ACLS Provider Manual Supplementary Material

American Heart Association.

Contents

Airway Management

Basic Airway Management

Devices to Provide Supplemental Oxygen

Oxygen administration is often necessary for patients with acute coronary syndromes (ACS), pulmonary distress, or stroke. Various devices can deliver supplemental oxygen from 21% to 100% (Table 1). This section describes 4 devices to provide supplemental oxygen:

- Nasal cannula
- Simple oxygen face mask
- Venturi mask
- Face mask with oxygen reservoir

Whenever you care for a patient receiving supplemental oxygen, quickly verify the proper function of the oxygen delivery system in use.

Table 1. Delivery of Supplemental Oxygen: Flow Rates and Percentage of Oxygen Delivered

Device	Flow rates (L/min)	Delivered oxygen (%)*
Nasal cannula	1 2 3 4 5 6	21-24 25-28 29-32 33-36 37-40 41-44
Simple oxygen face mask	6-10	35-60
Venturi mask	4-8 10-12	24-40 40-50
Face mask with oxygen reservoir (nonrebreathing mask)	10-15	95-100
*Percentages are approximate.		

Oxygen Supply

Oxygen supply refers to an oxygen cylinder or wall unit that connects to an administration device to deliver oxygen to the patient. When the patient is receiving oxygen from one of these systems, be sure to check the following equipment:

- Oxygen administration device
- Valve handles to open the cylinder
- Pressure gauge
- Flow meter

- Tubing that connects the oxygen supply to the patient's oxygen administration device

Trained advanced cardiovascular life support (ACLS) providers should be sure they are familiar with all emergency equipment before an emergency arises.

Nasal Cannula

Traditionally, the nasal cannula (Figure 1) is classified as a low-flow oxygen administration system designed to add oxygen to room air when the patient inspires. The ultimate inspired oxygen concentration is determined by the oxygen flow rate through the cannula and by how deeply and rapidly the patient breathes (minute ventilation), but the nasal cannula can provide up to 44% oxygen as inspired air mixes with room air. Increasing the oxygen flow by 1 L/min (starting with 1 L/min and limited to about 6 L/min) will increase the inspired oxygen concentration by approximately 4%.

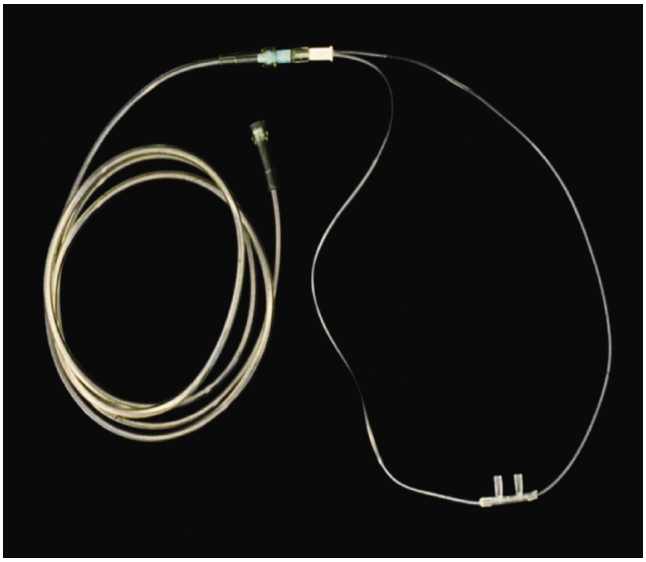
Recent years have seen the advent of high-flow nasal cannula systems, which allow for flow rates up to (and sometimes exceeding) 60 L/min. Inspired oxygen concentration can be set up to 100%.

Note that the use of the nasal cannula requires that the patient have adequate spontaneous respiratory effort, airway protective mechanism, and tidal volume.

Indications

- Patients with arterial oxyhemoglobin saturation 94% or less (less than 90% for ACS patients or 92% to 98% for post–cardiac arrest patients)
- Patients with minimal respiratory or oxygenation problems
- Patients who cannot tolerate a face mask

Figure 1. A nasal cannula used for supplemental oxygen delivery in spontaneously breathing patients.



Simple Oxygen Face Mask

The simple oxygen face mask delivers low-flow oxygen to the patient's nose and mouth. It can supply up to 60% oxygen with flow rates of 6 to 10 L/min, but the final oxygen concentration is highly dependent on the fit of the mask (Table 1). Oxygen flow rate of at least 6 L/min is needed to prevent rebreathing of exhaled carbon dioxide (CO_2) and to maintain increased inspired oxygen concentration.

Venturi Mask

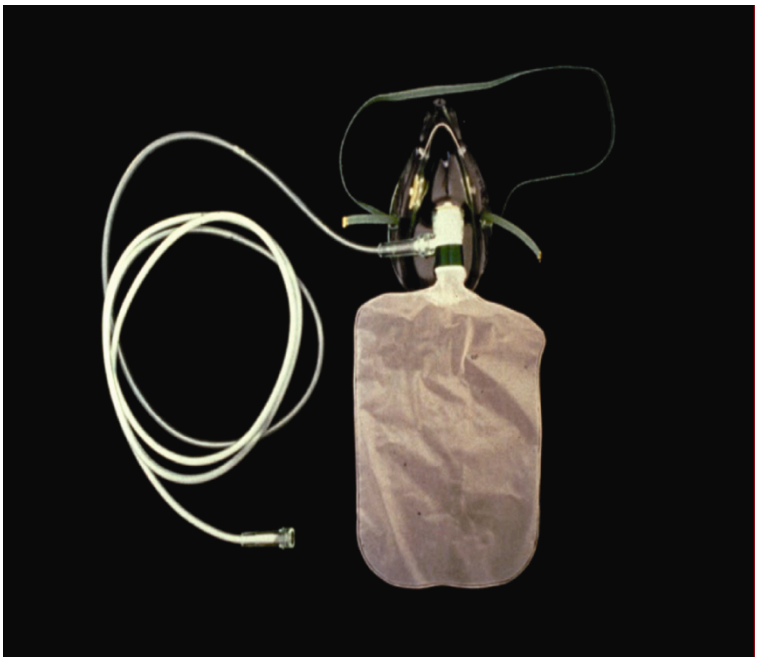
A Venturi mask enables a more reliable and controlled delivery of oxygen concentrations from 24% to 50% (Table 1). Delivered oxygen concentrations can be adjusted to 24%, 28%, 35%, and 40% by using a flow rate of 4 to 8 L/min and 40% to 50% by using a flow rate of 10 to 12 L/min. Observe the patient closely for respiratory depression. Use a pulse oximeter to titrate quickly to the preferred level of oxygen administration as long as peripheral perfusion is adequate and no shunting has occurred.

- A Venturi mask can accurately control the inspired oxygen concentration. Patients with chronic obstructive pulmonary disease (COPD), who usually have chronic hypercarbia (high CO_2) and mild to moderate hypoxemia, may benefit from this device.
- Administration of high oxygen concentrations to patients with COPD may produce respiratory depression because the increase in PaO_2 eliminates the stimulant effect of hypoxemia on the respiratory centers.
- Never withhold oxygen from patients who have respiratory distress and severe hypoxemia simply because you suspect a hypoxic ventilatory drive. If oxygen administration depresses respiration, support ventilation.

Face Mask With Oxygen Reservoir

The face mask below (Figure 2) is a partial rebreathing mask that consists of a face mask with an attached oxygen reservoir bag. A nonrebreathing face mask with an oxygen reservoir provides up to 95% to 100% oxygen with flow rates of 10 to 15 L/min (Table 1). In this system, a constant flow of oxygen enters an attached reservoir.

Figure 2. A face mask with oxygen reservoir used for supplemental oxygen delivery in spontaneously breathing patients.



Use of a face mask with a reservoir is indicated for patients who

- Are seriously ill, responsive, spontaneously breathing, have adequate tidal volume, and require high oxygen concentrations
- May avoid endotracheal (ET) intubation if acute interventions produce a rapid clinical effect (eg, patients with acute pulmonary edema, COPD, or severe asthma)
- Have relative indications for advanced airway management but maintain intact airway protective reflexes, such as gag and cough
- Are being prepared for advanced airway management

Caution

The above patients may have a diminished level of consciousness and be at risk for nausea and vomiting. A tight-fitting mask always requires close monitoring. Suctioning devices should be immediately available.

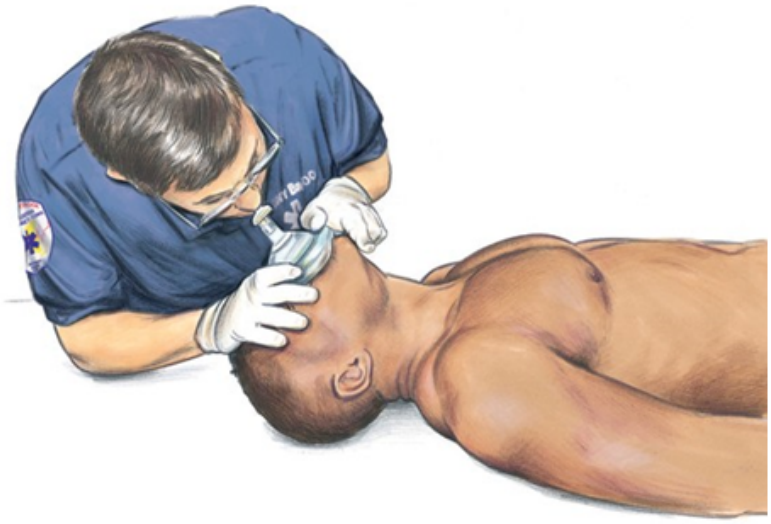
Giving Adult Mouth-to-Mask Breaths

To use a pocket mask, a healthcare provider who is alone should be positioned at the patient's side. This position is ideal when performing 1-rescuer cardiopulmonary resuscitation (CPR) because you can give breaths and perform chest compressions effectively without repositioning yourself every time you change from giving compressions to giving breaths. Follow these steps to open the airway with a head tilt–chin lift maneuver (use the jaw thrust method in patients with suspected neck or spinal cord injury) to give breaths to the patient:

1. Position yourself at the patient's side.
2. Place the pocket mask on the patient's face, using the bridge of the nose as a guide for correct position.

3. Seal the pocket mask against the face.
4. Using your hand that is closer to the top of the patient's head, place the index finger and thumb along the edge of the mask.
5. Place the thumb of your other hand along the edge of the mask.
6. Place the remaining fingers of your second hand along the bony margin of the jaw and lift the jaw. Perform a head tilt–chin lift to open the airway (Figure 3).
7. While you lift the jaw, press firmly and completely around the outside edge of the mask to seal the pocket mask against the patient's face.
8. Deliver each breath over 1 second, enough to make the patient's chest rise.

Figure 3. Mouth-to-mask ventilation, 1 rescuer. The single rescuer performs CPR from a position at the patient's side. Perform a head tilt–chin lift to open the airway while holding the mask tightly against the face.



Bag-Mask Ventilation

The bag-mask device typically consists of a self-inflating bag and a nonrebreathing valve; it may be used with a face mask or an advanced airway. Masks are made of transparent material to allow detection of regurgitation. They should be capable of creating a tight seal on the face, covering both mouth and nose. Bag-mask devices are available in adult and pediatric sizes. These devices are used to deliver high concentrations of oxygen by positive pressure to a patient who is not breathing effectively. Some devices allow the addition of a positive end-expiratory pressure valve.

Bag-mask ventilation is a challenging skill that requires considerable practice for competency. It is easier to provide by 2 trained and experienced rescuers. One rescuer opens the airway and seals the mask to the face while the other squeezes the bag, with both rescuers watching for visible chest rise. Healthcare providers can provide bag-mask ventilation with room air or oxygen if they use a self-inflating bag. This device provides positive-pressure ventilation when used without an advanced airway and, therefore, may produce gastric inflation and its consequent complications.

Tips for Performing Bag-Mask Ventilation

- Insert an oropharyngeal airway as soon as possible if the patient has no cough or gag reflex to help maintain an open airway.

- Use an adult (1- to 2-L) bag to deliver approximately 500- to 600-mL (6- to 7-mL/kg) tidal volume for adult patients. This amount is usually sufficient to produce visible chest rise and maintain oxygenation and normal carbon dioxide levels in apneic patients.
- To create a leak-proof mask seal, position yourself at the patient's head and perform a head tilt. Then, circle the thumb and index finger around the top of the mask (forming a "C"), pressing the edges of the mask to the face. Next, use the third, fourth, and fifth fingers (forming an "E") to lift the angle of the jaw and open the airway (Figure 4A). To maintain an effective mask seal, the hand holding the mask must perform multiple tasks simultaneously: maintaining the head-tilt position, pressing the mask against the face, and lifting the jaw.
- Two well-trained, experienced healthcare providers are preferred during bag-mask ventilation. The rescuer at the patient's head tilts the patient's head and seals the mask against the patient's face with the thumb and first finger of each hand (creating a "C") to provide a complete seal around the edges of the mask. The rescuer uses the remaining 3 fingers (the "E") to lift the jaw (this holds the airway open). The second rescuer slowly squeezes the bag over 1 second until the chest rises. Both rescuers should observe chest rise (Figure 4B).

The seal and volume problems do not occur when the bag-mask device is attached to the end of an advanced airway device (eg, laryngeal mask airway, laryngeal tube, esophageal-tracheal tube, or ET tube).

Figure 4. A, E-C clamp technique of holding the mask while lifting the jaw. B, Two-rescuer use of the bag-mask device.

A

B

B

Ventilation With an Advanced Airway and Chest Compressions

- When the patient has an advanced airway in place during CPR, provide continuous compressions and asynchronous ventilations once every 6 seconds.
- Avoid excessive ventilation (too many breaths or too large a volume).

Advanced Airway Management

Advanced Airway Adjuncts: Laryngeal Mask Airway

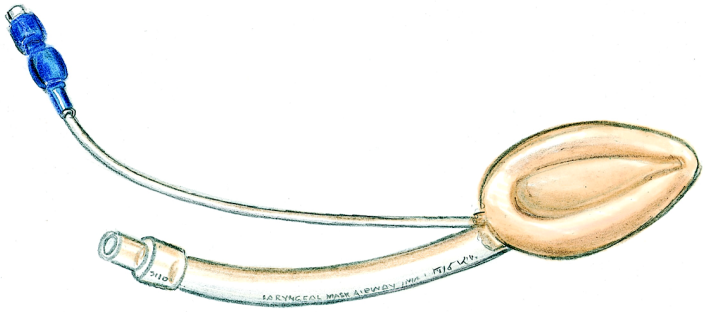
The laryngeal mask airway is composed of a tube with a cuffed, mask-like projection at the end of the tube (Figure 5). The laryngeal mask airway is an advanced airway device that is considered an acceptable alternative to the ET tube. When compared with the ET tube, the laryngeal mask airway provided equivalent ventilation during CPR in 72% to 97% of patients. A small proportion of patients cannot be ventilated with the laryngeal mask airway. Therefore, it is important for providers to have an alternative strategy for airway management.

The advantages of laryngeal mask airway are as follows:

- Regurgitation is less likely with the laryngeal mask airway than with the bag-mask device.

- Because insertion of the laryngeal mask airway does not require laryngoscopy and visualization of the vocal cords, training in its placement and use is simpler than for ET intubation.
- Laryngeal mask airway insertion is easier than ET tube insertion when access to the patient is limited, there is a possibility of unstable neck injury, or appropriate positioning of the patient for ET intubation is impossible.

Figure 5. Laryngeal mask airway.



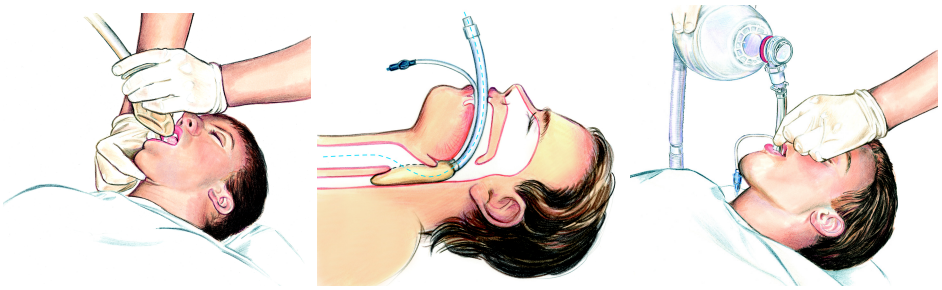
Insertion of the Laryngeal Mask Airway

The steps for insertion of the laryngeal mask airway (Figure 6) are as follows:

Step	Action
1	*Patient preparation:* Provide oxygenation and ventilation, and position the patient.
2	*Equipment preparation:* Check the integrity of the mask and tube according to the manufacturer's instructions. Lubricate only the posterior surface of the cuff to avoid blocking the airway aperture.
3	*Insertion technique:* • Introduce the laryngeal mask airway into the pharynx and advance it blindly until you feel resistance. Resistance indicates that the distal end of the tube has reached the hypopharynx. • Inflate the cuff of the mask. Cuff inflation pushes the mask up against the tracheal opening, allowing air to flow through the tube and into the trachea. • Ventilation through the tube is ultimately delivered to the opening in the center of the mask and into the trachea. • To avoid trauma, do not use force at any time during insertion of the laryngeal mask airway. • Avoid overinflating the cuff. Excessive intracuff pressure can result in misplacement of the device. It also can cause pharyngolaryngeal injury (eg, sore throat, dysphagia, or nerve injury).
4	. Consider inserting a bite-block (if the laryngeal mask airway does not have intrinsic bite-block), provide ventilation, and continue to monitor the patient's condition and the position of the laryngeal mask airway. A bite-block reduces the possibility of airway obstruction and tube damage from biting.

Step	Action
5	Secure the laryngeal mask airway tube in place (eg, with tape or other appropriate securing device).

Figure 6. Insertion of the laryngeal mask airway.



Cautions/Additional Information

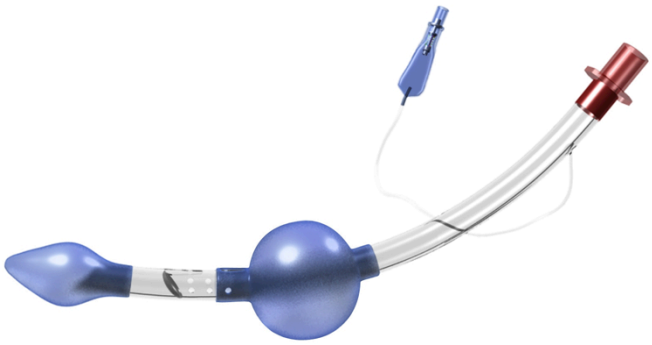
- Do not apply cricoid pressure because it may hinder the insertion of the laryngeal mask airway. Studies in anesthetized adults have shown that when cricoid pressure is used before insertion of a laryngeal mask airway, the proportion of tubes correctly positioned is reduced, and the incidence of failed insertion and impaired ventilation once the laryngeal mask airway had been placed is increased.
- In general, size 5 fits men and size 4 fits women.
- You may note a smooth swelling at the level of the cricoid cartilage in the neck. This is normal, and it confirms the proper positioning of the device.
- If you hear an air leak during ventilation with a bag for the next 3 or 4 breaths, reevaluate the position of the laryngeal mask airway for possible misplacement.
- To avoid displacement, limit the patient's head movement and avoid suctioning secretions in the pharynx once the laryngeal mask airway is in place.

Advanced Airway Adjuncts: Laryngeal Tube

The laryngeal tube (Figure 7) is a supraglottic airway device that is considered an acceptable alternative to an ET tube. The laryngeal tube is available in single- and dual-lumen versions. Only experienced providers should perform laryngeal tube insertion.

The advantages of the laryngeal tube are ease of training and ease of insertion due to its compact size. In addition, it isolates the airway, reduces the risk of aspiration compared with bag-mask ventilation, and provides reliable ventilation. Trained healthcare professionals may consider the laryngeal tube as an alternative to bag-mask ventilation or ET intubation for airway management in cardiac arrest.

Figure 7. Laryngeal tube.

