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ECC Guidelines

Part 8: Advanced Challenges in Resuscitation

Section 3: Special Challenges in ECC

▶ Hypothermia

Definition/Background

Severe hypothermia (body temperature below 30°C [86°F]) is associated with marked depression of cerebral blood flow and oxygen requirement, reduced cardiac output, and decreased arterial pressure.^{1A} Victims can appear to be clinically dead because of marked depression of brain function.^{1A 2A}

Hypothermia may exert a protective effect on the brain and organs in cardiac arrest.^{3A 4A} If the victim cools rapidly without hypoxemia, decreased oxygen consumption and metabolism may precede the arrest and reduce organ ischemia.^{5A} Although rare, full resuscitation with intact neurological recovery may be possible after hypothermic cardiac arrest.^{1A 6A}

The victim's pulses and respiratory efforts may be difficult to detect, but do not withhold lifesaving procedures on the basis of clinical presentation.^{1A} Transport victims as soon as possible to a center where monitored rewarming is possible.

Severe unintentional hypothermia is a serious and preventable health problem. Hypothermia in inner-city areas has a high association with mental illness, poverty, and use of drugs and alcohol.^{7A 8A} In some rural areas >90% of hypothermic deaths are associated with elevated blood alcohol levels.^{9A} Successful treatment of hypothermia requires optimal training of emergency personnel and appropriate resuscitation methods at each institution.

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The Figure^{5A} presents a recommended hypothermia treatment algorithm with recommended actions that should be taken for all possible victims of hypothermia.



Figure 1. Figure. Hypothermia treatment algorithm.

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Prevention of Arrest in Victims of Hypothermia: General Care for All Victims of Hypothermia

When the victim is extremely cold but has maintained a perfusing rhythm, interventions focus on prevention of further heat loss, careful transport, and rewarming:

- Prevent additional evaporative heat loss by removing wet garments, insulating the victim, and shielding the victim from wind.
- Carefully transport the victim to the hospital, taking care to avoid rough movement and activity, because these can precipitate VF.
- Monitor core temperature and cardiac rhythm. If the victim's skin is extremely cold, it may not be possible to obtain an ECG or monitor cardiac rhythm by use of adhesive electrodes. If necessary, needle electrodes may be used.
- Do not delay urgently needed procedures such as intubation or insertion of vascular catheters, but perform them gently and monitor cardiac rhythm closely.

Rewarm patients with a core temperature of $<34^{\circ}\text{C}$ ($<93^{\circ}\text{F}$). Passive rewarming can be achieved with blankets and a warm room. This form of rewarming will not be effective for a patient in cardiopulmonary arrest or with severe hypothermia.^{5A}

Active external rewarming uses heating devices (radiant heat, forced hot air, or warm bath water) or heated devices (warm packs). These devices require careful monitoring of patient and device and should be used with caution, if at all. Some researchers think that active external rewarming contributes to "afterdrop" (continued drop in core temperature when cold blood from the periphery is mobilized). In addition, topical application of warm devices may result in tissue

injury. If they are used, heated packs should be applied and only to truncal areas (neck, armpits, or groin).

Active internal rewarming techniques typically are reserved for patients with a core body temperature $<30^{\circ}\text{C}$ (86°F). They may include the administration of warmed (42°C to 46°C [108°F to 115°F]) humidified oxygen, use of extracorporeal membrane oxygenators, peritoneal lavage, intravenous administration of warmed (42°C to 44°C [108°F to 111°F]) saline, and esophageal rewarming tubes. No randomized, controlled clinical trials have been reported comparing the efficacy of these methods.^{5A}

Modifications of BLS for Hypothermia

When the victim is hypothermic, pulse and respiratory rates will be slow, breathing will be shallow, and peripheral vasoconstriction will make pulses difficult to feel. For these reasons the BLS rescuer should assess breathing and, later, pulse for a period of 30 to 45 seconds to confirm respiratory arrest, pulseless cardiac arrest, or bradycardia profound enough to require CPR.^{2A} If the victim is not breathing, initiate rescue breathing immediately. If possible, administer warmed (42°C to 46°C [108°F to 115°F]) humidified oxygen during bag-mask ventilation. If the victim is pulseless with no detectable signs of circulation, start chest compressions immediately. Do not withhold BLS until the victim is rewarmed.

To prevent further core heat loss from the victim, remove wet garments from the victim; insulate or shield the victim from wind, heat, or cold; and if possible ventilate with warm, humidified oxygen.^{1A 2A 10A} Avoid rough movement and do not apply external rewarming devices in the field. As soon as possible, carefully prepare the patient for transport to a hospital. All other field interventions require ACLS capability.

Treatment of severe hypothermia (temperature $<30^{\circ}\text{C}$ [86°F]) in the field remains controversial. Many providers do not have the equipment or time to assess core body temperature adequately or to institute rewarming with warm, humidified oxygen or warm fluids, although these methods should be initiated when available to help prevent temperature afterdrop.^{1A 10A 11A 12A} We recommend core temperature determinations in the field with either tympanic membrane sensors or rectal probes (for EMS systems so equipped), but these should not delay transfer. To prevent VF, avoid rough movement and excess activity. Transport the patient in the horizontal position to avoid aggravating hypotension.

If the hypothermic victim is in cardiac arrest, the general approach to BLS management still targets airway, breathing, and circulation, but some modifications in approach are required. If VT or VF is present, attempt defibrillation. Automated external defibrillators (AEDs) should be available on virtually all BLS rescue units, and if VF is detected, emergency personnel should be allowed to deliver up to 3 shocks to determine fibrillation responsiveness.^{2A} If VF persists after 3 shocks, further defibrillation attempts should be deferred. Emergency personnel should immediately begin CPR and rewarming (administer warmed, humidified oxygen and warmed intravenous saline) and attempt to stabilize the victim for transportation. If core temperature is

<30°C (86°F), successful conversion to normal sinus rhythm may not be possible until rewarming is accomplished.^{13A}

Some clinicians believe that patients who appear dead after prolonged exposure to cold temperatures should not be considered dead until they are near normal core temperature and are still unresponsive to CPR.^{2A 13A} Hypothermia may exert a protective effect on the brain and organs if the hypothermia develops rapidly in victims of cardiac arrest. When a victim of hypothermia is discovered, however, it may be impossible to separate primary from secondary hypothermia. If the victim is found in arrest in an extremely cold environment and the event was unwitnessed, emergency personnel and hospital providers will not know whether the arrest was due to hypothermia or whether hypothermia was a sequel to a normothermic arrest (eg, a man experiencing cardiac arrest while shoveling snow will develop hypothermia only after the arrest). In addition, the patient may have sustained additional organ insult. For example, successful resuscitation may be more difficult if drowning preceded hypothermia. When it is clinically impossible to know whether the arrest or the hypothermia occurred first, rescuers should attempt to stabilize the patient with CPR. If hypothermia is documented, initiate basic maneuvers to limit heat loss and begin rewarming. Physicians in the hospital should use their clinical judgment to decide when resuscitative efforts should cease in a hypothermic arrest victim.

