Part 7: Adult Advanced Cardiovascular Life Support

2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

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Introduction

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*[1] and was completed in February 2015.[2]

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,[3] 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.[4]

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 this 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,

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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.

Adjuncts to CPR

Oxygen Dose During CPR[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.

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 Po_2 measured during CPR and cardiac arrest outcomes.[5] In this study, during which all patients received maximal inspired oxygen concentration, patients were divided into low, intermediate, and high arterial Po_2 ranges (less than 61, 61–300, and greater than 300 mm Hg, respectively). The higher ranges of arterial Po_2 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.

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.

Monitoring Physiologic Parameters During CPR[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.[6] 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.

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 CO_2 ($ETCO_2$), 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.[7–13] These parameters can be monitored continuously, without interrupting chest compressions. An abrupt increase in any of these parameters is a sensitive indicator of ROSC.[14–31] There is evidence that these and other physiologic parameters can be modified by interventions aimed at improving CPR quality.[7,32–43]

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.

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.

Ultrasound During Cardiac Arrest[ALS 658]

Bedside cardiac and noncardiac ultrasound are frequently used as diagnostic and prognostic tools for critically ill patients.[44] 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.[45] However, it is unclear whether important clinical outcomes are affected by the routine use of ultrasound among patients experiencing cardiac arrest.

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.[46]

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).

Adjuncts for Airway Control and Ventilation

This portion of the 2015 Guidelines Update focuses on recommendations for airway management based on rate of survival and favorable neurologic outcome.

Bag-Mask Ventilation Compared With Any Advanced Airway During CPR[ALS 783]

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 bag-mask ventilation versus advanced airway placement on overall survival and neurologic outcome from cardiac arrest.

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[47–53] or other advanced airway devices.[47,49–51,54] 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[54,55] showed no difference in survival.

Advanced Airway Placement Choice

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.

2015 Evidence Summary

Endotracheal Intubation Versus Bag-Mask Ventilation

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.[47–53] 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.

Supraglottic Airway Devices

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 bag-mask ventilation[47,49–51] or endotracheal intubation.[47,49,50,54,56–61] Three observational studies demonstrated a lower rate of both overall survival and favorable neurologic outcome when SGA use was compared with bag-mask ventilation,[47,49,51] whereas another observational study demonstrated similar survival rates.[50]

In studies comparing SGA insertion to endotracheal intubation, no high-quality studies have demonstrated a difference in overall survival or favorable neurologic outcome.[50,54,56–58,61] Several retrospective observational studies show more favorable outcome with the use of an SGA device, whereas other studies favor the use of endotracheal intubation.[47,49,50,59–61]

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.

Clinical Assessment of Tracheal Tube Placement[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, CO_2 detection devices, esophageal detector device, tracheal ultrasound, fiberoptic bronchoscopy) have been proposed to confirm successful tracheal intubation in adults during cardiac arrest.

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.[62–64] Although the sensitivity of waveform capnography for detecting tracheal tube placement immediately after prehospital intubation was 100% in 1 study,[62] several other studies showed that the sensitivity of waveform capnography decreases after a prolonged cardiac arrest.[63–65] Differences in sensitivity can be explained by the low pulmonary blood flow during cardiac arrest, which will decrease $ETCO_2$ concentration.

Although exhaled CO_2 detection suggests correct tracheal tube placement, false-positive results (CO_2 detection with esophageal intubation) can occur after ingestion of carbonated liquids.[66] False-negative results (ie, absent exhaled CO_2 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.[15,67,68] The use of CO_2-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 CO_2 detectors can identify the presence of exhaled CO_2 from the respiratory tract, but there is no evidence that these devices are accurate for continued monitoring of ETT placement.[15,62,69–73] Moreover, because a minimal threshold of CO_2 must be reached to activate the detector and exhaled CO_2 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.[63–65,69,74,75]

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 CO_2 in exhaled gas.[76–78] 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%.[78] The usefulness of tracheal and pleural ultrasonography, like fiberoptic bronchoscopy, may be limited by abnormal anatomy, availability of equipment, and operator experience.

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 CO_2 detector, esophageal detector device, or ultrasound used by an experienced operator is a reasonable alternative (Class IIa, LOE C-LD).

