American Heart Association®

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ACLS Provider Manual Supplementary Material

Contents

Airway Management

Part 1—Basic Airway Management

Devices to Provide Supplementary Oxygen

Overview

Oxygen administration is always appropriate for patients with acute cardiac disease or pulmonary distress. Various devices can deliver supplementary oxygen from 21% to 100% (Table 1).This section describes 4 devices to provide supplementary oxygen:

- Oxygen supply (cylinder or wall unit)
- Nasal cannula
- Face mask
- Venturi mask

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

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

Device	Flow Rates	Delivered O_2*
Nasal cannula	1 L/min	21%-24%
	2 L/min	25%-28%
	3 L/min	29%-32%
	4 L/min	33%-36%
	5 L/min	37%-40%
	6 L/min	41%-44%
Simple oxygen face mask	6-10 L/min	35%-60%
Face mask with O_2 reservoir (nonrebreathing mask)	6 L/min	60%
	7 L/min	70%
	8 L/min	80%
	9 L/min	90%
	10-15 L/min	95%-100%
Venturi mask	4-8 L/min	24%-40%
	10-12 L/min	40%-50%

*Percentage is 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:

- Valve handles to open the cylinder, pressure gauge, and flow meter
- Tubing connecting the oxygen supply to the patient's oxygen administration device

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Nasal Cannula

The nasal cannula (Figure 1) is a low-flow oxygen administration system designed to add oxygen to room air when the patient inspires.

- A nasal cannula provides up to 44% oxygen.
- In this low-flow system, inspired air mixes with room air. The ultimate inspired oxygen concentration is determined by the oxygen flow rate through the cannula and how deeply the patient breathes (tidal volume).
- Increasing the oxygen flow by 1 L/min (starting with 1 L/min) will increase the inspired oxygen concentration by approximately 4%:
  - 1 L/min: 21% to 24%
  - 2 L/min: 25% to 28%
  - 3 L/min: 29% to 32%
  - 4 L/min: 33% to 36%
  - 5 L/min: 37% to 40%
  - 6 L/min: 41% to 44%

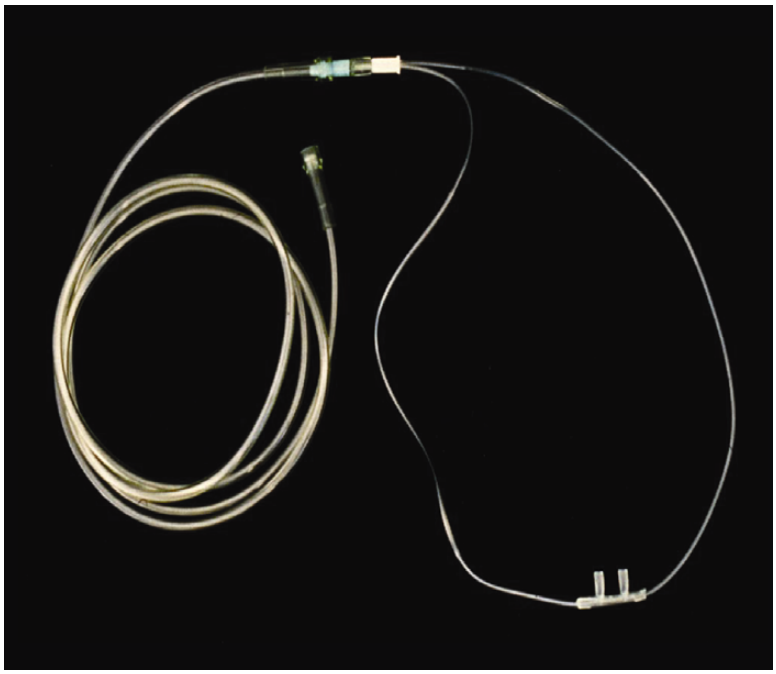


Figure 1. A nasal cannula used for supplementary oxygen delivery in spontaneously breathing patients.

Face Mask

A simple face mask delivers low oxygen flow to the patient's nose and mouth. A partial rebreathing mask consists of a face mask with an attached reservoir bag (Figure 2.)

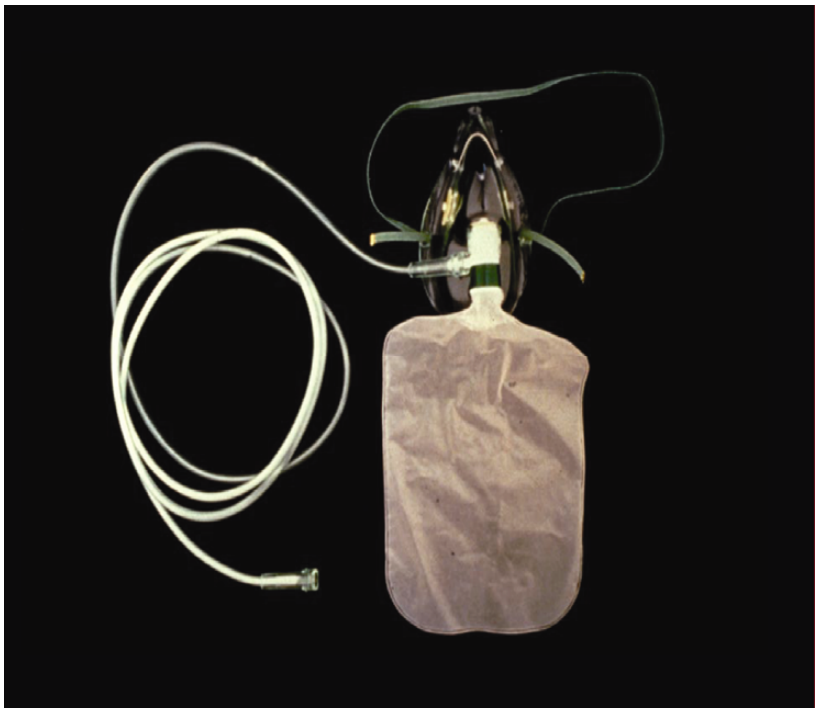


Figure 2. A face mask with oxygen reservoir used for supplementary oxygen delivery in spontaneously breathing patients.

A face mask can supply up to 60% oxygen with flow rates of 6 to 10 L/min (Table 1). A face mask with oxygen reservoir (nonrebreathing mask) provides up to 90% to 100% oxygen with flow rates of 9 to 15 L/min. In this system a constant flow of oxygen enters an attached reservoir.

Use a face mask with a reservoir for patients who

- Are seriously ill, responsive, and have adequate ventilation but require high oxygen concentrations
- May avoid endotracheal intubation if acute interventions produce a rapid clinical effect (eg, patients with acute pulmonary edema, chronic obstructive pulmonary disease [COPD], or severe asthma)
- Have relative indications for endotracheal intubation but maintain an intact gag reflex
- Have relative indications for intubation but have clenched teeth or other physical barriers to immediate intubation

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

Venturi Mask

A Venturi mask enables a more reliable and controlled delivery of oxygen concentrations from 24% to 50%. Use the Venturi mask for patients who retain carbon dioxide (CO_2). Patients who have chronic high levels of CO_2 in

their blood and moderate-to-severe hypoxemia may develop respiratory depression if the drive stimulating them to breathe (oxygen) is reduced.

- A Venturi mask can accurately control the inspired oxygen concentration. Use this mask in patients with COPD, who usually have chronic hypercarbia (high CO_2) and mild to moderate hypoxemia.
- Administration of high oxygen concentrations to patients with end-stage 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 ventilation, support ventilation.

Delivered oxygen concentrations can be adjusted to 24%, 28%, 35%, and 40% using a flow rate of 4-8 L/min and 40% to 50% using a flow rate of 10-12 L/min. Observe the patient closely for respiratory depression. Use a pulse oximeter to quickly titrate to the preferred level of oxygen administration.

Bag-Mask Ventilation

Overview

The bag-mask device, which typically consists of a self-inflating bag and a nonrebreathing valve, may be used with a face mask or an advanced airway (Figure 3). Bag-mask ventilation is a challenging skill that requires considerable practice for competency. 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 complications.

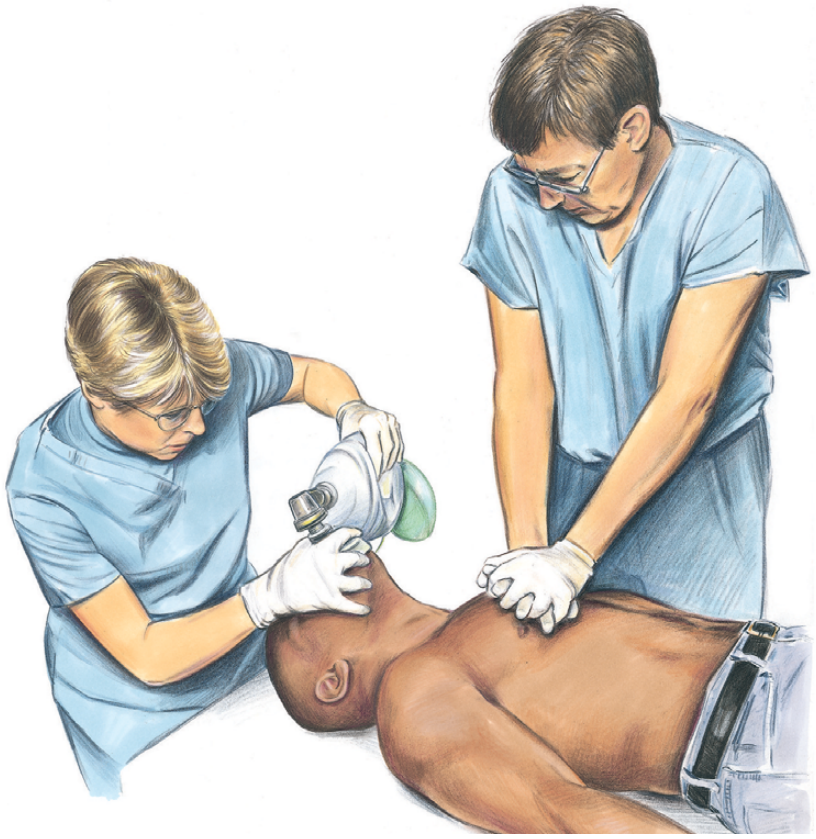


Figure 3. Bag-mask device with 2-rescuer CPR.

Use With an Advanced Airway

Advanced airway devices such as the laryngeal mask airway (LMA) and esophageal-tracheal Combitube are currently within the scope of EMS providers in several regions (with specific authorization from medical control). These devices are acceptable alternatives to bag-mask devices when used by healthcare providers who are well trained and have sufficient experience to use them. It is not clear that these devices are any more or less complicated to use than a pocket mask; training is needed for safe and effective use of both a bag-mask device and each of the advanced airways.

Tips for Performing Bag-Mask Ventilation

- Insert an oropharyngeal airway (OPA) as soon as possible if the patient has no cough or gag reflex to help maintain the airway.
- There is no specific tidal volume recommended for adults. Instead the tidal volume should be sufficient to achieve visible chest rise.
- Many healthcare providers cannot create a leakproof seal between the mask and face using 1 hand. The hand holding the mask must perform 2 tasks simultaneously: perform a head tilt and press the mask against the face while lifting the jaw. Perform and maintain a head tilt, and then use the thumb and index finger to make a “C,” pressing the edges of the mask to the face. Next use the remaining fingers to lift the angle of the jaw and open the airway (Figure 4A).
- For these reasons many experts recommend that 2 well-trained, experienced healthcare providers work together during bag-mask

ventilation. One provider should hold the mask with 2 hands, creating a leakproof seal between the mask and the face while lifting the patient's jaw. The second provider squeezes the bag slowly and gently over 1 second per ventilation (Figure 4B).

- These seal and volume problems do not occur when the bag-mask device is attached to the end of an advanced airway device (eg, endotracheal tube [ETT], Combitube, or LMA).

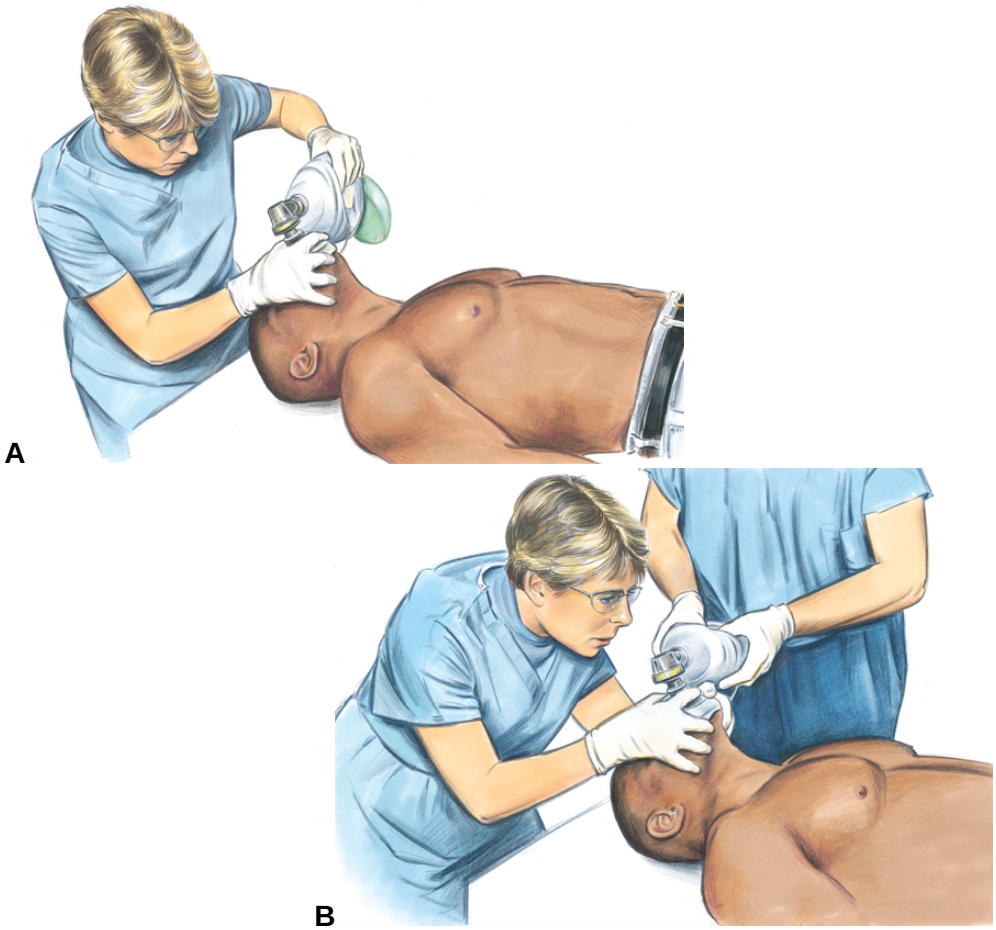


Figure 4. A, Mouth-to-mask E-C clamp technique of holding mask while lifting the jaw. Position yourself at the patient's head. Circle the thumb and first finger around the top of the mask (forming a "C") while using the third, fourth, and fifth fingers (forming an "E") to lift the jaw. **B,** Two-rescuer use of the bag mask. 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.

