SHIRLEY A. JONES

ACLS, CPR, AND PALS

Clinical Pocket Guide

- 2010 CPR Protocols for Adults, Children, & Infants
- 2010 Advanced Cardiac Life Support Concepts & Algorithms
- 2010 Pediatric Advanced Life Support Concepts & Algorithms
- Cardiac Anatomy & Physiology
- Comprehensive ECG Interpretation
- Critical Care Medications with Adult & Pediatric Dosages
- Megacode Teaching Scenarios for ACLS & PALS

Contacts • Phone/E-Mail

Name	
Ph:	e-mail:
Name	
Ph:	e-mail:
Name	
Ph:	e-mail:
Name	
Ph:	e-mail:
Name	
Ph:	e-mail:
Name	
Ph:	e-mail:
Name	
Ph:	e-mail:
Name	
Ph:	e-mail:
Name	
Ph:	e-mail:
Name	
Ph:	e-mail:
Name	
Ph:	e-mail:
Name	
Ph:	e-mail:

ACLS, CPR, and PALS

Clinical Pocket Guide

Shirley A. Jones, MS Ed, MHA, EMT-P, RN

Purchase additional copies of this book at your health science bookstore or directly from F.A. Davis by shopping online at www.fadavis.com or by calling 800-323-3555 (US) or 800-665-1148 (CAN)

F. A. Davis Company • Philadelphia

F. A. Davis Company
1915 Arch Street
Philadelphia, PA 19103
www.fadavis.com

Printed in China by Imago

Last digit indicates print number: 10 9 8 7 6 5 4 3 2 1

Publisher, Nursing: Lisa. B. Houck
Director of Content Development: Darlene D. Pedersen, MSN, APRN, BC
Content Project Manager: Victoria White
Design & Illustration Manager: Carolyn O'Brien
Reviewers: Dianna Bottoms, MS, RN, CCRN, CNE; Sue A. Bradbury, RN, MSN; Nita Jane Carrington, EdD, MSN, ANP, RN; Dr. Hazel Downing, RN, MN, EdD; Kara Jones, MSN, RN CPR instructor; Kathleen L. Slyh, RN, MSN; Beryl Stetson, RNBC, MSN, CNE, LCCE, CLC; Charlene Whiddon, MSN, RN.

Contributor: Carmen J. Petrin, MS, FNP-BC

Place 2⅞ x 2⅞ **Sticky Notes** here
For a convenient and refillable pad

√ HIPAA Compliant
√ OSHA Compliant

Waterproof and Reusable
Wipe-Free Pages

Write directly onto any page of *ACLS, CPR, and PALS: Clinical Pocket Guide* with a ballpoint pen. Wipe old entries off with an alcohol pad and reuse.

1

Tab 1: ECG

The body acts as a giant conductor of electrical current. Electrical activity that originates in the heart can be detected on the body's surface through an electrocardiogram (ECG). Electrodes are applied to the skin to measure voltage changes in the cells between the electrodes. These voltage changes are amplified and visually displayed on an oscilloscope and graph paper.

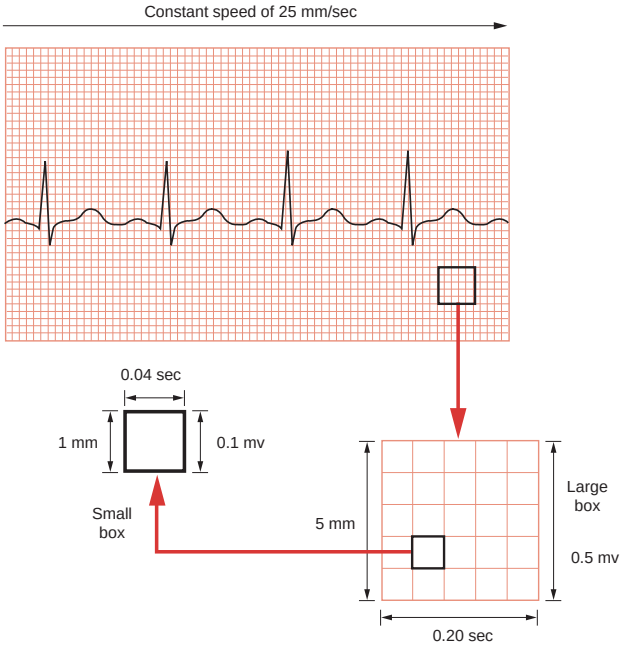
- An ECG is a series of waves and deflections recording the heart's electrical activity from a certain "view."
- Many views, each called a lead, monitor voltage changes between electrodes placed in different positions on the body.
- Leads I, II, and III are bipolar leads consisting of one positive and one negative electrode, with a third (ground) electrode to minimize electrical activity from other sources.
- Leads aVR, aVL, and aVF are unipolar leads consisting of a single positive electrode and a reference point (with zero electrical potential) that lies in the center of the heart's electrical field.
- Leads V_1–V_6 are unipolar leads consisting of a single positive electrode with a negative reference point found at the electrical center of the heart.
- An ECG tracing looks different in each lead because the recorded angle of electrical activity changes with each lead. Different angles allow a more accurate perspective than a single one would.
- The ECG machine can be adjusted to make any skin electrode positive or negative. The polarity depends on which lead the machine is recording.
- A cable attached to the patient is divided into several different-colored wires: three, four, or five for monitoring purposes, or ten for a 12-lead ECG.
- Incorrect placement of electrodes may turn a normal ECG tracing into an abnormal one.

♥ **Clinical Tip:** To obtain a 12-lead ECG, four wires are attached to each limb, and six wires are attached at different locations on the chest. The total of ten wires provides twelve views (12 leads).

♥ **Clinical Tip:** It is important to keep in mind that the ECG shows only electrical activity; it tells us nothing about how well the heart is working mechanically.

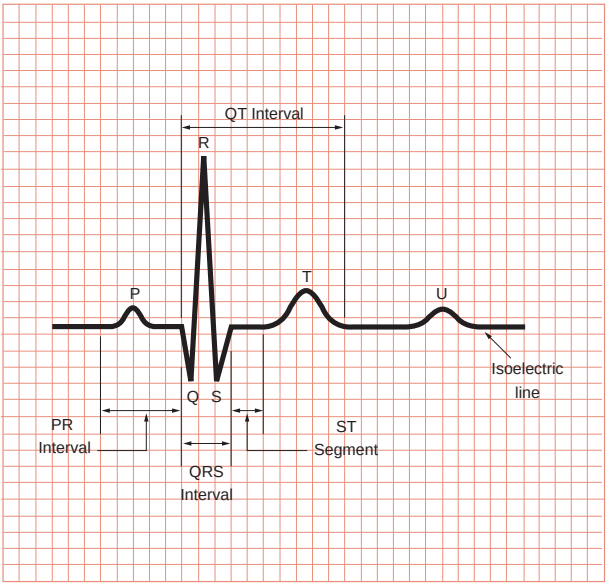
♥ **Clinical Tip:** Patients should be treated according to their symptoms, not merely their ECG.

Recording of the ECG



3

Components of an ECG Tracing



Electrical Activity

Term	Definition
Wave	A deflection, either positive or negative, away from the baseline (isoelectric line) of the ECG tracing
Complex	Several waves
Segment	A straight line between waves or complexes
Interval	A segment and a wave

♥ **Clinical Tip:** Between waves and cycles, the ECG records a baseline (isoelectric line), which indicates the absence of electrical activity.

Electrical Components

Deflection	Description
P Wave	First wave seen Small, rounded upright (positive) wave indicating atrial depolarization (and contraction)
PR Interval	Distance between beginning of P wave and beginning of QRS complex Measures time during which a depolarization wave travels from the atria to the ventricles
QRS Complex	Three deflections following the P wave Indicates ventricular depolarization (and contraction) Q Wave: First negative deflection R Wave: First positive deflection S Wave: First negative deflection after R wave
ST Segment	Distance between S wave and beginning of T wave Measures time between ventricular depolarization and beginning of repolarization
T Wave	Rounded upright (positive) wave following QRS Represents ventricular repolarization
QT Interval	Distance between beginning of QRS complex to end of T wave Represents total ventricular activity
U Wave	Small, rounded upright wave following T wave Most easily seen with a slow HR Represents repolarization of Purkinje fibers

ECG Interpretation

Analyzing a Rhythm

Component	Characteristic
Rate	The bpm is commonly the ventricular rate. If atrial and ventricular rates differ, as in a 3rd-degree block, measure both rates. Normal: 60–100 bpm Slow (bradycardia): <60 bpm Fast (tachycardia): >100 bpm
Regularity	Measure R-R intervals and P-P intervals. Regular: Intervals consistent Regularly irregular: Repeating pattern Irregular: No pattern
P Waves	If present: Same in size, shape, position? Does each QRS have a P wave? Normal: Upright (positive) and uniform Inverted: Negative Notched: P prime wave (P') None: Junctional, ventricular, or asystole
PR Interval	Constant: Intervals are the same Variable: Intervals differ Normal: 0.12–0.20 sec and constant
QRS Interval	Normal: 0.06–0.10 sec Wide: >0.10 sec None: Asystole
QT Interval	Beginning of QRS complex to end of T wave Varies with HR Normal: Less than half the RR interval
Dropped beats	Occur in AV blocks Occur in sinus arrest
Pause	Compensatory: Complete pause following a premature ventricular contraction (PVC) Noncompensatory: Incomplete pause following a PVC

Continued

Analyzing a Rhythm—cont'd

Component	Characteristic
QRS Complex grouping	Bigeminy: Repeating pattern of normal complex followed by a premature complex Trigeminy: Repeating pattern of 2 normal complexes followed by a premature complex Quadrigeminy: Repeating pattern of 3 normal complexes followed by a premature complex Couplet: 2 consecutive premature complexes Triplet: 3 consecutive premature complexes

Measuring the QT Interval

Prolonged QT: Caused by medications (amiodarone, droperidol, haldol, erythromycin, methadone, procainamide, tricyclics) or conditions (CHF, MI, hypocalcemia, hypomagnesemia, myocarditis)
Shortened QT: Caused by medications (digoxin, phenothiazines) or conditions (hypercalcemia, hyperkalemia)

Classification of Arrhythmias

Heart Rate	Classification
Slow	Bradyarrhythmia
Fast	Tachyarrhythmia
Absent	Pulseless arrest

Normal Heart Rate (bpm)

Age	Awake Rate	Mean	Sleeping Rate
Newborn to 3 mo	85–205	140	80–160
3 mo to 2 yr	100–190	130	75–160
2 to 10 yr	60–140	80	60–90
>10 yr	60–100	75	50–90

7

The 12-Lead ECG

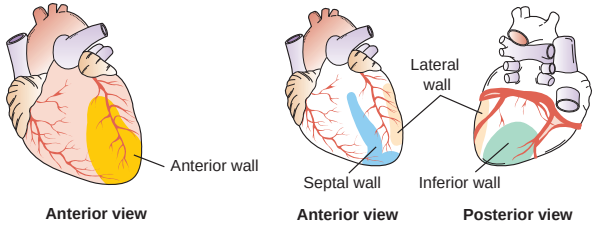
A standard 12-lead ECG provides views of the heart from 12 different angles. This diagnostic test helps to identify pathological conditions, especially bundle branch blocks and T wave changes associated with ischemia, injury, and infarction. The 12-lead ECG also uses ST segment analysis to pinpoint the specific location of an MI.

The 12-lead ECG is the type most commonly used in clinical settings. The following list highlights some of its important aspects:

- The 12-lead ECG consists of the six limb leads—I, II, III, aVR, aVL, and aVF—and the six chest leads—V1, V2, V3, V4, V5, and V6.
- The limb leads record electrical activity in the heart's frontal plane. This view shows the middle of the heart from top to bottom. Electrical activity is recorded from the anterior-to-posterior axis.
- The chest leads record electrical activity in the heart's horizontal plane. This transverse view shows the middle of the heart from left to right, dividing it into upper and lower portions. Electrical activity is recorded from either a superior or an inferior approach.
- Measurements are central to 12-lead ECG analysis. The height and depth of waves can offer important diagnostic information in certain conditions, including MI and ventricular hypertrophy.
- The direction of ventricular depolarization is an important factor in determining the axis of the heart.
- In an MI, multiple leads are necessary to recognize its presence and determine its location. If large areas of the heart are affected, the patient can develop cardiogenic shock and fatal arrhythmias.
- ECG signs of an MI are best seen in the reciprocal, or reflecting, leads—those facing the affected surface of the heart. Reciprocal leads are in the same plane but opposite the area of infarction; they show a "mirror image" of the electrical complex.
- Prehospital EMS systems may use 12-lead ECGs to discover signs of acute MI, such as ST segment elevation, in preparation for in-hospital administration of thrombolytic drugs.
- After a 12-lead ECG is performed, a 15-lead, or right-sided, ECG may be used for an even more comprehensive view if the right ventricle or the posterior portion of the heart appears to be affected.

Ischemia, Injury, and Infarction in Relation to the Heart

Ischemia, injury, and infarction of cardiac tissue are the three stages resulting from complete blockage in a coronary artery. The location of the MI is critical in determining the most appropriate treatment and predicting probable complications. Each coronary artery delivers blood to specific areas of the heart. Blockages at different sites can damage various parts of the heart. Characteristic ECG changes occur in different leads with each type of MI and can be correlated with the blockages.



Location of MI by ECG Leads

I lateral	aVR	V_1 septal	V_4 anterior
II inferior	aVL lateral	V_2 septal	V_5 lateral
III inferior	aVF inferior	V_3 anterior	V_6 lateral

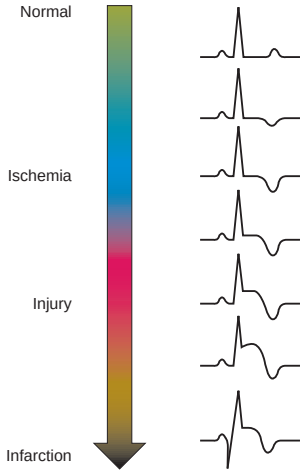
Clinical Tip: Lead aVR may not show any change in an MI.

Clinical Tip: An MI may not be limited to just one region of the heart. For example, if there are changes in leads V3 and V4 (anterior) and leads I, aVL, V5, and V6 (lateral), the MI is called an anterolateral infarction.

Progression of an Acute Myocardial Infarction

An acute MI is a continuum that extends from the normal state to a full infarction:

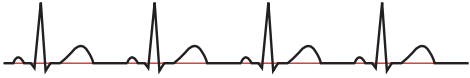
- **Ischemia**—Lack of oxygen to the cardiac tissue, represented by ST segment depression, T wave inversion, or both
- **Injury**—Arterial occlusion with ischemia, represented by ST segment elevation
- **Infarction**—Death of tissue, represented by a pathological Q wave



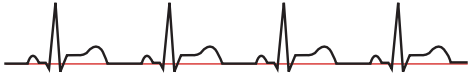
Clinical Tip: After the acute MI has ended, the ST segment returns to baseline, and the T wave becomes upright, but the Q wave remains abnormal because of scar formation.

ST Segment Elevation and Depression

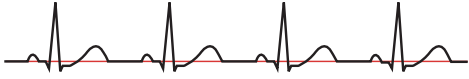
- A normal ST segment represents early ventricular repolarization.
- Displacement of the ST segment can be caused by the following various conditions:



ST segment is at baseline.



ST segment is elevated.



ST segment is depressed.

Primary Causes of ST Segment Elevation

- ST segment elevation exceeding 1 mm in the limb leads and 2 mm in the chest leads indicates an evolving acute MI or an **ST-elevation MI (STEMI)** until there is proof to the contrary. In a STEMI there is usually complete occlusion of an epicardial coronary artery. Other causes of ST segment elevation are:
  - Pericarditis, ventricular aneurysm
  - Pulmonary embolism, intracranial hemorrhage

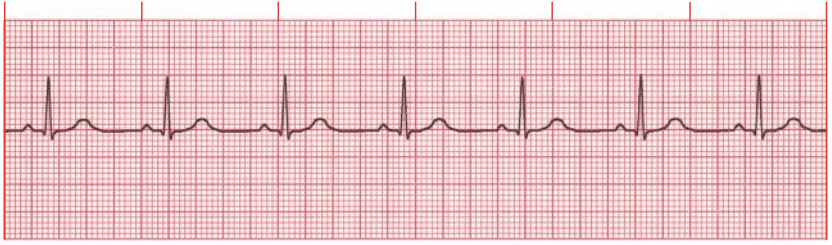
Primary Causes of ST Segment Depression

- Myocardial ischemia, or **non–ST-elevation MI (NSTEMI)**, is caused by a partial obstruction of an epicardial coronary artery.
  - Intraventricular conduction defects, left ventricular hypertrophy
  - Medication (e.g., digitalis)

Sinoatrial (SA) Node Arrhythmias

- Upright P waves all look similar. **Note: All ECG strips in Tab 1 were recorded in lead II.**
- PR intervals and QRS complexes are of normal duration.

Normal Sinus Rhythm (NSR)



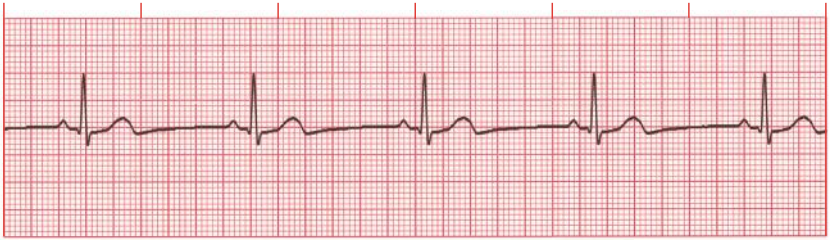
Rate: Normal (60–100 bpm)
Rhythm: Regular
P Waves: Normal (upright and uniform)
PR Interval: Normal (0.12–0.20 sec)
QRS: Normal (0.06–0.10 sec)

♥ **Clinical Tip:** A normal ECG does not exclude heart disease.

♥ **Clinical Tip:** This rhythm is generated by the sinus node, and its rate is within normal limits (60–80 bpm).

Sinus Bradycardia

- The sinoatrial node (sinus node, SA node) discharges more slowly than in NSR.

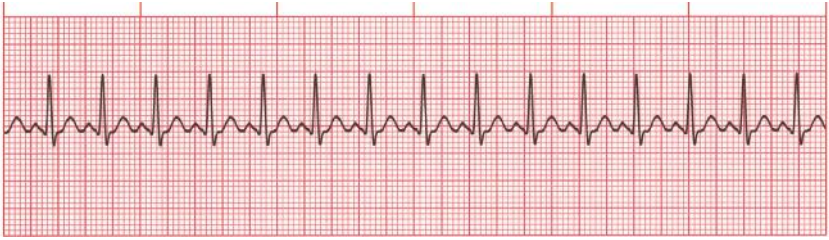


Rate: Slow (<60 bpm)
Rhythm: Regular
P Waves: Normal (upright and uniform)
PR Interval: Normal (0.12–0.20 sec)
QRS: Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Sinus bradycardia is normal in athletes and during sleep. In acute MI, the slow rate may be protective and beneficial or may compromise cardiac output (CO). Certain medications, such as beta blockers, may also cause sinus bradycardia. Sinus bradycardia may also be caused by vagal stimulation, such as gagging, straining, and endotracheal (ET) suctioning. Other causes are chronic ischemic heart disease, sick sinus syndrome, hypothyroidism, and increased intracranial pressure.

Sinus Tachycardia

- The sinus node discharges more frequently than in NSR.



13

Rate: Fast (>100 bpm)
Rhythm: Regular
P Waves: Normal (upright and uniform)
PR Interval: Normal (0.12–0.20 sec)
QRS: Normal (0.06–0.10 sec)

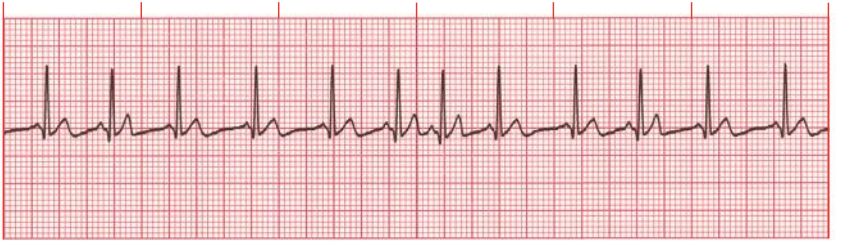
♥ **Clinical Tip:** Sinus tachycardia may be caused by conditions such as fear, pain, exercise, anxiety, or fever. More significant pathological causes include hypoxemia, hypovolemia/dehydration, cardiac failure or recent MI, CHF, beta blocker withdrawal, hyperthyroidism, or withdrawal from nicotine, caffeine, or alcohol.

Atrial Arrhythmias

- P waves differ in appearance from sinus P waves.
- QRS complexes are of normal duration if no ventricular conduction disturbances are present.

Multifocal Atrial Tachycardia (MAT)

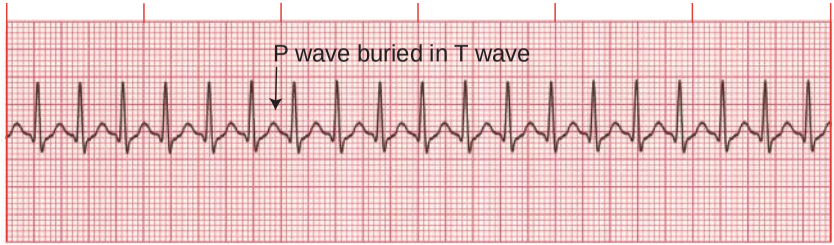
- This form of wandering atrial pacemaker (WAP) is associated with a ventricular response >100 bpm.
- MAT may be confused with atrial fibrillation (A-fib); however, MAT has a visible P wave.



Rate: Fast (>100 bpm)
Rhythm: Irregular
P Wave: At least three different forms, determined by the focus in the atria
PR Interval: Variable; determined by focus
QRS: Normal (0.06–0.10 sec)

Supraventricular Tachycardia (SVT)

- This arrhythmia has such a fast rate that the P waves may not be seen.



15

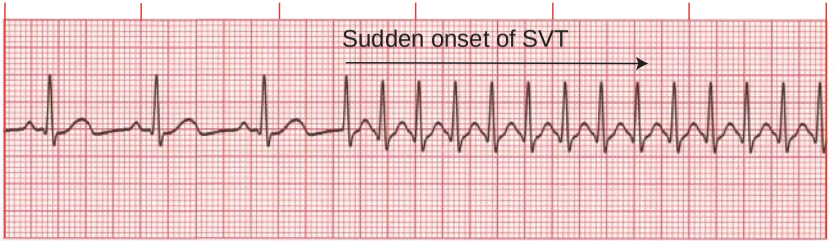
Rate: 150–250 bpm
Rhythm: Regular
P Waves: Frequently buried in preceding T waves and difficult to see
PR Interval: Usually not possible to measure
QRS: Normal (0.06–0.10 sec) but may be wide if abnormally conducted through ventricles

♥ **Clinical Tip:** SVT may be related to caffeine intake, nicotine, stress, or anxiety in healthy adults.

♥ **Clinical Tip:** Some patients may experience angina, hypotension, lightheadedness, palpitations, and intense anxiety.

Paroxysmal Supraventricular Tachycardia (PSVT)

- PSVT is a rapid rhythm that starts and stops suddenly.
- For accurate interpretation, the beginning or end of the PSVT must be seen.
- PSVT is sometimes called paroxysmal atrial tachycardia (PAT).

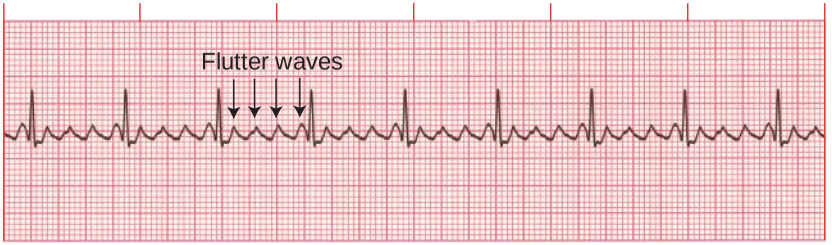


Rate: 150–250 bpm
Rhythm: Irregular
P Waves: Frequently buried in preceding T waves and difficult to see
PR Interval: Usually not possible to measure
QRS: Normal (0.06–0.10 sec) but may be wide if abnormally conducted through ventricles

Clinical Tip: The patient may feel palpitations, dizziness, lightheadedness, or anxiety.

Atrial Flutter (A-flutter)

- AV node conducts impulses to the ventricles at a ratio of 2:1, 3:1, 4:1, or greater (rarely 1:1).
- The degree of AV block may be consistent or variable.



17

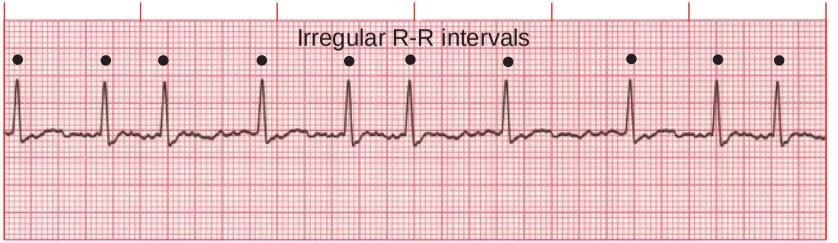
Rate: Atrial: 250–350 bpm; ventricular: variable
Rhythm: Atrial: regular; ventricular: variable
P Waves: Flutter waves have a saw-toothed appearance; some may be buried in the QRS and not visible
PR Interval: Variable
QRS: Usually normal (0.06–0.10 sec), but may appear widened if flutter waves are buried in QRS

Clinical Tip: A-flutter may be the first indication of cardiac disease.

Clinical Tip: Signs and symptoms depend on ventricular response rate.

Atrial Fibrillation (A-fib)

- Rapid, erratic electrical discharge comes from multiple atrial ectopic foci.
- No organized atrial depolarization is detectable.



Rate: Atrial: ⊠350 bpm; ventricular: variable
Rhythm: Irregular
P Waves: No true P waves; chaotic atrial activity
PR Interval: None
QRS: Normal (0.06–0.10 sec)

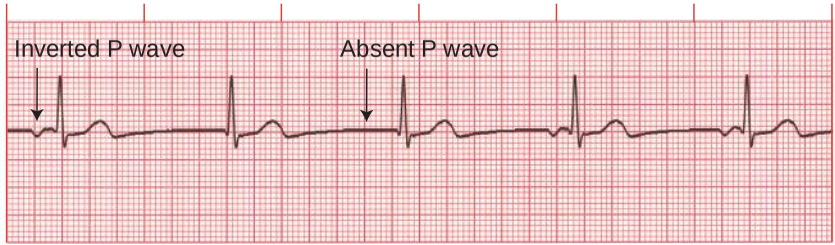
Clinical Tip: A-fib is usually a chronic arrhythmia associated with underlying heart disease.

Clinical Tip: Signs and symptoms depend on ventricular response rate.

Junctional Arrhythmias

- The atria and sinus node do not perform their normal pacemaking functions.
- A junctional escape rhythm begins.

Junctional Rhythm



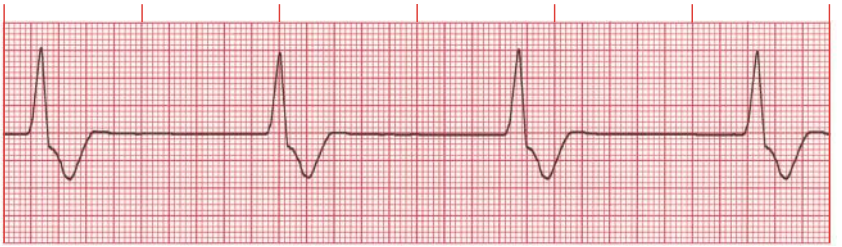
Rate: 40–60 bpm
Rhythm: Regular
P Waves: Absent, inverted, buried, or retrograde
PR Interval: None, short, or retrograde
QRS: Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Sinus node disease that causes inappropriate sinus node slowing may exacerbate this rhythm. Young, healthy adults, especially those with increased vagal tone during sleep, often have periods of junctional rhythm that is completely benign, not requiring intervention.

Ventricular Arrhythmias

- In all ventricular rhythms, the QRS complex is >0.10 sec. P Waves are absent or, if visible, have no consistent relationship to the QRS complex.

Idioventricular Rhythm

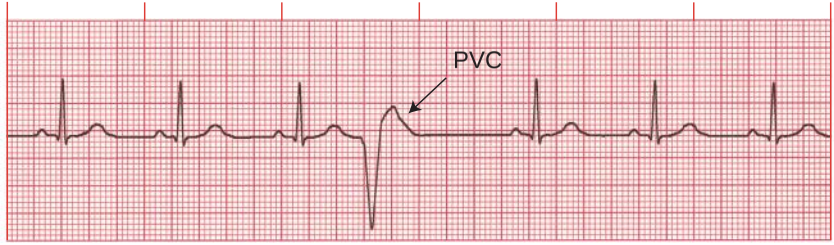


Rate: 20–40 bpm
Rhythm: Regular
P Waves: None
PR Interval: None
QRS: Wide (>0.10 sec), bizarre appearance

Clinical Tip: Diminished cardiac output is expected because of the slow heart rate. An idioventricular rhythm may be called an agonal rhythm when the heart rate drops below 20 bpm. An agonal rhythm is generally terminal and is usually the last rhythm before asystole.

Premature Ventricular Contraction (PVC)

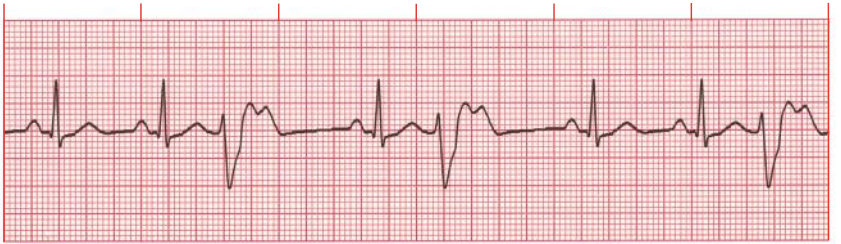
- PVCs result from an irritable ventricular focus.
- PVCs may be uniform (the same form) or multiform (different forms).
- Usually a PVC is followed by a full compensatory pause because the sinus node timing is not interrupted. In contrast, a PVC may be followed by a noncompensatory pause if the PVC enters the sinus node and resets its timing, enabling the following sinus P wave to appear earlier than expected.



Rate: Depends on rate of underlying rhythm
Rhythm: Irregular whenever a PVC occurs
P Waves: None associated with the PVC
PR Interval: None associated with the PVC
QRS: Wide (>0.10 sec), bizarre appearance

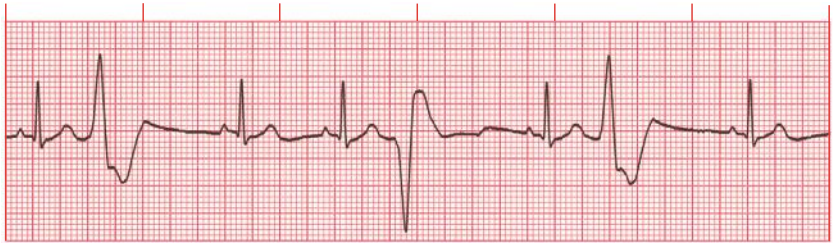
♥ **Clinical Tip:** Patients may sense PVCs as skipped beats. Because the ventricles are only partially filled, the PVC frequently does not generate a pulse.

Premature Ventricular Contraction: Uniform (same form)



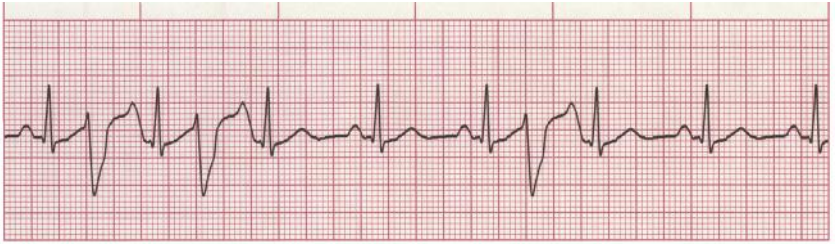
23

Premature Ventricular Contraction: Multiform (different forms)



Premature Ventricular Contraction: R-on-T Phenomenon

- The PVCs occur so early that they fall on the T wave of the preceding beat.
- These PVCs occur during the refractory period of the ventricles, a vulnerable period because the cardiac cells have not fully repolarized.

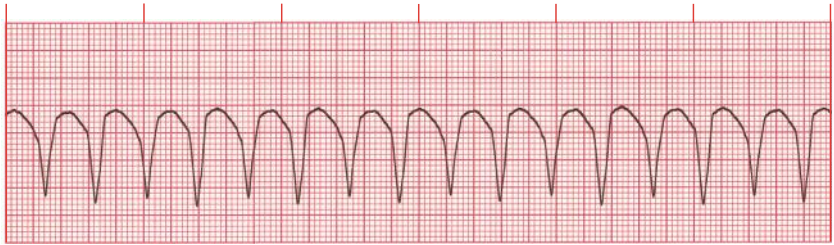


Rate: Depends on rate of underlying rhythm
Rhythm: Irregular whenever a PVC occurs
P Waves: None associated with the PVC
PR Interval: None associated with the PVC
QRS: Wide (>0.10 sec), bizarre appearance

Clinical Tip: In acute ischemia, R-on-T phenomenon may be especially dangerous because the ventricles may be more vulnerable to ventricular tachycardia (VT), ventricular fibrillation (VF), or torsade de pointes.

Ventricular Tachycardia (VT): Monomorphic

- In monomorphic VT, QRS complexes have the same shape and amplitude.



25

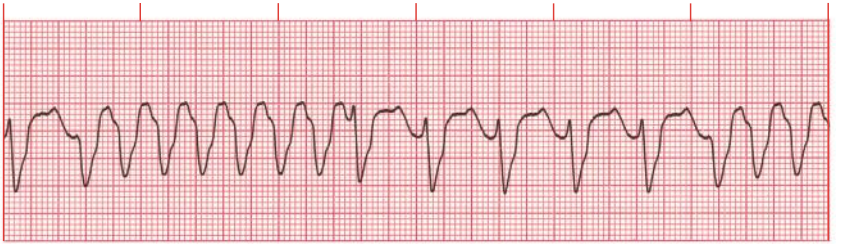
Rate: 100–250 bpm
Rhythm: Regular
P Waves: None or not associated with the QRS
PR Interval: None
QRS: Wide (>0.10 sec), bizarre appearance

Clinical Tip: It is important to confirm the presence or absence of pulses because monomorphic VT may be perfusing or nonperfusing.

Clinical Tip: Monomorphic VT will probably deteriorate into VF or unstable VT if sustained and not treated.

Ventricular Tachycardia (VT): Polymorphic

- In polymorphic VT, QRS complexes vary in shape and amplitude.
- The QT interval is normal or long.



Rate: 100–250 bpm
Rhythm: Regular or irregular
P Waves: None or not associated with the QRS
PR Interval: None
QRS: Wide (>0.10 sec), bizarre appearance

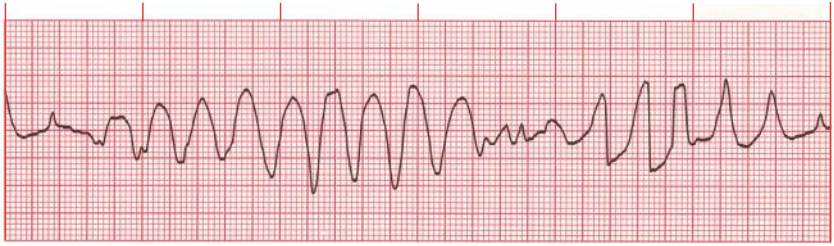
Clinical Tip: It is important to determine whether pulses are present because polymorphic VT may be perfusing or nonperfusing.

Clinical Tip: Consider electrolyte abnormalities as a possible cause.

Torsade de Pointes

- The QRS reverses polarity, and the strip shows a spindle effect.
- This rhythm is an unusual variant of polymorphic VT with long QT intervals.
- In French *torsade de pointes* means "twisting of points."

27



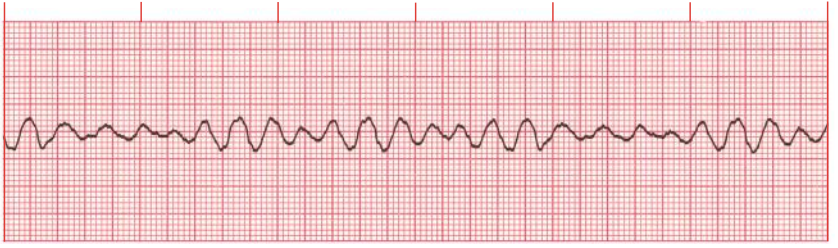
Rate: 200–250 bpm
Rhythm: Irregular
P Waves: None
PR Interval: None
QRS: Wide (>0.10 sec), bizarre appearance

♥ **Clinical Tip:** Torsade de pointes may deteriorate to VF or asystole.

♥ **Clinical Tip:** Frequent causes are drugs that prolong the QT interval, electrolyte abnormalities such as hypomagnesemia, and the R-on-T phenomenon.

Ventricular Fibrillation (VF)

- Chaotic electrical activity occurs with no ventricular depolarization or contraction.
- The amplitude and frequency of the fibrillatory activity can define the type of fibrillation as coarse, medium, or fine. Small baseline undulations are considered fine; large ones are coarse.

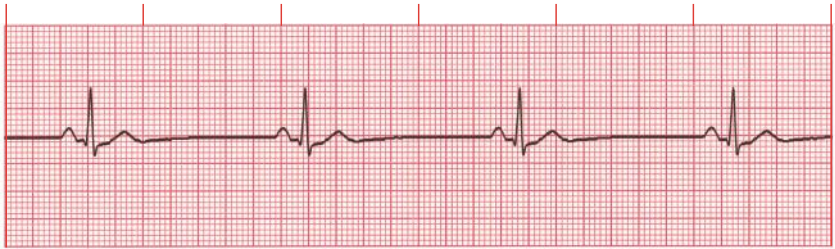


Rate: Indeterminate
Rhythm: Chaotic
P Waves: None
PR Interval: None
QRS: None

Clinical Tip: There is no pulse or cardiac output. Rapid intervention is critical. The longer the delay, the less the chance of conversion.

Pulseless Electrical Activity (PEA)

- The monitor shows an identifiable electrical rhythm, but no pulse is detected.
- The rhythm may be sinus, atrial, junctional, or ventricular.
- PEA is also called electromechanical dissociation (EMD).



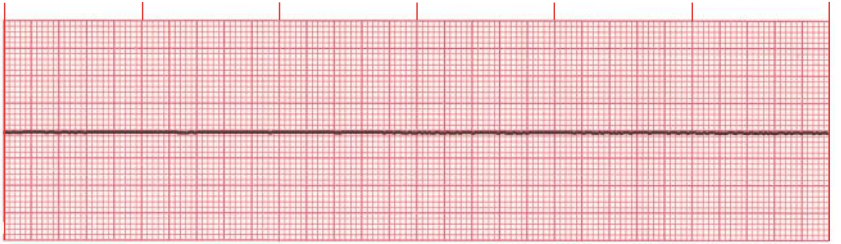
29

Rate: Reflects underlying rhythm
Rhythm: Reflects underlying rhythm
P Waves: Reflects underlying rhythm
PR Interval: Reflects underlying rhythm
QRS: Reflects underlying rhythm

♥ **Clinical Tip:** Potential causes of PEA are trauma, tension pneumothorax, thrombosis (pulmonary or coronary), cardiac tamponade, toxins, hypokalemia or hyperkalemia, hypovolemia, hypoxia, hypoglycemia, hypothermia, and hydrogen ion (acidosis).

Asystole

- Electrical activity in the ventricles is completely absent.



Rate: None
Rhythm: None
P Waves: None
PR Interval: None
QRS: None

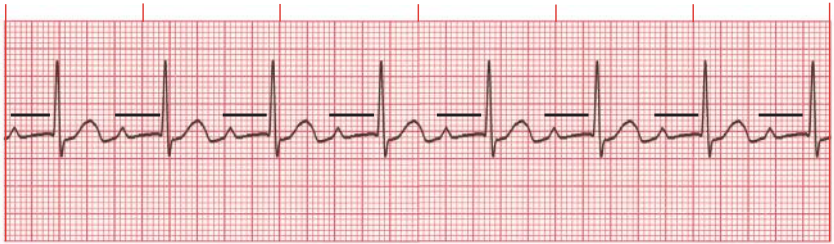
Clinical Tip: Rule out other causes such as loose leads, no power, or insufficient signal gain.

Clinical Tip: Seek to identify the underlying cause as in PEA. Also, search to identify VF.

Atrioventricular (AV) Blocks

- AV blocks are divided into three categories: first, second, and third degree.

First-Degree AV Block



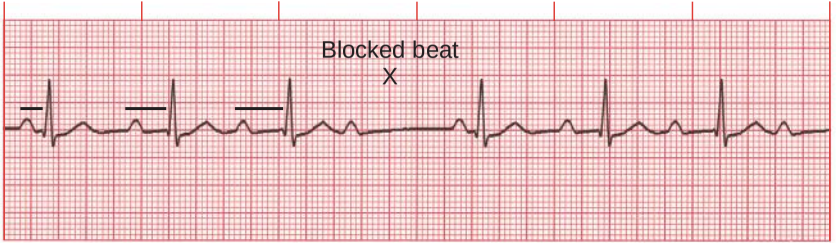
Rate: Depends on rate of underlying rhythm
Rhythm: Regular
P Waves: Normal (upright and uniform)
PR Interval: Prolonged (>0.20 sec)
QRS: Normal (0.06–0.10 sec)

♥ **Clinical Tip:** Usually a first-degree AV block is benign, but if associated with an acute MI, it may lead to further AV defects.

♥ **Clinical Tip:** Often AV block is caused by medications that prolong AV conduction; these include digoxin, calcium channel blockers, and beta blockers.

Second-Degree AV Block—Type I (Mobitz I or Wenckebach)

- PR intervals become progressively longer until one P wave is totally blocked and produces no QRS complex. After a pause, during which the AV node recovers, this cycle is repeated.



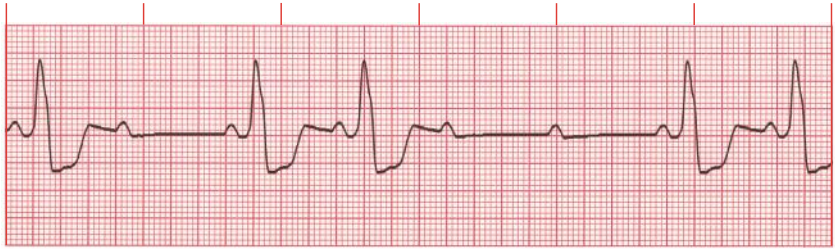
Rate: Depends on rate of underlying rhythm
Rhythm: Atrial: regular; ventricular: irregular
P Waves: Normal (upright and uniform), more P waves than QRS complexes
PR Interval: Progressively longer until one P wave is blocked and a QRS is dropped
QRS: Normal (0.06–0.10 sec)

♥ **Clinical Tip:** This rhythm may be caused by medication such as beta blockers, digoxin, and calcium channel blockers. Ischemia involving the right coronary artery is another cause.

Second-Degree AV Block—Type II (Mobitz II)

- Conduction ratio (P waves to QRS complexes) is commonly 2:1, 3:1, or 4:1, or variable.
- QRS complexes are usually wide because this block usually involves both bundle branches.

33

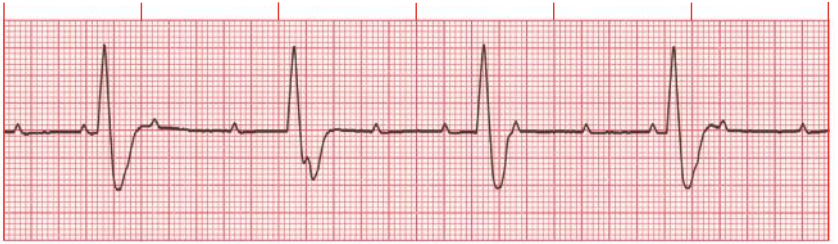


Rate: Atrial: usually 60–100 bpm; ventricular: slower than atrial rate
Rhythm: Atrial: regular; ventricular: regular or irregular
P Waves: Normal (upright and uniform); more P waves than QRS complexes
PR Interval: Normal or prolonged but constant
QRS: May be normal, but usually wide (>0.10 sec) if the bundle branches are involved

♥ **Clinical Tip:** Resulting bradycardia can compromise cardiac output and lead to complete AV block. This rhythm often occurs with cardiac ischemia or an MI.

