

Part 10: Special Circumstances of Resuscitation

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

Eric J. Lavonas, Chair; Ian R. Drennan; Andrea Gabrielli; Alan C. Heffner; Christopher O. Hoyte; Aaron M. Orkin; Kelly N. Sawyer; Michael W. Donnino

Introduction

This Part of the 2015 American Heart Association (AHA) Guidelines Update for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiovascular Care (ECC) addresses cardiac arrest in situations that require special treatments or procedures other than those provided during basic life support (BLS) and advanced cardiovascular life support (ACLS).

This Part summarizes recommendations for the management of resuscitation in several critical situations, including cardiac arrest associated with pregnancy (Part 10.1), pulmonary embolism (PE) (10.2), and opioid-associated resuscitative emergencies, with or without cardiac arrest (10.3). Part 10.4 provides recommendations on intravenous lipid emulsion (ILE) therapy, an emerging therapy for cardiac arrest due to drug intoxication. Finally, updated guidance for the management of cardiac arrest during percutaneous coronary intervention (PCI) is presented in Part 10.5. A table of all recommendations made in this 2015 Guidelines Update as well as those made in the 2010 Guidelines is contained in the Appendix.

The special situations of resuscitation section (Part 12) of the 2010 AHA Guidelines for CPR and ECC¹ covered 15 distinct topic areas. The following topics were last updated in 2010:

- Management of cardiac arrest associated with asthma (Part 12.1)
- Anaphylaxis (12.2)
- Morbid obesity (12.4)
- Electrolyte imbalance (12.6)
- Trauma (12.8)
- Accidental hypothermia (12.9)
- Avalanche (12.10)
- ACLS treatment of cardiac arrest due to drowning (12.11)
- Electric shock or lightning strikes (12.12)
- Cardiac tamponade (12.14)
- Cardiac surgery (12.15)
- Toxic effects of benzodiazepines, β -blockers, calcium channel blockers, digoxin, cocaine, cyclic antidepressants, carbon monoxide, and cyanide (12.7)

Additional information about drowning is presented in Part 5 of this publication, "Adult Basic Life Support and Cardiopulmonary Resuscitation Quality."

The recommendations in this 2015 Guidelines Update are based on an extensive evidence review process that was begun by the International Liaison Committee on Resuscitation (ILCOR) with the publication of the ILCOR 2010 International Consensus on CPR and ECC Science With Treatment Recommendations (CoSTR)² and was completed with the preparation of the 2015 CoSTR publication.^{3,4}

In the in-depth international evidence review process, the ILCOR task forces examined topics and then generated prioritized lists of questions for systematic review. The process by which topics were prioritized for review are described in the CoSTR publication.^{5,6} Questions were first formulated in PICO (population, intervention, comparator, outcome) format,⁷ the search strategy and inclusion and exclusion criteria were defined, and then a search for relevant articles was performed. The evidence was evaluated by using the standardized methodological approach proposed by the Grading of Recommendations Assessment, Development and Evaluation (GRADE) Working Group.⁸

The quality of the evidence was categorized based on the study methodologies and the 5 core GRADE domains of risk of bias, inconsistency, indirectness, imprecision, and other considerations (including publication bias). Then, where possible, consensus-based treatment recommendations were created. Further information about this international evidence evaluation process can be found in the 2015 CoSTR, "Part 2: Evidence Evaluation and Management of Conflicts of Interest."^{9,10}

To create this 2015 Guidelines Update, the AHA formed 15 writing groups, with careful attention to avoid or manage conflicts of interest, to assess the ILCOR treatment recommendations and to write AHA treatment recommendations by using the AHA Class of Recommendation and Level of Evidence (LOE) system. The recommendations made in this 2015 Guidelines Update are informed by the ILCOR recommendations and GRADE classification of the systematic reviews in the context of the delivery of medical care in North America. In the online version of this publication, live links

The American Heart Association requests that this document be cited as follows: Lavonas EJ, Drennan IR, Gabrielli A, Heffner AC, Hoyte CO, Orkin AM, Sawyer KN, Donnino MW. Part 10: special circumstances of resuscitation: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(suppl 2):S501–S518.

(*Circulation*. 2015;132[suppl 2]:S501–S518. DOI: 10.1161/CIR.0000000000000264.)

© 2015 American Heart Association, Inc.

Circulation is available at <http://circ.ahajournals.org>

DOI: 10.1161/CIR.0000000000000264

are provided so the reader can connect directly to those systematic reviews on the ILCOR Scientific Evidence Evaluation and Review System (SEERS) website. These links are indicated by a combination of letters and numbers (eg, ALS 436). We encourage readers to use the links and review the evidence and appendixes, such as the GRADE tables. Further information about this evidence evaluation process can be found in "Part 2: Evidence Evaluation and Management of Conflicts of Interest" of this 2015 Guidelines Update.

Contemporaneous with the ILCOR evidence-review process, the AHA ECC Committee; Council on Cardiopulmonary, Critical Care, Perioperative, and Resuscitation; Council on Cardiovascular Diseases in the Young; and Council on Clinical Cardiology have developed an AHA Scientific Statement on cardiac arrest in pregnancy.¹¹ While Part 10.1 of this 2015 Guidelines Update provides treatment recommendations for the intra-arrest management of pregnant patients, a full discussion of preparation, prevention, resuscitation, emergency delivery, and postresuscitation care are beyond the scope of this article. Readers are directed to the full Scientific Statement for more complete recommendations.

Part 10.1: Cardiac Arrest Associated With Pregnancy^{ALS 436}

Cardiac arrest associated with pregnancy is rare in high-income countries. Maternal cardiac arrest occurs in approximately 1:12 000 admissions for delivery in the United States.¹² Maternal cardiac arrest rates appear to be increasing in the United States, from 7.2 deaths per 100 000 live births in 1987 to 17.8 deaths per 100 000 live births in 2009.¹³ Maternal mortality rates are lower in Canada, where maternal mortality is reported as 6.1 deaths per 100 000 deliveries, with a decreasing trend from 2001 until 2011.^{14,15}

The best outcomes for both mother and fetus are likely to be achieved by successful maternal resuscitation. The most common causes of maternal cardiac arrest are hemorrhage, cardiovascular diseases (including myocardial infarction, aortic dissection, and myocarditis), amniotic fluid embolism, sepsis, aspiration pneumonia, PE, and eclampsia.^{12,16} Important iatrogenic causes of maternal cardiac arrest include hypermagnesemia from magnesium sulfate administration and anesthetic complications.

The 2015 ILCOR systematic review addressed the questions of patient positioning during CPR and the role of perimortem cesarean delivery (PMCD) in the management of pregnant women in cardiac arrest during the second half of pregnancy.

2015 Evidence Summary

The evidence regarding advanced treatment strategies for cardiac arrest in pregnancy is largely observational. As a result, the recommendations are based on application of physiologic principles and on close examination of observational studies that are susceptible to bias. The lack of high-quality studies examining treatment of cardiac arrest in late pregnancy represents a major scientific gap.

Patient Positioning During CPR

Patient position has emerged as an important strategy to improve the quality of CPR and resultant compression force

and cardiac output. The gravid uterus can compress the inferior vena cava, impeding venous return, thereby reducing stroke volume and cardiac output. In general, aortocaval compression can occur for singleton pregnancies at approximately 20 weeks of gestational age,¹⁷ at about the time when the fundus is at or above the umbilicus. Although chest compressions in the left lateral tilt position are feasible in a manikin study,¹⁸ they result in decreased CPR quality (less forceful chest compressions) than is possible in the supine position.¹⁹ Manual left lateral uterine displacement (LUD) effectively relieves aortocaval pressure in patients with hypotension²⁰ (Figure 1). No cardiac arrest outcome studies have been published examining the effect of LUD or other strategies to relieve aortocaval compression during resuscitation.

Emergency Cesarean Delivery in Cardiac Arrest

Evacuation of the gravid uterus relieves aortocaval compression and may improve resuscitative efforts.^{21–25} In the latter half of pregnancy, PMCD may be considered part of maternal resuscitation, regardless of fetal viability.²⁶ In a case series, 12 of 20 women for whom maternal outcome was recorded who underwent PMCD during resuscitation had return of spontaneous circulation (ROSC) immediately after delivery,

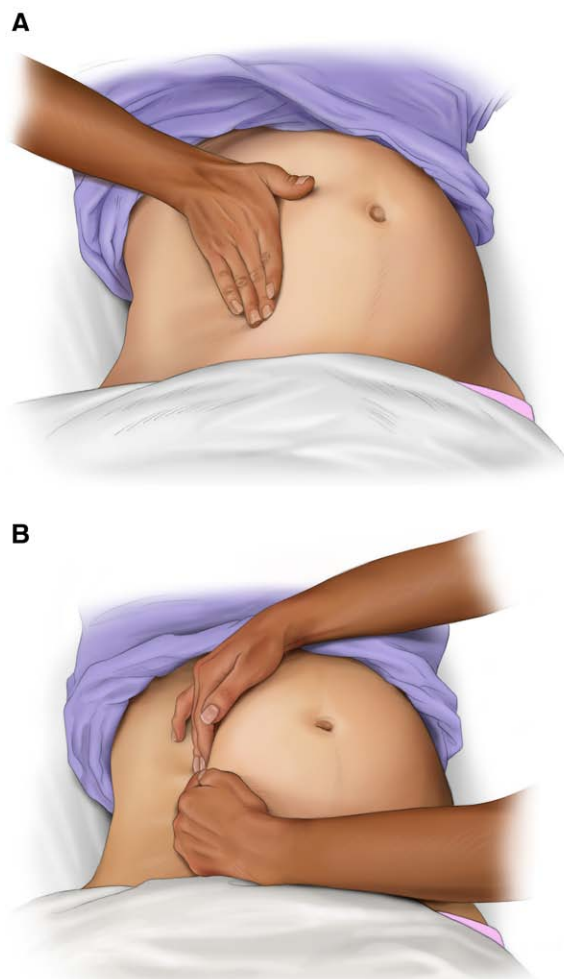


Figure 1. A, Manual LUD, performed with one-handed technique. B, Two-handed technique during resuscitation.

and no cases of worsening maternal status were reported.²⁷ A systematic review of the literature evaluated all case reports of cardiac arrest in pregnancy, but the wide range of case heterogeneity and reporting bias does not allow for any conclusions regarding the timing of PMCD.²⁸ Survival of the mother has been reported up to 15 minutes after the onset of maternal cardiac arrest.^{21,29–31} Neonatal survival has been documented with PMCD performed up to 30 minutes after the onset of maternal cardiac arrest.²¹

2015 Recommendations—New and Updated

BLS Modification: Relief of Aortocaval Compression

Priorities for the pregnant woman in cardiac arrest are provision of high-quality CPR and relief of aortocaval compression (Class I, LOE C-LD). If the fundus height is at or above the level of the umbilicus, manual LUD can be beneficial in relieving aortocaval compression during chest compressions (Class IIa, LOE C-LD).

ALS Modification: Emergency Cesarean Delivery in Cardiac Arrest

Because immediate ROSC cannot always be achieved, local resources for a PMCD should be summoned as soon as cardiac arrest is recognized in a woman in the second half of pregnancy (Class I, LOE C-LD). Systematic preparation and training are the keys to a successful response to such rare and complex events. Care teams that may be called upon to manage these situations should develop and practice standard institutional responses to allow for smooth delivery of resuscitative care (Class I, LOE C-EO).

During cardiac arrest, if the pregnant woman with a fundus height at or above the umbilicus has not achieved ROSC with usual resuscitation measures plus manual LUD, it is advisable to prepare to evacuate the uterus while resuscitation continues (Class I, LOE C-LD). In situations such as nonsurvivable maternal trauma or prolonged pulselessness, in which maternal resuscitative efforts are obviously futile, there is no reason to delay performing PMCD (Class I, LOE C-LD). PMCD should be considered at 4 minutes after onset of maternal cardiac arrest or resuscitative efforts (for the unwitnessed arrest) if there is no ROSC (Class IIa, LOE C-EO). The clinical decision to perform a PMCD—and its timing with respect to maternal cardiac arrest—is complex because of the variability in level of practitioner and team training, patient factors (eg, etiology of arrest, gestational age), and system resources.

Part 10.2: Cardiac Arrest Associated With Pulmonary Embolism^{ALS 435}

PE is a potentially reversible cause of shock and cardiac arrest. Acute increase in right ventricular pressure due to pulmonary artery obstruction and liberation of vasoactive mediators produces cardiogenic shock that may rapidly progress to cardiovascular collapse. Management of acute PE is determined by disease severity.³² Fulminant PE, characterized by cardiac arrest or severe hemodynamic instability, defines the subset of massive PE that is the focus of these recommendations.³³

Less than 5% of patients with acute PE progress to cardiac arrest. Disease of this severity is associated with mortality of

65% to 90%.^{34–36} PE-related cardiac arrests may occur within hours of symptom onset. Between 5% and 13% of unexplained cardiac arrests are associated with fulminant PE.^{37,38}

Because establishing the diagnosis of acute PE in cardiac arrest situations is often difficult, separate systematic reviews were performed for management of patients with suspected and confirmed PE. Although clinical markers specific to fulminant PE are limited, acute symptoms frequently prompt medical attention before cardiac arrest. Conventional thromboembolism risk factors, prodromal dyspnea or respiratory distress, and witnessed arrest are features associated with cardiac arrest due to PE.^{37,39} Pulseless electrical activity is the presenting rhythm in 36% to 53% of PE-related cardiac arrests, while primary shockable rhythms are uncommon.^{37,40,41} Specific recommendations about the use of diagnostic ultrasonography during resuscitation can be found in “Part 7: Adult Advanced Cardiovascular Life Support” in this 2015 Guidelines Update.

Prompt systemic anticoagulation is generally indicated for patients with massive and submassive PE to prevent clot propagation and support endogenous clot dissolution over weeks.⁴² Anticoagulation alone is inadequate for patients with fulminant PE. Pharmacologic and mechanical therapies to rapidly reverse pulmonary artery occlusion and restore adequate pulmonary and systemic circulation have emerged as primary therapies for massive PE, including fulminant PE.^{32,43} Current advanced treatment options include systemic thrombolysis, surgical or percutaneous mechanical embolectomy, and extracorporeal cardiopulmonary resuscitation (ECPR).

