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Part 8: Adult Advanced Cardiovascular Life Support

2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Robert W. Neumar, Chair; Charles W. Otto; Mark S. Link; Steven L. Kronick; Michael Shuster; Clifton W. Callaway; Peter J. Kudenchuk; Joseph P. Ornato; Bryan McNally; Scott M. Silvers; Rod S. Passman; Roger D. White; Erik P. Hess; Wanchun Tang; Daniel Davis; Elizabeth Sinz; Laurie J. Morrison

Advanced cardiovascular life support (ACLS) impacts multiple key links in the chain of survival that include interventions to prevent cardiac arrest, treat cardiac arrest, and improve outcomes of patients who achieve return of spontaneous circulation (ROSC) after cardiac arrest. ACLS interventions aimed at preventing cardiac arrest include airway management, ventilation support, and treatment of bradyarrhythmias and tachyarrhythmias. For the treatment of cardiac arrest, ACLS interventions build on the basic life support (BLS) foundation of immediate recognition and activation of the emergency response system, early CPR, and rapid defibrillation to further increase the likelihood of ROSC with drug therapy, advanced airway management, and physiologic monitoring. Following ROSC, survival and neurologic outcome can be improved with integrated post–cardiac arrest care.

Part 8 presents the 2010 Adult ACLS Guidelines: 8.1: "Adjuncts for Airway Control and Ventilation"; 8.2: "Management of Cardiac Arrest"; and 8.3: "Management of Symptomatic Bradycardia and Tachycardia." Post–cardiac arrest interventions are addressed in Part 9: "Post–Cardiac Arrest Care."

Key changes from the 2005 ACLS Guidelines include

- Continuous quantitative waveform capnography is recommended for confirmation and monitoring of endotracheal tube placement.
- Cardiac arrest algorithms are simplified and redesigned to emphasize the importance of high-quality CPR (including chest compressions of adequate rate and depth, allowing complete chest recoil after each compression, minimizing interruptions in chest compressions and avoiding excessive ventilation).
- Atropine is no longer recommended for routine use in the management of pulseless electrical activity (PEA)/asystole.
- There is an increased emphasis on physiologic monitoring to optimize CPR quality and detect ROSC.
- Chronotropic drug infusions are recommended as an alternative to pacing in symptomatic and unstable bradycardia.
- Adenosine is recommended as a safe and potentially effective therapy in the initial management of stable undifferentiated regular monomorphic wide-complex tachycardia.

Part 8.1: Adjuncts for Airway Control and Ventilation

Overview of Airway Management

This section highlights recommendations for the support of ventilation and oxygenation during CPR and the peri-arrest period. The purpose of ventilation during CPR is to maintain adequate oxygenation and sufficient elimination of carbon dioxide. However, research has not identified the optimal tidal volume, respiratory rate, and inspired oxygen concentration required during resuscitation from cardiac arrest.

Both ventilation and chest compressions are thought to be important for victims of prolonged ventricular fibrillation (VF) cardiac arrest and for all victims with other presenting rhythms. Because both systemic and pulmonary perfusion are substantially reduced during CPR, normal ventilation-perfusion relationships can be maintained with a minute ventilation that is much lower than normal. During CPR with an advanced airway in place, a lower rate of rescue breathing is needed to avoid hyperventilation.

Ventilation and Oxygen Administration During CPR

During low blood flow states such as CPR, oxygen delivery to the heart and brain is limited by blood flow rather than by arterial oxygen content.[1,2] Therefore, rescue breaths are less important than chest compressions during the first few minutes of resuscitation from witnessed VF cardiac arrest and could reduce CPR efficacy due to interruption in chest compressions and the increase in intrathoracic pressure that accompanies positive-pressure ventilation. Thus, during the first few minutes of witnessed cardiac arrest a lone rescuer should not interrupt chest

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compressions for ventilation. Advanced airway placement in cardiac arrest should not delay initial CPR and defibrillation for VF cardiac arrest (Class I, LOE C).

Oxygen During CPR

Oxygen Administration During CPR

The optimal inspired oxygen concentration during adult CPR has not been established in human or animal studies. In addition, it is unknown whether 100% inspired oxygen ($FIO_2 = 1.0$) is beneficial or whether titrated oxygen is better. Although prolonged exposure to 100% inspired oxygen ($FIO_2 = 1.0$) has potential toxicity, there is insufficient evidence to indicate that this occurs during brief periods of adult CPR.[3–5] Empirical use of 100% inspired oxygen during CPR optimizes arterial oxyhemoglobin content and in turn oxygen delivery; therefore, use of 100% inspired oxygen ($FIO_2 = 1.0$) as soon as it becomes available is reasonable during resuscitation from cardiac arrest (Class IIa, LOE C). Management of oxygen after ROSC is discussed in Part 9: "Post-Cardiac Arrest Care."

Passive Oxygen Delivery During CPR

Positive-pressure ventilation has been a mainstay of CPR but recently has come under scrutiny because of the potential for increased intrathoracic pressure to interfere with circulation due to reduced venous return to the heart. In the out-of-hospital setting, passive oxygen delivery via mask with an opened airway during the first 6 minutes of CPR provided by emergency medical services (EMS) personnel was part of a protocol of bundled care interventions (including continuous chest compressions) that resulted in improved survival.[6–8] When passive oxygen delivery using a fenestrated tracheal tube (Boussignac tube) during uninterrupted physician-managed CPR was compared with standard CPR, there was no difference in oxygenation, ROSC, or survival to hospital admission.[9,10] Chest compressions cause air to be expelled from the chest and oxygen to be drawn into the chest passively due to the elastic recoil of the chest. In theory, because ventilation requirements are lower than normal during cardiac arrest, oxygen supplied by passive delivery is likely to be sufficient for several minutes after onset of cardiac arrest with a patent upper airway.[2] **At this time there is insufficient evidence to support the removal of ventilations from CPR performed by ACLS providers.**

Bag-Mask Ventilation

Bag-mask ventilation is an acceptable method of providing ventilation and oxygenation during CPR but is a challenging skill that requires practice for continuing competency. All healthcare providers should be familiar with the use of the bag-mask device.[11,12] Use of bag-mask ventilation is not recommended for a lone provider. When ventilations are performed by a lone provider, mouth-to-mouth or mouth-to-mask are more efficient. When a second provider is available, bag-mask ventilation may be used by a trained and experienced provider. But bag-mask ventilation is most effective when performed by 2 trained and experienced providers. One provider opens the airway and seals the mask to the face while the other squeezes the bag. Bag-mask ventilation is particularly helpful when placement of an advanced airway is delayed or unsuccessful. The desirable components of a bag-mask device are listed in Part 5: "Adult Basic Life Support."

The provider should use an adult (1 to 2 L) bag and the provider should deliver approximately 600 mL of tidal volume sufficient to produce chest rise over 1 second.[13] This volume of ventilation is adequate for oxygenation and minimizes the risk of gastric inflation. The provider should be sure to open the airway adequately with a head tilt–chin lift, lifting the jaw against the mask and holding the mask against the face, creating a tight seal. During CPR give 2 breaths (each 1 second) during a brief (about 3 to 4 seconds) pause after every 30 chest compressions.

Bag-mask ventilation can produce gastric inflation with complications, including regurgitation, aspiration, and pneumonia. Gastric inflation can elevate the diaphragm, restrict lung movement, and decrease respiratory system compliance.[14–16]

Airway Adjuncts

Cricoid Pressure

Cricoid pressure in nonarrest patients may offer some measure of protection to the airway from aspiration and gastric insufflation during bag-mask ventilation.[17–20] However, it also may impede ventilation and interfere with placement of a supraglottic airway or intubation.[21–27] The role of cricoid pressure during out-of-hospital cardiac arrest and in-hospital cardiac arrest has not been studied. If cricoid pressure is used in special circumstances during cardiac arrest, the pressure should be adjusted, relaxed, or released if it impedes ventilation or advanced airway placement. The routine use of cricoid pressure in cardiac arrest is not recommended (Class III, LOE C).

Oropharyngeal Airways

Although studies have not specifically considered the use of oropharyngeal airways in patients with cardiac arrest, airways may aid in the delivery of adequate ventilation with a bag-mask device by preventing the tongue from occluding the airway. Incorrect insertion of an oropharyngeal airway can displace the tongue into the hypopharynx, causing airway obstruction. To facilitate delivery of ventilations with a bag-mask device, oropharyngeal airways can be used in unconscious (unresponsive) patients with no cough or gag reflex and should be inserted only by persons trained in their use (Class IIa, LOE C).

Nasopharyngeal Airways

Nasopharyngeal airways are useful in patients with airway obstruction or those at risk for developing airway obstruction, particularly when conditions such as a clenched jaw prevent placement of an oral airway. Nasopharyngeal airways are better tolerated than oral airways in patients who are not deeply unconscious. Airway bleeding can occur in up to 30% of patients following insertion of a nasopharyngeal airway.[28] Two case reports of inadvertent intracranial placement of a nasopharyngeal airway in patients with basilar skull fractures[29,30] suggest that nasopharyngeal airways should be used with caution in patients with severe craniofacial injury.

As with all adjunctive equipment, safe use of the nasopharyngeal airway requires adequate training, practice, and retraining. No studies have specifically examined the use of

nasopharyngeal airways in cardiac arrest patients. To facilitate delivery of ventilations with a bag-mask device, the nasopharyngeal airway can be used in patients with an obstructed airway. In the presence of known or suspected basal skull fracture or severe coagulopathy, an oral airway is preferred (Class IIa, LOE C).

Advanced Airways

Ventilation with a bag and mask or with a bag through an advanced airway (eg, endotracheal tube or supraglottic airway) is acceptable during CPR. All healthcare providers should be trained in delivering effective oxygenation and ventilation with a bag and mask. Because there are times when ventilation with a bag-mask device is inadequate, ideally ACLS providers also should be trained and experienced in insertion of an advanced airway.

Providers must be aware of the risks and benefits of insertion of an advanced airway during a resuscitation attempt. Such risks are affected by the patient's condition and the provider's expertise in airway control. There are no studies directly addressing the timing of advanced airway placement and outcome during resuscitation from cardiac arrest. Although insertion of an endotracheal tube can be accomplished during ongoing chest compressions, intubation frequently is associated with interruption of compressions for many seconds. Placement of a supraglottic airway is a reasonable alternative to endotracheal intubation and can be done successfully without interrupting chest compressions.

The provider should weigh the need for minimally interrupted compressions against the need for insertion of an endotracheal tube or supraglottic airway. There is inadequate evidence to define the optimal timing of advanced airway placement in relation to other interventions during resuscitation from cardiac arrest. In a registry study of 25 006 in-hospital cardiac arrests, earlier time to invasive airway (<5 minutes) was not associated with improved ROSC but was associated with improved 24-hour survival.[31] In an urban out-of-hospital setting, intubation that was achieved in <12 minutes was associated with better survival than intubation achieved in ≥ 13 minutes.[32]

In out-of-hospital urban and rural settings, patients intubated during resuscitation had a better survival rate than patients who were not intubated,[33] whereas in an in-hospital setting, patients who required intubation during CPR had a worse survival rate.[34] A recent study[8] found that delayed endotracheal intubation combined with passive oxygen delivery and minimally interrupted chest compressions was associated with improved neurologically intact survival after out-of-hospital cardiac arrest in patients with adult witnessed VF/pulseless VT. If advanced airway placement will interrupt chest compressions, providers may consider deferring insertion of the airway until the patient fails to respond to initial CPR and defibrillation attempts or demonstrates ROSC (Class IIb, LOE C).

For a patient with perfusing rhythm who requires intubation, pulse oximetry and electrocardiographic (ECG) status should be monitored continuously during airway placement. Intubation attempts should be interrupted to provide oxygenation and ventilation as needed.

To use advanced airways effectively, healthcare providers must maintain their knowledge and skills through frequent practice. It may be helpful for providers to master one primary method of airway control. Providers should have a second (backup) strategy for airway management and ventilation if they are unable to establish the first-choice airway adjunct. Bag-mask ventilation may serve as that backup strategy.

Once an advanced airway is inserted, providers should immediately perform a thorough assessment to ensure that it is properly positioned. This assessment should not interrupt chest compressions. Assessment by physical examination consists of visualizing chest expansion bilaterally and listening over the epigastrium (breath sounds should not be heard) and the lung fields bilaterally (breath sounds should be equal and adequate). A device also should be used to confirm correct placement (see the section "Endotracheal Intubation" below).

Continuous waveform capnography is recommended in addition to clinical assessment as the most reliable method of confirming and monitoring correct placement of an endotracheal tube (Class I, LOE A). Providers should observe a persistent capnographic waveform with ventilation to confirm and monitor endotracheal tube placement in the field, in the transport vehicle, on arrival at the hospital, and after any patient transfer to reduce the risk of unrecognized tube misplacement or displacement.

The use of capnography to confirm and monitor correct placement of supraglottic airways has not been studied, and its utility will depend on airway design. However, effective ventilation through a supraglottic airway device should result in a capnograph waveform during CPR and after ROSC.

Once an advanced airway is in place, the 2 providers should no longer deliver cycles of CPR (ie, compressions interrupted by pauses for ventilation) unless ventilation is inadequate when compressions are not paused. Instead the compressing provider should give continuous chest compressions at a rate of at least 100 per minute, without pauses for ventilation. The provider delivering ventilation should provide 1 breath every 6 to 8 seconds (8 to 10 breaths per minute). Providers should avoid delivering an excessive ventilation rate because doing so can compromise venous return and cardiac output during CPR. The 2 providers should change compressor and ventilator roles approximately every 2 minutes to prevent compressor fatigue and deterioration in quality and rate of chest compressions. When multiple providers are present, they should rotate the compressor role about every 2 minutes.

Supraglottic Airways

Supraglottic airways are devices designed to maintain an open airway and facilitate ventilation. Unlike endotracheal intubation, intubation with a supraglottic airway does not require visualization of the glottis, so both initial training and maintenance of skills are easier. Also, because direct visualization is not necessary, a supraglottic airway is inserted without interrupting compressions. Supraglottic airways that have been studied in cardiac arrest are the laryngeal mask airway (LMA), the esophageal-tracheal tube (Combitube) and the laryngeal tube

(Laryngeal Tube or King LT). When prehospital providers are trained in the use of advanced supraglottic airways such as the esophageal-tracheal tube, laryngeal tube, and the laryngeal mask airway, they appear to be able to use these devices safely and can provide ventilation that is as effective as that provided with a bag and mask or an endotracheal tube.[12,35–41]

Advanced airway interventions are technically complicated. Failure can occur; thus maintenance of skills through frequent experience or practice is essential.[42] It is important to remember that there is no evidence that advanced airway measures improve survival rates in the setting of out-of-hospital cardiac arrest. During CPR performed by providers trained in its use, the supraglottic airway is a reasonable alternative to bag-mask ventilation (Class IIa, LOE B) and endotracheal intubation (Class IIa, LOE A).

Esophageal-Tracheal Tube

The advantages of the esophageal-tracheal tube (Combitube) are similar to the advantages of the endotracheal tube when either is compared with bag-mask ventilation: isolation of the airway, reduced risk of aspiration, and more reliable ventilation. The advantages of the esophageal-tracheal tube over the endotracheal tube are related chiefly to ease of training.[12,43] Ventilation and oxygenation with the esophageal-tracheal tube compare favorably with those achieved with the endotracheal tube.[44]

In several controlled clinical trials involving both in-hospital and out-of-hospital resuscitation of adults, providers with all levels of experience were able to insert the esophageal-tracheal tube and deliver ventilation comparable to that achieved with endotracheal intubation.[35,45–48] In a retrospective study no difference in outcome was observed in patients treated with the esophageal-tracheal tube compared with those treated with endotracheal intubation.[38] The esophageal-tracheal tube is reported to provide successful ventilation during CPR in 62% to 100% of patients.[35,45–49] For healthcare professionals trained in its use, the esophageal-tracheal tube is an acceptable alternative to both bag-mask ventilation (Class IIa, LOE C) or endotracheal intubation (Class IIa, LOE A) for airway management in cardiac arrest.

Fatal complications may occur with use of the esophageal-tracheal tube if the position of the distal lumen of the esophageal-tracheal tube in the esophagus or trachea is identified incorrectly. For this reason, confirmation of tube placement is essential. Other possible complications related to the use of the esophageal-tracheal tube are esophageal trauma, including lacerations, bruising, and subcutaneous emphysema.[45,50,51]

Laryngeal Tube

The advantages of the laryngeal tube (Laryngeal Tube or King LT) are similar to those of the esophageal-tracheal tube; however, the laryngeal tube is more compact and less complicated to insert (unlike the esophageal-tracheal tube, the laryngeal tube can only go into the esophagus). At this time there are limited data published on the use of the laryngeal tube in cardiac arrest.[40,41,52,53] In one case series assessing 40 out-of-hospital cardiac arrest patients, insertion of the laryngeal tube by trained paramedics was successful and ventilation was effective in 85% of patients.[41] For 3 patients, ventilation was ineffective because of cuff rupture; for 3 other patients, ventilation was ineffective because of massive regurgitation and aspiration before laryngeal tube placement.

Another out-of-hospital assessment of 157 attempts at laryngeal tube placement revealed a 97% success rate in a mixed population of cardiac arrest and noncardiac arrest patients.[40] For healthcare professionals trained in its use, the laryngeal tube may be considered as an alternative to bag-mask ventilation (Class IIb, LOE C) or endotracheal intubation for airway management in cardiac arrest (Class IIb, LOE C).

Laryngeal Mask Airway

The laryngeal mask airway provides a more secure and reliable means of ventilation than the face mask.[54,55] Although the laryngeal mask airway does not ensure absolute protection against aspiration, studies have shown that regurgitation is less likely with the laryngeal mask airway than with the bag-mask device and that aspiration is uncommon. When compared with the endotracheal tube, the laryngeal mask airway provides equivalent ventilation[49,55]; successful ventilation during CPR has been reported in 72% to 97% of patients.[36,37,44,56–58]

Because insertion of the laryngeal mask airway does not require laryngoscopy and visualization of the vocal cords, training in its placement and use is simpler than that for endotracheal intubation. The laryngeal mask airway also may have advantages over the endotracheal tube when access to the patient is limited,[59,60] there is a possibility of unstable neck injury,[61] or appropriate positioning of the patient for endotracheal intubation is impossible.

