CHAPTER 55

Cardiopulmonary Resuscitation

Martin Giesecke, MD and Srikanth Hosur, MBBS, MD

KEY CONCEPTS

1. Cardiopulmonary resuscitation and emergency cardiac care should be considered any time an individual cannot adequately oxygenate or perfuse vital organs—not only following cardiac or respiratory arrest.
2. Regardless of which transtracheal jet ventilation system is chosen, it must be readily available, use low-compliance tubing, and have secure connections.
3. Chest compressions and ventilation should not be delayed for intubation if a patent airway is established by a jaw-thrust maneuver.
4. Attempts at intubation should not interrupt ventilation for more than 10 s.
5. Chest compressions should be immediately initiated in the pulseless patient.
6. Whether adult resuscitation is performed by a single rescuer or by two rescuers, two breaths are administered every 30 compressions (30:2), allowing 3–4 s for each two breaths. The cardiac compression rate should be 100/min regardless of the number of rescuers.
7. Health care personnel working in hospitals and ambulatory care facilities must be able to provide early defibrillation to collapsed patients with ventricular fibrillation as soon as possible. Shock should be delivered within 3 min (± 1 min) of arrest.
8. Lidocaine, epinephrine, atropine, naloxone, and vasopressin, but not sodium bicarbonate, can be delivered via a catheter whose tip extends past the tracheal tube. Dosages 2–2½ times higher than recommended for intravenous use, diluted in 10 mL of normal saline or distilled water, are recommended for adult patients.
9. If intravenous cannulation is difficult, an intraosseous infusion can provide emergency vascular access in children.
10. Because carbon dioxide, but not bicarbonate, readily crosses cell membranes and the blood–brain barrier, the resulting arterial hypercapnia will cause intracellular tissue acidosis.
11. A wide QRS complex following a pacing spike signals electrical capture, but mechanical (ventricular) capture must be confirmed by an improving pulse or blood pressure.

One goal of anesthesiology is to maintain the function of vital organ systems during surgery. It is not surprising, therefore, that anesthesiologists have played a major role in the development of cardiopulmonary resuscitation techniques outside the operating room.

1 Cardiopulmonary resuscitation and emergency cardiac care (CPR-ECC) should be considered any time an individual cannot adequately oxygenate or perfuse vital organs—not only following cardiac or respiratory arrest.

TABLE 55–1 Emergency cardiac care (ECC).

1. Recognition of impending event
2. Activation of emergency response system
3. Basic life support
4. Defibrillation
5. Ventilation
6. Pharmacotherapy

This chapter presents an overview of the American Heart Association (AHA) and the International Liaison Committee on Resuscitation (ILCOR) Year 2010 recommendations for establishing and maintaining the ABCDs of cardiopulmonary resuscitation: Airway, Breathing, Circulation, and Defibrillation (**Table 55–1**, **Figures 55–1** and **55–2**). The 2010 CPR-ECC guidelines have been updated with new evidence-based recommendations. Still of import to the layperson are that the pulse should not be checked, and chest compression without ventilation may be as effective as compression with ventilation for the first several minutes. If a lay rescuer is unwilling to perform mouth-to-mouth ventilation, chest compressions alone are preferred to doing nothing. For the health care provider, defibrillation using biphasic electrical current works best and tracheal tube (TT) placement should be confirmed with a quantitative capnographic waveform analysis. More importantly, in the new guidelines, emphasis has been placed on the quality and adequacy of compressions, minimizing interruption time of compressions and the preshock pause (the time taken from the last compression to the delivery of shock).

The sequence of steps in resuscitation has been changed in the 2010 guidelines from *ABC* (airway and breathing first, before compression) to *CAB* (compression first, with airway and breathing treated later). Emphasis has also been placed on physiological monitoring methods to optimize CPR quality and return of spontaneous circulation (ROSC). The rule of tens and multiples can be applied: less than 10 s to check for pulse, less than 10 s to place and secure the airway, target chest compression adequacy to maintain end-tidal pressure of carbon dioxide (P_{ETCO_2}) greater than 10, and target chest compression to maintain arterial diastolic blood pressure greater than 20 and central venous oxygen saturation (S_{CVO_2}) greater than 30.

Changes in drug recommendations are notable for exclusion of atropine in the settings of pulseless electrical activity (PEA) and asystole, addition of the use of chronotropic drug infusions as an alternative to pacing in unstable/symptomatic bradycardia, and recommendation for use of adenosine in the management of wide-complex monomorphic tachycardia.

This chapter is not intended as a substitute for a formal course in either life support without the use of special equipment (Basic Life Support [BLS]) or with the use of special equipment and drugs (Advanced Cardiac Life Support [ACLS]). The recommendations described are for infants, children, and adults; resuscitation of neonates is discussed in Chapter 42.

AIRWAY

Although the *A* of the mnemonic *ABC* stands for *airway,* it should also stand for the initial *assessment* of the patient. Before CPR is initiated, unresponsiveness is established and the emergency response system is activated. During low blood flow states such as cardiac arrest, oxygen delivery to the heart and brain is limited by blood flow rather than by arterial oxygen content; thus, in the new guidelines, greater emphasis is placed on immediate initiation of chest compressions than on rescuer breaths.

The patient is positioned supine on a firm surface. After initiation of chest compressions, the airway is evaluated. The airway is most commonly obstructed by posterior displacement of the tongue or epiglottis. If there is no evidence of cervical spine instability, a head-tilt chin-lift should be tried first (**Figure 55–3**). One hand (palm) is placed on the patient's forehead applying pressure to tilt the head back while lifting the chin with the forefinger and index finger of the opposite hand. The jaw-thrust may be more effective in opening the airway and is executed by placing both hands on either side of the patient's head, grasping the angles of the jaw, and lifting. Basic airway management is discussed in detail in Chapter 19, and the trauma patient is considered in Chapter 39.

Any vomitus or foreign body visible in the mouth of an unconscious patient should be removed. If the patient is conscious or if the foreign body

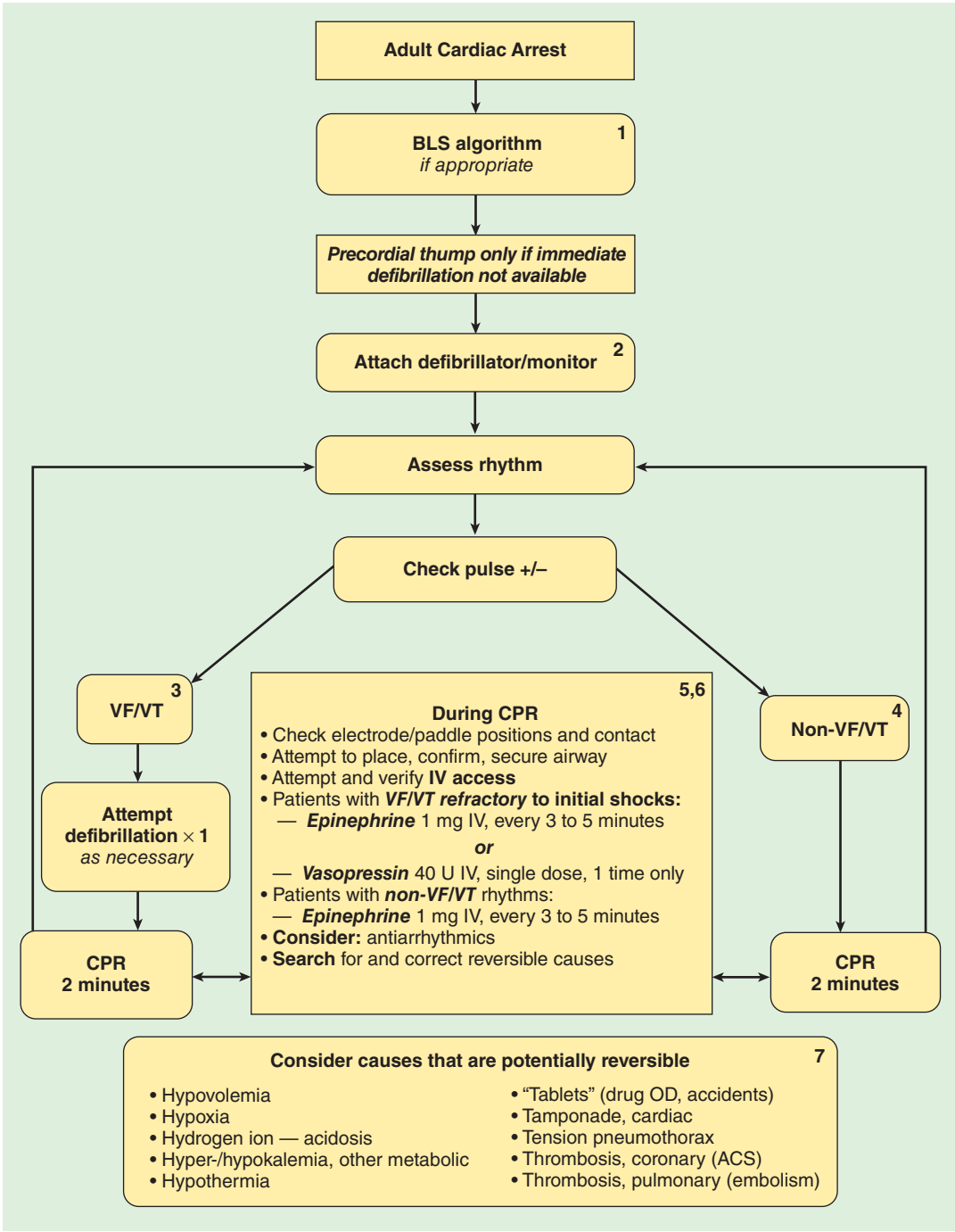


FIGURE 55–1 Universal algorithm for adult emergency cardiac care. BLS, basic life support; VF/VT, ventricular fibrillation and pulseless ventricular tachycardia; CPR, cardiopulmonary resuscitation.

(Data from The American Heart Association BLS and ACLS Guidelines 2010 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010;122:S685.)