Ventilation With an Advanced Airway and Chest Compressions

When the patient has an advanced airway in place during CPR, 2 rescuers no longer deliver cycles of CPR (ie, compressions interrupted by pauses for ventilation).

- Chest compressions are delivered at a rate of 100 per minute.
- The provider delivering ventilations delivers 1 ventilation every 6 to 8 seconds (8 to 10 per minute).
- Providers should switch roles every 2 minutes to prevent compressor fatigue and deterioration in the quality and rate of chest compressions.
- Minimize interruptions in chest compressions.
- Avoid excessive ventilation (too many breaths or too large a volume).

Advanced Airway Adjuncts: Combitube

Overview

The Combitube (Figure 5) is an advanced airway that is an acceptable alternative to the use of an ETT. The Combitube is an invasive airway device with 2 inflatable balloon cuffs. It is inserted without visualization of the vocal cords. The tube is more likely to enter the esophagus than the trachea. When the tube does enter the esophagus, ventilation occurs through side openings 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 Combitube and deliver ventilation comparable to that achieved with endotracheal intubation. The advantages of the Combitube are chiefly those related to ease of training. But only providers trained and experienced with the use of the Combitube should insert the device because fatal complications are possible.

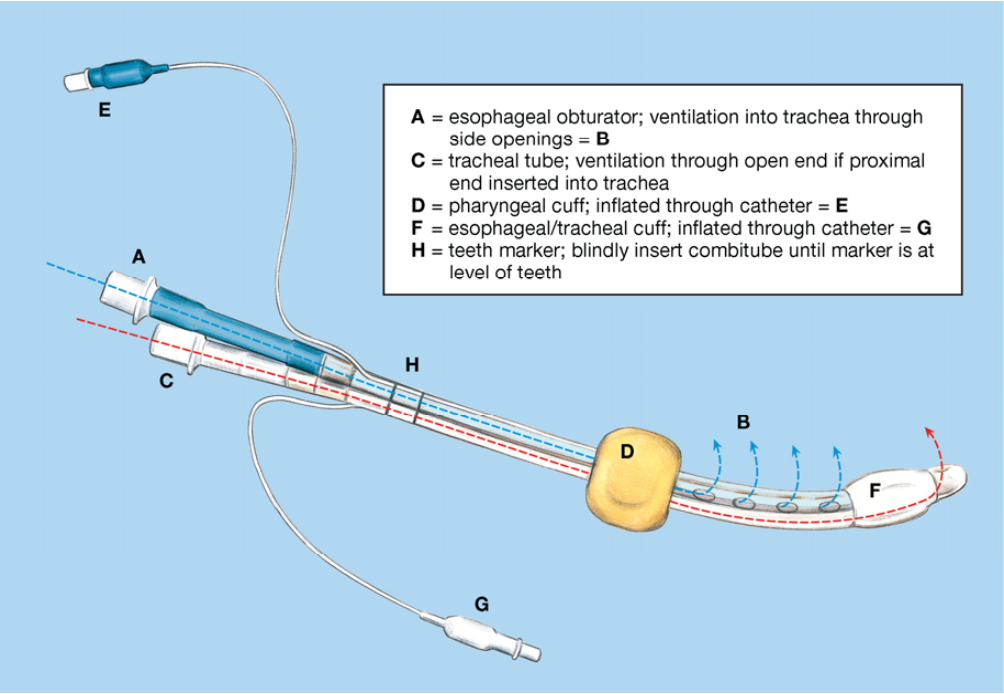


Figure 5. Esophageal-tracheal Combitube

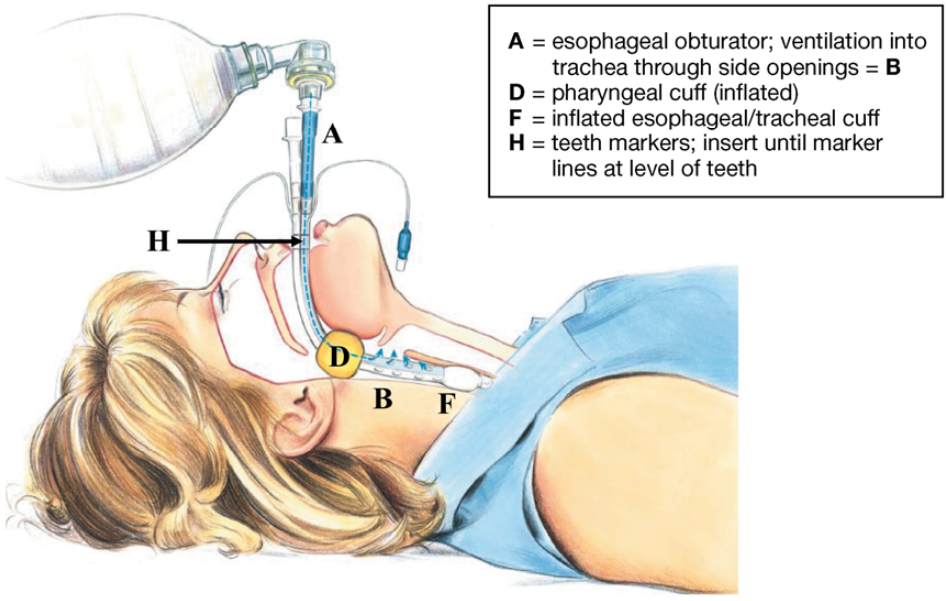


Figure 6. Esophageal-tracheal Combitube inserted in esophagus.

Insertion of the Combitube

The steps for blind insertion of the Combitube are as follows:

Step	Action
1	*Equipment preparation:* Check the integrity of both cuffs according to the manufacturer's instructions and lubricate the tube.
2	*Patient preparation:* Provide oxygenation and ventilation, sedate as clinically indicated, and position the patient. Rule out the following contraindications to insertion of the Combitube (according to the manufacturer's instructions): • Age younger than 16 years or height less than manufacturer's recommendation for adult and small adult sizes. • Gag reflex present • Known or suspected esophageal disease • Ingestion of a caustic substance

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 on the tube (Figure 5 H) are positioned between the patient's teeth. (Do not force, and do not attempt for more than 30 seconds.) • Inflate the proximal/pharyngeal (blue) cuff with 100 mL of air. (Inflate with 85 mL for the smaller Combitube.) Then inflate the distal (white or clear) cuff with 15 mL of air. (Inflate with 12 mL for the smaller Combitube.)
4	Confirm tube location and select the lumen for ventilation. To select the appropriate lumen to use 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:* Breath sounds should be present bilaterally with no epigastric sounds. Provide ventilation through the blue (proximal/pharyngeal) lumen. This action delivers ventilation through the pharyngeal side holes between the 2 cuffs, and air will enter the trachea. Because the tip of the tube rests in the esophagus, do not use the distal (white or clear) tube for ventilation. The distal cuff will also lie within the esophagus; inflation of this cuff prevents the ventilations that you deliver through the pharyngeal tube from entering the esophagus. • *Tracheal placement:* Breath sounds are absent and epigastric sounds are present when you attempt to provide ventilation through the blue (proximal/pharyngeal) lumen. Immediately stop providing ventilations through the blue lumen and provide them through the distal (white or clear) lumen that opens at the tip of the tube in the trachea. With endotracheal placement of the tube, the distal cuff performs the same function as a cuff on an ETT. Detection of exhaled CO_2 (through the ventilating white or clear lumen) should be used for confirmation, particularly if the patient has a perfusing rhythm. • *Unknown placement:* Breath sounds and epigastric sounds are absent. Deflate both cuffs and withdraw the tube slightly, reinflate the blue cuff, and then reinflate the white (or clear) cuff (see steps above). If breath sounds and epigastric sounds are still absent, remove the tube.
5	Insert a bite-block, provide ventilation, and continue to monitor the patient's condition and the position of the Combitube. A bite-block reduces the possibility of airway obstruction and tube damage. Keep the bite-block in place until you remove the Combitube.

Advanced Airway Adjuncts: Laryngeal Mask Airway

Overview

The LMA (Figure 7) is an advanced airway device that is considered an acceptable alternative to the ETT. The LMA is composed of a tube with a cuffed mask-like projection at the end of the tube.

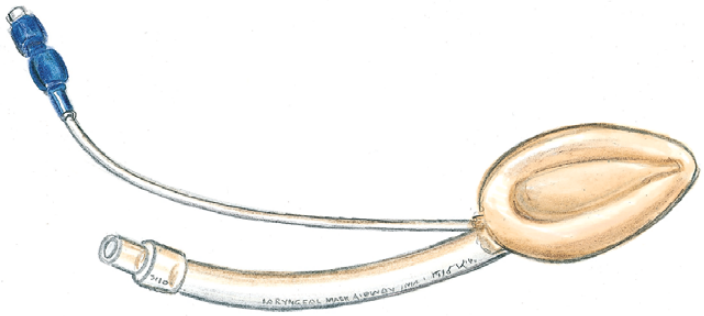
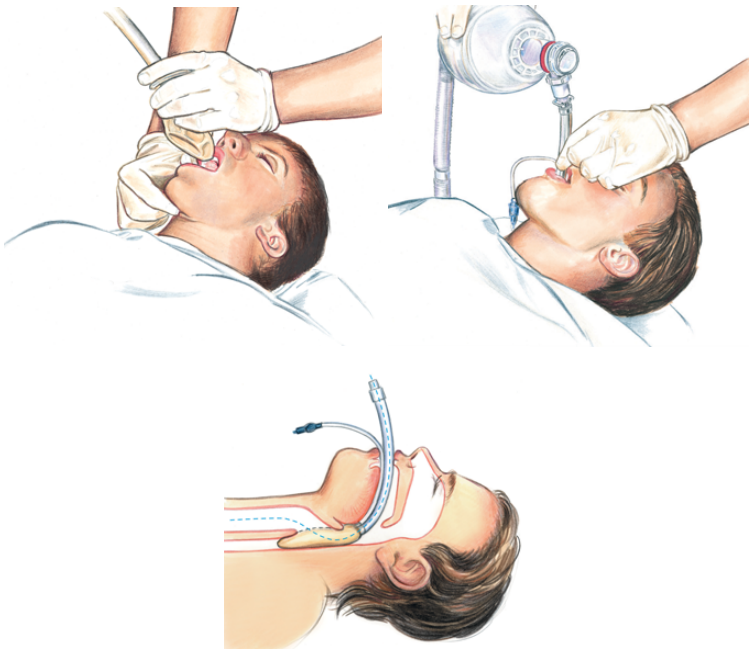


Figure 7. Laryngeal mask airway (LMA).

Insertion of the Laryngeal Mask Airway

The steps for blind insertion of the LMA (Figure 8) are as follows:

Step	Action
1	*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.
2	*Patient preparation:* Provide oxygenation and ventilation, sedate as indicated, and position the patient. Note that use of the LMA poses risks of regurgitation and aspiration in unresponsive patients. You must weigh these risks against the benefit of establishing an airway using this specific device.

3	*Insertion technique (Figure 8):* • Introduce the LMA 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 LMA. • Never overinflate the cuff after inflation. Excessive intracuff pressure can result in misplacement of the device. It also can cause pharyngolaryngeal injury (eg, sore throat, dysphagia, or nerve injury).
4	Insert a bite-block, provide ventilation, and continue to monitor the patient's condition and the position of the LMA. A bite-block reduces the possibility of airway obstruction and tube damage. Keep the bite-block in place until you remove the LMA.  **Figure 8.** Insertion of the laryngeal mask airway (LMA).

Endotracheal Intubation

Overview

Placement of an endotracheal tube (ETT), or endotracheal intubation, provides advanced airway management. The ETT

- Keeps the airway patent
- Enables delivery of a high concentration of oxygen
- facilitates delivery of a selected tidal volume to maintain adequate lung inflation
- 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
- Provides an alternative route for administration of resuscitation medications when intravenous (IV) or intraosseous (IO) access cannot be obtained. These medications are atropine, vasopressin, epinephrine, and lidocaine.. Note however that drug delivery and drug effects following endotracheal administration are less predictable than those delivered by the IV/IO route.

The Combitube and LMA are now considered acceptable alternatives to the ETT for advanced airway management.

Misplacement of an ETT can result in severe, even fatal, complications. For this reason only skilled, experienced personnel should perform endotracheal 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 the following criteria:

- Personnel are well trained.
- Personnel perform intubation frequently.
- Personnel receive frequent refresher training in this skill.
- ETT placement is included in the scope of practice defined by governmental regulations.

and

- Personnel participate in a process of continuous quality improvement to detect frequency of complications and minimize those complications.

Placement of an ETT 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, delivering defibrillation as needed and establishing IV/IO access.

Technique of Endotracheal 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 endotracheal intubation and the steps involved. Team members may assist with endotracheal intubation and must know how to integrate compressions and ventilations when an ETT is placed. This knowledge is often more important than knowing how to perform the procedure itself.

All ACLS providers must understand the following:

- When to intubate
- How to confirm successful tube placement
- How to integrate chest compressions and ventilations
- How to prevent and recognize tube dislodgment
- How to verify and monitor effective oxygenation and ventilation

Indications for Endotracheal Intubation

- Cardiac arrest when bag-mask ventilation is not possible or is ineffective
- Responsive patient in respiratory compromise is unable to oxygenate adequately despite noninvasive ventilatory measures
- Patient is unable to protect airway (eg, coma, areflexia, or cardiac arrest)

Cricoid Pressure Maneuver

During endotracheal intubation *in adults receiving CPR,* a third healthcare provider not involved in compressions or ventilations should apply cricoid pressure (Figure 9). This maneuver may protect against regurgitation of gastric contents and helps ensure tube placement in the tracheal orifice. This provider should maintain cricoid pressure until the ETT is inserted, the cuff of the ETT is inflated, and proper tube position is confirmed.

