Part 12: Cardiac Arrest in Special Situations: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

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Part 12: Cardiac Arrest in Special Situations

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

Terry L. Vanden Hoek, Chair; Laurie J. Morrison; Michael Shuster; Michael Donnino; Elizabeth Sinz; Eric J. Lavonas; Farida M. Jeejeebhoy; Andrea Gabrielli

This section of the *2010 AHA Guidelines for CPR and ECC* addresses cardiac arrest in situations that require special treatments or procedures beyond those provided during basic life support (BLS) and advanced cardiovascular life support (ACLS). We have included 15 specific cardiac arrest situations. The first several sections discuss cardiac arrest associated with internal physiological or metabolic conditions, such as asthma (12.1), anaphylaxis (12.2), pregnancy (12.3), morbid obesity (12.4), pulmonary embolism (PE) (12.5), and electrolyte imbalance (12.6).

The next several sections relate to resuscitation and treatment of cardiac arrest associated with external or environmentally related circumstances, such as ingestion of toxic substances (12.7), trauma (12.8), accidental hypothermia (12.9), avalanche (12.10), drowning (12.11), and electric shock/lightning strikes (12.12).

The last 3 sections review management of cardiac arrest that may occur during special situations affecting the heart, including percutaneous coronary intervention (PCI) (12.13), cardiac tamponade (12.14), and cardiac surgery (12.15).

Part 12.1: Cardiac Arrest Associated With Asthma

Asthma is responsible for more than 2 million visits to the emergency department (ED) in the United States each year, with 1 in 4 patients requiring admission to a hospital.[1] Annually there are 5,000 to 6,000 asthma-related deaths in the United States, many occurring in the prehospital setting.[2] Severe asthma accounts for approximately 2% to 20% of admissions to intensive care units, with up to one third of these patients requiring intubation and mechanical ventilation.[3] This section focuses on the evaluation and treatment of patients with near-fatal asthma.

Several consensus groups have developed excellent guidelines for the management of asthma that are available on the World Wide Web:

- http://www.nhlbi.nih.gov/about/naepp
- http://www.ginasthma.com

Pathophysiology

The pathophysiology of asthma consists of 3 key abnormalities:

- Bronchoconstriction
- Airway inflammation
- Mucous plugging

Complications of severe asthma, such as tension pneumothorax, lobar atelectasis, pneumonia, and pulmonary edema, can contribute to fatalities. Severe asthma exacerbations are commonly associated with hypercarbia and acidemia, hypotension due to decreased venous return, and depressed mental status, but the most common cause of death is asphyxia. Cardiac causes of death are less common.[4]

Clinical Aspects of Severe Asthma

Wheezing is a common physical finding, although the severity of wheezing does not correlate with the degree of airway obstruction. The absence of wheezing may indicate critical airway obstruction, whereas increased wheezing may indicate a positive response to bronchodilator therapy.

Oxygen saturation (Sao_2) levels may not reflect progressive alveolar hypoventilation, particularly if oxygen is being administered. Note that Sao_2 may fall initially during therapy because β_2-agonists produce both bronchodilation and vasodilation and initially may increase intrapulmonary shunting.

Other causes of wheezing are pulmonary edema,[5] chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxis,[6] foreign bodies, PE, bronchiectasis, and subglottic mass.[7]

Initial Stabilization

Patients with severe life-threatening asthma require urgent and aggressive treatment with simultaneous administration of oxygen, bronchodilators, and steroids. Healthcare providers must monitor these patients closely for deterioration. Although the pathophysiology of life-threatening asthma consists of bronchoconstriction, inflammation, and mucous plugging, only bronchoconstriction and inflammation are amenable to drug treatment.

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Primary Therapy

Oxygen

Oxygen should be provided to all patients with severe asthma, even those with normal oxygenation. As noted above, successful treatment with β_2-agonists may cause an initial decrease in oxygen saturation because the resultant bronchodilation can initially increase the ventilation-perfusion mismatch.

Inhaled β_2-Agonists

Short-acting β-agonists provide rapid, dose-dependent bronchodilation with minimal side effects. Because the dose delivered depends on the patient's lung volume and inspiratory flow rate, the same dose can be used in most patients regardless of age or size. Studies have shown no difference in the effects of continuous versus intermittent administration of nebulized albuterol[8,9]; however, continuous administration was more effective in a subset of patients with severe exacerbations of asthma.[8] A Cochrane meta-analysis showed no overall difference between the effects of albuterol delivered by metered-dose inhaler spacer or nebulizer.[10] If prior use of a metered-dose inhaler has not been effective, use of a nebulizer is reasonable.

Although albuterol is sometimes administered intravenously (IV) in severe asthma, a systematic review of 15 clinical trials found that IV β_2-agonists, administered by either bolus or infusion, did not lead to significant improvements in any clinical outcome measure.[9]

Levalbuterol is the R-isomer of albuterol. Comparisons with albuterol have produced mixed results, with some studies showing a slightly improved bronchodilator effect in the treatment of acute asthma in the ED.[11] There is no evidence that levalbuterol should be favored over albuterol.

One of the most common adjuncts used with β-agonist treatment, particularly in the first hours of treatment, include anticholinergic agents (see "Adjunctive Therapies" below for more detail). When combined with short-acting β-agonists, anticholinergic agents such as ipratropium can produce a clinically modest improvement in lung function compared with short-acting β-agonists alone.[12,13]

Corticosteroids

Systemic corticosteroids are the only treatment for the inflammatory component of asthma proven to be effective for acute asthma exacerbations. Because the antiinflammatory effects after administration may not be apparent for 6 to 12 hours, corticosteroids should be administered early. The early use of systemic steroids hastens the resolution of airflow obstruction and may reduce admission to the hospital.[14] Although there may be no difference in clinical effects between oral and IV formulations of corticosteroids,[15,16] the IV route is preferable in patients with severe asthma. In adults a typical initial dose of methylprednisolone is 125 mg (dose range: 40 mg to 250 mg); a typical dose of dexamethasone is 10 mg.

Adjunctive Therapies

Anticholinergics

Ipratropium bromide is an anticholinergic bronchodilator pharmacologically related to atropine. The nebulizer dose is 500 mcg.[15,16] Ipratropium bromide has a slow onset of action (approximately 20 minutes), with peak effectiveness at 60 to 90 minutes and no systemic side effects. The drug is typically given only once because of its prolonged onset of action, but some studies have shown that repeat doses of 250 mcg or 500 mcg every 20 minutes may be beneficial.[17] A recent meta-analysis indicated a reduced number of hospital admissions associated with treatment with ipratropium bromide, particularly in patients with severe exacerbations.[18]

Magnesium Sulfate

When combined with nebulized β-adrenergic agents and corticosteroids, IV magnesium sulfate can moderately improve pulmonary function in patients with asthma.[19] Magnesium causes relaxation of bronchial smooth muscle independent of serum magnesium level, with only minor side effects (flushing, lightheadedness). A Cochrane meta-analysis of 7 studies concluded that IV magnesium sulfate improves pulmonary function and reduces hospital admissions, particularly for patients with the most severe exacerbations of asthma.[20] The use of nebulized magnesium sulfate as an adjunct to nebulized β-adrenergic agents has been reported in a small case series to improve FEV1 and Spo_2,[21] although a prior meta-analysis demonstrated only a trend toward improved pulmonary function with nebulized magnesium.[22] For those with severe refractory asthma, providers may consider IV magnesium at the standard adult dose of 2 g administered over 20 minutes.

Epinephrine or Terbutaline

Epinephrine and terbutaline are adrenergic agents that can be given subcutaneously to patients with acute severe asthma. The dose of subcutaneous epinephrine (concentration 1:1000) is 0.01 mg/kg, divided into 3 doses of approximately 0.3 mg administered at 20-minute intervals. Although the nonselective adrenergic properties of epinephrine may cause an increase in heart rate, myocardial irritability, and increased oxygen demand, its use is well-tolerated, even in patients >35 years of age.[23] Terbutaline is given in a subcutaneous dose of 0.25 mg, which can be repeated every 20 minutes for 3 doses. There is no evidence that subcutaneous epinephrine or terbutaline has advantages over inhaled β_2-agonists. Epinephrine has been administered IV (initiated at 0.25 mcg/min to 1 mcg/min continuous infusion) in severe asthma; however, 1 retrospective investigation indicated a 4% incidence of serious side effects. There is no evidence of improved outcomes with IV epinephrine compared with selective inhaled β_2-agonists.[24]

Ketamine

Ketamine is a parenteral, dissociative anesthetic with bronchodilatory properties that also can stimulate copious bronchial secretions. One case series[25] suggested substantial efficacy, whereas 2 published randomized trials in children[26,27] found no benefit of ketamine when compared with standard care. Ketamine has sedative and analgesic properties that may be useful if intubation is planned.

Heliox

Heliox is a mixture of helium and oxygen (usually a 70:30 helium to oxygen ratio mix) that is less viscous than ambient air. Heliox has been shown to improve the delivery and deposition of nebulized albuterol[28]; however, a recent meta-analysis of clinical trials did not support its use as initial treatment for

patients with acute asthma.[29] Because the heliox mixture requires at least 70% helium for effect, it cannot be used if the patient requires >30% oxygen.

Methylxanthines

Although once considered a mainstay in the treatment of acute asthma, methylxanthines are no longer recommended because of their erratic pharmacokinetics, known side effects, and lack of evidence of benefit.[30]

Leukotriene Antagonists

Leukotriene antagonists improve lung function and decrease the need for short-acting β_2-agonists for long-term asthma therapy, but their effectiveness during acute exacerbations of asthma is unproven.

Inhaled Anesthetics

Case reports in adults[31] and children[32] suggest a benefit of the potent inhalation anesthetics sevoflurane and isoflurane for patients with life-threatening asthma unresponsive to maximal conventional therapy. These agents may have direct bronchodilator effects. In addition, the anesthetic effect of these drugs increases the ease of mechanical ventilation and reduces oxygen demand and carbon dioxide production. This therapy requires expert consultation in an intensive care setting, and its effectiveness has not been evaluated in randomized clinical studies.

Assisted Ventilation

Noninvasive Positive-Pressure Ventilation

Noninvasive positive-pressure ventilation (NIPPV) may offer short-term support for patients with acute respiratory failure and may delay or eliminate the need for endotracheal intubation.[33–35] This therapy requires that the patient is alert and has adequate spontaneous respiratory effort. Bilevel positive airway pressure (BiPAP), the most common method of delivering NIPPV, allows for separate control of inspiratory and expiratory pressures.

Endotracheal Intubation With Mechanical Ventilation

Endotracheal intubation is indicated for patients who present with apnea, coma, persistent or increasing hypercapnia, exhaustion, severe distress, and depression of mental status. Clinical judgment is necessary to assess the need for immediate endotracheal intubation for these critically ill patients. Endotracheal intubation does not solve the problem of small airway constriction in patients with severe asthma; thus, therapy directed toward relief of bronchoconstriction should be continued. Mechanical ventilation in the asthmatic patient can be difficult and associated risks require careful management. Intubation and positive-pressure ventilation can trigger further bronchoconstriction and complications such as breath stacking that result from incomplete expiration, air trapping, and buildup of positive end-expiratory pressure (ie, intrinsic or auto-PEEP). This breath stacking can cause barotrauma. Decreasing tidal volume may avoid auto-PEEP and high peak airway pressures. Optimal ventilator management requires expert consultation and ongoing careful review of ventilation flow and pressure curves. Although endotracheal intubation introduces risks, it should be performed when necessary based on clinical condition.

Rapid sequence intubation is the technique of choice and should be performed by an expert in airway management. The provider should use the largest endotracheal tube available (usually 8 or 9 mm) to decrease airway resistance. Immediately after intubation, endotracheal tube placement should be confirmed by clinical examination and waveform capnography. A chest radiograph should then be performed.

Troubleshooting After Intubation

When severe bronchoconstriction is present, breath stacking (so-called auto-PEEP) can develop during positive-pressure ventilation, leading to complications such as hyperinflation, tension pneumothorax, and hypotension. During manual or mechanical ventilation, a slower respiratory rate should be used with smaller tidal volumes (eg, 6 to 8 mL/kg),[36] shorter inspiratory time (eg, adult inspiratory flow rate 80 to 100 mL/min), and longer expiratory time (eg, inspiratory to expiratory ratio 1:4 or 1:5) than generally would be provided to patients without asthma.[37] Management of mechanical ventilation will vary based on patient-ventilation characteristics. Expert consultation should be obtained.

Mild hypoventilation (permissive hypercapnia) reduces the risk of barotrauma. Hypercapnia is typically well tolerated.[38,39] Sedation is often required to optimize ventilation, decrease ventilator dyssynchrony (and therefore auto-PEEP), and minimize barotrauma after intubation. Because delivery of inhaled medications may be inadequate before intubation, the provider should continue to administer inhaled albuterol treatments through the endotracheal tube.

Four common causes of acute deterioration in any intubated patient are recalled by the mnemonic **DOPE** (tube **D**isplacement, tube **O**bstruction, **P**neumothorax, **E**quipment failure). Auto-PEEP is another common cause of deterioration in patients with asthma. If the asthmatic patient's condition deteriorates or if it is difficult to ventilate the patient, check the ventilator for leaks or malfunction; verify endotracheal tube position; eliminate tube obstruction (eliminate any mucous plugs and kinks); evaluate for auto-PEEP; and rule out a pneumothorax.