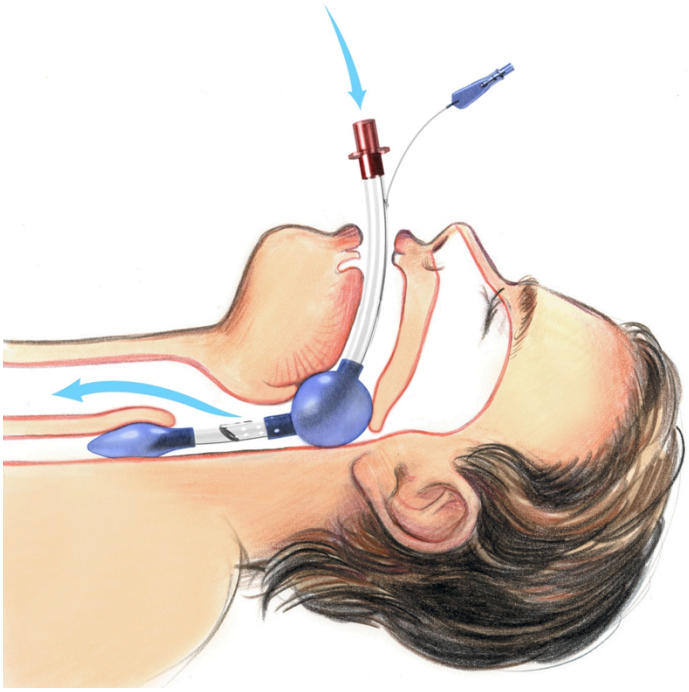


Insertion of the Laryngeal Tube

The steps for insertion of a laryngeal tube are as follows:

Step	Action
1	*Patient preparation:* Provide oxygenation and ventilation, and position the patient.
2	*Equipment preparation:* Check the integrity of the laryngeal tube according to the manufacturer's instructions.
3	*Insertion technique*: • Inspect the mouth and larynx of the patient before insertion of the laryngeal tube. • Open the mouth of the patient approximately 2 to 3 cm by using the thumb-and-index-finger technique. • Insert the laryngeal tube in the midline of the mouth along the palate until a slight resistance is felt (Figure 8). • In some cases, a slight head extension can facilitate mouth opening and tube placement. • Ensure that the ventilation holes of the laryngeal tube lie in front of the laryngeal inlet. • The insertion depth can be verified according to the teeth marks at the upper end of the tube. • The laryngeal tube is available in different sizes.
4	Consider inserting a bite-block (if the laryngeal tube does not have intrinsic bite-block), provide ventilation, and continue to monitor the patient's condition and the position of the laryngeal tube. A bite-block reduces the possibility of airway obstruction and tube damage from biting.
5	Secure the laryngeal tube in place (eg, with tape or other appropriate securing device).

Figure 8. Positioning of the laryngeal tube.



Advanced Airway Adjuncts: Esophageal-Tracheal Tube

The esophageal-tracheal tube (Figures 9 and 10) is an advanced airway that is an acceptable alternative to the ET tube. The esophageal-tracheal tube is an invasive airway device with 2 inflatable balloon cuffs. The tube is more likely to enter the esophagus than the trachea, thereby allowing ventilation to occur through side openings in the device adjacent to the vocal cords and trachea. If the tube enters the trachea, ventilation can still occur by an opening in the end of the tube.

Studies show that healthcare providers with all levels of experience can insert the esophageal-tracheal tube and deliver ventilation comparable to that achieved with ET intubation. Compared with bag-mask ventilation, the esophageal-tracheal tube is advantageous because it isolates the airway, reduces the risk of aspiration, and provides more reliable ventilation. The advantages of the esophageal-tracheal tube are chiefly related to ease of training when compared with the training needed for ET intubation. Only providers trained and experienced with the use of the esophageal-tracheal tube should insert the device because fatal complications are possible if the position of the distal lumen of the esophageal-tracheal tube in the esophagus or trachea is identified incorrectly. Other possible complications related to the use of the esophageal-tracheal tube are esophageal trauma, including lacerations, bruising, and subcutaneous emphysema. The esophageal-tracheal tube is supplied in 2 sizes: the smaller size (37F) is used in patients 4 to 5.5 feet tall, and the larger size (41F) is used in patients more than 5 feet tall.

Contraindications

- Responsive patients with cough or gag reflex
- Age 16 years or younger
- Height 4 feet or shorter

- Known or suspected esophageal disease
- Ingestion of a caustic substance

Figure 9. Esophageal-tracheal tube.

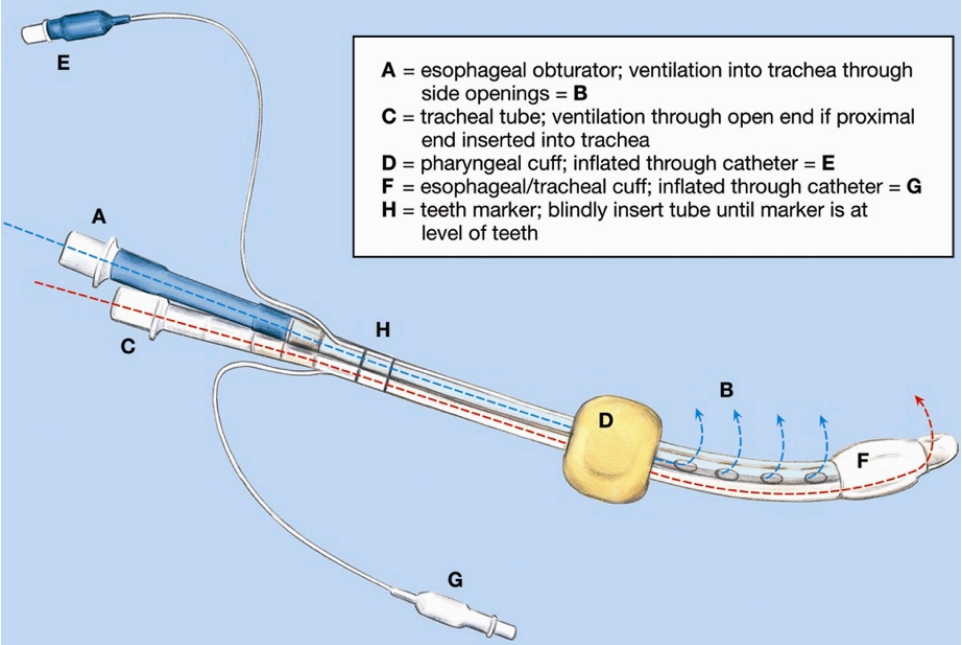
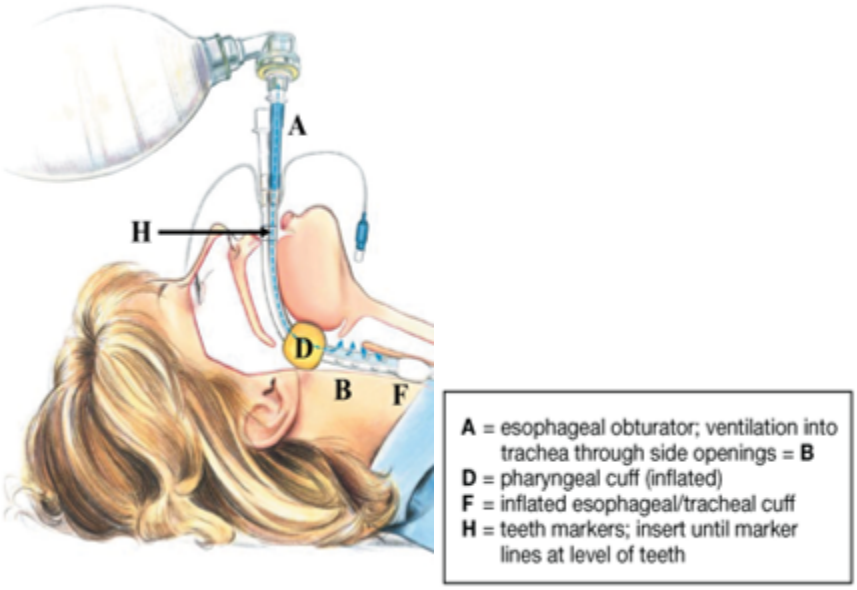


Figure 10. Esophageal-tracheal tube inserted in esophagus.



At the H point on Figures 9 and 10, rescuers' hands should be holding/anchoring the tube in place. With the bag-mask squeeze, potential movement of the invasive airways may occur.

Insertion of the Esophageal-Tracheal Tube

The steps for blind insertion of a esophageal-tracheal tube are as follows:

Step	Action
1	*Patient preparation:* Provide oxygenation and ventilation, and position the patient. Rule out the contraindications to insertion of the esophageal-tracheal tube.
2	*Equipment preparation:* Check the integrity of both cuffs according to the manufacturer's instructions, and lubricate the tube.
3	*Insertion technique:* • Hold the device with cuffs deflated so that the curvature of the tube matches the curvature of the pharynx. • Lift the jaw and insert the tube gently until the black lines (H) on the tube (Figures 9 and 10) are positioned between the patient's upper teeth. Do not force insertion and do not attempt for more than 30 seconds. • Inflate the proximal/pharyngeal (blue) cuff with approximately 100 mL of air; inflate with approximately 85 mL for the smaller esophageal-tracheal tube. Then, inflate the distal (white or clear) cuff with approximately 15 mL of air; inflate with approximately 12 mL for the smaller esophageal-tracheal tube. Manufacturers may differ in cuff volumes, so please read manufacturer's instructions.
4	Confirm tube location and select the lumen for ventilation. To select the appropriate lumen for ventilation, you must determine where the tip of the tube is located. The tip of the tube can rest in either the esophagus or the trachea. • *Esophageal placement:* To confirm esophageal placement, attach the bag-mask to the blue (proximal/pharyngeal) lumen. Squeezing the bag provides ventilation by forcing air through the openings in the tube between the 2 inflated cuffs. This action produces bilateral breath sounds. Epigastric sounds do not occur because the distal cuff, once inflated, obstructs the esophagus, thereby preventing airflow into the stomach. Because the tip of the tube rests in the esophagus, do not use the distal (white or clear) tube for ventilation. • *Tracheal placement:* If squeezing the bag attached to the blue (proximal/pharyngeal) lumen does not produce breath sounds, immediately disconnect the bag and reattach it to the distal (white or clear) lumen. Squeezing the bag should now produce breath sounds because this lumen goes to the trachea. With ET placement of the tube, the distal cuff performs the same function as a cuff on an ET tube. Detection of exhaled CO_2 (through the ventilating lumen) should be used for confirmation, particularly if the patient has a perfusing rhythm. • *Unknown placement:* If you are unable to hear breath sounds, deflate both cuffs and withdraw the tube slightly. Reinflate both cuffs (see previous steps) and attempt to ventilate the patient. If breath sounds and epigastric sounds are still absent, remove the tube. Make sure you have suction equipment available in case tube removal causes vomiting.
5	Secure the esophageal-tracheal tube in place (eg, with tape or other appropriate securing device).

Cautions/Additional Information

Do not apply cricoid pressure during insertion because it may hinder the insertion of the esophageal-tracheal tube.

Disadvantages

- Insertion of an esophageal-tracheal tube may cause esophageal trauma, including lacerations, bruising, and subcutaneous emphysema.
- Esophageal-tracheal tubes are available in only 2 sizes and cannot be used in any patient less than 4 feet tall.

Advanced Airway Adjuncts: ET Intubation

An ET tube is a single-use, cuffed tube that facilitates delivery of a high concentration of oxygen and selected tidal volume to maintain adequate ventilation; placement requires visualization of the patient's vocal cords.

The advantages of ET tube insertion are

- Maintains patent airway
- May protect the airway from aspiration of stomach contents or other substances in the mouth, throat, or upper airway
- Permits effective suctioning of the trachea
- Facilitates delivery of positive end-expiratory pressure
- Provides an alternative route for administering some resuscitation medications when intravenous (IV) or intraosseous (IO) access cannot be obtained
- Is ideal if longer-term ventilation is needed
- Provides the greatest compliance in situations that require higher airway pressure

There are several emergency medications that can be administered by ET tube, including naloxone, atropine, epinephrine, and lidocaine.

- The dose is approximately 2 to 2.5 times higher for ET tube administration than for IV/IO administration.
- Mix the dose of the drug with 5 to 10 mL of normal saline or sterile water. Studies with epinephrine and lidocaine showed that dilution with sterile water instead of 0.9% saline may achieve better drug absorption.
- After the medication has been administered through the ET tube, perform 1 to 2 ventilations to facilitate deposition of the drug into the airways.
- Hold chest compressions very briefly during the instillation of the drug into the ET tube because compressions will cause contents to come out of the ET tube if the bag-mask device is not reconnected.

There were no efficacy and safety data regarding ET administration of amiodarone at the time of writing the *2020 American Heart Association* (AHA) *Guidelines for CPR and Emergency Cardiac Care* (ECC).

Note that for drugs that can be administered by the ET route, optimal ET doses have not been established. IV/IO administration is preferred because it provides a more reliable drug delivery and pharmacologic effect.

ET tube insertion was once considered the optimal method of managing the airway during cardiac arrest. However, intubation attempts by unskilled providers can produce complications. Therefore, esophageal-tracheal tubes, laryngeal mask airways, and laryngeal tubes are now considered acceptable alternatives to the ET tube for advanced airway management.

Misplacement of an ET tube can result in severe, even fatal, complications. For this reason, only skilled, experienced personnel should perform ET intubation. In most states, medical practice acts specify the level of personnel allowed to perform this procedure. For clinical reasons, intubation should be restricted to healthcare providers who meet all of the following criteria:

- Personnel are well trained.
- Personnel perform intubations frequently.
- Personnel receive frequent refresher training in this skill.
- ET tube placement is included in the scope of practice defined by governmental regulations.
- Personnel participate in a process of continuous quality improvement to detect frequency of complications and minimize those complications.

Placement of an ET tube is an important part of a resuscitation attempt. But it is a much lower priority than providing high-quality continuous chest compressions with few interruptions and delivering defibrillation.

Technique of ET Intubation

Many ACLS providers do not perform intubation because of the professional restrictions noted above. Nonetheless, all members of the resuscitation team must understand the concept of ET intubation. Team members may assist with ET intubation and must know how to integrate compressions and ventilations when an ET tube is placed. This knowledge is often more important than knowing how to perform the procedure itself.

Step	Action
1	*Patient preparation:* Provide oxygenation and ventilation, and position the patient. Assess the likelihood of difficult ET tube placement based on the patient's anatomy. Remove any barriers to successful intubation, such as dentures or secretions.
2	*Equipment preparation:* Assemble and check all necessary equipment (ET tube and laryngoscope).
3	Insertion technique: • Choose an appropriate size of ET tube. In general, a 7.5- to 8.00-mm internal diameter tube is used for adult males, and a 7.0- to 7.5-mm internal diameter tube is used for adult females. • Choose the appropriate type (straight or curved) and size of laryngoscope blade (Figures 11A and B). • Test the ET tube cuff's integrity. • Secure the stylet inside the ET tube. • Lubricate the ET tube. • Place the head in the "sniffing" position.

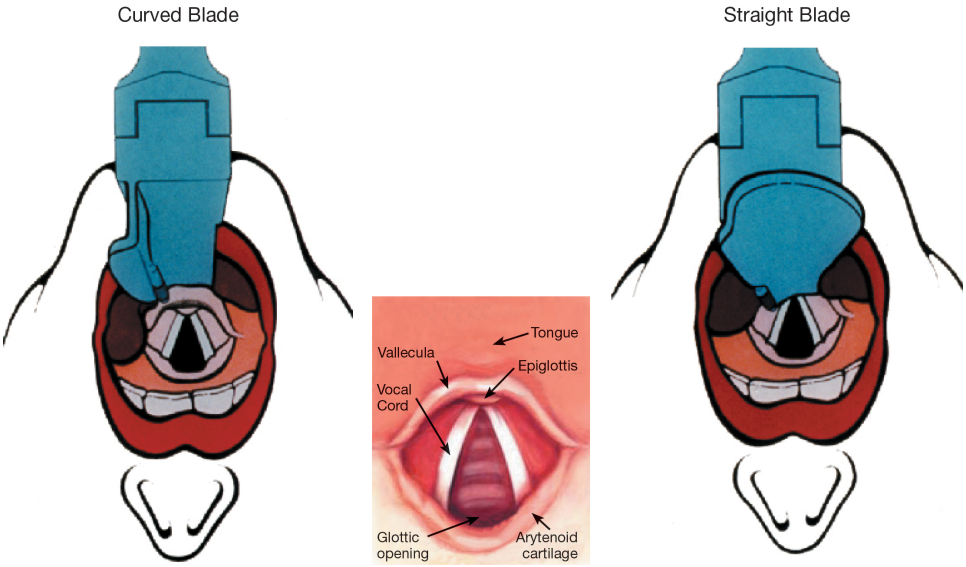
Step	Action
	• Open the mouth of the patient by using the thumb-and-index-finger technique. • Insert the laryngoscope blade and visualize the glottic opening (Figure 12). • Clear the airway if needed. • Insert the ET tube and watch it pass through the vocal cords. • Inflate the ET tube cuff with approximately 10 to 12 mL to achieve a proper seal.* • Remove the laryngoscope blade from the mouth. • Hold the tube with one hand and remove the stylet with the other hand. • Insert a bite-block. • Attach a bag to the tube. • Squeeze the bag to give breaths (1 second each) while watching for chest rise. • Assess proper placement by both a clinical assessment and device confirmation: – Auscultate for breath sounds. – Confirm correct positioning of the ET tube by continuous quantitative waveform capnography or, if not available, qualitative partial-pressure end-tidal CO_2 ($PETCO_2$) or esophageal detector device (EDD). • Secure the ET tube in place (eg, with tape or a tube holder). • Provide ventilation, and continue to monitor the patient's condition and the position of the ET tube by using continuous waveform capnography. *Manufacturers may differ in cuff volumes; please read manufacturer's instructions.

Figure 11. A, Curved laryngoscope blades. B, Straight laryngoscope blades.

A

B

Figure 12. View of the vocal cords.



Indications for ET Intubation

- Cardiac arrest when bag-mask ventilation is not possible or is ineffective, or if a protected airway is needed
- Responsive patient in respiratory distress or compromise
- Patient unable to protect airway (eg, coma, areflexia, or cardiac arrest)

Cautions/Additional Information

- The incidence of complications is unacceptably high when intubation is performed by inexperienced providers or monitoring of the tube placement is inadequate.
- Detailed assessment of out-of-hospital intubation attempts has concluded that ET tubes are much more difficult to place properly in that setting and highly susceptible to dislodgment.
- Remove any barriers to successful intubation, such as dentures or secretions, and position the patient.

- Healthcare providers can minimize interruptions in chest compressions for ET intubation with advanced preparation. Insert the laryngoscope blade with the tube ready at hand as soon as compressions are paused. Interrupt compressions only to visualize the vocal cords and insert the tube; this is ideally less than 10 seconds. Resume chest compressions immediately after passing the tube between the vocal cords. Verify the tube's placement. If the initial intubation attempt is unsuccessful, healthcare providers may make a second attempt, but they should consider using a supraglottic airway.

Ventilating With an ET Tube in Place During Chest Compressions

During cardiac arrest, provide the following:

- Volume: The volume should cause visible chest rise.
  - When practicing this skill, try to get a sense of what such a volume feels like when squeezing the ventilation bag.
  - Provide slightly more volume for very obese patients.
- Rate: Provide approximately 1 breath every 6 seconds when delivering ventilation during CPR or respiratory arrest.

Compression-ventilation cycles: Once an advanced airway is in place, or if the local protocol is continuous compressions with asynchronous bag-mask ventilation, the healthcare provider should provide continuous compressions and asynchronous ventilations once every 6 seconds. Providers should switch compressors about every 2 minutes.

With any advanced airway, ventilate once every 6 seconds during either cardiac arrest or respiratory arrest.

