Part 7: Adult Advanced Cardiovascular Life Support

Web-based Integrated 2010 & 2015 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Key Words: arrhythmia cardiac arrest drugs ventricular arrhythmia ventricular fibrillation

1 Highlights

Summary of Key Issues and Major Changes

Key issues and major changes in the 2015 Guidelines Update recommendations for advanced cardiac life support include the following:

- The combined use of vasopressin and epinephrine offers no advantage to using standard-dose epinephrine in cardiac arrest. Also, vasopressin does not offer an advantage over the use of epinephrine alone. Therefore, to simplify the algorithm, vasopressin has been removed from the Adult Cardiac Arrest Algorithm–2015 Update.
- Low end-tidal carbon dioxide ($ETCO_2$) in intubated patients after 20 minutes of CPR is associated with a very low likelihood of resuscitation. While this parameter should not be used in isolation for decision making, providers may consider low $ETCO_2$ after 20 minutes of CPR in combination with other factors to help determine when to terminate resuscitation.
- Steroids may provide some benefit when bundled with vasopressin and epinephrine in treating IHCA. While routine use is not recommended pending follow-up studies, it would be reasonable for a provider to administer the bundle for IHCA.
- When rapidly implemented, ECPR can prolong viability, as it may provide time to treat potentially reversible conditions or arrange for cardiac transplantation for patients who are not resuscitated by conventional CPR.
- In cardiac arrest patients with nonshockable rhythm and who are otherwise receiving epinephrine, the early provision of epinephrine is suggested.
- Studies about the use of lidocaine after ROSC are conflicting, and routine lidocaine use is not recommended. However, the initiation or continuation of lidocaine may be considered immediately after ROSC from VF/pulseless ventricular tachycardia (pVT) cardiac arrest.
- One observational study suggests that ß-blocker use after cardiac arrest may be associated with better outcomes than when ß-blockers are not used. Although this observational study is not strong-enough evidence to recommend routine use, the initiation or continuation of an oral or intravenous (IV) ß-blocker may be considered early after hospitalization from cardiac arrest due to VF/pVT.

Vasopressors for Resuscitation: Vasopressin

2015 (Updated): Vasopressin in combination with epinephrine offers no advantage as a substitute for standard-dose epinephrine in cardiac arrest.

2010 (Old): One dose of vasopressin 40 units IV/ intraosseously may replace either the first or second dose of epinephrine in the treatment of cardiac arrest.

Why: Both epinephrine and vasopressin administration during cardiac arrest have been shown to improve ROSC. Review of the available evidence shows that efficacy of the 2 drugs is similar and that there is no demonstrable benefit from administering both epinephrine and vasopressin as compared with epinephrine alone. In the interest of simplicity, vasopressin has been removed from the Adult Cardiac Arrest Algorithm.

Vasopressors for Resuscitation: Epinephrine

2015 (New): It may be reasonable to administer epinephrine as soon as feasible after the onset of cardiac arrest due to an initial nonshockable rhythm.

Why: A very large observational study of cardiac arrest with nonshockable rhythm compared epinephrine given

at 1 to 3 minutes with epinephrine given at 3 later time intervals (4 to 6, 7 to 9, and greater than 9 minutes). The study found an association between early administration of epinephrine and increased ROSC, survival to hospital discharge, and neurologically intact survival.

$ETCO_2$ for Prediction of Failed Resuscitation

2015 (New): In intubated patients, failure to achieve an $ETCO_2$ of greater than 10 mm Hg by waveform capnography after 20 minutes of CPR may be considered as one component of a multimodal approach to decide when to end resuscitative efforts but should not be used in isolation.

Why: Failure to achieve an $ETCO_2$ of 10 mm Hg by waveform capnography after 20 minutes of resuscitation has been associated with an extremely poor chance of ROSC and survival. However, the studies to date are limited in that they have potential confounders and have included relatively small numbers of patients, so it is inadvisable to rely solely on $ETCO_2$ in determining when to terminate resuscitation.

Extracorporeal CPR

2015 (New): ECPR may be considered among select cardiac arrest patients who have not responded to initial conventional CPR, in settings where it can be rapidly implemented.

Why: Although no high-quality studies have compared ECPR to conventional CPR, a number of lower-quality studies suggest improved survival with good neurologic outcome for select patient populations. Because ECPR is resource intensive and costly, it should be considered only when the patient has a reasonably high likelihood of benefit—in cases where the patient has a potentially reversible illness or to support a patient while waiting for a cardiac transplant.

Post–Cardiac Arrest Drug Therapy: Lidocaine

2015 (New): There is inadequate evidence to support the routine use of lidocaine after cardiac arrest. However, the initiation or continuation of lidocaine may be considered immediately after ROSC from cardiac arrest due to VF/pVT.

2Why: While earlier studies showed an association between giving lidocaine after myocardial infarction and increased mortality, a recent study of lidocaine in cardiac arrest survivors showed a decrease in the incidence of recurrent VF/pVT but did not show either long-term benefit or harm.

Post–Cardiac Arrest Drug Therapy: ß-Blockers

2015 (New): There is inadequate evidence to support the routine use of a ß-blocker after cardiac arrest. However, the initiation or continuation of an oral or IV ß-blocker may be considered early after hospitalization from cardiac arrest due to VF/pVT.

Why: In an observational study of patients who had ROSC after VF/pVT cardiac arrest, ß-blocker administration was associated with higher survival rates. However, this finding is only an associative relationship, and the routine use of ß-blockers after cardiac arrest is potentially hazardous because ß-blockers can cause or worsen hemodynamic instability, exacerbate heart failure, and cause bradyarrhythmias. Therefore, providers should evaluate patients individually for their suitability for ß-blockers.

2 Introduction - Updated

These Web-based Integrated Guidelines incorporate the relevant recommendations from 2010 and the new or updated recommendations from 2015.

Basic life support (BLS), advanced cardiovascular life support (ACLS), and post–cardiac arrest care are labels of convenience that each describe a set of skills and knowledge that are applied sequentially during the treatment of patients who have a cardiac arrest. There is overlap as each stage of care progresses to the next, but generally ACLS comprises the level of care between BLS and post–cardiac arrest care.

ACLS training is recommended for advanced providers of both prehospital and in-hospital medical care. In the past, much of the data regarding resuscitation was gathered from out-of-hospital arrests, but in recent years, data have also been collected from in-hospital arrests, allowing for a comparison of cardiac arrest and resuscitation in these 2 settings. While there are many similarities, there are also some differences between in-

and out-of-hospital cardiac arrest etiology, which may lead to changes in recommended resuscitation treatment or in sequencing of care. The consideration of steroid administration for in-hospital cardiac arrest (IHCA) versus out-of-hospital cardiac arrest (OHCA) is one such example discussed in this Part.

The recommendations in this *2015 American Heart Association* (AHA) *Guidelines Update for Cardiopulmonary Resuscitation* (CPR) *and Emergency Cardiovascular Care* (ECC) are based on an extensive evidence review process that was begun by the International Liaison Committee on Resuscitation (ILCOR) after the publication of the ILCOR *2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations* and was completed in February 2015.[1]

In this in-depth evidence review process, the ILCOR task forces examined topics and then generated prioritized lists of questions for systematic review. Questions were first formulated in PICO (population, intervention, comparator, outcome) format,[2] and then a search strategy and inclusion and exclusion criteria were defined and a search for relevant articles was performed. The evidence was evaluated by using the standardized methodological approach proposed by the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) Working Group.[3]

The quality of the evidence was categorized based on the study methodologies and the 5 core GRADE domains of risk of bias, inconsistency, indirectness, imprecision, and other considerations (including publication bias). Then, where possible, consensus-based treatment recommendations were created.

To create the 2015 Guidelines Update, the AHA formed 15 writing groups, with careful attention to avoid or manage conflicts of interest, to assess the ILCOR treatment recommendations and to write AHA treatment recommendations by using the AHA Class of Recommendation and Level of Evidence (LOE) system.

The recommendations made in this 2015 Guidelines Update are informed by the ILCOR recommendations and GRADE classification, in the context of the delivery of medical care in North America. The AHA ACLS writing group made new recommendations only on topics specifically reviewed by ILCOR in 2015. This chapter delineates any instances where the AHA writing group developed recommendations that are substantially different than the ILCOR statements. In the online version of this document, live links are provided so the reader can connect directly to the systematic reviews on the Scientific Evidence Evaluation and Review System (SEERS) website. These links are indicated by a superscript combination of letters and numbers (eg, ALS 433).

This update uses the newest AHA COR and LOE classification system, which contains modifications of the Class III recommendation and introduces LOE B-R (randomized studies) and B-NR (nonrandomized studies) as well as LOE C-LD (limited data) and LOE C-EO (consensus of expert opinion). All recommendations made in this 2015 Guidelines Update, as well as in the 2010 Guidelines, are listed in the Appendix. For further information, see "Part 2: Evidence Evaluation and Management of Conflicts of Interest." The ILCOR ACLS Task Force addressed 37 PICO questions related to ACLS care (presented in this Part) in 2015. These questions included oxygen dose during CPR, advanced airway devices, ventilation rate during CPR, exhaled carbon dioxide (CO_2) detection for confirmation of airway placement, physiologic monitoring during CPR, prognostication during CPR, defibrillation, antiarrhythmic drugs, and vasopressors. The 2 new topics are steroids and hormones in cardiac arrest, and extracorporeal CPR (ECPR), perhaps better known to the inpatient provider community as extracorporeal life support (ECMO). The 2010 Guidelines Part on electrical therapies (defibrillation and emergency pacing) has been eliminated, and relevant material from it is now included in this ACLS Part.

The major changes in the 2015 ACLS guidelines include recommendations about prognostication during CPR based on exhaled CO_2 measurements, timing of epinephrine administration stratified by shockable or nonshockable rhythms, and the possibility of bundling treatment of steroids, vasopressin, and epinephrine for treatment of in-hospital arrests. In addition, the administration of vasopressin as the sole vasoactive drug during CPR has been removed from the algorithm.

3 Adjuncts to CPR - Updated

3.1 Oxygen Dose During CPR - Updated ALS 889

The 2015 ILCOR systematic review considered inhaled oxygen delivery both during CPR and in the post–cardiac arrest period. This 2015 Guidelines Update evaluates the optimal inspired concentration of oxygen during CPR. The immediate goals of CPR are to restore the energy state of the heart so it can resume mechanical work and to maintain the energy state of the brain to minimize ischemic injury. Adequate oxygen delivery is necessary to achieve these goals. Oxygen delivery is dependent on both blood flow and arterial oxygen content. Because

blood flow is typically the major limiting factor to oxygen delivery during CPR, it is theoretically important to maximize the oxygen content of arterial blood by maximizing inspired oxygen concentration. Maximal inspired oxygen can be achieved with high-flow oxygen into a resuscitation bag device attached to a mask or an advanced airway.