High-end expiratory pressure can be reduced quickly by separating the patient from the ventilator circuit; this will allow PEEP to dissipate during passive exhalation. If auto-PEEP results in significant hypotension, assisting with exhalation by pressing on the chest wall after disconnection of the ventilator circuit will allow active exhalation and should lead to immediate resolution of hypotension. To minimize auto-PEEP, decrease the respiratory rate or tidal volume or both. If auto-PEEP persists and the patient displays ventilator dyssynchrony despite adequate sedation, paralytic agents may be considered.

In exceedingly rare circumstances, aggressive treatment for acute respiratory failure due to severe asthma will not provide adequate gas exchange. There are case reports that describe successful use of extracorporeal membrane oxygenation (ECMO) in adult and pediatric patients[40–43] with severe asthma after other aggressive measures have failed to reverse hyoxemia and hypercarbia.

BLS Modifications

BLS treatment of cardiac arrest in asthmatic patients is unchanged.

ACLS Modifications

When cardiac arrest occurs in the patient with acute asthma, standard ACLS guidelines should be followed.

Case series and case reports describe a novel technique of cardiopulmonary resuscitation (CPR) termed "lateral chest compressions"; however, there is insufficient evidence to recommend this technique over standard techniques.[44–50]

The adverse effect of auto-PEEP on coronary perfusion pressure and capacity for successful defibrillation has been described in patients in cardiac arrest without asthma.[51,52] Moreover, the adverse effect of auto-PEEP on hemodynamics in asthmatic patients who are not in cardiac arrest has also been well-described.[53–56] 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). 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).

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).

Part 12.2: Cardiac Arrest Associated With Anaphylaxis

Anaphylaxis is an allergic reaction characterized by multisystem involvement, including skin, airway, vascular system, and gastrointestinal tract. Severe cases may result in complete obstruction of the airway and cardiovascular collapse from vasogenic shock. Anaphylaxis accounts for about 500 to 1000 deaths per year in the United States.[57]

The term *classic anaphylaxis* refers to hypersensitivity reactions mediated by the immunoglobulins IgE and IgG. Prior sensitization to an allergen produces antigen-specific immunoglobulins. Subsequent reexposure to the allergen provokes the anaphylactic reaction, although many anaphylactic reactions occur with no documented prior exposure. Pharmacological agents, latex, foods, and stinging insects are among the most common causes of anaphylaxis described.

Signs and Symptoms

The initial symptoms of anaphylaxis are often nonspecific and include tachycardia, faintness, cutaneous flushing, urticaria, diffuse or localized pruritus, and a sensation of impending doom. Urticaria is the most common physical finding. The patient may be agitated or anxious and may appear either flushed or pale.

A common early sign of respiratory involvement is rhinitis. As respiratory compromise becomes more severe, serious upper airway (laryngeal) edema may cause stridor and lower airway edema (asthma) may cause wheezing. Upper airway edema can also be a sign in angiotensin converting enzyme inhibitor-induced angioedema or C1 esterase inhibitor deficiency with spontaneous laryngeal edema.[58–60]

Cardiovascular collapse is common in severe anaphylaxis. If not promptly corrected, vasodilation and increased capillary permeability, causing decreased preload and relative hypovolemia of up to 37% of circulating blood volume, can rapidly lead to cardiac arrest.[61,62] Myocardial ischemia and acute myocardial infarction, malignant arrhythmias, and cardiovascular depression can also contribute to rapid hemodynamic deterioration and cardiac arrest.[63] Additionally, cardiac dysfunction may result from underlying disease or development of myocardial ischemia due to hypotension or following administration of epinephrine.[64,65]

There are no randomized controlled trials evaluating alternative treatment algorithms for cardiac arrest due to anaphylaxis. Evidence is limited to case reports and extrapolations from nonfatal cases, interpretation of pathophysiology, and consensus opinion. Providers must be aware that urgent support of airway, breathing, and circulation is essential in suspected anaphylactic reactions.

Because of limited evidence, the management of cardiac arrest secondary to anaphylaxis should be treated with standard BLS and ACLS. The following therapies are largely consensus-based but commonly used and widely accepted in the management of the patient with anaphylaxis who is not in cardiac arrest.

BLS Modifications

Airway

Early and rapid advanced airway management is critical and should not be unnecessarily delayed. Given the potential for the rapid development of oropharyngeal or laryngeal edema,[66] immediate referral to a health professional with expertise in advanced airway placement is recommended (Class I, LOE C).

Circulation

The intramuscular (IM) administration of epinephrine (epinephrine autoinjectors, eg, the EpiPen™) in the anterolateral aspect of the middle third of the thigh provides the highest peak blood levels.[67] Absorption and subsequent achievement of maximum plasma concentration after subcutaneous administration is slower than the IM route and may be significantly delayed with shock.[67]

Epinephrine[68] 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). 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).[69] The adult epinephrine IM auto-injector will deliver 0.3 mg of epinephrine and the pediatric epinephrine IM auto-injector will deliver 0.15 mg of epinephrine. In both anaphylaxis and cardiac arrest the immediate use of an epinephrine autoinjector is recommended if available (Class I, LOE C).

ACLS Modifications

Airway

Early recognition of the potential for a difficult airway in anaphylaxis is paramount in patients who develop hoarseness, lingual edema, stridor, or oropharyngeal swelling. Planning for advanced airway management, including a surgical airway,[70] is recommended (Class I, LOE C).

Fluid Resuscitation

In a prospective evaluation of volume resuscitation after diagnostic sting challenge, repeated administration of 1000-mL

bolus doses of isotonic crystalloid (eg, normal saline) titrated to systolic blood pressure above 90 mm Hg was used successfully in patients whose hypotension did not respond immediately to vasoactive drugs.[61,71] Vasogenic shock from anaphylaxis may require aggressive fluid resuscitation (Class IIa, LOE C).

Vasopressors

There are no human trials establishing the role of epinephrine or preferred route of administration in anaphylactic shock managed by ACLS providers.[68] In an animal study of profound anaphylactic shock, IV epinephrine restored blood pressure to baseline; however, the effect was limited to the first 15 minutes after shock, and no therapeutic effect was observed with the same dose of epinephrine administered IM or subcutaneously.[72] Therefore, 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).

For patients not in cardiac arrest, IV epinephrine 0.05 to 0.1 mg (5% to 10% of the epinephrine dose used routinely in cardiac arrest) has been used successfully in patients with anaphylactic shock.[73] Because fatal overdose of epinephrine has been reported,[64,71,74,75] close hemodynamic monitoring is recommended (Class I, LOE B).

In a study of animals sensitized by ragweed, a continuous IV infusion of epinephrine maintained a mean arterial pressure at 70% of preshock levels better than no treatment or bolus epinephrine treatment (IV, subcutaneous, or IM).[76] Furthermore, a recent human study suggests that careful titration of a continuous infusion of IV epinephrine (5 to 15 mcg/min), based on severity of reaction and in addition to crystalloid infusion, may be considered in treatment of anaphylactic shock.[71] Therefore, 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) and may be considered in postarrest management (Class IIb, LOE C).

Recently vasopressin has been used successfully in patients with anaphylaxis (with or without cardiac arrest) who did not respond to standard therapy.[77–79] Other small case series described successful results with administration of alternative α-agonists such as norepinephrine,[80] methoxamine,[81,82] and metaraminol.[83–85] 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). No randomized controlled trials have evaluated epinephrine versus the use of alternative vasoactive drugs for cardiac arrest due to anaphylaxis.

Other Interventions

There are no prospective randomized clinical studies evaluating the use of other therapeutic agents in anaphylactic shock or cardiac arrest. Adjuvant use of antihistamines (H1 and H2 antagonist),[86,87] inhaled β-adrenergic agents,[88] and IV corticosteroids[89] has been successful in management of the patient with anaphylaxis and may be considered in cardiac arrest due to anaphylaxis (Class IIb, LOE C).

Extracorporeal Support of Circulation

Cardiopulmonary bypass has been successful in isolated case reports of anaphylaxis followed by cardiac arrest.[90,91] 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).

Part 12.3: Cardiac Arrest Associated With Pregnancy

Scope of the Problem

The Confidential Enquiries into Maternal and Child Health (CEMACH) data set constitutes the largest population-based data set on this target population.[92] The overall maternal mortality rate was calculated at 13.95 deaths per 100 000 maternities. There were 8 cardiac arrests with a frequency calculated at 0.05 per 1000 maternities, or 1:20 000. The frequency of cardiac arrest in pregnancy is on the rise with previous reports estimating the frequency to be 1:30 000 maternities.[93] Despite pregnant women being younger than the traditional cardiac arrest patient, the survival rates are poorer, with one case series reporting a survival rate of 6.9%.[93,94]

During attempted resuscitation of a pregnant woman, providers have 2 potential patients: the mother and the fetus. The best hope of fetal survival is maternal survival. For the critically ill pregnant patient, rescuers must provide appropriate resuscitation based on consideration of the physiological changes caused by pregnancy.

Key Interventions to Prevent Arrest

The following interventions are the standard of care for treating the critically ill pregnant patient (Class I, LOE C):

- Place the patient in the full left-lateral position to relieve possible compression of the inferior vena cava. Uterine obstruction of venous return can produce hypotension and may precipitate arrest in the critically ill patient.[95,96]
- Give 100% oxygen.
- Establish intravenous (IV) access above the diaphragm.
- Assess for hypotension; maternal hypotension that warrants therapy has been defined as a systolic blood pressure $<$100 mm Hg or $<$80% of baseline.[97,98] Maternal hypotension can result in reduced placental perfusion.[99–102] In the patient who is not in arrest, both crystalloid and colloid solutions have been shown to increase preload.[103]
- Consider reversible causes of critical illness and treat conditions that may contribute to clinical deterioration as early as possible.

Resuscitation of the Pregnant Patient in Cardiac Arrest (Figure 1)

There are no randomized controlled trials evaluating the effect of specialized obstetric resuscitation versus standard care in pregnant patients in cardiac arrest. There are reports in the literature of patients not in arrest that describe the science behind important physiological changes that occur in pregnancy that may influence treatment recommendations and guidelines for resuscitation from cardiac arrest in pregnancy.

BLS Modifications

Patient Positioning

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

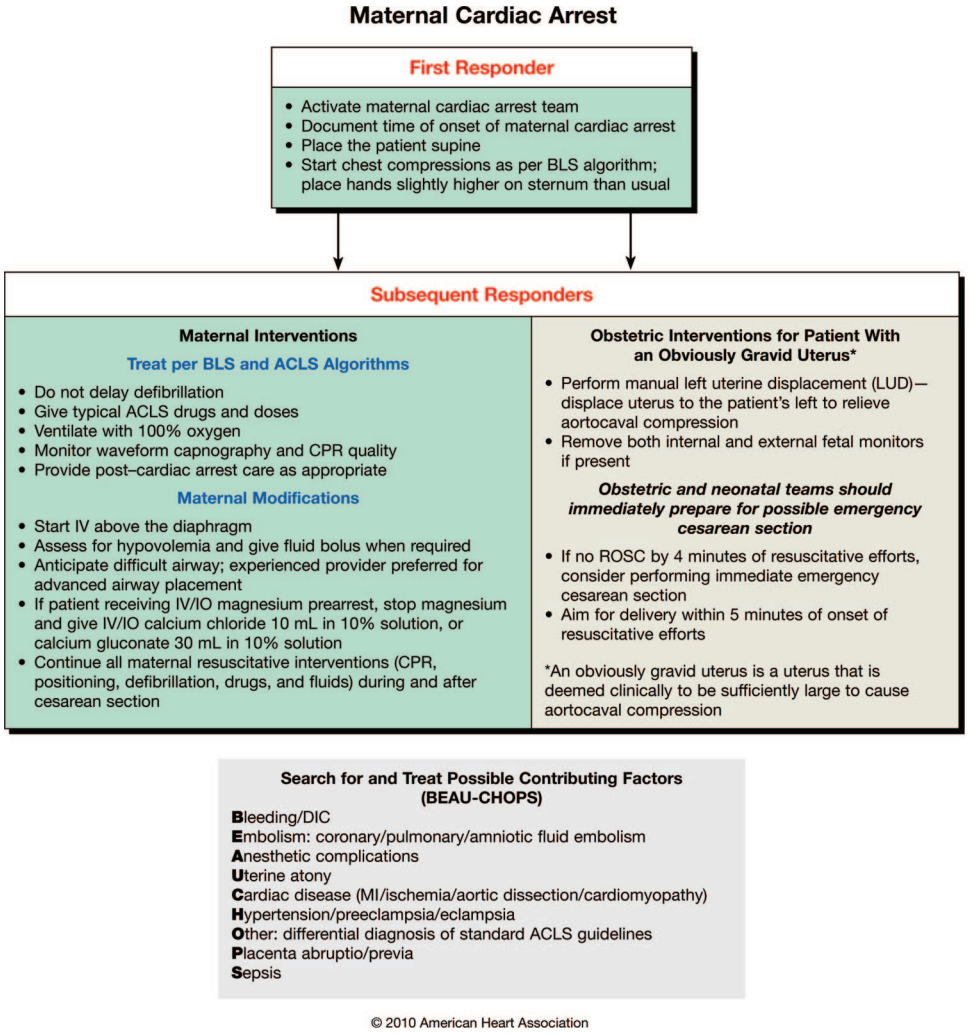

Figure 1. Maternal cardiac arrest algorithm.