Third-Degree AV Block

- Conduction between the atria and ventricles is totally absent because of complete electrical block at or below the AV node. This is known as AV dissociation.
- "Complete heart block" is another name for this rhythm.

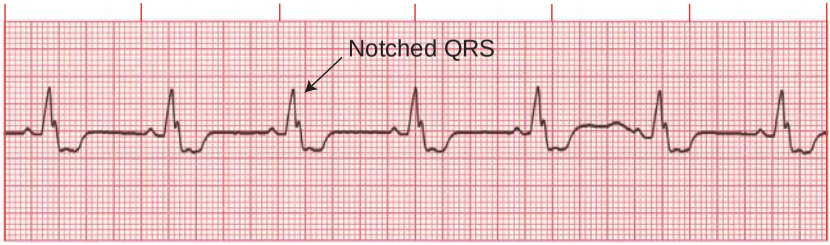


Rate: Atrial: 60–100 bpm; ventricular: 40–60 bpm if escape focus is junctional, <40 bpm if escape focus is ventricular
Rhythm: Usually regular, but atria and ventricles act independently
P Waves: Normal (upright and uniform); may be superimposed on QRS complexes or T waves
PR Interval: Varies greatly
QRS: Normal if ventricles are activated by junctional escape focus; wide if escape focus is ventricular

Clinical Tip: Third-degree AV block may be associated with ischemia involving the left coronary arteries.

Bundle Branch Block (BBB)

- Either the left or the right ventricle may depolarize late, creating a "wide" or "notched" QRS complex.



35

Rate: Depends on rate of underlying rhythm
Rhythm: Regular
P Waves: Normal (upright and uniform)
PR Interval: Normal (0.12–0.20 sec)
QRS: Wide (>0.10 sec) with a notched appearance

♥ **Clinical Tip:** Bundle branch block commonly occurs in coronary artery disease.

Healthcare Provider Guidelines for CPR

CPR Method	Compression/ Ventilation Ratio	Rate of Compressions (min)	Depth of Compressions	Pulse Check (artery)	Hand Position for Compressions
Adult, 1 rescuer	30:2	At least 100	At least 2.0 in	Carotid	Heels of 2 hands over center of chest between nipple line
Adult, 2 rescuers	30:2	At least 100	At least 2.0 in	Carotid	Heels of 2 hands over center of chest between nipple line
Child, 1 rescuer	30:2	At least 100	At least 1/3 depth of chest (about 2.0 in)	Carotid or femoral	Heel of 1 or 2 hands over center of chest between nipple line
Child, 2 rescuers	15:2	At least 100	At least 1/3 depth of chest (about 2.0 in)	Carotid or femoral	Heel of 1 or 2 hands over center of chest between nipple line
Infant, 1 rescuer	30:2	At least 100	At least 1/3 depth of chest (about 1.5 in)	Brachial	2 fingers over center of chest, just below nipple line
Infant, 2 rescuers	15:2	At least 100	At least 1/3 depth of chest (about 1.5 in)	Brachial	2 thumbs–encircling hands technique over lower third of sternum

CPR Overview

Every day around the world, cardiopulmonary resuscitation (CPR) is used in and out of the hospital to save lives. CPR has saved the lives of children who are drowning or have swallowed something accidentally, as well as those of adults suffering from a heart attack.

CPR encompasses a level of medical care that revives, resuscitates, or sustains a person who is in cardiac or respiratory arrest. The person's heartbeat and breathing may be compromised or stopped by a heart attack, drowning, choking, or other emergency. Healthcare personnel learn how to recognize emergencies, such as sudden cardiac arrest, and know how to respond. Skills taught in this tab include performing CPR and relieving choking (foreign-body airway obstruction) in all ages: adult, child, and infant. Also included are use of a bag-mask device and an automated external defibrillator (AED).

The adult and pediatric chains of survival ensure the proper steps for resuscitation.

Adult Chain of Survival

- Immediate recognition of cardiac or respiratory arrest with early access to the emergency medical response team
- Early CPR
- Early defibrillation
- Early access to advanced medical care
- Effective post-resuscitation care

Pediatric Chain of Survival

- Prevention of cardiac or respiratory arrest
- Early CPR
- Early access to the emergency medical response team
- Early access to advanced medical care
- Effective post-resuscitation care

What Is CPR?

CPR is performed when a person's breathing or heart has stopped. Its purpose is to move blood, and therefore oxygen, to the brain and heart. CPR involves the following three steps:

C-A-B	
C—Circulation	Compressing the chest to keep the blood circulating
A—Airway	Opening the airway (the passageway between the nose/mouth and the lungs)
B—Breathing	Giving rescue breaths that fill the lungs with air

Why Perform CPR?

- CPR is performed to prevent brain damage and death when a person is in cardiac arrest. The heart may stop because of heart disease, a motor vehicle accident, drowning, or choking.
- Anyone who has lost consciousness may need CPR. Also, confusion, weakness, and chest pain may signal that cardiac arrest is about to occur and that CPR may be needed.
- After the heart stops, even a few minutes' delay in starting CPR can mean the difference between life and death.
- Performing CPR supports the heart and brain with oxygen until medical help arrives.

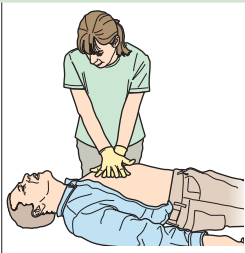
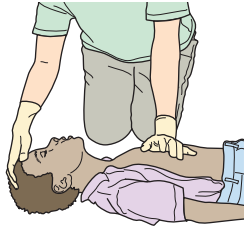
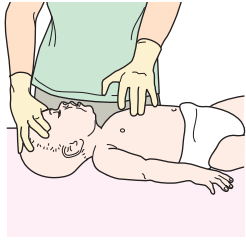
How Can I Tell Whether CPR Is Needed?

If the person is conscious but cannot talk and appears to be choking, CPR is not appropriate. Instead, follow the instructions for choking on pages 53–58.

If the person appears to have lost consciousness:

- Ask, "Are you OK?" Call out loudly. The person may be asleep or hard of hearing.
- If the person answers, ask how you can help.
- If there is no answer, gently tap the person's shoulder (or feet in an infant).
- If there is still no response, begin the three steps (circulation, airway, breathing) of CPR. The general technique for each step is described next. See the step-by-step instructions for CPR for adults (page 44), children (page 48), and infants (page 50).

How to Perform Chest Compressions

ADULT	• Place the heel of one hand over the center of the chest between the nipple line (lower half of sternum). • Place the heel of your other hand over the first. • Keep your arms straight and locked at the elbows. • Firmly compress the chest at least 2.0 in (5 cm). • Push hard and fast. • Allow complete recoil after each compression.	
CHILD	• Place the heel of one or both hands over the center of the chest between the nipple line (lower half of sternum). Keep your arm(s) straight and locked at the elbows. • Firmly compress the chest to at least 1/3 its depth (about 2.0 in [5 cm]). • Push hard and fast. • Allow complete recoil after each compression.	
INFANT	• Place two fingers just below the nipple line on the sternum. • Firmly compress the chest to at least 1/3 its depth (about 1.5 in [4 cm]). • Push hard and fast. • Allow complete recoil after each compression.	

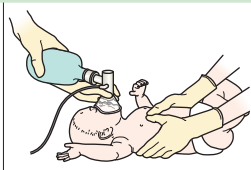
Continued

How to Perform Chest Compressions—cont'd

INFANT

Two thumbs–encircling hands technique:

- Encircle the infant's chest with both of your hands.
- Position your the thumbs just below the nipple line.
- Firmly compress the chest, with your thumbs, at least 1/3 the depth of the chest (about 1.5 in [4 cm]).
- Push hard and fast.
- Allow complete recoil after each compression.



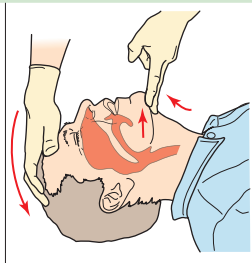
How Do I Open the Airway?

You must ensure an open airway. This does not require looking in the mouth, but instead requires straightening the angle of the head and neck so that the path of airflow is unobstructed (e.g., by the person's tongue).

How to Open the Airway

Head tilt–chin lift method:

- Place the person face up on a hard, flat surface.
- Lift the chin with one hand while pushing down on the forehead with the other hand. This aligns the airway structures.

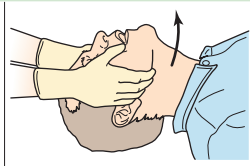


How to Open the Airway—cont'd

Jaw thrust method (if suspected spinal injury):

- Place the person face up on a hard, flat surface.
- Place the fingers of both your hands on each side of the person's jaw.
- Lift the jaw with both hands.

The jaw will be displaced forward, opening the airway.



How Do I Locate a Pulse?

The pulse points used are carotid, femoral, and brachial.

Carotid (adult or child):

- Locate the trachea.
- Using 2 or 3 fingers, feel for a pulse between the trachea and the muscles of the neck.

Femoral (child):

- Locate the inner thigh.
- Using 2 or 3 fingers, feel for a pulse midway between the pubic and hip bones.

Brachial (infant):

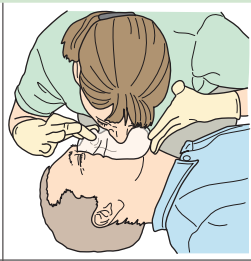
- Locate the medial portion of the upper arm.
- Using 2 or 3 fingers, feel for a pulse.

How Do I Perform Rescue Breathing?

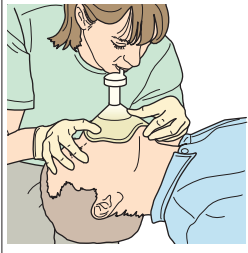
Rescue breathing delivers oxygen to the victim's lungs. The most common technique for single-rescuer breathing is a face mask or face shield. Use a bag-mask device when performing two-rescuer CPR.

Three Rescue Breathing Methods

Method 1, CPR face shield:
- Place the shield over the person's mouth with the airway tube between the lips.
- Hold the airway open and pinch the nose shut with your fingers.
- Give rescue breaths through the breathing tube.



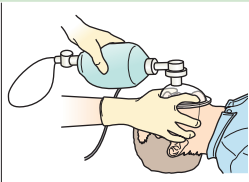
Method 2, CPR face mask:
- Put the mask over the person's nose and mouth as shown.
- Make sure the airway is open and press the mask against the face to create an airtight seal.
- Lift the jaw with one hand holding the mask in place and use your other hand to seal the mask around the nose.
- Give rescue breaths through the one-way valve.



Three Rescue Breathing Methods—cont'd

Method 3, Bag-mask device:

- Put the mask over the person's nose and mouth as shown.
- Make sure the airway is open and press the mask against the face to create an airtight seal.
- Lift the jaw with one hand using the **E-C clamp technique** to hold the mask in place. Circle the thumb and first finger around the top of the mask (forming a C) while using the third, fourth, and fifth fingers (forming an E).
- Use your other hand to squeeze the bag, giving rescue breaths through the one-way valve.
- A bag-valve device has an attachment for supplemental oxygen.



Do I Need an Automated External Defibrillator?

An automated external defibrillator (AED) can be used to "kick-start" a heart that has stopped beating. If CPR does not revive the person and an AED is available, you should use it. We explain how to use an AED in Tab 6: Skills.

What Is High-Quality CPR?

- Push hard and fast, delivering 30 compressions in less than 18 sec.
- Make sure you allow for complete chest recoil after each compression.
- Chest compressions should be interrupted infrequently and for no longer than 10 sec. Pulse checks, even to determine return of spontaneous circulation (ROSC), should be minimized during resuscitation.
- After every fifth cycle (2 min), rescuers should switch roles. This minimizes rescuer fatigue, which can reduce compression rates and/or depth to an inadequate level even if unrecognized. The switch should be accomplished in less than 5 sec.
- Avoid excessive ventilations.

CPR: Unconscious Adult (puberty or older)

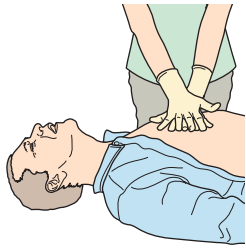
Emergency Actions

1. First, check to make sure the scene is safe.
2. **Check for unresponsiveness.**
   - Tap the person's shoulder.
   - Ask, "Are you OK?"
   - See if the person moves or makes a noise.

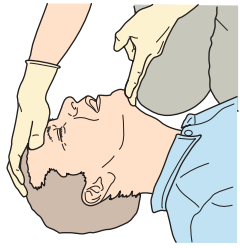
3. Check for breathing (no more than 10 sec). If the person is not breathing or is gasping for breath (agonal breaths), go immediately to step 4.
4. If there is no breathing or abnormal breathing (only agonal gasps) and you are alone, summon help, call a code, or phone 911 and get an AED. Send a second rescuer, if available, for help.
5. Position the person face up on a hard, flat surface.
6. Assess the carotid pulse (no more than 10 sec).

Emergency Actions—cont'd

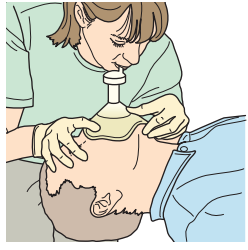
7. **Begin chest compressions.**
   - Place the heel of one hand over the center of the chest between the nipple line (lower half of sternum); place the heel of your other hand over the first. Keep your arms straight and locked at the elbows.
   - Firmly compress the chest at least 2.0 in (5 cm).
   - Deliver 30 compressions at a rate of at least 100/min.
   - Push hard and fast.
   - Allow complete recoil after each compression.
   - Count 1, 2, 3, 4 . . . up to 30.



8. After 30 compressions, open the airway by the head tilt–chin lift method or, if spinal injury is suspected, use the jaw thrust method, if possible.



9. If the person is not breathing, begin rescue breaths.
   - Use a face mask or face shield.
   - Give 2 breaths (1 sec each) with sufficient volume to make the chest rise.
   - Do not overventilate.



Continued

Emergency Actions—cont'd

10. Continue to deliver 30 compressions followed by 2 breaths until an AED arrives. Follow the instructions on how to use an AED in Tab 6: Skills. If an AED is unavailable, continue to give 30 compressions followed by 2 breaths.

11. When the **second rescuer** arrives:
    - Give cycles of 30 compressions and 2 breaths.
    - Always begin the switch with compressions.
    - Use a bag-mask device to deliver breaths.

What to Do Next

- If adequate breathing and circulation resume, place the person in the recovery position and monitor the person until help arrives.
- If circulation resumes but breathing does not or is inadequate, continue rescue breathing at the rate of 10–12 breaths/min (one breath every 5–6 sec) until help arrives. Check the pulse every 2 min.
- If neither circulation nor breathing occurs, continue CPR until help arrives. If an AED is available, set up and use it. See instructions in Tab 6: Skills.

♥ **Clinical Tip:** In two-rescuer CPR, after every fifth cycle (2 min), rescuers should switch roles. This minimizes rescuer fatigue, which can reduce compression rate and/or depth to an inadequate level even if unrecognized. The switch should be accomplished in less than 5 sec.

♥ **Clinical Tip:** Victims of asphyxial arrest (e.g., drowning, drug overdose, respiratory failure) should receive five cycles (2 min) of CPR before the lone rescuer leaves to call for help (to activate the EMS system).

♥ **Clinical Tip:** Push hard and fast, delivering 30 compressions in less than 18 sec.

♥ **Clinical Tip:** Chest compressions should be interrupted infrequently and for no longer than 10 sec. Pulse checks, even to determine ROSC, should be minimized during resuscitation.

CPR: Unconscious Adult (puberty or older) One and Two Rescuers With a Bag-Mask Device and AED

Emergency Actions

1. First, check to make sure the scene is safe.
2. Check for unresponsiveness. Tap the person's shoulder. Ask, "Are you OK?" See if the person moves or makes a noise.
3. Check for breathing (no more than 10 sec). If the person is not breathing or is only gasping for breath (agonal breaths), go immediately to step 4.
4. If there is no breathing or abnormal breathing (only agonal gasps) and you are alone, summon help, call a code, or phone 911 and get an AED. Send a second rescuer, if available, for help.
5. Position the person face up on a hard, flat surface.
6. Assess the carotid pulse (no more than 10 sec).
7. The **first rescuer** begins the sequence of 30 chest compressions followed by 2 breaths.
8. The **second rescuer** arrives with the bag-mask device, turns on the AED, and attaches the pads.
9. The **second rescuer** clears the area around the person while the AED analyzes the ECG rhythm.
10. If the AED identifies a shockable ECG rhythm, the **second rescuer** clears the area around the person and the AED delivers a shock.
11. After the shock is delivered, CPR is continued:
    - The **second rescuer** immediately delivers 30 compressions.
    - The **first rescuer** follows by delivering 2 breaths with the bag-mask device.

What to Do Next

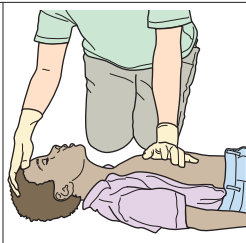
- If adequate breathing and circulation resume, place the person in the recovery position and monitor the person until help arrives.
- If circulation resumes but breathing does not or is inadequate, continue rescue breathing at the rate of 10–12 breaths/min (one breath every 5–6 sec) until help arrives. Check the pulse every 2 min.

CPR: Child (1 yr to puberty)

Emergency Actions

1. First, check to make sure the scene is safe.
2. **Check for unresponsiveness.**
   - Tap the child's shoulder.
   - Ask, "Are you OK?"
   - See if the child moves or makes a noise.

3. Check for breathing (no more than 10 sec). If the child is not breathing or is only gasping for breath (agonal breaths), go immediately to step 4.
4. If there is no breathing or abnormal breathing (only agonal gasps) and you are alone, begin the steps for CPR.
5. Position the child face up on a hard, flat surface.
6. Assess the carotid or femoral pulse (no more than 10 sec).
7. **Begin chest compressions.**
   - Place the heel of one hand or both hands over the center of the chest between the nipple line (lower half of sternum). Keep your arm(s) straight and locked at the elbows.
   - Firmly compress the chest to at least 1/3 its depth (about 2.0 in [5 cm]).
   - Deliver 30 compressions at a rate of at least 100/min.
   - Push hard and fast.
   - Allow complete recoil after each compression.
   - Count 1, 2, 3, 4 . . . up to 30.



Emergency Actions—cont'd

8. After 30 compressions, open the airway by the head tilt–chin lift method or, if spinal injury is suspected, use the jaw thrust method if possible.

9. If the child is not breathing, begin rescue breaths.
   - Use a face mask or face shield.
   - Give 2 breaths (1 sec each) with sufficient volume to make the chest rise.
   - Do not overventilate.

10. Continue to deliver cycles of 30 compressions followed by 2 breaths. After the fifth cycle of 30:2 (2 min), if you are still alone and no signs of circulation are present, summon help, call a code, or phone 911 and get an AED.

11. When the **second rescuer** arrives:
    - Give cycles of 15 compressions and 2 breaths.
    - Always begin the switch with compressions.
    - Use a bag-mask device to deliver breaths.

What to Do Next

- If adequate breathing and circulation resume, place the child in the recovery position and monitor the child until help arrives.
- If circulation resumes but breathing does not or is inadequate, continue rescue breathing at the rate of 12–20 breaths/min (one breath every 3–5 sec) until help arrives. Check the pulse every 2 min.
- If neither circulation nor breathing occurs, continue CPR until help arrives. If an AED is available, set up and use it. See instructions in Tab 6: Skills.

♥ **Clinical Tip:** If you are alone and know a child has had a **sudden collapse** due to heart failure, request immediate help including an AED. Do not delay defibrillation.

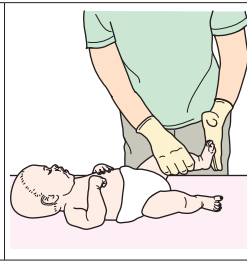
♥ **Clinical Tip:** When two rescuers are available, give cycles of 15 compressions and 2 breaths. Use a bag-mask device with supplemental oxygen to deliver breaths. After every fifth cycle (2 min), rescuers should switch roles. This minimizes rescuer fatigue, which can reduce compression rates and/or depth to an inadequate level even if unrecognized. The switch should be accomplished in less than 5 sec.

♥ **Clinical Tip:** Push hard and fast, delivering 30 compressions in less than 18 sec, or 15 compressions in less than 9 sec.

CPR: Infant (younger than 1 yr)

Emergency Actions

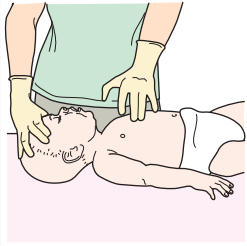
1. First, check to make sure the scene is safe.
2. **Check for unresponsiveness.**
   - Gently rub the infant's back or tap the feet.
   - Never shake an infant.
   - See if the infant moves or makes a noise.



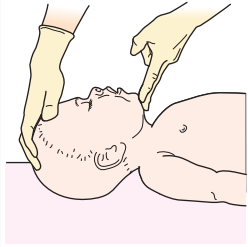
3. Check for breathing (no more than 10 sec). If the infant is not breathing or is only gasping for breath (agonal breaths), go immediately to step 4.
4. If there is no breathing or abnormal breathing (only agonal gasps) and you are alone, begin the steps for CPR.
5. Position the infant face up on a hard, flat surface.
6. Assess the brachial pulse (no more than 10 sec).

Emergency Actions—cont'd

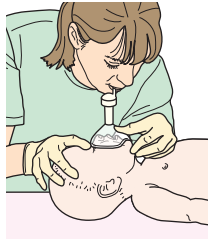
7. **Begin chest compressions.**
   - Place two fingers just below the nipple line on the sternum.
   - Firmly compress the chest to at least 1/3 its depth (about 1.5 in [4 cm]).
   - Deliver 30 compressions at a rate of at least 100/min.
   - Push hard and fast.
   - Allow complete recoil after each compression.
   - Count 1, 2, 3, 4 . . . up to 30.



8. After 30 compressions, open the airway by the head tilt–chin lift method or, if spinal injury is suspected, use the jaw thrust method if possible.
   - When using the head tilt–chin lift method, the infant's head should not be tilted too far back; it may close off the airway.



9. If the infant is not breathing, begin rescue breaths.
   - Use a face mask or face shield.
   - Give 2 breaths (1 sec each).
   - Use small puffs of air so you don't overinflate the lungs.



Continued

Emergency Actions—cont'd

10. Continue to deliver cycles of 30 compressions followed by 2 breaths. After the fifth cycle of 30:2 (2 min), if you are still alone and no signs of circulation are present, summon help, call a code, or phone 911 and get an AED.

11. When the **second rescuer** arrives:
    - Give cycles of 15 compressions and 2 breaths.
    - Always begin the switch with compressions.
    - Use the two thumbs–encircling hands technique for chest compressions, positioning the thumbs just below the nipple line.
    - Use a bag-mask device to deliver breaths.

What to Do Next

- If adequate breathing and circulation resume, place the infant in the recovery position and monitor the infant until help arrives.
- If circulation resumes but breathing does not or is inadequate, continue rescue breathing at the rate of 12–20 breaths/min (one breath every 3–5 sec) until help arrives. Check the pulse every 2 min.
- If neither circulation nor breathing occurs, continue CPR until help arrives. If an AED is available, set up and use it. See instructions in Tab 6: Skills.

♥ **Clinical Tip:** If you are alone and know an infant has had a **sudden collapse** due to heart failure, request immediate help including an AED. Do not delay defibrillation.

♥ **Clinical Tip:** When two rescuers are available, give cycles of 15 compressions and 2 breaths. Use a bag-mask device with supplemental oxygen to deliver breaths. After every 2 min, rescuers should switch roles. This minimizes rescuer fatigue, which can reduce compression rates and/or depth to an inadequate level even if unrecognized. The switch should be accomplished in less than 5 sec.

♥ **Clinical Tip:** Push hard and fast, delivering 30 compressions in less than 18 sec, or 15 compressions in less than 9 sec.

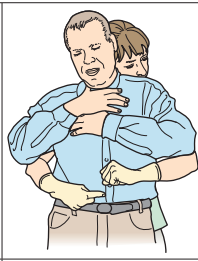
CHOKING: Conscious Adult or Child (1 yr or older)

Signs and Symptoms

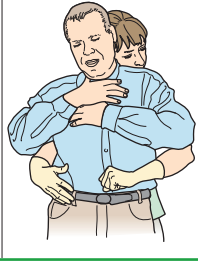
- Grabbing at the throat with one or both hands
- Noisy breathing, gagging, coughing
- Inability to speak or make a sound

Emergency Actions

1. Determine whether the airway is blocked. Ask, "Are you choking? Can you speak? Can I help you?"
2. If you determine the airway is blocked:
   - Stand behind the person and wrap your arms around the person's waist. Locate the navel.
   - If the person is obese or pregnant, wrap your arms around the chest.



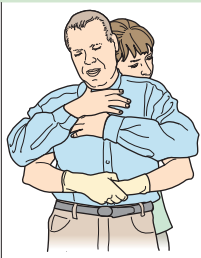
3. Make a fist.
   - Place the thumb side just above the navel.
   - Locate the middle of the chest (sternum) if the person is obese or pregnant.



Continued

Emergency Actions—cont'd

4. Grasp your fist with your other hand.
   - Press your fist abruptly into the abdomen and use an upward, inward thrust.
   - Use a straight thrust back if the person is obese or pregnant.
   - Your action will push air from the lungs like a cough. This can help dislodge an object.



What to Do Next

- Continue thrusts until the object is coughed out and the person can breathe or talk.
- If the object cannot be removed, the person will lose consciousness.
- If the person loses consciousness, lower the person to the ground and place face up. Treat as a choking adult or child who becomes unconscious (page 54).

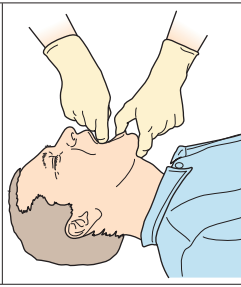
CHOKING: Unconscious Adult or Child (1 yr or older)

Signs and Symptoms

- Unconsciousness caused by choking
- Failure to breathe
- Cyanosis around lips or skin
- Inability to move air into the lungs with rescue breaths

Emergency Actions

1. Check for unresponsiveness. Tap the person's shoulder. Ask, "Are you OK?" See if the person moves or makes a noise.
2. If there is abnormal or no breathing and others are present, tell someone to summon help, call a code, or phone 911. Start CPR immediately, beginning with chest compressions.
3. Each time you open the person's mouth to give rescue breaths:
   - Look in the person's mouth for an object such as a piece of food that may have become stuck in the throat.
   - If you see an object, do not push it farther into the throat. Instead, use your finger to sweep the object out.
   - **If you do not see an object, never perform a blind finger sweep.**
   - Chest compressions from CPR can force air from the lungs to dislodge the object.



4. Continue 5 cycles (2 min) of 30 compressions followed by 2 breaths. Stop and look for signs of breathing, coughing, or movement. If these signs are not present, continue CPR until help arrives.
5. After a **second rescuer** arrives, continue the CPR ratio for an adult at 30 compressions followed by 2 breaths. The child–two rescuer ratio is 15 compressions followed by 2 breaths. Always begin the two-rescuer switch with compressions.

What to Do Next

- If the person moves but is still not breathing, continue rescue breathing at 10–12 breaths/min for an adult and 12–20 breaths/min for a child.
- If breathing begins, monitor the person until help arrives.

Clinical Tip: An airway obstruction is successfully removed if you see and remove the object or feel air movement and see the chest rise when you give breaths.

CHOKING: Conscious Infant (younger than 1 yr)

Signs and Symptoms

- Inability to breathe or cry
- High-pitched crowing sounds
- Sudden wheezing or noisy breathing

Emergency Actions

1. Determine whether the airway is blocked. Listen for noisy or high-pitched breathing.
2. If you determine the airway is blocked, support the infant's head and neck and lay the infant face down on your forearm. Using your leg or lap for support, keep the infant's head lower than the body.
3. **Deliver back slaps:**
   - With the heel of your free hand, strike 5 quick, forceful slaps between the infant's shoulder blades.

4. Turn the infant face up on your other arm. Using your leg or lap for support, keep the infant's head lower than the body.

Emergency Actions—cont'd

5. **Deliver chest thrusts:**
   - Place two fingers on the sternum just below the nipple line.
   - Deliver five quick thrusts downward, depressing the chest by 1/3 (1.5 in) to 1/2 its depth each time.

What to Do Next

- Continue the sequence of 5 back slaps and 5 chest thrusts until the object is coughed out or the infant cries.
- If the object cannot be removed, the infant will lose consciousness.
- If the infant loses consciousness, place the infant face up and follow the steps for choking infant who becomes unconscious (page 57).

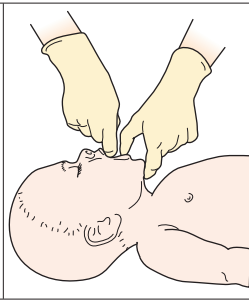
CHOKING: Unconscious Infant (younger than 1 yr)

Signs and Symptoms

- Unconsciousness caused by choking
- Failure to breathe
- Cyanosis around lips or skin
- Inability to move air into the lungs with rescue breaths

Emergency Actions

1. Check for unresponsiveness. Gently rub the infant's back or tap the feet. Never shake an infant. See if the infant moves or makes a noise.
2. If there is abnormal or no breathing and others are present, tell someone to summon help, call a code, or phone 911. Start CPR immediately, beginning with chest compressions.
3. Each time you open the infant's mouth to give rescue breaths:
   - Look in the infant's mouth for an object such as a piece of food or toy that may have become stuck in the throat.
   - If you see an object, do not push it farther into the throat. Instead, use your finger to sweep the object out.
   - **If you do not see an object, never perform a blind finger sweep.**
   - Chest compressions from CPR can force air from the lungs to dislodge the object.



4. Continue 5 cycles (2 min) of 30 compressions followed by 2 breaths. Stop and look for signs of breathing, coughing, or movement.
5. After a **second rescuer** arrives, continue the CPR ratio at 15 compressions followed by 2 breaths. Always begin the two-rescuer switch with compressions.

What to Do Next

- If the infant moves but is still not breathing, continue rescue breathing at 12–20 breaths/min.
- If breathing begins, monitor the infant until help arrives.

Tab 3: ACLS

Healthcare providers are expected to respond promptly and competently to cardiovascular emergencies and cardiopulmonary arrest. This expectation causes anxiety when the provider encounters such a situation. Knowledge of ECG rhythm interpretation, pharmacology, and emergency skills provides the foundation for Advanced Cardiac Life Support (ACLS) and instills confidence and competence in emergencies.

Systematic Approach

The immediate goal of resuscitation is the return of spontaneous circulation (ROSC). The ultimate goal is to ensure survival with intact neurological function by maintaining effective oxygenation, ventilation, and circulation. A systematic approach incorporates the basic life support (BLS) and ACLS surveys. Emphasis is on **high-quality CPR** and **early defibrillation** (for VF or pulseless VT).

When first encountering a cardiopulmonary emergency/arrest, always make sure the scene is safe before initiating BLS and ACLS.

BLS Survey

- **Check patient responsiveness.** If the patient is conscious, proceed directly to the ACLS survey.
- **Activate** the emergency response system and get an AED.
- **C–A–B**
  - **C—Circulation:** Check pulse (5–10 sec). If pulseless, start CPR, beginning with compressions (30 compressions/2 ventilations).
  - **A—Airway:** Open the airway.
  - **B—Breathing:** Provide two breaths.
- **Defibrillation**
  - If the rhythm is shockable, deliver shock promptly and resume CPR within 10 sec, starting with compressions.

ACLS Survey

- **A—Airway**
  - Maintain patent airway.
  - Maintain proper head position.
  - Use oropharyngeal or nasopharyngeal airway if indicated.
  - Use advanced airway if indicated (laryngeal mask airway [LMA], laryngeal tube, esophageal-tracheal tube, endotracheal tube [ET]).

- **B—Breathing**
  - Perform bag-mask ventilation.
  - Provide supplemental oxygen.
  - Monitor adequacy of ventilation and oxygenation.
    - Ensure adequate chest rise.
    - Use CO_2 detector or quantitative waveform capnography.
    - Measure oxygen saturation.
    - Avoid excessive ventilation.
- **C—Circulation**
  - Provide high-quality CPR.
  - Monitor cardiac rhythm.
  - Initiate prompt defibrillation/cardioversion when indicated.
  - Establish IV/IO access.
  - Administer medication when indicated.
  - Administer volume resuscitation when indicated.
  - Assess for ROSC.
- **D—Differential Diagnosis**
  - Identify and treat potentially reversible causes. Assess Hs and Ts (see Cardiac Arrest Algorithms):
    - **Hypokalemia/hyperkalemia:**
      - **Hypokalemia** should be suspected in patients on diuretics, those with a recent history of vomiting or diarrhea, and malnourished patients, especially alcoholic and elderly patients. A baseline ECG would reveal flattened T waves and prominent U waves, the latter best seen in leads V_2–V_3, ST depression, QT prolongation, and QRS widening. There could be increased ventricular ectopy: PVCs, nonsustained ventricular tachycardia (NSVT). Emergent treatment for a potassium level <2.5 mEq/L or significant ventricular ectopy includes potassium repletion with potassium chloride IV, maximum 20 mEq/hr by central line, or 10 mEq/hr by peripheral line. In cardiac arrest, potassium **cannot** be administered by IV bolus for more rapid repletion. IV magnesium sulfate should be administered because hypokalemia is often associated with hypomagnesemia. Magnesium is necessary for movement of potassium in and out of cells, and it stabilizes cell membranes. Hypomagnesemia must be corrected before the potassium level normalizes.
      - **Hyperkalemia** should be suspected in patients with a history of renal failure, diabetes with hyperglycemia, recent dialysis, metabolic acidosis, or use of potassium-sparing medications such as spironolactone, ACE inhibitors, or angiotensin-receptor blockers. A baseline ECG would reveal tall, peaked T waves; small P waves or loss of P waves; and QRS widening. Patients are at risk for fatal arrhythmias. With severe hyperkalemia, patients may present in cardiac arrest with sine waves (the P wave disappears and the QRS and T wave merge in an oscillating pattern) on the ECG. Emergent

treatment includes administration of IV calcium chloride to stabilize myocardial cell membranes, and IV sodium bicarbonate to shift potassium out of the vascular space and into body cells. In the absence of cardiac arrest, other treatments include IV insulin plus glucose or nebulized albuterol to shift potassium into cells, and IV furosemide, sodium polystyrene sulfonate (Kayexalate), or dialysis to increase potassium excretion.

- **Hypovolemia:** Hypovolemia should be suspected in patients taking diuretic medications or with a recent history of vomiting or diarrhea, gastrointestinal (GI) or other internal bleeding, traumatic blood loss, ruptured ectopic pregnancy, placenta previa or abruption, significant burns, or fluid shifts with peripheral edema (third-space shifting) but vascular dehydration. Physical findings include flattened neck veins; tachycardia; pallor; narrowing pulse pressure; cool, clammy skin; and eventual hypotension. The ECG may be normal with a rapid heart rate. Emergent treatment is rapid administration of IV fluid. In cases of external bleeding, direct pressure to the wound is indicated. In cardiac arrest, hypovolemia must be considered as a potential cause; this is easily and rapidly treated. **Resuscitation measures in cardiac arrest will be ineffective unless intravascular volume is replaced rapidly.**
- **Hypoxia:** Hypoxia (low arterial and tissue oxygen) should be suspected in patients with a history of asthma, COPD, or CHF. Causes of hypoxia include airway obstruction, pulmonary embolus, pulmonary edema, significant pleural effusions, pneumothorax or hemothorax, severe asthma attack, COPD exacerbation, or respiratory infection. Patients present with acute respiratory distress. Physical findings include tachypnea, dyspnea, tachycardia or bradycardia, and possible cyanosis (not present with low hemoglobin). Pulse oximetry would reveal low oxygen saturation. ABG would confirm low PaO_2, low oxygen content, and low oxygen saturation. Emergent treatment includes maintaining a patent airway (advanced airway if indicated), supplemental oxygen, and adequate ventilation. In cardiac arrest, patients require bag-mask ventilation with oxygen. If this is ineffective, an advanced airway should be rapidly inserted. Improved oxygenation enhances the efficacy of resuscitation measures.
- **Hypothermia:** Hypothermia should be suspected in patients with a history of exposure to cold temperatures or cold-water immersion. Very young, elderly, or debilitated patients and patients with alcohol or drug intoxication are at increased risk. A central body temperature of 34°–36°C (93.2°–96.8°F) is mild hypothermia; 30°–34°C (86–93.2°F) is moderate hypothermia; <30°C (<86°F) is severe hypothermia. The ECG may show sinus bradycardia with prolonged PR and QT intervals and a widened QRS complex. A-fib may occur. As the core body temperature decreases, a J or Osborne wave may be noted, appearing as an extra deflection between the end of the QRS and the beginning

of the ST segment, best seen in the inferior leads (II, III, aVF) and lateral precordial leads (V_4–V_6). The rhythm may deteriorate to VT/VF or asystole.

The patient should be removed from the cold environment and wet garments taken off. Mild hypothermia may respond to passive rewarming (warm room). Moderate hypothermia requires external rewarming with warm blankets, radiant heat sources, heating pads, hot-water bottles (use caution to prevent skin burns), or other devices. Severe hypothermia requires core rewarming with warm IV/IO fluids and warm humidified oxygen. In cardiac arrest, core rewarming is best accomplished with cardiopulmonary bypass. Alternatives include warm-water lavage of the thoracic cavity, extracorporeal (occurring outside the body) blood warming with partial bypass, or warm peritoneal lavage with a solution free of potassium chloride. These treatments are not available or feasible in all settings.

Cardiac arrest should be treated with high-quality CPR and with defibrillation for VT/VF. If cardiac arrest continues, an advanced airway should be inserted and warm (42°–46°C or 108°–115°F), humidified oxygen should be administered as soon as possible. Warm (43°C or 109°F) IV normal saline should be infused. Drug administration during cardiac arrest remains controversial. It may be reasonable to administer epinephrine or vasopressin (vasopressors) during resuscitation. Prolonged resuscitation may be required and should be continued until the patient has been adequately rewarmed. If there is no ROSC after rewarming to a core temperature ☒35°C (☒95°F), resuscitation measures may be withheld and the patient pronounced dead.

- **Hydrogen ion (acidosis):** Bicarbonate-responsive metabolic acidosis should be suspected in patients with diabetes (diabetic ketoacidosis [DKA]), renal failure, or drug overdose with tricyclic antidepressants, aspirin, cocaine, or diphenhydramine. ABG would reveal a low pH, low bicarbonate level, and normal $PaCO_2$ (indicating no respiratory compensation) or low $PaCO_2$ (more likely, indicating hyperventilation for respiratory compensation). A baseline ECG may reveal low-amplitude QRS complexes.

  Patients in cardiac arrest with respiratory or metabolic acidosis should receive adequate ventilation and oxygenation. IV sodium bicarbonate may be administered for bicarbonate-responsive acidosis as a buffering agent to raise blood pH and may enhance resuscitation measures. It should **not** be administered for respiratory acidosis, indicated by a low pH, high $PaCO_2$, and normal bicarbonate level (indicating no metabolic compensation) or high bicarbonate level (indicating chronic metabolic compensation). It also should **not** be administered for lactic acidosis (metabolic acidosis associated with shock or prolonged cardiac arrest). High-quality CPR and adequate

ventilation are the best "buffering agents" for these types of acidosis. **There are no quick remedies to correct lactic acidosis.**

- **Tension pneumothorax:** Intrapleural pressure is normally negative, allowing lung expansion. When the visceral pleura is penetrated, air enters the pleural space and accumulates with no way to exit, causing positive intrapleural pressure. The lung collapses partially or totally, causing hypoxia, respiratory distress, and possible mediastinal shift with decreased cardiac output and hypotension. Tension pneumothorax should be suspected in patients who develop acute respiratory distress. It may occur spontaneously in healthy patients or in patients with COPD, asthma, cystic fibrosis, lung cancer, lung infection, tuberculosis, Marfan syndrome, or AIDS, or after scuba diving. It may result from trauma associated with rib fractures, intubation, mechanical ventilation, central line placement, liver biopsy, thoracentesis, or pericardiocentesis. Physical findings include tachypnea, dyspnea, distended neck veins, absent or severely diminished breath sounds on the affected side, possible tracheal deviation toward the unaffected side, initial tachycardia, hypotension, and eventual bradycardia.

  The patient may go into cardiac arrest with PEA and have no pulse palpable during CPR, and it may be difficult to ventilate the patient. The ECG would probably show narrow QRS complexes with bradycardia caused by hypoxia. **Emergent treatment requires needle decompression and tube thoracostomy.** Resuscitation measures will probably be ineffective unless the chest is decompressed rapidly.
- **Thrombosis (pulmonary or coronary):**
  - **Pulmonary thrombosis** is one or more clots lodged in at least one pulmonary artery. Most clots are formed by blood but occasionally other substances such as fat from the marrow of a broken bone, part of a tumor, or air bubbles can form blockages. Thrombosis impedes blood gas exchange and decreases venous return to the left atrium. If large enough, clots can reduce cardiac output and cause hypoxia and hypotension. Thrombosis should be suspected in patients who develop sudden, acute respiratory distress. Patients may have a history of a hypercoagulable disorder, previous DVT, or previous PE. Patients may have A-fib or a mechanical heart valve, without anticoagulation therapy, or have a subtherapeutic international normalized ratio (INR). Other risk factors include oral contraceptives, pregnancy, immobility, advanced age, obesity, trauma (especially long-bone fractures, spinal injury, severe burns), surgery (especially orthopedic), chronic illness, cancer, and recent prolonged airline flight. Physical findings include tachypnea, focal wheeze, tachycardia, neck vein distention, possible hypotension, and shock. The ECG has narrow QRS complexes and a rapid heart rate. D-dimer (fibrin degradation product) level is elevated. An

echocardiogram may reveal right ventricular strain, pulmonary hypertension, tricuspid regurgitation, and septal flattening. A V/Q scan may show high probability for a PE. A CT chest angiogram would confirm a PE.

Treatment includes oxygen (ET intubation may be needed), anticoagulation therapy, and possible fibrinolytic therapy. Surgical embolectomy or pulmonary thromboendarterectomy may be considered for a massive PE. Massive PE may lead to cardiac arrest with a high mortality; resuscitation measures are likely to be ineffective. **There is no treatment to rapidly dissolve or remove the clot(s).**

– **Coronary thrombosis** is a blood clot in a coronary artery, obstructing blood flow and resulting in MI. It should be suspected in patients presenting with anginal symptoms; with a history of CAD, MI, previous PCI, or previous CABG; or with risk factors for CAD. The ECG may reveal T wave inversion and ST depression (unstable angina or NSTEMI) or ST elevation and possible abnormal Q waves (STEMI). Cardiac markers may be normal (early presentation) or elevated. Treatment includes oxygen, aspirin, nitroglycerin, morphine for unrelieved chest pain, heparin, antiplatelet therapy, and possible PCI. Fibrinolytic therapy may be administered for STEMI if there are no contraindications and PCI will be delayed. (See Acute Coronary Syndrome algorithm).

An MI may lead to cardiogenic shock requiring hemodynamic support and rapid revascularization if possible. A massive MI may lead to cardiac arrest with a high mortality; resuscitation measures are likely to be ineffective unless emergency PCI can be performed. This technology is not always rapidly available.

In some settings, emergency cardiopulmonary bypass may be initiated to stabilize the patient and facilitate PCI.

- **Tamponade, cardiac:** Cardiac tamponade results from rapid or excessive accumulation of fluid in the pericardial sac compressing the heart chambers and impeding ventricular filling during diastole. It decreases stroke volume during systole and decreases cardiac output. It should be suspected after blunt or penetrating chest trauma, large acute MI (rupture of ventricular free wall or ventricular aneurysm), or removal of epicardial pacing wires after CABG. Cardiac tamponade may be a complication of instrumentation during cardiac surgery, cardiac catheterization, electrophysiology procedures, or central line insertion. Other causes are pericardial effusion associated with CHF, end-stage renal disease, infection, malignancy, pericarditis, coagulopathies, collagen vascular disease, or massive fluid resuscitation. Patients typically present with dyspnea, vague chest pain, and possible abdominal discomfort, nausea, and vomiting. They may experience syncope or presyncope. Physical findings include

tachypnea, tachycardia, neck vein distention, distant heart sounds, narrowing pulse pressure, hypotension, and possible pulsus paradoxus (drop in systolic blood pressure ⊠10 mm Hg lower during inspiration than during expiration). The ECG reveals a narrow QRS complex with rapid HR. Diagnosis is confirmed by echocardiogram. The best treatment is echocardiogram-guided pericardiocentesis.