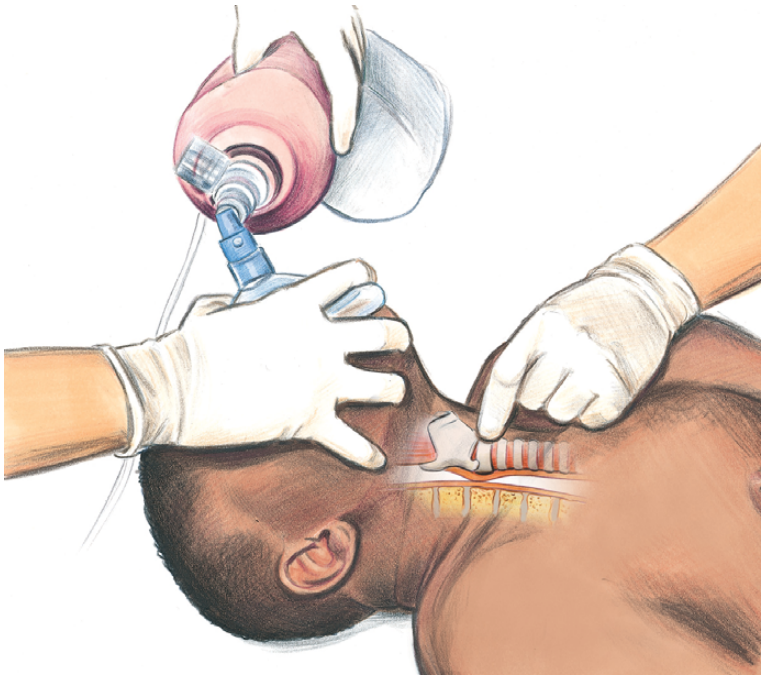


Figure 9. Cricoid pressure.

The steps of the cricoid pressure maneuver are as follows:

Step	Action
1	Find the prominent thyroid cartilage (Adam's apple).
2	Find the soft depression below the thyroid cartilage (cricothyroid membrane).
3	Find the hard prominence just below the depression (cricoid cartilage).
4	Apply firm pressure while pinching with the thumb and index finger while applying firm pressure toward the patient's back and somewhat toward the head. This action presses the trachea back against the esophagus, compressing the esophagus. Cricoid pressure facilitates intubation because it pushes the tracheal orifice into the visual field of the person performing intubation.
5	Release pressure *only* when proper tube placement is confirmed and the cuff is inflated or when instructed to do so by the person performing intubation.

Ventilating With an ETT in Place During Chest Compressions

During cardiac or respiratory 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 8 to 10 breaths per minute (approximately 1 breath every 6 to 8 seconds) when delivering ventilation during CPR and 10 to 12 breaths per minute (approximately 1 breath every 5 to 6 seconds) for ventilation without chest compressions (ie, for respiratory arrest without cardiac arrest). Each breath should last 1 second.
- **Compression-ventilation cycles:** Once an advanced airway is in place, the compressing rescuer provides chest compressions at a rate of at least 100 per minute without pauses for ventilations. Compressors should rotate every 2 minutes.

Once the patient is in the hospital, obtain a chest x-ray as soon as possible to determine the depth of the ETT insertion. Look for incorrect placement into a main bronchus.

Never wait for a chest x-ray to check misplacement of the tube in the esophagus. You must detect esophageal insertion immediately by checking tube placement immediately after tube insertion. Confirm proper placement by physical exam and the confirmation techniques discussed below (Clinical and Device Confirmation of ETT Placement).

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 (PEEP) 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.

Complications of ETT Placement

Several complications may occur with endotracheal intubation.

If the ETT is inserted into the esophagus, the patient will receive no ventilation or oxygenation unless he or she is still breathing spontaneously. If you or your team fails to recognize esophageal intubation, the patient could suffer permanent brain damage or die.

Use care when removing and replacing an incorrectly placed ETT. Use bag-mask ventilation and then reintubate after you address the higher priorities (ie, continuous chest compressions, defibrillation as needed, IV access). The ETT will help reduce the risk of gastric inflation, but the insertion process requires interruption of chest compressions and may produce additional complications.

If a laryngoscope and tube are not readily available or if the intubation attempt is not successful within 30 seconds, return to bag-mask ventilation. Provide 100% oxygen and attempt intubation again in 20 to 30 seconds.

Tube Trauma and Adverse Effects

Endotracheal intubation can cause significant trauma to the patient, including

- 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 ETT
- Injury to the vocal cords
- Pharyngeal-esophageal perforation
- Vomiting and aspiration of gastric contents into the lower airway
- Release of high levels of epinephrine and norepinephrine, which can cause elevated blood pressures, tachycardia, or arrhythmias

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Insertion of ETT Into One Bronchus

Insertion of the ETT 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.

To determine if the ETT has been inserted into a bronchus, listen to the chest for bilateral breath sounds. Also look for equal expansion of both sides during ventilation.

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.

- Recheck the patient's clinical signs, including chest expansion, breath sounds, and evidence of oxygenation.

You can also order a portable chest x-ray to check placement of the ETT. But remember, recognizing this complication is a clinical responsibility. You order an x-ray after clinical confirmation to assess correct ETT placement and tube position.

Endotracheal Administration of Resuscitation Medications

Endotracheal administration of medications is used if IV or IO access cannot be established. IV and IO are the preferred routes for drug administration. Providers use the memory aid NAVEL to recall naloxone, atropine, vasopressin, epinephrine, and lidocaine, each of which can be administered by ETT. Use the ETT route of administration *only if you cannot obtain IV/IO access.* In addition, you should use a dose that is approximately 2 to 2.5 times higher than the dose for IV/IO administration. Mix the dose of drug with 5 to 10 mL of normal saline or distilled water. (Note: Absorption of epinephrine and lidocaine is greater when these drugs are diluted with distilled water, but the water may cause more adverse effects on PaO_2.)

- As noted above, ETT doses of medications should be considerably higher than IV doses—in the range of 2 to 2.5 times the IV dose. For example, the recommended ETT dose of epinephrine is at least 2 to 2.5 mg.
- Once the medication has been administered through the ETT, perform 1 to 2 good ventilations to facilitate deposition of the drug into the airways.
- When equal amounts of the same drug are given by the IV and ETT routes, the serum concentration of ETT drugs is much lower than the serum concentration of IV drugs.

Confirmation of ETT Placement: Physical Exam

Confirm tube placement immediately, assessing the first breath delivered by the bag-mask device. This assessment should not require interruption of chest compressions. No single confirmation technique, including clinical signs or the presence of water vapor in the tube or device, is completely reliable, particularly when cardiac arrest is present. For this reason the AHA recommends the use of both clinical assessment and a device to confirm correct tube placement. Ideally you will attach a CO_2 detection device to enable detection of exhaled CO_2. 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 ETT at once. Then:

- Immediately resume 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 ventilations using 100% oxygen).
- If, following 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.
- If there is still doubt about correct tube placement, use the laryngoscope to see if the tube is passing through the vocal cords.
- 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 with a commercial device designed for this purpose or with tape.
- 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 ETT Placement: Qualitative and Quantitative Devices

The *2005 AHA Guidelines for CPR and ECC* recommend confirmation of ETT 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 that chest compressions must be interrupted.

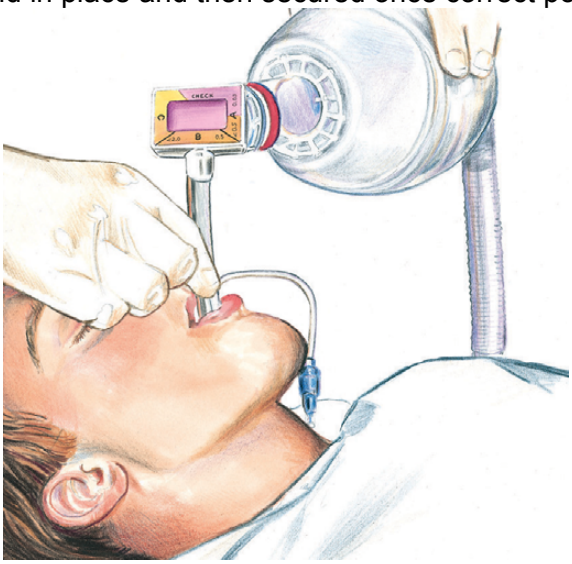
Providers should always use both clinical assessment and a device to confirm ETT location immediately after placement and each time the patient is moved. Detailed assessment of out-of-hospital intubation attempts has concluded that ETTs are (1) much more difficult to place properly in that setting and (2) 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.

A variety of electronic and mechanical devices are available for use in both the in-hospital and out-of-hospital settings. There are several models of end-tidal CO_2 detectors (qualitative, quantitative, and continuous) and of esophageal detector devices. These devices range from simple and inexpensive to complex and costly.

Exhaled (Qualitative) CO_2 Detectors

A number of commercial devices can react, usually with a color change, to CO_2 exhaled from the lungs. This simple method can be used as the initial method of detecting correct tube placement even in the patient in cardiac arrest (Figure 10). The qualitative detection device indicating exhaled CO_2 indicates proper ETT placement. The absence of a CO_2 response from the detector (ie, results are *negative* for CO_2) generally means that the tube is in the esophagus, particularly in patients with spontaneous circulation.

Figure 10. Confirmation of tracheal tube placement. **A,** End-tidal colorimetric carbon dioxide indicator: purple color indicates lack of carbon dioxide—probably in the esophagus. **B,** End-tidal colorimetric carbon dioxide indicators: yellow indicates the presence of carbon dioxide and tube in airway. 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.



A

B

No CO_2 *detected but tube in trachea:* The tube is actually in the trachea, but a negative reading for CO_2 leads to unnecessary removal of the tube. These negative readings most commonly occur because end-tidal CO_2 production is minimal in cardiac arrest. Chest compressions during CPR produce an estimated 20% to 33% of normal blood flow to the lungs, so little if any CO_2 is exhaled. Negative readings also occur in patients with a large amount of dead space (eg, a significant pulmonary embolus).

CO_2 *detected but tube in esophagus:* The tube is in the esophagus, yet CO_2 was detected, leading to prolonged esophageal intubation. These positive readings have been reported in animals that had ingested large amounts of carbonated liquids before the arrest. This results in CO_2 release from the stomach into the esophagus during CPR. To avoid this problem, manufacturers suggest that you evaluate CO_2 detector readings after delivery of about 5 or 6 breaths.

Quantitative End-Tidal CO_2 Monitors

The quantitative end-tidal CO_2 monitor is a hand-held confirmation device. This device is a *capnometer.* It provides a single quantitative readout of the concentration of CO_2 at a single point in time. The *capnograph* provides a continuous display of the level of CO_2 as it varies throughout the ventilation cycle.

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

Esophageal Detector Devices

Esophageal detector devices (EDDs) (Figure 11) apply a suction force to the inserted end of the ETT. The suction force is created when you pull back the plunger on a large syringe (60 to 100 mL) or completely compress a flexible aspiration bulb. Once compressed, the bulb is firmly attached to the end of the tube coming out of the mouth and then released. If the tip of the tube is in the esophagus, the suction will pull the esophageal mucosa against the tip of the tube, preventing movement of the plunger or reexpansion of the suction bulb. There will be either no expansion or very slow reexpansion.

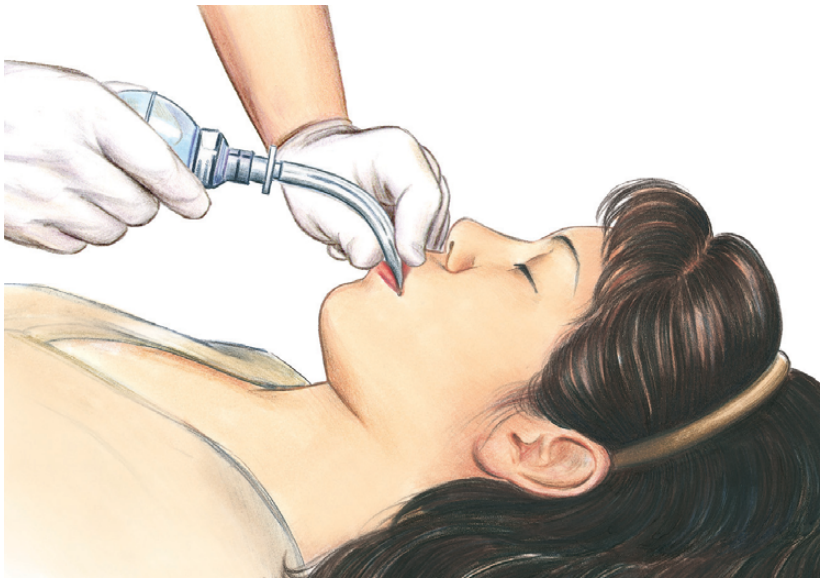


Figure 11. Esophageal detector device: aspiration bulb technique. Hold the tube in place until you confirm that it is in the correct position and then secure it.

Unlike the end-tidal CO_2 detector, the EDD does not depend on blood flow. However, although the device is generally sensitive for detection of ETT placement in the esophagus it is not specific for ETT placement in the trachea. In addition, it may yield misleading results in patients with morbid obesity, late pregnancy, or status asthmaticus. There is no evidence that the EDD is accurate for the continuous monitoring of ETT placement. For these reasons, the EDD should be considered one of several methods for confirmation of ETT placement.

Results suggest that the tube is not *in the esophagus when it* is *in the esophagus:* There are several ways in which the EDD can suggest that the tube is in the trachea (suction not maintained on bulb) when the tube is actually in the esophagus. The EDD indicates that the tube is in the trachea by rapid reexpansion of the suction bulb. But prior CPR or ventilations using a bag can fill the stomach or esophagus with air, causing the bulb to reexpand or the plunger to push out. The unwary rescuer, thinking the tube is in the trachea, may leave the tube in the esophagus, a potentially fatal error.

See Table 2 for a comparison of the qualitative performance of the EDD and end-tidal CO_2 device in terms of correct responses plus the most common causes of misleading results.

Causes of Misleading Results From End-Tidal CO_2 Detectors and EDDs

Table 2 lists possible causes of misleading results using end-tidal CO_2 detector devices and EDDs to confirm correct placement of the ETT. The columns (vertical) indicate the reading and actual location of the ETT. The rows (across) indicate the expected results from using either a colorimetric end-tidal CO_2 detector (A) or bulb-type esophageal detector device (B). With both devices assume that the rescuer made a conscientious intubation effort and thinks the ETT is in the trachea.

Table 2. Reasons for Misleading Results Using End-tidal CO_2 Detector and Esophageal Detector Device.