The pregnant uterus can compress the inferior vena cava, impeding venous return and thereby reducing stroke volume and cardiac output. Reports of noncardiac arrest parturients indicate that left-lateral tilt results in improved maternal hemodynamics of blood pressure, cardiac output, and stroke volume[96,98,104]; and improved fetal parameters of oxygenation, nonstress test, and fetal heart rate.[100–102]

Although chest compressions in the left-lateral tilt position are feasible in a manikin study,[105] they result in less forceful chest compressions than are possible in the supine position.[106] Two studies found no improvement in maternal hemodynamic or fetal parameters with 10° to 20° left-lateral tilt in patients not in arrest.[107,108] One study reported more aortic compression at 15° left-lateral tilt compared with a full left-lateral tilt.[97] In addition, aortic compression has been found at >30° of tilt,[109] however the majority of these patients were in labor.

If left-lateral tilt is used to improve maternal hemodynamics during cardiac arrest, the degree of tilt should be maximized. However, at a tilt ≥30° the patient may slide or roll off the inclined plane,[106] so this degree of tilt may not be practical during resuscitation. Although important, the degree of tilt is difficult to estimate reliably; 1 study reported that the degree of table tilt is often overestimated.[110] Using a fixed, hard wedge of a predetermined angle may help.

Two studies in pregnant women not in arrest found that manual left uterine displacement, which is done with the patient supine, is as good as or better than left-lateral tilt in relieving aortocaval compression (as assessed by the incidence of hypotension and use of ephedrine).[111,112]

Therefore, to relieve aortocaval compression during chest compressions and optimize the quality of CPR, it is reasonable to perform manual left uterine displacement in the supine position first (Class IIa, LOE C). Left uterine displacement can be performed from either the patient's left side with the 2-handed technique (Figure 2) or the patient's right side with the 1-handed technique (Figure 3), depending on the positioning of the resuscitation team. If this technique is unsuccessful, and an appropriate wedge is readily available, then providers may consider placing the patient in a left-lateral tilt of 27° to 30°,[106] using a firm wedge to support the pelvis and thorax (Figure 4) (Class IIb, LOE C).

If chest compressions remain inadequate after lateral uterine displacement or left-lateral tilt, immediate emergency cesarean section should be considered. (See "Emergency Cesarean Section in Cardiac Arrest," below.)

Airway

Airway management is more difficult during pregnancy (see "ACLS Modifications: Airway," below), and placing the

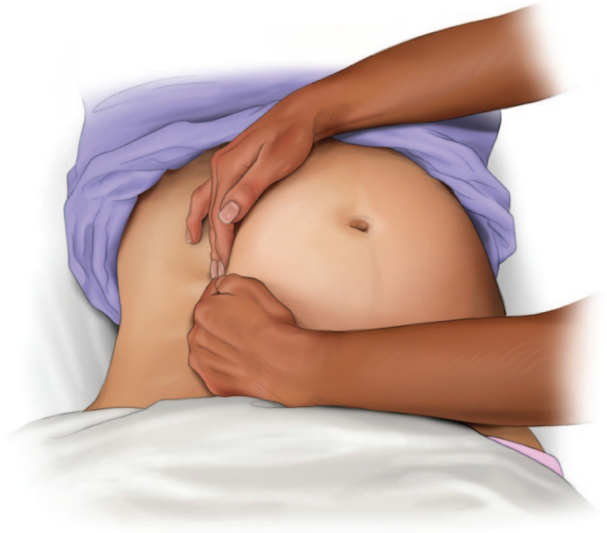

Figure 2. . Left uterine displacement with 2-handed technique.

patient in a tilt may increase the difficulty. In addition, altered airway anatomy increases the risks of aspiration and rapid desaturation. Therefore, optimal use of bag-mask ventilation and suctioning, while preparing for advanced airway placement (see "ACLS Modifications") is critical.

Breathing

Pregnant patients can develop hypoxemia rapidly because of decreased functional residual capacity and increased oxygen demand. One study in normal pregnancy reported increased intrapulmonary shunting of 12.8% to 15.3% compared with the nonpregnant state, in which the normal value is 2% to 5%,[113] which further increases the risk of hypoxemia. Ventilation volumes may need to be reduced because the mother's diaphragm is elevated. Providers should be prepared to support oxygenation and ventilation and monitor oxygen saturation closely.

Circulation

Chest compressions should be performed slightly higher on the sternum than normally recommended to adjust for the elevation of the diaphragm and abdominal contents caused by the gravid uterus.

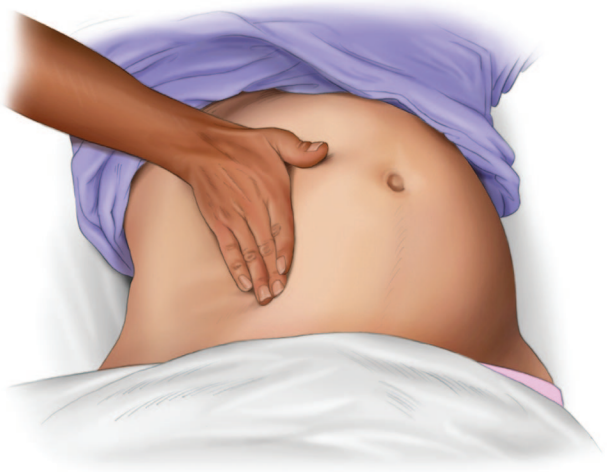

Figure 3. Left uterine displacement using 1-handed technique.

Figure 4. Patient in a 30° left-lateral tilt using a firm wedge to support pelvis and thorax.

Defibrillation

Use of an AED on a pregnant victim has not been studied but is reasonable.

ACLS Modifications

There should be no delay in delivering usual treatments during the management of cardiac arrest in pregnancy.

Airway

Pregnancy results in changes in airway mucosa, including edema, friability, hypersecretion, and hyperemia.[114,115] In addition, 1 study found that the upper airway in the third trimester of pregnancy is smaller compared with that of nonpregnant women and women in the postpartum period.[116] Therefore, airway management of the pregnant patient may be more difficult than airway management of the nonpregnant patient.

There is significant literature recognizing the issue of failed intubation in obstetric anesthesia as a major cause of maternal morbidity and mortality.[117,118] All providers involved in a resuscitation attempt should be aware of the increased risk for pregnancy-related complications in airway management. Intubation with an endotracheal tube or supraglottic airway should be performed only by experienced providers if possible.

Cheun et al[119] found that during apnea desaturation in pregnant patients is significantly faster than in nonpregnant patients. Bag-mask ventilation with 100% oxygen before intubation is especially important in pregnancy (Class IIa, LOE B).[120]

Circulation

Changes in Pharmacokinetics

One clinical pharmacokinetic study discovered an increase in the rate of glomerular filtration and volume of plasma during normal pregnancy.[121] There is no evidence, however, that current

medications or doses should be altered during management of cardiac arrest in pregnancy; therefore, current recommended drug dosages for use in resuscitation of adults should also be used in resuscitation of the pregnant patient.

Defibrillation

Defibrillation should be performed at the recommended ACLS defibrillation doses (Class I, LOE C).[122]

Although there are no studies documenting maternal or fetal complications with defibrillation, there are case reports[123–130] and case series[131–133] that describe potential harm to the fetus when an accidental electric shock (lightning, electric circuit) is delivered directly to the mother. After a pregnant woman receives an electric shock, the range of clinical presentations varies from the mother feeling only a strange sensation with no fetal effects to fetal death either immediately or a few days after the shock. Risk factors for adverse fetal outcomes include the magnitude of current and duration of contact. The greatest predictor of risk for adverse fetal outcome is if the current travels through the uterus, because amniotic fluid most likely transmits current in a manner similar to that transmitted via other body fluids, which could increase the risk of fetal death or burns.

Although there is a small risk of inducing fetal arrhythmias, cardioversion and defibrillation on the external chest are considered safe at all stages of pregnancy.[134–136]

Some experts have raised concern that electric arcing may occur if fetal monitors are attached during defibrillation of a pregnant woman, but there is no evidence to support this. Overall it is reasonable to assume that if the shock is delivered to the mother's thorax, there is very low to no risk of electric arcing to fetal monitors. 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).

Treatment of Reversible Causes

The same reversible causes of cardiac arrest that occur in nonpregnant women can occur during pregnancy. Providers should be familiar with pregnancy-specific diseases and procedural complications and during resuscitation attempts should try to identify common and reversible causes of cardiac arrest in pregnancy.[92]

Cardiac Disease

Cardiac disease is the primary cause of maternal mortality, according to the *2003 to 2005 Confidential Enquiries into Maternal and Child Health* report.[92] For example, the number of deaths from cardiac disease was 2.27 per 100,000 pregnancies, whereas the number of deaths from thrombosis and thromboembolism was 1.94 per 100,000 pregnancies.[92] The number of cardiac deaths during pregnancy has increased steadily since 1991. The most common causes of maternal death from cardiac disease are myocardial infarction, followed by aortic dissection.[92] A study completed in California also found that the incidence of myocardial infarction in pregnancy increased throughout the 1990s.[137] In addition, a nationwide review of myocardial infarction in pregnancy in the United States found that the risk of myocardial infarction in pregnancy is 3 to 4 times that of nonpregnant women of reproductive age.[138]

Women are deferring pregnancy to older ages, increasing the chance that they will have atherosclerotic heart disease. Because fibrinolytics are relatively contraindicated in pregnancy, PCI is the reperfusion strategy of choice for ST-elevation myocardial infarction.

The number of babies born with congenital heart disease who now survive to adulthood has increased exponentially over the last 3 decades.[139,140] It is estimated that 85% of neonates born with congenital heart disease will survive to adulthood. Therefore, more women with congenital heart disease are surviving to have children, which translates into higher risk for a cardiac event during pregnancy. In fact, illnesses related to congenital heart disease and pulmonary hypertension are the third most common cause of maternal cardiac deaths.[92]

Magnesium Sulfate Toxicity

Patients with magnesium toxicity present with cardiac effects ranging from ECG interval changes (prolonged PR, QRS and QT intervals) at magnesium levels of 2.5–5 mmol/L to AV nodal conduction block, bradycardia, hypotension and cardiac arrest at levels of 6–10 mmol/L. Neurological effects ranging from loss of tendon reflexes, sedation, severe muscular weakness, and respiratory depression are seen at levels of 4–5 mmol/L. Other signs of magnesium toxicity include gastrointestinal symptoms (nausea and vomiting), skin changes (flushing), and electrolyte/fluid abnormalities (hypophosphatemia, hyperosmolar dehydration). Patients with renal failure and metabolic derangements can develop toxicity after relatively lower magnesium doses.

Iatrogenic overdose is possible in the pregnant woman who receives magnesium sulfate, particularly if the woman becomes oliguric. Empirical calcium administration may be lifesaving in these cases.[141–143]

Preeclampsia/Eclampsia

Preeclampsia/eclampsia develops after the 20th week of gestation and can produce severe hypertension and ultimately diffuse organ-system failure. If untreated, maternal and fetal morbidity and mortality may result.

Life-Threatening Pulmonary Embolism (PE)

Successful use of fibrinolytics in pregnant women has been reported for massive, life-threatening PE[144–146] and ischemic stroke.[147] Pregnant women in cardiac arrest with suspected PE should be treated in accordance with the ACLS guidelines (see Part 12.5: "Cardiac Arrest Associated With Pulmonary Embolism").

Amniotic Fluid Embolism

Clinicians have reported successful use of cardiopulmonary bypass for pregnant women with a life-threatening amniotic fluid embolism during labor and delivery.[148] The use of perimortem cesarean section has resulted in maternal and neonatal survival.[149]

Anesthetic Complications

Anesthesia-related maternal morbidity and mortality continue to be a major concern, which has led to development of specialized obstetric anesthesia techniques.[118] Cardiac arrest may result from spinal shock as a result of regional anesthesia. Induction of general anesthesia may lead to loss of airway control or pulmonary aspiration, and emergence from anesthesia can be associated with hypoventilation or airway obstruction, leading to cardiac arrest.[150–155]

Maternal Cardiac Arrest Not Immediately Reversed by BLS and ACLS

Emergency Cesarean Section in Cardiac Arrest

Resuscitation team leaders should activate the protocol for an emergency cesarean delivery as soon as cardiac arrest is identified in a pregnant woman with an obviously gravid uterus. By the time the physician is ready to deliver the baby, standard ACLS should be underway and immediately reversible causes of cardiac arrest should be ruled out. When the gravid uterus is large enough to cause maternal hemodynamic changes due to aortocaval compression, emergency cesarean section should be considered, regardless of fetal viability.

What Defines a Gravid Uterus With the Potential to Cause Aortocaval Compression?

A study found that maternal aortocaval compression can occur for singleton pregnancies at ≥20 weeks of gestational age.[156] However, the exact gestational age at which aortocaval compression occurs is not consistent, especially with multiple-gestation pregnancies or intrauterine growth retardation, and gestational age and number of fetuses may not always be known in the emergency situation. Fundal height is often used to estimate gestational age. In a singleton gestation, by 20 weeks fundal height is approximately at the level of the umbilicus[157]; however the fundus may reach the umbilicus between 15 and 19 weeks of gestation.[158] Fundal height may also be skewed by other factors such as abdominal distention[157] and increased body mass index; therefore fundal height may be a poor predictor of gestational age.