Cardiac tamponade may lead to cardiac arrest with PEA. The pulse may not be palpable during CPR. Neck veins are distended. **Resuscitation measures will probably be unsuccessful unless the condition is rapidly identified and treated.** Emergent treatment includes rapid IV fluid bolus administration to enhance ventricular filling until emergency pericardiocentesis can be performed. A pericardial drain may be inserted and attached to gravity drainage.

- **Toxins:** Drug overdose or poisoning may cause critical instability and lead to cardiac arrest. Common agents include tricyclic antidepressants, benzodiazepines, opioids, cocaine, sedatives, amphetamines, antihistamines, anticholinergic agents, beta blockers, calcium channel blockers, digoxin, and cyanide. Signs, symptoms, physical findings, and ECG changes vary with each class of offending agents. Drug overdose should be suspected on the basis of clinical presentation, patient history (e.g., poor eyesight, confusion, memory loss, disorientation, mental illness, substance abuse, or suicidal ideation), empty medication bottles found at the scene, pharmacy records, or bystander information. It is helpful if the patient can state the drug(s), amount, route, and time of self-administration.

  Initial management should focus on maintaining a patent airway; providing oxygenation, ventilation, and hemodynamic support; and performing a neurological examination. The patient will probably require ET intubation. Seizures must be treated promptly. Knowledge of common toxidromes (toxic syndromes) and their appropriate antidotes is indispensable to effective treatment. Patients presenting in cardiac arrest pose a great challenge, especially when the offending agent remains unknown. **Resuscitation measures may be ineffective if the appropriate antidote is not administered, or if there is no known antidote.**

Enhancing Successful Resuscitation

The ultimate goal of resuscitation is the **survival of neurologically intact patients.** In cardiopulmonary emergencies, enhancing successful resuscitation requires the integration and coordination of rapid-response teams and processes.

Processes

- **CPR:** Rapid initiation of high-quality CPR is essential to survival. (Review Tab 2: CPR.)
- **Effective ACLS:** Implementation of appropriate algorithms is based on identification of the patient's cardiac rhythm/clinical presentation, as presented below.
- **Rapid transport** to an emergency facility (prehospital events) or critical care unit (hospital events) ensures timely, ongoing, comprehensive care.
- **Integrated post–cardiac arrest care:** Developing a comprehensive care plan increases the likelihood of survival to discharge of neurologically intact patients.

Teams

- **Rapid-Response Team (RRT) or Medical Emergency Team (MET)**
  - **Identify and treat patients** with early clinical signs of instability to prevent cardiac arrest. Activated by a nurse, physician, patient, or visitor who is concerned about a patient's clinical status.
  - **Composed of healthcare providers** with emergency/critical care knowledge, skills, and experience. Rapid assessment and treatment strategies to stabilize the patient and prevent cardiac arrest.
- **Resuscitation Team** (For details, see Tab 7: Megacode.)
  - ACLS providers responding to a cardiac arrest (code blue).
  - Composed of:
    - **Team leader** (typically a paramedic for prehospital events; physician for hospital events) responsible for directing the code
    - **Team members,** 3–5 persons performing the following:
      - Chest compressions
      - Monitor-defibrillator operation
      - Airway management
      - IV/IO medication administration
      - Observing/recording

ACLS Algorithms

When patients present in respiratory or cardiac arrest or with cardiovascular emergencies, they must be assessed rapidly and systematically. ACLS algorithms outline step-by-step protocols for dealing with these situations.

67

Cardiac Arrest: Ventricular Fibrillation With CPR and AED

Clinical Presentation

- Unresponsive state
- No respirations or only agonal respirations
- No pulse

Management

1. **Establish unresponsiveness:**
   - No respirations or only agonal respirations and no pulse
2. **Activate emergency response system and get AED** or direct second rescuer to do so.
3. **C–A–B: Compressions, airway, breathing:**
   - Check pulse: absent.
   - Start CPR, beginning with chest compressions.
4. **Defibrillation**
   - As soon as AED is available, turn power on and follow prompts.
   - Attach AED pads to patient's chest.
   - Stop CPR while AED analyzes rhythm and advises shock.
   - Clear patient and deliver shock as prompted.
5. **Immediately resume CPR**, starting with chest compressions.
   - Provide five cycles (2 min) of uninterrupted CPR.
6. When prompted, **stop CPR while AED analyzes rhythm**.
   - **Defibrillation:** If shock is advised, repeat **defibrillation** and resume CPR.
   - If no shock is advised, quickly assess patient for ROSC. If ROSC is absent, resume CPR.
   - If signs of spontaneous circulation are present, monitor and reevaluate patient. Arrange for transport to a hospital/critical care unit. The patient will need a comprehensive care plan. (See the Immediate Post–Cardiac Arrest Care algorithm.)

Clinical Tip: Do not delay defibrillation. If two or more rescuers are available, perform CPR while the second rescuer turns on the AED and applies the pads to the patient's chest.

Clinical Tip: Emphasis must be on high-quality CPR, ensuring adequate rate and depth of chest compressions with complete chest recoil after each compression, minimizing interruptions in chest compressions (no more than 10 sec when necessary), and avoiding excessive ventilation. Rotate chest compressor every 2 min to minimize fatigue.

Cardiac Arrest: Ventricular Fibrillation (VF) or Pulseless Ventricular Tachycardia (VT)

Clinical Presentation

- Unresponsive state
- No respirations or only agonal respirations
- No pulse

Management

1. **Establish unresponsiveness:**
   - No respirations or only agonal respirations and no pulse.
2. **Call for help.**
3. **C–A–B: Compressions, airway, breathing:**
   - Begin CPR, starting with compressions.
   - Provide oxygen.
4. **Defibrillation**
   - **Attach AED or manual monitor-defibrillator** as soon as available without interrupting compressions.
   - When device is attached, stop CPR and assess rhythm.
     - AED: If shock is advised, defibrillate following AED prompts.
     - Manual monitor-defibrillator:
       - Biphasic manual defibrillator: Defibrillate at 120–200 J (follow manufacturer's device-specific energy levels if known, or 200 J if unknown).
       - Monophasic manual defibrillator: Defibrillate at 360 J.
5. **Immediately resume CPR,** beginning with compressions.
   - Provide 5 cycles (2 min) of uninterrupted CPR.
   - During CPR, establish IV or IO access.
   - Prepare vasopressor dose (epinephrine or vasopressin).
6. **Defibrillation**
   - Stop CPR.
   - Assess rhythm.
   - If rhythm remains shockable, follow AED prompts or **defibrillate:**
     - Biphasic manual defibrillator: Defibrillate at same or higher energy.
     - Monophasic manual defibrillator: Defibrillate at 360 J.
7. **Immediately resume CPR,** beginning with compressions.
   - Provide 5 cycles (2 min) of uninterrupted CPR.
   - Insert an advanced airway (ET, LMA, King LT, or Combitube) if basic airway management is inadequate.
     - Confirm correct tube placement without interrupting CPR.
     - After correct placement is confirmed, deliver **uninterrupted** chest compressions at a rate of at least 100/min and deliver **8–10 breaths/min at a rate of 1 breath every 6–8 sec.**

8. **Drugs**
   - Administer **epinephrine** 1 mg.
     - Give 10 mL of 1:10,000 IV/IO.
     - Follow with 20 mL IV flush.
     - Repeat every 3–5 min as needed.
   - A single dose of **vasopressin** 40 units IV/IO can be administered to replace the first or second dose of epinephrine.
   - If no IV/IO access is available and the patient has an ET in place, stop compressions and inject 2.0–2.5 mg (1:1,000) epinephrine diluted in 5–10 mL normal saline or sterile water directly into ET tube every 3–5 min until IV/IO access is available. Follow ET drug administration with 5 consecutive ventilations to disperse drug into small airways for absorption into pulmonary vasculature and resume compressions.
9. **Continue CPR; check rhythm every 2 min.**
10. **Defibrillation**
    - If the rhythm remains shockable, follow AED prompts or **defibrillate** at same or higher energy biphasic; 360 J monophasic.
11. **Immediately resume CPR, check rhythm every 2 min.**
12. **Drugs**
    - Consider antiarrhythmic drugs for shock-refractory VF or pulseless VT.
      - Administer **amiodarone** 300 mg IV/IO.
      - If amiodarone is unavailable or the patient has a known allergy, administer **lidocaine** 1.0–1.5 mg/kg IV/IO.
    - Repeat antiarrhythmic therapy for shock-refractory VF or VT.
      - **Amiodarone** 150 mg IV/IO in 3–5 min (use only once).
      - If using **lidocaine**, administer 0.5–0.75 mg/kg IV/IO and repeat every 5–10 min if necessary; maximum dose 3 mg/kg.
    - Consider **magnesium sulfate** 1–2 g (2–4 mL of a 50% solution) diluted in 10 mL of D5W IV/IO, given over 1–2 min only for cardiac arrest caused by hypomagnesemia or torsade de pointes.
13. During CPR, consider and treat potentially reversible causes of VF/VT (Hs and Ts):
    - Hypokalemia/hyperkalemia
    - Hypovolemia
    - Hypoxia
    - Hypothermia
    - Hydrogen ion (acidosis)
    - Tension pneumothorax
    - Thrombosis (pulmonary or coronary)
    - Tamponade, cardiac
    - Toxins
14. If rhythm changes to asystole or PEA, follow algorithm for asystole or PEA.
15. If the rhythm converts to a stable ECG rhythm with ROSC:
    - Monitor and reevaluate the patient.
    - Arrange for transport to a critical care unit. The patient will need a comprehensive care plan. (See the Immediate Post–Cardiac Arrest Care algorithm.)

♥ **Clinical Tip:** Emphasis must be on high-quality CPR, ensuring adequate rate and depth of chest compressions with complete chest recoil after each compression, minimizing interruptions in chest compressions (no more than 10 sec when necessary), and avoiding excessive ventilation. Rotate chest compressor every 2 min to minimize fatigue.

♥ **Clinical Tip:** Do not delay defibrillation. If two or more rescuers are available, perform CPR while preparing the defibrillator. Perform defibrillation as soon as possible.

♥ **Clinical Tip:** Secure an advanced airway and verify placement by observing bilateral chest expansion, auscultation of bilateral breath sounds, and lack of epigastric sounds. Use a confirmatory device (exhaled CO_2 detector). Use continuous waveform capnography if available. If not available, use a colorimetric CO_2 detector. Monitor tube for displacement during transport or whenever the patient is moved.

♥ **Clinical Tip:** Once an advanced airway is in place, deliver chest compressions continuously without pausing for ventilations. Deliver one breath every 6–8 sec without regard to the phase of chest compressions (downstroke vs. upstroke).

♥ **Clinical Tip:** If an advanced airway cannot be secured, continue ventilations with a bag-mask device, delivering two breaths after each set of 30 compressions.

Cardiac Arrest: Pulseless Electrical Activity (PEA)

Clinical Presentation

- Unresponsive state
- No respirations or only agonal respirations
- No pulse
- Organized electrical rhythm on monitor but no pulse

Management

1. **Establish unresponsiveness:**
   - No respirations or only agonal respirations and no pulse.
2. **Call for help.**
3. **C–A–B: Compressions, airway, breathing:**
   - Begin CPR, starting with compressions.
   - Provide oxygen.
4. **Attach AED or manual monitor-defibrillator** as soon as available without interrupting compressions.
   - When device is attached, stop CPR to assess rhythm.
     - AED: **No shock advised.**

- Manual monitor-defibrillator: Organized rhythm (PEA). **Do not defibrillate.**

5. **Immediately resume CPR,** beginning with compressions.
   - Provide 5 cycles (2 min) of uninterrupted CPR.
   - During CPR, establish IV or IO access.
   - Prepare vasopressor dose (epinephrine or vasopressin).
6. **Stop CPR. Assess rhythm.**
   - AED: **No shock advised.**
   - Manual monitor-defibrillator: Organized rhythm (PEA). **Do not defibrillate.**
7. **If PEA persists, immediately resume CPR,** beginning with compressions.
   - Provide 5 cycles (2 min) of uninterrupted CPR.
   - Insert an advanced airway (ET tube, LMA, King LT, or Combitube) if basic airway management is inadequate.
     - Confirm correct tube placement without interrupting CPR.
     - After correct placement is confirmed, deliver uninterrupted chest compressions, at least 100/min for 2 min. Deliver 8–10 breaths/min at a rate of 1 breath every 6–8 sec.
8. **Drugs**
   - Administer **epinephrine** 1 mg.
     - 10 mL of 1:10,000 IV/IO.
     - Follow with 20 mL IV flush.
     - Repeat every 3–5 min as needed.
   - A single dose of **vasopressin** 40 units IV/IO can replace the first or second dose of epinephrine.
   - If no IV/IO access is available and the patient has an ET in place, stop compressions and inject 2.0–2.5 mg (1:1,000) epinephrine diluted in 5–10 mL normal saline or sterile water directly into the ET every 3–5 min until IV/IO access is available. Follow ET drug administration with 5 consecutive ventilations to disperse drug into small airways for absorption into pulmonary vasculature and resume compressions.
9. **Continue CPR, check rhythm every 2 min.**
   - If PEA persists, immediately resume CPR; check rhythm every 2 min.
   - Administer **epinephrine** every 3–5 min.
10. During CPR, consider and treat potentially reversible causes of PEA (Hs and Ts):
    - Hypokalemia/hyperkalemia
    - Hypovolemia
    - Hypoxia
    - Hypothermia
    - Hydrogen ion (acidosis)
    - Tension pneumothorax
    - Thrombosis (pulmonary or coronary)
    - Tamponade, cardiac
    - Toxins
11. If rhythm changes to VF, pulseless VT, or asystole; follow algorithm for VF, pulseless VT, or asystole.

12. If rhythm converts to a stable ECG rhythm with ROSC:
    - Monitor and reevaluate the patient.
    - Arrange for transport to a critical care unit. The patient will need a comprehensive care plan. (See the Immediate Post–Cardiac Arrest Care algorithm.)

♥ **Clinical Tip:** PEA is frequently caused by reversible conditions (Hs and Ts) and may be treated successfully if those conditions are identified and corrected early.

♥ **Clinical Tip:** Atropine is no longer recommended for bradycardic PEA.

Cardiac Arrest: Asystole

Clinical Presentation

- Unresponsive state
- No respirations or only agonal respirations
- No pulse
- Flat ECG tracing or only agonal rhythm; no electrical activity on monitor

Management

1. **Establish unresponsiveness:**
    - No respirations or only agonal respirations and no pulse
2. **Call for help.**
3. **C–A–B: Compressions, airway, breathing:**
    - Begin CPR, starting with compressions.
    - Provide oxygen.
4. **Attach AED or manual monitor-defibrillator** as soon as available without interrupting compressions.
    - When device is attached, stop CPR to assess rhythm.
        - AED: **No shock advised.**
        - Manual monitor-defibrillator: No electrical activity (flat line or only agonal rhythm). **Do not defibrillate.**
5. **Immediately resume CPR,** beginning with compressions.
    - Provide 5 cycles (2 min) of uninterrupted CPR.
    - During CPR, establish IV or IO access.
    - Prepare vasopressor dose (epinephrine or vasopressin).
6. **Stop CPR. Assess rhythm.**
    - AED: **No shock advised.**
    - Manual monitor-defibrillator: No electrical activity. **Do not defibrillate.**
7. **If asystole persists, immediately resume CPR,** beginning with compressions.
    - Provide 5 cycles (2 min) of uninterrupted CPR.
    - Insert an advanced airway (ET, LMA, King LT, or Combitube) if basic airway management is inadequate.

- Confirm correct tube placement without interrupting CPR.
- After correct placement is confirmed, deliver uninterrupted compressions, at least 100/min for 2 min, and deliver 8–10 breaths/min at a rate of 1 breath every 6–8 sec.

8. **Drugs**
   - Administer **epinephrine** 1 mg.
     - 10 mL of 1:10,000 IV/IO.
     - Follow with 20 mL IV flush.
     - Repeat every 3–5 min as needed.
   - A single dose of **vasopressin**, 40 units IV/IO, can replace the first or second dose of epinephrine.
   - If no IV/IO access is available and the patient has an ET in place, stop compressions and inject 2.0–2.5 mg (1:1,000) epinephrine diluted in 5–10 mL normal saline or sterile water directly into the ET every 3–5 min until IV/IO access is available. Follow ET drug administration with 5 consecutive ventilations to disperse drug into small airways and resume compressions.
9. Continue CPR, checking rhythm every 2 min.
   - If asystole persists, immediately resume CPR, check rhythm every 2 min.
   - Administer **epinephrine** every 3–5 min.
10. During CPR, consider and treat potentially reversible causes of asystole (Hs and Ts):
    - Hypokalemia/hyperkalemia
    - Hypovolemia
    - Hypoxia
    - Hypothermia
    - Hydrogen ion (acidosis)
    - Tension pneumothorax
    - Thrombosis (pulmonary or coronary)
    - Tamponade, cardiac
    - Toxins
11. If asystole persists, consider whether proper resuscitation protocols were followed and reversible causes identified. If procedures were performed correctly, follow local criteria for terminating resuscitation efforts.
12. If rhythm changes to VF or pulseless VT, follow algorithm for VF or pulseless VT.
13. If rhythm changes to an organized rhythm with no pulse, follow algorithm for PEA.
14. If rhythm converts to a stable ECG rhythm with ROSC:
    - Monitor and reevaluate the patient.
    - Arrange for transport to a critical care unit. The patient will need a comprehensive care plan. (See the Immediate Post–Cardiac Arrest Care algorithm.)

Clinical Tip: Transcutaneous pacing is not recommended for asystolic cardiac arrest.

Clinical Tip: Atropine is no longer recommended for asystole.

Clinical Tip: Study local policy to learn established criteria for stopping resuscitation efforts.

Bradycardia With a Pulse

Clinical Presentation

- Heart rate <50 bpm in symptomatic patient
- ECG tracing shows sinus bradycardia, junctional escape rhythm, or AV block
- Chest discomfort/pain, lightheadedness, dizziness, dyspnea, tachypnea, presyncope, syncope
- Hypoxemia, hypotension, diaphoresis, altered mental status, congestive heart failure (CHF), shock
- Medication history: rate-slowing/AV nodal–blocking agents (e.g., beta blockers, calcium channel blockers)
- Patient with permanent pacemaker: possible pacemaker failure

Management

1. Establish responsiveness.
2. Perform primary A–B–C–D survey.
   - Patent airway?
   - Breathing adequate?
     - Assess rate and depth of breathing.
     - Measure oxygen saturation.
     - Administer oxygen if oxygen saturation is <94%. Titrate to effect.
   - Circulation (perfusion) adequate?
     - Attach cardiac monitor to identify rhythm.
       - Obtain 12-lead ECG if available.
     - Measure pulse and BP.
     - Establish IV access.
   - Differential diagnosis?
     - Identify and treat causes of bradycardia.
3. **If patient is stable and asymptomatic with heart rate <50 bpm:**
   - Monitor and observe for any changes.
4. **Drugs**
   - If patient is symptomatic with signs of poor perfusion, initiate treatment:
     - Administer **atropine** 0.5 mg IV every 3–5 min, maximum total dose 3 mg.
       - In sinus bradycardia, junctional escape rhythm, or second-degree AV block Wenckebach/Mobitz type I, atropine is usually effective.
       - In second-degree Mobitz type II or third-degree AV block, atropine is likely to be ineffective; prepare for transcutaneous pacing (TCP).
5. **Transcutaneous pacing (TCP)**
   - If patient fails to respond to atropine, sedate patient and begin **TCP:**
     - TCP is a temporizing measure.
     - A conscious patient will require sedation and analgesia.

6. **Drugs**
   - If the patient is hypotensive with severe bradycardia despite administration of atropine, and TCP is unavailable or ineffective, initiate drug therapy:
     - Continuous **dopamine** infusion
       - Start at 2–10 mcg/kg/min (chronotropic or heart rate dose).
       - Mix 400 mg/250 mL in normal saline, lactated Ringer's solution, or D5W (1600 mcg/mL) or use commercially prepared premixed solution.
       - Titrate to patient response.
     - Alternative therapy: **Epinephrine** infusion, 2–10 mcg/min IV
       - Add 1 mg of 1:1,000 in 500 mL normal saline and infuse at 1–5 mL/min.
7. Obtain expert consultation.

Clinical Tip: If the patient is unstable, do not delay TCP while waiting for IV access or for atropine to take effect.

Clinical Tip: Use atropine with caution in a suspected acute MI; atropine may lead to rate-induced ischemia.

Tachycardia With a Pulse (Unstable)

Clinical Presentation

- Altered level of consciousness (LOC)
- Shortness of breath, diaphoresis, weakness, fatigue, syncope or presyncope, chest discomfort or pain, palpitations
- Hypotension, shock, CHF, ischemic ECG changes, poor peripheral perfusion
- Heart rate typically ≥150 bpm

Management

1. **Establish responsiveness.**
2. **Perform rapid primary A–B–C–D survey.**
   - Patent airway?
   - Breathing adequate?
     - Assess rate and depth of breathing.
     - Measure oxygen saturation.
     - Administer oxygen if oxygen saturation is <94%. Titrate to effect.
   - Circulation (perfusion) adequate?
     - Attach cardiac monitor to identify rhythm.
       - Obtain 12-lead ECG if available, but do not delay cardioversion if patient is very unstable.
     - Measure pulse and BP.
     - Establish IV access.

- Differential diagnosis?
  - Consider causes of unstable tachycardia.

3. **Establish that serious signs and symptoms are related to the tachycardia.**
   - If patient is **unstable with serious signs and symptoms** and a heart rate ☒150 bpm, prepare for immediate synchronized cardioversion.
   - Patients in good cardiac health are likely to be stable if the ventricular rate is <150 bpm; however, patients with cardiac disease may be unstable with heart rates <150 bpm.
   - It is important to assess the patient's symptoms in addition to monitoring heart rate as criteria for cardioversion.
4. **Cardioversion**
   - Premedicate with sedative plus analgesic whenever possible.
   - Place defibrillator in synchronized (Sync) mode.
   - Perform **synchronized cardioversion:**
     - **Regular narrow–QRS complex tachycardia** (SVT or atrial flutter) generally requires less energy; 50–100 J biphasic is often sufficient. For a monophasic device, initial energy is 200 J.
     - **A-fib (irregular narrow-complex tachycardia)** requires an initial energy dose of 120–200 J biphasic or 200 J monophasic.
     - **Regular wide–QRS complex tachycardia** (monomorphic VT) usually responds well to initial energy of 100 J biphasic or monophasic.
5. **Check monitor. If tachycardia persists:**
   - Reset Sync mode on monitor-defibrillator.
   - Ensure adequate sedation/analgesia.
   - Increase energy and repeat cardioversion.
   - Reassess monitor and patient.
6. **Drugs**
   - For **a regular narrow-complex reentry tachycardia** in an unstable patient, if cardioversion is not immediately available, consider administering **adenosine** before cardioversion.
     - Give **adenosine** 6 mg IV in the antecubital or another large vein rapidly over 1–3 sec, immediately followed by a 20-mL bolus of normal saline.
     - If the rhythm has not converted in 1–2 min, repeat **adenosine** at 12 mg IV.
     - If the rhythm still does not convert, a third dose of **adenosine** at 12 mg IV may be given after another 1–2 min, maximum 30 mg.
7. If pulseless arrest develops, identify rhythm and follow algorithm for VF/VT, PEA, or asystole.

♥ **Clinical Tip:** Reactivate the "Sync" mode before each attempted cardioversion. Observe marker on QRS complex.

♥ **Clinical Tip:** The "Sync" mode delivers energy synchronized with the timing of the QRS complex to avoid stimulation during the refractory, or vulnerable, period of the cardiac cycle, when a shock could produce VF.

♥ **Clinical Tip:** If initial cardioversion shock is ineffective, it is reasonable to increase the shock energy in steps.

♥ **Clinical Tip: Do not perform synchronized cardioversion to treat VF.** Because there is no QRS complex to detect and the device may not reliably sense any waveform, a shock may not be delivered. Synchronized cardioversion should also **not** be used for pulseless VT or polymorphic VT (irregular VT). These rhythms require high-energy unsynchronized shocks (i.e., defibrillation doses).

Tachycardia With a Pulse: Narrow Complex (Stable)

Clinical Presentation

- No *serious* signs or symptoms related to the tachycardia
- Regular or irregular ECG rhythm
- QRS narrow (<0.12 sec)
- Heart rate typically ☒150 bpm

Management

1. **Establish responsiveness.**
2. **Perform rapid primary A–B–C–D survey.**
   - Patent airway?
   - Breathing adequate?
     - Assess rate and depth of breathing.
     - Measure oxygen saturation.
     - Administer oxygen if oxygen saturation is <94%. Titrate to effect.
   - Circulation (perfusion) adequate?
     - Attach cardiac monitor to identify rhythm.
       - Obtain 12-lead ECG if available.
     - Measure pulse, blood pressure.
     - Establish IV access.
   - Differential diagnosis?
     - While treating patient, consider causes of stable tachycardia.
3. **Establish that patient is asymptomatic, or stable with minor symptoms.**
4. **Vagal Maneuvers**
   - **QRS <0.12 sec:**
   - Attempt **vagal maneuvers.**
     - Hold ice to patient's face.
     - Have patient hold breath while bearing down.
     - Have patient blow through an obstructed straw
     - Perform carotid sinus massage (only by a qualified physician).

5. **Drugs**
   - **QRS <0.12 sec and tachycardia persists:**
     - Administer **adenosine** 6 mg IV over 1–3 sec in antecubital or another large vein.
       - Immediately follow with 20-mL bolus of normal saline.
     - If rhythm converts, it was probably SVT.
     - If rhythm remains regular and has not converted in 1–2 min, repeat **adenosine** at 12 mg IV.
     - If rhythm still does not convert, a third dose of **adenosine** at 12 mg IV may be given after another 1–2 min, maximum 30 mg.
     - If rhythm still does not convert or is irregular, it may be A-flutter, atrial tachycardia, MAT, junctional tachycardia, or A-fib.
       - Consider rate control using IV **diltiazem** or IV **beta blockers**.
6. Obtain expert consultation.
7. If the rhythm converts, observe the patient and treat any recurrence with adenosine, diltiazem, or beta blockers. Obtain expert consultation.

♥ **Clinical Tip:** If the rhythm is thought to be sinus tachycardia, identify and treat the cause. **Sinus tachycardia is NOT an arrhythmia.**

♥ **Clinical Tip:** If the patient's condition becomes unstable during tachycardia, administer sedation or analgesia if possible and perform immediate synchronized cardioversion.

♥ **Clinical Tip:** Use beta blockers with caution in patients with decompensated CHF. Avoid use in patients with bronchospastic disease.

Tachycardia With a Pulse: Wide Complex (Stable)

Clinical Presentation

- No *serious* signs and symptoms related to the tachycardia
- Regular ECG rhythm
- Wide QRS (≥0.12 sec)
- Heart rate typically ≥150 bpm

Management

1. **Establish responsiveness.**
2. **Perform rapid primary A–B–C–D survey.**
   - Patent airway?
   - Breathing adequate?
     - Assess rate and depth of breathing.
     - Measure oxygen saturation.
     - Administer oxygen if oxygen saturation is <94%. Titrate to effect.

- Circulation (perfusion) adequate?
  - Attach cardiac monitor to identify rhythm.
    - Obtain 12-lead ECG if available.
  - Measure pulse, blood pressure.
  - Establish IV access.
- Differential diagnosis?
  - While treating patient, consider causes of stable tachycardia.

3. **Establish that patient is asymptomatic, or stable with minor symptoms.**
4. **Drugs**

- **QRS ≥0.12 sec:**
  - If monomorphic VT, administer **amiodarone** 150 mg IV/IO over 10 min.
    - May repeat every 10 min if needed.
    - Start infusion at 1 mg/min for 6 hr, followed by 0.5 mg/min for 18 hr. Do not exceed 2.2 g in 24 hr.
  - If regular, monomorphic, and suspected to be SVT with aberrancy, administer **adenosine** 6 mg IV over 1–3 sec in the antecubital or another large vein.
    - Immediately follow with 20-mL bolus of normal saline.
    - If rhythm transiently slows or converts to sinus rhythm, it probably was SVT.
    - If adenosine has no effect, rhythm is probably monomorphic VT or atrial fibrillation with preexcitation and should be treated with amiodarone.

5. Obtain expert consultation.

♥ **Clinical Tip:** If pulseless arrest develops, identify arrhythmia and follow algorithm for cardiac arrest.

♥ **Clinical Tip:** If unable to administer amiodarone for wide-complex stable tachycardia, consider alternative antiarrhythmic infusions, including procainamide or IV sotalol.

Immediate Post–Cardiac Arrest Care

Patients who experience **ROSC** following resuscitation measures need a comprehensive care plan. A structured, multidisciplinary approach is needed to achieve and maintain hemodynamic stability and intact neurological function.

After ROSC:

1. Maintain **oxygen saturation ≥94%–99%** for optimal oxygenation.
   - Avoid hyperoxygenation and oxygen toxicity.
2. Unless awake and alert, the patient may require an **advanced airway and monitoring with waveform capnography.**
   - Avoid hyperventilation.
   - Target end-tidal CO_2 ($PETCO_2$) should be 35–40 mm Hg, or $PaCO_2$ 40–45 mm Hg.

3. **Monitor vital signs.**
   - Systolic BP should be maintained above 90 mm Hg, mean arterial pressure ≥65 mm Hg to optimize BP, cardiac output, and perfusion.
   - **Treat hypotension.**
     - **Fluids**
       - If systolic BP is <90 mm Hg, administer a 1–2 L fluid bolus of **normal saline or lactated Ringer's solution.**
     - **Drugs**
       - If necessary, the patient may be treated with a vasopressor infusion: **epinephrine, dopamine, or norepinephrine.**
       - **Epinephrine:** IV infusion of 0.1–0.5 mcg/kg/min (in 70 kg adult, at 7–35 mcg/min)
       - **Dopamine:** IV infusion of 5–15 mcg/kg/min (in a 70-kg adult, at 350–1050 mcg/min)
       - **Norepinephrine:** IV infusion of 0.1–0.5 mcg/kg/min (in 70 kg adult, at 7–35 mcg/min)
4. Consider and treat potentially reversible causes of cardiac arrest (Hs and Ts):
   - Hypovolemia
   - Hypoxia
   - Hydrogen ion (acidosis)
   - Hypo-/hyperkalemia
   - Hypothermia
   - Tension pneumothorax
   - Tamponade, cardiac
   - Toxins
   - Thrombosis, pulmonary
   - Thrombosis, coronary
5. Obtain a 12-lead ECG as soon as possible.
6. If the patient remains unresponsive or comatose or cannot follow commands:
   - Initiate therapeutic hypothermia, cooling to a core temperature of 32°–34°C for 12–24 hr.
7. If the patient rules in (positive cardiac markers, ECG changes) for a STEMI or if there is high suspicion for an acute MI:
   - Arrange for prompt transport to a cardiac catheterization laboratory for coronary reperfusion with possible PCI, maintaining hypothermia (if indicated).
8. Maintain glycemic control.
   - Maintain blood glucose 144–180 mg/dL post-arrest.
   - Avoid hypoglycemia.
9. All cardiac arrest survivors need advanced critical care and a comprehensive care plan to optimize neurological, cardiopulmonary, and metabolic function.
   - Set a goal of return to pre-arrest functional status.
   - If patient requires continued advanced life support, use appropriate tools to establish prognosis before deciding to withdraw life support.

Suspected Stroke

For patients presenting with an **ischemic stroke,** the goal of care is rapid cerebral reperfusion to prevent permanent neurological deficits. For patients presenting with a **hemorrhagic stroke**, the focus is on stabilizing the patient to control bleeding and on considering advanced interventions. The importance of transporting patients to specialized stroke centers cannot be overemphasized.

Clinical Presentation

- Facial droop, arm or leg weakness/numbness, especially unilateral
- Difficulty speaking or understanding, confusion
- Sudden severe headache, visual disturbances, dizziness, loss of balance or coordination

Management

Rapid recognition of stroke symptoms, immediate activation of EMS, and rapid dispatch are critical.

EMS Response

1. Support airway, breathing, and circulation.
   - Administer oxygen if needed.
2. Perform prehospital stroke assessment.
   - Use Cincinnati Prehospital Stroke Scale.
3. Establish time when patient was last known to be normal or time of symptom onset if known.
4. Arrange rapid triage and transport to a center capable of providing acute stroke care if available.
5. Alert hospital.
   - Be sure hospital CT scan is functional.
6. Check patient's glucose level.

Emergency Department/Hospital

The following should be performed within 60 min after patient has arrived at the emergency department:

Immediate general assessment and stabilization within first 10 min of arrival:

1. Assess airway, breathing, circulation, and vital signs.
2. Administer oxygen if patient is hypoxemic.
3. Obtain IV access and blood samples.
4. Check glucose level.
   - Treat if indicated.
5. Perform initial neurological screening.
6. Activate stroke team.

7. Order emergent noncontrast CT scan or MRI of brain.
   - This is the most important test for a patient with a suspected stroke.
8. Obtain 12-lead ECG.

Immediate neurological assessment by stroke team or designee within first 25 min of arrival:

1. Review patient's history and perform general physical examination.
2. Establish time of symptom onset or last time patient was known to be normal.
3. Perform a neurological examination.
   - Use a stroke or neurological scale, such as the National Institutes of Health Stroke Scale (NIHSS) or the Canadian Neurological Scale.
4. Interpret CT scan within 45 min of arrival.
   - Does CT scan show hemorrhage?

If the CT scan does not show hemorrhage, perform the following within the first 45 min of arrival:

1. Review fibrinolytic inclusion, exclusion, and relative exclusion criteria.
2. Repeat neurological examination to determine whether patient's symptoms are improving/resolving.
3. If patient is a candidate for fibrinolytic therapy:
   - Review risks and benefits of fibrinolytic therapy with patient/family.
   - If patient/family agree, give rtPA within 60 min of arrival.
   - Do not give anticoagulants or antiplatelet treatment for 24 hr.
   - Begin post-rtPA stroke pathway.
   - Admit to stroke unit or intensive care unit.
   - Initiate supportive therapy.
   - Treat comorbidities.
4. If patient is not a candidate for fibrinolytic therapy:
   - Administer aspirin (orally if the patient can swallow, or rectally if the patient has difficulty swallowing).
   - Begin stroke pathway.
   - Admit to stroke unit or intensive care unit.
   - Initiate supportive therapy.
   - Treat comorbidities.

If the CT scan shows hemorrhage, perform the following within the first 45 min of arrival:

1. No aspirin, anticoagulation, or fibrinolytic therapy.
2. Consult neurologist or neurosurgeon.
   - Consider transfer if such expertise is not available.
3. Initiate supportive therapy.
4. Begin stroke pathway within 60 min of arrival.
5. Admit to stroke unit or intensive care unit within 3 hr of arrival.
6. Treat comorbidities.

83

General Stroke Care Pathway

1. Begin stroke pathway.
2. Continue to support airway, breathing, circulation.
   - Maintain oxygen saturation 94%–99%.
3. Maintain cardiac monitoring for first 24 hr or longer if indicated.
4. Avoid intravenous D5W or excessive fluid loading.
5. Monitor BP.
   - Manage hypertension if systolic BP is >220 mm Hg or diastolic BP is >120 mm Hg.
6. Monitor blood glucose.
   - Treat hyperglycemia.
7. Monitor temperature.
   - Treat fever with acetaminophen.
8. Perform dysphagia screening/swallow evaluation.
9. Monitor for complications of stroke and fibrinolytic therapy (if administered).

Cincinnati Prehospital Stroke Scale

Facial Droop: Have patient show teeth or smile:
- **Normal**—Both sides of face move equally well.
- **Abnormal**—One side of face does not move as well as the other side.

Arm Drift: Have patient close eyes and hold both arms straight out with palms up for 10 sec:
- **Normal**—Both arms move the same or do not move at all.
- **Abnormal**—One arm does not move or drifts down lower than the other.

Speech: Have patient say "You can't teach an old dog new tricks":
- **Normal**—Patient uses correct words with no slurring.
- **Abnormal**—Patient slurs words, uses inappropriate words, or is unable to speak.

Note: The presence of a single abnormality has a sensitivity of 59% and a specificity of 89% when scored by prehospital providers.

Glasgow Coma Scale

Observation	Response	Score
Eye response	• Opens spontaneously	4
	• Opens to verbal commands	3
	• Opens to pain	2
	• No response	1
Best verbal response	• Alert and oriented	5
	• Disoriented but converses	4
	• Uses inappropriate words	3
	• Makes incomprehensible sounds	2
	• No response	1
Best motor response	• Reacts to verbal commands	6
	• Reacts to localized pain	5
	• Withdraws from pain	4
	• Abnormal flexion	3
	• Abnormal extension	2
	• No response	1
Total score	Normal	15

Score can range from 3 (lowest neurological function) to 15 (highest function).
Score 14–15: Mild dysfunction
Score 11–13: Moderate to severe dysfunction
Score ⊠10: Severe dysfunction

Acute Coronary Syndrome

The goal of therapy for patients presenting with acute coronary syndrome (ACS) is myocardial salvage: limiting the amount of myocardial necrosis, preserving left ventricular function, preventing heart failure, and limiting other cardiovascular complications.

Clinical Presentation

- History of coronary artery disease, angina, or MI (or this may be a first event)

- Chest pain or discomfort
- Pain radiating to neck, jaw, shoulders, arms, or upper back
- Sudden unexplained shortness of breath, weakness, or fatigue with or without chest pain/discomfort
- Possible associated nausea, vomiting, diaphoresis, lightheadedness, presyncope or syncope

Management

Rapid recognition of potential ACS and prompt activation of EMS for rapid diagnosis and treatment are critical.

EMS Response

1. **Perform primary ABCD survey; support ABCs.**
2. Measure vital signs, including oxygen saturation.
   - Administer oxygen if oxygen saturation is <94%. Titrate to effect.
3. **Drugs**
   - Administer **aspirin** 160–325 mg PO if no history of true aspirin allergy.
     - Have patient chew 4 tablets non–enteric-coated aspirin 81 mg for more rapid antiplatelet effect.
     - Give within minutes of symptom onset.
4. Attach cardiac monitor.
5. Transport patient quickly to closest facility with PCI capabilities if possible.
6. Start an IV if possible.
   - Do not delay patient transport.
7. Obtain a 12-lead ECG.
   - Transmit ECG to receiving hospital if possible.
   - If 12-lead ECG shows STEMI:
     - Notify attending physician immediately.
     - Begin fibrinolytic checklist if PCI not readily available.
     - Hospital should mobilize cardiac catheterization team for rapid response.
8. **Drugs**
   - Administer sublingual **nitroglycerin**:
     - **Tablets:** 0.3–0.4 mg:
       - Repeat every 3–5 min if needed until chest pain is relieved.
       - Do not exceed three doses over a 15–min interval.
     - **Aerosol spray:** Administer spray for 0.5–1.0 sec (provides 0.4 mg/dose).
       - Repeat every 3–5 min if needed until chest pain is relieved.
       - Do not exceed 3 sprays in 15 min.
     - **Nitroglycerin administration requires a systolic BP ≥90 mm Hg.**
   - Administer **morphine** if chest pain is not relieved by nitroglycerin:
     - Administer morphine 2–4 mg IV (over 1–5 min).
     - If symptoms persist, administer 2–8 mg every 5–15 min if patient is hemodynamically stable.
     - Do not administer morphine if systolic BP is <90 mm Hg.

Emergency Department/Hospital: Perform Initial Assessment Within 10 Min

1. Vital signs, oxygen saturation
2. 12-lead ECG if not already obtained
3. IV access if not already obtained
4. Brief targeted history and physical examination
5. Initial laboratory studies including cardiac marker levels, electrolytes, and coagulation studies
6. Complete fibrinolytic checklist if appropriate (non–PCI-capable facilities).
7. Portable chest x-ray (within 30 min)
8. Immediate general treatment:
   - Oxygen if saturation <94%. Start oxygen at 4 L/min, titrate to effect.
   - Aspirin 160–325 mg, if not already administered
   - Nitroglycerin if indicated
   - Morphine if chest pain not relieved by nitroglycerin
9. STEMI, or new or presumably new left bundle branch block (LBBB) strongly suspicious for myocardial injury:
   - Start adjunctive medical therapies:
     - Use beta blockers in a hemodynamically stable patient.
     - Use IV or transdermal nitroglycerin for ongoing angina in a hemodynamically stable patient.
     - Antiplatelet therapy:
       - Clopidogrel, prasugrel, ticagrelor
       - Glycoprotein IIb/IIIa inhibitors
     - Anticoagulation therapy:
       - Unfractionated heparin
       - Low molecular weight heparin
       - Direct thrombin inhibitor: bivalirudin
       - Factor Xa inhibitor: fondaparinux (for patients with history of heparin-induced thrombocytopenia)
     - Start statin drug therapy for plaque stabilization and cholesterol management unless contraindicated.
   - Reperfusion therapy:
     - Patients with STEMI should be taken emergently for PCI if time of symptom onset is ⊠12 hr.
       - Door-to-balloon inflation goal is 90 min or less.
     - Fibrinolytic therapy if PCI not available if patient is appropriate candidate.
       - Door-to-needle goal is 30 min or less.
10. Patients with NSTEMI, unstable angina, or late-presentation MI (⊠12 hr) should be taken urgently for PCI if they:
    - Have refractory ischemic chest discomfort.
    - Have recurrent or persistent ST deviation.
    - Develop significant arrhythmia: VT or VF.

- Have signs of heart failure.
- Are hemodynamically unstable.
- Start adjunctive medical therapies, as above.

11. Patients with normal ECG or nondiagnostic ST–T wave changes on repeat ECGs in whom MI has been ruled out with serial negative troponin levels:
    - Should undergo noninvasive diagnostic stress testing for an ischemic evaluation.
    - If positive for ischemia, should initiate adjunctive medical therapies and consider diagnostic catheterization and possible PCI if indicated.
    - If negative for ischemia, may be discharged with appropriate follow-up.
12. Admit patient to monitored bed.
13. Perform an echocardiogram in patients in whom MI has been ruled in to evaluate left ventricular function and valve function.
14. Continue adjunctive drug therapy and start an ACE inhibitor or angiotensin-receptor blocker if patient has left ventricular dysfunction and is hemodynamically stable.

♥ **Clinical Tip:** Diabetic patients, elderly patients, and women frequently present with atypical symptoms (e.g., weakness, fatigue, complaints of indigestion, shortness of breath).

♥ **Clinical Tip:** GERD or history of GI bleeding with aspirin is an aspirin intolerance, not an allergy. These patients should receive aspirin. Only patients with true aspirin allergy (severe rash, anaphylaxis) should not receive aspirin.

♥ **Clinical Tip:** Do not delay rapid transport to the nearest facility with PCI capabilities. Consider fibrinolytic therapy within 30 min for patients with STEMI if transport to such a facility will be ⊠30 min.

♥ **Clinical Tip:** Patients should not be given nitroglycerin if they have taken sildenafil (**Viagra**) or vardenafil (**Levitra**) in the last 24 hr or tadalafil (**Cialis**) within 48 hr. The use of nitroglycerin with these medications may cause irreversible hypotension.

♥ **Clinical Tip:** Nitroglycerin should be used with caution in patients with an inferior MI with possible right ventricular involvement. It is contraindicated with right ventricular MI, tachycardia in absence of heart failure, and bradycardia <50 bpm.

Tab 4: PALS

Perhaps nothing is more frightening than dealing with an infant or child whose respiratory or circulatory system is compromised, or who presents in cardiopulmonary arrest. A systematic approach to assessment is essential. Just as important are knowledge of medication and pediatric dosages and the correct performance of skills in basic and advanced pediatric life support.

Systematic Approach

The goal of a systematic approach is to quickly recognize signs and symptoms of respiratory compromise or failure and circulatory compromise or shock. If not treated, these can lead to cardiopulmonary failure or arrest. **Most pediatric arrests are caused by respiratory failure, shock, or a combination of both.** Occasionally the cause is a lethal ventricular arrhythmia. Rapid intervention is key to improving survival rates.

