- Cardiac tamponade
- Tension pneumothorax
- Ductal-dependent congenital heart lesions

- Massive pulmonary embolism

Main Objectives

The early clinical presentation of obstructive shock may resemble hypovolemic shock. A reasonable initial approach may include administering a fluid challenge (10-20 mL/kg isotonic crystalloid) and assessing for response. Because children with obstructive shock can rapidly progress to cardiopulmonary failure and then cardiac arrest, immediately identifying obstructive shock by using the secondary assessment and diagnostic assessments is critical to effective treatment. The main objectives in managing obstructive shock are to correct the cause of cardiac output obstruction and restore tissue perfusion.

General Management Principles

In addition to considerations specific to the etiology of the obstruction, follow the initial management principles outlined in the section Fundamentals of Shock Management.

Specific Management of Cardiac Tamponade

Cardiac tamponade is caused by accumulating fluid, blood, or air in the pericardial space, limiting systemic venous return, impairing ventricular filling, and reducing cardiac output. Favorable outcome requires rapidly identifying and immediately treating tamponade. Children with cardiac tamponade may improve temporarily with small-volume fluid administration to augment cardiac output and tissue perfusion until pericardial drainage can be performed.

Consult appropriate specialists (eg, pediatric critical care, pediatric cardiology, pediatric surgery) early. Elective pericardial drainage (pericardiocentesis), often guided by echocardiography or fluoroscopy, should be performed by specialists who are trained and skilled in the procedure. Emergency pericardiocentesis may be performed in the setting of impending or actual pulseless arrest when there is a strong suspicion of pericardial tamponade.

Specific Management of Tension Pneumothorax

Tension pneumothorax is characterized by accumulating air under pressure in the pleural space with compression of the lung on the involved side and mediastinal shift to the other side of the chest. This prevents the lungs from expanding properly and applies pressure on the heart and great veins, compromising venous return and cardiac output. Favorable outcome depends on immediate diagnosis and urgent treatment. A tension pneumothorax should be identified through clinical examination, and treatment should not await confirmation with a chest radiograph.

Treatment of a tension pneumothorax is immediate needle decompression followed by chest tube placement as soon as possible. A trained professional can quickly perform an emergency needle decompression by inserting an 18- to 20-gauge

over-the-needle catheter over the top of the child's third rib (second intercostal space) in the midclavicular line. A gush of air is a sign that needle decompression has been successful. This indicates relief of pressure buildup in the pleural space. A chest tube can be placed by trained professionals thereafter.

Specific Management of Ductal-Dependent Lesions

Ductal-dependent lesions are a group of congenital cardiac abnormalities. These abnormalities result in pulmonary or systemic blood flow that must pass through an open/patent ductus arteriosus. The infant can deteriorate rapidly once the ductus begins to close during the first days or weeks of life.

Congenital heart defects with ductal-dependent pulmonary blood flow usually have a severe obstruction to pulmonary blood flow such that the ductus arteriosus supplies all or most blood flow into the lungs. When the ductus begins to close, the infant initially becomes hypoxemic and cyanotic, which can progress to shock if not recognized and intervened upon early.

Congenital heart defects with ductal-dependent systemic blood flow usually consist of an obstruction to systemic outflow such that all or most is through the ductus arteriosus. In these patients, when the ductus starts to close, signs of shock develop with severe deterioration in systemic perfusion.

For any infant with ductal-dependent pulmonary or systemic blood flow, immediate treatment with continuous infusion of prostaglandin E_1 can restore ductal openness/patency and may be lifesaving.

Other management actions for ductal-dependent obstructive lesions are

- Ventilatory support with O_2 administration
- Expert consultation to direct therapy
- Echocardiography to establish diagnosis and guide therapy
- Administration of inotropic agents to improve myocardial contractility
- Judicious administration of fluids to improve cardiac output
- Correction of metabolic derangements, including metabolic acidosis

Specific Management of Massive Pulmonary Embolism

Massive pulmonary embolism is a sudden block in the main or large-branch pulmonary artery. This block is usually caused by a blood clot that has traveled to the lungs from another part of the body. The block also can result from other substances, including fat, air, amniotic fluid, catheter fragment, or injected matter. Blood flow through the pulmonary circulation to the left side of the heart is obstructed, resulting in decreased left ventricular filling and inadequate cardiac output.

Initial treatment is supportive, including O_2 administration, ventilatory assistance, and fluid therapy if the child is poorly perfused. Consult a specialist who can perform echocardiography, a computed tomography scan with IV contrast, or angiography to confirm the diagnosis. Anticoagulants (eg, heparin, enoxaparin) are the definitive treatment for most children with pulmonary embolism who are not in shock. Because anticoagulants do not act immediately to relieve obstruction, consider thrombolytic agents (eg, recombinant tissue plasminogen activator, systemically or by image-guided direct thrombolysis) in children with severe cardiovascular compromise. Obtain expert consultation early upon the presentation of the patient as some may need interventional or surgical management.

Computed tomography angiography (specified by local protocol for PE) is the diagnostic assessment of choice because it can be rapidly obtained and does not require an invasive angiogram. Additional diagnostic studies that might be useful are an ABG, CBC, D-dimer, electrocardiography, chest x-ray, ventilation-perfusion scan, and echocardiography.

Resources for Managing Circulatory Emergencies

IO Access

IO cannulation is a relatively simple and effective method of rapidly establishing vascular access for emergency fluids or medications. It provides access to a noncollapsible marrow venous plexus, serving as a safe and reliable route for administering medications, crystalloids, colloids, and blood during resuscitation. One can achieve IO access in children of all ages, often in about 30 to 60 seconds. In certain circumstances (eg, cardiac arrest or severe shock with severe vasoconstriction), it may be the initial vascular access attempted. Any medication that can be administered IV can be given by the IO route. Fluids and medications delivered via an IO catheter can reach the central circulation within seconds. If you cannot readily obtain peripheral vascular access in a child with compensated or hypotensive shock, be prepared to establish IO access as soon as it is needed.

Sites for IO Access

Many sites, such as the proximal tibia (just below the growth plate), are appropriate for IO infusion. Other sites include the distal tibia (just above the medial malleolus), the distal femur, proximal humerus, and the anterior-superior iliac spine. IO drills are approved for use in infants, children, adolescents, and adults.

Contraindications

Contraindications to IO access include

- Fractures and crush injuries near the access site

- Conditions with fragile bones (eg, osteogenesis imperfecta)
- Previous attempts to establish IO access in the same bone

Avoid IO cannulation if an infection is present in the overlying tissues.

Procedure (Proximal Tibia)

Use the following procedure to establish IO access:

1. To establish access in the proximal tibia, position the leg with slight external rotation. Identify the tibial tuberosity just below the knee joint; the insertion site is the flat part of the tibia, about 1 to 3 cm (about 1 finger's width) below and medial to this bony prominence (Figure 40). Always use universal precautions when attempting vascular access. Disinfect the overlying skin and surrounding area.
2. Leave the stylet in the needle during insertion to prevent the needle from becoming clogged with bone or tissue. Stabilize the leg on a firm surface; do not place your hand behind the leg. *Note:* If a standard IO needle or bone marrow needle is not available, a large-bore (at least 18-gauge) standard hypodermic needle can be substituted. But the lumen may become clogged with bone or bone marrow during insertion. Short, wide-gauge spinal needles with internal stylets can be used in an emergency, but they tend to bend easily. Use a hemostat to help stabilize the needle during insertion.
3. Insert the needle through the skin over the anteromedial surface of the tibia perpendicular to the tibia; this avoids injury to the growth plate. Use a twisting motion with gentle but firm pressure. Continue inserting the needle through the cortical bone until there is a sudden decrease in resistance as the needle enters the marrow space. If the needle is placed correctly, it should stand easily without support and should not wiggle with gentle prodding.
4. Remove the stylet and attach a syringe. Aspiration of bone marrow and blood into the hub of the needle confirms correct placement; you may send blood to the lab for analysis. (*Note:* Blood or bone marrow may not always be aspirated despite correct needle placement.) Infuse a small volume of saline. It should infuse easily. Check for swelling at the insertion site or posteriorly, behind the insertion site. (Swelling occurs if the needle was inserted too deeply and penetrated through the posterior cortical bone.)
5. To stabilize the needle, place tape over the flange; you also may place gauze padding on both sides of the needle for support. Premanufactured devices are also available.
6. Tape IV tubing to the skin to avoid tension on the tubing that might displace the needle.
7. Infuse fluid with a syringe attached to a 3-way stopcock or by pressure infusion. When using a pressurized fluid bag, make sure no air gets into the system.

8. Any medication that can be administered IV can be given by the IO route, including vasoactive medication infusions such as epinephrine. Follow all bolus medications with a saline flush.

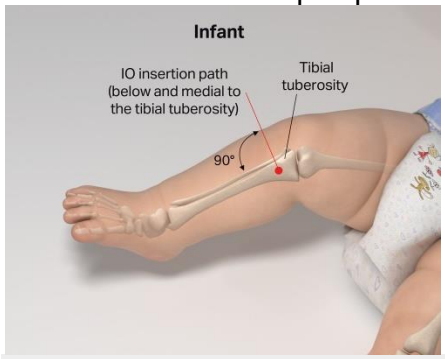


Figure 40A. Locations for IO insertion. A, General landmarks for IO insertion in the leg of an infant.

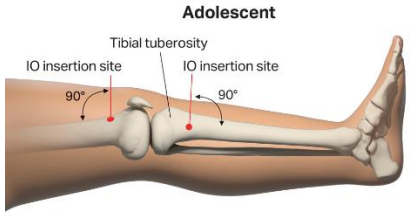


Figure 40B. Locations for IO insertion in the proximal tibia and distal femur in older children.

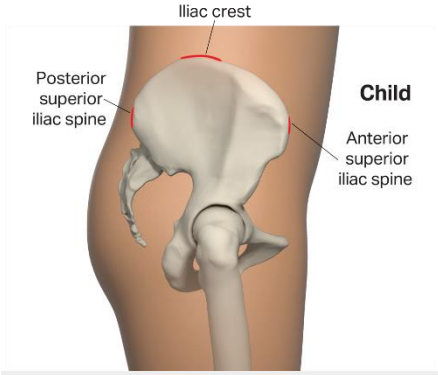


Figure 40C. Location for IO insertion in the iliac crest.

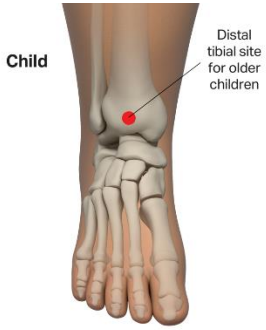


Figure 40D. Location for IO insertion in the distal tibia.

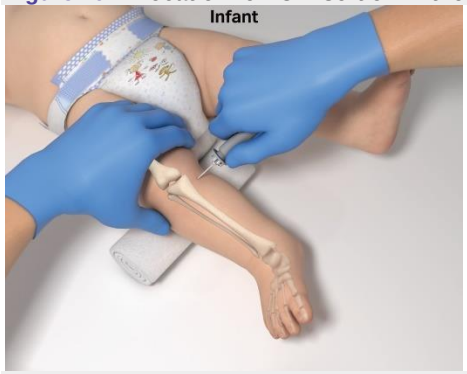


Figure 40E. Technique for immobilizing the leg while twisting the IO needle into the leg of an infant.

After IO Insertion

After inserting an IO needle/catheter, do the following:

- Check the site and the underside of the leg for signs of swelling and IO needle displacement; fluids or medications delivered via a displaced needle can cause severe complications (eg, tissue necrosis, compartment syndrome).

Because IO needles are intended for short-term use (generally less than 24 hours) and are often displaced early in a resuscitation, prioritize ongoing attempts to establish vascular access.

Removal of an IO

To remove an IO needle/catheter, do the following:

- Attach a syringe to the IO needle.

- Gently pull the needle, rotating clockwise if necessary, until the needle is removed.
- Maintain a 90° angle to the bone—avoid rocking or bending the needle during removal.
- Apply pressure to the site for several minutes.

Apply occlusive dressing to site as indicated.

Color-Coded Length-Based Resuscitation Tape

Use a color-coded length-based tape (Table 50) to select the correct size of resuscitation supplies and to determine the child's weight (if not known) for calculating medication doses.

Table 50. Color-Coded Length-Based Resuscitation Tape

Zone	**3 kg** <3 mos	**4 kg** <3 mos	**5 kg** <3 mos	**Pink** 6-7 kg 3-5 mos	**Red** 8-9 kg 6-11 mos	**Purple** 10-11 kg 12-24 mos	**Yellow** 12-14 kg 2 yrs	**White** 15-18 kg 3-4 yrs	**Blue** 19-23 kg 5-6 yrs	**Orange** 24-29 kg 7-9 yrs	**Green** 30-36 kg 10-11 yrs
ETT uncuffed (mm)	vw	3.5	3.5	3.5	3.5	4.0	4.5	5.0	5.5	NA	NA
ETT cuffed (mm)	3.0	3.0	3.0	3.0	3.0	3.5	4.0	4.5	5.0	5.5	6.0
Lip-tip (cm)	9-9.5	9.5-10	10-10.5	10-10.5	10.5-11	11-12	12.5-13.5	14-15	15.5-16.5	17-18	18.5-19.5
Suction (F)	8	8	8	8	8	8	10	10	10	10	12
L-scope blade	1 straight	1 straight	1 straight	1 straight	1 straight	1-1.5 straight	2 straight/curved	2 straight/curved	2 straight/curved	2-3 straight/curved	2-3 straight/curved
Stylet	6 F	6 F	6 F	6 F	6 F	6 F	10 F	10 F	10 F	14 F	14 F

OPA (mm)	50	50	50	50	50	60	60	60	70	80	80
NPA (F)	14	14	14	14	14	18	20	22	24	26	26
Bag-mask device (minimum mL)	450	450	450	450	450	450	450	450-750	750-1000	750-1000	1000
$ETCO_2$ detector	Ped	Ped	Ped	Ped	Ped	Ped	Ped	Adult	Adult	Adult	Adult
LMA	1	1	1	1.5	1.5	2	2	2	2-2.5	2.5	3
Tidal volume (mL)	20-30	24-40	30-50	40-65	50-85	65-105	80-130	100-165	125-210	160-265	200-330
Frequency	20-25/min	20-25/min	20-25/min	20-25/min	20-25/min	15-25/min	15-25/min	15-25/min	12-20/min	12-20/min	12-20/min

Abbreviations: ETT, endotracheal tube; F, French; LMA, laryngeal mask airway; NPA, nasopharyngeal airway; OPA, oropharyngeal airway; Ped, pediatric.

Part 9
Recognizing Arrhythmias

This Part discusses recognizing arrhythmias, such as bradycardia (slow heart rate) and tachycardia (fast heart rate) with a palpable pulse and adequate or inadequate perfusion in infants and children.

Learning Objective

After completing this Part, you should be able to differentiate between unstable and stable patients with arrhythmias.

You will be expected to identify bradycardic and tachycardic rhythms in an infant or child with and without cardiopulmonary compromise.

Bradycardia Definitions

Bradycardia is a heart rate that is slow in comparison with a normal heart rate range for the child's age, level of activity, and clinical condition. Refer to Table 18. Normal Heart Rates in Part 4.

Critical Concepts

Symptomatic Bradycardia and Cardiopulmonary Compromise

Symptomatic bradycardia is a heart rate slower than normal for the child's age (usually less than 60/min) associated with cardiopulmonary compromise.

Cardiopulmonary compromise is defined as hypotension, acutely altered mental status (ie, decreased level of consciousness), and signs of shock.

Bradycardia is an ominous sign of impending cardiac arrest in infants and children, especially if it is associated with hypotension or evidence of poor tissue perfusion. If, despite adequate oxygenation and ventilation, the heart rate is less than 60/min in an infant or child with signs of cardiopulmonary compromise, begin CPR.

Critical Concepts

Evaluating Heart Rate and Rhythm

Consider the following when evaluating the heart rate and rhythm in any seriously ill or injured child:

- The child's typical heart rate and baseline rhythm
- The child's activity level and clinical condition (including baseline cardiac function)

Children with congenital heart disease may have underlying conduction abnormalities. Interpret the child's heart rate and rhythm by comparing them to the child's baseline heart rate and rhythm. Children with poor baseline cardiac function are more likely to become symptomatic from arrhythmias than children with normal cardiac function.

Tissue hypoxia is the leading cause of symptomatic bradycardia in children. Therefore, symptomatic bradycardia in children is usually the result of (rather than the reason for) progressive hypoxemia and respiratory failure or shock. Priorities in initial assessment and management should be to support the airway and adequately oxygenate and ventilate.

Bradycardia may be classified as

- Primary bradycardia
- Secondary bradycardia

Primary bradycardia is the result of congenital or acquired heart conditions that slow the spontaneous depolarization rate of the heart's normal pacemaker cells or slow conduction through the heart's conduction system. Causes of primary bradycardia include

- Congenital abnormality of the conduction system
- Surgical injury to the conduction system
- Cardiomyopathy
- Myocarditis

Secondary bradycardia is the result of noncardiac conditions that alter the normal function of the heart (ie, slow the sinus node pacemaker or slow conduction through the AV junction). Causes of secondary bradycardia include

- Hypoxia
- Acidosis
- Hypotension
- Hypothermia
- Drug or medication effects
- Increased vagal tone (suctioning, gagging, vomiting)

Recognizing Bradycardia

Signs and Symptoms of Bradycardia

Cardiac output (the volume of blood pumped by the heart per minute) is the product of stroke volume (the volume of blood pumped with each ventricular contraction) and heart rate (number of times the ventricles contract per minute):

Cardiac Output = Stroke Volume × Heart Rate

When heart rate decreases, cardiac output can only be maintained by an increase in stroke volume. Because the heart's ability to increase stroke volume is limited during infancy and early childhood, cardiac output typically declines with bradycardia. An extremely slow heart rate results in critically low cardiac output that can be life threatening and lead to cardiopulmonary compromise. The signs of cardiopulmonary compromise associated with bradycardia are

- Hypotension
- Altered mental status: decreased level of consciousness or irritability
- Shock: poor end-organ perfusion with or without hypotension

Additional signs can include

- Respiratory distress or failure
- Chest pain or vague feeling of discomfort in older children
- Sudden collapse

ECG Characteristics of Bradycardia

The ECG characteristics of bradycardia include

- Heart rate: slow compared with normal heart rate for age
- P waves: may or may not be visible
- QRS complex: narrow or wide (depending on the origin of the rhythm and/or location of injury to the conduction system)
- P wave and QRS complex: may be unrelated (ie, AV dissociation) Refer to the Rhythm Recognition Review in the Appendix for examples.

Types of Bradyarrhythmias

Bradycardia, defined earlier in this Part, is a nonspecific term. If a patient has bradycardia due to slowing of the sinus node depolarization rate, the term sinus bradycardia is used.

Bradyarrhythmia is the appropriate term to use when the heart rate is abnormally low and the rhythm is abnormal. Types of bradyarrhythmias include AV block, sinus node arrest, junctional escape rhythms, and ventricular escape rhythms. Only AV block will be reviewed; the other bradyarrhythmias are more complex and are not discussed in the PALS Provider Course.

Sinus Bradycardia

Sinus bradycardia is a sinus node depolarization rate that is slower than normal for the child's age (refer to Table 18. Normal Heart Rates in Part 4). Sinus bradycardia is not necessarily problematic. It is often present in healthy children at rest when metabolic demands of the body are relatively low (eg, during sleep). Well-conditioned athletes often have sinus bradycardia because they have high stroke volume and increased vagal tone. However, sinus bradycardia can also develop in response to hypoxia, hypotension, and acidosis. As discussed above, it is often the result of progressive respiratory failure or shock and indicates impending cardiac arrest. Sinus bradycardia also may result from drug or medication effects. Therefore, evaluating sinus bradycardia must always involve assessing the clinical status of the child.

Rarely, children have a primary bradycardia with an intrinsic disorder of the sinus node that impairs the sinus node to depolarize at an adequate rate. These children usually have a history of surgery for complex congenital heart disease. Additional causes of sinus node disorders include congenital abnormalities of the conduction system, cardiomyopathy, and myocarditis.

AV Block

AV block is a disturbance of electrical conduction through the AV node. AV block is classified as follows and in Table 51:

- First degree: A prolonged PR interval representing slowed conduction through the AV node (Figure 41A)
- Second degree: Block of some, but not all, atrial impulses before they reach the ventricles. This block can be further classified as Mobitz type I or Mobitz type II second-degree AV block.
    - –In Mobitz type I or Wenckebach-type second-degree AV block, there is successive prolongation of the PR interval preceding non-conducted P waves (Figure 41B).
    - –Mobitz type II is identified by intermittently nonconducted P waves, with a constant PR interval on conducted beats (Figure 41C).
- Third degree: None of the atrial impulses conduct to the ventricles. This block may also be referred to as *complete heart block* or *complete AV block* (Figure 41D).

Table 51.AV Block Classification

Type	Causes	Characteristics	Symptoms
First degree	• *Note:* May be present in healthy children • Enhanced vagal tone • Myocarditis • Electrolyte disturbances (eg, hyperkalemia) • Hypoxemia • Myocardial infarction • Cardiac surgery • Medications (eg, calcium channel blockers, β-adrenergic blockers, digoxin) • Acute rheumatic fever • Intrinsic AV nodal disease	Prolonged PR interval with 1:1 P:QRS	Asymptomatic
Second-degree Mobitz type I (Wenckebach phenomenon)	• *Note:* May be present in healthy children • Medications (eg, calcium channel and β-adrenergic blockers, digoxin) • Any condition that stimulates vagal (parasympathetic) tone • Myocardial infarction • Lyme disease	In Mobitz type I or Wenckebach-type second-degree AV block there is successive prolongation of the PR interval preceding nonconducted P waves	Occasionally, may cause presyncope (light-headedness)

Second degree Mobitz type II	• Typically results from intrinsic conduction system abnormalities • Rarely caused by increased parasympathetic tone or medications • Cardiac surgery • Myocardial infarction • Lyme disease	Mobitz type II is identified by intermittently nonconducted P waves with a constant PR interval on conducted beats	May cause • Sensed irregularities of heartbeat (palpitations) • Presyncope (light-headedness) • Syncope
Third degree	• Extensive conduction system disease or injury, including myocarditis • Cardiac surgery • Congenital complete heart block • Myocardial infarction • Can also result from increased parasympathetic tone, toxic drug or medication effects, or severe hypoxia/acidosis • Lyme disease	• No relationship between P waves and QRS complexes • No atrial impulses reach ventricles • Ventricular rhythm maintained by a slower pacemaker	• Most frequent symptoms are • Fatigue • Light-headedness • Syncope

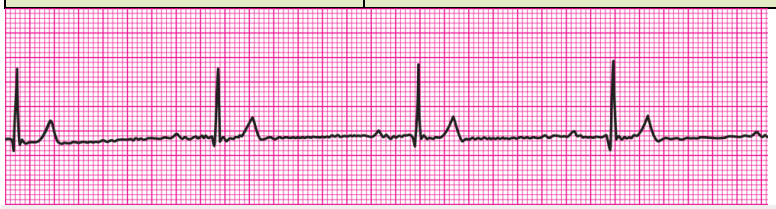


Figure 41A. Examples of AV block. A, Sinus bradycardia with first-degree AV block.

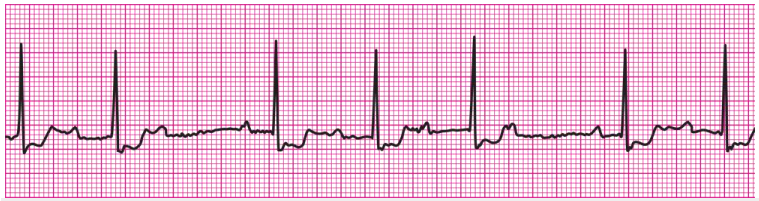


Figure 41B. **Second-degree AV block Mobitz type I (Wenckebach phenomenon).**

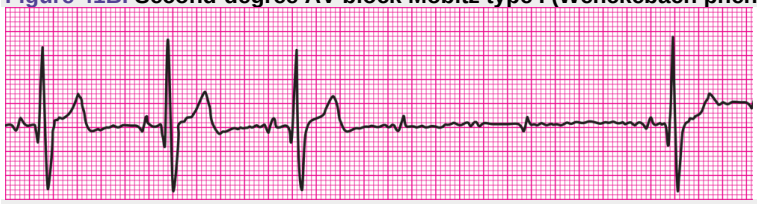


Figure 41C. **Second-degree AV block Mobitz type II.**

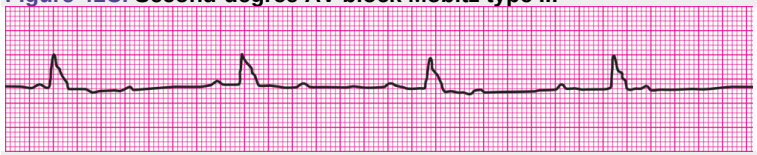


Figure 41D. **Third-degree AV block.**

Tachyarrhythmias

Tachycardia is a heart rate that is fast compared with the normal heart rate for the child's age. Refer to Table 18. Normal Heart Rates in Part 4. ST is a normal response to anxiety, pain, fever, or other physiologic stressors.

Tachyarrhythmias are rapid abnormal rhythms originating either in the atria or the ventricles of the heart. Tachyarrhythmias can be tolerated without symptoms for a variable period of time, especially if cardiac function is good. However, tachyarrhythmias can also cause acute hemodynamic compromise, such as shock or deterioration to cardiac arrest. Rapid deterioration is more likely to occur if cardiac function is poor when the arrhythmia develops.

Recognizing Tachyarrhythmias

Signs and Symptoms

Tachyarrhythmias may cause nonspecific signs and symptoms that differ according to the child's age. Clinical findings may include palpitations, light-headedness, and syncope. In infants who are at home, the tachyarrhythmia may be undetected for long periods (eg, for hours or days) until cardiac output is significantly compromised and the infant develops signs of CHF such

as irritability, poor feeding, and rapid breathing. Episodes of extremely rapid heart rate may be life threatening if cardiac output is compromised. When a tachyarrhythmia develops in a child with poor baseline cardiovascular function, clinical signs can develop quickly and deterioration can be rapid.