The 2015 ILCOR systematic review addressed the treatment of PE as the known or suspected cause of cardiac arrest. The role of thrombolytic medications in the management of undifferentiated cardiac arrest was last reviewed in the 2010 Guidelines and is not reviewed again here.⁴⁴

2015 Evidence Summary

The evidence regarding advanced treatment strategies for fulminant PE is largely observational. The lack of high-quality studies examining treatment of cardiac arrest due to PE represents a major scientific gap.

Confirmed Pulmonary Embolism

Systemic thrombolysis is associated with ROSC and short-term survival in PE-related cardiac arrest in nonrandomized observational studies.^{37,45–54}

There is no consensus on the ideal dose of thrombolytic therapy in PE-associated cardiac arrest. Contemporary examples of accelerated emergency thrombolysis dosing regimens for fulminant PE include alteplase 50 mg intravenous (IV) bolus with an option for repeat bolus in 15 minutes, or single-dose weight-based tenecteplase; thrombolytics are administered with or followed by systemic anticoagulation.^{55–57} Early administration of systemic thrombolysis is associated with improved resuscitation outcomes compared with use after failure of conventional ACLS.⁴⁶

Successful surgical and percutaneous mechanical embolectomy in cases of PE-related cardiac arrest have been reported in limited series.^{58–60} Many of these patients developed cardiac arrest before or during embolectomy. The feasibility of embolectomy under uncontrolled CPR conditions is not known.

Suspected Pulmonary Embolism

No evidence is available to support or refute the effectiveness of empiric thrombolysis in suspected but unconfirmed PE.

2015 Recommendations—New and Updated**ALS Modification: Confirmed Pulmonary Embolism**

In patients with confirmed PE as the precipitant of cardiac arrest, thrombolysis, surgical embolectomy, and mechanical embolectomy are reasonable emergency treatment options (Class IIa, LOE C-LD). Comparative data are not available to recommend one strategy over another. Patient location, local intervention options, and patient factors (including thrombolysis contraindications) are recognized elements to be considered. Thrombolysis can be beneficial even when chest compressions have been provided (Class IIa, LOE C-LD). Given the poor outcomes associated with fulminant PE in the absence of clot-directed therapy, standard contraindications to thrombolysis may be superseded by the need for potentially lifesaving intervention.

ALS Modifications: Suspected Pulmonary Embolism

Thrombolysis may be considered when cardiac arrest is suspected to be caused by PE (Class IIb, LOE C-LD). There is no consensus on inclusion criteria (eg, risk factors, signs, or symptoms that constitute suspected PE), thrombolytic timing, drug, or dose in this situation. There are insufficient data on surgical and mechanical embolectomy to evaluate these therapies for cardiac arrest associated with suspected but unconfirmed PE.

Part 10.3: Cardiac or Respiratory Arrest Associated With Opioid Overdose^{ALS 441,BLS 811,BLS 891}

In the United States in 2013, 16235 people died of prescription opioid toxicity, and an additional 8257 died of heroin overdose.^{61,62} In the United States in 2012, opioid overdose became the leading cause of unintentional injurious death in people aged 25 to 60 years, accounting for more deaths than motor vehicle collisions.⁶³ A majority of these deaths are associated with prescription opioids. Statistics are similar in Canada.⁶⁴

Isolated opioid toxicity is associated with central nervous system (CNS) and respiratory depression that can progress to respiratory and cardiac arrest. Most opioid deaths involve the co-ingestion of multiple drugs or medical and mental health comorbidities.^{65–68} In addition, methadone and propoxyphene can cause *torsades de pointes*, and cardiotoxicity has been reported with other opioids.^{69–75} Except in specific clinical settings (eg, unintended opioid overdose during a medical procedure), rescuers cannot be certain that the patient's clinical condition is due to opioid-induced CNS and respiratory depression toxicity alone, and might therefore misidentify opioid-associated cardiac arrest as unconsciousness or *vice versa*. This is particularly true in the first aid and BLS contexts, where determination of the presence or absence of a pulse is unreliable.^{76,77} Any treatment recommendations intended for use in the first aid or BLS settings must therefore have benefit that exceeds harm

when applied to a mixed patient population that may include people with severe CNS and respiratory depression, respiratory arrest, and cardiac arrest.

In creating this 2015 Guidelines Update, the writing group considered the difficulty in accurately differentiating opioid-associated resuscitative emergencies from other causes of cardiac and respiratory arrest. Opioid-associated resuscitative emergencies are defined by the presence of cardiac arrest; respiratory arrest; or severe life-threatening instability (such as severe CNS or respiratory depression, hypotension, or cardiac arrhythmia) that is suspected to be due to opioid toxicity. The term “opioid-associated life-threatening emergency” is used for first aid and non-healthcare providers.

Naloxone is a potent opioid receptor antagonist in the brain, spinal cord, and gastrointestinal system. Naloxone has an excellent safety profile and can rapidly reverse CNS and respiratory depression in a patient with an opioid-associated resuscitative emergency. Based on the rescuer's training and clinical circumstance, naloxone can be administered intravenously,^{78–81} intramuscularly,^{78,79,82} intranasally,^{80,82–86} or subcutaneously⁸⁷; nebulized for inhalation^{88,89}; or instilled into the bronchial tree via endotracheal tube.⁹⁰ Appropriate dose and concentrations differ by route.

There are no known harms or major clinical effects associated with the administration of naloxone in typical doses to patients who are not opioid-intoxicated or dependent.^{91,92} Naloxone administration may precipitate acute withdrawal syndrome in patients with opioid dependency, with signs and symptoms including hypertension, tachycardia, piloerection, vomiting, agitation, and drug cravings. These signs and symptoms are rarely life-threatening, and they may be minimized by using the lowest effective dose of naloxone.⁹³ Pulmonary edema has been reported with naloxone administration, but it also may be caused primarily by opioid toxicity.⁹³

The ideal dose of naloxone is not known. In the 2010 Guidelines, an empiric starting dose of 0.04 to 0.4 mg IV or intramuscular (IM) was recommended to avoid provoking severe opioid withdrawal in patients with opioid dependency and to allow for consideration of a range of doses, depending on the clinical scenario.¹ Repeat doses or dose escalation to 2 mg IV or IM was recommended if the initial response was inadequate. Few comparative data exist about the appropriate dose of intranasal (IN) naloxone; most studies used a fixed dose of 2 mg, repeated in 3 to 5 minutes if necessary.^{80,82–86,94} Nebulized naloxone has been studied and well-tolerated in opioid-intoxicated patients at a dose of 2 mg diluted in 3 mL normal saline.^{88,89} Regardless of the care setting and route of administration, the initial goal of therapy is to restore and maintain patent airway and ventilation, preventing respiratory and cardiac arrest, without provoking severe opioid withdrawal.

The 2015 ILCOR systematic review addressed the questions of whether opioid overdose response education (with or without naloxone distribution) improves outcomes related to opioid overdose and whether naloxone administration or any other therapy improves outcomes in the patients with opioid-associated cardio/respiratory arrest in the first aid, BLS, or ACLS settings.

2015 Evidence Summary

Opioid Overdose Response Education and Naloxone Training and Distribution

Several studies have shown that community-based opioid overdose response education and naloxone distribution programs are feasible and that naloxone administration occurs frequently by persons trained by these programs.⁹⁵ Because patients who have CNS and respiratory depression from opioid overdose cannot self-administer naloxone, naloxone is typically administered in the first aid setting by friends, family, or bystanders.^{96,97}

In 2014, the US Food and Drug Administration approved of the use of a naloxone autoinjector by lay rescuers⁹⁸ as well as healthcare providers. Both the IM and IN⁹⁵ routes of administration have been successfully used in first aid settings, with commercially available devices or kits containing a naloxone vial or prefilled syringe and a nasal atomizer or other administration device. IM, IN, and nebulized routes of administration have also been used to treat opioid-associated resuscitative emergencies in the BLS and ACLS settings.^{79,80,88,99} Recent recommendations by an international working group called for uniform training standards based on simplified (first aid) resuscitation principles for community-based naloxone distribution programs.¹⁰⁰

Administration of Naloxone in Opioid-Associated Resuscitation Emergencies

Respiratory Arrest

Two clinical trials and 12 observational studies examined outcomes after naloxone treatment for opioid-induced respiratory arrest or severe CNS and respiratory depression. Of these, 5 studies compared routes of naloxone administration,^{80,82,83,87,101} and 9 assessed the safety of naloxone use or were observational studies of naloxone use alone.^{79,102–109} All studies reported improvement in level of consciousness and spontaneous breathing after naloxone administration in the majority of patients treated, and complication rates were low. No study compared resuscitation outcomes achieved with naloxone with those achieved through standard therapy alone (eg, manual or mechanical ventilation).

Cardiac Arrest

One small observational study noted an improvement in cardiac rhythm in some patients after naloxone administration, but it did not compare outcomes in patients managed with and without naloxone administration.¹¹⁰

2015 Recommendations—New

Opioid Overdose Response Education and Naloxone Training and Distribution

It is reasonable to provide opioid overdose response education, either alone or coupled with naloxone distribution and training, to persons at risk for opioid overdose (Class IIa, LOE C-LD). Some populations that may benefit from opioid overdose response interventions are listed in Table 1. It is reasonable to base this training on first aid and non-healthcare provider BLS recommendations rather than on more advanced practices intended for healthcare providers (Class IIa, LOE C-EO).

Table 1. Groups That May Benefit From Opioid Overdose Response Education and/or Naloxone Distribution and Training^{100,111–119}

-
- Persons who abuse prescription opioids or heroin
 - Patients who have required emergency care for opioid overdose
 - Patients enrolled in opioid dependence treatment programs, including methadone and buprenorphine maintenance programs, particularly at high-risk periods, such as induction or discharge
 - Persons with a history of opioid abuse or dependence who are being released from prison
 - Patients receiving prescription opioid therapy with risk factors for adverse effects
 - Coprescriptions of benzodiazepines or other sedatives
 - Ongoing alcohol use
 - High-dose prescription opioid therapy
 - Persons living with or in frequent contact with those listed above
-

First Aid and Non-Healthcare Provider BLS Modification: Administration of Naloxone

Although naloxone has no clear role in the management of confirmed cardiac arrest, first aid and other non-healthcare providers are not instructed to attempt to determine whether an unresponsive person is pulseless. Empiric administration of IM or IN naloxone to all unresponsive opioid-associated life-threatening emergency patients may be reasonable as an adjunct to standard first aid and non-healthcare provider BLS protocols (Class IIb, LOE C-EO). Standard resuscitation, including activation of emergency medical services, should not be delayed for naloxone administration. However, family members and friends of those known to be addicted to opiates are likely to have naloxone available and ready to use if someone known or suspected to be addicted to opiates is found unresponsive and not breathing normally or only gasping (see sequence in Figure 2). Victims who respond to naloxone administration should access advanced healthcare services (Class I, LOE C-EO).

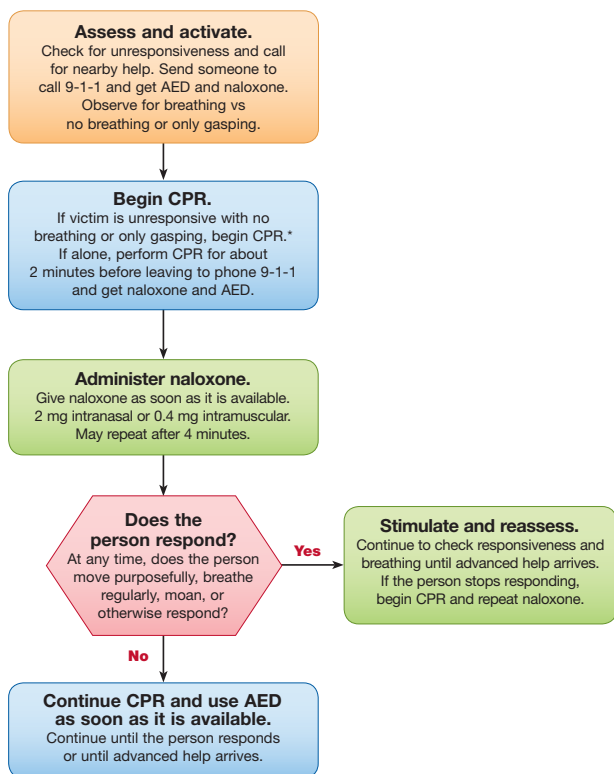
Healthcare Provider BLS Modification: Administration of Naloxone

Respiratory Arrest

For patients with known or suspected opioid overdose who have a definite pulse but no normal breathing or only gasping (ie, a respiratory arrest), in addition to providing standard BLS care, it is reasonable for appropriately trained BLS healthcare providers to administer IM or IN naloxone (Class IIa, LOE C-LD). For further information, see “Part 5: Adult Basic Life Support and Cardiopulmonary Resuscitation Quality.”

Cardiac Arrest

Patients with no definite pulse may be in cardiac arrest or may have an undetected weak or slow pulse. These patients should be managed as cardiac arrest patients. Standard resuscitative measures should take priority over naloxone administration (Class I, LOE C-EO), with a focus on high-quality CPR (compressions plus ventilation). It may be reasonable to administer IM or IN naloxone based on the possibility that the patient is not in cardiac arrest (Class IIb, LOE C-EO). Responders should not delay access to more-advanced medical services while

Opioid-Associated Life-Threatening Emergency (Adult) Algorithm—New 2015

*CPR technique based on rescuer's level of training.

© 2015 American Heart Association

Figure 2. Opioid-Associated Life-Threatening Emergency (Adult) Algorithm.

awaiting the patient's response to naloxone or other interventions (Class I, LOE C-EO). Unless the patient refuses further care, victims who respond to naloxone administration should access advanced healthcare services (Class I, LOE C-EO).

ACLS Modification: Administration of Naloxone

Respiratory Arrest

ACLS providers should support ventilation and administer naloxone to patients with a perfusing cardiac rhythm and opioid-associated respiratory arrest or severe respiratory depression. Bag-mask ventilation should be maintained until spontaneous breathing returns, and standard ACLS measures should continue if return of spontaneous breathing does not occur (Class I, LOE C-LD).