Results from studies in anesthetized patients comparing the laryngeal mask airway with endotracheal intubation, as well as additional studies comparing it with other airways or ventilation techniques support the use of the laryngeal mask airway for airway control in a variety of settings by nurses, respiratory therapists, and EMS personnel, many of whom had not previously used this device.[12,39,44,55,62–65]

After successful insertion, a small proportion of patients cannot be ventilated with the laryngeal mask airway.[12,44,55] With this in mind, it is important for providers to have an alternative strategy for airway management. Providers who insert the laryngeal mask airway should receive adequate initial training and then should practice insertion of the device regularly. Success rates and the occurrence of complications should be monitored closely. For healthcare professionals trained in its use, the laryngeal mask airway is an acceptable alternative to bag-mask ventilation (Class IIa, LOE B) or endotracheal intubation (Class IIa, LOE C) for airway management in cardiac arrest.

Endotracheal Intubation

The endotracheal tube was once considered the optimal method of managing the airway during cardiac arrest. However, intubation attempts by unskilled providers can produce complications, such as trauma to the oropharynx, interruption of compressions and ventilations for unacceptably long periods, and hypoxemia from prolonged intubation attempts or failure to recognize tube misplacement or displacement. It is now clear that the incidence of complications is unacceptably high when intubation is performed by inexperienced providers or monitoring of tube placement is inadequate. The optimal method of managing the airway during cardiac arrest will vary based on provider experience, characteristics of the

EMS or healthcare system, and the patient's condition. Frequent experience or frequent retraining is recommended for providers who perform endotracheal intubation (Class I, LOE B).[31,66] EMS systems that perform prehospital intubation should provide a program of ongoing quality improvement to minimize complications (Class IIa, LOE B).

No prospective randomized clinical trials have performed a direct comparison of bag-mask ventilation versus endotracheal intubation in adult victims of cardiac arrest. One prospective, randomized controlled trial in an EMS system with short out-of-hospital transport intervals[67] showed no survival advantage for endotracheal intubation over bag-mask ventilation in children; providers in this study had limited training and experience in intubation.

The endotracheal tube keeps the airway patent, permits suctioning of airway secretions, enables delivery of a high concentration of oxygen, provides an alternative route for the administration of some drugs, facilitates delivery of a selected tidal volume, and, with use of a cuff, may protect the airway from aspiration.

Indications for emergency endotracheal intubation are (1) the inability of the provider to ventilate the unconscious patient adequately with a bag and mask and (2) the absence of airway protective reflexes (coma or cardiac arrest). The provider must have appropriate training and experience in endotracheal intubation.

During CPR providers should minimize the number and duration of interruptions in chest compressions, with a goal to limit interruptions to no more than 10 seconds. Interruptions for supraglottic airway placement should not be necessary at all, whereas interruptions for endotracheal intubation can be minimized if the intubating provider is prepared to begin the intubation attempt—ie, insert the laryngoscope blade with the tube ready at hand—as soon as the compressing provider pauses compressions. Compressions should be interrupted only for the time required by the intubating provider to visualize the vocal cords and insert the tube; this is ideally less than 10 seconds. The compressing provider should be prepared to resume chest compressions immediately after the tube is passed through the vocal cords. If the initial intubation attempt is unsuccessful, a second attempt may be reasonable, but early consideration should be given to using a supraglottic airway.

In retrospective studies, endotracheal intubation has been associated with a 6% to 25% incidence of unrecognized tube misplacement or displacement.[68–72] This may reflect inadequate initial training or lack of experience on the part of the provider who performed intubation, or it may have resulted from displacement of a correctly positioned tube when the patient was moved. The risk of tube misplacement, displacement, or obstruction is high,[67,70] especially when the patient is moved.[73] Thus, even when the endotracheal tube is seen to pass through the vocal cords and tube position is verified by chest expansion and auscultation during positive-pressure ventilation, providers should obtain additional confirmation of placement using waveform capnography or an exhaled CO_2 or esophageal detector device (EDD).[74]

The provider should use both clinical assessment and confirmation devices to verify tube placement immediately after insertion and again when the patient is moved. However, no single confirmation technique is completely reliable.[75,76] Continuous waveform capnography is recommended in addition to clinical assessment as the most reliable method of confirming and monitoring correct placement of an endotracheal tube (Class I, LOE A).

If waveform capnography is not available, an EDD or nonwaveform exhaled CO_2 monitor in addition to clinical assessment is reasonable (Class IIa, LOE B). Techniques to confirm endotracheal tube placement are further discussed below.

Clinical Assessment to Confirm Tube Placement

Providers should perform a thorough assessment of endotracheal tube position immediately after placement. This assessment should not require interruption of chest compressions. Assessment by physical examination consists of visualizing chest expansion bilaterally and listening over the epigastrium (breath sounds should not be heard) and the lung fields bilaterally (breath sounds should be equal and adequate). A device should also be used to confirm correct placement in the trachea (see below). If there is doubt about correct tube placement, use the laryngoscope to visualize the tube passing through the vocal cords. If still in doubt, remove the tube and provide bag-mask ventilation until the tube can be replaced.

Use of Devices to Confirm Tube Placement

Providers should always use both clinical assessment and devices to confirm endotracheal tube location immediately after placement and throughout the resuscitation. Two studies of patients in cardiac arrest[72,77] demonstrated 100% sensitivity and 100% specificity for waveform capnography in identifying correct endotracheal tube placement in victims of cardiac arrest. However, 3 studies demonstrated 64% sensitivity and 100% specificity when waveform capnography was first used for victims with prolonged resuscitation and transport times.[78–80] All confirmation devices should be considered adjuncts to other confirmation techniques.

Exhaled CO_2 Detectors. Detection of exhaled CO_2 is one of several independent methods of confirming endotracheal tube position. Studies of waveform capnography to verify endotracheal tube position in victims of cardiac arrest have shown 100% sensitivity and 100% specificity in identifying correct endotracheal tube placement.[72,77,81–88] Continuous waveform capnography is recommended in addition to clinical assessment as the most reliable method of confirming and monitoring correct placement of an endotracheal tube (Class I, LOE A).

Given the simplicity of colorimetric and nonwaveform exhaled CO_2 detectors, these methods can be used in addition to clinical assessment as the initial method for confirming correct tube placement in a patient in cardiac arrest when waveform capnography is not available (Class IIa, LOE B). However, studies of colorimetric exhaled CO_2 detectors[89–94] and nonwaveform P_{ETCO_2} capnometers[77,89,90,95] indicate that the accuracy of these devices does not exceed that of auscultation and direct visualization for confirming the tracheal position of an endotracheal tube in victims of cardiac arrest.

When exhaled CO_2 is detected (positive reading for CO_2) in cardiac arrest, it is usually a reliable indicator of tube

position in the trachea. False-positive readings (ie, CO_2 is detected but the tube is located in the esophagus) have been observed in animals after ingestion of large amounts of carbonated liquids before the arrest; however, the waveform does not continue during subsequent breaths.[96]

False-negative readings (defined in this context as failure to detect CO_2 despite tube placement in the trachea) may be present during cardiac arrest for several reasons. The most common is that blood flow and delivery of CO_2 to the lungs is low. False-negative results also have been reported in association with pulmonary embolus because pulmonary blood flow and delivery of CO_2 to the lungs are reduced. If the detector is contaminated with gastric contents or acidic drugs (eg, endotracheally administered epinephrine), a colorimetric device may display a constant color rather than breath-to-breath color change. In addition, elimination and detection of CO_2 can be drastically reduced with severe airway obstruction (eg, status asthmaticus) and pulmonary edema.[93,97,98] For these reasons, if CO_2 is not detected, we recommend that a second method be used to confirm endotracheal tube placement, such as direct visualization or the esophageal detector device.

Use of CO_2-detecting devices to determine the correct placement of other advanced airways (eg, Combitube, laryngeal mask airway) has not been studied; their utility will depend on airway design. However, effective ventilation through a supraglottic airway device should result in capnograph waveform during CPR and after ROSC.

Esophageal Detector Devices. The EDD consists of a bulb that is compressed and attached to the endotracheal tube. If the tube is in the esophagus (positive result for an EDD), the suction created by the EDD will collapse the lumen of the esophagus or pull the esophageal tissue against the tip of the tube, and the bulb will not re-expand. The EDD may also consist of a syringe that is attached to the endotracheal tube; the provider attempts to pull the barrel of the syringe. If the tube is in the esophagus, it will not be possible to pull the barrel (aspirate air) with the syringe.

However, studies of the syringe aspiration EDD[79,99] and the self-inflating bulb EDD[78–80] indicate that the accuracy of these devices does not exceed that of auscultation and direct visualization for confirming the tracheal position of an endotracheal tube in victims of cardiac arrest. Given the simplicity of the EDD, it can be used as the initial method for confirming correct tube placement in addition to clinical assessment in the victim of cardiac arrest when waveform capnography is not available (Class IIa, LOE B).

The EDD may yield misleading results in patients with morbid obesity, late pregnancy, or status asthmaticus, or when there are copious endotracheal secretions,[100,101] because the trachea tends to collapse in the presence of these conditions. There is no evidence that the EDD is accurate for the continued monitoring of endotracheal tube placement.

Thoracic Impedance. Transthoracic impedance is slightly but significantly higher during inspiration than during exhalation.[102] Air is a poor electric conductor. Preliminary studies suggest that changes in thoracic impedance, as measured through standard defibrillation pads, may distinguish tracheal from esophageal intubations.[103–105]

There are 2 published reports involving 6 patients where ventilation-induced changes in thoracic impedance disappeared after esophageal intubation.[106,107] There is little evidence for the use of thoracic impedance in diagnosing adequacy of ventilation during CPR. Treatment decisions should not be based solely on thoracic impedance measurements until further study has confirmed its utility and accuracy in this population.

Postintubation Airway Management

After inserting and confirming correct placement of an endotracheal tube, the provider should record the depth of the tube as marked at the front teeth or gums and secure it. There is significant potential for endotracheal tube movement with head flexion and extension[108–110] and when the patient is moved from one location to another.[111,112] Continuous monitoring of endotracheal tube placement with waveform capnography is recommended as discussed above. The endotracheal tube should be secured with tape or a commercial device (Class I, LOE C). Devices and tape should be applied in a manner that avoids compression of the front and sides of the neck, which may impair venous return from the brain.

One out-of-hospital study[113] and 2 studies in an intensive-care setting[114,115] indicate that backboards, commercial devices for securing the endotracheal tube, and other strategies provide equivalent methods for preventing inadvertent tube displacement when compared with traditional methods of securing the tube (tape). These devices may be considered during patient transport (Class IIb, LOE C). After tube confirmation and fixation, obtain a chest x-ray (when feasible) to confirm that the end of the endotracheal tube is properly positioned above the carina.

Ventilation After Advanced Airway Placement

Except for respiratory rate, it is unknown whether monitoring ventilatory parameters (eg, minute ventilation, peak pressure) during CPR will influence outcome. However, positive-pressure ventilation increases intrathoracic pressure and may reduce venous return and cardiac output, especially in patients with hypovolemia or obstructive airway disease. Ventilation at high respiratory rates (>25 breaths per minute) is common during resuscitation from cardiac arrest.[116,117] In animal models, slower ventilation rates (6 to 12 breaths per minute) are associated with improved hemodynamic parameters and short-term survival.[116,118–124]

Because cardiac output is lower than normal during cardiac arrest, the need for ventilation is reduced. Following placement of an advanced airway, the provider delivering ventilations should perform 1 breath every 6 to 8 seconds (8 to 10 breaths per minute) without pausing in applying chest compressions (unless ventilation is inadequate when compressions are not paused) (Class IIb, LOE C). Monitoring respiratory rate coupled with real-time feedback during CPR may result in better compliance with ventilation guidelines.[125]

Automatic Transport Ventilators

In both out-of-hospital and in-hospital settings, automatic transport ventilators (ATVs) can be useful for ventilation of adult patients in noncardiac arrest who have an advanced airway in place (Class IIb, LOE C). There are very few studies evaluating the use of ATVs attached to advanced airways during ongoing resuscitative efforts. During prolonged resuscitative efforts the use of an ATV (pneumatically powered and time- or pressure-cycled) may allow the EMS team to perform other tasks while providing adequate ventilation and oxygenation (Class IIb, LOE C).[126,127] Providers should always have a bag-mask device available for backup.

Suction Devices

Both portable and installed suction devices should be available for resuscitation emergencies. Portable units should provide adequate vacuum and flow for pharyngeal suction. The suction device should be fitted with large-bore, nonkinking suction tubing and semirigid pharyngeal tips. Several sterile suction catheters of various sizes should be available for suctioning the lumen of the advanced airway, along with a nonbreakable collection bottle and sterile water for cleaning tubes and catheters. The installed suction unit should be powerful enough to provide an airflow of >40 L/min at the end of the delivery tube and a vacuum of >300 mm Hg when the tube is clamped. The amount of suction should be adjustable for use in children and intubated patients.

Summary

All basic and advanced healthcare providers should be able to provide ventilation with a bag-mask device during CPR or when the patient demonstrates cardiorespiratory compromise. Airway control with an advanced airway, which may include an endotracheal tube or a supraglottic airway device, is a fundamental ACLS skill. Prolonged interruptions in chest compressions should be avoided during advanced airway placement. All providers should be able to confirm and monitor correct placement of advanced airways; this key skill is required to ensure the safe and effective use of these devices. Training, frequency of use, and monitoring of success and complications are more important than the choice of a specific advanced airway device for use during CPR.

Part 8.2: Management of Cardiac Arrest

Overview

This section details the general care of a patient in cardiac arrest and provides an overview of the 2010 ACLS Adult Cardiac Arrest Algorithms (Figures 1 and 2). Cardiac arrest can be caused by 4 rhythms: ventricular fibrillation (VF), pulseless ventricular tachycardia (VT), pulseless electric activity (PEA), and asystole. VF represents disorganized electric activity, whereas pulseless VT represents organized electric activity of the ventricular myocardium. Neither of these rhythms generates significant forward blood flow. PEA encompasses a heterogeneous group of organized electric rhythms that are associated with either absence of mechanical ventricular activity or mechanical ventricular activity that is insufficient to generate a clinically detectable pulse. Asystole (perhaps better described as ventricular asystole) represents absence of detectable ventricular electric activity with or without atrial electric activity.

Survival from these cardiac arrest rhythms requires both basic life support (BLS) and a system of advanced cardiovascular life support (ACLS) with integrated post–cardiac arrest care. The foundation of successful ACLS is high-quality CPR, and, for VF/pulseless VT, attempted defibrillation within minutes of collapse. For victims of witnessed VF arrest, early CPR and rapid defibrillation can significantly increase the chance for survival to hospital discharge.[128–133] In comparison, other ACLS therapies such as some medications and advanced airways, although associated with an increased rate of ROSC, have not been shown to increase the rate of survival to hospital discharge.[31,33,134–138] The majority of clinical trials testing these ACLS interventions, however, preceded the recently renewed emphasis on high-quality CPR and advances in post–cardiac arrest care (see Part 9: "Post–Cardiac Arrest Care"). Therefore, it remains to be determined if improved rates of ROSC achieved with ACLS interventions might better translate into improved long-term outcomes when combined with higher-quality CPR and post–cardiac arrest interventions such as therapeutic hypothermia and early percutaneous coronary intervention (PCI).

The 2010 ACLS Adult Cardiac Arrest Algorithms (Figures 1 and 2) are presented in the traditional box-and-line format and a new circular format. The 2 formats are provided to facilitate learning and memorization of the treatment recommendations discussed below. Overall these algorithms have been simplified and redesigned to emphasize the importance of high-quality CPR that is fundamental to the management of all cardiac arrest rhythms. Periodic pauses in CPR should be as brief as possible and only as necessary to assess rhythm, shock VF/VT, perform a pulse check when an organized rhythm is detected, or place an advanced airway. Monitoring and optimizing quality of CPR on the basis of either mechanical parameters (chest compression rate and depth, adequacy of relaxation, and minimization of pauses) or, when feasible, physiologic parameters (partial pressure of end-tidal CO_2 [P_{ETCO_2}], arterial pressure during the relaxation phase of chest compressions, or central venous oxygen saturation [S_{cvO_2}]) are encouraged (see "Monitoring During CPR" below). In the absence of an advanced airway, a synchronized compression–ventilation ratio of 30:2 is recommended at a compression rate of at least 100 per minute. After placement of a supraglottic airway or an endotracheal tube, the provider performing chest compressions should deliver at least 100 compressions per minute continuously without pauses for ventilation. The provider delivering ventilations should give 1 breath every 6 to 8 seconds (8 to 10 breaths per minute) and should be particularly careful to avoid delivering an excessive number of ventilations (see Part 8.1: "Adjuncts for Airway Control and Ventilation").