Initial Observation: Use of Your Vision and Hearing

- **Level of consciousness:** Is the patient awake and alert, restless/irritable, lethargic, obtunded, or unresponsive?
- **Breathing:** Is breathing normal, labored, slow or rapid, deep or shallow, irregular? Is the patient using accessory muscles? Do you see sternal retractions? Is breathing agonal or apneic?
- **Skin color:** Is the skin normal, pale, flushed, or cyanotic?

If the patient is unresponsive and apneic: shout for help, activate the emergency response system, and initiate the infant/child CPR algorithm in Tab 2: CPR.

Gather Information

- **Make sure the scene is safe** before initiating patient assessment.
- **Evaluate:** Initiate primary and secondary assessment, use appropriate diagnostic studies.
- **Identify:** Identify the type of problem (respiratory, circulatory, or both) and the severity (respiratory distress or failure, compensated or hypotensive shock, cardiopulmonary failure or arrest).
- **Intervene:** Select appropriate interventions within your scope of practice.

Primary Patient Assessment

Airway

Is the airway patent? Is the chest or abdomen moving? Are breath sounds audible? Can airway patency be maintained by head tilt–chin lift maneuver? Does the airway need to be suctioned? Is intubation needed?

Breathing

Assess respiratory rate, depth, and effort; breath sounds; and oxygen saturation.

- **Abnormal breathing:** Decreasing respiratory rate is associated with worsening clinical condition.
  - **Tachypnea:** Excessive respiratory rate (relative to age) may be the first sign of respiratory distress, or may be a physiological response to distress or serious illness (e.g., pain, high fever, severe anemia, infection/sepsis, early CHF, cyanotic congenital heart defects, dehydration, shock, DKA).
  - **Bradypnea:** Slow respiratory rate (relative to age) is often irregular and associated with respiratory muscle fatigue, CNS infection or injury, hypothermia, or drugs that suppress the respiratory drive.
  - **Apnea:** Breathing is absent for ⊠20 sec, or <20 sec but associated with bradycardia, pallor, and cyanosis.
- **Respiratory effort:** Increased work of breathing may be caused by conditions that increase airflow resistance (e.g., asthma, bronchiolitis), restrictive lung disease (e.g., pneumonia, pneumothorax, pleural effusion, pulmonary edema), or extrapulmonary conditions (e.g., severe metabolic acidosis as with salicylate poisoning or DKA, inborn errors of metabolism). Clinical presentation is marked by:
  - Use of accessory muscles
  - Prolonged inspiration or expiration
  - Open-mouth breathing
  - Nasal flaring: dilatation of nostrils during inspiration.
  - Retractions: inward movement of tissues, neck, sternum, chest wall, abdomen below sternum; associated with upper or lower airway obstruction and lung tissue disease.
  - Head bobbing: Use of neck muscles during breathing, marked by chin lift and neck extension during inspiration and chin falling forward during expiration. Possible sign of respiratory failure.
  - Seesaw respirations: Chest retraction with abdominal expansion during inspiration, chest expansion with abdominal retraction during expiration, leading to fatigue. Associated with conditions such as upper airway obstruction, severe lower airway obstruction, interstitial pulmonary disease, neuromuscular disease.
  - Decreased or asymmetrical chest wall expansion; associated with excessive fatigue, airway obstruction, restrictive lung disease, aspiration, mucus plug.
  - Decreased air movement during auscultation of breath sounds.
- **Abnormal airway and lung sounds:** Respirations are markedly noisy and may include:
  - **Grunting:** Low-pitched, short expiratory sound, associated with interstitial pulmonary disease, lung contusion, acute respiratory distress

syndrome (ARDS), pulmonary edema; may indicate pain associated with appendicitis, peritonitis, bowel obstruction, perforated bowel.
- **Stridor:** High-pitched, coarse inspiratory and possibly expiratory sound, associated with upper airway obstruction. Causes include foreign body airway obstruction, infection, upper airway edema, congenital or acquired airway abnormality.
- **Wheezing:** High- or low-pitched whistling or sighing expiratory and possibly inspiratory sound, associated with lower airway obstruction caused by asthma, bronchiolitis. Localized wheezing may indicate partial obstruction of trachea or upper airway.
- **Crackles:** Sharp, discrete crackling or bubbling inspiratory sounds. Dry crackles may indicate atelectasis or interstitial pulmonary disease. Moist crackles indicate alveolar fluid and are associated with pneumonia, pulmonary edema, or interstitial pulmonary disease.

- **Oxygen saturation:** Evaluate by pulse oximetry, preferably continuously. Hypoxemia may precede bradycardia or cyanosis. Reading may be normal despite respiratory distress or failure during supplemental oxygen administration. Reading may be inaccurate in poor perfusion states. It does not indicate oxygen tissue delivery.

Normal Respiratory Rates By Age

Patient Age	Normal Respiratory Rate
Infant (<12 mo)	30–60 breaths/min
Toddler (1–3 yr)	24–40 breaths/min
Preschool (4–5 yr)	22–34 breaths/min
School age (6–12 yr)	18–30 breaths/min
Adolescence (13–18 yr)	12–16 breaths/min

Circulation

Assess heart rate and rhythm, peripheral and central pulses, capillary refill time, skin color and temperature, and BP. Adequate circulation is also assessed by LOC and urine output.

Normal Heart Rates By Age

Patient Age	Rate While Awake	Mean Rate	Rate While Asleep
Birth–3 mo	85–205 bpm	140 bpm	80–160 bpm
3 mo–2 yr	100–190 bpm	130 bpm	75–160 bpm
2–10 yr	60–140 bpm	80 bpm	60–90 bpm
>10 yr	60–100 bpm	75 bpm	50–90 bpm

- **Arrhythmia:** Abnormal heart rhythm is caused by conduction disturbance or abnormality in cardiac tissue, or may result from hypoxia or shock.
  - **Bradycardia:** Heart rate below normal for age. If associated with clinical signs of deterioration, may indicate impending cardiac arrest. Typically caused by hypoxia. Heart rate may improve with oxygen administration.
  - **Tachycardia:** Heart rate above normal for age. Sinus tachycardia is a compensatory response to the patient's condition. If the rhythm is not sinus, it may be a narrow-complex tachycardia (SVT), wide-complex tachycardia (VT), or SVT with bundle branch block.
- **Pulses:** Is the pulse normal, fast, or slow? Regular or irregular? Bounding or weak?
  - **Central pulses:** Femoral, brachial (infants), carotid (older children), axillary. Larger vessels, closer to the heart, usually have stronger pulses than peripheral vessels. Weak central pulses may indicate impending cardiac arrest.
  - **Peripheral pulses:** Radial, dorsalis pedis, posterior tibial. Weak pulses are associated with peripheral vasoconstriction as in shock.
  - **Beat-to-beat variation:** Associated with premature atrial and/or ventricular contractions.
  - **Fluctuations in pulse volume with respiratory cycle:** Pulsus paradoxus, associated with severe asthma or pericardial (cardiac) tamponade.
- **Capillary refill time:** Assessment of the time blood flow takes to return after pressure is applied to blanch tissues and then released, normally <2 sec. Capillary refill time indicates adequacy of tissue perfusion. Time increases with decreased perfusion, as in dehydration, hypothermia, shock with vasoconstriction. Prolonged capillary refill time is associated with low cardiac output (CO).

- **Skin color and temperature:** These should be normal and consistent over the torso and extremities, with pink mucous membranes, nail beds, palms, and soles of feet. Decreased perfusion is first indicated by changes in the hands and feet, progressing to the extremities and torso with worsening perfusion.
  - **Pallor:** Decreased perfusion associated with hypovolemic and cardiogenic shock or with anemia. Central pallor is noted in the lips, tongue, mucous membranes of the mouth, lining of the eyes.
  - **Mottling:** Irregular, patchy skin discoloration; may be associated with hypoxemia, hypovolemia, or shock.
  - **Cyanosis:** Bluish skin or mucous membrane discoloration, associated with circulation of deoxygenated blood.
  - **Acrocyanosis:** Cyanosis of hands and feet, common in newborns, normal.
  - **Peripheral cyanosis:** Cyanosis of hands and feet associated with poor tissue perfusion in shock, heart failure, peripheral arterial disease, or venous stasis.
  - **Central cyanosis:** Cyanosis of lips and mucous membranes when there is ⊠5 g/dL of unsaturated hemoglobin. It is more apparent when hypoxemia is associated with cyanotic congenital heart disease, such as intracardiac shunt or polycythemia. Patients with significant anemia with low hemoglobin may not have cyanosis. Other causes include alveolar hypoventilation with head injury or drug overdose; alveolar diffusion defect as in pneumonia; ventilation-perfusion defect as in asthma, bronchiolitis, or ARDS; altitude sickness with low ambient oxygen tension.
- **Blood pressure:** Normal values vary with age.

Normal Blood Pressures By Age

Age	Systolic BP		Diastolic BP	
	Male	Female	Male	Female
Neonate–1 day	60–74 mm Hg	60–76 mm Hg	30–44 mm Hg	31–45 mm Hg
Neonate–4 days	68–84 mm Hg	67–83 mm Hg	35–53 mm Hg	37–53 mm Hg
Infant–1 mo	74–94 mm Hg	73–91 mm Hg	37–55 mm Hg	36–56 mm Hg
Infant–3 mo	81–103 mm Hg	78–100 mm Hg	45–65 mm Hg	44–64 mm Hg
Infant–6 mo	87–105 mm Hg	82–102 mm Hg	48–68 mm Hg	46–66 mm Hg
Infant–12 mo	67–103 mm Hg	68–104 mm Hg	20–58 mm Hg	22–60 mm Hg
Child–2 yr	70–106 mm Hg	71–105 mm Hg	25–63 mm Hg	27–65 mm Hg
Child–7 yr	79–115 mm Hg	79–113 mm Hg	38–78 mm Hg	39–77 mm Hg
Adolescent–15 yr	95–131 mm Hg	93–127 mm Hg	45–85 mm Hg	47–85 mm Hg

- **Hypotension:** Systolic pressure below threshold for age. A drop in systolic pressure of ≥10 mm Hg from baseline is significant. Even a pressure in the low-normal range may be considered abnormal in seriously ill or injured infants or children. Hypotension signals a failure of compensation (except in vasodilatory or septic shock). When accompanied by bradycardia, it requires aggressive airway management with support of ventilation and oxygenation and IV fluid resuscitation. Quick guideline to determining hypotension by age:
  - **Newborns 0–28 days:** Systolic BP <60 mm Hg
  - **Infants 1–12 months:** Systolic BP <70 mm Hg
  - **Children 1–10 yr:** Systolic BP <70 + (age in years – 2)
  - **Children >10 yr:** Systolic BP <90 mm Hg

- **Disability:** Rapidly assess neurological function and adequacy of brain perfusion. Assess LOC, muscle tone, and pupil response. Evaluation methods include:
  - **AVPU Pediatric Scale:** Assesses LOC to evaluate cerebral cortex function:
    - **A**lert: Patient is awake, alert, active.
    - **V**oice: Patient responds only to voice.
    - **P**ain: Patient responds only to painful stimulus.
    - **U**nresponsive: Patient does not respond to any stimuli.
  - **Glasgow Coma Scale:** Assesses LOC and neurological status. Evaluates and scores best eye opening, best verbal response, and best motor response. Total score reflects severity of injury or illness. Use a modified scale for preverbal or nonverbal children.
  - **Pupil response to light:** Assesses brainstem function. Evaluate pupil size, equality, constriction to light.
- **Exposure:** Remove clothing, perform a focused physical examination. Assess core temperature for fever. Examine for signs of trauma, both accidental and nonaccidental: bleeding, wounds, burns, bruising, unusual markings, deformities. Look for petechiae, purpura, rashes. Palpate extremities for tenderness.
- If a life-threatening problem is identified, activate the emergency response system and initiate lifesaving interventions. Otherwise, proceed with the secondary patient assessment.

Secondary Patient Assessment

- **History:** Obtain a targeted history focused on the presenting signs and symptoms, using the **SAMPLE** mnemonic:
  - **S**igns and symptoms at onset of illness
  - **A**llergies to foods, medications, latex, materials
  - **M**edications including name, last dose, time of last dose

- **P**ast medical history including medical illness, injuries, surgeries, immunizations
- **L**ast meal including type, time, amount
- **E**vents preceding illness or injury, hazards present at scene, treatments administered, estimated time of arrival if prehospital.

- **Physical examination:** Perform a brief head-to-toe examination focused on the area of concern regarding illness or injury.
- **Diagnostic tests:** Diagnostic tests can identify and determine the presence and severity of respiratory and circulatory dysfunction. They may include:
  - **Arterial blood gas (ABG):** Assesses partial pressure of oxygen (PaO_2) and carbon dioxide ($PaCO_2$) in arterial blood, as well as the acid–base status (pH, bicarbonate [HCO_3^-]) concentration. Identifies respiratory failure relative to hypoxemia (oxygenation failure), hypercarbia (ventilation failure), or metabolic acid–base imbalances.
  - **Venous blood gas (VBG):** Assesses partial pressure of oxygen (PvO_2) and carbon dioxide ($PvCO_2$) and pH in venous blood. Should correlate results with ABG results if possible. A widening gradient between $PaCO_2$ and $PvCO_2$ indicates poor perfusion. A central venous sample is preferred over a peripheral sample.
  - **Hemoglobin concentration:** Evaluates oxygen-carrying capacity of blood; most oxygen in blood is bound to hemoglobin. SaO_2 measures the hemoglobin oxygen saturation.
  - **Central venous oxygen saturation:** Evaluates balance between oxygen delivery (from arterial sample) and tissue oxygen consumption. Measures oxygen reserve via oxygen saturation in the venous sample (SvO_2) just before pulmonary capillary gas exchange.
  - **Arterial lactate:** Increases in metabolic acidosis associated with tissue hypoxia and anaerobic metabolism (as in shock) and with stress hyperglycemia.
  - **Central venous pressure monitoring:** Assesses right ventricular preload to guide fluid management and vasoactive drug therapy. High pressures indicate poor myocardial contractility or cardiac compression, as in tension pneumothorax or cardiac tamponade. Other causes include excessive PEEP (positive end-expiratory pressure) with mechanical ventilation, severe pulmonary hypertension, and massive pulmonary embolism.
  - **Invasive arterial pressure monitoring:** Continuous monitoring of systemic arterial pressures. Waveform analysis can identify pulsus paradoxus, which may indicate cardiac tamponade.
  - **Chest x-ray:** Can identify upper and lower airway obstruction, lung disease, pleural effusions, pneumothorax, hemothorax, barotrauma caused by mechanical ventilation, interstitial pulmonary edema, cardiomegaly, pericardial effusion, depth of ET placement.
  - **12-lead ECG:** Assesses cardiac rhythm and rate, arrhythmias, electrical conduction disorders, myocardial ischemia or infarction, pericarditis.

- **Echocardiogram:** Sonogram of the heart assesses atrial and ventricular chamber size, wall thickness, wall motion, valve structure and function, congenital anomalies, abnormal masses, pericardial space and fluid.
- **Peak expiratory flow rate (PEFR):** Measures maximal flow rate during forced expiration; decreases with airway obstruction as in asthma.

Respiratory Arrest and Failure

Alterations in gas exchange caused by alveolar dysfunction or airway disease can impair oxygenation, ventilation, or both, causing respiratory problems. Respiratory distress can lead to respiratory failure and cardiac arrest. Early recognition and aggressive management are essential to preventing this progression.

Impairment of Oxygenation and Ventilation

Hypoxemia

Hypoxemia is low arterial oxygen content, identified by low PaO_2 and low SaO_2, which indicate inadequate oxygenation. Hypoxemia causes tissue hypoxia. Compensatory mechanisms to improve tissue hypoxia include increased respiratory rate and depth (hyperventilation) and tachycardia, which increases CO.

- **Signs of tissue hypoxia include:**
  - Tachycardia (early compensatory mechanism)
  - Tachypnea
  - Nasal flaring
  - Restlessness, agitation, irritability, anxiety
  - Pallor
  - Retractions
  - Decreased LOC, bradypnea, bradycardia (late signs)
- **Hypoxemia results from:**
  - **Alveolar hypoventilation** may be caused by CNS infection, traumatic brain injury, respiratory muscle weakness from neuromuscular disease, drug overdose, and apnea. It leads to hypercarbia, displacing alveolar oxygen and resulting in hypoxemia.
    - **Management:** Give supplemental oxygen and treat the underlying condition to restore normal ventilation.
  - **Diffusion defect** may be caused by pulmonary edema, interstitial pneumonia, alveolar proteinosis. Impaired oxygen diffusion leads to hypoxemia. Impaired carbon dioxide diffusion leads to hypercarbia.
    - **Management:** Give supplemental oxygen with CPAP, ventilate with an advanced airway and PEEP.

- **Ventilation-perfusion (V/Q) imbalance** may be caused by asthma, bronchiolitis, pneumonia, atelectasis, ARDS, foreign body obstruction. Blood flows through poorly ventilated areas of the lung, causing hypoxemia and possible hypercarbia.
  - **Management:** Support ventilation, increase mean airway pressure using an advanced airway with supplemental oxygen and PEEP.
- **Right-to-left shunt** may be caused by cyanotic congenital heart disease or extracardiac (anatomical) vascular shunt, as well as causes listed above for V/Q imbalances. Deoxygenated blood is shunted directly from the right to the left side of the heart, or from the pulmonary artery to the aorta. It mixes with oxygenated blood and lowers arterial blood oxygen, resulting in hypoxemia.
  - **Management:** The defect must be corrected. Supplemental oxygen is provided but is not adequate in itself.
- **Low atmospheric PO_2** is caused by high altitudes with decreased barometric pressure, resulting in hypoxemia.
  - **Management:** Give supplemental oxygen; bring the patient down if still at high altitude.

Hypercarbia

Hypercarbia is increased $PaCO_2$ caused by inadequate ventilation (decreased elimination of carbon dioxide), resulting in respiratory acidosis.

- Causes include airway disease, lung disease, and decreased respiratory effort or ventilatory drive due to narcotic overdose, CNS disease, or respiratory muscle weakness.
- Signs and symptoms of respiratory distress may include tachypnea, restlessness, agitation, anxiety, inadequate respiratory rate, nasal flaring, retractions, poor respiratory effort, and decreased LOC.
- Impaired ventilation may occur with normal oxygen saturation (no hypoxemia).

Identification of Respiratory Problems by Severity

Respiratory Distress

Respiratory distress may be mild to severe. Respiratory distress may lead to respiratory failure.

- **Clinical presentation:**
  - Tachypnea, tachycardia, nasal flaring, retractions
  - Pallor, cool skin
  - Hypoventilation, bradypnea, abnormal respiratory sounds
  - Change in LOC

Respiratory Failure

Respiratory failure is caused by inadequate oxygenation or ventilation and may occur without preceding respiratory distress. Rapid, aggressive intervention is needed to prevent cardiac arrest.

- **Clinical presentation**:
  - Initial marked tachypnea with increased respiratory effort and tachycardia
  - Bradypnea with decreased respiratory effort and bradycardia
  - Cyanosis
  - Decreased LOC
  - Lethargy, stupor, coma, apnea

♥ **Clinical Tip:** Signs of impending respiratory failure include tachypnea, nasal flaring, intercostal and sternal retractions, grunting, and seesaw breathing.

Causes and Management of Respiratory Problems

Rapid recognition and appropriate, aggressive management of respiratory problems in infants and children are essential to support and restore oxygenation and ventilation and prevent cardiac arrest.

- Perform rapid, focused evaluation to identify type and severity of respiratory problem.
- Initiate interventions to support and maintain oxygenation and ventilation.
  - **Airway**
    - Use the head tilt–chin lift maneuver or use jaw thrust without head tilt if cervical spine trauma is suspected.
    - Clear the airway if necessary: suction, remove foreign body if visible.
    - Insert an oropharyngeal or nasopharyngeal airway to maintain airway patency.
  - **Breathing**
    - Check SaO_2, administer supplemental oxygen.
    - Administer inhaled medications (inhalers, nebulizers) if indicated.
    - Use bag-mask ventilation if indicated.
    - Use an advanced airway (LMA, ET) if indicated.
  - **Circulation**
    - Monitor heart rate and rhythm, BP.
    - Establish vascular access.
    - Start chest compressions if indicated.
- **Identify cause of respiratory problem and perform specific interventions.**

Upper Airway Obstruction

An upper airway obstruction may be in the nose, pharynx, or larynx. It may be mild to severe. Causes include:

- Foreign-body obstruction
- Airway edema caused by anaphylaxis, tonsillar hypertrophy, croup, or epiglottitis

- Mass such as an abscess or tumor
- Thick secretions
- Congenital upper airway anomaly
- Airway trauma following intubation

- **Clinical presentation**:
  - Tachypnea, increased respiratory effort
  - Hoarseness, barking cough or cry, stridor
  - Decreased breath sounds, inadequate chest rise
- **Management:**
  - Proper head position to maintain airway patency or position of comfort to facilitate breathing
  - Removal of visible foreign body from airway
  - Suctioning of nose and mouth
  - Nebulized epinephrine; corticosteroids IV, IM, PO, or inhaled
  - Medications to reduce airway inflammation or edema
  - Airway adjunct or advanced airway
  - Management of restlessness, agitation, anxiety
  - Surgical airway if indicated (tracheostomy, cricothyroidotomy)
- Management of foreign-body airway obstruction (See Tab 2: CPR) may include:
  - Partial obstruction with ability to cough: allow child to cough
  - Complete airway obstruction but conscious, unable to cough, no sound, no breathing:
    - Infant <12 months: 5 back blows, then 5 chest thrusts
    - Child ≥1 year: abdominal thrusts
    - Complete airway obstruction and unconscious: start CPR
- Management of anaphylaxis may include:
  - Epinephrine IM every 10–15 min as needed
  - Albuterol MDI or nebulized for wheezing and bronchospasm
  - If persistent airway swelling, prepare for intubation
  - Diphenhydramine and H_2 blocker IV
  - Corticosteroid IV
  - Treat hypotension:
    - 20 mL/kg bolus normal saline or lactated Ringer's IV; repeat as needed
    - Trendelenburg position
    - Epinephrine IV infusion if needed
- Management of moderate to severe croup may include:
  - Fasting state
  - Humidified oxygen
  - Nebulized epinephrine
  - Dexamethasone
  - Heliox (helium-oxygen mixture) for severe croup
  - Vigilant monitoring for improvement or deterioration

- Management of croup with impending respiratory failure may include:
  - High oxygen concentration via nonrebreather mask
  - Bag-mask ventilation if indicated
  - Dexamethasone IV or IM
  - ET with tube slightly smaller than predicted for child
  - Surgical airway if indicated

Lower Airway Obstruction

The trachea is obstructed below the larynx in the bronchi, bronchioles, or both. Causes include asthma and bronchiolitis.

- **Clinical presentation**:
  - Tachypnea, wheezing, cough
  - Increased respiratory effort, prolonged expiration
- **Management** includes support of oxygenation and ventilation
- **Management of bronchiolitis** may include:
  - Suction as needed
  - Supplemental oxygen if needed
  - Nebulized epinephrine or albuterol if needed
- **Management of asthma** may include:
  - For mild to moderate cases:
    - High-concentration humidified oxygen
    - Albuterol MDI or nebulizer
    - Corticosteroids PO
  - For moderate to severe cases:
    - High-concentration humidified oxygen
    - Albuterol MDI with spacer or nebulizer; may require continuous nebulizer
    - Ipratropium nebulizer
    - Corticosteroids PO or IV
    - Magnesium sulfate IV bolus over 15–30 min if needed
    - ABGs, chest x-ray if indicated
  - For impending respiratory failure:
    - High-concentration humidified oxygen via nonrebreather mask
    - Continuous albuterol nebulizer
    - Corticosteroid IV
    - Consider terbutaline SQ or continuous IV, or epinephrine SQ or IM
    - Bilevel positive airway pressure if alert and cooperative
    - ET if indicated

Lung Disease

Lung disease typically occurs in small airways and alveoli. It compromises oxygenation and possibly ventilation, reduces lung compliance, and may lead to pulmonary infiltrates. Causes include pneumonia, pulmonary edema, pulmonary contusion, allergic reaction, infiltrative disease, vasculitis, or lung trauma caused by toxins.

- **Clinical presentation**:
  - Tachypnea, increased respiratory effort, tachycardia
  - Pulmonary crackles, grunting, diminished breath sounds
  - Hypoxemia
- **Management**:
  - For infectious pneumonia:
    - Diagnostic tests: ABG, chest x-ray, CBC, sputum and blood cultures, viral studies
    - Appropriate antibiotic therapy
    - Albuterol MDI or nebulizer for wheezing
    - CPAP or noninvasive ventilation if indicated
    - ET and mechanical ventilation if indicated
    - Management of fever
    - Supportive measures to reduce work of breathing
  - For chemical pneumonitis:
    - Nebulized bronchodilator for wheezing
    - CPAP or noninvasive ventilation if indicated
    - Expert consultation, specialized care if indicated
  - For aspiration pneumonia:
    - CPAP or noninvasive ventilation
    - ET and mechanical ventilation
    - Antibiotics if fever and infiltrate on chest x-ray
  - For cardiogenic pulmonary edema:
    - Noninvasive ventilation
    - ET and mechanical ventilation
    - Diuretics
    - Inotropic IV infusions
    - Management of fever
    - Supportive measures to reduce work of breathing
  - Noncardiogenic pulmonary edema—ARDS:
    - Diagnostic tests: ABG, chest x-ray, central venous blood gas, CBC
    - Noninvasive ventilation
    - ET and mechanical ventilation with PEEP, low tidal volumes
    - Correction of hypoxemia with permissive hypercarbia

Disordered Control of Breathing

Respiratory rate, effort, or both are inadequate, leading to abnormal breathing patterns. Causes may include neurological disorders such as CNS infection, head trauma, seizures, brain tumor, narcotic overdose, neuromuscular disease, hydrocephalus.

- **Clinical presentation**:
  - Irregular breathing pattern with tachypnea alternating with bradypnea
  - Irregular respiratory effort, shallow respirations
  - Hypoxemia, hypercarbia
  - Central apnea (no respirations or respiratory effort)

- **Management:**
  - For increased intracranial pressure:
    - Stabilizing cervical spine if trauma is suspected
    - Maintaining a patent airway; adequate oxygenation and ventilation
    - 20 mL/kg IV normal saline or lactated Ringer's if signs of poor perfusion
    - Hypertonic saline or osmotic agents for increased ICP
    - Treating agitation and pain
    - Avoiding hyperthermia
    - Neurosurgical consultation
  - For poisoning or drug overdose:
    - Contacting poison control center
    - Appropriate antidote as indicated
    - Suctioning airway if vomiting
    - Diagnostic tests: ABG, drug screens, complete metabolic profile, ECG, serum osmolality
  - For neuromuscular disease:
    - Treating the disorder
    - Maintaining a patent airway; adequate oxygenation and ventilation
    - Suctioning if indicated
    - Noninvasive ventilation with CPAP or BiPAP
    - ET and mechanical ventilation if indicated

Recognition and Management of Shock

Shock is a state in which the delivery of oxygen and nutrients is inadequate to meet cellular metabolic demand. It may occur with normal or decreased BP, CO, and perfusion. Inadequate tissue perfusion leads to tissue hypoxia and anaerobic metabolism, causing increased lactate production and metabolic acidosis. Cellular and organ damage result. Shock must be rapidly identified and treated to prevent cardiopulmonary failure and eventual cardiac arrest.

Pathophysiology

- **Compensated shock:** BP is normal. Perfusion to the heart and brain is adequate, but peripheral tissue perfusion is inadequate. Compensatory mechanisms include increased heart rate, increased myocardial contractility, and peripheral arterial and venous vascular constriction. Stroke volume, preload, afterload, and CO are all increased.
  - **Clinical presentation:** Tachycardia, weak peripheral pulses, decreased pulse pressure, cool and pale skin, diaphoresis, skin mottling, decreased urine output, decreased splanchnic perfusion possibly causing nausea, vomiting, and ileus.

- **Hypotensive shock:** Compensatory mechanisms fail, causing decreased BP with further tissue hypoxia and progressive deterioration.
  - **Clinical presentation:** Altered LOC, hypotension, signs and symptoms of multiple organ dysfunction.

Types of Shock and Management

There are four major categories of shock: hypovolemic shock, distributive shock, cardiogenic shock, and obstructive shock.

Hypovolemic Shock

Hypovolemic shock is the most common cause of shock in children. Loss of volume decreases preload, which decreases stroke volume and CO, decreasing BP and tissue perfusion. **Causes include:**

- Diarrhea (most common cause in children)
- Vomiting
- Internal or external blood loss
- Large burns
- Severe dehydration
- Osmotic diuresis, as with DKA
- Third-space losses: fluid shifting into interstitial spaces

- **Clinical presentation:**
  - Tachypnea causing respiratory alkalosis, compensating for metabolic acidosis; nonlabored breathing
  - Tachycardia, weak or absent peripheral pulses, narrow pulse pressure, delayed capillary refill, hypotension
  - Skin cool, pale, dusky, cyanotic, mottled, diaphoretic, especially in extremities
  - Changes in level of consciousness
  - Oliguria
- **Management of nonhemorrhagic hypovolemic shock:**
  - Identify and treat source of volume loss
  - Administer isotonic crystalloid solution with boluses of 20 mL/kg IV to replace volume
  - Replace ongoing fluid losses
  - Administer colloids such as albumin for large interstitial fluid shifts (third spacing) or albumin deficit
- **Management of hemorrhagic hypovolemic shock:**
  - Identify and treat source of blood loss
  - Estimate percentage of total blood volume loss
  - Administer isotonic crystalloid solution with boluses of 20 mL/kg IV. Rule of thumb: Give 3 mL for every 1 mL of blood loss
  - If patient is still hypotensive after 3 boluses, consider blood replacement with 10 mL/kg boluses of warmed packed RBCs

Perform diagnostic studies for hypovolemic shock: may include CBC with differential, type and crossmatch, ABG, basic metabolic panel, serum or plasma lactate, imaging studies.

♥ **Clinical Tip:** Shock can cause lactic metabolic acidosis with tachypnea, resulting in respiratory alkalosis as a compensatory mechanism to raise blood pH. The primary treatment for hypovolemic shock is fluid resuscitation to improve perfusion. Sodium bicarbonate should not be given unless metabolic acidosis results from loss of bicarbonate caused by GI or renal disease.

Distributive Shock

Distributive shock is a maldistribution of blood volume leading to inadequate cellular and organ perfusion. Some tissues receive excessive perfusion; others, especially splanchnic tissues, are poorly perfused, causing lactic acidosis. Preload and myocardial contractility may be normal or decreased. Afterload is variable. CO may be normal, increased, or decreased.

- **Warm shock:** This early phase is marked by decreased SVR from vasodilation, hypotension, wide pulse pressure, increased skin perfusion, warm extremities, bounding peripheral pulses, brisk capillary refill.
- **Cold shock:** This later phase is marked by increased SVR from vasoconstriction, hypotension, narrow pulse pressure, decreased tissue perfusion, cold extremities, weak or thready peripheral pulses, delayed capillary refill.

Types of distributive shock include septic, anaphylactic, and neurogenic shock.

Septic Shock

In septic shock, infectious organisms cause systemic inflammation, initiating a cascade of physiological responses. These include inflammatory mediators (cytokines), which cause vasodilation, increased capillary permeability, microvascular thrombosis leading to DIC, myocardial dysfunction, and reduced CO. Tissue perfusion is inadequate. Relative or absolute adrenal insufficiency may develop, contributing to reduced SVR.

- **Clinical presentation:**
  - Signs and symptoms of "warm shock" above
  - Signs and symptoms of "cold shock" above
  - Altered mental status
  - Possible fever and leukocytosis
- **Management:**

1. Establish responsiveness.
2. Perform primary ABCDE survey.
3. Provide oxygen; support ventilation.
4. Measure vital signs, including oxygen saturation.
5. Obtain IV/IO access and monitor-defibrillator.
6. Obtain blood samples for arterial or venous blood gas, CBC, glucose, ionized calcium, lactate, and blood cultures.

7. During first hour:
   - Treat shock with repeated IV boluses of 20 mL/kg of crystalloid solution as needed. Discontinue fluid boluses if patient develops signs of respiratory distress, pulmonary crackles, or hepatomegaly.
   - Treat hypocalcemia and hypoglycemia.
   - Administer first dose of antibiotics immediately.
   - Obtain additional vascular access if vasoactive infusion is required.
8. Assess response to fluid administration. If hemodynamics and perfusion normalize, admit to ICU. Identify and treat causes of sepsis. If patient remains hemodynamically unstable with poor perfusion:
   - Establish arterial access for direct invasive BP monitoring.
   - Begin vasoactive IV infusion.
     - If normotensive with poor perfusion, administer dopamine.
     - If hypotensive and vasodilated (warm shock), administer norepinephrine.
     - If hypotensive and vasoconstricted (cold shock), administer epinephrine.
   - Establish central venous access for central venous oxygen saturation monitoring.
     - The goal of central venous oxygen saturation ($S_{cv}O_2$) is >70%.
     - Optimize arterial oxygen saturation (SaO_2).
     - If $S_{cv}O_2$ >70% with hypotension (warm shock), administer additional fluid boluses and consider norepinephrine. Vasopressin may be added if necessary.
     - If $S_{cv}O_2$ is <70% with normal BP but poor perfusion, transfuse to increase hemoglobin >10 g/dL if indicated, administer additional fluid boluses, and consider dobutamine, milrinone, or nitroprusside.
     - If $S_{cv}O_2$ is <70% with low BP and poor perfusion (cold shock), transfuse to increase hemoglobin >10 g/dL if indicated, administer additional fluid boluses, and consider epinephrine or dobutamine plus norepinephrine.
   - Titrate medication(s) to achieve an adequate BP and restore perfusion.
9. Patients who continue in shock refractory to fluids and dependent on dopamine or norepinephrine are at risk for adrenal insufficiency.
   - Draw baseline cortisol.
   - Consider ACTH stimulation test if unsure of need for steroids.
   - Administer hydrocortisone 2 mg/kg IV bolus (maximum dose 100 mg) if adrenal insufficiency is suspected.

Anaphylactic Shock

Anaphylactic shock is a severe reaction to an allergen causing an acute multi-system allergic reaction. It causes arterial and venous vasodilation; increased capillary permeability and pulmonary vasoconstriction, which increase right ventricular afterload, reduce pulmonary blood flow, decrease left ventricular preload, and reduce CO.

- **Clinical presentation:**
  - Restlessness, anxiety, agitation
  - Tachycardia
  - Hypotension caused by vasodilation (relative hypovolemia), decreased CO, and capillary leak (absolute hypovolemia)
  - Urticaria
  - Respiratory distress, wheezing, stridor
  - Nausea, vomiting
  - Angioedema
  - Partial or complete upper airway obstruction
- **Management:**
  - Anticipate angioedema, be prepared to assist ventilation
  - Administer epinephrine 1:1,000 IM or autoinjector 0.01 mg/kg, may repeat in 10–15 min if needed
  - Administer isotonic crystalloid solution IV for hypotension
  - Administer epinephrine IV infusion if needed
  - Administer albuterol MDI or nebulizer for bronchospasm
  - Administer diphenhydramine, an H_1 blocker; it may be more effective when given with an H_2 blocker such as famotidine
  - Administer corticosteroids

Neurogenic Shock

Neurogenic shock is the sudden lack of sympathetic nervous system innervation to vascular smooth muscle, resulting in inappropriate vasodilation and hypotension. Heart rate cannot increase to compensate for hypotension. Causes include cervical spine or high thoracic spine injury.

- **Clinical presentation:**
  - Hypotension
  - Wide pulse pressure with low diastolic pressure
  - Normal heart rate or bradycardia
- **Management:**
  - Place patient in supine or Trendelenburg position to improve venous return
  - Consider IV isotonic crystalloid: may produce minimal response because hypotension is from vasodilation, not volume loss
  - Administer vasopressors: norepinephrine or epinephrine infusion
  - Control ambient temperature: warming or cooling as indicated

Cardiogenic Shock

Cardiogenic shock is caused by myocardial dysfunction resulting in reduced CO and inadequate tissue perfusion. Causes include congenital heart anomalies, cardiomyopathy, myocarditis, arrhythmias, drug toxicity or poisoning, myocardial contusion or trauma, and sepsis.

- **Clinical presentation:**
  - Tachypnea

- Tachycardia
- Increased work of breathing
- Pulmonary crackles, pulmonary edema
- Normal or low BP
- Weak or absent peripheral pulses
- Delayed capillary refill
- Cool, diaphoretic, mottled skin
- Oliguria
- Jugular venous distention
- Hepatomegaly, ascites
- Peripheral edema

- **Management:**
  - Administer fluids cautiously: 5–10 mL/kg IV over 10–20 min, monitoring for improvement. Too much fluid may cause pulmonary edema, hypoxemia, respiratory distress, jugular venous distention, hepatomegaly, cardiomegaly.
  - Give supplemental oxygen; support ventilation.
  - Perform diagnostic studies: CBC, ABG, cardiac serum markers, thyroid function tests, lactate level, ECG, CXR, echocardiogram.
  - Use central venous catheter and pulmonary artery catheter to measure preload, contractility, and afterload.
  - If patient is normotensive, diuretic and vasodilator therapy are indicated.
  - If patient is in shock, treatment may include cautious vasodilator therapy, inotropic therapy if indicated, phosphodiesterase enzyme inhibitor therapy with an inodilator such as milrinone.
  - Reduce metabolic demand with ventilatory support, antipyretics if indicated, analgesics and sedatives if indicated, mechanical circulatory support using ECLS, VAD, or ECMO if indicated and available.

Obstructive Shock

In obstructive shock, CO is impeded by obstruction of blood flow. A significantly reduced CO may cause hypotension and shock. Obstructive shock may be caused by pericardial (cardiac) tamponade and tension pneumothorax.

Pericardial (Cardiac) Tamponade

In pericardial (cardiac) tamponade, an excess amount of fluid, blood, or air accumulates in the pericardial sac. External pressure around the heart impedes ventricular filling, decreasing stroke volume and CO. Causes include penetrating trauma, cardiac surgery, infection, inflammatory disorders, tumors and malignancies, and excessive leukocytosis.

- **Clinical presentation:**
  - Tachypnea, increased work of breathing
  - Tachycardia
  - Weak, thready peripheral pulses
  - Delayed capillary refill

- Narrow pulse pressure
- Distant or muffled heart sounds
- Pulsus paradoxus
- Cool, clammy, mottled extremities
- Distended neck veins
- Oliguria
- Change in LOC

- **Management:**
  - Use initial IV volume administration until pericardial drainage can be done.
  - Use echocardiogram- or fluoroscopy-guided pericardiocentesis

Tension Pneumothorax

In **tension pneumothorax,** air enters the pleural space and accumulates with no opportunity to exit. Causes include internal lung injury, such as ruptured bullae or excessive positive-pressure ventilation, and external penetrating chest injury. Pressure builds up in the pleural space, compressing underlying lung tissue. Eventually, mediastinal shift pushes the lung and heart to the opposite side of the chest. This condition leads to respiratory failure, decreased venous return, decreased CO, and hypotension.

- **Clinical presentation:**
  - Tachypnea
  - Increased respiratory effort
  - Hypoxemia
  - Diminished breath sounds on affected side
  - Hyperresonance and hyperexpansion of chest on affected side
  - Tracheal deviation toward unaffected side
  - Jugular vein distention
  - Pulsus paradoxus
  - Initial tachycardia deteriorating to bradycardia
  - Hypotension
  - Cool, pale extremities
  - Change in LOC
- **Management:**
  - Perform immediate needle decompression at second intercostal space over third rib in midclavicular line
  - Perform thoracostomy, chest tube placement

PALS Algorithms

When pediatric patients present in respiratory or cardiac arrest or with cardiovascular emergencies, they must be assessed rapidly and systematically. PALS algorithms outline step-by-step protocols for dealing with these situations.

Ventricular Fibrillation (VF) or Pulseless Ventricular Tachycardia (VT)

Clinical Presentation

- Unresponsive state
- No respirations or only agonal respirations
- No pulse

Management

1. **Establish unresponsiveness:**
   - No respirations or only agonal respirations and no pulse.
2. **Call for help.**
3. **C–A–B: Compressions, airway, breathing:**
   - Begin CPR, starting with compressions.
   - Provide oxygen.
4. **Defibrillation**
   - **Attach AED or manual monitor-defibrillator** as soon as available without interrupting compressions. Use pediatric pads or paddles if available and indicated.
     - When device is attached, stop CPR and assess rhythm.
     - AED: If shock is advised, defibrillate following AED prompts.
     - Manual monitor-defibrillator: Defibrillate at 2 J/kg using a biphasic or monophasic defibrillator.
5. **Immediately resume CPR,** beginning with compressions.
   - Provide 5 cycles (2 min) of uninterrupted CPR.
   - During CPR, establish IV or IO access.
   - Prepare vasopressor dose (epinephrine).
6. **Defibrillation**
   - Stop CPR.
   - Assess rhythm.
   - If the rhythm remains shockable, follow AED prompts or **defibrillate** at 4 J/kg.
7. **Immediately resume CPR,** beginning with compressions.
   - Provide 5 cycles (2 min) of uninterrupted CPR.
   - Insert an advanced airway (ET) if basic airway management is inadequate.
     - Confirm correct tube placement without interrupting CPR.
     - After correct placement is confirmed, deliver **uninterrupted** chest compressions at a rate of at least 100/min and deliver **8–10 breaths/min at a rate of 1 breath every 6–8 sec.**

8. **Drugs**
   - Administer **epinephrine** 0.01 mg/kg.
     - Give 0.1 mL/kg of 1:10,000 IV/IO.
     - Follow with 20 mL IV flush.
     - Repeat every 3–5 min as needed.
   - If no IV/IO access is available and the patient has an ET in place, stop compressions and inject 0.1 mg/kg (0.1 mL/kg of 1:1,000) epinephrine directly into ET followed by a 5-mL saline flush. Follow ET drug administration with ventilations to disperse drug into small airways for absorption into pulmonary vasculature and resume compressions.
9. **Continue CPR; check the rhythm every 2 min.**
10. **Defibrillation**
    - If the rhythm remains shockable, follow AED prompts or **defibrillate** at 4 J/kg.
    - May increase energy with subsequent shocks, if needed, to a maximum of 10 J/kg or adult maximum energy dose.
11. **Immediately resume CPR,** check rhythm every 2 min.
12. **Drugs**
    - Consider antiarrhythmic drugs for shock-refractory VF or pulseless VT.
      - Administer **amiodarone** 5 mg/kg IV/IO, or **lidocaine** 1 mg/kg IV/IO if amiodarone is not available.
      - May repeat **amiodarone** 5 mg/kg IV/IO up to 2 times for shock-refractory VF or VT to a maximum of 15 mg/kg.
    - If the arrhythmia is torsade de pointes, consider **magnesium sulfate** 25–50 mg/kg IV/IO (maximum dose 2 g) bolus.
13. During CPR, consider and treat potentially reversible causes (Hs and Ts):
    - Hypokalemia/hyperkalemia
    - Hypovolemia
    - Hypoxia or ventilation problems
    - Hypoglycemia
    - Hypothermia
    - Hydrogen ion (acidosis)
    - Trauma (hypovolemia, increased ICP)
    - Tension pneumothorax
    - Tamponade (cardiac)
    - Toxins
    - Thrombosis (pulmonary or coronary)
14. If rhythm changes to asystole or PEA, follow algorithm for asystole or PEA.
15. If rhythm converts to a stable ECG rhythm with ROSC:
    - Monitor and reevaluate the patient.
    - Arrange for transport to a critical care unit. The patient will need a comprehensive care plan. (See the Immediate Post–Cardiac Arrest Care algorithm.)

♥ **Clinical Tip:** In infants and young children, cardiac arrest is more likely caused by progressive respiratory failure, or shock leading to an asphyxial arrest, than by cardiac disease. Early recognition and management of impending respiratory failure may prevent cardiac arrest.

♥ **Clinical Tip:** Use infant defibrillation pads or paddles for infants weighing <10 kg. Use adult pads or paddles for infants/children weighing >10 kg.