Cardiac Arrest

We can make no recommendation regarding the administration of naloxone in confirmed opioid-associated cardiac arrest. Patients with opioid-associated cardiac arrest are managed in accordance with standard ACLS practices.

Observation and Post-Resuscitation Care

After ROSC or return of spontaneous breathing, patients should be observed in a healthcare setting until the risk of recurrent opioid toxicity is low and the patient's level of consciousness and vital signs have normalized (Class I, LOE C-LD). If recurrent opioid toxicity develops, repeated small

doses or an infusion of naloxone can be beneficial in health-care settings (Class IIa, LOE C-LD).

Patients who respond to naloxone administration may develop recurrent CNS and/or respiratory depression. Although abbreviated observation periods may be adequate for patients with fentanyl, morphine, or heroin overdose,^{102,109,120–123} longer periods of observation may be required to safely discharge a patient with life-threatening overdose of a long-acting or sustained-release opioid.^{93,124,125}

Naloxone administration in post-cardiac arrest care may be considered in order to achieve the specific therapeutic goals of reversing the effects of long-acting opioids (Class IIb, LOE C-EO).

Part 10.4: Role of Intravenous Lipid Emulsion Therapy in Management of Cardiac Arrest Due to Poisoning^{ALS 834}

The use of ILE therapy was first developed as a treatment for cardiac arrest resulting from the local anesthetic bupivacaine.^{126–128} Local anesthetics inhibit voltage at the cell membrane sodium channels, limiting action potential and the conduction of nerve signals. Local anesthetic systemic toxicity (LAST) can present with fulminant cardiovascular collapse that is refractory to standard resuscitative measures. A CNS toxicity phase (agitation evolving to frank seizures or CNS depression) may precede cardiovascular collapse. A recent review of peripheral nerve anesthetic blocks estimated the incidence of LAST equal to 0.87/1000 patients.¹²⁹ When a local anesthetic is administered, professional organizations recommend continuous neurologic and cardiovascular monitoring, dose fractionation, slow injection, concurrent use of an intravascular marker of systemic absorption (epinephrine 10 to 15 µg), and the use of ultrasound techniques.¹³⁰

Administration of ILE creates a lipid compartment in the serum, reducing by sequestration the concentration of lipophilic medications in the tissues.¹³¹ Administration of ILE also increases cardiac inotropy by other mechanisms.^{132–134}

Over time, common use of this modality has been expanded to include poisoning by other local anesthetics and other medications.^{135–138}

The 2015 ILCOR systematic review addressed the question of whether administration of lipid emulsion improves outcomes for patients who develop cardiac arrest due to drug toxicity, including that caused by local anesthetics and other drugs.

2015 Evidence Summary

To date, we identified no human studies that compared outcomes of patients in cardiac arrest treated with ILE plus supportive care versus supportive care alone. A small controlled trial of adults with poisoning from drugs other than local anesthetics showed a more rapid improvement in level of consciousness in the group that received ILE, but all patients survived in both groups.¹³⁹ Patients with glyphosate-surfactant herbicide ingestion treated with ILE had less hypotension and fewer arrhythmias than historic controls, but there was no difference in survival outcomes.¹⁴⁰ Registry studies of patients receiving ILE are difficult to interpret because of a lack of comparison groups.^{141,142}

Animal studies in rats consistently show a benefit of ILE in LAST caused by bupivacaine.^{137,143} Studies are less consistently positive in porcine models of LAST and from poisoning by drugs other than local anesthetics.¹³⁸ In a recent systematic review of human case reports, the majority (81/103) reported clinical improvement, such as ROSC, relief of hypotension, resolution of dysrhythmia, improved mental status, or termination of status epilepticus, after ILE administration.¹³⁸ In this review, all 21 published cases of the use of ILE to treat LAST from bupivacaine demonstrated clinical improvement after ILE administration.

Comparative dose studies are not available. The most commonly reported strategy is to use a 20% emulsion of long-chain triglycerides, giving an initial bolus of 1.5 mL/kg lean body mass over 1 minute followed by an infusion of 0.25 mL/kg per minute for 30 to 60 minutes. The bolus can be repeated once or twice as needed for persistent cardiovascular collapse; the suggested maximum total dose is 10 mL/kg over the first hour.^{137,144–146} The safety of prolonged infusions (beyond 1 hour) has not been established.¹⁴⁷

The most common adverse effect of ILE therapy is interference with diagnostic laboratory testing¹⁴⁸; rare cases of pancreatitis¹⁴⁸ and pulmonary changes similar to those observed with acute respiratory distress syndrome¹⁴⁹ have also been reported. There appear to be complex pharmacodynamic interactions between ILE and epinephrine given during resuscitation, and in some situations, treatment with ILE alters the effectiveness of epinephrine and vasopressin in animal resuscitation studies.¹⁵⁰ Although some organizations recommend modification of the pharmacologic treatment of cardiac arrest after ILE administration,^{151,152} there are no human data to support a modification in ACLS recommendations. More recently, concern has been raised that ILE administration may increase the absorption of lipophilic medications from the gastrointestinal tract¹⁵³ and interfere with the operation of venoarterial extracorporeal membrane oxygenation circuits.¹⁵⁴

2015 Recommendations—New and Updated

ACLS Modifications

It may be reasonable to administer ILE, concomitant with standard resuscitative care, to patients with local anesthetic systemic toxicity and particularly to patients who have premonitory neurotoxicity or cardiac arrest due to bupivacaine toxicity (Class IIb, LOE C-EO). It may be reasonable to administer ILE to patients with other forms of drug toxicity who are failing standard resuscitative measures (Class IIb, LOE C-EO).

Part 10.5: Cardiac Arrest During Percutaneous Coronary Intervention^{ALS 479}

Cardiac arrest during PCI is rare, occurring in approximately 1.3% of catheterization procedures.^{155,156} Although the risk of cardiac arrest during PCI is present in both elective and emergency procedures, the incidence is higher in emergency cases.¹⁵⁷

In general, patients who develop cardiac arrest during PCI have superior outcomes to patients in cardiac arrest that occurs in other settings, including in-hospital units.¹⁵⁸ Many

patients will respond to standard ACLS resuscitation, including high-quality CPR and rapid defibrillation. Rapid defibrillation (within 1 minute) is associated with survival to hospital discharge rates as high as 100% in this population.¹⁵⁹

A subset of patients who develop cardiac arrest during PCI will require prolonged resuscitation efforts. Providing effective prolonged resuscitation in the catheterization laboratory has unique challenges, and a number of interventions and adjuncts for management of cardiac arrest during PCI have been described. Inconsistent availability and lack of comparative studies limit recommendations of one approach over another.

The 2015 ILCOR systematic review addressed the question of whether any special interventions or changes in care, compared with standard ACLS resuscitation alone, can improve outcomes in patients who develop cardiac arrest during PCI.

There are a number of mechanical devices available to provide hemodynamic support during cardiac catheterization in high-risk patients presenting with cardiogenic shock. The use of these devices in cardiogenic shock was not reviewed by ILCOR in 2015. Therefore, the *2015 AHA Guidelines Update for CPR and ECC* does not make recommendations on the use of mechanical support devices in patients presenting in cardiogenic shock who undergo PCI. Recent recommendations for the use of mechanical support devices in these situations can be found in the *2013 American College of Cardiology Foundation (ACCF)/AHA Guideline for the Management of ST-Elevation Myocardial Infarction*.¹⁶⁰

2015 Evidence Summary

The feasibility of using mechanical CPR devices during PCI has been demonstrated in both animal¹⁶¹ and human^{162–165} studies. No comparative studies have examined the use of mechanical CPR devices compared with manual chest compressions during PCI procedures. However, a number of case reports^{161,162,166} and case series^{164,165} have reported the use of mechanical CPR devices to facilitate prolonged resuscitation in patients who have a cardiac arrest during PCI. One study demonstrated that the use of a mechanical CPR device for cardiac arrest during PCI was feasible; however, no patients survived to hospital discharge.¹⁶⁴ Other studies have reported good patient outcomes, including ROSC, survival to discharge, and functional outcome at hospital discharge, after use of mechanical devices in resuscitation from cardiac arrest during PCI.^{161,165} Mechanical CPR devices may also allow the use of fluoroscopy during chest compressions without direct irradiation of personnel.

Patients in cardiogenic shock or with other high-risk features (eg, multivessel coronary disease) may be at increased risk for adverse outcomes during or after PCI. Ventricular assist devices, intraaortic balloon pumps (IABP), and ECPR are all rescue treatment options available to support circulation and permit completion of the PCI. Not all interventions are available or can be rapidly deployed in all centers.

Rapid initiation of ECPR or cardiopulmonary bypass is associated with good patient outcomes in patients with hemodynamic collapse and cardiac arrest in the

catheterization lab.^{167–173} The use of ECPR is also feasible and associated with good outcomes when used as a bridge to coronary artery bypass grafting.^{167,173–175} The combination of ECPR and IABP has been associated with increased survival when compared with IABP alone for patients who present with cardiogenic shock, including those who have a cardiac arrest while undergoing PCI.^{168,172,176} Available observational studies often implement ECPR 20 to 30 minutes after cardiac arrest.^{168,170}

IABP counterpulsation increases coronary perfusion, decreases myocardial oxygen demand, and improves hemodynamics in cardiogenic shock states, but it is not associated with improved patient survival in cardiogenic shock.^{177–185} The role of IABP in patients who have a cardiac arrest in the catheterization laboratory is not known.

Several case series have reported on the use of emergency coronary artery bypass graft surgery after failed PCI.^{186,187} In patients with cardiogenic shock or cardiac arrest and failed PCI, mechanical CPR devices and/or ECPR have been used as rescue bridges to coronary artery bypass graft. Although no comparison studies have examined the use of this therapy as an adjunct to PCI, survival

to hospital discharge rates as high as 64% have been reported.^{167,168,173,175}

2015 Recommendations—New and Updated

ACLS Modifications

It may be reasonable to use mechanical CPR devices to provide chest compressions to patients in cardiac arrest during PCI (Class IIb, LOE C-EO).

It may be reasonable to use ECPR as a rescue treatment when initial therapy is failing for cardiac arrest that occurs during PCI (Class IIb, LOE C-LD). Because patients can remain on ECPR support for extended periods of time without possibility of recovery, practical and ethical considerations must be taken into account in determining which victims of cardiac arrest should receive ECPR support. Institutional guidelines should include the selection of appropriate candidates for use of mechanical support devices to ensure that these devices are used as a bridge to recovery, surgery or transplant, or other device (Class I, LOE C-EO).

Due to a lack of comparative studies, it is not possible to recommend one approach (manual CPR, mechanical CPR, or ECPR) over another when options exist.

Disclosures

Part 10: Special Circumstances of Resuscitation: 2015 Guidelines Update Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/Advisory Board	Other
Eric J. Lavonas	Rocky Mountain Poison & Drug Center; Denver Health and Hospital Authority	None	None	None	None	None	None	BTG International, Inc. – relationship between Employer and BTG†
Ian R. Drennan	St. Michael's Hospital Rescu	None	None	None	None	None	None	None
Andrea Gabrielli	University of Florida College of Medicine	None	None	None	For intraoperative cardiac ischemia*	None	None	None
Alan C. Heffner	UNC School of Medicine	None	None	Edwards Lifesciences*	Defense and plaintiff consultant and causation expert in cases focused on airway management and CPR*	None	None	None
Christopher O. Hoyte	University of Colorado School of Medicine	None	None	None	None	None	None	None
Aaron M. Orkin	University of Toronto	Canadian Institutes of Health Research*	None	None	None	None	None	Remote Health Initiative*
Kelly N. Sawyer	William Beaumont Hospital	None	None	None	None	None	None	None
Consultant								
Michael W. Donnino	Beth Israel Deaconess Med Center	None	None	None	None	None	American Heart Association†	None

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

*Modest.
†Significant.

Appendix

2015 Guidelines Update: Part 10 Recommendations

Year Last Reviewed	Topic	Recommendation	Comments
2015	Cardiac Arrest Associated With Pregnancy	Priorities for the pregnant woman in cardiac arrest are provision of high-quality CPR and relief of aortocaval compression (Class I, LOE C-LD).	new for 2015
2015	Cardiac Arrest Associated With Pregnancy	If the fundus height is at or above the level of the umbilicus, manual LUD can be beneficial in relieving aortocaval compression during chest compressions (Class IIa, LOE C-LD).	new for 2015
2015	Cardiac Arrest Associated With Pregnancy	Because immediate ROSC cannot always be achieved, local resources for a PMCD should be summoned as soon as cardiac arrest is recognized in a woman in the second half of pregnancy (Class I, LOE C-LD).	new for 2015
2015	Cardiac Arrest Associated With Pregnancy	Systematic preparation and training are the keys to a successful response to such rare and complex events. Care teams that may be called upon to manage these situations should develop and practice standard institutional responses to allow for smooth delivery of resuscitative care (Class I, LOE C-E0).	new for 2015

(Continued)