Signs of hemodynamic instability associated with tachyarrhythmias are

- Hypotension
- Altered mental status (ie, decreased level of consciousness)
- Signs of shock (poor end-organ perfusion) with or without hypotension

Additional signs may include

- Sudden collapse with rapid, weak pulses
- Respiratory distress/failure

Effect on Cardiac Output

An increased heart rate can produce increased cardiac output, up to a point. If that point is exceeded (ie, the heart rate is extremely rapid), stroke volume decreases because diastole is shortened and there is insufficient time for the ventricles to fill during diastole. Cardiac output then decreases substantially. In addition, coronary perfusion (blood flow to the heart muscle) occurs chiefly during diastole; the decrease in duration of diastole that occurs with a very rapid heart rate reduces coronary perfusion. Finally, a fast heart rate increases myocardial O_2 demand. In infants, prolonged episodes of rapid heart rate (SVT) can cause myocardial dysfunction, leading to CHF. In any child, an extremely rapid heart rate can result in inadequate cardiac output and, ultimately, cardiogenic shock.

Classifying Tachycardia and Tachyarrhythmias

Tachycardia and tachyarrhythmias are classified according to the width of the QRS complex; the arrhythmias are divided into those with narrow (0.09 second or less) vs wide (greater than 0.09 second) QRS complexes. ST, SVT, and atrial flutter are classified as narrow complex, and VT and SVT with aberrant interventricular conduction are classified as wide complex.

Sinus Tachycardia

ST is a sinus node depolarization rate faster than normal for the child's age. It typically develops in response to the body's need for increased cardiac output or O_2 delivery. ST is a normal physiologic response and is not considered an arrhythmia (Figure 42). In ST, the heart rate is not fixed but varies with activity and other factors (eg, the child's sleep/awake state, temperature) that influence O_2 demand.

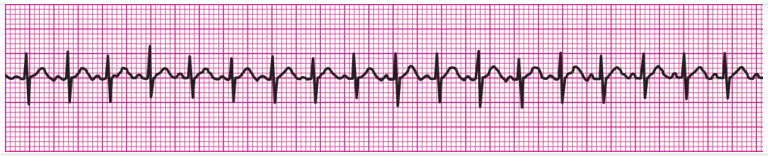


Figure 42. ST (heart rate 180/min) in a febrile 10-month-old infant.

Common causes of ST include exercise, pain, anxiety, tissue hypoxia, hypovolemia (hemorrhagic and nonhemorrhagic fluid loss), shock, fever, metabolic stress, injury, toxins, poisons, medications, and anemia. Cardiac tamponade, tension pneumothorax, and thromboembolism are less common causes of ST.

ECG Characteristics of ST

ECG characteristics of ST include the following:

- Heart rate: beat-to-beat variability with changes in activity or stress level
    - –Usually less than 220/min in infants
    - –Usually less than 180/min in children
- P waves: present/normal
- PR interval: constant, normal duration
- R-R interval: variable
- QRS complex: narrow (0.09 second or less)

Supraventricular Tachycardia

SVT is an abnormally fast rhythm originating above the ventricles. In infants and children, the most common cause is a reentry mechanism that occurs through an accessory pathway or within the AV node. SVT is the most common tachyarrhythmia that causes cardiovascular compromise during infancy. In addition to an accessory pathway reentry or AV nodal reentry, other mechanisms that can cause SVT are atrial flutter and ectopic atrial focus.

Two outdated terms for SVT are *paroxysmal atrial tachycardia* and *paroxysmal SVT*. SVT was labeled paroxysmal because it occurs episodically (in paroxysms). The rapid rhythm starts and stops suddenly, often without warning.

Clinical Presentation of SVT

SVT (Figure 43) is a rapid, regular rhythm that often appears abruptly and may be episodic. During episodes of SVT, cardiopulmonary function is influenced by the child's age, duration of the tachycardia, baseline ventricular function, and ventricular rate. In infants with normal ventricular function, SVT may be present but undetected for long periods (hours or days)

until cardiac output is significantly impaired. However, if baseline myocardial function is impaired (eg, in a child with congenital heart disease or cardiomyopathy), SVT can produce signs of shock in a short time.

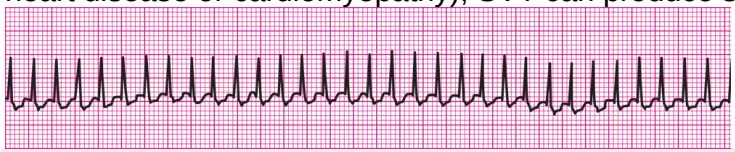


Figure 43. SVT in a 10-month-old infant.

In infants, SVT is often diagnosed when symptoms of CHF develop. Common signs and symptoms of SVT in infants include irritability; poor feeding; rapid breathing; unusual sleepiness; vomiting; and pale, mottled, gray, or cyanotic skin. Common signs and symptoms of SVT in older children include palpitations, shortness of breath, chest pain or discomfort, light-headedness, and fainting.

SVT is initially well tolerated in most infants and older children. However, it can lead to CHF and clinical evidence of shock when baseline myocardial function is impaired (eg, in a child with congenital heart disease or cardiomyopathy) or in an infant having prolonged episodes over hours to days. Ultimately, SVT can cause cardiovascular collapse.

Signs

SVT may be identified as the result of its effect on systemic perfusion. SVT with cardiopulmonary compromise can produce the signs and symptoms listed in Table 52.

Table 52.Signs and Symptoms of SVT With Cardiopulmonary Compromise

Evaluate	Signs and symptoms
Airway	Usually open/patent unless level of consciousness is significantly impaired

Breathing	• Tachypnea • Increased work of breathing • Crackles (or wheezing in infants) if CHF develops • Grunting if CHF develops
Circulation	• Tachycardia beyond the typical range for ST and characterized by fixed rate and/or abrupt onset • Delayed capillary refill time • Weak peripheral pulses • Cool extremities • Diaphoretic, pale, mottled, gray, or cyanotic skin • Hypotension • Jugular venous distension (difficult to observe in young children) or hepatomegaly if CHF develops
Disability	• Altered mental status • Sleepiness or lethargy • Irritability
Exposure	Defer evaluation of temperature until ABCs are supported

ECG Characteristics of SVT

ECG characteristics of SVT include the following:

- Heart rate: no beat-to-beat variability with activity
    - –Usually 220/min or greater in infants
    - –Usually 180/min or greater in children
- P waves: absent or abnormal (may appear after the QRS complex)
- PR interval: because P waves are usually absent, PR interval cannot be determined; in ectopic atrial tachycardia, a short PR interval may be seen
- R-R interval: often constant
- QRS complex: usually narrow; wide complex uncommon

Narrow-QRS-complex SVT: In more than 90% of children with SVT, the QRS complex is narrow (Figure 43), ie, 0.09 second or less.

Wide-QRS-complex SVT: SVT with aberrant conduction (uncommon in the pediatric age group) produces a wide QRS complex (ie, greater than 0.09 second). This form of SVT most often occurs as a result of rate-related bundle branch block (within the

ventricles) or preexisting bundle branch block. It also may be caused by an accessory pathway in which electrical impulses are conducted from the atria to the ventricles through the accessory pathway rather than through the AV node. The impulse then returns to the atria through the AV node (or through a different accessory pathway).

It can be difficult to differentiate SVT with aberrant conduction from VT. This usually requires careful analysis of at least one 12-lead ECG. Both SVT with aberrant conduction and VT can cause similar hemodynamic instability, have similar rates, and have wide QRS complexes (greater than 0.09 second). In the pediatric age group, unless patient history or previous ECGs suggest the likelihood of SVT with aberrant conduction (eg, preexisting bundle branch block), assume that a tachycardia with a wide QRS complex is due to VT.

Comparison of ST and SVT

It may be difficult to differentiate SVT with shock from shock resulting from another etiology with compensatory ST. The characteristics in Table 53 may aid in differentiating ST from SVT. Note that signs of heart failure and other signs and symptoms of poor perfusion may be absent early after the onset of SVT.

Table 53.Characteristics of ST and SVT

Characteristic	ST	SVT
History	Gradual onset; Compatible with ST (eg, history of fever, pain, dehydration, hemorrhage)	Abrupt onset or termination or both Infant: Symptoms of CHF Child: Sudden onset of palpitations
Physical examination	Signs of underlying cause of ST (eg, crackles, fever, hypovolemia, anemia)	Infant: Signs of CHF (eg, rales, hepatomegaly, edema)
Heart rate	Infant: Usually <220/min Child: Usually <180/min	Infant: Usually ≥220/min Child: Usually ≥180/min
Monitor	Variability in heart rate with changes in level of activity or stimulation; slowing of heart rate with rest or treatment of underlying cause (eg, administering IV fluids for hypovolemia)	Minimal or no variability in heart rate with changes in level of activity or stimulation
ECG	P waves present/normal/upright in leads I/aVF	P waves absent/abnormal/inverted (negative) in leads II/III/aVF; if present, they may follow the QRS complex

Chest x-ray	Usually small heart and clear lungs unless ST is caused by pneumonia, pericarditis, or underlying heart disease	Signs of CHF (eg, enlarged heart, pulmonary edema) may be present

P waves may be difficult to identify in both ST and SVT once the ventricular rate exceeds 200/min.

Atrial Flutter

Atrial flutter is a narrow-complex tachyarrhythmia that can develop in newborn infants with normal hearts (Figure 44). It also can develop in children with congenital heart disease, especially after cardiac surgery. A reentrant mechanism typically is present in children with enlarged atria or with anatomic barriers resulting from cardiac surgery (eg, atriotomy scars, surgical anastomoses). A reentry circuit within the atria allows a wave of depolarization to travel in a circle within the atria. Because the AV node is not part of the circuit, AV conduction may be variable. The atrial rate can exceed 300/min, whereas the ventricular rate is slower and may be irregular. Classically, a “sawtooth” pattern of the P waves is present on the ECG.

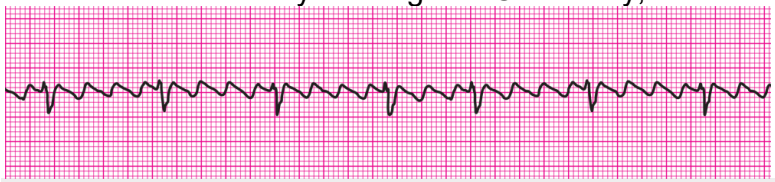


Figure 44. Atrial flutter in an adolescent (atrial rate about 270/min; ventricular rate approximately 70/min).

Ventricular Tachycardia

VT is a wide-QRS-complex tachyarrhythmia generated within the ventricles (Figure 45). VT is uncommon in children. When VT with pulses is present, the ventricular rate may vary from near normal to greater than 200/min. Rapid ventricular rates compromise ventricular filling, stroke volume, and cardiac output and may deteriorate into pVT or VF.

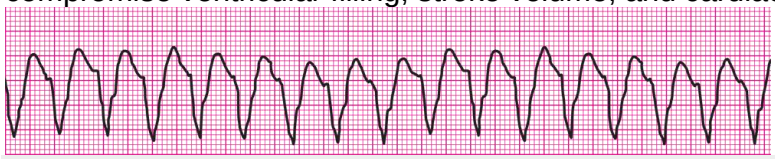


Figure 45A. VT; A, Monomorphic.

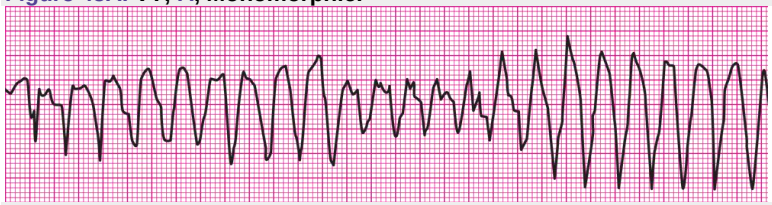


Figure 45B. Polymorphic (torsades de pointes).

Most children who develop VT have underlying heart disease (or have had surgery for heart disease), long QT syndrome, or myocarditis/cardiomyopathy. They may have a family history of a sudden, unexplained death in a child or young adult, suggesting cardiomyopathy or an inherited cardiac ion “channelopathy.” Other causes of VT in children include electrolyte disturbances (eg, hyperkalemia, hypocalcemia, hypomagnesemia), drug toxicity (eg, tricyclic antidepressants, cocaine, methamphetamines), and acute systemic illnesses (eg, severe sepsis) or coronary artery abnormalities (eg, Kawasaki syndrome) causing myocardial ischemia.

ECG Characteristics of VT

ECG characteristics of VT include the following:

- Ventricular rate: at least 120/min and regular
- QRS complex: wide (greater than 0.09 second)
- P waves: often not identifiable; when present, may not be related to QRS (AV dissociation); at slower rates, atria may be depolarized in a retrograde manner, resulting in a 1:1 ventricular-to-atrial association
- T waves: typically opposite in polarity from QRS

It may be difficult to differentiate SVT with aberrant conduction from VT. Fortunately, aberrant conduction is present in less than 10% of children with SVT. In general, the health care professional should initially assume that a wide-complex rhythm is VT unless the child is known to have aberrant conduction or previous episodes of wide-QRS-complex SVT.

Polymorphic VT, Including Torsades de Pointes

VT may be monomorphic (QRS complexes are uniform in appearance) or polymorphic (QRS complexes vary in appearance). If the ventricular rate is slow enough, the patient with monomorphic VT can maintain pulses. In contrast, polymorphic VT is typically associated with loss of pulses at onset or within a very short time after onset. Torsades de pointes is a distinctive form of polymorphic VT. The term *torsades de pointes* is French and means “turning on a point.” In torsades de pointes, the QRS complexes change in polarity and amplitude, appearing to rotate around the ECG isoelectric line (Figure 45B). The ventricular rate can range from 150 to 250/min. Torsades de pointes can be seen in conditions associated with a prolonged QT interval, including congenital long QT syndrome, electrolyte abnormalities, and drug toxicity. The prolonged QT interval is identified during sinus rhythm; it cannot be evaluated during the tachycardia. A rhythm strip may show the child’s baseline QT prolongation because torsades de pointes sometimes occurs in bursts that convert spontaneously to sinus rhythm.

Conditions and agents that predispose to torsades de pointes include

- Long QT syndromes (often congenital and inherited)
- Hypomagnesemia

- Hypokalemia
- Drugs that prolong the QTc (eg, Class IA, quinidine, procainamide, disopyramide; Class IC, flecainide; Class III, sotalol, amiodarone; phenothiazines)

It is important to recognize that VT, especially polymorphic VT (including torsades de pointes), can rapidly deteriorate to VF. The long QT syndromes and other inherited arrhythmia syndromes (ie, channelopathies) are associated with sudden death from either primary VF or torsades de pointes. Polymorphic VT not associated with a prolonged QT interval during sinus rhythm is treated as generic VT.

Part 10
Managing Arrhythmias

This Part discusses managing bradycardia (slow heart rate) with a pulse and cardiopulmonary compromise and tachycardia (fast heart rate) with a palpable pulse and cardiopulmonary compromise. Health care professionals should quickly treat symptomatic arrhythmias before they result in shock or cardiac arrest.

Learning Objective

After completing this Part, you should be able to describe clinical characteristics of instability in patients with arrhythmias.

You will also be expected to manage a child as outlined in the algorithms for bradycardia with a pulse and cardiopulmonary compromise and tachycardia with a pulse and cardiopulmonary compromise.

Principles of Managing Pediatric Arrhythmias

Whenever the child has an abnormal heart rate or rhythm, you must quickly determine if the arrhythmia is causing hemodynamic instability or other signs of deterioration. The signs of instability in a patient with arrhythmia include the following:

- Respiratory distress or failure
- Shock with poor end-organ perfusion, which may occur with or without hypotension
- Irritability or a decreased level of consciousness
- Chest pain or a vague feeling of discomfort in older children
- Sudden collapse

Priorities in initially managing arrhythmias are the same as they are for all critically ill children: support the ABCs—airway, breathing, and circulation—and treat the underlying cause.

Management: Pediatric Bradycardia With a Pulse

The Pediatric Bradycardia With a Pulse Algorithm (Figure 46) outlines the steps for evaluating and managing the child who presents with symptomatic bradycardia (bradycardia with a pulse). Refer to the Critical Concepts box Symptomatic Bradycardia

and Cardiopulmonary Compromise in Part 9 for more information about symptomatic bradycardia and cardiopulmonary compromise.

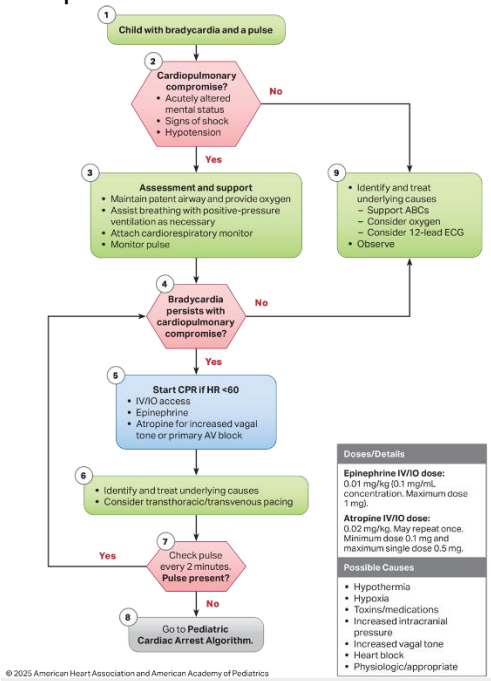


Figure 46. Pediatric Bradycardia With a Pulse Algorithm

Abbreviation: HR, heart rate.

Algorithm legend: Red hexagon = decision or question; green box = action/steps to consider; orange box = assessment; gray box = assessment/action; blue box = CPR; white box with gray header = notes; and red, bold text = possible answers or decisions.

For a child with bradycardia, once you identify symptomatic bradycardia, you need to determine if the child is stable or unstable. Looking for signs of cardiopulmonary compromise is imperative in determining appropriate interventions for the child.

Critical Concepts

Assess for Cardiopulmonary Compromise

Cardiopulmonary compromise includes

- Acutely altered mental status: acutely decreased level of consciousness
- Signs of shock

Hypotension: Do not delay initiating emergency treatment, including high-quality CPR, if signs of cardiopulmonary compromise are present.

Supporting the ABCs is important. Consider oxygen or ventilatory support. If the patient is stable, obtain a 12-lead ECG. If bradycardia is associated with cardiopulmonary compromise, and if heart rate is less than 60/min despite effective oxygenation and ventilation, start CPR.

If the bradycardia persists, proceed with medication therapy and possible pacing. Pacing can be accomplished with

- Transcutaneous pads and leads using the defibrillator
- Transvenous pacing wire placement through internal jugular vein
- Surgically placed epicardial leads

It is imperative to be sure that there is capture of the myocardium by continuously monitoring for a pulse. It can be difficult to determine capture of skeletal muscle instead of myocardium and feel skeletal muscle contraction when feeling for a pulse. Temporary transcutaneous/transthoracic/transvenous pacing may be lifesaving in selected cases of bradycardia caused by complete heart block or abnormal sinus node function. For example, pacing is indicated for AV block after surgical correction of congenital heart disease. A child with primary bradycardia may benefit from evaluation by a pediatric cardiologist.

Reassess the child frequently in response to each therapy provided (Table 54).

Table 54.Managing Symptomatic Bradycardia With Cardiopulmonary Compromise

Evaluate	Interventions
Airway	Support the airway (position the child or allow the child to assume a position of comfort) or open the airway (perform manual airway maneuver) if needed
Breathing	• Give O_2 in high concentration • Provide positive pressure ventilation as indicated (eg, bag-mask ventilation) • Attach a pulse oximeter to assess oxygenation
Circulation	• Monitor blood pressure and assess perfusion • Attach a monitor/defibrillator (with transcutaneous pacing capability if available) • Establish vascular access (IV or IO) • Check electrode pad position and skin contact to ensure that there is no artifact and the ECG tracing is accurate • Record a 12-lead ECG if available (do not delay therapy)

	• Obtain appropriate laboratory studies (eg, potassium, glucose, ionized calcium, magnesium, blood gas for pH, toxicology screen)

A child with primary bradycardia may benefit from evaluation by a pediatric cardiologist.

Medications

If bradycardia and cardiopulmonary compromise continue despite oxygenation, ventilation, and CPR, administer epinephrine. Consider atropine for increased vagal tone or primary AV block.

Epinephrine

Epinephrine is indicated for symptomatic bradycardia. Epinephrine has both α- and β-adrenergic activity. β-adrenergic activity increases heart rate and cardiac contractility, and α-adrenergic activity causes vasoconstriction. The effects of epinephrine and other catecholamines may be reduced by acidosis and hypoxia. This makes support of the airway, ventilation, oxygenation, and perfusion (with chest compressions) essential.

- For IV/IO route, give 0.01 mg/kg (0.1 mg/mL concentration)
- Repeat every 3 to 5 minutes as needed
- *Note:* IV/IO administration is preferred, but if it is not available, epinephrine can be administered by ET tube.
- For ET route, give 0.1 mg/kg (1 mg/mL concentration)
- For persistent bradycardia, consider a continuous infusion of epinephrine (0.1 to 0.3 mcg/kg per minute). A continuous epinephrine infusion may be useful, particularly if the child has responded to a bolus of epinephrine. Titrate the infusion dose to clinical response.

Atropine

Atropine sulfate is a parasympatholytic (or anticholinergic) medication that accelerates sinus or atrial pacemakers and enhances AV conduction. Administer atropine instead of epinephrine for bradycardia caused by increased vagal tone, cholinergic medication toxicity (eg, organophosphates), or complete AV block. Atropine (and pacing) are preferred over epinephrine as the first-choice treatment of symptomatic AV block due to primary bradycardia. Atropine is not indicated for AV block from secondary bradycardia (ie, treatable causes such as hypoxia or acidosis). The rationale for using atropine rather than epinephrine in these situations is that epinephrine can cause ventricular arrhythmias if the myocardium is chronically abnormal or hypoxic/ischemic. If the child does not respond to atropine, use epinephrine.

Atropine may be used for the treatment of second-degree AV block (Mobitz types I and II) and third-degree AV block. The health care professional should recognize, however, that symptomatic AV block may not respond to atropine and the child may require pacing.

- For IV/IO route, give 0.02 mg/kg; minimum 0.1 mg, maximum 0.5 mg.
    - –May repeat dose once, in 5 minutes.
    - –*Note:* Larger doses may be required for organophosphate poisoning.
- For ET route, give 0.04 to 0.06 mg/kg.
    - –*Note:* IV/IO administration is preferred, but if it is not available, atropine can be administered by ET tube. Because absorption of atropine given by the ET route is unreliable, a larger dose (2-3 times the IV dose) may be required.

Tachycardia may follow atropine administration, but it is generally well tolerated in the pediatric patient.

Identify and Treat Underlying Causes

Identify and treat potentially reversible causes and special circumstances that can cause bradycardia. The 2 most common potentially reversible causes of bradycardia are hypoxia and increased vagal tone. Be aware that after heart transplantation, sympathetic nerve fibers are no longer attached to the heart, so the response to sympathomimetic medications may be unpredictable. For the same reason, anticholinergic medications, such as atropine, may be ineffective. Early cardiac pacing may be indicated in such patients.

Treat potentially reversible causes of bradycardia as shown in Table 55.

Table 55.Treatment of Bradycardia Causes

Reversible cause	Treatment
Hypoxia	Give high-concentration supplemental O_2 with assisted ventilation as necessary
Hydrogen ion (acidosis)	Provide ventilation to treat respiratory acidosis secondary to hypercarbia
Hyperkalemia	Restore normal potassium concentration • Stop all fluids that contain potassium and medications that increase potassium • Stabilize myocardium: calcium (chloride or gluconate) • Shift intracellularly: albuterol, sodium bicarbonate, insulin with glucose administration • Excrete: loop diuretics, potassium binders

	• Remove: dialysis
Hypothermia	Warm the child as needed, but avoid hyperthermia if the patient has experienced a cardiac arrest
Heart block	For AV block, consider atropine, chronotropic medications, and electrical pacing; obtain expert consultation
Toxins/poisons/medications	Treat with a specific antidote and provide supportive care. Some toxicologic causes of bradyarrhythmias are • Cholinesterase inhibitors (organophosphates, carbamates, and nerve agents) • Calcium channel blockers • β-adrenergic blockers • Digoxin and other cardiac glycosides • Clonidine and other centrally acting α_2-adrenergic agonists • Opioids • Succinylcholine
Trauma	Head trauma: Bradycardia in a child with head trauma is an ominous sign of high ICP. Provide oxygenation and ventilation. A brief period of mild hyperventilation may occasionally be used as temporizing rescue therapy in response to signs of impending herniation (eg, irregular respirations or apnea, bradycardia, hypertension, unequal or dilated pupil[s] not responsive to light, decerebrate or decorticate posturing). Obtain immediate expert assistance for relief of increased ICP.

Managing Tachyarrhythmias

Pulseless Arrest Initial Management Questions

Answer the following questions to direct your initial management of a critically ill or injured child with a rapid heart rate:

Does the child have a pulse (or signs of circulation)?

- If no, initiate the Pediatric Cardiac Arrest Algorithm (refer to Part 11: Recognizing and Managing Cardiac Arrest).
    - –*Note:* Because the accuracy of a pulse check is poor, recognizing cardiac arrest may require that you identify the absence of signs of circulation (ie, the child is unresponsive, is not breathing or only gasping). With invasive monitoring of arterial pressure, absence of arterial waveform is observed.
- If yes, proceed with the Pediatric Tachyarrhythmia With a Pulse Algorithm (Figure 49).

Initial Management Priorities

As soon as you recognize a tachyarrhythmia in an infant or child, assess and look for signs of hypotension, altered mental status, shock (ie, poor perfusion), or life-threatening hemodynamic instability. Initial management priorities include the following:

- Maintain a patent airway; assist breathing as necessary.
- Use a cardiac monitor to identify the rhythm, and monitor pulse, blood pressure, and oximetry.
- Establish IV/IO access.
- Obtain a 12-lead ECG if available (but do not delay urgent intervention).
- Assess neurologic status.
- Anticipate the need for medications depending on the type of rhythm disturbance (ie, supraventricular vs ventricular).
- Simultaneously try to identify and treat reversible causes.
- Obtain laboratory studies (eg, potassium, glucose, ionized calcium, magnesium, blood gas to assess pH and cause of pH changes) as appropriate. *Note:* Do not delay urgent intervention for these studies.

Emergency Interventions

Specific emergency interventions used to treat tachyarrhythmias with a pulse are dictated by the severity of the child's condition. Treatments also vary based on the width of the observed QRS complex (narrow vs wide). Interventions may include the following:

- Vagal maneuvers (if the child with a narrow-complex tachycardia is stable)
- Medication therapy
- Synchronized cardioversion

Vagal Maneuvers

In normal infants and children, the heart rate decreases when the vagus nerve is stimulated. In patients with SVT, vagal stimulation may terminate the tachycardia by slowing conduction through the AV node. Several maneuvers can stimulate vagal activity. The success rates of these maneuvers in terminating tachyarrhythmias vary, depending on the child's age, level of cooperation, and underlying condition.