♥ **Clinical Tip:** Secure an advanced airway and verify its placement by observing bilateral chest expansion, auscultating bilateral breath sounds and lack of epigastric sounds, and using a confirmatory device (exhaled CO_2 detector). Use continuous waveform capnography if available. If not available, use a colorimetric CO_2 detector. Monitor ET for displacement during transport or whenever the patient is moved.

♥ **Clinical Tip:** Once an advanced airway is in place, deliver chest compressions continuously without pause for ventilations. Deliver one breath every 6–8 sec without regard to the phase of chest compressions (downstroke vs. upstroke).

♥ **Clinical Tip:** If an advanced airway cannot be secured, and a second rescuer has arrived, continue ventilations with a bag-mask device, delivering two breaths after each set of 15 compressions.

Pulseless Electrical Activity (PEA)

Clinical Presentation

- Unresponsive state
- No respirations or only agonal respirations
- Organized electrical rhythm on monitor but no pulse

Management

1. **Establish unresponsiveness:**
   - No respirations or only agonal respirations and no pulse.
2. **Call for help.**
3. **C–A–B: Compressions, airway, breathing:**
   - Begin CPR, starting with compressions.
   - Provide oxygen.
4. **Attach AED or manual monitor-defibrillator** as soon as available without interrupting compressions. Use pediatric pads or paddles if available and indicated.
   - When device is attached, stop CPR to assess rhythm.
     - AED: **No shock advised.**
     - Manual monitor-defibrillator: Organized rhythm (PEA). **Do not defibrillate.**
5. **Immediately resume CPR,** beginning with compressions.
   - Provide 5 cycles (2 min) of uninterrupted CPR.
   - During CPR, establish IV or IO access.
   - Prepare vasopressor dose (epinephrine).

6. **Stop CPR. Assess rhythm.**
   - AED: **No shock advised.**
   - Manual monitor-defibrillator: Organized rhythm (PEA). **Do not defibrillate.**
7. **If PEA persists, immediately resume CPR,** beginning with compressions.
   - Provide 5 cycles (2 min) of uninterrupted CPR.
   - Insert an advanced airway (ET) if basic airway management is inadequate.
     - Confirm correct tube placement without interrupting CPR.
     - After correct placement is confirmed, deliver uninterrupted chest compressions, at least 100/min for 2 min. Deliver 8–10 breaths/min at a rate of 1 breath every 6–8 sec.
8. **Drugs**
   - Administer **epinephrine** 0.01 mg/kg.
     - Give 0.1 mL/kg of 1:10,000 IV/IO.
     - Follow with 20 mL IV flush.
     - Repeat every 3–5 min as needed.
   - If no IV/IO access is available and the patient has an ET in place, stop compressions and inject 0.1 mg/kg (0.1 mL/kg of 1:1,000) epinephrine directly into ET followed by a 5-mL saline flush. Follow ET drug administration with ventilations to disperse drug into small airways for absorption into pulmonary vasculature and resume compressions.
9. **Continue CPR, check rhythm every 2 min.**
   - If PEA persists, immediately resume CPR; check rhythm every 2 min.
   - Administer **epinephrine** every 3–5 min.
10. During CPR, consider and treat potentially reversible causes of PEA (Hs and Ts):
    - Hypokalemia/hyperkalemia
    - Hypovolemia
    - Hypoxia or ventilation problems
    - Hypoglycemia
    - Hypothermia
    - Hydrogen ion (acidosis)
    - Trauma (hypovolemia, increased ICP)
    - Tension pneumothorax
    - Tamponade, cardiac
    - Toxins
    - Thrombosis (pulmonary or coronary)
11. If rhythm changes to VF, pulseless VT, or asystole, follow algorithm for VF, pulseless VT, or asystole.
12. If rhythm converts to a stable ECG rhythm with ROSC:
    - Monitor and reevaluate the patient.
    - Arrange for transport to a critical care unit. The patient will need a comprehensive care plan. (See the Immediate Post–Cardiac Arrest Care algorithm.)

Clinical Tip: PEA may be caused by potentially reversible conditions (Hs and Ts) and may be treated successfully if those conditions are identified and corrected early.

Asystole

Clinical Presentation

- Unresponsive state
- No respirations or only agonal respirations
- No pulse
- Flat ECG tracing or only agonal rhythm; no electrical activity on monitor

Management

1. **Establish unresponsiveness:**
   - No respirations or only agonal respirations and no pulse.
2. **Call for help.**
3. **C–A–B: Compressions, airway, breathing:**
   - Begin CPR, starting with compressions.
   - Provide oxygen.
4. **Attach AED or manual monitor-defibrillator** as soon as available without interrupting CPR. Use pediatric pads or paddles if available and indicated.
   - When device is attached, stop CPR to assess rhythm.
     - AED: **No shock advised.**
     - Manual monitor-defibrillator: No electrical activity (flat line or only agonal rhythm). **Do not defibrillate.**
5. **Immediately resume CPR,** beginning with compressions.
   - Provide 5 cycles (2 min) of uninterrupted CPR.
   - During CPR, establish IV or IO access.
   - Prepare vasopressor dose (epinephrine).
6. **Stop CPR. Assess rhythm.**
   - AED: **No shock advised.**
   - Manual monitor-defibrillator: No electrical activity. **Do not defibrillate.**
7. If asystole persists, immediately resume CPR, beginning with compressions.
   - Provide 5 cycles (2 min) of uninterrupted CPR.
   - Insert an advanced airway (ET) if basic airway management is inadequate.
     - Confirm correct tube placement without interrupting CPR.
     - After correct placement is confirmed, deliver uninterrupted compressions, at least 100/min for 2 min, and deliver 8–10 breaths/min at a rate of 1 breath every 6–8 sec.
8. **Drugs**
   - Administer **epinephrine** 0.01 mg/kg.
     - Give 0.1 mL/kg of 1:10,000 IV/IO.
     - Follow with 20 mL IV flush.
     - Repeat every 3–5 min as needed.
   - If no IV/IO access is available and the patient has an ET in place, stop compressions and inject 0.1 mg/kg (0.1 mL/kg of 1:1,000) epinephrine

directly into ET followed by a 5-mL saline flush. Follow ET drug administration with ventilations to disperse drug into small airways for absorption into pulmonary vasculature and resume compressions.

9. **Continue CPR, check rhythm every 2 min.**
   - If asystole persists, immediately resume CPR, check rhythm every 2 min.
   - Administer **epinephrine** every 3–5 min.
10. During CPR, consider and treat potentially reversible causes of asystole (Hs and Ts):
    - Hypokalemia/hyperkalemia
    - Hypovolemia
    - Hypoxia or ventilation problems
    - Hypoglycemia
    - Hypothermia
    - Hydrogen ion (acidosis)
    - Trauma (hypovolemia, increased ICP)
    - Tension pneumothorax
    - Tamponade, cardiac
    - Toxins
    - Thrombosis (pulmonary or coronary)
11. If asystole persists, consider whether proper resuscitation protocols were followed and reversible causes identified. If procedures were performed correctly, follow local criteria for terminating resuscitation efforts.
12. If rhythm changes to VF or pulseless VT, follow algorithm for VF or pulseless VT.
13. If rhythm changes to an organized rhythm with no pulse, follow algorithm for PEA.
14. If rhythm converts to a stable ECG rhythm with ROSC:
    - Monitor and reevaluate the patient.
    - Arrange for transport to a critical care unit. The patient will need a comprehensive care plan. (See the Immediate Post–Cardiac Arrest Care algorithm.)

♥ **Clinical Tip:** Emphasis must be on high-quality CPR, ensuring adequate rate and depth of chest compressions with complete chest recoil after each compression, minimizing interruptions in chest compressions (no more than 10 sec when necessary) and avoiding excessive ventilation. Rotate chest compressor every 2 min to minimize fatigue.

♥ **Clinical Tip:** Family presence during resuscitation may be beneficial, providing an opportunity to say goodbye and facilitating the grieving process when resuscitation attempts are unsuccessful. Families should be offered this opportunity whenever possible.

Bradycardia With a Pulse

Clinical Presentation

- Heart rate <60 bpm
- Respiratory distress or failure
- Signs of shock with hypotension, diaphoresis, altered mental status

Management

1. **Establish responsiveness.**
2. **Perform primary ABCDE survey.**
3. Measure vital signs, including oxygen saturation.
4. Administer oxygen, establish IV/IO access, and attach manual monitor-defibrillator to identify rhythm.
5. Obtain 12-lead ECG if possible.
6. **Assess for signs and symptoms.**
   - If the patient is stable and asymptomatic with a heart rate <60 bpm, support oxygenation and ventilation, monitor and observe for any changes, and seek expert consultation.
   - If the patient is symptomatic with a heart rate <60 bpm and signs of poor perfusion despite oxygenation and ventilation, perform CPR.
7. **Drugs**
   - If symptomatic bradycardia persists, administer **epinephrine** 0.01 mg/kg IV/IO (0.1 mL/kg of 1:10,000); follow with 20 mL IV flush.
     - Repeat every 3–5 min as needed.
     - If no IV/IO access is available and the patient has an ET in place, inject 0.1 mg/kg (0.1 mL/kg of 1:1,000) epinephrine directly into ET followed by a 5-mL saline flush. Follow ET drug administration with ventilations to disperse drug into small airways for absorption into pulmonary vasculature.
8. **Drugs**
   - For increased vagal tone or primary AV block, administer a first dose of **atropine** 0.02 mg/kg IV/IO.
     - May repeat every 3–5 min. Minimum single dose 0.1 mg, maximum single dose 0.5 mg, maximum total dose 1 mg.
9. **Pacing**
   - If the patient fails to respond to atropine, consider **transthoracic or transvenous pacing.**
10. Identify and treat the cause of bradycardia.

Tachycardia With Pulse, Narrow-Complex QRS (0.09 sec) With Poor Perfusion

Clinical Presentation

- Altered LOC
- Shortness of breath, diaphoresis, fatigue, syncope, poor perfusion

Management

1. **Establish responsiveness.**
2. **Perform primary ABCDE survey.**
3. Measure vital signs, including oxygen saturation.

4. Administer oxygen, establish IV/IO access, and attach manual monitor-defibrillator to identify rhythm.
5. Obtain a 12-lead ECG if possible.
6. **Assess for signs and symptoms.**
   - If the patient is stable and asymptomatic, with a heart rate <180 bpm for a child and <220 bpm for an infant, normal P waves, and varying R–R intervals, the rhythm is probably sinus tachycardia, not SVT. Search for and treat the underlying cause.
7. **Vagal Maneuvers**
   - If the heart rate is ≥180 bpm for a child and ≥220 bpm for an infant with absent P waves, regular rhythm, and signs of poor perfusion, the rhythm is probably SVT; consider **vagal maneuvers.**
8. **Drugs**
   - If vagal maneuvers are ineffective and IV/IO access is available, give **adenosine** 0.1 mg/kg IV/IO rapid push.
     - Maximum first dose is 6 mg.
     - May double first dose and give 0.2 mg/kg IV/IO rapid push.
     - Maximum second dose is 12 mg.
     - Immediately follow each dose with 5–10 mL normal saline flush.
9. **Cardioversion**
   - If IV/IO access is not available or adenosine was ineffective, attempt **synchronized cardioversion** at 0.5–1.0 J/kg.
   - If unstable tachycardia persists, increase to 2 J/kg.
   - Premedicate with a sedative plus an analgesic whenever possible, but do not delay cardioversion.
10. If cardioversion is unsuccessful, seek expert consultation.
11. **Drugs**
    - Prepare to administer either **amiodarone** 5 mg/kg IV/IO over 20–60 min or **procainamide** 15 mg/kg IV/IO over 30–60 min.
      - **Do not administer** amiodarone and procainamide together.

Clinical Tip: If there are visible P waves with a constant PR interval and somewhat variable R–R intervals in an infant with a heart rate <220 bpm or a child with a heart rate <180 bpm, the rhythm is probably sinus tachycardia. The heart rate increases gradually rather than abruptly. Correlate with the patient's clinical history and always search for and treat the cause.

Clinical Tip: If there are no visible P waves and therefore no measurable PR interval, with a regular R–R interval in an infant with a heart rate ≥220 bpm or a child with a heart rate ≥180 bpm, the rhythm is probably SVT. The rate typically changes abruptly. Treat according to the algorithm above.

Tachycardia With Pulse, Wide-Complex QRS (>0.09 sec) With Poor Perfusion

Clinical Presentation

- Altered LOC
- Shortness of breath, diaphoresis, fatigue, syncope, poor perfusion

Management

1. **Establish responsiveness.**
2. **Perform primary ABCDE survey.**
3. Measure vital signs, including oxygen saturation.
4. Administer oxygen, establish IV/IO access, and attach manual monitor-defibrillator to identify rhythm.
5. Obtain a 12-lead ECG.
6. **Cardioversion**
   - If the patient has cardiopulmonary compromise with a rapid heart rate and wide QRS complex, the rhythm is presumed to be VT.
     - Prompt **synchronized cardioversion** is indicated.
     - Attempt synchronized cardioversion at 0.5–1 J/kg; if unstable tachycardia persists, increase to 2 J/kg.
     - Premedicate with a sedative plus an analgesic whenever possible, but do not delay cardioversion.
7. **Drugs**
   - If the patient does not show signs of cardiopulmonary compromise, consider **adenosine** if wide-complex tachycardia is regular and monomorphic.
     - Give **adenosine** 0.1 mg/kg IV/IO rapid push (maximum first dose 6 mg).
     - May double the first dose and give 0.2 mg/kg IV/IO rapid push.
     - Maximum second dose is 12 mg.
8. If cardioversion or adenosine is unsuccessful, seek expert consultation.
9. **Drugs**
   - Prepare to administer either **amiodarone** 5 mg/kg IV/IO over 20–60 min or **procainamide** 15 mg/kg IV/IO over 30–60 min.
     - **Do not administer** amiodarone and procainamide together.

Clinical Tip: Wide-complex tachycardia is relatively uncommon in children. However, it can occur in children with heart disease, drug ingestion (tricyclic antidepressants), and hyperkalemia.

Tachycardia With Pulse, Narrow-Complex QRS (0.09 sec) With Adequate Perfusion

Clinical Presentation

- Rapid heart rate
- No serious symptoms with signs of adequate perfusion

Management

1. **Establish responsiveness.**
2. **Perform primary ABCDE survey.**
3. Measure vital signs, including oxygen saturation.
4. Administer oxygen, establish IV/IO access, and attach manual monitor-defibrillator to identify rhythm.
5. Obtain a 12-lead ECG if possible.
6. **Assess for signs and symptoms.**
   - If the patient is stable and asymptomatic, with a heart rate <180 bpm for a child and <220 bpm for an infant, the rhythm is probably sinus tachycardia, not SVT. Search for and treat the underlying cause.
7. **Vagal Maneuvers**
   - If the heart rate is ≥180 bpm for a child and ≥220 bpm for an infant, the rhythm is probably SVT; consider **vagal maneuvers.**
8. **Drugs**
   - If vagal maneuvers are ineffective and IV/IO access is available, give **adenosine** 0.1 mg/kg IV/IO rapid push:
   - Maximum first dose is 6 mg.
   - May double the first dose and give 0.2 mg/kg IV/IO rapid push.
   - Maximum second dose is 12 mg.
9. Search for and treat reversible causes.
10. **Drugs**
    - If rhythm has not converted to sinus, consider **amiodarone** 5 mg/kg IV over 20–60 min or **procainamide** 15 mg/kg IV over 30–60 min.
      - **Do not administer** amiodarone and procainamide together.
10. **Cardioversion**
    - If medications are ineffective, or if patient becomes unstable, attempt **synchronized cardioversion** at 0.5–1.0 J/kg.
      - If unstable tachycardia persists, increase to 2 J/kg.
      - Premedicate with a sedative plus an analgesic whenever possible but do not delay cardioversion.

Tachycardia With Pulse, Wide-Complex QRS (>0.09 sec) With Adequate Perfusion

Clinical Presentation

- Rapid heart rate
- No serious symptoms with signs of adequate perfusion

Management

1. **Establish responsiveness.**
2. **Perform primary ABCDE survey.**
3. Measure vital signs, including oxygen saturation.
4. Administer oxygen, establish IV/IO access, and attach manual monitor-defibrillator to identify rhythm.
5. Obtain a 12-lead ECG if possible.
6. **Drugs**
   - If the rhythm is suspected to be SVT with aberrancy (wide QRS), seek expert consultation.
     - Give **adenosine** 0.1 mg/kg IV/IO rapid push.
     - Maximum first dose is 6 mg.
     - May double the first dose and give 0.2 mg/kg IV/IO rapid push.
     - Maximum second dose is 12 mg.
7. Search for and treat reversible causes.
8. **Drugs**
   - If the rhythm is suspected to be VT, seek expert consultation.
     - Prepare to administer **amiodarone** 5 mg/kg IV over 20–60 min or **procainamide** 15 mg/kg IV over 30–60 min.
     - **Do not administer** amiodarone and procainamide together.
9. **Cardioversion**
   - If medications are ineffective or if patient becomes unstable, attempt **synchronized cardioversion** at 0.5–1.0 J/kg.
     - If unstable tachycardia persists, increase to 2 J/kg.
     - Premedicate with a sedative plus an analgesic whenever possible but do not delay cardioversion.

Clinical Tip: To calculate medication doses, use the patient's weight if known. If weight is not known, use a pediatric body-length tape with precalculated doses for a reasonable estimate.

Immediate Post–Cardiac Arrest Care

Management

1. **If there is ROSC,** assess responsiveness, perform primary ABCDE survey and secondary assessment. Measure vital signs, including oxygen saturation.

2. **Airway, breathing:**
   - Provide oxygen to maintain oxygen saturation 94%–99% for optimal oxygenation.
   - Wean oxygen down if saturation is 100% to prevent hyperoxemia and associated oxidative injury.
   - Unless awake and alert, the patient may require an advanced airway and monitoring with waveform capnography.
   - Avoid hyperventilation. Target end-tidal CO_2 should be 35–40 mm Hg.
3. **Circulation:**
   - Assess for shock.
   - Treat persistent shock with 20-mL/kg IV/IO boluses of normal saline or lactated Ringer's. Consider smaller boluses (10 mL/kg) if poor cardiac function is suspected.
   - If hypotensive shock persists, consider vasopressor infusion with epinephrine, dopamine, or norepinephrine.
   - If normotensive shock, consider dobutamine, dopamine, epinephrine, or milrinone.
4. Consider and treat potentially reversible causes of cardiac arrest (Hs and Ts):
   - Hypokalemia/hyperkalemia
   - Hypovolemia
   - Hypoxia or ventilation problems
   - Hypoglycemia
   - Hypothermia
   - Hydrogen ion (acidosis)
   - Trauma (hypovolemia, increased ICP)
   - Tension pneumothorax
   - Tamponade, cardiac
   - Toxins
   - Thrombosis (pulmonary or coronary)
5. Obtain a chest x-ray to confirm ET placement, assess pulmonary status, and evaluate heart size.
6. Obtain a 12-lead ECG as soon as possible.
7. If the patient remains unresponsive or cannot follow commands, initiate therapeutic hypothermia, cooling to a core temperature of 32°–34°C (89.6°–93.2°F) for 12–24 hr.
8. Assess arterial blood gases, serum electrolytes, and calcium level.
9. Monitor the patient for pain, agitation and seizures; initiate appropriate treatment if present.
10. Monitor the patient for hypoglycemia and initiate appropriate treatment if present.
11. All cardiac arrest survivors should receive advanced critical care and a comprehensive care plan to optimize neurological, cardiopulmonary, and metabolic function.

Tab 5: Emergency Medications

This is a reference list only. It is not meant to be exhaustive in clinical content. Drug dosages follow Advanced Cardiac Life Support (ACLS) guidelines for adult patients and Pediatric Advanced Life Support (PALS) guidelines for pediatric patients.

Before administering medications, especially IV medications, always consult an authoritative, current reference about dose, dilution, route, rate of administration, and interactions. Have a second licensed person independently check dose calculations, preparation, original orders, and infusion pump programming.

Ace Inhibitors

Class: Angiotensin-converting enzyme inhibitors
Common Agents: Captopril, enalapril, lisinopril, ramipril.
Indications: MI, especially with ST elevation and with left ventricular dysfunction; HTN; heart failure without hypotension.

Adult Dose: See individual order and drug for route and dosage. Usually not started in the Emergency Department for an acute MI, but within 24 hr after reperfusion therapy has been completed and BP has stabilized.

Contraindications: Lactation, pregnancy, angioedema, hypersensitivity to ACE inhibitors, hyperkalemia, hypotension.
Side Effects: Cough, dizziness, headache, fatigue, hypotension, hyperkalemia, renal insufficiency.
Precautions: Reduce dose in renal insufficiency. Caution in severe aortic stenosis, hypertrophic cardiomyopathy, unstented renal artery stenosis, severe CHF.

Adenosine (Adenocard)

Class: Antiarrhythmic
Indications: Regular narrow-complex tachycardia, PSVT, and wide-complex tachycardia only if regular and monomorphic.

Adult Dose: 6 mg IV in the antecubital or another large vein given rapidly over 1–3 sec followed by a 20-mL bolus of normal saline. If the rhythm does not convert, give 12 mg by rapid IVP in 1–2 min if needed. A third dose of 12 mg IVP may be given in another 1–2 min, maximum total dose 30 mg.

Pediatric Dose: 0.1 mg/kg (max. 6 mg) IV/IO given rapidly over 1–3 sec followed by a 5–10-mL bolus of normal saline. If the rhythm does not convert, give 0.2 mg/kg (max. 12 mg) IV/IO in 1–2 min if needed.

Continued

Contraindications: Hypersensitivity, sick sinus syndrome, second- or third-degree AV block (unless a functioning pacemaker is present), A-fib/A-flutter with underlying Wolff-Parkinson-White syndrome, drug- or poison-induced tachycardia, bronchospastic lung disease.
Side Effects: Flushing, nausea, dizziness, headache, dyspnea, bronchospasm, chest pain or tightness, discomfort in neck, throat, or jaw, bradycardia, AV block, asystole, ventricular ectopic beats, VF.
Precautions: Ineffective in treating A-fib, A-flutter, or VT. Less effective in patients taking theophylline or caffeine. Reduce dose in patients taking dipyridamole or carbamazepine and in heart transplant patients.

Adenosine Diphosphate (ADP) Antagonists

Class: Antiplatelet agents—thienopyridines (clopidogrel and prasugrel), cyclopentyltriazolopyrimidine (ticagrelor)
Common Agents: Clopidogrel (Plavix), prasugrel (Effient), ticagrelor (Brilinta).
Indications: Antiplatelet therapy for acute coronary syndromes (ACS) managed with percutaneous coronary intervention (PCI). Clopidogrel: ACS, recent stroke, or peripheral arterial disease.
Adult Dose: See individual order and drug for dosage.
Contraindications: Acute pathological bleeding (e.g., peptic ulcer, intracranial bleeding). Prasugrel: also history of TIA or stroke. Ticagrelor: also history of intracranial hemorrhage, hepatic impairment.
Side Effects: Bleeding, thrombocytopenia purpura. Ticagrelor: dyspnea, increased serum creatinine. Stent thrombosis with premature discontinuation of therapy.
Precautions: Increased risk for bleeding (chronic NSAID use, anticoagulation therapy, thrombocytopenia, trauma/surgery), thienopyridine hypersensitivity, severe hepatic impairment, severe renal impairment. Prasugrel: caution in patients ≥75 years old or patients <60 kg. Ticagrelor: patients with hyperuricemia or gouty arthritis, patients at risk for bradycardia without pacemaker. Drugs must be withheld prior to CABG or elective surgery—clopidogrel and ticagrelor: 5 days; prasugrel: 7 days. In patients at risk for stent thrombosis, consider bridging with an IV glycoprotein IIb/IIIa inhibitor such as eptifibatide (Integrilin) while patient is off ADP antagonist; resume oral therapy as soon as possible after surgery when risk for postoperative bleeding is reduced.

Albuterol (ProAir, Proventil, Ventolin)

Class: Adrenergic beta$_2$-agonist, bronchodilator
Indications: Asthma, COPD, anaphylaxis (bronchospasm), hyperkalemia.

Adult Dose: *For bronchospasm,* metered-dose inhaler (MDI): 2 puffs every 4–6 hr prn; nebulizer: 2.5 mg 3–4 times daily prn or 1.25–5 mg every 4–8 hr prn. *For severe bronchospasm and status asthmaticus*: 5 mcg/min IV, titrate up every 15–30 min to 10–20 mcg/min. *For severe acute asthma exacerbation,* MDI: 4–8 puffs every 20 min up to 4 hr, then every 1–4 hr prn; nebulizer: 2.5–5 mg every 20 min for 3 doses, then 2.5–10 mg every 1–4 hr prn or 10–15 mg/hr by continuous nebulizer.

Pediatric Dose: *For bronchospasm*, MDI 2 puffs every 4–6 hr prn; nebulizer children 2–12 yr: 0.63–1.25 mg 3–4 times daily prn; nebulizer children ≥12 yr: 2.5 mg 3–4 times daily prn. *For mild to moderate asthma or anaphylaxis, hyperkalemia,* MDI: 4–8 puffs every 20 min prn; nebulizer, <20 kg: 2.5 mg every 20 min, >20 kg: 5 mg every 20 min. *For severe asthma exacerbation,* MDI children <12 yr: 4–8 puffs every 20 min for 3 doses, then every 1–4 hr prn; MDI children ≥12 yr: 4–8 puffs every 20 min for up to 4 hr, then every 1–4 hr prn; nebulizer children <12 yr: 0.15 mg/kg every 20 min for 3 doses, then 0.15–0.3 mg/kg every 1–4 hr prn, or 0.5 mg/kg/hr by continuous nebulizer; nebulizer children ≥12 yr: 2.5–5 mg every 20 min for 3 doses, then 2.5–10 mg ever 1–4 hr prn, or 10–15 mg/hr by continuous nebulizer.

Contraindications: Hypersensitivity, tachyarrhythmias, risk for abortion during first or second trimester.

Side Effects: Angina, arrhythmias, palpitations, tachycardia, flushing, dizziness, headache, insomnia, irritability, angioedema, rash, urticaria, hypokalemia, hyperglycemia, asthma exacerbation, cough.

Precautions: Use of spacer with MDI is recommended. Caution in cardiovascular disease (arrhythmias, HTN, heart failure), diabetes (may increase serum glucose), glaucoma (increased intraocular pressure), hyperthyroidism (may stimulate thyroid activity), hypokalemia (decreased serum potassium), seizure disorders (CNS stimulation/excitation).

Amiodarone (Cordarone, Pacerone)

Class: Antiarrhythmic, class III

Indications: Management of life-threatening shock-refractory VF or VT, recurrent hemodynamically unstable VT. Conversion of A-fib, SVT. Control of rapid ventricular rate in pre-excited atrial arrhythmias. Control of hemodynamically stable VT, polymorphic VT with normal QT interval, or wide-complex tachycardia of uncertain origin.

Adult Dose: *Cardiac arrest:* 300 mg IV/IO; consider additional 150 mg IV/IO in 3–5 min if needed. *Wide- and narrow-complex tachycardia (stable):* 150 mg IV over first 10 min (15 mg/min)—may repeat infusion of 150 mg IV every 10 min as needed; slow infusion of 360 mg IV over next 6 hr (1 mg/min); maintenance infusion of 540 mg over next 18 hr (0.5 mg/min). Maximum cumulative dose 2.2 g IV in 24 hr.

Continued

Pediatric Dose: *Cardiac arrest:* 5 mg/kg IV/IO bolus (max. 300 mg), may repeat to maximum of 15 mg/kg (or 2.2 g in adolescents) in 24 hr; *Wide- and narrow-complex tachycardia (stable):* 5 mg/kg IV/IO load over 20–60 min (max. 300 mg), may repeat to maximum of 15 mg/kg (2.2 g in adolescents) per day.

Contraindications: Hypersensitivity, cardiogenic shock, symptomatic bradycardia or second- or third-degree AV block without functioning pacemaker, severe sinus node dysfunction.

Side Effects: Vasodilation, hypotension, bradycardia, proarrhythmic effects, visual impairment, hepatotoxicity, pulmonary toxicity, CHF. May prolong QT interval, producing torsade de pointes.

Precautions: Avoid concurrent use with procainamide. Correct hypokalemia and hypomagnesemia, if possible, before use. Draw up amiodarone through a large-gauge needle to reduce foaming. For slow or maintenance IV infusion, mix the medication only in a glass bottle containing D5W or NS and administer through an in-line filter using a volumetric pump. Use with caution in thyroid disease, pulmonary disease, or hepatic impairment, and in patients on warfarin.

Aspirin (Acetylsalicylic Acid, ASA)

Class: Antiplatelet

Indications: Acute coronary syndrome, symptoms suggestive of cardiac ischemia, post-percutaneous coronary interventions, A-fib, stroke, peripheral arterial disease.

Adult Dose: *Acute coronary syndrome*: 160–325 mg PO. Chewing the tablet is preferable; use non–enteric-coated tablets for more rapid antiplatelet effect. Give within minutes of onset of ischemic symptoms. Other indications: 81–325 mg PO daily.

Contraindications: Hypersensitivity to salicylates, active bleeding, avoid in third trimester of pregnancy.

Side Effects: Anorexia, nausea, epigastric pain, bleeding, anaphylaxis.

Precautions: GERD, erosive gastritis, peptic ulcer disease, asthma, bleeding disorders, or thrombocytopenia.

Atropine Sulfate

Class: Anticholinergic, parasympatholytic, vagolytic

Indications: Symptomatic sinus bradycardia, junctional escape rhythm, or second-degree type I block. Not likely to be effective in second-degree type II or third-degree AV block with wide QRS complex.

Adult Dose: 0.5 mg IV given every 3–5 min as needed, maximum total dose 3 mg (0.04 mg/kg).

Pediatric Dose: 0.02 mg/kg IV/IO (min. dose 0.1 mg, max. single dose child 0.5 mg, max. single dose adolescent 1 mg), may repeat dose once, maximum total dose child 1 mg, maximum total dose adolescent 3 mg. May give 0.04–0.06 mg/kg, flush with 5 mL normal saline if administering by ET. Use ET route only if IV/IO access is not available.

Contraindications: Hypersensitivity, acute angle-closure glaucoma, asthma, prostatic hypertrophy, myasthenia gravis.
Side Effects: Tachycardia, headache, dry mouth, nausea, constipation, dilated pupils, flushing, hypotension.
Precautions: Use caution in myocardial ischemia and hypoxia. Avoid in hypothermic bradycardia and in second-degree (Mobitz type II) and third-degree AV block with wide QRS complex, asystole, bradycardic PEA. Caution in colon disease, hepatic or renal impairment, hiatal hernia, obstructive uropathy, hyperthyroidism.

Beta Blockers

Class: Beta blockers, antihypertensive, class II antiarrhythmic, antianginal
Common Agents: Atenolol, esmolol, labetalol, metoprolol, propranolol.
Indications: MI, unstable angina, PSVT, A-fib, A-flutter, HTN, CHF.

Adult Dose: See individual order and drug for route and dosage.

Contraindications: Heart rate <50 bpm, systolic BP <100 mm Hg, second- or third-degree AV block or sick sinus syndrome without functioning pacemaker, severe decompensated left ventricular failure, cardiogenic shock. Nonselective beta blockers are contraindicated in bronchospastic disease.
Side Effects: Hypotension, dizziness, bradycardia, headache, fatigue, nausea and vomiting, depression.
Precautions: Concurrent use with calcium channel blockers, such as verapamil or diltiazem, can cause hypotension. Use beta-1 selective agents with caution in patients with a history of bronchospasm. Use caution in thyroid disease, peripheral arterial disease, and diabetes (monitor blood glucose levels frequently).

Calcium Chloride

Class: Minerals, electrolytes, calcium salt
Indications: Hyperkalemia, hypocalcemia, hypermagnesemia; antidote for calcium channel blocker or beta blocker overdose.

Adult Dose: 500–1000 mg (5–10 mL of a 10% solution) over 2–5 min IV; may be repeated as needed.

Continued

Pediatric Dose: 20 mg/kg (0.2 mL/kg) IV/IO slow push during arrest or if severe hypotension, repeat as needed.

Contraindications: Hypercalcemia, hypophosphatemia, VF, digoxin toxicity.
Side effects: Bradycardia, hypotension, hypomagnesemia, hypercalcemia, VF, syncope, nephrolithiasis, flushing, dizziness, nausea and vomiting.
Precautions: Incompatible with sodium bicarbonate (precipitates). Caution in patients with renal impairment, respiratory acidosis, hypokalemia, hyperparathyroidism.

Digoxin (Lanoxin)

Class: Antiarrhythmic, cardiac glycoside
Indications: To slow ventricular response in A-fib or A-flutter; rarely, as a positive inotrope in CHF.

Adult Dose: Loading dose of 4–6 mcg/kg IV over 5 min, followed by 2–3 mcg/kg IV in 4–8 hr × 2. Maintenance dose determined by body size and renal function.

Contraindications: Hypersensitivity, uncontrolled ventricular arrhythmias, AV block without functioning pacemaker, idiopathic hypertrophic subaortic stenosis (IHSS), constrictive pericarditis, A-fib with Wolff-Parkinson-White syndrome.
Side Effects: Accelerated junctional rhythm, atrial tachycardia with block, AV block, asystole, VT, VF, ventricular bigeminy and trigeminy, dizziness, weakness, fatigue, nausea and vomiting, blurred or yellow vision, headache, rash, urticaria, hypokalemia.
Precautions: Avoid electrical cardioversion of stable patients. If the patient's condition is unstable, use lower current settings such as 10–20 J. Use cautiously in elderly patients and patients with heart failure, acute MI, renal impairment, and hypothyroidism. Correct electrolyte abnormalities, monitor digoxin levels at least 4 hr after IV dose, monitor for clinical signs of toxicity. Hypokalemia, hypomagnesemia, and hypercalcemia may precipitate digitalis toxicity. Reduce digoxin dose by 50% in patients on amiodarone.

Digoxin Immune FAB (Fragment Antigen Binding) (DigiFab)

Class: Antidote to digoxin and digitoxin
Indications: Symptomatic digoxin toxicity or acute ingestion of unknown amount of digoxin.

Adult Dose: Depends on serum digoxin levels. One 40-mg vial binds to approximately 0.5 mg of digoxin. Dose is typically administered over 30 min.

Contraindications: Allergy only, otherwise none known. Allergy to sheep proteins or other sheep products.
Side Effects: Worsening of CHF, rapid ventricular response in patients with A-fib, hypokalemia, postural hypotension, increased serum digoxin levels due to bound complexes (clinically misleading because bound complex cannot interact with receptors).
Precautions: Heart failure, renal impairment.

Diltiazem (Cardizem)

Class: Calcium channel blocker, class IV antiarrhythmic
Indications: To control ventricular rate in A-fib and A-flutter; to terminate PSVT (reentry SVT) refractory to adenosine with narrow QRS complex and adequate BP.

Adult Dose: 15–20 mg (0.25 mg/kg) IV given over 2 min. May repeat in 15 min at 20–25 mg (0.35 mg/kg) IV given over 2 min. Start maintenance drip at 5–15 mg/hr and titrate to HR and BP.

Contraindications: Drug- or poison-induced tachycardia, wide-complex tachycardia of uncertain origin, rapid A-fib and A-flutter with Wolff-Parkinson-White syndrome, sick sinus syndrome, second- or third-degree AV block (unless a functioning pacemaker is present), hypotension with systolic BP less than 90 mm Hg, acute MI with pulmonary congestion.
Side Effects: Hypotension, bradycardia (including AV block), chest pain, ventricular arrhythmias, peripheral edema, flushing, heart failure, syncope.
Precautions: Severe hypotension in patients receiving beta blockers. Caution in patients with hepatic or renal disease, heart failure, hypertrophic cardiomyopathy.

Dobutamine

Class: Direct-acting beta$_1$ adrenergic agonist, inotrope
Indications: To increase myocardial contractility in patients with decompensated heart failure with systolic BP 70–100 mm Hg and no signs of shock.

Adult Dose: Continuous infusion (titrate to patient response): 2–20 mcg/kg/min, maximum 40 mcg/kg/min.

Pediatric Dose: Same as adult dose, titrate to patient response.

Contraindications: Hypersensitivity, idiopathic hypertrophic subaortic stenosis (IHSS), suspected or known poison- or drug-induced shock. Do not mix with sodium bicarbonate.
Side Effects: Tachycardia, HTN, hypotension, increased ventricular ectopy, chest pain, palpitations, restlessness, headache, nausea, vomiting.

Continued

Precautions: Avoid in patients with systolic BP <100 mm Hg and signs of shock; correct hypovolemia before use, if needed. MI: may increase myocardial oxygen demand.

Dopamine (Intropin)

Class: Alpha-beta$_1$ adrenergic agonist, inotrope, vasopressor
Indications: Symptomatic bradycardia and hypotension, cardiogenic shock.

Adult Dose: Continuous infusion (titrate to patient response): low dose 1–5 mcg/kg/min (renal dose); moderate dose 5–15 mcg/kg/min (cardiac dose); high dose >15 mcg/kg/min (vasopressor dose). Mix 400 mg/250 mL in normal saline, lactated Ringer's solution, or D5W (1600 mcg/mL).

Pediatric Dose: *Cardiogenic shock, distributive shock:* 2–20 mcg/kg/min IV/IO infusion, titrate to desired effect.

Contraindications: Hypersensitivity to sulfites, pheochromocytoma, VF.
Side Effects: Tachyarrhythmias, angina, hypotension, palpitations, vasoconstriction, dyspnea, headache, nausea and vomiting.
Precautions: Hypovolemia, MI. Adjust dosage in elderly patients and in those with occlusive vascular disease. Ensure adequate IV volume repletion with normal saline before infusion. Taper slowly. Do not mix with sodium bicarbonate. Use care with peripheral administration; infiltration with extravasation can cause tissue necrosis. A central line is preferred. Use a volumetric infusion pump. Caution in patients with occlusive vascular disease and patients taking MAO inhibitors.

Epinephrine (Adrenalin)

Class: Alpha-beta adrenergic agonist (sympathomimetic: inotrope, vasopressor, bronchodilator)
Indications: Cardiac arrest: PEA, asystole, pulseless VT, VF; hypotension with severe bradycardia. Anaphylaxis, severe asthma exacerbation.

Adult Dose: *Cardiac arrest:* 1 mg IV/IO (10 mL of 1:10,000 solution) every 3–5 min prn; follow each dose with 20 mL IV flush. Give 2.0–2.5 mg (1:1,000) diluted in 5–10 mL normal saline or sterile water if administering by ET. *Profound bradycardia or hypotension:* 2–10 mcg/min IV infusion; add 1 mg (1 mL of a 1:1,000 solution) to 500 mL normal saline or D5W. *Anaphylaxis:* 0.2–0.5 mg (1:1,000 solution) IM (1:1,000 solution) every 5–15 min prn or 0.1–0.25 mg (1:10,000 solution) IV every 5–15 min, then 1–4 mcg/min IV prn. *Severe asthma exacerbation:* 0.3–0.5 mg (1:1,000 solution) SQ/IM every 20 min × 3 doses prn. Maximum 1 mg/dose.

Pediatric Dose: *Cardiac arrest or symptomatic bradycardia:* 0.01 mg/kg (0.1 mL/kg) 1:10,000 IV/IO every 3–5 min as needed (maximum dose: 1 mg or 10 mL). Give 0.1 mg/kg (0.1 mL/kg) 1:1,000, flush with 5 mL normal saline if administering by ET. Use ET route only if IV/IO access is not available. Repeat every 3–5 min as needed. *Anaphylaxis:* 0.01 mg/kg (1:1,000 solution) SQ/IM every 5–20 min × 3 doses prn or 0.01 mg/kg (1:10,000 solution) IV × 1, then 0.1 mcg/kg/min IV prn. *Severe asthma exacerbation:* 0.01 mg/kg (1:1,000 solution) SQ/IM every 20 min × 3 doses prn. Maximum 0.5 mg/dose.

Contraindications: Hypersensitivity to adrenergic amines, hypovolemic shock, coronary insufficiency. No contraindication in cardiac arrest.

Side Effects: Angina, HTN, tachycardia, VT, VF, nervousness, restlessness, palpitations, tremors, weakness, diaphoresis, anxiety, headache, nausea.

Precautions: Use caution in HTN and increasing heart rate (may cause increased myocardial oxygen demand). Higher doses can contribute to post-arrest cardiac impairment but may be needed to treat poison- or drug-induced shock. Avoid mixing with alkaline solutions.

Fibrinolytic Agents

Class: Thrombolytic, fibrinolytic

Common Agents: Alteplase (Activase, rt-PA), reteplase (Retavase), streptokinase (Streptase), tenecteplase (TNKase).

Indications: Acute ST elevation MI with onset within 12 hr. Alteplase is the only fibrinolytic agent approved for acute ischemic stroke and must be started less than 3 hr from the onset of symptoms.

Adult Dose: See individual order and drug for route and dosage.

Contraindications: Active internal bleeding within 21 days (except menses), neurovascular event within 3 months, major surgery or trauma within 2 weeks, aortic dissection, severe (uncontrolled) HTN, bleeding disorders, prolonged CPR, lumbar puncture within 1 week. History of any intracranial bleeding, oral anticoagulation therapy, severe stroke.

Side Effects: Hypotension, reperfusion arrhythmias, heart failure, headache, increased bleeding time, deep or superficial hemorrhage, flushing, urticaria, anaphylaxis.

Precautions: Use cautiously in patients with severe renal or hepatic disease. Initiate bleeding precautions. Monitor patient for bleeding complications.

Fondaparinux (Arixtra)

Class: Factor Xa inhibitor, anticoagulant

Indications: To inhibit thrombin generation by inhibiting factor Xa in patients with ACS; anticoagulation in patients with history of heparin-induced

Continued

thrombocytopenia (HIT); deep-vein thrombosis (DVT) prophylaxis in patients undergoing orthopedic surgery or abdominal surgery; pulmonary embolism (PE); acute DVT without PE.

Adult Dose: *STEMI*: 2.5 mg IV bolus followed by 2.5 mg SQ daily for up to 8 days. *Acute DVT/PE, acute thrombosis*: 5–10 mg SQ daily (based on body weight) up to 5–9 days, start coumadin therapy on first or second day, discontinue fondaparinux when INR ⊠2 for at least 24 hr. *Other uses*: 2.5 mg SQ daily for up to 8 days (up to 10 days for abdominal surgery, up to 11 days for hip replacement or total knee replacement surgery, up to 14 days for total hip or total knee arthroplasty or hip fracture surgery).

Contraindications: Creatinine clearance <30 mL/min, hypersensitivity, body weight <50 kg when used for prophylaxis, active major bleeding, bacterial endocarditis, thrombocytopenia associated with positive *in vitro* test for antiplatelet antibody in presence of fondaparinux.

Side Effects: Bleeding, edema, hypotension, insomnia, dizziness, headache, rash, constipation, vomiting, diarrhea, urinary retention, moderate thrombocytopenia.

Precautions: Increased risk for bleeding, creatinine clearance 30–50 mL/min, patients >75 years old, patients <50 kg being treated for DVT/PE. Discontinue 24 hr before CABG and administer unfractionated heparin.

Furosemide (Lasix)

Class: Loop diuretic

Indications: CHF with acute pulmonary edema, hypertensive crisis, post-arrest cerebral edema, edema associated with hepatic or renal disease.

Adult Dose: 0.5–1 mg/kg IV given over 1–2 min; may repeat at 2 mg/kg IV given over 1–2 min. Alternative: 20–40 mg IV, increase by 20 mg IV every 2 hr until desired response is obtained, maximum 160–200 mg/dose.

Pediatric Dose: 0.5–1 mg/kg IV/IO, may increase by 1 mg/kg IV every 2 hr until desired response is obtained, maximum 6 mg/kg/dose.

Contraindications: Hypersensitivity (cross-sensitivity with thiazides and sulfonamides may occur), uncontrolled electrolyte imbalance, hepatic coma, anuria, hypovolemia.

Side Effects: Severe dehydration, hypovolemia, hypotension, hypokalemia, hyponatremia, hypochloremia, hyperglycemia, dizziness, ototoxicity.

Precautions: Use cautiously in severe liver disease accompanied by cirrhosis or ascites, electrolyte depletion, diabetes mellitus, pregnancy, lactation, severe renal disease, gout. Risk for ototoxicity with increased dose or rapid injection. Monitor electrolytes closely.