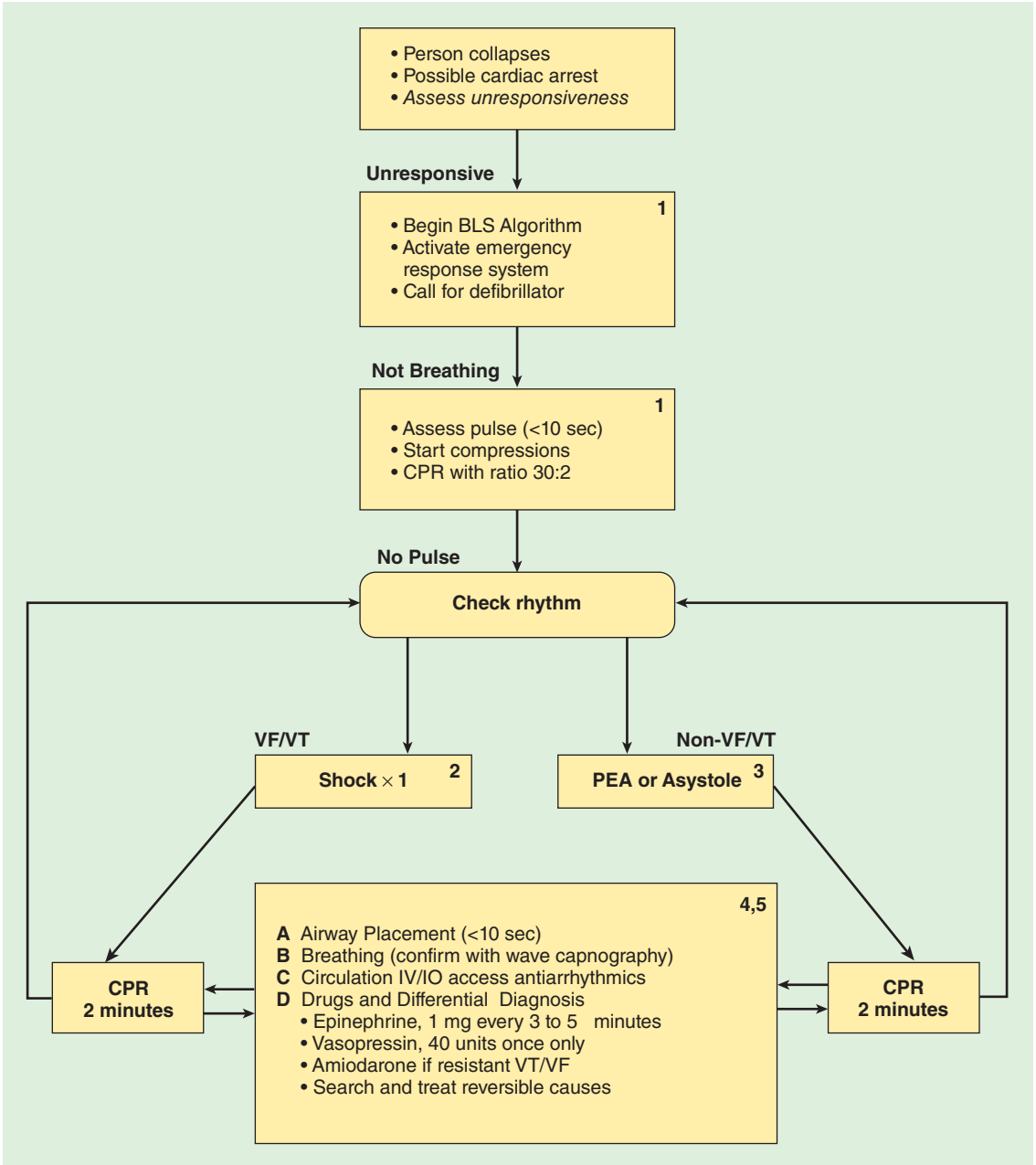


FIGURE 55–2 Comprehensive emergency cardiac care algorithm. BLS, basic life support; VF/VT, ventricular fibrillation and pulseless ventricular tachycardia; PEA, pulseless electrical activity; CPR, cardiopulmonary resuscitation. (Data from The American Heart Association BLS and ACLS Guidelines 2010 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010;122:S685.)

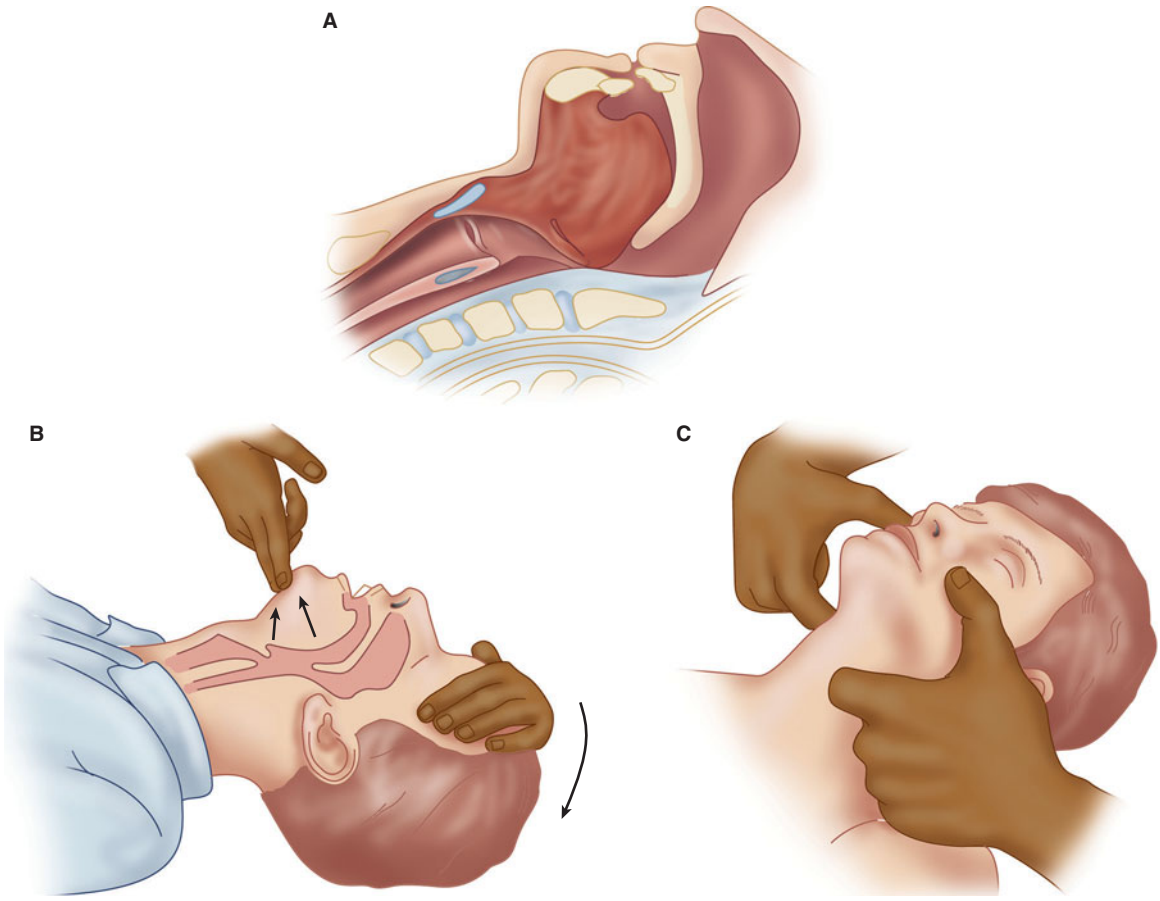


FIGURE 55–3 Loss of consciousness is often accompanied by loss of submandibular muscle tone (**A**). Occlusion of the airway by the tongue can be relieved by a head-tilt chin-lift (**B**) or a jaw-thrust (**C**). In patients with possible cervical spine injury, the angles of the jaw should be lifted anteriorly without hyperextending the neck.

cannot be removed by a finger sweep, the Heimlich maneuver is recommended. This subdiaphragmatic abdominal thrust elevates the diaphragm, expelling a blast of air from the lungs that displaces the foreign body (Figure 55–4). Complications of the Heimlich maneuver include rib fracture, trauma to the internal viscera, and regurgitation. A combination of back blows and chest thrusts is recommended to clear foreign body obstruction in infants (Table 55–2).

If after opening the airway there is no evidence of adequate breathing, the rescuer should initiate assisted ventilation, by inflating the victim's lungs with each breath using mouth-to-mouth, mouth-to-nose, mouth-to-stoma, mouth-to-barrier device, mouth-to-face shield, or mouth-to-mask rescue breathing or by using a bag-mask device (see Chapter 19). Breaths are delivered slowly (inspiratory time of ½–1 s) with a smaller tidal volume [VT] (approximately 700–1000 mL, smaller [400–600 mL] if supplemental O_2 is used) than was recommended in the past.

With positive-pressure ventilation, even with a small VT, gastric inflation with subsequent regurgitation and aspiration are possible. Therefore, as soon as it is feasible, the airway should be secured with a TT, or, if that is not possible, an alternative airway should be inserted. There is inadequate evidence to support the optimal timing of the placement of an artificial airway; however, chest compressions

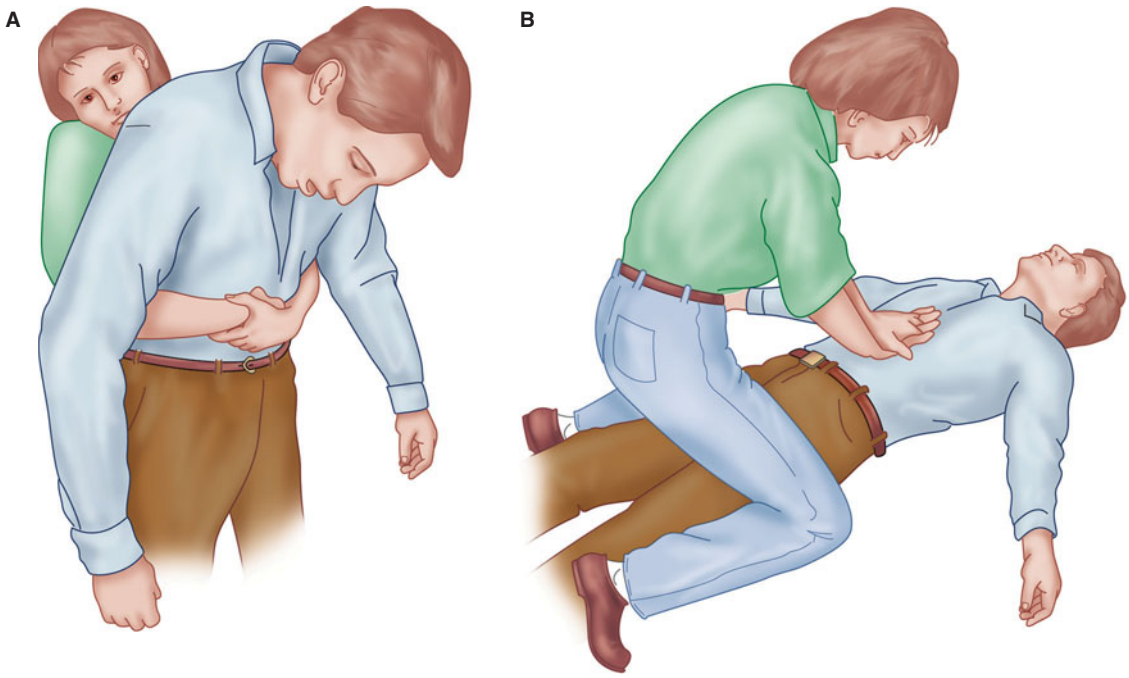


FIGURE 55–4 The Heimlich maneuver can be performed with the victim standing (**A**) or lying down (**B**). The hands are positioned slightly above the navel and well below the xiphoid process and then pressed into the abdomen with a quick upward thrust. The maneuver may need to be repeated.

should not be interrupted for more than 10 seconds to place any airway. Alternative airways include the esophageal–tracheal Combitube (ETC), laryngeal mask airway (LMA), pharyngotracheal lumen airway, King laryngeal tube, and cuffed oropharyngeal airway. The ETC and LMA, along with oral and nasopharyngeal airways, face masks, laryngoscopes, and TTs, are discussed in Chapter 19. Of these, the LMA is increasingly preferred for in-hospital arrests. The 2010 CPR-ECC guidelines recommend a TT as the airway adjunct of choice if personnel skilled in placing it are available.