One review of emergency cesarean sections in maternal cardiac arrest before the third trimester concluded that if the fundus extends above the level of the umbilicus, aortocaval compression can occur, and emergency cesarean section should be performed regardless of gestational age.[158]

Two cases of maternal cardiac arrest in early pregnancy of 13 to 15 weeks were reported in which the mother was resuscitated without an emergency cesarean section being performed and the pregnancy continued to successful delivery of a live infant at term.[159,160] Not every pregnant woman in cardiac arrest is a candidate for an emergency cesarean section; the decision depends on whether or not the gravid uterus is thought to interfere with maternal hemodynamics.

Why Perform an Emergency Cesarean Section in Cardiac Arrest?

Several case reports of emergency cesarean section in maternal cardiac arrest indicate a return of spontaneous circulation or improvement in maternal hemodynamic status only after the uterus has been emptied.[94–96,143,149,161–166] In a case series of 38 cases of perimortem cesarean section, 12 of 20 women for whom maternal outcome was recorded had return of spontaneous circulation immediately after delivery. No cases of worsened maternal status after cesarean section were reported.[166] The critical point to remember is that both mother and infant may die if the provider cannot restore blood flow to the mother's heart.[94]

The Importance of Timing With Emergency Cesarean Section

The 5-minute window that providers have to determine if cardiac arrest can be reversed by BLS and ACLS was first described in 1986 and has been perpetuated in specialty guidelines.[143,166] The rescue team is not required to wait 5 minutes before initiating emergency hysterotomy, and there are circumstances that support an earlier start.[157] For instance, in an obvious nonsurvivable injury,[166,167–169] when the maternal prognosis is grave and resuscitative efforts appear futile, moving straight to an emergency cesarean section may be appropriate, especially if the fetus is viable.

Many reports document long intervals between an urgent decision for hysterotomy and actual delivery of the infant, far exceeding the obstetric guideline of 30 minutes for patients not in arrest.[170,171] Very few cases of perimortem cesarean section fall within the recommended 5-minute period.[94,166] Survival of the mother has been reported with perimortem cesarean section performed up to 15 minutes after the onset of maternal cardiac arrest.[94,172–174] If emergency cesarean section cannot be performed by the 5-minute mark, it may be advisable to prepare to evacuate the uterus while the resuscitation continues. (Class IIb, LOE C).

At >24 to 25 weeks of gestation, the best survival rate for the infant occurs when the infant is delivered no more than 5 minutes after the mother's heart stops beating.[175–178] Typically this requires that the provider begin the hysterotomy about 4 minutes after cardiac arrest. At gestational ages ≥30 weeks, infant survival has been seen even when delivery occurred after 5 minutes from onset of maternal cardiac arrest.[166] In a recent retrospective cohort series, neonatal survival was documented when delivery occurred within 30 minutes after onset of maternal cardiac arrest.[94]

When there is an obvious gravid uterus, the emergency cesarean section team should be activated at the onset of maternal cardiac arrest (Class I, LOE B). Emergency cesarean section may be considered at 4 minutes after onset of maternal cardiac arrest if there is no return of spontaneous circulation (Class IIb, LOE C).

Institutional Preparation for Maternal Cardiac Arrest

Experts and organizations have emphasized the importance of preparation.[143,179] Providers at medical centers must review whether performance of an emergency hysterotomy is feasible, and if so, they must identify the best means of accomplishing this procedure rapidly. Team planning should be done in collaboration with the obstetric, neonatal, emergency, anesthesiology, intensive care, and cardiac arrest services (Class I, LOE C).

Post–Cardiac Arrest Care

One case report showed that post–cardiac arrest hypothermia can be used safely and effectively in early pregnancy without emergency cesarean section (with fetal heart monitoring), with favorable maternal and fetal outcome after a term delivery.[159] No cases in the literature have reported the use of therapeutic hypothermia with perimortem cesarean section. Therapeutic hypothermia may be considered on an individual basis after cardiac arrest in a comatose pregnant patient based on current recommendations for the nonpregnant patient (Class IIb, LOE C). 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).

Part 12.4: Cardiac Arrest in the Morbidly Obese

Morbid obesity can provide challenges during the resuscitation attempt. Airway management may be more challenging, and changes to the thorax may make resuscitative efforts more demanding. Evidence from 2 case studies,[180,181] 1 case series,[182] and 1 related clinical study[183] indicated no differences in survival based on patient weight. However, one large case series demonstrated lower survival for morbidly obese children who required in-hospital pediatric CPR.[184]

BLS and ACLS Modifications

No modifications to standard BLS or ACLS care have been proven efficacious, although techniques may need to be adjusted due to the physical attributes of individual patients.

Part 12.5: Cardiac Arrest Associated With Pulmonary Embolism

Pulmonary embolism (PE) can result in cardiovascular collapse and cardiac arrest. Although cardiac arrest caused by PE often presents as pulseless electric activity (PEA), not all cases of PEA are caused by PE.

ACLS Modifications

In patients with cardiac arrest and without known PE, routine fibrinolytic treatment given during CPR shows no benefit[185,186] and is not recommended (Class III, LOE A).

In patients with cardiac arrest and presumed PE, however, the use of fibrinolytics during CPR may improve the patient's chance of survival.[187–194] Despite the potential to increase the risk of severe bleeding, fibrinolytics may improve survival to discharge and long-term neurological function in patients with presumed PE-induced cardiac arrest.[193–196] Emergency echocardiography may be helpful in determining the presence of thrombus or PE.

In a small number of patients, percutaneous mechanical thromboembolectomy during CPR has been performed successfully.[189] Surgical embolectomy has also been used successfully in some patients with PE-induced cardiac arrest.[191,197,198]

In patients with cardiac arrest due to presumed or known PE, it is reasonable to administer fibrinolytics (Class IIa, LOE B). Survival has been described with percutaneous mechanical thrombectomy or surgical embolectomy with or without prior treatment with fibrinolysis.

Part 12.6: Cardiac Arrest Associated With Life-Threatening Electrolyte Disturbances

Electrolyte abnormalities can be associated with cardiovascular emergencies and may cause or contribute to cardiac arrest, hinder resuscitative efforts, and affect hemodynamic recovery after cardiac arrest. An evidence-based review in 2010 focused on electrolyte abnormalities most often associated with cardiac arrest.

Early consideration may be given to using selective methods of therapeutic management in addition to standard ACLS protocols that can be provided rapidly and have been shown to be effective in patients with cardiovascular instability as outlined below. Current BLS and ACLS should be used to manage cardiac arrest associated with all electrolyte disturbances.

Potassium (K+)

Potassium is maintained mainly in the intracellular compartment through the action of the Na+/K+ ATPase pump. The magnitude of the potassium gradient across cell membranes determines excitability of nerve and muscle cells, including the myocardium.

Potassium is tightly regulated. Under normal conditions potential differences across membranes, especially cardiac, are not affected by alterations in potassium level. Rapid or significant changes in serum concentrations of potassium result from the shifting of potassium from one space to another and may have life-threatening consequences.

Hyperkalemia

Hyperkalemia is one of the few potentially lethal electrolyte disturbances. Severe hyperkalemia (defined as a serum potassium concentration >6.5 mmol/L) occurs most commonly from renal failure or from release of potassium from cells and can cause cardiac arrhythmias and cardiac arrest. In 1 retrospective in-hospital study of 29 063 patients, hyperkalemia was found to be directly responsible for sudden cardiac arrest in 7 cases.[199] Acute kidney injury was present in all the arrest cases, accompanied by acute pancreatitis in 3 cases and acute hepatic failure in 2 cases. Overall renal failure and drug treatment were the most common causes of hyperkalemia, with the most severe cases occurring when excessive IV potassium was administered to a patient with renal insufficiency.

Although severe hyperkalemia may cause flaccid paralysis, paresthesia, depressed deep tendon reflexes, or respiratory difficulties,[200–202] the first indicator of hyperkalemia may be the presence of peaked T waves (tenting) on the electrocardiogram (ECG). As serum potassium rises, the ECG may progressively develop flattened or absent P waves, a prolonged PR interval, widened QRS complex, deepened S waves, and merging of S and T waves (Figure 5). If hyperkalemia is left untreated, a sine-wave pattern, idioventricular rhythms, and asystolic cardiac arrest may develop.[203,204]

ACLS Modifications in Management of Severe Cardiotoxicity or Cardiac Arrest Due to Hyperkalemia

Treatment of severe hyperkalemia aims at protecting the heart from the effects of hyperkalemia by antagonizing the effect of potassium on excitable cell membranes, forcing potassium into cells to remove it promptly from the circulation, and removing potassium from the body. Therapies that shift potassium will act rapidly but are temporary and thus may need to be repeated. In order of urgency, treatment includes the following:

- Stabilize myocardial cell membrane:
  - Calcium chloride (10%): 5 to 10 mL (500 to 1000 mg) IV over 2 to 5 minutes or calcium gluconate (10%): 15 to 30 mL IV over 2 to 5 minutes
- Shift potassium into cells:
  - Sodium bicarbonate: 50 mEq IV over 5 minutes

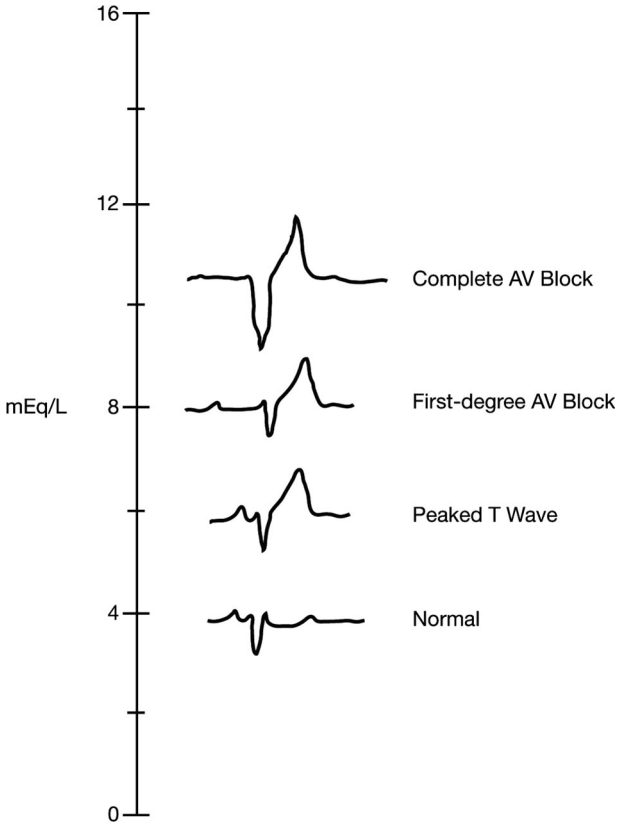

Figure 5. ECG changes in hyperkalemia.

- Glucose plus insulin: mix 25 g (50 mL of D50) glucose and 10 U regular insulin and give IV over 15 to 30 minutes
- Nebulized albuterol: 10 to 20 mg nebulized over 15 minutes

- Promote potassium excretion:
  - Diuresis: furosemide 40 to 80 mg IV
  - Kayexalate: 15 to 50 g plus sorbitol per oral or per rectum
  - Dialysis

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).

ACLS Modifications in Management of Severe Cardiotoxicity Due to Hypokalemia

Life-threatening hypokalemia is uncommon but can occur in the setting of gastrointestinal and renal losses and is associated with hypomagnesemia. Severe hypokalemia will alter cardiac tissue excitability and conduction. Hypokalemia can produce ECG changes such as U waves, T-wave flattening, and arrhythmias (especially if the patient is taking digoxin), particularly ventricular arrhythmias,[205,206] which, if left untreated, deteriorate to PEA or asystole.

Several studies reported an association with hypokalemia and development of ventricular fibrillation,[207–210] whereas a single animal study reported that hypokalemia lowered the ventricular fibrillation threshold.[211] However, the management of hypokalemia in the setting of cardiotoxicity, specifically torsades de pointes, is largely based on historical case reports that report slow infusion of potassium over hours.[212] 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).

Sodium (Na^+)

Sodium is the major intravascular ion that influences serum osmolality. Sodium abnormalities are unlikely to lead to cardiac arrest, and there are no specific recommendations for either checking or treating sodium during cardiac arrest. Disturbances in sodium level are unlikely to be the primary cause of severe cardiovascular instability.

Magnesium (Mg^{++})

Magnesium is an essential electrolyte and an important cofactor for multiple enzymes, including ATPase. Magnesium is necessary for the movement of sodium, potassium, and calcium into and out of cells and plays an important role in stabilizing excitable membranes. The presence of a low plasma magnesium concentration has been associated with poor prognosis in cardiac arrest patients.[208,213–216]

Hypermagnesemia

Hypermagnesemia is defined as a serum magnesium concentration >2.2 mEq/L (normal: 1.3 to 2.2 mEq/L). Neurological symptoms of hypermagnesemia include muscular weakness, paralysis, ataxia, drowsiness, and confusion. Hypermagnesemia can produce vasodilation and hypotension.[217] Extremely high serum magnesium levels may produce a depressed level of consciousness, bradycardia, cardiac arrhythmias, hypoventilation, and cardiorespiratory arrest.[208,215,216]

ACLS Modifications in Management of Cardiac Arrest and Severe Cardiotoxicity Due to Hypermagnesemia

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).