Take care to avoid air trapping in patients with conditions associated with increased resistance to exhalation, such as severe obstructive lung disease and asthma. Air trapping could result in a positive end-expiratory pressure effect that may significantly lower blood pressure. In these patients, use slower ventilation rates to allow more complete exhalation. In cases of hypovolemia, restore intravascular volume.

Tube Trauma and Adverse Effects

ET intubation can cause significant trauma to a patient, including

- Brain damage or death
  - If the ET tube is inserted into a patient's esophagus, the patient will receive no ventilation or oxygenation unless they are still breathing spontaneously. If you or your team fails to recognize esophageal intubation, the patient could suffer permanent brain damage or die.
- Lacerated lips or tongue from forceful pressure between the laryngoscope blade and the tongue or cheek
- Chipped teeth
- Lacerated pharynx or trachea from the end of the stylet or ET tube
- Injury to the vocal cords
- Pharyngeal-esophageal perforation
- Vomiting and aspiration of gastric contents into the lower airway

Insertion of ET Tube Into One Bronchus

Insertion of the ET tube into the right (most common) or left main bronchus is a frequent complication. Unrecognized and uncorrected intubation of a bronchus can result in hypoxemia due to underinflation of the uninvolved lung or overinflation of the ventilated lung.

To determine if the ET tube has been inserted into a bronchus, listen to the chest for bilateral breath sounds. Also, look for equal expansion of both sides during ventilation. Waveform capnography is not sensitive enough to detect main bronchus intubation.

If you suspect that the tube has been inserted into either the left or right main bronchus, take these actions:

- Deflate the tube cuff.
- Withdraw the tube back 1 to 2 cm.
- Confirm correct tube placement by both clinical assessment and device confirmation.
- Reinflate the cuff and secure the ET tube in place.
- Recheck the patient's clinical signs, including chest expansion, breath sounds, and evidence of oxygenation.

Even when the ET tube is seen to pass through the vocal cords and the tube position is verified by chest expansion and auscultation during positive-pressure ventilation, you should obtain additional confirmation of placement by using continuous quantitative waveform capnography or a qualitative device like a colorimetric P_{ETCO_2} detector or EDD.

Once the patient is more stable, an x-ray may be obtained to optimize the ET tube position and assess lung pathology. An x-ray takes too long to be used as a means of confirming tracheal placement of an ET tube. Recognizing misplacement of an ET tube is a clinical responsibility.

After inserting and confirming the correct placement of an ET tube, you should record the depth of the tube as marked at the front teeth or gums and secure it. Because there is significant potential for ET tube movement with head flexion and extension and when the patient is moved from one location to another, secure the ET tube with tape or a commercial device. Devices and tape should be applied in a manner that avoids compression of the front and sides of the neck to protect against impairment of venous return from the brain.

Confirmation of ET Tube Placement: Physical Exam

Confirm tube placement immediately, assessing the first breath delivered by the bag-mask device. You should use both clinical assessment and confirmation devices to verify tube placement immediately after insertion and again when the patient is moved. However, because no single confirmation technique is completely reliable, particularly when cardiac arrest is present, the AHA recommends the use of continuous waveform capnography, in addition to clinical assessment, as the most reliable method of confirming and monitoring the correct placement of an ET tube. If waveform capnography is not available, EDD or nonwaveform P_{ETCO_2} monitor, in addition to clinical assessment, should be used to confirm ET tube placement.

Assessment by physical examination consists of visualizing chest expansion bilaterally and listening over the epigastrium (breath sounds should not be heard) and the lung fields bilaterally (breath sounds should be equal and adequate).

As the bag is squeezed, listen over the epigastrium and observe the chest wall for movement. If you hear stomach gurgling and see no chest-wall expansion, you have intubated the esophagus. Stop ventilations. Remove the ET tube at once. Then do the following:

- Continue chest compressions if CPR is in progress.
- Resume bag-mask ventilation or consider an alternate advanced airway.
- Reattempt intubation only after reoxygenating the patient (approximately 30 seconds of bag-mask ventilation with 100% oxygen).
- If, after intubation, the chest wall rises appropriately and stomach gurgling is not heard, listen to the lung fields with 5-point auscultation: over the stomach, left and right anterior lung fields, and left and right midaxillary lung fields. Document the location of breath sounds in the patient's medical record. If you have any doubt, stop ventilations through the tube, and use the laryngoscope to see if the tube is passing through the vocal cords. If the tube is too high, the cuff may not be properly sealed, leading to difficulty ventilating.
- If still in doubt, remove the tube and provide bag-mask ventilation until the tube can be replaced. If the ET tube is too small, the inflated cuff will not seal the patient. If the ET tube is too high, the cuff often won't seal as it is herniating through the vocal cords.
- If the ET tube is in the trachea and will not seal after inflating the cuff, the ET tube could be too small. Recheck the appropriate size for the patient.
- If the tube seems to be in place, reconfirm the tube mark at the front teeth (previously noted after inserting the tube 1– to 2 cm past the vocal cords).
- Secure the tube (ideally with a commercial device designed for this purpose), avoiding compression of the front and sides of the neck.
- Once the tube is secured, insert a bite-block if the commercial device used to secure the tube does not prevent the patient from biting down and occluding the airway.

Confirmation of ET Tube Placement: Qualitative and Quantitative Devices

The *2020 AHA Guidelines for CPR and ECC* recommend confirmation of the ET tube with both clinical assessment and a device. If the device is attached to the bag before it is joined to the tube, it will increase efficiency and decrease the time in which chest compressions must be interrupted.

Detailed assessment of out-of-hospital intubation attempts has concluded that ET tubes are much more difficult to place properly in that setting and highly susceptible to misplacement and displacement. Proper training, supervision, frequent clinical experience, and a process of quality improvement are the keys to achieving successful intubation.

Waveform Capnography

Continuous waveform capnography, in addition to physical assessment, is recommended as the most reliable method of confirming and monitoring correct placement of an ET tube. Providers should observe a persistent

capnographic waveform with ventilation to confirm and monitor ET tube placement in the field, in the transport vehicle, on arrival at the hospital, and after any patient transfer to reduce the risk of unrecognized tube misplacement or displacement. Studies of waveform capnography to verify ET tube position in patients in cardiac arrest have shown high sensitivity and specificity in identifying correct ET tube placement (Figures 13 and 14A-C).

The use of capnography to confirm and monitor correct placement of supraglottic airways has not been studied. However, effective ventilation through a supraglottic airway device should result in a capnographic waveform during CPR and after return of spontaneous circulation.

Figure 13. Waveform capnography with ET tube.

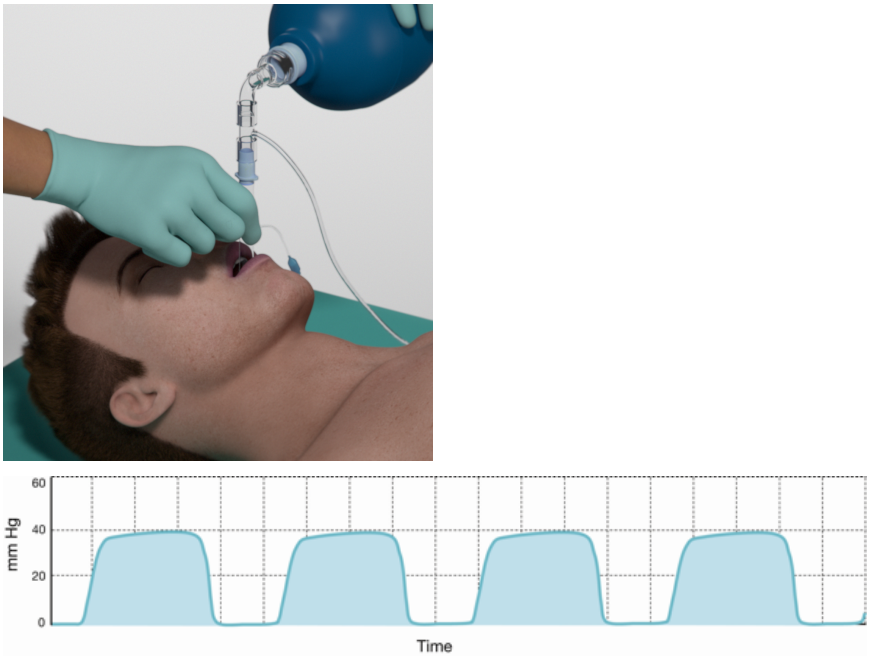
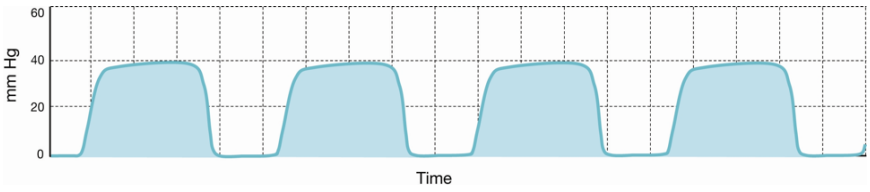
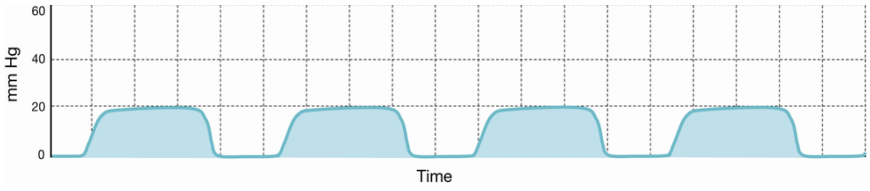


Figure 14. Waveform capnography. A, Normal range (approximately 35 to 45 mm Hg). B, Expected waveform with adequate chest compressions in cardiac arrest (approximately 20 mm Hg). C, ET tube incorrectly placed or dislodged (0 mm Hg).

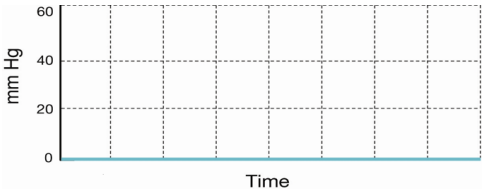
A



B



C



Quantitative End-Tidal CO_2 Monitors (Capnometry)

The quantitative end-tidal CO_2 monitor or capnometer provides a single quantitative readout of the concentration of CO_2 at a single point in time. The device provides a continuous display of the level of CO_2 as it varies throughout the ventilation cycle.

These monitors can help confirm successful ET tube placement within seconds of an intubation attempt. They also can detect a patient's deterioration associated with declining clinical status or ET tube displacement. ET tube displacement is an adverse event that is alarmingly common during out-of-hospital transport of a patient.

Exhaled (Qualitative) CO_2 Detectors

A number of commercial devices can react, usually with a color change (different colors for different CO_2 detectors), to CO_2 exhaled from the lungs. This simple method, when used by an experienced operator, can be a reasonable alternative for detecting correct tube placement if continuous waveform capnography is not available. There is, however, no evidence that these devices are accurate for continued monitoring of ET tube placement.

Note that the carbon dioxide detection cannot ensure proper *depth* of tube insertion. The tube should be held in place and then secured once correct position is verified. Different manufacturers may use different color indicators.

Note also that these devices expire, and if they are not maintained in a sealed bag (or if the seal is broken), they will no longer work. Out of the bag, they will be yellow when they should be blue or purple.

Additionally, acidic drugs such as epinephrine will create a false positive. They will cause the device to turn yellow and not change with each breath.

You must ensure that with each breath, the color of the device changes.

Esophageal Detector Devices

EDDs use simple anatomical principles to determine the location of the distal end of the ET tube. Unlike the end-tidal CO_2 detector, the EDD does not depend on blood flow. Providers should completely compress the bulb-style EDD before attaching it to the ET tube. The EDD should be used before any breaths are given. After the provider releases the bulb, if the tube is resting in the esophagus, reinflation of the bulb produces a vacuum, which pulls the esophageal mucosa against the tip of tube. This results in slow or no reexpansion of the bulb.

With the syringe-style EDDs, the vacuum occurs when the rescuer pulls back on the syringe plunger. Esophageal placement results in the inability of the rescuer to pull back on the plunger.

If the tube rests in the trachea, the vacuum will allow smooth reexpansion of the bulb or aspiration of the syringe.

If continuous waveform capnography is not available, an EDD used by an experienced operator is also a reasonable alternative for confirming correct ET tube placement. However, while observational studies and a small randomized controlled trial of these devices report a low false-positive rate for confirming tracheal placement, there is no evidence that an EDD is accurate or practical for the continued monitoring of ET tube placement.

Note that the EDD may yield misleading results in patients with morbid obesity, late pregnancy, or status asthmaticus.

Table 2 lists correct responses plus the most common causes of misleading results from using end-tidal CO_2 detector devices and EDDs to confirm the correct placement of the ET tube. The table's columns (vertical) indicate the reading and actual location of the ET tube. The rows (across) indicate the expected results from using either a colorimetric end-tidal CO_2 detector (A) or bulb-type EDD (B). With both devices, assume that the rescuer made a conscientious intubation effort and thinks the ET tube is in the trachea.

Table 2. Comparison of Qualitative Performance: EDD and P_{ETCO_2} Devices

A: Colorimetric end-tidal CO_2 detector		
Reading	**Actual location of ET tube: trachea**	**Actual location of ET tube: esophagus (or hypopharynx)**
Carbon dioxide detected Color change: positive = CO_2 present (or as specified by manufacturer)	*ET tube in trachea* Proceed with ventilations.	*Reasons for apparent CO_2 detection despite tube in esophagus* Causes: Distended stomach, recent ingestion of carbonated beverage, nonpulmonary sources of CO_2 Consequences: Unrecognized esophageal intubation; can lead to iatrogenic death
No CO_2 detected No color change: negative = CO_2 absent (or as specified by manufacturer)	*No CO_2 detection with tube in trachea* Causes: Low or no blood flow state (eg, cardiac arrest); any cardiac arrest with no, prolonged, or poor CPR Consequences: Leads to unnecessary removal of properly placed ET tube; reintubation attempts increase chances of other adverse consequences	*No CO_2 detection and tube is not in trachea (ie, tube is in esophagus)* Causes: Rescuer has inserted ET tube in esophagus/hypopharynx. A life-threatening adverse event has been detected. Consequences: Rescuer recognizes ET tube is not in trachea; properly and rapidly identified; tube is removed at once; patient is reintubated.
B: EDD		
Reading	**Actual location of ET tube: esophagus**	**Actual location of ET tube: trachea**

Consistent with tube in esophagus Bulb does not refill or refills slowly (>10 seconds × 2), or syringe cannot be aspirated	*Device suggests tube in esophagus when it is in esophagus* Causes: Rescuer has inserted tube in esophagus/hypopharynx. A potentially life-threatening adverse event has been detected. Consequences: Rescuer correctly recognizes ET tube is in esophagus; ET tube is removed at once; patient is reintubated.	*Device suggests tube in esophagus when it is in trachea* Causes: Secretions in trachea (mucus, gastric contents, acute pulmonary edema); insertion in right main bronchus; pliable trachea (morbid obesity, late-term pregnancy) Consequences: This leads to unnecessary removal of properly placed ET tube. Reintubation attempts increase chances of other adverse consequences.
Consistent with tube in trachea Bulb fills immediately or syringe can be aspirated	*Results suggest that tube is* not *in esophagus (ie, that it is in trachea) when tube* is *in esophagus.* Causes: • Conditions that cause increased lung expansion (eg, COPD, status asthmaticus) • Conditions that fill stomach with air (eg, recent bag-mask ventilation, mouth-to-mask or mouth-to-mouth breathing) • Conditions that cause poor tone in esophageal sphincter or increased gastric pressure (late pregnancy) Consequences: Unrecognized esophageal intubation can lead to death.	*Results suggest that tube is* not *in the esophagus (ie, that it is in the trachea) when it* is *in the trachea.* EDD indicates ET tube is in trachea. Proceed with ventilations.

ACLS Core Rhythms

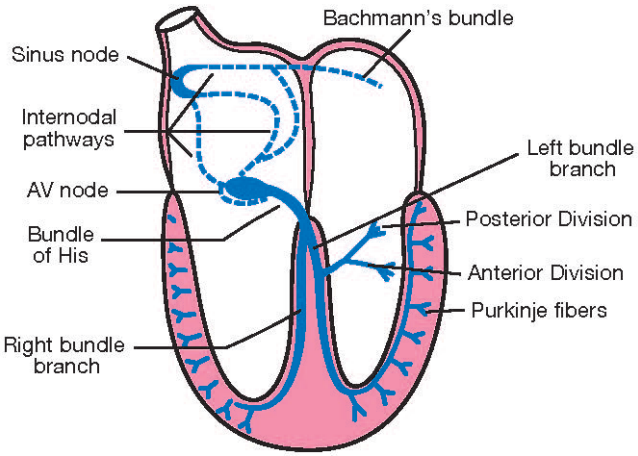
Recognition of Core Electrocardiogram Arrest Rhythms

The Basics

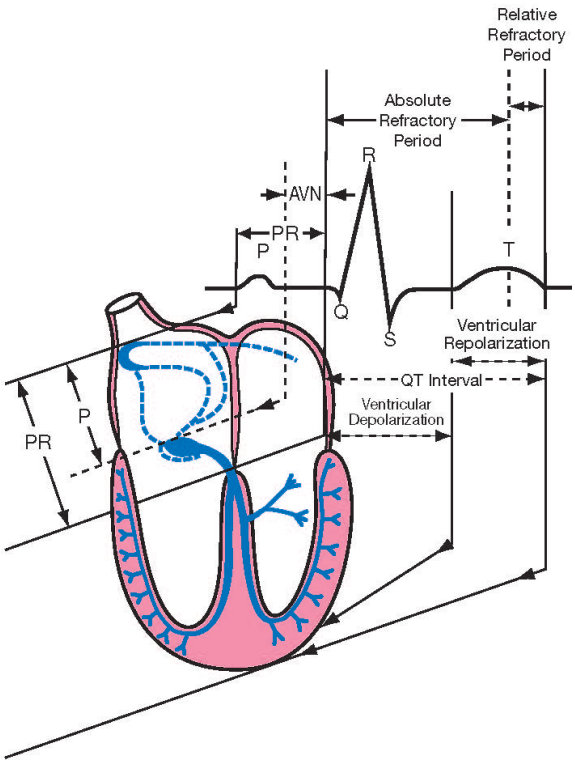
Figure 15 shows the anatomy of the cardiac conduction system and its relationship to the electrocardiogram (ECG) cardiac cycle.

Figure 15. Anatomy of the cardiac conduction system: relationship to the ECG cardiac cycle. A, Heart: anatomy of conduction system. B, Relation of cardiac cycle to conduction system anatomy.

A



B



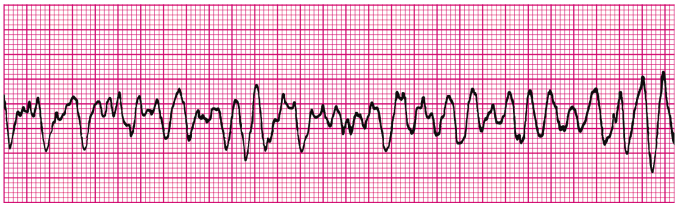
Cardiac Arrest Rhythms and Conditions

The ECG rhythms/conditions for patients who are in cardiac arrest are ventricular fibrillation (VF; Figure 16A and B), pulseless ventricular tachycardia (pVT), asystole (Figure 17), or pulseless electrical activity (PEA), which presents with a variety of rhythms.