Critical Concepts

Vagal Maneuvers

Ice to the face is a vagal maneuver that can be performed in infants (Figure 47). Fill a small plastic bag with a mixture of ice and water. Apply it to the upper half of the child's face for 15 to 20 seconds. Do not occlude the nose or mouth.

Figure 47. Vagal maneuvers. Note that the bag of ice water does not cover the nose or mouth and does not obstruct ventilation.

- Children old enough to cooperate can perform a Valsalva maneuver by blowing through a narrow straw.
- Do not use ocular pressure because it may produce retinal injury.

Support the child's airway, breathing, and circulation. If possible, obtain a 12-lead ECG before and after the maneuver; record and monitor the ECG continuously during the maneuver. If the child is stable and the rhythm does not convert, you may repeat the attempt. If the second attempt fails, select another method or provide medication therapy. If the child is unstable, attempt vagal maneuvers only while making preparations for pharmacologic or electrical cardioversion. Do not delay definitive treatment to perform vagal maneuvers.

Medication Therapy

Table 56 reviews common agents used in managing tachyarrhythmias.

Table 56.Medication Therapy Used in the Pediatric Tachyarrhythmia With a Pulse Algorithm

Medication	Indications/precautions	Dosage/administration
Adenosine	**Indications** • Medication of choice for treating SVT • Effective for SVT caused by reentry at the AV node (both accessory pathway and AV nodal reentry mechanisms) • May be helpful in distinguishing atrial flutter from SVT	**Dose** • With continuous ECG monitoring, administer 0.1 mg/kg (maximum initial dose 6 mg) as a rapid IV bolus

	• Not effective for treating atrial flutter, atrial fibrillation, or tachycardias caused by mechanisms other than reentry through the AV node **Mechanism of Action** • Blocks conduction through the AV node temporarily (for about 10 seconds) **Precautions** • A common cause of adenosine cardioversion "failure" is that the medication is administered too slowly or with inadequate IV flush • A brief period (10-15 seconds) of bradycardia (asystole or third-degree heart block) may ensue after administration of adenosine (Figure 48); consider warning caregiver and patient, if age appropriate, that bradycardia can be very uncomfortable	• If the medication is effective, the rhythm will convert to sinus rhythm within 15 to 30 seconds after administration (Figure 48) • If there is no effect, give 1 dose of 0.2 mg/kg (maximum second dose 12 mg); this dose is more likely to be needed when the medication is administered into a peripheral (rather than central) vein • Decrease the initial dose by approximately 75% for patients receiving carbamazepine or dipyridamole or those with transplanted hearts **Administration** • Because adenosine has a short half-life (<10 seconds), administer as rapidly as possible • The medication is rapidly taken up by vascular endothelial cells and red blood cells and metabolized by an enzyme on the surface of red blood cells (adenosine deaminase) • To enhance delivery to the site of action in the heart, use a rapid flush technique (5-10 mL normal saline; preferably in a proximal IV) • Adenosine may be given by the IO route
Amiodarone	**Indications** • May be considered when treating hemodynamically unstable SVT refractory to vagal maneuvers, adenosine, and cardioversion and for whom expert consultation is not available • Can be used for treating a wide variety of atrial and ventricular tachyarrhythmias in children • Effective for hemodynamically unstable VT in children **Mechanism of Action**	**Dose** • For supraventricular and ventricular arrhythmias with poor perfusion, a loading dose of 5 mg/kg infused over 20 to 60 minutes is recommended (maximum single dose: 300 mg). Because this medication can cause hypotension and decrease cardiac contractility, a slower rate of delivery is recommended for treating a

	• Inhibits α-and β-adrenergic receptors, producing vasodilation and AV nodal suppression (this slows conduction through the AV node) • Inhibits the outward potassium current so it prolongs the QT duration • Inhibits sodium channels, which slows conduction in the ventricles and prolongs QRS duration **Precautions** • Medication effects may be beneficial in some patients but may also increase the risk for polymorphic VT (torsades de pointes) by prolonging the QT interval • Potential significant acute side effects of amiodarone include bradycardia, hypotension, and polymorphic VT • Use with caution if hepatic failure is present • Because the pharmacology of amiodarone is complex and it has slow and incomplete oral absorption, long half-life, and potential for long-term adverse effects, a pediatric cardiologist should direct long-term amiodarone therapy	perfusing rhythm than for cardiac arrest. Health care professionals must weigh the potential for causing hypotension against the need to achieve a rapid medication effect • Repeat doses of 5 mg/kg may be given up to a maximum of 15 mg/kg per day as needed (should not exceed the maximum recommended adult cumulative daily dose of 2.2 g over 24 hours) **Administration** • Rapid administration of amiodarone may cause vasodilation and hypotension; it may also cause heart block or polymorphic VT • Monitor blood pressure frequently during administration • Seek expert consultation when using amiodarone • Routine use of amiodarone and another agent that prolongs the QT interval (eg, procainamide) is not recommended

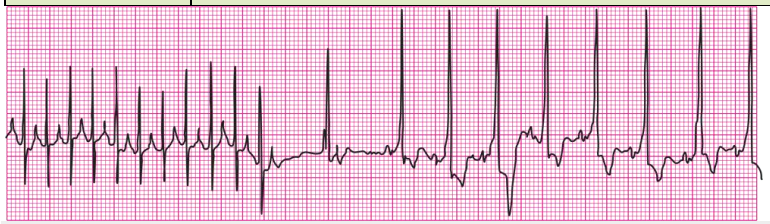


Figure 48. SVT converting to sinus rhythm with administration of adenosine.

Synchronized Cardioversion

Electrical cardioversion is painful. Whenever possible, establish vascular access and provide procedural sedation and analgesia before cardioversion, especially in a hemodynamically stable infant or child. If the child's condition is unstable, do not delay synchronized cardioversion to achieve vascular access. Sedation in the setting of an arrhythmia carries increased risk.

Medications given for procedural sedation should be carefully selected to minimize hemodynamic effects. If your patient is stable and you are considering cardioversion, seek expert consultation.

The next section discusses the following important concepts about cardioversion:

- Definition of synchronized cardioversion
- Potential problems with synchronized shocks
- Indications for the use of synchronized cardioversion
- Energy doses

Delivering Synchronized Shocks

Manual defibrillators are capable of delivering both unsynchronized and synchronized shocks. If the shock is unsynchronized, it is delivered at any time in the cardiac cycle. Synchronized shocks are used for cardioversion from SVT and VT with a pulse. If the shock is synchronized, shock delivery is timed to coincide with the R wave of the patient's QRS complex. The goal is to prevent VF that could result from delivery of the shock during the vulnerable period of the T wave. When you press the Shock button to deliver a synchronized shock, the defibrillator/cardioverter may seem to pause before it delivers a shock because it is waiting to synchronize shock delivery with the next QRS complex. For pulseless rhythms, it clinically does not matter whether the shock is synchronized; accordingly, unsynchronized shocks (eg, defibrillations) are used for both VF and pVT. Refer to the Critical Concepts box Cardioversion (for Unstable SVT or VT With a Pulse) later in this Part for a description of the procedure.

Important considerations: Each manufacturer is different and it is important to follow their individual directions. In theory, synchronization is simple. The operator pushes the Sync button on the defibrillator, charges the device, and delivers the shock. However, in practice there can be potential problems, such as

- In most units, the Sync button must be activated each time synchronized cardioversion is attempted. Most devices will default to an unsynchronized shock immediately after delivery of a synchronized shock.
- If the R waves of a tachycardia are undifferentiated or of low amplitude, the monitor sensors may be unable to identify them and therefore will not deliver the shock. If this occurs, increase the gain of the ECG lead being monitored or select a different ECG lead.
- Synchronization may take extra time (eg, if it is necessary to attach separate ECG electrodes or if the operator is unfamiliar with the equipment).

Indications: Use synchronized cardioversion for

- Hemodynamically unstable patients (poor perfusion, hypotension, or heart failure) with tachyarrhythmias (SVT, atrial flutter, VT), but with palpable pulses

- Elective cardioversion, under the direction of a pediatric cardiologist, for children with hemodynamically stable SVT, atrial flutter, or VT with a pulse

Energy dose: In general, cardioversion requires less energy than defibrillation does. Start with an energy dose of 0.5 to 1 J/kg for cardioversion of SVT or VT with a pulse. If the initial dose is ineffective, increase the dose to 2 J/kg. The experienced professional may increase the shock dose more gradually (eg, 0.5 J/kg, and then 1 J/kg, followed by 2 J/kg for subsequent doses). If the rhythm does not convert to sinus rhythm, reevaluate the diagnosis of SVT vs ST. Sedate if needed, but don't delay cardioversion if the child is hemodynamically unstable.

Critical Concepts

Cardioversion (for Unstable SVT or VT With a Pulse)

Consider expert consultation for suspected VT.

1. Turn on the defibrillator.
2. Set lead switch to paddles (or lead I, II, or III if monitor leads are used).
3. Select adhesive pads or paddles. Use the largest pads or paddles that can fit on the patient's chest without touching each other.
4. If using paddles, apply conductive gel or paste. Be sure cables are attached to the defibrillator.
5. Consider sedation.
6. Select synchronized mode.
7. Look for markers on R waves indicating that sync mode is operative. If necessary, adjust monitor gain until sync markers occur with each R wave.
8. Select energy dose: Initial dose: 0.5 to 1 J/kg; Subsequent doses: 2 J/kg
9. Announce "Charging defibrillator," and press charge on the defibrillator controls or apex paddle.
10. When the defibrillator is fully charged, firmly state, "I am going to shock on 3." Then count and say, "All clear!"
11. After confirming all personnel are clear of the patient, press and hold the Shock button on the defibrillator or press both paddle discharge buttons simultaneously. Hold paddles in place until the shock is delivered.
12. Check the monitor. If tachycardia persists, prepare to attempt cardioversion again.
13. Reset the sync mode and increase the energy dose. You must reset the sync mode after each synchronized cardioversion because most defibrillators default back to unsynchronized mode after synchronized shock delivery. This default allows an immediate defibrillation (nonsynchronized) shock if the cardioversion produces VF.

Note: If VF develops, immediately begin CPR and prepare to deliver an unsynchronized shock as soon as possible. Refer to the Critical Concepts box Manual Defibrillation (for VF or pVT) in Part 11.

Other Emergency Interventions

Many other interventions (eg, digoxin, short-acting β-blockers, overdrive pacing) have been used to treat children with SVT but should be reserved for expert consultation.

Verapamil, a calcium channel blocking agent, should not be used routinely to treat SVT in infants because refractory hypotension and cardiac arrest have been reported after administration. Use verapamil with caution in children because it may cause hypotension and myocardial depression. If using verapamil in children 1 year or older, infuse the medication in a dose of 0.1 mg/kg (up to 5 mg) over at least 2 minutes with continuous ECG monitoring.

Summary of Emergency Interventions

The specific emergency interventions listed in Table 57 are used to treat tachyarrhythmias with pulses, based on the width of the observed QRS complex (narrow vs wide).

Table 57.Emergency Interventions for Tachyarrhythmias With Pulses

Intervention	Narrow-complex tachyarrhythmia	Wide-complex tachyarrhythmia
Vagal maneuvers	Used for SVT	Used for SVT with aberrancy
Medication therapy	Used for SVT: • Adenosine • Amiodarone (seek expert consultation) • Procainamide (seek expert consultation) • Verapamil for children <1 year of age (seek expert consultation) Used for other SVT with a pulse (eg, atrial flutter): Seek expert consultation	Used for VT with palpable pulses: • Amiodarone (seek expert consultation) • Procainamide (seek expert consultation) Used for torsades de pointes: • Magnesium Used for SVT with abnormal/aberrant intraventricular conduction: • Adenosine • Amiodarone (seek expert consultation) • Procainamide (seek expert consultation)
Synchronized cardioversion	Used for • SVT • Atrial flutter (seek expert consultation)	Used for VT with palpable pulses

Pediatric Tachyarrhythmia With a Pulse Algorithm

The Pediatric Tachyarrhythmia With a Pulse Algorithm (Figure 49) outlines the steps for assessing and managing a child presenting with symptomatic tachycardia with or without cardiopulmonary compromise.

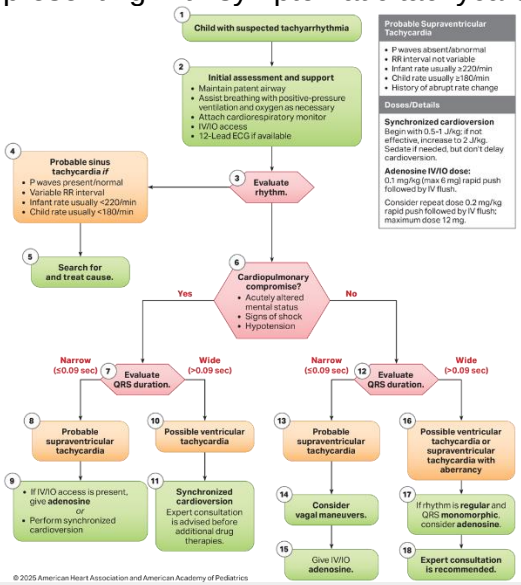


Figure 49. Pediatric Tachyarrhythmia With a Pulse Algorithm.

Algorithm legend: Red hexagon = decision or question; green box = action/steps to consider; orange box = assessment; gray box = assessment/action; blue box = CPR; white box with gray header = notes; and red, bold text = possible answers or decisions.

Initial assessment and support: Initial assessment and support focuses on the ABCs and the placement of monitoring equipment.

Evaluate rhythm with 12-lead ECG or monitor: To determine if this is sinus tachycardia or a tachyarrhythmia (such as SVT or VT). Assess for cardiopulmonary compromise. Determine if the child is stable or unstable (has cardiac compromise). Evaluate the QRS duration as either narrow or wide. If narrow, treat as probable SVT.

Administer adenosine or perform synchronized cardioversion:

- If IV/IO access is present, give adenosine. Adenosine is the medication of choice for most common forms of SVT caused by a reentrant pathway involving the AV node. For IV/IO route, give 0.1 mg/kg (maximum first dose 6 mg). If the first dose is ineffective, you may give 1 dose of 0.2 mg/kg (maximum second dose 12 mg). Use a rapid bolus

with a rapid flush 2-syringe technique (5-10 mL normal saline). It is helpful to run a rhythm strip on the ECG machine while administering adenosine.

- If child is unstable, perform synchronized cardioversion. In general, cardioversion requires less energy than defibrillation does. Start with an energy dose of 0.5 to 1 J/kg for cardioversion of SVT. If the initial dose is ineffective, increase the dose to 2 J/kg. If the rhythm does not convert to sinus rhythm, reevaluate the diagnosis of SVT vs ST. Sedate if needed but don't delay cardioversion.

If there are signs of cardiopulmonary compromise and the QRS complex is wide (greater than 0.09 second), and regular with a pulse, treat as possible VT and perform synchronized cardioversion.

- Expert consultation is advised before any additional medication therapies are administered for treatment of a wide-complex tachycardia.

If there are no signs of cardiopulmonary compromise, and the QRS complex is narrow (0.09 second or less) and regular, treat as probable SVT.

Consider vagal maneuvers.

In the stable patient with SVT, try the following:

- Place a bag with ice water over the upper half of an infant's face (without obstructing the airway).
- Ask older children to bear down, blow through an obstructed straw, or try to push the plunger out of a 10 mL syringe by blowing into the tip.
- Monitor and record the ECG continuously before, during, and after attempted vagal maneuvers. If the maneuvers fail, you may attempt them a second time. Do not apply ocular pressure. For more information, refer to the Critical Concepts box Vagal Maneuvers earlier in this Part.

If IV/IO access is present, give adenosine:

- For SVT resistant to vagal maneuvers, establish vascular access and administer adenosine

If wide complex (greater than 0.09 second), treat as possible VT.

If rhythm is regular and QRS monomorphic, consider adenosine:

- There is a potential for SVT with aberrancy.
- Avoid adenosine if rhythm is irregular as this may result in an unstable rhythm.

Expert consultation is recommended. If a child with a wide-complex tachycardia is hemodynamically stable, early consultation with a pediatric cardiologist or other health care professional with appropriate expertise is recommended.

For unstable SVT after attempting vagal maneuvers, IV adenosine, electric synchronized cardioversion and for whom expert consultation is not available, it may be reasonable to consider either procainamide or amiodarone.

Establish vascular access and consider administering one of the following medications:

- Amiodarone: For IV/IO route, give 5 mg/kg over 20 to 60 minutes.
- Procainamide: For IV/IO route, give 15 mg/kg over 30 to 60 minutes.

Do not routinely administer amiodarone and procainamide together or with other medications that prolong the QT interval. If these initial efforts do not terminate the rapid rhythm, reevaluate the rhythm. Seek expert consultation when giving amiodarone or procainamide.

If not already administered, consider adenosine because a wide-complex, regular tachycardia could be SVT with aberrant ventricular conduction.

Part 11

Recognizing and Managing Cardiac Arrest

This section discusses how to recognize and promptly intervene in life-threatening emergencies and cardiac arrest in infants and children. It covers reversible causes, treatments, and the Pediatric Cardiac Arrest Algorithm.

Learning Objective

After completing this Part, you should be able to recognize cardiopulmonary arrest immediately and begin CPR within 10 seconds.

During the course, you will practice and be tested on CPR skills. Your performance will also be tested in 2 case scenarios.

Rapidly Intervene to Prevent Cardiac Arrest

If not appropriately treated, children with respiratory failure and shock can quickly develop cardiopulmonary failure and even cardiac arrest (Figure 50). In infants and children, most cardiac arrests result from progressive respiratory failure, shock, or both. Less commonly, pediatric cardiac arrests can occur without warning (ie, with sudden collapse) secondary to an arrhythmia (VF or VT).

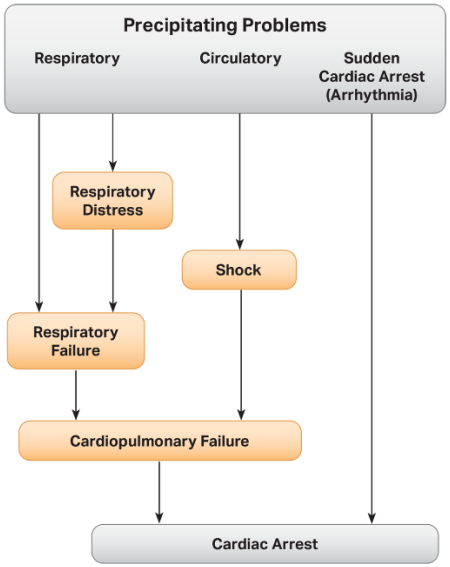


Figure 50. Pathways to pediatric cardiac arrest.

Once cardiac arrest occurs, even with optimal resuscitation efforts, the outcome is generally poor. Cardiac arrest survival rates in the in-hospital setting are poor, and rates for the out-of-hospital setting are more dismal. The concepts in this course will help you identify signs of respiratory failure and shock and rapidly intervene to prevent progression to cardiac arrest.

Respiratory conditions may progress to respiratory failure with or without signs of respiratory distress when the child fails to maintain an open airway or adequate respiratory effort, typically because of a decreased level of consciousness. Sudden cardiac arrest in children is less common than in adults and typically results from arrhythmias such as VF or VT. During sports activities, sudden cardiac arrest can occur in children with underlying cardiac conditions that may or may not have been previously diagnosed.

Interventions

Activate the emergency response system as indicated in your practice setting and begin lifesaving interventions in the following circumstances:

- If the child has a life-threatening condition.
- If you are uncertain or “something feels wrong.”

If the child does not have a life-threatening condition, begin the secondary assessment and diagnostic assessments.

Cardiac Arrest in Infants and Children

In contrast to adult cardiac arrest, cardiac arrest in infants and children usually results from progressive respiratory failure or shock rather than a primary cardiac cause (ie, sudden cardiac arrest). This hypoxic/asphyxial arrest occurs most often in infants and young children, especially those with underlying diseases.

It is important to identify and treat respiratory distress, respiratory failure, and shock before progression to cardiopulmonary failure and cardiac arrest. Early identification and treatment are crucial to saving the lives of seriously ill or injured children.

Sudden cardiac arrest from ventricular arrhythmia occurs in about 5% to 15% of all pediatric in-hospital cardiac arrests (IHCAs) and out-of-hospital cardiac arrests (OHCAs). Although a shockable rhythm (ie, VF or pVT) is the presenting rhythm in only about 14% of pediatric in-hospital arrests, it is present in up to 27% of such arrests at some point during the resuscitation. The incidence of cardiac arrest from VF/pVT increases with age and should be suspected in any patient with a sudden collapse. Increasing evidence suggests that sudden unexpected death in young people is often associated with underlying cardiac conditions.

Despite the improved outcome of in-hospital CPR, most children with IHCA, and an even larger percentage of children with OHCA, do not survive or survive with significant neurologic impairment. Because outcome from cardiac arrest is so poor, focus on preventing cardiac arrest through

- Preventing disease processes and injuries that can lead to cardiac arrest
- Recognizing and managing respiratory distress, respiratory failure, and shock before they cause cardiac arrest

Definition of Cardiac Arrest

Cardiac arrest occurs when blood circulation ceases because of absent or ineffective cardiac mechanical activity. Clinically, the child is unresponsive and is not breathing or is only gasping, and you cannot detect a pulse. Cerebral hypoxia causes the child to lose consciousness and stop breathing, although you may observe agonal gasps during the first minutes after sudden arrest. When circulation stops, the resulting organ and tissue ischemia can cause cell, organ, and patient death if not rapidly reversed.

Pathways to Cardiac Arrest

The 2 pathways to cardiac arrest in children are

- Hypoxic/asphyxial
- Sudden cardiac arrest

Hypoxic/Asphyxial Arrest

Hypoxic/asphyxial arrest is the most common cause of cardiac arrest in infants, children, and adolescents and is the end result of progressive tissue hypoxia and acidosis caused by respiratory failure or hypotensive shock. Regardless of the initiating event or disease process, the final common pathway preceding cardiac arrest is cardiopulmonary failure (Figure 50).

Critical Concepts

Prioritize Airway and Breathing in Children in Cardiac Arrest

In contrast to adults, infants and children usually arrest as the result of progression of respiratory failure or shock. As a result, the child's arterial oxygen content and oxygen delivery to the tissues are often low at the time of cardiac arrest. Although C-A-B sequence for CPR is used for both adults and children, establishing an adequate airway, along with adequate oxygenation and ventilation, should be a particularly high priority in children during CPR.

Sudden Cardiac Arrest

Sudden cardiac arrest, less common in children than it is in adults, is most often caused by the sudden development of VF or pVT. Predisposing conditions or causes for sudden cardiac arrest may include

- Hypertrophic cardiomyopathy
- Anomalous coronary artery
- Long QT syndrome or other channelopathies
- Myocarditis
- Drug intoxication (eg, digoxin, opiates, sodium channel blockers, cocaine)
- Commotio cordis (ie, sharp blow to the chest)

For primary prevention of pediatric cardiac arrest, health care professionals may use cardiovascular screening (eg, for hypertrophic cardiomyopathy or long QT syndrome) and treat predisposing problems (eg, myocarditis, anomalous coronary artery). Some cases of sudden cardiac arrest in children and young adults are associated with genetic mutations that cause cardiac ion channelopathies. A channelopathy is a disorder of the ion channels in myocardial cells that predisposes the heart to arrhythmias. These genetic mutations are known as *familial channelopathies*, so conduct a careful family history to identify syncopal episodes, seizures, and sudden and unexplained death (including sudden infant death syndrome/sudden unexplained infant death, drowning, and even a motor vehicle crash).

For secondary prevention of death from sudden cardiac arrest, health care professionals must perform prompt and effective resuscitation, including timely defibrillation. Most episodes of sudden cardiac arrest in children occur during athletic activity. Coaches, trainers, parents, and the general public must know about sudden cardiac arrest in children so that they can ensure prompt treatment. In addition, bystanders must be prepared and willing to activate the emergency response system, provide high-quality CPR, and use an AED as soon as one is available. Prompt CPR and defibrillation can double or triple your chance of surviving a cardiac arrest.

Causes of Cardiac Arrest

Causes of cardiac arrest in children vary based on the child's age and underlying health as well as on event location (in-hospital vs out-of-hospital). Most OHCA in infants and children occurs at or near the home.

Co-bedding is an increasing cause of death in the infant population. Therefore, instruct parents that infants should have their own sleep space. Trauma is the predominant cause of death in children 6 months of age through young adulthood. Causes of traumatic cardiac arrest include airway compromise, tension pneumothorax, hemorrhagic shock, and brain injury.

The most common causes of pediatric cardiac arrest are respiratory failure and hypotensive shock. Arrhythmia is a less common cause of arrest. Cardiac arrest in children may be associated with a reversible condition. If you don't think about reversible causes or complicating factors, you are likely to miss them. Review the following H's and T's to help you identify potentially reversible causes of cardiac arrest or factors that may be complicating resuscitative efforts.

H's

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypoglycemia
- Hypo-/hyperkalemia
- Hypothermia

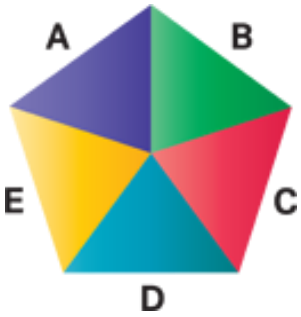
T's

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

- Thrombosis, coronary

Also consider unrecognized trauma (eg, abdominal injury, hemorrhage) as a cause of cardiac arrest, especially in infants and young children.

Identifying a Child at Risk for Cardiac Arrest



Children with the combination of severe respiratory failure and shock will likely develop cardiac arrest within minutes unless you intervene immediately. Be alert for signs of inadequate oxygenation, ventilation, and tissue perfusion.

Recognizing Cardiac Arrest

Signs of cardiac arrest are

- Unresponsiveness
- No normal breathing or only gasping (agonal gasps aren't effective breathing)
- No pulse felt (assess for no less than 5 seconds and no more than 10 seconds)

Arrest rhythm may be noted on the cardiac monitor, but monitoring is not mandatory for recognizing cardiac arrest.

If a child is unresponsive and does not have normal breathing (or is only gasping), try to palpate a central pulse (brachial in an infant, carotid or femoral in a child). Because even health care professionals cannot always detect a pulse, take no more than 10 seconds to try to palpate the pulse. If there is no pulse felt or you are not sure if a pulse is present, start CPR, beginning with chest compressions.