Independent of which airway adjunct is used, the guidelines state that rescuers must confirm TT

TABLE 55–2 Summary of recommended basic life support techniques.

	Infant (1–12 mo)	Child (>12 mo)	Adult
Breathing rate	20 breaths/min	20 breaths/min	10–12 breaths/min[1]
Pulse check	Brachial	Carotid	Carotid
Compression rate	>100/min	100/min	100/min
Compression method	Two or three fingers	Heel of one hand	Hands interlaced
Compression/ventilation ratio	30:2	30:2	30:2
Foreign body obstruction	Back blows and chest thrusts	Heimlich maneuver	Heimlich maneuver

[1]Decrease to 8–10 breaths/min if the airway is secured with a tracheal tube.

placement with a P_{ETCO_2} detector—an indicator, a capnograph, or a capnometric device. The best choice for confirmation of TT placement is continuous capnographic waveform analysis. All confirmation devices are considered adjuncts to clinical conformation techniques (eg, auscultation). Once an artificial airway is successfully placed, *it must be carefully secured with a tie or tape* (25% of airways are displaced during transportation).

Some causes of airway obstruction may not be relieved by conventional methods. Furthermore, tracheal intubation may be technically impossible to perform (eg, severe facial trauma), or repeated attempts may be unwise (eg, cervical spine trauma). In these circumstances, cricothyrotomy or tracheotomy may be necessary. Cricothyrotomy involves placing a large intravenous catheter or a commercially available cannula into the trachea through the midline of the cricothyroid membrane (**Figure 55–5**). Proper location is confirmed by aspiration of air. A 12- or 14-gauge catheter requires a driving pressure of 50 psi to generate sufficient gas flow (for transtracheal jet ventilation). The catheter must be adequately secured to the skin, as the jet ventilation pressure can otherwise easily propel the catheter out of the trachea.

Various systems are available that connect a high-pressure source of oxygen (eg, central wall oxygen, tank oxygen, or the anesthesia machine fresh gas outlet) to the catheter (**Figure 55–6**). A hand-operated jet injector or the oxygen flush valve of an anesthesia machine controls ventilation. The addition of a pressure regulator minimizes the risk of barotrauma.

2 Regardless of which transtracheal jet ventilation system is chosen, it must be readily available, use low-compliance tubing, and have secure connections. Direct connection of a 12- or 14-gauge intravenous catheter to the anesthesia circle system does not allow adequate ventilation because of the high compliance of the corrugated breathing tubing and breathing bag. One cannot reliably deliver acceptable ventilation through a 12- or 14-gauge catheter with a self-inflating resuscitation bag.

Adequacy of ventilation—particularly expiration—is judged by observation of chest wall movement and auscultation of breath sounds. Acute complications include pneumothorax, subcutaneous emphysema, mediastinal emphysema, bleeding, esophageal puncture, aspiration, and respiratory acidosis. Long-term complications include tracheomalacia, subglottic stenosis, and vocal cord changes. Cricothyrotomy is not generally recommended in children younger than 10 years of age.

Tracheotomy can be performed in a more controlled environment after oxygenation has been restored by cricothyrotomy. A detailed description of tracheotomy, however, is beyond the scope of this text.

BREATHING

Assessment of spontaneous breathing should immediately follow the opening or the establishment of

3 the airway. Chest compressions and ventilation should not be delayed for intubation if a patent airway is established by a jaw-thrust maneuver. Apnea is confirmed by lack of chest movement, absence of breath sounds, and lack of airflow. Regardless of the airway and breathing methods employed, a specific regimen of ventilation has been proposed for the apneic patient. Initially, two breaths are slowly administered (2 s per breath in adults, 1–1½ s in infants and children). If these breaths cannot be delivered, either the airway is still obstructed and the head and neck need repositioning or a foreign body is present that must be removed.

Mouth-to-mouth or mouth-to-mask (mouth-to-barrier-device) rescue breathing should be instituted in the apneic patient, even in the hospital setting when the crash cart is on its way. Pinching the nose allows formation of an airtight seal between the rescuer's lips and the outside of the victim's mouth. Successful rescue breathing (700–1000 mL V_T, 8–10 times per minute in an adult with a secured airway and a ratio of 30 compressions to 2 ventilations if the airway is unsecured) is confirmed by observing the chest rising and falling with each breath and hearing and feeling the escape of air during expiration. The most common cause of inadequate mouth-to-mouth ventilation is insufficient airway control. Mouth-to-mouth-and-nose breathing is more effective in infants and small children than in adults.

A rescuer's exhaled air has an oxygen concentration of only 16–17% and contains significant CO_2.

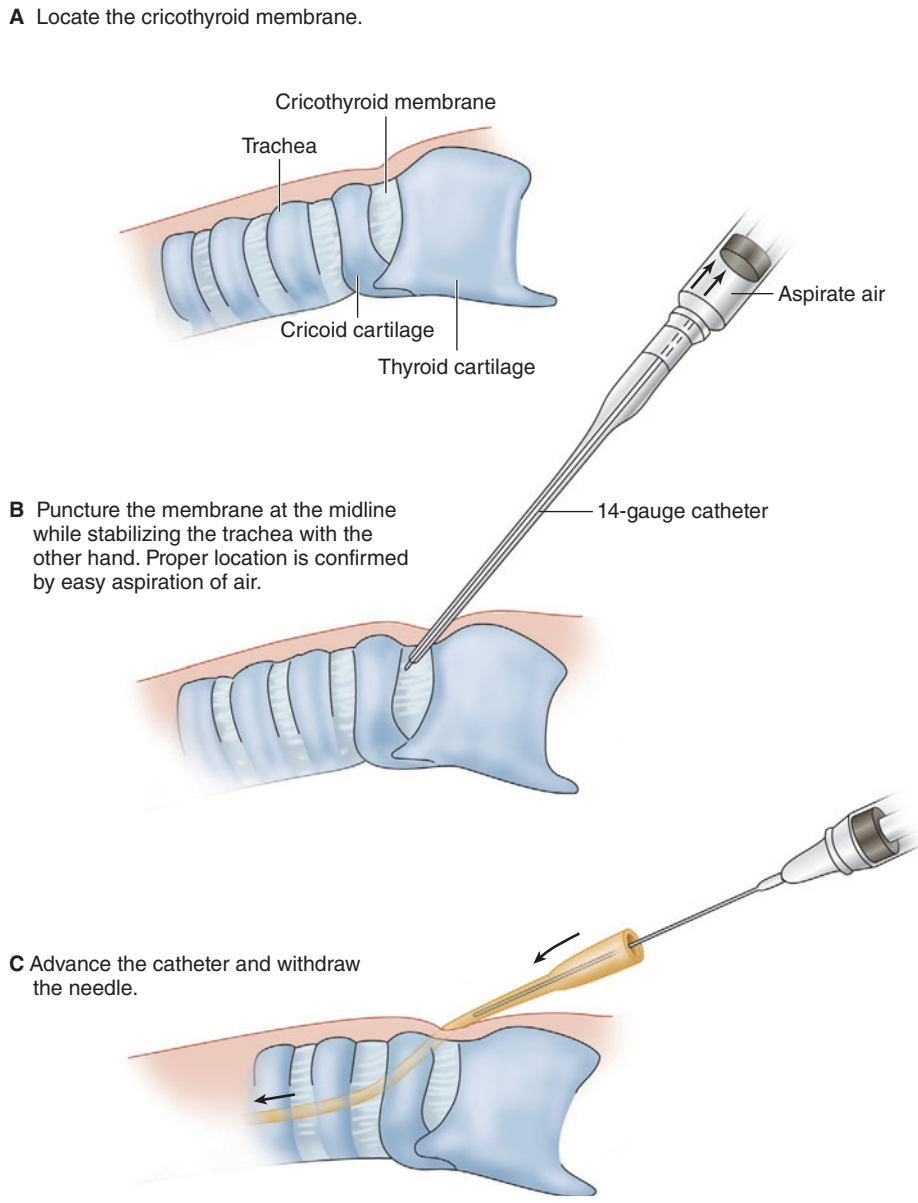


FIGURE 55–5 Percutaneous cricothyrotomy with a 14-gauge over-the-needle intravenous catheter.

Supplemental oxygen, preferably 100%, should always be used if available. If supplemental oxygen is used, a smaller VT of 400–700 mL is recommended.

Mouth-to-mask or barrier device breathing has a hygienic advantage over mouth-to-mouth breathing as the rescuer's lips form a seal with an intervening device. Devices that avoid mouth-to-mouth contact should be immediately available everywhere in the hospital. Ventilation with a mask may be performed more easily in some patients because the rescuer may be able to adjust the airway or make an airtight seal more effectively. Furthermore, some mouth-to-mask devices allow the delivery of supplemental oxygen.

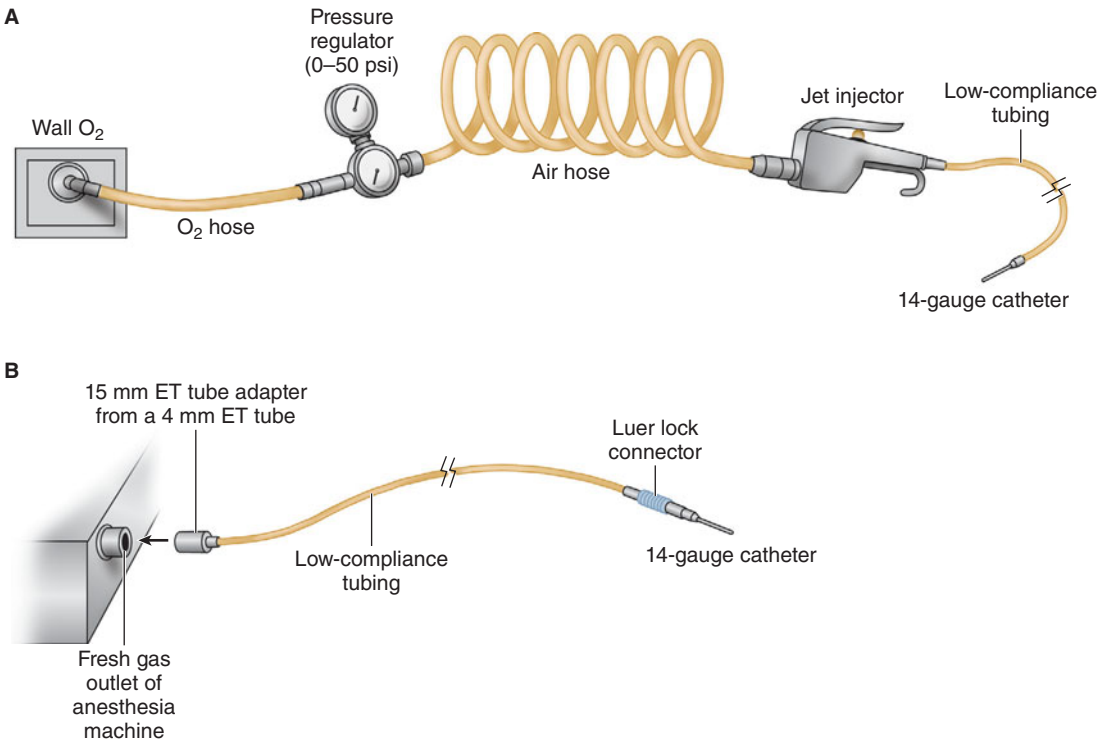


FIGURE 55–6 **A,B**: Two systems for transtracheal jet ventilation after cricothyrotomy (see Figure 55–5). A jet ventilator and pressure regulator (as shown in A) provide better control of the inspiratory cycle. Both systems use low-compliance tubing and a high-pressure source of oxygen.