Ventilation After Advanced Airway Placement[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. This 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.[79,80] There is concern that excessive ventilation in the setting of cardiac arrest may be associated with worse outcome.

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[79,81–89] and 1 human observational study[90] 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.

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).

Management of Cardiac Arrest

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.

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%)[91] of biphasic shocks using 200 J or less for the first shock.[91] 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.

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).

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 this 2015 Guidelines Update.

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.[92] 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.[93] 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.[94]

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).

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.

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).[95] An RCT published in 2010 showed no difference in frequency of VF recurrence when comparing the 2 treatment protocols.[96] 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.[97] 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.[98] 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.[99]

2015 Recommendation—Updated

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

Antiarrhythmic Drugs During and Immediately After Cardiac Arrest[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.

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

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 this 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.

2015 Evidence Summary

Amiodarone

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[100] or 1.5 mg/kg lidocaine with polysorbate.[101] 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.

Lidocaine

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.[102,103] 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.[101]

Procainamide

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.[104]

Magnesium

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,[105] including VF/pVT.[106,107] In these RCTs and in 1 additional randomized clinical trial, the use of magnesium in cardiac arrest for any rhythm presentation of cardiac arrest[105,108] or strictly for VF arrest[106,107] did not improve survival to hospital discharge or neurologic outcome.[108]

2015 Recommendations—Updated

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.

Antiarrhythmic Drugs During and Immediately After Cardiac Arrest: Antiarrhythmic Drugs After Resuscitation[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 β-adrenergic blocking drugs and lidocaine, and the evidence for their use is limited.

2015 Evidence Summary

β-Adrenergic Blocking Drugs

β-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.[109] Conversely, β-blockers can cause or worsen hemodynamic instability, exacerbate heart failure, and cause bradyarrhythmias, making their routine administration after cardiac arrest potentially hazardous. There is no evidence addressing the use of β-blockers after cardiac arrest precipitated by rhythms other than VF/pVT.

Lidocaine

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.[110,111] 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.[112] 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.

2015 Recommendations—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 (Class IIb, LOE C-LD).

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

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

Vasopressors in Cardiac Arrest

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.

Vasopressors in Cardiac Arrest: Standard-Dose Epinephrine[ALS 788]

Epinephrine produces beneficial effects in patients during cardiac arrest, primarily because of its α-adrenergic (ie, vasoconstrictor) effects. These α-adrenergic effects of epinephrine can increase coronary perfusion pressure and cerebral perfusion pressure during CPR. The value and safety of the β-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.

2015 Evidence Summary

One trial[113] 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.[114,115]

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).

Vasopressors in Cardiac Arrest: Standard Dose Epinephrine Versus High-Dose Epinephrine[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 intra-arrest 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 β-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.

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),[116,117] survival to discharge,[116–120] or survival to hospital admission.[116–118,121] There was, however, a demonstrated ROSC advantage with high-dose epinephrine.[116–121]

2015 Recommendation—New

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

Vasopressors in Cardiac Arrest: Epinephrine Versus Vasopressin[ALS 659]

Vasopressin is a nonadrenergic peripheral vasoconstrictor that also causes coronary[122,123] and renal vasoconstriction.[124]

2015 Evidence Summary

A single RCT[125] 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.

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 (Figure 1).

Vasopressors in Cardiac Arrest: Epinephrine Versus Vasopressin in Combination With Epinephrine[ALS 789]

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,[126–128] no benefit for survival to hospital discharge or hospital admission in 2438 patients,[126–130] and no benefit for ROSC.[126–131]

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 (Figure 1).

Vasopressors in Cardiac Arrest: Timing of Administration of Epinephrine[ALS 784]

2015 Evidence Summary: IHCA

One large (n=25 905 patients) observational study of IHCA with nonshockable rhythms was identified,[132] 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.