Glycoprotein IIb/IIIa Inhibitors

Class: Antiplatelet agents, GP IIb/IIIa inhibitors
Common Agents: Abciximab (ReoPro), eptifibatide (Integrilin), tirofiban (Aggrastat)
Indications: Acute coronary syndromes that are managed medically (Eptifibatide and Tirofiban) and patients undergoing PCI (all three agents).

Adult Dose: See individual order and drug for dosage.

Contraindications: Hypersensitivity, active internal bleeding or bleeding disorder within past 30 days, history of bleeding diathesis, history of stroke within 30 days, history of hemorrhagic stroke, uncontrolled HTN (systolic BP >200 mm Hg, diastolic pressure >110 mm Hg), major surgery or trauma within 1 month, concomitant use of another GP IIb/IIIa inhibitor, dependency on hemodialysis.
Side Effects: Bleeding, hypotension, thrombocytopenia.
Precautions: Patients at increased risk for bleeding, patients <70 kg, platelet count <150,000/mm^3, renal impairment. Discontinue 2–4 hr prior to CABG. Abciximab (ReoPro) must be administered with aspirin and heparin.

Heparin (Unfractionated Heparin [UFH])

Class: Anticoagulant
Indications: Acute coronary syndromes (ACS): STEMI, NSTEMI, unstable angina (UA), during PCI; prophylaxis and treatment of thromboembolic disorders such as DVT, pulmonary embolus; anticoagulant for extracorporeal and dialysis procedures.

Adult Dose: *ACS*: 60 units/kg IV bolus, maximum 4,000 units, followed by continuous IV infusion of 12 units/kg/hr, maximum 1,000 units/hr, check APTT every 4–6 hr, adjust infusion to maintain APTT 50–70 sec for 48 hr or until angiography. *Thromboprophylaxis*: 5,000 units SQ every 8–12 hr. Treatment of DVT/PE: 80 units/kg or 5,000 unit IV bolus, followed by continuous IV infusion of 18 units/kg/hr; adjust infusion to maintain therapeutic APTT.

Pediatric Dose: *Systemic heparinization for infants <1 yr*: 75 units/kg IV over 10 min, followed by initial maintenance infusion of 28 units/kg/hr; check APTT every 4 hr and adjust heparin dose to maintain APTT 60–85 sec. *Children >1 yr*: 75 units/kg over 10 min, followed by initial maintenance infusion of 20 units/kg/hr, adjust heparin to maintain APTT 60–85 sec.

Contraindications: Hypersensitivity, heparin-induced thrombocytopenia (HIT), severe thrombocytopenia, uncontrolled active bleeding unless due to DIC; recent intracranial, intraspinal, or eye surgery; uncontrolled HTN.
Side Effects: Bleeding, HIT, thrombocytopenia, hyperkalemia, osteoporosis with use >6 months.

Continued

Precautions: Patients at increased risk for bleeding; patients with heparin resistance (antithrombin deficiency, increased heparin clearance, elevations in heparin-binding proteins, elevations in factor VIII and/or fibrinogen). Female patients >60 may require lower doses. Check platelet count daily.

Ibutilide (Corvert)

Class: Antiarrhythmic, class III
Indications: SVT, including A-fib and A-flutter; most effective for conversion of A-fib or A-flutter of short duration (⊠48 hr).

Adult Dose: *Patients weighing 60 kg or more:* 1 mg IV given over 10 min; may repeat the same dose in 10 min if arrhythmia does not terminate. *Patients weighing less than 60 kg:* 0.01 mg/kg IV given over 10 min; may repeat the same dose in 10 min if arrhythmia does not terminate.

Contraindications: Known hypersensitivity, history of polymorphic VT, QTc greater than 440 msec.
Side Effects: Nonsustained or sustained monomorphic or polymorphic VT, torsade de pointes, AV block, CHF, HTN, headache, tachycardia, hypotension, nausea and vomiting.
Precautions: Continuous ECG monitoring for 4–6 hr after administration or until QTc returns to baseline. Monitor for AV block. Skilled personnel and resuscitative equipment must be readily available. Correct electrolyte abnormalities prior to use. If A-fib has lasted longer than 48 hr, anticoagulation is required before cardioversion with ibutilide. Monitor QTc. Not recommended for chronic atrial fibrillation. Caution in patients with heart failure or hepatic impairment.

Isoproterenol (Isuprel)

Class: Beta adrenergic agonist
Indications: Medically refractory symptomatic bradycardia when transcutaneous or transvenous pacing is not available, refractory torsade de pointes unresponsive to magnesium, bradycardia in heart transplant patients, beta blocker poisoning.

Adult Dose: IV infusion: mix 1 mg/250 mL in normal saline, lactated Ringer's solution, or D5W, run at 2–10 mcg/min, and titrate to patient response. In torsade de pointes, titrate to increase heart rate until VT is suppressed.

Contraindications: Hypersensitivity to drug or sulfites, digitalis intoxication, angina, tachyarrhythmias, concurrent use with epinephrine (can cause VF or VT).
Side Effects: Arrhythmias, cardiac arrest, hypotension, angina, anxiety, tachycardia, palpitations, skin flushing, palpitations, dizziness, tremors, headache, nausea, vomiting, restlessness.

Precautions: May increase myocardial ischemia. Use caution in patients with renal impairment, cardiovascular disease, distributive shock, hyperthyroidism, diabetes. High doses are harmful except in beta blocker overdose.

Lidocaine (Xylocaine)

Class: Class Ib Antiarrhythmic, local anesthetic
Indications: Alternative to amiodarone in VF or pulseless VT. Use in stable VT, wide-complex tachycardia of uncertain origin.

Adult Dose: *Cardiac arrest from VF or VT:* 1.0–1.5 mg/kg IV/IO (or 2–4 mg/kg via ET); may repeat 0.5–0.75 mg/kg IV/IO every 5–10 min, maximum dose 3 mg/kg. *Stable VT, wide-complex tachycardia of uncertain origin:* 0.50–0.75 mg/kg up to 1.0–1.5 mg/kg; may repeat 0.50–0.75 mg/kg every 5–10 min, maximum total dose 3.0 mg/kg. If conversion is successful, start an IV infusion of 1–4 mg/min (30–50 mcg/kg/min) in normal saline or D5W.

Pediatric Dose: 1 mg/kg IV/IO bolus. Give 2–3 mg/kg, flush with 5 mL normal saline if administering by ET. Use ET route only if IV/IO access is not available. Maintenance: 20–50 mcg/kg/min IV/IO infusion (repeat bolus [0.5–1 mg/kg IV/IO] when infusion is initiated if bolus has not been given within previous 15 min).

Contraindications: Prophylactic use in acute MI, advanced AV block without functioning pacemaker, hypotension, Wolff-Parkinson-White syndrome, hypersensitivity to amide local anesthetics.
Side Effects: Confusion, agitation, anxiety, tinnitus, blurred vision, dizziness, tremors, hallucinations, seizures, hypotension, bradycardia, cardiovascular collapse, respiratory arrest, slurred speech.
Precautions: CHF, respiratory depression, shock. Reduce maintenance dose (not loading dose) in presence of impaired liver function or left ventricular dysfunction or in the elderly. Stop infusion if signs of CNS toxicity develop.

Magnesium Sulfate

Class: Electrolyte, antiarrhythmic
Indications: Torsade de pointes, hypomagnesemia, life-threatening ventricular arrhythmias due to digitalis toxicity, status asthmaticus, seizures.

Adult Dose: *Torsade de pointes (cardiac arrest):* 1–2 g IV (2–4 mL of a 50% solution) diluted in 10 mL of D5W over 1–2 min. *Torsade de pointes (non–cardiac arrest with pulse):* load with 1–2 g mixed in 50–100 mL of D5W infused over 5–60 min IV, then infuse 0.5–1.0 g/hr IV (titrate to control torsade). *Seizures: 2 g IV diluted in 10 mL of D5W over 10 min.*

Continued

Pediatric Dose: *Torsade de pointes (cardiac arrest—pulseless VT):* 25–50 mg/kg IV/IO bolus. *Torsade de pointes (non–cardiac arrest with pulses):* 25–50 mg/kg IV/IO over 10–20 min. *Status asthmaticus:* 25–50 mg/kg/IV/IO over 15–30 min.

Contraindications: Hypermagnesemia, hypocalcemia, AV block.
Side Effects: HTN, bradycardia, cardiac arrest, respiratory depression, altered LOC, flushed skin, diaphoresis, hypocalcemia, hyperkalemia, hypophosphatemia.
Precautions: Renal insufficiency, occasional fall in BP with rapid administration. Monitor serum magnesium levels. Caution in patients with myasthenia gravis. Correct concurrent hypokalemia and hypocalcemia.

Naloxone (Narcan)

Class: Opioid antagonist
Indications: Reversal of opioid overdose/toxicity unresponsive to oxygen and ventilator support, such as respiratory and neurological depression.

Adult dose: *Opioid overdose*: 2 mg IV, IM or SQ, may need to repeat every 2–3 min up to 10 mg. Reversal of respiratory depression with therapeutic opioid doses: 0.04–0.4 mg IV, IM or SQ, may repeat until ventilation is adequate, up to 0.8 mg. *Postoperative reversal*: 0.1–0.2 mg IV every 2–3 min until adequate ventilation.

Pediatric dose: *For total reversal, birth to 5 yr*: 0.1 mg/kg IV every 2–3 min as needed, maximum 2 mg; *>5 yrs*: 2 mg IV every 2–3 min prn up to 10 mg. *For partial reversal*: 0.001–0.005 mg/kg IV (1–5 mcg/kg), repeat as needed every 2–3 min. For postoperative reversal: 0.01 mg/kg IV every 2–3 min prn.

Contraindications: Hypersensitivity, meperidine-induced seizures.
Side Effects: Secondary to reversal (withdrawal) of narcotic analgesia and sedation. Recurrent respiratory depression, pain, hypertension, hypotension, irritability, agitation, diaphoresis, seizures.
Precautions: May precipitate symptoms of acute withdrawal in opioid-dependent patients. Use caution in patients with a history of seizures and patients with cardiovascular disease. Abrupt postoperative reversal may cause nausea, vomiting, diaphoresis, tachycardia, hypertension, seizures, pulmonary edema, arrhythmias.

Morphine Sulfate

Class: Opiate narcotic analgesic
Indications: Chest pain unrelieved by nitroglycerin; CHF and dyspnea associated with pulmonary edema.

Adult Dose: 2–4 mg IV (given over 1–5 min), administer every 5–30 min as needed if hemodynamically stable; may repeat dose of 2–8 mg at 5- to 15-minute intervals if needed.

Contraindications: Hypersensitivity, heart failure due to chronic lung disease, respiratory depression, hypercarbia, hypotension, bowel obstruction, severe asthma, acute or severe hypercarbia. Avoid in patients with RV infarction.

Side Effects: Respiratory depression, hypotension, nausea and vomiting, bradycardia, altered LOC, seizures, somnolence, dizziness, diaphoresis, flushing, pruritus, dry mouth, urinary retention.

Precautions: Administer slowly and titrate to effect. Reverse with naloxone (0.4–2.0 mg IV) if necessary. Use caution in cerebral edema and pulmonary edema with compromised respiration. Use caution with hypovolemic patients; be prepared to administer volume. Use caution in renal and hepatic impairment, seizure disorder, CNS depression, head injury, hypothyroidism, adrenal insufficiency, prostatic hypertrophy, shock.

Nitroglycerin (Nitrostat, Nitrolingual [Pump Spray])

Class: Antianginal, nitrate, vasodilator

Indications: Acute coronary syndrome, angina, CHF associated with acute MI, hypertensive urgency with ACS.

Adult Dose: Sublingual route, 0.3–0.4 mg (1 tablet); repeat every 3–5 min if chest pain is not relieved, maximum 3 doses/15 min. Aerosol, spray for 0.5–1.0 sec at 3- to 5-min intervals (provides 0.4 mg/dose), maximum 3 sprays/15 min. IV bolus administration at 12.5–25.0 mcg (if no sublingual or spray used). IV infusion: mix 25 mg/250 mL (100 mcg/mL) in D5W, start at 5 mcg/min and titrate by 5 mcg/min every 3–5 min to 20 mcg/min. If patient remains symptomatic, titrate by 10–20 mcg/min every 3–5 min, maximum 200 mcg/min.

Pediatric Dose: 0.25–0.50 mcg/kg/min IV/IO infusion, titrate by 0.5–1 mcg/kg/min every 3–5 min as needed to typical dose range of 1–5 mcg/kg/min (max.20 mcg/kg/min).

Contraindications: Hypersensitivity, systolic BP less than 90 mm Hg, pericardial tamponade, constrictive pericarditis, severe bradycardia or severe tachycardia associated with hypotension; sildenafil (Viagra) or vardenafil (Levitra) within 24 hr, tadalafil (Cialis) within 48 hr; right ventricular infarction, increased intracranial pressure, hypertrophic cardiomyopathy with outflow tract obstruction, restrictive cardiomyopathy, increased intracranial pressure.

Side Effects: Hypotension with reflex tachycardia, syncope, headache, flushed skin, dizziness, paradoxical bradycardia.

Continued

Precautions: Do not mix with other medications; titrate IV to maintain systolic BP above 90 mm Hg. Mix only in glass IV bottles and infuse only through non-PVC tubing; standard polyvinyl chloride (PVC) tubing can bind up to 80% of the medication, making it necessary to infuse higher doses. Do not shake aerosol spray (affects metered dose).

Norepinephrine (Levophed)

Class: Alpha-beta adrenergic agonist, vasopressor
Indications: Treatment of persistent shock after adequate volume replacement, cardiogenic shock, low systemic vascular resistance shock, septic shock, hemodynamically significant hypotension.

Adult Dose: Start at 0.1–0.5 mcg/kg/min, titrate to response up to 8–12 mcg/min, maintenance infusion usually 2–4 mcg/min. Use volumetric infusion pump.

Pediatric Dose: Start at 0.05–0.1 mcg/kg/min, titrate to response, up to 2 mcg/kg/min. Use volumetric infusion pump.

Contraindications: Hypovolemic shock prior to adequate volume replacement, mesenteric or peripheral vascular thrombosis except as emergency measure to maintain coronary and cerebral perfusion. Do not administer in same line as alkaline solutions.
Side Effects: Arrhythmias, hypertension, headache, anxiety, dyspnea, skin necrosis with extravasation.
Precautions: Use caution in patients on MAO inhibitors because drug may cause prolonged hypertension; infuse into large vein and avoid extravasation; use caution in patients with ischemic heart disease: increases myocardial oxygen consumption, may induce arrhythmias, tachycardia, hypertension.

Oxygen

Class: Gas
Indications: Cardiopulmonary emergencies with shortness of breath and chest pain, cardiac or respiratory arrest, hypoxemia. Used to optimize oxygen saturation <94%.

Adult and Pediatric Dose: Nasal cannula 1–6 L/min (21%–44% oxygen), Venturi mask 4–12 L/min (24%–50% oxygen), simple mask 5–8 L/min (40%–60% oxygen), partial rebreathing mask 6–10 L/min (35%–60% oxygen), non-rebreathing mask 6–15 L/min (60%–100% oxygen), bag-valve mask 15 L/min (95%–100% oxygen).

Contraindications: None reported.
Side Effects: Drying of respiratory mucosa, possible bronchospasm if oxygen is extremely cold and dry. Oxygen supports combustion and can fuel a fire.

Hypoventilation in patients with severe COPD, pulmonary fibrosis, oxygen toxicity.

Precautions: Respiratory arrest in patients with hypoxic respiratory drive. The patient needs an airway and adequate ventilation before oxygen is effective.

Procainamide (Pronestyl)

Class: Antiarrhythmic, class I_a

Indications: Recurrent VT or VF, PSVT refractory to adenosine and vagal stimulation, rapid A-fib with Wolff-Parkinson-White syndrome, stable wide-complex tachycardia of uncertain origin, maintenance after conversion. Stable monomorphic VT with normal QTc and preserved LV function.

Adult Dose: 20 mg/min IV infusion or up to 50 mg/min under urgent conditions, until arrhythmia is suppressed, maximum 17 mg/kg loading dose. Maintenance IV infusion: mix 1 g/250 mL (4 mg/mL) in normal saline or D5W, run at 1–4 mg/min.

Pediatric Dose: *Atrial flutter, SVT, VT (with pulses):* 15 mg/kg IV/IO load over 30–60 min.

Contraindications: Hypersensitivity, second- and third-degree AV block (unless a functioning pacemaker is in place), prolonged QT interval, torsade de pointes, hypersensitivity, systemic lupus erythematosus.

Side Effects: Hypotension, widening QRS, headache, nausea and vomiting, flushed skin, seizures, ventricular arrhythmias, AV block, cardiovascular collapse, arrest.

Precautions: Monitor BP every 2–3 min while administering procainamide. If QRS width increases by 50% or more, or if systolic BP decreases to less than 90 mm Hg, stop the drug. Monitor for prolonged PR interval and AV block. Monitor for QT prolongation. May precipitate or exacerbate CHF. Reduce the total dose to 12 mg/kg and maintenance infusion to 1–2 mg/min if cardiac or renal dysfunction is present. Use cautiously in heart failure, myasthenia gravis, and hepatic or renal disease. Avoid concurrent use with drugs that prolong the QT interval (e.g., amiodarone, sotalol).

Sodium Bicarbonate

Class: Alkalinizing agent, buffer

Indications: Known preexisting hyperkalemia, bicarbonate-responsive acidosis such as DKA or tricyclic antidepressant overdose, metabolic acidosis associated with prolonged resuscitation with effective ventilation.

Continued

Adult Dose: 1 mEq/kg IV; may repeat 0.5 mEq/kg every 10 min. Dosing is best guided by calculated base deficits or bicarbonate concentration with arterial blood gas analysis if available.

Pediatric Dose: 1 mEq/kg IV/IO slow bolus. Dosing is best guided by calculated base deficits or bicarbonate concentration with arterial blood gas analysis if available.

Contraindications: Metabolic and respiratory alkalosis, hypochloremia, hypocalcemia, hypokalemia, hypercarbic acidosis, hypernatremia, severe pulmonary edema.

Side Effects: Hypokalemia, hypocalcemia, hypernatremia, metabolic alkalosis, edema, seizures, tetany, exacerbation of CHF, tissue hypoxia, intracellular acidosis.

Precautions: CHF, renal disease, cirrhosis, hypernatremia, hypervolemia, toxemia, concurrent corticosteroid therapy. Not recommended for routine use in cardiac arrest because adequate ventilation and CPR are the major "buffer agents" in this case. Incompatible with many drugs; flush the line before and after administration.

Vasopressin (Pitressin)

Class: Vasopressor, hormone

Indication: Cardiac arrest: an alternative to epinephrine in shock-refractory VF and pulseless VT, PEA, and asystole. Vasodilatory shock/septic shock.

Adult Dose: *Cardiac arrest:* 40 units IV/IO single dose to replace first or second dose of epinephrine as an alternative. *Vasodilatory shock:* 0.01–0.04 units/min continuous IV infusion.

Pediatric Dose: *Cardiac arrest:* 0.4–1 unit/kg IV/IO, maximum 40 units. *Vasodilatory shock:* 0.0002–0.002 unit/kg/min continuous IV infusion.

Contraindications: Hypersensitivity.

Side Effects: Bradycardia, HTN, angina, MI, arrhythmias, dizziness, headache, nausea and vomiting, abdominal cramps, diaphoresis, bronchoconstriction, anaphylaxis.

Precautions: Coronary artery disease (may precipitate angina or MI), CHF, hepatic or renal impairment; seizure disorders, asthma, vascular disease.

Verapamil (Calan, Isoptin)

Class: Calcium channel blocker, class IV antiarrhythmic, antihypertensive

Indications: PSVT (with narrow QRS and adequate BP) refractory to adenosine; rapid ventricular rates in A-fib, A-flutter, and MAT.

Adult Dose: 2.5–5.0 mg IV over 2 min; may give second dose, if needed, of 5–10 mg IV in 15–30 min, maximum dose 20 mg. An alternative second dose is 5 mg IV every 15 min, maximum dose 30 mg.

Contraindications: A-fib with Wolff-Parkinson-White syndrome, wide-complex tachycardia of uncertain origin, second- or third-degree AV block (unless a functioning pacemaker is in place), sick sinus syndrome, hypotension, severe CHF, cardiogenic shock, concurrent IV beta blocker, VT.

Side Effects: Hypotension, exacerbation of CHF with left ventricular dysfunction, bradycardia, AV block, constipation, peripheral edema, headache, dizziness, fatigue, paralytic ileus, hepatotoxicity.

Precautions: Concurrent oral beta blockers, CHF, impaired hepatic or renal function, myasthenia gravis, muscular dystrophy, hypertrophic cardiomyopathy with outflow tract obstruction; may decrease myocardial contractility. In geriatric patients administer slowly over 3 min.

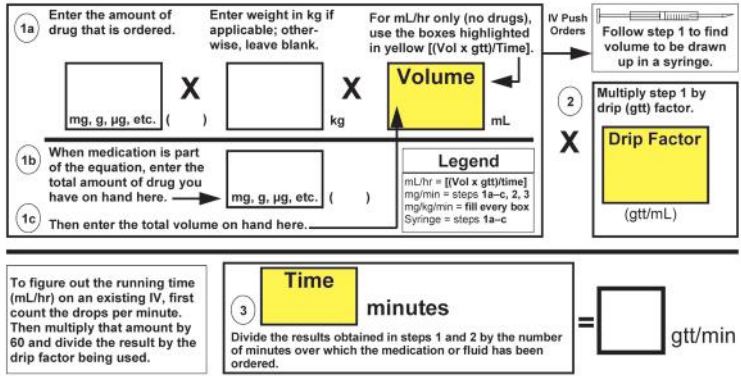
Common Medication Formulas

Syringe: Amount to be drawn up	$\frac{\text{Desired dose of drug} \times \text{Total volume}}{\text{Total dose of drug on hand}}$
IV: Calculating gtt/min	$\frac{\text{Volume to be infused} \times \text{Drop (gtt) factor}}{\text{Total time in minutes}}$
IV: Calculating infusion rate (mg/min or mcg/min)	$\frac{\text{Volume on hand} \times \text{gtt factor} \times \text{Desired dose}}{\text{Total dose of drug on hand}} = \text{gtt/min}$ Example: Administer 2 mg/min of lidocaine. To prepare the infusion mix 2 g (2,000 mg) of lidocaine in 500 mL of D5W with a drip set of 60 gtt/mL. Calculate the infusion rate. $\frac{500\text{ mL} \times 60\text{ gtt/mL} \times 2\text{ mg}}{2{,}000\text{ mg}} = 30\text{ gtt/min}$
IV: Rate of an existing IV in mL/hr	1. Count drops (gtt)/min and multiply by 60 min. 2. Divide result by the drop (gtt) factor being used.

IV Fluid Drip Rate Table (gtt/min)

Rate: (mL/hr)	TKO	50	75	100	125	150	175	200	250
10 gtt/mL set	5	8	13	17	21	25	29	33	42
12 gtt/mL set	6	10	15	20	25	30	35	40	50
15 gtt/mL set	8	13	19	25	31	37	44	50	62
20 gtt/mL set	10	17	25	33	42	50	58	67	83
60 gtt/mL set	30	50	75	100	125	150	175	200	250

Universal Formula—Figure Out Drip Rates and Drug Amounts



Note: The abbreviation mcg (microgram) means the same as µg (used in the above formula); mcg is the more commonly used to prevent medication errors.

Tab 6: Emergency Medical Skills

Electrical Therapy

Automated External Defibrillator

An automated external defibrillator (AED) is a small, lightweight device used by both professionals and laypersons to assess heart rhythm by computer analysis. Using voice and visual prompts, it administers an electric shock, if necessary, to restore a normal rhythm in patients with sudden cardiac arrest. A shock is administered only if the rhythm detected is VF or pulseless VT.

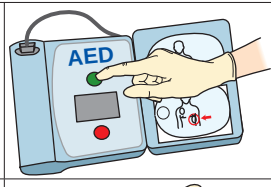
Indications: VF or pulseless VT in adults, children, and infants.

Energy Levels: The AED automatically selects the energy level for each defibrillation. Most devices are equipped with pediatric systems that include a **dose attenuator**. A dose attenuator is a pad–cable system or a key that delivers energy in a reduced dose suitable for children.

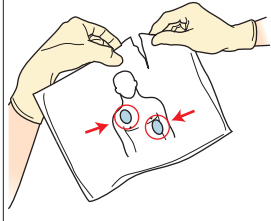
Emergency Actions

1. Verify that the patient is in cardiac arrest, with no pulse or respiration. Have someone provide CPR while the AED is obtained and placed next to the patient.

2. Power on the AED. Follow the voice prompts and visual messages.



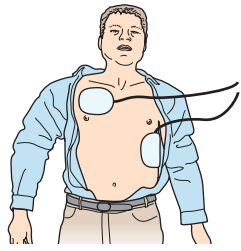
3. Open the package of adhesive electrode pads and attach pads to the person's bare chest.
   - Check for medication patches on the chest (use gloves).
   - Shave the chest (with razor in AED pack) if the person has chest hair where pads are placed.
   - Do not place pads over an implanted pacemaker or defibrillator.
   - If the chest is moist or wet, dry it so electricity will not arc when the shock is given.



Emergency Actions—cont'd

4. Use **adult pads for an adult and child pads for a child or infant.** If there are no child pads available, you may use adult pads on a child or infant, but be sure the pads do not touch each other (leave at least 1 in [2.54 cm] space between pads). Never use child pads on an adult; they do not supply enough energy.

5. Attach the sticky side of the pads directly to the person's bare chest.
   - Attach one pad to the right sternal border (superior–anterior right chest).
   - Place the second pad over the left apex (inferior–lateral left chest).
   - Alternatively, follow the diagrams on each of the AED electrodes.
   - On an infant or small child, you can place one pad on the anterior chest and the other pad on the posterior chest.



6. Connect the pad cables to the AED.

7. Clear the patient and stop CPR. Remove oxygen, if applicable.

8. The AED will automatically analyze the patient's rhythm or may be equipped with an "Analyze" button. If the latter, press the "Analyze" button.

9. If a shock is indicated and other people are present, warn them to keep clear. Also, make sure your body is not touching the person.
   - Say, "I'm going to shock on three. One, I'm clear; two, you're clear; three, everybody's clear."
   - Perform a visual sweep to ensure rescue personnel are not touching the patient or equipment and **oxygen is removed,** if applicable.

Continued

Emergency Actions—cont'd

10. If the AED is semiautomated, press the "Shock" button.

What to Do Next

- Once the shock is delivered, continue CPR beginning with chest compressions.
- After 2 min of CPR, the AED will prompt you with further verbal and visual cues.

♥ **Clinical Tip:** A fully automated AED analyzes the rhythm and delivers a shock, if one is indicated, without operator intervention once the pads are applied to the patient.

♥ **Clinical Tip:** A semiautomated AED analyzes the rhythm and tells the operator that a shock is indicated. If it is, the operator initiates the shock by pushing the shock button.

Manual Defibrillation

A manual defibrillator is used to restore a normal heart rhythm in VF or pulseless VT by delivering an electric shock to the heart, allowing the sinus node to regain control of the heart's electrical system.

Indications: VF or pulseless VT.

Energy Levels: If using an adult biphasic monitor-defibrillator, defibrillate shock at 120–200 J (use manufacturer's device-specific energy levels if known, or 200 J if unknown). Continue at a biphasic energy level of 120–200 J or escalate to higher energy for further shocks. **If using a monophasic defibrillator,** shock at 360 J. Continue at a monophasic energy level of 360 J.

Application: Dry moisture off skin; quickly shave excessive chest hair, if necessary. Use remote adhesive pads or handheld paddles. Always use a conducting gel with paddles and apply firm pressure (15–25 lb per paddle) to the chest to ensure good skin contact.

Methods: Methods may be manual or automated.

Precautions: Place pads or paddles several inches away from an implanted pacemaker or implanted cardioverter-defibrillator (ICD). Check for medication patches on the chest (use gloves).

Emergency Actions

1. Continue CPR while the defibrillator is obtained and placed next to the patient or left on the crash cart.
2. Turn on the device; verify that all cables are connected.
3. Turn the lead select switch to "paddles" or to the clearest "lead" if monitor leads are used.
   - **Paddles:** Place conducting gel or gel pads on the right sternal border (superior-anterior right chest), and place the second paddle over the left apex (inferior-lateral left chest). Apply firm pressure with both paddles.
   - **Pads:** Place in locations specified for paddles. Roll pads on from top to bottom edges to ensure complete gel contact with the skin and prevent air pockets.
4. **Remove oxygen from the patient and from the immediate vicinity.**
5. Select the initial energy level for an adult to a biphasic energy level of 120–200 J (use the manufacturer's device-specific energy levels if known, or 200 J if unknown) or a monophasic energy level of 360 J. For a child or infant, select the initial energy dose to 2 J/kg (acceptable range, 2–4 J/kg). Use subsequent doses of 4 J/kg or higher, not to exceed 10 J/kg or the standard adult dose.
6. Verify rhythm as VF or pulseless VT.
7. Say, "Charging defibrillator, stand clear!"
8. Charge the defibrillator.
9. Say, "I'm going to shock on three. One, I'm clear; two, you're clear; three, everybody's clear." Perform a visual sweep to ensure rescue personnel are not touching the patient or equipment and oxygen is removed, if applicable.
10. Discharge the defibrillator, reassess the rhythm, and refer to the appropriate ACLS protocol.

Clinical Tip: Oxygen increases the risk for fire and should be turned off before defibrillation.

Clinical Tip: Defibrillation may be used on infants (younger than 1 yr) and on children (1 yr to puberty). Use pediatric pads or paddles whenever possible and follow pediatric protocols. If pediatric pads or paddles are not available, use adult pads or paddles and ensure adequate spacing (at least 1 in [2.54 cm] between pads or paddles). For infants or children with small chests, place the sternal pad or paddle on the anterior chest. Place the apex pad or paddle with flat adapter on the posterior chest.

Cardioversion (Synchronized)

Indications: Unstable tachycardias with a ventricular rate >150 bpm with a perfusing rhythm. Immediate cardioversion is generally not needed for a heart rate of ☒150 bpm. The patient may present with an acutely altered LOC, angina, acute heart failure, hypotension, or signs of shock.

Energy Levels:

- Regular, narrow QRS tachycardia: 50–100 J biphasic is often sufficient. For a monophasic device, initial energy is 200 J. (SVT or A-flutter generally require less energy.)
- A-fib (irregular narrow QRS tachycardia): initial energy dose of 120–200 J biphasic or 200 J monophasic.
- Regular wide QRS tachycardia (monomorphic VT): usually responds well to initial energy of 100 J biphasic or monophasic.
- Initial pediatric energy dose: 0.5–1 J/kg with subsequent doses of 2 J/kg.

If there is no response to the first shock, it may be reasonable to increase the dose in steps. Synchronized cardioversion must not be used to treat VF. Because there is no QRS complex, the device may not reliably sense any waveform and may not deliver a shock. Synchronized cardioversion should also not be used for pulseless VT or polymorphic VT (irregular VT). These rhythms require high-energy unsynchronized shocks (i.e., defibrillation doses).

Application: Use remote adhesive pads or handheld paddles. Always use a conducting gel or gel pads with paddles. For conscious patients, explain the procedure and use medication for sedation and analgesia. Consider 2.5–5.0 mg of midazolam **(Versed),** 5.0 mg diazepam **(Valium),** fentanyl 1–2 mcg/kg/min IV, or anesthesia, if available.

Methods: Remove oxygen, if applicable. Place the defibrillator in synchronized (Sync) mode. Observe the R wave marker to confirm proper synchronization. Charge to the appropriate level. Say, "I'm going to shock on three. One, I'm clear; two, you're clear; three, everybody's clear." Perform a visual sweep and ensure oxygen is removed. Press and hold the shock button until a shock is delivered. If using paddles, press and hold both discharge buttons simultaneously until shock is delivered. Reassess the patient and treat according to the appropriate ACLS support protocol.

Precautions: Reactivate the "Sync" mode after each attempted cardioversion; defibrillators default to the unsynchronized mode after shock delivery. Place pads or paddles several inches away from an implanted pacemaker or ICD.

Clinical Tip: The "Sync" mode synchronizes energy delivery with the timing of the QRS complex. This avoids stimulation during the refractory, or vulnerable, period of the cardiac cycle, when a shock could potentially produce VF.

Transcutaneous Pacing

Indications: A temporizing measure for symptomatic bradycardia (with a pulse) unresponsive to atropine, bradycardia with ventricular escape rhythms, symptomatic second-degree AV block type II, or third-degree AV block.

Pacing Modes: *Demand-mode (synchronous)* pacemakers sense the person's heart rate and pace only when it falls below the level set by the clinician. *Fixed-mode (asynchronous)* pacemakers cannot sense the heart rate and always operate at the rate set by the clinician. Rate selections vary between 30 and 180 bpm. Output is adjustable between 0 and 200 mA. Pulse duration varies from 20–40 ms.

Application: Pacemaker pads work most effectively if placed in an anterior-posterior position.

Contraindications: Not effective in VF, pulseless VT, or asystole.

Side Effects: Chest muscle contraction, burns, and chest discomfort.

Precautions: Make sure pads have good skin contact to achieve capture and avoid burns.

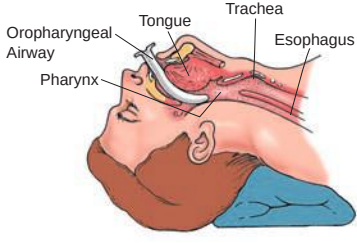
Airway Management

Oropharyngeal Airway

An oropharyngeal airway (OPA) is indicated for unconscious people who do not have a gag reflex and are at risk for developing airway obstruction from the tongue.

Emergency Actions

- Select an airway of the correct size. This is done by measuring from the earlobe to the corner of the mouth.
- Insert the airway: rotate it 180 degrees as it approaches the posterior wall of the pharynx, or insert it sideways into the mouth and turn it 90 degrees downward so it angles toward the posterior pharynx.

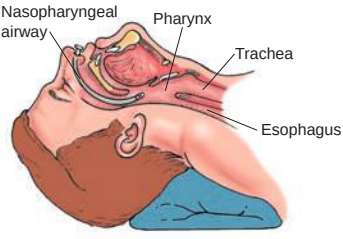


Nasopharyngeal Airway

A nasopharyngeal airway (NPA) is indicated for people who may have lockjaw, are comatose with spontaneous respirations, or have a gag reflex.

Emergency Actions

- Select an airway of the correct size. Measure from the earlobe to the tip of the nose.
- Never insert an NPA if the person has facial trauma.
- Lubricate the airway with a water-soluble lubricant.
- Insert the NPA by passing it gently along the floor of the nasopharynx with a slight rotation.



Laryngeal Mask Airway

An LMA is indicated for people who have no spontaneous respiration. It serves as an alternative to an ET airway. Only experienced providers should use this airway.

Laryngeal Tube Airway

A laryngeal tube airway, or King tube, is indicated for people who have no spontaneous respiration. It provides an alternative to an ET airway. Only experienced providers should use this airway.

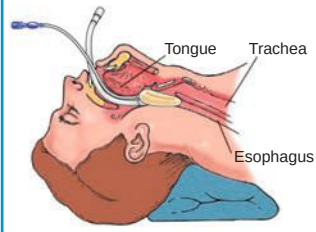
Esophageal–Tracheal Tube

An esophageal–tracheal tube, or Combitube airway, is indicated for people who have no spontaneous respiration. It serves as an alternative to an ET airway. Only experienced providers should use this airway.

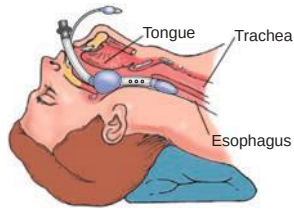
Endotracheal Tube Airway

An ET airway is indicated for people who have no spontaneous respiration. It is the most effective advanced airway and should only be used by experienced providers.

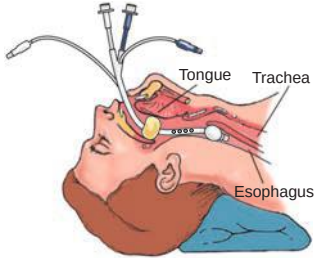
Laryngeal Mask Airway



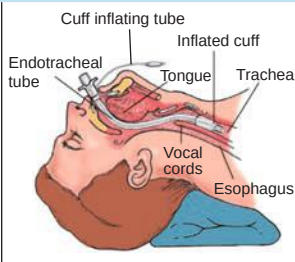
Laryngeal Tube Airway



Esophageal–Tracheal Tube



Endotracheal Tube Airway



Suction

Protecting the airway from blood, mucus, and vomitus is important regardless of the type of airway used. Suction, coupled with body position, provides a valuable tool for airway management. Suction employs negative pressure to withdraw foreign material from the posterior pharynx. Suction can also be used to remove blood and mucus from an ET or other such device.

Tonsil Tip

The tonsil tip, or Yankauer device, is rigid, allowing easy placement in the posterior pharynx. Larger in diameter, it can aspirate larger and thicker substances. The tonsil tip handle has a hole, and there are numerous vents near the tip. When the hole in the handle is left open, little or no suction is exerted at the tip of the strip. Occluding the hole with a thumb or finger initiates suction. The vents are placed near the tip to prevent tissue destruction in case the tip accidentally touches the delicate mucosa.

Suction Catheter

Because of its flexibility, a suction catheter is difficult to maneuver into the posterior pharynx, where secretions accumulate. Its smaller diameter causes it to become clogged more easily. Its primary use is to suction blood or mucus from ETs and other such devices. Suction catheters are also useful in children whose mouth and pharynx are too small to accommodate a tonsil tip.

Oxygen Delivery

Oxygen

In treating cardiopulmonary emergency (i.e., shortness of breath and chest pain, cardiac or respiratory arrest, hypoxemia), increasing oxygen concentration in the inspired air is an important factor. Supplemental oxygen is used to optimize oxygen saturation <94%.

Oxygen Delivery Devices

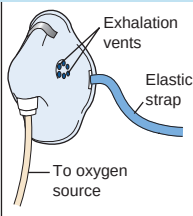
Several types of oxygen delivery devices are available. Selection is based on concentration of oxygen required, adequacy of the patient's ventilation, and patient tolerance.

Nasal Cannula

- Most people tolerate cannulas well.
- Cannulas may be used in all cases unless the patient's nostrils are obstructed.
- Because dead air space in the nasopharynx acts as an oxygen reservoir, there is no need to breathe through the nose; oxygen is entrained through the device even with mouth breathing.
- **Flow rates of 1–6 L/min deliver 21%–44% fraction of inspired oxygen (FiO_2).**

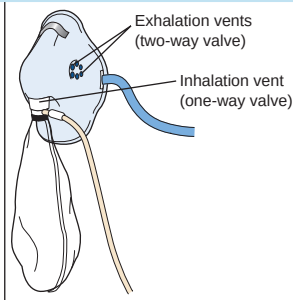
Simple Mask

- Face masks are used when a higher oxygen concentration is required or when nostrils are obstructed.
- Simple masks contain a delivery inlet, and small vents on either side of the mask allow exhaled gas to escape.
- **Flow rates of 5–8 L/min deliver 40%–60% FiO_2.**



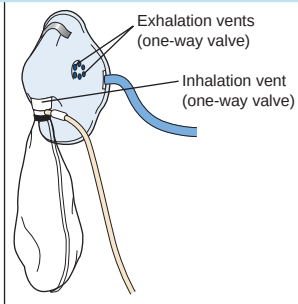
Partial Rebreathing Mask

- Partial rebreathing masks allow a maximum flow rate of 10 L/min.
- As the person inhales, oxygen is drawn up from the bag into the mask.
- Small vents on either side of the mask allow exhaled gas to escape.
- **Flow rates of 6–10 L/min deliver 35%–60% FiO_2.**



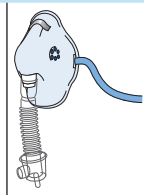
Nonrebreather Mask

- As the person inhales, oxygen is drawn up from the bag into the mask.
- A one-way valve directs oxygen into the mask from the bag during inhalation and prevents other gases from accumulating in the bag during exhalation.
- Flapper valves cover air vents in the mask and close when the person inhales.
- **A flow rate of 6 L/min delivers 60% FiO_2; a flow rate of 10–15 L/min delivers close to 100% FiO_2.**



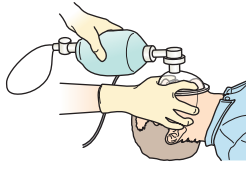
Venturi Mask

- This specialized mask enables delivery of precise oxygen concentrations.
- This delivery is valuable during transport of people, e.g., those with COPD, who are sensitive to oxygen.
- **Flow rates of 4–8 L/min deliver 24–40% FiO_2.**



Bag-Valve-Mask

- This type of mask is used if respirations are too slow or shallow to provide adequate ventilation.
- Supplemental oxygen must be delivered in tandem with supplemental ventilation.
- An oxygen reservoir must be used.
- **A flow rate of 10–15 L/min delivers close to 100% FiO_2.**



Oxygenation Assessment

Pulse Oximetry

A pulse oximeter is a simple, convenient technique for continuous monitoring of arterial oxygen saturation.

How to Use

- Clean sensor and finger with an alcohol swab.
- Place sensor on finger.
- Record reading.

Readings May be Affected by

- Movement, caused by twitching or agitation
- Dirty sensor
- Dark nail polish
- Vasoconstrictive drugs
- Fungal infection of the nails
- Cold hands and fingers

Critical Thinking: Although a pulse oximetry reading may be normal, cardiac output or hemoglobin levels may be low. In the field or clinical setting, always assess systemic perfusion and hemoglobin concentration.

Carbon Dioxide Assessment

Exhaled CO_2 is assessed in field and clinical settings to aid in correct placement of an ET. Evidence of exhaled of CO_2 almost always indicates that the lungs are being ventilated. Absence of exhaled CO_2 may be caused by esophageal intubation or may indicate cardiac arrest with no perfusion to the lungs (i.e., hemothorax or pneumothorax)

Capnometry

Capnometry detects exhaled CO_2 in the air by using colorimetric capnometers, devices that produce color on a strip of paper. The exhaled CO_2 reacts with a chemical detector in the paper. A CO_2 concentration of >2% reacts with the chemical reagent to change the color in a **colorimetric CO_2 detector**. It is important to read the manufacturer's instructions on which colors are "good" (successful intubation and ventilation) and which are "poor" (unsuccessful intubation or lack of ventilation). In the absence of exhaled CO_2 the color will remain unchanged.

Many advanced training materials suggest standardized colors, such as purple for low CO_2 and yellow for high CO_2. As new devices are introduced to the market, however, their color schemes still depend on the manufacturer. Always read the instructions.

Capnography

The most common **capnography devices** are infrared absorption detectors. Air flows past a sensor, usually attached to the top of the ET or LMA, that detects the amount of CO_2. The CO_2 concentration is typically displayed as a continuous exhaled waveform with a digital display of partial pressure of end-tidal CO_2 ($PETCO_2$). Capnography is also an early indicator of ROSC.

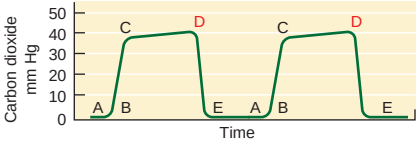
The waveform $PETCO_2$ device monitors CO_2 production continuously, whereas the colorimetric device provides only a single-point evaluation. The continuous waveform enables the healthcare provider to detect a dislodged ET minutes before the pulse oximeter registers a change. Also, the waveform device measures the quantity of CO_2, which correlates directly with cerebral perfusion. For

example, if CO_2 is less than 35 mm Hg, cerebral perfusion becomes impaired. This is critical in head injury patients and in any patients who have signs and symptoms of increased intracranial pressure.

The $PETCO_2$ tracing goes up on expiration and down (back to zero) on inspiration. The normal $PETCO_2$ is 35–45 mm Hg.

Normal $PETCO_2$

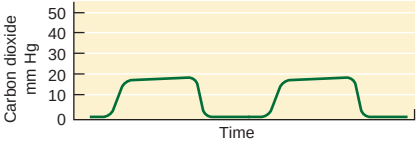
The upper tracing goes to 40 mm Hg.