A self-inflating bag-valve-mask device is described in Chapter 3 (see the section on Resuscitation Breathing Systems). These devices can be less effective than mouth-to-mask or bag-valve-TT ventilation because of the difficulty inexperienced personnel may have in maintaining an airway and seal with one hand and simultaneously delivering an adequate VT with the other. Use of cricoid pressure to prevent regurgitation during cardiac arrest resuscitation may be considered; however, there are no data to support its efficacy in this circumstance and its routine use is not recommended in the new guidelines.

Tracheal intubation should be attempted as soon as practical. Attempts at intubation should not interrupt ventilation for more than 10 s. After intubation, the patient can be ventilated with a self-inflating bag capable of delivering high oxygen concentrations. Because two hands are now available to squeeze the bag, ventilation should be satisfactory. A ratio of 8–10 breaths/min in a secure airway should be maintained, as high respiratory rates can impede cardiac output in a cardiac arrest situation.

The ratio of physiological dead space to tidal volume (VD/VT) reflects the efficiency of CO_2 elimination. VD/VT increases during CPR as a result of low pulmonary blood flow and high alveolar pressures. Thus, minute ventilation may need to be increased by 50–100% once circulation is restored as CO_2 from the periphery is brought back to the lungs.

CIRCULATION

Circulation takes precedence over airway and breathing in a cardiac arrest situation. In this scenario, as previously noted, *chest compressions should begin prior to the initial breaths.* Subsequent actions to assess circulation may then vary depending on whether the responder is a lay person

or health care provider. Although lay rescuers should assume that an unresponsive patient is in cardiac arrest and need not check pulse; health care providers should assess for presence or absence of a pulse.

After successful delivery of two initial breaths (each 2 s in duration), circulation is rapidly assessed. If the patient has an adequate pulse (carotid artery in an adult or child, brachial or femoral artery in an infant) or blood pressure, breathing is continued at 10–12 breaths/min for an adult or a child older than 8 years, and 20 breaths/min for an infant or a child younger than 8 years of age (Table 55–2). If the patient is pulseless or severely hypotensive, the circulatory system must be supported by a combination of external chest compressions, intravenous drug administration, and defibrillation when appropriate. Initiation of chest compressions is mandated by the inadequacy of peripheral perfusion, and drug choices and defibrillation energy levels often depend on electrocardiographic diagnosis of arrhythmias.

External Chest Compression

Chest compressions force blood to flow either by increasing intrathoracic pressure (thoracic pump) or by directly compressing the heart (cardiac pump). During CPR of short duration, the blood flow is created more by the cardiac pump mechanism; as CPR continues, the heart becomes less compliant and the thoracic pump mechanism becomes more important. As important as the rate and force of compression are for maintaining blood flow, effective perfusion of the heart and brain is best achieved when chest compression consumes 50% of the duty cycle, with the remaining 50% devoted to the relaxation phase (allowing blood return into the chest and heart).

To perform chest compressions in the unresponsive or pulseless patient, the xiphoid process is located and the heel of the rescuer's hand is placed over the lower half of the sternum. The other hand is placed over the hand on the sternum with the fingers either interlaced or extended, but off the chest. The rescuer's shoulders should be positioned directly over the hands with the elbows locked into position and arms extended, so that the weight of the upper body is used for compressions. With a straight downward thrust, the sternum is depressed 1½–2 in. (4–5 cm) in adults, 1–1½ in. (2–4 cm) in children, and then allowed to return to its normal position. For an infant, compressions ½–1 in. (1½–2½ cm) in depth are made with the middle and ring fingers on the sternum one finger-breadth below the nipple line. Compression and release times should be equal.

Whether adult resuscitation is performed by a single rescuer or by two rescuers, two breaths are administered every 30 compressions (30:2), allowing 3–4 s for the two breaths. The cardiac compression rate should be 100/min regardless of the number of rescuers. A slightly higher compression rate of more than 100/min is suggested for infants, with two breaths delivered every 30 compressions.

Assessing the Adequacy of Chest Compressions

Cardiac output can be estimated by monitoring end-tidal CO_2 (P_{ETCO_2} >10 mm Hg, S_{CVO_2} >30%) or arterial pulsations (with an arterial diastolic relaxation pressure >20 mm Hg). Arterial pulsations during resuscitation are not a good measure of adequate chest compression; however, spontaneous arterial pulsations are an indicator of ROSC. There is new emphasis in the 2010 guidelines on physiological parameters, such as P_{ETCO_2}, S_{CVO_2}, and diastolic arterial pressure, to assess the adequacy of chest compressions.

1. P_{ETCO_2}—In an intubated patient, a P_{ETCO_2} greater than 10 mm Hg indicates good-quality chest compressions; a P_{ETCO_2} less than 10 mm Hg has been shown to be a predictor of poor outcomes of CPR (decreased chance of ROSC). A transient increase in P_{ETCO_2} may be seen with administration of sodium bicarbonate; however, an abrupt and sustained rise of P_{ETCO_2} is an indicator of ROSC.

2. Coronary perfusion pressure (CPP)—This is the difference between the aortic diastolic pressure and the right atrial diastolic pressure. Arterial diastolic pressure in the radial, brachial, or femoral artery is a good indicator of CPP. Arterial diastolic pressure greater than 20 mm Hg is an indicator of adequate chest compressions.

3. S_{CVO_2}—An S_{CVO_2} less than 30% in the jugular vein is associated with poor outcomes. If the S_{CVO_2} is less than 30%, attempts to improve the quality of CPR, either by improving the quality of compressions or

through administration of medications, should be considered.

DEFIBRILLATION

Ventricular fibrillation develops most commonly in adults who experience nontraumatic cardiac arrest. The time from collapse to defibrillation is the most important determinant of survival. The chances for survival decline 7–10% for every minute without defibrillation (Figure 55–7). Therefore, patients who have cardiac arrest should be defibrillated at the earliest possible moment. Health care personnel working in hospitals and ambulatory care facilities must be able to provide early defibrillation to collapsed patients with ventricular fibrillation as soon as possible. Shock should be delivered within 3 min (±1 min) of arrest.

There is no definite relationship between the energy requirement for successful defibrillation and body size. A shock with too low an energy (current) level will not successfully defibrillate; conversely, too high an energy level may result in functional and morphological injury. Defibrillators deliver energy in either monophasic or biphasic waveforms. Increasingly, biphasic waveforms are recommended for cardioversion as they achieve the same degree of success but with less energy and theoretically less myocardial damage.

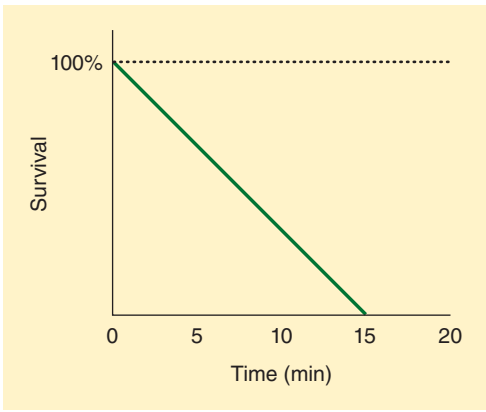


FIGURE 55–7 Success of defibrillation versus time. The chance of successful defibrillation of a patient in ventricular fibrillation decreases 7–10% per minute.

In many institutions, automated external defibrillators (AEDs) are available. Such devices are increasingly being used throughout the community by police, firefighters, security personnel, sports marshals, ski patrol members, and airline flight attendants, among others. They are placed in any public location where 20,000 or more people pass by every day. AEDs are technologically advanced, microprocessor-based devices that are capable of electrocardiographic analysis with very high specificity and sensitivity in differentiating shockable from nonshockable rhythms. All AEDs manufactured today deliver some type of biphasic waveform shock. Compared with monophasic shocks, biphasic shocks deliver energy in two directions with equivalent efficacy at lower energy levels and possibly with less myocardial injury. These devices deliver impedance-compensating shocks employing either biphasic truncated exponential (BTE) or rectilinear (RBW) morphology. Biphasic shocks delivering low energy for defibrillation (120–200 joule [J]) have been found to be as or more effective than 200–360 J monophasic damped sine (MDS) waveform shocks. When using AEDs, one electrode pad is placed beside the upper right sternal border, just below the clavicle, and the other pad is placed just lateral to the left nipple, with the top of the pad a few inches below the axilla.

A decrease in time delay between the last compression and the delivery of a shock (the preshock pause) has received special emphasis in the new guidelines. Stacking shocks increases the time to next compression, and it has been noted that the first shock is usually associated with a 90% efficacy. Thus, stacked shocks have been replaced by a recommendation for a single shock, followed by immediate resumption of chest compressions.

For cardioversion of atrial fibrillation (Table 55–3), 120–200 J can be used initially with escalation if needed. For atrial flutter or paroxysmal supraventricular tachycardia (PSVT), an initial energy level of 50–100 J is often adequate. All monophasic shocks should start with 200 J.

Ventricular tachycardia, particularly monomorphic ventricular tachycardia, responds well to shocks at initial energy levels of 100 J. For polymorphic ventricular tachycardia or for ventricular fibrillation, initial energy can be set at 120–200 J,

TABLE 55–3 Energy requirements for cardioversion using biphasic truncated exponential (BTE) or rectilinear morphology.[1]

Indications	Shocks (J)
Unstable atrial fibrillation	120–200
Unstable atrial flutter/tachycardia	50–100
Monomorphic ventricular tachycardia	100
Polymorphic ventricular tachycardia or ventricular fibrillation	120–200

depending upon the type of biphasic waveform being used. Stepwise increases in energy levels should be used if the first shock fails, although some AEDs operate with a fixed-energy protocol of 150 J with very high success in terminating ventricular fibrillation (Table 55–3).

Cardioversion should be synchronized with the QRS complex and is recommended for hemodynamically stable, wide-complex tachycardia requiring cardioversion, PSVT, atrial fibrillation, and atrial flutter. Polymorphic VT should be treated as VF with unsynchronized shocks.