Modifications of ACLS for Hypothermia

If the hypothermic victim has not yet developed cardiac arrest, focus attention on assessment and support of oxygenation and ventilation, assessment and support of circulation, warming, and prevention of further heat loss. Handle the victim gently for all procedures; many physical manipulations (including endotracheal or nasogastric intubation, temporary pacing, or insertion of a pulmonary artery catheter) have been reported to precipitate VF.^{1A 12A} When specifically and urgently indicated, however, do not withhold such procedures. In a prospective multicenter study of hypothermia victims, careful endotracheal intubation did not result in a single incident of VF.^{14A} In fact, the fear of precipitating VF during endotracheal intubation may be exaggerated,^{5A} and it should not prevent or delay performance of careful intubation.

Endotracheal intubation is required if the hypothermic victim is unconscious or if ventilation is inadequate. The intubation will serve 2 purposes: it will enable provision of effective ventilation with warm, humidified oxygen, and it can isolate the airway to reduce the likelihood of aspiration. We recommend ventilation with 100% oxygen via bag-mask before any intubation attempt.

Conscious victims who are cold with only mild symptoms of hypothermia may be rewarmed with external active and passive rewarming techniques (eg, warm packs, warmed sleeping bags, and warm baths).

ACLS management of cardiac arrest due to hypothermia is quite different from management of normothermic arrest. Active core rewarming techniques are the primary therapeutic modality in hypothermic victims in cardiac arrest or unconscious with a slow heart rate. The hypothermic

heart may be unresponsive to cardioactive drugs, pacemaker stimulation, and defibrillation,^{12A} and drug metabolism is reduced. Although administration of epinephrine and vasopressin has been shown to improve coronary artery perfusion pressure in animals,^{15A} there is concern that administered medications, including epinephrine, lidocaine, and procainamide, can accumulate to toxic levels in the peripheral circulation if they are administered repeatedly in the severely hypothermic victim. For these reasons intravenous drugs are often withheld if the victim's core body temperature is $<30^{\circ}\text{C}$ (86°F). If the victim's core body temperature is $>30^{\circ}\text{C}$ (86°F), intravenous medications may be administered but with increased intervals between doses.

The temperature at which defibrillation should first be attempted and how often it should be tried in the severely hypothermic patient have not been firmly established. In general, an attempt at defibrillation is appropriate if VT/VF is present. If the patient fails to respond to 3 initial defibrillation attempts or initial drug therapy, subsequent defibrillation attempts or additional boluses of medication should be deferred until the core temperature rises above 30°C (86°F).^{16A} Bradycardia may be physiological in severe hypothermia, and cardiac pacing is usually not indicated unless bradycardia persists after rewarming.

Treatment of severely hypothermic victims (core temperature $<30^{\circ}\text{C}$ [86°F]) in cardiac arrest in the hospital should be directed at rapid core rewarming. Techniques that can be used for in-hospital controlled rewarming include

- Administration of warmed, humidified oxygen (42°C to 46°C [108°F to 115°F])
- Administration of warmed intravenous fluids (normal saline) at 43°C (109°F) infused centrally at rates of approximately 150 to 200 mL/h (to avoid overhydration)
- Peritoneal lavage with warmed (43°C [109°F]) potassium-free fluid administered 2 L at a time

Note the following:

- Extracorporeal blood warming with partial bypass is the preferred method of active internal rewarming because it ensures adequate support of oxygenation and ventilation while the core body temperature is gradually rewarmed.^{1A 10A 12A 17A 18A}
- The use of esophageal rewarming tubes in the United States has not been reported, although they have been used extensively and successfully in Europe.^{19A}
- Pleural lavage with warm saline instilled through a chest tube has also been used successfully.^{14A}

During rewarming, patients who have been hypothermic for >45 to 60 minutes are likely to require volume administration because their vascular space expands with vasodilation. Careful monitoring of heart rate and hemodynamic monitoring are important at this time. The routine administration of steroids, barbiturates, or antibiotics has not been documented to help increase survival or decrease postresuscitative damage.^{20A 21A}

During rewarming, significant hyperkalemia may develop. Extreme hyperkalemia has been reported in avalanche victims who sustained crushing injuries and hypothermia.^{5A} Severe hyperkalemia has also been reported among hypothermic patients in North America who did not sustain crushing injuries.^{22A} In fact, severity of hyperkalemia has been linked with mortality. Management of hyperkalemia should include the traditional ACLS approach, with administration of calcium chloride, sodium bicarbonate, glucose plus insulin, and Kayexalate enema. More aggressive measures to reduce extremely high serum potassium may include dialysis or exchange transfusion.

If drowning preceded the victim's hypothermia, successful resuscitation will be rare. 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 the hypothermia. If the victim appears malnourished or has a history of chronic alcoholism, administer thiamine (100 mg IV) early during the rewarming procedures.

Withholding and Cessation of Resuscitative Efforts

In the field, resuscitation may be withheld if the victim has obvious lethal injuries or if the body is frozen so completely that chest compression is impossible and the nose and mouth are blocked with ice.^{22A}

Physicians in the hospital should use their clinical judgment to decide when resuscitative efforts should cease in a hypothermic arrest victim. Complete rewarming is not indicated for all victims. Predictors of outcome may be unreliable in the face of injury or other complicating factors. A high (nonhemolyzed) serum potassium has been associated with a poor outcome, but these results will be unreliable in the presence of crushing injuries, hemolysis, or succinylcholine administration.^{5A}

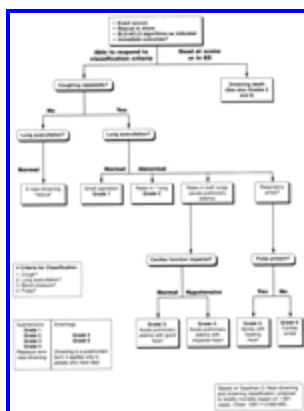


Figure 2. Figure. Uniform reporting of submersion episodes.

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View this table: **Table 1.** Clinical Factors Associated With Submersion Mortality
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Figure 3. Figure. Left lateral position for pregnant woman.

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View this table: **Table 1A.** Primary and Secondary ABCD Surveys: Modifications for
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► Footnotes

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▶ Submersion or Near-Drowning

Submersion: Overview

The most important and detrimental consequence of submersion without ventilation is hypoxia. The duration of hypoxia is the critical factor in determining the victim's outcome. Therefore, oxygenation, ventilation, and perfusion should be restored as rapidly as possible. Immediate resuscitation at the scene is essential for survival and neurological recovery after submersion. This will require bystander provision of CPR plus immediate activation of the EMS system. Victims who have spontaneous circulation and breathing when they reach the hospital usually recover with good outcomes.

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Hypoxia can produce multisystem insult and complications, including hypoxic encephalopathy and acute respiratory distress syndrome (ARDS). These complications are relevant to the care of the victim after resuscitation and will not be addressed here.