2015 Guidelines Update: Part 10 Recommendations, *Continued*

Year Last Reviewed	Topic	Recommendation	Comments
2015	Cardiac Arrest Associated With Pregnancy	During cardiac arrest, if the pregnant woman with a fundus height at or above the umbilicus has not achieved ROSC with usual resuscitation measures plus manual LUD, it is advisable to prepare to evacuate the uterus while resuscitation continues (Class I, LOE C-LD).	new for 2015
2015	Cardiac Arrest Associated With Pregnancy	In situations such as nonsurvivable maternal trauma or prolonged pulselessness, in which maternal resuscitative efforts are obviously futile, there is no reason to delay performing PMCD (Class I, LOE C-LD).	new for 2015
2015	Cardiac Arrest Associated With Pregnancy	PMCD should be considered at 4 minutes after onset of maternal cardiac arrest or resuscitative efforts (for the unwitnessed arrest) if there is no ROSC (Class IIa, LOE C-E0).	updated for 2015
2015	Cardiac Arrest Associated With Pulmonary Embolism	In patients with confirmed PE as the precipitant of cardiac arrest, thrombolysis, surgical embolectomy, and mechanical embolectomy are reasonable emergency treatment options (Class IIa, LOE C-LD).	new for 2015
2015	Cardiac Arrest Associated With Pulmonary Embolism	Thrombolysis can be beneficial even when chest compressions have been provided (Class IIa, LOE C-LD).	new for 2015
2015	Cardiac Arrest Associated With Pulmonary Embolism	Thrombolysis may be considered when cardiac arrest is suspected to be caused by PE (Class IIb, LOE C-LD).	updated for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	It is reasonable to provide opioid overdose response education, either alone or coupled with naloxone distribution and training, to persons at risk for opioid overdose (Class IIa, LOE C-LD).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	It is reasonable to base this training on first aid and non–healthcare provider BLS recommendations rather than on more advanced practices intended for healthcare providers (Class IIa, LOE C-E0).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	Empiric administration of IM or IN naloxone to all unresponsive opioid-associated life-threatening emergency patients may be reasonable as an adjunct to standard first aid and non–healthcare provider BLS protocols (Class IIb, LOE C-E0).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	Victims who respond to naloxone administration should access advanced healthcare services (Class I, LOE C-E0).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	For patients with known or suspected opioid overdose who have a definite pulse but no normal breathing or only gasping (ie, a respiratory arrest), in addition to providing standard BLS care, it is reasonable for appropriately trained BLS healthcare providers to administer IM or IN naloxone (Class IIa, LOE C-LD).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	Standard resuscitative measures should take priority over naloxone administration (Class I, LOE C-E0), with a focus on high-quality CPR (compressions plus ventilation).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	It may be reasonable to administer IM or IN naloxone based on the possibility that the patient is not in cardiac arrest (Class IIb, LOE C-E0).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	Responders should not delay access to more-advanced medical services while awaiting the patient's response to naloxone or other interventions (Class I, LOE C-E0).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	Unless the patient refuses further care, victims who respond to naloxone administration should access advanced healthcare services (Class I, LOE C-E0).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	Bag-mask ventilation should be maintained until spontaneous breathing returns, and standard ACLS measures should continue if return of spontaneous breathing does not occur (Class I, LOE C-LD).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	After ROSC or return of spontaneous breathing, patients should be observed in a healthcare setting until the risk of recurrent opioid toxicity is low and the patient's level of consciousness and vital signs have normalized (Class I, LOE C-LD).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	If recurrent opioid toxicity develops, repeated small doses or an infusion of naloxone can be beneficial in healthcare settings (Class IIa, LOE C-LD).	new for 2015
2015	Cardiac or Respiratory Arrest Associated With Opioid Overdose	Naloxone administration in post–cardiac arrest care may be considered in order to achieve the specific therapeutic goals of reversing the effects of long-acting opioids (Class IIb, LOE C-E0).	new for 2015

(Continued)

2015 Guidelines Update: Part 10 Recommendations, *Continued*

Year Last Reviewed	Topic	Recommendation	Comments
2015	Role of Intravenous Lipid Emulsion Therapy in Management of Cardiac Arrest Due to Poisoning	It may be reasonable to administer ILE, concomitant with standard resuscitative care, to patients with local anesthetic systemic toxicity and particularly to patients who have premonitory neurotoxicity or cardiac arrest due to bupivacaine toxicity (Class IIb, LOE C-E0).	updated for 2015
2015	Role of Intravenous Lipid Emulsion Therapy in Management of Cardiac Arrest Due to Poisoning	It may be reasonable to administer ILE to patients with other forms of drug toxicity who are failing standard resuscitative measures (Class IIb, LOE C-E0).	new for 2015
2015	Cardiac Arrest During Percutaneous Coronary Intervention	It may be reasonable to use mechanical CPR devices to provide chest compressions to patients in cardiac arrest during PCI (Class IIb, LOE C-E0).	updated for 2015
2015	Cardiac Arrest During Percutaneous Coronary Intervention	It may be reasonable to use ECPR as a rescue treatment when initial therapy is failing for cardiac arrest that occurs during PCI (Class IIb, LOE C-LD).	new for 2015
2015	Cardiac Arrest During Percutaneous Coronary Intervention	Institutional guidelines should include the selection of appropriate candidates for use of mechanical support devices to ensure that these devices are used as a bridge to recovery, surgery or transplant, or other device (Class I, LOE C-E0).	new for 2015
The following recommendations were not reviewed in 2015. For more information, see the <i>2010 AHA Guidelines for CPR and ECC</i> , "Part 12: Cardiac Arrest in Special Situations."			
2010	Cardiac Arrest Associated With Asthma	Therefore, since the effects of auto-PEEP in an asthmatic patient with cardiac arrest are likely quite severe, a ventilation strategy of low respiratory rate and tidal volume is reasonable (Class IIa, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Asthma	During arrest a brief disconnection from the bag mask or ventilator may be considered, and compression of the chest wall to relieve air-trapping can be effective (Class IIa, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Asthma	For all asthmatic patients with cardiac arrest, and especially for patients in whom ventilation is difficult, the possible diagnosis of a tension pneumothorax should be considered and treated (Class I, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	Given the potential for the rapid development of oropharyngeal or laryngeal edema, immediate referral to a health professional with expertise in advanced airway placement is recommended (Class I, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	Epinephrine should be administered early by IM injection to all patients with signs of a systemic allergic reaction, especially hypotension, airway swelling, or difficulty breathing (Class I, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	The recommended dose is 0.2 to 0.5 mg (1:1000) IM to be repeated every 5 to 15 minutes in the absence of clinical improvement (Class I, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	In both anaphylaxis and cardiac arrest the immediate use of an epinephrine autoinjector is recommended if available (Class I, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	Planning for advanced airway management, including a surgical airway, is recommended (Class I, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	Vasogenic shock from anaphylaxis may require aggressive fluid resuscitation (Class IIa, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	When an IV line is in place, it is reasonable to consider the IV route as an alternative to IM administration of epinephrine in anaphylactic shock (Class IIa, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	Because fatal overdose of epinephrine has been reported, close hemodynamic monitoring is recommended (Class I, LOE B).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	IV infusion of epinephrine is a reasonable alternative to IV boluses for treatment of anaphylaxis in patients not in cardiac arrest (Class IIa, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	Alternative vasoactive drugs (vasopressin, norepinephrine, methoxamine, and metaraminol) may be considered in cardiac arrest secondary to anaphylaxis that does not respond to epinephrine (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	Adjuvant use of antihistamines (H1 and H2 antagonist), inhaled β -adrenergic agents, and IV corticosteroids has been successful in management of the patient with anaphylaxis and may be considered in cardiac arrest due to anaphylaxis (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Anaphylaxis	Cardiopulmonary bypass has been successful in isolated case reports of anaphylaxis followed by cardiac arrest. Use of these advanced techniques may be considered in clinical situations where the required professional skills and equipment are immediately available (Class IIb, LOE C).	not reviewed in 2015

(Continued)

2015 Guidelines Update: Part 10 Recommendations, *Continued*

Year Last Reviewed	Topic	Recommendation	Comments
2010	Cardiac Arrest Associated With Pregnancy	Bag-mask ventilation with 100% oxygen before intubation is especially important in pregnancy (Class IIa, LOE B).	not reviewed in 2015
2010	Cardiac Arrest Associated With Pregnancy	If internal or external fetal monitors are attached during cardiac arrest in a pregnant woman, it is reasonable to remove them (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Pregnancy	Team planning should be done in collaboration with the obstetric, neonatal, emergency, anesthesiology, intensive care, and cardiac arrest services (Class I, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Pregnancy	During therapeutic hypothermia of the pregnant patient, it is recommended that the fetus be continuously monitored for bradycardia as a potential complication, and obstetric and neonatal consultation should be sought (Class I, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Pulmonary Embolism	In patients with cardiac arrest and without known PE, routine fibrinolytic treatment given during CPR shows no benefit and is not recommended (Class III, LOE A).	not reviewed in 2015
2010	Cardiac Arrest Associated With Life-Threatening Electrolyte Disturbances	When cardiac arrest occurs secondary to hyperkalemia, it may be reasonable to administer adjuvant IV therapy as outlined above for cardiotoxicity in addition to standard ACLS (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Life-Threatening Electrolyte Disturbances	The effect of bolus administration of potassium for cardiac arrest suspected to be secondary to hypokalemia is unknown and ill advised (Class III, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Life-Threatening Electrolyte Disturbances	Administration of calcium (calcium chloride [10%] 5 to 10 mL or calcium gluconate [10%] 15 to 30 mL IV over 2 to 5 minutes) may be considered during cardiac arrest associated with hypermagnesemia (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Life-Threatening Electrolyte Disturbances	For cardiotoxicity and cardiac arrest, IV magnesium 1 to 2 g of MgSO ₄ bolus IV push is recommended (Class I, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Life-Threatening Electrolyte Disturbances	Empirical use of calcium (calcium chloride [10%] 5 to 10 mL OR calcium gluconate [10%] 15 to 30 mL IV over 2 to 5 minutes) may be considered when hyperkalemia or hypermagnesemia is suspected as the cause of cardiac arrest (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	The administration of flumazenil to patients with undifferentiated coma confers risk and is not recommended (Class III, LOE B).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	The recommended dose of glucagon is a bolus of 3 to 10 mg, administered slowly over 3 to 5 minutes, followed by an infusion of 3 to 5 mg/h (0.05 to 0.15 mg/kg followed by an infusion of 0.05 to 0.10 mg/kg per hour) (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	Administration of high-dose insulin in patients with shock refractory to other measures may be considered (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	Administration of calcium in patients with shock refractory to other measures may be considered (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	High-dose insulin, in the doses listed in the β -blocker section above, may be effective for restoring hemodynamic stability and improving survival in the setting of severe cardiovascular toxicity associated with toxicity from a calcium channel blocker overdose (Class IIb, LOE B).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	Administration of calcium in patients with shock refractory to other measures may be considered (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	Antidigoxin Fab antibodies should be administered to patients with severe life-threatening cardiac glycoside toxicity (Class I, LOE B).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	It may be reasonable to try agents that have shown efficacy in the management of acute coronary syndrome in patients with severe cardiovascular toxicity. β -Blockers (phentolamine), benzodiazepines (lorazepam, diazepam), calcium channel blockers (verapamil), morphine, and sublingual nitroglycerin may be used as needed to control hypertension, tachycardia, and agitation (Class IIb, LOE B).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	The available data do not support the use of 1 agent over another in the treatment of cardiovascular toxicity due to cocaine (Class IIb, LOE B).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	For cocaine-induced hypertension or chest discomfort, benzodiazepines, nitroglycerin, and/or morphine can be beneficial (Class IIa, LOE B).	not reviewed in 2015

(Continued)

2015 Guidelines Update: Part 10 Recommendations, *Continued*

Year Last Reviewed	Topic	Recommendation	Comments
2010	Cardiac Arrest Associated With Toxic Ingestions	Although contradictory evidence exists, current recommendations are that pure β -blocker medications in the setting of cocaine are not indicated (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	Administration of sodium bicarbonate for cardiac arrest due to cyclic antidepressant overdose may be considered (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	Sodium bicarbonate boluses of 1 mL/kg may be administered as needed to achieve hemodynamic stability (adequate mean arterial blood pressure and perfusion) and QRS narrowing (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	Because hyperbaric oxygen therapy appears to confer little risk, the available data suggest that hyperbaric oxygen therapy may be helpful in treatment of acute carbon monoxide poisoning in patients with severe toxicity (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Associated With Toxic Ingestions	Based on the best evidence available, a treatment regimen of 100% oxygen and hydroxocobalamin, with or without sodium thiosulfate, is recommended (Class I, LOE B).	not reviewed in 2015
2010	Cardiac Arrest in Accidental Hypothermia	It may be reasonable to perform further defibrillation attempts according to the standard BLS algorithm concurrent with rewarming strategies (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest in Accidental Hypothermia	It may be reasonable to consider administration of a vasopressor during cardiac arrest according to the standard ACLS algorithm concurrent with rewarming strategies (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest in Avalanche Victims	Full resuscitative measures, including extracorporeal rewarming when available, are recommended for all avalanche victims without the characteristics outlined above that deem them unlikely to survive or with any obvious lethal traumatic injury (Class I, LOE C).	not reviewed in 2015
2010	Drowning	All victims of drowning who require any form of resuscitation (including rescue breathing alone) should be transported to the hospital for evaluation and monitoring, even if they appear to be alert and demonstrate effective cardiorespiratory function at the scene (Class I, LOE C).	not reviewed in 2015
2010	Drowning	Routine stabilization of the cervical spine in the absence of circumstances that suggest a spinal injury is not recommended (Class III, LOE B).	not reviewed in 2015
2010	Drowning	The routine use of abdominal thrusts or the Heimlich maneuver for drowning victims is not recommended (Class III, LOE C).	not reviewed in 2015
2010	Cardiac Arrest During Percutaneous Coronary Intervention	It is reasonable to use cough CPR during PCI (Class IIa, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Caused by Cardiac Tamponade	In the arrest setting, in the absence of echocardiography, emergency pericardiocentesis without imaging guidance can be beneficial (Class IIa, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Caused by Cardiac Tamponade	Emergency department thoracotomy may improve survival compared with pericardiocentesis in patients with pericardial tamponade secondary to trauma who are in cardiac arrest or who are prearrest, especially if gross blood causes clotting that blocks a pericardiocentesis needle (Class IIb, LOE C).	not reviewed in 2015
2010	Cardiac Arrest Following Cardiac Surgery	For patients with cardiac arrest following cardiac surgery, it is reasonable to perform re sternotomy in an appropriately staffed and equipped intensive care unit (Class IIa, LOE B).	not reviewed in 2015
2010	Cardiac Arrest Following Cardiac Surgery	Despite rare case reports describing damage to the heart possibly due to external chest compressions, chest compressions should not be withheld if emergency re sternotomy is not immediately available (Class IIa, LOE C).	not reviewed in 2015