Invasive Cardiopulmonary Resuscitation

Thoracotomy and open-chest cardiac massage are not part of routine CPR because of the high incidence of severe complications. Nonetheless, these invasive techniques can be helpful in specific life-threatening circumstances that preclude effective closed-chest massage. Possible indications include cardiac arrest associated with penetrating or blunt chest trauma, penetrating abdominal trauma, severe chest deformity, pericardial tamponade, or pulmonary embolism.

Intravenous Access

Some resuscitation drugs are fairly well absorbed following administration through a TT. Lidocaine, epinephrine, atropine, naloxone, and vasopressin (but *not* sodium bicarbonate) can be delivered via a catheter whose tip extends past the TT. Dosages 2–2½ times higher than recommended for intravenous use, diluted in 10 mL of normal saline or distilled water, are recommended for adult patients. Even though establishing reliable intravenous access is a high priority, it *should not take precedence* over initial chest compressions, airway management, or defibrillation. A preexisting internal jugular or subclavian line is ideal for venous access during resuscitation. If there is no central line access, an attempt should be made to establish peripheral intravenous access in either the antecubital or the external jugular vein. Peripheral intravenous sites are associated with a significant delay of 1–2 min between drug administration and delivery to the heart, as peripheral blood flow is drastically reduced during resuscitation. Administration of drugs given through a peripheral intravenous line should be followed by an intravenous flush (eg, a 20-mL fluid bolus in adults) and/or elevation of the extremity for 10–20 s. Establishing central vein access can potentially cause interruption of CPR but should be considered if an inadequate response is seen to peripherally administered drugs.

If intravenous cannulation is difficult, an intraosseous infusion can provide emergency vascular access in children. The success rate is lower in older children, but even in adults intraosseous cannulas have been successfully placed in the tibia and in the distal radius and ulna. A rigid 18-gauge spinal needle with a stylet or a small bone marrow trephine needle can be inserted into the distal femur or proximal tibia. If the tibia is chosen, a needle is inserted 2–3 cm below the tibial tuberosity at a 45° angle away from the epiphyseal plate (Figure 55–8). Once the needle is advanced through the cortex, it should stand upright without support. Proper placement is confirmed by the ability to aspirate marrow through the needle and a smooth infusion of fluid. A network of venous sinusoids within the medullary cavity of long bones drains into the systemic circulation by way of nutrient or emissary veins. This route is very effective for administration of drugs, crystalloids, colloids, and blood and can achieve flow rates exceeding 100 mL/h under gravity. Much higher flow rates are possible if the fluid is placed under pressure (eg, 300 mm Hg) with an infusion bag. The onset of drug action may be slightly delayed compared with intravenous or tracheal

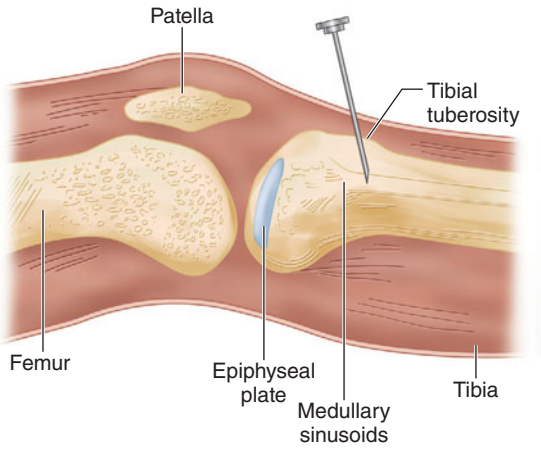


FIGURE 55–8 Intraosseous infusions provide emergency access to the venous circulation in pediatric patients by way of the large medullary venous channels. The needle is directed away from the epiphyseal plate to minimize the risk of injury.

administration. The intraosseous route may require a higher dose of some drugs (eg, epinephrine) than recommended for intravenous administration. The use of intraosseous infusion for induction and maintenance of general anesthesia, antibiotic therapy, seizure control, and inotropic support has been described. (Note that most studies have evaluated the placement of intraosseous access in patients with intact hemodynamics or hypovolemic states, not in cardiac arrest situations.) Because of the risks of osteomyelitis and compartment syndrome, however, intraosseous infusions should be replaced by a conventional intravenous route as soon as possible. In addition, because of the theoretical risk of bone marrow or fat emboli, intraosseous infusions should be avoided if possible in patients with right-to-left shunts, pulmonary hypertension, or severe pulmonary insufficiency.

Arrhythmia Recognition

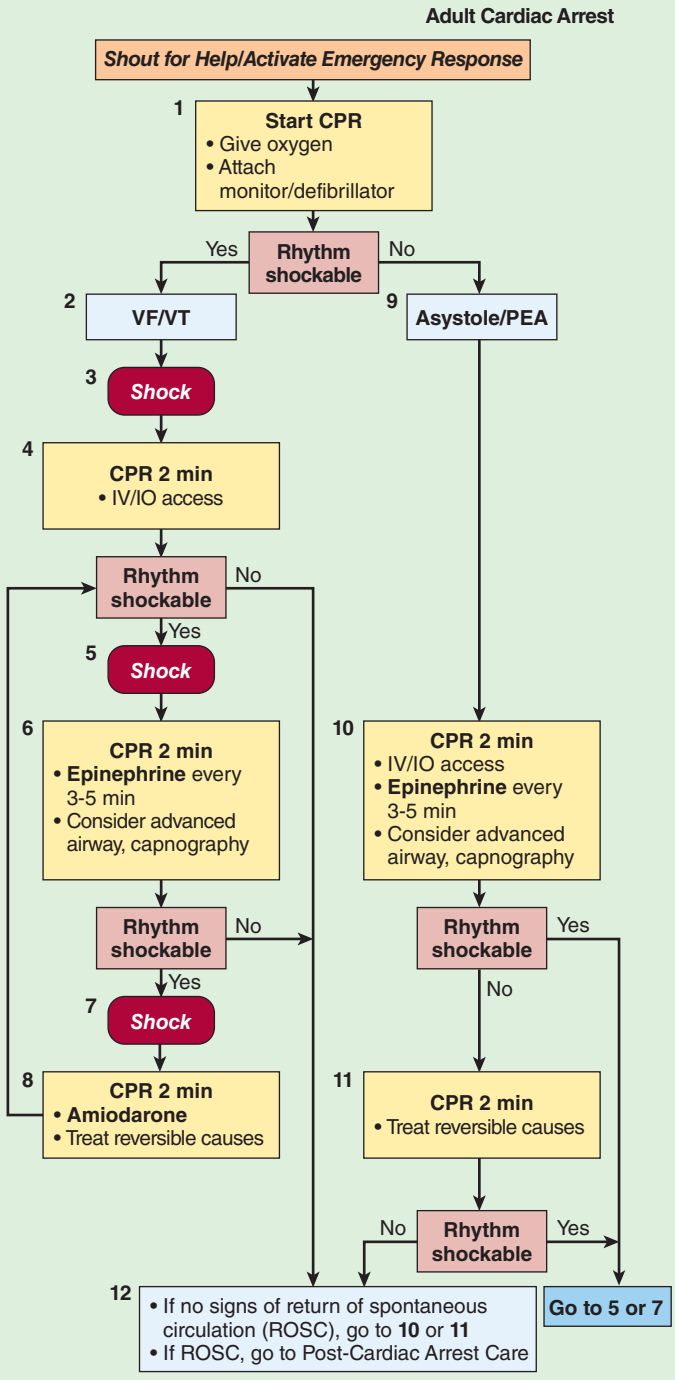
Successful pharmacological and electrical treatment of cardiac arrest (**Figure 55–9**) depends on definitive identification of the underlying arrhythmia. Interpreting rhythm strips in the midst of a resuscitation situation is complicated by artifacts and variations in monitoring techniques (eg, lead systems, equipment).

Drug Administration

Many of the drugs administered during CPR have been described elsewhere in this text. **Table 55–4** summarizes the cardiovascular actions, indications, and dosages of drugs commonly used during resuscitation.

Atropine is not included as a drug for PEA/asystole in the new CPR-ECC guidelines; however, its use is retained for symptomatic bradycardia. Infusions of chronotropic drugs (eg, dopamine, epinephrine, isoproterenol) can be considered as an alternative to pacing if atropine is ineffective in the setting of symptomatic bradycardia. Calcium chloride, sodium bicarbonate, and bretylium are conspicuously absent from this table. Calcium (2–4 mg/kg of the chloride salt) is helpful in the treatment of documented hypocalcemia, hyperkalemia, hypermagnesemia, or a calcium channel blocker overdose. When used, 10% calcium chloride can be given at 2–4 mg/kg every 10 min. Sodium bicarbonate (0.5–1 mEq/kg) is not recommended in the guidelines and should be considered only in specific situations such as preexisting metabolic acidosis or hyperkalemia, or in the treatment of tricyclic antidepressant or barbiturate overdose. Sodium bicarbonate elevates plasma pH by combining with hydrogen ions to form carbonic acid, which readily dissociates into carbon dioxide and water. Because carbon dioxide, but not bicarbonate, readily crosses cell membranes and the blood–brain barrier, the resulting arterial hypercapnia will cause intracellular tissue acidosis. Although successful defibrillation is not related to *arterial* pH, increased *intramyocardial* carbon dioxide may reduce the possibility of cardiac resuscitation. Furthermore, bicarbonate administration can lead to detrimental alterations in osmolality and the oxygen–hemoglobin dissociation curve. Therefore, effective alveolar ventilation and adequate tissue perfusion are the treatments of choice for the respiratory and metabolic acidosis that accompany resuscitation.

Intravenous fluid therapy with either colloid or balanced salt solutions is indicated in patients with intravascular volume depletion (eg, acute blood loss, diabetic ketoacidosis, thermal burns). Dextrose-containing solutions may lead to a hyperosmotic



CPR Quality

- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy

- **Biphasic:** Manufacturer recommendation (e.g., initial dose of 120–200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy

- **Epinephrine IV/IO Dose:** 1 mg every 3-5 minutes
- **Vasopression IV/IO Dose:** 40 units can replace first or second dose of epinephrine
- **Amiodarone IV/IO Dose:** First dose: 300 mg bolus Second dose: 150 mg

Advanced Airway

- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

FIGURE 55–9 Algorithm for treating ventricular fibrillation and pulseless ventricular tachycardia (VF/VT). Pulseless ventricular tachycardia should be treated in the same way as ventricular fibrillation. Note: This figure and Figures 55–1 and 55–2 emphasize the concept that rescuers and health care providers must assume that all unmonitored adult cardiac arrests are due to VF/VT. In each figure, the flow of the algorithm assumes that the arrhythmia is continuing. (Reproduced, with permission, from Neumar RW, Otto CW, Link MS, et al: Part 8: Adult Advanced Cardiovascular Life Support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2012;122(18 Suppl 3): S729-S767.)