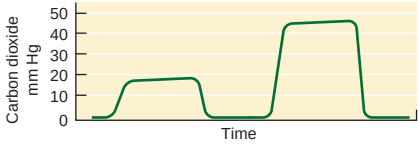
A–B: Beginning of exhalation
B–C: Continuation of exhalation
C–D: Final exhalation
D: $ETCO_2$ level (reading on the monitor)
D–E: Beginning of inhalation
E–A: Final inhalation

High-Quality Chest Compressions

High-quality chest compressions are achieved when $PETCO_2$ is at least 10–20 mm Hg. In cardiac arrest, good CPR delivers CO_2 to the lungs, so you will see a waveform with ventilations.



ROSC



When ROSC occurs, $PETCO_2$ increases significantly (35–45 mm Hg). This increase represents improvement in blood flow and a resulting increase in CO_2 entering the lungs.

Esophageal Detection Device

An **esophageal detection device** is a disposable tool that uses the anatomical differences between the trachea and the esophagus to verify proper ET placement. The trachea, because of the presence of tracheal rings, is like a hollow but noncollapsible tube. In contrast, the esophagus is like a balloon, which can collapse in the presence of a vacuum. When a vacuum pressure is applied to a tube in the esophagus, the esophagus literally collapses around the ET and prevents air from being aspirated from the tube (bulb does not fill). In the trachea, the rings prevent this collapse, and air is easily drawn back (bulb fills).

Access Routes for Drugs

Intravenous

A peripheral IV is the preferred method for drug or fluid administration unless central line access is already available. Starting a peripheral IV does not require interruption of CPR. The administration of drugs through an IV should be followed by a 20 mL IV flush. This facilitates drug delivery to the central circulation.

Intraosseous

If a peripheral IV cannot be started or is not accessible, the IO route is an effective and safe way to give IV drugs and fluids. IO access can be used in all age groups and is preferred over the ET route. Any ACLS drug or fluid that can be administered by IV can be given IO. As with the peripheral IV, follow a drug bolus with a 20-mL IV flush.

Endotracheal

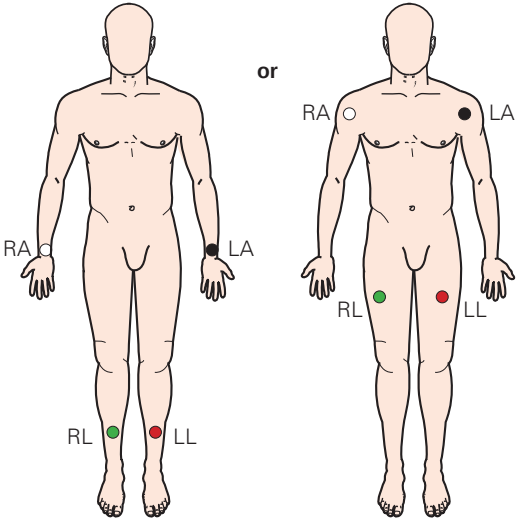
The endotracheal route is not the optimal method for delivering most drugs. Endotracheal absorption of drugs is poor, and a typical dose is 2–2.5 times the IV or IO dose. If giving drugs by ET, be sure to dilute the dose in 5–10 mL of sterile water or normal saline. Inject the drug directly into the trachea via the ET.

Cardiac Rhythm Monitoring

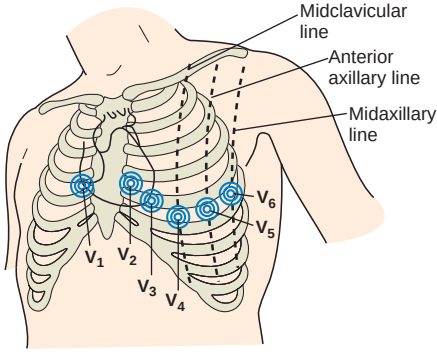
Lead Application

Standard Limb Lead Electrode Placement

Electrodes are placed on the right arm (RA), left arm (LA), right leg (RL), and left leg (LL). With only four electrodes, six leads are viewed. These leads include the standard leads—I, II, and III—and the augmented leads—aVR, aVL, and aVF.



Standard Chest Lead Electrode Placement
The chest leads are identified as V_1, V_2, V_3, V_4, V_5, and V_6. Each electrode placed in a "V" position is positive.

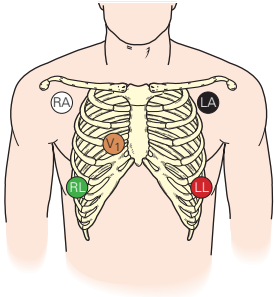


Elements of Chest Leads

Lead	Positive Electrode Placement	View of Heart
V_1	4th Intercostal space to right of sternum	Septum
V_2	4th Intercostal space to left of sternum	Septum
V_3	Directly between V_2 and V_4	Anterior
V_4	5th Intercostal space at left midclavicular line	Anterior
V_5	Level with V_4 at left anterior axillary line	Lateral
V_6	Level with V_5 at left midaxillary line	Lateral

159

Electrode Placement Using a 5-Wire Cable



Clinical Tip: Five-wire telemetry units are commonly used to monitor leads I, II, III, aVR, aVL, aVF, and V_1 in critical care settings.

Vagal Maneuvers

Vagal maneuvers are nonpharmacological interventions that terminate and diagnose tachyarrhythmias. Vagal maneuvers stimulate the vagus nerve and slow conduction through the AV node. They slow SVT, or even convert SVT to NSR, without severe hemodynamic compromise.

The most common methods for stimulating the vagus nerve are Valsalva's maneuver (bearing down or blowing through a straw) and carotid sinus massage. Facial immersion in ice water is an acceptable method for pediatric patients. Vagal maneuvers should be performed only when the ECG is being monitored and venous access has been established.

Valsalva's Maneuver

Valsalva's maneuver is performed by a conscious person.

- Document the arrhythmia before starting treatment.
- Instruct the person to inhale and hold his/her breath.
- Ask the person bear down as if for a bowel movement and to hold this position for 20–30 sec.

OR

- Document the arrhythmia before starting treatment.
- Have the person blow forcefully through a straw (IV catheter or similar device) for as long as possible (at least 20 sec).

Stop the maneuver immediately if:

- The person becomes confused.
- HR drops below 100 bpm.
- Asystole occurs.

Carotid Sinus Massage

Indications: Should be performed only by qualified physicians because of stroke risk.

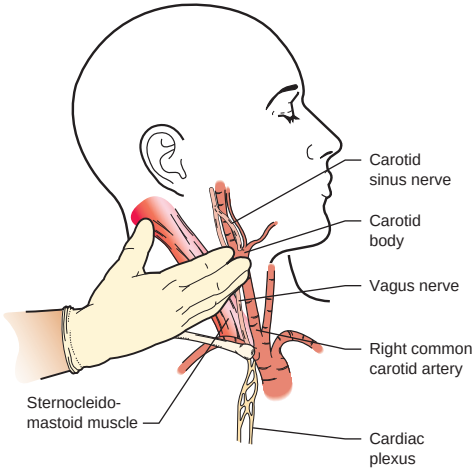
Method: Place the patient in a supine position, head tilted to either side with the neck hyperextended. Place your index and middle fingers over the carotid artery, just below the angle of the jaw, as high on the neck as possible. Massage the artery for 5–10 sec by pressing it firmly against the vertebral column and rubbing.

Contraindications: Unequal carotid pulses, carotid bruits, cervical spine injury, or history of cerebrovascular accident or carotid atherosclerosis.

Side Effects: Slow HR or AV block, PVCs, VT, VF, syncope, seizure, hypotension, nausea or vomiting, stroke.

Precautions: Be sure the patient is receiving oxygen and an IV is in place. Never massage both arteries simultaneously. Resuscitation equipment must be readily available.

Clinical Tip: Carotid sinus massage should be performed only by qualified physicians. Each carotid artery should be gently palpated and auscultated before the procedure to evaluate for contraindications. Proceed only if bilateral carotid pulses are palpable and auscultate for bruits over both carotid arteries. **Do not perform the procedure if a bruit is heard on either side.**



Carotid sinus massage

Tab 7: Megacode

A **megacode** is a group practice that teaches each of the resuscitation team roles. Students practice rapid patient assessments and basic resuscitation skills. They must demonstrate knowledge of ECG rhythm interpretation, pharmacology, and core resuscitation principles. They apply appropriate algorithms to multiple critical scenarios presented by certified instructors. Mannequins and resuscitation equipment are used to simulate cases and practice responses to emergencies in a nonthreatening environment. Students learn from practice, observation, discussions, and scenario debriefings.

Resuscitation Team

A knowledgeable, experienced, and well-organized **team** is critical to achieving optimal outcomes in response to cardiac arrest and cardiopulmonary emergencies. Team members have defined roles and responsibilities and perform lifesaving skills and interventions promptly and efficiently.

Roles of the Team

Team Leader

In clinical practice, the **team leader** is usually a physician or advanced practitioner in the hospital setting, or a paramedic in the prehospital setting, who possesses the knowledge and leadership skills required to effectively run a code. In ACLS and PALS courses, each course participant will function as a team leader. The team leader has the following responsibilities:

- Organize the resuscitation team, assign roles and responsibilities within each member's scope of practice, and monitor each member's performance.
- Direct the code following current, approved protocols and guidelines, ensuring that interventions are performed correctly, promptly, and efficiently and focusing on a comprehensive care plan.
- Communicate explanations and rationales for interventions based on sound knowledge.
- Lead a critique at the conclusion of the code for quality improvement.
- Train and coach team members.

Team Members

Team members include healthcare providers from the medical, nursing, prehospital, and respiratory therapy disciplines who are proficient in the knowledge and skills required in ACLS or PALS. Team members have the following responsibilities:

- Function within their individual scope of practice and be clear about their assignments in a code.
- Respond immediately to a code, be prepared to fulfill their roles and responsibilities, and back up other team members when needed.
- Demonstrate knowledge of current, approved protocols and guidelines.
- Contribute to the conduct of the code, making appropriate suggestions and pointing out potential errors or omissions when indicated.

Team Dynamics

To ensure a smooth, well-coordinated resuscitation response and optimize the chance for a successful outcome, teams must use the following eight elements of communication and team dynamics:

Clear Roles and Responsibilities

- There are six roles in a resuscitation team:
  - Team leader
  - Chest compressor
  - Airway manager
  - Monitor-defibrillator operator
  - IV/IO access and medication administrator
  - Observer/recorder
- If there are fewer than six team members, all the roles must still be performed, requiring some team members to perform more than one. The team leader should clearly assign the roles to team members within their scope of practice and level of competence or expertise.
- Team members must clearly inform the team leader if they are unable to perform their assigned roles.

Clear Messages

- The team leader and team members should speak distinctly, calmly, and loudly enough to be heard without shouting or yelling.
- One person should speak at a time to avoid confusion, miscommunication, and error, and should look directly at the person for whom the communication is intended.
- Team members should validate orders by repeating them, and should question any ambiguity or doubt about an order.

Closed-Loop Communication

- The team leader should order one task at a time (e.g., “Give epinephrine 1 mg IV”) and seek confirmation that the order was received and implemented.
- The team member should confirm that the task was performed (e.g., “epinephrine 1 mg IV given”).

Constructive Intervention

Errors should not be allowed to occur and should not be ignored. The team leader or team member should tactfully correct a colleague who:

- Is about to make a mistake (e.g., administering atropine 1 mg for VF instead of epinephrine 1 mg) and suggest appropriate action (e.g., "Did you mean epinephrine 1 mg instead of atropine?").
- Is not performing a task correctly (e.g., inadequate chest compressions or airway management).
- Fails to perform a task (e.g., not calling out "All clear," not ensuring that everyone is clear from the patient and bed/stretcher before delivering a defibrillation shock).

It is important not to ridicule the colleague or cause confrontation with team members. Issues should be discussed further during a debriefing session following the code to identify areas for improvement.

Knowledge Sharing

Team members possess knowledge, skills, and expertise and should provide helpful suggestions during a code. A team leader focused on an intervention strategy may fail to recognize alternative approaches or changes in the patient's condition.

- If resuscitative efforts remain ineffective, the entire team should go back to the basics, review what has been done so far, and determine whether anything has been missed.
- The team leader should ask for suggestions and ideas, collaborate with team members to evaluate suggestions, and decide whether to act on them or not.

Mutual Respect

An effective team works collaboratively in a friendly, collegial, and supportive manner. Team members must:

- Demonstrate mutual respect when interacting.
- Speak in controlled voices, avoiding yelling and aggressive or demeaning behavior.

The team leader should express gratitude for good performance.

Knowing One's Limitations

All team members should perform only those tasks for which they have been educated and trained, working within their scope of practice.

- The team leader should be aware of limitations and must not direct team members to perform tasks they cannot or should not perform.
- Early on, the team leader should evaluate team resources and request any needed backup before the situation deteriorates.

Reevaluation and Summary

The team leader should:

- Monitor all aspects of the code.
- Periodically reevaluate the patient's status and assessment findings.
- Change strategies when indicated by change in the patient's clinical status.
- Summarize and reevaluate the efficacy of interventions performed, focusing on decisions about differential diagnoses.

Team members should:

- Know the patient's current status and plans for further interventions.
- Assess and report changes in the patient's clinical status.

The code observer/recorder must:

- Document all code assessments and interventions.
- Be prepared to provide data for code summarization.

ACLS Megacode Practice Scenarios

Megacode practice scenarios present simulated adult cardiac arrest cases. A mannequin and resuscitation equipment are used to practice implementing ACLS algorithms in response to patient assessment findings.

- Students function in each role during practice scenarios while an ACLS instructor monitors and evaluates performance.
- Students must demonstrate knowledge of core case material and skills, knowledge of ACLS algorithms, correct ECG rhythm interpretation, appropriate use of ACLS drug therapy, and effective performance in the role of team member and team leader.

The ACLS Megacode section will present six adult cardiac arrest cases, demonstrating correct implementation of ACLS algorithms and promoting critical thinking by a team leader to prepare students for successful completion of an ACLS course.

PALS Megacode Practice Scenarios

Core case practice scenarios present simulated pediatric emergency and cardiac arrest cases. A mannequin and resuscitation equipment are used to practice implementing PALS protocols and algorithms in response to patient assessment findings.

- Students function in each role during practice scenarios while a PALS instructor monitors and evaluates performance.
- Students must demonstrate knowledge of core case material and skills, knowledge of a systematic approach to pediatric assessment and of PALS algorithms, correct ECG rhythm interpretation, appropriate use of PALS

drug therapy, and effective performance in the role of team member and team leader.

The PALS Megacode section will present six pediatric emergency or cardiac arrest cases, demonstrating correct implementation of a systematic approach to patient assessment and of PALS protocols and algorithms and promoting critical thinking by a team leader to prepare students for successful completion of a PALS course.

ACLS Megacode Case 1: Bradycardia ⊘ VF ⊘ Asystole ⊘ ROSC

Scenario: 84-year-old woman with history of HTN and CHF; recently hospitalized for A-fib with rapid ventricular response and started on metoprolol, diltiazem, hydrochlorothiazide, and warfarin; discharged 4 days ago in controlled A-fib. She now presents with complaints of significant lightheadedness, generalized weakness, and shortness of breath. She is pale, cool, diaphoretic, and dyspneic. HR 35 bpm, BP 76/42, RR 22/min, O_2 sat 90%.

Team Leader	Team Members
Assess responsiveness: patient responsive	
Assign team member roles and responsibilities, monitor quality of performance	
Perform primary **ABCD** survey:	
A: Airway is patent	Maintain patent airway, support ventilation if needed
B: Tachypneic, dyspneic, O_2 sat low: order O_2	Administer O_2 by nonrebreather mask, maintain O_2 sat 94%–99%
C: Slow, weak, thready pulse, hypotension; order monitor	Attach monitor, evaluate rhythm: third-degree (complete) heart block, wide QRS
IV/IO access	Establish IV/IO access, start IV NS
D: Shock not warranted	
Obtain pertinent history to establish cause of bradycardia	

Team Leader	Team Members
Rhythm: Third-degree heart block	
Symptomatic bradycardia	
Order 12-lead ECG if available	Obtain 12-lead ECG
Order atropine 0.5 mg IVP	Give atropine 0.5 mg IVP, confirm when given
Order preparation for TCP	Apply pads to patient's chest
Consider chronotropic infusion (epinephrine or dopamine) if needed to increase HR	
Rhythm: Third-degree heart block	
Order atropine 0.5 mg IVP every 3–5 min, max. 3 mg, or may initiate TCP or chronotropic infusion.	Give atropine 0.5 mg IVP every 3–5 min, confirm when given if ordered, or initiate TCP or chronotropic infusion if ordered
Patient becomes unresponsive.	
Rhythm: VF	
C-A-B: No pulse, no breathing	
Order defibrillation: 200 J biphasic (360 J monophasic)	Clear area, remove O_2, deliver shock
Order 2 min of CPR; monitor for **high-quality CPR**	Start CPR, insert OPA
Order vasopressor: epinephrine 1 mg IVP or vasopressin 40 units IVP	Give vasopressor drug ordered, confirm when given
	Recorder: Indicate when 2 min of CPR is ended

Continued

Team Leader	Team Members
Stop CPR, assess **rhythm: VF**	
Order defibrillation: same energy as above, or escalate energy	Clear area, remove O_2, deliver shock
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR
If basic airway management is inadequate, order advanced airway with waveform capnography	Insert advanced airway with waveform capnography if ordered
Order amiodarone 300 mg IVP	Give amiodarone 300 mg IVP, confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: Asystole**	
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR
Order epinephrine 1 mg IVP	Give epinephrine 1 mg IVP, confirm when given
Consider possible causes: Hs and Ts, treat cause if possible	Contribute to discussion of Hs and Ts
Order preparation of epinephrine 1 mg IV	Prepare epinephrine 1 mg IV
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: NSR**	
Assess patient: remains unresponsive	
C-A-B assessment:	
C: Pulse palpable: ROSC	
A, B: Agonal respirations	

Team Leader	Team Members
Order continued support of ventilation and oxygenation: ET intubation if not already performed, mechanical ventilation, waveform capnography, O_2 to maintain O_2 sat 94%–99%	Support ventilation and oxygenation as ordered, initiate waveform capnography, assess O_2 sat
Order vital signs	HR 90, BP 84/62, O_2 sat 98%
Order 12-lead ECG	Perform 12-lead ECG: Sinus rhythm, no ischemic changes
Order IV fluid resuscitation	Administer IV NS bolus
Order blood glucose level and appropriate lab tests	Obtain blood samples for ordered lab tests
Order therapeutic hypothermia	Initiate therapeutic hypothermia
Get cardiology consult	Call for cardiology consult
Order transfer to critical care unit	Transfer patient to critical care unit
Formulate comprehensive care plan and treat cause(s)	Initiate care plan

Critical Thinking:

1. **What do you suspect is the cause of this patient's bradycardia?**
   The patient probably converted spontaneously to sinus rhythm following her hospital discharge 4 days ago. Her beta blocker and calcium channel blocker doses are now excessive, causing complete heart block. You should reevaluate her medications and doses. In addition, the patient is elderly and may have cardiac electrical conduction system disease, in which case you should consider implantation of a permanent pacemaker.
2. **Why administer atropine for complete heart block?**
   Atropine suppresses vagal innervation, allowing HR to accelerate. However, complete heart block may be infranodal (below the AV node) and not related to increased vagal tone, in which case atropine is ineffective. Prepare for TCP.
3. **Why defibrillate first instead of providing 2 min of CPR once the rhythm changed to VF?**
   Defibrillation delivers an electrical shock to disrupt VF and restore sinus rhythm. CPR provides perfusion but will probably not restore sinus rhythm

without defibrillation. In a witnessed VF, immediate defibrillation is warranted. CPR should be performed until an AED or manual defibrillator is charged and ready for use. In unwitnessed VF, when suspected cardiac arrest time has exceeded 4 or 5 min, initial CPR may improve perfusion to make defibrillation more effective, but this has not been proven.

4. **When should an ET be inserted during CPR?**
   If basic airway management is effective, there is no need to insert an ET early. However, if basic airway management is difficult or ineffective, or prolonged CPR is required, you should consider inserting an advanced airway. Only skilled personnel can insert an ET. All equipment should be prepared and checked before stopping CPR to perform intubation. It should be performed rapidly, without interrupting CPR for more than 10 sec if possible. Proper tube placement must be confirmed and the tube secured. Waveform capnography is highly recommended.
5. **Why is atropine not administered in asystole?**
   Atropine blocks parasympathetic (vagal) innervation of the heart. Asystole is rarely due to excessive vagal tone; therefore atropine will probably not restore sinus rhythm in asystole.
6. **Why is TCP not recommended in asystole?**
   TCP has not been found effective in restoring sinus rhythm in asystole. It requires interruption of CPR, during which time coronary perfusion drops to critically low levels. If a patient has preexisting transvenous or epicardial pacing wires, an external pacemaker should be attached to attempt pacing.

ACLS Megacode Case 2: Bradycardia ➪ Pulseless VT ➪ Asystole ➪ ROSC

Scenario: 48-year-old man with a history of HTN, dyslipidemia, ongoing tobacco use, and GERD. He is on hydrochlorothiazide, simvastatin, and famotidine. He presents with complaints of chest burning, nausea, and shortness of breath. He is pale, cool, diaphoretic. HR 39 bpm, BP 80/48, RR 18/min, O_2 sat 94%.

Team Leader	Team Members
Assess responsiveness: patient responsive	
Assign team member roles and responsibilities, monitor quality of performance	

Team Leader	Team Members
Perform primary **ABCD** survey:	
A: Airway is patent	Maintain patent airway, support ventilation if needed
B: Normal, O_2 sat normal; order O_2	Administer O_2 by nonrebreather mask, maintain O_2 sat 94%–99%
C: Slow, weak, thready pulse, hypotension; order monitor	Attach monitor, evaluate rhythm: Sinus bradycardia
IV/IO access	Establish IV/IO access, start IV NS
D: Shock not warranted	
Obtain pertinent history to establish cause of bradycardia	
Rhythm: Sinus bradycardia	
Symptomatic bradycardia	
Order 12-lead ECG if available: **Inferior STEMI**	Obtain 12-lead ECG: sinus bradycardia with ST elevation in leads II, III, aVF
Order right precordial ECG to assess for RV infarction	Obtain right precordial ECG
Order chewable aspirin 324 mg PO	Give chewable aspirin 324 mg PO, confirm when given
Order atropine 0.5 mg IVP	Give atropine 0.5 mg IVP, confirm when given
Order IV fluid resuscitation	Administer IV NS, titrate to BP response
Order preparation for TCP	Apply pads to patient's chest
Consider chronotropic infusion (epinephrine, dopamine) if necessary to increase HR	
Patient becomes unresponsive.	
Rhythm: Pulseless VT	
C-A-B: No pulse, no breathing	
Order **defibrillation:** 200 J biphasic (360 J monophasic)	Clear area, remove O_2, deliver shock
Order 2 min of CPR; monitor for **high-quality CPR**	Start CPR, insert OPA

Continued

Team Leader	Team Members
Order vasopressor: epinephrine 1 mg IVP or vasopressin 40 units IVP	Give vasopressor drug ordered, confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: Pulseless VT**	
Order **defibrillation:** same energy as above, or escalate energy	Clear area, remove O_2, deliver shock
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR
If airway inadequate, order advanced airway with waveform capnography	Insert advanced airway with waveform capnography if ordered
Order amiodarone 300 mg IVP	Give amiodarone 300 mg IVP, confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: Asystole**	
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR
Order epinephrine 1 mg IVP	Give epinephrine 1 mg IVP, confirm when given
Consider possible causes: Hs and Ts, treat cause if possible	Contribute to discussion of Hs and Ts
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: NSR**	
Patient becomes responsive, responds to commands	

Team Leader	Team Members
ABCD survey:	
A: Airway patent	
B: Breathing with adequate chest rise	
C: Pulse palpable: ROSC	
Order continued support of oxygenation: O_2 to maintain O_2 sat 94%–99%	Support oxygenation as ordered, assess O_2 sat
Order vital signs	HR 88, BP 108/62, O_2 sat 96%
Order 12-lead ECG: **Inferior STEMI**	Perform 12-lead ECG: NSR, ST elevation in leads II, III, aVF
Order maintenance IV fluid	Administer IV NS
Order blood glucose level and appropriate lab tests	Obtain blood samples for ordered lab tests
Get cardiology consult	Call for cardiology consult
Order transport to cardiac catheterization lab if available, or to critical care unit	Transport patient to cardiac catheterization lab if available, or to critical care unit
Formulate comprehensive care plan. Treat cause(s)	Initiate care plan

Critical Thinking:

1. **What do you suspect as the cause of this patient's bradycardia?**
   The patient had anginal symptoms. His ECG revealed ST elevation in the inferior leads, consistent with inferior STEMI, which can be associated with bradycardia.
2. **Why administer chewable aspirin?**
   The patient is presenting with inferior STEMI. Antiplatelet therapy with chewable aspirin for rapid absorption is essential to decrease platelet aggregation in the obstructed coronary artery.
3. **Why administer atropine for this patient's sinus bradycardia?**
   Sinus bradycardia may be caused by excessive parasympathetic (vagal) stimulation of the SA node. Atropine suppresses vagal innervation of the heart, allowing the HR to accelerate, increasing CO and BP, and increasing myocardial perfusion.

4. **Why not administer nitroglycerin or morphine to this patient presenting with an inferior STEMI?**
   The patient is hypotensive and bradycardic. Nitroglycerin or morphine would further decrease his blood pressure. In addition, RV infarction should be suspected when a patient presents with an inferior STEMI and hypotension. A right precordial ECG may help to identify RV infarction. Nitroglycerin and morphine decrease preload and are contraindicated in RV infarctions because the RV is preload dependent. The patient would benefit more from IV fluids.
5. **Why transport the patient to a cardiac catheterization laboratory, if available, instead of a critical care unit?**
   With STEMI, the occluded coronary artery must be opened as soon as possible to restore myocardial blood flow and salvage myocardial function.
6. **Why not initiate therapeutic hypothermia?**
   The patient regained consciousness and responded to commands, indicating adequate cerebral function.

ACLS Megacode Case 3: Tachycardia → VF → PEA → ROSC

Scenario: 66-year-old woman with history of HTN and tobacco use. She presents with complaints of chest palpitations, chest pressure, and shortness of breath. She is pale, cool, and diaphoretic. HR 156 bpm, BP 74/52, RR 20/min, O_2 sat 93%.

Team Leader	Team Members
Assess responsiveness: patient responsive	
Assign team member roles and responsibilities, monitor quality of performance	
Perform primary **ABCD** survey:	
A: Airway is patent	Maintain patent airway, support ventilation if needed
B: Tachypneic, O_2 sat low	Administer O_2 by nonrebreather mask, maintain O_2 sat 94%–99%

Team Leader	Team Members
C: Rapid, weak, thready pulse, hypotension: order monitor, IV/IO access	Attach monitor, evaluate **rhythm: regular monomorphic wide-complex tachycardia: VT** Establish IV/IO access, start IV NS
D: Cardioversion warranted	
Obtain pertinent history to establish cause of tachycardia; do not delay cardioversion	
Order preparation for synchronized cardioversion	Apply pads to chest, press Sync button on monitor-defibrillator
Order chewable aspirin 324 mg PO while preparing for cardioversion	Give chewable aspirin 324 mg PO; confirm when given.
Order sedative and analgesic	Give sedative and analgesic ordered, confirm when given
Order **cardioversion** at 100 J for VT	Clear area, remove O_2, deliver shock
Consider IV antiarrhythmic drug therapy	
Patient becomes unresponsive.	
Rhythm: VF	
C-A-B: No pulse, no breathing	
Order **defibrillation:** 200 J biphasic (360 J monophasic)	Clear area, remove O_2, deliver shock
Order 2 min of CPR; monitor for **high-quality CPR**	Start CPR, insert OPA
Order vasopressor: epinephrine 1 mg IVP or vasopressin 40 units IVP	Give vasopressor drug ordered, confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: VF**	
Order **defibrillation:** same energy as above, or escalate energy	Clear area, remove O_2, deliver shock
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR

Continued

Team Leader	Team Members
If airway inadequate, order advanced airway with waveform capnography	Insert advanced airway with waveform capnography if ordered
Order amiodarone 300 mg IVP	Give amiodarone 300 mg IVP, confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: NSR**	
Patient remains unresponsive	
C-A-B:	
C: No pulse palpable: **PEA**	
A-B: No respirations	
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR
Order epinephrine 1 mg IVP	Give epinephrine 1 mg IVP, confirm when given
Consider possible causes: Hs and Ts, treat cause if possible	Contribute to discussion of Hs and Ts
Order IV fluid resuscitation	Administer IV NS bolus
Order preparation of epinephrine 1 mg IV	Prepare epinephrine 1 mg IV
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: NSR**	
Patient remains unresponsive	
C-A-B assessment:	
C: Pulse palpable: **ROSC**	
A, B: Agonal respirations	
Order continued support of ventilation and oxygenation: ET intubation if not already performed, mechanical ventilation, waveform capnography, O_2 to maintain O_2 sat 94%–99%	Support ventilation and oxygenation as ordered, initiate waveform capnography, assess O_2 sat

Team Leader	Team Members
Order vital signs	HR 90, BP 68/40, O_2 sat 98%
Order 12-lead ECG: **Unstable angina or NSTEMI**	Perform 12-lead ECG: NSR, ST depression in leads V_3–V_5
Order IV fluid resuscitation	Administer IV NS bolus
Order blood glucose level and appropriate lab tests	Obtain blood samples for ordered lab tests
Order therapeutic hypothermia	Initiate therapeutic hypothermia
Get cardiology consult	Call for cardiology consult
Order transport to cardiac catheterization lab if available, or to critical care unit	Transport patient to cardiac catheterization lab if available, or to critical care unit
Formulate comprehensive care plan. Treat cause(s)	Initiate care plan

Critical Thinking:

1. **What do you suspect as the cause of this patient's wide-complex tachycardia?**
   The patient has ACS, presenting with chest pressure and an irritable cardiac rhythm. Her ECG post-arrest showed ST depression in the anterior leads, consistent with unstable angina or non–ST-elevation myocardial infarction (NSTEMI). Myocardial ischemia causes unstable rhythms.
2. **Why give the patient aspirin initially?**
   If ACS is suspected, it is appropriate to promptly administer aspirin for antiplatelet therapy.
3. **Why perform synchronized cardioversion so soon?**
   Ischemic heart disease causes ventricular irritability, potentially leading to unstable rhythms such as VT. A rapid HR increases O_2 demand, causing more irritability and leading to cardiac arrest. Synchronized cardioversion delivers a shock on the QRS complex during depolarization to terminate monomorphic VT, restore sinus rhythm, and prevent cardiac arrest. In polymorphic VT, the QRS complexes change, making synchronization difficult and possibly delaying shock delivery. Unsynchronized defibrillation is more appropriate for polymorphic VT.
4. **What is the most important key to successful resuscitation and optimal patient outcomes?**
   It is most important to provide high-quality CPR to optimize perfusion to the heart and brain. The role of compressor should change every 2 min to avoid fatigue, which leads to inadequate compressions and decreased perfusion. High-quality CPR must be monitored by the team leader, but

should also be the responsibility of each team member. If inadequate CPR is observed, it should be pointed out and corrected.

5. **Is basic airway management sufficient?**
   Initially, basic airway management is usually sufficient. If basic airway management is inadequate, a qualified practitioner should place an advanced airway.
6. **Do you have to administer vasopressin during cardiac arrest?**
   No. You can give epinephrine 1 mg IVP every 3–5 min during cardiac arrest and not give vasopressin at all. If you want to give vasopressin 40 units IVP, it should substitute for epinephrine as the first or second vasopressor dose. It is given only once. It has not been found more effective than epinephrine to increase ROSC or survival to discharge.

ACLS Megacode Case 4: Tachycardia ↯ VF ↯ PEA ↯ ROSC

Scenario: 65-year-old woman with history of NSTEMI and a drug-coated stent to her right coronary artery (RCA) 9 months ago. She is on aspirin, clopidogrel, metoprolol, lisinopril, and atorvastatin. She presented with complaints of rapid chest palpitations, slight shortness of breath, and anxiety. She denies chest discomfort, dyspnea, dizziness, diaphoresis, or nausea. She has normal skin color and appears to be in no distress. HR 165 bpm, BP 168/74, RR 16/min, O_2 sat 98%.

Team Leader	Team Members
Assess responsiveness: patient responsive	
Assign team member roles and responsibilities, monitor quality of performance	
Perform primary **ABCD** survey:	
A: Airway is patent	Maintain patent airway, support ventilation if needed
B: Breathing normal, O_2 sat normal	Administer O_2 by nasal cannula, if needed, to maintain O_2 sat 94%–99%
C: Rapid pulse: order monitor, IV/IO access	Attach monitor, evaluate **rhythm: regular narrow-complex tachycardia** Establish IV/IO access, start IV NS

Team Leader	Team Members
D: Stable: cardioversion not warranted	
Obtain pertinent history to establish cause of tachycardia	
Patient remains stable, no serious signs or symptoms	
Rhythm: SVT	
Order 12-lead ECG if available	Obtain 12-lead ECG: regular narrow-complex tachycardia
Attempt vagal maneuvers (patient has no carotid bruits): No change in rhythm	
Order adenosine 6 mg rapid IVP	Give adenosine 6 mg rapid IVP, flush with 20 mL NS IV; confirm when given
Patient becomes unresponsive.	
Rhythm: VF	
C-A-B: No pulse, no breathing	
Order **defibrillation:** 200 J biphasic (360 J monophasic)	Clear area, remove O_2, deliver shock
Order 2 min of CPR; monitor for **high-quality CPR**	Start CPR, insert OPA
Order vasopressor: epinephrine 1 mg IVP or vasopressin 40 units IVP	Give vasopressor drug ordered, confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: VF**	
Order **defibrillation:** same energy as above, or escalate energy	Clear area, remove O_2, deliver shock
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR

Continued

Team Leader	Team Members
If airway inadequate, order advanced airway with waveform capnography	Insert advanced airway with waveform capnography if ordered
Order amiodarone 300 mg IVP	Give amiodarone 300 mg IVP; confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: NRS**	
Patient remains unresponsive	
C-A-B	
C: No pulse palpable: **PEA**	
A, B: No respirations	
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR
Order epinephrine 1 mg IVP	Give epinephrine 1 mg IVP, confirm when given
Consider possible causes: Hs and Ts, treat cause if possible	Contribute to discussion of Hs and Ts
Order IV fluid resuscitation	Administer IV NS bolus
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: NSR**	
Patient remains unresponsive	
C-A-B assessment:	
C: Pulse palpable: **ROSC**	
A, B: Agonal respirations	
Order continued support of ventilation and oxygenation: ET intubation if not already performed, mechanical ventilation, waveform capnography, O_2 to maintain O_2 sat 94%–99%	Support ventilation and oxygenation as ordered, initiate waveform capnography, assess O_2 sat

Team Leader	Team Members
Order vital signs	HR 90, BP 84/62, O_2 sat 98%
Order 12-lead ECG	Perform 12-lead ECG: NSR, no ischemic ECG changes.
Order IV fluid resuscitation	Administer IV NS bolus
Order blood glucose level and appropriate lab tests	Obtain blood samples for ordered lab tests
Order therapeutic hypothermia	Initiate therapeutic hypothermia
Get cardiology consult	Call for cardiology consult
Order transport to critical care unit	Transport patient to critical care unit
Formulate comprehensive care plan. Treat cause(s)	Initiate care plan

Critical Thinking:

1. **Why did this patient not require initial cardioversion?**
   The patient presented in SVT and was hemodynamically stable, without serious signs and symptoms. SVT frequently responds to vagal maneuvers, adenosine, or both.
2. **Why attempt vagal maneuvers?**
   SVT typically involves a reentry circuit in the AV node. Vagal maneuvers enhance vagal tone to slow AV nodal conduction and halt the reentry circuit to restore sinus rhythm.
3. **Why must adenosine be administered by rapid IVP?**
   Adenosine has a short half-life, less than 10 sec. It must be administered over 1–2 sec and followed by a 20-mL IV NS flush. It is better to use a peripheral IV site as proximal to the trunk as possible, such as the upper arm. See Tab 5: Emergency Medications for further information.

ACLS Megacode Case 5: Tachycardia ⇄ Pulseless VT ⇄ PEA ⇄ ROSC

Scenario: 70-year-old man with no significant medical history. He takes occasional calcium carbonate (Tums) and ibuprofen, but no other medications. He developed indigestion at home and took an antacid without relief. His discomfort grew worse. He presents with complaints of chest palpitations, chest tightness, and shortness of breath. He is flushed and diaphoretic. HR 174 bpm, BP 162/98, RR 20/min, O_2 sat 92%.

Team Leader	Team Members
Assess responsiveness: patient responsive	
Assign team members role and responsibilities, monitor quality of performance	
Perform primary **ABCD** survey:	
A: Airway is patent	Maintain patent airway, support ventilation if needed
B: Tachypneic, O_2 sat low	Administer O_2 by nonrebreather mask, maintain O_2 sat 94%–99%
C: Rapid pulse: order monitor, IV/IO access	Attach monitor, evaluate **rhythm: regular narrow-complex tachycardia** Establish IV/IO access, start IV NS
D: Unstable SVT: cardioversion warranted	
Obtain pertinent history to establish cause of tachycardia; do not delay cardioversion	
Order preparation for cardioversion	Apply pads to chest, press Sync button on monitor-defibrillator
Order chewable aspirin 324 mg PO and nitroglycerin 0.04 mg sl while preparing for cardioversion if time allows	Give chewable aspirin 324 mg PO and nitroglycerin 0.04 mg sl if ordered; confirm when given
Order sedative and analgesic	Give sedative and analgesic ordered, confirm when given
Order **cardioversion** at 100 J	Clear area, remove O_2, deliver shock
Patient becomes unresponsive.	
Rhythm: Pulseless VT	
C-A-B: No pulse, no breathing	
Order **defibrillation:** 200 J biphasic (360 J monophasic)	Clear area, remove O_2, deliver shock

Team Leader	Team Members
Order 2 min of CPR; monitor for **high-quality CPR**	Start CPR, insert OPA
Order vasopressor: epinephrine 1 mg IVP or vasopressin 40 units IVP	Give vasopressor drug ordered, confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: Pulseless VT**	
Order **defibrillation:** same energy as above, or escalate energy	Clear area, remove O_2, deliver shock
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR
If airway inadequate, order advanced airway with waveform capnography	Insert advanced airway with waveform capnography if ordered
Order amiodarone 300 mg IVP	Give amiodarone 300 mg IVP, confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: NSR**	
Patient remains unresponsive	
C-A-B	
C: No pulse palpable: **PEA**	
A-B: No respirations	
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR
Order epinephrine 1 mg IVP	Give epinephrine 1 mg IVP, confirm when given
Consider possible causes: Hs and Ts, treat cause if possible	Contribute to discussion of Hs and Ts
Order IV fluid resuscitation	Administer IV NS bolus
	Recorder: Indicate when 2 min of CPR is done

Continued

Team Leader	Team Members
Stop CPR, assess **rhythm: NSR**	
Patient becomes responsive	
A-B-C assessment:	
A: Airway is patent	
B: Breathing labored	
C: Pulse palpable: **ROSC**	
Order continued support of ventilation and oxygenation: ET intubation if not already performed, mechanical ventilation, waveform capnography, O_2 to maintain O_2 sat 94%–99%	Support ventilation and oxygenation as ordered, initiate waveform capnography, assess O_2 sat
Order vital signs	HR 98, BP 85/68, O_2 sat 88%
Order 12-lead ECG: **Unstable angina or NSTEMI**	Perform 12-lead ECG: Sinus rhythm, ST depression in leads I, aVL, V_5, V_6
Order IV fluid resuscitation	Administer IV NS bolus
Order blood glucose level and appropriate lab tests	Obtain blood samples for ordered lab tests
Order therapeutic hypothermia	Initiate therapeutic hypothermia
Get cardiology consult	Call for cardiology consult
Order transport to cardiac catheterization lab if available, or to critical care unit	Transport patient to cardiac catheterization lab if available, or to critical care unit
Formulate comprehensive care plan. Treat cause(s)	Initiate care plan

Critical Thinking:

1. **Why should this patient be cardioverted given a blood pressure of 162/98?**
   Although this patient was not hypotensive, he was seriously symptomatic, with chest tightness, shortness of breath, and hypoxemia. He is probably experiencing ACS, and his tachycardia is increasing myocardial O_2 demand, putting him at risk for significant myocardial damage and cardiac arrest.

2. **Which is preferable, IV access or IO access?**
   IV access is preferable, using a large peripheral vein such as a brachial vein using an 18- or 16-gauge needle. Avoid using veins in the hand. To hasten drug delivery to the central circulation, follow each dose with a 20-mL bolus of IV normal saline flush and elevate the extremity for 10–20 sec if possible for gravity assistance.
3. **Are there changes in drug and fluid administration when IO access is used?**
   If IV access cannot be obtained, IO access can be used without interrupting chest compressions. Recommended ACLS drug doses and fluid administration are the same as with IV access. Anything that can be administered IV can be administered IO.
4. **If this were an inpatient with a preexisting central venous line, could it have been used?**
   If a patient has a functioning central venous line, it should be used for more rapid drug and fluid delivery. IV medication already infusing through the central line before arrest should be discontinued during cardiac arrest unless it is a plain IV solution or a vasopressor agent. Vasodilators should be discontinued because the patient is hypotensive. Other drugs may be incompatible with ACLS drugs.
5. **Should a central venous line be inserted for more rapid drug delivery during cardiac arrest?**
   A central venous line should not be initiated during cardiac arrest. The procedure may require interruption of chest compressions for longer than 10 sec, causing a critical drop in coronary perfusion. The procedure can be performed only by qualified, skilled practitioners and may cause serious complications such as vascular damage, bleeding, hematoma, pneumothorax, or cardiac tamponade.

ACLS Megacode Case 6: Tachycardia ↯ VF ↯ PEA ↯ ROSC

Scenario: 36-year-old man with history of HTN and type 2 diabetes mellitus. He is on aspirin, lisinopril, and metformin. He suddenly developed rapid chest palpitations but otherwise felt well. After 30 min he presented with continuing chest palpitations but no other complaints. His skin color is normal, and he appears in no distress. HR 162 bpm, BP 148/76, RR 14/min, O_2 sat 99%.

Team Leader	Team Members
Assess responsiveness: patient responsive	
Assign team member role and responsibilities, monitor quality of performance	
Perform primary **ABCD** survey:	
A: Airway is patent	Maintain patent airway, support ventilation if needed
B: Breathing normal, O_2 sat normal	Administer O_2 by nasal cannula, if needed, to maintain O_2 sat 94%–99%
C: Rapid pulse: order monitor, IV/IO access	Attach monitor-defibrillator, evaluate **rhythm: regular monomorphic wide-complex tachycardia** Establish IV/IO access, start IV NS
D: Stable: cardioversion not warranted	
Obtain pertinent history to establish cause of tachycardia	
Patient remains stable, no serious signs or symptoms	
Rhythm: stable regular monomorphic wide-complex tachycardia: probably VT	
Order 12-lead ECG if available	Obtain 12-lead ECG: regular monomorphic wide-complex tachycardia, probably VT
Order amiodarone 150 mg IV given over 10 min	Give amiodarone 150 mg IV over 10 min, confirm when given
Patient becomes unresponsive.	
Rhythm: VF	
C-A-B: No pulse, no breathing	
Order **defibrillation:** 200 J biphasic (360 J monophasic)	Clear area, remove O_2, deliver shock

Team Leader	Team Members
Order 2 min of CPR; monitor for **high-quality CPR**	Start CPR, insert OPA
Order vasopressor: epinephrine 1 mg IVP or vasopressin 40 units IVP	Give vasopressor drug ordered, confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: VF**	
Order **defibrillation:** same energy as above, or escalate energy	Clear area, remove O_2, deliver shock
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR
If airway inadequate, order advanced airway with waveform capnography	Insert advanced airway with waveform capnography if ordered
Order amiodarone 300 mg IVP	Give amiodarone 300 mg IVP, confirm when given
	Recorder: Indicate when 2 min of CPR is done
Stop CPR, assess **rhythm: NSR**	
Patient remains unresponsive	
C-A-B	
C: No pulse palpable: **PEA**	
A-B: No respirations	
Order 2 min of CPR; monitor for **high-quality CPR**	Change compressor; immediately resume CPR
Order epinephrine 1 mg IVP	Give epinephrine 1 mg IVP, confirm when given
Consider possible causes: Hs and Ts, treat cause if possible	Contribute to discussion of Hs and Ts
Order IV fluid resuscitation	Administer IV NS bolus
	Recorder: Indicate when 2 min of CPR is done

Continued

Team Leader	Team Members
Stop CPR, assess **rhythm: NSR**	
Patient becomes responsive, able to follow commands	
A-B-C assessment:	
A: Airway is patent	
B: Breathing labored	
C: Pulse palpable: **ROSC**	
Order continued support of ventilation and oxygenation: ET intubation if not already performed, mechanical ventilation, waveform capnography, O_2 to maintain O_2 sat 94%–99%	Support ventilation and oxygenation as ordered, initiate waveform capnography, assess O_2 sat
Order vital signs	HR 112, BP 85/68, O_2 sat 88%
Order 12-lead ECG: Anterior STEMI	Perform 12-lead ECG: Sinus rhythm, ST elevation in leads V_1 to V_4
Order IV fluid resuscitation	Administer IV NS bolus
Order blood glucose level and appropriate lab tests	Obtain blood samples for ordered lab tests
Get cardiology consult	Call for cardiology consult
Order transport to cardiac catheterization lab if available, or to critical care unit	Transport patient to cardiac catheterization lab if available, or to critical care unit
Formulate comprehensive care plan. Treat cause(s)	Initiate care plan

Critical Thinking:

1. **Why order a 12-lead ECG initially for this patient?**
   A 12-lead ECG may help differentiate between VT and SVT with aberrancy. Negative wide QRS complexes from V_1 through V_6 support the diagnosis of VT. A negative wide QRS in V_1 with a positive wide QRS in V_6 may support the diagnosis of SVT with a left bundle branch block. A wide QRS with rSR′ morphology in V_1 with a wide QRS with qRS morphology in V_6 supports a diagnosis of SVT with a right bundle branch block. If SVT is suspected, adenosine may be administered.