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[133] 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[134] 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[135] 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

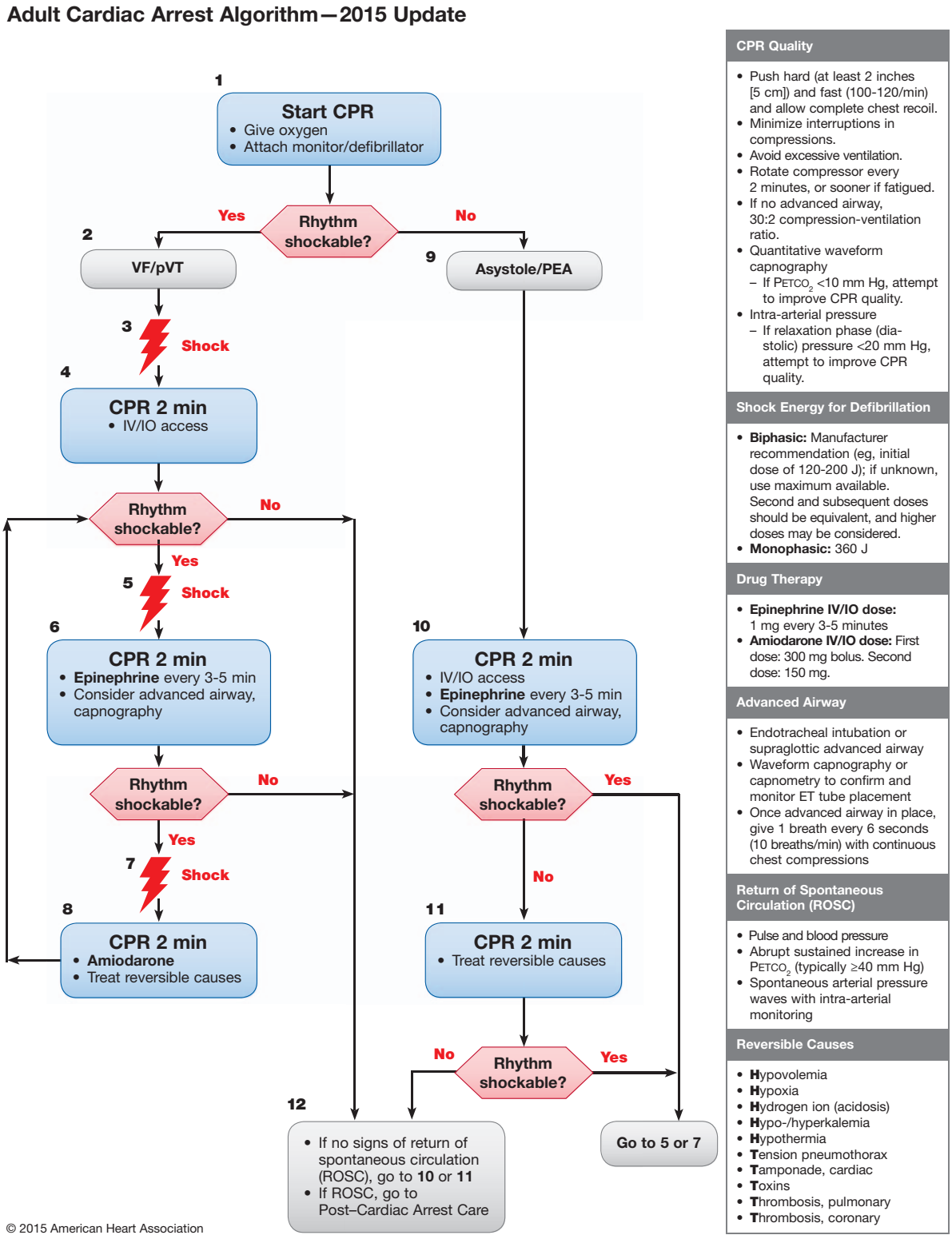

Figure 1. Adult Cardiac Arrest Algorithm—2015 Update.

with late administration. When neurologically intact survival to discharge was assessed,[133,134,136] 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.[120,133,135,137] 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.

2015 Recommendations—Updated

It may be reasonable to administer epinephrine as soon as feasible after the onset of cardiac arrest due to an initial nonshockable 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.

Steroids[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.

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.[138] In a subsequent 3-center study published in 2013,[138] 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.[138,139]

2015 Evidence Summary: OHCA

In OHCA, steroids have been evaluated in 1 RCT[140] and 1 observational study.[141] 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.[140] The observational study[141] showed no benefit in survival to discharge but did show an association of improved ROSC with hydrocortisone compared with no hydrocortisone.