Hypomagnesemia

Hypomagnesemia, defined as a serum magnesium concentration <1.3 mEq/L, is far more common than hypermagnesemia. Hypomagnesemia usually results from decreased absorption or increased loss of magnesium from either the kidneys or intestines (diarrhea). Alterations in thyroid hormone function, certain medications (eg, pentamidine, diuretics, alcohol), and malnourishment can also induce hypomagnesemia.

ACLS Modifications in Management of Cardiac Arrest and Severe Cardiotoxicity Due to Hypomagnesemia

Hypomagnesemia can be associated with polymorphic ventricular tachycardia, including torsades de pointes, a pulseless form (polymorphic) of ventricular tachycardia. For cardiotoxicity and cardiac arrest, IV magnesium 1 to 2 g of $MgSO_4$ bolus IV push is recommended (Class I, LOE C).

Calcium (Ca^{++})

Calcium abnormality as an etiology of cardiac arrest is rare. There are no studies evaluating the treatment of hypercalcemia or hypocalcemia during arrest. However, 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).

Part 12.7: Cardiac Arrest Associated With Toxic Ingestions

Poisoning has been likened to trauma on the cellular level, destroying the natural workings of a victim's physiology.[218] Severe poisoning alters the function of a cellular receptor, ion channel, organelle, or chemical pathway to the extent that critical organ systems can no longer support life.

As with any patient in cardiac arrest, management of the patient with a toxic exposure begins with support of airway, breathing, and circulation. Cardiac arrest due to toxicity is managed in accordance with the current standards of BLS and ACLS. With few exceptions, there are no unique antidotes or toxin-specific interventions that are recommended during resuscitation from cardiac arrest.

Once return of spontaneous circulation is achieved, urgent consultation with a medical toxicologist or certified regional poison center is recommended, as the postarrest management of the critically poisoned patient may benefit from a thorough understanding of the toxic agent. Consultation is also recommended early in the management of a patient with potentially life-threatening poisoning, when appropriate interventions might prevent deterioration to cardiac arrest. In the United States a certified poison center can be reached by calling 1-800-222-1222; in Canada, call 1-800-268-9017.

It is extremely difficult to conduct clinical trials of acute life-threatening poisoning. Challenges include the infrequency with which most specific conditions occur, the heterogeneity of presentation, and ethical challenges related to withholding established care from patients who are unable to provide informed consent because the patient has an altered mental status, the patient is suicidal, or there is a lack of time to explain treatment alternatives.[219]

The majority of questions addressing cardiac arrest due to drug toxicity remain unanswered. Epidemiological studies are required to document the incidence rate of cardiac arrests secondary to drug toxicity and the safety and efficacy baseline rates for current therapeutic strategies. This section presents recommendations for the care of the patient with a toxicological problem causing cardiac arrest or severe cardiovascular instability (respiratory depression, hypotension, life-threatening alterations of cardiac conduction, etc). Some recommendations are evidence-based, but most research in this area consists of case reports, small case series, animal studies, and pharmacokinetic studies in healthy volunteers. Virtually no toxicology research involves human cardiac arrest. Thus, many of these recommendations are based on expert consensus, and further research is needed to validate them.

Initial Approach to the Critically Poisoned Patient

Management of the critically poisoned patient begins with airway protection, support of respiration and circulation, and rapid assessment. Patients may or may not be able to provide an accurate history of exposure to a toxic substance. Whenever possible, history gathering should include questioning of persons who accompany the patient, evaluation of containers, review of pharmacy records, and examination of the patient's prior medical record.[220] Many patients who ingest medications in a suicide attempt take more than 1 substance, and the number of substances ingested is greater in fatal than in nonfatal suicide attempts.[221] Comprehensive toxicology laboratory testing is virtually never available in a time frame that supports early resuscitation decisions.[222]

Poisoned patients may deteriorate rapidly. Care for all adult patients who are critically ill or under evaluation for possible toxin exposure or ingestion, particularly when the history is uncertain, should begin in a monitored treatment area where the development of central nervous system depression, hemodynamic instability, or seizures can be rapidly recognized and addressed.[223]

Gastrointestinal decontamination, once a mainstay in the management of ingested toxins, has a less significant role in poisoning treatment today. With rare exceptions, gastric lavage, whole bowel irrigation, and administration of syrup of ipecac are no longer recommended.[224–226] Administration of single-dose activated charcoal to adsorb ingested toxins is generally recommended for the ingestion of life-threatening poisons for which no adequate antidotal therapy is available and when the charcoal can be administered within 1 hour of poisoning.[228] Multiple-dose activated charcoal should be considered for patients who have ingested a life-threatening amount of specific toxins (eg, carbamazepine, dapsone, phenobarbital, quinine, or theophylline) for which a benefit of this strategy has been established.[229] Charcoal should not be administered for ingestions of caustic substances, metals, or hydrocarbons.[228]

Charcoal should only be administered to patients with an intact or protected airway. In patients who are at risk for aspiration, endotracheal intubation and head-of-bed elevation should be performed before charcoal administration.[229,230] Because the decision to perform gastrointestinal decontamination is complex, multifactorial, and associated with risk, expert advice can be helpful.

Toxidromes

A "toxidrome" is a clinical syndrome—a constellation of signs, symptoms, and laboratory findings—suggestive of the effects of a specific toxin. By recognizing these presentations, the clinician can establish a working diagnosis that guides initial management. Some common toxidromes are presented in the Table. Practically every sign and symptom observed in poisoning can be produced by natural disease, and many clinical presentations associated with natural disease can be mimicked by some poison.[231] It is important to maintain a broad differential diagnosis, particularly when the history of toxic chemical exposure is unclear.

Opioid Toxicity

There are no data to support the use of specific antidotes in the setting of cardiac arrest due to opioid overdose. Resuscitation from cardiac arrest should follow standard BLS and ACLS algorithms.

Naloxone is a potent antagonist of the binding of opioid medications to their receptors in the brain and spinal cord. Administration of naloxone can reverse central nervous system

Table. Common Toxidromes*

Cardiac Signs		
Tachycardia and/or Hypertension	Bradycardia and/or Hypotension	Cardiac Conduction Delays (Wide QRS)
Amphetamines	Beta blockers	Cocaine
Anticholinergic drugs	Calcium channel blockers	Cyclic antidepressants
Antihistamines	Clonidine	Local anesthetics
Cocaine	Digoxin and related glycosides	Propoxyphene
Theophylline/caffeine	Organophosphates and carbamates	Antiarrhythmics (e.g., quinidine, flecainide)
Withdrawal states		

CNS/Metabolic Signs		
Seizures	CNS and/or Respiratory Depression	Metabolic Acidosis
Cyclic antidepressants	Antidepressants (several classes)	Cyanide
Isoniazid	Benzodiazepines	Ethylene glycol
Selective and non-selective norepinephrine reuptake inhibitors (eg, bupropion)	Carbon monoxide	Metformin
Withdrawal states	Ethanol	Methanol
	Methanol	Salicylates
	Opioids	
	Oral hypoglycemics	

*Differential diagnosis lists are partial.

and respiratory depression caused by opioid overdose. Naloxone has no role in the management of cardiac arrest.

In the patient with known or suspected opioid overdose with respiratory depression who is not in cardiac arrest, ventilation should be assisted by a bag mask,[232–238] followed by administration of naloxone and placement of an advanced airway if there is no response to naloxone (Class I, LOE A).

Administration of naloxone can produce fulminate opioid withdrawal in opioid-dependent individuals, leading to agitation, hypertension, and violent behavior. For this reason, naloxone administration should begin with a low dose (0.04 to 0.4 mg), with repeat dosing or dose escalation to 2 mg if the initial response is inadequate.[239] Some patients may require much higher doses to reverse intoxication with atypical opioids, such as propoxyphene, or following massive overdose ingestions.[240,241] Naloxone can be given IV,[235,236,242,243] IM,[232,235,236] intranasally,[232,242] and into the trachea.[244]

The duration of action of naloxone is approximately 45 to 70 minutes, but respiratory depression caused by ingestion of a long-acting opioid (eg, methadone) may last longer. Thus, the clinical effects of naloxone may not last as long as those of the opioid, and repeat doses of naloxone may be needed.

Patients with life-threatening central nervous system or respiratory depression reversed by naloxone administration should be observed for resedation. Although a brief period of observation may be appropriate for patients with morphine or heroin overdose,[245] a longer period of observation may be required to safely discharge a patient with life-threatening overdose of a long-acting or sustained-release opioid.[239,246]

Benzodiazepines

There are no data to support the use of specific antidotes in the setting of cardiac arrest due to benzodiazepine overdose. Resuscitation from cardiac arrest should follow standard BLS and ACLS algorithms.

Flumazenil is a potent antagonist of the binding of benzodiazepines to their central nervous system receptors. Administration of flumazenil can reverse central nervous system and respiratory depression caused by benzodiazepine overdose. Flumazenil has no role in the management of cardiac arrest.

The administration of flumazenil to patients with undifferentiated coma confers risk and is not recommended (Class III, LOE B). Flumazenil administration can precipitate seizures in benzodiazepine-dependent patients and has been associated with seizures, arrhythmia, and hypotension in patients with coingestion of certain medications, such as tricyclic antidepressants.[247,248] However, flumazenil may be used safely to reverse excessive sedation known to be due to the use of benzodiazepines in a patient without known contraindications (eg, procedural sedation).[249]

β-Blockers

There are no data to support the use of specific antidotes in the setting of cardiac arrest due to β-blocker overdose. Resuscitation from cardiac arrest should follow standard BLS and ACLS algorithms.

β-Blocker medication overdose may cause such severe inhibition of β-adrenergic receptors that high-dose vasopressors cannot effectively restore blood pressure, cardiac output, or perfusion. Therapeutic options in the treatment of refractory hemodynamic instability due to β-blocker overdose include administration of glucagon, high-dose insulin, or IV calcium salts.

Glucagon

Administration of glucagon may be helpful for severe cardiovascular instability associated with β-blocker toxicity that is refractory to standard measures, including vasopressors. 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).[250–262] The infusion rate is titrated to achieve an adequate hemodynamic response (appropriate mean arterial pressure and evidence of good perfusion). Because the amount of glucagon required to sustain this therapy may exceed 100 mg in a 24-hour period, plans should be made early to ensure that an adequate supply of glucagon is available. Glucagon commonly causes vomiting. In patients with central nervous system depression, the airway must be protected before glucagon administration. Animal studies have suggested that the concomitant use of dopamine alone or in combination with isoproterenol and milrinone may decrease the effectiveness of glucagon.[263–265]

Insulin

Animal studies suggest that high-dose IV insulin, accompanied by IV dextrose supplementation and electrolyte monitoring, may improve hemodynamic stability and survival in β-blocker overdose by improving myocardial energy utilization.[266,267] A single human case report[268] showed improved hemodynamic stability

and survival to discharge following administration of high-dose insulin in refractory shock due to a massive overdose of metoprolol. Administration of high-dose insulin in patients with shock refractory to other measures may be considered (Class IIb, LOE C).

Although the ideal human dose has not been determined, a commonly used protocol calls for IV administration of 1 U/kg regular insulin as a bolus, accompanied by 0.5 g/kg dextrose, followed by continuous infusions of 0.5 to 1 U/kg per hour of insulin and 0.5 g/kg per hour of dextrose.[269] The insulin infusion is titrated as needed to achieve adequate hemodynamic response, whereas the dextrose infusion is titrated to maintain serum glucose concentrations of 100 to 250 mg/dL (5.5 to 14 mmol/L). Very frequent serum glucose monitoring (up to every 15 minutes) may be needed during the initial phase of dextrose titration. Sustained infusions of concentrated dextrose solutions (<10%) require central venous access. Insulin causes potassium to shift into the cells. Moderate hypokalemia is common during high-dose insulin-euglycemia therapy, and animals treated with aggressive potassium repletion developed asystole.[266] To avoid overly aggressive potassium repletion, 1 human protocol targets potassium levels of 2.5 to 2.8 mEq/L.[269]

Calcium

One human case report[270] and a large-animal study[271] suggest that calcium may be helpful in β-blocker overdose. Administration of calcium in patients with shock refractory to other measures may be considered (Class IIb, LOE C).

One approach is to administer 0.3 mEq/kg of calcium (0.6 mL/kg of 10% calcium gluconate solution or 0.2 mL/kg of 10% calcium chloride solution) IV over 5 to 10 minutes, followed by an infusion of 0.3 mEq/kg per hour.[269] The infusion rate is titrated to adequate hemodynamic response. Serum ionized calcium levels should be monitored, and severe hypercalcemia (ionized calcium levels greater than twice the upper limits of normal) should be avoided. Sustained infusions of IV calcium require central venous access.

Other Therapies

Case reports have suggested that in patients who remain critically hypotensive despite maximal vasopressor therapy, specific interventions using intra-aortic balloon counterpulsation, ventricular assist devices, and extracorporeal membrane oxygenation or other extra corporeal life support (ECLS) devices may be lifesaving.[272–274] While evidence remains weak, at least two human case reports indicate a possible benefit from lipid emulsion infusion for overdose by β-blockers.[275,276] Animal studies are mixed.[277–280] Because this area of therapy is rapidly evolving,[281–283] prompt consultation with a medical toxicologist or other specialists with up-to-date knowledge is recommended when managing treatment-refractory hypotension from β-blocker overdosage.