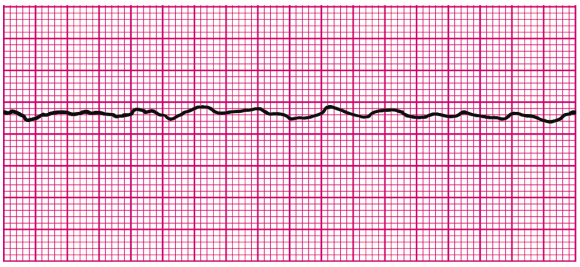
Ventricular fibrillation	
Pathophysiology	• Ventricles consist of areas of normal myocardium alternating with areas of ischemic, injured, or infarcted myocardium, leading to a chaotic asynchronous pattern of ventricular depolarization and repolarization. Without organized ventricular depolarization, the ventricles cannot contract as a unit, and they produce no cardiac output. The heart "quivers" and does not pump blood.
Defining criteria per ECG	• **Rate/QRS complex:** Unable to determine; no recognizable P, QRS, or T waves; baseline undulations occur between 150 and 500 per minute • **Rhythm:** Indeterminate; pattern of sharp up (peak) and down (trough) deflections • **Amplitude:** Measured from peak to trough; often used subjectively to describe VF as *fine* (peak to trough 2 to <5 mm), *medium or moderate* (5 to <10 mm), *coarse* (10 to <15 mm), or *very coarse* (>15 mm)
Clinical manifestations	• Pulse disappears with onset of VF – The pulse may disappear before the onset of VF if a common precursor to VF, rapid VT, develops before the VF. • Collapse, unresponsiveness • Agonal gasps or apnea • Sudden death
Common etiologies	• ACS leading to ischemic areas of myocardium • Stable to unstable VT, untreated • Premature ventricular complexes (PVCs) with R-on-T phenomenon • Multiple drug, electrolyte, or acid-base abnormalities that prolong the relative refractory period • Primary or secondary QT prolongation • Electrocution, hypoxia, many others

Figure 16. A, Coarse VF. Note high-amplitude waveforms, which vary in size, shape, and rhythm, representing chaotic ventricular electrical activity. B, Fine VF. Note the complete absence of QRS complexes.

A



B



The ECG criteria for VF are as follows:

- QRS complexes: No normal-looking QRS complexes are recognizable; a regular negative-positive-negative pattern (Q-R-S) cannot be seen.
- Rate: Uncountable; electrical deflections are very rapid and too disorganized to count.
- Rhythm: No regular rhythmic pattern can be discerned; the electrical waveforms vary in size and shape; the pattern is completely disorganized.

In comparison with Figure 16A, the amplitude of electrical activity in Figure 16B is much reduced. In terms of electrophysiology, prognosis, and the likely clinical response to attempted defibrillation, adrenergic agents, or antiarrhythmics, this rhythm pattern may be difficult to distinguish from that of asystole.

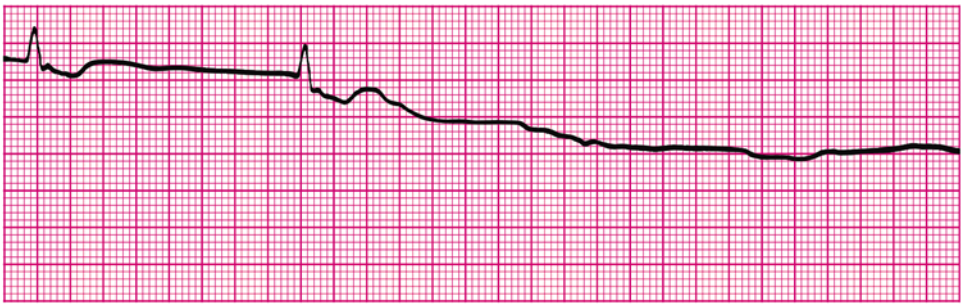
Pulseless electrical activity	
Pathophysiology	• Cardiac conduction impulses occur in an organized pattern but do not produce myocardial contraction (this condition was formerly called *electromechanical dissociation*), insufficient ventricular filling during diastole, or ineffective contractions.
Defining criteria per ECG	• Rhythm that displays organized electrical activity (not VF/pVT) • Usually not as organized as normal sinus rhythm • Can be narrow (QRS <0.12 second) or wide (QRS ≥0.12 second); fast (>100/min) or slow (<60/min) • Narrow QRS and fast heart rate mostly caused by noncardiac etiology; wide QRS and slow heart rate mostly caused by cardiac etiology
Clinical manifestations	• Collapse, unresponsiveness • Agonal gasps or apnea • No pulse detectable by palpation Very low systolic blood pressure could still be present in such cases.
Common etiologies	Use the H's and T's mnemonic to recall possible underlying causes: • Hypovolemia • Hypoxia • Hydrogen ion (acidosis) • Hypo-/hyperkalemia

Pulseless electrical activity	
	• Hypothermia • Tension pneumothorax • Tamponade, cardiac • Toxins (ie, drug overdose, ingestion) • Thrombosis, pulmonary (embolism) • Thrombosis, coronary (ACS)

Asystole	
Defining criteria per ECG Classically, asystole presents as a "flat line"; defining criteria are virtually nonexistent.	• **Rate:** No ventricular activity seen; so-called *P-wave asystole* occurs with only atrial impulses present (P waves) • **Rhythm:** No ventricular activity seen • **PR:** Cannot be determined; occasionally P wave is seen, but by definition R wave must be absent • **QRS complex:** No deflections seen that are consistent with a QRS complex
Clinical manifestations	• Collapse; unresponsiveness • Agonal gasps (early) or apnea • No pulse or blood pressure • Death
Common etiologies	• End of life (death) • Cardiac ischemia • Acute respiratory failure/hypoxia from many causes (no oxygen, apnea, asphyxiation) • Massive electrical shock (eg, electrocution, lightning strike) • May represent "stunning" of the heart immediately after defibrillation (shock delivery that eliminates VF) before resumption of spontaneous rhythm

Figure 17. The "rhythm" of ventricular asystole. This patient is pulseless and unresponsive. Note the 2 QRS-like complexes at the start of this rhythm display. These complexes represent a minimum of electrical activity,

probably ventricular escape beats. Note the long section in which electrical activity is completely absent. This patient is in asystole at this point.

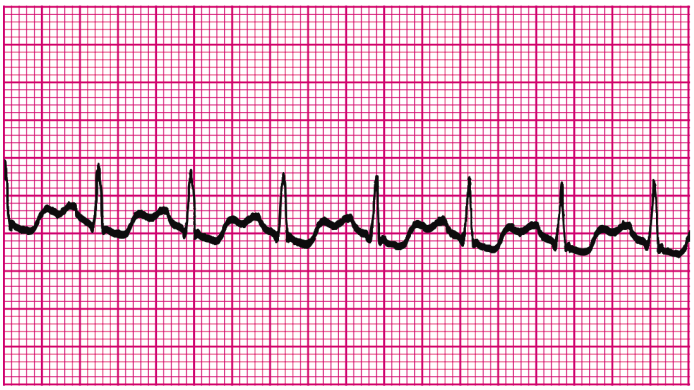


Recognition of Selected Nonarrest ECG Rhythms

Recognition of Supraventricular Tachyarrhythmias (SVTs)

Sinus tachycardia (Figure 18)	
Pathophysiology	• More of a physical response or sign than an actual arrhythmia or pathologic condition • Normal impulse formation and conduction
Defining criteria and ECG features	• **Rate:** >100/min • **Rhythm:** Sinus • **PR:** Usually <0.20 second • **P for every QRS complex** • **QRS complex:** May be normal or wide if there is an underlying abnormality
Clinical manifestations	• None specific for the tachycardia • Symptoms may be present due to the cause of the tachycardia (fever, hypovolemia, etc)
Common etiologies	• Normal exercise • Hypoxemia • Fever • Hypovolemia • Adrenergic stimulation, anxiety • Hyperthyroidism • Anemia • Pain

Figure 18. Sinus tachycardia.



Atrial fibrillation (Figure 19) and atrial flutter (Figure 20)			
Pathophysiology	• Atrial impulses are faster than sinoatrial (SA node) impulses. • Atrial fibrillation: Impulses take multiple chaotic, random pathways through atria. • Atrial flutter: Impulses take a circular course around atria, setting up flutter waves.		
Defining criteria and ECG features Distinctions between atrial fibrillation and atrial flutter; all other characteristics are the same **Atrial fibrillation key:** A classic clinical axiom is "Irregularly irregular rhythm—with variation in both interval and amplitude from R wave to R wave—is atrial fibrillation." This one is usually dependable and can also be observed in multifocal atrial tachycardia. **Atrial flutter key:** Flutter waves in classic "sawtooth" pattern		**Atrial fibrillation**	**Atrial flutter**
	Rate	• Wide-ranging ventricular response to atrial undulations that occur between 300 and 400 per minute • May be normal or slow if atrioventricular (AV) nodal conduction is abnormal (eg, "sick sinus syndrome")	• Atrial rate is 220 to 350/min. • Ventricular response is a function of AV node block or conduction of atrial impulses. • Ventricular response is rarely >150 to 180 because of AV nodal conduction limits.
	Rhythm	Irregular (classic "irregularly irregular")	• Regular • Ventricular rhythm often regular • Set ratio to atrial rhythm, eg, 2:1 or 4:1
	P waves	• Chaotic atrial fibrillatory waves only • Creates variable baseline	• No true P waves seen • Flutter waves in classic "sawtooth" pattern
	PR	• Cannot be measured	

Atrial fibrillation (Figure 19) and atrial flutter (Figure 20)		
	QRS	• Remains <0.12 second unless QRS complex is distorted by fibrillation or flutter waves or by conduction defects through ventricles
Clinical manifestations	• Signs and symptoms are a function of the rate of ventricular response to atrial fibrillation waves; "atrial fibrillation with rapid ventricular response" may be characterized by dyspnea on exertion, shortness of breath, and sometimes acute pulmonary edema. • Loss of "atrial kick" may lead to drop in cardiac output and decreased coronary perfusion. • Irregular rhythm is often perceived as "palpitations." • Atrial fibrillation and atrial flutter can be asymptomatic.	
Common etiologies	• ACS, coronary artery disease, congestive heart failure • Disease at mitral or tricuspid valve • Hypoxia, acute pulmonary embolism • Drug-induced: digoxin or quinidine; β-agonists, theophylline • Sepsis • Hypertension • Hyperthyroidism	

Figure 19. Atrial fibrillation.

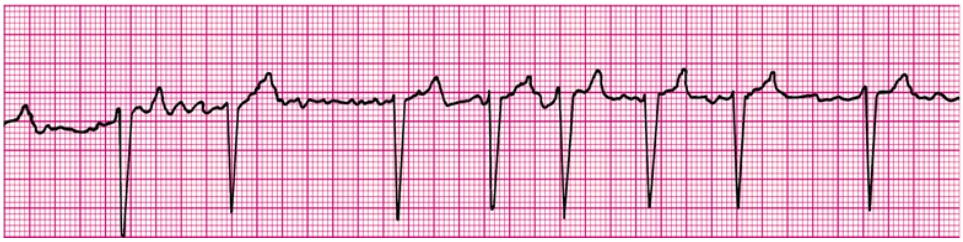
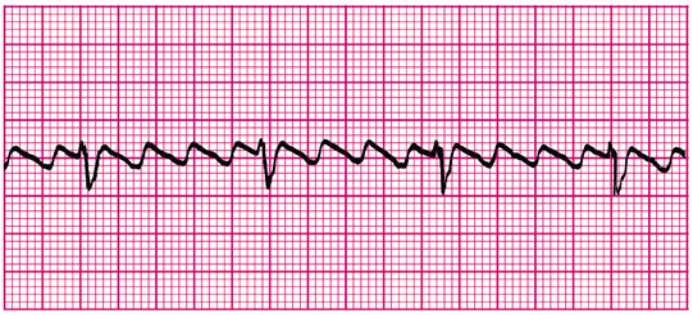
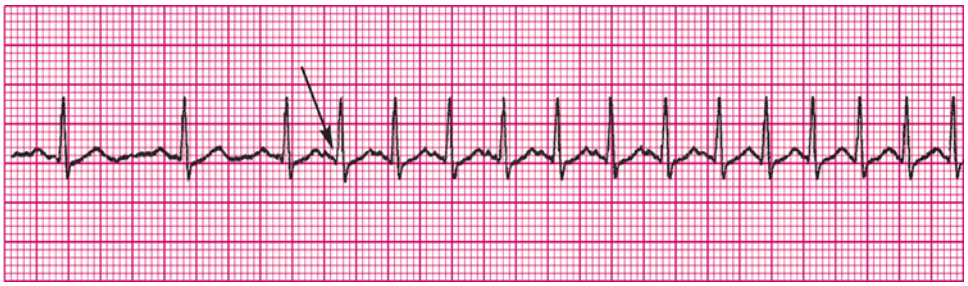


Figure 20. Atrial flutter (sawtooth pattern).



Accessory-mediated SVT (Figure 21) May include AV nodal reentrant tachycardia or AV reentry tachycardia	
Pathophysiology	• Reentry phenomenon: Impulses recycle repeatedly in the AV node because an abnormal rhythm circuit allows a wave of depolarization to travel in a circle. Usually the depolarization travels antegrade (forward) through the abnormal pathway and then circles back retrograde through the "normal" conduction tissue.
Defining criteria and ECG features **Key:** Regular, narrow-complex tachycardia without P waves and sudden onset or cessation ***Note:*** To merit the diagnosis of reentry SVT, some experts require capture of the abrupt onset or cessation on a monitor strip.	• **Rate:** Exceeds upper limit of sinus tachycardia at rest (>220/min), seldom <150/min, often up to 250/min • **Rhythm:** Regular • **P waves:** Seldom seen because rapid rate causes P wave to be "hidden" in preceding T waves or to be difficult to detect because the origin is low in the atrium • **QRS complex:** Normal, narrow
Clinical manifestations	• Palpitations felt by patient at onset; becomes anxious, uncomfortable • Low exercise tolerance with very high rates • Symptoms of unstable tachycardia may occur
Common etiologies	• Accessory conduction pathway occurs in many SVT patients. • For such otherwise healthy people, many factors can provoke the reentry SVT: caffeine, hypoxia, cigarettes, stress, anxiety, sleep deprivation, numerous medications. • Frequency of SVT increases in unhealthy patients with coronary artery disease, chronic obstructive pulmonary disease, and congestive heart failure.

Figure 21. Sinus rhythm with a reentry SVT.

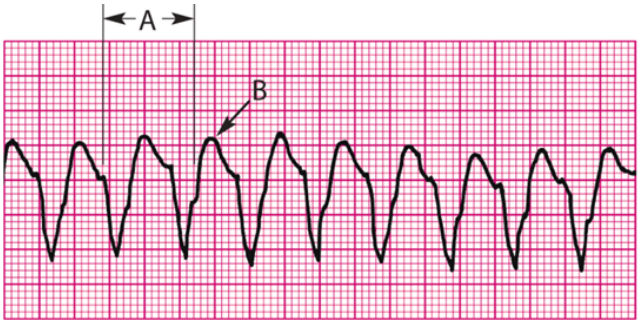


Recognition of Ventricular Tachyarrhythmias

Monomorphic VT (Figure 22)	
Pathophysiology	• Impulse conduction is slowed around areas of ventricular injury, infarct, or ischemia.

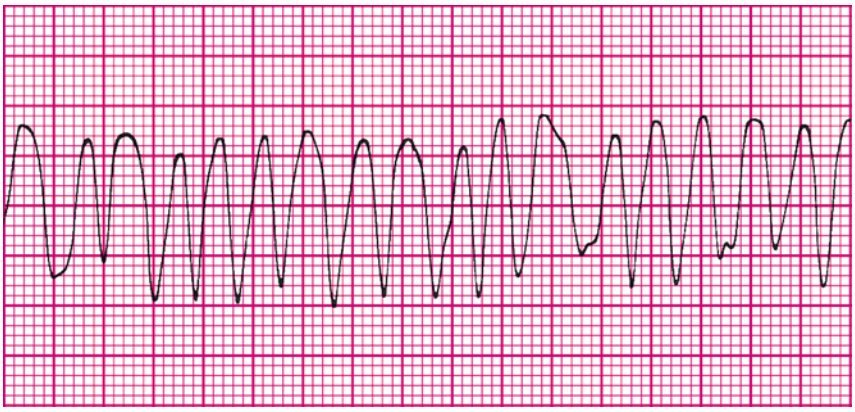
Monomorphic VT (Figure 22)	
	• These areas also serve as sources of ectopic impulses (irritable foci). • These areas of injury can cause the impulse to take a circular course, leading to the reentry phenomenon and rapid repetitive depolarizations.
Defining criteria per ECG **Key:** The same morphology, or shape, is seen in every QRS complex. ***Notes:*** • Three or more consecutive PVCs indicate VT. • VT of less than 30 seconds duration is nonsustained VT. • VT of more than 30 seconds duration is sustained VT.	• Rate: Ventricular rate >100/min; typically 120 to 250/min • Rhythm: Regular ventricular rhythm • PR: Absent (rhythm is AV dissociated) • P waves: Seldom seen but present; VT is a form of AV dissociation, a defining characteristic for wide-complex tachycardias of ventricular origin versus supraventricular tachycardias with aberrant conduction • QRS complex: Wide and bizarre, "PVC-like" complexes ≥0.12 second, with large T wave of opposite polarity from QRS • Fusion beats: Occasional chance capture of a conducted P wave. Resulting QRS "hybrid" complex, part normal, part ventricular • Nonsustained VT: Lasts <30 seconds and does not require intervention
Clinical manifestations	• Typically, symptoms of decreased cardiac output (orthostasis, hypotension, syncope, exercise limitations, etc) do develop. • Monomorphic VT can be asymptomatic despite widespread belief that sustained VT always produces symptoms. • Untreated and sustained VT will deteriorate to unstable VT and often to VF.
Common etiologies	• An acute ischemic event (see Pathophysiology) with areas of ventricular irritability leading to PVCs • Low ejection fraction due to chronic systolic heart failure • PVCs that occur during relative refractory period of cardiac cycle (R-on-T phenomenon) • Drug-induced prolonged QT interval (tricyclic antidepressants, procainamide, sotalol, amiodarone, ibutilide, dofetilide, some antipsychotics, digoxin, some long-acting antihistamines, certain antibiotics)

Figure 22. Monomorphic VT at a rate of 150/min; wide QRS complexes (arrow A) with opposite polarity T waves (arrow B).



Polymorphic VT (Figure 23)	
Pathophysiology	• Impulse conduction is slowed around multiple areas of ventricular injury, infarct, or ischemia. • These areas also serve as the source of ectopic impulses (irritable foci); irritable foci occur in multiple areas of the ventricles and thus are polymorphic. • These areas of injury can cause impulses to take a circular course, leading to the reentry phenomenon and rapid repetitive depolarizations.
Defining criteria per ECG **Key:** Marked variation and inconsistency seen in QRS complexes	• **Rate:** Ventricular rate >100/min; typically 120 to 250/min • **Rhythm:** Regular or irregular ventricular; no atrial activity • **PR:** Nonexistent • **P waves:** Seldom seen but present; VT is a form of AV dissociation • **QRS complexes:** Marked variation and inconsistency seen in QRS complexes
Clinical manifestations	• Typically will rapidly deteriorate to pVT or VF • Symptoms of decreased cardiac output (orthostasis, hypotension, poor perfusion, syncope, etc) present before pulseless arrest
Common etiologies	• Acute ischemic event (see Pathophysiology) with areas of ventricular irritability • PVCs that occur during relative refractory period of cardiac cycle (R-on-T phenomenon) • Drug-induced prolonged QT interval (tricyclic antidepressants, procainamide, sotalol, amiodarone, ibutilide, dofetilide, some antipsychotics, digoxin, some long-acting antihistamines, certain antibiotics) • Hereditary long QT interval syndromes

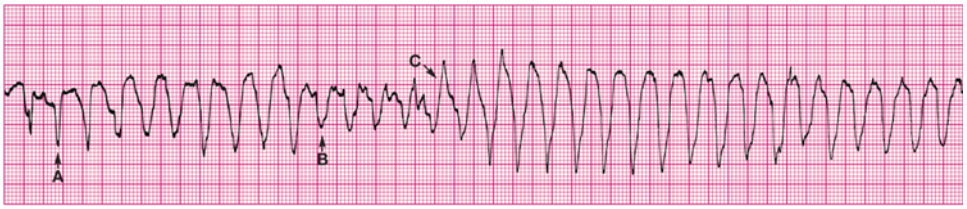
Figure 23. Polymorphic VT; QRS complexes display multiple morphologies.