Victims of submersion may develop primary or secondary hypothermia. If the submersion occurs in icy water (<5°C [41°F]), hypothermia may develop rapidly and provide some protection against hypoxia. Such effects, however, have typically been reported only after submersion of *small* victims in icy water.^{1B} Hypothermia may also develop as a secondary complication of the submersion and subsequent heat loss through evaporation during attempted resuscitation. In these victims the hypothermia is not protective (see Hypothermia earlier in this section).

All victims of submersion who require resuscitation should be transported to the hospital for evaluation and monitoring. The hypoxic insult can produce an increase in pulmonary capillary permeability with resultant pulmonary edema.

Definitions, Classifications, and Prognostic Indicators

A number of terms are used to describe submersion. Clinicians and others who report about submersion often apply the misunderstood term *drowning* to victims *who die within 24 hours of a submersion episode*. They apply the term *near-drowning* to submersion victims who survive >24 hours after the episode if the victim also requires active intervention for one or more submersion complications. Complications can include pneumonia, ARDS, or neurological sequelae. Rescuers and emergency personnel find these definitions irrelevant, because the drowning versus near-

drowning distinction often cannot be made for 24 hours.

Pending the future recommendations of an ILCOR Task Force revising the Utstein Guidelines, the Guidelines 2000 Conference recommends these terms:

Water rescue: a person who is alert but experiences some distress while swimming. The victim may receive some help from others and displays minimal, transient symptoms, such as coughing, that clear quickly. In general the person is left on shore and is not transported for further evaluation and care.

Submersion: a person who experiences some swimming-related distress that is sufficient to require support in the field plus transportation to an emergency facility for further observation and treatment.

Drowning: this is a "mortal" event; this refers to submersion events in which the victim is pronounced dead at the scene of the attempted resuscitation, in the Emergency Department (ED), or in the hospital. With drowning, the victim suffers cardiopulmonary arrest and cannot be resuscitated. Death can be pronounced at the scene, in the ED, or within 24 hours of the event. If death occurs after 24 hours, the term *drowning* is still used as in "drowning-related death." Up until the time of drowning-related death, refer to the victim as a *submersion victim*.

We recommend that the term *near-drowning* no longer be used.

We recommend that clinicians, managers, and research teams stop the classification of submersion victims by submersion fluid (salt water versus fresh water). Although there are theoretical differences between the effects of salt-water and fresh-water submersion in the laboratory, these differences are not clinically significant. The single most important factor that determines outcome of submersion is the duration of the submersion and the duration and severity of the hypoxia.

Although survival is uncommon in victims who have undergone prolonged submersion and require prolonged resuscitation,^{2B 3B} successful resuscitation with full neurological recovery has occasionally occurred in near-drowning victims with prolonged submersion in extremely cold water.^{4B 5B 6B} Therefore, resuscitation should be initiated by rescuers at the scene unless there is obvious physical evidence of death, such as putrefaction, dependent lividity, or rigor mortis. The victim should be transported with continued CPR to an emergency facility. In many European countries a physician will be available on scene as part of the EMS team.

Prognostic indicators after submersion in children and adolescents (up to 20 years of age) include 3 factors associated with 100% mortality in one study^{1B} :

- Submersion duration >25 minutes
- Resuscitation duration >25 minutes

- Pulseless cardiac arrest on arrival in the ED

Additional factors associated with poor prognosis in the same study^{1B} included

- Presence of VT/VF on initial ECG (93% mortality)
- Fixed pupils noted in the ED (89% mortality)
- Severe acidosis (89% mortality) in the ED
- Respiratory arrest (87% mortality) in the ED
- In a more recent study of adults and children from the same investigators, level of consciousness and responsiveness correlated with survival. Deaths occurred only among victims who remained comatose at the scene and comatose on arrival at the hospital. No deaths occurred among victims who were alert or lethargic but responsive either at the scene or in the hospital.^{7B}
- A number of classification systems have been proposed to link clinical findings with outcome of submersion victims.^{8B 9B} In a recent analysis of 1831 submersion episodes from the beaches of Brazil, mortality was related to severity of cardiopulmonary involvement as assessed by an on-scene physician with the drowning response team needing only 4 variables: coughing (yes or no), auscultation, blood pressure, and heart rate. Unlike other researchers in this area, Szpilman^{9B} did not start with an implicitly derived classification scheme into which he forces each case. Instead, the classification grades were derived retrospectively by asking what simple list of criteria had the best association with severe pulmonary compromise. As displayed in the Table⁹, increasing mortality correlated with ascending grades of clinical severity.^{9B}
- Auscultation of breath sounds will not be applicable to the BLS provider. For the ACLS provider, however, auscultatory findings provide a helpful classification of the severity of cardiopulmonary failure after submersion. The algorithm we have developed (Figure⁹) is largely a translation of Szpilman's results and an algorithm he derived in a manner that can be used by epidemiologists to support a prospective database of submersion victims.
- Any prognostic approach should consider the temperature of the submersion fluid (icy versus nonicy) and the size and age of the victim. Aggressive attempts at resuscitation in the hospital may be continued for the small victim of icy-water submersion and hypothermia.

Modifications to Guidelines for BLS for Resuscitation From Submersion

No modification of standard BLS sequencing is necessary. There are, however, cautions and emphasis that should be considered when beginning CPR for the submersion victim.

Recovery From the Water

When attempting to rescue a near-drowning victim, the rescuer should get to the victim as quickly as possible, preferably by some conveyance (boat, raft, surfboard, or flotation device). The rescuer must always be aware of personal safety and should minimize the danger to the rescuer and the victim. Treat all victims as potential victims of spinal cord injury, and immobilize the cervical and thoracic spine. Spinal injury is particularly likely after submersion associated

with diving or involving recreational equipment, but it should be suspected if the submersion episode was not witnessed.

If first-responding rescuers suspect a spinal cord injury, they should use their hands to stabilize the victim's neck in a neutral position (without flexion or extension). They should float the victim, supine, onto a horizontal back support device before removing the victim from the water. The rescue from the water should be done quickly to ensure timely application of CPR if required. If the victim must be turned, align and support the head, neck, chest, and body. Carefully log-roll the victim to a horizontal and supine position. Provide rescue breathing while maintaining the head in a neutral position, using the jaw thrust without head tilt or chin lift to open the airway.

Rescue breathing should begin as quickly as possible (see below). Provision of chest compression typically will have to wait until the victim has been removed from the water. External chest compressions cannot be performed in the water unless the victim is extremely small and can be supported on the rescuer's forearm or unless flotation devices are used. Proper use of in-water resuscitation flotation devices requires training.

Rescue Breathing

The first and most important treatment of the near-drowning victim is provision of immediate mouth-to-mouth ventilation. Prompt initiation of rescue breathing has a positive association with survival.^{10B}

Start rescue breathing as soon as the victim's airway can be opened and the rescuer's safety ensured. This is usually achieved when the victim is in shallow water or out of the water. If it is difficult for the rescuer to pinch the victim's nose and support the head and open the airway in the water, mouth-to-nose ventilation may be used as an alternative to mouth-to-mouth ventilation.