Calcium Channel Blockers

There are no data to support the use of specific antidotes in the setting of cardiac arrest due to calcium channel blocker overdose. Resuscitation from cardiac arrest should follow standard BLS and ACLS algorithms.

Calcium channel blocker overdose also may cause life-threatening hypotension and bradycardia that are refractory to standard agents. Treatment with high-dose insulin has been described in a number of clinical case reports[284–295] and animal studies.[296–299] 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).

Limited evidence supports the use of calcium in the treatment of hemodynamically unstable calcium channel blocker overdose refractory to other treatments.[285,286,289,290,292–294,297,300–303] Administration of calcium in patients with shock refractory to other measures may be considered (Class IIb, LOE C).

There is insufficient and conflicting evidence to recommend the use of glucagon[289,290,294,296,297,300,303–306] in the treatment of hemodynamically unstable calcium channel blocker overdose.

Digoxin and Related Cardiac Glycosides

Digoxin poisoning can cause severe bradycardia and life-threatening arrhythmias, including ventricular tachycardia, ventricular fibrillation, and high degrees of AV nodal blockade. Other plant- and animal-derived cardiac glycosides may produce similar effects, including those found in oleander, lily-of-the-valley, toad skin, and some herbal medications. There are no data to support the use of specific antidotes in the setting of cardiac arrest due to digoxin overdose. Resuscitation from cardiac arrest should follow standard BLS and ACLS algorithms, with specific antidotes used in the post-cardiac arrest phase if severe cardiotoxicity is encountered.

Antidigoxin Fab antibodies should be administered to patients with severe life-threatening cardiac glycoside toxicity (Class I, LOE B).[307–316] One vial of antidigoxin Fab is standardized to neutralize 0.5 mg of digoxin. Although the ideal dose is unknown, a reasonable strategy is as follows:

- If the ingested dose of digoxin is known, administer 2 vials of Fab for every milligram of digoxin ingested.
- In cases of chronic digoxin toxicity or when the ingested dose is not known, calculate the number of vials to administer by using the following formula: serum digoxin concentration (ng/mL)×weight (kg)/100.
- In critical cases in which therapy is required before a serum digoxin level can be obtained or in cases of life-threatening toxicity due to cardiac glycosides, administer empirically 10 to 20 vials.

Hyperkalemia is a marker of severity in acute cardiac glycoside poisoning and is associated with poor prognosis.[317] Antidigoxin Fab may be administered empirically to patients with acute poisoning from digoxin or related cardiac glycosides whose serum potassium level exceeds 5.0 mEq/L.[318]

Cocaine

There are no data to support the use of cocaine-specific interventions in the setting of cardiac arrest due to cocaine overdose. Resuscitation from cardiac arrest should follow standard BLS and ACLS algorithms, with specific antidotes used in the postresuscitation phase if severe cardiotoxicity or neurotoxicity is encountered. A single case series demon-

strated excellent overall and neurologically intact survival (55%) in patients with cardiac arrest associated with cocaine overdose who were treated with standard therapy.[319]

Cocaine-induced tachycardia and hypertension are predominantly caused by central nervous system stimulation. Treatment strategies are extrapolated from acute coronary syndrome studies, small case series, and experiments in cocaine-naïve human volunteers. It may be reasonable to try agents that have shown efficacy in the management of acute coronary syndrome in patients with severe cardiovascular toxicity. α-Blockers (phentolamine),[320] benzodiazepines (lorazepam, diazepam),[321] calcium channel blockers (verapamil),[322] morphine,[323] and sublingual nitroglycerin[324,325] may be used as needed to control hypertension, tachycardia, and agitation (Class IIb, LOE B). 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).

There is clear evidence that cocaine can precipitate acute coronary syndromes.[326] For cocaine-induced hypertension or chest discomfort, benzodiazepines, nitroglycerin, and/or morphine can be beneficial (Class IIa, LOE B).[321,324,327] Because the effects of cocaine and other stimulant medications are transient, drugs and doses should be chosen carefully to minimize the risk of producing hypotension after the offending agent has been metabolized. Catheterization laboratory studies demonstrate that cocaine administration leads to reduced coronary artery diameter. This effect is reversed by morphine,[323] nitroglycerin,[325] phentolamine,[320] and verapamil[322]; is not changed by labetalol[328]; and is exacerbated by propranolol.[329] Several studies suggest that administration of β-blockers may worsen cardiac perfusion and/or produce paradoxical hypertension when cocaine is present.[329,330] Although contradictory evidence exists,[331,332] current recommendations are that pure β-blocker medications in the setting of cocaine are not indicated (Class IIb, LOE C).[333]

In severe overdose, cocaine acts as a Vaughan-Williams class Ic antiarrhythmic, producing wide-complex tachycardia through several mechanisms, including blockade of cardiac sodium channels.[107] Although there is no human evidence in cocaine poisoning, extrapolation from evidence in the treatment of wide-complex tachycardia caused by other class Ic agents (flecainide) and tricyclic antidepressants suggests that administration of hypertonic sodium bicarbonate may be beneficial.[334] A typical treatment strategy used for these other sodium channel blockers involves administration of 1 mL/kg of sodium bicarbonate solution (8.4%, 1 mEq/mL) IV as a bolus, repeated as needed until hemodynamic stability is restored and QRS duration is $\leq$120 ms.[335–342] Current evidence neither supports nor refutes a role for lidocaine in the management of wide-complex tachycardia caused by cocaine.

Cyclic Antidepressants

Many drugs can prolong the QRS interval in overdose. These include Vaughan-Williams class Ia and Ic antiarrhythmics (eg, procainamide, quinidine, flecainide), cyclic antidepressants (eg, amitriptyline), and cocaine. Type Ia and Ic antiarrhythmics were not reviewed in 2010. Similar to the type Ia antiarrhythmics, cyclic antidepressants block cardiac sodium channels, leading to hypotension and wide-complex arrhythmia in overdose.

Cardiac arrest caused by cyclic antidepressant toxicity should be managed by current BLS and ACLS treatment guidelines. A small case series of cardiac arrest patients demonstrated improvement with sodium bicarbonate and epinephrine,[343] but the concomitant use of physostigmine in the prearrest period in this study reduces the ability to generalize this study. Administration of sodium bicarbonate for cardiac arrest due to cyclic antidepressant overdose may be considered (Class IIb, LOE C).

Therapeutic strategies for treatment of severe cyclic antidepressant cardiotoxicity include increasing serum sodium, increasing serum pH, or doing both simultaneously. The relative contributions of hypernatremia and alkalemia are controversial, but in practice most experience involves administration of hypertonic sodium bicarbonate solution (8.4% solution, 1 mEq/mL). 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).[335–342] Serum sodium levels and pH should be monitored, and severe hypernatremia (sodium $>$155 mEq/L) and alkalemia (pH $>$7.55) should be avoided. A number of vasopressors and inotropes have been associated with improvement in the treatment of tricyclic-induced hypotension, ie, epinephrine,[239,344,345] norepinephrine,[345–348] dopamine,[348–350] and dobutamine.[349]

Local Anesthetic Toxicity

Inadvertent intravascular administration of local anesthetics, such as bupivacaine, mepivacaine, or lidocaine, can produce refractory seizures and rapid cardiovascular collapse leading to cardiac arrest. Clinical case reports[351–355] and controlled animal studies[356–360] have suggested that rapid IV infusion of lipids may reverse this toxicity either by redistributing the local anesthetic away from its site of action or by augmenting metabolic pathways within the cardiac myocyte.

Case reports have shown return of spontaneous circulation in patients with prolonged cardiac arrest unresponsive to standard ACLS measures,[361,362] suggesting a role for administration of IV lipids during cardiac arrest. Although ideal dosing has not been determined, because dosage varied across all studies, it may be reasonable to consider 1.5 mL/kg of 20% long-chain fatty acid emulsion as an initial bolus, repeated every 5 minutes until cardiovascular stability is restored (Class IIb, LOE C).[363] After the patient is stabilized, some papers suggest a maintenance infusion of 0.25 mL/kg per minute for at least 30 to 60 minutes. A maximum cumulative dose of 12 mL/kg has been proposed.[363]

Some animal data suggest that lipid infusion alone may be more effective than standard doses of epinephrine or vasopressin.[357,360] Although there is limited evidence to change routine care for severe cardiotoxicity, several professional societies advocate protocolized clinical use.[364–366] Because this is a rapidly evolving clinical area,[367,368] prompt consultation with a medical toxicologist, anesthesiologist, or other specialist with up-to-date knowledge is strongly recommended.

Carbon Monoxide

Apart from complications from deliberate drug abuse, carbon monoxide is the leading cause of unintentional poisoning death in the United States.[369] In addition to reducing the ability of hemoglobin to deliver oxygen, carbon monoxide causes direct cellular damage to the brain and myocardium.[370] Survivors of carbon monoxide poisoning are at risk for lasting neurological injury.[370]

Several studies have suggested that very few patients who develop cardiac arrest from carbon monoxide poisoning survive to hospital discharge, regardless of treatment administered following return of spontaneous circulation.[371–373] Routine care of patients in cardiac arrest and severe cardiotoxicity from carbon monoxide poisoning should comply with standard BLS and ACLS recommendations.

Hyperbaric Oxygen

Two studies suggest that neurological outcomes were improved in patients with carbon monoxide toxicity of all severity (excluding "moribund" patients)[374] and mild to moderate severity (excluding loss of consciousness and cardiac instability)[375] who received hyperbaric oxygen therapy for carbon monoxide poisoning. Other studies found no difference in neurologically intact survival.[376,377] A systematic review[378,379] and a recent evidence-based clinical policy review[380] concluded that, based on the available evidence, improvement in neurologically intact survival following treatment for carbon monoxide poisoning with hyperbaric oxygen is possible but unproven.

Hyperbaric oxygen therapy is associated with a low incidence of severe side effects. Because hyperbaric oxygen therapy appears to confer little risk,[380] 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).

Patients with carbon monoxide poisoning who develop a cardiac injury have an increased risk of cardiovascular and all-cause mortality for at least 7 years after the event, even if hyperbaric oxygen is administered.[381,382] Although data about effective interventions in this population are lacking, it is reasonable to advise enhanced follow-up for these patients.

On the basis of this conflicting evidence, the routine transfer of patients to a hyperbaric treatment facility following resuscitation from severe cardiovascular toxicity should be carefully considered, weighing the risk of transport against the possible improvement in neurologically intact survival.

Cyanide

Cyanide is a surprisingly common chemical. In addition to industrial sources, cyanide can be found in jewelry cleaners, electroplating solutions, and as a metabolic product of the putative antitumor drug amygdalin (laetrile). Cyanide is a major component of fire smoke, and cyanide poisoning must be considered in victims of smoke inhalation who have hypotension, central nervous system depression, metabolic acidosis, or soot in the nares or respiratory secretions.[383] Cyanide poisoning causes rapid cardiovascular collapse, which manifests as hypotension, lactic acidosis, central apnea, and seizures.

Patients in cardiac arrest[383–385] or those presenting with cardiovascular instability[383–389] caused by known or suspected cyanide poisoning should receive cyanide-antidote therapy with a cyanide scavenger (either IV hydroxocobalamin or a nitrate such as IV sodium nitrite and/or inhaled amyl nitrite), followed as soon as possible by IV sodium thiosulfate.[387,390,391]

Both hydroxocobalamin[383–389] and sodium nitrite[387,390,391] serve to rapidly and effectively bind cyanide in the serum and reverse the effects of cyanide toxicity. Because nitrites induce methemoglobin formation[390] and can cause hypotension,[392] hydroxocobalamin has a safety advantage, particularly in children and victims of smoke inhalation who might also have carbon monoxide poisoning. A detailed comparison of these measures has been recently published.[393]

Sodium thiosulfate serves as a metabolic cofactor, enhancing the detoxification of cyanide to thiocyanate. Thiosulfate administration enhances the effectiveness of cyanide scavengers in animal experimentation[394–397] and has been used successfully in humans with both hydroxocobalamin[383,389] and sodium nitrite.[387,390,391] Sodium thiosulfate is associated with vomiting but has no other significant toxicity.[398] Therefore, 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).

Part 12.8: Cardiac Arrest Associated With Trauma

BLS and ACLS for the trauma patient are fundamentally the same as that for the patient with primary cardiac arrest, with focus on support of airway, breathing, and circulation. In addition, reversible causes of cardiac arrest need to considered. While CPR in the pulseless trauma patient has overall been considered futile, several reversible causes of cardiac arrest in the context of trauma are correctible and their prompt treatment could be life-saving. These include hypoxia, hypovolemia, diminished cardiac output secondary to pneumothorax or pericardial tamponade, and hypothermia.

BLS Modifications

When multisystem trauma is present or trauma involves the head and neck, the cervical spine must be stabilized. A jaw thrust should be used instead of a head tilt–chin lift to establish a patent airway. If breathing is inadequate and the patient's face is bloody, ventilation should be provided with a barrier device, a pocket mask, or a bag-mask device while maintaining cervical spine stabilization. Stop any visible hemorrhage using direct compression and appropriate dressings. If the patient is completely unresponsive despite rescue breathing, provide standard CPR and defibrillation as indicated.

ACLS Modifications

After initiation of BLS care, if bag-mask ventilation is inadequate, an advanced airway should be inserted while maintaining cervical spine stabilization. If insertion of an advanced airway is not possible and ventilation remains

inadequate, experienced providers should consider a cricothyrotomy.