Arrest Rhythms

Cardiac arrest is associated with one of the following rhythms, also known as *arrest rhythms* or *states*:

- Asystole
- PEA
- VF
- pVT, including torsades de pointes

Asystole and PEA are the most common initial rhythms seen in both pediatric IHCA and OHCA, especially in children younger than 12 years. Slow wide QRS complex rhythms that immediately precede asystole are often referred to as *agonal rhythms* (Figure 51). VF and pVT are more likely terminal rhythms in older children with sudden collapse or in children with underlying cardiovascular conditions.

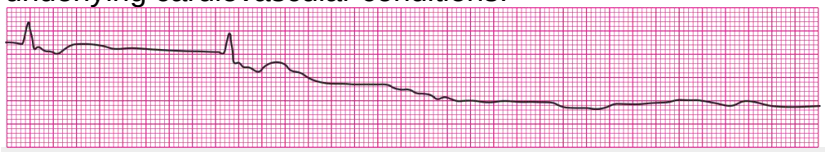


Figure 51. Agonal rhythm (slow ventricular rhythm progressing to asystole).

Asystole

Asystole is cardiac standstill without discernable electrical activity represented by a straight (flat) line on the ECG. Do not rely on the ECG to diagnose cardiac arrest; always confirm it clinically because a “flat line” on the ECG also can be caused by a loose ECG lead. If the gain is low, VF may be overlooked as it might appear to be asystole. Always confirm asystole in 2 or more leads.

Recall potentially reversible causes of asystole by remembering the H’s and T’s.

PEA is not a specific rhythm but a term describing any organized electrical activity (ie, not VF/pVT or asystole) on an ECG or cardiac monitor that is associated with no palpable pulses; an arterial waveform or Doppler study may detect pulsations, but these pulses are not palpable. The rate of electrical activity may be slow (most common), normal, or fast. You may refer to very slow PEA as *agonal*.

In PEA, the ECG may display normal or wide QRS complexes or other abnormalities, including

- Low- or high-amplitude T waves
- Prolonged PR and QT intervals
- AV dissociation, complete heart block, or ventricular complexes without P waves

Assess the monitored rhythm and note the rate and width of the QRS complexes.

Unless you can quickly identify and treat the cause of PEA (recall the H’s and T’s), the rhythm will likely deteriorate to asystole.

Ventricular Fibrillation

Once a monitor is in place, evaluate the rhythm during pulse checks to assess for shockable rhythms (VF, pVT). Be sure that the amplitude is large enough to discern asystole from VF. When VF is present, the heart has no organized rhythm and no coordinated contractions (Figure 52). Electrical activity is chaotic, and the heart quivers and does not pump blood. Therefore, pulses are not palpable. VF may be preceded by a brief period of VT with or without pulses.

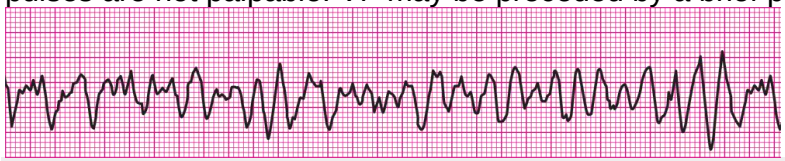


Figure 52A. Ventricular fibrillation. A, Coarse VF. High-amplitude electrical activity varies in size and shape, representing chaotic ventricular electrical activity with no identifiable P, ORS, or T waves.

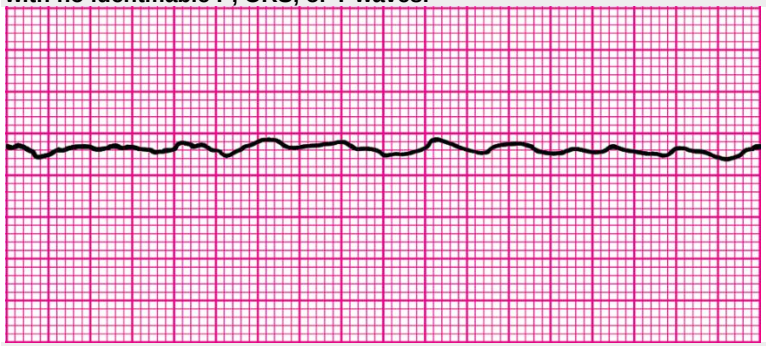


Figure 52B. Fine VF. Electrical activity is reduced as compared with previous (A) rhythm strip.

Primary VF is uncommon in children. In studies of pediatric cardiac arrest, VF was the initial rhythm in 5% to 15% of both OHCAs and IHCAs, but overall prevalence may be higher because VF may occur early during an arrest and quickly deteriorate to asystole. It has been reported in up to 27% of pediatric in-hospital arrests at some point during the resuscitation.

VF without a previously known underlying cause may occasionally occur in otherwise healthy teens during sports activities because of an undiagnosed cardiac abnormality or channelopathy, such as long QT syndrome. In addition, sudden impact to the chest from a collision or moving object may result in commotio cordis that leads to VF. Consider the H’s and T’s for other potential reversible causes.

Survival and outcome of patients with VF or pVT as the initial arrest rhythm are generally better than those of patients presenting with asystole or PEA. Outcome may be improved by promptly recognizing and providing CPR and defibrillation.

Pulseless Ventricular Tachycardia

VT may produce pulses or may be a form of pulseless arrest. Because the treatment of pVT differs from the treatment of VT with a pulse, you must assess the pulse to determine appropriate treatment. Almost any cause of VT can present without detectable pulses. Unlike VF, pVT is characterized by organized, wide QRS complexes. As shown in Figure 53A, the ventricular rhythm is rapid and regular at a rate of 158/min (greater than the minimum 120/min characteristic of VT). The QRS is wide (greater than 0.09 second), and there is no evidence of atrial depolarization. The complexes are uniform in appearance, so the VT is monomorphic. This form of pulseless arrest is usually brief before it deteriorates into VF. Refer to Part 9: Recognizing Arrhythmias for more information.

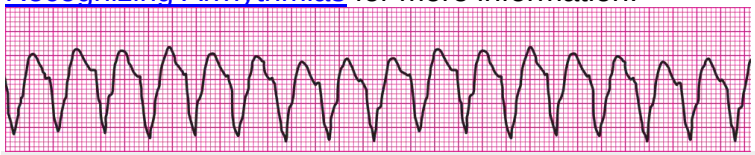


Figure 53A. VT. A, VT in a child with muscular dystrophy and known cardiomyopathy.

Treat pVT exactly the same as VF. Refer to the Pediatric Cardiac Arrest Algorithm (Figure 56) in Part 11: Recognizing and Managing Cardiac Arrest.

Torsades de Pointes (Turning on a Point)

pVT may be monomorphic (ventricular complexes appear uniform) or polymorphic (ventricular complexes do not look alike). Torsades de pointes is a distinctive form of polymorphic VT (Figure 53B), in which the complexes appear to be “turning on a point.” This arrhythmia is seen in conditions distinguished by a prolonged QT interval, including congenital long QT syndrome, medication toxicity, and electrolyte abnormalities (eg, hypomagnesemia). Refer to Part 9: Recognizing Arrhythmias for more information.

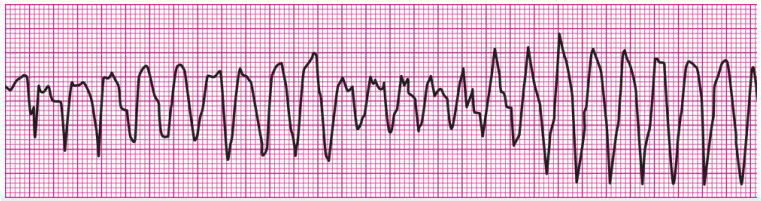


Figure 53B. Torsades de pointes in a child with hypomagnesemia.

Managing Cardiac Arrest

The success of any resuscitation is built on high-quality CPR, timely shock delivery for any shockable rhythm, and good teamwork.

Critical Concepts

CPR in Hypoxic/Asphyxial Arrest

Conventional CPR (chest compressions and rescue breaths) should be provided for pediatric cardiac arrests by all health care professionals and trained lay rescuers. The asphyxial nature of the majority of pediatric cardiac arrests requires ventilation as part of effective CPR.

ALS in Pediatric Cardiac Arrest

The immediate goal of therapeutic interventions for cardiac arrest is ROSC, which occurs when an organized cardiac electrical rhythm on the monitor and palpable central pulses resume. Corresponding clinical evidence of perfusion will also be apparent (eg, sudden increase in end-tidal CO_2 [P_{ETCO_2}], measurable blood pressure, improved color).

PALS treatment of cardiac arrest may include the following:

- Rhythm analysis (shockable vs nonshockable)
- Defibrillation of a shockable rhythm
- Establishment of vascular access
- Medication therapy
- Advanced airway management

Review the fundamentals of BLS in Table 1. These recommendations are based on the *2025 AHA Guidelines for CPR and ECC*.

In-Hospital Cardiac Arrest

Many in-hospital patients, especially those in an intensive care unit, have advanced monitoring in place; some have advanced airways and receive mechanical ventilation. Continuously monitoring the child's P_{ETCO_2} can provide indirect evidence of chest compression quality (Figure 54). If P_{ETCO_2} is low, cardiac output during CPR is probably low, meaning not much blood is delivered to the lungs. Verify that cardiac compressions are effective. If the child has an indwelling arterial catheter, use the waveform as feedback to evaluate hand position and chest compression depth. Minorly adjusting hand position or compression depth can significantly improve the amplitude of the arterial waveform, reflecting better chest compression–induced stroke volume and cardiac output. Verify that ventilation is not excessive. Both the P_{ETCO_2} and arterial waveform may be useful in identifying ROSC.

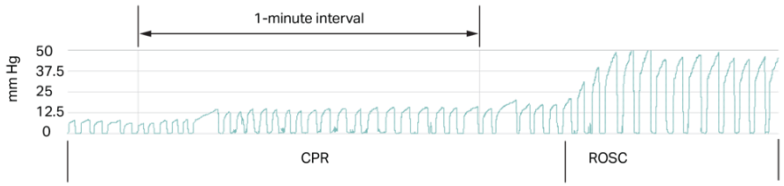


Figure 54. Capnography to monitor effectiveness of resuscitative efforts.

This capnography tracing displays the P_{ETCO_2} in mm Hg on the vertical axis over time (depicted horizontally). This patient is intubated and receiving CPR. Note that the ventilation rate is approximately 20 to 30 breaths per min. Chest compressions in this patient were given continuously at a rate slightly faster than 100/min but are not visible with this tracing. The initial P_{ETCO_2} is less than 12.5 mm Hg during the first minute, indicating very low blood flow. In this example, the P_{ETCO_2} increases to between 12.5 and 25 mm Hg during the second and third minutes, consistent with the increase in blood flow with ongoing resuscitation.

ROSC occurs during the fourth minute and is identifiable by the abrupt increase in the P_{ETCO_2} (visible just after the fourth vertical line) to over 40 mm Hg, which is consistent with a substantial improvement in blood flow with ROSC.

Arrest Rhythm Assessment

The Pediatric Cardiac Arrest Algorithm outlines the recommended sequence of CPR, shocks, and medication administration for both shockable and nonshockable pediatric cardiac arrest rhythms, so identifying the arrest rhythm as shockable or nonshockable determines which pathway to follow. Although the algorithm depicts actions sequentially, many actions (eg, compressions, medication administration) are typically performed simultaneously when multiple rescuers are present.

Vascular Access

Priorities for medication delivery routes during PALS are (in order of preference)

1. Intravenous
2. Intraosseous
3. Endotracheal

When the critically ill child develops cardiac arrest, vascular access may already be established. If vascular access is not present, establish it immediately. Peripheral IV access is the first choice during resuscitation if you can place it rapidly, but placement may be difficult in critically ill or injured children. Limit the time you spend trying to obtain IV access in a seriously ill or injured child. If IV access is not already present and you cannot achieve it immediately, establish IO access. IO access is

useful as the initial vascular access in cases of cardiac arrest. If neither IV nor IO access is available for medication delivery, the ET route is a third option.

Critical Concepts

Vascular Access

Rapid initiation of vascular access (IV/IO) is recommended for medication administration in infants and children with cardiac arrest. Health care professionals should choose the initial type of vascular access route (IV or IO) based on availability, expertise, and timeliness.

IV Route

Although a central venous catheter provides a more secure route of vascular access than a peripheral catheter, central venous access is not needed during the resuscitation attempts, and its placement requires interruptions in chest compressions. Trying to place the central catheter during chest compressions may cause vascular lacerations, hematomas, pneumothorax, and bleeding. If a central venous catheter is already in place, it may be used, however the resistance makes pushing volumes more difficult. A short large-bore IV for volume administration may be preferable.

Establishing peripheral venous access does not require interrupting CPR, but it may delay medication delivery to the central circulation. To improve medication delivery, do the following when administrating medications into a peripheral IV catheter infusion system:

- Give the medication by bolus injection if appropriate.
- Give the medication while chest compressions are being performed; not during pulse checks.
- Follow with a 5-mL flush of normal saline to move the medication from the peripheral to the central circulation.

IO Route

If IV access is not available, medications and fluids can be delivered safely and effectively via the IO route. In fact, the IO route is useful as the initial route of vascular access in cases of cardiac arrest. Important points about IO access are

- IO access can be established in all age groups
- IO access can often be achieved in 30 to 60 seconds
- The IO route is preferred over the ET route
- Any medication or fluid that can be administered IV can be administered IO

IO cannulation provides access to a noncollapsible marrow venous plexus, which serves as a rapid, safe, and reliable route for administering resuscitation medications and fluids. The technique uses a rigid needle, preferably a specially designed IO or

bone marrow needle. Although an IO needle with a stylet is preferred to prevent obstruction of the needle with cortical bone during insertion, butterfly needles, standard hypodermic needles, and spinal needles can be inserted and used effectively. Powered IO insertion devices are commercially available and are widely used in the United States. Refer to the Resources for Managing Circulatory Emergencies in Part 8 of this manual for more information on IO access.

ET Route

The IV and IO routes are preferable to the ET route for administering medications, but lipid-soluble medications can be given by the ET route. These include naloxone, atropine, vasopressin, epinephrine, and lidocaine (NAVEL). However, there are limited human studies about ET vasopressin administration and limited studies to provide dosing guidelines for most medications given by the ET route.

When considering medication administration via the ET route during CPR, keep these concepts in mind:

- Medication absorption from the tracheobronchial tree is unpredictable, so medication concentrations and medication effects will be unpredictable.
- The optimal medication dose given by the ET route is unknown.
    - –Medication administration into the trachea results in lower blood concentrations than the same dose given via IV or IO routes.
    - –Animal data suggest that the lower epinephrine concentrations achieved when the medication is delivered by the ET route may produce transient but detrimental β-adrenergic–mediated vasodilation.
- Recommended medication doses administered by the ET route are higher than for the IV/IO route.
    - –The recommended ET dose of epinephrine is 10 times the IV/IO dose.
    - –Use epinephrine 0.1 mg/kg (1 mg/mL concentration).
    - –The typical ET dose of other medications is 2 to 3 times the IV/IO dose.

To give a medication by the ET route, administer it as follows:

- Instill the medication into the ET tube (briefly pause compressions during instillation).
- Follow with a minimum of 5 mL normal saline flush; a smaller volume may be used in neonates.
- Provide 5 rapid positive-pressure breaths after the medication is instilled.

Defibrillation

A defibrillation shock “stuns” the heart by depolarizing a critical mass of the myocardium. If a shock is successful, it terminates VF/VT, which allows the heart’s natural pacemaker cells to resume an organized rhythm. The return of an organized rhythm alone, however, does not ensure survival. The organized rhythm must ultimately produce effective cardiac mechanical activity

that results in ROSC, defined by the presence of palpable central pulses. If the child's P_{ETCO_2} or intra-arterial pressure is being monitored, it also can indicate ROSC (Figure 55).

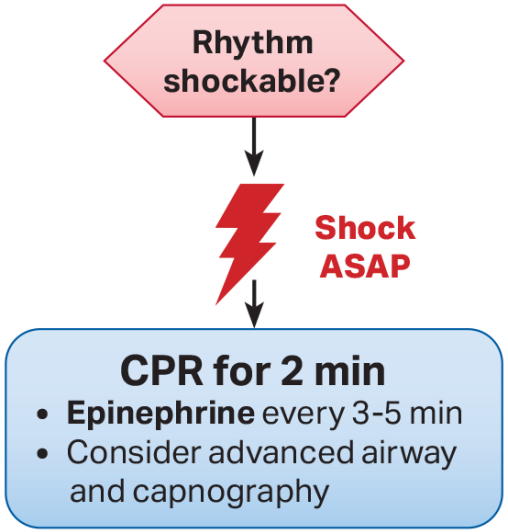


Figure 55. Immediately resume CPR after delivering a shock.

When attempting defibrillation, provide compressions until the defibrillator is charged, deliver 1 shock, and immediately resume CPR, starting with chest compressions. Chest compressions are needed to maintain blood flow to the heart (the coronary circulation) and brain until effective cardiac contractility resumes. No evidence indicates that performing chest compressions in a child with spontaneous cardiac activity is harmful. When the rhythm has reverted to normal sinus rhythm, many patients have very poor contractility for the first 1 to 2 minutes following shock, requiring a minimum of 2 minutes of CPR. If the shock does not eliminate VF, the heart is probably ischemic. Resuming chest compressions is likely to be of greater value to the child than immediate delivery of a second shock. Stacked shocks are not recommended (see Part 12: Post–Cardiac Arrest Care).

In an out-of-hospital or unmonitored setting, do not look for a shockable rhythm or try to palpate a pulse immediately after shock delivery; neither is likely to be present. Resume high-quality CPR, beginning with chest compressions, for 1 to 2 minutes to regain spontaneous cardiac output.

Health care professionals may modify this sequence in hospital units with invasive arterial monitoring. In in-hospital settings with invasive monitoring in place, return of an arterial waveform or a sudden increase in P_{ETCO2} suggests ROSC. When monitored parameters indicate ROSC, confirm by palpating a central pulse.

For more information about the manual defibrillation procedure, refer to the Critical Concepts box Manual Defibrillation (for VF or pVT) later in this Part.

Critical Concepts
Defibrillation and CPR

If a shock eliminates VF, continue CPR because most people/patients have asystole or PEA immediately after shock delivery.

Defibrillation devices for children are the

- AED (able to distinguish pediatric shockable from nonshockable rhythms and ideally equipped with a pediatric dose attenuator)
- Manual cardioverter/defibrillator (capable of variable shock doses)

Institutions that care for children at risk for arrhythmias and cardiac arrest (eg, hospitals, EDs) ideally have defibrillators available that can adjust the energy dose for children.

Manual Defibrillator

The optimal electrical energy dose for pediatric defibrillation is unknown, but an initial dose of 2 to 4 J/kg is acceptable. For ease of teaching, consider a 2 J/kg dose (biphasic or monophasic waveform). If VF or pVT persist at the next rhythm check, deliver a 4 J/kg dose for the second shock. If VF/pVT persist after the second shock, use at least 4 J/kg or higher, but do not exceed 10 J/kg or the maximum adult dose (200 J biphasic, 360 J monophasic). Successful resuscitation using shock doses up to 9 J/kg have been reported in children.

Refer to the Critical Concepts box Manual Defibrillation (for VF or pVT) later in this Part for the universal steps for operating a manual defibrillator.

Pads/Paddles

Use either self-adhesive electrode pads or paddles to deliver shocks with a manual defibrillator. Self-adhesive pads are preferred because they are easy to apply and reduce the risk of current arcing and can be used to monitor the heart rhythm.

Use the largest self-adhering electrode pads that will fit on the chest wall while ensuring that the pads don’t touch.

The selection of pediatric pads vs adult pads is manufacturer specific. Refer to package instructions to determine the appropriate size.

Some defibrillator manufacturers recommend placing self-adhesive electrode pads on the front and back (AP) position. This placement may be necessary in an infant, particularly if only large electrode pads are available. Place the electrode pads according to the recommendations of the defibrillator manufacturer that are typically illustrated on the pads themselves. If you use paddles, apply a conducting gel, cream, or paste, or place an electrode pad between the paddle and the child's chest to reduce transthoracic impedance. Do not use saline-soaked gauze pads, sonographic gels, or alcohol pads because alcohol pads may pose a fire hazard and cause chest burns.

There are 2 paddle sizes based on the child's weight/age. Refer to manufacturer recommendations for device specific information.

- Place the electrode pads/paddles so that the heart is between them. Place the right electrode pad/paddle vertically on the upper right side of the child's chest just below the clavicle and the lateral pad/paddle horizontally, centered at the mid-axillary line. The top of the pad should be on the lower ribcage, in line with the sternal border (inferior portion of the xyphoid process.) Make sure they don't touch. Allow at least 3 cm between paddles, and apply firm pressure to create good contact with the skin.

Modifications may be required in special situations (eg, if the child has an implanted defibrillator or abnormal anatomy, such as dextrocardia).

Clearing for Defibrillation

To ensure rescuer safety during defibrillation, visually check the child and the resuscitation team just before you deliver a shock. Make sure that O_2 is not directed across the child's chest. Warn others that you are about to deliver a shock and that everyone must stand clear. (This entire sequence should take less than 5 seconds.) Refer to the Critical Concepts box Manual Defibrillation (for VF or pVT).

Critical Concepts

Manual Defibrillation (for VF or pVT) (Step 3)

Continue CPR without interruption during all steps until the defibrillator is fully charged. Minimize interval between the last compression and shock delivery (do not deliver breaths between last compression and shock delivery).

1. Turn on the defibrillator.
2. Set lead switch to paddles (or lead I, II, or III if monitor leads are used).
3. Select adhesive pads or paddles; use the largest pads or paddles that can fit on the child's chest without touching each other.
4. If using paddles, apply conductive gel or paste. Be sure cables are attached to the defibrillator.

5. Position the adhesive pads on the child's chest: right anterior chest wall and left axillary positions. If using paddles, apply firm pressure. If the child has an implanted pacemaker, do not place the pads/paddles directly over the device. Be sure that oxygen is not directed over the child's chest.
6. Select energy dose: Initial dose: 2 J/kg (acceptable range 2-4 J/kg); Subsequent doses: 4 J/kg or higher (not to exceed 10 J/kg or standard adult dose).
7. Announce "Charging defibrillator," and press Charge on the defibrillator controls or apex paddle. Continue chest compressions during charging unless charging occurs immediately.
8. When the defibrillator is fully charged, state a firm chant, such as "I am going to shock on 3." Then count. This chant can be shortened to "Clear for shock." (Continue chest compressions until this announcement.)
9. After confirming all personnel and oxygen are clear of the patient, press the Shock button on the defibrillator or press the 2 paddle discharge buttons simultaneously.
10. Immediately after shock delivery, resume CPR beginning with compressions for 5 cycles (about 2 minutes), and then recheck the rhythm. Minimize interruptions to compressions.

Medication Therapy

The objectives for medication administration during cardiac arrest are to

- Increase coronary and cerebral perfusion pressures and blood flow
- Stimulate spontaneous or more forceful myocardial contractility
- Accelerate heart rate
- Correct and treat the possible cause of cardiac arrest
- Suppress or treat arrhythmias

Medications that may be used while treating pediatric cardiac arrest are listed in Table 58.

Table 58.Pediatric Cardiac Arrest Medications

Agent	Indication	Mechanism(s) of action	Clinical data
Vasopressors			
Epinephrine	• Used for cardiac arrest associated with VF/pVT as well as asystole/PEA	• Causes α-adrenergic–mediated vasoconstriction that increases aortic diastolic pressure and	• Both beneficial and toxic physiologic effects during CPR have been

	• High doses may be considered for special resuscitation circumstances, such as β-blocker overdose	coronary perfusion pressure, a critical determinant of successful resuscitation • Has β_1 and β_2 receptor activity resulting in increased heart rate and contractility, which in lower doses as an infusion can help in the periarrest and shock states	demonstrated in animal and human studies • Prompt administration of epinephrine is associated with improved outcomes • High doses may be harmful, particularly in hypoxic/asphyxial arrest • High-dose IV/IO epinephrine is not recommended because it offers no survival advantage
Antiarrhythmics			
Amiodarone	• May be used for shock-refractory VF or pVT	• α-Adrenergic and β-adrenergic blocking activity • Affects sodium, potassium, and calcium channels • Slows AV conduction • Prolongs the AV refractory period and QT interval • Slows ventricular conduction (widens the QRS)	• Increased survival to hospital admission but not to hospital discharge compared with placebo or lidocaine for shock-resistant VF in adults • No association between amiodarone use and ROSC, 24-hour survival, or survival to hospital discharge in pediatric observational study
Lidocaine	• May be used to treat shock-refractory VF or pVT in children	• Decreases automaticity and suppresses ventricular arrhythmias	• Pediatric observational data showed lidocaine improved ROSC as compared with no lidocaine

			• No association between lidocaine use and survival to hospital discharge
Magnesium sulfate	• Used to treat torsades de pointes • Used for hypomagnesemia	• Used to treat arrhythmias associated with hypomagnesemia or prolonged QTc	• Insufficient evidence to recommend for or against routine use in pediatric cardiac arrest not associated with torsades de pointes or hypomagnesemia
Other Agents			
Atropine	• Indicated to treat bradycardia, especially if it results from excessive vagal tone, cholinergic medication toxicity (eg, organophosphates), or complete AV block	• Increases heart rate	• No published studies suggesting efficacy for treating cardiac arrest in pediatric patients • Refer to complete discussion in Part 10: Managing Arrhythmias
Calcium	• Routine use in cardiac arrest is not recommended • Indicated for documented ionized hypocalcemia (relatively common in critically ill children, particularly during sepsis or after cardiopulmonary bypass) and hyperkalemia, particularly in children with hemodynamic compromise • May also be considered for hypermagnesemia or calcium channel blocker overdose	• Restores calcium • Helps maintain the cell membrane action potential threshold • Helps maintain the gradient between intracellular potassium and extracellular sodium	• Does not improve survival in cardiac arrest and may be harmful

Sodium bicarbonate	• Routine administration in cardiac arrest is not recommended • Recommended for symptomatic patients with hyperkalemia, tricyclic antidepressant overdose, diphenhydramine, or an overdose of other sodium channel blocking agents	• Helpful in treating arrhythmias due to sodium channel blockers such as tricyclic or diphenhydramine overdose • Rapidly reduces potassium concentrations in hyperkalemia	• Does not improve survival in cardiac arrest

Advanced Airway Management

When managing the airway and ventilation in pediatric patients of cardiac arrest, consider the following:

- Avoid excessive ventilation during resuscitation.
    - –Excessive ventilation can be harmful because it impedes cerebral perfusion, venous return and decreases cardiac output.
    - –Increased intrathoracic pressure from positive-pressure ventilation also elevates right atrial pressure and thus reduces coronary perfusion pressure.
    - –When providing ventilation with a bag and mask (in cycles of 15 compressions and 2 breaths for 2 rescuers), give each breath over 1 second and provide just enough volume to make the chest rise.
    - –Excessive tidal volume or pressure during bag-mask ventilation may distend the stomach; gastric distention impedes ventilation and increases the risk of regurgitation and aspiration.
- Avoid routine use of cricoid pressure if it interferes with intubation or ventilation.
- Use waveform capnography or capnometry with clinical examination to confirm and monitor ET tube placement.
- Colorimetric exhaled CO_2 devices may fail to detect the presence of exhaled CO_2 (ie, lack of a color change indicates no CO_2 detected) during cardiac arrest despite correctly placing the ET tube. Use direct laryngoscopy to confirm tube placement if exhaled CO_2 is not detected and there is evidence that the tube is in the trachea (eg, chest rise, bilateral breath sounds).
- When providing ventilation via an advanced airway during CPR, provide 1 breath every 2 to 3 seconds (20-30 breaths/min) without pausing chest compressions. Chest compressions are delivered without interruption at a rate of 100 to 120/min.