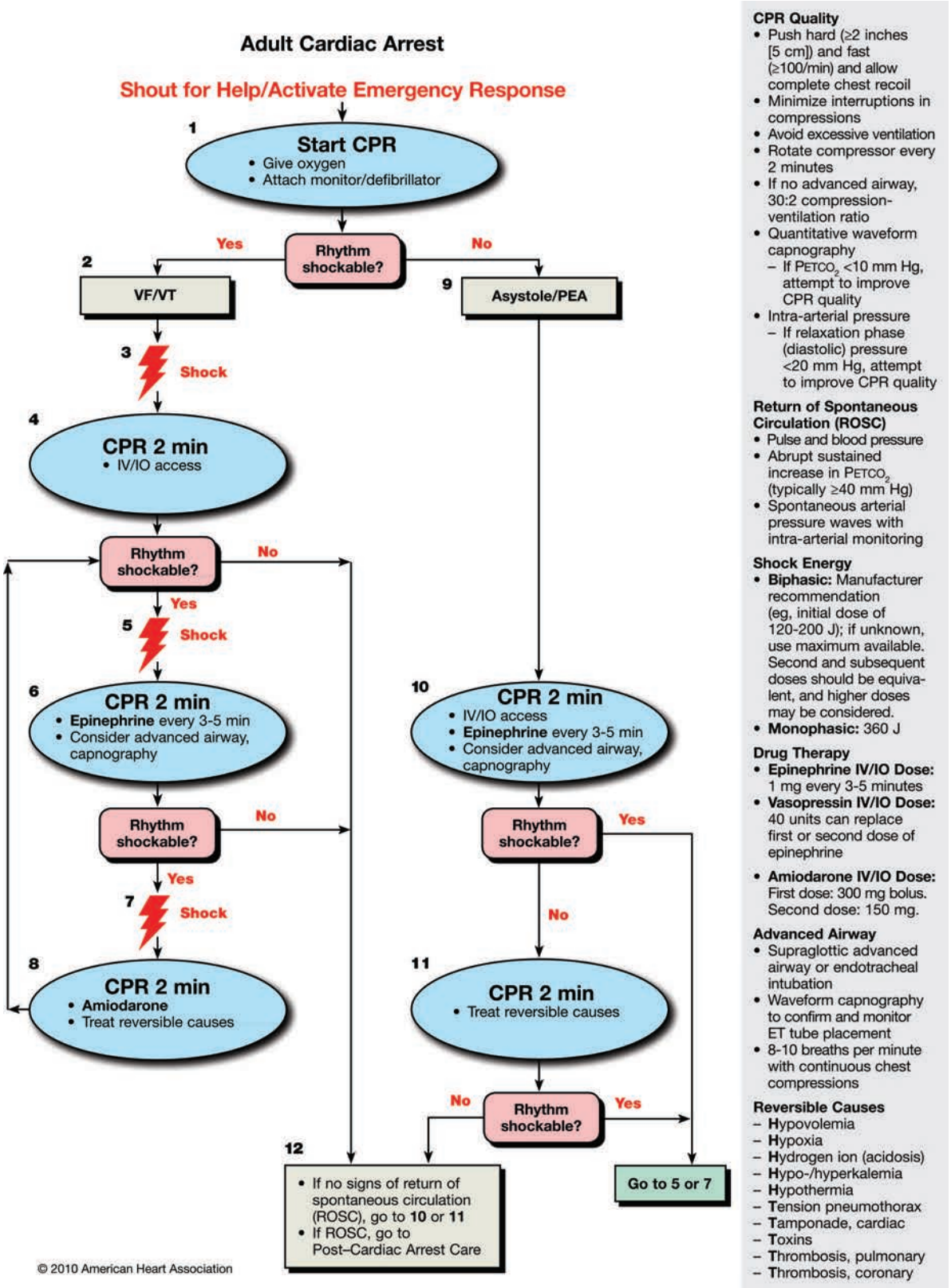
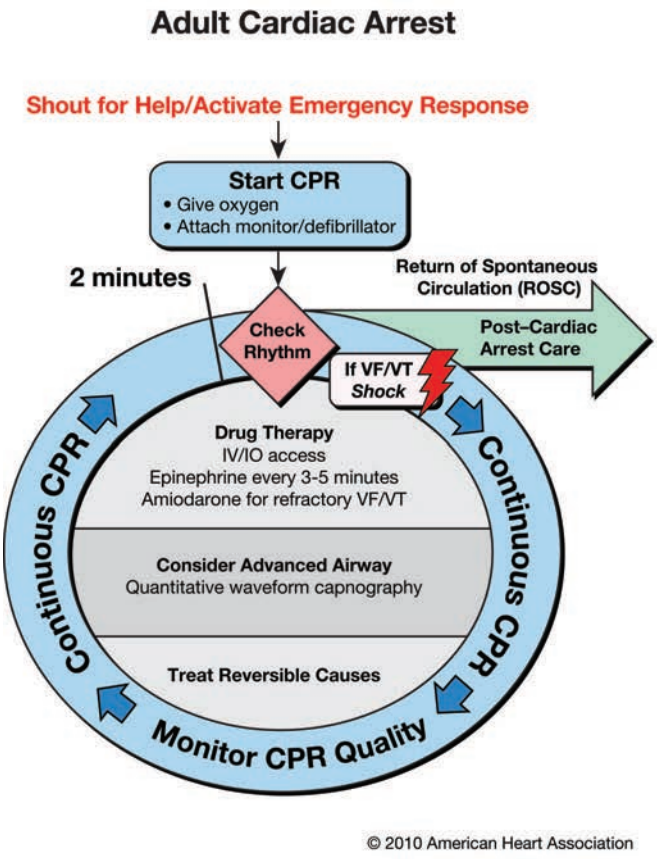


Figure 1. ACLS Cardiac Arrest Algorithm.

In addition to high-quality CPR, the only rhythm-specific therapy proven to increase survival to hospital discharge is defibrillation of VF/pulseless VT. Therefore, this intervention is included as an integral part of the CPR cycle when the rhythm check reveals VF/pulseless VT. Other ACLS interventions during cardiac arrest may be associated with an increased rate of ROSC but have not yet been proven to increase survival to hospital discharge. Therefore, they are



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 (eg, 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
- **Vasopressin 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 2. ACLS Cardiac Arrest Circular Algorithm.

recommended as considerations and should be performed without compromising quality of CPR or timely defibrillation. In other words, vascular access, drug delivery, and advanced airway placement should not cause significant interruptions in chest compression or delay defibrillation. There is insufficient evidence to recommend a specific timing or sequence (order) of drug administration and advanced airway placement during cardiac arrest. In most cases the timing and sequence of these secondary interventions will depend on the number of providers participating in the resuscitation and their skill levels. Timing and sequence will also be affected by whether vascular access has been established or an advanced airway placed before cardiac arrest.

Understanding the importance of diagnosing and treating the underlying cause is fundamental to management of all cardiac arrest rhythms. During management of cardiac arrest the provider should consider the H's and T's to identify and treat any factor that may have caused the arrest or may be complicating the resuscitative effort (Table 1).

It is common for the arrest rhythm to evolve during the course of resuscitation. In such cases management should shift smoothly to the appropriate rhythm-based strategy. In particular, providers should be prepared to deliver a timely shock when a patient who presented with asystole or PEA is found to be in VF/pulseless VT during a rhythm check. There is no evidence that the resuscitation strategy for a new cardiac arrest rhythm should necessarily be altered based on the characteristics of the previous rhythm. Medications administered during resuscitation should be monitored and total doses tabulated to avoid potential toxicity.

If the patient achieves ROSC, it is important to begin post–cardiac arrest care immediately to avoid rearrest and optimize the patient's chance of long-term survival with good neurologic function (see Part 9). Finally, the reality is that the majority of resuscitative efforts do not result in ROSC. Criteria for ending unsuccessful resuscitative efforts are addressed briefly below (see "When Should Resuscitative Efforts Stop?") and in more detail in Part 3: "Ethics."

Rhythm-Based Management of Cardiac Arrest

In most cases of witnessed and unwitnessed cardiac arrest the first provider should start CPR with chest compressions and the second provider should get or turn on the defibrillator,

Table 1. Treatable Causes of Cardiac Arrest: The H's and T's

H's	T's
Hypoxia	Toxins
Hypovolemia	Tamponade (cardiac)
Hydrogen ion (acidosis)	Tension pneumothorax
Hypo-/hyperkalemia	Thrombosis, pulmonary
Hypothermia	Thrombosis, coronary

For further explanation of the H's and T's, see Part 12: "Special Resuscitation Situations."

place the adhesive pads or paddles, and check the rhythm. Paddles and electrode pads should be placed on the exposed chest in an anterior-lateral position. Acceptable alternative positions are anterior-posterior, anterior-left infrascapular, and anterior-right infrascapular. Rhythm checks should be brief, and if an organized rhythm is observed, a pulse check should be performed. If there is any doubt about the presence of a pulse, chest compressions should be resumed immediately. If a cardiac monitor is attached to the patient at the time of arrest, the rhythm can be diagnosed before CPR is initiated.

VF/Pulseless VT

When a rhythm check by an automated external defibrillator (AED) reveals VF/VT, the AED will typically prompt to charge, "clear" the victim for shock delivery, and then deliver a shock, all of which should be performed as quickly as possible. CPR should be resumed immediately after shock delivery (without a rhythm or pulse check and beginning with chest compressions) and continue for 2 minutes before the next rhythm check.

When a rhythm check by a manual defibrillator reveals VF/VT, the first provider should resume CPR while the second provider charges the defibrillator. Once the defibrillator is charged, CPR is paused to "clear" the patient for shock delivery. After the patient is "clear," the second provider gives a single shock as quickly as possible to minimize the interruption in chest compressions ("hands-off interval"). The first provider resumes CPR immediately after shock delivery (without a rhythm or pulse check and beginning with chest compressions) and continues for 2 minutes. After 2 minutes of CPR the sequence is repeated, beginning with a rhythm check.

The provider giving chest compressions should switch at every 2-minute cycle to minimize fatigue. CPR quality should be monitored based on mechanical or physiologic parameters (see "Monitoring During CPR" below).

Defibrillation Strategies

Waveform and Energy

If a biphasic defibrillator is available, providers should use the manufacturer's recommended energy dose (eg, initial dose of 120 to 200 J) for terminating VF (Class I, LOE B). If the provider is unaware of the effective dose range, the provider may use the maximal dose (Class IIb, LOE C). Second and subsequent energy levels should be at least equivalent, and higher energy levels may be considered if available (Class IIb, LOE B). If a monophasic defibrillator is used, providers should deliver an initial shock of 360 J and use that dose for all subsequent shocks. If VF is terminated by a shock but then recurs later in the arrest, deliver subsequent shocks at the previously successful energy level.

Automatic Versus Manual Modes for Multimodal Defibrillators

Use of a multimodal defibrillator in manual mode may reduce the duration of interruption of CPR required for rhythm analysis compared with automatic mode but could increase the frequency of inappropriate shock.[139,140] Current evidence indicates that the benefit of using a multimodal defibrillator in manual instead of automatic mode during cardiac arrest is uncertain (Class IIb, LOE C).

CPR Before Defibrillation

During treatment of VF/pulseless VT healthcare providers must ensure that coordination between CPR and shock delivery is efficient. When VF is present for more than a few minutes, the myocardium is depleted of oxygen and metabolic substrates. A brief period of chest compressions can deliver oxygen and energy substrates and "unload" the volume-overloaded right ventricle, increasing the likelihood that a perfusing rhythm will return after shock delivery.[141]

Performing CPR while a defibrillator is readied for use is strongly recommended for all patients in cardiac arrest (Class I, LOE B). Analyses of VF waveform characteristics predictive of shock success have documented that the shorter the time interval between the last chest compression and shock delivery, the more likely the shock will be successful.[141] A reduction of even a few seconds in the interval from pausing compressions to shock delivery can increase the probability of shock success.[142]

The value of intentionally delaying defibrillation to perform CPR is less clear. One randomized controlled trial (RCT)[143] and one clinical trial[144] involving adults with out-of-hospital cardiac arrest not witnessed by EMS personnel showed that survival was improved by a period of CPR performed before the first defibrillation shock when the EMS response interval was >4 to 5 minutes. But 2 RCTs[145,146] demonstrated no improvement in ROSC or survival to hospital discharge in patients with out-of-hospital VF or pulseless VT who received CPR from EMS personnel for 1.5 to 3 minutes before defibrillation, regardless of EMS response interval. At this time the benefit of delaying defibrillation to perform CPR before defibrillation is unclear (Class IIb, LOE B).

VF Waveform Analysis to Predict Defibrillation Success

Retrospective analysis of VF waveforms in multiple clinical studies suggests that it is possible to predict the success of defibrillation from the fibrillation waveform with varying reliability.[141,147–166] No prospective human studies have specifically evaluated whether treatment altered by predicting success of defibrillation can improve successful defibrillation, rate of ROSC, or survival from cardiac arrest. The value of VF waveform analysis to guide management of defibrillation in adults with in-hospital and out-of-hospital cardiac arrest is uncertain (Class IIb, LOE C).

Drug Therapy in VF/Pulseless VT

When VF/pulseless VT persists after at least 1 shock and a 2-minute CPR period, a vasopressor can be given with the primary goal of increasing myocardial blood flow during CPR and achieving ROSC (see "Medications for Arrest Rhythms" below for dosing) (Class IIb, LOE A). The peak effect of an intravenous (IV)/intraosseous (IO) vasopressor given as a bolus dose during CPR is delayed for at least 1 to 2 minutes. The optimal timing of vasopressor administration during the 2-minute period of uninterrupted CPR has not been established. If a shock fails to generate a perfusing rhythm, then giving a vasopressor soon after the shock will optimize

the potential impact of increased myocardial blood flow before the next shock. However, if a shock results in a perfusing rhythm, a bolus dose of vasopressor at any time during the subsequent 2-minute period of CPR (before rhythm check) could theoretically have detrimental effects on cardiovascular stability. This may be avoided by using physiologic monitoring such as quantitative waveform capnography, intra-arterial pressure monitoring, and continuous central venous oxygen saturation monitoring to detect ROSC during chest compressions.[93,167–177] However, adding an additional pause for rhythm and pulse check after shock delivery but before vasopressor therapy will decrease myocardial perfusion during the critical postshock period and could reduce the chance of achieving ROSC.

Amiodarone is the first-line antiarrhythmic agent given during cardiac arrest because it has been clinically demonstrated to improve the rate of ROSC and hospital admission in adults with refractory VF/pulseless VT. Amiodarone may be considered when VF/VT is unresponsive to CPR, defibrillation, and vasopressor therapy (Class IIb, LOE A). If amiodarone is unavailable, lidocaine may be considered, but in clinical studies lidocaine has not been demonstrated to improve rates of ROSC and hospital admission compared with amiodarone (Class IIb, LOE B). Magnesium sulfate should be considered only for torsades de pointes associated with a long QT interval (Class IIb, LOE B).

Treating Potentially Reversible Causes of VF/Pulseless VT

The importance of diagnosing and treating the underlying cause of VF/pulseless VT is fundamental to the management of all cardiac arrest rhythms. As always, the provider should recall the H's and T's to identify a factor that may have caused the arrest or may be complicating the resuscitative effort (see Table 1 and Part 12: "Special Resuscitation Situations"). In the case of refractory VF/pulseless VT, acute coronary ischemia or myocardial infarction should be considered as a potential etiology. Reperfusion strategies such as coronary angiography and PCI during CPR or emergency cardiopulmonary bypass have been demonstrated to be feasible in a number of case studies and case series but have not been evaluated for their effectiveness in RCTs.[178–187] Fibrinolytic therapy administered during CPR for acute coronary occlusion has not been shown to improve outcome.[188]

ROSC After VF/Pulseless VT

If the patient has ROSC, post–cardiac arrest care should be started (Part 9). Of particular importance are treatment of hypoxemia and hypotension, early diagnosis and treatment of ST-elevation myocardial infarction (STEMI) (Class I, LOE B) and therapeutic hypothermia in comatose patients (Class I, LOE B).

PEA/Asystole

When a rhythm check by an AED reveals a nonshockable rhythm, CPR should be resumed immediately, beginning with chest compressions, and should continue for 2 minutes before the rhythm check is repeated. When a rhythm check using a manual defibrillator or cardiac monitor reveals ***an organized rhythm***, a pulse check is performed. If a pulse is detected, post–cardiac arrest care should be initiated immediately (see Part 9). If the rhythm is asystole or the pulse is absent (eg, PEA), CPR should be resumed immediately, beginning with chest compressions, and should continue for 2 minutes before the rhythm check is repeated. The provider performing chest compressions should switch every 2 minutes. CPR quality should be monitored on the basis of mechanical or physiologic parameters (see "Monitoring During CPR" below).

Drug Therapy for PEA/Asystole

A vasopressor can be given as soon as feasible with the primary goal of increasing myocardial and cerebral blood flow during CPR and achieving ROSC (see "Vasopressors" below for dosing) (Class IIb, LOE A). Available evidence suggests that the routine use of atropine during PEA or asystole is unlikely to have a therapeutic benefit (Class IIb, LOE B). For this reason atropine has been removed from the cardiac arrest algorithm.

Treating Potentially Reversible Causes of PEA/Asystole

PEA is often caused by reversible conditions and can be treated successfully if those conditions are identified and corrected. During each 2-minute period of CPR the provider should recall the H's and T's to identify factors that may have caused the arrest or may be complicating the resuscitative effort (see Table 1 and Part 12: "Special Resuscitation Situations"). Given the potential association of PEA with hypoxemia, placement of an advanced airway is theoretically more important than during VF/pulseless VT and might be necessary to achieve adequate oxygenation or ventilation. PEA caused by severe volume loss or sepsis will potentially benefit from administration of empirical IV/IO crystalloid. A patient with PEA caused by severe blood loss will potentially benefit from a blood transfusion. When pulmonary embolism is presumed or known to be the cause of cardiac arrest, empirical fibrinolytic therapy can be considered (Class IIa, LOE B; see Part 12). Finally, if tension pneumothorax is clinically suspected as the cause of PEA, initial management includes needle decompression. If available, echocardiography can be used to guide management of PEA because it provides useful information about intravascular volume status (assessing ventricular volume), cardiac tamponade, mass lesions (tumor, clot), left ventricular contractility, and regional wall motion.[189] See Part 12 for management of toxicological causes of cardiac arrest.

Asystole is commonly the end-stage rhythm that follows prolonged VF or PEA, and for this reason the prognosis is generally much worse.

ROSC After PEA/Asystole

If the patient has ROSC, post–cardiac arrest care should be initiated (see Part 9). Of particular importance is treatment of hypoxemia and hypotension and early diagnosis and treatment of the underlying cause of cardiac arrest. Therapeutic hypothermia may be considered when the patient is comatose (Class IIb, LOE C).

Monitoring During CPR

Mechanical Parameters

CPR quality can be improved by using a number of nonphysiologic techniques that help the provider adhere to recom-

mended CPR parameters such as rate and depth of compression and rate of ventilation. The most simple are auditory or visual metronomes to guide providers in performing the recommended rate of chest compressions or ventilations. More sophisticated devices actually monitor chest compression rate, depth, relaxation, and pauses in real time and provide visual and auditory feedback. When recorded, this information can also be useful in providing feedback to the entire team of providers after the resuscitation has ended. This type of CPR quality monitoring is discussed in more detail in Part 5: "Adult Basic Life Support" and Part 16: "Education, Implementation and Teams."