References

- Vanden Hoek TL, Morrison LJ, Shuster M, Donnino M, Sinz E, Lavonas EJ, Jeejeebhoy FM, Gabrielli A. Part 12: cardiac arrest in special situations: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(suppl 3):S829–S861. doi: 10.1161/CIRCULATIONAHA.110.971069.
- Deakin CD, Morrison LJ, Morley PT, Callaway CW, Kerber RE, Kronick SL, Lavonas EJ, Link MS, Neumar RW, Otto CW, Parr M, Shuster M, Sunde K, Peberdy MA, Tang W, Hoek TL, Böttiger BW, Drajer S, Lim SH, Nolan JP; Advanced Life Support Chapter Collaborators. Part 8: advanced life support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation*. 2010;81 suppl 1:e93–e174. doi: 10.1016/j.resuscitation.2010.08.027.
- Hazinski MF, Nolan JP, Aickin R, Bhanji F, Billi JE, Callaway CW, Castren M, de Caen AR, Ferrer JME, Finn JC, Gent LM, Griffin RE, Iverson S, Lang E, Lim SH, Maconochie IK, Montgomery WH, Morley PT, Nadkarni VM, Neumar RW, Nikolaou NI, Perkins GD, Perlman JM, Singletary EM, Soar J, Travers AH, Welsford M, Wyllie J, Zideman DA. Part 1: executive summary: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation*. 2015;132(suppl 1):S2–S39. doi: 10.1161/CIR.0000000000000270.
- Nolan JP, Hazinski MF, Aickin R, Bhanji F, Billi JE, Callaway CW, Castren M, de Caen AR, Ferrer JME, Finn JC, Gent LM, Griffin RE, Iverson S, Lang E, Lim SH, Maconochie IK, Montgomery WH, Morley PT, Nadkarni VM, Neumar RW, Nikolaou NI, Perkins GD, Perlman JM, Singletary EM, Soar J, Travers AH, Welsford M, Wyllie J, Zideman DA. Part 1: executive summary: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation*. 2015. In press.
- Callaway CW, Soar J, Aibiki M, Böttiger BW, Brooks SC, Deakin CD, Donnino MW, Drajer S, Kloeck W, Morley PT, Morrison LJ, Neumar RW, Nicholson TC, Nolan JP, Okada K, O'Neil BJ, Paiva EF, Parr MJ, Wang TL, Witt J; on behalf of the Advanced Life Support Chapter Collaborators. Part 4: advanced life support: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation*. 2015;132(suppl 1):S84–S145. doi: 10.1161/CIR.0000000000000273.
- Soar J, Callaway CW, Aibiki M, Böttiger BW, Brooks SC, Deakin CD, Donnino MW, Drajer S, Kloeck W, Morley PT, Morrison LJ, Neumar RW, Nicholson TC, Nolan JP, Okada K, O'Neil BJ, Paiva EF, Parr MJ, Wang TL, Witt J; on behalf of the Advanced Life Support Chapter Collaborators. Part 4: advanced life support: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation*. 2015. In press.
- O'Connor D, Green S, Higgins J, eds. Chapter 5: defining the review questions and developing criteria for including studies. In: *The Cochrane Collaboration*. Higgins JPT, Green S, eds. *Cochrane Handbook for Systematic Reviews of Interventions*. Version 5.1.0. 2011. <http://handbook.cochrane.org/>. Accessed May 6, 2015.
- Schünemann H, Brożek J, Guyatt G, Oxman A. *GRADE Handbook*. 2013. <http://www.guidelinedevelopment.org/handbook/>. Accessed May 6, 2015.
- Morley PT, Lang E, Aickin R, Billi JE, Eigel B, Ferrer JME, Finn JC, Gent LM, Griffin RE, Hazinski MF, Maconochie IK, Montgomery WH, Morrison LJ, Nadkarni VM, Nikolaou NI, Nolan JP, Perkins GD, Sayre MR, Travers AH, Wyllie J, Zideman DA. Part 2: evidence evaluation and management of conflicts of interest: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation*. 2015;132(suppl 1):S40–S50. doi: 10.1161/CIR.0000000000000271.
- Lang E, Morley PT, Aickin R, Billi JE, Eigel B, Ferrer JME, Finn JC, Gent LM, Griffin RE, Hazinski MF, Maconochie IK, Montgomery WH, Morrison LJ, Nadkarni VM, Nikolaou NI, Nolan JP, Perkins GD, Sayre MR, Travers AH, Wyllie J, Zideman DA. Part 2: evidence evaluation and management of conflicts of interest: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation*. 2015. In press.
- Jeejeebhoy FM, Zelop CM, Lipman S, Carvalho B, Joglar J, Mhyre JM, Katz VL, Lapinsky SE, Einav S, Warnes CA, Page RL, Griffin RE, Jain A, Dainty KN, Arafeh J, Windrim R, Koren G, Callaway C; on behalf of the American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation, Council on Cardiovascular Diseases in the Young, and Council on Clinical Cardiology. Cardiac arrest in pregnancy: a scientific statement from the American Heart Association. *Circulation*. 2015. In press. doi: 10.1161/CIR.0000000000000300.
- Mhyre JM, Tsen LC, Einav S, Kuklina EV, Leffert LR, Bateman BT. Cardiac arrest during hospitalization for delivery in the United States, 1998–2011. *Anesthesiology*. 2014;120:810–818. doi: 10.1097/ALN.0000000000000159.
- Centers for Disease Control and Prevention. Pregnancy mortality surveillance system <http://www.cdc.gov/reproductivehealth/maternalinfanthealth/pmss.html>. Accessed April 2, 2015.
- Public Health Agency of Canada. Maternal mortality in Canada. http://sogc.org/wp-content/uploads/2014/05/REVISED_Mortality-EN-Final-PDF.pdf. Accessed April 2, 2015.
- Hogan MC, Foreman KJ, Naghavi M, Ahn SY, Wang M, Makela SM, Lopez AD, Lozano R, Murray CJL. Maternal mortality for 181 countries, 1980–2008: a systematic analysis of progress towards Millennium Development Goal 5. 2010. http://cdrwww.who.int/pmnch/topics/maternal/20100402_ihmarticle.pdf. Accessed April 2, 2015.
- Creanga AA, Berg CJ, Ko JY, Farr SL, Tong VT, Bruce FC, Callaghan WM. Maternal mortality and morbidity in the United States: where are we now? *J Womens Health (Larchmt)*. 2014;23:3–9. doi: 10.1089/jwh.2013.4617.
- Ueland K, Novy MJ, Peterson EN, Metcalfe J. Maternal cardiovascular dynamics. IV. The influence of gestational age on the maternal cardiovascular response to posture and exercise. *Am J Obstet Gynecol*. 1969;104:856–864.
- Goodwin AP, Pearce AJ. The human wedge. A manoeuvre to relieve aortocaval compression during resuscitation in late pregnancy. *Anaesthesia*. 1992;47:433–434.
- Rees GA, Willis BA. Resuscitation in late pregnancy. *Anaesthesia*. 1988;43:347–349.
- Cyna AM, Andrew M, Emmett RS, Middleton P, Simmons SW. Techniques for preventing hypotension during spinal anaesthesia for caesarean section. *Cochrane Database Syst Rev*. 2006;CD002251.
- Dijkman A, Huisman CM, Smit M, Schutte JM, Zwart JJ, van Roosmalen JJ, Oepkes D. Cardiac arrest in pregnancy: increasing use of perimortem caesarean section due to emergency skills training? *BJOG*. 2010;117:282–287. doi: 10.1111/j.1471-0528.2009.02461.x.
- Page-Rodriguez A, Gonzalez-Sanchez JA. Perimortem cesarean section of twin pregnancy: case report and review of the literature. *Acad Emerg Med*. 1999;6:1072–1074.
- Cardosi RJ, Porter KB. Cesarean delivery of twins during maternal cardiopulmonary arrest. *Obstet Gynecol*. 1998;92(4 Pt 2):695–697.
- Rees SG, Thurlow JA, Gardner IC, Scrutton MJ, Kinsella SM. Maternal cardiovascular consequences of positioning after spinal anaesthesia for Caesarean section: left 15 degree table tilt vs. left lateral. *Anaesthesia*. 2002;57:15–20.
- Mendonca C, Griffiths J, Ateleanu B, Collis RE. Hypotension following combined spinal-epidural anaesthesia for Caesarean section. Left lateral position vs. tilted supine position. *Anaesthesia*. 2003;58:428–431.
- Svinos H. Towards evidence based emergency medicine: best BETs from the Manchester Royal Infirmary. BET 1. Emergency caesarean section in cardiac arrest before the third trimester. *Emerg Med J*. 2008;25:764–765. doi: 10.1136/emj.2008.066860.
- Katz V, Balderston K, DeFreest M. Perimortem cesarean delivery: were our assumptions correct? *Am J Obstet Gynecol*. 2005;192:1916–1920; discussion 1920. doi: 10.1016/j.ajog.2005.02.038.
- Einav S, Kaufman N, Sela HY. Maternal cardiac arrest and perimortem caesarean delivery: evidence or expert-based? *Resuscitation*. 2012;83:1191–1200. doi: 10.1016/j.resuscitation.2012.05.005.
- Kam CW. Perimortem caesarean sections (PMCS). *J Accid Emerg Med*. 1994;11:57–58.
- Kupas DF, Harter SC, Vosk A. Out-of-hospital perimortem cesarean section. *Prehosp Emerg Care*. 1998;2:206–208.
- Oates S, Williams GL, Rees GA. Cardiopulmonary resuscitation in late pregnancy. *BMJ*. 1988;297:404–405.
- Jaff MR, McMurtry MS, Archer SL, Cushman M, Goldenberg N, Goldhaber SZ, Jenkins JS, Kline JA, Michaels AD, Thistlethwaite P, Vedantham S, White RJ, Zierler BK; American Heart Association Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation; American Heart Association Council on Peripheral Vascular Disease; American Heart Association Council on Arteriosclerosis, Thrombosis and Vascular Biology. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientific statement from the American