Appliances (such as a snorkel for the mouth-to-snorkel technique or buoyancy aids) may permit specially trained rescuers to perform rescue breathing in deep water. But rescue breathing should not be delayed for lack of such equipment if it can otherwise be provided safely. Untrained rescuers should not attempt to use such adjuncts.

Management of the airway and breathing of the submersion victim is similar to that of any victim with potential trauma in cardiopulmonary arrest. The airway can be managed with adjuncts in the near-drowning victim.^{2B 11B}

There is no need to clear the airway of aspirated water.^{12B} Some victims aspirate nothing because of laryngospasm or breath-holding.^{3B 8B 13B} At most only a modest amount of water is aspirated by the majority of drowning victims, and it is rapidly absorbed into the central circulation.^{3B} An attempt to remove water from the breathing passages by any means other than suction is unnecessary and dangerous. Abdominal thrusts, for example, cause regurgitation of gastric contents and subsequent aspiration and have been associated with other injuries.^{12B} Do not

routinely perform the Heimlich maneuver for resuscitation of submersion victims. It delays the initiation of ventilation and produces complications.^{12B} Use of the Heimlich maneuver as the first step in resuscitation of submersion victims is not evidence-based. Use the Heimlich maneuver *only* if the rescuer suspects foreign-body airway obstruction.^{11B 12B 14B 15B} If foreign-body airway obstruction is suspected, consider chest compressions rather than the Heimlich maneuver. There is recent evidence that chest compressions are superior to the Heimlich maneuver in generating increases in intrathoracic pressure to assist with the expulsion of foreign material.^{16B}

Chest Compressions

As soon as the victim is removed from the water, check for signs of circulation. The lay rescuer will look for general signs of circulation (breathing, coughing, or movement in response to the rescue breaths). The healthcare provider will look for signs of circulation, including the presence of a central pulse. The pulse may be difficult to appreciate in a near-drowning victim, particularly if the victim is cold. If signs of circulation (including a pulse, if appropriate) are not present, start chest compressions at once. Chest compressions should not be attempted in the water.

If there are no signs of circulation, an AED should be used to evaluate rhythm for victims older than 8 years of age. Attempt defibrillation if a shockable rhythm is identified. If hypothermia is present in a victim of VF and the victim's core body temperature is $\leq 30^{\circ}\text{C}$ (86°F), give a maximum of 3 defibrillation attempts (shocks). If a total of 3 defibrillation attempts are unsuccessful, return to BLS and ACLS care until the core body temperature rises above 30°C (86°F).

Vomiting During Resuscitation

Vomiting is likely to occur when chest compressions or rescue breathing is performed, and it will complicate efforts to maintain a patent airway. In fact, in a 10-year study in Australia, vomiting occurred in half of submersion victims who required no interventions after removal from the water. Vomiting occurred in two thirds of victims who received rescue breathing and 86% of victims who required compression and ventilation.^{17B} If vomiting occurs, turn the victim's mouth to the side and remove the vomitus with the finger sweep or use a cloth to wipe the mouth or use suction. If spinal cord injury is possible, log-roll the victim so that the head, neck, and torso are turned as a unit to remove the vomitus.

Modifications to Guidelines for ACLS for Arrest After Submersion

The submersion victim in cardiac arrest requires ACLS including intubation without delay. Every submersion victim, even one who requires only minimal resuscitation and regains consciousness at the scene, should be transferred to a medical facility for follow-up care. Monitoring of life support measures must be continued en route with oxygen administered in the transport vehicle.

Victims in cardiac arrest may present with asystole, pulseless electrical activity, or pulseless VT/VF. PALS and ACLS guidelines should be followed for the treatment of these rhythms. If severe hypothermia is present (core body temperature $\leq 30^{\circ}\text{C}$ [86°F]), defibrillation attempts are typically limited to 3, and intravenous medications are withheld until the core body temperature

rises above these levels. If moderate hypothermia is present, intravenous medications are spaced at longer than standard intervals (see Hypothermia earlier in this section). In children and adolescents, VT/VF on initial ECG is an extremely poor prognostic sign.^{1B}

Attempts have been made to improve neurological outcome in the intensive care unit with the use of barbiturates, intracranial pressure (ICP) monitoring, induced hypothermia, and steroid administration. None of these interventions has been shown to alter outcome. In fact, signs of ICP serve as a symptom of significant neurological hypoxic injury, and there is no evidence that attempts to alter the ICP will affect outcome.

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▶ Near-Fatal Asthma

Introduction

This section of the International Guidelines 2000 focuses on near-fatal asthma. The recommendations deviate immediately from routine asthma care and address the sequence of action steps needed to prevent death. Some recommendations, such as use of aminophylline, permissive hypercarbia, and early tracheal intubation, do not reflect routine asthma attacks. Nevertheless, in the desperate race to prevent cardiopulmonary arrest, heroic measures must be considered and considered early.

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Severe exacerbation of asthma can lead to several forms of sudden death. One classification scheme categorizes asthma on the basis of the onset of symptoms. Signs of rapid-onset asthma develop in <2.5 hours, signs and symptoms of slow-onset asthma develop over several days.^{1C} Cardiac arrest in patients with severe asthma has been linked to

- Severe bronchospasm and mucous plugging leading to asphyxia^{2C} (this condition causes the vast majority of asthma-related deaths).
- Cardiac arrhythmias due to hypoxia, which is the common cause of asthma-related arrhythmia. In addition, arrhythmias are caused by use of β -adrenergic agonists. (In rare instances these arrhythmias may be due to prolongation of the QT interval resulting from β -adrenergic agonists^{3C 4C 5C} or toxicity caused by medications such as theophylline.)
- Auto-PEEP (positive end-expiratory pressure) occurs in some patients who are intubated and mechanically ventilated. Patients fail to expire as much air as they took in; gradual buildup of pressure occurs and reduces blood flow and blood pressure. Auto-PEEP is secondary to air trapping and "breath stacking" (breathed air entering and being unable to escape).
- Tension pneumothorax (often bilateral)

Most asthma-related deaths occur outside the hospital. The number of patients with severe attacks of asthma who present to the Emergency Department at night is 10 times greater than the number presenting during the day.^{6C} Multiple factors affect the outcome of therapy in asthmatic patients. Constantly review these issues during evaluation and treatment:

- Determine whether the patient has true acute asthma. When a patient presents with dyspnea in extremis you may not be able to obtain the recent history.
- Depending on the patient's history and medication use, other conditions may be present.

These include —Cardiac disease (congestive heart failure, myocarditis), pulmonary disease (emphysema, pneumonia, upper-airway obstruction—structural or psychogenic), acute allergic bronchospasm or anaphylaxis (aspirin, foods, or idiopathic), pulmonary embolism, or vasculitis (Churg-Strauss syndrome).^{7C} —Medications and drugs of abuse: bronchospasm as sequelae to medications (β -blockers)^{8C} or drugs of abuse (cocaine and opiates).^{9C 10C} —Discontinuation of corticosteroids. Patients who have used corticosteroids for a long time may have relative adrenal insufficiency. With the stress of discontinuation of the steroids and the adrenal insufficiency, these patients may present with near-fatal asthma.

