Part 7: Adult Advanced Cardiovascular Life Support

Web-based Integrated 2015 & 2018 American Heart Association Guidelines for CPR and ECC

Key Words: arrhythmia cardiac arrest drugs ventricular arrhythmia ventricular fibrillation

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1 Highlights

2018 Summary of Key Issues and Major Changes

The review considered the use of Amiodarone, Lidocaine, Magnesium and Beta-blockers for antiarrhythmic therapy during and immediately after adult ventricular fibrillation (VF) and pulseless ventricular tachycardia (pVT) cardiac arrest. As a result, the Adult writing group's recommendations for CPR and ECC have been updated and now provide further clarity regarding the application of antiarrhythmics during cardiac arrest. The recommendations are as follows:

Adult Recommendations

- Use of antiarrhythmic drugs during resuscitation from adult VF/pVT cardiac arrest
    - **Amiodarone or lidocaine** may be considered for VF/pVT that is unresponsive to defibrillation. These

drugs may be particularly useful for patients with witnessed arrest, for whom time to drug administration may be shorter.
    - The routine use of magnesium for cardiac arrest is not recommended in adult patients. Magnesium may be considered for torsades de pointes (ie, polymorphic VT associated with long-QT interval). The wording of this recommendation is consistent with the AHA's 2010 Guidelines.
- Use of antiarrhythmic drugs immediately following return of spontaneous circulation (ROSC) following adult cardiac arrest
    - There is insufficient evidence to support or refute the routine use of a β- blocker early (within the first hour) after ROSC.
    - There is insufficient evidence to support or refute the routine use of lidocaine early (within the first hour) after ROSC.

Antiarrhythmic Drugs Immediately After ROSC Following Adult Cardiac Arrest: β-Blocker Recommendation

2018 (Updated): There is insufficient evidence to support or refute the routine use of a β-blocker early (within the first hour) after ROSC.

2015 (Old): 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).

Why: The 2018 CoSTR summary and systematic review considered the use of prophylactic antiarrhythmic drugs immediately (within the first hour) after ROSC. Although no new studies were reviewed for this topic, detailed

evaluation of the literature led to the simplification of the recommendation. There is no Class or LOE listed because the writing group agreed that there was insufficient evidence to make any recommendation.

Antiarrhythmic Drugs Immediately After ROSC Following Adult Cardiac Arrest: Lidocaine Recommendations

2018 (Updated): There is insufficient evidence to support or refute the routine use of lidocaine early (within the first hour) after ROSC. In the absence of contraindications, the prophylactic use of lidocaine may be considered in specific circumstances (such as during emergency medical services transport) when treatment of recurrent VF/pVT might prove to be challenging (Class IIb, LOE C-LD).

2015 (Old): 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).

Why: The 2018 CoSTR summary and systematic review considered the use of prophylactic antiarrhythmic drugs immediately (within the first hour) after ROSC. Although no new studies were reviewed for this topic, the writing group acknowledged that while there is insufficient evidence to support the routine use of lidocaine, there are situations for which recurrence of VF/pVT would be logistically challenging to manage (eg, during emergency medical services transport); in such situations, lidocaine administration may be considered.

Use of Antiarrhythmic Drugs During Resuscitation From Adult VF/pVT Cardiac Arrest: Amiodarone and Lidocaine Recommendation

2018 (Updated): Amiodarone or lidocaine may be considered for VF/pVT that is unresponsive to defibrillation. These drugs may be particularly useful for patients with witnessed arrest, for whom time to drug administration may be shorter (Class IIb, LOE B-R).

2015 (Old): 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).

Why: The 2018 CoSTR summary and systematic review considered the use of amiodarone or lidocaine during VF/pVT cardiac arrest refractory after at least 1 shock. The writing group evaluated a new large, out-of-hospital randomized controlled trial that compared a Captisol-based formulation of amiodarone with lidocaine or placebo for patients with refractory VF/pVT. Although the available studies did not demonstrate an improvement in survival to hospital discharge (or neurologically intact survival to discharge) associated with either drug, ROSC was higher in patients receiving lidocaine compared with placebo, and survival to hospital admission was higher with either drug compared with placebo. As a result, lidocaine is now recommended as an alternative to amiodarone and has now been added to the ACLS Cardiac Arrest Algorithm for treatment of shock-refractory VF/pVT (see the Figure 2 and ACLS Cardiac Arrest Algorithm Update section).

Use of Antiarrhythmic Drugs During Resuscitation From Adult VF/pVT Cardiac Arrest: Magnesium Recommendations

2018 (Updated): The routine use of magnesium for cardiac arrest is not recommended in adult patients (Class III: No Benefit, LOE C-LD). Magnesium may be considered for torsades de

pointes (ie, polymorphic VT associated with long QT interval) (Class IIb, LOE C-LD). The wording of this recommendation is consistent with the AHA's 2010 ACLS guidelines.

2015 (Old): The routine use of magnesium for VF/pVT is not recommended in adult patients (Class III: No Benefit, LOE B-R).