An advanced airway (eg, ET tube, SGA) can be placed during CPR. However, in a study of OHCA, ET intubation demonstrated no survival advantage over effective bag-mask ventilation when emergency medical services transport time was short, and

health care professionals had limited ongoing experience in pediatric intubation. This study does not address ET intubation in the in-hospital setting but suggests that immediate intubation may not be necessary.

If the resuscitation attempt takes place in a critical care setting and the child has continuous intra-arterial monitoring in place, the diastolic blood pressure can be used to guide CPR quality. Invasive arterial blood pressure monitoring during CPR provides insight to blood pressures generated with compressions and medications.

Pediatric Cardiac Arrest Algorithm

The Pediatric Cardiac Arrest Algorithm (Figure 56) outlines steps for assessing and managing an infant or child in cardiac arrest who does not respond to BLS interventions. The expert consensus–based Pediatric Cardiac Arrest Algorithm maximizes uninterrupted periods of CPR while enabling efficient delivery of electrical therapy and medications as appropriate. Although the actions appear sequentially, when several rescuers are involved, some actions will occur simultaneously.

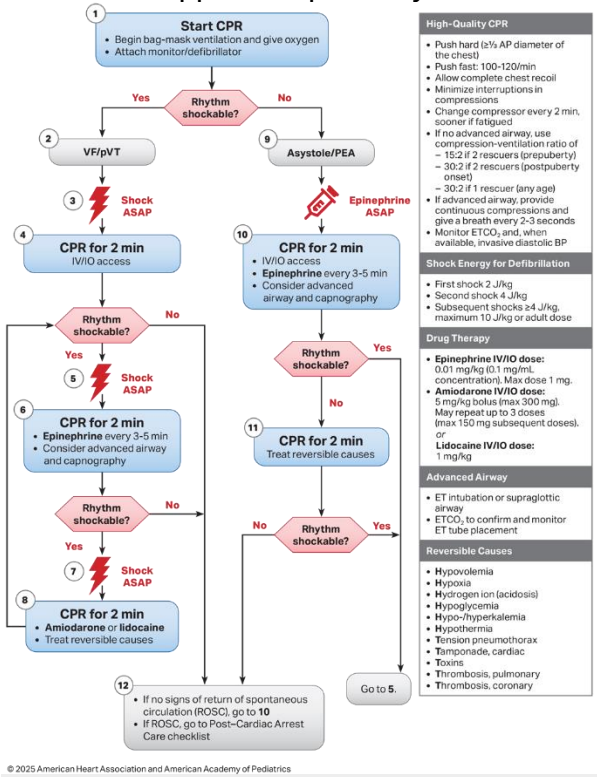


Figure 56. Pediatric Cardiac Arrest Algorithm.

Algorithm legend: Red hexagon = decision or question; green box = action/steps to consider; orange box = assessment; gray box = assessment/action; blue box = CPR; white box with gray header = notes; and red, bold text = possible answers or decisions.

Critical Concepts

Coordinating Team Members During Resuscitation

Using the Pediatric Cardiac Arrest Algorithm, health care professionals should structure assessments and interventions around 2-minute periods of uninterrupted high-quality CPR, rotating chest compressors after every 2-minute period. This requires organization so that all team members know their responsibilities. When all team members are familiar with the algorithm, they can anticipate next steps and prepare the equipment and proper doses of medications.

The algorithm consists of 2 pathways, depending on the cardiac rhythm as identified on a monitor or interpreted by an AED. A shockable rhythm (VF/pVT) pathway is displayed on one side of the algorithm while a nonshockable rhythm (asystole/PEA) pathway is displayed on the other side of the algorithm.

Step numbers in the following sections refer to the corresponding steps in the algorithm.

Start CPR (Step 1)

- Start CPR
    - –As soon as the child is found to be unresponsive with no breathing (or only gasping), shout for nearby help and activate the emergency response system. Use a compression-to-ventilation ratio of 30:2 for 1 rescuer (any age), 15:2 for 2 rescuers (prepuberty), and 30:2 for 2 rescuers (post–puberty onset).
- Determine whether the rhythm is shockable (VF/pVT) or nonshockable (asystole/PEA). If the rhythm is shockable, follow the VF/pVT pathway of the algorithm.

Shockable rhythm: VF/pVT (Step 2)

If the rhythm is shockable, deliver 1 unsynchronized shock (Step 3). Perform CPR while the defibrillator is charging, if possible, because the shorter the interval is between the last compression and shock delivery, the higher the potential shock success (elimination of VF). Therefore, try to keep that interval as short as possible, ideally less than 10 seconds.

Resume CPR, establish IV/IO access, check rhythm (Step 4)

Before the rhythm check, the Team Leader should ensure that the team is prepared to

- Rotate compressors
- Calculate appropriate shock dose to administer if VF/pVT persists
- Prepare medications for administration if indicated

If the rhythm is nonshockable, it may be

- Organized with a pulse and ROSC
- Organized (Figure 57) without a pulse (PEA)
- Not organized without a pulse (asystole). If asystole, do not perform a pulse check. A rhythm without a pulse requires initiation of compressions.

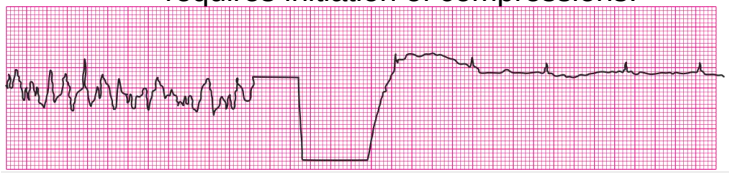


Figure 57. VF converted to organized rhythm after defibrillation (successful shock).

Chest compressions are unlikely to harm a child with a spontaneous rhythm and weak pulses.

In specialized environments (eg, an intensive care unit) with intra-arterial or other hemodynamic monitoring in place, health care professionals may alter this sequence.

Persistent VF/pVT (Step 5)

A second shock may be delivered with a persistent shockable rhythm. Administer epinephrine while compressions continue after the second shock. Consider inserting an advanced airway if one is not already in place.

If VF/pVT persists, administer epinephrine every 3 to 5 minutes while compressions continue. For the IV/IO route, give epinephrine 0.01 mg/kg (0.1 mg/mL concentration) dose. For the ET route, give epinephrine 0.1 mg/kg (1 mg/mL concentration) dose. This will generally result in epinephrine delivery after every other rhythm check. *Note:* IV/IO administration is preferred, but if it is not available, epinephrine can be administered via the ET tube.

The Pediatric Cardiac Arrest Algorithm does not state a specific time for delivering the first dose of epinephrine. However, the *2025 AHA Guidelines for CPR and ECC* states that the earlier epinephrine is administered after CPR initiation, the more likely the patient is to survive. The 2025 Guidelines state the following:

1. For pediatric patients in cardiac arrest with initial nonshockable rhythm, it is reasonable to administer the initial dose of epinephrine as early as possible.
2. For pediatric patients in cardiac arrest with initial shockable rhythm, it may be reasonable to administer epinephrine after 2 attempts at defibrillation or sooner if rapid defibrillation is not possible.
3. For pediatric patients in any setting, it may be reasonable to administer epinephrine every 3 to 5 minutes until ROSC is achieved.

When health care professionals identify VF/pVT during the rhythm check, they can administer epinephrine during CPR that immediately follows the shock delivery. Conversely, if an organized rhythm is present during the rhythm check, it is reasonable to check for a pulse to avoid an unnecessary dose of epinephrine because it can produce adverse effects. For example, if the initial VF/pVT was related to a cardiomyopathy, myocarditis, or medication toxicity, epinephrine administration immediately after eliminating VF/pVT could induce recurrent VF/pVT.

If a shockable rhythm persists, an antiarrhythmic medication should be administered. Immediately after resuming chest compressions, administer amiodarone or lidocaine. Give amiodarone 5 mg/kg IV/IO bolus; you may repeat 5 mg/kg IV/IO bolus up to 3 total doses (max 150 mg subsequent doses) for refractory VF/pVT, or give lidocaine IV/IO at an initial 1 mg/kg loading dose. If torsades de pointes presents without a pulse, give magnesium 25 to 50 mg/kg IV/IO bolus, maximum dose 2 g.

When an advanced airway is in place during CPR, provide continuous chest compressions. Refer to the Critical Concepts box CPR With an Advanced Airway.

Critical Concepts

CPR With an Advanced Airway

Once an advanced airway (eg, ET tube, SGA) is in place, change the CPR sequence from “cycles” to continuous chest compressions and a regular ventilation rate. While one team member compresses the chest at a rate of 100 to 120/min, another team member ventilates with 1 breath every 2 to 3 seconds (a rate of about 20-30 breaths per minute).

Summary of the VF/pVT Sequence

Prepare the next medication before rhythm check. Administer medication during chest compressions, as soon as possible after the rhythm check confirms VF/pVT. Do not delay shock. Continue CPR while the medications are prepared and administered and the defibrillator is charging. Ideally, interrupt chest compressions only for ventilation (until advanced airway is placed), rhythm check, and actual shock delivery.

Establish vascular access (Step 10)

For treating asystole or PEA, provide high-quality CPR, deliver epinephrine as soon as possible, and try to identify and treat potentially reversible causes of the arrest. If the rhythm becomes shockable, immediately provide a shock. If the rhythm returns with a pulse, utilize the Post–Cardiac Arrest Care Checklist (see Part 12). If there are no signs of ROSC, go to Step 10. If a shockable rhythm presents at any time, go to Step 7.

As soon as ROSC develops after cardiac arrest or resuscitation from severe shock or respiratory failure, a systematic approach to assessment and support of the respiratory, cardiovascular, and neurologic systems, including providing targeted temperature

management, is critical. Ultimate outcome is often determined by the subsequent care the child receives, which includes safe transport to a center with expertise in caring for seriously ill or injured children. Refer to the Post–Cardiac Arrest Care Checklist in Part 12.

Summary of Asystole/PEA Treatment Sequence

Managing any pulseless arrest includes providing nearly continuous CPR, interrupted by only brief rhythm checks. Do not interrupt CPR for medication preparation and administration. Administer IV/IO medications during chest compressions. Refer to the Critical Concepts box CPR With an Advanced Airway earlier in this Part.

Identifying and Treating Potentially Reversible Causes of Cardiac Arrest

The outcome of hypoxic-ischemic pediatric cardiac arrest is generally poor. Rapid recognition, immediate high-quality CPR, and correcting contributing factors and potentially reversible causes offer the best chance for a successful resuscitation. If you can quickly identify the condition and treat it, your resuscitative efforts may be successful.

When searching for potentially reversible causes or contributing factors, continue to support the ABCs.

ROSC

If resuscitative efforts successfully restore an organized rhythm (or you find other evidence of ROSC, such as an abrupt and sustained increase in P_{ETCO_2} or visible pulsations on an arterial waveform), check the child's pulse for a perfusing rhythm. If a pulse is present, continue with post–cardiac arrest care.

Pediatric Cardiac Arrest: Special Circumstances

The following special circumstances resulting in pediatric cardiac arrest require specific management:

- Trauma
- Drowning
- Anaphylaxis
- Poisoning
- Congenital heart disease: single ventricle
- Pulmonary hypertension

Cardiac Arrest Due to Trauma

Trauma-associated cardiac arrest in children represents a significant subgroup of pediatric OHCAs. Improper resuscitation (including inadequate volume resuscitation) is a major cause of preventable pediatric trauma deaths. Despite rapid and effective out-of-hospital and trauma center response, the survival rate is low for pediatric OHCA due to trauma and very low for pediatric OHCA due to blunt trauma. Factors that may improve outcome from traumatic OHCA include treatable penetrating injuries and prompt transport (typically 10 minutes or less) to a trauma care facility.

Traumatic cardiac arrest in children has multiple possible causes, including

- Hypoxia secondary to respiratory arrest, airway obstruction, or tracheobronchial injury
- Injury to vital structures (eg, heart, aorta, pulmonary arteries)
- Severe brain injury with secondary cardiovascular collapse
- Upper cervical spinal cord injury with respiratory arrest, which may be accompanied by spinal shock, progressing to cardiac arrest
- Diminished cardiac output or PEA from tension pneumothorax, cardiac tamponade, or massive hemorrhage

BLS and ALS techniques for the pediatric trauma patient in cardiac arrest are fundamentally the same as for the child with nontraumatic cardiac arrest: support of circulation, airway, and breathing. The focus of a resuscitation attempt in an out-of-hospital setting is to

- Provide high-quality CPR, if needed
- Maintain adequate airway, ventilation, and circulation
- Anticipate airway obstruction by dental fragments, blood, or other debris (use a suction device, if necessary)
- Minimize cervical spine motion (if indicated), while opening the airway (jaw thrust) and providing ventilation
- Control hemorrhage with direct pressure, elevation, hemostatic dressings, and/or by using a tourniquet as indicated
- Minimize interventions that delay transport to definitive care
- Transport infants and children with multisystem trauma to a trauma center with pediatric expertise
- Establish IV/IO access and initiate volume resuscitation as needed

The following is a summary of key management principles for traumatic cardiac arrest in children (Table 59).

Table 59.Managing Traumatic Cardiac Arrest

Technique	Intervention
CPR	• Perform high-quality CPR

	• Attach a monitor/defibrillator • Provide defibrillation as needed • Control visible hemorrhage with direct pressure or tourniquet
Airway	• Open and maintain the airway by using a jaw-thrust maneuver • Restrict cervical spine motion by manually stabilizing the head and neck if cervical spine injury is suspected
Breathing	• Avoid excessive ventilation • Ventilate with a bag-mask device using 100% O_2; use a 2-person bag-mask ventilation technique to maintain manual stabilization of the head and neck (if spinal motion restriction is indicated) • One rescuer should stabilize the head and neck in a neutral position (if spinal motion restriction is indicated) during airway placement • Perform unilateral or bilateral needle decompressions for suspected tension pneumothoraxes • Seal any significant open pneumothorax and insert a thoracotomy tube
Circulation	• Assume that the patient is hypovolemic; establish IV/IO access and replace fluids rapidly. Consider non-crossmatched type O-negative blood for female patients (and either O-negative or O-positive for male patients) • Consider pericardiocentesis for possible cardiac tamponade (if suspected) • Consider spinal shock (ie, loss of sympathetic innervation) resulting in fluid-refractory hypotension and bradycardia. Vasopressor therapy is indicated if spinal shock is suspected

Cardiac Arrest Following Drowning

Immediate high-quality CPR is the single most important factor influencing survival in drowning (Table 60). You may have difficulty performing chest compressions while the person is still in the water, but you can provide in-water breaths immediately if you are trained to do so and it does not compromise your own safety. Start chest compressions as soon as you can do so safely and when the child is lying faceup on a firm surface.

Table 60.Managing Cardiac Arrest Following Drowning

Technique	Intervention
CPR	• Perform high-quality CPR with breaths • Attach a monitor/defibrillator; provide rapid defibrillation if needed. Do not delay initiation of CPR to obtain or apply an AED in cardiac arrest following drowning

	• If defibrillation is indicated and the chest is covered with water, quickly wipe the child's chest to minimize electrical arcing between the defibrillation pads or paddles

Airway	• If there is no reason to suspect a cervical spine injury, open the airway with head tilt–chin lift
	• If there is reason to suspect a cervical spine injury (eg, diving injury)
	  ○ –Restrict spinal motion
	  ○ –Use jaw thrust to open the airway
	• During advanced airway insertion, one rescuer should stabilize the head and neck in a neutral position

Breathing	• Ventilate with a bag-mask device by using 100% O_2 • Be prepared to suction the airway, because people who have drowned often vomit swallowed water; decompress the stomach with a nasogastric/orogastric tube after the advanced airway is inserted
Circulation	• Continue high-quality CPR if needed
Exposure	• Evaluate core body temperature and attempt rewarming if the child is severely hypothermic (core temperature <30 °C)

Except for consideration of cervical spine injury and hypothermia as contributing factors, BLS and ALS techniques for cardiac arrest due to drowning are fundamentally the same as for other children in cardiac arrest: support of airway, breathing, and circulation. Rescuers should remove a child who has drowned from the water and begin the resuscitation attempt as quickly as possible.

In hypothermia-associated cardiac arrest, it is often difficult to know when to terminate resuscitative efforts. People who have drowned in icy water can possibly survive after prolonged submersion (as long as 40 minutes) and CPR (longer than 2 hours). When drowning occurs in icy water, experts recommend rewarming to a core temperature of at least 30 °C before abandoning CPR efforts. The heart may not respond to resuscitative efforts until it achieves this core temperature.

Extracorporeal circulation is the most rapid and effective technique for rewarming severely hypothermic cardiac arrest patients after submersion in icy water. Although you may rewarm the patient with passive techniques or body cavity irrigation in the out-of-hospital or community hospital setting, you should rapidly transfer to a facility that is capable of performing pediatric extracorporeal CPR (ECPR) or ECMO.

Cardiac Arrest Due to Anaphylaxis

Near-fatal anaphylaxis produces airway edema and obstruction and profound vasodilation, which significantly increases intravascular capacity and produces relative hypovolemia. Anaphylaxis often accompanies bronchoconstriction, which

compromises oxygenation and further impairs tissue O_2 delivery. If cardiac arrest develops, provide CPR as the primary therapy and establish and maintain an adequate airway, bolus fluid administration, and epinephrine. Children with anaphylaxis are often young with healthy hearts and cardiovascular systems. They may respond to adequate oxygenation and ventilation along with rapid correction of vasodilation and low intravascular volume. Effective CPR may maintain sufficient O_2 delivery until the catastrophic effects of the anaphylactic reaction resolve.

Managing cardiac arrest that results from anaphylaxis may include the following critical therapies (Table 61):

Table 61 Managing Cardiac Arrest Due to Anaphylaxis

Technique	Intervention
CPR	• Perform high-quality CPR and rapid defibrillation as needed
Airway	• Open and maintain the airway by using manual maneuvers • If you perform ET intubation, be prepared for possible airway edema and the need to use a smaller ET tube than predicted by the child's age or length
Breathing	• Perform bag-mask ventilation, until an advanced airway is inserted, using 100% O_2

Circulation	• Insert 2 large-bore IVs or 2 IO catheters
	• Administer boluses of isotonic crystalloid to treat shock
	• Administer epinephrine every 3-5 minutes:
	  ○ –0.01 mg/kg (0.1 mg/mL concentration)
	  ○ –*Note:* IV/IO administration is preferred, but if it is not available, epinephrine can be administered by ET tube
	  ○ –For ET route, give 0.1 mg/kg (1 mg/mL concentration)
	• Infuse with epinephrine as needed

Antihistamines and corticosteroids are considered adjuncts in the treatment of anaphylaxis, with varying degrees of evidence to support their use. In the setting of cardiac arrest due to anaphylaxis, the most important medication to give, and to continue to give if indicated, is epinephrine. If ROSC is achieved, administering antihistamines or corticosteroids, or both, can then be considered.

Cardiac Arrest Associated With Poisoning

Drug overdose or poisoning may cause cardiovascular collapse from direct cardiotoxicity, arrhythmias, or peripheral vasodilation. A person with poisoning often has a healthy myocardium, but temporary cardiac dysfunction may be present until the effects of the drug or toxin have been reversed or metabolized. This takes a variable amount of time, often several hours, depending on the nature of the toxin, drug, or poison. Because the toxicity may be temporary, prolonged resuscitation efforts and use of advanced support techniques such as extracorporeal life support may result in good long-term survival.

Initiate ALS measures according to the Pediatric Cardiac Arrest Algorithm. Check glucose as soon as possible. If the patient is in cardiac arrest and hypoglycemic (eg, from a β-blocker or alcohol overdose), normalize glucose as soon as possible to improve the chances of a successful cardiac and neurologic outcome. PALS treatment for patients of a suspected poisoning should include searching for and treating reversible causes including antidote administration. We recommend early consultation with a poison control center or toxicologist.

Congenital Heart Disease: Single Ventricle

While the prevalence of complex cyanotic congenital heart disease in children is low, those with single ventricle (tricuspid/pulmonary atresia, hypoplastic left heart syndrome, and their variants) represent a large proportion of children who have cardiac arrest, particularly in the in-hospital setting. More infants and children are surviving after palliative surgical procedures; these patients may require resuscitation postoperatively or during readmission for critical illness.

Single-ventricle physiology is complex and varies with the specific lesion and stage of surgical repair. Therefore, it is important to obtain a history from the caretakers to determine the child's baseline hemodynamic status and arterial O_2 saturation. During cardiac arrest, standard resuscitation care is indicated for all infants with single-ventricle anatomy after stage I (Norwood) palliation or those with a univentricular heart and aortopulmonary shunt to provide pulmonary blood flow. In addition to standard resuscitation, specific measures include the following:

- Consider administering heparin for children with aortopulmonary or right ventricular-pulmonary artery shunt if you are concerned about shunt openness/patency.
- After resuscitation, titrate administered O_2 to achieve an O_2 saturation appropriate to maintain the optimum pulmonary-to-systemic blood flow ratio and adequate systemic perfusion and oxygenation (must be individualized for each patient).
- Do not rely on P_{ETCO_2} to indicate CPR quality in a single-ventricle patient because pulmonary blood flow in these patients does not always reflect cardiac output (ie, it is influenced by additional factors).
- Consider permissive hypoventilation strategies or even negative-pressure ventilation in the periarrest state to improve cardiac output.

- Consider extracorporeal life support or ECMO for patients in cardiac arrest who have undergone stage I palliation (Norwood) or Fontan-type procedures.

Pulmonary Hypertension

In pulmonary hypertension, increased resistance to blood flow through the lungs may impair cardiac output, so follow standard PALS recommendations during cardiac arrest. Other measures include the following:

- Correct hypercarbia and acidosis if present.
- Consider giving a bolus of isotonic crystalloid (eg, normal saline) to maintain ventricular preload.
- If the patient received pulmonary vasodilators such as nitric oxide or prostacyclin immediately before the arrest, be sure that medication administration continues and add vasopressors to support blood pressure as needed.
- Consider administering inhaled nitric oxide or prostacyclin (or IV prostacyclin) to reduce pulmonary vascular resistance.
- Consider instituting ECPR early during resuscitation.

Critical Concepts

ECPR

You may consider ECPR for pediatric patients with cardiac diagnoses who have IHCA in settings with existing ECMO protocols, expertise, and equipment. While evidence shows no overall benefit to using ECPR, observational data from a registry of pediatric IHCA showed improved survival to hospital discharge with the use of ECPR in patients with surgical cardiac diagnoses. For children with underlying cardiac disease, when ECPR is initiated in a critical care setting, long-term survival has been reported after prolonged CPR. When you use ECPR during cardiac arrest, the outcome for children with underlying cardiac disease is better than for those with noncardiac disease.

During the course, you will learn about the phases of post–cardiac arrest care. This will include optimizing oxygenation, ventilation, and perfusion, stabilizing cardiopulmonary function, and providing neurologic care, including targeted temperature management.

Part 12
Post–Cardiac Arrest Care

As soon as ROSC develops after cardiac arrest or resuscitation from severe shock or respiratory failure, a systematic approach to assessment and support of the respiratory, cardiovascular, and neurologic systems, including providing targeted temperature management, is critical. Although effective resuscitation is a major focus of the PALS Provider Course, ultimate outcome is often determined by the subsequent care the child receives. This includes safe transport to a center with expertise in caring for seriously ill or injured children.

One objective of optimal post–cardiac arrest care is to avoid common causes of both early and late morbidity and mortality. Early mortality can be caused by hemodynamic instability and respiratory complications. Late morbidity and mortality can result from multiorgan failure, including brain injury.

The extent of post–cardiac arrest evaluation and management is influenced by the PALS provider's scope of practice and available resources.

Learning Objective

After completing this Part, you should be able to implement post–cardiac arrest care.

During the course, you will learn about the phases of post–cardiac arrest care. This will include optimizing oxygenation, ventilation, and perfusion, stabilizing cardiopulmonary function, and providing neurologic care, including targeted temperature management.

Goals of Therapy

For optimal post–cardiac arrest care, identify and treat organ system dysfunction. This includes

- Providing adequate oxygenation and ventilation
- Supporting tissue perfusion and cardiovascular function
- Avoiding hypotension
- Correcting acid-base and electrolyte imbalances
- Maintaining appropriate glucose concentration

- Providing targeted temperature management: avoiding hyperthermia and considering need for therapeutic hypothermia
- Ensuring adequate analgesia and sedation

Post–cardiac arrest management consists of 2 general phases to stabilize the child.

The first phase is immediate post–cardiac arrest management. During this phase you will continue to provide ALS for immediate life-threatening conditions and focus on the ABCs:

- Airway and Breathing: Assess and support airway, oxygenation, and ventilation. At this time, you will typically use diagnostic equipment and assessments—such as monitoring end-tidal CO_2 by capnography, ABG analysis, and chest x-ray—to further establish the adequacy of oxygenation and ventilation and to confirm ET tube position in the midtrachea.
- Circulation: Assess and maintain adequate blood pressure and perfusion. Treat arrhythmias. Diagnostic assessments—such as lactate concentration, central venous O_2 saturation, and base deficit—provide information on adequacy of tissue perfusion. As you proceed with evaluation, identify and treat any reversible or contributing causes of the arrest or critical illness.

In the second phase of post–cardiac arrest management, provide broader multiorgan supportive care, including targeted temperature management. After the child is stabilized, coordinate transfer or transport to a tertiary care setting as appropriate.