3.1.1 2015 Evidence Summary

There were no adult human studies identified that directly compared maximal inspired oxygen with any other inspired oxygen concentration. However, 1 observational study of 145 OHCA patients evaluated arterial Po2 measured during CPR and cardiac arrest outcomes.[4] In this study, during which all patients received maximal inspired oxygen concentration, patients were divided into low, intermediate, and high arterial Po2 ranges (less than 61, 61–300, and greater than 300 mmHg, respectively). The higher ranges of arterial Po2 during CPR were associated with an increase in hospital admission rates (low, 18.8%; intermediate, 50.6%; and high, 83.3%). However, there was no statistical difference in overall neurologic survival (low, 3.1%; intermediate, 13.3%; and high, 23.3%). Of note, this study did not evaluate the provision of various levels of inspired oxygen, so differences between groups likely reflect patient-level differences in CPR quality and underlying pathophysiology. This study did not find any association between hyperoxia during CPR and poor outcome.

3.1.2 2015 Recommendation - Updated

When supplementary oxygen is available, it may be reasonable to use the maximal feasible inspired oxygen concentration during CPR. (Class IIb, LOE C-EO)

Evidence for detrimental effects of hyperoxia that may exist in the immediate post–cardiac arrest period should not be extrapolated to the low-flow state of CPR where oxygen delivery is unlikely to exceed demand or cause an increase in tissue Po_2 . Therefore, until further data are available, physiology and expert consensus support providing the maximal inspired oxygen concentration during CPR.

3.2 Passive Oxygen Delivery During CPR

This topic was updated in 2015 and is discussed in Part 5: Adult Basic Life Support and Cardiopulmonary Resuscitation Quality.

3.3 Monitoring Physiologic Parameters During CPR - Updated ALS 656

Monitoring both provider performance and patient physiologic parameters during CPR is essential to optimizing CPR quality. The 2010 Guidelines put a strong emphasis on CPR quality. In 2013, the AHA published a Consensus Statement focused on strategies to improve CPR quality.[5] In 2015, the ILCOR ACLS Task Force evaluated the available clinical evidence to determine whether using physiologic feedback to guide CPR quality improved survival and neurologic outcome.

3.3.1 2015 Evidence Summary

Animal and human studies indicate that monitoring physiologic parameters during CPR provides valuable information about the patient's condition and response to therapy. Most important, end-tidal CO2 (etco2), coronary perfusion pressure, arterial relaxation pressure, arterial blood pressure, and central venous oxygen saturation correlate with cardiac output and myocardial blood flow during CPR, and threshold values have been reported below which return of spontaneous circulation (ROSC) is rarely achieved.[6-12] These parameters can be monitored continuously, without interrupting chest compressions. An abrupt increase in any of these parameters is a sensitive indicator of ROSC.[13-30] There is evidence that these and other physiologic parameters can be modified by interventions aimed at improving CPR quality.[6,31-42]

The 2015 ILCOR systematic review was unable to identify any clinical trials that have studied whether titrating resuscitative efforts to a single or combined set of physiologic parameters during CPR results in improved survival or neurologic outcome.

3.3.2 2015 Recommendation - Updated

Although no clinical study has examined whether titrating resuscitative efforts to physiologic

parameters during CPR improves outcome, it may be reasonable to use physiologic parameters (quantitative waveform capnography, arterial relaxation diastolic pressure, arterial pressure monitoring, and central venous oxygen saturation) when feasible to monitor and optimize CPR quality, guide vasopressor therapy, and detect ROSC. (Class IIb, LOE C-EO)

Previous guidelines specified physiologic parameter goals; however, because the precise numerical targets for these parameters during resuscitation have not as yet been established, these were not specified in 2015.

3.4 Ultrasound During Cardiac Arrest - Updated ALS 658

Bedside cardiac and noncardiac ultrasound are frequently used as diagnostic and prognostic tools for critically ill patients.[43] Ultrasound may be applied to patients receiving CPR to help assess myocardial contractility and to help identify potentially treatable causes of cardiac arrest such as hypovolemia, pneumothorax, pulmonary thromboembolism, or pericardial tamponade.[44] However, it is unclear whether important clinical outcomes are affected by the routine use of ultrasound among patients experiencing cardiac arrest.

3.4.1 2015 Evidence Summary

One limited study with a small sample size was identified that specifically addressed the utility of ultrasound during cardiac arrest. This study evaluated bedside cardiac ultrasound use during ACLS among adult patients in pulseless electrical activity arrest and found no difference in the incidence of ROSC when ultrasound was used.[45]

3.4.2 2015 Recommendations - Updated

Ultrasound (cardiac or noncardiac) may be considered during the management of cardiac arrest, although its usefulness has not been well established. (Class IIb, LOE C-EO)

If a qualified sonographer is present and use of ultrasound does not interfere with the standard cardiac arrest treatment protocol, then ultrasound may be considered as an adjunct to standard patient evaluation. (Class IIb, LOE C-EO)

4 Adjuncts for Airway Control and Ventilation - Updated

4.1 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.

4.2 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.[46, 47] 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 compressions for ventilation. Advanced airway placement in cardiac arrest should not delay initial CPR

and defibrillation for VF cardiac arrest. (Class I, LOE C)

4.3 Bag-Mask Ventilation - Updated

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.[48,49] 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 and Cardiopulmonary Resuscitation Quality."

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.[50-52]

4.3.1 Bag-Mask Ventilation Compared With Any Advanced Airway During CPR - Updated ALS 783

As stated above, bag-mask ventilation is a commonly used method for providing oxygenation and ventilation in patients with respiratory insufficiency or arrest. When cardiac arrest occurs, providers must determine the best way to support ventilation and oxygenation. Options include standard bag-mask ventilation versus the placement of an advanced airway (ie, endotracheal tube [ETT], supraglottic airway device [SGA]). Previous guidelines recommended that prolonged interruptions in chest compressions should be avoided during transitions from bag-mask ventilation to an advanced airway device. In 2015, ILCOR evaluated the evidence comparing the effect of bagmask ventilation versus advanced airway placement on overall survival and neurologic outcome from cardiac arrest.

4.3.1.1 2015 Evidence Summary

There is inadequate evidence to show a difference in survival or favorable neurologic outcome with the use of bag-mask ventilation compared with endotracheal intubation[53-59] or other advanced airway devices.[53,55-57,60] The majority of these retrospective observational studies demonstrated slightly worse survival with the use of an advanced airway when compared with bag-mask ventilation. However, interpretation of these results is limited by significant concerns of selection bias. Two additional observational studies[60,61] showed no difference in survival.

4.4 Airway Adjuncts

4.4.1 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.[62-65] However, it also may impede ventilation and interfere with placement of a supraglottic airway or intubation.[66-72] 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)

4.4.2 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)

4.4.3 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.[73] Two case reports of inadvertent intracranial placement of a nasopharyngeal airway in patients with basilar skull fractures[74, 75] 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)

4.5 Advanced Airways - Updated

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.

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.[76] 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.[77]

In out-of-hospital urban and rural settings, patients intubated during resuscitation had a better survival rate than patients who were not intubated,[78] whereas in an in-hospital setting, patients who required intubation during CPR had a worse survival rate.[79] A recent study[80] 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).

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 100/min to 120/min, without pauses for ventilation. The provider delivering ventilation should provide 1 breath every 6 seconds (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.

4.5.1 Advanced Airway Placement Choice - Updated

Advanced airway devices are frequently placed by experienced providers during CPR if bag-mask ventilation is inadequate or as a stepwise approach to airway management. Placement of an advanced airway may result in interruption of chest compressions, and the ideal timing of placement to maximize outcome has not been adequately studied. The use of an advanced airway device such as an ETT or SGA and the effect of ventilation technique on overall survival and neurologic outcome was evaluated in 2015.

4.5.1.1 2015 Evidence Summary

4.5.1.1.1 Endotracheal Intubation Versus Bag-Mask Ventilation - Updated

There is no high-quality evidence favoring the use of endotracheal intubation compared with bag-mask ventilation or an advanced airway device in relation to overall survival or favorable neurologic outcome.[53-59] Evaluating retrospective studies that compare bag-mask ventilation to endotracheal intubation is challenging because patients with more severe physiologic compromise will typically receive more invasive care (including endotracheal intubation) than patients who are less compromised and more likely to survive. Within that context, a number of retrospective studies show an association of worse outcome in those who were intubated as compared with those receiving bag-mask ventilation. While the studies did attempt to control for confounders, bias still may have been present, limiting the interpretation of these investigations. These studies illustrate that endotracheal intubation can be associated with a number of complications and that the procedure requires skill and experience. Risks of endotracheal intubation during resuscitation include unrecognized esophageal intubation and increased hands-off time.

4.5.1.1.2 Supraglottic Airway Devices - Updated

Several retrospective studies compared a variety of supraglottic devices (laryngeal mask airway, laryngeal tube, Combitube, esophageal obturator airway) to both bag-mask ventilation and endotracheal intubation. There is no high-quality evidence demonstrating a difference in survival rate or favorable neurologic outcome from use of an SGA compared with bagmask ventilation[53,55-57] or endotracheal intubation.[53,55,56,60,81-86] Three

observational studies demonstrated a lower rate of both overall survival and favorable neurologic outcome when SGA use was compared with bag-mask ventilation,[53,55,57] whereas another observational study demonstrated similar survival rates.[56]

In studies comparing SGA insertion to endotracheal intubation, no high-quality studies have demonstrated a difference in overall survival or favorable neurologic outcome.[56,60,81-83,86] Several retrospective observational studies show more favorable outcome with the use of an SGA device, whereas other studies favor the use of endotracheal intubation.[53,55,56,84-86]

4.5.1.2 2015 Recommendations - Updated

Either a bag-mask device or an advanced airway may be used for oxygenation and ventilation during CPR in both the in-hospital and out-of-hospital setting. (Class IIb, LOE C-LD)

For healthcare providers trained in their use, either an SGA device or an ETT may be used as the initial advanced airway during CPR. (Class IIb, LOE C-LD)

Recommendations for advanced airway placement presume that the provider has the initial training and skills as well as the ongoing experience to insert the airway and verify proper position with minimal interruption in chest compressions. Bag-mask ventilation also requires skill and proficiency. The choice of bag-mask device versus advanced airway insertion, then, will be determined by the skill and experience of the provider.

Frequent experience or frequent retraining is recommended for providers who perform endotracheal intubation.[76,87] (Class I, LOE B)

EMS systems that perform prehospital intubation should provide a program of ongoing quality improvement to minimize complications. (Class IIa, LOE B)

4.5.2 Clinical Assessment of Tracheal Tube Placement - Updated ALS 469

The 2015 ILCOR systematic review considered tracheal tube placement during CPR. This section evaluates methods for confirming correct tracheal tube placement.

Attempts at endotracheal intubation during CPR have been associated with unrecognized tube misplacement or displacement as well as prolonged interruptions in chest compression. Inadequate training, lack of experience, patient physiology (eg, low pulmonary blood flow, gastric contents in the trachea, airway obstruction), and patient movement may contribute to tube misplacement. After correct tube placement, tube displacement or obstruction may develop. In addition to auscultation of the lungs and stomach, several methods (eg, waveform capnography, CO2 detection devices, esophageal detector device, tracheal ultrasound, fiberoptic bronchoscopy) have been proposed to confirm successful tracheal intubation in adults during cardiac arrest.