- Heart Association. *Circulation*. 2011;123:1788–1830. doi: 10.1161/CIR.0b013e318214914f.
33. Schmid C, Zietlow S, Wagner TO, Laas J, Borst HG. Fulminant pulmonary embolism: symptoms, diagnostics, operative technique, and results. *Ann Thorac Surg*. 1991;52:1102–1105; discussion 1105.
 34. Kucher N, Rossi E, De Rosa M, Goldhaber SZ. Massive pulmonary embolism. *Circulation*. 2006;113:577–582. doi: 10.1161/CIRCULATIONAHA.105.592592.
 35. Kasper W, Konstantinides S, Geibel A, Olschewski M, Heinrich F, Grosser KD, Rauber K, Iversen S, Redecker M, Kienast J. Management strategies and determinants of outcome in acute major pulmonary embolism: results of a multicenter registry. *J Am Coll Cardiol*. 1997;30:1165–1171.
 36. Janata K. Managing pulmonary embolism. *BMJ*. 2003;326:1341–1342. doi: 10.1136/bmj.326.7403.1341.
 37. Kürkciyan I, Meron G, Sterz F, Janata K, Domanovits H, Holzer M, Berzlanovich A, Bankl HC, Lagner AN. Pulmonary embolism as a cause of cardiac arrest: presentation and outcome. *Arch Intern Med*. 2000;160:1529–1535.
 38. Hess EP, Campbell RL, White RD. Epidemiology, trends, and outcome of out-of-hospital cardiac arrest of non-cardiac origin. *Resuscitation*. 2007;72:200–206. doi: 10.1016/j.resuscitation.2006.06.040.
 39. Courtney DM, Kline JA. Identification of prearrest clinical factors associated with outpatient fatal pulmonary embolism. *Acad Emerg Med*. 2001;8:1136–1142.
 40. Courtney DM, Kline JA. Prospective use of a clinical decision rule to identify pulmonary embolism as likely cause of outpatient cardiac arrest. *Resuscitation*. 2005;65:57–64. doi: 10.1016/j.resuscitation.2004.07.018.
 41. Comess KA, DeRook FA, Russell ML, Tognazzi-Evans TA, Beach KW. The incidence of pulmonary embolism in unexplained sudden cardiac arrest with pulseless electrical activity. *Am J Med*. 2000;109:351–356.
 42. Aghayev A, Furlan A, Patil A, Gumus S, Jeon KN, Park B, Bae KT. The rate of resolution of clot burden measured by pulmonary CT angiography in patients with acute pulmonary embolism. *AJR Am J Roentgenol*. 2013;200:791–797. doi: 10.2214/AJR.12.8624.
 43. Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. *Chest*. 2002;121:877–905.
 44. Neumar RW, Otto CW, Link MS, Kronick SL, Shuster M, Callaway CW, Kudenchuk PJ, Ornato JP, McNally B, Silvers SM, Passman RS, White RD, Hess EP, Tang W, Davis D, Sinz E, Morrison LJ. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(suppl 3):S729–S767. doi: 10.1161/CIRCULATIONAHA.110.970988.
 45. Böttiger BW, Böhrer H, Bach A, Motsch J, Martin E. Bolus injection of thrombolytic agents during cardiopulmonary resuscitation for massive pulmonary embolism. *Resuscitation*. 1994;28:45–54.
 46. Er F, Nia AM, Gassanov N, Caglayan E, Erdmann E, Hoppe UC. Impact of rescue-thrombolysis during cardiopulmonary resuscitation in patients with pulmonary embolism. *PLoS One*. 2009;4:e8323. doi: 10.1371/journal.pone.0008323.
 47. Lederer W, Lichtenberger C, Pechlaner C, Kroesen G, Baubin M. Recombinant tissue plasminogen activator during cardiopulmonary resuscitation in 108 patients with out-of-hospital cardiac arrest. *Resuscitation*. 2001;50:71–76.
 48. Böttiger BW, Martin E. Thrombolytic therapy during cardiopulmonary resuscitation and the role of coagulation activation after cardiac arrest. *Curr Opin Crit Care*. 2001;7:176–183.
 49. Ruiz-Bailén M, Aguayo-de-Hoyos E, Serrano-Córcoles MC, Díaz-Castellanos MA, Fierro-Rosón JL, Ramos-Cuadra JA, Rodríguez-Elvira M, Torres-Ruiz JM. Thrombolysis with recombinant tissue plasminogen activator during cardiopulmonary resuscitation in fulminant pulmonary embolism. A case series. *Resuscitation*. 2001;51:97–101.
 50. Janata K, Holzer M, Kürkciyan I, Losert H, Riedmüller E, Pikula B, Lagner AN, Laczika K. Major bleeding complications in cardiopulmonary resuscitation: the place of thrombolytic therapy in cardiac arrest due to massive pulmonary embolism. *Resuscitation*. 2003;57:49–55.
 51. Lederer W, Lichtenberger C, Pechlaner C, Kinzl J, Kroesen G, Baubin M. Long-term survival and neurological outcome of patients who received recombinant tissue plasminogen activator during out-of-hospital cardiac arrest. *Resuscitation*. 2004;61:123–129. doi: 10.1016/j.resuscitation.2003.12.016.
 52. Stadlbauer KH, Krismer AC, Arntz HR, Mayr VD, Lienhart HG, Böttiger BW, Jahn B, Lindner KH, Wenzel V. Effects of thrombolysis during out-of-hospital cardiopulmonary resuscitation. *Am J Cardiol*. 2006;97:305–308. doi: 10.1016/j.amjcard.2005.08.045.
 53. Renard A, Verret C, Jost D, Meynard JB, Tricheureau J, Hersan O, Fontaine D, Briche F, Benner P, de Stabenrath O, Bartou C, Segal N, Domanski L. Impact of fibrinolysis on immediate prognosis of patients with out-of-hospital cardiac arrest. *J Thromb Thrombolysis*. 2011;32:405–409. doi: 10.1007/s11239-011-0619-0.
 54. Dirican A, Ozkaya S, Atas AE, Ulu EK, Kitapci I, Ece F. Thrombolytic treatment (alteplase; rt-PA) in acute massive pulmonary embolism and cardiopulmonary arrest. *Drug Des Devel Ther*. 2014;8:759–763. doi: 10.2147/DDDT.S61679.
 55. Böttiger BW, Arntz HR, Chamberlain DA, Bluhmki E, Belmans A, Danays T, Carli PA, Adgey JA, Bode C, Wenzel V; TROICA Trial Investigators; European Resuscitation Council Study Group. Thrombolysis during resuscitation for out-of-hospital cardiac arrest. *N Engl J Med*. 2008;359:2651–2662. doi: 10.1056/NEJMoa070570.
 56. Böttiger BW, Bode C, Kern S, Gries A, Gust R, Glätzer R, Bauer H, Motsch J, Martin E. Efficacy and safety of thrombolytic therapy after initially unsuccessful cardiopulmonary resuscitation: a prospective clinical trial. *Lancet*. 2001;357:1583–1585. doi: 10.1016/S0140-6736(00)04726-7.
 57. Logan JK, Pantle H, Huiras P, Bessman E, Bright L. Evidence-based diagnosis and thrombolytic treatment of cardiac arrest or periarrest due to suspected pulmonary embolism. *Am J Emerg Med*. 2014;32:789–796. doi: 10.1016/j.ajem.2014.04.032.
 58. Konstantinov IE, Saxena P, Koniuszko MD, Alvarez J, Newman MA. Acute massive pulmonary embolism with cardiopulmonary resuscitation: management and results. *Tex Heart Inst J*. 2007;34:41–45; discussion 45.
 59. Doerge HC, Schoendube FA, Loeser H, Walter M, Messmer BJ. Pulmonary embolectomy: review of a 15-year experience and role in the age of thrombolytic therapy. *Eur J Cardiothorac Surg*. 1996;10:952–957.
 60. Fava M, Loyola S, Bertoni H, Dougnac A. Massive pulmonary embolism: percutaneous mechanical thrombectomy during cardiopulmonary resuscitation. *J Vasc Interv Radiol*. 2005;16:119–123. doi: 10.1097/01.RVI.0000146173.85401.BA.
 61. Centers for Disease Control and Prevention. Injury prevention and control: prescription drug overdose. <http://www.cdc.gov/drugoverdose/index.html>. Accessed March 17, 2015.
 62. Hedegaard H, Chen LH, Warner M. Drug-poisoning deaths involving heroin: United States, 2000–2013. <http://www.cdc.gov/nchs/data/databriefs/db190.htm>. Accessed March 17, 2015.
 63. Centers for Disease Control and Prevention. Fatal injury data. <http://www.cdc.gov/injury/wisqars/fatal.html>. Accessed April 4, 2015.
 64. Carter CI, Graham B. Opioid overdose prevention and response in Canada. http://drugpolicy.ca/wp-content/uploads/2014/07/CDPC_OverdosePreventionPolicy_Final_July2014.pdf. Accessed March 17, 2015.
 65. Jones CM, Paulozzi LJ, Mack KA; Centers for Disease Control and Prevention (CDC). Alcohol involvement in opioid pain reliever and benzodiazepine drug abuse-related emergency department visits and drug-related deaths - United States, 2010. *MMWR Morb Mortal Wkly Rep*. 2014;63:881–885.
 66. Madadi P, Hildebrandt D, Lauwers AE, Koren G. Characteristics of opioid-users whose death was related to opioid-toxicity: a population-based study in Ontario, Canada. *PLoS One*. 2013;8:e60600. doi: 10.1371/journal.pone.0060600.
 67. Webster LR, Cochella S, Dasgupta N, Fakata KL, Fine PG, Fishman SM, Grey T, Johnson EM, Lee LK, Passik SD, Peppin J, Porucznik CA, Ray A, Schnoll SH, Stieg RL, Wakeland W. An analysis of the root causes for opioid-related overdose deaths in the United States. *Pain Med*. 2011;12 suppl 2:S26–S35. doi: 10.1111/j.1526-4637.2011.01134.x.
 68. Paulozzi LJ, Logan JE, Hall AJ, McKinstry E, Kaplan JA, Crosby AE. A comparison of drug overdose deaths involving methadone and other opioid analgesics in West Virginia. *Addiction*. 2009;104:1541–1548. doi: 10.1111/j.1360-0443.2009.02650.x.
 69. Krantz MJ, Kutinsky IB, Robertson AD, Mehler PS. Dose-related effects of methadone on QT prolongation in a series of patients with torsade de pointes. *Pharmacotherapy*. 2003;23:802–805.
 70. Eap CB, Crettol S, Rougier JS, Schlöpfer J, Sintra Grilo L, Déglon JJ, Besson J, Croquette-Krokar M, Carrupt PA, Abriel H. Stereoselective block of hERG channel by (S)-methadone and QT interval prolongation in CYP2B6 slow metabolizers. *Clin Pharmacol Ther*. 2007;81:719–728. doi: 10.1038/sj.clpt.6100120.
 71. Krantz MJ, Martin J, Stimmel B, Mehta D, Haigney MC. QTc interval screening in methadone treatment. *Ann Intern Med*. 2009;150:387–395.

72. Stallvik M, Nordstrand B, Kristensen Ø, Bathen J, Skogvoll E, Spigset O. Corrected QT interval during treatment with methadone and buprenorphine—relation to doses and serum concentrations. *Drug Alcohol Depend.* 2013;129:88–93. doi: 10.1016/j.drugalcdep.2012.09.016.
73. Chou R, Weimer MB, Dana T. Methadone overdose and cardiac arrhythmia potential: findings from a review of the evidence for an American Pain Society and College on Problems of Drug Dependence clinical practice guideline. *J Pain.* 2014;15:338–365. doi: 10.1016/j.jpain.2014.01.495.
74. Lipski J, Stimmel B, Donoso E. The effect of heroin and multiple drug abuse on the electrocardiogram. *Am Heart J.* 1973;86:663–668.
75. Labi M. Paroxysmal atrial fibrillation in heroin intoxication. *Ann Intern Med.* 1969;71:951–959.
76. Bahr J, Klingler H, Panzer W, Rode H, Kettler D. Skills of lay people in checking the carotid pulse. *Resuscitation.* 1997;35:23–26.
77. Eberle B, Dick WF, Schneider T, Wissner G, Doetsch S, Tzanova I. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. *Resuscitation.* 1996;33:107–116.
78. Leach M. Naloxone: a new therapeutic and diagnostic agent for emergency use. *JACEP.* 1973;2:21–23.
79. Sporer KA, Firestone J, Isaacs SM. Out-of-hospital treatment of opioid overdoses in an urban setting. *Acad Emerg Med.* 1996;3:660–667.
80. Robertson TM, Hendey GW, Stroh G, Shalit M. Intranasal naloxone is a viable alternative to intravenous naloxone for prehospital narcotic overdose. *Prehosp Emerg Care.* 2009;13:512–515. doi: 10.1080/10903120903144866.
81. Evans LE, Swainson CP, Roscoe P, Prescott LF. Treatment of drug overdosage with naloxone, a specific narcotic antagonist. *Lancet.* 1973;1:452–455.
82. Kelly AM, Kerr D, Dietze P, Patrick I, Walker T, Koutsogiannis Z. Randomised trial of intranasal versus intramuscular naloxone in prehospital treatment for suspected opioid overdose. *Med J Aust.* 2005;182:24–27.
83. Barton ED, Colwell CB, Wolfe T, Fosnocht D, Gravitz C, Bryan T, Dunn W, Benson J, Bailey J. Efficacy of intranasal naloxone as a needleless alternative for treatment of opioid overdose in the prehospital setting. *J Emerg Med.* 2005;29:265–271. doi: 10.1016/j.jemermed.2005.03.007.
84. Wolfe TR, Braude DA. Intranasal medication delivery for children: a brief review and update. *Pediatrics.* 2010;126:532–537. doi: 10.1542/peds.2010-0616.
85. Loimer N, Hofmann P, Chaudhry HR. Nasal administration of naloxone is as effective as the intravenous route in opiate addicts. *Int J Addict.* 1994;29:819–827.
86. Doe-Simkins M, Walley AY, Epstein A, Moyer P. Saved by the nose: bystander-administered intranasal naloxone hydrochloride for opioid overdose. *Am J Public Health.* 2009;99:788–791. doi: 10.2105/AJPH.2008.146647.
87. Wanger K, Brough L, Macmillan I, Goulding J, MacPhail I, Christenson JM. Intravenous vs subcutaneous naloxone for out-of-hospital management of presumed opioid overdose. *Acad Emerg Med.* 1998;5:293–299.
88. Baumann BM, Patterson RA, Parone DA, Jones MK, Glaspey LJ, Thompson NM, Stauss MP, Haroz R. Use and efficacy of nebulized naloxone in patients with suspected opioid intoxication. *Am J Emerg Med.* 2013;31:585–588. doi: 10.1016/j.ajem.2012.10.004.
89. Weber JM, Tataris KL, Hoffman JD, Aks SE, Mycyk MB. Can nebulized naloxone be used safely and effectively by emergency medical services for suspected opioid overdose? *Prehosp Emerg Care.* 2012;16:289–292. doi: 10.3109/10903127.2011.640763.
90. Greenberg MI, Roberts JR, Baskin SI. Endotracheal naloxone reversal of morphine-induced respiratory depression in rabbits. *Ann Emerg Med.* 1980;9:289–292.
91. Posner J, Burke CA. The effects of naloxone on opiate and placebo analgesia in healthy volunteers. *Psychopharmacology (Berl).* 1985;87:468–472.
92. Borrás MC, Becerra L, Ploghaus A, Gostic JM, DaSilva A, Gonzalez RG, Borsook D. fMRI measurement of CNS responses to naloxone infusion and subsequent mild noxious thermal stimuli in healthy volunteers. *J Neurophysiol.* 2004;91:2723–2733. doi: 10.1152/jn.00249.2003.
93. Clarke SF, Dargan PI, Jones AL. Naloxone in opioid poisoning: walking the tightrope. *Emerg Med J.* 2005;22:612–616. doi: 10.1136/emj.2003.009613.
94. Walley AY, Doe-Simkins M, Quinn E, Pierce C, Xuan Z, Ozonoff A. Opioid overdose prevention with intranasal naloxone among people who take methadone. *J Subst Abuse Treat.* 2013;44:241–247. doi: 10.1016/j.jsat.2012.07.004.
95. Clark AK, Wilder CM, Winstanley EL. A systematic review of community opioid overdose prevention and naloxone distribution programs. *J Addict Med.* 2014;8:153–163. doi: 10.1097/ADM.0000000000000034.
96. Lagu T, Anderson BJ, Stein M. Overdoses among friends: drug users are willing to administer naloxone to others. *J Subst Abuse Treat.* 2006;30:129–133. doi: 10.1016/j.jsat.2005.05.010.
97. Tracy M, Piper TM, Ompad D, Bucciarelli A, Coffin PO, Vlahov D, Galea S. Circumstances of witnessed drug overdose in New York City: implications for intervention. *Drug Alcohol Depend.* 2005;79:181–190. doi: 10.1016/j.drugalcdep.2005.01.010.
98. US Food and Drug Administration. *FDA News Release: FDA approves new hand-held auto-injector to reverse opioid overdose.* 2015. <http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm391465.htm>. Accessed May 11, 2015.
99. Tataris KL, Weber JM, Stein-Spencer L, Aks SE. The effect of prehospital nebulized naloxone on suspected heroin-induced bronchospasm. *Am J Emerg Med.* 2013;31:717–718. doi: 10.1016/j.ajem.2012.11.025.
100. Bingham K, Klaiman M, Leece P, Orkin A, Buick J. An agenda for naloxone distribution research and practice: Meeting report of the Surviving Opioid Overdose with Naloxone (SOON) International Working Group. *J Addict Res Ther.* 2015;6:212.
101. Kerr D, Kelly AM, Dietze P, Jolley D, Barger B. Randomized controlled trial comparing the effectiveness and safety of intranasal and intramuscular naloxone for the treatment of suspected heroin overdose. *Addiction.* 2009;104:2067–2074. doi: 10.1111/j.1360-0443.2009.02724.x.
102. Boyd JJ, Kuisma MJ, Alaspää AO, Vuori E, Repo JV, Randell TT. Recurrent opioid toxicity after pre-hospital care of presumed heroin overdose patients. *Acta Anaesthesiol Scand.* 2006;50:1266–1270. doi: 10.1111/j.1399-6576.2006.01172.x.
103. Buajordet I, Naess AC, Jacobsen D, Brørs O. Adverse events after naloxone treatment of episodes of suspected acute opioid overdose. *Eur J Emerg Med.* 2004;11:19–23.
104. Cantwell K, Dietze P, Flander L. The relationship between naloxone dose and key patient variables in the treatment of non-fatal heroin overdose in the prehospital setting. *Resuscitation.* 2005;65:315–319. doi: 10.1016/j.resuscitation.2004.12.012.
105. Cetrullo C, Di Nino GF, Melloni C, Pieri C, Zanoni A. [Naloxone antagonism toward opiate analgesic drugs. Clinical experimental study]. *Minerva Anestesiol.* 1983;49:199–204.
106. Nielsen K, Nielsen SL, Siersma V, Rasmussen LS. Treatment of opioid overdose in a physician-based prehospital EMS: frequency and long-term prognosis. *Resuscitation.* 2011;82:1410–1413. doi: 10.1016/j.resuscitation.2011.05.027.
107. Osterwalder JJ. Naloxone—for intoxications with intravenous heroin and heroin mixtures—harmless or hazardous? A prospective clinical study. *J Toxicol Clin Toxicol.* 1996;34:409–416.
108. Stokland O, Hansen TB, Nilsen JE. [Prehospital treatment of heroin intoxication in Oslo in 1996]. *Tidsskr Nor Lægeforen.* 1998;118:3144–3146.
109. Wampler DA, Molina DK, McManus J, Laws P, Manifold CA. No deaths associated with patient refusal of transport after naloxone-reversed opioid overdose. *Prehosp Emerg Care.* 2011;15:320–324. doi: 10.3109/10903127.2011.569854.
110. Saybolt MD, Alter SM, Dos Santos F, Calello DP, Rynn KO, Nelson DA, Merlin MA. Naloxone in cardiac arrest with suspected opioid overdoses. *Resuscitation.* 2010;81:42–46. doi: 10.1016/j.resuscitation.2009.09.016.
111. Stoové MA, Dietze PM, Jolley D. Overdose deaths following previous non-fatal heroin overdose: record linkage of ambulance attendance and death registry data. *Drug Alcohol Rev.* 2009;28:347–352. doi: 10.1111/j.1465-3362.2009.00057.x.
112. Chan GM, Stajic M, Marker EK, Hoffman RS, Nelson LS. Testing positive for methadone and either a tricyclic antidepressant or a benzodiazepine is associated with an accidental overdose death: analysis of medical examiner data. *Acad Emerg Med.* 2006;13:543–547. doi: 10.1197/j.aem.2005.12.011.
113. Substance Abuse and Mental Health Services Administration. *Drug Abuse Warning Network, 2010: national estimates of drug-related emergency department visits.* 2010. <http://archive.samhsa.gov/data/2k13/DAWN2k10ED/DAWN2k10ED.pdf>. Accessed May 13, 2013.
114. Binswanger IA, Stern MF, Deyo RA, Heagerty PJ, Cheadle A, Elmore JG, Koepsell TD. Release from prison—a high risk of death for former inmates. *N Engl J Med.* 2007;356:157–165. doi: 10.1056/NEJMs064115.
115. Davoli M, Bargagli AM, Perucci CA, Schifano P, Belleudi V, Hickman M, Salamina G, Diecidue R, Vigna-Taglianti F, Faggiano