Primary Goals

The primary goals of post–cardiac arrest management are to

- Optimize and stabilize airway, oxygenation, ventilation, and cardiopulmonary function with emphasis on restoring and maintaining vital organ perfusion and function (especially of the brain)
- Prevent secondary organ injury
- Identify and treat the cause of acute illness
- Institute measures that may improve long-term, neurologically intact survival
- Minimize the risk of deterioration of the child during transport to a higher level of care

Post–Cardiac Arrest Care Checklist

This checklist (Figure 58) includes a systematic approach to assessing and caring for the child after cardiac arrest. Assess the child by using the systematic approach (refer to Part 4: Systematic Approach to the Seriously Ill or Injured Child). In addition to the primary assessment, your evaluation will often include the secondary assessment as well as diagnostic evaluation. The

secondary assessment is a review of patient history and a focused physical examination. Diagnostic evaluation includes invasive and noninvasive monitoring and appropriate laboratory and nonlaboratory tests.

Components of Post–Cardiac Arrest Care	Check
Ensure normoxia and normocapnia.	
Monitor Spo_2 and target 94%-99% (or child's normal/appropriate oxygen saturation).	☐
Measure and target $Paco_2$ appropriate to the patient's underlying condition and limit exposure to severe hypercapnia or hypocapnia.	☐
Optimize cardiac function and end-organ perfusion.	
Monitor with cardiac telemetry.	☐
Monitor arterial blood pressure.	☐
Review hemodynamic goals daily.	☐
Monitor serum lactate and urine output to help guide therapies.	☐
Use parenteral fluid bolus with or without inotropes or vasopressors to maintain a systolic blood pressure and mean arterial blood pressure greater than the 10th percentile for age.	☐
Consider echocardiography to assess for myocardial dysfunction.	☐
Maintain electrolytes within normal ranges to avoid possible life-threatening arrhythmias.	☐
Apply targeted temperature management (TTM).	
Continuously monitor core temperature.	☐
Prevent and treat fever immediately after cardiac arrest and after rewarming.	☐
If patient is comatose, apply TTM (32 °C-34 °C) followed by (36 °C-37.5 °C) or only TTM (36 °C-37.5 °C) for up to 5 days.	☐
Prevent shivering.	☐
Remember that rewarming is a high-risk time for hypotension, electrolyte abnormalities, hypoglycemia, and seizures.	☐
Provide neuromonitoring.	
If patient is not at neurologic baseline and resources are available, monitor with continuous electroencephalogram.	☐
Treat seizures.	☐
Measure glucose.	
Measure blood glucose and avoid hypoglycemia.	☐
Prevent agitation and pain.	
Treat with sedatives, anxiolytics, and analgesics to a sedation score target.	☐
Consider prognosis.	
Always consider multiple modalities (clinical and other) over any single predictive factor.	☐
Delay prognostication until at least 72 hours after cardiac arrest.	☐
Remember that prognostics assessments may be modified by TTM.	☐

Figure 58. Post–Cardiac Arrest Care Checklist.

This Part discusses evaluation and management of the following systems during the post–cardiac arrest period:

- Respiratory system
- Cardiovascular system
- Neurologic system

Respiratory System

Management Priorities

Continue to monitor and support the child's airway, oxygenation, and ventilation. Look for clinical signs and objective measurements of adequate oxygenation and ventilation. (Refer to Part 5: Recognizing Respiratory Problems for more information on assessment of the respiratory system.) During resuscitation, high-flow O_2, inhaled medications, and ET intubation may be required. In the post–cardiac arrest phase, elective intubation may be appropriate to achieve airway control and support the child during diagnostic studies, such as a computed tomography scan. If the child is being manually ventilated, transition to mechanical ventilation.

The goals of respiratory management in the immediate post–cardiac arrest period are listed in Table 62.

Table 62.Goals of Respiratory Management in Immediate Post–Cardiac Arrest Period

Goal	Considerations
Maintain adequate oxygenation (generally an O_2 saturation of 94%-99%) to reduce the risk of reperfusion injury	Once ROSC is achieved, titrate O_2 administration to target normoxemia while ensuring that hypoxemia is strictly avoided. Maintain an O_2 saturation of 94%-99%, avoiding hypoxemia and hyperoxia (an O_2 saturation of 100% can correspond to a PaO_2 of anywhere between 80 and approximately 500 mm Hg). Determining optimal PaO_2 and O_2 saturation requires evaluation of the child's arterial O_2 content because it is an important determinant of tissue O_2 delivery. If the child is anemic, tissue O_2 delivery may be better maintained by achieving a high PaO_2 and O_2 saturation. In comparison, an O_2 saturation of 94%-100% is typically adequate in a child with a normal hemoglobin concentration and normal O_2 consumption and no cyanotic heart disease. Thus, oxygen is titrated to a value appropriate to the specific patient condition
Maintain adequate ventilation and $PaCO_2$ appropriate to the patient	It is reasonable to target a $PaCO_2$ (or end-tidal CO_2 if $PaCO_2$ is unavailable) that is appropriate to each child's clinical condition and to limit exposure to severe hypercapnia or hypocapnia. For example, for most patients with neurologic injury, a normal $PaCO_2$ is desirable to avoid hypocapnia (hypocarbia) or hypercapnia (hypercarbia). However, in children with asthma and respiratory failure, rapid correction of hypercarbia is unnecessary. Efforts to achieve normocarbia with mechanical ventilation in a child with asthma could result in complications such as pneumothorax. Conversely, in children with congenital heart disease and pulmonary hypertension, hypercarbia must be avoided

General Recommendations

General recommendations for assessment and management of the respiratory system may include the following:

Monitoring: Continuously monitor the following parameters (at a minimum):

- SpO_2 and heart rate by pulse oximetry (compare pulse oximetry heart rate with ECG and pulse rate to ensure that pulse oximeter values are accurate)
- Heart rate and rhythm
- If the patient is intubated, monitor end-tidal CO_2 by capnography if equipment and expertise are available, or intermittently confirm exhaled CO_2 by colorimetric device. Always monitor exhaled CO_2 by either capnography or colorimetric device during intrahospital and interhospital transport to aid in immediate detection of inadvertent extubation.
- If the child is already intubated, verify tube position, openness/patency, and security.
- After proper tube position is confirmed, ensure that the tube is well taped and that tube position at the lip or gum is documented.
- Health care professionals must use both clinical assessment and confirmatory devices (such as monitoring of exhaled CO_2) to verify proper tube placement immediately after intubation, during transport, and when the child is moved (eg, from gurney to bed).

Physical examination:

- Observe for adequate and equal chest rise bilaterally and auscultate for abnormal or asymmetric breath sounds.
- Monitor for evidence of respiratory compromise (eg, tachypnea, increased work of breathing, agitation, decreased responsiveness, poor air exchange, cyanosis) or inadequate respiratory effort.

Laboratory tests: Obtain an arterial sample for ABG analysis if possible. If the child is mechanically ventilated, obtain the ABG 10 to 15 minutes after establishing initial ventilator settings; ideally, correlate blood gases with capnographic end-tidal CO_2 to enable noninvasive monitoring of ventilation.

Other tests: Obtain a chest x-ray to verify correct depth of the ET tube insertion and position in the mid-trachea, and to identify pulmonary conditions that may require specific treatment (eg, pneumothorax, aspiration).

Oxygenation:

- If the child is not intubated, provide supplemental O_2 with a partial or a nonrebreathing mask until you confirm adequate SpO_2.
- After ROSC, adjust inspired O_2 concentration to achieve an SpO_2 of 94%-99%.
- If the child has an SpO_2 of <90% while receiving 100% inspired O_2, consider noninvasive ventilatory support or ET intubation with mechanical ventilation and PEEP.

- If the child has a cyanotic cardiac lesion, adjust the O_2 saturation goal to the child's baseline SpO_2 and clinical status.

Ventilation: Assist ventilation as needed, targeting a normal $PaCO_2$ (or $ETCO_2$ if $PaCO_2$ is unavailable) (ie, 35-45 mm Hg) if the child's lung function was previously normal. Remember that normalizing $PaCO_2$ may not be appropriate in all situations. Avoid routine hyperventilation in children with neurologic problems unless there are signs of impending cerebral herniation. Limit exposure to severe hypercapnia or hypocapnia.

Respiratory failure:

- Intubate the trachea if O_2 administration and other interventions do not achieve adequate oxygenation and ventilation. Intubate if needed to maintain an open/patent airway and adequate oxygenation and ventilation in the child with decreased level of consciousness. In some patients, CPAP or noninvasive ventilation may be adequate.
- Use age- and weight-appropriate ventilator settings.
- Verify ET tube position, openness/patency, and security; retape, if needed, before transport.
- Assess for a large glottic air leak. Consider reintubation with a cuffed tube or a larger tube if the glottic air leak prevents adequate chest rise, oxygenation, or ventilation. Weigh the risk of removing the advanced airway against the benefit of improving tidal volume, oxygenation, and ventilation.
- If a cuffed ET tube is in place and is inflated, check the cuff pressure (goal for most tubes is <20 to 25 cm H_2O; follow the manufacturer's recommendations) or assess for the presence of a minimal glottic air leak at an inspiratory pressure of <20 to 25 cm H_2O.
- Insert a gastric tube to relieve and help prevent gastric inflation.

Use the "DOPE" mnemonic to troubleshoot acute deterioration in a mechanically ventilated patient. (Refer to the Sudden Deterioration in an Intubated Patient section in Part 6: Managing Respiratory Problems.)

Analgesia and sedation:

- Control pain with analgesics (eg, fentanyl, morphine) and anxiety with sedatives (eg, lorazepam, midazolam) as needed.
- Administer sedation and analgesia to all responsive intubated patients.

Use lower doses of sedatives or analgesics if the child is hemodynamically unstable; titrate the dose while stabilizing hemodynamic function. When used in equipotent doses, morphine is more likely than fentanyl to cause hypotension because morphine causes histamine release.

Neuromuscular blockade: For the intubated patient with poor oxygenation and ventilation despite adequate sedation and analgesia, assess for acute causes of deterioration by using the DOPE mnemonic. Then consider neuromuscular blocking agents (eg, vecuronium, cisatracurium) with sedation. Indications for use of neuromuscular blocking agents include

- High peak or mean airway pressure caused by high airway resistance or reduced lung compliance
- Patient ventilator asynchrony
- Difficult airway

Neuromuscular blockade may reduce the risk of ET tube displacement. Be aware that neuromuscular blockers do not provide sedation or analgesia and will mask seizures. Neuromuscular blockers will also eliminate many signs of agitation that may signal inadequate oxygenation and ventilation. When using neuromuscular blockers, always ensure that the child is adequately sedated by evaluating for signs of stress, such as tachycardia, hypertension, pupil dilation, or tearing.

Cardiovascular System

Management Priorities

Ischemia resulting from cardiac arrest and subsequent reperfusion can cause circulatory dysfunction that can last for hours after ROSC. Compromised tissue perfusion and oxygenation from shock and respiratory failure can have secondary adverse effects on cardiovascular function. Health care professionals should maintain adequate blood pressure, cardiac output, and distribution of blood flow to restore or maintain tissue oxygenation and delivery of metabolic substrates. Circulatory management priorities include

- Restoring and maintaining intravascular volume (preload)
- Treating myocardial dysfunction
- Controlling arrhythmias
- Maintaining normal blood pressure and adequate systemic perfusion
- Maintaining adequate Sp_{O_2} and Pa_{O_2}
- Maintaining adequate hemoglobin concentration
- Considering therapies to reduce metabolic demand (eg, support ventilation, reduce temperature)

This section includes

- General recommendations for advanced evaluation and management of the cardiovascular system
- The Cardiovascular Management After ROSC Algorithm
- Information about administering maintenance fluids

Review Part 7: Recognizing Shock and Part 8: Managing Shock for more information about the pathophysiology of shock and the use of fluid therapy and medications to maintain cardiac output and tissue perfusion.

General Recommendations for Assessing and Managing the Cardiovascular System

Assessment

Monitoring: Monitor the following frequently or continuously:

- Heart rate and rhythm by cardiac monitor
- Blood pressure and pulse pressure (noninvasively or invasively)
- SpO_2 by pulse oximetry
- Urine output by urinary catheter
- Temperature

In the critical care setting, also consider monitoring

- Central venous pressure by central venous catheter
- Central venous O_2 saturation by catheter providing continuous $ScvO_2$ or by intermittent blood draw
- Trends in regional venous oxygenation by near infrared spectroscopy
- Cardiac function (eg, echocardiogram) and/or cardiac output by noninvasive monitoring

Noninvasive blood pressure monitoring (ie, by automated blood pressure devices) is often unreliable in children with poor perfusion or frequent arrhythmias. Blood pressure monitoring with an indwelling arterial catheter and monitoring system is more reliable in these children, provided the catheter is open/patent and the transducer is appropriately zeroed and leveled.

Physical examination:

- Repeat the physical examination (eg, evaluate quality of central and peripheral pulses, heart rate, capillary refill, blood pressure, extremity temperature and color) frequently until the child is stable.
- Monitor end-organ function (eg, neurologic function, renal function, skin perfusion) to assess circulatory function.

Laboratory tests:

- ABG or VBG
- Hemoglobin and hematocrit
- Serum glucose, electrolytes, blood urea nitrogen, creatinine, calcium
- Consider monitoring lactate and central venous O_2 saturation

In addition to pH, note the magnitude of any metabolic acidosis (base deficit). A persistent metabolic (lactic) acidosis suggests inadequate cardiac output and O_2 delivery. Serum electrolytes can help identify an anion gap acidosis. If the child has an elevated anion gap but normal lactate, consider other causes of acidosis, such as toxins or uremia.

The difference in O_2 saturation between an arterial and superior vena caval blood sample [$S(a\text{-}v)O_2$] provides information about the balance of oxygen supply vs demand. Assuming that O_2 consumption remains constant, a high $S(a\text{-}v)O_2$ difference (greater than 35-40) suggests low oxygen delivery. This may be caused by a fall in cardiac output, or arterial oxygen content. When oxygen delivery falls, there will be increased O_2 extraction in the tissues (ie, blood flow and O_2 delivery are decreased, so O_2 extraction must increase), producing a fall in the superior vena caval oxygen saturation. Alternatively, increased O_2 consumption with stable cardiac output can result in a widened $a\text{-}vO_2$ difference as well.

- If there's a clinical indication to check a troponin, realize that troponin concentrations are frequently elevated after cardiac arrest. This may be due to the arrest itself, chest compressions, or defibrillation.

Nonlaboratory tests:

- Perform a chest x-ray to evaluate ET tube insertion depth and position in the mid-trachea, assess heart size, and identify pulmonary edema or other pathology.
- Evaluate a 12-lead ECG for arrhythmias or evidence of myocardial ischemia.
- Consider echocardiography if there is concern about pericardial tamponade or myocardial dysfunction.

A small heart is often present with reduced cardiac preload or severe lung hyperinflation. A large heart may be associated with normal or increased cardiac preload, pericardial effusion, CHF, or when the patient is unable to take a deep breath (eg, with severe abdominal distension).

Management

Intravascular volume:

- Establish secure vascular access (if possible, 2 catheters, either IV or IO).
- Administer fluid boluses (10-20 mL/kg of isotonic crystalloid over 5-20 minutes) as needed to establish adequate intravascular volume. Smaller boluses of fluid (5-10 mL/kg) administered over 10 to 20 minutes may be appropriate in the setting of heart failure. Adjust the fluid administration rate to replace fluid deficits and meet ongoing requirements. Avoid excessive fluid administration in the presence of myocardial dysfunction, heart failure, or respiratory failure.
- Consider the need for colloid or blood administration (especially in trauma).
- Calculate maintenance fluid requirements and administer as appropriate.

Do not use boluses of hypotonic or dextrose-containing fluids for volume resuscitation.

Refer to Administer Maintenance Fluids later in this Part.

Blood pressure:

- Treat hypotension aggressively, titrating volume and vasoactive medications as appropriate.
- Post–cardiac arrest systolic blood pressure must be maintained above the 10th percentile for age.
- If hypotension is due to excessive vasodilation (eg, sepsis), early use of a vasopressor may be indicated.
- The use of adrenergic agents during resuscitation may produce elevation in SVR and cause hypertension. Because the half-life of these agents is relatively short, assess for other causes of hypertension in the post–cardiac arrest phase (eg, pain, anxiety, seizures).

Treatment of hypotension is crucial to avoid secondary multisystem injury. Refer to the Cardiovascular Management After ROSC Algorithm for more information about management of hypotensive and normotensive shock.

Tissue oxygenation:

- Provide supplemental O_2 in a sufficient concentration to ensure adequate oxygenation.
- After ROSC following cardiac arrest, titrate O_2 to maintain adequate SpO_2 (94%-99%).
- Support adequate perfusion.
- Consider transfusion with PRBCs for patients with low hematocrit and signs of inadequate O_2 delivery.

Metabolic demand:

- Consider ET intubation and assisted ventilation to reduce the work of breathing.
- Control pain with analgesia (eg, morphine, fentanyl).
- Control agitation as needed with sedation (eg, lorazepam, midazolam); rule out hypoxemia, hypercarbia, or poor perfusion as potential causes of agitation.
- Control fever with antipyretics and a cooling blanket if needed (neuromuscular blockade may be appropriate to control shivering).

Caution: Sedatives or analgesics may cause hypotension. Consider expert consultation before elective ET intubation. The use of sedatives or analgesics, ET intubation, and initiating positive-pressure ventilation can all precipitate cardiovascular collapse in a child with poor myocardial function.

Arrhythmias:

- Monitor for tachyarrhythmias and bradyarrhythmias and treat aggressively.

- If bradycardia develops, first ensure adequate oxygenation and ventilation; if the heart rate remains <60/min with signs of poor perfusion despite adequate oxygenation and ventilation, initiate CPR. Bradycardia can also occur in the setting of hypothermia; consider whether perfusion and blood pressure are adequate.
- If arrhythmia persists, control with medications or electrical therapy per algorithms.
- Seek expert consultation for arrhythmia management.

Refer to Part 9: Recognizing Arrhythmias and Part 10: Managing Arrhythmias for more information.

Postarrest myocardial dysfunction:

- Anticipate postarrest myocardial dysfunction in the first 24 hours after ROSC.
- Consider vasoactive agents to improve contractility and/or decrease afterload if blood pressure is adequate.
- Correct metabolic abnormalities that can contribute to poor myocardial function (eg, acidosis, hypocalcemia, hypoglycemia).
- Consider positive-pressure ventilation (noninvasive ventilation or via ET tube) to improve left ventricular function.

Myocardial dysfunction is common in children after resuscitation from cardiac arrest. Postarrest myocardial dysfunction can produce hemodynamic instability and secondary organ injury and may precipitate another cardiac arrest.

Treatment of Shock

After resuscitation from cardiac arrest or shock, hemodynamic compromise may result from a combination of

- Inadequate intravascular volume
- Decreased cardiac contractility
- Either increased or decreased SVR or pulmonary vascular resistance

Children with cardiogenic shock typically have poor myocardial function and a compensatory increase in systemic and pulmonary vascular resistances in an attempt to maintain an adequate blood pressure. The increased SVR may become detrimental because it increases left ventricular afterload. Very low SVR most commonly occurs in children with early septic shock. When children with septic shock do not respond to bolus fluid administration (ie, the shock is fluid refractory), they may have high rather than low SVR and poor myocardial function, similar to cardiogenic shock.

Support of Systemic Perfusion

The parameters listed in Table 63 can be manipulated to optimize systemic perfusion.

Table 63.Parameters to Optimize Systemic Perfusion

Parameters to optimize	Action (when needed)
Preload	• Administer fluid bolus
Contractility	• Administer inotropes • Correct hypoxia, electrolyte and acid-base imbalances, and hypoglycemia/hypocalcemia • Treat poisonings (eg, administer antidotes if available)
Afterload (SVR)	• Administer vasopressors or vasodilators as appropriate
Heart rate	• Administer chronotropes for bradycardia (eg, epinephrine) • Administer antiarrhythmics • Correct hypoxia • Consider pacing

Refer to the Pathophysiology of Shock section in Part 7 for a discussion of preload, afterload, and contractility.

Managing Shock After ROSC

The Cardiovascular Management After ROSC Algorithm (Figure 59) outlines evaluation and management steps after cardiac arrest.

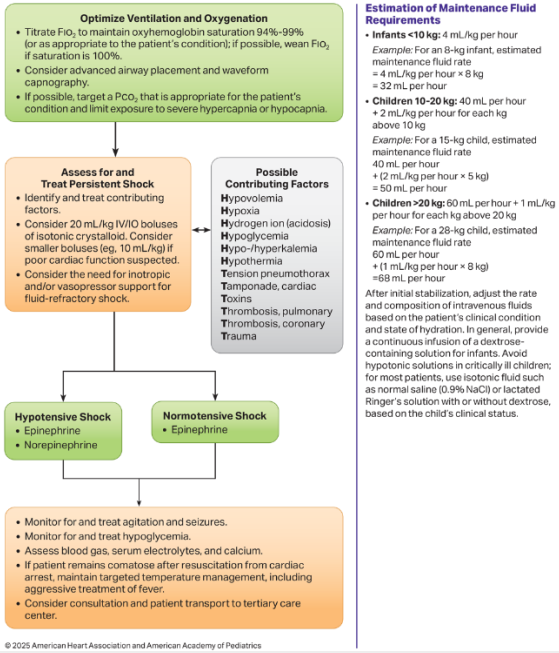


Figure 59. Cardiovascular Management After ROSC Algorithm.

Algorithm legend: Red hexagon = decision or question; green box = action/steps to consider; orange box = assessment; gray box = assessment/action; blue box = CPR; white box with gray header = notes; and red, bold text = possible answers or decisions.

Optimize oxygenation and ventilation

Maintain an O_2 saturation of 94% to 99%. Consider placing an advanced airway and using waveform capnography if not yet established. If possible, target a PCO_2 that is appropriate for the patient's conditions and limit exposure to severe hypercapnia or hypocapnia.

Fluid therapy

Consider administering a fluid bolus of 20 mL/kg IV/IO bolus of isotonic crystalloid. If you suspect postarrest myocardial dysfunction, consider administering a smaller fluid bolus (5-10 mL/kg) over 10 to 20 minutes, and then reassess. Excessive fluid administration can worsen cardiopulmonary function. Consider the need for inotropic and/or vasopressor support for fluid-refractory shock.

Hypotensive shock

If the child remains hypotensive after bolus fluid administration, consider an infusion of one or a combination of the following medications:

- Epinephrine: for IV/IO route, give 0.03 to 0.2 mcg/kg per minute* *and/or*
- Norepinephrine: for IV/IO route, give 0.05 to 0.5 mcg/kg per minute*

*Upper limit dosing range can be highly variable and should be based on clinical scenarios.

Ensure that cardiac preload is adequate. Base your choice of medication on the most likely cause of hypotension (inadequate heart rate, poor contractility, excessive vasodilation, or a combination of factors). If the heart rate is abnormally low, administering catecholamine may increase the heart rate and cardiac output. However, when catecholamines cause extreme tachycardia, they increase myocardial O_2 demand. Continue to carefully assess the child with each intervention.

Epinephrine

Epinephrine is a potent vasoactive agent that can either lower or increase SVR depending on the infusion dose. Low-dose infusions generally produce β-adrenergic effects (increased heart rate and contractility and vasodilation); higher doses generally produce α-adrenergic effects (vasoconstriction). Because there is great interpatient variability, titrate the medication to the desired clinical effect. Epinephrine may be preferable to dopamine in children (especially infants) with marked circulatory instability and hypotensive shock.

Norepinephrine

Norepinephrine is a potent inotropic and peripheral vasoconstricting agent. Titrate the infusion to treat shock with low SVR (septic, anaphylactic, spinal) that is unresponsive to bolus fluid administration.

Normotensive shock

If the child is normotensive but remains poorly perfused after bolus fluid administration, consider administering low-dose epinephrine: for IV/IO route, give 0.03 to 0.05 mcg/kg per minute.

Post–cardiac arrest considerations

ROSC affects multiple organ systems.

- Monitor for and treat agitation and seizures with appropriate medications.
- Monitor for and treat hypoglycemia.
- Assess blood gas, serum, electrolytes, and calcium.
- Correct metabolic derangements, noting that treating metabolic acidosis is best accomplished by treating the underlying cause of the acidosis (ie, restore perfusion in shock).

- Provide targeted temperature management including continuous monitoring of central temperature and prevention or aggressive treatment of temperatures greater than 37.5°C.
- Consider the need for therapeutic hypothermia.
- Arrange for transfer to an appropriate pediatric critical care unit.

Administer Maintenance Fluids

Maintenance Fluid Composition

After initial stabilization, adjust the rate and composition of IV fluids based on the patient's condition. If cardiovascular function is adequate, consider administering maintenance fluids once intravascular volume has been restored and fluid deficits have been replaced. When you calculate the maintenance fluid requirements, include the total fluids of continuous infusions.

In the first hours after resuscitation, the appropriate composition of IV fluids is an isotonic crystalloid with or without dextrose, based on the child's condition and age. Avoid hypotonic fluids in critically ill children in the post–cardiac arrest phase. Recent evidence suggests that buffered solutions (eg, lactated Ringer's, other electrolyte-containing isotonic solution) may be preferred because hyperchloremia has been associated with poorer outcomes. However, if you use one of these solutions, you must ensure that the child is receiving some source of dextrose because hypoglycemia will likely occur.

You may add specific components to maintenance fluids based on the clinical condition:

- Include dextrose in IV fluids for infants and for children who are hypoglycemic or at risk for hypoglycemia.
- Add potassium chloride (KCl) 10 to 20 mEq/L for children with adequate renal function and documented urine output once periodic monitoring of potassium is available. Do not add KCl to maintenance fluid in children with hyperkalemia, renal failure, muscle injury, or severe acidosis.

Maintenance Fluid Calculation by 4-2-1 Method

Use the 4-2-1 method as a practical approach to estimating hourly maintenance fluid requirements in children (Table 64).

Table 64.Estimating Maintenance Fluid Requirements

Weight (kg)	Estimated hourly fluid requirements	Sample collection
<10	4 mL/kg per hour	8-kg infant: 4 mL/kg per hour × 8 kg = 32 mL/h

10-20	40 mL/h + 2 mL/kg per hour for each kilogram between 10 and 20 kg	15-kg child: 40 mL/h + 2 mL/kg per hour × 5 kg = 50 mL/h
>20	60 mL/h + 1 mL/kg per hour for each kilogram above 20 kg*	30-kg child: 60 mL/h + 1 mL/kg per hour × 10 kg = 70 mL/h

*An alternate calculation of maintenance hourly fluid rate for patients weighing more than 20 kg is weight in kilograms + 40 mL/h.