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[139] 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).

Prognostication During CPR: End-Tidal CO_2[ALS 459]

The 2015 ILCOR systematic review considered one intra-arrest 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.

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 mm Hg immediately after intubation and 20 minutes after the initial resuscitation is associated with extremely poor chances for ROSC and survival.[9,13,16,19,142]

A prospective observational study of 127 IHCA patients found that an $ETCO_2$ less than 10 mm Hg at any point during the resuscitation was predictive of mortality, and only 1 patient with an $ETCO_2$ value less than 10 mm Hg survived to discharge.[142] In that same study, an $ETCO_2$ greater than 20 mm Hg after 20 minutes of resuscitation was associated with improved survival to discharge.[142] Another prospective observational study of 150 OHCA patients reported no survival to hospital admission when the $ETCO_2$ was less than 10 mm Hg after 20 minutes of resuscitation.[9] 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 mm Hg 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.

2015 Recommendations—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 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 $ETCO_2$ cutoff value at any time during CPR *should not* be used as an indication to end resuscitative efforts (Class III: Harm, LOE C-EO).

Overview of Extracorporeal CPR[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

Table 1. Inclusion and Exclusion Criteria for Key Extracorporeal CPR Articles

Study	CA Type	Inclusion Criteria	Exclusion Criteria
Chen, 2008[143]	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
Shin, 2011[144]	IHCA	Witnessed CA of cardiac origin	Age less than 18 years or greater than 80 years
		No 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[145]	IHCA	Witnessed CA of cardiac origin	Age less than 18 years or greater than 75 years
		No sustained (20 minutes or more) ROSC during first 10 minutes of conventional CPR	Known severe irreversible brain damage
			Terminal malignancy
			Severe trauma
			Uncontrolled bleeding
Maekawa, 2013[146]	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)
Sakamoto, 2014[147]	OHCA	VF/pVT on initial ECG	Age less than 20 years or 75 years or older
		CA of presumed cardiac origin on hospital arrival with or without prehospital ROSC	Poor level of activities of daily living before onset of CA
		Arrival to hospital 45 minutes or less after reception of emergency call or onset of CA	Noncardiac origin (trauma, drug intoxication, primary cerebral disorders, acute aortic dissection, terminal malignancy)
		No ROSC (1 minute or more of continuing confirmation of pulsation) during first 15 minutes of conventional CPR in hospital	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.

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.

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.[143]

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.[144]

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.[145]

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.[146]

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.[147]

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 1), which may affect the generalizability of their results and could explain some of the inconsistencies in outcomes between studies.

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).

Disclosures

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

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
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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.

Appendix

2015 Guidelines Update: Part 7 Recommendations

Year Last Reviewed	Topic	Recommendation	Comments
2015	Adjuncts to CPR	When supplementary oxygen is available, it may be reasonable to use the maximal feasible inspired oxygen concentration during CPR (Class IIb, LOE C-EO).	updated for 2015
2015	Adjuncts to CPR	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).	updated for 2015
2015	Adjuncts to CPR	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).	updated for 2015
2015	Adjuncts to CPR	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).	updated for 2015
2015	Adjuncts for Airway Control and Ventilation	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).	updated for 2015
2015	Adjuncts for Airway Control and Ventilation	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).	updated for 2015
2015	Adjuncts for Airway Control and Ventilation	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).	updated for 2015
2015	Adjuncts for Airway Control and Ventilation	If continuous waveform capnometry is not available, a nonwaveform CO_2 detector, esophageal detector device, or ultrasound used by an experienced operator is a reasonable alternative (Class IIa, LOE B-NR).	updated for 2015
2015	Adjuncts for Airway Control and Ventilation	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).	updated for 2015
2015	Management of Cardiac Arrest	Defibrillators (using BTE, RLB, or monophasic waveforms) are recommended to treat atrial and ventricular arrhythmias (Class I, LOE B-NR).	updated for 2015
2015	Management of Cardiac Arrest	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).	updated for 2015
2015	Management of Cardiac Arrest	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).	updated for 2015
2015	Management of Cardiac Arrest	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).	updated for 2015
2015	Management of Cardiac Arrest	If using a manual defibrillator capable of escalating energies, higher energy for second and subsequent shocks may be considered (Class IIb, LOE C-LD).	updated for 2015
2015	Management of Cardiac Arrest	A single-shock strategy (as opposed to stacked shocks) is reasonable for defibrillation (Class IIa, LOE B-NR).	updated for 2015
2015	Management of Cardiac Arrest	Amiodarone may be considered for VF/pVT that is unresponsive to CPR, defibrillation, and a vasopressor therapy (Class IIb, LOE B-R).	updated for 2015
2015	Management of Cardiac Arrest	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).	updated for 2015
2015	Management of Cardiac Arrest	The routine use of magnesium for VF/pVT is not recommended in adult patients (Class III: No Benefit, LOE B-R).	updated for 2015
2015	Management of Cardiac Arrest	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).	new for 2015
2015	Management of Cardiac Arrest	There is inadequate evidence to support the routine use of a β-blocker after cardiac arrest. However, the initiation or continuation of an oral or intravenous β-blocker may be considered early after hospitalization from cardiac arrest due to VF/pVT (Class IIb, LOE C-LD).	new for 2015