Key Interventions to Prevent Arrest

The major clinical action is to treat the severe asthmatic crisis aggressively, before deterioration to full arrest. The specific agents and the treatment sequence will vary according to local practice. Emergency treatment will include some combination of the agents and interventions discussed below. The challenge of most concern for the ALS provider is the patient who deteriorates progressively, unresponsive to multiple therapeutic efforts.

Oxygen

Use a concentration of inspired oxygen to achieve a PaO₂ of ≥ 92 mm Hg. High-flow oxygen by mask is sometimes necessary. In patients with an asthmatic crisis, the following signs indicate that the need for rapid tracheal intubation is imminent:

- Findings of obtundation
- Profuse diaphoresis
- Poor ("floppy") muscle tone (clinical signs of hypercarbia)
- Findings of severe agitation, confusion, and fighting against the oxygen mask (clinical signs of hypoxemia)^{11C}
- Elevation of the PCO₂ by itself is not indicative of a need for tracheal intubation. Elevated PCO₂ does indicate severity of the episode. Reserve intubation for patients with the clinical findings mentioned above or a clearly rising PCO₂. *Treat the patient, not the numbers.*

Nebulized β_2 -Agonists

Albuterol (salbutamol) is the cornerstone of therapy for acute asthma in most of the world. Standard practice in Emergency Departments is a dose of 2.5 to 5.0 mg every 15 to 20 minutes given up to 3 times in 1 hour (total dose of 7.5 to 15 mg/h). Patients who do not respond to albuterol may respond well to subcutaneous epinephrine or terbutaline.^{12C}

Intravenous Corticosteroids

By 2000 it became a common practice in accident and emergency departments to begin corticosteroid therapy early (in the first 30 minutes) for patients with life-threatening asthma. Corticosteroids should be started early, but oxygen and β -agonists always have priority as the initial agents. Clinicians typically use 125 mg of methylprednisolone (or equivalent

hydrocortisone 200 mg IV) as a starting dose in cases of severe asthma.^{13C 14C 15C} Doses can range as low as 40 mg to as high as 250 mg IV or its equivalent.

Nebulized Anticholinergics

Use ipratropium, an inhaled anticholinergic agent, as a moist nebulizing agent in combination with albuterol at a dose of 0.5 mg.^{16C} Unlike β_2 -agonists, which have an immediate onset of action, nebulized anticholinergic agents have a delayed onset of approximately 20 minutes.

Intravenous Aminophylline

Aminophylline, now used as secondary therapy after β_2 -agonists and corticosteroids, can enhance the effects of those agents. As a bronchodilator aminophylline is approximately one third as potent as β_2 -agonists. Clinicians use aminophylline much more frequently in children than in adults. A loading dose of 5 mg/kg is given over 30 to 45 minutes followed by an infusion of 0.5 to 0.7 mg/kg per hour, but this loading dose is not advised in people already taking theophylline, who should receive either half loading doses or maintenance doses. Addition of this agent to high doses of β_2 -agonists is thought to increase side effects more than it increases bronchodilation.

This is most evident in patients already taking theophyllines. The risk-benefit ratio may be different in patients not taking theophyllines.^{17C}

Intravenous Magnesium Sulfate

A number of authors have reported success with magnesium sulfate in patients refractory to inhaled adrenergic agents and corticosteroids. Although not consistently effective, magnesium is widely available and can be administered with few if any side effects at a dose of 2 to 3 g IV at rates as fast as 1 g/min (1 g magnesium sulfate=98 mg of elemental magnesium).^{18C 19C}

Parenteral or Subcutaneous or Intramuscular Epinephrine or Terbutaline

Subcutaneous administration of epinephrine or terbutaline may prevent the need for artificial ventilation in cases of life-threatening asthma, especially in patients who do not respond to inhaled β_2 -agonists. The total epinephrine dose (at a concentration of 1:1000) is 0.01 mg/kg, usually divided into 3 doses at 20-minute intervals. For convenience and easy recall a non-weight-based dose of 0.3 mg usually is given to adults. This dose of epinephrine (0.3 mg) can be repeated twice at 20-minute intervals to a total of 3 injections.

The dose of terbutaline is 0.25 mg SC every 30 minutes; up to 3 doses may be given. At this time there is no good evidence of advantages for IV β -agonists over inhaled bronchodilators.^{20C} The value of IV bronchodilators, however, compared with that of inhaled bronchodilators merits further study.

Ketamine

Ketamine is a parenteral dissociative anesthetic that has been found to be a useful bronchodilator. Most experts think that ketamine is the anesthetic agent of choice for intubation of severe asthmatics. Ketamine potentiates catecholamines and directly induces relaxation of smooth muscle. It also increases bronchial secretions and can cause emergent reactions. Because of the

effect of ketamine on bronchial secretions, atropine (0.01 mg/kg, minimum dose of 0.1 mg) also should be administered if this agent is used. Benzodiazepines help to minimize emergent reactions, although hallucinations may occur after the patient awakes. The initial dose of ketamine is 0.1 to 0.2 mg/kg followed by an infusion of 0.5 mg/kg per hour. In intubated patients or in those being prepared for intubation, the usual dose of ketamine is a bolus of 0.5 to 1.5 mg/kg, repeated 20 minutes later, or infusion of 1 to 5 mg/kg per hour.^{21C}

Heliox

Heliox is a mixture of helium and oxygen (usually 70:30) that may delay the need for intubation by decreasing the work of breathing while the other medications are beginning to take effect.^{22C}

Bilevel Positive Airway Pressure

Bilevel positive airway pressure intermittently provides assisted ventilation. Like a combination of positive-pressure ventilation and PEEP, bilevel airway pressure helps to delay or abort the need for tracheal intubation. This ventilation counteracts the effects of auto-PEEP, thereby reducing the work of breathing. Begin with an inspiratory positive airway pressure of 8 to 10 cm H₂O and an expiratory positive airway pressure of 3 to 5 cm H₂O.^{23C}

Tracheal Intubation With Artificial Ventilation

In some patients oxygenation and ventilation can be achieved only after sedation, general anesthesia, muscle paralysis, and tracheal intubation. Patients with severe asthma experience some obstruction of inspiration and marked obstruction of expiration. This results in auto-PEEP, which is secondary to air trapping and "breath stacking" (breathed air entering and being unable to escape).

The following critical points relate to tracheal intubation for life-threatening asthma:

- Provide adequate sedation with ketamine, a benzodiazepine, or a barbiturate.
- Paralyze the patient with succinylcholine or vecuronium.
- Once intubated some patients may need permissive hypercarbia with elective hypoventilation.^{24C}
- Inhaled volatile anesthetics, although no longer widely used or available outside the operating room, are powerful relaxants of bronchial smooth muscle. Agents such as halothane, isoflurane, enflurane, and ether have been used to reverse status asthmaticus refractory to all other treatments.^{25C} Use these agents with extreme caution because (except for ether) they are also vasodilators and myocardial depressants. Some anesthetics sensitize the myocardium to catecholamines, leading to life-threatening arrhythmias.
- Extracorporeal membrane oxygenation has been used as a lifesaving measure for severe refractory asthma when all else has failed, but this technique is not generally available.
- For most experts ketamine is the intravenous anesthetic of choice for patients with status asthmaticus. In titrated doses ketamine has a mild bronchodilator effect and does not cause vasodilatation, circulatory collapse, or myocardial depression.