- F; VEdeTTE Study Group. Risk of fatal overdose during and after specialist drug treatment: the VEdeTTE study, a national multi-site prospective cohort study. *Addiction*. 2007;102:1954–1959. doi: 10.1111/j.1360-0443.2007.02025.x.
116. Caplehorn JR. Deaths in the first two weeks of maintenance treatment in NSW in 1994: identifying cases of iatrogenic methadone toxicity. *Drug Alcohol Rev*. 1998;17:9–17. doi: 10.1080/09595239800187551.
 117. Woody GE, Kane V, Lewis K, Thompson R. Premature deaths after discharge from methadone maintenance: a replication. *J Addict Med*. 2007;1:180–185. doi: 10.1097/ADM.0b013e318155980e.
 118. Gomes T, Mamdani MM, Dhalla IA, Paterson JM, Juurlink DN. Opioid dose and drug-related mortality in patients with nonmalignant pain. *Arch Intern Med*. 2011;171:686–691. doi: 10.1001/archinternmed.2011.117.
 119. Degenhardt L, Bucello C, Mathers B, Briegleb C, Ali H, Hickman M, McLaren J. Mortality among regular or dependent users of heroin and other opioids: a systematic review and meta-analysis of cohort studies. *Addiction*. 2011;106:32–51. doi: 10.1111/j.1360-0443.2010.03140.x.
 120. Vilke GM, Sloane C, Smith AM, Chan TC. Assessment for deaths in out-of-hospital heroin overdose patients treated with naloxone who refuse transport. *Acad Emerg Med*. 2003;10:893–896.
 121. Rudolph SS, Jehu G, Nielsen SL, Nielsen K, Siersma V, Rasmussen LS. Prehospital treatment of opioid overdose in Copenhagen—is it safe to discharge on-scene? *Resuscitation*. 2011;82:1414–1418. doi: 10.1016/j.resuscitation.2011.06.027.
 122. Moss ST, Chan TC, Buchanan J, Dunford JV, Vilke GM. Outcome study of prehospital patients signed out against medical advice by field paramedics. *Ann Emerg Med*. 1998;31:247–250.
 123. Christenson J, Etherington J, Grafstein E, Innes G, Pennington S, Wanger K, Fernandes C, Spinelli JJ, Gao M. Early discharge of patients with presumed opioid overdose: development of a clinical prediction rule. *Acad Emerg Med*. 2000;7:1110–1118.
 124. Etherington J, Christenson J, Innes G, Grafstein E, Pennington S, Spinelli JJ, Gao M, Lahiffé B, Wanger K, Fernandes C. Is early discharge safe after naloxone reversal of presumed opioid overdose? *CJEM*. 2000;2:156–162.
 125. Zuckerman M, Weisberg SN, Boyer EW. Pitfalls of intranasal naloxone. *Prehosp Emerg Care*. 2014;18:550–554. doi: 10.3109/10903127.2014.896961.
 126. Weinberg GL, VadeBoncouer T, Ramaraju GA, Garcia-Amaro MF, Cwik MJ. Pretreatment or resuscitation with a lipid infusion shifts the dose-response to bupivacaine-induced asystole in rats. *Anesthesiology*. 1998;88:1071–1075.
 127. Weinberg G, Ripper R, Feinstein DL, Hoffman W. Lipid emulsion infusion rescues dogs from bupivacaine-induced cardiac toxicity. *Reg Anesth Pain Med*. 2003;28:198–202. doi: 10.1053/rapm.2003.50041.
 128. Rosenblatt MA, Abel M, Fischer GW, Itzkovich CJ, Eisenkraft JB. Successful use of a 20% lipid emulsion to resuscitate a patient after a presumed bupivacaine-related cardiac arrest. *Anesthesiology*. 2006;105:217–218.
 129. Barrington MJ, Kluger R. Ultrasound guidance reduces the risk of local anesthetic systemic toxicity following peripheral nerve blockade. *Reg Anesth Pain Med*. 2013;38:289–297. doi: 10.1097/AAP.0b013e318292669b.
 130. Neal JM, Mulroy MF, Weinberg GL; American Society of Regional Anesthesia and Pain Medicine. American Society of Regional Anesthesia and Pain Medicine checklist for managing local anesthetic systemic toxicity: 2012 version. *Reg Anesth Pain Med*. 2012;37:16–18. doi: 10.1097/AAP.0b013e31822e0d8a.
 131. Weinberg G, Lin B, Zheng S, Di Gregorio G, Hiller D, Ripper R, Edelman L, Kelly K, Feinstein D. Partitioning effect in lipid resuscitation: further evidence for the lipid sink. *Crit Care Med*. 2010;38:2268–2269. doi: 10.1097/CCM.0b013e3181f17d85.
 132. Weinberg GL, Palmer JW, VadeBoncouer TR, Zuechner MB, Edelman G, Hoppel CL. Bupivacaine inhibits acylcarnitine exchange in cardiac mitochondria. *Anesthesiology*. 2000;92:523–528.
 133. Fettiplace MR, Akpa BS, Ripper R, Zider B, Lang J, Rubinstein I, Weinberg G. Resuscitation with lipid emulsion: dose-dependent recovery from cardiac pharmacotoxicity requires a cardiotoxic effect. *Anesthesiology*. 2014;120:915–925. doi: 10.1097/ALN.0000000000000142.
 134. Harvey M, Cave G. Lipid rescue: does the sink hold water? And other controversies. *Br J Anaesth*. 2014;112:622–625. doi: 10.1093/bja/aeu010.
 135. Turner-Lawrence DE, Kerns Ii W. Intravenous fat emulsion: a potential novel antidote. *J Med Toxicol*. 2008;4:109–114.
 136. Jamaty C, Bailey B, Larocque A, Notebaert E, Sanogo K, Chauny JM. Lipid emulsions in the treatment of acute poisoning: a systematic review of human and animal studies. *Clin Toxicol (Phila)*. 2010;48:1–27. doi: 10.3109/15563650903544124.
 137. Weinberg GL. Lipid emulsion infusion: resuscitation for local anesthetic and other drug overdose. *Anesthesiology*. 2012;117:180–187. doi: 10.1097/ALN.0b013e31825ad8de.
 138. Cao D, Heard K, Foran M, Koyfman A. Intravenous lipid emulsion in the emergency department: a systematic review of the literature. *J Emerg Med*. 2015;48:387–397. doi: 10.1016/j.jemermed.2014.10.009.
 139. Taftachi F, Sanaei-Zadeh H, Sepehrian B, Zamani N. Lipid emulsion improves Glasgow coma scale and decreases blood glucose level in the setting of acute non-local anesthetic drug poisoning—a randomized controlled trial. *Eur Rev Med Pharmacol Sci*. 2012;16 suppl 1:38–42.
 140. Gil HW, Park JS, Park SH, Hong SY. Effect of intravenous lipid emulsion in patients with acute glyphosate intoxication. *Clin Toxicol (Phila)*. 2013;51:767–771. doi: 10.3109/15563650.2013.821129.
 141. Cave G, Harvey M, Willers J, Uncles D, Meek T, Picard J, Weinberg G. LIPAEMIC report: results of clinical use of intravenous lipid emulsion in drug toxicity reported to an online lipid registry. *J Med Toxicol*. 2014;10:133–142. doi: 10.1007/s13181-013-0375-y.
 142. Downes MA, Calver LA, Isbister GK. Intralipid therapy does not improve level of consciousness in overdoses with sedating drugs: a case series. *Emerg Med Australas*. 2014;26:286–290. doi: 10.1111/1742-6723.12237.
 143. Weinberg GL, Di Gregorio G, Ripper R, Kelly K, Massad M, Edelman L, Schwartz D, Shah N, Zheng S, Feinstein DL. Resuscitation with lipid versus epinephrine in a rat model of bupivacaine overdose. *Anesthesiology*. 2008;108:907–913. doi: 10.1097/ALN.0b013e31816d91d2.
 144. Weinberg G. LipidRescue resuscitation for drug toxicity. <http://www.lipidrescue.org/>. Accessed March 19, 2015.
 145. American Society of Regional Anesthesia and Pain Medicine. Checklist for treatment of local anesthetic systemic toxicity. <https://www.asra.com/advisory-guidelines/article/3/checklist-for-treatment-of-local-anesthetic-systemic-toxicity>. Accessed March 19, 2015.
 146. American College of Medical Toxicology. ACMT position statement: interim guidance for the use of lipid resuscitation therapy. *J Med Toxicol*. 2011;7:81–82.
 147. Fettiplace MR, Akpa BS, Rubinstein I, Weinberg G. Confusion about infusion: rational volume limits for intravenous lipid emulsion during treatment of oral overdoses. *Ann Emerg Med*. 2015;66:185–188. doi: 10.1016/j.annemergmed.2015.01.020.
 148. Levine M, Skolnik AB, Ruha AM, Bosak A, Menke N, Pizon AF. Complications following antidotal use of intravenous lipid emulsion therapy. *J Med Toxicol*. 2014;10:10–14. doi: 10.1007/s13181-013-0356-1.
 149. Carreiro S, Blum J, Jay G, Hack JB. Intravenous lipid emulsion alters the hemodynamic response to epinephrine in a rat model. *J Med Toxicol*. 2013;9:220–225. doi: 10.1007/s13181-013-0291-1.
 150. Harvey M, Cave G, Prince G, Lahner D. Epinephrine injection in lipid-based resuscitation from bupivacaine-induced cardiac arrest: transient circulatory return in rabbits. *Anesth Analg*. 2010;111:791–796. doi: 10.1213/ANE.0b013e3181e66050.
 151. Neal JM, Bernards CM, Butterworth JF 4th, Di Gregorio G, Drasner K, Hejtmanek MR, Mulroy MF, Rosenquist RW, Weinberg GL. ASRA practice advisory on local anesthetic systemic toxicity. *Reg Anesth Pain Med*. 2010;35:152–161. doi: 10.1097/AAP.0b013e3181d22fed.
 152. American Society of Regional Anesthesia and Pain Medicine. Checklist for Treatment of Local Anesthetic Systemic Toxicity. 2011. <https://www.asra.com/content/documents/checklist-for-local-anesthetic-toxicity-treatment-1-18-12.pdf>. Accessed May 11, 2015.
 153. Cole JB, Stellpflug SJ, Engebretsen KM. Asystole immediately following intravenous fat emulsion for overdose. *J Med Toxicol*. 2014;10:307–310. doi: 10.1007/s13181-014-0382-7.
 154. Lee HM, Archer JR, Dargan PI, Wood DM. What are the adverse effects associated with the combined use of intravenous lipid emulsion and extracorporeal membrane oxygenation in the poisoned patient? *Clin Toxicol (Phila)*. 2015;53:145–150. doi: 10.3109/15563650.2015.1004582.
 155. Webb JG, Solankhi NK, Chugh SK, Amin H, Buller CE, Ricci DR, Humphries K, Penn IM, Carere R. Incidence, correlates, and outcome of cardiac arrest associated with percutaneous coronary intervention. *Am J Cardiol*. 2002;90:1252–1254.
 156. Mehta RH, Harjai KJ, Grines L, Stone GW, Boura J, Cox D, O'Neill W, Grines CL; Primary Angioplasty in Myocardial Infarction (PAMI) Investigators. Sustained ventricular tachycardia or fibrillation in the cardiac catheterization laboratory among patients receiving primary percutaneous coronary intervention: incidence, predictors, and outcomes. *J Am Coll Cardiol*. 2004;43:1765–1772. doi: 10.1016/j.jacc.2003.09.072.
 157. Martin-Yuste V, Alvarez-Contreras L, Brugaletta S, Ferreira-Gonzalez I, Cola C, Garcia-Picart J, Martí V, Sabate M. Emergent versus elective