4.5.2.1 2015 Evidence Summary

The evidence regarding the use of tracheal detection devices during cardiac arrest is largely observational. Observational studies and 1 small randomized study of waveform capnography to verify ETT position in victims of cardiac arrest report a specificity of 100% for correct tube placement.[88-90] Although the sensitivity of waveform capnography for detecting tracheal tube placement immediately after prehospital intubation was 100% in 1 study,[88] several other studies showed that the sensitivity of waveform capnography decreases after a prolonged cardiac arrest. [88-90] Differences in sensitivity can be explained by the low pulmonary blood flow during cardiac arrest, which will decrease ETCO2 concentration.

Although exhaled CO2 detection suggests correct tracheal tube placement, false-positive results (CO2 detection with esophageal intubation) can occur after ingestion of carbonated liquids.66 False-negative results (ie, absent exhaled CO2 in the presence of tracheal intubation) can occur in the setting of pulmonary embolism, significant hypotension, contamination of the detector with gastric contents, and severe airflow obstruction.[14,91,92] The use

of CO2 -detecting devices to determine the correct placement of other advanced airways (eg, Combitube, laryngeal mask airway) has not been studied, but, as with an ETT, effective ventilation should produce a capnography waveform during CPR and after ROSC.

Colorimetric and nonwaveform CO2 detectors can identify the presence of exhaled CO2 from the respiratory tract, but there is no evidence that these devices are accurate for continued monitoring of ETT placement.[14,88,93-97] Moreover, because a minimal threshold of CO2 must be reached to activate the detector and exhaled CO2 is low in cardiac arrest, proper placement of an ETT may not be confirmed with this qualitative methodology.

While observational studies and a small randomized controlled trial (RCT) of esophageal detector devices report a low false-positive rate for confirming tracheal placement, there is no evidence that these devices are accurate or practical for the continued monitoring of ETT placement.[89-98,93,99,100]

An ultrasound transducer can be placed transversely on the anterior neck above the suprasternal notch to identify endotracheal or esophageal intubation. In addition, ultrasound of the thoracic cavity can identify pleural movement as lung sliding. Unlike capnography, confirmation of ETT placement via ultrasonography is not dependent on adequate pulmonary blood flow and CO2 in exhaled gas.[101-103] One small prospective study of experienced clinicians compared tracheal ultrasound to waveform capnography and auscultation during CPR and reported a positive predictive value for ultrasound of 98.8% and negative predictive value of 100%.[103] The usefulness of tracheal and pleural ultrasonography, like fiberoptic bronchoscopy, may be limited by abnormal anatomy, availability of equipment, and operator experience.

4.5.2.2 2015 Recommendations - Updated

Continuous waveform capnography is recommended in addition to clinical assessment as the most reliable method of confirming and monitoring correct placement of an ETT. (Class I, LOE C-LD)

If continuous waveform capnometry is not available, a nonwaveform CO2 detector, esophageal detector device, or ultrasound used by an experienced operator is a reasonable alternative. (Class IIa, LOE C-LD)

4.5.3 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[104-106] and when the patient is moved from one location to another.[107,108] 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[109] and 2 studies in an intensive-care setting[110,111] 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.

4.5.4 Ventilation After Advanced Airway Placement - Updated ALS 808

The 2015 ILCOR systematic review addressed the optimal ventilation rate during continuous chest compressions among adults in cardiac arrest with an advanced airway. The 2015 Guidelines Update for ACLS applies only to patients who have been intubated and are in cardiac arrest. The effect of tidal volume and any other ventilation

parameters during CPR are not addressed in this recommendation.

Except for respiratory rate, it is unknown whether monitoring ventilatory parameters (eg, minute ventilation, peak pressure) during CPR can 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 inappropriately high respiratory rates (greater than 25 breaths/ min) is common during resuscitation from cardiac arrest.[112,113] There is concern that excessive ventilation in the setting of cardiac arrest may be associated with worse outcome.

4.5.4.1 2015 Evidence Summary

No human clinical trials were found addressing whether a ventilation rate of 10 breaths/min, compared with any other ventilation rate, changes survival with favorable neurologic or functional outcome. Although there have been a number of animal studies[112,114-122] and 1 human observational study[123] evaluating ventilation rates during CPR, the design and data from these studies did not allow for the assessment of the effect of a ventilation rate of 10 per minute compared with any other rate for ROSC or other outcomes.

4.5.4.2 2015 Recommendation - Updated

After placement of an advanced airway, it may be reasonable for the provider to deliver 1 breath every 6 seconds (10 breaths/min) while continuous chest compressions are being performed.
(Class IIb, LOE C-LD)

4.5.5 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.[124,125](Class IIb, LOE C)

Providers should always have a bag-mask device available for backup.

4.6 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.

5 Management of Cardiac Arrest - Updated

5.1 Overview

This section details the general care of a patient in cardiac arrest and provides an overview of the ACLS Adult Cardiac Arrest Algorithms (Figure 1 and Figure 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.

Figure 1: Adult Cardiac Arrest Algorithm?2015 Update

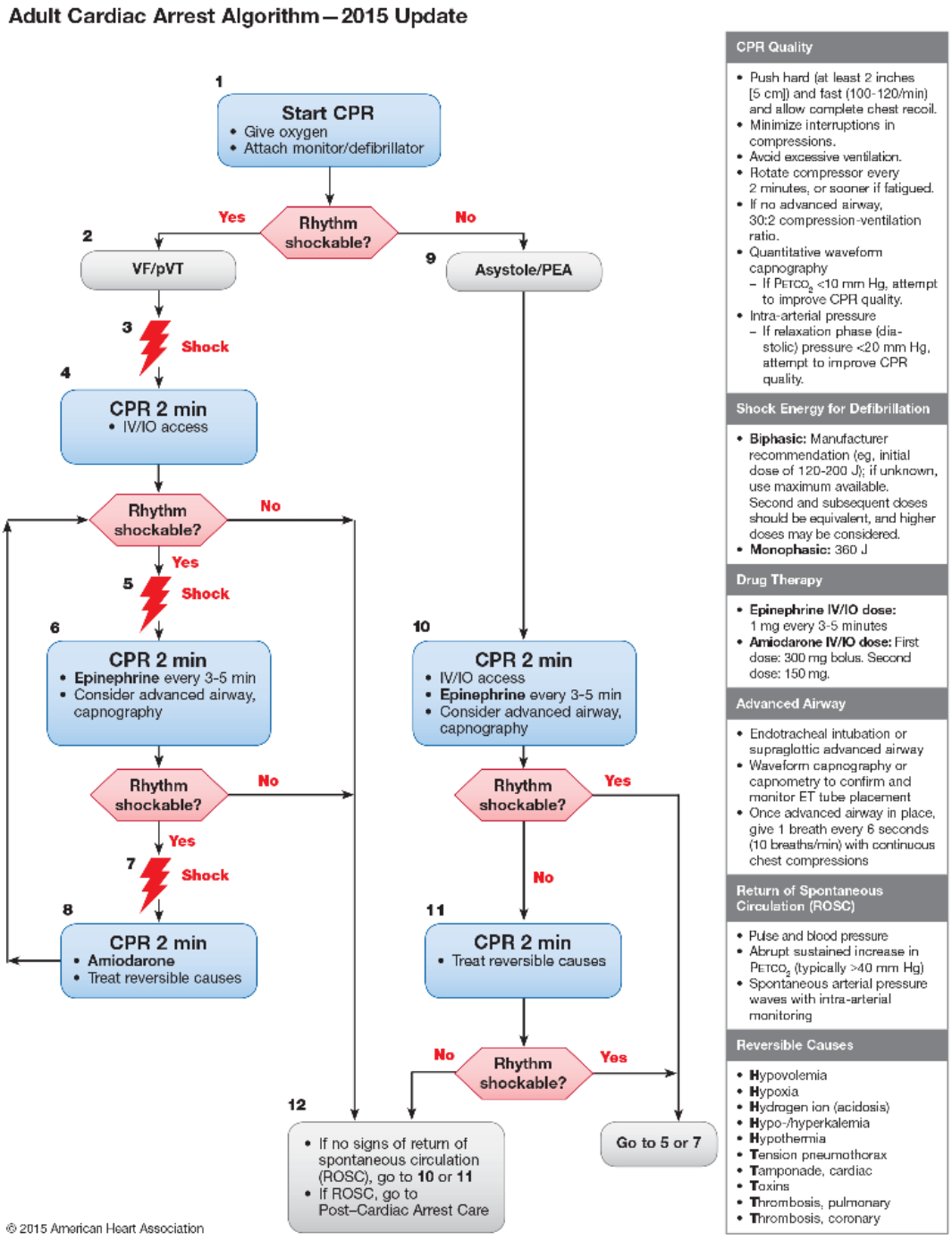
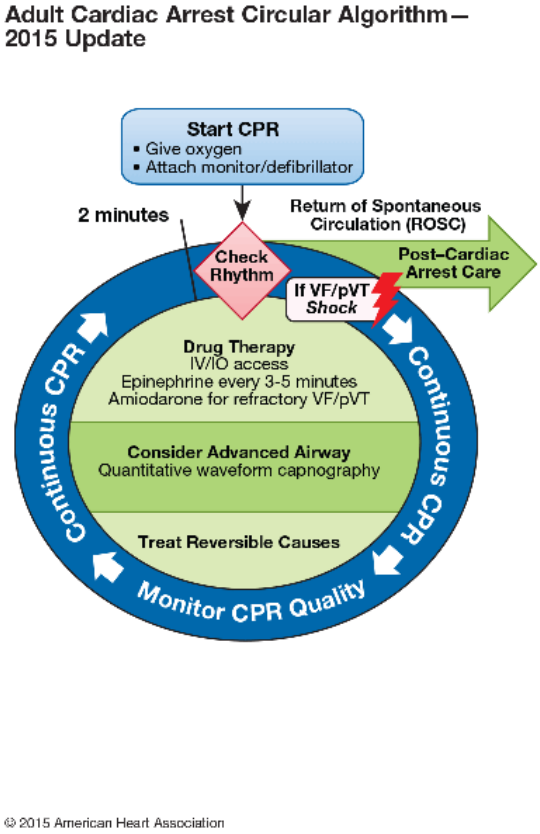


Figure 2: ACLS Cardiac Arrest Circular Algorithm - 2015 Update



CPR Quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Rotate compressor every 2 minutes, or sooner if fatigued.
- 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.

Shock Energy for Defibrillation

- **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
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

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

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

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.[126-131] 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.[76,78,132-136]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 8: 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 ACLS Adult Cardiac Arrest Algorithms (Figure 1) 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 [$PETCO_2$], arterial pressure during the relaxation phase of chest compressions, or central venous oxygen saturation [$ScvO_2$]) 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 seconds (10 breaths per minute) and should be particularly careful to avoid delivering an excessive number of ventilations.