Steps to Take Immediately After Intubation

Tracheal intubation only provides more external mechanical power to the patient's failing ventilation efforts; it does not solve the problem. Patients with severe asthma may be extremely difficult to preoxygenate manually before intubation and even once the tube is in place.

Because breathing efforts may be uncoordinated, the patient may not have inhaled an adequate amount of β_2 -agonist before intubation. Immediately after intubation inject 2.5 to 5.0 mg of albuterol directly into the tracheal tube. Confirm correct placement of the tracheal tube by the following newly recommended sequence:

- *Primary tracheal tube confirmation.* Visualize the tube past the vocal cords; perform 5-point auscultation; watch chest rise; condensation in tube. Auscultation can be misleading because poor ventilation and air movement may result in inaudible breath sounds in patients with severe refractory asthma.
- *Secondary confirmation of tube placement.* Qualitative end-tidal CO_2 detectors; esophageal detector device; dynamic pulse oximetry readings; quantitative and continuous CO_2 measurements using capnometers or capnographs.

Ventilate the patient with 100% oxygen.

The absence of any significant obstruction to airflow immediately after intubation suggests that the diagnosis of acute asthma may have been incorrect, and the problem may have been more in the upper airway (eg, vocal cord dysfunction, tumor, or a foreign body). The person who performs manual ventilation after intubation should be instructed *beforehand* to ventilate at a rate of only 8 to 10 breaths per minute to avoid auto-PEEP and its consequences (eg, sudden severe hypotension). The drop in blood pressure with hyperventilation may be extremely sudden. Prevention of this problem is clearly better than treatment of it.

Acute asthma can be confused with exacerbation of emphysema, especially in the elderly. For different reasons hyperventilation immediately after intubation can cause dire consequences in elderly patients with emphysema. (See the textbook *Advanced Cardiovascular Life Support* for an in-depth discussion of this topic.)

Intubated, Critically Ill Asthmatic: Ventilator-Dependent Permissive Hypercapnia

Adequately sedate and paralyze the patient to allow a "passive" ventilator-patient interaction. Allow PCO_2 to rise (permissive hypercapnia) to values as high as 80 mm Hg. The ensuing drop in pH can be controlled with bicarbonate if needed.

To set the ventilator for permissive hypercapnia:

- Rate: provide mechanical ventilation, 8 to 10 breaths per minute
- Volume: tidal volume, 5 to 7 mL/kg
- Peak flow: 60 L/min with a decelerating pattern

- Inspired oxygen: FIO_2 , 1.0 (ie, 100%)

If the patient's airway is extremely difficult to ventilate, perform the following procedures *in order* until ventilation is adequate:

1. Ensure that the patient is adequately sedated or paralyzed so that there is passive patient-ventilator interaction.
2. Check the patency of the tube for obstructions caused by kinking, mucous plugging, or biting. Aspirate the tube as needed.
3. To ensure that the patient is receiving adequate tidal volume, increase the time for exhalation, decrease the time for inhalation, and increase the peak pressure.
4. Reduce the respiratory rate to 6 to 8 breaths per minute to reduce auto-PEEP to ≤ 15 mm Hg.
5. Reduce the tidal volume to 3 to 5 mL/kg to reduce auto-PEEP to ≤ 15 mm Hg.
6. Increase peak flow to >60 L/min (90 to 120 L/min is commonly used) to further shorten inspiratory time and increase the ratio of inspired to expired air (I:E).^{26C}

Troubleshooting: Hypotension or Desaturation Immediately After Intubation^{26C}

Ensure that the tracheal tube is in the correct position. The tube should be inserted to 21 to 23 cm (measured at the incisors) in most men and to 20 cm in most women. These values may need to be reduced in a small person.

Incorrect placement of the tube must be addressed *immediately*. Do not take the time to obtain a chest x-ray, although an x-ray of the chest after intubation is always appropriate. The immediate consequences of insertion of the tube incorrectly in a patient with severe refractory asthma may be fatal.

If the patient is difficult to ventilate, check the patency of the tube for obstructions caused by kinking, mucous plugging, or biting. Aspirate the tube.

The differential diagnosis of hypotension or desaturation immediately after intubation, once tube position is confirmed, includes tension pneumothorax and massive auto-PEEP buildup. In patients with severe refractory asthma the chest often is silent to auscultation because of poor airflow and hyperinflation of the chest wall.

Tension Pneumothorax

Evidence of a tension pneumothorax includes unilateral expansion of the chest wall, shifting of the trachea, and subcutaneous emphysema. The lifesaving action is to release air from the pleural space with needle decompression. Slowly insert a 16-gauge cannula in the second intercostal space along the midclavicular line, being careful to avoid direct puncture of the lung. If air is emitted, insert a chest tube.

Caution! Insertion of a chest tube in a patient with severe refractory asthma without

pneumothorax will have dire consequences because the visceral pleura of the hyperinflated lung could be punctured, iatrogenically producing pneumothorax. The person inserting the tube would not realize that this has occurred because puncture of the lung would cause a release of air under pressure through the needle catheter or thoracostomy tube, just as would occur with relief of tension pneumothorax. Because of the high pressures experienced by the contralateral mechanically ventilated lung and coexisting auto-PEEP, contralateral pneumothorax would be generated, most likely under tension.

Massive Auto-PEEP Buildup

The most common cause of profound hypotension after intubation is a massive buildup of auto-PEEP. Stop ventilating the patient for a brief period (<1 minute) and allow the auto-PEEP to dissipate. At the same time observe the patient's oxygenation.

Hypotension may also be due to the intubation sedatives, which should respond to volume infusion.

If high auto-PEEP is not present, reconsider alternative explanations.

- Obtain an ECG: Exclude myocardial ischemia or infarction as a consequence of acute respiratory failure (ie, hypoxemia, intubation, and medications). [27C 28C](#)
- Request emergency consultation from a pulmonologist.
- Request emergency consultation from an anesthesiologist.
- Admit the patient to the Critical Care Unit.

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▶ Anaphylaxis

Background

Anaphylactic and anaphylactoid reactions lack universally accepted definitions.

- The term anaphylaxis is typically applied to hypersensitivity reactions mediated by the IgE and IgG4 subclass of antibodies. Some may be mediated by complement (eg, allergic reactions to blood products). Signs of an anaphylactic reaction develop after reexposure to a sensitizing antigen within minutes.
- *Anaphylactoid reactions* look exactly the same, but they are not mediated by an antigen-antibody reaction.
- The manifestations and management of anaphylactic and anaphylactoid reactions are similar so that the distinction is unimportant in relation to treatment of an acute attack.