A unilateral decrease in breath sounds during positive-pressure ventilation should prompt the rescuer to consider the possibility of pneumothorax, hemothorax, or rupture of the diaphragm.

When the airway, oxygenation, and ventilation are adequate, evaluate and support circulation. Control ongoing bleeding where possible and replace lost volume if the losses appear to have significantly compromised circulating blood volume. Cardiac arrest resuscitation will likely be ineffective in the presence of uncorrected severe hypovolemia.

Treatment of PEA requires identification and treatment of reversible causes, such as severe hypovolemia, hypothermia, cardiac tamponade, or tension pneumothorax.[399] Development of bradyasystolic rhythms often indicates the presence of severe hypovolemia, severe hypoxemia, or cardiorespiratory failure. Ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT) are treated with CPR and defibrillation. For treatment recommendations regarding cardiac tamponade in traumatic cardiac arrest, see Part 12.14: "Cardiac Arrest Caused by Cardiac Tamponade."

Resuscitative thoracotomy may be indicated in selected patients. A review of the literature from 1966 to 1999, carried out by the American College of Surgeons Committee on Trauma, found a survival rate of 7.8% (11.2% for penetrating injuries and 1.6% for blunt lesions) in trauma victims who would otherwise have 100% mortality.[400] Practitioners should consult the guidelines for withholding or terminating resuscitation, which were developed for victims of traumatic cardiac arrest by a joint committee of the National Association of EMS Physicians and the American College of Surgeons Committee on Trauma.[401,402]

Commotio Cordis

Commotio cordis is VF triggered by a blow to the anterior chest during a cardiac repolarization.[403,404] Blunt cardiac injury may result in cardiac contusion with injured myocardium and risk of ECG changes and arrhythmias. Even a small blow to the anterior chest during a cardiac repolarization, such as that imparted by the strike of a baseball or hockey puck, may trigger VF, so-called commotio cordis.[405] Events causing commotio cordis are most commonly seen in young persons up to 18 years of age who are engaged in sports but may occur during daily activities. Prompt recognition that a precordial blow may cause VF is critical. Rapid defibrillation is often life-saving for these frequently young victims of cardiac arrest. Provision of immediate BLS care using an automated external defibrillator (AED) and ACLS for VF in this setting is appropriate.

Part 12.9: Cardiac Arrest in Accidental Hypothermia

Unintentional or accidental hypothermia is a serious and preventable health problem. Severe hypothermia (body temperature <30°C [86°F]) is associated with marked depression of critical body functions, which may make the victim appear clinically dead during the initial assessment. Therefore, life-saving procedures should be initiated unless the victim is obviously dead (eg, rigor mortis, decomposition, hemisection, decapitation). The victim should be transported as soon as possible to a center where aggressive rewarming during resuscitation is possible.

Initial Care for Victims of Accidental Hypothermia

When the victim is extremely cold but has maintained a perfusing rhythm, the rescuer should focus on interventions that prevent further loss of heat and begin to rewarm the victim immediately. Additional interventions include the following:

- Preventing additional evaporative heat loss by removing wet garments and insulating the victim from further environmental exposures. Passive rewarming is generally adequate for patients with mild hypothermia (temperature >34°C [93.2°F]).
- For patients with moderate (30°C to 34°C [86°F to 93.2°F]) hypothermia with a perfusing rhythm, external warming techniques are appropriate.[406] Passive rewarming alone will be inadequate for these patients.[407]
- For patients with severe hypothermia (<30°C [86°F]) with a perfusing rhythm, core rewarming is often used, although some have reported successful rewarming with active external warming techniques.[408,409] Active external warming techniques include forced air or other efficient surface-warming devices.
- Patients with severe hypothermia and cardiac arrest can be rewarmed most rapidly with cardiopulmonary bypass.[406,410–415] Alternative effective core rewarming techniques include warm-water lavage of the thoracic cavity[413,416–420] and extracorporeal blood warming with partial bypass.[421–423]
- Adjunctive core rewarming techniques include warmed IV or intraosseous (IO) fluids and warm humidified oxygen.[424] Heat transfer with these measures is not rapid, and should be considered supplementary to active warming techniques.
- Do not delay urgent procedures such as airway management and insertion of vascular catheters. Although these patients may exhibit cardiac irritability, this concern should not delay necessary interventions.

Beyond these critical initial steps, the treatment of severe hypothermia (temperature <30°C [86°F]) in the field remains controversial. Many providers do not have the time or equipment to assess core body temperature or to institute aggressive rewarming techniques, although these methods should be initiated when available.

BLS Modifications

When the victim is hypothermic, pulse and respiratory rates may be slow or difficult to detect,[425,426] and the ECG may even show asystole. If the hypothermic victim has no signs of life, begin CPR without delay. If the victim is not breathing, start rescue breathing immediately.

The temperature at which defibrillation should first be attempted in the severely hypothermic patient and the number of defibrillation attempts that should be made have not been

established. There are case reports of refractory ventricular arrhythmias with severe hypothermia; however, in a recent animal model it was found that an animal with a temperature of as low as 30°C had a better response to defibrillation than did normothermic animals in arrest.[427,428]

If VT or VF is present, defibrillation should be attempted. If VT or VF persists after a single shock, the value of deferring subsequent defibrillations until a target temperature is achieved is uncertain. It may be reasonable to perform further defibrillation attempts according to the standard BLS algorithm concurrent with rewarming strategies (Class IIb, LOE C).

To prevent further loss of core heat, remove wet garments and protect the victim from additional environmental exposure. Insofar as possible, this should be done while providing initial BLS therapies. Rewarming should be attempted when feasible.

ACLS Modifications

For unresponsive patients or those in arrest, advanced airway insertion is appropriate as recommended in the standard ACLS guidelines. Advanced airway management enables effective ventilation with warm, humidified oxygen and reduces the likelihood of aspiration in patients in periarrest.

ACLS management of cardiac arrest due to hypothermia focuses on aggressive active core rewarming techniques as the primary therapeutic modality. Conventional wisdom indicates that the hypothermic heart may be unresponsive to cardiovascular drugs, pacemaker stimulation, and defibrillation; however, the data to support this are essentially theoretical.[429] In addition, drug metabolism may be reduced, and there is a theoretical concern that medications could accumulate to toxic levels in the peripheral circulation if given repeatedly to the severely hypothermic victim. For these reasons, previous guidelines suggest withholding IV drugs if the victim's core body temperature is <30°C (86°F).

In the last decade a number of animal investigations have been performed evaluating both vasopressors and antiarrhythmic medications that could challenge some of this conventional wisdom.[430–435] In a meta-analysis of these studies, Wira et al[436] found that vasopressor medications (ie, epinephrine or vasopressin) increased rates of return of spontaneous circulation (ROSC) when compared with placebo (62% versus 17%; $P<0.0001$, n=77). Coronary perfusion pressures were increased in groups that received vasopressors compared with placebo. But groups given antiarrhythmics showed no improvement in ROSC when compared with control groups, although sample sizes were relatively small (n=34 and n=40, respectively).

One small-animal investigation suggested that the application of standard normothermic ACLS algorithms using both drugs (ie, epinephrine and amiodarone) and defibrillation improved ROSC compared with a placebo arm of defibrillation only (91% versus 30%; $P<0.01$; n=21). Human trials of medication use in accidental hypothermia do not exist, although case reports of survival with use of intra-arrest medication have been reported.[414,418,437]

Given the lack of human evidence and relatively small number of animal investigations, the recommendation for administration or withholding of medications is not clear. 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).

After ROSC

After ROSC, patients should continue to be warmed to a goal temperature of approximately 32° to 34°C; this can be maintained according to standard postarrest guidelines for mild to moderate hypothermia in patients for whom induced hypothermia is appropriate. For those with contraindications to induced hypothermia, rewarming can continue to normal temperatures.

Because severe hypothermia is frequently preceded by other disorders (eg, drug overdose, alcohol use, or trauma), the clinician must look for and treat these underlying conditions while simultaneously treating hypothermia.

Withholding and Cessation of Resuscitative Efforts

Multiple case reports indicate survival from accidental hypothermia even with prolonged CPR and downtimes.[410,422] Thus, patients with severe accidental hypothermia and cardiac arrest may benefit from resuscitation even in cases of prolonged downtime and prolonged CPR. Low serum potassium may indicate hypothermia, and not hypoxemia, as the primary cause of the arrest.[438] Patients should not be considered dead before warming has been provided.

Part 12.10: Cardiac Arrest in Avalanche Victims

Avalanche-related deaths are on the rise in North America due to winter recreational activities, including backcountry skiing and snowboarding, helicopter and snowcat skiing, snowmobiling, out-of-bounds skiing, ice climbing, mountaineering, and snowshoeing. The most common causes of avalanche-related death are asphyxia, trauma, and hypothermia, or combinations of the 3. Rescue and resuscitation strategies focus on management of asphyxia and hypothermia, because most field research has been done on these 2 conditions.

Avalanches occur in areas that are difficult to access by rescuers in a timely manner, and burials frequently involve multiple victims. The decision to initiate full resuscitative measures should be determined by the number of victims, resources available, and likelihood of survival. Studies of avalanche victims demonstrate a progressive nonlinear reduction in survival as the time of avalanche burial lengthens.[439–442] The likelihood of survival is minimal when avalanche victims are buried >35 minutes with an obstructed airway and in cardiac arrest on extrication[440,441,443–449] or are buried for any length of time and in cardiac arrest on extrication with an obstructed airway and an initial core temperature of <32°C.[441–443,447,450]

It may be difficult to know with any certainty how long an avalanche victim has been buried. The core temperature at time of extrication provides a proxy for duration of burial. A case series[450] of buried avalanche victims showed a maximum cooling rate of 8°C per hour, whereas a case report[447] described a maximum cooling rate of 9°C per hour. These

cooling rates suggest that at 35 minutes of burial, the core temperature may drop as low as 32°C.

If information on the duration of burial or the state of the airway on extrication is not available to the receiving physician, a serum potassium level of <8 mmol/L on hospital admission is a prognostic marker for ROSC[444] and survival to hospital discharge.[443,450] High potassium values are associated with asphyxia,[443,450–452] and there is an inverse correlation between admission K+ and survival to discharge in all-cause hypothermic patients.[443,453–456] In a series of 32 avalanche survivors the highest serum K+ was 6.4 mmol/L,[450] but there is a single case report of a 31- month-old child with a K+ of 11.8 mmol/L presenting with hypothermia from exposure unrelated to an avalanche who survived.[457] This suggests that the upper survivable limit of potassium is unknown for children who are hypothermic and victims of avalanche.

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).

Part 12.11: Drowning

Each year drowning is responsible for more than 500 000 deaths worldwide.[458] Drowning is a leading preventable cause of unintentional morbidity and mortality.[459,460] 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).

A number of terms are used to describe drowning.[461] To aid in use of consistent terminology and uniform reporting of data, use of the Utstein definitions and style of data reporting specific to drowning is recommended.[462,463]

Although survival is uncommon in victims who have undergone prolonged submersion and require prolonged resuscitation,[464,465] successful resuscitation with full neurological recovery has occurred occasionally after prolonged submersion in icy water[466–469] and, in some instances, warm water.[470,471] For this reason, scene resuscitation should be initiated and the victim transported to the ED unless there is obvious death (eg, rigor mortis, decomposition, hemisection, decapitation, lividity).

BLS Modifications

The most important and detrimental consequence of submersion is hypoxia; therefore, oxygenation, ventilation, and perfusion should be restored as rapidly as possible. This will require immediate bystander CPR plus activation of the EMS system. With the *2010 AHA Guidelines for CPR and ECC*, CPR now begins with chest compressions in a C-A-B sequence. However, the guidelines recommend individualization in sequence based upon the presumed etiology of the arrest. CPR for drowning victims should use the traditional A-B-C approach in view of the hypoxic nature of the arrest. Victims with only respiratory arrest usually respond after a few artificial breaths are given.

Recovery From the Water

When attempting to rescue a drowning victim, the rescuer should get to the victim as quickly as possible. It is crucial, however, that the rescuer pays constant attention to his or her own personal safety during the rescue process.

The reported incidence of cervical spine injury in drowning victims is low (0.009%).[472,473] Unnecessary cervical spine immobilization can impede adequate opening of the airway and delay delivery of rescue breaths. Routine stabilization of the cervical spine in the absence of circumstances that suggest a spinal injury is not recommended (Class III, LOE B).[473,474]

Rescue Breathing

The first and most important treatment of the drowning victim is the immediate provision of ventilation. Prompt initiation of rescue breathing increases the victim's chance of survival.[475] Rescue breathing is usually performed once the unresponsive victim is in shallow water or out of the water. Mouth-to-nose ventilation may be used as an alternative to mouth-to-mouth ventilation if it is difficult for the rescuer to pinch the victim's nose, support the head, and open the airway in the water.

Management of the drowning victim's airway and breathing is similar to that recommended for any victim of cardiopulmonary arrest. Some victims aspirate no water because they develop laryngospasm or breath-holding.[465,476] Even if water is aspirated, there is no need to clear the airway of aspirated water, because only a modest amount of water is aspirated by the majority of drowning victims, and aspirated water is rapidly absorbed into the central circulation.[465,477] Attempts to remove water from the breathing passages by any means other than suction (eg, abdominal thrusts or the Heimlich maneuver) are unnecessary and potentially dangerous.[477] The routine use of abdominal thrusts or the Heimlich maneuver for drowning victims is not recommended (Class III, LOE C).