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 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).

Table 1: 2010 - Treatable Causes of Cardiac Arrest: The H's and T's

Open table in a new window

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

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

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 8: Post–Cardiac Arrest Care”). Finally, the reality is that the majority of resuscitative efforts do not result in ROSC. Criteria for ending unsuccessful resuscitative efforts are addressed in *Part 3: Ethical Issues*.

5.2 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, 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.

5.2.1 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)**.

5.2.1.1 Defibrillation Strategies for Ventricular Fibrillation or Pulseless Ventricular Tachycardia: Waveform Energy and First-Shock Success ALS 470

Currently manufactured manual and automated external defibrillators use biphasic waveforms of 3 different designs: biphasic truncated exponential (BTE), rectilinear biphasic (RLB), and pulsed biphasic waveforms; they deliver different peak currents at the same programmed energy setting and may adjust their energy output in relation to patient impedance in differing ways. These factors can make comparisons of shock efficacy between devices from different manufacturers challenging even when the same programmed energy setting is used. A substantial body of evidence now exists for the efficacy of BTE and RLB waveforms, with a smaller body of evidence for the pulsed waveform. An impedance-compensated version of the pulsed biphasic waveform is now clinically available, but no clinical studies were identified to define its performance characteristics.

5.2.1.1.1 2015 Evidence Summary

There is no evidence indicating superiority of one biphasic waveform or energy level for the termination of ventricular fibrillation (VF) with the first shock (termination is defined as absence of VF at 5 seconds after shock). All published studies support the effectiveness (consistently in the range of 85%–98%)[137] of biphasic shocks using 200 J or less for the first shock.[137] Defibrillators using the RLB waveform typically deliver more shock energy than selected, based on patient impedance. Thus, in the single study in which a manufacturer's nonescalating energy device was programmed to deliver 150 J shocks, comparison with other devices was not possible because shock energy delivery in other devices is adjusted for measured patient impedance. For the RLB, a selected energy dose of 120 J typically provides nearly 150 J for most patients.

5.2.1.1.2 2015 Recommendations - Updated

Defibrillators (using BTE, RLB, or monophasic waveforms) are recommended to treat atrial and ventricular arrhythmias. (Class I, LOE B-NR)

Based on their greater success in arrhythmia termination, defibrillators using biphasic waveforms (BTE or RLB) are preferred to monophasic defibrillators for treatment of both atrial and ventricular arrhythmias. (Class IIa, LOE B-R)

In the absence of conclusive evidence that 1 biphasic waveform is superior to another in termination of VF, it is reasonable to use the manufacturer's recommended energy dose for the first shock. If this is not known, defibrillation at the maximal dose may be considered. (Class IIb, LOE C-LD)

5.2.1.2 Defibrillation Strategies for Ventricular Fibrillation or Pulseless Ventricular Tachycardia: Energy Dose for Subsequent Shocks

The 2010 Guidelines regarding treatment of VF/pulseless ventricular tachycardia (pVT) recommended that if the first shock dose did not terminate VF/pVT, the second and subsequent doses should be equivalent, and higher doses may be considered. The evidence supporting energy dose for subsequent shocks was evaluated for the 2015 Guidelines Update.

5.2.1.2.1 2015 Evidence Summary

Observational data indicate that an automated external defibrillator administering a high peak current at 150 J biphasic fixed energy can terminate initial, as well as persistent or recurrent VF, with a high rate of conversion.[138] In fact, the high conversion rate achieved with all biphasic waveforms for the first shock makes it difficult to study the energy requirements for second and subsequent shocks when the first shock is not successful. A 2007 study attempted to determine if a fixed lower energy dose or escalating higher doses were associated with better outcome in patients requiring more than 1 shock. Although termination of VF at 5 seconds after shock was higher in the escalating higher-energy group (82.5% versus 71.2%), there were no significant differences in ROSC, survival to discharge, or survival with favorable neurologic outcome between the 2 groups. In this study, only 1 manufacturer's nonescalating energy device, programmed to deliver 150-J shocks, was used. Thus, it is not possible to compare this 150-J shock with that delivered by any other device set to deliver 150 J.

There is a decline in shock success with repeated shocks. One nonrandomized trial that used a BTE waveform reported a decline in shock success when repeated shocks at the same energy were administered.[139] For the

RLB waveform, 1 observational study reported an initial VF termination rate of 87.8% at a selected energy setting of 120 J and an 86.4% termination rate for persistent VF. Recurrence of VF did not affect ultimate shock success, ROSC, or discharge survival.[140]

5.2.1.2.2 2015 Recommendations - Updated

It is reasonable that selection of fixed versus escalating energy for subsequent shocks be based on the specific manufacturer's instructions. (Class IIa, LOE C-LD)

If using a manual defibrillator capable of escalating energies, higher energy for second and subsequent shocks may be considered. (Class IIb, LOE C-LD)

5.2.1.3 Defibrillation Strategies for Ventricular Fibrillation or Pulseless Ventricular Tachycardia: Single Shocks Versus Stacked Shocks

The 2010 Guidelines recommended a 2-minute period of CPR after each shock instead of immediate successive shocks for persistent VF. The rationale for this is at least 3-fold: First, VF is terminated with a very high rate of success with biphasic waveforms. Second, when VF is terminated, a brief period of asystole or pulseless electrical activity (PEA) typically ensues and a perfusing rhythm is unlikely to be present immediately. Third, this provides for a period of uninterrupted CPR following a shock before repeat rhythm analysis. The evidence for single versus stacked shocks was reviewed again in 2015.

5.2.1.3.1 2015 Evidence Summary

One RCT that comprised 845 OHCA patients found no difference in 1-year survival when a single shock protocol with 2 minutes of CPR between successive shocks was compared against a previous resuscitation protocol employing 3 initial stacked shocks with 1 minute of CPR between subsequent shocks (odds ratio, 1.64; 95% confidence interval, 0.53– 5.06).[141] An RCT published in 2010 showed no difference in frequency of VF recurrence when comparing the 2 treatment protocols.[142] In that study, increased time in recurrent VF was associated with decreased favorable neurologic survival under either protocol.

There is evidence that resumption of chest compressions immediately after a shock can induce recurrent VF, but the benefit of CPR in providing myocardial blood flow is thought to outweigh the benefit of immediate defibrillation for the VF.[143] Another study of patients presenting in VF after a witnessed arrest concluded that recurrence of VF within 30 seconds of a shock was not affected by the timing of resumption of chest compressions.[144] Thus, the effect of chest compressions on recurrent VF is not clear. It is likely that in the future, algorithms that recognize recurrent VF during chest compressions with high sensitivity and specificity will allow us to deliver a shock earlier in the CPR cycle, thereby reducing the length of time the myocardium is fibrillating and the duration of postshock CPR.[145]

5.2.1.3.2 2015 Recommendation - Updated

A single-shock strategy (as opposed to stacked shocks) is reasonable for defibrillation. (Class IIa, LOE B-NR)

5.2.1.4 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.[146,147]

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)

5.2.1.5 CPR Before Defibrillation

This topic now covered in Part 5: Adult Basic Life Support and Cardiopulmonary Resuscitation Quality.

5.2.1.6 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.[148,149-168] 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)

5.2.2 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 8: Post–Cardiac Arrest Care). 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).

5.2.2.1 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 10: Special Circumstances of Resuscitation”). 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)

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.[169] See “Part 10: Special Circumstances of Resuscitation” 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.

5.2.2.2 ROSC After PEA/Asystole

If the patient has ROSC, post–cardiac arrest care should be initiated (see Part 8: Post–Cardiac Arrest Care). 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)

5.3 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 [136] 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 [135] 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.

5.3.1 Ventricular Fibrillation (VF) or Pulseless Ventricular Tachycardia (pVT)

5.3.1.1 Treating Potentially Reversible Causes of VF/pVT

The importance of diagnosing and treating the underlying cause of VF/pVT 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 10: Special Circumstances of Resuscitation"). 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.[170-179] Fibrinolytic therapy administered during CPR for acute coronary occlusion has not been shown to improve outcome.[180]

5.3.1.2 ROSC After VF/pVT

If the patient has ROSC, post–cardiac arrest care should be started. 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)

5.3.2 Antiarrhythmic Drugs During and Immediately After Cardiac Arrest - Updated ALS 428

The 2015 ILCOR systematic review addressed whether the administration of antiarrhythmic drugs for cardiac arrest due to refractory VF or pVT results in better outcome.

5.3.2.1 Antiarrhythmic Drugs During and Immediately After Cardiac Arrest: Antiarrhythmic Therapy for Refractory VF/pVT Arrest - Updated

Refractory VF/pVT refers to VF or pVT that persists or recurs after 1 or more shocks. It is unlikely that an antiarrhythmic drug will itself pharmacologically convert VF/pVT to an organized perfusing rhythm. Rather, the principal objective of antiarrhythmic drug therapy in shock-refractory VF/pVT is to facilitate the restoration and maintenance of a spontaneous perfusing rhythm in concert with the shock termination of VF. Some antiarrhythmic drugs have been associated with increased rates of ROSC and hospital admission, but none have yet been proven to increase long-term survival or survival with good neurologic outcome. Thus, establishing vascular access to enable drug administration should not compromise the quality of CPR or timely defibrillation, which are known to improve survival. The optimal sequence of ACLS interventions, including administration of antiarrhythmic drugs during resuscitation and the preferred manner and timing of drug administration in relation to shock delivery, is not known. Previous ACLS guidelines addressed the use of magnesium in cardiac arrest with polymorphic ventricular tachycardia (ie, torsades de pointes) or suspected hypomagnesemia, and this has

not been reevaluated in the 2015 Guidelines Update. These previous guidelines recommended defibrillation for termination of polymorphic VT (ie, torsades de pointes), followed by consideration of intravenous magnesium sulfate when secondary to a long QT interval.

The 2015 ILCOR systematic review did not specifically address the selection or use of second-line antiarrhythmic medications in patients who are unresponsive to a maximum therapeutic dose of the first administered drug, and there are limited data available to direct such treatment.

5.3.2.1.1 2015 Evidence Summary

5.3.2.1.1.1 Amiodarone - Updated

Intravenous amiodarone is available in 2 approved formulations in the United States, one containing polysorbate 80, a vasoactive solvent that can provoke hypotension, and one containing captisol, which has no vasoactive effects. In blinded RCTs in adults with refractory VF/pVT in the out-of-hospital setting, paramedic administration of amiodarone in polysorbate (300 mg or 5 mg/kg) after at least 3 failed shocks and administration of epinephrine improved hospital admission rates when compared to placebo with polysorbate[181] or 1.5 mg/kg lidocaine with polysorbate.[182] Survival to hospital discharge and survival with favorable neurologic outcome, however, was not improved by amiodarone compared with placebo or amiodarone compared with lidocaine, although these studies were not powered for survival or favorable neurologic outcome.