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Incidence

The annual incidence of anaphylaxis is unknown. Recent US estimates have averaged 30 per 100 000.^{1D} A study in the United Kingdom has reported a frequency of 1 of every 2300 attendees at a hospital Emergency Department.^{2D} The annual international incidence of fatal anaphylactic reactions seems to be 154 per 1 million hospitalized patients per year.^{3D}

Etiology

Insect stings, drugs, contrast media, and some foods (milk, eggs, fish, and shellfish) are the most common causes of anaphylaxis. When hypersensitivity to insect stings is present, 35% to 60% of affected patients will experience anaphylaxis to a subsequent sting.^{4D} Peanut and tree nut (Brazil, almond, hazel, and macadamia nuts) allergies have recently been recognized as particularly dangerous.^{5D} Aspirin and other nonsteroidal anti-inflammatory agents, parenteral penicillins, many other drugs and toxins, vaccines, and beer have become notorious causes of anaphylaxis. Latex-associated anaphylaxis has become a major problem in medical centers. An exercise-induced anaphylaxis (especially after ingestion of certain foods) has been reported. Anaphylaxis may even be idiopathic, typically managed with long-term use of oral steroids. β -Blockers may increase the incidence and severity of anaphylaxis and can produce a paradoxical response to epinephrine.

Signs and Symptoms

The manifestations of anaphylaxis are related to release of chemical mediators from mast cells. The most important mediators of anaphylaxis are histamines, leukotrienes, prostaglandins, thromboxanes, and bradykinins. These mediators contribute to vasodilation, increased capillary permeability, and airway constriction and produce the clinical signs of hypotension, bronchospasm, and angioedema.

The location and concentration of mast cells determine the organ(s) affected. Typically 2 or more of the following systems are involved: cutaneous, respiratory, cardiovascular, and gastrointestinal. The sooner the reaction occurs after exposure, the more likely it is to be severe.

- Upper airway (laryngeal) edema, lower airway edema (asthma), or both may develop acutely and become life-threatening.
- *Cardiovascular collapse* is the most common periarrest manifestation. It is caused by an absolute and a relative hypovolemia. Vasodilation produces a relative hypovolemia, and the intravascular volume loss associated with increased capillary permeability contributes to the absolute volume loss. Cardiac dysfunction is due principally to hypotension but may be complicated by presence of underlying disease or development of myocardial ischemia from epinephrine administration.
- Other symptoms include urticaria, rhinitis, conjunctivitis, abdominal pain, vomiting, diarrhea, and a sense of impending doom.
- The patient may appear either flushed or pale.

Differential Diagnosis

The diagnosis of anaphylaxis is challenging because there is a wide variety of presentations, and no single finding is pathognomonic. Many conditions, including vasovagal reactions (from parenteral injections), functional vocal cord dysfunction, and panic attacks, have been misdiagnosed as anaphylaxis, whereas patients with genuine anaphylaxis do not always receive appropriate therapy.

Angioedema (diffuse soft-tissue swelling) is often present in anaphylaxis. It is typically associated with urticaria, with small to even giant-sized lesions observed. There are, however, many other potential causes of angioedema and urticaria that should be considered.

Scombroid poisoning, which often develops within 30 minutes of eating spoiled tuna, mackerel, or dolphin (mahi-mahi), typically presents with urticaria, nausea, vomiting, diarrhea, and headache. It is treated with antihistamines.

Hereditary angioedema (in which there is a family history of angioedema) presents with no urticaria, but gastrointestinal mucosal edema produces severe abdominal pain, and respiratory mucosal edema produces airway compromise. This form of angioedema is treated with fresh-frozen plasma.

ACE inhibitors are associated with a reactive angioedema predominantly of the upper airway. This reaction can develop days or years after ACE inhibitor therapy is begun. The best medical treatment of this form of angioedema is unclear, but aggressive early airway management is critical.^{6D}

Finally, in some forms of *panic disorder*, functional stridor develops as a result of forced adduction of the vocal cords. In a panic attack there is no urticaria, angioedema, or hypotension.

Key Interventions to Prevent Arrest^{7D}

The approach to therapy is difficult to standardize because etiology, clinical presentation (including severity and course), and organ involvement vary widely. Few randomized trials of treatment approaches have been reported. The following recommendations are commonly used and widely accepted but are based more on consensus than on evidence:

- **Position.** Place victims in a position of comfort. If hypotension is present, elevate the legs until replacement fluids and vasopressors restore the blood pressure.
- **Oxygen.** Administer oxygen at high flow rates.
- **Epinephrine.** Administer epinephrine to all patients with clinical signs of shock, airway swelling, or definite breathing difficulty. Administer intravenous epinephrine if anaphylaxis is profound and life-threatening and vascular access is available. If vascular access is not available or if anaphylaxis is not profound and life-threatening, administer epinephrine by intramuscular injection. Subcutaneous administration may be used but absorption and subsequent achievement of maximum plasma concentration may be delayed with shock.^{8D}—The IM dose of 0.3 to 0.5 mg (1:1000; 1 mL) may be repeated after 5 to 10 minutes if no clinical improvement. —Intravenous epinephrine (1:10 000; 10 mL) 1 to 5 mL or 0.1 to 0.5 mg over 5 minutes should be used only for profound, immediately life-threatening manifestations and when there are no delays in intravenous access. Epinephrine may be diluted to a 1:10 000 solution before infusion. An intravenous infusion (1 mg in 250 mL D₅W [4 µg/mL]) at rates of 1 to 4 µg/min may avoid frequent repeat epinephrine injections.^{9D}
- **Antihistamines.** Administer antihistamines slowly intravenously or intramuscularly (eg, 25 mg of diphenhydramine).
- **H₂ blockers.** Administer H₂ blockers, such as cimetidine (300 mg PO, IM, or IV).^{10D}
- **Isotonic solutions.** Give isotonic crystalloid (normal saline) if hypotension is present and does not respond rapidly to epinephrine. A rapid infusion of 1 to 2 L or even 4 L may be needed initially.
- **Inhaled β-adrenergic agents.** Provide inhaled albuterol if bronchospasm is a major feature. If hypotension is present, administer parenteral epinephrine before inhaled albuterol to prevent a possible further decrease in blood pressure. Inhaled ipratropium may be especially useful for treatment of bronchospasm in patients on β-blockers.
- **Corticosteroids.** Infuse high-dose intravenous corticosteroids slowly or administer intramuscularly after severe attacks, especially for asthmatic patients and those already receiving steroids. The beneficial effects are delayed at least 4 to 6 hours.
- **Envenomation.** Rarely insect envenomation by bees, but not wasps, leaves a venom sac. Immediately scrape away any insect parts at the site of the sting.^{11D} Squeezing is alleged to increase envenomation. Judicious local application of ice may also slow antigen absorption. The application of papain (available in meat tenderizers) to the stinger site is a common home remedy that appears to have no therapeutic value.^{12D}
- **Glucagon.** For patients unresponsive to epinephrine, especially those receiving β-blockers, glucagon may be effective. This agent is short-acting (1 to 2 mg every 5 minutes IM or IV).

Nausea, vomiting, and hyperglycemia are common side effects.