Chest Compressions

As soon as the unresponsive victim is removed from the water, the rescuer should open the airway, check for breathing, and if there is no breathing, give 2 rescue breaths that make the chest rise (if this was not done previously in the water). After delivery of 2 effective breaths, the lay rescuer should immediately begin chest compressions and provide cycles of compressions and ventilations according to the BLS guidelines. Once the victim is out of the water, if he or she is unresponsive and not breathing after delivery of 2 rescue breaths, rescuers should attach an AED and attempt defibrillation if a shockable rhythm is identified. It is only necessary to dry the chest area before applying the defibrillation pads and using the AED. If hypothermia is present, follow the recommendations in Part 12.9: "Cardiac Arrest in Accidental Hypothermia."

Vomiting by the Victim During Resuscitation

The victim may vomit when the rescuer performs chest compressions or rescue breathing. In fact, in a 10-year study in Australia, two thirds of victims who received rescue breathing and 86% of those who required compressions and ventilations vomited.[478] If vomiting occurs, turn the victim to the side and remove the vomitus using your finger, a cloth, or suction. If spinal cord injury is suspected, the victim should be logrolled so

that the head, neck, and torso are turned as a unit to protect the cervical spine.

ACLS Modifications

Victims in cardiac arrest may present with asystole, PEA, or pulseless VT/VF. For treatment of these rhythms, follow the appropriate PALS or ACLS guidelines. Case reports of pediatric patients document the use of surfactant for fresh water–induced respiratory distress, but further research is needed.[479–482] The use of extracorporeal membrane oxygenation in patients with severe hypothermia after submersion has been documented in case reports.[468,469,483]

Part 12.12: Cardiac Arrest Associated With Electric Shock and Lightning Strikes

Injuries from electric shock and lightning strike result from the direct effects of current on the heart and brain, cell membranes, and vascular smooth muscle. Additional injuries result from the conversion of electric energy into heat energy as current passes through body tissues.[484]

Electric Shock

Fatal electrocutions may occur with household current; however, high-tension current generally causes the most serious injuries.[485] Contact with alternating current (the type of current commonly present in most North American households and commercial settings) may cause tetanic skeletal muscle contractions, "locking" the victim to the source of the electricity and thereby leading to prolonged exposure. The frequency of alternating current increases the likelihood of current flow through the heart during the relative refractory period, which is the "vulnerable period" of the cardiac cycle. This exposure can precipitate VF, which is analogous to the R-on-T phenomenon that occurs in nonsynchronized cardioversion.[486]

Lightning Strike

The National Weather Service estimates that an average of 70 deaths and 630 injuries occur due to lightning strikes in the United States each year.[487] Lightning strike injuries can vary widely, even among groups of people struck at the same time. Symptoms are mild in some victims, whereas fatal injuries occur in others.[488,489]

The primary cause of death in victims of lightning strike is cardiac arrest, which may be associated with primary VF or asystole.[488–491] Lightning acts as an instantaneous, massive direct-current shock, simultaneously depolarizing the entire myocardium.[489,492] In many cases intrinsic cardiac automaticity may spontaneously restore organized cardiac activity and a perfusing rhythm. However, concomitant respiratory arrest due to thoracic muscle spasm and suppression of the respiratory center may continue after ROSC. Unless ventilation is supported, a secondary hypoxic (asphyxial) cardiac arrest will develop.[493]

Lightning also can have myriad effects on the cardiovascular system, producing extensive catecholamine release or autonomic stimulation. The victim may develop hypertension, tachycardia, nonspecific ECG changes (including prolongation of the QT interval and transient T-wave inversion), and myocardial necrosis with release of creatinine kinase-MB fraction.

Lightning can produce a wide spectrum of peripheral and central neurological injuries. The current can produce brain hemorrhages, edema, and small-vessel and neuronal injury. Hypoxic encephalopathy can result from cardiac arrest.

Victims are most likely to die of lightning injury if they experience immediate respiratory or cardiac arrest and no treatment is provided. Patients who do not suffer respiratory or cardiac arrest, and those who respond to immediate treatment, have an excellent chance of recovery. Therefore, when multiple victims are struck simultaneously by lightning, rescuers should give the highest priority to patients in respiratory or cardiac arrest.

For victims in cardiac arrest, treatment should be early, aggressive, and persistent. Victims with respiratory arrest may require only ventilation and oxygenation to avoid secondary hypoxic cardiac arrest. Resuscitation attempts may have high success rates and efforts may be effective even when the interval before the resuscitation attempt is prolonged.[493]

BLS Modifications

The rescuer must first be certain that rescue efforts will not put him or her in danger of electric shock. When the scene is safe (ie, the danger of shock has been removed), determine the victim's cardiorespiratory status. If spontaneous respiration or circulation is absent, immediately initiate standard BLS resuscitation care, including the use of an AED to identify and treat VT or VF.

Maintain spinal stabilization during extrication and treatment if there is a likelihood of head or neck trauma.[494,495] Both lightning and electric shock often cause multiple trauma, including injury to the spine,[495] muscular strains, internal injuries from being thrown, and fractures caused by the tetanic response of skeletal muscles.[496] Remove smoldering clothing, shoes, and belts to prevent further thermal damage.

ACLS Modifications

No modification of standard ACLS care is required for victims of electric injury or lightning strike, with the exception of paying attention to possible cervical spine injury. Establishing an airway may be difficult for patients with electric burns of the face, mouth, or anterior neck. Extensive soft-tissue swelling may develop rapidly, complicating airway control measures. Thus, early intubation should be performed for patients with evidence of extensive burns even if the patient has begun to breathe spontaneously.

For victims with significant tissue destruction and in whom a pulse is regained, rapid IV fluid administration is indicated to counteract distributive/hypovolemic shock and to correct ongoing fluid losses due to third spacing. Fluid administration should be adequate to maintain diuresis and facilitate excretion of myoglobin, potassium, and other byproducts of tissue destruction (this is particularly true for patients with electric injury).[492] Regardless of the extent of external injuries after electrothermal shock, the underlying tissue damage can be far more extensive.

Part 12.13: Cardiac Arrest During Percutaneous Coronary Intervention

During both elective and emergent percutaneous coronary intervention (PCI), there is risk of cardiac arrest. Although high-quality chest compressions improve the chance of successful resuscitation and survival, it is difficult to perform effective, high-quality chest compressions during PCI. Therefore, resuscitation adjuncts have been explored for the treatment of cardiac arrest during PCI. There are no randomized controlled trials evaluating alternative treatment strategies as opposed to standard care for cardiac arrest during PCI.

Mechanical CPR During PCI

Mechanical chest compression devices have been used successfully in an animal model[497] and adult humans[497–501] to provide maintenance of circulation in cardiac arrest while continuing a percutaneous coronary procedure. It is reasonable to use mechanical CPR during PCI (Class IIa, LOE C).

Emergency Cardiopulmonary Bypass

One case series[502] describes the use of emergency cardiopulmonary bypass to stabilize and facilitate emergency coronary angioplasty in patients with cardiac arrest unresponsive to ACLS during PCI. It is reasonable to use emergency cardiopulmonary bypass during PCI (Class IIb, LOE C).

Cough CPR

Multiple case reports[503–507] describe the use of cough CPR to temporarily maintain adequate blood pressure and level of consciousness in patients who develop ventricular arrhythmias during PCI while definitive therapy for malignant arrhythmias is instituted. It is reasonable to use cough CPR during PCI (Class IIa, LOE C).

Intracoronary Verapamil

One large case series[508] describes the successful use of intracoronary verapamil to terminate reperfusion-induced VT following mechanical revascularization therapy. Verapamil was not successful in terminating VF.

Part 12.14: Cardiac Arrest Caused by Cardiac Tamponade

Cardiac tamponade can be a life-threatening event. Increasing fluid and pressure in the pericardium reduces atrial and ventricular filling. As filling is reduced, stroke volume and cardiac output fall, with associated hypotension leading to cardiac arrest. Rapid diagnosis and drainage of the pericardial fluid are required to avoid cardiovascular collapse.

Pericardiocentesis guided by echocardiography is a safe and effective method of relieving tamponade in a nonarrest setting, especially when used in conjunction with a pericardial drain, and may obviate the need for subsequent operating room treatment.[509–513] In the arrest setting, in the absence of echocardiography, emergency pericardiocentesis without imaging guidance can be beneficial (Class IIa, LOE C).

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,[514–516] especially if gross blood causes clotting that blocks a pericardiocentesis needle (Class IIb, LOE C).[517]

Part 12.15: Cardiac Arrest Following Cardiac Surgery

The incidence of cardiac arrest following cardiac surgery is in the range of 1–3%. Causes include conditions that may be readily reversed such as ventricular fibrillation, hypovolemia, cardiac tamponade, or tension pneumothorax. Pacing wires, if present, may reverse symptomatic bradycardia or asystole. A recent review may be helpful for those seeking additional information.[518]

Resternotomy

Studies of patients with cardiac arrest after cardiac surgery who are treated with resternotomy and internal cardiac compression have reported improved outcome compared with a standard protocol[519–529] when patients are treated by experienced personnel in intensive care units. Findings of similar quality studies[530–534] reported no difference in outcomes when resternotomy was compared with standard management of cardiac arrest after cardiac surgery. Resternotomy performed outside an intensive care unit generally has a very poor outcome.[519,526,533]

For patients with cardiac arrest following cardiac surgery, it is reasonable to perform resternotomy in an appropriately staffed and equipped intensive care unit (Class IIa, LOE B). Despite rare case reports describing damage to the heart possibly due to external chest compressions,[535,536] chest compressions should not be withheld if emergency resternotomy is not immediately available (Class IIa, LOE C).

Mechanical Circulatory Support

Nine case series have reported survival of some post–cardiac surgery patients during cardiac arrest refractory to standard resuscitation measures following the use of extracorporeal membrane oxygenation[537–541] and cardiopulmonary bypass.[529,542–544] In post–cardiac surgery patients who are refractory to standard resuscitation procedures, mechanical circulatory support (eg, extracorporeal membrane oxygenation and cardiopulmonary bypass) may be effective in improving outcome (Class IIb, LOE B).

Pharmacological Intervention

Rebound hypertension following administration of pressors during resuscitation has the potential to induce significant bleeding in this group of patients. Results from a single study of epinephrine[545] and another study evaluating the choice of antiarrhythmics[546] in patients with cardiac arrest following cardiac surgery were neutral. There is insufficient evidence on epinephrine dose, antiarrhythmic use, and other routine pharmacological interventions to recommend deviating from standard resuscitation guidelines when cardiac arrest occurs after cardiac surgery.

Disclosures

Guidelines Part 12: Cardiac Arrest in Special Situations: Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Terry L. Vanden Hoek	The University of Chicago-Associate Professor	*Vanden Hoek, Principal Investigator, Department of Defense, Office of Naval Research, "Proteomic Development of Molecular Vital Signs: Mapping a Mitochondrial Injury Severity Score to Triage and Guide Resuscitation of Hemorrhagic Shock." Research grant awarded to the University of Chicago	None	None	None	None	None
Laurie J. Morrison	St. Michaels Clinician scientist	None	None	None	None	None	None
Michael Shuster	Self-employed–emergency physician	None	None	None	None	None	None
Michael Donnino	Harvard Medical Faculty Physicians–Physician	†Corticosteroids in Post-arrest Shock (American Heart Association, Scientist Development Grant); Thiamine as a Metabolic Resuscitator in Septic Shock (NIH pending); *Statins in Sepsis (Eleanor Shore) Clinical Correlates to Influenza Genome (NIH); Thiamine Deficiency in Critically Ill (Harvard Medical School/NIH); Thiamine for Congestive Heart Failure (Baystate Incubator Fund-NON-industry, academic hospital funding)	None	None	None	None	None
Elizabeth Sinz	Penn State Hershey Medical Center–Professor of Anesthesiology and Neurosurgery; AHA–Associate Science Editor	None	None	None	None	None	None
Eric J. Lavonas	Rocky Mountain Poison & Drug Center; (RMPDC) Denver, Colo. Associate Director	†RMPDC performed research related to hydroxocobalamin prior to its licensure in the United States. This occurred prior to my arrival at RMPDC. RMPDC-DH performed work related to the development of hydroxocobalamin (CyanoKit, Dey LP) as a cyanide antidote. Various projects were completed in 2001, 2005, and 2006. Some of the sponsors of this research (EMD; Merck KGA) either no longer exist or no longer have an interest in hydroxocobalamin. I was not involved in this research, which was performed long before my arrival. RMPDC-DH does not have any current or pending hydroxocobalamin-related projects. Neither I nor any other DHHA employee derives personal financial benefit from these relationships. I don't get a bonus of any sort. My salary is supported by general institutional funds and an unrelated research endowment. Also, my performance evaluation is not related the performance of any of these contracts. My role: PI on one portion of the project, collaborator on the rest 2008–2009 (ongoing)	None	None	None	None	None
Farida M. Jeejeebhoy	Self employed cardiologist, affiliate with University Health Network/Mt Sinai and University of Toronto	None	None	None	None	None	None
Andrea Gabrielli	University of Florida–Professor of Anesthesiology and Surgery	†NIH-Biomarkers in Traumatic Brain Injury	None	None	None	None	None

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

*Modest.

†Significant.

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