Once you have calculated the estimated maintenance fluid requirements, adjust the actual rate of fluid administration to the child's clinical condition (eg, pulse, blood pressure, systemic perfusion, urine output) and hydration level.

Neurologic System

Management Priorities

During the post–cardiac arrest period, health care professionals should aim to preserve brain function and prevent secondary neuronal injury. Neurologic management priorities include

- Maintaining adequate brain perfusion
- Maintaining normoglycemia
- Providing targeted temperature management: prevent or aggressively treat fever and consider therapeutic hypothermia if indicated
- Treating increased ICP
- Treating seizures; searching for and treating cause

General Recommendations

Refer to Table 65 for general recommendations for assessment and management of the neurologic system.

Table 65.General Recommendations for Assessing and Managing the Neurologic System

	Assessment
Monitoring	• Monitor heart rate and systemic blood pressure • Monitor temperature

	In children with poor peripheral perfusion, reliable monitoring of core temperature requires invasive devices (rectal, bladder, esophageal thermometer)
Physical examination	• Perform frequent, brief neurologic assessments (eg, Glasgow Coma Scale, pupil responses, gag reflex, corneal reflexes, oculocephalic reflexes) • Identify signs of impending cerebral herniation • Identify seizure activity • Identify abnormal neurologic findings, including abnormal movements (posturing/myoclonus/hyperreflexia) Signs of impending cerebral herniation include unequal or dilated unresponsive pupils, posturing, hypertension, bradycardia, respiratory irregularities or apnea, and reduced response to stimulation. A sudden increase in ICP (if monitoring is in place) is often observed. Other causes of central nervous system dysfunction are hypoxic-ischemic brain injury, hypoglycemia, convulsive or nonconvulsive seizures, toxins/medications, electrolyte abnormalities, hypothermia, traumatic brain injury, stroke or intracranial hemorrhage, and central nervous system infection. Refer to the section Disability in Part 4 for more information on neurologic assessment.
Laboratory tests	• Perform point-of-care glucose testing; repeat measurement after treatment of hyperglycemia or hypoglycemia • Obtain serum electrolytes, point-of-care glucose, and serum ionized calcium concentration if seizure activity is present; measure concentrations of anticonvulsant medications if the child was receiving these agents • Consider toxicologic studies if poisoning or overdose is suspected • Consider cerebral spinal fluid studies if central nervous system infection is suspected, but defer a lumbar puncture if the patient's cardiopulmonary status is not stable
Nonlaboratory tests	• Consider a computed tomography scan if central nervous system dysfunction or neurologic deterioration is present • Consider an electroencephalogram (EEG) if nonconvulsive status epilepticus is suspected or seizures are a concern during administration or duration of effect of neuromuscular blockers. EEGs performed up to 72 hours after pediatric cardiac arrest may be considered in prognosticating neurologic outcome at the time of hospital discharge but should not be used as the sole criterion. For more information, see the Appendix. Additional details and guidance can be accessed through PALS Plus at **shopcpr.heart.org/pals-plus**
Management	
Brain perfusion	• Optimize brain perfusion by supporting cardiac output and arterial O_2 content • Avoid hyperventilation unless there are signs of impending cerebral herniation

	Support cardiac output by optimizing heart rate, preload, afterload, and contractility. Refer to the section Support of Systemic Perfusion earlier in this Part for more information.
Blood glucose	• Treat hypoglycemia. • Monitor glucose concentration. In general, try to avoid causing or worsening hyperglycemia. • In the critical care setting, consider treating persistent hyperglycemia; careful monitoring is needed to prevent hypoglycemia. Although hyperglycemia is associated with poor outcome in critically ill children, the relative benefits of active treatment of hyperglycemia vs risks of hypoglycemia in critically ill children remains uncertain. In most animal studies, hyperglycemia at the time of cerebral ischemia produces a worse outcome, but the effect of hyperglycemia occurring after ROSC is less clear.
Targeted temperature management	Provide targeted temperature management. This includes preventing or aggressively treating fever and considering therapeutic hypothermia. **Prevent/treat fever** • Prevent fever; adjust environmental temperature as needed (cooling devices may be used to maintain a target temperature and prevent fever). • Aggressively treat fever (temperature 37.5 °C or higher) with antipyretics and cooling devices (eg, cooling blanket) or procedures. • Do not actively rewarm a post–cardiac arrest patient who has a temperature between 32 °C and 37 °C after ROSC unless hypothermia is contributing to hemodynamic instability. Fever adversely influences recovery from ischemic brain injury and is associated with poor outcome after resuscitation from cardiac arrest. Metabolic O_2 demand increases by 10% to 13% for each degree Celsius elevation of temperature above normal. Increased metabolic demand may worsen neurologic injury. Furthermore, fever increases the release of inflammatory mediators, cytotoxic enzymes, and neurotransmitters, which increase brain injury. **Therapeutic hypothermia** • For infants and children who remain comatose after out-of-hospital cardiac arrest, it is reasonable either to maintain 5 days of continuous normothermia (36 °C-37.5 °C) or to maintain 2 days of initial continuous hypothermia (32 °C-34 °C), followed by 3 days of continuous normothermia. • For infants and children remaining comatose after in-hospital cardiac arrest, there is insufficient evidence to recommend cooling over normothermia, but all patients should receive targeted temperature management that prevents or aggressively treats fever. During cooling, treatment or prevention of shivering is often required. • Monitor for and treat complications of hypothermia, including diminished cardiac output, arrhythmia, infection, pancreatitis, coagulopathy, thrombocytopenia, hypophosphatemia, and hypomagnesemia.

Increased ICP	• Elevate the head of bed to 30° if blood pressure is adequate and no spinal precautions are in place. • Keep head in midline. • Decrease environmental stimuli such as buzzers, bells, or alarms. Dim lights to decrease stimulation if able. • Support adequate ventilation to maintain normocapnia. • If signs of impending cerebral herniation develop (eg, irregular respirations or apnea, bradycardia, hypertension, unequal or dilated pupil[s] not responsive to light, decerebrate or decorticate posturing), a brief period of mild hyperventilation may occasionally be used as temporizing rescue therapy. • Consider mannitol or hypertonic saline for acute herniation syndrome. • For children with neurosurgical conditions (eg, traumatic brain injury, intracranial hemorrhage), obtain expert consultation about indications for monitoring of ICP and/or neurosurgical intervention. Prolonged hyperventilation is not effective to treat increased ICP, and excessive hyperventilation may worsen neurologic outcome. Hypocarbia results in cerebral vasoconstriction, reducing cerebral blood flow. Hyperventilation also reduces venous return and cardiac output, contributing to cerebral ischemia.
Seizures	• Treat seizures aggressively. Therapeutic options include a benzodiazepine (eg, lorazepam, midazolam), fosphenytoin/phenytoin, levetiracetam or a barbiturate (eg, phenobarbital). Monitor blood pressure carefully if you use phenytoin or phenobarbital because these medications may cause hypotension. • Search for a correctable metabolic cause, such as hypoglycemia, hyponatremia, or hypocalcemia. • Consider toxins or metabolic disease as the etiology. • Consult a neurologist if available.

Appendix

BLS Competency Testing

BLS Skills Testing Checklists

The Child CPR and AED Skills Testing Checklist and the Infant CPR Skills Testing Checklist provide detailed descriptions of the CPR skills that you will be expected to perform. Your instructor will evaluate your CPR skills during the skills test on the basis of these descriptions.

If you perform a specific skill exactly as described in the critical performance criteria details, the instructor will check that specific skill as “passing.” If you do not perform a specific skill exactly as it is described, the skill will not be checked off and you will require remediation in that skill.

Study the BLS skills testing checklists so that you will be able to perform each skill correctly.

Pediatric Advanced Life Support

Child CPR and AED Skills Testing Checklist

American Heart Association. | American Academy of Pediatrics
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Student Name _____ Date of Test _____

Directions: In groups of 3 or 4 students, assign a CPR Coach for each group. The CPR Coach will perform the role of the AED/Monitor/Defibrillator and will not switch from that position. Have groups perform 5-minute rounds of CPR with at least 1 Compressor switch. Total number of 5-minute intervals is equal to the number of students in the group. Ensure that every student has a chance to practice the CPR Coach role.

Hospital Scenario: "You are working in a hospital or clinic, and you see a child who has suddenly collapsed in the hallway. You check that the scene is safe and then approach the patient. Demonstrate what you would do next."

Prehospital Scenario: "You arrive on the scene for a child who is not breathing. No bystander CPR has been provided. You approach the scene and ensure that it is safe. Demonstrate what you would do next."

Assessment and Activation

- ☐ Checks responsiveness
- ☐ Shouts for help/Activates emergency response system/Sends for AED
- ☐ Checks breathing
- ☐ Checks pulse

Once student shouts for help, instructor says, "Here's the barrier device. I am going to get the AED."

Student must complete 2 cycles of CPR (30:2, approximately 1 minute) as a single rescuer before the second rescuer and CPR Coach arrive to help.

Cycle 1 of CPR (30:2) ***CPR feedback devices are required for accuracy****

Child Compressions

- ☐ *Performs high-quality compressions:
  - Hand placement on lower half of sternum
  - 30 compressions in no less than 15 and no more than 18 seconds
  - Compresses at least one third the AP diameter of the chest, approximately 2 inches (5 cm)
  - Complete recoil after each compression

Child Breaths

- ☐ Gives 2 breaths with a barrier device:
  - Each breath given over 1 second
  - Visible chest rise with each breath
  - Resumes compressions in less than 10 seconds

Cycle 2 of CPR (repeats steps in Cycle 1) ***Only check box if step is successfully performed***

- ☐ Compressions
- ☐ Breaths
- ☐ Resumes compressions in less than 10 seconds

Rescuer 2 says, "Here is the AED. I'll take over compressions, and you use the AED."

AED (follows prompts of AED)

- ☐ Powers on AED
- ☐ Correctly attaches pads
- ☐ Clears for analysis
- ☐ Clears to safely deliver a shock
- ☐ Safely delivers a shock

Resumes Compressions

- ☐ Ensures that compressions are resumed immediately after shock delivery
  - Student directs instructor to resume compressions *or*
  - Second student resumes compressions

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

Pediatric Advanced Life Support

Child CPR and AED

Skills Testing Critical Skills Descriptors

1. **Assesses victim and activates emergency response system (this must precede starting compressions) within a maximum of 30 seconds. After determining that the scene is safe:**
    - Checks for responsiveness by tapping and shouting
    - Shouts for help/directs someone to call for help and get AED/defibrillator
    - Checks for no breathing or no normal breathing (only gasping)
        - –Scans from the head to the chest for a minimum of 5 seconds and no more than 10 seconds
    - Checks carotid pulse
        - –Can be done simultaneously with check for breathing
        - –Checks for a minimum of 5 seconds and no more than 10 seconds
2. **Performs high-quality chest compressions (initiates compressions immediately after recognition of cardiac arrest)**
    - Correct hand placement
        - –Lower half of sternum
        - –2-handed (second hand on top of the first or grasping the wrist of the first hand) or 1-handed
    - Compression rate of 100 to 120/min
        - –Delivers 30 compressions in 15 to 18 seconds
    - Compression depth and recoil—compress at least one third the AP diameter of the chest, approximately 2 inches (5 cm)
        - –Use of a commercial feedback device or high-fidelity manikin is required
        - –Complete chest recoil after each compression
    - Minimizes interruptions in compressions
        - –Delivers 2 breaths so that less than 10 seconds elapses between last compression of one cycle and first compression of next cycle
        - –Compressions resumed immediately after shock/no shock indicated
3. **Provides 2 breaths by using a barrier device**
    - Opens airway adequately
        - –Uses a head tilt–chin lift maneuver or jaw thrust
    - Delivers each breath over 1 second

- Delivers breaths that produce visible chest rise
- Avoids excessive ventilation
- Resumes chest compressions in less than 10 seconds

4. **Performs same steps for compressions and breaths for Cycle 2**
5. **AED use**
    - Powers on AED
        - –Turns AED on by pushing button or lifting lid as soon as it arrives
    - Correctly attaches pads
        - –Places proper-sized pads for victim's age in correct location
    - Clears for analysis
        - –Clears rescuers from victim for AED to analyze rhythm (pushes analyze button if required by device)
        - –Communicates clearly to all other rescuers to stop touching victim
    - Clears to safely deliver shock
        - –Communicates clearly to all other rescuers to stop touching victim
    - Delivers a shock
        - –Resumes chest compressions immediately after shock delivery
        - –Does not turn off AED during CPR
6. **Resumes compressions**
    - Ensures that high-quality chest compressions are resumed immediately after shock delivery
    - Performs same steps for compressions

Pediatric Advanced Life Support

Infant CPR Skills Testing Checklist

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Student Name _____ Date of Test _____

Directions: In groups of 3 or 4 students, assign a CPR Coach for each group. The CPR Coach will perform the role of the AED/Monitor/Defibrillator and will not switch from that position. Have groups perform 5-minute rounds of CPR with at least 1 Compressor switch. Total number of 5-minute intervals is equal to the number of students in the group. Ensure that every student has a chance to practice the CPR Coach role.

Hospital Scenario: "You are working in a hospital or clinic when a woman runs through the door, carrying an infant. She shouts, 'Help me! My baby's not breathing.' You have gloves and a pocket mask. You send your coworker to activate the emergency response system and to get the emergency equipment."

Prehospital Scenario: "You arrive on the scene for an infant who is not breathing. No bystander CPR has been provided. You approach the scene and ensure that it is safe. Demonstrate what you would do next."

Assessment and Activation

☐ Checks responsiveness ☐ Shouts for help/Activates emergency response system
☐ Checks breathing ☐ Checks pulse

Once student shouts for help, instructor says, "Here's the barrier device."

Student must complete 2 cycles of CPR (30:2, approximately 1 minute) as a single rescuer before the second rescuer and CPR Coach arrive to help.

Cycle 1 of CPR (30:2) ***CPR feedback devices are preferred for accuracy****

Infant Compressions

☐ *Performs high-quality compressions:
- Placement of 2 thumbs or the heel of one hand in the center of the chest
- 30 compressions in no less than 15 and no more than 18 seconds
- Compresses at least one third the AP diameter of the chest, approximately 1½ inches (4 cm)
- Complete recoil after each compression

Infant Breaths

☐ Gives 2 breaths with a barrier device:
- Each breath given over 1 second
- Visible chest rise with each breath
- Resumes compressions in less than 10 seconds

Cycle 2 of CPR (repeats steps in Cycle 1) ***Only check box if step is successfully performed***

☐ Compressions ☐ Breaths ☐ Resumes compressions in less than 10 seconds

Rescuer 2 arrives with bag-mask device and begins ventilation while Rescuer 1 continues compressions with 2 thumb–encircling hands technique.

Cycle 3 of CPR

Rescuer 1: Infant Compressions

☐ *Performs high-quality compressions:
- 15 compressions with 2 thumb–encircling hands technique or heel-of-1-hand technique
- 15 compressions in no less than 7 and no more than 9 seconds
- Compresses at least one third the AP diameter of the chest, approximately 1½ inches (4 cm)
- Complete recoil after each compression

Rescuer 2: Infant Breaths

This rescuer is not evaluated.

Cycle 4 of CPR

Rescuer 2: Infant Compressions

This rescuer is not evaluated.

Rescuer 1: Infant Breaths

☐ Gives 2 breaths with a bag-mask device:
- Each breath given over 1 second
- Visible chest rise with each breath
- Resumes compressions in less than 10 seconds

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

Pediatric Advanced Life Support

Infant CPR

Skills Testing Critical Skills Descriptors

1. **Assesses victim and activates emergency response system (this must precede starting compressions) within a maximum of 30 seconds. After determining that the scene is safe:**
    - Checks for responsiveness by tapping and shouting
    - Shouts for help/directs someone to call for help and get emergency equipment
    - Checks for no breathing or no normal breathing (only gasping)
        - –Scans from the head to the chest for a minimum of 5 seconds and no more than 10 seconds
    - Checks brachial pulse
        - –Can be done simultaneously with check for breathing
        - –Checks for a minimum of 5 seconds and no more than 10 seconds
2. **Performs high-quality chest compressions during 1-rescuer CPR (initiates compressions within 10 seconds of identifying cardiac arrest)**
    - Correct placement of thumbs/hand in center of chest
        - –1 rescuer: 2 thumbs or heel of one hand
    - Compression rate of 100 to 120/min
        - –Delivers 30 compressions in 15 to 18 seconds
    - Adequate depth for age
        - –Infant: at least one third the AP diameter of the chest (approximately 1½ inches [4 cm])
        - –Use of a commercial feedback device or high-fidelity manikin is required
    - Complete chest recoil after each compression
    - Appropriate ratio for age and number of rescuers
        - –1 rescuer: 30 compressions to 2 breaths
    - Minimizes interruptions in compressions
        - –Delivers 2 breaths so that less than 10 seconds elapses between last compression of one cycle and first compression of next cycle
3. **Provides effective breaths with bag-mask device during 2-rescuer CPR**
    - Opens airway adequately
    - Delivers each breath over 1 second
    - Delivers breaths that produce visible chest rise

- Avoids excessive ventilation
- Resumes chest compressions in less than 10 seconds

4. **Performs high-quality chest compressions during 2-rescuer CPR**
    - Correct placement of thumbs/hand in center of chest
        - –2 rescuers: 2 thumb–encircling hands just below the nipple line
    - Compression rate of 100 to 120/min
        - –Delivers 15 compressions in 7 to 9 seconds
    - Adequate depth for age
        - –Infant: at least one third the AP diameter of the chest (approximately 1½ inches [4 cm])
    - Complete chest recoil after each compression
    - Appropriate ratio for age and number of rescuers
        - –2 rescuers: 15 compressions to 2 breaths
    - Minimizes interruptions in compressions
        - –Delivers 2 breaths so that less than 10 seconds elapses between last compression of one cycle and first compression of next cycle

Initial Assessment—Pediatric Assessment Triangle*

Appearance

From the doorway, your initial observation of the patient. Observe for

- Abnormal tone
- Decreased interaction
- Inconsolable

- Abnormal look/gaze
- Abnormal speech/cry

Work of Breathing

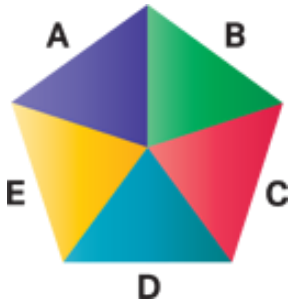
- Abnormal sounds
- Abnormal positioning
- Retractions
- Flaring
- Apnea/gasping

Circulation to the Skin

- Pallor
- Mottling
- Dusky
- Cyanosis

*If the patient is unresponsive, not breathing, or only gasping, initiate the Pediatric BLS Algorithm (Infants to Puberty) for Health Care Professionals—Single Rescuer or Pediatric BLS Algorithm (Infants to Puberty) for Health Care Professionals—2 or More Rescuers in Part 2.

Primary Assessment



A: Airway

Assessment

- Is the airway maintainable?
- Is the airway clear?
- If no to any of these, below are the interventions*

Interventions

- Maintain airway patency by positioning, using OPA
- Suction as indicated
- Advanced airway (eg, supraglottic airway or endotracheal tube)
- If inserting an advanced airway, verify correct placement with waveform capnography

B: Breathing

Assessment

- Adequate depth and rate of respirations
- Chest rise
- Noisy breathing (eg, grunting, stridor, wheezing)
- Use of accessory muscles, nasal flaring
- Pulse oximetry*

Interventions

- Provide high-flow O_2
- Bag-mask device with or without OPA
- Advanced airway
- Avoid excessive ventilation

C: Circulation

Assessment

- Adequate peripheral and/or central pulse
- Heart rate

- Blood pressure*
- Capillary refill—peripheral and/or central
- Skin color and temperature
- Level of consciousness

Interventions

- Obtain IV/IO access
- Consider fluid resuscitation

D: Disability

Assessment

- Quickly assess for responsiveness, level of consciousness, and pupillary response to light
- AVPU: Alert, Voice, Pain, and Unresponsive
- Check point-of-care glucose

Interventions

- Spinal motion restrictions
- Correct hypoglycemia
- Consider naloxone for acute opioid toxicity

E: Exposure

Assessment

- Remove clothing to perform a physical examination (anterior and posterior), looking for obvious signs of trauma, bleeding, burns, unusual markings, rashes, or medical alert bracelets
- Temperature

Interventions

- Ensure normothermia
- Control bleeding
- Decontamination

*If at any part of this sequence you find that a patient has a life-threatening condition, correction of that condition takes precedence over establishing baseline vital sign measures, such as blood pressure or pulse oximetry. When the primary assessment is completed and after life-threatening problems have been addressed, the health care professional proceeds to the secondary assessment (Consensus Statement: Emergency Medical Services for Children—Definitions and Pediatric Assessment Approaches. April 2005. Updated July 2015).

Remediation

Anyone who does not pass both skills tests will practice and undergo remediation during the remediation lesson at the end of the course.

Students who require remediation and retesting will be tested in the entire skill.

Skills Station Competency Checklists

Airway Management Skills Station Competency Checklist

Critical Performance Steps

- Verbalizes difference between high-flow and low-flow O_2 delivery systems
    - –High flow: O_2 flow exceeds patient inspiratory flow, preventing entrainment of room air if system is tight-fitting; delivers nearly 1.00 (100%) FIO_2 (eg, nonrebreathing mask with reservoir high-flow nasal cannula)
    - –Low flow (≤10 L/min): patient inspiratory flow exceeds O_2 flow, allowing entrainment of room air; delivers 0.22 (22%) to 0.60 (60%) FIO_2 (eg, nasal cannula, simple O_2 mask)
- Verbalizes maximum nasal cannula flow rate for standard nasal cannula (4 L/min)
- Opens airway by using head tilt–chin lift maneuver while keeping mouth open (jaw thrust for trauma victim)
    - –For more information, see instructor demonstration
- Verbalizes different indications for OPA and NPA
    - –OPA only for unconscious victim without a gag reflex
    - –NPA for conscious or semiconscious victim
- Selects correctly sized airway by measuring
    - –OPA from corner of mouth to angle of mandible
- Inserts OPA correctly
- Verbalizes assessment for adequate breathing after insertion of OPA

- Suctions with OPA in place; states suctioning not to exceed 10 seconds
- Selects correct mask size for ventilation
    - –For more information, see Part 6: Managing Respiratory Problems
- Assembles bag-mask device, opens airway, and creates seal by using E-C clamp technique
- With bag-mask device, gives 1 breath every 2 to 3 seconds for about 30 seconds. Gives each breath in approximately 1 second; each breath should cause chest rise
- Endotracheal Intubation
    - –States equipment needed for endotracheal (ET) tube intubation procedure
    - –Demonstrates technique to confirm proper ET tube placement by physical exam and by using an exhaled CO_2 device
    - –Secures ET tube
    - –Suctions with ET tube in place
    - –For more information, see Table 33. Pre-event Equipment Checklist for Endotracheal Intubation

The following steps are optional. They are demonstrated and evaluated only when the student's scope of practice involves ET intubation.

- Endotracheal intubation
    - –Prepares equipment for ET intubation
    - –Inserts ET tube correctly if within scope of practice

Rhythm Disturbances/Electrical Therapy Skills Station Competency Checklist

Critical Performance Steps

- Applies 3 ECG leads correctly (or local equipment if >3 leads are used)
    - –Negative (white) lead: to right shoulder
    - –Positive (red) lead: to left lower ribs
    - –Ground (black, green, brown) lead: to left shoulder
    - –For more information, see instructor demonstration
- Demonstrates correct operation of monitor
    - –Turns monitor on
    - –Adjusts device to manual mode (not AED mode) to display rhythm in standard limb leads (I, II, III) or paddles/electrode pads
    - –For more information, see instructor demonstration

- Verbalizes correct electrical therapy for appropriate core rhythms
    - –Synchronized cardioversion for unstable SVT, VT with pulses
    - –Defibrillation for pulseless VT, VF
    - –For more information, see Part 9: Recognizing Arrhythmias and Part 10: Managing Arrhythmias
- Selects correct paddle/electrode pad for infant or child; places paddles/electrode pads in correct position
    - –For more information, see Part 10: Managing Arrhythmias
- Demonstrates correct and safe synchronized cardioversion
    - –Places device in synchronized mode
    - –Selects appropriate energy (0.5 to 1.0 J/kg for initial shock)
    - –Charges, clears, and delivers current
    - –For more information, see Part 10: Managing Arrhythmias
- Demonstrates correct and safe manual defibrillation
    - –Places device in unsynchronized mode
    - –Selects energy (2 to 4 J/kg for initial shock)
    - –Charges, clears, and delivers current
    - –For more information, see Part 10: Managing Arrhythmias

Vascular Access Skills Station Competency Checklist

Critical Performance Steps

- Verbalizes indications for IO insertion
    - –For more information, see Resources for Managing Circulatory Emergencies in Part 8
- Verbalizes sites for IO insertion (anterior tibia, distal femur, medial malleolus, anterior-superior iliac spine)
- Verbalizes contraindications for IO placement
    - –Fracture in extremity
    - –Previous insertion attempt in the same bone
    - –Infection overlying bone
    - –Osteogenesis imperfecta
- Inserts IO catheter safely
- Verbalizes how to confirm IO catheter is in correct position; verbalizes how to secure IO catheter
- Attaches IV line to IO catheter; demonstrates giving IO fluid bolus by using 3-way stopcock and syringe
    - –For more information, see Instructor demonstration

- Shows how to determine correct drug doses by using a color-coded length-based tape or other resource
    - –For more information, see Table 50. Color-Coded Length-Based Resuscitation Tape
- *Optional:* Verbalizes correct procedure for establishing IV access

Rhythm Recognition Review

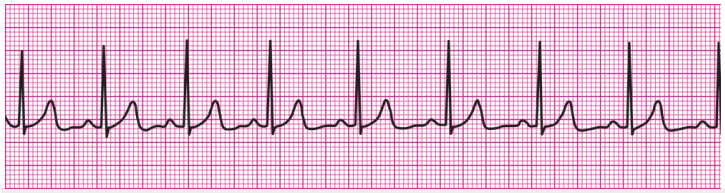


Figure 60. Rhythm Strip 1: Normal sinus rhythm, rate 100/min.

Note that every P wave in Figure 60 conducts to the ventricle, resulting in a QRS complex. Be aware that normal heart rates are age dependent in the pediatric population. For example, a heart rate of 75/min would be normal for a 10-year-old but bradycardic for a neonate. Likewise, a rate of 140/min would be normal for an infant but tachycardic for an adolescent.