(*Continued*)

2015 Guidelines Update: Part 7 Recommendations, *Continued*

Year Last Reviewed	Topic	Recommendation	Comments
2015	Management of Cardiac Arrest	Standard-dose epinephrine (1 mg every 3 to 5 minutes) may be reasonable for patients in cardiac arrest (Class IIb, LOE B-R).	updated for 2015
2015	Management of Cardiac Arrest	High-dose epinephrine is not recommended for routine use in cardiac arrest (Class III: No Benefit, LOE B-R).	new for 2015
2015	Management of Cardiac Arrest	Vasopressin offers no advantage as a substitute for epinephrine in cardiac arrest (Class IIb, LOE B-R).	updated for 2015
2015	Management of Cardiac Arrest	Vasopressin in combination with epinephrine offers no advantage as a substitute for standard-dose epinephrine in cardiac arrest (Class IIb, LOE B-R).	new for 2015
2015	Management of Cardiac Arrest	It may be reasonable to administer epinephrine as soon as feasible after the onset of cardiac arrest due to an initial nonshockable rhythm (Class IIb, LOE C-LD).	updated for 2015
2015	Management of Cardiac Arrest	In IHCA, the combination of intra-arrest vasopressin, epinephrine, and methylprednisolone and post-arrest hydrocortisone as described by Mentzelopoulos et al may be considered; however, further studies are needed before recommending the routine use of this therapeutic strategy (Class IIb, LOE C-LD).	new for 2015
2015	Management of Cardiac Arrest	For patients with OHCA, use of steroids during CPR is of uncertain benefit (Class IIb, LOE C-LD).	new for 2015
2015	Management of Cardiac Arrest	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 (Class IIb, LOE C-LD).	new for 2015
2015	Management of Cardiac Arrest	In nonintubated patients, a specific $ETCO_2$ cutoff value at any time during CPR should not be used as an indication to end resuscitative efforts (Class III: Harm, LOE C-EO).	new for 2015
2015	Management of Cardiac Arrest	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).	new for 2015
The following recommendations were not reviewed in 2015. For more information, see the *2010 AHA Guidelines for CPR and ECC*, "Part 8: Adult Advanced Cardiovascular Life Support."			
2010	Cricoid Pressure	The routine use of cricoid pressure in cardiac arrest is not recommended (Class III, LOE C).	not reviewed in 2015
2010	Oropharyngeal Airways	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).	not reviewed in 2015
2010	Nasopharyngeal Airways	In the presence of known or suspected basal skull fracture or severe coagulopathy, an oral airway is preferred (Class IIa, LOE C).	not reviewed in 2015
2010	Postintubation Airway Management	The endotracheal tube should be secured with tape or a commercial device (Class I, LOE C).	not reviewed in 2015
2010	Postintubation Airway Management	One out-of-hospital study and 2 studies in an intensive care setting 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).	not reviewed in 2015
2010	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).	not reviewed in 2015
2010	Automatic Transport Ventilators	During prolonged resuscitative efforts the use of an ATV (pneumatically powered and time- or pressure-cycled) may allow the EMS team to perform other tasks while providing adequate ventilation and oxygenation (Class IIb, LOE C).	not reviewed in 2015
2010	Automatic Versus Manual Modes for Multimodal Defibrillators	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).	not reviewed in 2015
2010	CPR Before Defibrillation	Performing CPR while a defibrillator is readied for use is strongly recommended for all patients in cardiac arrest (Class I, LOE B).	not reviewed in 2015
2010	CPR Before Defibrillation	At this time the benefit of delaying defibrillation to perform CPR before defibrillation is unclear (Class IIb, LOE B).	not reviewed in 2015
2010	Drug Therapy for PEA/ Asystole	Available evidence suggests that the routine use of atropine during PEA or asystole is unlikely to have a therapeutic benefit (Class IIb, LOE B).	not reviewed in 2015