2010 (Old): When VF/pVT cardiac arrest is associated with torsades de pointes, providers may administer IV/IO bolus of magnesium sulfate at a dose of 1 to 2 g diluted in 10 mL D5W (Class IIb, LOE C).

Why: The 2018 CoSTR summary and systematic review considered the use of magnesium during resuscitation from cardiac arrest. No new studies were reviewed for this topic, and only a handful of small, nonrandomized studies have been identified in past reviews. The current recommendation reaffirms that magnesium should not be routinely used for cardiac arrest and notes that it may be considered for the treatment of torsades de pointes (ie, polymorphic VT associated with long QT interval).

2015 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.

Introduction

These *Web-based Integrated Guidelines* incorporate all relevant recommendations from 2010, 2015 and 2018. This *2018 American Heart Association* (AHA) focused update on the advanced cardiovascular life support (ACLS) guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) is based on the systematic review of antiarrhythmic therapy and the resulting "*2018 International Consensus on CPR and ECC Science With Treatment Recommendations*" (CoSTR) from the Advanced Life Support (ALS) Task Force of the International Liaison Committee on Resuscitation (ILCOR). The draft ALS CoSTR was posted online for public comment, and a

summary containing the final wording of the CoSTR has been published simultaneously with this focused update. AHA guidelines and focused updates are developed in concert with the ILCOR systematic evidence review process. In 2015, the ILCOR process transitioned to a continuous one, with systematic reviews performed as new published evidence warrants them or when the ILCOR ALS Task Force prioritizes a topic. Once the ILCOR ALS Task Force develops a CoSTR statement, AHA ACLS science experts review the relevant topics and update the AHA's ACLS guidelines as needed, typically on an annual basis. A description of the ILCOR continuous evidence review process is available in the 2017 CoSTR summary. The ILCOR systematic reviews use the Grading of Recommendations Assessment, Development, and Evaluation methodology and its associated nomenclature to determine the quality of evidence and strength of recommendations in the published CoSTR statement. The expert writing group for this 2018 ACLS guidelines focused update reviewed the studies and analysis of the 2018 CoSTR summary and carefully considered the ILCOR consensus recommendations in light of the structure and resources of the out-of-hospital and in-hospital resuscitation systems and the providers who use AHA guidelines. In addition, the writing group determined Classes of Recommendation and Levels of Evidence according to the most recent recommendations of the American College of Cardiology/AHA Task Force on Clinical Practice Guidelines (Table) by using the process detailed in "" in the "*2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care*." This 2018 ACLS guidelines focused update includes updates only to the recommendations for the use of antiarrhythmics during and immediately after adult ventricular fibrillation (VF) and pulseless ventricular tachycardia (pVT) cardiac arrest. All

other recommendations and algorithms published in “” in the 2015 guidelines update and “” in the “*2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care*” remain the official ACLS recommendations of the AHA ECC Science Subcommittee and writing groups. In addition, the “*2017 American Heart Association Focused Update on Adult Basic Life Support and Cardiopulmonary Resuscitation Quality: An Update to the American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care*” contains updated AHA recommendations for CPR delivered to adult patients in cardiac arrest. Through this systematic evaluation process, several issues have been identified in related areas that may be the subject of future systematic reviews.

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. 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.

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 .

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. 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. These parameters can be monitored continuously, without interrupting chest compressions. An abrupt increase in any of these parameters is a sensitive indicator of ROSC. There is evidence that

these and other physiologic parameters can be modified by interventions aimed at improving CPR quality.[1]

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.

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. 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. 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.

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.

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.

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.[,] 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.

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.[,] 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 “.”

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.

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 or other advanced airway devices.[,,] 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[,] 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. However, it also may impede ventilation and interfere with placement of a supraglottic airway or intubation. 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.

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.

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. Two case reports of inadvertent intracranial placement of a nasopharyngeal airway in patients with basilar skull fractures，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.

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. 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.

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

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. 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, or endotracheal intubation.[,,,,] Three observational studies demonstrated a lower rate of both overall survival and favorable neurologic outcome when

SGA use was compared with bag-mask ventilation,[,], whereas another observational study demonstrated similar survival rates.

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

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.

For healthcare providers trained in their use, either an SGA device or an ETT may be used as the initial advanced airway during CPR.

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.,

EMS systems that perform prehospital intubation should provide a program of ongoing quality improvement to minimize complications.

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. Although the sensitivity of

waveform capnography for detecting tracheal tube placement immediately after prehospital intubation was 100% in 1 study, several other studies showed that the sensitivity of waveform capnography decreases after a prolonged cardiac arrest. 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.,, 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.,, 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. , , ,

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. 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%. 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.

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.

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 and when the patient is moved from one location to another.[,] 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.

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 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.

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.[,] 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[,] and 1 human observational study 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.

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.

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.,

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 (and). 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—2018 Update

Figure 2: ACLS Cardiac Arrest Circular Algorithm - 2018 Update

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. 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.[,] 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 “”). 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 () 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 100-120 per minute. After placement of a supraglottic airway or an endotracheal tube, the provider performing chest compressions should deliver at least 100-120 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: 2010 - Treatable Causes of Cardiac Arrest: The H's and T's

Open table in a

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 “”). Finally, the reality is that the majority of resuscitative efforts do not result in ROSC. Criteria for ending unsuccessful resuscitative efforts are addressed in .