Torsades de pointes (a unique subtype of polymorphic VT) (Figure 24)	
Pathophysiology	Specific pathophysiology of classic torsades: • QT interval is abnormally long (baseline ECG) and leads to an increase in the relative refractory period ("vulnerable period") of the cardiac cycle. This increases the probability that an irritable focus (PVC) will occur on the T wave (vulnerable period or R-on-T phenomenon). • The R-on-T phenomenon often induces VT.
Defining criteria per ECG **Key:** QRS complexes display a "spindle-node" pattern, in which VT amplitude increases and then decreases in a regular pattern, creating the spindle. The initial deflection at the start of one spindle (eg, negative) will be followed by complexes of opposite (eg, positive) polarity or deflection at the start of the next spindle, creating the node.	• **Atrial rate:** Cannot be determined • **Ventricular rate:** 150 to 250 complexes per minute • **Rhythm:** Only irregular ventricular rhythm • **PR:** Nonexistent • **P waves:** Nonexistent • **QRS complexes:** Display classic spindle-node pattern (see Key at left)
Clinical manifestations	• Tendency toward sudden deterioration to pVT or VF • Symptoms of decreased cardiac output typical (orthostasis, hypotension, syncope, signs of poor perfusion, etc) • "Stable" torsades; sustained torsades is uncommon
Common etiologies	• Most commonly occurs in patients with prolonged QT interval, due to many causes: – Drug-induced: tricyclic antidepressants, procainamide, sotalol, amiodarone, ibutilide, dofetilide, some antipsychotics, digoxin, some long-acting antihistamines, certain antibiotics

Torsades de pointes (a unique subtype of polymorphic VT) (Figure 24)	
	– Electrolyte and metabolic alterations (hypomagnesemia is the prototype) ▪ Inherited forms of long QT syndrome ▪ Acute ischemic events (see Pathophysiology)

Figure 24. Torsades de pointes, a unique type of polymorphic VT. A, Start of a spindle. Note negative initial deflection and increasing QRS amplitude. B, End of a spindle and start of a node. C, End of a node and start of the next spindle. Note the positive initial deflection and spindling in QRS amplitude.

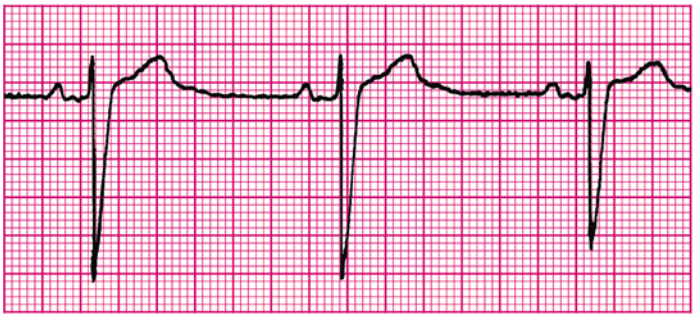


Recognition of Sinus Bradycardia

Sinus bradycardia (Figure 25)	
Pathophysiology	• Impulses originate at SA node at a slow rate • May be physiologic • Can be a physical sign, as in sinus tachycardia
Defining criteria per ECG **Key:** Regular P waves followed by regular QRS complexes at rate <60/min ***Note:*** Often a physical sign rather than an abnormal rhythm	• **Rate:** <60/min (when symptomatic and bradycardia is the cause of symptoms, the rate is generally <50/min) • **Rhythm:** Regular sinus • **PR:** Regular, 0.12 to 0.20 second • **P waves:** Size and shape normal; every P wave followed by a QRS complex; every QRS complex preceded by a P wave • **QRS complex:** Narrow; <0.12 second (often <0.11 second) in absence of intraventricular conduction defect
Clinical manifestations	• Person is usually asymptomatic when at rest. • With increased activity and sinus node dysfunction, a persistent slow rate can lead to symptoms of easy fatigue, shortness of breath, dizziness or light-headedness, syncope, hypotension, diaphoresis, pulmonary congestion, and frank pulmonary edema. • The ECG can independently display acute ST-segment or T-wave deviation or ventricular arrhythmias.
Common etiologies	• Can be normal for well-conditioned people • Vasovagal event, such as vomiting, Valsalva maneuver, rectal stimuli, inadvertent pressure on carotid sinus (“shaver’s syncope”)

Sinus bradycardia (Figure 25)	
	• ACS that affect circulation to SA node (right coronary artery); most often inferior acute myocardial infarctions (AMIs) • Adverse drug effects, eg, β-blockers or calcium channel blockers, digoxin, quinidine

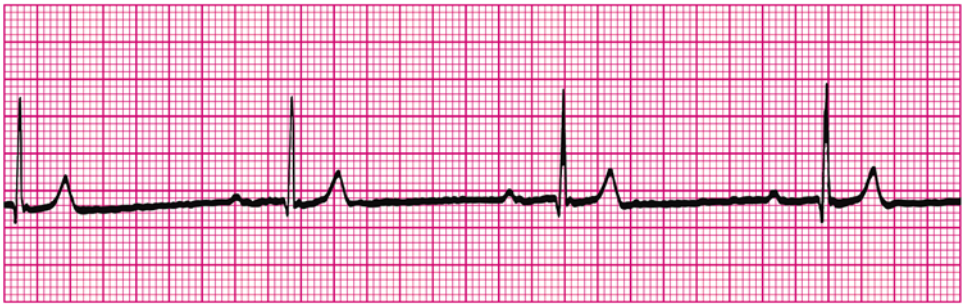
Figure 25. Sinus bradycardia.



Recognition of AV Block

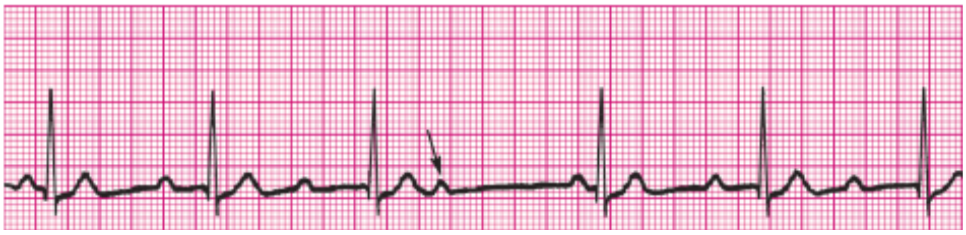
First-degree AV block (Figure 26)	
Pathophysiology	• Impulse conduction slowed *(partial block)* at AV node for a fixed interval • May be a sign of another problem or a primary conduction abnormality
Defining criteria per ECG **Key:** PR interval >0.20 second	• **Rate:** First-degree AV block seen with rhythms with both sinus bradycardia and sinus tachycardia as well as a normal sinus mechanism • **Rhythm:** Sinus, regular, both atria and ventricles • **PR:** Prolonged, >0.20 second but does not vary *(fixed)* • **P waves:** Size and shape normal; every P wave followed by a QRS complex; every QRS complex preceded by a P wave • **QRS complex:** Narrow, <0.12 second in absence of intraventricular conduction defect
Clinical manifestations	• Usually asymptomatic
Common etiologies	• Many first-degree AV blocks due to drugs, usually the AV nodal blockers: β-blockers, nondihydropyridine calcium channel blockers, and digoxin • Any condition that stimulates the parasympathetic nervous system (eg, vasovagal reflex) • AMI that affects circulation to the AV node (right coronary artery); most often inferior AMI

Figure 26. First-degree AV block.



Type I second-degree AV block (Mobitz I–Wenckebach) (Figure 27)	
Pathophysiology	• Site of pathology is AV node. • AV node blood supply comes from branches of right coronary artery (right dominant circulation). • Impulse conduction is progressively slowed at AV node (causing increasing PR interval) until one sinus impulse is completely blocked and QRS complex fails to follow.
Defining criteria per ECG **Key:** There is progressive lengthening of PR interval until one P wave is not followed by QRS complex (dropped beat).	• **Rate:** Atrial rate just slightly faster than ventricular (because of dropped conduction), usually within normal range. • **Rhythm:** Atrial complexes are regular and ventricular complexes are irregular in timing (because of dropped beats); can show regular P waves marching through irregular QRS. • **PR:** Progressive lengthening of PR interval occurs from cycle to cycle; and then one P wave is not followed by QRS complex ("dropped beat"). • **P waves:** Size and shape remain normal; occasional P wave is not followed by QRS complex ("dropped beat"). • **QRS complex:** <0.12 second most often, but a QRS "drops out" periodically.
Clinical manifestations—rate-related	**Due to bradycardia** • Most often asymptomatic • **Symptoms:** Chest pain, shortness of breath, decreased level of consciousness • **Signs:** Hypotension, shock, pulmonary congestion, congestive heart failure, angina
Common etiologies	• AV nodal blocking agents: β-blockers, nondihydropyridine calcium channel blockers, digoxin • Conditions that stimulate the parasympathetic nervous system • ACS that involves right coronary artery

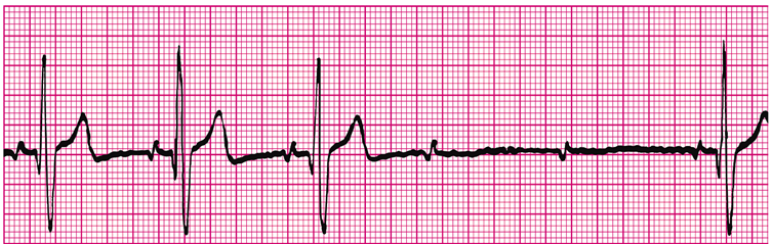
Figure 27. Type I second-degree AV block. Note the progressive lengthening of the PR interval until one P wave (arrow) is not followed by a QRS.



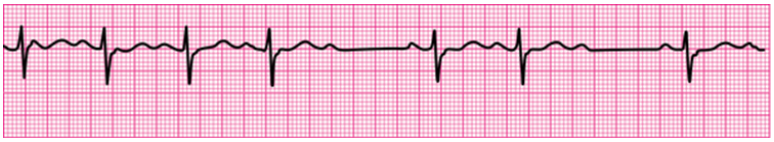
Type II second-degree AV block (infranodal; Mobitz II) (Figure 28)	
Pathophysiology	• The site of the block is most often below the AV node (infranodal) at the bundle of His (infrequent) or at bundle branches. • Impulse conduction is normal through node, thus no first-degree block and no prior PR prolongation.
Defining criteria per ECG	• **Atrial rate:** Usually 60 to 100/min • **Ventricular rate:** By definition (because of blocked impulses), slower than atrial rate • **Rhythm:** Atrial is regular; ventricular is irregular (because of blocked impulses); ventricular is regular if there is consistent 2:1 or 3:1 block • **PR:** Constant and set; no progressive prolongation as with Type I second-degree AV block Mobitz—*a distinguishing characteristic* • **P waves:** Typical in size and shape; by definition, some P waves will not be conducted and therefore not followed by a QRS complex • **QRS complex:** Narrow (<0.12 second) implies high block relative to AV node; wide (≥0.12 second) implies low block relative to AV node
Clinical manifestations—rate-related	**Due to bradycardia** • **Symptoms:** Chest pain, shortness of breath, decreased level of consciousness • **Signs:** Hypotension, shock, pulmonary congestion, congestive heart failure, AMI
Common etiologies	• ACS that involves branches of *left* coronary artery

Figure 28. A, Type II (high block): regular PR-QRS intervals until 2 dropped beats occur; borderline normal QRS complexes indicate high nodal or nodal block. B, Type II (low block): regular PR-QRS intervals until dropped beats; wide QRS complexes indicate infranodal block.

A



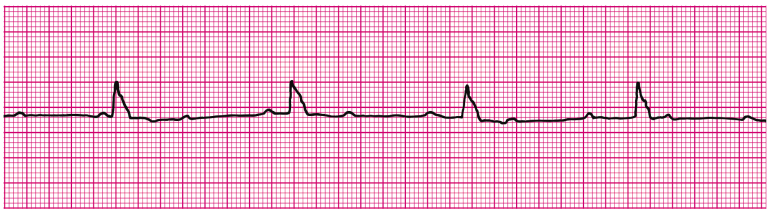
B



Third-degree AV block and AV dissociation (Figure 29)	
Pathophysiology pearl *AV dissociation* is the defining class; *third-degree* or *complete AV block* is one type of AV dissociation.	• Injury or damage to cardiac conduction system so that no impulses *(complete block)* pass between atria and ventricles (neither antegrade nor retrograde) • This complete block can occur at several different anatomic areas: – AV node (high-, supra-, or junctional nodal block) – Bundle of His – Bundle branches (low-nodal or infranodal block)
Defining criteria per ECG **Key:** Third-degree block (see Pathophysiology) causes atria and ventricles to depolarize independently, with no relationship between the two (AV dissociation).	• **Atrial rate:** Usually 60 to 100/min; impulses completely independent (dissociated) from the slower ventricular rate • **Ventricular rate:** Depends on rate of ventricular escape beats that arise: – Ventricular escape rate slower than atrial rate = third-degree AV block (rate = 20 to 40/min) – Ventricular escape rate faster than atrial rate = AV dissociation (rate = 40 to 55/min) • **Rhythm:** Both atrial rhythm and ventricular rhythm regular but independent (dissociated) • **PR:** By definition, no relationship between P wave and R wave • **P waves:** Typical in size and shape • **QRS complex:** Narrow (<0.12 second) implies high block relative to AV node; wide (≥0.12 second) implies low block relative to AV node

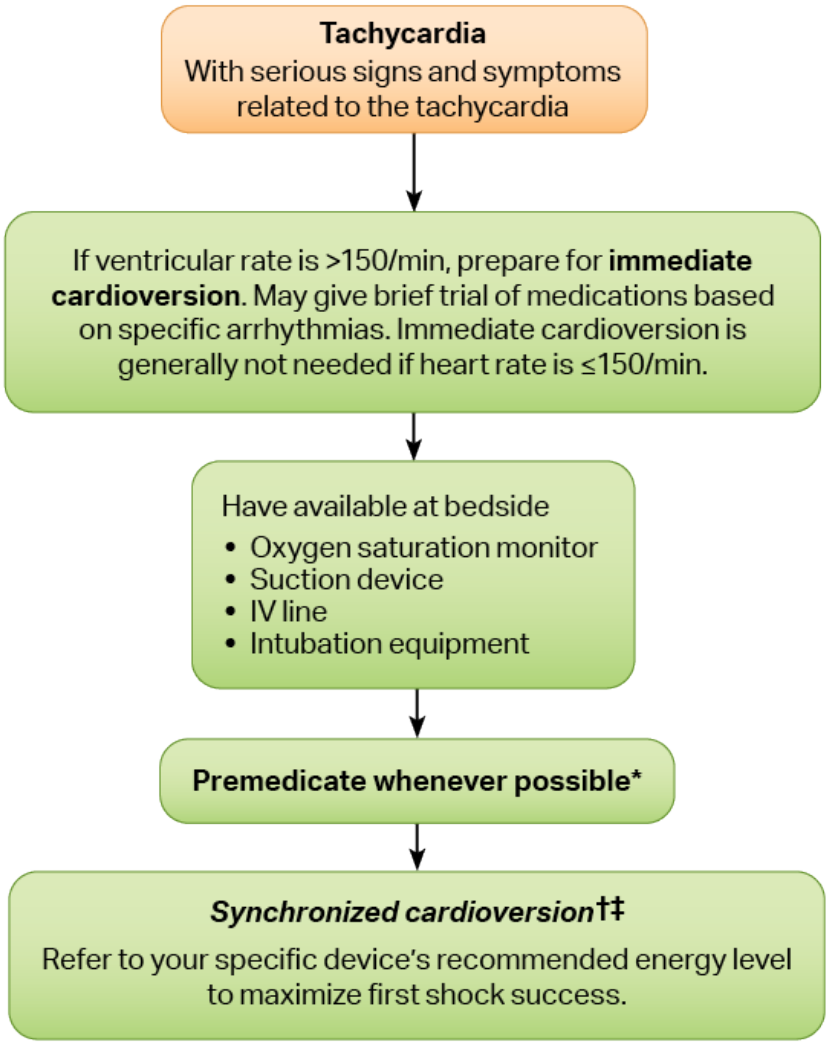
Third-degree AV block and AV dissociation (Figure 29)	
Clinical manifestations—rate-related	**Due to bradycardia** • **Symptoms:** Chest pain, shortness of breath, decreased level of consciousness • **Signs:** Hypotension, shock, pulmonary congestion, congestive heart failure, AMI
Common etiologies	• ACS that involves branches of *left* coronary artery • In particular, involves left anterior descending artery and branches to interventricular septum (supply bundle branches)

Figure 29. Third-degree AV block. Regular P waves at 50 to 55/min; regular ventricular "escape beats" at 35 to 40/min; no relationship between P waves and escape beats.



The Electrical Cardioversion Algorithm (Figure 30) is used for unstable SVT or unstable monomorphic VT.

Figure 30. Electrical Cardioversion Algorithm.



Notes

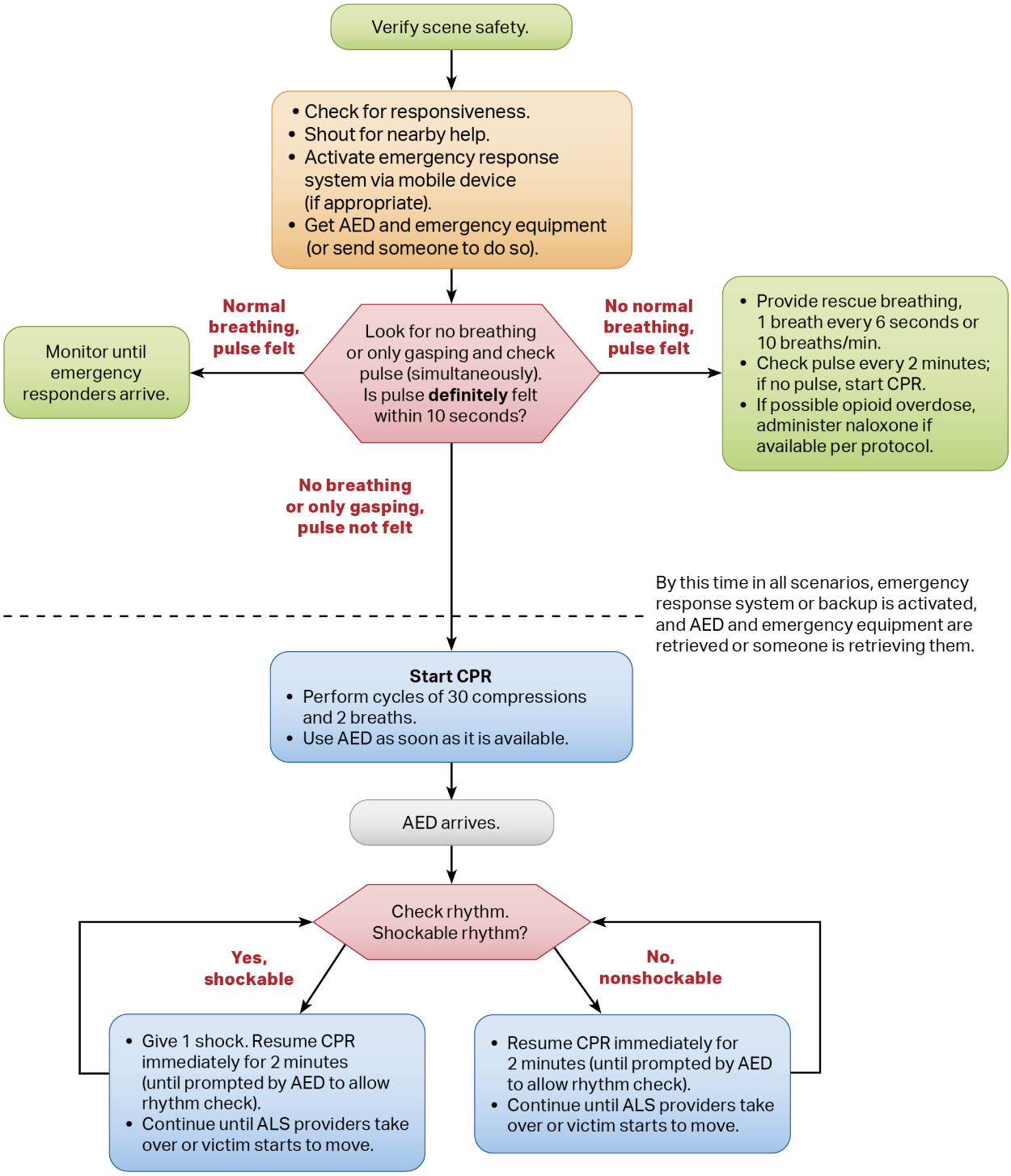
*Effective regimens have included a sedative **(eg, diazepam, midazolam, etomidate, methohexital, propofol)** with or without an analgesic agent **(eg, fentanyl, morphine)**. Many experts recommend anesthesia if service is readily available.