- percutaneous stent implantation in the unprotected left main: long-term outcomes from a single-center registry. *J Invasive Cardiol*. 2011;23:392–397.
158. Sprung J, Ritter MJ, Rihal CS, Warner ME, Wilson GA, Williams BA, Stevens SR, Schroeder DR, Bourke DL, Warner DO. Outcomes of cardiopulmonary resuscitation and predictors of survival in patients undergoing coronary angiography including percutaneous coronary interventions. *Anesth Analg*. 2006;102:217–224. doi: 10.1213/01.ane.0000189082.54614.26.
 159. Addala S, Kahn JK, Moccia TF, Harjai K, Pellizon G, Ochoa A, O'Neill WW. Outcome of ventricular fibrillation developing during percutaneous coronary interventions in 19,497 patients without cardiogenic shock. *Am J Cardiol*. 2005;96:764–765. doi: 10.1016/j.amjcard.2005.04.057.
 160. O'Gara PT, Kushner FG, Ascheim DD, Casey DE Jr, Chung MK, de Lemos JA, Ettinger SM, Fang JC, Fesmire FM, Franklin BA, Granger CB, Krumholz HM, Linderbaum JA, Morrow DA, Newby LK, Ornato JP, Ou N, Radford MJ, Tamis-Holland JE, Tommaso CL, Tracy CM, Woo YJ, Zhao DX. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2013;127:e362–e425. doi: 10.1161/CIR.0b013e3182742cf6.
 161. Groggaard HK, Wik L, Eriksen M, Brekke M, Sunde K. Continuous mechanical chest compressions during cardiac arrest to facilitate restoration of coronary circulation with percutaneous coronary intervention. *J Am Coll Cardiol*. 2007;50:1093–1094. doi: 10.1016/j.jacc.2007.05.028.
 162. Agostoni P, Cornelis K, Vermeersch P. Successful percutaneous treatment of an intraprocedural left main stent thrombosis with the support of an automatic mechanical chest compression device. *Int J Cardiol*. 2008;124:e19–e21. doi: 10.1016/j.ijcard.2006.11.175.
 163. Steen S, Sjöberg T, Olsson P, Young M. Treatment of out-of-hospital cardiac arrest with LUCAS, a new device for automatic mechanical compression and active decompression resuscitation. *Resuscitation*. 2005;67:25–30. doi: 10.1016/j.resuscitation.2005.05.013.
 164. Larsen AI, Hjørnevik AS, Ellingsen CL, Nilsen DW. Cardiac arrest with continuous mechanical chest compression during percutaneous coronary intervention. A report on the use of the LUCAS device. *Resuscitation*. 2007;75:454–459. doi: 10.1016/j.resuscitation.2007.05.007.
 165. Wagner H, Terkelsen CJ, Friberg H, Harnek J, Kern K, Lassen JF, Olivecrona GK. Cardiac arrest in the catheterisation laboratory: a 5-year experience of using mechanical chest compressions to facilitate PCI during prolonged resuscitation efforts. *Resuscitation*. 2010;81:383–387. doi: 10.1016/j.resuscitation.2009.11.006.
 166. Linder R, Abdollahi P, Wennersten G. [Life-saving mechanical compression during percutaneous coronary intervention]. *Lakartidningen*. 2006;103:2390–2392.
 167. Ladowski JS, Dillon TA, Deschner WP, DeRiso AJ 2nd, Peterson AC, Schatzlein MH. Durability of emergency coronary artery bypass for complications of failed angioplasty. *Cardiovasc Surg*. 1996;4:23–27.
 168. Redle J, King B, Lemole G, Doorey AJ. Utility of rapid percutaneous cardiopulmonary bypass for refractory hemodynamic collapse in the cardiac catheterization laboratory. *Am J Cardiol*. 1994;73:899–900.
 169. Overlie PA. Emergency use of portable cardiopulmonary bypass. *Cathet Cardiovasc Diagn*. 1990;20:27–31.
 170. Shawl FA, Domanski MJ, Wish MH, Davis M, Punja S, Hernandez TJ. Emergency cardiopulmonary bypass support in patients with cardiac arrest in the catheterization laboratory. *Cathet Cardiovasc Diagn*. 1990;19:8–12.
 171. Bagai J, Webb D, Kasasbeh E, Crenshaw M, Salloum J, Chen J, Zhao D. Efficacy and safety of percutaneous life support during high-risk percutaneous coronary intervention, refractory cardiogenic shock and in-laboratory cardiopulmonary arrest. *J Invasive Cardiol*. 2011;23:141–147.
 172. Sheu JJ, Tsai TH, Lee FY, Fang HY, Sun CK, Leu S, Yang CH, Chen SM, Hang CL, Hsieh YK, Chen CJ, Wu CJ, Yip HK. Early extracorporeal membrane oxygenator-assisted primary percutaneous coronary intervention improved 30-day clinical outcomes in patients with ST-segment elevation myocardial infarction complicated with profound cardiogenic shock. *Crit Care Med*. 2010;38:1810–1817. doi: 10.1097/CCM.0b013e3181e8ac7f.
 173. Arlt M, Philipp A, Voelkel S, Schopka S, Husser O, Hengstenberg C, Schmid C, Hilker M. Early experiences with miniaturized extracorporeal life-support in the catheterization laboratory. *Eur J Cardiothorac Surg*. 2012;42:858–863. doi: 10.1093/ejcts/ezs176.
 174. Grambow DW, Deeb GM, Pavlides GS, Margulis A, O'Neill WW, Bates ER. Emergent percutaneous cardiopulmonary bypass in patients having cardiovascular collapse in the cardiac catheterization laboratory. *Am J Cardiol*. 1994;73:872–875.
 175. Mooney MR, Arom KV, Joyce LD, Mooney JF, Goldenberg IF, Von Rueden TJ, Emery RW. Emergency cardiopulmonary bypass support in patients with cardiac arrest. *J Thorac Cardiovasc Surg*. 1991;101:450–454.
 176. Tsao NW, Shih CM, Yeh JS, Kao YT, Hsieh MH, Ou KL, Chen JW, Shyu KG, Weng ZC, Chang NC, Lin FY, Huang CY. Extracorporeal membrane oxygenation-assisted primary percutaneous coronary intervention may improve survival of patients with acute myocardial infarction complicated by profound cardiogenic shock. *J Crit Care*. 2012;27:530.e1–530.11. doi: 10.1016/j.jcrc.2012.02.012.
 177. Thiele H, Zeymer U, Neumann FJ, Ferenc M, Olbrich HG, Hausleiter J, de Waha A, Richardt G, Hennemerdorf M, Empen K, Fuernau G, Desch S, Eitel I, Hambrecht R, Lauer B, Böhm M, Ebel H, Schneider S, Werdan K, Schuler G; Intra-aortic Balloon Pump in cardiogenic shock II (IABP-SHOCK II) trial investigators. Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II): final 12 month results of a randomised, open-label trial. *Lancet*. 2013;382:1638–1645. doi: 10.1016/S0140-6736(13)61783-3.
 178. Ohman EM, George BS, White CJ, Kern MJ, Gurbel PA, Freedman RJ, Lundergan C, Hartmann JR, Talley JD, Frey MJ. Use of aortic counterpulsation to improve sustained coronary artery patency during acute myocardial infarction. Results of a randomized trial. The Randomized IABP Study Group. *Circulation*. 1994;90:792–799.
 179. Kaul U, Sahay S, Bahl VK, Sharma S, Wasir HS, Venugopal P. Coronary angioplasty in high risk patients: comparison of elective intra-aortic balloon pump and percutaneous cardiopulmonary bypass support—a randomized study. *J Interv Cardiol*. 1995;8:199–205.
 180. Stone GW, Marsalese D, Brodie BR, Griffin JJ, Donohue B, Costantini C, Balestrini C, Wharton T, Esente P, Spain M, Moses J, Nobuyoshi M, Ayres M, Jones D, Mason D, Grines L, O'Neill WW, Grines CL. A prospective, randomized evaluation of prophylactic intra-aortic balloon counterpulsation in high risk patients with acute myocardial infarction treated with primary angioplasty. Second Primary Angioplasty in Myocardial Infarction (PAMI-II) Trial Investigators. *J Am Coll Cardiol*. 1997;29:1459–1467.
 181. Ohman EM, Nanas J, Stomel RJ, Leeser MA, Nielsen DW, O'Dea D, Rogers FJ, Harber D, Hudson MP, Fraulo E, Shaw LK, Lee KL; TACTICS Trial. Thrombolysis and counterpulsation to improve survival in myocardial infarction complicated by hypotension and suspected cardiogenic shock or heart failure: results of the TACTICS Trial. *J Thromb Thrombolysis*. 2005;19:33–39. doi: 10.1007/s11239-005-0938-0.
 182. Prondzinsky R, Lemm H, Swyter M, Wegener N, Unverzagt S, Carter JM, Russ M, Schlitt A, Buerke U, Christoph A, Schmidt H, Winkler M, Thiery J, Werdan K, Buerke M. Intra-aortic balloon counterpulsation in patients with acute myocardial infarction complicated by cardiogenic shock: the prospective, randomized IABP SHOCK Trial for attenuation of multiorgan dysfunction syndrome. *Crit Care Med*. 2010;38:152–160. doi: 10.1097/CCM.0b013e3181b78671.
 183. Unverzagt S, Buerke M, de Waha A, Haerting J, Pietzner D, Seyfarth M, Thiele H, Werdan K, Zeymer U, Prondzinsky R. Intra-aortic balloon pump counterpulsation (IABP) for myocardial infarction complicated by cardiogenic shock. *Cochrane Database Syst Rev*. 2015;3:CD007398. doi: 10.1002/14651858.CD007398.pub3.
 184. Sjauw KD, Engström AE, Vis MM, van der Schaaf RJ, Baan J Jr, Koch KT, de Winter RJ, Piek JJ, Tijssen JG, Henriques JP. A systematic review and meta-analysis of intra-aortic balloon pump therapy in ST-elevation myocardial infarction: should we change the guidelines? *Eur Heart J*. 2009;30:459–468. doi: 10.1093/eurheartj/ehn602.
 185. Lee JM, Park J, Kang J, Jeon KH, Jung JH, Lee SE, Han JK, Kim HL, Yang HM, Park KW, Kang HJ, Koo BK, Kim SH, Kim HS. The efficacy and safety of mechanical hemodynamic support in patients undergoing high-risk percutaneous coronary intervention with or without cardiogenic shock: Bayesian approach network meta-analysis of 13 randomized controlled trials. *Int J Cardiol*. 2015;184:36–46. doi: 10.1016/j.ijcard.2015.01.081.
 186. Reul GJ, Cooley DA, Hallman GL, Duncan JM, Livesay JJ, Frazier OH, Ott DA, Angelini P, Massumi A, Mathur VS. Coronary artery bypass for unsuccessful percutaneous transluminal coronary angioplasty. *J Thorac Cardiovasc Surg*. 1984;88(5 Pt 1):685–694.
 187. Andreasen JJ, Mortensen PE, Andersen LI, Arendrup HC, Ilkjaer LB, Kjølner M, Thyssen P. Emergency coronary artery bypass surgery after failed percutaneous transluminal coronary angioplasty. *Scand Cardiovasc J*. 2000;34:242–246.

KEY WORDS: cardiac arrest ■ defibrillation ■ emergency

Part 10: Special Circumstances of Resuscitation: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Eric J. Lavonas, Ian R. Drennan, Andrea Gabrielli, Alan C. Heffner, Christopher O. Hoyte, Aaron M. Orkin, Kelly N. Sawyer and Michael W. Donnino

Circulation. 2015;132:S501-S518

doi: 10.1161/CIR.0000000000000264

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 2015 American Heart Association, Inc. All rights reserved.

Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://circ.ahajournals.org/content/132/18_suppl_2/S501

An erratum has been published regarding this article. Please see the attached page for:

</content/134/9/e122.full.pdf>

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the [Permissions and Rights Question and Answer](#) document.

Reprints: Information about reprints can be found online at:
<http://www.lww.com/reprints>

Subscriptions: Information about subscribing to *Circulation* is online at:
<http://circ.ahajournals.org/subscriptions/>

Correction to: Part 10: Special Circumstances of Resuscitation: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

In the article by Lavonas et al “Part 10: Special Circumstances of Resuscitation: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care,” which published online October 15, 2015, and appeared in the November 3, 2015, issue of the journal (*Circulation*. 2015;132[suppl 2]:S501–S518. DOI: 10.1161/CIR.0000000000000264), a correction was needed.

1. On page S517, in the Reference section, reference 138 read, “138. Cao H, Zhou X, Zhang J, Huang X, Zhai Y, Zhang X, Chu L. Hydrogen sulfide protects against bleomycin-induced pulmonary fibrosis in rats by inhibiting NF- κ B expression and regulating Th1/Th2 balance. *Toxicol Lett*. 2014;224:387–394. doi: 10.1016/j.toxlet.2013.11.008.” It has been replaced with, “Cao D, Heard K, Foran M, Koyfman A. Intravenous lipid emulsion in the emergency department: a systematic review of the literature. *J Emerg Med*. 2015;48:387–397. doi:10.1016/j.jemermed.2014.10.009.”

This correction has been made to the current online version of the article, which is available at http://circ.ahajournals.org/content/132/18_suppl_2/S501.

Circulation is available at
<http://circ.ahajournals.org>.

© 2016 American Heart
Association, Inc.