Physiologic Parameters

In humans cardiac arrest is the most critically ill condition, yet it is typically monitored by rhythm assessment using selected electocardiographic (ECG) leads and pulse checks as the only physiologic parameters to guide therapy. Animal and human studies indicate that monitoring of P_{ETCO_2}, coronary perfusion pressure (CPP), and central venous oxygen saturation (Scv_{O_2}) provides valuable information on both the patient's condition and response to therapy. Most importantly, P_{ETCO_2}, CPP, and Scv_{O_2} correlate with cardiac output and myocardial blood flow during CPR, and threshold values below which ROSC is rarely achieved have been reported.[168,190–195] Furthermore, an abrupt increase in any of these parameters is a sensitive indicator of ROSC that can be monitored without interrupting chest compressions.[91,93,167–175,177,196–201] Although no clinical study has examined whether titrating resuscitative efforts to these or other physiologic parameters improves outcome, it is reasonable to consider using these parameters when feasible to optimize chest compressions and guide vasopressor therapy during cardiac arrest (Class IIb, LOE C).

Pulse

Clinicians frequently try to palpate arterial pulses during chest compressions to assess the effectiveness of compressions. No studies have shown the validity or clinical utility of checking pulses during ongoing CPR. Because there are no valves in the inferior vena cava, retrograde blood flow into the venous system may produce femoral vein pulsations.[202] Thus, palpation of a pulse in the femoral triangle may indicate venous rather than arterial blood flow. Carotid pulsations during CPR do not indicate the efficacy of myocardial or cerebral perfusion during CPR. Palpation of a pulse when chest compressions are paused is a reliable indicator of ROSC but is potentially less sensitive than other physiologic measures discussed below.

Healthcare providers also may take too long to check for a pulse[203,204] and have difficulty determining if a pulse is present or absent.[203–205] There is no evidence, however, that checking for breathing, coughing, or movement is superior for detection of circulation.[206] Because delays in chest compressions should be minimized, the healthcare provider should take no more than 10 seconds to check for a pulse, and if it is not felt within that time period chest compressions should be started.[205,207]

End-Tidal CO_2

End-tidal CO_2 is the concentration of carbon dioxide in exhaled air at the end of expiration. It is typically expressed as a partial pressure in mm Hg (P_{ETCO_2}). Because CO_2 is a trace gas in atmospheric air, CO_2 detected by capnography in exhaled air is produced in the body and delivered to the lungs by circulating blood. Under normal conditions P_{ETCO_2} is in the range of 35 to 40 mm Hg. During untreated cardiac arrest CO_2 continues to be produced in the body, but there is no CO_2 delivery to the lungs. Under these conditions P_{ETCO_2} will approach zero with continued ventilation. With initiation of CPR, cardiac output is the major determinant of CO_2 delivery to the lungs. If ventilation is relatively constant, P_{ETCO_2} correlates well with cardiac output during CPR. The correlation between P_{ETCO_2} and cardiac output during CPR can be transiently altered by giving IV sodium bicarbonate.[208] This is explained by the fact that the bicarbonate is converted to water and CO_2, causing a transient increase in delivery of CO_2 to the lungs. Therefore, a transient rise in P_{ETCO_2} after sodium bicarbonate therapy is expected and should not be misinterpreted as an improvement in quality of CPR or a sign of ROSC. Animal and human studies have also shown that P_{ETCO_2} correlates with CPP and cerebral perfusion pressure during CPR.[209,210] The correlation of P_{ETCO_2} with CPP during CPR can be altered by vasopressor therapy, especially at high doses (ie, >1 mg of epinephrine).[211–214] Vasopressors cause increased afterload, which will increase blood pressure and myocardial blood flow during CPR but will also decrease cardiac output. Therefore, a small decrease in P_{ETCO_2} after vasopressor therapy may occur but should not be misinterpreted as a decrease in CPR quality.

Persistently low P_{ETCO_2} values (<10 mm Hg) during CPR in intubated patients suggest that ROSC is unlikely.[171,173,174,190,191,215,216] Similar data using quantitative monitoring of P_{ETCO_2} are not available for patients with a supraglottic airway or those receiving bag-mask ventilation during CPR. One study using colorimetic end-tidal CO_2 detection in nonintubated patients during CPR found that low end-tidal CO_2 was not a reliable predictor of failure to achieve ROSC.[217] An air leak during bag-mask ventilation or ventilation with a supraglottic airway could result in lower measured P_{ETCO_2} values. Although a P_{ETCO_2} value of <10 mm Hg in intubated patients indicates that cardiac output is inadequate to achieve ROSC, a specific target P_{ETCO_2} value that optimizes the chance of ROSC has not been established. Monitoring P_{ETCO_2} trends during CPR has the potential to guide individual optimization of compression depth and rate and to detect fatigue in the provider performing compressions.[201,218,219] In addition, an abrupt sustained increase in P_{ETCO_2} during CPR is an indicator of ROSC.[91,177,196,198–201] Therefore, it is reasonable to consider using quantitative waveform capnography in intubated patients to monitor CPR quality, optimize chest compressions, and detect ROSC during chest compressions or when rhythm check reveals an organized rhythm (Class IIb, LOE C). If P_{ETCO_2} is <10 mm Hg, it is reasonable to consider trying to improve CPR quality by optimizing chest compression parameters (Class IIb, LOE C). If P_{ETCO_2} abruptly increases to a normal value (35 to 40 mm Hg), it is reasonable to consider that this is an indicator of ROSC (Class IIa, LOE B). The

value of using quantitative waveform capnography in nonintubated patients to monitor and optimize CPR quality and detect ROSC is uncertain (Class IIb, LOE C).

Coronary Perfusion Pressure and Arterial Relaxation Pressure

CPP (coronary perfusion pressure=aortic relaxation ["diastolic"] pressure minus right atrial relaxation ["diastolic"] pressure) during CPR correlates with both myocardial blood flow and ROSC.[168,192,220] Relaxation pressure during CPR is the trough of the pressure waveform during the relaxation phase of chest compressions and is analogous to diastolic pressure when the heart is beating. Increased CPP correlates with improved 24-hour survival rates in animal studies[193] and is associated with improved myocardial blood flow and ROSC in animal studies of epinephrine, vasopressin, and angiotensin II.[193–195] In one human study ROSC did not occur unless a CPP ≥15 mm Hg was achieved during CPR.[168] However, monitoring of CPP during CPR is rarely available clinically because measurement and calculation require simultaneous recording of aortic and central venous pressure.

A reasonable surrogate for CPP during CPR is arterial relaxation ("diastolic") pressure, which can be measured using a radial, brachial, or femoral artery catheter. These closely approximate aortic relaxation pressures during CPR in humans.[211,221] The same study that identified a CPP threshold of ≥15 mm Hg for ROSC also reported that ROSC was not achieved if aortic relaxation "diastolic" pressure did not exceed 17 mm Hg during CPR.[168] A specific target arterial relaxation pressure that optimizes the chance of ROSC has not been established. It is reasonable to consider using arterial relaxation "diastolic" pressure to monitor CPR quality, optimize chest compressions, and guide vasopressor therapy. (Class IIb, LOE C). If the arterial relaxation "diastolic" pressure is <20 mm Hg, it is reasonable to consider trying to improve quality of CPR by optimizing chest compression parameters or giving a vasopressor or both (Class IIb, LOE C). Arterial pressure monitoring can also be used to detect ROSC during chest compressions or when a rhythm check reveals an organized rhythm (Class IIb, LOE C).

Central Venous Oxygen Saturation

When oxygen consumption, arterial oxygen saturation (SaO_2), and hemoglobin are constant, changes in $Scvo_2$ reflect changes in oxygen delivery by means of changes in cardiac output. $Scvo_2$ can be measured continuously using oximetric tipped central venous catheters placed in the superior vena cava. $Scvo_2$ values normally range from 60% to 80%. During cardiac arrest and CPR these values range from 25% to 35%, indicating the inadequacy of blood flow produced during CPR. In one clinical study the failure to achieve $Scvo_2$ of 30% during CPR was associated with failure to achieve ROSC.[169] $Scvo_2$ also helps to rapidly detect ROSC without interrupting chest compressions to check rhythm and pulse. When available, continuous $Scvo_2$ monitoring is a potentially useful indicator of cardiac output and oxygen delivery during CPR. Therefore, when in place before cardiac arrest, it is reasonable to consider using continuous $Scvo_2$ measurement to monitor quality of CPR, optimize chest compressions, and detect ROSC during chest compressions or when rhythm check reveals an organized rhythm (Class IIb, LOE C). If $Scvo_2$ is <30%, it is reasonable to consider trying to improve the quality of CPR by optimizing chest compression parameters (Class IIb, LOE C).

Pulse Oximetry

During cardiac arrest, pulse oximetry typically does not provide a reliable signal because pulsatile blood flow is inadequate in peripheral tissue beds. But the presence of a plethysmograph waveform on pulse oximetry is potentially valuable in detecting ROSC, and pulse oximetry is useful to ensure appropriate oxygenation after ROSC.

Arterial Blood Gases

Arterial blood gas monitoring during CPR is not a reliable indicator of the severity of tissue hypoxemia, hypercarbia (and therefore adequacy of ventilation during CPR), or tissue acidosis.[222] Routine measurement of arterial blood gases during CPR has uncertain value (Class IIb, LOE C).

Echocardiography

No studies specifically examine the impact of echocardiography on patient outcomes in cardiac arrest. However, a number of studies suggest that transthoracic and transesophageal echocardiography have potential utility in diagnosing treatable causes of cardiac arrest such as cardiac tamponade, pulmonary embolism, ischemia, and aortic dissection.[223–227] In addition, 3 prospective studies[228–230] found that absence of cardiac motion on sonography during resuscitation of patients in cardiac arrest was highly predictive of inability to achieve ROSC: of the 341 patients from the 3 studies, 218 had no detectable cardiac activity and only 2 of these had ROSC (no data on survival-to-hospital discharge were reported). Transthoracic or transesophageal echocardiography may be considered to diagnose treatable causes of cardiac arrest and guide treatment decisions (Class IIb, LOE C).

Access for Parenteral Medications During Cardiac Arrest

Timing of IV/IO Access

During cardiac arrest, provision of high-quality CPR and rapid defibrillation are of primary importance and drug administration is of secondary importance. After beginning CPR and attempting defibrillation for identified VF or pulseless VT, providers can establish IV or IO access. This should be performed without interrupting chest compressions. The primary purpose of IV/IO access during cardiac arrest is to provide drug therapy. Two clinical studies[134,136] reported data suggesting worsened survival for every minute that antiarrhythmic drug delivery was delayed (measured from time of dispatch). However, this finding was potentially biased by a concomitant delay in onset of other ACLS interventions. In one study[136] the interval from first shock to administration of an antiarrhythmic drug was a significant predictor of survival.

One animal study[231] reported lower CPP when delivery of a vasopressor was delayed. Time to drug administration was also a predictor of ROSC in a retrospective analysis of swine cardiac arrest.[232] Thus, although time to drug treatment appears to have importance, there is insufficient evidence to specify exact time parameters or the precise sequence with which drugs should be administered during cardiac arrest.

Peripheral IV Drug Delivery

If a resuscitation drug is administered by a peripheral venous route, it should be administered by bolus injection and followed with a 20-mL bolus of IV fluid to facilitate the drug flow from the extremity into the central circulation.[233] Briefly elevating the extremity during and after drug administration theoretically may also recruit the benefit of gravity to facilitate delivery to the central circulation but has not been systematically studied.

IO Drug Delivery

IO cannulation provides access to a noncollapsible venous plexus, enabling drug delivery similar to that achieved by peripheral venous access at comparable doses. Two prospective trials in children[234] and adults[235] and 6 other studies[236–242] suggest that IO access can be established efficiently; is safe and effective for fluid resuscitation, drug delivery, and blood sampling for laboratory evaluation; and is attainable in all age groups. However, many of these studies were conducted during normal perfusion states or hypovolemic shock or in animal models of cardiac arrest. Although virtually all ACLS drugs have been given intraosseously in the clinical setting without known ill effects, there is little information on the efficacy and effectiveness of such administration in clinical cardiac arrest during ongoing CPR. It is reasonable for providers to establish IO access if IV access is not readily available (Class IIa, LOE C). Commercially available kits can facilitate IO access in adults.

Central IV Drug Delivery

The appropriately trained provider may consider placement of a central line (internal jugular or subclavian) during cardiac arrest, unless there are contraindications (Class IIb, LOE C). The primary advantage of a central line is that peak drug concentrations are higher and drug circulation times shorter compared with drugs administered through a peripheral IV catheter.[243–245] In addition, a central line extending into the superior vena cava can be used to monitor $Scvo_2$ and estimate CPP during CPR, both of which are predictive of ROSC.[168,169] However, central line placement can interrupt CPR. Central venous catheterization is a relative (but not absolute) contraindication for fibrinolytic therapy in patients with acute coronary syndromes.

Endotracheal Drug Delivery

One study in children,[246] 5 studies in adults,[247–251] and multiple animal studies[252–254] have shown that lidocaine,[248,255] epinephrine,[256] atropine,[257] naloxone, and vasopressin[254] are absorbed via the trachea. There are no data regarding endotracheal administration of amiodarone. Administration of resuscitation drugs into the trachea results in lower blood concentrations than when the same dose is given intravascularly. Furthermore, the results of recent animal studies[258,259] suggest that the lower epinephrine concentrations achieved when the drug is delivered endotracheally may produce transient β-adrenergic effects, resulting in vasodilation. These effects can be detrimental, causing hypotension, lower CPP and flow, and reduced potential for ROSC. Thus, although endotracheal administration of some resuscitation drugs is possible, IV or IO drug administration is preferred because it will provide more predictable drug delivery and pharmacologic effect.

In one nonrandomized cohort study of out-of-hospital cardiac arrest in adults[260] using a randomized control, IV administration of atropine and epinephrine was associated with a higher rate of ROSC and survival to hospital admission than administration by the endotracheal route. Five percent of those who received IV drugs survived to hospital discharge, but no patient survived in the group receiving drugs by the endotracheal route.

If IV or IO access cannot be established, epinephrine, vasopressin, and lidocaine may be administered by the endotracheal route during cardiac arrest (Class IIb, LOE B). The optimal endotracheal dose of most drugs is unknown, but typically the dose given by the endotracheal route is 2 to 2½ times the recommended IV dose. In 2 animal CPR studies the equipotent epinephrine dose given endotracheally was approximately 3 to 10 times higher than the IV dose.[261,262] Providers should dilute the recommended dose in 5 to 10 mL of sterile water or normal saline and inject the drug directly into the endotracheal tube.[256] Studies with epinephrine[263] and lidocaine[251] showed that dilution with sterile water instead of 0.9% saline may achieve better drug absorption.

Advanced Airway

There is inadequate evidence to define the optimal timing of advanced airway placement in relation to other interventions during resuscitation from cardiac arrest. There are no prospective studies that directly address the relationship between timing or type of advanced airway placement during CPR and outcomes. In an urban out-of-hospital setting, intubation in $<$12 minutes has been associated with a better rate of survival than intubation in $\geq$13 minutes.[32] In a registry study of 25 006 in-hospital cardiac arrests, earlier time to advanced airway ($<$5 minutes) was not associated with increased ROSC but was associated with improved 24-hour survival.[31] In out-of-hospital urban and rural settings, patients intubated during resuscitation had better survival rates than patients who were not intubated.[33] In an in-hospital setting patients requiring intubation during CPR had worse survival rates.[34] A recent study[8] found that delayed endotracheal intubation combined with passive oxygen delivery and minimally interrupted chest compressions was associated with improved neurologically intact survival after out-of-hospital cardiac arrest in patients with witnessed VF/VT.

Advantages of advanced airway placement include elimination of the need for pauses in chest compressions for

ventilation, potentially improved ventilation and oxygenation, reduction in the risk of aspiration, and ability to use quantitative waveform capnography to monitor quality of CPR, optimize chest compressions, and detect ROSC during chest compressions or when a rhythm check reveals an organized rhythm. The primary disadvantages are interruptions in chest compression during placement and the risk of unrecognized esophageal intubation.

When an advanced airway (eg, endotracheal tube or supraglottic airway) is placed, 2 providers no longer deliver cycles of compressions interrupted with pauses for ventilation. Instead, the provider performing compressions should deliver at least 100 compressions per minute continuously without pauses for ventilation. The provider delivering ventilations should give 1 breath every 6 to 8 seconds (8 to 10 breaths per minute) and should be careful to avoid delivering an excessive number of ventilations.

When Should Resuscitative Efforts Stop?

The final decision to stop can never rest on a single parameter, such as duration of resuscitative efforts. Rather, clinical judgment and respect for human dignity must enter into decision making. In the out-of-hospital setting, cessation of resuscitative efforts in adults should follow system-specific criteria under direct medical control. There are limited clinical data to guide this decision in neonatal and pediatric out-of-hospital or in-hospital cardiac arrest. A more detailed discussion is provided in Part 3: "Ethics."

Medications for Arrest Rhythms

The primary goal of pharmacologic therapy during cardiac arrest is to facilitate restoration and maintenance of a perfusing spontaneous rhythm. Toward this goal, ACLS drug therapy during CPR is often associated with increased rates of ROSC and hospital admission but not increased rates of long-term survival with good neurologic outcome. One study[138] randomized patients to IV or no IV medications during management of adult out-of-hospital cardiac arrest. The study demonstrated higher rates of ROSC in the IV group (40% IV versus 25% no IV [odds ratio (OR) 1.99; 95% confidence interval (CI) 1.48 to 2.67]), but there was no statistical difference in survival to hospital discharge (10.5% IV versus 9.2% no IV [OR 1.16; 95% CI 0.74 to 1.82]) or survival with favorable neurologic outcome (9.8% IV versus 8.1% no IV [OR 1.24; 95% CI 0.77 to 1.98]). This study was not adequately powered to detect clinically important differences in long-term outcomes. Evidence from one nonrandomized trial[137] found that the addition of ACLS interventions including IV drugs in a previously optimized BLS system with rapid defibrillation resulted in an increased rate of ROSC (18.0% with ACLS versus 12.9% before ACLS, $P<0.001$) and hospital admission (14.6% with ACLS versus 10.9% before ACLS, $P<0.001$) but no statistical difference in survival to hospital discharge (5.1% with ACLS versus 5.0% before ACLS). Whether optimized high-quality CPR and advances in post–cardiac arrest care will enable the increased rates of ROSC with ACLS medications to be translated into increased long-term survival remains to be determined.