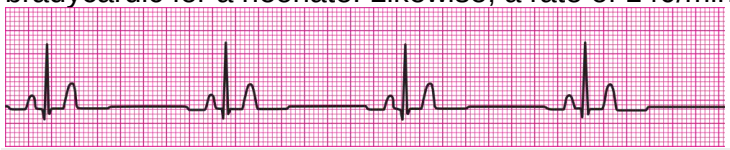


Figure 61. Rhythm Strip 2: Sinus bradycardia.

Note that the P waves in Figure 61 result in a QRS complex (conducted to the ventricle). The rate is very slow, especially in infants and small children (approximately 45/min). Sinus bradycardia is often a manifestation of hypoxemia and acidosis. It may be seen in healthy children, particularly during sleep.

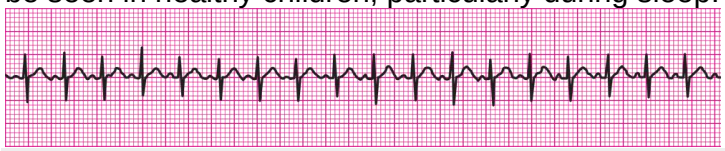


Figure 62. Rhythm Strip 3: Sinus tachycardia, 180/min.

Note that P waves in Figure 62 are visible preceding every QRS. The rate for sinus tachycardia may vary according to age. In an infant, sinus tachycardia could be as high as 220/min.

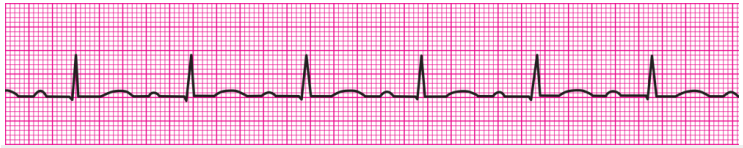


Figure 63. **Rhythm Strip 4: Sinus rhythm with first-degree heart block.**

Note that the PR interval in Figure 63 is prolonged (0.3 second). This is often a reflection of increased vagal tone and may be seen in healthy children. Less often, it can be a sign of intrinsic atrioventricular node disease, myocarditis, electrolyte disturbances (such as hyperkalemia), hypoxemia, drug toxicity (such as digoxin, β-blocker, or calcium channel blocker), or acute rheumatic fever.

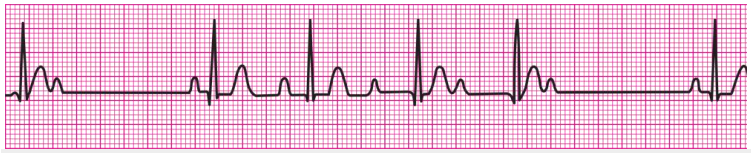


Figure 64. **Rhythm Strip 5: Second-degree heart block, Mobitz type I, or Wenckebach.**

Note that the PR interval in Figure 64 progressively prolongs until a P wave fails to conduct to the ventricle. Like first-degree heart block, this is often seen in healthy children, especially during sleep. It may also be a manifestation of drug toxicity, such as digoxin, β-blocker, or calcium channel blocker.

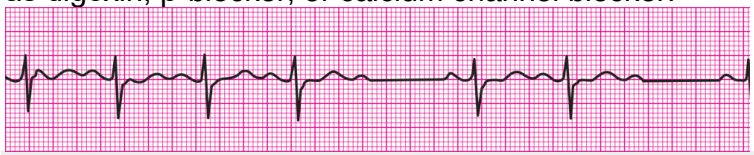


Figure 65. **Rhythm Strip 6: Second-degree heart block, Mobitz type II.**

Note that some, but not all, of the P waves in Figure 65 do not conduct to the ventricle. There is no progressive prolongation of the PR interval. This is a sign of intrinsic conduction system disease, typically related to cardiac surgery or myocardial inflammation or infarction.

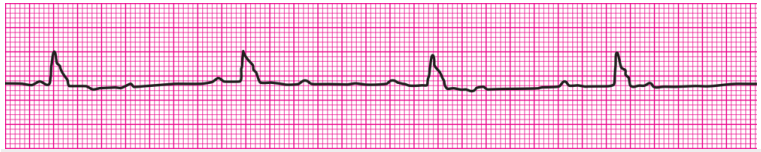


Figure 66. **Rhythm Strip 7: Third-degree (complete) heart block with ventricular escape rhythm.**

Note that none of the P waves in Figure 66 conduct to the ventricle. Often the QRS complex “marches” at a constant interval because of junctional or ventricular escape rhythm. There is no relation between P waves and QRS complexes. Occasionally

this is a result of severe hypoxemia and acidosis. This may also be a manifestation of damage to the atrioventricular node or extensive conduction system disease, such as that seen after cardiac surgery, or myocarditis, or with congenital complete heart block.

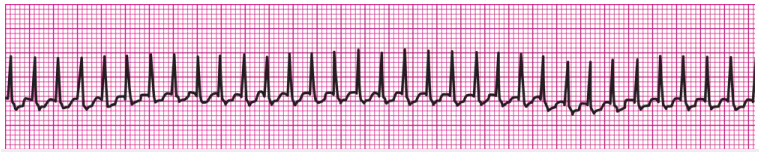


Figure 67. Rhythm Strip 8: Supraventricular tachycardia, 230/min.

Note that the QRS complexes in Figure 67 are narrow and regular, the rate is very fast (>200/min), and P waves are not obvious.

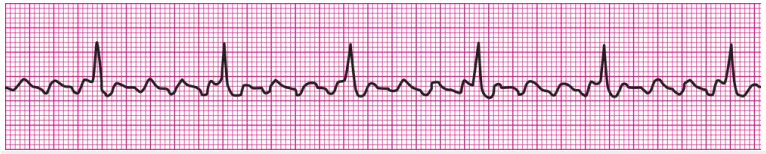


Figure 68. Rhythm Strip 9: Atrial flutter.

Note that there is a “sawtooth” pattern to the P waves in Figure 68, reflecting an extremely rapid atrial rate. Conduction of the P waves to the ventricle may be variable, resulting in an irregular QRS rate.

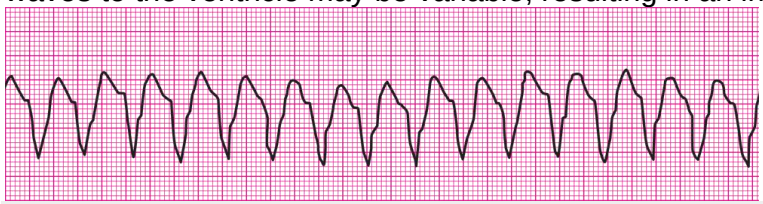


Figure 69. Rhythm Strip 10: Ventricular tachycardia, 150/min.

Note that the QRS complexes in Figure 69 are wide (greater than 0.09 second), regular, and fast. The QRS morphologies are all identical, characterizing it as monomorphic ventricular tachycardia. P waves are not identifiable.

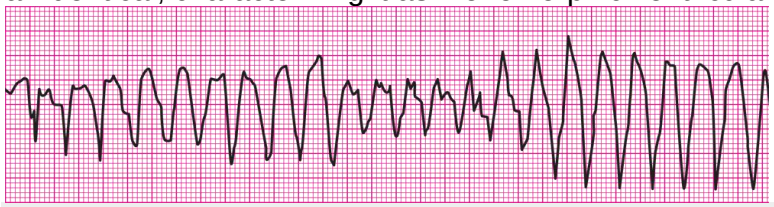


Figure 70. Rhythm Strip 11: Polymorphic ventricular tachycardia.

Note that the QRS complexes in Figure 70 are wide, irregular, and very fast (greater than 200/min). The QRS complexes vary in appearance, characterizing it as polymorphic. In fact, there is a phase change during this recording where the QRS complexes are initially positive and then become negative before returning to positive polarity. This is a torsades de pointes ("turning of points") type of polymorphic ventricular tachycardia, seen most often in states where the QT interval is prolonged during the baseline.

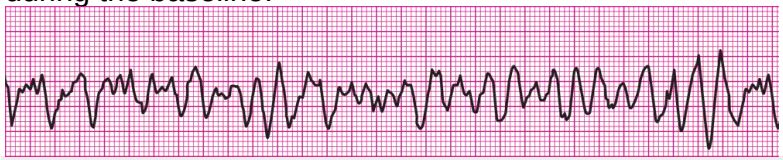


Figure 71. Rhythm Strip 12: Ventricular fibrillation.

Note that there are no definable QRS complexes in Figure 71, just irregular disorganized electrical activity.

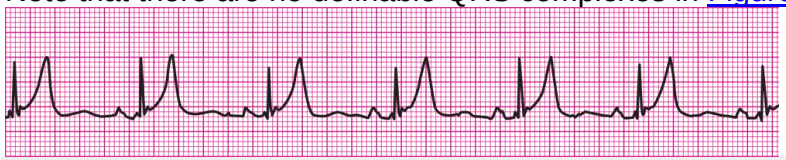


Figure 72. Rhythm Strip 13: Sinus rhythm with peaked T waves.

Note how the T waves in Figure 72 have increased amplitude and are even larger than the QRS complexes. This may be indicative of hyperkalemia.

Learning Station Competency Checklists and PALS Case Scenario Testing Checklists

The learning station competency checklists provide detailed descriptions of the critical performance steps that you will perform at each learning/skills station.

Additionally, the case scenario testing checklists provide the steps you will perform during each case scenario test. Your instructor will evaluate your skills on the basis of these descriptions.

If you perform a specific skill exactly as described in the critical performance criteria details, the instructor will check that specific skill as "passing." If you do not perform a specific skill exactly as it is described, the skill will not be checked off and you will require remediation in that skill.

Study these checklists so that you will be able to perform each skill correctly.

Airway Management
Skills Station Competency Checklist

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Verbalizes difference between high-flow and low-flow O_2 delivery systems • High flow: O_2 flow exceeds patient inspiratory flow, preventing entrainment of room air if system is tight-fitting; delivers nearly 1.00 (100%) FIO_2, eg, nonrebreathing mask with reservoir, high-flow nasal cannula • Low flow (≤10 L/min): patient inspiratory flow exceeds O_2 flow, allowing entrainment of room air; delivers 0.22 (22%) to 0.60 (60%) FIO_2, eg, standard nasal cannula, simple O_2 mask	
Verbalizes maximum nasal cannula flow rate for standard nasal cannula (4 L/min)	
Opens airway by using head tilt–chin lift maneuver while keeping mouth open (jaw thrust for trauma victim)	
Verbalizes different indications for OPA and NPA • OPA only for unconscious victim without a gag reflex • NPA for conscious or semiconscious victim	
Selects correctly sized airway by measuring • OPA from corner of mouth to angle of mandible	
Inserts OPA correctly	
Verbalizes assessment for adequate breathing after insertion of OPA	
Suctions with OPA in place; states suctioning not to exceed 10 seconds	
Selects correct mask size for ventilation	
Assembles bag-mask device, opens airway, and creates seal by using E-C clamp technique	
With bag-mask device, gives 1 breath every 2-3 seconds for 30 seconds; gives each breath in approximately 1 second; each breath should cause chest rise	
Endotracheal Intubation • States equipment needed for endotracheal (ET) tube intubation procedure • Demonstrates technique to confirm proper ET tube placement by physical exam and by using an exhaled CO_2 device • Secures ET tube • Suctions with ET tube in place	
The following steps are optional. They are demonstrated and evaluated only when the student's scope of practice involves ET intubation.	
Endotracheal Intubation • Prepares equipment for ET intubation • Inserts ET tube correctly, if within scope of practice	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

Rhythm Disturbances/ Electrical Therapy Skills Station Competency Checklist

American Heart Association. | American Academy of Pediatrics
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Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Applies 3 ECG leads correctly (or local equipment if >3 leads are used) • Negative (white) lead: to right shoulder • Positive (red) lead: to left lower ribs • Ground (black, green, brown) lead: to left shoulder	
Demonstrates correct operation of monitor • Turns monitor on • Adjusts device to manual mode (not AED mode) to display rhythm in standard limb leads (I, II, III) or paddles/electrode pads	
Verbalizes correct electrical therapy for appropriate core rhythms • Synchronized cardioversion for unstable SVT, VT with pulses • Defibrillation for pulseless VT, VF	
Selects correct paddle/electrode pad for infant or child; places paddles/electrode pads in correct position	
Demonstrates correct and safe synchronized cardioversion • Places device in synchronized mode • Selects appropriate energy (0.5-1 J/kg for initial shock) • Charges, clears, delivers current	
Demonstrates correct and safe manual defibrillation • Places device in unsynchronized mode • Selects energy (2-4 J/kg for initial shock) • Charges, clears, delivers current	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

Vascular Access Skills Station Competency Checklist

American Heart Association. | American Academy of Pediatrics
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Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Verbalizes indications for IO insertion	
Verbalizes sites for IO insertion (anterior tibia, distal femur, medial malleolus, anterior-superior iliac spine)	
Verbalizes contraindications for IO placement • Fracture in extremity • Previous insertion attempt in the same bone • Infection overlying bone • Osteogenesis imperfecta	
Inserts IO catheter safely	
Verbalizes how to confirm IO catheter is in correct position; verbalizes how to secure IO catheter	
Attaches IV line to IO catheter; demonstrates giving IO fluid bolus by using 3-way stopcock and syringe	
Shows how to determine correct drug doses by using a color-coded length-based tape or other resource	
The following is optional:	
Verbalizes correct procedure for establishing IV access	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Respiratory Case Scenario
Upper Airway Obstruction

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Directs assessment of airway, breathing, circulation, disability, and exposure, including vital signs	
Directs administration of 100% oxygen or supplemental oxygen as needed to support oxygenation	
Directs application of cardiac monitor and pulse oximetry	
Identifies signs and symptoms of upper airway obstruction	
Categorizes as respiratory distress or failure	
Directs administration of nebulized epinephrine and corticosteroid (for croup), or IM epinephrine and IV corticosteroid (for anaphylaxis)	
States indications for bag-mask ventilation and/or other airway or ventilation support	
If the student does not verbalize the above, prompt the student with the following question: "What are the indications for bag-mask ventilation and/or other airway or ventilation support?"	
Directs establishment of IV or IO access, if indicated	
Directs reassessment of patient in response to treatment	
Case Conclusion/Debriefing	
The following step is evaluated only if the student's scope of practice applies	
Describes how to estimate correct endotracheal tube size for this patient	
If the student does not verbalize the above, prompt the student with the following question: "How would you estimate the endotracheal tube size for this infant with upper airway obstruction?"	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Respiratory Case Scenario
Lower Airway Obstruction

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Directs assessment of airway, breathing, circulation, disability, and exposure, including vital signs	
Directs administration of 100% oxygen or supplemental oxygen as needed to support oxygenation	
Directs application of cardiac monitor and pulse oximetry	
Identifies signs and symptoms of lower airway obstruction	
Categorizes as respiratory distress or failure	
Directs administration of albuterol and corticosteroids (for asthma) or suctioning or possible additional laboratory studies (for bronchiolitis)	
States indications for bag-mask ventilation and/or other airway or ventilation support	
If the student does not verbalize the above, prompt the student with the following question: "What are the indications for bag-mask ventilation and/or other airway or ventilation support?"	
Directs establishment of IV or IO access, if appropriate	
Directs reassessment of patient in response to treatment	
Case Conclusion/Debriefing	
The following step is evaluated only if the student's scope of practice applies	
States indications for advanced airway management	
If the student does not verbalize the above, prompt the student with the following question: "What are the indications for endotracheal intubation?"	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Respiratory Case Scenario
Lung Tissue Disease

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Directs assessment of airway, breathing, circulation, disability, and exposure, including vital signs	
Directs administration of 100% oxygen (or supplemental oxygen as needed to support oxygenation) and evaluates response	
Identifies indications for bag-mask ventilation and/or additional airway or ventilation support	
Describes methods to verify that bag-mask ventilation is effective	
Directs application of cardiac monitor and pulse oximetry	
Identifies signs and symptoms of lung tissue disease	
Categorizes as respiratory distress or failure	
Directs establishment of IV or IO access	
Directs reassessment of patient in response to treatment	
Identifies need for involvement of advanced provider with expertise in pediatric intubation and mechanical ventilation	
Case Conclusion/Debriefing	
The following step is evaluated only if the student's scope of practice applies	
States indications for advanced airway management	
If the student does not verbalize the above, prompt the student with the following question: "What are the indications for advanced airway management?"	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results	Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Respiratory Case Scenario
Disordered Control of Breathing

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Directs assessment of airway, breathing, circulation, disability, and exposure, including vital signs	
Directs administration of 100% oxygen (or supplemental oxygen as needed to support oxygenation) and evaluates response	
Identifies indications for bag-mask ventilation and/or additional airway or ventilation support	
Describes methods to verify that bag-mask ventilation is effective	
Directs application of cardiac monitor and pulse oximetry	
Identifies signs of disordered control of breathing	
Categorizes as respiratory distress or failure	
Directs establishment of IV or IO access	
Directs reassessment of patient in response to treatment	
Identifies need for involvement of advanced provider with expertise in pediatric intubation and mechanical ventilation	
Case Conclusion/Debriefing	
The following step is evaluated only if the student's scope of practice applies	
States indications for advanced airway management	
If the student does not verbalize the above, prompt the student with the following question: "What are the indications for advanced airway management?"	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Shock Case Scenario
Hypovolemic Shock

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Directs assessment of airway, breathing, circulation, disability, and exposure, including vital signs	
Directs administration of 100% oxygen	
Directs application of cardiac monitor and pulse oximetry	
Identifies signs and symptoms of hypovolemic shock	
Categorizes as compensated or hypotensive shock	
Directs establishment of IV or IO access	
Directs rapid administration of a 20 mL/kg fluid bolus of isotonic crystalloid over 10 minutes if hypotensive and over 20 minutes if compensated; repeats as needed to treat signs of shock	
Reassesses patient during and after each fluid bolus; stops fluid bolus if signs of heart failure develop (ie, if vital signs fail to improve, respiratory distress worsens, hepatomegaly or rales/crackles develop)	
Directs reassessment of patient in response to each treatment	
Case Conclusion/Debriefing	
States therapeutic end points during shock management	
If the student does not verbalize the above, prompt the student with the following question: "What are the therapeutic end points during shock management?"	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Shock Case Scenario
Obstructive Shock

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Directs assessment of airway, breathing, circulation, disability, and exposure, including vital signs	
Directs application of cardiac monitor and pulse oximetry	
Verbalizes DOPE mnemonic for intubated patient who deteriorates	
If the student does not verbalize the above, prompt the student with the following questions: "What mnemonic is helpful to recall when the intubated patient deteriorates? What does this mnemonic mean?"	
Identifies signs and symptoms of obstructive shock	
States at least 2 causes of obstructive shock	
If the student does not state the above, prompt the student with the following statement: "Tell me at least 2 causes of obstructive shock."	
Categorizes as compensated or hypotensive shock	
Directs establishment of IV or IO access, if needed	
Directs rapid administration of a fluid bolus of 5-20 mL/kg over 10-20 minutes of isotonic crystalloid, if needed (ie, for cardiac tamponade, massive pulmonary embolus)	
Directs appropriate treatment for obstructive shock (needle decompression for tension pneumothorax; fluid bolus, and pericardiocentesis for cardiac tamponade; oxygen, ventilatory support, fluid bolus, and expert consultation for massive pulmonary embolus; prostaglandin infusion and expert consultation for neonate with ductal-dependent congenital heart disease and constriction/closure of the ductus arteriosus)	
Directs reassessment of patient in response to treatment	
Case Conclusion/Debriefing	
States therapeutic end points during shock management	
If the student does not verbalize the above, prompt the student with the following question: "What are the therapeutic end points during shock management?"	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results	Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Shock Case Scenario
Distributive Shock

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Directs assessment of airway, breathing, circulation, disability, and exposure, including vital signs	
Directs administration of 100% oxygen	
Directs application of cardiac monitor and pulse oximetry	
Identifies signs and symptoms of distributive (septic) shock	
Categorizes as compensated or hypotensive shock	
Directs establishment of IV or IO access	
Directs rapid administration of a 10-20 mL/kg fluid bolus of isotonic crystalloid for septic shock and 20 mL/kg fluid bolus of isotonic crystalloid over 5-20 minutes for anaphylactic shock; repeats as needed (with careful reassessment) to treat shock	
Reassesses patient during and after each fluid bolus; stops fluid bolus if signs of heart failure develop (ie, if vital signs fail to improve, respiratory distress worsens, hepatomegaly or rales/ crackles develop)	
Directs initiation of vasoactive drug therapy within first hour of care for fluid-refractory shock	
Directs reassessment of patient in response to treatment	
Directs early administration of antibiotics (within first hour after shock is identified)	
Case Conclusion/Debriefing	
States therapeutic end points during shock management	
If the student does not verbalize the above, prompt the student with the following question: "What are the therapeutic end points during shock management?"	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Shock Case Scenario
Cardiogenic Shock

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Directs assessment of airway, breathing, circulation, disability, and exposure, including vital signs	
Directs administration of 100% oxygen	
Directs application of cardiac monitor and pulse oximetry	
Identifies signs and symptoms of cardiogenic shock	
Categorizes as compensated or hypotensive shock	
Directs establishment of IV or IO access	
Directs slow administration of a 5 to 10 mL/kg fluid bolus of isotonic crystalloid over 10 to 20 minutes and reassesses patient during and after fluid bolus; stops fluid bolus if signs of heart failure worsen (ie, if vital signs fail to improve, respiratory distress worsens, hepatomegaly or rales/crackles develop)	
Directs reassessment of patient in response to treatment	
Recognizes the need to obtain expert consultation from pediatric cardiologist	
Identifies need for inotropic/vasoactive drugs during treatment of cardiogenic shock	
If the student does not indicate the above, prompt the student with the following question: "What are the indications for inotropic/vasoactive drugs during cardiogenic shock?"	
Case Conclusion/Debriefing	
States therapeutic end points during shock management	
If the student does not verbalize the above, prompt the student with the following question: "What are the therapeutic end points during shock management?"	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Cardiac Case Scenario
Supraventricular Tachycardia

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Directs assessment of airway, breathing, circulation, disability, and exposure, including vital signs	
Directs application of cardiac monitor and pulse oximetry	
Directs administration of supplemental oxygen	
Identifies narrow-complex tachycardia (ie, SVT with adequate perfusion) and verbalizes how to distinguish between ST and SVT	
If the student does not verbalize the above, prompt the student with the following question: "How do you distinguish between ST and SVT?"	
Directs performance of appropriate vagal maneuvers	
Directs establishment of IV or IO access	
Directs preparation and administration of appropriate doses (first and, if needed, second) of adenosine	
States the rationale for the strong recommendation for expert consultation before providing synchronized cardioversion if the stable child with SVT fails to respond to vagal maneuvers and adenosine	
Directs or describes appropriate indications for and safe delivery of attempted cardioversion at 0.5-1 J/kg (subsequent doses increased by 0.5-1 J/kg, not to exceed 2 J/kg)	
Performs reassessment of patient in response to treatment	
Case Conclusion/Debriefing	
Discusses indications and appropriate energy doses for synchronized cardioversion	
If the student does not verbalize the above, prompt the student with the following question: "What are the indications and appropriate energy doses for synchronized cardioversion?"	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results	Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Cardiac Case Scenario
Bradycardia

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Directs assessment of airway, breathing, circulation, disability, and exposure, including vital signs	
Identifies bradycardia associated with cardiopulmonary compromise/failure	
Directs initiation of bag-mask ventilation with 100% oxygen	
Directs application of cardiac monitor and pulse oximetry	
Reassesses heart rate and systemic perfusion after initiation of bag-mask ventilation	
Recognizes indications for high-quality CPR (chest compressions plus ventilation) in a bradycardic patient	
If the student does not indicate the above, prompt the student with the following question: "What are the indications for high-quality CPR in a bradycardic patient?"	
Directs establishment of IV or IO access	
Directs or discusses preparation for and appropriate administration and dose (0.01 mg/kg IV/IO [0.1 mg/kg concentration]) of epinephrine	
Performs reassessment of patient in response to treatment	
Case Conclusion/Debriefing	
Verbalizes consideration of 3 potential causes of bradycardia in infants and children	
If the student does not verbalize the above, prompt the student with the following statement: "Tell me 3 potential causes of bradycardia in infants and children."	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results	Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Cardiac Case Scenario
Asystole/PEA

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Identifies cardiac arrest	
Directs immediate initiation of high-quality CPR, and ensures performance of high-quality CPR at all times	
Directs placement of pads/leads and activation of monitor/defibrillator	
Identifies asystole or PEA	
Directs establishment of IO or IV access	
Directs preparation and administration of appropriate dose of epinephrine at appropriate intervals	
Directs checking rhythm approximately every 2 minutes while minimizing interruptions in chest compressions	
Case Conclusion/Debriefing	
Verbalizes at least 3 reversible causes of PEA or asystole	
If the student does not verbalize the above, prompt the student with the following statement: "Tell me at least 3 reversible causes of PEA or asystole."	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____

PALS Case Scenario Testing Checklist
Cardiac Case Scenario
VF/Pulseless VT

American Heart Association. | American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN®

Student Name _____ Date of Test _____

Critical Performance Steps	Check if done correctly
Team Leader	
Assigns team member roles	
Uses effective communication throughout	
Patient Management	
Identifies cardiac arrest	
Directs immediate initiation of high-quality CPR, and ensures performance of high-quality CPR at all times	
Directs placement of pads/leads and activation of monitor/defibrillator	
Identifies VF or pulseless VT cardiopulmonary arrest	
Directs safe performance of attempted defibrillation at 2 J/kg	
After delivery of every shock, directs immediate resumption of CPR, beginning with chest compressions	
Directs establishment of IO or IV access	
Directs preparation and administration of appropriate dose of epinephrine at appropriate intervals	
Directs safe delivery of second shock at 4 J/kg (subsequent doses 4-10 J/kg, not to exceed 10 J/kg or standard adult dose for that defibrillator)	
Directs preparation and administration of appropriate dose of antiarrhythmic (amiodarone or lidocaine) at appropriate time	
Case Conclusion/Debriefing	
Verbalizes possible need for additional doses of epinephrine and antiarrhythmic (amiodarone or lidocaine), and consideration of reversible causes of arrest (H's and T's)	
If the student does not verbalize the above, prompt the student with the following question: "If VF persists despite the therapies provided, what else should you administer or consider?"	

STOP TEST

Instructor Notes

- Place a check in the box next to each step the student completes successfully.
- If the student does not complete all steps successfully (as indicated by at least 1 blank check box), the student must receive remediation. Make a note here of which skills require remediation (refer to instructor manual for information about remediation).

Test Results	Circle **PASS** or **NR** to indicate pass or needs remediation:	**PASS**	**NR**

Instructor Initials _____ Instructor Number _____ Date _____