5.3.2.1.1.2 Lidocaine - Updated

Intravenous lidocaine is an alternative antiarrhythmic drug of long-standing and widespread familiarity. Compared with no antiarrhythmic drug treatment, lidocaine did not consistently increase ROSC and was not associated with improvement in survival to hospital discharge in observational studies.[183,184] In a prospective, blinded, randomized clinical trial, lidocaine was less effective than amiodarone in improving hospital admission rates after OHCA due to shock-refractory VF/pVT, but there were no differences between the 2 drugs in survival to hospital discharge.[182]

5.3.2.1.1.3 Procainamide - Updated

Procainamide is available only as a parenteral formulation in the United States. In conscious patients, procainamide can be given only as a controlled infusion (20 mg/min) because of its hypotensive effects and risk of QT prolongation, making it difficult to use during cardiac arrest. Procainamide was recently studied as a second-tier antiarrhythmic agent in patients with OHCA due to VF/pVT that was refractory to lidocaine and epinephrine. In this study, the drug was given as a rapid infusion of 500 mg (repeated as needed up to 17 mg/kg) during ongoing CPR. An unadjusted analysis showed lower rates of hospital admission and survival among the 176 procainamide recipients as compared with 489 nonrecipients. After adjustment for patients' clinical and resuscitation characteristics, no association was found between use of the drug and hospital admission or survival to hospital discharge, although a modest survival benefit from the drug could not be excluded.[185]

5.3.2.1.1.4 Magnesium - Updated

Magnesium acts as a vasodilator and is an important cofactor in regulating sodium, potassium, and calcium flow across cell membranes. In 3 randomized clinical trials, magnesium was not found to increase rates of ROSC for cardiac arrest due to any presenting rhythm,[186] including VF/pVT.[187,188] In these RCTs and in 1 additional randomized clinical trial, the use of magnesium in cardiac arrest for any rhythm presentation of cardiac arrest[186,189] or strictly for VF arrest[187,188] did not improve survival to hospital discharge or neurologic outcome.[189]

5.3.2.1.2 2015 Recommendations - Unchanged

Amiodarone may be considered for VF/pVT that is unresponsive to CPR, defibrillation, and a vasopressor therapy. (Class IIb, LOE B-R)

Lidocaine may be considered as an alternative to amiodarone for VF/pVT that is unresponsive to CPR, defibrillation, and vasopressor therapy. (Class IIb, LOE C-LD)

The routine use of magnesium for VF/pVT is not recommended in adult patients. (Class III: No Benefit, LOE B-R)

No antiarrhythmic drug has yet been shown to increase survival or neurologic outcome after cardiac arrest due to VF/pVT. Accordingly, recommendations for the use of antiarrhythmic medications in cardiac arrest are based primarily on the potential for benefit on short-term outcome until more definitive studies are performed to address their effect on survival and neurologic outcome.

5.3.2.2 Antiarrhythmic Drugs During and Immediately After Cardiac Arrest: Antiarrhythmic Drugs After Resuscitation - Updated ALS 493

The 2015 ILCOR systematic review addressed whether, after successful termination of VF or pVT cardiac arrest, the prophylactic administration of antiarrhythmic drugs for cardiac arrest results in better outcome. The only medications studied in this context are ?-adrenergic blocking drugs and lidocaine, and the evidence for their use is limited.

5.3.2.2.1 2015 Evidence Summary

5.3.2.2.1.1 ?-Adrenergic Blocking Drugs - Updated

?-Adrenergic blocking drugs blunt heightened catecholamine activity that can precipitate cardiac arrhythmias. The drugs also reduce ischemic injury and may have membrane-stabilizing effects. In 1 observational study of oral or intravenous metoprolol or bisoprolol during hospitalization after cardiac arrest due to VF/pVT, recipients had a significantly higher adjusted survival rate than nonrecipients at 72 hours after ROSC and at 6 months.[190] Conversely, ?-blockers can cause or worsen hemodynamic instability, exacerbate heart failure, and cause bradyarrhythmias, making their routine adminis- tration after cardiac arrest potentially hazardous. There is no evidence addressing the use of ?-blockers after cardiac arrest precipitated by rhythms other than VF/pVT.

5.3.2.2.1.2 Lidocaine - Updated

Early studies in patients with acute myocardial infarction found that lidocaine suppressed premature ventricular complexes and nonsustained VT, rhythms that were believed to presage VF/pVT. Later studies noted a disconcerting association between lidocaine and higher mortality after acute myocardial infarction, possibly due to a higher incidence of asystole and bradyarrhythmias; the routine practice of administering prophylactic lidocaine during acute myocardial infarction was abandoned.[191,192] The use of lidocaine was explored in a multivariate and propensity score–adjusted analysis of patients resuscitated from out-of-hospital VF/pVT arrest. In this observational study of 1721 patients, multivariate analysis found the prophylactic administration of lidocaine before hospitalization was associated with a significantly lower rate of recurrent VF/ pVT and higher rates of hospital admission and survival to hospital discharge. However, in a propensity score–adjusted analysis, rates of hospital admission and survival to hospital discharge did not differ between recipients of prophylactic lidocaine as compared with nonrecipients, although lidocaine was associated with less recurrent VF/pVT and there was no evidence of harm.[193] Thus, evidence supporting a role for prophylactic lidocaine after VF/pVT arrest is weak at best, and nonexistent for cardiac arrest initiated by other rhythms.

5.3.2.2.2 Recommendations - Updated

There is inadequate evidence to support the routine use of lidocaine after cardiac arrest. However, the initiation or continuation of lidocaine may be considered immediately after ROSC from cardiac arrest due to VF/pVT. (Class IIb, LOE C-LD)

There is inadequate evidence to support the routine use of a ?-blocker after cardiac arrest. However, the initiation or continuation of an oral or intravenous ?-blocker may be considered early after hospitalization from cardiac arrest due to VF/pVT. (Class IIb, LOE C-LD)

Available evidence suggests that the routine use of atropine during PEA or asystole is unlikely to have a

therapeutic benefit. (Class IIb, LOE B)

There is insufficient evidence to recommend for or against the routine initiation or continuation of other antiarrhythmic medications after ROSC from cardiac arrest.

5.3.3 Vasopressors in Cardiac Arrest - Updated

The 2015 ILCOR systematic review addresses the use of the vasopressors epinephrine and vasopressin during cardiac arrest. The new recommendations in this 2015 Guidelines Update apply only to the use of these vasopressors for this purpose.

In 2010 it was noted that, 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.

5.3.3.1 Vasopressors in Cardiac Arrest: Standard-Dose Epinephrine - Updated ALS 788

Epinephrine produces beneficial effects in patients during cardiac arrest, primarily because of its ?-adrenergic (ie, vasoconstrictor) effects. These ?-adrenergic effects of epinephrine can increase coronary perfusion pressure and cerebral perfusion pressure during CPR. The value and safety of the ?-adrenergic effects of epinephrine are controversial because they may increase myocardial work and reduce subendocardial perfusion. The 2010 Guidelines stated that it is reasonable to consider administering a 1-mg dose of IV/IO epinephrine every 3 to 5 minutes during adult cardiac arrest.

5.3.3.1.1 2015 Evidence Summary

One trial[194] assessed short-term and longer-term outcomes when comparing standard-dose epinephrine to placebo. Standard-dose epinephrine was defined as 1 mg given IV/ IO every 3 to 5 minutes. For both survival to discharge and survival to discharge with good neurologic outcome, there was no benefit with standard-dose epinephrine; however, the study was stopped early and was therefore underpowered for analysis of either of these outcomes (enrolled approximately 500 patients as opposed to the target of 5000). There was, nevertheless, improved survival to hospital admission and improved ROSC with the use of standard-dose epinephrine. Observational studies were performed that evaluated epinephrine, with conflicting results.[195,196]

5.3.3.1.2 2015 Recommendation - Updated

Standard-dose epinephrine (1 mg every 3 to 5 minutes) may be reasonable for patients in cardiac arrest. (Class IIb, LOE B-R)

5.3.3.2 Vasopressors in Cardiac Arrest: Standard Dose Epinephrine Versus High-Dose Epinephrine - Updated ALS 778 ALS 778

High doses of epinephrine are generally defined as doses in the range of 0.1 to 0.2 mg/kg. In theory, higher doses of epinephrine may increase coronary perfusion pressure, resulting in increased ROSC and survival from cardiac arrest. However, the adverse effects of higher doses of epinephrine in the postarrest period may negate potential advantages during the intraarrest period. Multiple case series followed by randomized trials have been performed to evaluate the potential benefit of higher doses of epinephrine. In the 2010 Guidelines, the use of high-dose epinephrine was not recommended except in special circumstances, such as for ?-blocker overdose, calcium channel blocker overdose, or when titrated to real-time physiologically monitored parameters. In 2015, ILCOR evaluated the use of high-dose epinephrine compared with standard doses.

5.3.3.2.1 2015 Evidence Summary

A number of trials have compared outcomes from standard-dose epinephrine with those of high-dose epinephrine. These trials did not demonstrate any benefit for high-dose epinephrine over standard-dose epinephrine for survival to discharge with a good neurologic recovery (ie, Cerebral Performance Category score),[197,198] survival to discharge,[197-201] or survival to hospital admission.[197-199,202] There was, however, a demonstrated ROSC advantage with highdose epinephrine.[197-202]

5.3.3.2.2 2015 Recommendation—New

High-dose epinephrine is not recommended for routine use in cardiac arrest.
(Class III: No Benefit, LOE B-R)

5.3.3.3 Vasopressors in Cardiac Arrest: Epinephrine Versus Vasopressin - Updated ALS 659

Vasopressin is a nonadrenergic peripheral vasoconstrictor that also causes coronary[203,204] and renal vasoconstriction.[205]

5.3.3.3.1 2015 Evidence Summary

A single RCT[206] enrolling 336 patients compared multiple doses of standard-dose epinephrine with multiple doses of standard dose vasopressin (40 units IV) in the emergency department after OHCA. The trial had a number of limitations but showed no benefit with the use of vasopressin for ROSC or survival to discharge with or without good neurologic outcome.

5.3.3.3.2 2015 Recommendation—Updated

Vasopressin offers no advantage as a substitute for epinephrine in cardiac arrest. (Class IIb, LOE B-R)

The removal of vasopressin has been noted in the Adult Cardiac Arrest Algorithm above (Figure 1).

5.3.3.4 Vasopressors in Cardiac Arrest: Epinephrine Versus Vasopressin in Combination With Epinephrine - Updated ALS 789

5.3.3.4.1 2015 Evidence Summary

A number of trials have compared outcomes from standard dose epinephrine to those using the combination of epinephrine and vasopressin. These trials showed no benefit with the use of the epinephrine/vasopressin combination for survival to hospital discharge with Cerebral Performance Category score of 1 or 2 in 2402 patients,[207-209] no benefit for survival to hospital discharge or hospital admission in 2438 patients,[207-211] and no benefit for ROSC.[207-212]

5.3.3.4.2 2015 Recommendation—New

Vasopressin in combination with epinephrine offers no advantage as a substitute for standard-dose epinephrine in cardiac arrest. (Class IIb, LOE B-R)

The removal of vasopressin has been noted in the Adult Cardiac Arrest Algorithm above (Figure 1).