2. **Should this patient receive an amiodarone maintenance infusion?**
   Following ROSC, it is appropriate to start an amiodarone maintenance infusion for patients successfully resuscitated from VF or pulseless VT, after successful conversion to NSR. Patients are at risk for recurrent VF or VT. See Tab 5: Emergency Medications for dosing information.
3. **If an ET is inserted during cardiac arrest, does that change the ratio of compressions to ventilations?**
   Following successful ET intubation, compressions should be performed continuously for 2 min during each cycle, without pause for ventilations. One breath should be delivered every 6–8 sec for a total of 8–10 breaths/min. Deliver only sufficient volume for the chest to rise. Further volume forces extra air into the esophagus and stomach. This elevates the diaphragm and decreases thoracic excursion, making it more difficult to adequately oxygenate and ventilate the patient. Excessive volume also increases thoracic pressure and impedes ventricular filling, reducing stroke volume from chest compressions.
4. **Is there any advantage to delivering more frequent breaths during cardiac arrest, e.g., 1 breath every 3–4 sec?**
   No. It is ineffective and may even be harmful. Each positive pressure ventilation impedes venous return to the right side of the heart, decreasing ventricular filling and reducing the stroke volume and CO of chest compressions. During chest compressions, CO is already less than normal. Further reductions reduce perfusion pressure. Lung perfusion is also reduced during cardiac arrest, even with effective chest compressions, affecting alveolar gas exchange. More frequent ventilations may blow off excessive carbon dioxide, causing hypocapnia (hypocarbia) and respiratory alkalosis. Alkalosis impedes release of oxygen from hemoglobin, leading to cellular hypoxia and cell death.
5. **During CPR, your intubated patient's waveform capnogram indicates an end-tidal CO_2 ($PETCO_2$) of 8 mm Hg. What does that indicate?**
   $PETCO_2$ measures expired carbon dioxide from the lungs. It is determined by lung perfusion and alveolar gas exchange. The normal value is 35–45 mm Hg. A low value may be due to incorrect ET placement. Therefore, you should first assess ET placement. If a right mainstem intubation is suspected, the tube should be withdrawn a few centimeters and placement reassessed. If esophageal intubation is suspected, the tube should be immediately withdrawn and bag-mask ventilation initiated. Correct ET placement and $PETCO_2$ less than 10 mm Hg indicate poor gas exchange from decreased lung perfusion. This may result from inadequate chest compressions. First assess the quality of chest compressions and take steps to improve CPR. Ensure that a vasopressor (e.g., epinephrine) is administered every 3–5 min to increase perfusion pressure. A persistent low value despite correct ET placement and high-quality CPR indicates that ROSC is unlikely. You may then consider terminating resuscitation efforts.

6. **When should ACLS drugs be administered during cardiac arrest?** Administration at the beginning of CPR allows 2 min for the drug to circulate. The drug should be prepared during the previous cycle so that it can be given just as compressions are resumed. Alternatively, it can be prepared during a cycle and administered at the end of the cycle during a pause to analyze the rhythm, just before delivering a shock if rhythm is VF or pulseless VT. There is no clear evidence that drugs improve survival to discharge.

PALS Megacode Case 1: Respiratory Distress/Respiratory Failure

Scenario: An 8-year-old girl presents with difficulty breathing.

Team Leader	Team Members
Initial Observation	
Evaluate:	
LOC: Awake, restless	
Breathing: Labored, shallow, use of accessory muscles, stridor	
Skin color: Pale	
Identify:	
Respiratory distress or respiratory failure	
Intervention:	**Perform interventions as ordered:**
Activate emergency response	Call for a rapid response
Airway, Breathing: Order airway management, O_2	Open airway Apply pulse oximeter, evaluate O_2 sat Apply nonrebreather mask Administer 100% O_2
Circulation: Order monitor	Get cardiac monitor, apply ECG leads or pads

Team Leader	Team Members
Primary Assessment	**Perform primary assessment as ordered. Findings:**
Evaluate:	
Airway: Inspiratory stridor	**Airway:** Inspiratory stridor
Breathing: Order assessment of breathing	**Breathing:** RR 34, nasal flaring, suprasternal retractions, diminished breath sounds, O_2 sat 88% on room air
Circulation: Order assessment of circulation	**Circulation:** HR 128, BP 110/72, peripheral pulses 2+, capillary refill 2 sec, skin pale and cool
Disability: Order neurological assessment	**Disability:** Awake, alert, anxious, normal pupil response
Exposure: Order removal of clothing, rapid examination of body	**Exposure:** Remove clothing, examine body: no signs of trauma, wounds, deformities, bruising, bleeding, rashes, petechiae, purpura; extremities nontender; temperature normal, afebrile
Identify:	
Respiratory distress	
Upper airway obstruction	
Intervention:	**Perform interventions as ordered. Findings:**
Assess response to 100% O_2	O_2 sat 97% on 100% O_2
Analyze heart **rhythm: Sinus tachycardia**	Monitor: Sinus tachycardia
Secondary Assessment	
Evaluate:	
SAMPLE history	
Signs and symptoms: Developed shortness of breath 10 min after eating a chocolate bar with peanuts	
Allergies: Peanut allergy	

Continued

Team Leader	Team Members
Medications: Children's vitamin	
Past medical history: Healthy, previous chicken pox, previous allergic reaction to peanuts	
Last meal: Breakfast this morning; chocolate bar 30 min ago	
Events: Difficulty breathing began 10 min after eating chocolate bar	
Physical Exam:	
Order repeat vital signs	Vital signs: HR 118, RR 26, BP 112/76, O_2 sat 97%
General: Well nourished, appears anxious	
Head and neck: Normal findings except for inspiratory stridor	
Pulmonary: Pulmonary rhonchi bilaterally	
Heart: Normal heart sounds, no murmurs, rubs, gallops	
Abdomen: Normal, bowel sounds active, nontender	
Extremities: Distal pulses palpable, capillary refill 2 sec, normal movement and strength	
Back: Normal	
Neurological: Normal reflexes	
Identify:	
Respiratory distress	
Upper airway obstruction probably due to airway edema, anaphylactic reaction (peanut allergy)	

Team Leader	Team Members
Intervention:	**Perform interventions as ordered:**
Airway patency: Place patient in most comfortable position	Assist patient to assume most comfortable position
Order epinephrine 0.01 mg/kg IM; may repeat every 10–15 min	Administer epinephrine 0.01 mg/kg IM, confirm when given
	Establish patient's weight from family, or use color-coded resuscitation tape to estimate weight for appropriate dose
Order IV/IO access, NS IV	Establish IV/IO access, start NS IV
Consider other treatments if needed: Racemic epinephrine, dexamethasone IV/IM, helium-oxygen mixture, CPAP	
Order preparation for ET intubation	Prepare for ET intubation
Diagnostic Tests	
Imaging: If you suspect infection, order AP/lateral neck and chest x-ray (CXR); evaluate for subglottic narrowing, rule out retropharyngeal abscess, evaluate for pulmonary infiltrates, edema	Assist in obtaining ordered diagnostic tests
Lab data: Defer for first 5–10 min; if appropriate, order ABGs, electrolytes, CBC with differential, blood culture, BUN, creatinine, blood glucose	
Reevaluate: Evaluate efficacy of interventions	Discuss team dynamics; analyze and summarize megacode

Critical Thinking:

1. **What was the cause of this patient's respiratory distress?**
   The cause is airway edema resulting from ingestion of a chocolate bar with peanuts in a patient with a history of peanut allergy.

2. **Was this patient in respiratory distress or respiratory failure?**
   The patient was in respiratory distress. Signs and symptoms included tachypnea, tachycardia, hypoxemia, nasal flaring, suprasternal retractions, inspiratory stridor, diminished breath sounds, and pallor. The patient remained conscious with normal peripheral pulses, blood pressure, and capillary refill. Oxygen saturation improved with oxygen administration.
3. **What signs and symptoms would indicate respiratory failure?**
   Signs and symptoms of deterioration such as bradypnea, decreased respiratory effort, bradycardia, poor to absent distal air movement, hypoxemia despite oxygen administration, possible cyanosis, decreased LOC, lethargy, stupor, or coma.
4. **What are additional interventions for respiratory distress or failure caused by anaphylaxis?**
   Additional interventions include albuterol MDI or nebulizer for patients with wheezing/bronchospasm, IV diphenhydramine and H_2 blocker, and IV corticosteroid. If airway edema persists, prepare for intubation or emergency tracheostomy or cricothyroidotomy. If the patient becomes hypotensive, administer 20-mL/kg bolus of NS or lactated Ringer's IV; repeat as needed.
5. **What are other causes of upper airway obstruction?**
   Other causes include foreign body airway obstruction, airway trauma, an abscess or tumor, thick secretions, congenital upper airway anomaly, and airway edema caused by tonsillar hypertrophy, croup, or epiglottitis.
6. **How should a foreign body airway obstruction be managed?**
   For a partial obstruction, coach the patient to cough, which may dislodge the obstruction. For a complete obstruction in a conscious patient with inability to cough effectively and inability to make vocal sounds, perform back blows and chest thrusts on infants younger than 12 months, or abdominal thrusts on children 1 year or older. Be prepared to suction the airway. If the patient becomes unresponsive, perform CPR. The patient may require intubation, or if unsuccessful, emergency tracheostomy or cricothyroidotomy.
7. **If the patient's upper airway obstruction was caused by croup, how should the patient be managed?**
   Administer a high concentration of oxygen using a nonrebreather mask. If the patient requires ventilatory support, assist with bag-mask ventilations. Consider administration of dexamethasone IV or IM. Consider inserting an ET slightly smaller than predicted for that child. If necessary, prepare for emergency tracheostomy or cricothyroidotomy.

PALS Megacode Case 2: Shock

Scenario: A 13-year-old boy presents after a 2-day bout with fever, abdominal pain, nausea, vomiting, and diarrhea.

Team Leader	Team Members
Initial Observation	
Evaluate:	
LOC: Lethargic	
Breathing: Normal effort, rate slightly increased	
Skin color: Pale	
Identify:	
Possible dehydration, possible shock	
Intervention:	**Perform interventions as ordered:**
Activate emergency response	Call for a rapid response
Airway, Breathing: Order airway management, O_2	Maintain open airway Apply pulse oximeter, evaluate O_2 sat Apply nasal cannula Administer O_2
Circulation: Order monitor	Get cardiac monitor, apply ECG leads or pads
Primary Assessment	**Perform primary assessment as ordered. Findings:**
Evaluate:	
Airway: Patent	**Airway:** Patent
Breathing: Order assessment of breathing	**Breathing:** RR 22, normal respiratory effort, normal breath sounds, O_2 sat 99% on 2 L/min nasal cannula
Circulation: Order assessment of circulation	**Circulation:** HR 130, BP 84/62, peripheral pulses weak, capillary refill 4 sec, skin pale, cool, clammy
Disability: Order neurological assessment	**Disability:** Lethargic, mumbling, does not answer questions

Continued

Team Leader	Team Members
Exposure: Order removal of clothing, rapid examination of body	**Exposure:** Remove clothing, examine body: no signs of trauma, wounds, deformities, bruising, bleeding, rashes, petechiae, purpura; extremities nontender; temperature 38.4°C (101.2°F)
Identify:	
Hypotensive shock	
Hypovolemic shock	
Intervention:	**Perform interventions as ordered:**
Analyze heart **rhythm: Sinus tachycardia**	Monitor: Sinus tachycardia
Order IV/IO access	Establish IV/IO access
Order rapid 20 mL/kg IV bolus of isotonic crystalloid	Administer 20 mL/kg IV isotonic crystalloid by rapid IV bolus
	(If weight unknown, use color-guided resuscitation tape)
Assess response to O_2 and IV volume resuscitation	
Secondary Assessment:	
Evaluate:	
SAMPLE history	
Signs and symptoms: Second day of persistent fever, nausea, vomiting, diarrhea and abdominal pain	
Allergies: No known allergies	
Medications: Children's vitamin	
Past medical history: Healthy, tonsillectomy at age 9 years	
Last meal: Ginger ale last night; no solid food for 2 days	

Team Leader	Team Members
Events: Developed anorexia and malaise 3 days ago; fever, vomiting, diarrhea and abdominal pain started 2 days ago	
Physical Exam:	
Order repeat vital signs	Vital signs: HR 144, RR 25, BP 78/50, O_2 sat 98%
General: Lethargic	
Head and neck: Dry mucous membranes, eyes sunken	
Pulmonary: Breath sounds clear bilaterally	
Heart: Normal heart sounds, no murmurs, rubs, gallops	
Abdomen: Hyperactive bowel sounds, diffusely tender to palpation without guarding	
Extremities: Distal pulses weak, capillary refill 4 sec; no rashes, no edema, dry, cool to touch; normal movement, weak	
Back: Normal	
Neurological: Normal reflexes	
Identify:	
Hypotensive shock	
Hypovolemic shock: identify and treat the cause	
Intervention:	**Perform interventions as ordered:**
Order repeat IV fluid bolus	Administer 20 mL/kg IV isotonic crystalloid by rapid IV bolus
Reevaluate vital signs, order repeat IV fluid bolus as needed	Assess vital signs; administer repeat IV fluid bolus if needed

Continued

Team Leader	Team Members
Order measurement of intake and output	Measure intake and output: Urine output decreased
Diagnostic Tests:	
Lab data: Blood glucose: 78 mg/dL; ABG: pH 7.30, PCO_2 33 mm Hg, HCO_3 16 mmol/L, PO_2 218 mm Hg; CBC with differential, electrolytes, calcium, magnesium, BUN, creatinine, blood culture, serum lactate, urinalysis	Assist in obtaining ordered diagnostic tests
Imaging: CXR: Lungs clear, heart normal size	
Reevaluate: Evaluate efficacy of interventions	Discuss team dynamics; analyze and summarize megacode

Critical Thinking:

1. **Does this scenario represent a case of compensated shock or decompensated shock?**
   This scenario represents a case of decompensated shock because the patient is hypotensive. A normal BP for a boy this age should be systolic 95–131 mm Hg and diastolic 45–85 mm Hg. This patient's BP was 84/62 with a narrowed pulse pressure of 22 mm Hg. He is lethargic because of inadequate cerebral perfusion.
2. **What are other causes of hypovolemic shock?**
   Other causes include internal or external blood loss, large burns, osmotic diuresis related to DKA, and third-space losses.
3. **What do this patient's arterial blood gas results indicate?**
   The patient has a partially compensated metabolic acidosis (decreased peripheral perfusion has caused hypoxemia and lactic acidosis), indicated by a low bicarbonate level, and a low PCO_2 level (respiratory alkalosis, hypocarbia, hypocapnia caused by tachypnea trying to compensate for metabolic acidosis). Metabolic acidosis will be corrected when blood pressure is adequate for both peripheral and central perfusion.
4. **The patient is being treated with isotonic crystalloid fluid. Should albumin be administered?**
   No. Albumin is a colloid with a higher osmolality and would draw fluid from the interstitium into the vascular space, increasing blood volume and BP. Although it would be helpful to raise BP, it is not indicated because the patient is dehydrated from vomiting and diarrhea and has decreased

interstitial fluid volume. Albumin is appropriate for interstitial edema ("third spacing") with hypotension. It would shift fluid from the interstitium into the vascular space, increasing BP and reducing interstitial edema.

5. **How should a patient presenting with hemorrhagic shock be treated?** Interventions should begin with 20 mL/kg of isotonic crystalloid fluid IV, using the rule of 3 mL for every 1 mL of estimated blood loss. If the patient remains hypotensive after three boluses, consider blood replacement with 10 mL/kg boluses of warmed PRBCs. The source of blood loss should be identified and treated.

PALS Megacode Case 3: Cardiac—SVT

Scenario: An 8-month-old infant girl presents with acute respiratory distress.

Team Leader	Team Members
Initial Observation	
Evaluate:	
LOC: Arousable, eyes closing, sleepy	
Breathing: Increased respiratory effort, increased rate	
Skin color: Pale, mottled	
Identify:	
Respiratory distress	
Possible shock	
Intervention:	**Perform interventions as ordered:**
Activate emergency response	Call for a rapid response
Airway, Breathing: Order airway management, O_2	Maintain open airway Apply pulse oximeter, evaluate O_2 sat Apply nonrebreather mask Administer high-flow O_2
Circulation: Order monitor	Get cardiac monitor, apply ECG leads or pads

Continued

Team Leader	Team Members
Primary Assessment	**Perform primary assessment as ordered. Findings:**
Evaluate:	
Airway: Patent	**Airway:** Patent
Breathing: Order assessment of breathing	**Breathing:** RR 45, increased respiratory effort, slight nasal flaring and intercostal retractions, O_2 sat 93% on room air, increased to 98% on O_2
Circulation: Order assessment of circulation	**Circulation:** HR 240, BP 82/56, normal central pulses, peripheral pulses adequate, capillary refill 2 sec, skin cool, diaphoretic
Disability: Order neurological assessment	**Disability:** Awake, sleepy
Exposure: Order removal of clothing, rapid examination of body	**Exposure:** Remove clothing, examine body: no signs of trauma, wounds, deformities, bruising, bleeding, rashes, petechiae, purpura; extremities nontender; temperature 37.4°C (99.4°F)
Identify:	
Respiratory distress	
Identify type of tachycardia	
Intervention:	**Perform intervention as ordered:**
Analyze heart rhythm: **Narrow-complex tachycardia, HR 240: SVT**	Monitor: Narrow-complex tachycardia, HR 240, regular
Secondary Assessment	
Evaluate:	
SAMPLE history	
Signs and symptoms: Awake, sleepy, rapid respiratory rate, diaphoretic	
Allergies: No known allergies	

Team Leader	Team Members
Medications: None	
Past medical history: Healthy full-term infant	
Last meal: 5 oz of formula 2 hr ago	
Events: Sudden onset of respiratory distress	
Physical Exam:	
Order repeat vital signs	Vital signs: HR 240, RR 45, BP 78/54, O_2 sat 98%
General: Remains sleepy, arousable, weak cry in response to painful stimuli	
Head and neck: Normal except for slight nasal flaring	
Pulmonary: Breath sounds clear bilaterally, mild intercostal retractions	
Heart: Normal heart sounds, no murmurs, rubs, gallops	
Abdomen: Normal bowel sounds, nontender	
Extremities: Distal pulses adequate, capillary refill 2 sec; no rashes, no edema, cool, diaphoretic	
Back: Normal	
Neurological: Normal reflexes	
Identify:	
Respiratory distress	
Cardiac arrhythmia: **SVT with adequate perfusion**	
Intervene:	**Perform interventions as ordered:**
Order vagal maneuvers, monitor response	Perform vagal maneuvers

Continued

Team Leader	Team Members
Order IV/IO access	Establish IV/IO access
Order adenosine 0.1 mg/kg rapid IV/IO push	Give adenosine 0.1 mg/kg rapid IV/IO push, flush with 5–10 mL IV NS; confirm when given (If weight unknown, use color-guided resuscitation tape)
Assess rhythm; if SVT persists, order adenosine 0.2 mg/kg rapid IV/IO push	Give adenosine 0.2 mg/kg rapid IV/IO push, flush with 510 mL IV NS; confirm when given
Assess rhythm; if SVT persists, seek expert consultation	
Order 12-lead ECG	Obtain 12-lead ECG
Prepare to administer amiodarone 5 mg/kg IV over 20–60 min or procainamide 15 mg/kg IV over 30–60 min	Give amiodarone or procainamide if ordered; confirm when given
Assess rhythm; if SVT persists, order sedation and analgesia	Give sedation and analgesia as ordered; confirm when given
Order **cardioversion** at 0.5–1 J/kg	Apply pads, press Sync button on monitor-defibrillator, clear area, remove O_2, deliver shock
Assess rhythm; if SVT persists, order **cardioversion** at 2 J/kg	Repeat cardioversion with increased energy
Diagnostic Tests	
Lab data: ABGs, electrolytes, glucose, calcium, magnesium, BUN, creatinine **Imaging:** CXR, 12-lead ECG	Assist in obtaining ordered diagnostic tests
Reevaluate: Evaluate efficacy of interventions	Discuss team dynamics; analyze and summarize megacode

Critical Thinking:

1. **How can you tell the difference between sinus tachycardia and SVT?**
   Sinus tachycardia is marked by visible P waves with a constant PR interval and somewhat variable R-R intervals. HR increases gradually rather than abruptly. HR in infants is less than 220 bpm, and in children it is less than

180 bpm. SVT has no visible P waves and has regular R-R intervals. Onset is abrupt. HR in infants is 220 bpm or more and 180 bpm or more in children.

2. **Should sinus tachycardia be treated with adenosine?**
   No. Sinus tachycardia is not an arrhythmia. It is a physiological response to increased metabolic demands and is a compensatory response to hypotension to maintain cardiac output. It may also be a response to certain medications, such as beta-adrenergic agents. The cause of sinus tachycardia should be identified and treated.
3. **What are examples of vagal maneuvers and why are they indicated for treatment of stable SVT?**
   Examples of vagal maneuvers include applying ice to the patient's face, having the patient bear down as if having a bowel movement, or having the patient blow through an obstructed straw. The last two require the patient's cooperation and are clearly not indicated for an infant. Another example is carotid massage, which must be performed by a qualified provider. Vagal maneuvers cause parasympathetic innervation of the AV node, slowing down AV node conduction; this interrupts the reentry circuit or pathway causing SVT and restores sinus rhythm.
4. **How does adenosine convert SVT to sinus rhythm?**
   Adenosine slows AV node conduction to interrupt the reentry circuit or pathway causing SVT and restore sinus rhythm.
5. **How should adenosine be administered?**
   Adenosine has a very short half-life, less than 10 sec. It should be administered over 1–2 sec and followed by a 5–10-mL IV NS flush. Try to use a peripheral IV site as proximal to the trunk as possible, such as the upper arm. See Tab 5: Emergency Medications for further information.

PALS Megacode Case 4: Cardiac—Bradycardia

Scenario: A 5-year-old boy presents with decreased LOC.

Team Leader	Team Members
Initial Observation	
Evaluate:	
LOC: Decreased responsiveness	
Breathing: Shallow, slow respirations	
Skin color: Pale skin and mucous membranes	

Continued

Team Leader	Team Members
Identify:	
Acute, life-threatening problem	
Intervention:	**Perform interventions as ordered:**
Activate emergency response	Call for a rapid response
Airway, Breathing: Order airway management, O_2	Open airway Apply pulse oximeter, evaluate O_2 sat Apply nonrebreather mask Administer 100% O_2
Circulation: Order monitor	Get cardiac monitor, apply ECG leads or pads
Primary Assessment	**Perform primary assessment as ordered. Findings:**
Evaluate:	
Airway: Noisy, snoring	**Airway:** Noisy, snoring
Breathing: Order assessment of breathing; order bag-mask ventilation	**Breathing:** RR 5, shallow, inadequate chest rise, O_2 sat 75% on room air Deliver assisted ventilation: O_2 sat increases to 100% with adequate chest rise, adequate bilateral breath sounds
Circulation: Order assessment of circulation	**Circulation:** HR 45, central pulses palpable, peripheral pulses weak, capillary refill 5 sec, skin cool, diaphoretic Defer BP assessment
Disability: Very decreased responsiveness; defer neurological assessment	
Exposure: Defer	
Identify:	
Respiratory failure	
Cardiopulmonary failure	

Team Leader	Team Members
Bradycardia with poor perfusion	
Intervention:	
Analyze heart rhythm: **Sinus bradycardia**	Monitor: Sinus bradycardia
Order 12-lead ECG to be done when heart rate and perfusion improve	
Instruct team to begin **high-quality CPR**, starting with oxygenation and ventilation	Provide **high-quality CPR**, starting with oxygenation and ventilation
Assess HR response to O_2 and ventilation; if heart rate remains <60 with poor perfusion after effective oxygenation and ventilation, continue chest compressions and bag-mask ventilation; consider advanced airway if indicated	HR: 50 Continue **high-quality CPR** Reevaluate HR and vital signs after 2 min of CPR
Secondary Assessment	
Evaluate:	
SAMPLE history	
Signs and symptoms: Lethargic, minimally responsive to pain stimuli	
Allergies: No known allergies	
Medications: None	
Past medical history: Healthy child	
Last meal: Dinner last evening	
Events: Slept at grandmother's house last night; found in bed this morning with empty bottle of grandmother's pills	
Physical Exam:	
Order repeat vital signs. If heart rate <60 bpm, order resumption of **high-quality CPR**	Vital signs: HR 50, RR 16 by bag-mask ventilation, BP 72/46, O_2 sat 96%

Continued

Team Leader	Team Members
General: Remains poorly responsive	
Head and neck: Normal	
Pulmonary: Breath sounds clear bilaterally	
Heart: Normal heart sounds, no murmurs, rubs, gallops	
Abdomen: Normal bowel sounds, nontender, nondistended, no palpable masses	
Extremities: Hands and feet cool, pale; no edema, no rash, weak peripheral pulses, capillary refill 5 sec	
Back: Normal	
Neurological: Pupils equal, reactive; minimally responsive to pain	
Identify:	
Respiratory failure	
Cardiac arrhythmia: **Bradycardia with poor perfusion**	
Intervene:	**Perform interventions as ordered:**
Order **high-quality CPR** Reevaluate HR/vital signs every 2 min	Continue **high-quality CPR**
Order IV/IO access	Establish IV/IO access
Order epinephrine 0.01 mg/kg IV/IO push; repeat every 3–5 min as needed	Give epinephrine 0.01 mg/kg IV/IO push, flush with 5–10 mL IV NS; confirm when given
	(If weight unknown, use color-guided resuscitation tape)
Identify ingested pills, order appropriate antidote if needed	Administer antidote if ordered
Prescription label: Metoprolol tartrate 25 mg twice daily	

Team Leader	Team Members
Antidote: Atropine, calcium chloride, or calcium gluconate	
Order preparation for ET intubation	Prepare for ET intubation
Obtain expert consultation	
Reevaluate vital signs every 2 min; if HR >60 bpm, stop CPR	Vital signs: HR 72, RR 20, BP 88/60, O_2 sat 98%; stop CPR
Diagnostic Tests:	
Lab data: ABGs, electrolytes, glucose, calcium, BUN, creatinine, magnesium, phosphorus, toxicology screen **Imaging:** CXR, 12-lead ECG, CT of brain	Assist in obtaining ordered diagnostic tests
Reevaluate: Evaluate efficacy of interventions	Discuss team dynamics; analyze and summarize megacode

Critical Thinking:

1. **Why should oxygenation and assisted ventilation be performed before chest compressions start?**
   Sinus bradycardia may be caused by hypoxia. HR may improve with oxygen administration and ventilatory support.
2. **When should chest compressions be performed for an infant or child presenting with bradycardia?**
   When an infant's or child's HR is less than 60 bpm with signs of poor perfusion, high-quality CPR should be performed to improve circulation.
3. **Why should epinephrine be administered for sinus bradycardia that does** not improve with oxygenation and ventilation?
   Epinephrine stimulates beta-adrenergic receptors in the heart to increase HR. It also stimulates alpha-adrenergic receptors in peripheral arteries to improve perfusion.
4. **What other medication may be administered if epinephrine is ineffective in increasing HR?**
   Atropine is appropriate if the bradycardia is caused by increased vagal tone or primary AV block, and is appropriate for beta blocker and calcium channel blocker overdose. Administer atropine 0.02 mg/kg IV/IO push (minimum dose 0.1 mg, maximum dose 0.5 mg). It may be repeated once in 3–5 min if necessary.

5. **If epinephrine and atropine are ineffective in increasing HR, what other interventions can be performed?**
   TCP or transvenous pacing should be initiated to increase HR. Pacing may be required for bradycardia caused by complete heart block or by sinus node dysfunction that remains unresponsive to oxygenation, ventilation, and medication. Pacing may also be required when bradycardia is associated with congenital or acquired heart disease. The cause of the bradycardia should be identified and appropriately treated.

PALS Megacode Case 5: Cardiac—Asystole

Scenario: A 4-year-old girl who presented with initial respiratory distress has now become unresponsive.

Team Leader	Team Members
Initial Observation	
Evaluate:	
LOC: Unresponsive, no spontaneous movement, body limp	
Breathing: No spontaneous respirations	
Skin color: Pallor and some cyanosis in extremities and lips, mottling	
Identify:	
Life-threatening condition	
Intervention:	**Perform interventions as ordered:**
Activate emergency response	Call a code or call 911
Order initiation of **high-quality CPR**, 100% O_2	Initiate **high-quality CPR** Open airway
	Bag-mask ventilation with 100% O_2
Circulation: Order cardiac monitor and pulse oximeter	Get cardiac monitor and pulse oximeter, apply ECG leads or pads, evaluate O_2 sat

Team Leader	Team Members
Primary Assessment	**Perform primary assessment as ordered. Findings:**
Evaluate:	
Airway: Order assessment of airway patency	**Airway:** Patent with head tilt–chin lift
Breathing: Order assessment of breathing; order bag-mask ventilation	**Breathing:** Apneic; adequate chest rise with bag-mask ventilation, 100% O_2
Circulation: Order assessment of circulation	**Circulation:** No central pulse, defer BP assessment
Disability: Defer	
Exposure: Defer	
Identify:	
Cardiac arrest	
Intervention:	
Analyze heart **rhythm: Asystole**	Monitor: Asystole
Order resumption of **high-quality CPR**	Continue **high-quality CPR**
Secondary Assessment	
Evaluate:	
Consider Hs and Ts: Attempt to identify reversible causes of asystole	
SAMPLE history: Deferred until ROSC	
Physical Exam: Deferred	
Identify:	
Cardiac arrest: **Asystole**	
Intervene:	**Perform interventions as ordered:**
Ensure provision of continuous **high-quality CPR**	Perform 2 min of **high-quality CPR**

Continued

Team Leader	Team Members
Ensure adequate ventilation and oxygenation; if inadequate with bag-mask ventilation, order ET intubation	Provide ventilation and oxygenation, observe for adequate chest rise
Order IV/IO access	Establish IV/IO access
Order epinephrine 0.01 mg/kg IV/IO	Give epinephrine 0.01 mg/kg IV/IO, flush with 5–10 mL IV NS; confirm when given
	Recorder: Indicate when 2 min of CPR is done
Order to stop CPR, analyze **rhythm: Asystole**	Stop CPR; monitor: Asystole
Order resumption of **high-quality CPR**	Change compressor, resume **high-quality CPR**
Order repeat epinephrine 0.01 mg/kg IV/IO every 3–5 min	Give epinephrine every 3–5 min as ordered; confirm when given
	Recorder: Indicate when each 2 min of CPR is done
Order to stop CPR, analyze **rhythm: Asystole**	Stop CPR; monitor: Asystole
Order resumption of **high-quality CPR**	Change compressor, resume **high-quality CPR**
Reassess rhythm every 2 min	Check rhythm
Identify and treat reversible causes	Participate in discussion to identify reversible causes
Assess for ROSC if rhythm changes to organized rhythm	Perform interventions if ordered
Diagnostic Tests:	
Lab data: ABGs, electrolytes, glucose, calcium, BUN, creatinine, magnesium **Imaging:** CXR, 12-lead ECG	Assist in obtaining ordered diagnostic tests
Reevaluate: Evaluate efficacy of interventions	Discuss team dynamics; analyze and summarize megacode

Critical Thinking:

1. **What is the maximum single dose of epinephrine when given IV/IO?**
   The maximum single dose of epinephrine is 1 mg.
2. **What is the maximum number of doses of epinephrine that can be administered in cardiac arrest?**
   There is no maximum number of doses. It should be administered every 3–5 min until ROSC, or until a decision is made to terminate resuscitation efforts.
3. **Should atropine be administered for asystole?**
   Atropine is not recommended for asystole. It works by decreasing vagal tone to the heart. Asystole is rarely caused by excessive vagal tone.
4. **How should CPR be performed after insertion of an advanced airway?**
   Insertion of an advanced airway should be considered if basic airway management is ineffective. Following insertion, compressions should be delivered continuously for 2 min, without interruption for breaths, at 100 compressions/min. One breath should be delivered every 6–8 sec. After 2 min the rhythm should be checked. If cardiac arrest continues, change compressor to avoid fatigue and resume another 2 min of CPR.

PALS Megacode Case 6: Cardiac—VF

Scenario: An 18-month-old boy was found listless, cyanotic, and apneic in his crib.

Team Leader	Team Members
Initial Observation	
Evaluate:	
LOC: Unresponsive, no spontaneous movement, body limp	
Breathing: No spontaneous respirations	
Skin color: Pallor and some cyanosis in extremities and lips, mottling	
Identify:	
Life-threatening condition	

Continued

Team Leader	Team Members
Intervention:	**Perform interventions as ordered:**
Activate emergency response	Call a code or call 911
Order initiation of **high-quality CPR**, 100% O_2	Initiate **high-quality CPR** Open airway
	Bag-mask ventilation with 100% O_2
Circulation: Order cardiac monitor and pulse oximeter	Get cardiac monitor and pulse oximeter, apply ECG leads or pads, evaluate O_2 sat
Primary Assessment	**Perform primary assessment as ordered. Findings:**
Evaluate:	
Airway: Order assessment of airway patency	**Airway:** Patent with head tilt–chin lift
Breathing: Order assessment of breathing; order bag-mask ventilation	**Breathing:** Apneic; adequate chest rise with bag-mask ventilation, 100% O_2
Circulation: Order assessment of circulation	**Circulation:** No central pulse; continue chest compressions; defer BP assessment
Disability: Defer	
Exposure: Defer	
Identify:	
Cardiac arrest	
Intervention:	**Perform interventions as ordered:**
Analyze heart rhythm: VF	Monitor: VF
Order **defibrillation** at 2 J/kg using pediatric pads or paddles	Remove O_2, clear the area, deliver shock
(If weight unknown, use color-guided resuscitation tape)	
Order resumption of **high-quality CPR**	Change compressor; immediately resume **high-quality CPR**
Secondary Assessment	

Team Leader	Team Members
Evaluate:	
Consider Hs and Ts: Attempt to identify reversible causes of VF	Participate in discussion to identify reversible causes
SAMPLE history: Deferred until ROSC	
Physical Exam: Deferred	
Identify:	
Cardiac arrest: **VF**	
Intervene:	**Perform interventions as ordered:**
Ensure **high-quality CPR**	Perform 2 min of **high-quality CPR**
Ensure adequate ventilation and oxygenation; if inadequate with bag-mask ventilation, order ET intubation	Provide ventilation and oxygenation, observe for adequate chest rise
Order IV/IO access	Establish IV/IO access
Order epinephrine 0.01 mg/kg IV/IO	Give epinephrine 0.01 mg/kg IV/IO, flush with 5–10 mL IV NS; confirm when given
	Recorder: Indicate when 2 min of CPR is done
Order to stop CPR, **analyze rhythm: VF**	Stop CPR; Monitor: VF
Order **defibrillation** at 4 J/kg	Remove O_2, clear the area, deliver shock
Order resumption of **high-quality CPR**	Change compressor; immediately resume **high-quality CPR**
Order amiodarone 5 mg/kg IV/IO	Give amiodarone 5 mg/kg IV/IO, flush with 5–10 mL IV NS; confirm when given
	Recorder: Indicate when 2 min of CPR is done
Order to stop CPR, analyze **rhythm: Sinus rhythm**	**Stop CPR**; Monitor: Sinus rhythm, pulse palpable

Continued

Team Leader	Team Members
Order vital signs	HR 126, RR 22, BP 75/52, O_2 sat 99%
Transport patient to pediatric critical care unit	Transport patient to pediatric critical care unit
Formulate comprehensive care plan	Initiate care plan
Identify and treat cause(s) of VF	Discuss and treat cause(s) of VF
Diagnostic Tests	
Lab data: ABGs, electrolytes, glucose, calcium, BUN, creatinine, magnesium **Imaging:** CXR, 12-lead ECG	Assist in obtaining ordered diagnostic tests
Reevaluate Evaluate efficacy of interventions	Discuss team dynamics; analyze and summarize megacode

Critical Thinking:

1. **What is the maximum energy recommended to defibrillate for VF in pediatric patients?**
   The maximum is 10 J/kg up to 360 J.
2. **If VF occurs in an infant younger than 1 year, can an AED be used?**
   A manual defibrillator is preferred, but if one is not available, an AED may be used. Preferably it should have a dose attenuator for pediatric defibrillation in infants and children up to age 8 or weighing up to 25 kg. If necessary, an AED without a dose attenuator may be used even in infants because it is urgent to deliver a shock to terminate VF or pulseless VT.
3. **If pediatric pads are not available, can adult pads be used to defibrillate an infant?**
   Pediatric pads are preferred for infants weighing up to 10 kg (up to approximately 1 year old). If pediatric pads are not available, adult pads may be used but must be spaced at least 3 cm apart on the infant's chest. Adult pads are used for children weighing more than 10 kg (1 year to adulthood).
4. **When should medications be administered during CPR?**
   Medications should be prepared during the 2 min of CPR, before stopping for a rhythm check. They can be administered at the beginning of the next 2 min of CPR during chest compressions to optimize delivery.

Tab 8: Tools

Abbreviations

ABG	arterial blood gas
ACE	angiotensin-converting enzyme
ACLS	advanced cardiovascular life support
ACS	acute coronary syndrome
AED	automated external defibrillator
A-fib	atrial fibrillation
A-flutter	atrial flutter
APTT	activated partial thromboplastin time
ARDS	acute respiratory distress syndrome
AV	atrioventricular
BiPAP	[noninvasive ventilation with both inspiratory and expiratory pressures above atmospheric levels]
BLS	basic life support
BP	blood pressure
BUN	blood urea nitrogen
CABG	coronary artery bypass graft
CAD	coronary artery disease
CBC	complete blood count
CHF	congestive heart failure
CNS	central nervous system
CO	cardiac output
CO_2	carbon dioxide
COPD	chronic obstructive pulmonary disease
CPAP	continuous positive airway pressure
CPR	cardiopulmonary resuscitation
CT	computed tomography
CXR	chest x-ray
DIC	disseminated intravascular coagulation
DKA	diabetic ketoacidosis
DVT	deep-vein thrombosis
ECG	electrocardiogram
ECLS	extracorporeal life support
ECMO	extracorporeal membrane oxygenation
EMS	emergency medical services
ET	endotracheal tube
FiO_2	fraction of inspired oxygen
GERD	gastroesophageal reflux disease
H_1	histamine 1
H_2	histamine 2
Hs and Ts	[mnemonic for possible causes of cardiac arrest]

HCO_3^-	bicarbonate
HIT	heparin-induced thrombocytopenia
HR	heart rate
HTN	hypertension
ICD	implanted cardioverter–defibrillator
ICP	intracranial pressure
ICU	intensive care unit
IHSS	idiopathic hypertrophic subaortic stenosis
IM	intramuscular
INR	international normalized ratio
IO	intraosseous
IV	intravenous
IVP	intravenous push
LA	left arm
LBBB	left bundle branch block
LL	left leg
LMA	laryngeal mask airway
LOC	level of consciousness
LT	laryngeal tube
MAT	multifocal atrial tachycardia
MDI	metered-dose inhaler
MET	medical emergency team
MI	myocardial infarction
MRI	magnetic resonance imaging
NIHSS	National Institutes of Health Stroke Scale
NPA	nasopharyngeal airway
NS	normal saline
NSAID	nonsteroidal anti-inflammatory drug
NSR	normal sinus rhythm
NSTEMI	non–ST-elevation myocardial infarction
O_2	oxygen
OPA	oropharyngeal airway
$PaCO_2$	partial pressure of carbon dioxide in arterial blood
PALS	pediatric advanced life support
PaO_2	partial pressure of oxygen in arterial blood
PCI	percutaneous coronary intervention
PE	pulmonary embolism
PEA	pulseless electrical activity
PEEP	positive end-expiratory pressure
PEFR	peak expiratory flow rate
$PETCO_2$	partial pressure of end-tidal carbon dioxide
PO	by mouth (per os)
PO_2	partial pressure of oxygen
prn	as needed (pro re nata)
PSVT	paroxysmal supraventricular tachycardia

PVC	premature ventricular contraction
$PvCO_2$	partial pressure of carbon dioxide in venous blood
PvO_2	partial pressure of oxygen in venous blood
QTc	duration of QT interval corrected for heart rate
RA	right arm
RBBB	right bundle branch block
RBC	red blood cell
RCA	right coronary artery
RL	right leg
ROSC	return of spontaneous circulation
RR	respiratory rate
RRT	rapid response team
rtPA	recombinant tissue-type plasminogen activator
RV	right ventricle
SaO_2	hemoglobin oxygen saturation in arterial blood
sat	saturation
sc	subcutaneously
$S_{cv}O_2$	central venous hemoglobin oxygen saturation
sl	sublingual
SQ	subcutaneous
STEMI	ST-elevation myocardial infarction
SvO_2	venous oxygen hemoglobin saturation
SVR	systemic vascular resistance
SVT	supraventricular tachycardia
Sync	synchronized
TCP	transcutaneous pacing
TIA	transient ischemic attack
UA	unstable angina
UFH	unfractionated heparin
VAD	ventricular assist device
VBG	venous blood gas
VF	ventricular fibrillation
V/Q	ventilation-perfusion
VT	ventricular tachycardia
WAP	wandering atrial pacemaker

Illustration Credits

ECG strips on pages 11–35 from Jones, SA: Author's personal collection.

Pages 2, 3, 8, 9, 10, 157–159, 161 from Jones, SA: ECG Notes, ed 2. F. A. Davis, Philadelphia, 2010.

Pages 39, 40 (bottom), 41–42, 44–45, 48–51, 53–58, 142–144 from Jones, SA: First Aid, Survival, and CPR. F. A. Davis, Philadelphia, 2012.

Page 141, 151 (top) from Myers E: RNotes, ed 3. F. A. Davis, Philadelphia, 2011.

Selected References

1. American Heart Association: Advanced Life Support for Healthcare Providers (Student Manual). American Heart Association, Dallas, TX, 2011.
2. American Heart Association: Basic Life Support for Healthcare Providers (Student Manual). American Heart Association, Dallas, TX, 2011.
3. American Heart Association: Pediatric Life Support for Healthcare Providers (Student Manual). American Heart Association, Dallas, TX, 2011.
4. American Heart Association: Guidelines for CPR and ECG. Supplement to *Circulation* 122(18), November 2, 2010.
5. Jones, SA: ECG Notes, ed 2. F. A. Davis, Philadelphia, 2013.
6. Jones SA: First Aid, Survival, and CPR. F. A. Davis, Philadelphia, 2012.
7. Myers E: RN Notes, ed 3. F. A. Davis, Philadelphia, 2011.
8. Hopkins T: Med Surg Notes, ed 3. F. A. Davis, Philadelphia, 2011.

Index

Page numbers followed by "f" denote figures and "t" denote tables