A: Colorimetric End-Tidal CO_2 Detector		
Reading	**Actual Location of ETT: Trachea**	**Actual Location of ETT: Esophagus (or Hypopharynx)**
Carbon Dioxide Detected Color change (or as specified by manufacturer) (positive = CO_2 present)	***ETT 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 (or as specified by manufacturer) (negative = CO_2 absent)	***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 ETT. 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 ETT in esophagus/hypopharynx. A life-threatening adverse event has occurred. **Consequences:** Rescuer recognizes ETT is not in trachea; properly and rapidly identified; tube is removed at once; patient is reintubated.
B: Esophageal Detector Device		
Reading	**Actual Location of ETT: Esophagus**	**Actual Location of ETT: Trachea**
Consistent With Tube in	***Device suggests tube in esophagus when it is***	***Device suggests tube in esophagus when it is in***

Esophagus Bulb does not refill or refills slowly (>5 seconds × 2), or syringe cannot be aspirated, suggesting that tip of ETT is in esophagus	***in esophagus*** **Causes:** Rescuer has inserted tube in esophagus/hypopharynx. A life-threatening adverse event has occurred. **Consequences:** Rescuer correctly recognizes ETT is in esophagus; ETT is removed at once; patient is reintubated.	***trachea*** **Causes:** Secretions in trachea (mucus, gastric contents, acute pulmonary edema); insertion in right main bronchus; pliable trachea (morbid obesity, late-term pregnancy). **Consequences:** Leads to unnecessary removal of properly placed ETT. Reintubation attempts increase chances of other adverse consequences.
Consistent With Tube in Trachea Bulb fills immediately or syringe can be aspirated, suggesting that ETT is in trachea	***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.*** Esophageal detector device indicates ETT is in trachea. Proceed with ventilations.

ACLS Core Rhythms

Part 1—Recognition of Core ECG Arrest Rhythms

The Basics Figure 12 shows the anatomy of the cardiac conduction system and its relationship to the ECG cardiac cycle.

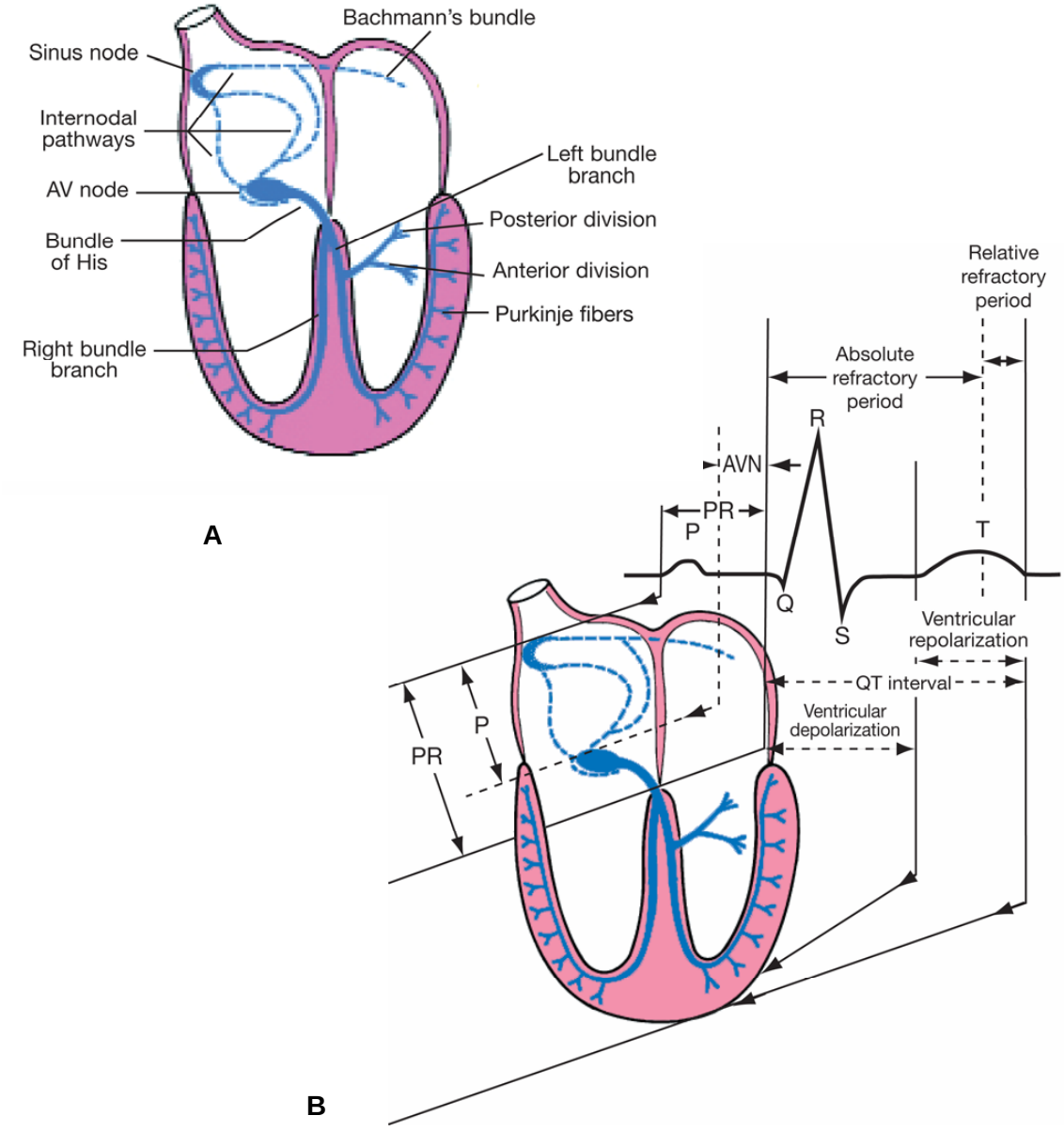


Figure 12. Anatomy of the cardiac conduction system: relationship to the ECG cardiac cycle. **A,** Heart: anatomy of conduction system. **B,** Relation of cardiac cycle to conduction system anatomy.

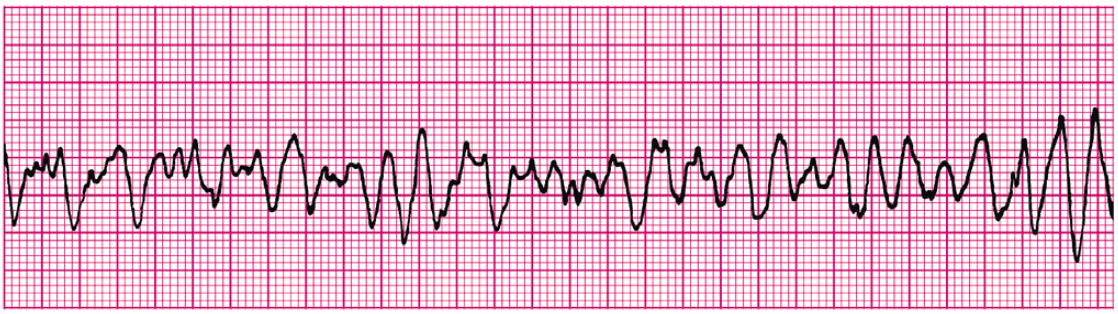
Cardiac Arrest Rhythms

The ECG rhythms for patients who are in cardiac arrest are

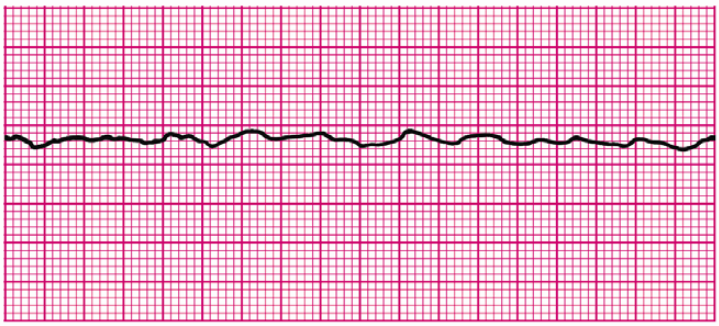
- Ventricular fibrillation (VF)/pulseless ventricular tachycardia (VT)
- Pulseless electrical activity (PEA)
- Asystole

These ECG rhythms are shown below:

Ventricular Fibrillation (Figure 13)	
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 prior to the VF) • Collapse, unresponsiveness • Agonal gasps • Onset of *irreversible death*
Common Etiologies	• Acute coronary syndromes (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



A



B

Figure 13. A, Coarse ventricular fibrillation. Note high-amplitude waveforms, which vary in size, shape, and rhythm, representing chaotic ventricular electrical activity. The ECG criteria for VF are as follows: (1) QRS complexes: no normal-looking QRS complexes are recognizable; a regular "negative-positive-negative pattern (Q-R-S) cannot be seen. (2) Rate: uncountable; electrical deflections are very rapid and too disorganized to count. (3) Rhythm: no regular rhythmic pattern can be discerned; the electrical waveforms vary in size and shape; the pattern is completely disorganized. **B,** Fine ventricular fibrillation. In comparison with Figure 13A, the amplitude of electrical activity is much reduced. Note the complete absence of QRS complexes. 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.

PEA	
Pathophysiology	• Cardiac conduction impulses occur in an organized pattern but do not produce myocardial contraction (this condition was formerly called electromechanical dissociation); or insufficient ventricular filling during diastole; or ineffective contractions
Defining Criteria per ECG	• Rhythm displays organized electrical activity (not VF/pulseless VT) • Usually not as organized as normal sinus rhythm • Can be narrow (QRS <0.10 mm) or wide (QRS >0.12 second); fast (>100 per minute) or slow (<60 per minute) • May be narrow (noncardiac etiology) or wide (often cardiac etiology) and can be slow (cardiac etiology) or fast (often

	noncardiac etiology).
Clinical Manifestations	• Collapse, unresponsive • Agonal gasps or apnea • No pulse detectable by palpation (very low systolic blood pressure could still be present in such cases termed *pseudo-PEA*)
Common Etiologies	Use the H's and T's mnemonic to recall possible causes of PEA: • Hypovolemia • Hypoxia • Hydrogen ion (acidosis) • Hypo-/hyperkalemia • Hypoglycemia • Hypothermia • Toxins ("tablets," ie, drug overdose, ingestion) • Tamponade, cardiac • Tension pneumothorax • Thrombosis, coronary (ACS) or pulmonary (embolism) • Trauma

Asystole (Figure 14)	
Defining Criteria per ECG Classically asystole presents as a "flat line"; defining criteria are virtually nonexistent	• **Rate:** no ventricular activity seen or ≤6 complexes per minute; so-called "P-wave asystole" occurs with only atrial impulses present (P waves) • **Rhythm:** no ventricular activity seen or ≤6 complexes per minute • **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	• May have agonal gasps (early); unresponsive • No pulse or blood pressure • Cardiac arrest
Common Etiologies	• End of life (death) • Ischemia/hypoxia from many causes • Acute respiratory failure (no oxygen, apnea, asphyxiation) • Massive electrical shock (eg, electrocution, lightning strike) • May represent "stunning" of the heart immediately after defibrillation (shock deliver that eliminates VF), prior to resumption of spontaneous rhythm

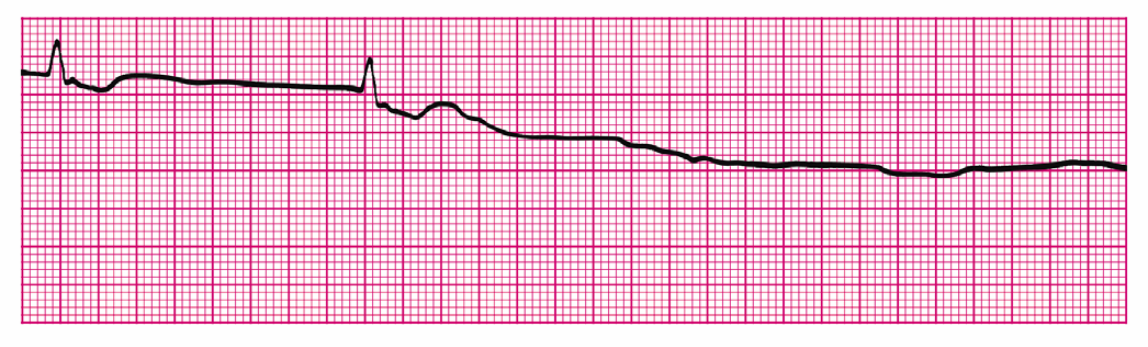


Figure 14. The "rhythm" of ventricular asystole. This patient is pulseless and unresponsive. Note the 2 QRS-like complexes at the start of this rhythm display. These complexes represent a minimum of electrical activity, probably ventricular escape beats. Does this pattern represent *pulseless electrical activity*? Note the long section in which electrical activity is completely absent. This patient is in asystole at this point.

Part 2—Recognition of Selected Nonarrest ECG Rhythms

Recognition of Supraventricular Tachyarrhythmias

Sinus Tachycardia (Figure 15)	
Pathophysiology	• None—more a physical sign than an arrhythmia or pathologic condition • Normal impulse formation and conduction
Defining Criteria and ECG Features	• **Rate:** >100 per minute • **Rhythm:** sinus • **PR:** usually <0.20 second • **P for every QRS Complex** • **QRS complex:** normal
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 • Fever • Hypovolemia • Adrenergic stimulation, anxiety • Hyperthyroidism

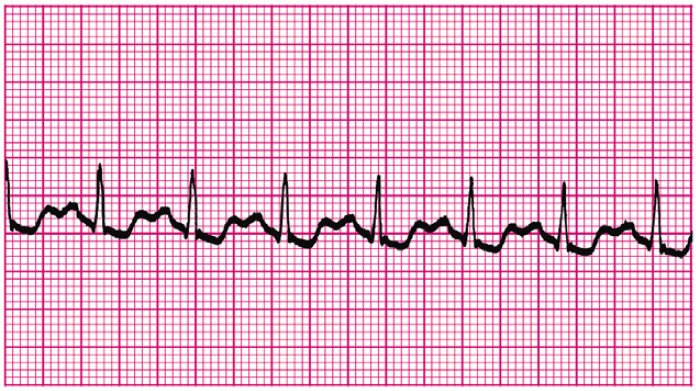


Figure 15. Sinus tachycardia.

<table>
<tr><th colspan="4">Atrial Fibrillation (Figure 16) and Atrial Flutter (Figure 17)</th></tr>
<tr><td>Pathophysiology</td><td colspan="3">• Atrial impulses 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</td></tr>
<tr><td rowspan="5">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: “Irregularly irregular rhythm—with variation in both interval and amplitude from R wave to R wave—is atrial fibrillation.” This one is usually dependable. Can also be observed in multifocal atrial tachycardia (MAT).