Vasopressors

To date no placebo-controlled trials have shown that administration of any vasopressor agent at any stage during management of VF, pulseless VT, PEA, or asystole increases the rate of neurologically intact survival to hospital discharge. There is evidence, however, that the use of vasopressor agents is associated with an increased rate of ROSC.

Epinephrine

Epinephrine hydrochloride produces beneficial effects in patients during cardiac arrest, primarily because of its α-adrenergic receptor-stimulating (ie, vasoconstrictor) properties.[264] The α-adrenergic effects of epinephrine can increase CPP and cerebral perfusion pressure during CPR.[265] The value and safety of the β-adrenergic effects of epinephrine are controversial because they may increase myocardial work and reduce subendocardial perfusion.[266]

There are no RCTs that adequately compare epinephrine with placebo in treatment of and outcomes related to out-of-hospital cardiac arrest. A retrospective study[267] compared epinephrine to no epinephrine for sustained VF and PEA/asystole and found improved ROSC with epinephrine but no difference in survival between the treatment groups. A meta-analysis and other studies have found improved ROSC, but none have demonstrated a survival benefit of high-dose epinephrine versus standard-dose epinephrine in cardiac arrest.[135,268–272]

It is reasonable to consider administering a 1 mg dose of IV/IO epinephrine every 3 to 5 minutes during adult cardiac arrest (Class IIb, LOE A). Higher doses may be indicated to treat specific problems, such as a β-blocker or calcium channel blocker overdose. Higher doses can also be considered if guided by hemodynamic monitoring such as arterial relaxation "diastolic" pressure or CPP. If IV/IO access is delayed or cannot be established, epinephrine may be given endotracheally at a dose of 2 to 2.5 mg.

Vasopressin

Vasopressin is a nonadrenergic peripheral vasoconstrictor that also causes coronary and renal vasoconstriction.[273] Three RCTs and a meta-analysis of the trials[274–277] demonstrated no difference in outcomes (ROSC, survival to discharge, or neurologic outcome) with vasopressin (40 units IV) versus epinephrine (1 mg) as a first-line vasopressor in cardiac arrest. Two RCTs[278,279] demonstrated no difference in outcomes (ROSC, survival to discharge, or neurologic) when comparing epinephrine in combination with vasopressin versus epinephrine alone in cardiac arrest. One RCT found that repeated doses of vasopressin during cardiac arrest did not improve survival rates compared with repeated doses of epinephrine.[280]

Because the effects of vasopressin have not been shown to differ from those of epinephrine in cardiac arrest, 1 dose of vasopressin 40 units IV/IO may replace either the first or second dose of epinephrine in the treatment of cardiac arrest (Class IIb, LOE A).

Other Vasopressors

There are no alternative vasopressors (norepinephrine, phenylephrine) with proven survival benefit compared with epinephrine.[268,281,282]

Antiarrhythmics

There is no evidence that any antiarrhythmic drug given routinely during human cardiac arrest increases survival to hospital discharge. Amiodarone, however, has been shown to increase short-term survival to hospital admission when compared with placebo or lidocaine.

Amiodarone

IV amiodarone affects sodium, potassium, and calcium channels and has α- and β-adrenergic blocking properties. It can be considered for treatment of VF or pulseless VT unresponsive to shock delivery, CPR, and a vasopressor. In blinded randomized controlled clinical trials in adults with refractory VF/pulseless VT in the out-of-hospital setting,[134,136] paramedic administration of amiodarone (300 mg[134] or 5 mg/kg[136]) improved hospital admission rates when compared with administration of placebo[134] or 1.5 mg/kg of lidocaine.[136] Additional studies[283–287] documented consistent improvement in termination of arrhythmias when amiodarone was given to humans or animals with VF or hemodynamically unstable VT. A higher incidence of bradycardia and hypotension was reported for amiodarone in one out-of-hospital study.[134] A canine study[288] noted that administration of a vasoconstrictor before amiodarone prevented hypotension. The adverse hemodynamic effects of the IV formulation of amiodarone are attributed to vasoactive solvents (polysorbate 80 and benzyl alcohol). When administered in the absence of these solvents, an analysis of the combined data of 4 prospective clinical trials of patients with VT (some hemodynamically unstable) showed that amiodarone produced no more hypotension than lidocaine.[286] A formulation of IV amiodarone without these vasoactive solvents was approved for use in the United States.

Amiodarone may be considered for VF or pulseless VT unresponsive to CPR, defibrillation, and a vasopressor therapy (Class IIb, LOE B). An initial dose of 300 mg IV/IO can be followed by 1 dose of 150 mg IV/IO. Although anecdotally administered IO without known adverse effects, there is limited experience with amiodarone given by this route.

Lidocaine

A retrospective review[289] demonstrated an association between improved hospital admission rates and use of lidocaine (compared with standard treatment) in patients with out-of-hospital VF cardiac arrest. But there is inadequate evidence to recommend the use of lidocaine in patients who have refractory VT/VF, defined as VT/VF not terminated by defibrillation or that continues to recur after defibrillation during out-of-hospital cardiac arrest or in-hospital cardiac arrest.

Lidocaine is an alternative antiarrhythmic of long-standing and widespread familiarity with fewer immediate side effects than may be encountered with other antiarrhythmics. Lidocaine, however, has no proven short- or long-term efficacy in cardiac arrest. Lidocaine may be considered if amiodarone is not available (Class IIb, LOE B). The initial dose is 1 to 1.5 mg/kg IV. If VF/pulseless VT persists, additional doses of 0.5 to 0.75 mg/kg IV push may be administered at 5- to 10-minute intervals to a maximum dose of 3 mg/kg.

Magnesium Sulfate

Two observational studies[290,291] showed that IV magnesium sulfate can facilitate termination of torsades de pointes (irregular/polymorphic VT associated with prolonged QT interval). Magnesium sulfate is not likely to be effective in terminating irregular/polymorphic VT in patients with a normal QT interval.[291]

A number of doses of magnesium sulfate have been used clinically, and an optimal dosing regimen has not been established. When VF/pulseless VT cardiac arrest is associated with torsades de pointes, providers may administer an IV/IO bolus of magnesium sulfate at a dose of 1 to 2 g diluted in 10 mL D_5W (Class IIb, LOE C). See Part 8.3: "Management of Symptomatic Bradycardia and Tachycardia" for additional information about management of torsades de pointes not associated with cardiac arrest.

Three RCTs[292–294] did not identify a significant benefit from use of magnesium compared with placebo among patients with VF arrest in the prehospital, intensive care unit, and emergency department setting, respectively. Thus, routine administration of magnesium sulfate in cardiac arrest is not recommended (Class III, LOE A) unless torsades de pointes is present.

Interventions Not Recommended for Routine Use During Cardiac Arrest

Atropine

Atropine sulfate reverses cholinergic-mediated decreases in heart rate and atrioventricular nodal conduction. No prospective controlled clinical trials have examined the use of atropine in asystole or bradycardic PEA cardiac arrest. Lower-level clinical studies provide conflicting evidence of the benefit of routine use of atropine in cardiac arrest.[34,295–304] There is no evidence that atropine has detrimental effects during bradycardic or asystolic cardiac arrest. Available evidence suggests that routine use of atropine during PEA or asystole is unlikely to have a therapeutic benefit (Class IIb, LOE B). For this reason atropine has been removed from the cardiac arrest algorithm.

Sodium Bicarbonate

Tissue acidosis and resulting acidemia during cardiac arrest and resuscitation are dynamic processes resulting from no blood flow during arrest and low blood flow during CPR. These processes are affected by the duration of cardiac arrest, level of blood flow, and arterial oxygen content during CPR. Restoration of oxygen content with appropriate ventilation with oxygen, support of some tissue perfusion and some cardiac output with high-quality chest compressions, then rapid ROSC are the mainstays of restoring acid-base balance during cardiac arrest.

Two studies demonstrated[305,306] increased ROSC, hospital admission, and survival to hospital discharge associated with use of bicarbonate. However, the majority of studies showed no benefit[307–309] or found a relationship with poor outcome.[304,310–312]

There are few data to support therapy with buffers during cardiac arrest. There is no evidence that bicarbonate improves the likelihood of defibrillation or survival rates in animals with VF cardiac arrest. A wide variety of adverse effects have been linked to administration of bicarbonate during cardiac arrest. Bicarbonate may compromise CPP by reducing systemic vascular resistance.[313] It can create extracellular alkalosis that will shift the oxyhemoglobin saturation curve and inhibit oxygen release. It can produce hypernatremia and therefore hyperosmolarity. It produces excess CO_2, which freely diffuses into myocardial and cerebral cells and may paradoxically contribute to intracellular acidosis.[314] It can exacerbate central venous acidosis and may inactivate simultaneously administered catecholamines.

In some special resuscitation situations, such as preexisting metabolic acidosis, hyperkalemia, or tricyclic antidepressant overdose, bicarbonate can be beneficial (see Part 12: "Cardiac Arrest in Special Situations"). However, routine use of sodium bicarbonate is not recommended for patients in cardiac arrest (Class III, LOE B). When bicarbonate is used for special situations, an initial dose of 1 mEq/kg is typical. Whenever possible, bicarbonate therapy should be guided by the bicarbonate concentration or calculated base deficit obtained from blood gas analysis or laboratory measurement. To minimize the risk of iatrogenically induced alkalosis, providers should not attempt complete correction of the calculated base deficit. Other non–CO_2-generating buffers such as carbicarb, THAM, or tribonate have shown potential for minimizing some adverse effects of sodium bicarbonate, including CO_2 generation, hyperosmolarity, hypernatremia, hypoglycemia, intracellular acidosis, myocardial acidosis, and "overshoot" alkalosis.[315–317] But clinical experience is greatly limited and outcome studies are lacking.

Calcium

Studies of calcium during cardiac arrest have found variable results on ROSC, and no trial has found a beneficial effect on survival either in or out of hospital.[301,304,318–323] Routine administration of calcium for treatment of in-hospital and out-of-hospital cardiac arrest is not recommended (Class III, LOE B).

Fibrinolysis

Fibrinolytic therapy was proposed for use during cardiac arrest to treat both coronary thrombosis (acute coronary syndrome) with presumably complete occlusion of a proximal coronary artery and major life-threatening pulmonary embolism. Ongoing CPR is not an absolute contraindication to fibrinolysis. Initial studies were promising[324–330] and suggested benefit from fibrinolytic therapy in the treatment of victims of cardiopulmonary arrest unresponsive to standard therapy. But 2 large clinical trials[188,331] failed to show any improvement in outcome with fibrinolytic therapy during CPR. One of these showed an increased risk of intracranial bleeding associated with the routine use of fibrinolytics during cardiac arrest.[188]

Fibrinolytic therapy should not be routinely used in cardiac arrest (Class III, LOE B). When pulmonary embolism is presumed or known to be the cause of cardiac arrest, empirical fibrinolytic therapy can be considered (Class IIa, LOE B; see Part 12).

IV Fluids

No published human study directly compares the outcome of routine IV fluid administration to no fluid administration during CPR. Most human and animal studies of fluid infusion during CPR did not have a control group,[332–343] and 2 animal studies showed that normothermic fluid infusion during CPR caused a decrease in CPP.[344–346] In addition to normothermic fluid, hypertonic and chilled fluids have been studied in animal and small human studies without a survival benefit.[332,334,336–338,341–343] If cardiac arrest is associated with extreme volume losses, hypovolemic arrest should be suspected. These patients present with signs of circulatory shock advancing to PEA. In these settings intravascular volume should be promptly restored.

Pacing

Electric pacing is generally not effective in cardiac arrest, and no studies have observed a survival benefit from pacing in cardiac arrest.[347–350] Existing evidence suggests that pacing by transcutaneous, transvenous, or transmyocardial means in cardiac arrest does not improve the likelihood of ROSC or survival outcome regardless of the timing of pacing administration (early or delayed in established asystole), location of arrest (in-hospital or out-of-hospital), or primary cardiac rhythm (asystole, PEA) targeted for treatment. Electric pacing is not recommended for routine use in cardiac arrest (Class III, LOE B).

Precordial Thump

The potential utility of precordial thump in cardiac arrest has not been well studied. When hemodynamically unstable ventricular tachyarrhythmias were induced during electrophysiological testing, initial administration of a precordial thump appeared to be safe but rarely effective in terminating ventricular arrhythmias.[351] In a prospective observational study of patients with out-of-hospital cardiac arrest, precordial thump was associated with ROSC when administered promptly to patients with responder-witnessed asystolic arrest. When administered for VF/VT or PEA arrest it was ineffective but resulted in no apparent harm.[352] In 3 case series[353–355] VF or pulseless VT was converted to a perfusing rhythm by a precordial thump. Conversely, other case series documented deterioration in cardiac rhythm, such as rate acceleration of VT, conversion of VT to VF, or development of complete AV block or asystole following the thump.[354,356–361]

The precordial thump may be considered for termination of witnessed monitored unstable ventricular tachyarrhythmias when a defibrillator is not immediately ready for use (Class IIb, LOE B), but should not delay CPR and shock delivery. There is insufficient evidence to recommend for or against the use of the precordial thump for witnessed onset of asystole, and there is insufficient evidence to recommend percussion pacing during typical attempted resuscitation from cardiac arrest.

Summary

Intervention to prevent cardiac arrest in critically ill patients is ideal. When cardiac arrest occurs, high-quality CPR is fundamental to the success of any subsequent ACLS intervention. During resuscitation healthcare providers must perform chest compressions of adequate rate and depth, allow complete recoil of the chest after each compression, minimize interruptions in chest compressions, and avoid excessive ventilation, especially with an advanced airway. Quality of CPR should be continuously monitored. Physiologic monitoring may prove useful to optimize resuscitative efforts. For patients in VF/pulseless VT, shocks should be delivered promptly with minimal interruptions in chest compressions. The increased rates of ROSC associated with ACLS drug therapy have yet to be translated into long-term survival benefits. However, improved quality of CPR, advances in post–cardiac arrest care, and improved overall implementation through comprehensive systems of care may provide a pathway to optimize the outcomes of cardiac arrest patients treated with ACLS interventions.

Part 8.3: Management of Symptomatic Bradycardia and Tachycardia

Overview

This section highlights recommendations for management of patients with acute symptomatic arrhythmias. Electrocardiographic (ECG) and rhythm information should be interpreted within the context of total patient assessment. Errors in diagnosis and treatment are likely to occur if advanced cardiovascular life support (ACLS) providers base treatment decisions solely on rhythm interpretation and neglect clinical evaluation. Providers must evaluate the patient's symptoms and clinical signs, including ventilation, oxygenation, heart rate, blood pressure, level of consciousness, and signs of inadequate organ perfusion.

Unstable and *symptomatic* are terms typically used to describe the condition of patients with arrhythmias. Generally, *unstable* refers to a condition in which vital organ function is acutely impaired or cardiac arrest is ongoing or imminent. When an arrhythmia causes a patient to be unstable, immediate intervention is indicated. *Symptomatic* implies that an arrhythmia is causing symptoms, such as palpitations, lightheadedness, or dyspnea, but the patient is stable and not in imminent danger. In such cases more time is available to decide on the most appropriate intervention. In both unstable and symptomatic cases the provider must make an assessment as to whether it is the arrhythmia that is causing the patient to be unstable or symptomatic. For example, a patient in septic shock with sinus tachycardia of 140 beats per minute is unstable; however, the arrhythmia is a physiologic compensation rather than the cause of instability. Therefore, electric cardioversion will not improve this patient's condition. Additionally, if a patient with respiratory failure and severe hypoxemia becomes hypotensive and develops a bradycardia, the bradycardia is not the primary cause of instability. Treating the bradycardia without treating the hypoxemia is unlikely to improve the patient's condition. It is critically important to determine the cause of the patient's instability in order to properly direct treatment. In general, sinus tachycardia is a response to other factors and, thus, it rarely (if ever) is the cause of instability in and of itself.

The *2010 AHA Guidlines for CPR and ECC* emphasize the importance of clinical evaluation and highlight principles of therapy with algorithms that have been refined and streamlined since publication of the *2005 AHA Guidelines for CPR and ECC*.[362] The key principles of arrhythmia recognition and management in adults are as follows:

If bradycardia produces signs and symptoms of instability (eg, acutely altered mental status, ischemic chest discomfort, acute heart failure, hypotension, or other signs of shock that persist despite adequate airway and breathing), the initial treatment is atropine (Class IIa, LOE B). If bradycardia is unresponsive to atropine, intravenous (IV) infusion of β-adrenergic agonists with rate-accelerating effects (dopamine, epinephrine) or transcutaneous pacing (TCP) can be effective (Class IIa, LOE B) while the patient is prepared for emergent transvenous temporary pacing if required.

If the tachycardic patient is unstable with severe signs and symptoms related to a suspected arrhythmia (eg, acute altered mental status, ischemic chest discomfort, acute heart failure, hypotension, or other signs of shock), immediate cardioversion should be performed (with prior sedation in the conscious patient) (Class I, LOE B). In select cases of regular narrow-complex tachycardia with unstable signs or symptoms, a trial of adenosine before cardioversion is reasonable to consider (Class IIb, LOE C).

If the patient with tachycardia is stable, determine if the patient has a narrow-complex or wide-complex tachycardia, whether the rhythm is regular or irregular, and for wide complexes whether the QRS morphology is monomorphic or polymorphic. Therapy is then tailored accordingly (Table 2).

Know when to call for expert consultation regarding complicated rhythm interpretation, drugs, or management decisions.

A comprehensive presentation of the evaluation and management of bradyarrhythmias and tachyarrhythmias is beyond the scope of the *2010 AHA Guidelines for CPR and ECC*. The following selected rhythm scenarios are meant to aid with the management of periarrest rhythm disorders. If cardiac arrest develops at any time, see the ACLS Cardiac Arrest Algorithms in Part 8.2: "Management of Cardiac Arrest."