TABLE 55–4 Cardiovascular effects, indications, and dosages of resuscitation drugs.[1]

			Initial Dose		
Drug	**Cardiovascular Effects**	**Indications**	**Adult**	**Pediatric**	**Comments**
Adenosine	Slows AV nodal conduction	Narrow complex tachycardias, stable supraventricular tachycardia, and wide-complex tachycardias if supraventricular in origin	6 mg over 1–3 s; 12 mg repeat dose	Initial dose 0.1–0.2 mg/kg; subsequent doses doubled to maximum single dose of 12 mg	Recommended as diagnostic or therapeutic maneuver for supraventricular tachycardias; give as rapid IV bolus. Vasodilates, BP may decrease. Theoretical risk of angina, bronchospasm, proarrhythmic action. Drug–drug interaction with theophylline, dipyridamole.
Atropine	Anticholinergic (parasympatholytic). Increases sinoatrial node rate and automaticity; increases AV node conduction	Symptomatic brachycardia, AV block	0.5–1.0 mg repeated every 3–5 min	0.02 mg/kg	Repeat atropine doses every 5 min to a total dose of 3 mg in adults or 0.5 mg in children, 1.0 mg in adolescents. The minimum pediatric dose is 0.1 mg. Do not use for infranodal (Mobitz II) block.
Epinephrine	α-Adrenergic effects increase myocardial and cerebral blood flow. β-Adrenergic effects may increase myocardial work and decrease subendocardial perfusion and cerebral blood flow	VF/VT, electromechanical dissociation, ventricular asystole, severe bradycardia unresponsive to atropine or pacing Severe hypotension	1 mg IV 0.03 mcg/kg/min in an infusion increased to effect	Initial dose 0.01 mg/kg IV; repeat same for subsequent doses or up to 0.1–0.2 mg/kg IV 1 mcg/kg	Repeat doses every 3–5 min as necessary. An infusion of epinephrine (eg., 1 mg in 250 mL D_5W or NS, 4 mcg/mL) can be titrated to effect in adults (1–4 mcg/min) or children (0.1–1 mcg/kg/min). Administration down a tracheal tube requires higher doses (2–2.5 mg in adults, 0.1 mg/kg in children). High-dose epinephrine (0.1 mg/kg) in adults is recommended only after standard therapy has failed.
Lidocaine	Decreases rate of phase 4 depolarization (decreases automaticity); depresses conduction in reentry pathways. Elevates VF threshold. Reduces disparity in action potential duration between normal and ischemic tissue. Reduces action potential and effective refractory period duration	VT that has not responded to defibrillation; premature ventricular contractions. Use only as second-line therapy; thus, consider only if amiodarone is unavailable	1–1.5 mg/kg	1 mg/kg	Doses of 0.5–1.5 mg/kg can be repeated every 5–10 min to a total dose of 3 mg/kg. After infarction or successful resuscitation, a continuous infusion (eg, 1 g in 500 mL D_5W, 2 mg/mL) should be run at a rate of 20–50 mcg/kg/min (2–4 mg/min in most adults). Therapeutic blood levels are usually 1.5–6 mcg/mL.

(*continued*)

TABLE 55–4 Cardiovascular effects, indications, and dosages of resuscitation drugs.[1] (continued)

			Initial Dose		
Drug	**Cardiovascular Effects**	**Indications**	**Adult**	**Pediatric**	**Comments**
Vasopressin	Nonadrenergic peripheral vasoconstrictor; direct stimulation of V_1 receptors	Bleeding esophageal varices; adult shock-refractory VF; hemodynamic support in vasodilatory (septic) shock	40 units IV, single dose, 1 time only	Not recommended	Newly recommended as equivalent to epinephrine in VF and PEA; may be more effective in asystole; used only one time; has a 10–20 min half-life.
Procainamide	Suppresses both atrial and ventricular arrhythmias	AF/flutter; preexcited atrial arrhythmias with rapid ventricular response; wide-complex tachycardia that cannot be distinguished as SVT or VT	20 mg/min until arrhythmia suppressed, hypotension develops, QRS complex increases by >50%, or total dose of 17 mg/kg has infused. In urgent situation, 50 mg/min may be used to maximum of 17 mg/kg. Maintenance infusion, 1–4 mg/min	Loading dose: 15 mg/kg; infusion over 30–60 min; routine use in combination with drugs that prolong QT interval is not recommended	Contraindicated in overdose of tricyclic antidepressants or other antiarrhythmic drugs. Bolus doses can result in toxicity. Should not be used in preexisting QT prolongation or torsades de pointes. Blood levels should be monitored in patients with impaired renal function and when constant infusion >3 mg/min for >24 h.
Amiodarone	Complex drug with effects on sodium, potassium, and calcium channels as well as α- and β-adrenergic blocking properties	SVT with accessory pathway conduction; unstable VT and VF; stable VT, polymorphic VT, wide-complex tachycardia of uncertain origin; AF/flutter with CHF; preexcited AF/flutter; adjunct to electrical cardioversion in refractory PSVTs, atrial tachycardia, and AF	150 mg over 10 min, followed by 1 mg/min for 6 h, then 0.5 mg/min, with supplementary infusion of 150 mg as necessary up to 2 g. For pulseless VT or VF, initial administration is 300 mg rapid infusion diluted in 20–30 mL of saline or dextrose in water	5 mg/kg for pulseless VT/VF; for perfusing tachycardia loading dose, 5 mg/kg IV/IO; maximum dose, 15 mg/kg/d	Antiarrhythmic of choice if cardiac function is impaired, EF <40%, or CHF. Routine use in combination with drugs prolonging QT interval is not recommended. Most frequent side effects are hypotension and bradycardia.
Verapamil	Calcium channel blocking agent used to slow conduction and increase refractoriness in AV node, terminating reentrant arrhythmias that require AV nodal conduction for continuation	Controls ventricular response rate in AF/flutter and MAT; rate control in AF; terminating narrow-complex PSVT	2.5–5 mg IV over 2 min; without response, repeat dose with 5–10 mg every 15–30 min to a max of 20 mg		Use only in patients with narrow-complex PSVT or supraventricular arrhythmia. Do not use in presence of impaired ventricular function or CHF

Diltiazem	Calcium channel blocking agent used to slow conduction and increase refractoriness in AV node, terminating reentrant arrhythmias that require AV nodal conduction for continuation	Slows conduction and increases refractoriness in AV node. May terminate reentrant arrhythmias. Controls ventricular response rate in AF/flutter and MAT	0.25 mg/kg, followed by second dose of 0.35 mg/kg if necessary; maintenance infusion of 5–15 mg/h in AF/flutter		May exacerbate CHF in severe LV dysfunction; may decrease myocardial contractility, but less so than verapamil.
Dobutamine	Synthetic catecholamine and potent inotropic agent with predominant β-adrenergic receptor-stimulating effects that increase cardiac contractility in a dose-dependent manner, accompanied by a decrease in LV filling pressures.	Severe systolic heart failure	5–20 mcg/kg/min		Hemodynamic end points rather than specific dose is goal. Elderly have significantly reduced response. May induce or exacerbate myocardial ischemia with increases in heart rate.
Flecainide	Potent sodium channel blocker with significant conduction-slowing effects	AF/flutter, ventricular arrhythmias and supraventricular arrhythmias without structural heart disease, ectopic atrial heart disease, AV nodal reentrant tachycardia, SVTs associated with an accessory pathway, including preexcited AF	2 mg/kg at 10 mg/min (IV use not approved in the United States)		Should not be used in patients with impaired LV function, or when coronary artery disease is suspected.
Ibutilide	Short-acting antiarrhythmic, prolongs the action potential duration and increases refractory period	Acute conversion or adjunct to electrical cardioversion of AF/flutter of short duration	In patients >60 kg, 1 mg (10 mL) over 10 min; a second similar dose may be repeated in 10 min. In patients <60 kg, initial dose is 0.01 mg/kg		Patients should be monitored for arrhythmias for 4–6 h, and longer in those with hepatic dysfunction.
Magnesium	Hypomagnesemia associated with arrhythmias, cardiac insufficiency, and sudden death; can precipitate refractory VF; can hinder K^+ replacement	Torsades de pointes with prolonged QT, even with normal serum levels of magnesium	1–2 g in 50–100 mL D_5W over 15 min	500 mg/mL–IV/IO: 25–50 mg/kg; maximum dose: 2 g per dose	Rapid IV infusion for torsades de pointes or suspected hypomagnesemia not recommended in cardiac arrest except when arrhythmia suspected.

(*continued*)

TABLE 55–4 Cardiovascular effects, indications, and dosages of resuscitation drugs.[1] (continued)

			Initial Dose		
Drug	**Cardiovascular Effects**	**Indications**	**Adult**	**Pediatric**	**Comments**
Propafenone	Significant conduction slowing and negative inotropic effects. Nonselective β-adrenergic blocking properties	AF/flutter, ventricular arrhythmias and supraventricular arrhythmias without structural heart disease, ectopic atrial heart disease, AV nodal reentrant tachycardia, SVTs associated with an accessory pathway	2.0 mg/kg at 10 mg/min (IV use not approved in the United States)		Should be avoided with impaired LV function or when CAD suspected.
Sotalol	Prolongs action potential duration and increases cardiac tissue refractoriness. Nonselective β-adrenergic blocking properties	Preexcited AF/flutter, ventricular and supraventricular arrhythmias	1.0–1.5 mg/kg at a rate of 10 mg/min		Limited by need to be infused slowly.

[1]AV, atrioventricular; BP, blood pressure; VF, ventricular fibrillation; VT, ventricular tachycardia; PEA, pulseless electrical activity; AF, atrial fibrillation; SVT, supraventricular tachycardia; CHF, congestive heart failure; PSVT, paroxysmal supraventricular tachycardia; IV/IO, intravenous/intraosseous; EF, ejection fraction; MAT, multifocal atrial tachycardia; LV, left ventricular; CAD, coronary artery disease.

diuresis and may worsen neurological outcome. They should be avoided unless hypoglycemia is suspected. Likewise, administration of free water (eg, D_5W) may lead to cerebral edema.

Emergency Pacemaker Therapy

Transcutaneous cardiac pacing (TCP) is a noninvasive method of rapidly treating arrhythmias caused by conduction disorders or abnormal impulse. TCP is not routinely recommended in cardiac arrest. TCP use may be considered to treat asystole, bradycardia caused by heart block, or tachycardia from a reentrant mechanism. If there is concern about the use of atropine in high-grade block, TCP is always appropriate. If the patient is unstable with marked bradycardia, TCP should be implemented immediately while awaiting treatment response to drugs. The pacer unit has become a built-in feature of some defibrillator models. Disposable pacing electrodes are usually positioned on the patient in an anterior–posterior manner. The placement of the negative electrode corresponds to a V_2 electrocardiograph position, whereas the positive electrode is placed on the left posterior chest beneath the scapula and lateral to the spine. Note that this positioning does not interfere with paddle placement during defibrillation. Failure to capture may be due to electrode misplacement, poor electrode-to-skin contact, or increased transthoracic impedance (eg, barrel-shaped chest, pericardial effusion). Current output is slowly increased until the pacing stimuli obtain electrical and mechanical capture. A wide QRS complex following a pacing spike signals *electrical* capture, but *mechanical* (ventricular) capture must be confirmed by an improving pulse or blood pressure. Conscious patients may require sedation to tolerate the discomfort of skeletal muscle contractions. Transcutaneous pacing can provide effective temporizing therapy until transvenous pacing or other definitive treatment can be initiated. TCP has many advantages over transvenous pacing because it can be used by almost all electrocardiogram providers and can be started quickly and conveniently at the bedside.