†Note possible need to resynchronize after each cardioversion.

‡If delays in synchronization occur and clinical condition is critical, go immediately to unsynchronized shocks.

VF Treated With CPR and Automated External Defibrillator

Figure 31. Adult BLS Algorithm for Healthcare Providers.



Defibrillation

Automated External Defibrillator

Operation

ACLS providers will typically use a manual defibrillator to shock a patient in VF or pVT. In some cases, only an automated external defibrillator (AED) will be available. The information below will discuss the use of AEDs within the framework of the Adult BLS Algorithm for Healthcare Providers (Figure 31).

Use AEDs only when patients have the following 3 clinical findings:

- No response
- Absent or abnormal breathing (ie, no breathing or only gasping)
- No pulse

In the first few minutes after the onset of sudden cardiac arrest (SCA), the patient may demonstrate agonal gasps, which are not adequate breathing. A nonresponsive patient with agonal gasping who has no pulse is in cardiac arrest.

Know Your AED

You must be familiar with the AED used in your clinical setting and be ready to use it at any time. Review the troubleshooting checklist supplied by the AED manufacturer. Learn to perform daily maintenance checks. Not only are these checks an effective review of the steps of operation, but they are also a means of verifying that the AED is ready for use.

Universal Steps for Operating an AED

After the AED arrives, place it at the patient's side, next to the rescuer who will operate it. This position provides ready access to the AED controls and easy placement of electrode pads. It also allows a second rescuer to perform CPR from the opposite side of the patient without interfering with AED operation.

AEDs are available in different models. Although there are small differences from model to model, all AEDs operate in basically the same way. The following table lists the universal steps for operating an AED:

Step	Action
1	**Open** the carrying case. **Power on the AED** if needed. • Some devices will power on automatically when you open the lid or case. • Follow the AED prompts as a guide to next steps.
2	**Attach AED pads** to the patient's bare chest. Choose adult pads (not child pads or a child system) victims 8 years of age and older. • Peel the backing away from the AED pads.

Step	Action
	• Attach the adhesive AED pads to the patient's bare chest. Follow the placement diagrams on the pad (Figure 32). See Foundational Facts: Alternative AED Electrode Pad Placement Positions for common placement options. • Attach the AED connecting cables to the AED device (some AED cables are already preconnected to the device).
3	Clear the patient and allow the AED to **analyze** the rhythm**.** • When the AED prompts you, clear the victim during analysis. Be sure that no one is touching the patient, not even the rescuer in charge of giving breaths. • Some AEDs will tell you to push a button to allow the AED to begin analyzing the heart rhythm; others will do that automatically. The AED may take a few seconds to analyze. • The AED then tells you if a shock is needed.
4	If the AED advises a shock, it will tell you to **clear** the victim and then deliver a shock. • Clear the patient before delivering the shock; be sure that no one is touching the patient. • Loudly state a "clear the patient" message, such as "Everybody clear" or simply "Clear." • Look to be sure that no one is in contact with the victim. • Press the shock button. • The shock will produce a sudden contraction of the patient's muscles.
5	If no shock is needed, and after any shock delivery, **immediately resume CPR**, starting with chest compressions.
6	After about 2 minutes of CPR, the AED will prompt you to repeat steps 3 and 4.

Immediately resume high-quality CPR, starting with chest compressions, after

- A shock is delivered or
- The AED prompts "no shock advised"

After 2 minutes of high-quality CPR, the AED will prompt you to repeat steps 3 and 4. Continue until advanced life support providers take over or the patient begins to breathe, move, or otherwise react.

Figure 32. AED electrode pad placement on the patient.

Alternative AED Electrode Pad Placement Positions

There are 4 acceptable AED electrode pad positions:

- Anterolateral
- Anteroposterior
- Anterior–left infrascapular
- Anterior–right infrascapular

All 4 positions are equally effective in shock success and are reasonable for defibrillation. For ease of placement, anterolateral is a reasonable default electrode placement. Providers may consider alternative pad positions based on individual patient characteristics.

Troubleshooting the AED

Studies of AED failures have shown that most problems are caused by operator error rather than by AED defects. Operator error is less likely if the operator is experienced in using the AED, has had recent training or practice with the AED, and is using a well-maintained AED.

If the AED does not promptly analyze the rhythm, do the following:

- Resume high-quality chest compressions and ventilation.
- Check all connections between the AED and the patient to make sure that they are intact.

Never delay chest compressions to troubleshoot the AED.

Shock First vs CPR First

When you care for an adult patient in cardiac arrest, should you attempt to shock first with an AED or provide CPR first?

Healthcare providers who treat cardiac arrest in hospitals and other facilities should provide immediate CPR until the AED/defibrillator is ready for use. Use the AED as soon as it is available.

At this time, the benefit of delaying defibrillation to perform CPR before defibrillation is unclear. EMS system medical directors may consider implementing a protocol that allows EMS responders to provide CPR while preparing for defibrillation of patients found by EMS personnel to be in VF. In practice, however, CPR can be initiated while the AED is being readied.

With in-hospital SCA, there is insufficient evidence to support or refute CPR before defibrillation. However, in monitored patients, the time from VF to defibrillation should be under 3 minutes. When 2 or more rescuers are present, one rescuer should begin CPR while the other activates the emergency response system and prepares the defibrillator.

AED Use in Special Situations

The following special situations may require the operator to take extra care in placing the electrode pads when using an AED.

Hairy Chest

If the patient has a hairy chest, the AED pads may stick to the hair and not the skin on the chest. If this occurs, the AED will not properly analyze the patient's heart rhythm. The AED will give a "check electrodes" or "check electrode pads" message. If this happens, complete the following steps and actions while minimizing interruptions in chest compressions.

Step	Action
1	If the pads stick to the hair instead of the skin, press down firmly on each pad.
2	If the AED continues to prompt you to check pads or check electrodes, quickly pull off the pads. This will remove much of the hair.
3	If too much hair remains where you will put the pads, shave the area with the razor in the AED carrying case, if available.
4	Put on a new set of pads. Follow the AED voice prompts.

Water

Do not use an AED in water. If water is present on the patient's chest, it may conduct the shock electricity across the skin of the chest. This will prevent the delivery of an adequate shock dose to the heart.

If	Then
The patient is in the water	Pull the patient out of the water.

If	Then
The patient's chest is covered with water	Wipe the chest quickly before attaching the electrodes.
The patient is lying on snow or ice or in a small puddle	Use the AED.

Implanted Pacemaker

Patients known to be at high risk for SCA may have implanted defibrillators/pacemakers that automatically deliver shocks directly to the heart muscle if a life-threatening arrhythmia is detected. You can immediately identify these devices because they create a hard lump beneath the skin of the upper chest or abdomen. The lump ranges from the size of a silver dollar to half the size of a deck of cards, with a small overlying scar. The presence of an implanted defibrillator or pacemaker is not a contraindication to attaching and using an AED. Avoid placing the AED electrode pads directly over the device because the devices may interfere with each other.

If you identify an implanted defibrillator/pacemaker, take the following steps:

- If possible, place the AED electrode pad to either side and not directly on top of the implanted device.
- Follow the normal steps for operating an AED.

Note that a pacemaker spike may confuse the AED's interpretation of the patient's real heart rhythm. Occasionally, the analysis and shock cycles of implanted defibrillators and AEDs will conflict. If the implanted defibrillator is delivering shocks to the patient (the patient's muscles contract in a manner like that observed after an AED shock), allow 30 to 60 seconds for the implanted defibrillator to complete the treatment cycle before delivering a shock from the AED.

Transdermal Medication Patches

Do not place AED electrodes directly on top of a medication patch (eg, a patch of nitroglycerin, nicotine, pain medication, hormone replacement therapy, or antihypertensive medication). The medication patch may block the transfer of energy from the electrode pad to the heart or cause small burns to the skin. To prevent these complications, remove the patch and wipe the area clean before attaching the AED electrode pad. Try to minimize interruptions in chest compressions, and do not delay shock delivery.

Defibrillation and Safety

Manual Defibrillation

Using a Manual Defibrillator/Monitor

When using a manual defibrillator/monitor, perform a rhythm check as indicated by the ACLS Cardiac Arrest Algorithm. This can be performed by attaching the adhesive defibrillator electrode pads or placing the defibrillator paddles on the chest (with appropriate conduction surface or gel) to reduce transthoracic impedance, and by using the paddle quick-look feature.

At the time of writing of the *2020 AHA Guidelines for CPR and ECC,* there were no data to suggest that one of these modalities is better than the others are in reducing impedance. Because adhesive monitor/defibrillator electrode pads are as effective as paddles and gel pads or paste, and the pads can be placed before cardiac arrest to allow for monitoring and rapid administration of a shock when necessary, adhesive pads should be used routinely instead of standard paddles.

For adult defibrillation, both handheld paddles and self-adhesive pads (8 to 12 cm in diameter) perform well, although defibrillation success may be higher with electrodes 12 cm in diameter than with those 8 cm in diameter, whereas small electrodes (4.3 cm) may be harmful and may cause myocardial necrosis. When using handheld paddles and gel or pads, you must ensure that the paddle is in full contact with the skin. Even smaller pads have been found to be effective in VF of brief duration. Use of the smallest (pediatric) pads, however, can result in unacceptably high transthoracic impedance in larger children.

Early defibrillation is critical to survival from SCA. A common initial rhythm in out-of-hospital witnessed SCA is VF, the treatment for which is defibrillation. The probability of successful defibrillation diminishes rapidly over time, and VF tends to deteriorate to asystole within 10 to 15 minutes. Therefore, whether the adhesive electrode pads or paddles are being used, you should be very careful not to delay the shock during CPR to minimize the time between last compression and shock delivery. Intervals between pausing chest compressions and shock delivery have been shown to last approximately 20 to 30 seconds, which is no longer acceptable. If CPR is in progress, chest compressions should continue until the defibrillator electrode adhesive pads are attached to the chest and the manual defibrillator is ready to analyze the rhythm.

For every minute that passes between collapse and defibrillation, survival rates from witnessed VF SCA decrease 7% to 10% if no CPR is provided. When bystander CPR is provided, the decrease in survival rates is more gradual and averages 3% to 4% from collapse to defibrillation. CPR can double or triple survival from witnessed SCA at most intervals to defibrillation.

When any rescuer witnesses an out-of-hospital arrest and an AED is immediately available onsite, the rescuer should start CPR and use the AED as soon as possible. Healthcare providers who treat cardiac arrest in hospitals and other facilities with AEDs onsite should provide immediate CPR and should use the AED/defibrillator as soon as it becomes available.

When you identify VF/pVT, *immediately* deliver 1 shock, using the following energy levels:

- **Biphasic:** Device-specific; the first dose is typically a selected energy of 120 J with a rectilinear biphasic waveform and a first dose selected energy of 120 J to 200 J with a biphasic truncated exponential waveform. If you do not know the device or the manufacturer's recommendation regarding the specific dose shown to be effective for elimination of VF, defibrillation at the maximal dose may be considered.
- **Monophasic:** 360 J; if VF persists after the first shock, second and subsequent shocks of 360 J should be given.

After delivering a single shock, immediately resume CPR, pushing hard and fast at a rate of 100 to 120 compressions per minute. Minimize interruption of CPR, and allow full chest recoil after each compression.

Safety and Clearing the Patient

Clearing: You and Your Team

To ensure the safety of defibrillation, whether manual or automated, the defibrillator operator must always announce that a shock is about to be delivered and perform a visual check to make sure no one is in contact with the patient. The operator is responsible for clearing the patient and rescuers before each shock is delivered. Whenever you use a defibrillator, firmly state a defibrillation clearing or warning before each shock. The purpose of this warning is to ensure that no one has any contact with the patient and that no oxygen is flowing across the patient's chest or openly flowing across the electrode pads. You should state the warning quickly to minimize the time from last compression to shock delivery. For example, state, *"Clear. Shocking,"* and then perform a visual check to make sure you have no contact with the patient, the stretcher, or other equipment.

You do not need to use those exact words. But it is imperative that you warn others that you are about to deliver a shock and that everyone stand clear.

Make sure all personnel step away from the patient, remove their hands from the patient, and end contact with any device or object touching the patient. Any personnel in indirect contact with the patient, such as the team member holding a ventilation bag attached to an ET tube, must also disconnect the bag and end contact with the patient. The person responsible for airway support and ventilation should ensure that oxygen is not openly flowing around the electrode pads (or paddles) or across the patient's chest.

A Final Note About Defibrillators

Most modern AEDs and manual defibrillators use biphasic waveforms. Take the time to learn to operate the defibrillator used in your workplace and its energy settings. Remember, *early* defibrillation in the presence of shockable rhythm increases the patient's chance of survival. This principle holds true regardless of the type of defibrillator or waveform.

Access for Medications

Intravenous Access

Using Peripheral Veins for IV Access

The most common sites for IV access are in the hands and arms. Favored sites are the dorsum of the hands, the wrists, and the antecubital fossae. The antecubital vein is the preferred location for IV drug administration during CPR.

Anatomy: Upper Extremities

Starting at the radial side of the wrist, a thick vein, the superficial radial vein, runs laterally up to the antecubital fossa and joins the median cephalic vein to form the cephalic vein. Superficial veins on the ulnar aspect of the forearm run to the elbow and join the median basilic vein to form the basilic vein. The cephalic vein of the forearm bifurcates into a Y in the antecubital fossa, becoming the median cephalic (laterally) and the median basilic (medially).

Technique: Antecubital Venipuncture

The largest surface veins of the arm are in the antecubital fossa. Select these veins first for access if the patient is in circulatory collapse or cardiac arrest. Select a point between the junctions of 2 antecubital veins (Figure 33). The vein is more stable here, and venipuncture is more often successful.

If peripheral access is impossible, consider central access via the femoral veins since chest compressions and other resuscitation interventions should not be interrupted, and potential vascular injuries can be better controlled at this site.

If upper extremity access is impossible and a central line is not an option, consider a peripheral leg vein.

Figure 33. Antecubital venipuncture. A, Scene perspective from a distance. B, Close-up view of antecubital area: anatomy of veins of upper extremity.

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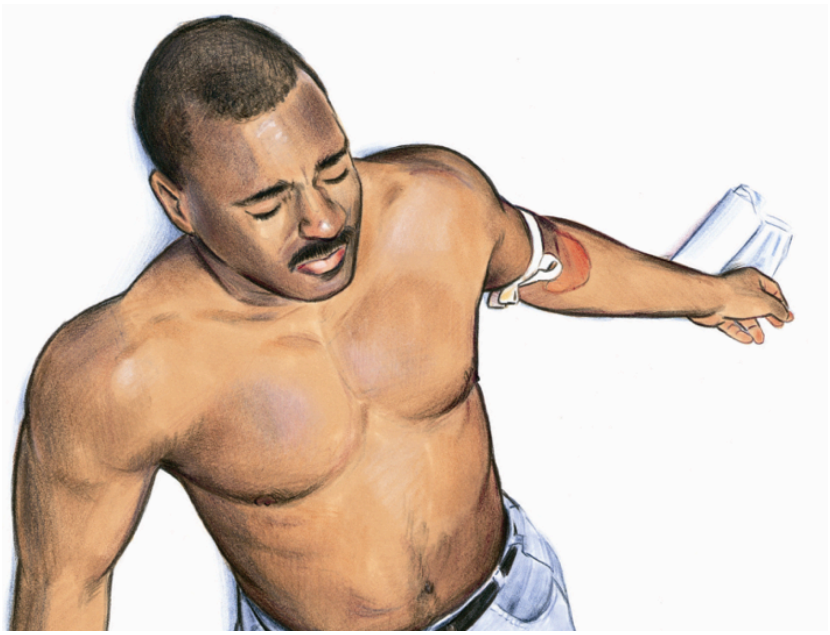

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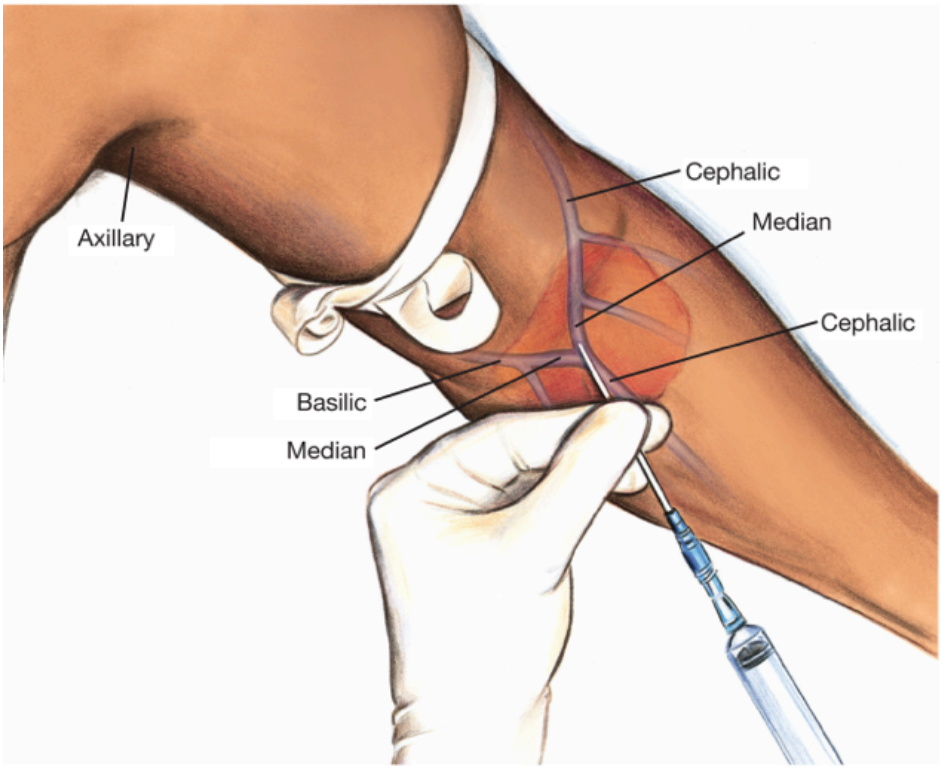


General IV Principles

Once you gain vascular access, follow these important principles for administering IV therapy:

- After a cardiac arrest patient becomes stable, remove the cannula inserted emergently, and replace it with a new one under sterile conditions. Strict aseptic technique is compromised in most emergency venipunctures, where speed is essential. This compromise is particularly likely when emergency vascular access is established outside the hospital because personnel and equipment are limited.
- IV solutions are usually packaged in nonbreakable plastic bottles or bags. Squeeze plastic bags before use to detect punctures that may lead to contamination of the contents.
- Avoid adding drugs that may be adsorbed by the plastic bag or tubing (eg, IV nitroglycerin). If you must administer these drugs without specialty infusion systems, allow for drug adsorption when you titrate the drug administration rate.
- Ideally, set the rate of infusion to at least 10 mL/h to keep the IV line open.
- Saline lock catheter systems are particularly useful for patients who have spontaneous circulation and require drug injections but not IV volume infusion.
- Most contemporary systems use needleless injection sites. These systems permit drug and flush infusions without the use of needles and the associated risk of needle sticks.
- Avoid letting the arm with the IV access hang off the bed. Place the arm at the level of the heart, or slightly above the heart, to facilitate delivery of fluids and medications to the central circulation.
- During cardiac arrest, follow all peripherally administered drugs with a bolus of at least 20 mL of IV flush solution. This flush will facilitate delivery to the central circulation. Elevate the extremity for 10 to 20 seconds to facilitate drug delivery to the central circulation.
- Be aware of complications common to all IV techniques. Local complications include hematomas, cellulitis, thrombosis, infiltration, and phlebitis. Systemic complications include sepsis, pulmonary thromboembolism, air embolism, and catheter fragment embolism.