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2015 Guidelines Update: Part 7 Recommendations, *Continued*

Year Last Reviewed	Topic	Recommendation	Comments
2010	Coronary Perfusion Pressure and Arterial Relaxation Pressure	It is reasonable to consider using arterial relaxation "diastolic" pressure to monitor CPR quality, optimize chest compressions, and guide vasopressor therapy (Class IIb, LOE C).	not reviewed in 2015
2010	Coronary Perfusion Pressure and Arterial Relaxation Pressure	If the arterial relaxation "diastolic" pressure is <20 mm Hg, it is reasonable to consider trying to improve quality of CPR by optimizing chest compression parameters or giving a vasopressor or both (Class IIb, LOE C).	not reviewed in 2015
2010	Coronary Perfusion Pressure and Arterial Relaxation Pressure	Arterial pressure monitoring can also be used to detect ROSC during chest compressions or when a rhythm check reveals an organized rhythm (Class IIb, LOE C).	not reviewed in 2015
2010	Central Venous Oxygen Saturation	Therefore, when in place before cardiac arrest, it is reasonable to consider using continuous $Scvo_2$ measurement to monitor quality of CPR, optimize chest compressions, and detect ROSC during chest compressions or when rhythm check reveals an organized rhythm (Class IIb, LOE C).	not reviewed in 2015
2010	Central Venous Oxygen Saturation	If $Scvo_2$ is <30%, it is reasonable to consider trying to improve the quality of CPR by optimizing chest compression parameters (Class IIb, LOE C).	not reviewed in 2015
2010	Arterial Blood Gases	Routine measurement of arterial blood gases during CPR has uncertain value (Class IIb, LOE C).	not reviewed in 2015
2010	IO Drug Delivery	It is reasonable for providers to establish IO access if IV access is not readily available (Class IIa, LOE C).	not reviewed in 2015
2010	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).	not reviewed in 2015
2010	Endotracheal Drug Delivery	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).	not reviewed in 2015
2010	Atropine	Available evidence suggests that routine use of atropine during PEA or asystole is unlikely to have a therapeutic benefit (Class IIb, LOE B).	not reviewed in 2015
2010	Sodium Bicarbonate	Routine use of sodium bicarbonate is not recommended for patients in cardiac arrest (Class III, LOE B).	not reviewed in 2015
2010	Calcium	Routine administration of calcium for treatment of in-hospital and out-of-hospital cardiac arrest is not recommended (Class III, LOE B).	not reviewed in 2015
2010	Precordial Thump	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.	not reviewed in 2015
2010	Management of Symptomatic Bradycardia and Tachycardia	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).	not reviewed in 2015
2010	Management of Symptomatic Bradycardia and Tachycardia	If bradycardia is unresponsive to atropine, intravenous (IV) infusion of β-adrenergic agonists with rate-accelerating effects (dopamine, epinephrine) or transcutaneous pacing (TCP) can be effective (Class IIa, LOE B) while the patient is prepared for emergent transvenous temporary pacing if required.	not reviewed in 2015
2010	Management of Symptomatic Bradycardia and Tachycardia	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).	not reviewed in 2015
2010	Management of Symptomatic Bradycardia and Tachycardia	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).	not reviewed in 2015
2010	Atropine	Atropine remains the first-line drug for acute symptomatic bradycardia (Class IIa, LOE B).	not reviewed in 2015
2010	Pacing	It is reasonable for healthcare providers to initiate TCP in unstable patients who do not respond to atropine (Class IIa, LOE B).	not reviewed in 2015
2010	Pacing	Immediate pacing might be considered in unstable patients with high-degree AV block when IV access is not available (Class IIb, LOE C).	not reviewed in 2015
2010	Pacing	If the patient does not respond to drugs or TCP, transvenous pacing is probably indicated (Class IIa, LOE C).	not reviewed in 2015
2010	Dopamine	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).	not reviewed in 2015
2010	Wide-Complex Tachycardia - Evaluation	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).	not reviewed in 2015