Table 2. IV Drugs Used for Tachycardia

Drug	Characteristics	Indication(s)	Dosing	Side Effects	Precautions or Special Considerations
Intravenous Drugs Used to Treat Supraventricular Tachyarrhythmias					
Adenosine	Endogenous purine nucleoside; briefly depresses sinus node rate and AV node conduction; vasodilator	• Stable, narrow-complex regular tachycardias • Unstable narrow-complex regular tachycardias while preparations are made for electrical cardioversion • Stable, regular, monomorphic, wide complex tachycardia as a therapeutic and diagnostic maneuver	6 mg IV as a rapid IV push followed by a 20 mL saline flush; repeat if required as 12 mg IV push	Hypotension, bronchospasm, chest discomfort	Contraindicated in patients with asthma; may precipitate atrial fibrillation, which may be very rapid in patients with WPW; thus a defibrillator should be readily available; reduce dose in post–cardiac transplant patients, those taking dipyridamole or carbamazepine and when administered via a central vein
Diltiazem, Verapamil	Non-dihydropyridine calcium channel blockers; slow AV node conduction and increase AV node refractoriness; vasodilators, negative inotropes	• Stable, narrow-complex tachycardias if rhythm remains uncontrolled or unconverted by adenosine or vagal maneuvers or if SVT is recurrent • Control ventricular rate in patients with atrial fibrillation or atrial flutter	Diltiazem: Initial dose 15 to 20 mg (0.25 mg/kg) IV over 2 minutes; additional 20 to 25 mg (0.35 mg/kg) IV in 15 minutes if needed; 5 to 15 mg/h IV maintenance infusion (titrated to AF heart rate if given for rate control) Verapamil: Initial dose 2.5 to 5 mg IV given over 2 minutes; may repeat as 5 to 10 mg every 15 to 30 minutes to total dose of 20 to 30 mg	Hypotension, bradycardia, precipitation of heart failure	Should only be given to patients with narrow-complex tachycardias (regular or irregular). Avoid in patients with heart failure and pre-excited AF or flutter or rhythms consistent with VT
Atenolol, Esmolol, Metoprolol, Propranolol	β-Blockers; reduce effects of circulating catecholamines; reduce heart rate, AV node conduction and blood pressure; negative inotropes	• Stable, narrow-complex tachycardias if rhythm remains uncontrolled or unconverted by adenosine or vagal maneuvers or if SVT is recurrent • Control ventricular rate in patients with atrial fibrillation or atrial flutter • Certain forms of polymorphic VT (associated with acute ischemia, familial LQTS, catecholaminergic)	Atenolol (β1 specific blocker) 5 mg IV over 5 minutes; repeat 5 mg in 10 minutes if arrhythmia persists or recurs Esmolol (β1 specific blocker with 2- to 9-minute half-life) IV loading dose 500 mcg/kg (0.5 mg/kg) over 1 minute, followed by an infusion of 50 mcg/kg per minute (0.05 mg/kg per minute); if response is inadequate, infuse second loading bolus of 0.5 mg/kg over 1 minute and increase maintenance infusion to 100 mcg/kg (0.1 mg/kg) per minute; increment; increase in this manner if required to maximum infusion rate of 300 mcg/kg [0.3 mg/kg] per minute Metoprolol (β1 specific blocker) 5 mg over 1 to 2 minutes repeated as required every 5 minutes to maximum dose of 15 mg Propranolol (nonselective β-blocker) 0.5 to 1 mg over 1 minute, repeated up to a total dose of 0.1 mg/kg if required	Hypotension, bradycardia, precipitation of heart failure	Avoid in patients with asthma, obstructive airway disease, decompensated heart failure and pre-excited artrial fibrillation or flutter
Procainamide	Sodium and potassium channel blocker	• Pre-excited atrial fibrillation	20 to 50 mg/min until arrhythmia suppressed, hypotension ensues, or QRS prolonged by 50%, or total cumulative dose of 17 mg/kg; or 100 mg every 5 minutes until arrhythmia is controlled or other conditions described above are met	Bradycardia, hypotension, torsades de pointes	Avoid in patients with QT prolongation and CHF

(*Continued*)

Table 2. Continued

Drug	Characteristics	Indication(s)	Dosing	Side Effects	Precautions or Special Considerations
Amiodarone	Multichannel blocker (sodium, potassium, calcium channel, and noncompetitive α/β-blocker)	• Stable irregular narrow complex tachycardia (atrial fibrillation) • Stable regular narrow-complex tachycardia • To control rapid ventricular rate due to accessory pathway conduction in pre-excited atrial arrhythmias	150 mg given over 10 minutes and repeated if necessary, followed by a 1 mg/min infusion for 6 hours, followed by 0.5 mg/min. Total dose over 24 hours should not exceed 2.2 g.	Bradycardia, hypotension, phlebitis	
Digoxin	Cardiac glycoside with positive inotropic effects; slows AV node conduction by enhancing parasympathetic tone; slow onset of action	• Stable, narrow-complex regular tachycardias if rhythm remains uncontrolled or unconverted by adenosine or vagal maneuvers or if SVT is recurrent • Control ventricular rate in patients with atrial fibrillation or atrial flutter	8 to 12 mcg/kg total loading dose, half of which is administered initially over 5 minutes, and remaining portion as 25% fractions at 4- to 8- hour intervals	Bradycardia	Slow onset of action and relative low potency renders it less useful for treatment of acute arrhythmias
Intravenous Drugs Used to Treat Ventricular Tachyarrhythmias					
Procainamide	Sodium and potassium channel blocker	• Hemodynamically stable monomorphic VT	20 to 50 mg/min until arrhythmia suppressed, hypotension ensues, or QRS prolonged by 50%, or total cumulative dose of 17 mg/kg; or 100 mg every 5 minutes until arrhythmia is controlled or other conditions described above are met	Bradycardia, hypotension, torsades de pointes	Avoid in patients with QT prolongation and CHF
Amiodarone	Multichannel blocker (sodium, potassium, calcium channel, α- and noncompetitive β-blocker)	• Hemodynamically stable monomorphic VT • Polymorphic VT with normal QT interval	150 mg given over 10 minutes and repeated if necessary, followed by a 1 mg/min infusion for 6 hours, followed by 0.5 mg/min. Total dose over 24 hours should not exceed 2.2 g.	Bradycardia, hypotension, phlebitis	
Sotalol	Potassium channel blocker and nonselective β-blocker	• Hemodynamically stable monomorphic VT	In clinical studies 1.5 mg/kg infused over 5 minutes; however, US package labeling recommends any dose of the drug should be infused slowly over a period of 5 hours	Bradycardia, hypotension, torsades de pointes	Avoid in patients with QT prolongation and CHF
Lidocaine	Relatively weak sodium channel blocker	• Hemodynamically stable monomorphic VT	Initial dose range from 1 to 1.5 mg/kg IV; repeated if required at 0.5 to 0.75 mg/kg IV every 5 to 10 minutes up to maximum cumulative dose of 3 mg/kg; 1 to 4 mg/min (30 to 50 mcg/kg per minute) maintenance infusion	Slurred speech, altered consciousness, seizures, bradycardia	
Magnesium	Cofactor in variety of cell processes including control of sodium and potassium transport	• Polymorphic VT associated with QT prolongation (torsades de pointes)	1 to 2 g IV over 15 minutes	Hypotension, CNS toxicity, respiratory depression	Follow magnesium levels if frequent or prolonged dosing required, particularly in patients with impaired renal function

Bradycardia

This section summarizes the management of bradyarrhythmias. Following the overview of bradyarrhythmias and summary of the initial evaluation and treatment of bradycardia, drugs used in the treatment of bradycardia are presented. See the Bradycardia Algorithm, Figure 3. Box numbers in the text refer to the numbered boxes in the algorithm.

Evaluation

Bradycardia is defined as a heart rate of <60 beats per minute. However, when bradycardia is the cause of symptoms, the rate is generally <50 beats per minute, which is the working definition of bradycardia used here (Figure 3, **Box 1**). A slow heart rate may be physiologically normal for some patients, whereas a heart rate of >50 beats per minute may be inadequate for others. The Bradycardia Algorithm focuses on management of clinically significant bradycardia (ie, bradycardia that is inappropriate for the clinical condition).

Because hypoxemia is a common cause of bradycardia, initial evaluation of any patient with bradycardia should focus on signs of increased work of breathing (tachypnea, intercostal retractions, suprasternal retractions, paradoxical abdominal breathing) and oxyhemoglobin saturation as determined by pulse oximetry (Figure 3, **Box 2**). If oxygenation is

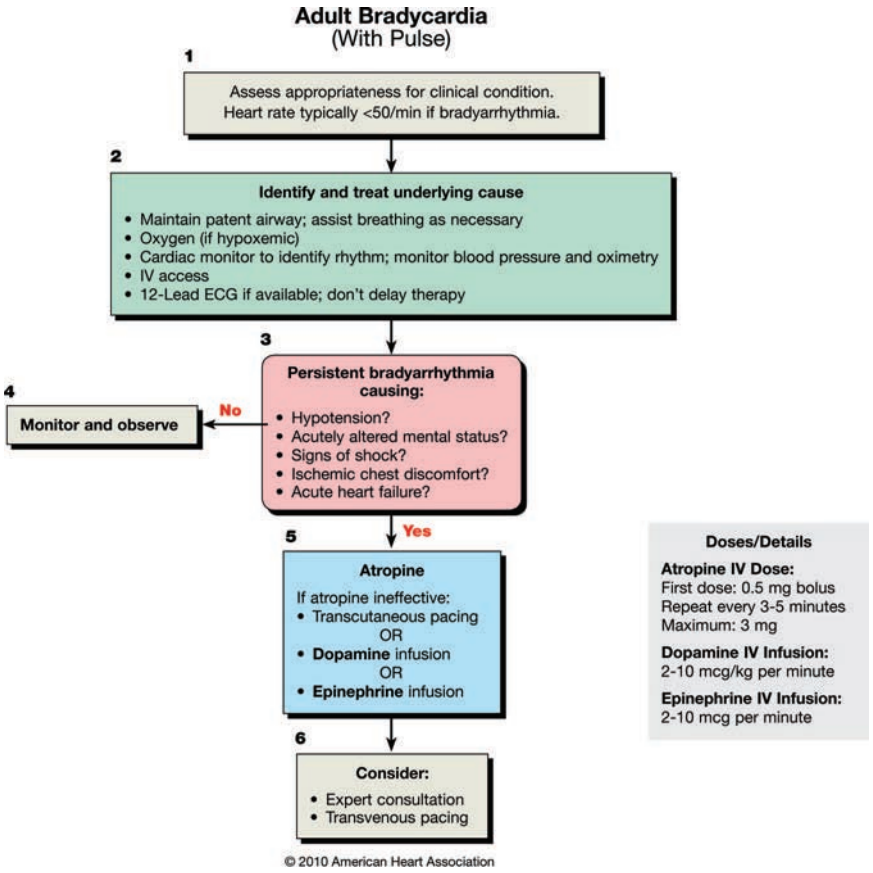


Figure 3. Bradycardia Algorithm.

inadequate or the patient shows signs of increased work of breathing, provide supplementary oxygen. Attach a monitor to the patient, evaluate blood pressure, and establish IV access. If possible, obtain a 12-lead ECG to better define the rhythm. While initiating treatment, evaluate the patient's clinical status and identify potentially reversible causes.

The provider must identify signs and symptoms of poor perfusion and determine if those signs are likely to be caused by the bradycardia (Figure 3, **Box 3**). If the signs and symptoms are not due to bradycardia, the provider should reassess the underlying cause of the patient's symptoms. Remember that signs and symptoms of bradycardia may be mild; asymptomatic or minimally symptomatic patients do not necessarily require treatment (Figure 3, **Box 4**) unless there is suspicion that the rhythm is likely to progress to symptoms or become life-threatening (eg, Mobitz type II second-degree AV block in the setting of acute myocardial infarction [AMI]). If the bradycardia is suspected to be the cause of acute altered mental status, ischemic chest discomfort, acute heart failure, hypotension, or other signs of shock, the patient should receive immediate treatment.

Atrioventricular (AV) blocks are classified as first-, second-, and third-degree. Blocks may be caused by medications or electrolyte disturbances, as well as structural problems resulting from AMI or other myocardial diseases. A first-degree AV block is defined by a prolonged PR interval (>0.20 second) and is generally benign. Second-degree AV block is divided into Mobitz types I and II. In Mobitz type I block, the block is at the AV node; the block is often transient and asymptomatic. In Mobitz type II block, the block is usually below the AV node within the His-Purkinje system; this block is often symptomatic, with the potential to progress to complete (third-degree) AV block. Third-degree AV block may occur at the AV node, bundle of His, or bundle branches. When third-degree AV block is present, no impulses pass between the atria and ventricles. Third-degree AV block can be permanent or transient, depending on the underlying cause.

Therapy (Figure 3, Box 5)

Atropine

Atropine remains the first-line drug for acute symptomatic bradycardia (Class IIa, LOE B). Clinical trials in adults[363–367] showed that IV atropine improved heart rate, symptoms, and signs associated with bradycardia. Atropine sulfate reverses cholinergic-mediated decreases in heart rate and should be considered a temporizing measure while awaiting a transcutaneous or transvenous pacemaker for patients with symptomatic sinus bradycardia, conduction block at the level of the AV node, or sinus arrest.[367]

The recommended atropine dose for bradycardia is 0.5 mg IV every 3 to 5 minutes to a maximum total dose of 3 mg. Doses of atropine sulfate of <0.5 mg may paradoxically result in further slowing of the heart rate.[368] Atropine administration should not delay implementation of external pacing for patients with poor perfusion.

Use atropine cautiously in the presence of acute coronary ischemia or MI; increased heart rate may worsen ischemia or increase infarction size. Atropine will likely be ineffective in

patients who have undergone cardiac transplantation because the transplanted heart lacks vagal innervation. One small uncontrolled study documented paradoxical slowing of the heart rate and high-degree AV block when atropine was administered to patients after cardiac transplantation.[369]

Avoid relying on atropine in type II second-degree or third-degree AV block or in patients with third-degree AV block with a new wide-QRS complex where the location of block is likely to be in non-nodal tissue (such as in the bundle of His or more distal conduction system). These bradyarrhythmias are not likely to be responsive to reversal of cholinergic effects by atropine and are preferably treated with TCP or β-adrenergic support as temporizing measures while the patient is prepared for transvenous pacing (Figure 3, **Box 6**).

Pacing

TCP may be useful for the treatment of symptomatic bradycardias. There are limited studies comparing TCP with drug therapy for the treatment of symptomatic bradycardia. A randomized controlled trial in which atropine and glycopyrrolate were compared with TCP showed few differences in outcome and survival, although the TCP group obtained a more consistent heart rate.[363] In a study evaluating the feasibility of treatment with dopamine as compared with TCP, no differences were observed between treatment groups in survival to hospital discharge.[370] TCP is, at best, a temporizing measure. TCP is painful in conscious patients, and, whether effective or not (achieving inconsistent capture), the patient should be prepared for transvenous pacing and expert consultation should be obtained. It is reasonable for healthcare providers to initiate TCP in unstable patients who do not respond to atropine (Class IIa, LOE B). Immediate pacing might be considered in unstable patients with high-degree AV block when IV access is not available (Class IIb, LOE C). If the patient does not respond to drugs or TCP, transvenous pacing is probably indicated (Class IIa, LOE C) (Figure 3, **Box 6**).

Alternative Drugs to Consider

Although not first-line agents for treatment of symptomatic bradycardia, dopamine, epinephrine, and isoproterenol are alternatives when a bradyarrhythmia is unresponsive to or inappropriate for treatment with atropine, or as a temporizing measure while awaiting the availability of a pacemaker. Alternative drugs may also be appropriate in special circumstances such as the overdose of a β-blocker or calcium channel blocker.

Dopamine. Dopamine hydrochloride is a catecholamine with both α- and β-adrenergic actions. It can be titrated to more selectively target heart rate or vasoconstriction. At lower doses dopamine has a more selective effect on inotropy and heart rate; at higher doses (>10 mcg/kg per minute), it also has vasoconstrictive effects. Dopamine infusion may be used for patients with symptomatic bradycardia, particularly if associated with hypotension, in whom atropine may be inappropriate or after atropine fails (Class IIb, LOE B). Begin dopamine infusion at 2 to 10 mcg/kg per minute and titrate to patient response.[370] Use of vasoconstrictors requires that the recipient be assessed for adequate intravascular volume and volume status supported as needed.

Epinephrine. Epinephrine is a catecholamine with α- and β-adrenergic actions. Epinephrine infusion may be used for patients with symptomatic bradycardia, particularly if associated with hypotension, for whom atropine may be inappropriate or after atropine fails (Class IIb, LOE B). Begin the infusion at 2 to 10 mcg/min and titrate to patient response. Use of vasoconstrictors requires that the recipient be assessed for adequate intravascular volume and volume status supported as needed.

Isoproterenol. Isoproterenol is a β-adrenergic agent with β-1 and β-2 effects, resulting in an increase in heart rate and vasodilation. The recommended adult dose is 2 to 10 mcg/min by IV infusion, titrated according to heart rate and rhythm response.

Tachycardia

This section summarizes the management of a wide variety of tachyarrhythmias. Following the overview of tachyarrhythmias and summary of the initial evaluation and treatment of tachycardia, common antiarrhythmic drugs used in the treatment of tachycardia are presented. See the Tachycardia Algorithm, Figure 4. Box numbers in the text refer to the numbered boxes in the algorithm.

Classification of Tachyarrhythmias

Tachycardias can be classified in several ways, based on the appearance of the QRS complex, heart rate, and regularity. ACLS professionals should be able to recognize and differentiate between sinus tachycardia, narrow-complex supraventricular tachycardia (SVT), and wide-complex tachycardia. Because ACLS providers may be unable to distinguish between supraventricular and ventricular rhythms, they should be aware that most wide-complex (broad-complex) tachycardias are *ventricular* in origin.