11

Precordial Thump

The precordial thump is to be considered only in witnessed, monitored unstable VT when a defibrillator is not immediately available.

RECOMMENDED RESUSCITATION PROTOCOLS

A resuscitation team leader integrates the assessment of the patient, including electrocardiographic diagnosis, with the electrical and pharmacological therapy (Table 55–5). This person must have a firm grasp of the guidelines for cardiac arrest presented in the CPR-ECC algorithms (Figures 55–9 to 55–13).

TABLE 55–5 Steps for synchronized cardioversion.[1]

1. Consider sedation.
2. Turn on defibrillator (monophasic or biphasic).
3. Attach monitor leads to the patient ("white to right, red to ribs, what's left over to the left shoulder") and ensure proper display of the patient's rhythm.
4. Engage the synchronization mode by pressing the "sync" control button.
5. Look for markers on R waves indicating sync mode.
6. If necessary, adjust monitor gain until sync markers occur with each R wave.
7. Select appropriate energy level.
8. Position conductor pads on patient (or apply gel to paddles).
9. Position paddle on patient (sternum-apex).
10. Announce to team members: "Charging defibrillator—stand clear!"
11. Press "charge" button on apex paddle (right hand).
12. When the defibrillator is charged, begin the final clearing chant. State firmly in a forceful voice the following chant before each shock:
    - *"I am going to shock on three. One, I'm clear."* (Check to make sure you are clear of contact with the patient, the stretcher, and the equipment.)
    - *"Two, you are clear."* (Make a visual check to ensure that no one continues to touch the patient or stretcher. In particular, do not forget about the person providing ventilation. That person's hands should not be touching the ventilatory adjuncts, including the tracheal tube!).
    - *"Three,* everybody's *clear."* (Check yourself one more time before pressing the "shock" buttons.)
13. Apply 25 lb pressure on both paddles.
14. Press the "discharge" buttons simultaneously.
15. Check the monitor. If tachycardia persists, increase the joules according to the electrical cardioversion algorithm.
16. Reset the sync mode after each synchronized cardioversion because most defibrillators default back to unsynchronized mode. This default allows an immediate defibrillation if the cardioversion produces ventricular fibrillation.

[1]Data from The American Heart Association Guidelines 2010 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010;122:S706.

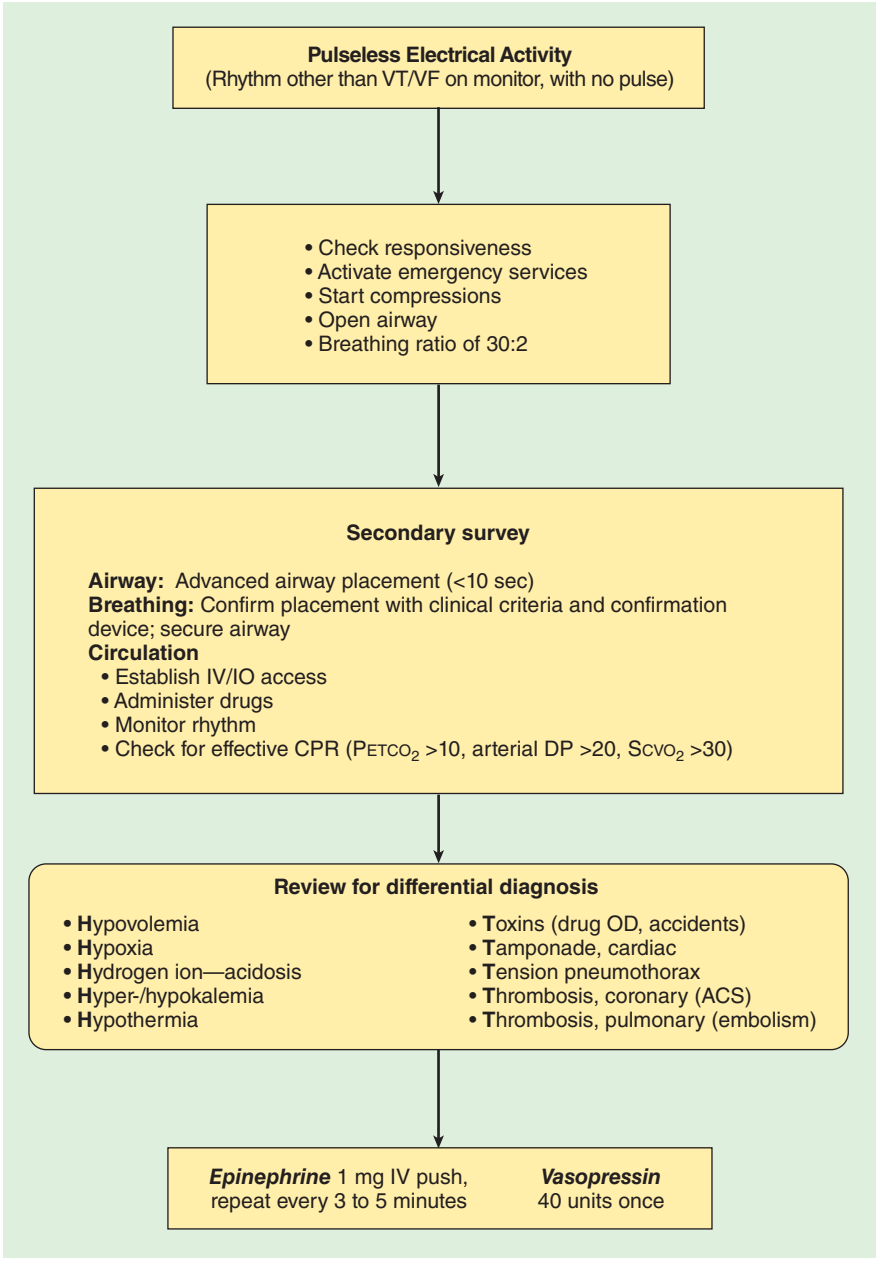


FIGURE 55–10 Pulseless electrical activity algorithm. VF/VT, ventricular fibrillation and pulseless ventricular tachycardia. P_{ETCO_2}, end-tidal carbon dioxide; DP, diastolic pressure; S_{CVO_2}, central venous oxygen saturation. (Data from the American Heart Association BLS and ACLS Guidelines 2010 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010.122;S729.)

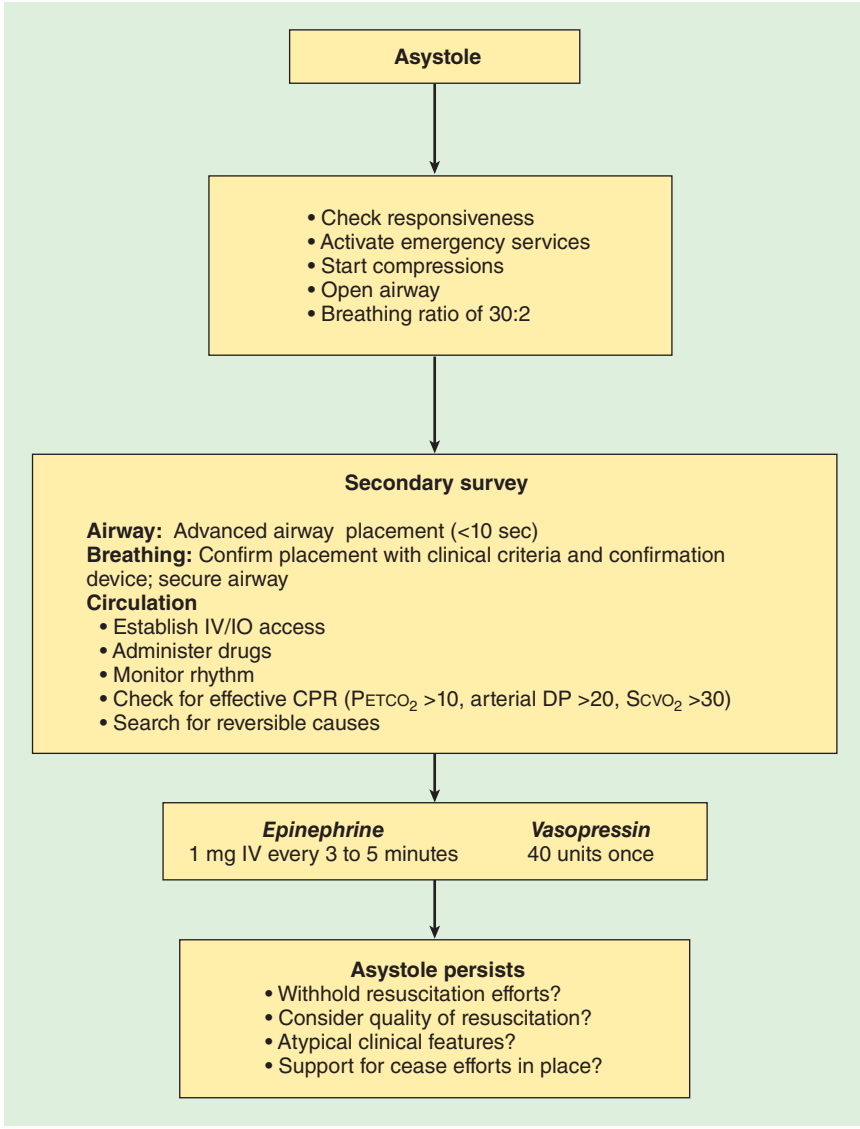


FIGURE 55–11 Asystole: The silent heart algorithm. VF/VT, ventricular fibrillation and pulseless ventricular tachycardia; P_{ETCO_2}, end-tidal carbon dioxide; DP, diastolic pressure; S_{CVO_2}, central venous oxygen saturation. (Data from The American Heart Association BLS and ACLS Guidelines 2010 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010:122;S729.)