5.3.3.5 Vasopressors in Cardiac Arrest: Timing of Administration of Epinephrine - Updated ALS 784

5.3.3.5.1 2015 Evidence Summary: IHCA

One large (n=25 905 patients) observational study of IHCA with nonshockable rhythms was identified,[213] in which outcomes from early administration of epinephrine (1 to 3 minutes) were compared with outcomes from administration of epinephrine at 4 to 6 minutes, 7 to 9 minutes, and greater than 9 minutes. In this study, the early administration of epinephrine in nonshockable rhythms was associated with increased ROSC, survival to hospital discharge, and neurologically intact survival. No studies were identified specifically examining the effect of timing of administration of epinephrine after IHCA with shockable rhythms.

5.3.3.5.2 2015 Evidence Summary: OHCA

For nonshockable rhythms, 3 studies showed improved survival to hospital discharge with early administration of epinephrine. A study of 209 577 OHCA patients[214] showed improved 1-month survival when outcomes from administration of epinephrine at less than 9 minutes of EMS-initiated CPR were compared with those in which epinephrine was administered at greater than 10 minutes. Another study enrolling 212 228 OHCA patients[215] showed improved survival to discharge with early epinephrine (less than 10 minutes after EMS-initiated CPR) compared with no epinephrine. A smaller study of 686 OHCA patients[216] showed improved rates of ROSC with early epinephrine (less than 10 minutes after 9-1-1 call) when the presenting rhythm was pulseless electrical activity. For shockable rhythms, there was no benefit with early administration of epinephrine, but there was a negative association of outcome with late administration. When neurologically intact survival to discharge was assessed,[214,215,217] however, there was variable benefit with early administration of epinephrine for both shockable and nonshockable rhythms. Later administration of epinephrine was associated with a worse outcome. ROSC was generally improved with early administration of epinephrine in studies of more than 210 000 patients.[201,214,216,218] Design flaws in the majority of these observational OHCA studies, however, included the use of a “no epinephrine” control arm as the comparator (thus not allowing for estimates on the effect of timing), and the lack of known timing of epinephrine administration upon arrival in the emergency department. In addition, the relationship of timing of defibrillation to timing of epinephrine is unknown for studies that included shockable rhythms.

5.3.3.5.3 2015 Recommendations—Updated

It may be reasonable to administer epinephrine as soon as feasible after the onset of cardiac arrest due to an initial non- shockable rhythm. (Class IIb, LOE C-LD)

There is insufficient evidence to make a recommendation as to the optimal timing of epinephrine, particularly in relation to defibrillation, when cardiac arrest is due to a shockable rhythm, because optimal timing may vary based on patient factors and resuscitation conditions.

5.3.4 Steroids - Updated ALS 433

The use of steroids in cardiac arrest has been assessed in 2 clinical settings: IHCA and OHCA. In IHCA, steroids were combined with a vasopressor bundle or cocktail of epinephrine and vasopressin. Because the results of IHCA and OHCA were so different, these situations are discussed separately.

5.3.4.1 2015 Evidence Summary: IHCA

In an initial RCT involving 100 IHCA patients at a single center, the use of a combination of methylprednisolone, vasopressin, and epinephrine during cardiac arrest and hydrocortisone after ROSC for those with shock significantly improved survival to hospital discharge compared with the use of only epinephrine and placebo.[219] In a subsequent 3-center study published in 2013,[219] of 268 patients with IHCA (the majority coming from the same center as in the first study), the same combination of methylprednisolone, vasopressin, and epinephrine during cardiac arrest, and hydrocortisone for those with post-ROSC shock, significantly improved survival to discharge with good neurologic outcome compared with only epinephrine and placebo.

The same 2 RCTs provided evidence that the use of methylprednisolone and vasopressin in addition to epinephrine improved ROSC compared with the use of placebo and epinephrine alone.[219,220]

5.3.4.2 2015 Evidence Summary: OHCA

In OHCA, steroids have been evaluated in 1 RCT[221] and 1 observational study.[222] In these studies, steroids were not bundled as they were in the IHCA but studied as a sole treatment. When dexamethasone was given

during cardiac arrest, it did not improve survival to hospital discharge or ROSC as compared with placebo.[221] The observational study[222] showed no benefit in survival to discharge but did show an association of improved ROSC with hydrocortisone compared with no hydrocortisone.

5.3.4.3 2015 Recommendations—New

There are no data to recommend for or against the routine use of steroids alone for IHCA patients.

In IHCA, the combination of intra-arrest vasopressin, epinephrine, and methylprednisolone and post-arrest hydrocortisone as described by Mentzelopoulos et al [220] may be considered; however, further studies are needed before recommending the routine use of this therapeutic strategy.

(Class IIb, LOE C-LD)

For patients with OHCA, use of steroids during CPR is of uncertain benefit. (Class IIb, LOE C-LD)

5.4 Access for Parenteral Medications During Cardiac Arrest

5.4.1 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[132,134] 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[134] the interval from first shock to administration of an antiarrhythmic drug was a significant predictor of survival. One animal study[223] 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.[224] 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.

5.4.2 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.[225] 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.

5.4.3 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[226] and adults[227] and 6 other studies[228-234] 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.

5.4.4 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.[235-237] In addition, a central line extending into the superior vena cava can be used to monitor ScvO2 and estimate CPP during CPR, both of which are predictive of ROSC.[238,239] 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.

5.4.5 Endotracheal Drug Delivery

One study in children,[240] 5 studies in adults,[241-245] and multiple animal studies[246-248] have shown that lidocaine,[242,249] epinephrine,[250] atropine,[251] naloxone, and vasopressin[248] 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[252,253] suggest that the lower epinephrine concentrations achieved when the drug is delivered endotracheally may produce transient ?-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[254] 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.[255,256] 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.[250] Studies with epinephrine[257] and lidocaine[245] showed that dilution with sterile water instead of 0.9% saline may achieve better drug absorption.

5.5 Prognostication During CPR:End-Tidal CO2 - Updated ALS 459 ALS 459

The 2015 ILCOR systematic review considered one intraarrest modality, $ETCO_2$ measurement, in prognosticating outcome from cardiac arrest. This section focuses on whether a specific $ETCO_2$ threshold can reliably predict ROSC and survival or inform a decision to terminate resuscitation efforts. The potential value of using $ETCO_2$ as a physiologic monitor to optimize resuscitation efforts is discussed elsewhere (See Monitoring Physiologic Parameters During CPR, earlier in this Part).

$ETCO_2$ is the partial pressure of exhaled carbon dioxide at the end of expiration and is determined by CO_2 production, alveolar ventilation, and pulmonary blood flow. It is most reliably measured using waveform capnography, where the visualization of the actual CO_2 waveform during ventilation ensures accuracy of the measurement. During low-flow states with relatively fixed minute ventilation, pulmonary blood flow is the primary determinant of $ETCO_2$. During cardiac arrest, $ETCO_2$ levels reflect the cardiac output generated by chest compression. Low $ETCO_2$ values may reflect inadequate cardiac output, but $ETCO_2$ levels can also be low as a result of bronchospasm, mucous plugging of the ETT, kinking of the ETT, alveolar fluid in the ETT, hyperventilation, sampling of an SGA, or an airway with an air leak. It is particularly important to recognize that all of the prognostication studies reviewed in this section included only intubated patients. In nonintubated patients (those with bag-mask ventilation or SGA), $ETCO_2$ may not consistently reflect the true value, making the measurement less reliable as a prognostication tool.

5.5.1 2015 Evidence Summary

Studies on the predictive capacity of $ETCO^2$ among intubated patients during cardiac arrest resuscitation are observational, and none have investigated survival with intact neurologic outcome. An $ETCO_2$ less than 10 mmHg immediately after intubation and 20 minutes after the initial resuscitation is associated with extremely poor chances for ROSC and survival.[8,12,15,18,258]

A prospective observational study of 127 IHCA patients found that an $ETCO_2$ less than 10 mmHg at any point during the resuscitation was predictive of mortality, and only 1 patient with an $ETCO_2$ value less than 10 mmHg survived to discharge.[258] In that same study, an $ETCO_2$ greater than 20 mmHg after 20 minutes of resuscitation was associated with improved survival to discharge.[258] Another prospective observational study of 150 OHCA patients reported no survival to hospital admission when the $ETCO_2$ was less than 10 mmHg after 20 minutes of resuscitation.[8] Although these results suggest that $ETCO_2$ can be a valuable tool to predict futility during CPR, potential confounding reasons for a low $ETCO_2$ as listed above and the relatively small numbers of patients in these studies suggest that the $ETCO_2$ should not be used alone as an indication to terminate resuscitative efforts. However, the failure to achieve an $ETCO_2$ greater than 10 mmHg despite optimized resuscitation efforts may be a valuable component of a multimodal approach to deciding when to terminate resuscitation.

There are no studies that assess the prognostic value of $ETCO_2$ measurements sampled from an SGA or bag-mask airway in predicting outcomes from a cardiac arrest.

5.5.2 2015 Recommendations—New

In intubated patients, failure to achieve an ETCO2 of greater than 10 mm Hg by waveform capnography after 20 minutes of CPR may be considered as one component of a multimodal approach to decide when to end resuscitative efforts, but it should not be used in isolation. (Class IIb, LOE C-LD)

The above recommendation is made with respect to $ETCO_2$ in patients who are intubated, because the studies examined included only those who were intubated.

In nonintubated patients, a specific ETCO2 cutoff value at any time during CPR should not be used as an indication to end resuscitative efforts. (Class III: Harm, LOE C-EO)

5.6 Overview of Extracorporeal CPR - Updated ALS 723

The 2015 ILCOR systematic review compared the use of ECPR (or ECMO) techniques for adult patients with IHCA and OHCA to conventional (manual or mechanical) CPR, in regard to ROSC, survival, and good neurologic outcome. The recommendations in this update apply only to the use of ECPR in this context.

ECPR refers to venoarterial extracorporeal membrane oxygenation during cardiac arrest, including extracorporeal membrane oxygenation and cardiopulmonary bypass. These techniques require adequate vascular access and specialized equipment. The use of ECPR may allow providers additional time to treat reversible underlying causes of cardiac arrest (eg, acute coronary artery occlusion, pulmonary embolism, refractory VF, profound hypothermia, cardiac injury, myocarditis, cardiomyopathy, congestive heart failure, drug intoxication etc) or serve as a bridge for left ventricular assist device implantation or cardiac transplantation.

5.6.1 2015 Evidence Summary

All of the literature reviewed in the 2015 ILCOR systematic review comparing ECPR to conventional CPR was in the form of reviews, case reports, and observational studies. The low-quality evidence suggests a benefit in regard to survival and favorable neurologic outcome with the use of ECPR when compared with conventional CPR. There are currently no data from RCTs to support the use of ECPR for cardiac arrest in any setting.