Intraosseous Access

IO access can serve as a rapid, safe, and reliable route for administration of drugs, crystalloids, and colloids (including blood).

Needles

The technique uses a rigid needle, preferably a specially designed IO or Jamshidi-type bone marrow needle. In the past, the higher bone density in older children and adults made it difficult for smaller IO needles to penetrate the bone without bending. With the development of IO cannula systems and newer, drill-type devices, IO access is now easier to obtain in older children and adults.

Sites

Many sites are appropriate for IO infusion. In older children and adults, these general sites include the humeral head, proximal tibia, medial malleolus, sternum, distal radius, distal femur, and anterior-superior iliac spine.

Indications and Administration

Resuscitation drugs, fluids, and blood products can be administered safely by the IO route. Continuous catecholamine infusions can also be provided by this route.

The onset of action and drug levels after IO infusion during CPR are comparable to those for vascular routes of administration, including central venous access. When providing drugs and fluids by the IO route, remember the following:

- Flush all IO medications with normal saline to facilitate delivery into the central circulation.
- Administer viscous drugs and solutions and fluid for rapid volume resuscitation under pressure by using an infusion pump, pressure bag, or forceful manual pressure to overcome the resistance of the emissary veins.
- Some have expressed concern that high-pressure infusion of blood might induce hemolysis, but animal studies have failed to document this problem.

Contraindications

Absolute contraindications to IO access are as follows:

- Fractures and crush injuries near or proximal to the access site
- Conditions in which the bone is fragile, such as osteogenesis imperfecta
- Previous attempts to establish access in the same bone
- Presence of infection of the overlying tissues

Complications

Complications of IO infusion include tibial fracture, lower extremity compartment syndrome or severe extravasation of drugs, and osteomyelitis. But less than 1% of patients have complications after IO infusion. Careful technique helps to prevent complications.

Equipment Needed

The following equipment is needed to establish IO access:

- Gloves
- Skin disinfectant
- IO needle (15 to 18 gauge) or bone marrow needle
- Tape
- Syringe
- Isotonic crystalloid fluid
- Intravenous tubing

Procedure

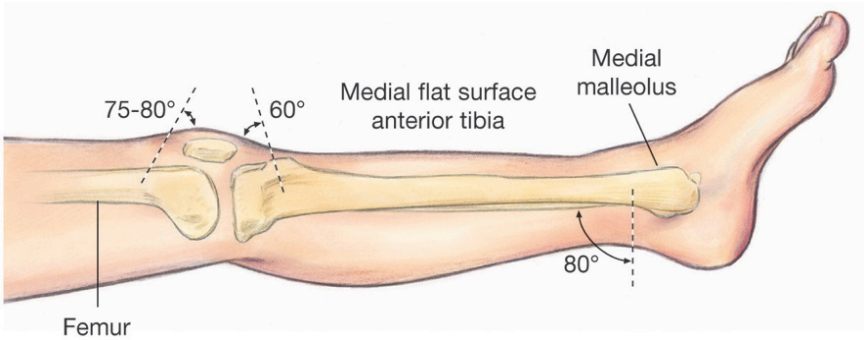
The steps to establish IO by using the tibial tuberosity as an example of an access site are described below. Commercial kits (eg, newer, drill-type IO devices) are currently available, and providers should follow the manufacturer's steps provided with the kit.

Step	Action
1	• Always use universal precautions when attempting vascular access. Disinfect the overlying skin and surrounding area with an appropriate agent. • Identify the tibial tuberosity just below the knee joint. The insertion site is the flat part of the tibia, 1 or 2 finger widths below and medial to this bony prominence. Figure 34 shows some of the sites for IO access.
2	• The stylet should remain in place during insertion to prevent the needle from becoming clogged with bone or tissue. • Stabilize the leg to facilitate needle insertion. *Do not place your hand behind the leg.*
3	• Insert the needle so that it is perpendicular to the tibia. (When placing an IO needle in other locations, aim slightly away from the nearest joint space to reduce the risk of injury to the epiphysis or joint, but keep the needle as perpendicular to the bone as possible to avoid bending.) – *Twist, do not push, the needle.* • Use a twisting motion with gentle but firm pressure. Some IO needles have threads. These threads must be turned clockwise and screwed into the bone.
4	• Continue inserting the needle through the cortical bone until there is a sudden release of resistance. This release occurs as the needle enters the marrow space. If the needle is placed correctly, it will stand easily without support.
5	• Remove the stylet and attach a syringe. • Aspiration of bone marrow contents and blood in the hub of the needle confirms appropriate placement. You may send this blood to the lab for study. (*Note:* Blood or bone marrow may *not* be aspirated in every case.) • Infuse a small volume of saline and observe for swelling at the insertion site. Also check the extremity behind the insertion site in case the needle has penetrated into and through the posterior cortical bone. Fluid should easily infuse with saline injection from the syringe with no evidence of swelling at the site. • If the test injection is unsuccessful (ie, you observe infiltration/swelling at or near the insertion site), remove the needle and attempt the procedure on *another bone*. If the cortex of the bone is penetrated, placing another needle in the same extremity will permit fluids and drugs to escape from the original hole and infiltrate the soft tissues, potentially causing injury.
6	There are a number of methods to stabilize the needle. Place tape over the flange of the needle to provide support. Position gauze padding on both sides of the needle for additional support.

Step	Action
7	• When connecting IV tubing, tape it to the skin to avoid displacing the needle by placing tension on the tubing.
8	• Volume resuscitation can be delivered via a stopcock attached to extension tubing or by infusion of fluid under pressure. When using a pressurized fluid bag, take care to avoid air embolism. Other methods include the following: • Use a syringe bolus via a medication port in the IV tubing (3-way stopcock not needed). • Attach a saline lock to the IO cannula, and then provide syringe boluses through the lock.
9	Any medication that can be administered by the IV route can be given by the IO route, including vasoactive drug infusions (eg, epinephrine drip). • All medications should be followed with a saline flush.

Figure 34. A, Locations for IO insertion in the distal femur, proximal tibia, and medial malleolus. B, Location for IO insertion in the anterior-superior iliac spine.

A



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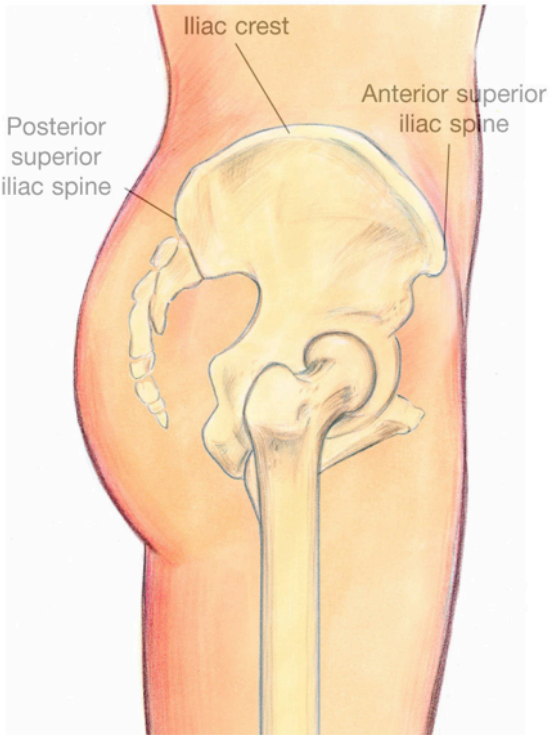


Follow-up

Follow-up is important after you establish IO access. Use these guidelines:

- Check the site frequently for signs of swelling.
- Check the site often for needle displacement. Delivery of fluids or drugs through a displaced needle may cause severe complications (eg, tissue necrosis or compartment syndrome).
- Replace the IO access with vascular access as soon as reasonable. IO needles are intended for short-term use, generally less than 24 hours. Replacement with long-term vascular access is usually done in the intensive care unit.

Acute Coronary Syndromes

ST-Segment Elevation Myocardial Infarction Location and AV Block

Right Ventricular Infarction

Patients with inferior or right ventricular (RV) infarction often present with excess parasympathetic tone. Inappropriate parasympathetic discharge can cause symptomatic bradycardia and hypotension. If hypotension is present, it is usually due to a combination of hypovolemia (decreased left ventricular filling pressure) and bradycardia.

- Administer normal saline (250 to 500 mL) and reassess the patient.
- If there is improvement and no symptoms or signs of heart failure or volume overload, repeat fluid administration (typically up to 1 to 2 L).
- Reassess the patient before *each* fluid administration.
- For patients with RV infarct and hypotension, volume administration may be lifesaving.

When hypotension is present, a slow heart rate is inappropriate. The heart rate should be faster in the presence of low blood pressure. The fluid bolus increases RV filling pressures, which cause an increase in the strength of RV contractions (Starling mechanism), blood flow through the lungs, and, ultimately, left ventricular filling pressure and cardiac output.

AV Block With Inferior Wall Myocardial Infarction

Acute inferior wall myocardial infarction (usually a right coronary artery event) may result in symptomatic second-degree or third-degree AV block with a junctional, narrow-complex escape rhythm. However, if the patient remains asymptomatic and hemodynamically stable, transcutaneous pacing (TCP) and a transvenous pacemaker are not indicated. Monitor the patient and prepare for TCP if high-degree block develops and the patient becomes symptomatic or unstable before expert cardiology evaluation.

- AV block frequently develops from excess vagal tone and AV nodal ischemia. The patient may be stable if junctional pacemaker cells can function and maintain an adequate ventricular rate. This rhythm usually has a narrow-complex QRS and a ventricular rate of 40 to 60/min. Unless a large amount of myocardium is nonfunctional or comorbid conditions exist, the patient is often stable.
- If the bradycardia is symptomatic, follow the Adult Bradycardia Algorithm.
- Prepare for TCP.
- Use atropine to increase heart rate and blood pressure if the patient becomes symptomatic. The initial recommended atropine dose is 1 mg IV bolus. Repeat every 3 to 4 minutes, not exceeding the maximum dose of 3 mg. Use only the dose necessary to stabilize the patient. Excess atropine may increase ischemia by excessively increasing heart rate and contractility—major determinants of myocardial oxygen consumption.
- If there is no response to atropine in an unstable patient, initiate TCP or infusion of chronotropic drug, such as epinephrine (2 to 10 mcg/min) or dopamine (5– to 20 mcg/kg per minute). Titrate to patient response.
- If the patient does not respond to drugs or TCP, start transvenous pacing.

Evaluation of AV block with AMI can be difficult. Obtain immediate expert consultation for evaluation and recommendation (eg, transvenous temporary pacemaker).

Human, Ethical, and Legal Dimensions of ECC and ACLS

Rescuer and Witness Issues

How Often Will CPR, Defibrillation, and ACLS Succeed?

Cardiac arrest occurs both in and out of the hospital. In the United States, more than 565 000 people per year have a cardiac arrest and undergo a resuscitation attempt. The estimated incidence of out-of-hospital cardiac arrest assessed by emergency medical services (EMS) in the United States is about 141 adults per 100 000. Extrapolation of the incidence of in-hospital cardiac arrest reported by Get With The Guidelines®-Resuscitation to the total population of hospitalized patients in the United States suggests that each year, 209 000 people are treated for in-hospital cardiac arrest. Cardiac arrest continues to be an all-too-common cause of premature death, and small, incremental improvements in survival can translate into thousands of lives saved every year.

Many public health experts consider CPR training to be the most successful public health initiative of modern times. Millions of people have prepared themselves to take action to save the life of a fellow human being. But despite our best efforts, in most locations, half or more of out-of-hospital resuscitation attempts do not succeed.

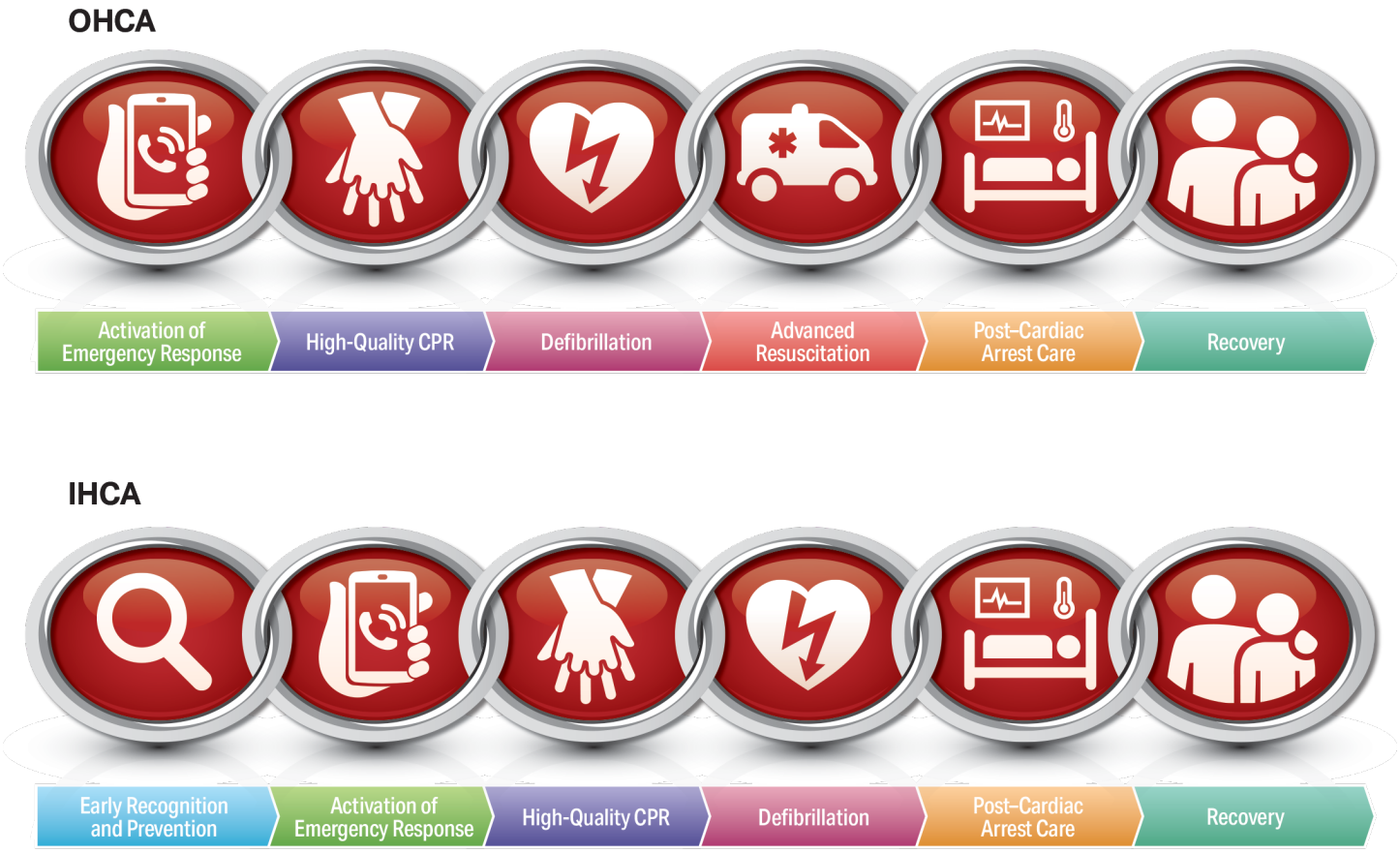
Tragically, even when return of spontaneous circulation occurs, few VF cardiac arrest patients admitted to the emergency department and hospital survive and go home. This means that most CPR attempts will be unsuccessful in terms of neurologically intact survival to hospital discharge. In 2014, survival after bystander-witnessed VF was 38.6% for patients of any age. Survival to hospital discharge after nontraumatic, EMS-treated cardiac arrest with any first recorded rhythm was only 12%. In the hospital setting, only 24.8% of cardiac arrest patients survived to hospital discharge. We must consider and plan for the emotional reactions from rescuers and witnesses to any resuscitation attempt. This is particularly true when their efforts appear to have failed.

Successful resuscitation after cardiac arrest requires an integrated set of coordinated actions represented by the links in the AHA Chain of Survival (Figure 35). The links include the following:

- Immediate recognition of cardiac arrest and activation of the emergency response system
- Early CPR with an emphasis on chest compressions
- Rapid defibrillation
- Effective advanced life support
- Integrated post–cardiac arrest care

Emergency systems that can effectively implement these links can achieve witnessed VF cardiac arrest survival to discharge of almost 50%. In most emergency systems, however, survival is lower, indicating that there is an opportunity for improvement by carefully examining the links and strengthening those that are weak. The individual links are interdependent, and the success of each link is dependent on the effectiveness of those that precede it.

Figure 35. System-specific Chains of Survival.



Take Pride in Your Skills as an ACLS Provider

You should be proud that you are learning to become an ACLS provider. Now, you can be confident that you will be better prepared to do the right thing when your professional skills are needed. Of course, these emergencies can have negative outcomes. You and the other emergency personnel who arrive to help in the resuscitation may not succeed in restoring life. Some people have a cardiac arrest simply because they have reached the end of their lives. Your success will not be measured by whether a cardiac arrest patient lives or dies, but rather by the fact that you tried and worked well together as a team. Simply by taking action, making an effort, and trying to help, you will be judged a success.

Stress Reactions After Resuscitation Attempts

A cardiac arrest is a dramatic and emotional event, especially if the patient is a friend or loved one. The emergency may involve disagreeable physical details, such as bleeding, vomiting, or poor hygiene. The emergency can produce strong emotional reactions in physicians, nurses, bystanders, lay rescuers, and EMS professionals. Failed attempts at resuscitation can impose even more stress on rescuers. This stress can result in a variety of emotional reactions and physical symptoms that may last long after the original emergency.

It is common for a person to experience emotional "aftershocks" following an unpleasant event. Usually, such stress reactions occur immediately or within the first few hours after the event. Sometimes, the emotional response occurs later. These reactions are frequent and normal. There is nothing wrong with you or with someone who has such reactions after an event.

Psychologists working with professional emergency personnel have learned that rescuers may experience grief, anxiety, anger, and guilt. Typical physical reactions include difficulty sleeping, fatigue, irritability, changes in eating habits, and confusion. Many people say they are unable to stop thinking about the event. Remember that these reactions are *common* and *normal*. They do not mean that you are disturbed or weak. Strong reactions simply indicate that this particular event had a powerful impact on you. With the understanding and support of friends and loved ones, the stress reactions usually pass.

Techniques to Reduce Stress in Rescuers and Witnesses

Psychologists tell us that one of the most successful ways to reduce stress after a rescue effort is simple: *talk about it.* Sit down with other people who witnessed the event and talk it over. EMS personnel who respond to calls from lay rescuer defibrillation sites are encouraged to offer emotional support to lay rescuers and bystanders. More formal discussions, called *critical event debriefings*, should include not only the lay rescuers but also the professional responders.

In these discussions, you will be encouraged to describe what happened. Do not be afraid of reliving the event. It is natural and healthy to talk about the event. Describe what went through your mind during the rescue effort. Describe how it made you feel at the time. Describe how you feel now. Be patient with yourself. Understand that many reactions will diminish within a few days. Sharing your thoughts and feelings with your companions at work, fellow rescuers, EMS personnel, or friends will help reduce stress reactions and help you recover.

Other sources of psychological and emotional support are local clergy, police chaplains, fire service chaplains, and hospital and emergency department social workers. Your course instructor may be able to tell you what plans are established for critical event debriefings in your professional setting.