Atrial Flutter Key: Flutter waves in classic “sawtooth” pattern.</td><td></td><td>Atrial Fibrillation</td><td>Atrial Flutter</td></tr>
<tr><td>Rate</td><td>• Wide-ranging ventricular response to atrial rate
• May be normal or slow if AV nodal conduction is abnormal (eg “sick sinus syndrome”)</td><td>• Atrial rate 220 to 350 per minute
• Ventricular response is a function of AV node block or conduction of atrial impulses
• Ventricular response rarely >150 to 180 beats because of AV nodal conduction limits</td></tr>
<tr><td>Rhythm</td><td>• Irregular (classic “irregularly irregular”)</td><td>• Regular (unlike atrial fibrillation)
• Ventricular rhythm often regular
• Set ratio to atrial rhythm, eg, 2-to-1 or 4-to-1</td></tr>
<tr><td>P waves</td><td>• Chaotic atrial fibrillatory waves only
• Creates variable baseline</td><td>• No true P waves seen
• Flutter waves in “sawtooth” pattern is classic</td></tr>
<tr><td>PR</td><td colspan="2">• Cannot be measured</td></tr>
<tr><td>QRS</td><td colspan="2">• Remains ≤0.10 to 0.12 second unless QRS complex is distorted by fibrillation or flutter waves or by conduction defects through ventricles</td></tr>
<tr><td>Clinical Manifestations</td><td colspan="3">• 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 (DOE), shortness of breath (SOB), and sometimes acute pulmonary edema
• Loss of “atrial kick” may lead to drop in cardiac output and decreased coronary perfusion
• Irregular rhythm often perceived as “palpitations”
• Can be asymptomatic</td></tr>
</table>

Common Etiologies	• Acute coronary syndromes, coronary artery disease, congestive heart failure • Disease at mitral or tricuspid valve • Hypoxia, acute pulmonary embolism • Drug-induced: digoxin or quinidine; β agonists, theophylline • Hypertension • Hyperthyroidism

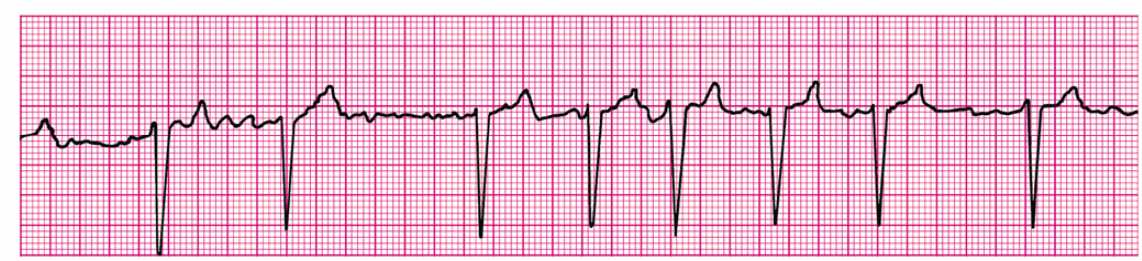


Figure 16. Atrial fibrillation.

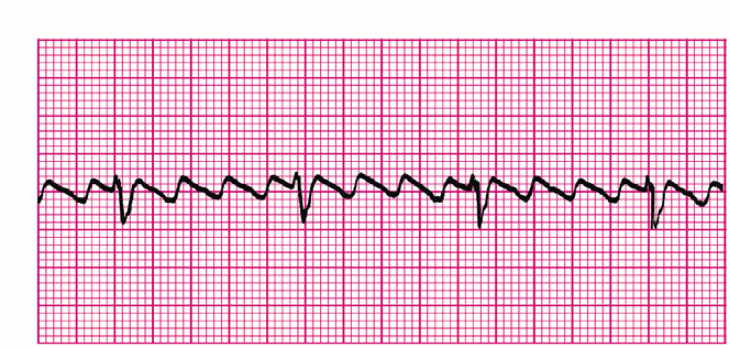


Figure 17. Atrial flutter.

Accessory-Mediated SVT (Figure 18); 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 (>120 to 130 per minute), seldom <150 per minute, often up to 250 per minute **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 (usually ≤0.10 second)
Clinical Manifestations	• Palpitations felt by patient at onset; becomes anxious, uncomfortable • Exercise tolerance low with very high rates • Symptoms of unstable tachycardia may occur
Common Etiologies	• Accessory conduction pathway 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 increased in unhealthy patients with coronary artery disease, chronic obstructive pulmonary disease, and congestive heart failure

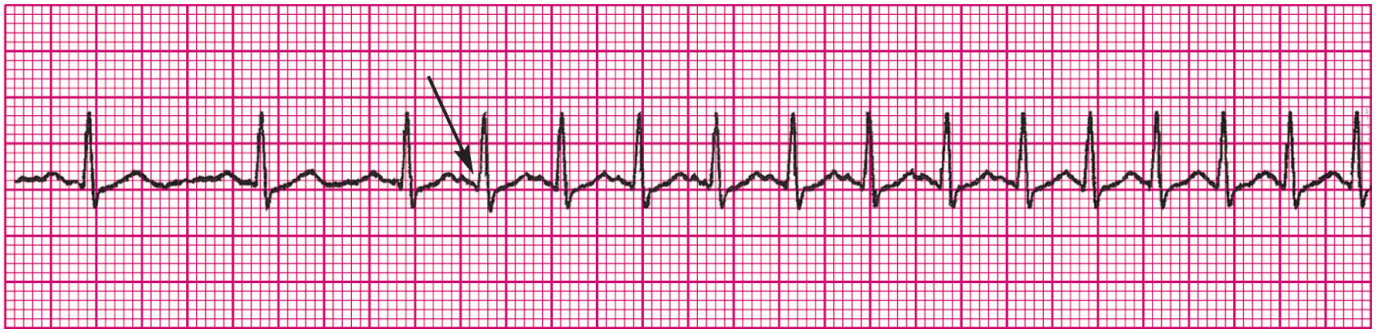


Figure 18. Sinus rhythm with a reentry supraventricular tachycardia (SVT).

Recognition of Ventricular Tachyarrhythmias

Monomorphic VT (Figure 19)	
Pathophysiology	• Impulse conduction is slowed around areas of ventricular injury, infarct, or ischemia • 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:** 3 or more consecutive PVCs indicate *VT* ◦ VT <30 seconds duration is *nonsustained VT* ◦ VT >30 seconds duration is *sustained VT*	• **Rate:** ventricular rate >100 per minute; typically 120 to 250 per minute • **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 seconds, 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, often VF
Common Etiologies	• An acute ischemic event (see Pathophysiology) with areas of "ventricular irritability" leading to PVCs • PVCs that occur during relative refractory period of cardiac cycle ("R-on-T phenomenon") • Drug-induced, prolonged QT interval (tricyclic antidepressants, procainamide, digoxin, some long-acting antihistamines)

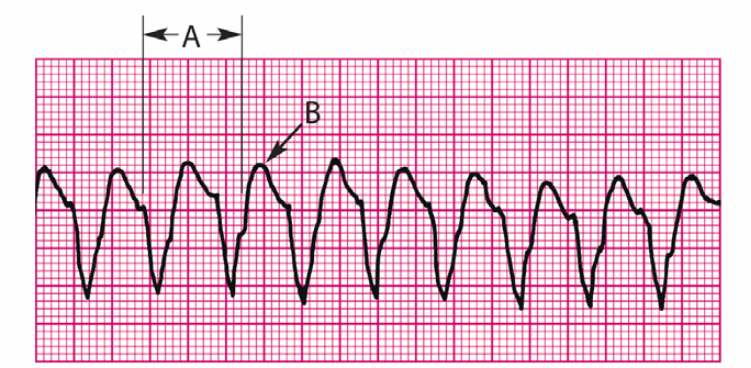


Figure 19. Monomorphic VT at a rate of 150 per minute: wide QRS complexes (arrow A) with opposite polarity T waves (arrow B).

Polymorphic VT (Figure 20)	
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 per minute; typically 120 to 250 per minute • **Rhythm:** only regular ventricular • **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 pulseless VT or VF • Symptoms of decreased cardiac output (orthostasis, hypotension, poor perfusion, syncope, etc) present before pulseless arrest • Seldom sustained VT
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) • Hereditary long QT interval syndromes

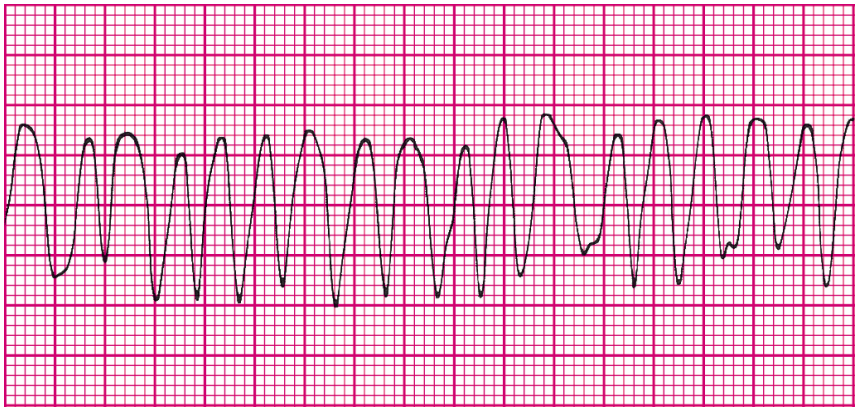


Figure 20. Polymorphic VT: QRS complexes display multiple morphologies.

Torsades de Pointes (a Unique Subtype of Polymorphic VT) (Figure 21)	
Pathophysiology	Specific pathophysiology of classic torsades: • QT interval is abnormally long (baseline ECG) (see the Maximum QT Interval table in the ECC Handbook) • Leads to increase in relative refractory period ("vulnerable period") of cardiac cycle. This increases probability that an irritable focus (PVC) will occur on T wave (vulnerable period or R-on-T phenomenon) • 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 next spindle (creating the "node").	• **Atrial rate:** cannot determine • **Ventricular rate:** 150 to 250 complexes/min • **Rhythm:** only irregular ventricular rhythm • **PR:** nonexistent • **P waves:** nonexistent • **QRS complexes:** display classic spindle-node pattern (see "Key" at left)

Clinical Manifestations	• Tends toward sudden deterioration to pulseless VT or VF • Symptoms of decreased cardiac output are typical (orthostasis, hypotension, syncope, signs of poor perfusion, etc) • "Stable" torsades, sustained torsades is uncommon • Treated with unsynchronized high-energy (defibrillation) shocks
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 • Electrolyte and metabolic alterations (hypomagnesemia is the prototype) • Inherited forms of long QT syndrome • Acute ischemic events (see Pathophysiology)

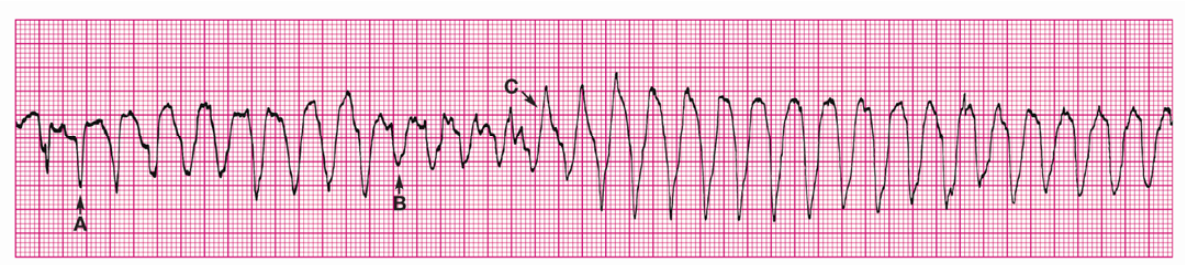


Figure 21. Torsades de pointes: a unique type of polymorphic VT. **A,** Start of a "spindle." Note negative initial deflection and increasing QRS amplitude. **B,** End of a spindle and start of a "node." **C,** End of a node and start of the next spindle. Note the positive initial deflection and "spindling" in QRS amplitude.

Recognition of Sinus Bradycardia

Sinus Bradycardia (Figure 22)	
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 per minute **Note:** Often a physical sign rather than an abnormal rhythm	• **Rate:** <60 per minute • **Rhythm:** regular sinus • **PR:** regular, <0.20 second • **P waves:** size and shape normal; every P wave is followed by a QRS complex, every QRS complex is preceded by a P wave • **QRS complex:** narrow; ≤0.10 second in absence of intraventricular conduction defect
Clinical Manifestations	• Usually asymptomatic 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 lightheadedness, syncope, hypotension
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”) • Acute coronary syndromes that affect circulation to SA node (right coronary artery); most often inferior AMIs • Adverse drug effects, eg, β-blockers or calcium channel blockers, digoxin, quinidine

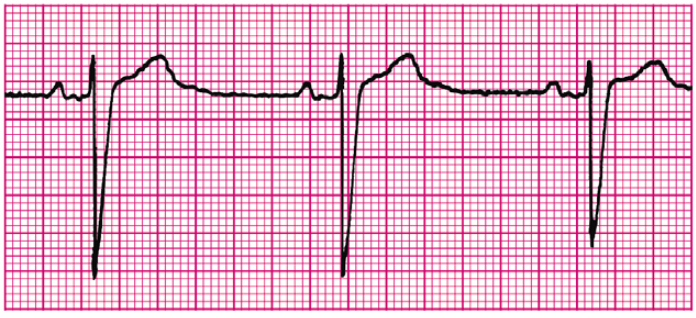


Figure 22. Sinus bradycardia.

Recognition of Atrioventricular (AV) Block

First-Degree AV Block (Figure 23)	
Pathophysiology	• Impulse conduction is 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 greater than 0.20 second	• **Rate:** first-degree heart block can be 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 is followed by a QRS complex, every QRS complex is preceded by P wave • **QRS complex:** narrow, ≤0.10 second in absence of intraventricular conduction defect
Clinical Manifestations	• Usually asymptomatic
Common Etiologies	• Many first-degree AV blocks are due to drugs, usually the AV nodal blockers: β-blockers, non-dihydropyridine calcium channel blockers, and digoxin • Any condition that stimulates the parasympathetic nervous system (eg, vasovagal reflex) • AMIs that affect circulation to the AV node (right coronary artery); most often inferior AMIs

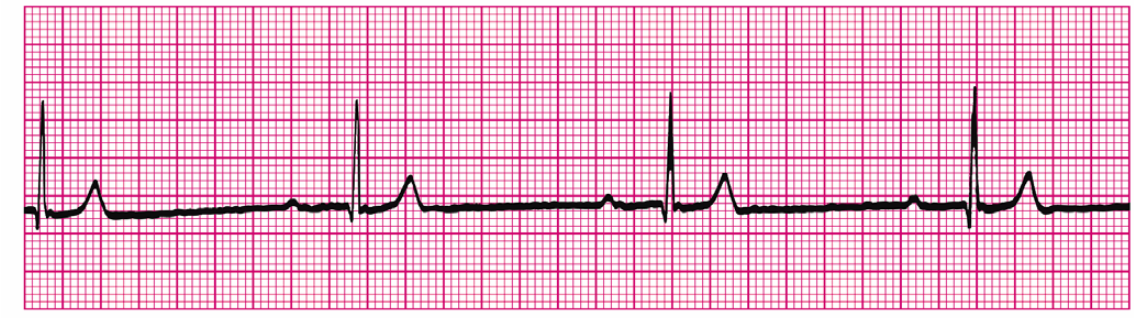


Figure 23. First-degree AV block.