- Narrow–QRS-complex (SVT) tachycardias (QRS <0.12 second), in order of frequency
  - Sinus tachycardia
  - Atrial fibrillation
  - Atrial flutter
  - AV nodal reentry
  - Accessory pathway–mediated tachycardia
  - Atrial tachycardia (including automatic and reentry forms)
  - Multifocal atrial tachycardia (MAT)
  - Junctional tachycardia (rare in adults)
- Wide–QRS-complex tachycardias (QRS ≥0.12 second)
  - Ventricular tachycardia (VT) and ventricular fibrillation (VF)
  - SVT with aberrancy
  - Pre-excited tachycardias (Wolff-Parkinson-White [WPW] syndrome)
  - Ventricular paced rhythms

Irregular narrow-complex tachycardias are likely atrial fibrillation or MAT; occasionally atrial flutter is irregular. The management of atrial fibrillation and flutter is discussed in the section "Irregular Tachycardias" below.

Initial Evaluation and Treatment of Tachyarrhythmias

Tachycardia is defined as an arrhythmia with a rate of >100 beats per minute, although, as with defining bradycardia, the

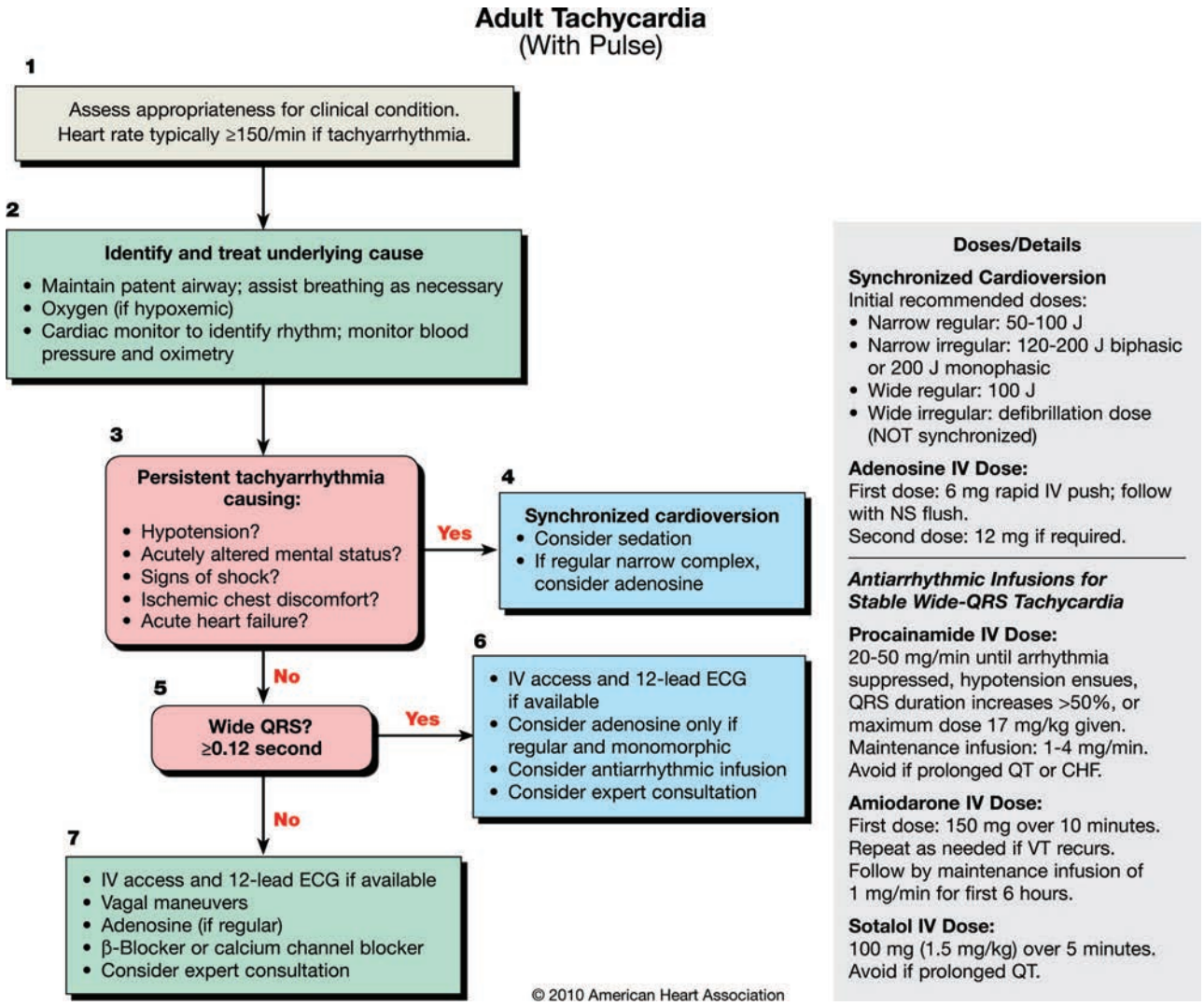


Figure 4. Tachycardia Algorithm.

rate of a tachycardia takes on clinical significance at its greater extremes and is more likely attributable to an arrhythmia rate of ≥150 beats per minute (Figure 4, **Box 1**). A rapid heart rate is an appropriate response to a physiologic stress (eg, fever, dehydration) or other underlying conditions. When encountering patients with tachycardia, efforts should be made to determine whether the tachycardia is the primary cause of the presenting symptoms or secondary to an underlying condition that is causing both the presenting symptoms and the faster heart rate. Many experts suggest that when a heart rate is <150 beats per minute, it is unlikely that symptoms of instability are caused primarily by the tachycardia unless there is impaired ventricular function.

The evaluation and management of tachyarrhythmias is depicted in the ACLS Tachycardia With Pulse Algorithm (Figure 4). Box numbers in the text refer to numbered boxes in this algorithm. If cardiac arrest develops at any time, see the ACLS Cardiac Arrest Algorithms in Part 8.2: "Management of Cardiac Arrest."

Because hypoxemia is a common cause of tachycardia, initial evaluation of any patient with tachycardia should focus on signs of increased work of breathing (tachypnea, intercostal retractions, suprasternal retractions, paradoxical abdominal breathing) and oxyhemoglobin saturation as determined by pulse oximetry (Figure 4, **Box 2**). If oxygenation is inadequate or the patient shows signs of increased work of breathing, provide supplementary oxygen. Attach a monitor to the patient, evaluate blood pressure, and establish IV access. If available, obtain a 12-lead ECG to better define the rhythm, but this should not delay immediate cardioversion if the patient is unstable. While initiating treatment, evaluate the patient's clinical status and identify potential reversible causes of the tachycardia.

If signs and symptoms persist despite provision of supplementary oxygen and support of airway and ventilation, the provider should assess the patient's degree of instability and determine if the instability is related to the tachycardia (Figure 4, **Box 3**). If the patient demonstrates rate-related cardiovascular compromise with signs and symptoms such as acute altered mental status, ischemic chest discomfort, acute heart failure, hypotension, or other signs of shock suspected to be due to a tachyarrhythmia, proceed to immediate syn-

chronized cardioversion (Figure 4, **Box 4**). However, with ventricular rates <150 beats per minute in the absence of ventricular dysfunction, it is more likely that the tachycardia is secondary to the underlying condition rather than the cause of the instability. If not hypotensive, the patient with a regular narrow-complex SVT (likely due to suspected reentry, paroxysmal supraventricular tachycardia, as described below) may be treated with adenosine while preparations are made for synchronized cardioversion (Class IIb, LOE C).

If the patient with tachycardia is stable (ie, no serious signs related to the tachycardia), the provider has time to obtain a 12-lead ECG, evaluate the rhythm, determine if the width of the QRS complex is ≥0.12 second (Figure 4, **Box 5**), and determine treatment options. Stable patients may await expert consultation because treatment has the potential for harm.

Cardioversion

If possible, establish IV access before cardioversion and administer sedation if the patient is conscious. Do not delay cardioversion if the patient is extremely unstable. For further information about defibrillation and cardioversion, see Part 6: "Electrical Therapies."

Synchronized Cardioversion and Unsynchronized Shocks (Figure 4, Box 4)

Synchronized cardioversion is shock delivery that is timed (synchronized) with the QRS complex. This synchronization avoids shock delivery during the relative refractory period of the cardiac cycle when a shock could produce VF.[371] If cardioversion is needed and it is impossible to synchronize a shock, use high-energy unsynchronized shocks (defibrillation doses).

Synchronized cardioversion is recommended to treat (1) unstable SVT, (2) unstable atrial fibrillation, (3) unstable atrial flutter, and (4) unstable monomorphic (regular) VT. Shock can terminate these tachyarrhythmias by interrupting the underlying reentrant pathway that is responsible for them.

Waveform and Energy

The recommended initial biphasic energy dose for cardioversion of atrial fibrillation is 120 to 200 J (Class IIa, LOE A).[372–376] If the initial shock fails, providers should increase the dose in a stepwise fashion.

Cardioversion of atrial flutter and other SVTs generally requires less energy; an initial energy of 50 J to 100 J is often sufficient.[376] If the initial 50-J shock fails, the provider should increase the dose in a stepwise fashion.[377] Cardioversion with monophasic waveforms should begin at 200 J and increase in stepwise fashion if not successful (Class IIa, LOE B).[372–374]

Monomorphic VT (regular form and rate) with a pulse responds well to monophasic or biphasic waveform cardioversion (synchronized) shocks at initial energies of 100 J. If there is no response to the first shock, it may be reasonable to increase the dose in a stepwise fashion. No studies were identified that addressed this issue. Thus, this recommendation represents expert opinion (Class IIb, LOE C).

Arrhythmias with a polymorphic QRS appearance (such as torsades de pointes) will usually not permit synchronization. Thus, if a patient has polymorphic VT, treat the rhythm as VF and deliver high-energy *unsynchronized* shocks (ie, defibrillation doses). If there is any doubt whether monomorphic or polymorphic VT is present in the *unstable* patient, do not delay shock delivery to perform detailed rhythm analysis: provide high-energy unsynchronized shocks (ie, defibrillation doses). Use the ACLS Cardiac Arrest Algorithm (see Part 8.2: "Management of Cardiac Arrest").

Regular Narrow-Complex Tachycardia

Sinus Tachycardia

Sinus tachycardia is common and usually results from a physiologic stimulus, such as fever, anemia, or hypotension/shock. Sinus tachycardia is defined as a heart rate >100 beats per minute. The upper rate of sinus tachycardia is age-related (calculated as approximately 220 beats per minute, minus the patient's age in years) and may be useful in judging whether an apparent sinus tachycardia falls within the expected range for a patient's age. If judged to be sinus tachycardia, no specific drug treatment is required. Instead, therapy is directed toward identification and treatment of the underlying cause. When cardiac function is poor, cardiac output can be dependent on a rapid heart rate. In such compensatory tachycardias, stroke volume is limited, so "normalizing" the heart rate can be detrimental.

Supraventricular Tachycardia (Reentry SVT)

Evaluation. Most SVTs are regular tachycardias that are caused by reentry, an abnormal rhythm circuit that allows a wave of depolarization to repeatedly travel in a circle in cardiac tissue. The rhythm is considered to be of supraventricular origin if the QRS complex is narrow (<120 milliseconds or <0.12 second) or if the QRS complex is wide (broad) and preexisting bundle branch block or rate-dependent aberrancy is *known* to be present. Reentry circuits resulting in SVT can occur in atrial myocardium (resulting in atrial fibrillation, atrial flutter, and some forms of atrial tachycardia). The reentry circuit may also reside in whole or in part in the AV node itself. This results in AV nodal reentry tachycardia (AVNRT) if both limbs of the reentry circuit involve AV nodal tissue. Alternatively, it may result in AV reentry tachycardia (AVRT) if one limb of the reentry circuit involves an accessory pathway and the other involves the AV node. The characteristic abrupt onset and termination of each of the latter groups of reentrant tachyarrhythmias (AVNRT and AVRT) led to the original name, paroxysmal supraventricular tachycardia (PSVT). This subgroup of reentry arrhythmias, due to either AVNRT or AVRT, is characterized by abrupt onset and termination and a regular rate that exceeds the typical upper limits of sinus tachycardia at rest (usually >150 beats per minute) and, in the case of an AVNRT, often presents without readily identifiable P waves on the ECG.

Distinguishing the forms of reentrant SVTs that are based in atrial myocardium (such as atrial fibrillation) versus those with a reentry circuit partly or wholly based in the AV node itself (PSVT) is important because each will respond differently to therapies aimed at impeding conduction through the AV node. The ventricular rate of reentry arrhythmias based in atrial myocardium will be slowed but not terminated by drugs that slow conduction through the AV node. Conversely, reentry arrhythmias for which at least one limb of the circuit resides in the AV node (PSVT attributable to AVNRT or AVRT) can be terminated by such drugs.

Yet another group of SVTs is referred to as automatic tachycardias. These arrhythmias are not due to a circulating circuit but to an excited automatic focus. Unlike the abrupt pattern of reentry, the characteristic onset and termination of these tachyarrhythmias are more gradual and analogous to how the sinus node behaves in gradually accelerating and slowing heart rate. These automatic arrhythmias include ectopic atrial tachycardia, MAT, and junctional tachycardia. These arrhythmias can be difficult to treat, are not responsive to cardioversion, and are usually controlled acutely with drugs that slow conduction through the AV node and thereby slow ventricular rate.

Therapy

Vagal Maneuvers. Vagal maneuvers and adenosine are the preferred initial therapeutic choices for the termination of stable PSVT (Figure 4, **Box 7**). Vagal maneuvers alone (Valsalva maneuver or carotid sinus massage) will terminate up to 25% of PSVTs.[378–380] For other SVTs, vagal maneuvers and adenosine may transiently slow the ventricular rate and potentially assist rhythm diagnosis but will not usually terminate such arrhythmias.

Adenosine. If PSVT does not respond to vagal maneuvers, give 6 mg of IV adenosine as a rapid IV push through a large (eg, antecubital) vein followed by a 20 mL saline flush (Class I, LOE B). If the rhythm does not convert within 1 to 2 minutes, give a 12 mg rapid IV push using the method above. Because of the possibility of initiating atrial fibrillation with rapid ventricular rates in a patient with WPW, a defibrillator should be available when adenosine is administered to any patient in whom WPW is a consideration. As with vagal maneuvers, the effect of adenosine on other SVTs (such as atrial fibrillation or flutter) is to transiently slow ventricular rate (which may be useful diagnostically) but not afford their termination or meaningful lasting rate control.

A number of studies[381–398] support the use of adenosine in the treatment of stable PSVT. Although 2 randomized clinical trials[383,386] documented a similar PSVT conversion rate between adenosine and calcium channel blockers, adenosine was more rapid and had fewer severe side effects than verapamil. Amiodarone as well as other antiarrhythmic agents can be useful in the termination of PSVT, but the onset of action of amiodarone is slower than that of adenosine,[399] and the potential proarrhythmic risks of these agents favor the use of safer treatment alternatives.

Adenosine is safe and effective in pregnancy.[400] However, adenosine does have several important drug interactions. Larger doses may be required for patients with a significant blood level of theophylline, caffeine, or theobromine. The initial dose should be reduced to 3 mg in patients taking dipyridamole or carbamazepine, those with transplanted hearts, or if given by central venous access. Side effects with adenosine are common but transient; flushing, dyspnea, and chest discomfort are the most frequently observed.[401] Adenosine should not be given to patients with asthma.

After conversion, monitor the patient for recurrence and treat any recurrence of PSVT with adenosine or a longer-acting AV nodal blocking agent (eg, diltiazem or β-blocker). If adenosine or vagal maneuvers disclose another form of SVT (such as atrial fibrillation or flutter), treatment with a longer-acting AV nodal blocking agent should be considered to afford more lasting control of ventricular rate.

Calcium Channel Blockers and β-Blockers. If adenosine or vagal maneuvers fail to convert PSVT (Figure 4, **Box 7**), PSVT recurs after such treatment, or these treatments disclose a different form of SVT (such as atrial fibrillation or flutter), it is reasonable to use longer-acting AV nodal blocking agents, such as the nondihydropyridine calcium channel blockers (verapamil and diltiazem) (Class IIa, LOE B) or β-blockers (Class IIa, LOE C). These drugs act primarily on nodal tissue either to terminate the reentry PSVTs that depend on conduction through the AV node or to slow the ventricular response to other SVTs by blocking conduction through the AV node. The alternate mechanism of action and longer duration of these drugs may result in more sustained termination of PSVT or afford more sustained rate control of atrial arrhythmias (such as atrial fibrillation or flutter). A number of studies have established the effectiveness of verapamil[381,383,384,386,394, 398,402–405] and diltiazem[402,406,407] in converting PSVT to normal sinus rhythm.

For verapamil, give a 2.5 mg to 5 mg IV bolus over 2 minutes (over 3 minutes in older patients). If there is no therapeutic response and no drug-induced adverse event, repeated doses of 5 mg to 10 mg may be administered every 15 to 30 minutes to a total dose of 20 mg. An alternative dosing regimen is to give a 5 mg bolus every 15 minutes to a total dose of 30 mg. Verapamil should be given *only* to patients with narrow-complex reentry SVT or arrhythmias known with certainty to be of supraventricular origin. Verapamil should not be given to patients with wide-complex tachycardias. It should not be given to patients with impaired ventricular function or heart failure.

For diltiazem, give a dose of 15 mg to 20 mg (0.25 mg/kg) IV over 2 minutes; if needed, in 15 minutes give an additional IV dose of 20 mg to 25 mg (0.35 mg/kg). The maintenance infusion dose is 5 mg/hour to 15 mg/hour, titrated to heart rate.

A wide variety of IV β-blockers are available for treatment of supraventricular tachyarrhythmias. These include metoprolol, atenolol, propranolol, esmolol, and labetolol (the latter more commonly used for acute management of hypertension than for arrhythmias). In principle these agents exert their effect by antagonizing sympathetic tone in nodal tissue, resulting in slowing of conduction. Like calcium channel blockers, they also have negative inotropic effects and further reduce cardiac output in patients with heart failure. More detailed information is provided below. Side effects of β-blockers can include bradycardias, AV conduction delays, and hypotension. β-blockers should be used with caution in patients with obstructive pulmonary disease or congestive heart failure.