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2015 Guidelines Update: Part 7 Recommendations, *Continued*

Year Last Reviewed	Topic	Recommendation	Comments
2010	Therapy for Regular Wide-Complex Tachycardias	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 (Class IIb, LOE B).	not reviewed in 2015
2010	Therapy for Regular Wide-Complex Tachycardias	Adenosine should not be given for unstable or for irregular or polymorphic wide-complex wide-complex tachycardias, as it may cause degeneration of the arrhythmia to VF (Class III, LOE C).	not reviewed in 2015
2010	Therapy for Regular Wide-Complex Tachycardias	Verapamil is contraindicated for wide-complex tachycardias unless known to be of supraventricular origin (Class III, LOE B).	not reviewed in 2015
2010	Therapy for Regular Wide-Complex Tachycardias	If IV antiarrhythmics are administered, procainamide (Class IIa, LOE B), amiodarone (Class IIb, LOE B), or sotalol (Class IIb, LOE B) can be considered.	not reviewed in 2015
2010	Therapy for Regular Wide-Complex Tachycardias	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).	not reviewed in 2015
2010	Therapy for Regular Wide-Complex Tachycardias	If antiarrhythmic therapy is unsuccessful, cardioversion or expert consultation should be considered (Class IIa, LOE C).	not reviewed in 2015
2010	Rate Control	IV β-blockers and nondihydropyridine calcium channel blockers such as diltiazem are the drugs of choice for acute rate control in most individuals with atrial fibrillation and rapid ventricular response (Class IIa, LOE A).	not reviewed in 2015
2010	Polymorphic (Irregular) VT	In the absence of a prolonged QT interval, the most common cause of polymorphic VT is myocardial ischemia. In this situation IV amiodarone and β-blockers may reduce the frequency of arrhythmia recurrence (Class IIb, LOE C).	not reviewed in 2015
2010	Polymorphic (Irregular) VT	Magnesium is unlikely to be effective in preventing polymorphic VT in patients with a normal QT interval (Class IIb, LOE C), but amiodarone may be effective (Class IIb, LOE C).	not reviewed in 2015
2010	Ventilation and Oxygen Administration During CPR	Advanced airway placement in cardiac arrest should not delay initial CPR and defibrillation for VF cardiac arrest (Class I, LOE C).	not reviewed in 2015
2010	Advanced Airways	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).	not reviewed in 2015
2010	Endotracheal Intubation	EMS systems that perform prehospital intubation should provide a program of ongoing quality improvement to minimize complications (Class IIa, LOE B).	not reviewed in 2015
2010	VF Waveform Analysis to Predict Defibrillation Success	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).	not reviewed in 2015
2010	Fibrinolysis	Fibrinolytic therapy should not be routinely used in cardiac arrest (Class III, LOE B).	not reviewed in 2015
2010	Pacing	Electric pacing is not recommended for routine use in cardiac arrest (Class III, LOE B).	not reviewed in 2015
2010	Epinephrine	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).	not reviewed in 2015
2010	Initial Evaluation and Treatment of Tachyarrhythmias	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).	not reviewed in 2015
2010	Therapy	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).	not reviewed in 2015
2010	Therapy	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 β-blockers (Class IIa, LOE C).	not reviewed in 2015
2010	Therapy	Therefore, AV nodal blocking drugs should not be used for pre-excited atrial fibrillation or flutter (Class III, LOE C).	not reviewed in 2015

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

Part 7: Adult Advanced Cardiovascular Life Support: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

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