Second-Degree Block Type I (Mobitz I–Wenckebach) (Figure 24)	
Pathophysiology	• Site of pathology: 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 see regular P waves marching through irregular QRS • **PR:** progressive lengthening of PR interval occurs from cycle to cycle; then one P wave is not followed by QRS complex ("dropped beat") • **P waves:** size and shape remain normal; occasional P wave not followed by QRS complex ("dropped beat") • **QRS complex:** ≤0.10 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 (CHF), angina
Common Etiologies	• AV nodal blocking agents: β-blockers, non-dihydropyridine calcium channel blockers, digoxin • Conditions that stimulate the parasympathetic nervous system • Acute coronary syndrome that involves right coronary artery

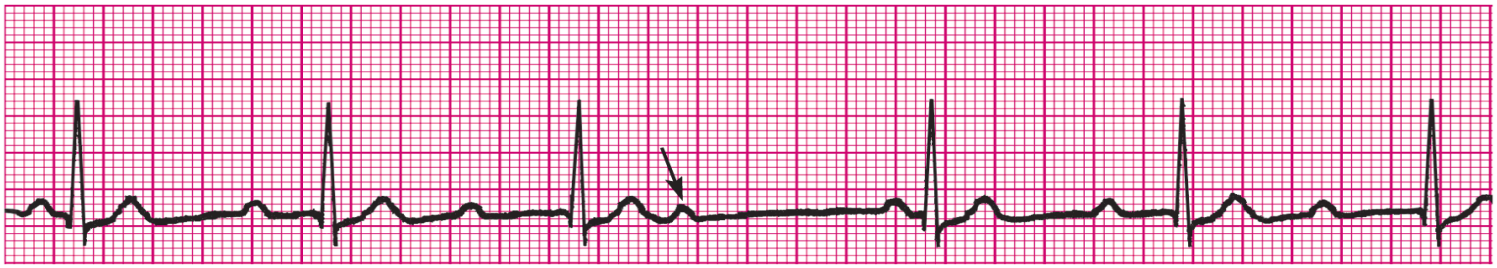
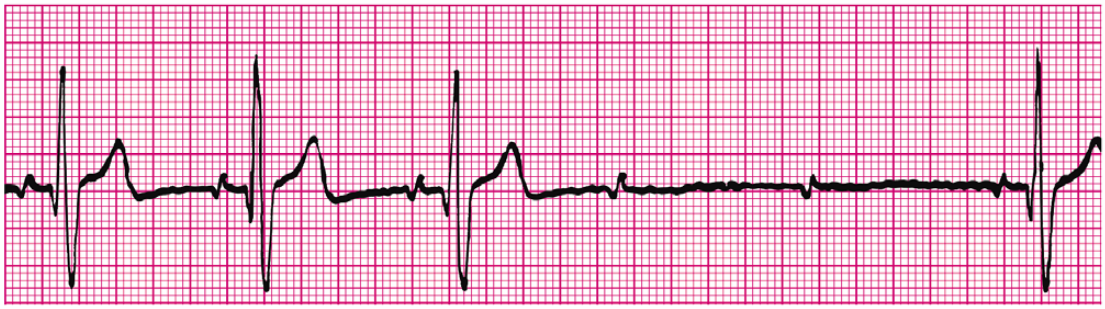


Figure 24. Second-degree AV block Type I. Note the progressive lengthening of the PR interval until one P wave (arrow) is not followed by a QRS.

Second-Degree AV Block Type II (Infranodal) (Mobitz II) (Figures 25)	
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 per minute • **Ventricular rate:** by definition (because of blocked impulses) slower than atrial rate • **Rhythm:** atrial = regular, ventricular = irregular (because of blocked impulses) • **PR:** constant and set; no progressive prolongation as with Mobitz Type I second-degree block—a distinguishing characteristic • **P waves:** typical in size and shape; by definition some P waves will not be followed by a QRS complex • **QRS complex:** narrow (≤0.10 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, CHF, AMI
Common Etiologies	• Acute coronary syndrome that involves branches of *left* coronary artery



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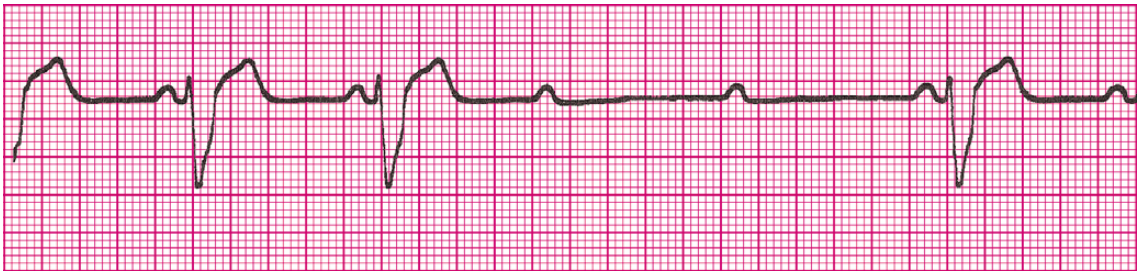


B

Figure 25. 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.

Third-Degree AV Block and AV Dissociation (Figure 26)	
Pathophysiology **Pearl:** *AV dissociation* is the defining class; *third-degree* or *complete AV block* is one type of AV dissociation. By convention (outdated), if ventricular escape depolarization is faster than atrial rate, *AV dissociation* is present; if ventricular rate is slower than atrial rate, *third-degree AV block* is present.	• 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	• **Atrial rate:** usually 60 to 100 per minute; impulses completely independent (“dissociated”) from the slower ventricular rate • **Ventricular rate:** depends on rate of ventricular escape beats that arise:

block (see Pathophysiology) causes atria and ventricles to depolarize independently, with no relationship between the two (AV dissociation).	— Ventricular escape rate slower than atrial rate = third-degree AV block (rate = 20 to 40 per minute) — Ventricular escape rate faster than atrial rate = AV dissociation (rate = 40 to 55 per minute) • **Rhythm:** both atrial rhythm and ventricular rhythm are regular but independent ("dissociated") • **PR:** by definition there is no relationship between P wave and R wave • **P waves:** typical in size and shape • **QRS complex:** narrow (≤0.10 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, CHF, AMI
Common Etiologies	• Acute coronary syndrome that involves branches of *left* coronary artery In particular, involves left anterior descending (LAD) artery and branches to interventricular septum (supply bundle branches)

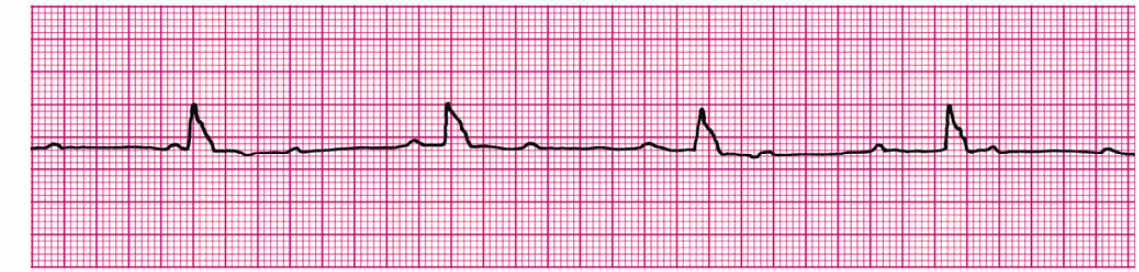


Figure 26. Third-degree AV block: regular P waves at 50 to 55 per minute; regular ventricular "escape beats" at 35 to 40 per minute; no relationship between P waves and escape beats.

Defibrillation

Manual Defibrillation

Using a Manual Defibrillator/ Monitor

When using a manual defibrillator/monitor, perform a rhythm check as indicated by the Pulseless 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) and using the paddle "quick look" feature.

Because adhesive monitor/defibrillator electrode pads are as effective as gel pads or paste and paddles, 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. Whether using the adhesive electrode pads or paddles, the ACLS provider should be very careful not to delay the shock and during CPR to minimize the time between last compression and shock delivery. Delays in delivery of the first shock 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.

When you identify VF/pulseless VT, *immediately* deliver 1 shock. Use the following energy levels:

- Manual biphasic: device-specific (typically a selected energy of 120 J with a rectilinear biphasic waveform and a selected energy of 150 J to 200 J with a biphasic truncated exponential waveform); if you do not know the device-specific dose shown to be effective for elimination of VF, use 200 J
- Monophasic: 360 J

After delivering the shock, immediately resume CPR, pushing hard and fast (compression rate 100 per minute). Allow full chest recoil after each compression, and minimize interruptions in compressions.

Attaching the 3 Monitor Leads

Most monitors use three leads: white, red, and black.

"WHITE to RIGHT"
"RED to RIBS"
"*The LEAD LEFT OVER goes to LEFT SHOULDER*"

The following table explains these directions in more detail.

Attach…	Where…
WHITE lead to RIGHT	Right side of the chest, just beneath the right clavicle
RED lead to RIBS	Left midaxillary line, below the expected point of maximum impulse of the heart
The LEAD [that is] LEFT OVER goes to LEFT SHOULDER	Left side of the torso, just beneath the distal end of the left clavicle

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:

- *"I am going to shock on three. One, I am clear."* (Check to make sure you have no contact with the patient, the stretcher, or other equipment.)
- *"Two, you are clear."* (Check to make sure no one is touching the patient. "No one" includes providers performing chest compressions, starting IVs, inserting catheters, or performing ventilation and airway maintenance.)

  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 endotracheal tube, must also 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.

- *"Three, everybody is clear."* (Perform a visual check to make sure no one has contact with the patient or stretcher.)

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

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 increases the patient's chance of survival. This principle holds true regardless of the type of defibrillator or waveform.

Part 2—AED Checklist

AED Readiness-for-Use Checklist
Daily/Weekly Checklist
Date __________ Covering Period _____________ to ______________

Organization Name/Identifier ____
Manufacturer/Model No. _______________________
Serial/ID No. ________________

At the beginning of each shift or at the scheduled time, inspect the device using this checklist. Note any inconsistencies, problems, and corrective actions taken. If the device is not ready for use or is out of service, write OOS on the "day of month" line and note problems in the corrective action log.

	Corrective Action Log	***Day of Month/Signature/Unit No.***
1. Defibrillator unit		
a. Clean, no spills, unobstructed	_________________	1. __________________________
b. Casing intact	_________________	2. __________________________
2. Defibrillation cables and connectors	_________________	3. __________________________
a. Inspect for cracks, broken wires, or damage	_________________	4. __________________________
b. Connectors engage securely	_________________	5. __________________________
3. Supplies available	_________________	6. __________________________
a. Two sets of unexpired hands-free defibrillator	_________________	7. __________________________
pads in sealed packages	_________________	8. __________________________
b. Personal protection equipment—gloves, barrier	_________________	9. __________________________
device (or equivalent)	_________________	10. _________________________
c. Razor and scissors	_________________	11. _________________________
d. Hand towel	_________________	12. _________________________
e. Spare event documentation device*	_________________	13. _________________________
f. ECG paper*	_________________	14. _________________________
g. ECG monitoring electrodes*	_________________	15. _________________________
h. ALS module/key (or equivalent)*	_________________	16. _________________________
4. Power supply	_________________	17. _________________________
a. Verify fully charged battery(ies) in place	_________________	18. _________________________
b. Spare charged battery available*	_________________	19. _________________________
c. Rotate batteries per manufacturer's	_________________	20. _________________________
specifications*	_________________	21. _________________________
d. AC power plugged into live outlet*	_________________	22. _________________________
5. Indicators and screen display	_________________	23. _________________________
a. POWER ON display and self-test OK*	_________________	24. _________________________
b. ECG monitor display functional*	_________________	25. _________________________
c. No error or service required indicator/message*	_________________	26. _________________________
d. Correct time displayed/set; synchronized with	_________________	27. _________________________
dispatch center	_________________	28. _________________________
6. ECG paper and event documentation device	_________________	29. _________________________
a. Event documentation device in place and	_________________	30. _________________________
functional*	_________________	31. _________________________
b. Adequate ECG paper*	_________________	
c. ECG recorder functional*		***Example entry:*** *5. John Jones (signature)/Aid 2 checked Aid 2's device on the 5th day of this month and found it ready for use.*
7. Charge/display cycle for defibrillation		
a. Test per manufacturer's recommended test procedure		
b. Identifies shockable rhythm*		
c. Charges to appropriate energy level*		
d. Acceptable discharge detected*		
8. AED returned to patient-ready status		
**Applicable only if the device has this capability or feature or if required by medical authority.*		

Access for Medications

Correct Priorities

Historically in ACLS, drugs were administered by the intravenous (IV) or endotracheal (ET) route. But new science and consensus opinion have prioritized both access routes and drug administration. Remember, no drug given during cardiac arrest has been shown to improve survival to hospital discharge or improve neurologic function after cardiac arrest.

- High-quality CPR and early defibrillation are the top priorities during cardiac arrest.
- Drug administration is of secondary importance.
- Insertion of an advanced airway whether for drug administration or ventilation, unless bag-mask ventilation is ineffective, is of secondary importance. Absorption of drugs given by the ET route is unpredictable, and optimal dosing is unknown. For this reason the IO route is preferred when IV access is not available.

Intravenous Route

A peripheral IV is preferred for drug and fluid administration. Central line access is not needed during most resuscitation attempts. Attempts to insert a central line may interrupt CPR. In addition, CPR can cause complications during central line insertion, such as vascular laceration, hematomas, and bleeding. Insertion of a central line in a noncompressible area of a vein is a relative contraindication to fibrinolytic therapy (eg, for the patient with an STEMI and sudden cardiac arrest).

Establishing a peripheral line should not require interruption of CPR. And drugs typically require 1 to 2 minutes to reach the central circulation when given by the peripheral IV route. Keep this in mind during CPR. The drug you give based on a rhythm check will not take effect until it is flushed into the patient and has been circulated by the blood flow generated during CPR.