Psychological Barriers to Action

Performance Anxiety

The ACLS Provider Course helps prepare you to respond appropriately to a future emergency. ACLS providers have expressed some common concerns about responding to sudden cardiac emergencies: *Will I be able to take action? Will I remember the steps of the ACLS approach? Will I remember how to perform the skills of CPR, defibrillation, and intubation; the details of drug doses; and the steps in the algorithms? Will I really have what it takes to respond to a true emergency?* Any emergency involving a patient you have grown close to, a friend, or a family member will produce a strong emotional reaction.

Disagreeable Aspects of CPR

What about the unpleasant and disagreeable aspects of performing CPR in either the in-hospital or out-of-hospital setting? Will you really be able to perform mouth-to-mouth rescue breathing on a stranger? What if the patient is bleeding from facial injuries? Would this not pose a risk of disease for a rescuer without a CPR barrier device? CPR and defibrillation require that the rescuer remove clothing from the patient's chest. You

cannot attach defibrillation electrodes unless the pads are placed directly on the skin. The rescuer must open the patient's shirt or blouse and remove the undergarments. Common courtesy and modesty may cause some people to hesitate before removing the clothing of strangers, especially in front of many other people in a public location.

Everyone is familiar with the concept of defibrillation shocks as shown in television shows and movies. These shocks appear painful. Can you overcome your natural tendency not to hurt others, even in an emergency when your actions could be lifesaving? Often, friends and relatives will be at the scene of an emergency. If you respond and take action, these people will look to you to perform quickly, effectively, and confidently.

These barriers can hinder a quick emergency response, especially in settings where such events are rare. There are no easy solutions to help overcome these barriers. Your instructor will encourage you to anticipate many of the scenes described above. The case scenarios will include role-playing and rehearsals. Think through how you would respond when confronted with such a circumstance. Mental practice, even without hands-on practice, may help improve your future performance. The best preparation, however, is frequent practice with manikins in realistic scenarios and situations.

Leaders of all courses that follow the AHA guidelines are aware of the mental and emotional challenge of rescue efforts. You will have support if you ever participate in a resuscitation attempt. You may not know for several days whether the patient lives or dies. If the person you try to resuscitate does not live, take comfort from knowing that, in taking action, you did your best.

Legal and Ethical Issues

The Right Thing to Do

The AHA has supported community CPR training for more than 3 decades. Citizen CPR responders have helped save thousands of lives. The AHA believes that training in the use of CPR and AEDs will dramatically increase the number of survivors of cardiac arrest.

Studies of lay rescuer AED programs in airports and casinos and of first-responder programs with police officers have shown survival rates of 41% to 74% from out-of-hospital witnessed VF SCA when immediate bystander CPR is provided and defibrillation occurred within 3 to 5 minutes of collapse. Other studies have demonstrated decreased time intervals from collapse to delivery of the first shock when AEDs were used during adult out-of-hospital cardiac arrest. However, if no decrease in time to defibrillation is achieved, then high survival rates are not observed.

Anyone can perform emergency CPR without fear of legal action.

Chest compressions and rescue breathing require direct physical contact between rescuer and patient. Often, these 2 people are strangers. Too often, the arrest patient dies. In the United States, people may take legal action when they think that one person has harmed another, even unintentionally. Despite this legal environment, CPR remains widely used and remarkably free of legal issues and lawsuits. Although attorneys have included rescuers who performed CPR in lawsuits, no Good Samaritan has ever been found guilty of doing harm while performing CPR.

All 50 states have Good Samaritan laws that grant immunity to any volunteer or lay rescuer who attempts CPR in an honest, good faith effort to save a life. A person is considered a Good Samaritan if the following apply:

- The person is genuinely trying to help.
- The help is reasonable (you cannot engage in gross misconduct, ie, actions that a reasonable person with your training would never do).
- The rescue effort is voluntary and not part of the person's job requirements.

Most Good Samaritan laws protect laypersons who perform CPR even if they have had no formal training. The purpose of this protection is to encourage broad awareness of resuscitative techniques and to remove a barrier to involving more people. As the details of these laws may vary across states, please check with your state legislation agency for further information.

Principle of Futility

If the purpose of medical treatment cannot be achieved, it is considered *futile*. The key determinants of medical futility are length and quality of life. An intervention that cannot establish any increase in length or quality of life is futile. An objective criterion for medical futility was defined in 1990 for interventions and drug therapy as imparting a less than 1% chance of survival. Although this criterion may be controversial, it remains a basis for current futility research.

Patients or families may ask physicians to provide care that is inappropriate. However, physicians have no obligation to provide such care when there is scientific and social consensus that the treatment is ineffective. An obvious example of an inappropriate or futile intervention is CPR for patients with signs of irreversible death. Other healthcare providers also have no obligation to provide CPR or ACLS if no benefit can be expected (ie, CPR would not restore effective circulation) and/or in the presence of advance directives, including do not attempt resuscitation (DNAR) orders or living wills. In the absence of these clinical circumstances and personal desires, healthcare providers should attempt resuscitation.

A careful balance of the patient's prognosis for both length and quality of life will determine whether CPR is appropriate. CPR is inappropriate when survival is not expected.

When the likelihood of survival is borderline, or when the likelihood of morbidity and burden to the patient is relatively high, rescuers should support the patient's desires. If the patient's desires are unknown, healthcare providers may follow the preferences of the legally authorized surrogate decision maker. Noninitiation of resuscitation and discontinuation of life-sustaining treatment during or after resuscitation are ethically equivalent. When the patient's prognosis is uncertain, consider a trial of treatment while gathering more information to determine the likelihood of survival and the expected clinical course.

Terminating Resuscitative Efforts

The decision to stop resuscitative efforts rests with the treating physician in the hospital. The physician bases this decision on many factors, including time to CPR, time to defibrillation, comorbid disease, prearrest state, initial arrest rhythm, clinical factors, and intra-arrest physiologic parameters. None of these factors alone is clearly predictive of outcome. The chance of discharge from the hospital alive and neurologically intact may diminish as resuscitation time increases but must be considered in context with the other factors mentioned.

The responsible clinician should stop the resuscitation when they determine with a high degree of certainty that the patient will not respond to further ACLS efforts.

It is important to consider the circumstances of the cardiac arrest when deciding whether to continue resuscitative efforts. Resuscitation efforts may be prolonged beyond what would be appropriate for prolonged asystole in the following conditions:

- Young patient
- Presence of toxins or electrolytes abnormalities
- Profound hypothermia
- Victims of cold water submersion
- Therapeutic or illicit drug overdose
- Suicide attempt
- Nearby family member or loved ones expressing opposition to stopping efforts

Delayed or token efforts such as so-called *slow codes* (knowingly providing ineffective resuscitative efforts) are inappropriate. This practice compromises the ethical integrity of healthcare providers, uses deception to create a false impression, and may undermine the provider-patient relationship. The practice of "pseudo-resuscitation" was self-reported by paramedics to occur in 27% of cardiac arrests in a community where a termination-of-resuscitation protocol was not in place.

When Not to Start CPR

Few criteria can accurately predict the futility of CPR. In light of this uncertainty, all patients in cardiac arrest should receive resuscitation unless any of the following apply:

- The patient has a valid DNAR order.
- The patient has signs of irreversible death (eg, rigor mortis, decapitation, decomposition, or dependent lividity).
- No physiologic benefit can be expected because vital functions have deteriorated despite maximal therapy (eg, progressive septic or cardiogenic shock).

Withholding vs Withdrawing CPR

Basic life support (BLS) training urges the first lay responder at a cardiac arrest to begin CPR. Healthcare providers are expected to provide BLS and ACLS as part of their duty to respond. There are a few exceptions to this rule:

- A person lies dead with obvious clinical signs of irreversible death (eg, rigor mortis, dependent lividity, decapitation, or decomposition).
- Attempts to perform CPR would place the rescuer at risk of physical injury.
- The patient or surrogate has indicated that resuscitation is not desired with an advance directive (DNAR order).

- No physiologic benefit can be expected because vital functions have deteriorated despite maximal therapy (eg, progressive sepsis or cardiogenic shock).

No rescuer should make a judgment about the present or future quality of life of a patient of cardiac arrest on the basis of current (ie, during the attempted resuscitation) or anticipated neurologic status. Such snap judgments are often inaccurate. Conditions such as irreversible brain damage or brain death cannot be reliably assessed or predicted during an emergency.

Out-of-hospital DNAR protocols must be clear to all involved (eg, physicians, patients, family members, loved ones, and out-of-hospital healthcare providers). Advance directives can take many forms (eg, written bedside orders from physicians, wallet identification cards, and identification bracelets).

The ideal EMS DNAR form is portable in case the patient is transferred. In addition to including out-of-hospital DNAR orders, the form should provide direction to EMS about initiating or continuing life-sustaining interventions for the patient who is not pulseless and apneic.

Withdrawal of Life Support

Withdrawal of life support is an emotionally complex decision for family and staff. Withholding and withdrawing life support are ethically similar. The decision to withdraw life support is justifiable when it is determined that the patient is dead, if the physician and patient or surrogate agree that treatment goals cannot be met, or the burden to the patient of continued treatment would exceed any benefits.

Patients who are unconscious or unresponsive after cardiac arrest should be directed to an inpatient critical-care facility with a comprehensive care plan that includes acute cardiovascular interventions, use of targeted temperature management (TTM), standardized medical goal-directed therapies, and advanced neurological monitoring and care. The earliest time for prognostication in patients treated with TTM by using clinical examination where sedation and paralysis could be a confounder may be 72 hours after return to normothermia. In patients not treated with TTM, the earliest time to prognosticate a poor neurologic outcome by using clinical examination is 72 hours after cardiac arrest. This time can even be longer if the residual effect of sedation or paralysis confounds the clinical examination. Many initially comatose survivors of cardiac arrest have the potential for full recovery such that they are able to lead normal lives. Between 20% and 50% (or more) of survivors of out-of-hospital cardiac arrest who are comatose on arrival at the hospital may have good 1-year neurological outcome. Therefore, it is important to place patients in a hospital critical-care unit where expert care and neurological evaluation can be performed and where appropriate testing to aid prognosis is available and performed in a timely manner.

The following 3 factors are associated with poor outcome:

- Absence of pupillary response to light on the third day
- Absence of motor response to pain on the third day
- Bilateral absence of cortical response to median somatosensory evoked potentials when used in normothermic patients who are comatose for at least 72 hours after the cardiac arrest and resuscitation

Withdrawal of life support is ethically permissible under these circumstances.

Patients in the end stage of an incurable disease, whether responsive or unresponsive, should receive care that ensures their comfort and dignity. The goal of such care is to minimize the suffering associated with pain, dyspnea, delirium, convulsions, and other terminal complications. It is ethically acceptable to gradually increase the dose of narcotics and sedatives to relieve pain and other symptoms, even to levels that might shorten the patient's life. Nursing and comfort care (eg, oral hygiene, skin care, patient positioning, and measures to relieve pain and suffering) should be continued.

Advance Directives, Living Wills, and Patient Self-Determination

An *advance directive* is any expression of a person's thoughts, wishes, or preferences for their end-of-life care. Advance directives can be based on conversations, written directives, living wills, or durable powers of attorney for health care. The legal validity of various forms of advance directives varies from jurisdiction to jurisdiction. Courts consider written advance directives to be more trustworthy than recollections of conversations.

A *living will* provides written direction to physicians about medical care the patient would approve if they become terminally ill and unable to make decisions. A living will constitutes clear evidence of the patient's wishes and can be legally enforced in most areas.

Patients should periodically reevaluate their living wills and advance directives. Desires and medical conditions may change over time. In the United States, the Patient Self-Determination Act of 1991 requires healthcare institutions and managed-care organizations to ask if patients have advance directives. Healthcare institutions are required to facilitate the completion of advance directives if patients request them.

Out-of-Hospital DNAR Orders

In many settings, *Allow Natural Death* is becoming a preferred term to replace DNAR, to emphasize that the order is to allow natural consequences of a disease or injury, and to emphasize ongoing end-of-life care.

Many patients for whom EMS is called because of cardiac arrest are chronically ill, have a terminal illness, or have a written advance directive (DNAR order). Countries, individual states in the United States, and individual jurisdictions worldwide have different laws for out-of-hospital DNAR orders and advance directives. Even if a patient has a DNAR order, it may be difficult to determine whether to start resuscitation. It is especially difficult if family members have differing opinions. You should initiate CPR and ACLS if you have reason to believe that

- There is reasonable doubt about the validity of a DNAR order or advance directive
- The patient may have changed their mind
- The best interests of the patient are in question

Sometimes, within a few minutes of initiation of CPR, relatives or other medical personnel arrive and confirm that the patient had clearly expressed a wish that healthcare providers not attempt resuscitation. CPR or other life support measures may be discontinued, with approval of medical direction, when further information becomes available.

Some EMS systems in the United States have extended the DNAR protocol to include verbal requests from family members as grounds to withhold therapy from cardiac arrest patients with a history of a terminal illness and under the care of a physician.

The number of patients for whom resuscitation was withheld doubled after implementation of this law from 45 to 99 patients a year. This is an important step in expanding the clinical decision rule pertaining to when to start resuscitation in out-of-hospital cardiac arrest.

When you cannot obtain clear information about the patient's wishes, you should initiate resuscitative measures.

EMS No-CPR Programs

In the United States, a number of states have adopted "no-CPR" programs. These programs allow patients and family members to call EMS for emergency care, support, and treatment for end-of-life distress (ie, shortness of breath, bleeding, or uncontrolled pain). Patients do not have to fear unwanted resuscitative efforts.

In a no-CPR program, the patient, who usually has a terminal illness, signs a document requesting "no heroics" if there is a loss of pulse or if breathing stops. In some states, the patient must wear a no-CPR identification bracelet. In an emergency, the bracelet or other documentation signals rescuers that CPR efforts, including use of an AED, are not recommended.

If an ACLS provider arrives at the side of a person in apparent cardiac arrest (unresponsive, no pulse, no breathing) and sees that the person is wearing a no-CPR bracelet (or has some other indication of no-CPR status), the provider should respect the person's wishes. Report the problem as a "collapsed, unresponsive person wearing a no-CPR bracelet." State that you think CPR should not be performed.

Check with your state or ask your instructor to see what the law is in your jurisdiction regarding no-CPR orders in the out-of-hospital setting.

Legal Aspects of AED Use

Defibrillators, including many AEDs, are restricted medical devices. In the United States, most states have legislation that requires a physician to authorize the use of restricted medical devices. Lay rescuer CPR and defibrillation programs that make AEDs available to lay rescuers (and, in some cases, EMS providers) may be required to have a medical authority or a healthcare provider who oversees the purchase of AEDs, treatment protocols, training, and contact with EMS providers. In a sense, the medical authority prescribes the AED for use by the lay responder and therefore complies with medical regulations.

In the United States, malpractice accusations and product liability lawsuits increase every year. In the past, fear of malpractice suits hindered innovative programs to bring early CPR and early defibrillation into every community, but such fears have proven unfounded.

To solve this problem of fear of litigation, all states have changed existing laws and regulations to provide limited immunity for lay rescuers who use AEDs in the course of attempting resuscitation. These are the Good Samaritan laws. Many states have amended Good Samaritan laws to include the use of AEDs by lay rescuers.

This means that the legal system will consider lay rescuers to be Good Samaritans when they attempt CPR and defibrillation for someone in cardiac arrest. As a Good Samaritan, you cannot be successfully sued for any harm or damage that occurs during the rescue effort (except in cases of gross negligence).

In 2006, the AHA published a statement detailing recommended legislation to promote lay rescuer AED programs and to assist legislators and policymakers in removing impediments to these programs. You can find this statement at **http://circ.ahajournals.org/content/early/2006/01/16/CIRCULATIONAHA.106.172289.full.pdf**.

Lay rescuer CPR and AED programs should implement processes of continuous quality improvement, including evaluation of the following:

- Performance of the emergency response plan, including accurate time intervals for key interventions (such as collapse to shock or no shock advisory to initiation of CPR) and patient outcome
- Responder performance
- AED function
- Battery status and function
- Electrode pad function and readiness, including expiration date

Providing Emotional Support for the Family

Conveying News of a Sudden Death to Family Members

- Before talking to the family, obtain as much information as possible about the patient and the circumstances surrounding the death. Be ready to refer to the patient by name.
- Call the family if they have not been notified. Explain that their loved one has been admitted to the emergency department or critical care unit and that the situation is serious. If possible, family members should be told of the death in person, not over the telephone.
- When family members arrive, ask someone to take them to a private area. Walk in, introduce yourself, and sit down. Address the closest relative. Maintain eye contact and position yourself at the same level as family members (ie, sitting or standing).
- Enlist the aid of a social worker or a member of the clergy if possible.
- Briefly describe the circumstances leading to the death. Summarize the sequence of events. Avoid euphemisms such as “he’s passed on,” “she’s no longer with us,” or “he’s left us.” Instead, use the words “death,” “dying,” or “dead.”
- Allow time for family members to process the information. Make eye contact and touch. Convey your feelings with a simple phrase such as “You have my (our) sincere sympathy.”
- Determine the patient’s suitability for and wishes about tissue donation (use driver’s license and patient records). Follow local protocols on when to discuss with family. Consent for donation should be requested by a trained individual who is not part of the care team. (See Organ and Tissue Donation for more information.)
- Allow as much time as necessary for questions and discussion. Review the events several times if needed.

- Allow family members the opportunity to see the patient. Prepare the family for what they will see. If equipment is still connected to the patient, tell the family. Equipment must be left in place for coroner's cases or when an autopsy is performed.
- Determine in advance what happens next and who will sign the death certificate. Physicians may impose burdens on staff and family if they fail to understand policies about death certification and disposition of the body.
- Offer to contact the patient's attending or family physician and to be available if there are further questions. Arrange for follow-up and continued support during the grieving period.

Family Presence During Resuscitation

According to surveys in the United States and the United Kingdom, most family members state that they would like to be present during the attempted resuscitation of a loved one. Parents and care providers of chronically ill patients are often knowledgeable about and comfortable with medical equipment and emergency procedures.

Even family members with no medical background report that it is comforting to be at the side of a loved one and say goodbye during the final moments of life. These are those who choose to be at the bedside and who have a designated support person with them to answer questions, clarify information, and comfort the family.

Family members often do not ask if they can be present, but healthcare providers should offer the opportunity whenever possible.

Relatives and friends who are present and are provided counseling during the attempted resuscitation of a loved one report fewer incidences of posttraumatic avoidance behaviors, fewer grieving symptoms, and less intrusive imagery.

When family members are present during resuscitative efforts, sensitivity is heightened among resuscitation team members. A team member who is knowledgeable about resuscitation practices should be available to answer questions, provide comfort, and help the family during the resuscitation. Even when the resuscitation outcome is not optimal, families feel comforted to know they can be present to say goodbye, give comfort to their dying loved one, and begin the grieving process.

Organ and Tissue Donation

Most communities do not optimize the retrieval of organ and tissue donations. This has created protracted waiting and greater suffering for patients awaiting organ transplantation. The ECC community of the AHA supports efforts to optimize the ethical acquisition of organ and tissue donations. Studies suggest no difference in functional outcomes of organs transplanted from patients who are determined to be brain dead as a consequence of cardiac arrest when compared with donors who are brain dead from other causes. Therefore, it is reasonable to suggest that all communities should optimize retrieval of tissues and organ donations in brain-dead post–cardiac arrest patients in-hospital and those pronounced dead in the out-of-hospital setting.

Most important to this process are advanced planning and infrastructure support to allow organ donation to occur in a manner sensitive to the needs of the donor's family and without undue burden on the staff. Medical directors of EMS agencies, emergency departments, and critical care units should develop protocols and

implementation plans with the regional organ and tissue donation program to optimize donation after a cardiac arrest death. These include

- A process by which permission for organ and tissue donations will be obtained for in- and out-of-hospital settings
- The establishment of clearly defined guidelines for organ and tissue procurement that will be available to all healthcare providers both in- and out-of-hospital
- Availability of information to address the possible difference between applicable laws and social values in procedures for organ procurement
- The emotional support to be offered to providers after the event