One propensity-matched prospective observational study enrolling 172 patients with IHCA reported greater likelihood of ROSC and improved survival at hospital discharge, 30-day follow-up, and 1-year follow-up with the use of ECPR among patients who received more than 10 minutes of CPR. However, this study showed no difference in neurologic outcomes.[259]

A single retrospective, observational study enrolling 120 patients with witnessed IHCA who underwent more than 10 minutes of CPR reported a modest benefit over historic controls with the use of ECPR over continued

conventional CPR in both survival and neurologic outcome at discharge and 6-month follow-up.[260]

A single propensity-matched, retrospective, observational study enrolling 118 patients with IHCA who underwent more than 10 minutes of CPR and then ECPR after cardiac arrest of cardiac origin showed no survival or neurologic benefit over conventional CPR at the time of hospital discharge, 30-day follow-up, or 1-year follow-up.[261]

One post hoc analysis of data from a prospective, observational cohort of 162 patients with OHCA who did not achieve ROSC with more than 20 minutes of conventional CPR, including propensity score matching, showed that ECPR was associated with a higher rate of neurologically intact survival than continued conventional CPR at 3-month follow-up.[262]

A single prospective, observational study enrolling 454 patients with OHCA who were treated with ECPR if they did not achieve ROSC with more than 15 minutes of conventional CPR after hospital arrival demonstrated improved neurologic outcomes at 1-month and 6-month follow-up.[263]

The key articles reviewed in the 2015 ILCOR systematic review comparing ECPR to conventional CPR feature some variability in their inclusion and exclusion criteria (Table 2), which may affect the generalizability of their results and could explain some of the inconsistencies in outcomes between studies.

Table 2: 2015 - Inclusion and Exclusion Criteria for Key Extracorporeal CPR Articles

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Inclusion and Exclusion Criteria for Key Extracorporeal CPR Articles

Study	CA Type	Inclusion Criteria	Exclusion Criteria
Chen, 2008[reference id="1291" range="" /]	IHCA	Witnessed CA of cardiac origin (elevated cardiac enzymes before CA, sudden collapse without obvious cause, or sudden collapse with pre-existing cardiovascular disease)	Age less than 18 years or greater than 75 years Known severe irreversible brain damage Terminal malignancy
		No ROSC during first 10 minutes of conventional CPR	Traumatic origin with uncontrolled bleeding Postcardiotomy shock with inability to be weaned from cardiopulmonary bypass

Study	CA Type	Inclusion Criteria	Exclusion Criteria
Shin, 2011[reference id="1292" range="" /]	IHCA	Witnessed CA of cardiac origin No ROSC during first 10 minutes of conventional CPR	Age less than 18 years or greater than 80 years No sustained (20 minutes or more) ROSC during first 10 minutes of conventional CPR Known severe neurologic damage Current intracranial hemorrhage Terminal malignancy Traumatic origin with uncontrolled bleeding Noncardiac origin* (submersion, drug overdose, asphyxia, exsanguination, sepsis) Irreversible organ failure (liver failure, late stage of adult respiratory distress syndrome, etc)
Lin, 2010[reference id="1293" range="" /]	IHCA	• Witnessed CA of cardiac origin • No ROSC during first 10 minutes of conventional CPR	Age less than 18 years or greater than 75 years Known severe irreversible brain damage Terminal malignancy Severe trauma Uncontrolled bleeding
Maekawa, 2013[reference id="1294" range="" /]	OHCA	Witnessed CA of presumed cardiac origin	Age less than 16 years Terminal malignancy
		No ROSC during first 20 minutes of conventional CPR	Poor level of activities of daily living before onset of CA Noncardiac origin (trauma, submersion, hypothermia, drug overdose, asphyxia, exsanguination, intracranial hemorrhage, acute aortic dissection)

Study	CA Type	Inclusion Criteria	Exclusion Criteria
Sakamoto, 2014[reference id="1295" range="" /]	OHCA	VF/pVT on initial ECG CA of presumed cardiac origin on hospital arrival with or without prehospital ROSC Arrival to hospital 45 minutes or less after reception of emergency call or onset of CA No ROSC (1 minute or more of continuing confirmation of pulsation) during first 15 minutes of conventional CPR in hospital	Age less than 20 years or 75 years or older Poor level of activities of daily living before onset of CA Noncardiac origin (trauma, drug intoxication, primary cerebral disorders, acute aortic dissection, terminal malignancy) Core body temperature less than 30°C

CA indicates cardiac arrest; CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; IHCA, in-hospital cardiac arrest; OHCA, out-of-hospital cardiac arrest; pVT, pulseless ventricular tachycardia; ROSC, return of spontaneous circulation; and VF, ventricular fibrillation.*Postcardiotomy bleeding considered to be of cardiac origin.

5.6.2 2015 Recommendation—New

There is insufficient evidence to recommend the routine use of ECPR for patients with cardiac arrest. In settings where it can be rapidly implemented, ECPR may be considered for select cardiac arrest patients for whom the suspected etiology of the cardiac arrest is potentially reversible during a limited period of mechanical cardiorespiratory support. (Class IIb, LOE C-LD)

5.7 Interventions Not Recommended for Routine Use During Cardiac Arrest

5.7.1 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.[79,264-273] 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)

5.7.2 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 [274,275] increased ROSC, hospital admission, and survival to hospital discharge associated with use of bicarbonate. However, the majority of studies showed no benefit [276-278] or found a relationship with poor outcome.[273,279-281]

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.[282] 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.[283] 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 10: Special Circumstances of Resuscitation).

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.[284-286] But clinical experience is greatly limited and outcome studies are lacking.

5.7.3 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.[270,273,287-292]

Routine administration of calcium for treatment of in-hospital and out-of-hospital cardiac arrest is not recommended. (Class III, LOE B)

5.7.4 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 [293-299] and suggested benefit from fibrinolytic therapy in the treatment of victims of cardiopulmonary arrest unresponsive to standard therapy. But 2 large clinical trials [180,300] 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.[180]

Fibrinolytic therapy should not be routinely used in cardiac arrest. (Class III, LOE B)

5.7.5 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,[301-312] and 2 animal studies showed that normothermic fluid infusion during CPR caused a decrease in CPP.[313-315] In addition to normothermic fluid, hypertonic and chilled fluids have been studied in animal and small human studies without a survival benefit.[301,303,305-307,310-312] 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.

5.8 Pacing

Electric pacing is generally not effective in cardiac arrest, and no studies have observed a survival benefit from pacing in cardiac arrest.[316-319] 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)

5.9 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.[320] 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.[321] In 3 case series[322-324] 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.[323,325-330]

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.

5.10 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: Ethical Issues.

5.11 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.

6 Management of Symptomatic Bradycardia and Tachycardia

6.1 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*.[331] 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 ?-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 3).

Table 3: 2010 - IV Drugs Used for Tachycardia

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IV Drugs Used for Tachycardia					
Drug	**Characteristics**	**Indication(s)**	**Dosing**	**Side Effects**	**Precautions or Special Considerations**
Intravenous Drugs Used to Treat Supraventricular Tachyarrhythmias					

Drug	Characteristics	Indication(s)	Dosing	Side Effects	Precautions or Special Considerations
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 cardioversio • Stable, regular, monomorphi 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

Drug	Characteristics	Indication(s)	Dosing	Side Effects	Precautions or Special Considerations
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

Drug	Characteristics	Indication(s)	Dosing	Side Effects	Precautions or Special Considerations
Atenolol, Esmolol, Metoprolol, Propranolol	?-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, catecholami	Atenolol (?1 specific blocker) 5 mg IV over 5 minutes; repeat 5 mg in 10 minutes if arrhythmia persists or recurs Esmolol (?1 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 (?1 specific blocker) 5 mg over 1 to 2 minutes repeated as required every 5 minutes to maximum dose of 15 mg Propranolol (nonselective ?-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

Drug	Characteristics	Indication(s)	Dosing	Side Effects	Precautions or Special Considerations
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
Amiodarone	Multichannel blocker (sodium, potassium, calcium channel, and noncompetitive ?/?-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	

Drug	Characteristics	Indication(s)	Dosing	Side Effects	Precautions or Special Considerations
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

Drug	Characteristics	Indication(s)	Dosing	Side Effects	Precautions or Special Considerations
Amiodarone	Multichannel blocker (sodium, potassium, calcium channel, ?- and noncompetitive ?-blocker)	• Hemodynam stable monomorphi 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 ?-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	• Hemodynam stable monomorphi 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	

Drug	Characteristics	Indication(s)	Dosing	Side Effects	Precautions or Special Considerations
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

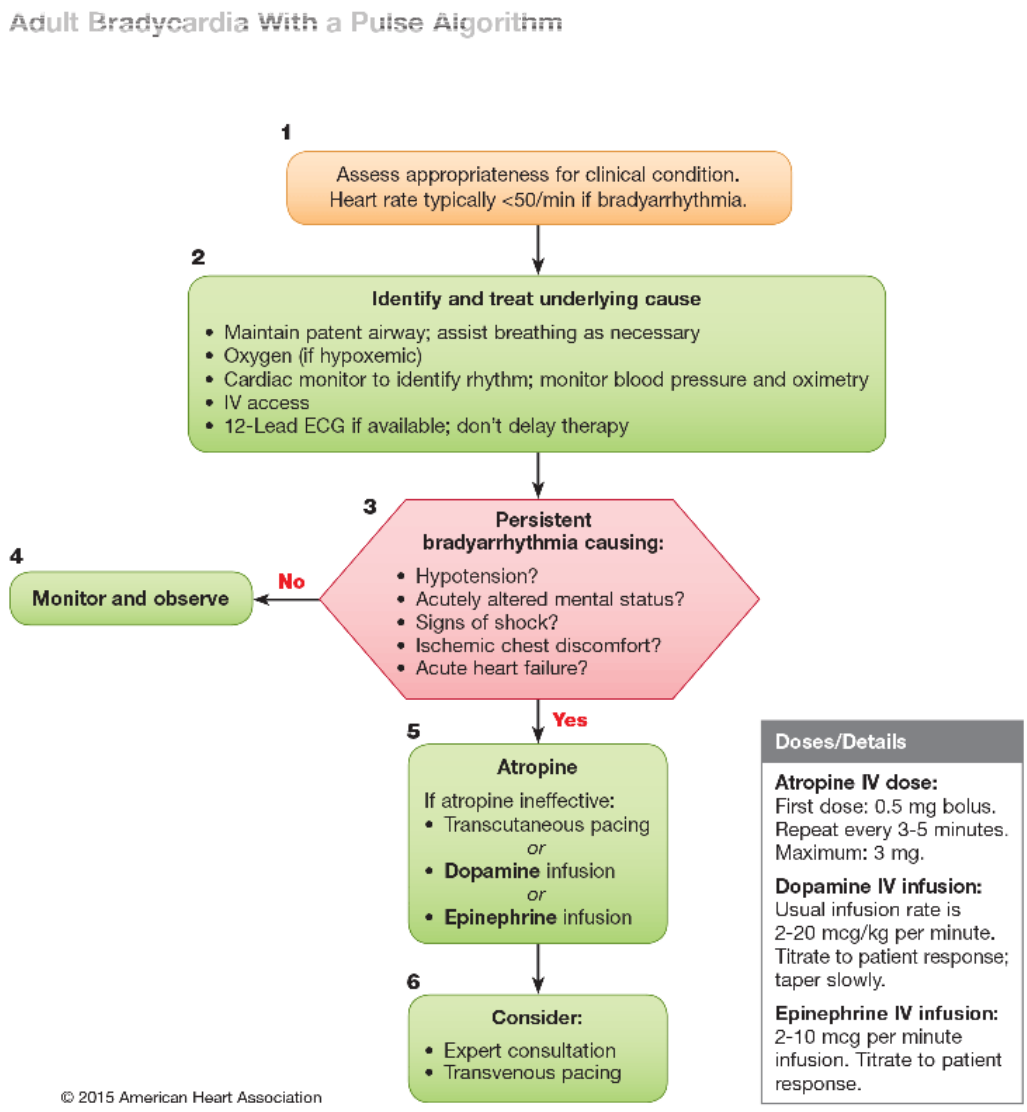
Know when to call for expert consultation regarding complicated rhythm interpretation, drugs, or management decisions.