If you choose the peripheral venous route, give the drug by bolus injection and follow with a 20-mL bolus of IV fluid. Elevate the extremity for 10 to 20 seconds to facilitate delivery of the drug to the central circulation.

Intraosseous Route

Use the IO route to deliver drugs and fluids during resuscitation if IV access is unavailable. IO access is safe and effective for fluid resuscitation, drug delivery, and blood sampling for laboratory evaluation. IO access can be established in all age groups.

Any drug or fluid that can be given by the IV route can also be given by the IO route. The IO route is preferred over the ET route.

IO cannulation provides access to a noncollapsible venous plexus in bone marrow. This vascular network provides a rapid, safe, and reliable route for administration of drugs, crystalloids, colloids, and blood during resuscitation. It is often possible to achieve IO access in 30 to 60 seconds. The technique uses a rigid needle, preferably a specially designed IO or bone marrow needle. Use of an IO needle with stylet may be preferred to use of a needle without stylet because the stylet prevents obstruction of the needle with cortical bone during insertion. Butterfly needles and standard hypodermic needles also can be used.

Endotracheal Route

The IV and IO routes of administration are preferred over the ET route of administration during CPR. When considering use of the ET route during CPR, keep these concepts in mind:

- The optimal dose of most drugs given by the ET route is unknown.
- The typical dose of drugs administered by the ET route is 2 to 2½ times the dose given by the IV route.
- To give drugs via the ET route, dilute the dose in 5 to 10 mL of water or normal saline and inject the drug directly into the endotracheal tube. Follow with several positive-pressure breaths.
- You can give the following drugs by the ET route during cardiac arrest: atropine, vasopressin, epinephrine, and lidocaine. The memory aid NAVEL is often used to recall drugs that can be given by the ET route. Arrest drugs that can be given are: atropine (A), epinephrine (E), vasopressin (V) and lidocaine (L). “N” stands for naloxone, which is often used for respiratory depressions due to opioids. Note that the drug absorption and drug effect are much less predictable when drugs are administered by the ET rather than by the IV/IO route.

Part 2—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. Ideally only the antecubital veins should be used for drug administration during CPR.

Anatomy: Upper Extremities (Figure 27)

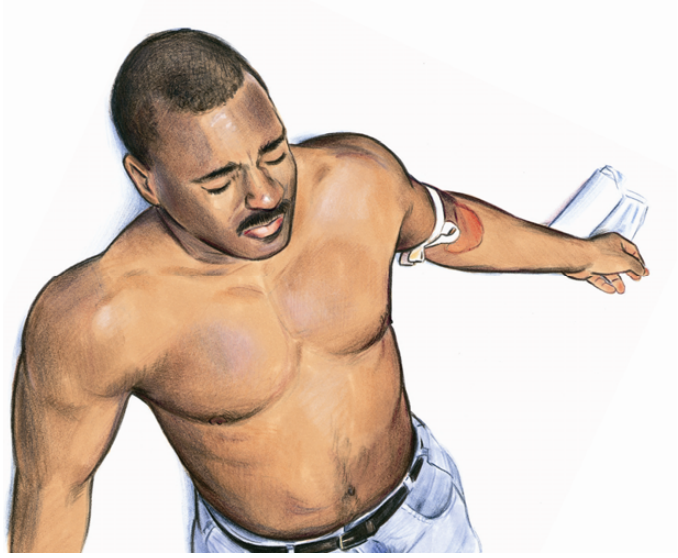
Starting at the radial side of the wrist, a thick vein, the superficial radial vein, runs laterally up to the antecubital fossa and joins the median cephalic vein to form the cephalic vein. Superficial veins on the ulnar aspect of the forearm run to the elbow and join the median basilic vein to form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

The basilic vein passes up the inner side of the arm, where it joins the brachial vein to become the axillary vein. The cephalic vein continues laterally up the arm, crosses anteriorly, and courses deep between the pectoralis major and deltoid muscles. After a sharp angulation it joins the axillary vein at a 90° angle. This sharp angulation makes the cephalic vein unsuitable for insertion of central venous pulmonary artery catheters.

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 (Figure 27). Select a point between the junctions of 2 antecubital veins. The vein is more stable here, and venipuncture is more often successful.

Self-contained kits allow easy central venous access, so today providers rarely use peripheral leg veins for vascular access.



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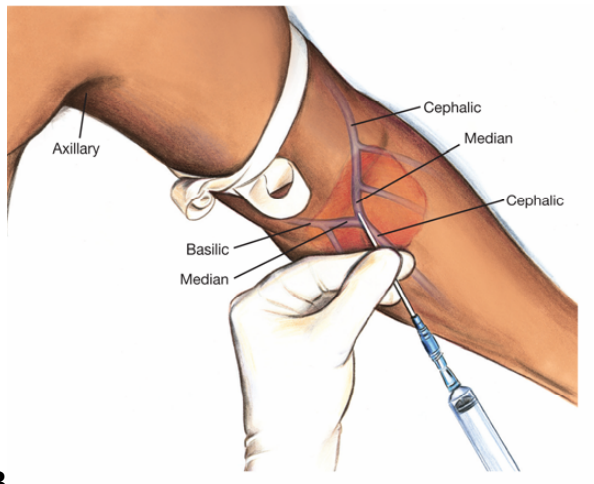

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Figure 27. Antecubital venipuncture. **A,** Scene perspective from a distance. **B,** Close-up view of antecubital area: anatomy of veins of upper extremity.

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 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, and phlebitis. Systemic complications include sepsis, pulmonary thromboembolism, air embolism, and catheter fragment embolism.

Part 3—Intraosseous Access

Introduction

When venous access cannot be rapidly achieved, intraosseous (IO) access can serve as a rapid, safe, and reliable route for administration of drugs, crystalloids, colloids, and blood. IO cannulation provides access to a noncollapsible venous plexus in bone marrow and can often be achieved in 30 to 60 seconds. This vascular access technique is suitable for all age groups, from preterm neonates through adulthood.

Needles

The technique uses a rigid needle, preferably a specially designed IO or Jamshidi-type bone marrow needle. An IO needle with stylet is preferred to one without a stylet because the stylet can prevent obstruction of the needle by cortical bone during insertion. Commercial kits with specially designed needles are available.

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 for adults, IO access is now easier to obtain in older children and adults.

Sites

Many sites are appropriate for IO infusion. For young children, the proximal tibia, just below the growth plate, is the most common site used. In older children and adults, successful IO insertion sites include the sternum, the distal tibia just above the medial malleolus, the lateral or medial malleolus, the distal radius and distal ulna, the distal femur, and the 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 following 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 5 to 10 mL of normal saline to facilitate delivery into the central circulation.
- Administer viscous drugs and solutions and fluid for rapid volume resuscitation under pressure 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.

Complications

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

Contraindications

Absolute contraindications to IO access are as follows:

- Fractures and crush injuries near the access site
- Conditions in which the bone is fragile, such as osteogenesis imperfecta
- Previous attempts to establish access in the same bone

Avoid IO cannulation if infection is present in overlying tissues.

Equipment Needed

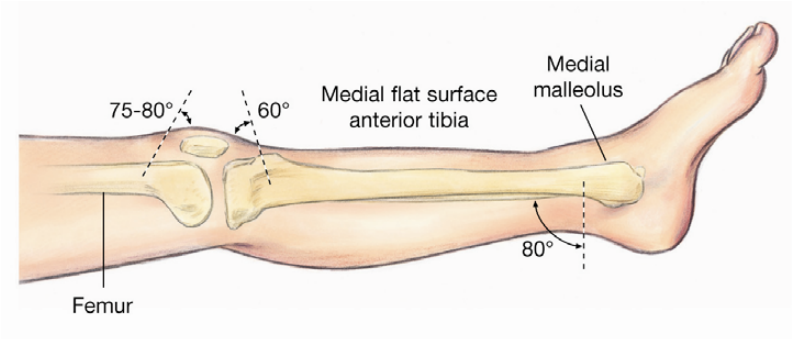
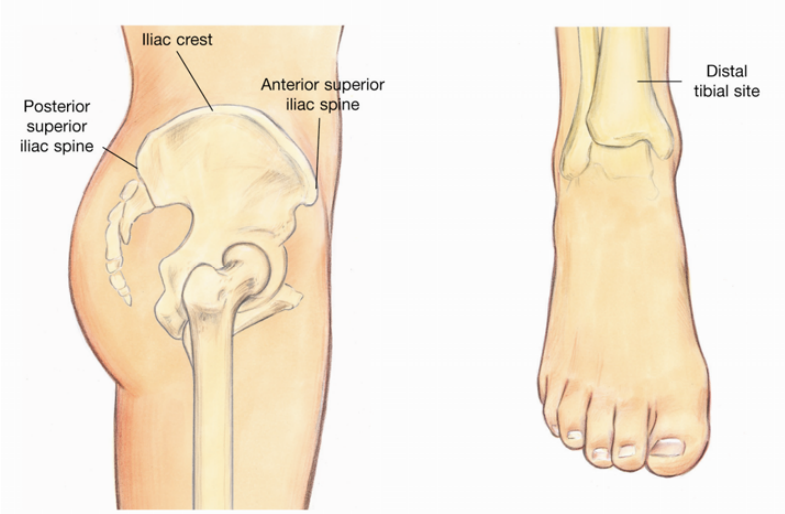
The following equipment is needed to establish IO access:

- Gloves
- Skin disinfectant
- IO needle (16 or 18 gauge) or bone marrow needle
- Tape
- Syringe
- Isotonic crystalloid fluid and intravenous tubing

Procedure

The steps to establish IO using the tibial tuberosity as an access site example are as follows:

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 28 shows 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, DON'T 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.
	A  **B** **C**  **Figure 28. A,** Locations for IO insertion in the distal tibia and the femur. **B,** Location for IO insertion in the iliac crest. **C,** Location for IO insertion in the distal tibia.

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.
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.

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 <24 hours. Replacement with long-term vascular access is usually done in the intensive care unit.

Acute Coronary Syndromes

Part 1—STEMI Infarct Location and Heart Block

Right Ventricular Infarction

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

- Give a careful fluid challenge with normal saline (250 to 500 mL based on clinical assessment). Repeat fluid administration (typically up to 1 to 2 L) if there is improvement and no symptoms or signs of heart failure or volume overload. 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 causes an increase in the strength of RV contractions (Starling mechanism), blood flow through the lungs, and ultimately LV filling pressure and cardiac output.

AV Block With Inferior MI

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

- Heart block frequently develops from excess vagal tone and atrioventricular 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 per minute. Unless a large amount of myocardium is nonfunctional or comorbid conditions exist, the patient is often stable.
- If the bradycardia is symptomatic, follow the 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 0.5 mg IV. Avoid excessive use of atropine. Use only the dose necessary to stabilize the patient. Excess atropine may increase ischemia by increasing heart rate and contractility – major determinants of myocardial oxygen consumption.
- If there is no response to atropine and TCP, follow the Bradycardia Algorithm and consider epinephrine (2 to 10 µg/min) or dopamine

(2 to 10 μg/kg per minute) infusion.

- The conduction defect is often transient. But you should keep TCP on standby for these patients.
- Evaluation of AV block with AMI can be difficult. Obtain immediate expert consultation for evaluation and recommendation (eg, transvenous temporary pacemaker).

Part 2—Fibrinolytic Checklist

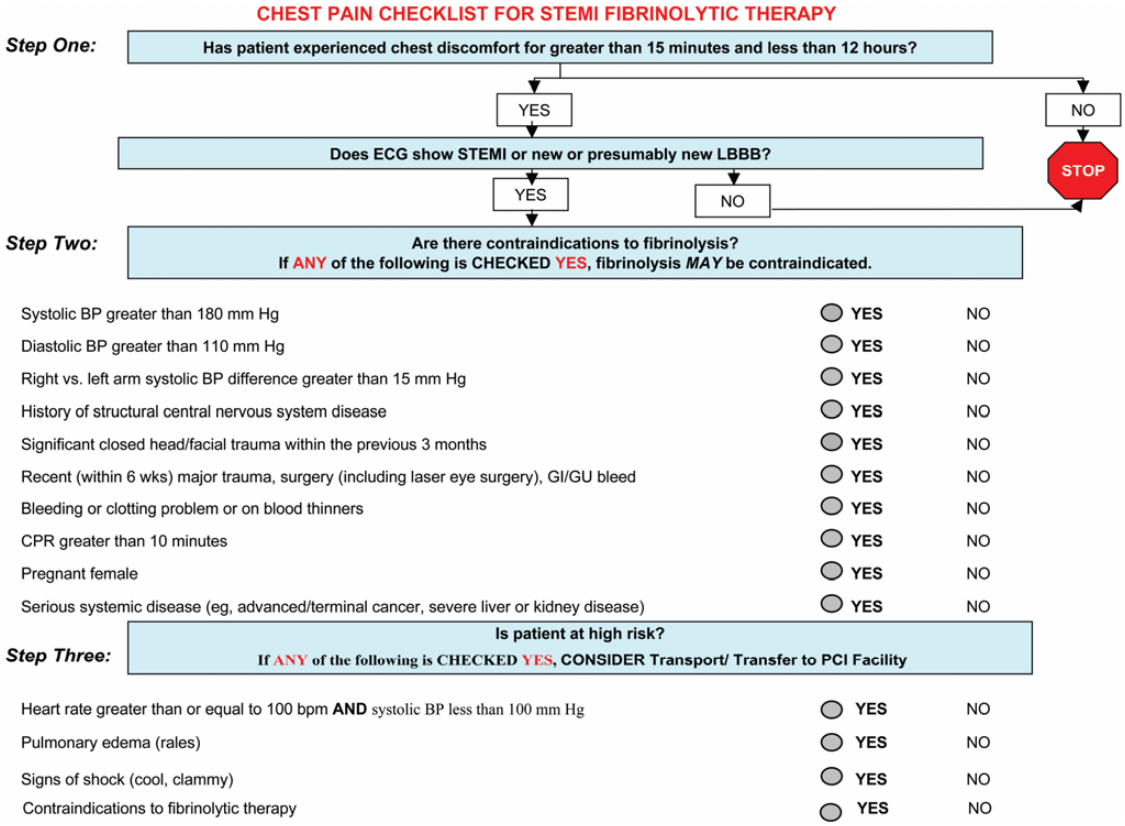


Figure 29. Fibrinolytic checklist.

Stroke

Stroke Fibrinolytic Checklist

Table 3. Fibrinolytic Checklist for Patients With Acute Ischemic Stroke

All boxes must be checked before tPA can be given.

Note: The following checklist includes FDA-approved indications and contraindications for tPA administration for acute ischemic stroke. A physician with expertise in acute stroke care may modify this list.