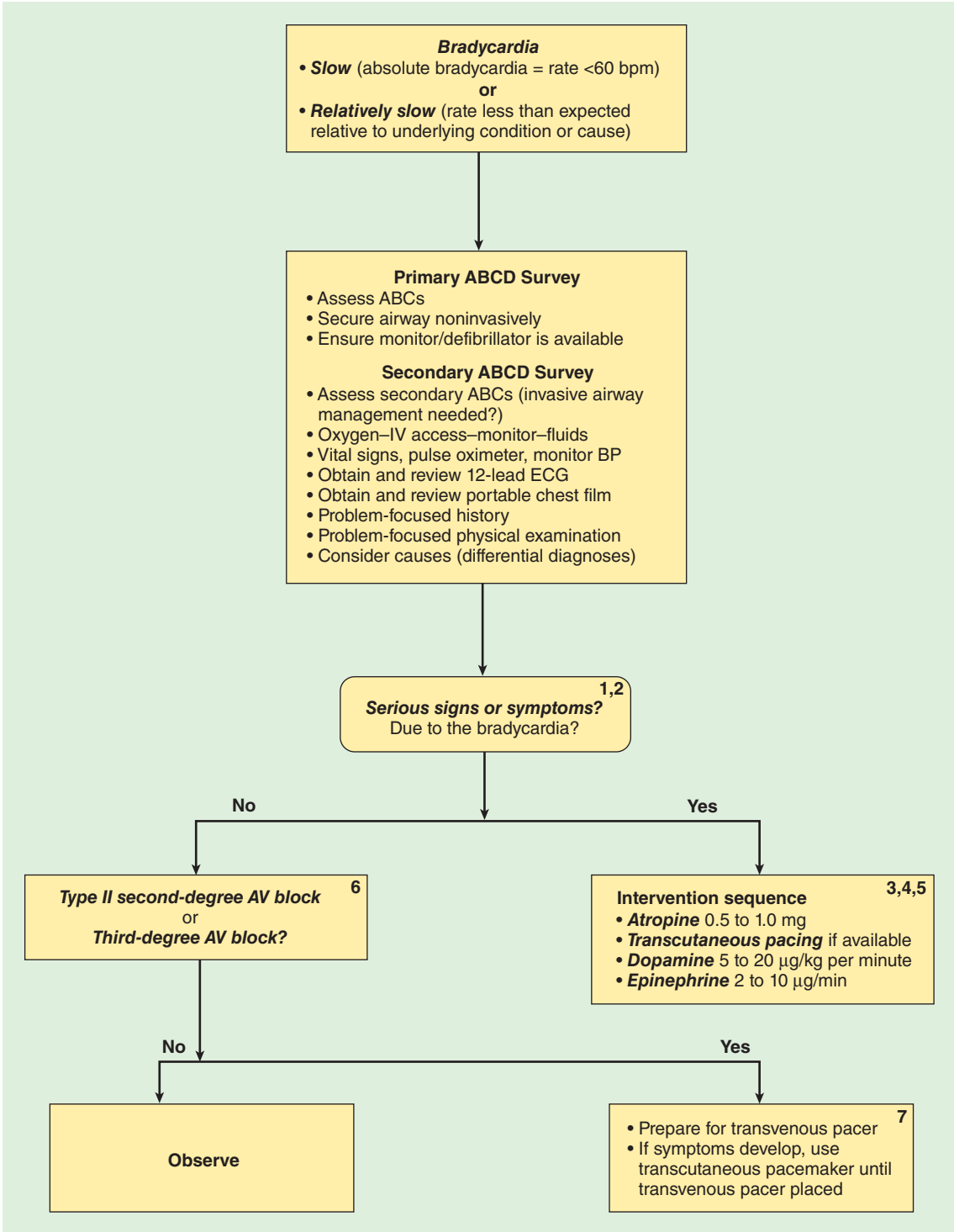


FIGURE 55–12 Bradycardia algorithm. AV, atrioventricular. (Data from The American Heart Association BLS and ACLS Guidelines 2010 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010:122;S729.)

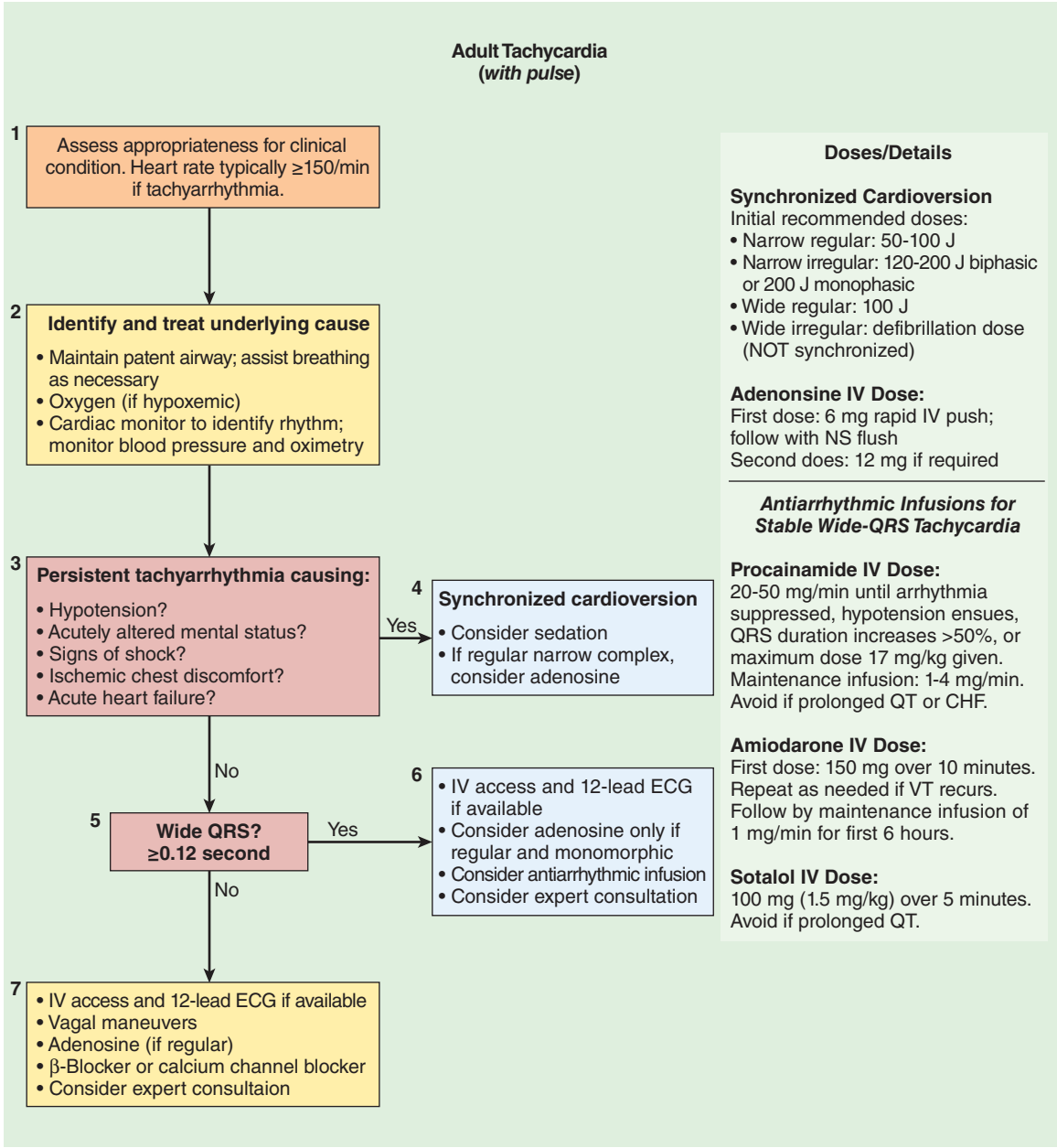


FIGURE 55–13 Tachycardia overview algorithm. VT, ventricular tachycardia; CHF, congestive heart failure. WPW, Wolff-Parkinson-White syndrome. (Reproduced, with permission, from The American Heart Association BLS and ACLS Guidelines 2010 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010:122;S729.)

CASE DISCUSSION

Intraoperative Hypotension & Cardiac Arrest

A 16-year-old boy is rushed to the operating room for emergency laparotomy and thoracotomy after suffering multiple abdominal and thoracic stab wounds. In the field, paramedics intubated the patient, started two large-bore intravenous lines, began fluid resuscitation, and inflated a pneumatic antishock garment. Upon arrival in the operating room, the patient's blood pressure is unobtainable, heart rate is 128 beats/min (sinus tachycardia), and respirations are being controlled by a bag-valve device.

What should be done immediately?

Cardiopulmonary resuscitation must be initiated immediately: external chest compressions should be started as soon as the arterial blood pressure is found to be inadequate for vital organ perfusion. Because the patient is already intubated, the location of the tracheal tube should be confirmed with chest auscultation and quantitative waveform capnography (if available, to assist in both confirmation of tube placement as well as to assess the adequacy of CPR) and 100% oxygen should be delivered.

Which CPR sequence best fits this situation?

Pulselessness in the presence of sinus rhythm suggests severe hypovolemia, cardiac tamponade, ventricular rupture, dissecting aortic aneurysm, tension pneumothorax, profound hypoxemia and acidosis, or pulmonary embolism. Epinephrine, 1 mg, should be administered intravenously.

What is the most likely cause of this patient's profound hypotension?

The presence of multiple stab wounds strongly suggests hypovolemia. Abdominal ultrasound can rapidly identify a collapsed vena cava, which is pathognomonic of hypovolemia. Fluids, preferably warmed, should be rapidly administered. Additional venous access can be sought as other members of the operating room team administer fluid through blood pumps or other rapid infusion devices. Five percent albumin or lactated Ringer's solution is acceptable until blood products are available. Activation of a massive transfusion protocol is often indicated.

What are the signs of tension pneumothorax and pericardial tamponade?

The signs of **tension pneumothorax**—the presence of air under pressure in the pleural space—include increasing peak inspiratory pressures, tachycardia and hypotension (decreased venous return), hypoxia (atelectasis), distended neck veins, unequal breath sounds, tracheal deviation, and mediastinal shift away from the pneumothorax.

Pericardial tamponade—cardiac compression from pericardial contents—should be suspected in any patient with narrow pulse pressure; pulsus paradoxus (>10 mm Hg drop in systolic blood pressure with inspiration); elevated central venous pressure with neck vein distention; equalization of central venous pressure, atrial pressures, and ventricular end-diastolic pressures; distant heart sounds; tachycardia; and hypotension. Many of these signs may be masked by concurrent hypovolemic shock.

Fluid administration and properly performed external cardiac compressions do not result in satisfactory carotid or femoral pulsations. What else should be done?

Because external chest compressions are often ineffective in trauma patients, an emergency thoracotomy should be performed as soon as possible to clamp the thoracic aorta, relieve a tension pneumothorax or pericardial tamponade, identify possible intrathoracic hemorrhage, and perform open-chest cardiac compressions. Cross-clamping of the thoracic aorta increases brain and heart perfusion and decreases subdiaphragmatic hemorrhage. Lack of response to cross-clamping is a good predictor of demise.

What is the function of the pneumatic antishock garment, and how should it be removed?

Inflation of the bladders within a pneumatic antishock garment increases arterial blood

pressure by elevating peripheral vascular resistance. Functionally, the suit has the same effect as thoracic aorta cross-clamping by decreasing blood flow and hemorrhage in the lower half of the body. Complications of inflating the abdominal section of the pneumatic antishock garment include renal dysfunction, altered lung volumes, and visceral injury during external chest compressions. The suit should be deflated only after restoration of hemodynamic parameters. Even then, deflation should be gradual, as it may be accompanied by marked hypotension and metabolic acidosis caused by reperfusion of ischemic tissues.

GUIDELINES

The American Heart Association in Collaboration with the International Liaison Committee on Resuscitation (ILCOR): Guidelines 2010 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010;122:S250.

SUGGESTED READING

Adult ACLS. Circulation 2010;122:S729.
Adult BLS. Circulation 2010;122:S685.
Adult CPR overview. Circulation 2010;122:S675.
CPR techniques and devices. Circulation 2010;122:S720.
Electrical therapies: AED, defibrillation, pacing. Circulation 2010;122:S706.
Executive summary ACLS. Circulation 2010;122:S640.