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

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.

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.

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. 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. 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.

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.

If using a manual defibrillator capable of escalating energies, higher energy for second and subsequent shocks may be considered.

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). An RCT published in 2010 showed no difference in frequency of VF recurrence when comparing the 2 treatment protocols. 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. 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. 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.

5.2.1.3.2 ***2015 Recommendation - Updated***

A single-shock strategy (as opposed to stacked shocks) is reasonable for defibrillation.

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.,

Current evidence indicates that the benefit of using a multimodal defibrillator in manual instead of automatic mode during cardiac arrest is uncertain.

5.2.1.5 ***CPR Before Defibrillation***

This topic now covered in .

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., 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.

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). 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 and “”). 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.

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. See “” 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). 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.

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 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 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 and “”). 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. Fibrinolytic therapy administered during CPR for acute coronary occlusion has not been shown to improve outcome.

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)

and therapeutic hypothermia in comatose patients.

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 or 1.5 mg/kg lidocaine with polysorbate. 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.[,] 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.

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.

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

5.3.2.1.2 ***Recommendations - Updated***

Amiodarone or lidocaine may be considered for VF/pVT that is unresponsive to defibrillation. These drugs maybe particularly useful for patients with witnessed arrest, for whom time to drug administration may be shorter.

The routine use of magnesium for cardiac arrest is not recommended in adult patients.

Magnesium may be considered for torsades de pointes (ie, polymorphic VT associated with long QT interval)

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 β-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 β-Adrenergic Blocking Drugs - Updated

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. Conversely, β-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 β-

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.[,] 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. 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 insufficient evidence to support or refute the routine use of a β-blocker early (within the first hour) after ROSC.

There is insufficient evidence to support or refute the routine use of lidocaine early (within the first hour) after ROSC.

In the absence of contraindications, the prophylactic use of lidocaine may be considered in specific circumstances (such as during emergency medical services transport) when treatment of recurrent VF/pVT might prove to be challenging.

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

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*

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.

5.3.3.1.1 *2015 Evidence Summary*

One trial 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.[,]

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.

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 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.

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), survival to discharge, or survival to hospital admission. There was, however, a demonstrated ROSC advantage with highdose epinephrine.

5.3.3.2.2 ***2015 Recommendation—New***

High-dose epinephrine is not recommended for routine use in cardiac arrest.

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

Vasopressin is a nonadrenergic peripheral vasoconstrictor that also causes coronary, and renal vasoconstriction.

5.3.3.3.1 ***2015 Evidence Summary***

A single RCT 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.

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

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, no benefit for survival to hospital discharge or hospital admission in 2438 patients, and no benefit for ROSC.

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.

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

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, 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 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 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 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,[,,] 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.[,,,] 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.

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. In a subsequent 3-center study published in 2013, 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.[1]

5.3.4.2 *2015 Evidence Summary: OHCA*

In OHCA, steroids have been evaluated in 1 RCT and 1 observational study. 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. The observational study 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 may be considered; however, further studies are needed before recommending the routine use of this therapeutic strategy.

For patients with OHCA, use of steroids during CPR is of uncertain benefit.

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, 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 the interval

from first shock to administration of an antiarrhythmic drug was a significant predictor of survival. One animal study 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. 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. 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 and adults and 6 other studies 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.

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.

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. 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.[,] 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, 5 studies in adults, and multiple animal studies have shown that lidocaine,[,] epinephrine, atropine, naloxone, and vasopressin 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, suggest that the lower epinephrine concentrations achieved when the drug is delivered endotracheally may produce transient β-adrenergic effects, resulting in vasodilation. These effects can be detrimental, causing hypotension, lower CPP and flow, and reduced potential for ROSC. Thus, although endotracheal administration of some resuscitation drugs is possible, IV or IO drug administration is preferred because it will provide more predictable drug delivery and pharmacologic effect.

In one nonrandomized cohort study of out-of-hospital cardiac arrest in adults 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.

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., 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. Studies

with epinephrine and lidocaine showed that dilution with sterile water instead of 0.9% saline may achieve better drug absorption.

5.5 ***Prognostication During CPR:End-Tidal CO_2 - 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.[1,2,3,4]

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. In that same study, an $ETCO_2$ greater than 20 mmHg after 20 minutes of resuscitation was associated with improved survival to discharge. 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. 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.

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.

5.6 ***Overview of Extracorporeal CPR - Updated***

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.

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.

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.

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.

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.

The key articles reviewed in the 2015 ILCOR systematic review comparing ECPR to conventional CPR feature some variability in their inclusion and exclusion criteria (), 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 Arti

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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.

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.[,] 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.

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 , increased ROSC, hospital admission, and survival to hospital discharge associated with use of bicarbonate. However, the majority of studies showed no benefit or found a relationship with poor outcome. ,

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. It can create extracellular alkalosis that will shift the oxyhemoglobin saturation curve and inhibit oxygen release. It can produce hypernatremia and theref