Caution is advised when encountering pre-excited atrial fibrillation or flutter that conducts to the ventricles via both the AV node and an accessory pathway. Treatment with an AV nodal blocking agent (including adenosine, calcium blockers, β-blockers, or digoxin) is unlikely to slow the ventricular rate and in some instances may accelerate the ventricular response. Therefore, AV nodal blocking drugs should not be used for pre-excited atrial fibrillation or flutter (Class III, LOE C).

Caution is also advised to avoid the combination of AV nodal blocking agents that have a longer duration of action. For example, the short elimination half-life of adenosine affords follow-up treatment, if required, with a calcium channel blocker or β-blocker. Conversely the longer half-life of a calcium channel or β-blocker means their effects will overlap; profound bradycardia can develop if they are given serially.

Although antiarrhythmic medications (eg, amiodarone, procainamide, or sotalol) can also be used to treat SVTs, the higher toxicity and risk for proarrhythmia make these medications less desirable alternatives to the described AV nodal blocking agents. A possible exception is in patients with pre-excited atrial arrhythmias; the typical AV nodal blocking drugs are contraindicated in these patients and rate control may be achieved with antiarrhythmic medications. Importantly, use of these agents for atrial-based SVTs, such as atrial fibrillation and flutter can result in their termination, which may be undesirable in the absence of precautions to prevent the thromboembolic complications that may result from such conversion.

Wide-Complex Tachycardia (Figure 4, Boxes 5, 6, and 7)

Evaluation

The first step in the management of any tachycardia is to determine if the patient's condition is stable or unstable (Figure 4, **Box 3**). An unstable patient with a wide-complex tachycardia should be presumed to have VT and immediate cardioversion should be performed (Figure 4, **Box 4** and see above). Precordial thump may be considered for patients with witnessed, monitored, unstable ventricular tachycardia if a defibrillator is not immediately ready for use (Class IIb, LOE C).

If the patient is stable, the second step in management is to obtain a 12-lead ECG (Figure 4, **Boxes 6 and 7**) to evaluate the rhythm. At this point the provider should consider the need to obtain expert consultation. If the patient becomes unstable at any time, proceed with synchronized cardioversion or unsynchronized defibrillation should the arrhythmia deteriorate to VF or be due to a polymorphic VT.

Wide-complex tachycardias are defined as those with a QRS $\geq$0.12 second. The most common forms of wide-complex tachycardia are

- VT or VF
- SVT with aberrancy
- Pre-excited tachycardias (associated with or mediated by an accessory pathway)
- Ventricular paced rhythms

The third step in management of a tachycardia is to determine if the rhythm is regular or irregular. A *regular* wide-complex tachycardia is likely to be VT or SVT with aberrancy. An *irregular* wide-complex tachycardia may be atrial fibrillation with aberrancy, pre-excited atrial fibrillation (ie, atrial fibrillation using an accessory pathway for antegrade conduction), or polymorphic VT/torsades de pointes. Providers should consider the need for expert consultation when treating wide-complex tachycardias.

Therapy for Regular Wide-Complex Tachycardias

In patients with stable undifferentiated wide-QRS complex tachycardia, a reasonable approach is to try to identify the wide-complex tachycardia as SVT or VT and treat based on the algorithm for that rhythm.

If the etiology of the rhythm cannot be determined, the rate is regular, and the QRS is monomorphic, recent evidence suggests that IV adenosine is relatively safe for both treatment and diagnosis[47] (Class IIb, LOE B). However, adenosine should ***not*** be given for unstable or for ***irregular or polymorphic*** wide-complex tachycardias, as it may cause degeneration of the arrhythmia to VF (Class III, LOE C). If the wide-complex tachycardia proves to be SVT with aberrancy, it will likely be transiently slowed or converted by adenosine to sinus rhythm; if due to VT there will be no effect on rhythm (except in rare cases of idiopathic VT), and the brevity of the transient adenosine effect should be reasonably tolerated hemodynamically. Because close attention to these varying responses may help to diagnose the underlying rhythm, whenever possible, continuous ECG recording is strongly encouraged to provide such written documentation. This documentation can be invaluable in helping to establish a firm rhythm diagnosis even if after the fact. Typically, adenosine is administered in a manner similar to treatment of PSVT: as a 6 mg rapid IV push; providers may follow the first dose with a 12 mg bolus and a second 12 mg bolus if the rate fails to convert. When adenosine is given for undifferentiated wide-complex tachycardia, a defibrillator should be available.

Depending on the underlying rhythm, the response to adenosine challenge can be variable. Some studies[408–412] showed that adenosine converted an undifferentiated wide-complex tachycardia to sinus rhythm. Another study[413] showed poor rates of conversion to sinus rhythm in patients known to have VT. The following adverse effects were reported in patients with pre-excited atrial fibrillation treated with adenosine: conversion to atrial fibrillation with a rapid ventricular response in one patient later found to have preexcitation, conversion to VF in one patient with known WPW,[414] conversion to VF in 4 patients with pre-excited atrial fibrillation,[415] conversion to VF in 2 patients with WPW,[416] and a single case of VF in a patient with VT.[417]

Verapamil is contraindicated for wide-complex tachycardias unless known to be of supraventricular origin (Class III, LOE B). Adverse effects when the rhythm was due to VT were shown in 5 small case series.[414–418] Profound hypotension was reported in 11 of 25 patients known to have VT treated with verapamil.[418]

For patients who are stable with likely VT, IV antiarrhythmic drugs or elective cardioversion is the preferred treatment strategy. If IV antiarrhythmics are administered, procainamide (Class IIa, LOE B), amiodarone (Class IIb, LOE B), or sotalol (Class IIb, LOE B) can be considered. Procainamide and sotalol should be avoided in patients with prolonged QT. If one of these antiarrhythmic agents is given, a second agent should not be given without expert consultation (Class III, LOE B). If antiarrhythmic therapy is unsuccessful, cardioversion or expert consultation should be considered (Class IIa, LOE C).

One randomized comparison found procainamide (10 mg/kg) to be superior to lidocaine (1.5 mg/kg) for termination of hemodynamically stable monomorphic VT.[419] Procainamide can be administered at a rate of 20 to 50 mg/min until the arrhythmia is suppressed, hypotension ensues, QRS duration increases >50%, or the maximum dose of 17 mg/kg is given. Maintenance infusion is 1 to 4 mg/min. Procainamide should be avoided in patients with prolonged QT and congestive heart failure.

IV sotalol (100 mg IV over 5 minutes) was found to be more effective than lidocaine (100 mg IV over 5 minutes) when administered to patients with spontaneous hemodynamically stable sustained monomorphic VT in a double-blind randomized trial within a hospital setting.[420] In a separate study of 109 patients with a history of spontaneous and inducible sustained

ventricular tachyarrhythmias, infusing 1.5 mg/kg of sotalol over ≤5 minutes was found to be relatively safe and effective, causing hypotension in only 2 patients, both of whom responded to IV fluid.[421] Package insert recommends slow infusion, but the literature supports more rapid infusion of 1.5 mg/kg over 5 minutes or less. Sotalol should be avoided in patients with a prolonged QT interval.

Amiodarone is also effective in preventing recurrent monomorphic VT or treating refractory ventricular arrhythmias[286,422–424] in patients with coronary artery disease and poor ventricular function. It is given 150 mg IV over 10 minutes; dosing should be repeated as needed to a maximum dose of 2.2 g IV per 24 hours. Higher doses (300 mg) were associated with an increased frequency of hypotension, although some reports[422,424] attributed the hypotension to the vasoactive solvents that are not present in a new form of the drug recently approved for use in the US.

By comparison, lidocaine is less effective in terminating VT than procainamide, sotalol, and amiodarone,[286,419,420] and when given to patients with or without a history of MI with spontaneous sustained stable VT in the hospital setting.[413,425,426] Lidocaine has been reported to variably terminate VT when administered intramuscularly to patients with AMI and VT in the out-of-hospital setting.[427,428] Thus, while occasionally effective, lidocaine should be considered second-line antiarrhythmic therapy for monomorphic VT. Lidocaine can be administered at a dose of 1 to 1.5 mg/kg IV bolus. Maintenance infusion is 1 to 4 mg/min (30 to 50 mcg/kg per minute).

Irregular Tachycardias

Atrial Fibrillation and Flutter

Evaluation

An irregular narrow-complex or wide-complex tachycardia is most likely atrial fibrillation (with or without aberrant conduction) with an uncontrolled ventricular response. Other diagnostic possibilities include MAT or sinus rhythm/tachycardia with frequent atrial premature beats. When there is doubt about the rhythm diagnosis and the patient is stable, a 12-lead ECG with expert consultation is recommended.

Therapy

General management of atrial fibrillation should focus on control of the rapid ventricular rate (rate control), conversion of hemodynamically unstable atrial fibrillation to sinus rhythm (rhythm control), or both. Patients with an atrial fibrillation duration of >48 hours are at increased risk for cardioembolic events, although shorter durations of atrial fibrillation do not exclude the possibility of such events. Electric or pharmacologic cardioversion (conversion to normal sinus rhythm) should *not be attempted* in these patients unless the patient is unstable. An alternative strategy is to perform cardioversion following anticoagulation with heparin *and* performance of transesophageal echocardiography to ensure the absence of a left atrial thrombus; see the ACC/AHA Guidelines for Management of Patients with Atrial Fibrillation.[429]

Rate Control

Patients who are hemodynamically unstable should receive prompt electric cardioversion. More stable patients require ventricular rate control as directed by patient symptoms and hemodynamics. IV β-blockers and nondihydropyridine calcium channel blockers such as diltiazem[430–433] are the drugs of choice for acute rate control in most individuals with atrial fibrillation and rapid ventricular response (Class IIa, LOE A). Digoxin[434–436] and amiodarone[437,438] may be used for rate control in patients with congestive heart failure; however, the potential risk of conversion to sinus rhythm with amiodarone should be considered before treating with this agent.

A wide-complex irregular rhythm should be considered pre-excited atrial fibrillation. Expert consultation is advised. Avoid AV nodal blocking agents such as adenosine, calcium channel blockers, digoxin, and possibly β-blockers in patients with pre-excitation atrial fibrillation because these drugs may cause a paradoxical increase in the ventricular response. Typically, patients with pre-excited atrial fibrillation present with very rapid heart rates and require emergent electric cardioversion. When electric cardioversion is not feasible or effective, or atrial fibrillation is recurrent, use of rhythm control agents (discussed below) may be useful for both rate control and stabilization of the rhythm.

Rhythm Control

A variety of agents have been shown to be effective in terminating atrial fibrillation (pharmacologic or chemical cardioversion), although success between them varies and not all are available as parenteral formulations. Expert consultation is recommended.

Polymorphic (Irregular) VT

Polymorphic (irregular) VT requires immediate defibrillation with the same strategy used for VF.

Pharmacologic treatment to prevent recurrent polymorphic VT should be directed by the underlying cause of VT and the presence or absence of a long QT interval during sinus rhythm.

If a long QT interval is observed during sinus rhythm (ie, the VT is torsades de pointes), the first step is to stop medications known to prolong the QT interval. Correct electrolyte imbalance and other acute precipitants (eg, drug overdose or poisoning: see Part 12.7: "Cardiac Arrest Associated With Toxic Ingestions"). Although magnesium is commonly used to treat torsades de pointes VT (polymorphic VT associated with long QT interval), it is supported by only 2 observational studies[107,170] that showed effectiveness in patients with prolonged QT interval. One adult case series[439] showed that isoproterenol or ventricular pacing can be effective in terminating torsades de pointes associated with bradycardia and drug-induced QT prolongation. Polymorphic VT associated with familial long QT syndrome may be treated with IV magnesium, pacing, and/or β-blockers; isoproterenol should be avoided. Polymorphic VT associated with acquired long QT syndrome may be treated with IV magnesium. The addition of pacing or IV isoproterenol may be considered when polymorphic VT is accompanied by bradycardia or appears to be precipitated by pauses in rhythm.

In the absence of a prolonged QT interval, the most common cause of polymorphic VT is myocardial ischemia. In this situation IV amiodarone and β-blockers may reduce the frequency of arrhythmia recurrence (Class IIb, LOE C). Myocar-

dial ischemia should be treated with β-blockers and consideration be given to expeditious cardiac catheterization with revascularization. Magnesium is unlikely to be effective in preventing polymorphic VT in patients with a normal QT interval (Class IIb, LOE C),[107] but amiodarone may be effective (Class IIb, LOE C).[440]

Other causes of polymorphic VT apart from ischemia and long QT syndrome are catecholaminergic VT (which may be responsive to β-blockers) and Brugada syndrome (which may be responsive to isoproterenol).

Summary

The goal of therapy for bradycardia or tachycardia is to rapidly identify and treat patients who are hemodynamically unstable or symptomatic due to the arrhythmia. Drugs or, when appropriate, pacing may be used to control unstable or symptomatic bradycardia. Cardioversion or drugs or both may be used to control unstable or symptomatic tachycardia. ACLS providers should closely monitor stable patients pending expert consultation and should be prepared to aggressively treat those with evidence of decompensation.

Disclosures

Guidelines Part 8: ACLS Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Robert W. Neumar	University of Pennsylvania-Associate Professor of Emergency Medicine	None	None	None	None	None	None
Charles W. Otto	University of Arizona–Professor	None	None	None	None	None	None
Mark S. Link	Tufts Medical Center–Physician	None	None	None	None	None	None
Steven L. Kronick	University of Michigan–Assistant Professor	None	None	None	None	None	None
Michael Shuster	Self-employed–emergency physician	None	None	None	None	None	None
Clifton W. Callaway	University of Pittsburgh School of Medicine–Associate Professor; UPMC Health System—Physician *American Heart Association-Work Sheet Editor for 2010 Guidelines. My effort on this project is paid to University of Pittsburgh as a "contracted services agreement," and not paid directly to me	†Grants to University of Pittsburgh: NHLBI-Resuscitation Outcomes Consortium HRSA-Development and Dissemination of Program Tools for Uncontrolled Donation After Cardiac Death (UDCD)	*Loan of an Arctic Sun cooling device (without disposables) to human physiology laboratory for experiments on hypothermia by Medivance, Inc.	None	†Co-inventor on patent about ventricular fibrillation waveform analysis, licensed by University of Pittsburgh to Medtronic ERS, Inc.	None	None
Peter J. Kudenchuk	University of Washington–Professor of Medicine	†Resuscitation Outcomes Consortium (NIH/NHLBI)	None	Network for Continuing Medical Education, Academy for Healthcare Education, Sanofi-Aventis, with honoraria	Sanofi-Aventis, Novartis	None	None
Joseph P. Ornato	Richmond Ambulance Authority–Medical Director; Virginia Commonwealth University-Prof & Chmn, Emergency Medicine	†Consultant and Cardiac Co-Chairman, NIH Resuscitation Outcomes Consortium Principal Investigator, VCU site for NIH Neurological Emergency Treatment Trials Network	None	*Hospital grand rounds presentations funded by ZOLL Circulation *Occasional hospital grand rounds supported by unrestricted educational grants from Squibb/Sanofi, ZOLL	None	*ZOLL Circulation Science Advisory Board (UNPAID, only receive travel reimbursement)	None

(Continued)

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Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Bryan McNally	Emory University–Assistant Professor of Emergency Medicine	†Center for Disease Control and Prevention, CARES-Cardiac Arrest Registry to Enhance Survival, Money comes to Emory University School of Medicine as part of a cooperative agreement through American Association of Medical Colleges	None	None	None	None	None
Scott M. Silvers	Mayo Clinic–Chair, Department of Emergency Medicine	None	None	None	None	None	None
Rod S. Passman	Northwestern University–Associate Professor	None	None	None	None	*Steering Committee member for Medtronic Crystal AF study	None
Roger D. White	Mayo Clinic–staff physician	None	None	None	None	None	None
Erik P. Hess	Mayo Clinic–Senior Associate Consultant	None	None	None	None	None	None
Wanchun Tang	Weil Institute of Critical Care Medicine–Professor and president	None	None	*47th Weil Critical Care Symposium: $1,500	None	None	None
Daniel Davis	UC San Diego–Faculty physician	†Zoll Medical (Air Medical Advanced Monitoring Strategies)	*Bispectral EEG Analyzer (Zoll Medical)	*Continuous Renal Replacement Therapy Conference 2009 Hospital Medicine National and Regional Meeting 2009 Grand Rounds Redding Medical Center	None	†Cardinal Health (Development of a Prehospital Ventilator)	*Derek White Law Firm John Anderson Law Firm Otorowski Johnston Diamond & Golden Law Firm
Elizabeth Sinz	Penn State Hershey Medical Center–Professor of Anesthesiology and Neurosurgery; AHA: Paid Consultant Associate Science Editor	None	None	None	None	None	None
Laurie J. Morrison	St Michaels Hosp. Clinician Scientist	None	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

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KEY WORDS: arrhythmia ■ cardiac arrest ■ drugs ■ ventricular arrhythmia ■ ventricular fibrillation

Correction

In the article by Neumar et al, "Part 8: Adult Advanced Cardiovascular Life Support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care," which published ahead of print on October 18, 2010, and appeared with the November 2, 2010, issue of the journal (*Circulation.* 2010;122[suppl 3]:S729–S767), several corrections were needed.

1. On page S736, in Figure 1, in gray text box on the right, under "Shock Energy," the bullet for "Biphasic" read, "**Biphasic:** Manufacturer recommendation (120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered." It has been updated to read, "**Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered."

2. On page S737, in Figure 2, in gray text box on the right, under "Shock Energy," the bullet for "Biphasic" read, "**Biphasic:** Manufacturer recommendation (120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered." It has been updated to read, "**Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered."

3. On page S738, in the left column, the first paragraph under "Waveform and Energy," the first sentence read, "If a biphasic defibrillator is available, providers should use the manufacturer's recommended energy dose (120 to 200 J) for terminating VF (Class I, LOE B)." It has been updated to read, "If a biphasic defibrillator is available, providers should use the manufacturer's recommended energy dose (eg, initial dose of 120 to 200 J) for terminating VF (Class I, LOE B)."

These corrections have been made to the current online version of the article, which is available at http://circ.ahajournals.org/cgi/content/full/122/18_suppl_3/S729.

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