A comprehensive presentation of the evaluation and management of bradyarrhythmias and tachyarrhythmias is beyond the scope of this document. 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 above (Figure 1,Figure 2).

6.1.1 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.

Figure 3: Adult Bradycardia With a Pulse Algorithm



6.1.1.1 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: Bradycardia Algorithm, Box 1). A slow heart rate may be physiologically normal for some patients, whereas a heart rate of >50 beats per minut1e 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 (**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 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 (**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 (**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.

6.1.1.2 Therapy (Figure 3 Box 5)

6.1.1.2.1 Atropine

Atropine remains the first-line drug for acute symptomatic bradycardia. (Class IIa, LOE B)

Clinical trials in adults[332-336] 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.[336]

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.[337] 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.[338]

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 ?-adrenergic support as temporizing measures while the patient is prepared for transvenous pacing (Figure 3, **Box 6**).

6.1.1.2.2 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.[332] 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.[339] 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 (Figure 3, Box 6). (Class IIa, LOE C)

6.1.1.2.3 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 ?-blocker or calcium channel blocker.

6.1.1.2.3.1 Dopamine

Dopamine hydrochloride is a catecholamine with both ?- and ?-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.[339] Use of vasoconstrictors requires that the recipient be assessed for adequate intravascular volume and volume status supported as needed.

6.1.1.2.3.2 Epinephrine

Epinephrine is a catecholamine with ?- and ?-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.

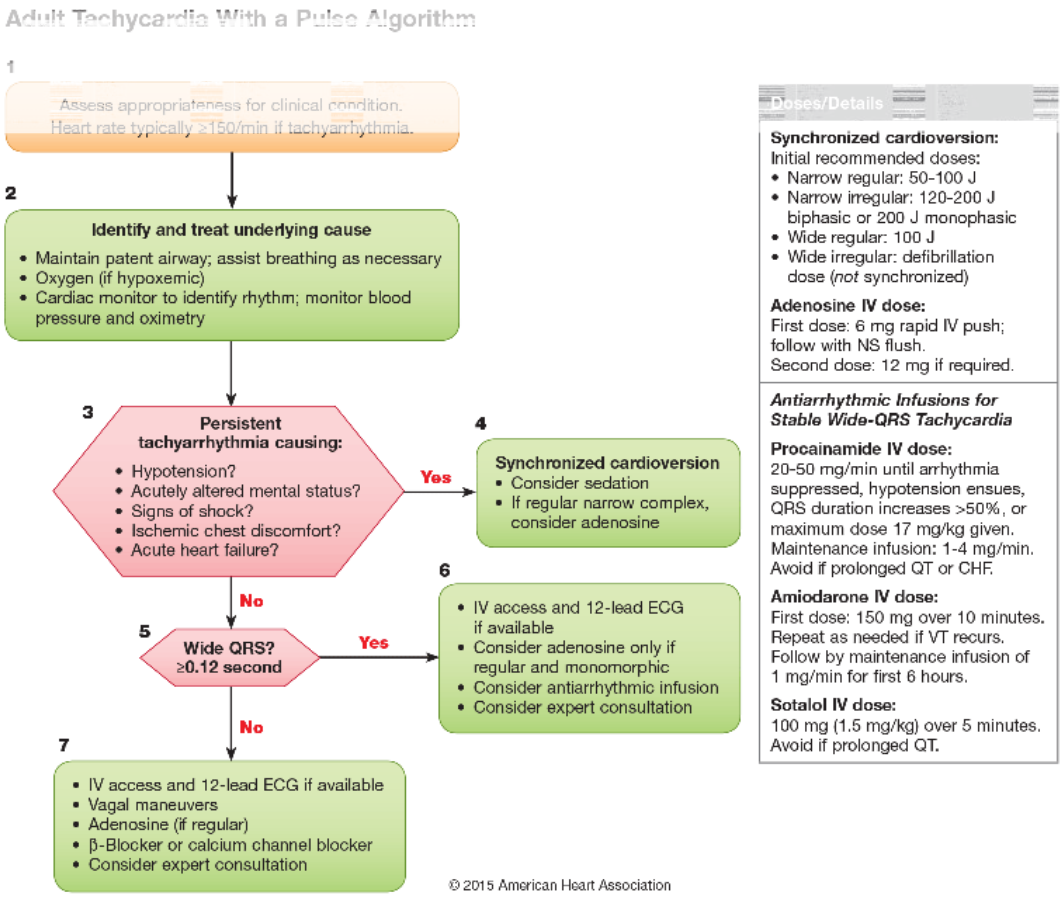
6.1.1.2.3.3 Isoproterenol

Isoproterenol is a ?-adrenergic agent with ?-1 and ?-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.

6.2 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.

Figure 4: Adult Tachycardia With a Pulse Algorithm



6.2.1 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.

6.2.2 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 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: Tachycardia Algorithm, **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: Tachycardia Algorithm). 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 this document above under 4.1: “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 (**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 (**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 synchronized cardioversion (**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 (**Box 5**), and determine treatment options. Stable patients may await expert consultation because treatment has the potential for harm.

6.2.3 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.

6.2.3.1 Synchronized Cardioversion and Unsynchronized Shocks

Refer to Figure 4: Tachycardia Algorithm – **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.[340] 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.

6.2.3.2 Waveform and Energy

The recommended initial biphasic energy dose for cardioversion of atrial fibrillation is 120 to 200 J.[341-345] (Class IIa, LOE A)

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.[345] If the initial 50-J shock fails, the provider should increase the dose in a stepwise fashion.[346]

Cardioversion of atrial fabrillation with monophasic waveforms should begin at 200 J and increase in stepwise fashion if not successful.[341-343] (Class IIa, LOE B)

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 Algorithms in this document above under 4.1: “Management of Cardiac Arrest.”

6.2.4 Regular Narrow-Complex Tachycardia

6.2.4.1 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.

6.2.4.2 Supraventricular Tachycardia (Reentry SVT)

6.2.4.2.1 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.

6.2.4.2.2 Therapy

6.2.4.2.2.1 Vagal Maneuvers

Vagal maneuvers and adenosine are the preferred initial therapeutic choices for the termination of stable PSVT (Figure 4: Tachycardia Algorithm, **Box 7**). Vagal maneuvers alone (Valsalva maneuver or carotid sinus massage) will terminate up to 25% of PSVTs.[347-349] 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.

6.2.4.2.2.2 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[350-367] support the use of adenosine in the treatment of stable PSVT. Although 2 randomized clinical trials[352,355] 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,[368] and the potential proarrhythmic risks of these agents favor the use of safer treatment alternatives.

Adenosine is safe and effective in pregnancy.[369] 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.[370] 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 ?-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.

6.2.4.2.2.3 Calcium Channel Blockers and ?-Blockers

If adenosine or vagal maneuvers fail to convert PSVT, 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 ?-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[350,352,353,355,363, 367,371-374] and diltiazem[371,375,376] 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 ?-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 ?-blockers can include bradycardias, AV conduction delays, and hypotension. ?-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, ?-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 ?-blocker. Conversely the longer half-life of a calcium channel or ?-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.

6.2.5 Wide-Complex Tachycardia

6.2.5.1 Evaluation

The first step in the management of any tachycardia is to determine if the patient's condition is stable or unstable (Figure 4: Tachycardia Algorithm, **Box 3**). An unstable patient with a wide-complex tachycardia should be presumed to have VT and immediate cardioversion should be performed (**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 (**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 ?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.

6.2.5.2 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.[377] (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[378-382] showed that adenosine converted an undifferentiated wide-complex tachycardia to sinus rhythm. Another study [383] 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,[384] conversion to VF in 4 patients with pre-excited atrial fibrillation,[385] conversion to VF in 2 patients with WPW,[386] and a single case of VF in a patient with VT.[387]

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.[384-388] Profound hypotension was reported in 11 of 25 patients known to have VT treated with verapamil.[388]

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, or sotalol can be considered. (Class IIb, LOE B)

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.[389] 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.[390]

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.[391] 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[392,393-395] 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[393,395] 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,[392,389,390] and when given to patients with or without a history of MI with spontaneous sustained stable VT in the hospital setting.[383,396,397] Lidocaine has been reported to variably terminate VT when administered intramuscularly to patients with AMI and VT in the out-of-hospital setting.[398,399] 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).

6.3 Irregular Tachycardias

6.3.1 Atrial Fibrillation and Flutter

6.3.1.1 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.

6.3.1.2 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.[400]

6.3.1.3 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 ?-blockers and nondihydropyridine calcium channel blockers such as diltiazem[401-404] are the drugs of choice for acute rate control in most individuals with atrial fibrillation and rapid ventricular response. (Class IIa, LOE A)

Digoxin[405-407] and amiodarone[408,409] 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 ?-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.

6.3.1.4 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.

6.3.2 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[410,411] that showed effectiveness in patients with prolonged QT interval. One adult case series[412] 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 ?-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 ?-blockers may reduce the frequency of arrhythmia recurrence. (Class IIb, LOE C)

Myocardial ischemia should be treated with ?-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,[410] but amiodarone may be effective.[413] (Class IIb, LOE C)

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

6.4 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.

7 Authorship and Disclosures

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Table 4: Part 7: Adult Advanced Cardiovascular Life Support: 2015 Guidelines Update Writing Group Disclosures

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Part 7: Adult Advanced Cardiovascular Life Support: 2015 Guidelines Update Writing Group Disclosures

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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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Table 5: 2010 - Guidelines Part 7: CPR Techniques and Devices: Writing Group Disclosures

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- ?* Modest.
- ?† Significant.

8 Footnotes

The American Heart Association requests that this document be cited as follows:

American Heart Association. Web-based Integrated Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care – Part 7: Adult Advanced Cardiovascular Life Support. ECCguidelines.heart.org

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