Inclusion Criteria *(all* ***Yes*** *boxes in this section must be checked):*

Yes

- ❑ Age 18 years or older?
- ❑ Clinical diagnosis of ischemic stroke with a measurable neurologic deficit?
- ❑ Time of symptom onset (when patient was last seen normal) well established as <180 minutes (3 hours) before treatment would begin?

Exclusion Criteria *(all* ***No*** *boxes in "Contraindications" section must be checked):*

Contraindications:

No

- ❑ Evidence of intracranial hemorrhage on pretreatment noncontrast head CT?
- ❑ Clinical presentation suggestive of subarachnoid hemorrhage even with normal CT?
- ❑ CT shows multilobar infarction (hypodensity greater than one third cerebral hemisphere)?
- ❑ History of intracranial hemorrhage?
- ❑ Uncontrolled hypertension: At the time treatment should begin, systolic pressure remains >185 mm Hg or diastolic pressure remains >110 mm Hg despite repeated measurements?
- ❑ Known arteriovenous malformation, neoplasm, or aneurysm?
- ❑ Witnessed seizure at stroke onset?
- ❑ Active internal bleeding or acute trauma (fracture)?
- ❑ Acute bleeding diathesis, including but not limited to
  - — Platelet count <100 000/mm^3?
  - — Heparin received within 48 hours, resulting in an activated partial thromboplastin time (aPTT) that is greater than upper limit of normal for laboratory?
  - — Current use of anticoagulant (eg, warfarin sodium) that has produced anelevated international normalized ratio (INR) >1.7 or prothrombin time (PT) >15 seconds?*
- ❑ Within 3 months of intracranial or intraspinal surgery, serious head trauma, or previous stroke?
- ❑ Arterial puncture at a noncompressible site within past 7 days?

Relative Contraindications/Precautions:

Recent experience suggests that under some circumstances—with careful consideration and weighing of risk-to-benefit ratio—patients may receive fibrinolytic therapy despite one or more relative contraindications. Consider the pros and cons of tPA administration carefully if any of these relative contraindications is present:

- Only minor or rapidly improving stroke symptoms (clearing spontaneously)
- Within 14 days of major surgery or serious trauma
- Recent gastrointestinal or urinary tract hemorrhage (within previous 21 days)
- Recent acute myocardial infarction (within previous 3 months)
- Postmyocardial infarction pericarditis
- Abnormal blood glucose level (<50 or >400 mg/dL [<2.8 or >22.2 mmol/L])

**In patients without recent use of oral anticoagulants or heparin, treatment with tPA can be initiated before availability of coagulation study results but should be discontinued if the INR is >1.7 or the partial thromboplastin time is elevated by local laboratory standards.*

Medical Emergency (or Rapid Response) Teams to Prevent In-Hospital Cardiac Arrest

Introduction

Mortality from in-hospital cardiac arrest remains high. The average survival rate is approximately 17% despite significant advances in treatments. Survival rates are particularly poor for arrest associated with rhythms other than ventricular fibrillation (VF)/ventricular tachycardia (VT) rhythms. Non-VF/VT rhythms are present in more than 75% of arrests in the hospital.

Many in-hospital arrests are preceded by easily recognizable physiologic changes, many of which are evident with routine monitoring of vital signs. In recent studies nearly 80% of hospitalized patients with cardiorespiratory arrest had abnormal vital signs documented for up to 8 hours before the actual arrest. This finding suggests that there is a period of increasing instability before the arrest.

Of the small percentage of in-hospital cardiac arrest patients who experience return of spontaneous circulation and are admitted to the intensive care unit, 80% ultimately die before discharge. In comparison, only 44% of nonarrest patients admitted to intensive care urgently from the floor (ie, before an arrest occurs) die before discharge.

Cardiac Arrest Teams (In Hospital)

Cardiac arrest teams are unlikely to prevent arrests because their focus has traditionally been to respond only after the arrest has occurred. Once the arrest occurs, the mortality rate is greater than 80%.

There has been a major shift in focus for in-hospital cardiac arrest over the past few years, with patient safety and prevention of arrest now the focus. The best way to improve a patient's chance of survival from a cardiorespiratory arrest is to prevent it from happening. For this reason recognizing clinical deterioration and intervening at once to prevent arrest are now being stressed. Rapid assessment and intervention for a number of abnormal physiologic variables can decrease the number of arrests occurring in the hospital. The majority of cardiorespiratory arrests in the hospital should be classified as a "failure to rescue" rather than an isolated, unexpected, random occurrence. This new thinking requires a significant cultural shift within institutions. Actions and interventions need to be proactive with the goal of improving rates of morbidity and mortality rather than reacting to a catastrophic event.

Rapid Response Systems

- **Rapid Response Team (RRT)**
- **Medical Emergency Team (MET)**

Over the past decade hospitals in several countries have designed systems to identify and treat early clinical deterioration in patients. The purpose of these rapid response systems is to improve patient outcomes by bringing critical care expertise to ward patients. There are several names for these systems, such as medical emergency team (MET), rapid response team (RRT), and rapid assessment team.

There are common basic components to all rapid response systems. Success depends on many factors. Initially success depends on activation of the MET by the floor or ward nurse or physician, who uses specific physiologic criteria to decide when to call the team. The following list gives examples of such "calling criteria" for adult patients:

- Threatened airway
- Respiratory rate <6 or >30 breaths per minute
- Heart rate <40 per minute or >140 per minute
- Systolic blood pressure <90 mm Hg
- Symptomatic hypertension
- Sudden decrease in level of consciousness
- Unexplained agitation
- Seizure
- Significant fall in urine output
- Nurse or provider concerned about patient
- Subjective criteria also may be used

The system is critically dependent on the primary nurse's identifying and acting on the specified criteria to immediately summon the MET to the patient's bedside. The MET typically consists of healthcare providers with critical care or emergency care experience and skills that support immediate intervention for critical care situations. The MET is responsible for performing a rapid patient assessment and beginning appropriate treatment to reverse physiologic deterioration and prevent a poor outcome.

Published Studies

The majority of published "before and after" studies of METs or rapid response systems have reported a 17% to 65% drop in the rate of cardiac arrests after the intervention. Other documented benefits of these systems are a decrease in unplanned emergency transfers to the intensive care unit (ICU), decreased ICU and total hospital length of stay, reductions in postoperative morbidity and mortality rates, and improved rates of survival from cardiac arrest.

The recently published MERIT trial is the only randomized controlled trial comparing hospitals with a MET and those without one. The study did not show a difference in the composite primary outcome (cardiac arrest, unexpected death, unplanned ICU admission) between the 12 hospitals in which a MET system was

introduced and 11 hospitals that had no MET system in place. Further research is needed about the critical details of implementation and the potential effectiveness of METs in preventing cardiac arrest or improving other important patient outcomes.

Implementation of a Rapid Response System

Implementing any type of rapid response system will require a significant cultural change in most hospitals. Those who design and manage the system must pay particular attention to issues that may prevent the system from being used effectively. Examples of such issues are insufficient resources, poor education, fear of calling the team, fear of losing control over patient care, and resistance from team members.

Implementation of a rapid response system or MET requires ongoing education, impeccable data collection and review, and feedback. Development and maintenance of these programs requires a long-term cultural and financial commitment from the hospital administration, which must understand that the potential benefits from the system (decreased resource use and improved survival rates) may have independent positive financial ramifications. Hospital administrators and healthcare professionals need to reorient their approach to emergency medical events and develop a culture of patient safety with a primary goal of decreasing morbidity and mortality.

Human, Ethical, and Legal Dimensions of ECC and ACLS

Part 1—Rescuer and Witness Issues

How Often Will CPR, Defibrillation, and ACLS Succeed?

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. CPR at home or in public results in return of spontaneous circulation (ROSC)—ie, even temporary return of a perfusing rhythm—only about 50% of the time.

Tragically even when ROSC occurs, only about half of VF cardiac arrest patients admitted to the emergency department and hospital survive and go home. This means that 3 of 4 prehospital CPR attempts will be "unsuccessful" in terms of neurologically intact survival to hospital discharge. Also, there is a > 80% mortality for in-hospital arrest. 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."

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 life. 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 following 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 and 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. Everyone knows to expect the "jump" and muscle contractions whenever a character yells "clear" and delivers a shock. 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 psychological barriers can hinder a quick emergency response, especially in settings where such events are rare. There are no easy solutions to help overcome these psychological 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.

Part 2—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.

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 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. Unless you are expected to perform CPR as part of your job responsibilities, you are under no *legal* obligation to attempt CPR for a patient of cardiac arrest. Failure to attempt CPR when there is no danger to the rescuer and the rescuer has the ability is not a legal violation, but it might be considered an *ethical* violation by some.

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.

Patients or families may ask physicians to provide care that is inappropriate. But physicians have no obligation to provide such care when there is scientific and social consensus that the treatment is ineffective. An example 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). Beyond these clinical circumstances, and in the absence of advance directives (including

DNAR) or living wills with statements to the contrary, 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 are 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, and initial arrest rhythm. None of these factors alone or in combination is clearly predictive of outcome. *The most important factor associated with poor outcome in adults with normothermic cardiac arrest is the duration of resuscitative efforts.* The chance of discharge from the hospital alive and neurologically intact diminishes as resuscitation time increases. The responsible clinician should stop the resuscitation when he or she determines with a high degree of certainty that the patient will not respond to further ACLS efforts.

In the absence of mitigating factors (eg, drug toxicity, hypothermia), prolonged resuscitative efforts are unlikely to be successful. If ROSC of any duration occurs, it may be appropriate to extend resuscitative efforts. It is important to consider the circumstances of the cardiac arrest (eg, drug overdose or submersion in icy water) when deciding whether to continue resuscitative efforts.

For the newly born infant, discontinuation of resuscitation can be justified after 10 minutes with no signs of life despite continuous and adequate resuscitative efforts. The prognosis for survival or survival without disability has been shown to be extremely poor when there is lack of response to intensive resuscitative efforts for >10 minutes.

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

- The patient has a valid Do Not Attempt Resuscitation (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 CPR for newly born infants in the delivery room may be appropriate under circumstances such as the following:

- Confirmed gestation <23 weeks
- Birth weight <400 g
- Confirmed anencephaly
- Confirmed trisomy 13
- Other congenital anomalies that are incompatible with life

Withholding vs Withdrawing CPR

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.

Some patients do not regain consciousness after cardiac arrest and (ROSC). In most cases the prognosis for adults who remain deeply comatose (Glasgow Coma Scale score <5) after cardiac arrest can be predicted with accuracy within 2 to 3 days of resuscitation. Specific physical findings or laboratory tests may be helpful to assist with this process. 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.

Advance Directives, Living Wills, and Patient Self-Determination

An advance directive is any expression of a person's thoughts, wishes, or preferences for his or her 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 he or she becomes 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. 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

Many patients for whom 911 is called because of cardiac arrest are chronically ill, have a terminal illness, or have a written advance directive (DNAR order). States and other jurisdictions 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 his or her mind
- The best interests of the patient are in question

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

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

EMS No-CPR Programs

A number of states have adopted "no-CPR" programs. These programs allow patients and family members to call 911 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.

Transport

If an EMS system does not allow nonphysicians to pronounce death and stop all resuscitative efforts, personnel may be forced to transport a deceased patient of cardiac arrest to the hospital. Such an action is unethical. If carefully executed BLS and ACLS treatment protocols fail in the out-of-hospital setting, then how could the same treatment succeed in the emergency department? A number of studies have consistently shown that <1% of patients transported with continuing CPR survive to hospital discharge.

Delayed or token efforts to provide CPR and ACLS—or so-called “slow codes” (knowingly providing ineffective resuscitation)—are *inappropriate*. These practices compromise the ethical integrity of healthcare providers and undermine the provider-patient relationship.

Many EMS systems authorize the termination of a resuscitation attempt in the out-of-hospital setting. EMS systems should establish protocols for pronouncement of death and appropriate transport of the body. EMS systems should also train personnel to deal sensitively with family and friends.

Legal Aspects of AED Use

Defibrillators, including many AEDs, are restricted medical devices. 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. 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). By the year 2000 plaintiffs and attorneys had started filing lawsuits against some facilities for failing to train and equip their employees to perform CPR and use an AED, but as of 2005 no lawsuits were identified involving a lawsuit for an attempted resuscitation in which a lay rescuer used an AED.

Some states grant limited immunity for lay rescuer use of AEDs only when specific recommendations are fulfilled. These recommendations may require that the rescuer must

- Have formal training in CPR and use of an AED (eg, the AHA Heartsaver AED Course or equivalent)
- Use treatment protocols approved by a recognized medical authority
- Perform routine checks and maintenance of the AED as specified by the manufacturer
- Notify local EMS authorities of the placement of the AED so that EMS personnel, particularly the dispatchers, will know when emergency calls are

made from a setting with an AED

The AHA recently published a statement detailing recommended legislation to promote lay rescuer CPR and AED programs and to assist legislators and policymakers in removing impediments to these programs: (http://circ.ahajournals.org/cgi/reprint/CIRCULATIONAHA.106.172289v1)

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

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Part 3—Providing Emotional Support for the Family

Notifying Survivors of the Death of a Loved One

Despite our best efforts, most resuscitation attempts fail. Notifying the family of the death of a loved one is an important aspect of resuscitation. It should be done compassionately, with sensitivity to the cultural and religious beliefs and practices of the family.

Family members have often been excluded from the resuscitation of a loved one. Surveys suggest that healthcare providers hold a range of opinions concerning the presence of family members during a resuscitation attempt. Several commentaries have expressed concern that family members may interfere with procedures or faint. Exposure of the institution and providers to legal liability is another concern.

But several surveys conducted before resuscitative efforts were observed showed that most family members wished to be present during a resuscitation attempt. Family members have reported that being at a loved one's side and saying goodbye during their final moments of life was comforting. In addition, being present during the resuscitation attempt helped them adjust to the death of their loved one, and most indicated they would attend again. Several retrospective reports note positive reactions from family members, many of whom said that they felt a sense of having helped their loved one and of easing their own grieving process. Most parents wanted to be given the option to decide whether to be present at the resuscitation of a child.

Given the absence of data suggesting that family presence is harmful, and in light of data suggesting that it may be helpful, it seems reasonable to offer selected relatives the option to be present during a resuscitation attempt. This recommendation assumes that the patient, if an adult, has not previously raised an objection. Parents seldom ask if they can be present unless encouraged to do so by healthcare providers.

Resuscitation team members should be sensitive to the presence of family members. It is helpful to have one team member available to answer questions from the family, clarify information, and otherwise offer comfort.