- **Observation.** Observe closely up to 24 hours. Many patients do not respond promptly to therapy, and symptoms may recur in some patients (up to 20%) within 1 to 8 hours despite an intervening asymptomatic period. [13D](#) [14D](#) [15D](#)

Special Considerations

Rapid Progression to Lethal Airway Obstruction

Close observation is required during conventional therapy (see above). Early, elective intubation is indicated for patients with hoarseness, lingual edema, and posterior or oropharyngeal swelling. If respiratory function deteriorates, perform semielective (awake, sedated) tracheal intubation without paralytic agents.

Angioedema. Patients with angioedema pose a particularly worrisome problem because they are at high risk for rapid deterioration. Most will present with some degree of labial or facial swelling. Patients with hoarseness, lingual edema, and posterior or oropharyngeal swelling are at particular risk for respiratory compromise.

Early tracheal intubation. If intubation is delayed, patients can deteriorate over a brief period of time (0.5 to 3 hours), with development of progressive stridor, severe dysphonia or aphonia, laryngeal edema, massive lingual swelling, facial and neck swelling, and hypoxemia. At this point both tracheal intubation and cricothyrotomy may be difficult or impossible. Attempts at tracheal intubation may only further increase laryngeal edema or compromise the airway with bleeding into the oropharynx and narrow glottic opening. The patient may become agitated as a result of hypoxia and may be uncooperative with oxygen therapy.

Paralysis followed by an attempt at tracheal intubation may prove lethal, because the glottic opening is narrow and difficult to see because of the lingual and oropharyngeal edema and the patient is iatrogenically apneic. If tracheal intubation is not successful, even bag-mask ventilation may be impossible, because laryngeal edema will prevent air entry and facial edema will prevent creation of an effective seal between the face and bag mask. Pharmacological paralysis at this point may deprive the patient of the sole mechanism for ventilation, ie, spontaneous breathing attempts.

During Arrest: Key Interventions and Modifications of BLS/ALS Therapy

Death from anaphylaxis may be associated with profound vasodilation, intravascular collapse, tissue hypoxia, and asystole. No data is available on how cardiac arrest procedures should be modified, but difficulties in achieving adequate volume replacement and ventilation are frequent. Reasonable recommendations can be based on experience with nonfatal cases.

Airway, Oxygenation, and Ventilation

Death may result from angioedema and upper or lower airway obstruction. Bag-mask ventilation and tracheal intubation may fail. Cricothyrotomy may be difficult or impossible because severe swelling will obliterate landmarks. In these desperate circumstances, consider the following airway techniques:

- Fiberoptic tracheal intubation
- Digital tracheal intubation, in which the fingers are used to guide insertion of a small (≤ 7 mm) tracheal tube
- Needle cricothyrotomy followed by transtracheal ventilation
- Cricothyrotomy as described for the patient with massive neck swelling^{16D}

Support of Circulation

Support of circulation requires rapid volume resuscitation and administration of vasopressors to support blood pressure. Epinephrine is the drug of choice for treatment of both vasodilation/hypotension and cardiac arrest.

- **Rapid volume expansion** is an absolute requirement. —When anaphylaxis occurs, it can produce profound vasodilation that significantly increases intravascular capacity. Very large volumes should be administered over very short periods; typically 2 to 4 L of isotonic crystalloid should be given.
- **High-dose epinephrine IV** (ie, rapid progression to high dose) should be used without hesitation in patients in full cardiac arrest. —A commonly used sequence: 1 to 3 mg IV (3 minutes), 3 to 5 mg IV (3 minutes), then 4 to 10 $\mu\text{g}/\text{min}$.
- **Antihistamines IV.** There is little data about the value of antihistamines in anaphylactic cardiac arrest, but it is reasonable to assume that little additional harm could result.
- **Steroid therapy.** Although steroids should have no effect if given during a cardiac arrest, they may be of value in the postresuscitation period.
- **Asystole/PEA Algorithms.** Because the arrest rhythm in anaphylaxis is often PEA or asystole, the ILCOR panel recommended adding the other steps in the Asystole and PEA Algorithms. These include —Administration of atropine —Transcutaneous pacing
- **Prolonged CPR.** Cardiac arrest associated with anaphylaxis may respond to longer therapy than usual. —In these circumstances the patient is often a young person with a healthy heart and cardiovascular system. Rapid correction of vasodilation and low blood volume is required. —Effective CPR may maintain sufficient oxygen delivery until the catastrophic effects of the anaphylactic reaction resolve.

Summary

The management of anaphylaxis includes early recognition, anticipation of deterioration, and aggressive support of airway, oxygenation, ventilation, and circulation. Prompt, aggressive therapy may be successful even if cardiac arrest develops.

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▶ Cardiac Arrest Associated With Trauma

Introduction

Survival from out-of-hospital cardiac arrest secondary to blunt trauma is uniformly low in children and adults. [1E](#) [2E](#) [3E](#)

In some out-of-hospital and Emergency Department settings, resuscitative efforts are withheld when patients with blunt trauma are found in asystole or agonal electrical cardiac activity. Survival after cardiac arrest resulting from penetrating trauma is only slightly better; rapid transport to a trauma center is associated with better outcome than

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resuscitation attempts in the field.^{4E}

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BLS and ALS for the trauma patient are fundamentally the same as the care of the patient with a primary cardiac or respiratory arrest. In trauma resuscitation, a "Primary Survey" is performed, with rapid evaluation and stabilization of airway, breathing, and circulation. The trauma rescuer must anticipate, rapidly identify, and immediately treat life-threatening conditions that will interfere with establishing effective airway, oxygenation, ventilation, and circulation.

Cardiopulmonary deterioration associated with trauma has several possible causes, and the management plan may vary for each. Potential causes of cardiopulmonary deterioration and arrest include

1. Severe central neurological injury with secondary cardiovascular collapse
2. Hypoxia secondary to respiratory arrest resulting from neurological injury, airway obstruction, large open pneumothorax, or severe tracheobronchial laceration or crush
3. Direct and severe injury to vital structures, such as the heart, aorta, or pulmonary arteries
4. Underlying medical problems or other conditions that led to the injury, such as sudden VF in the driver of a motor vehicle or the victim of an electric shock
5. Severely diminished cardiac output from tension pneumothorax or pericardial tamponade
6. Exsanguination leading to hypovolemia and severely diminished oxygen delivery
7. Injuries in a cold environment (eg, fractured leg) complicated by secondary severe hypothermia

In cases of cardiac arrest associated with uncontrolled internal hemorrhage or pericardial tamponade, a favorable outcome requires that the victim be rapidly transported to an emergency facility with immediate operative capabilities.^{4E 5E} Despite a rapid and effective out-of-hospital and trauma center response, patients with out-of-hospital cardiopulmonary arrest due to multiple-organ hemorrhage (as commonly seen with blunt trauma) will rarely survive neurologically intact.^{5E 6E 7E 8E}

Patients who survive out-of-hospital cardiopulmonary arrest associated with trauma generally are young, have penetrating injuries, have received early (out-of-hospital) endotracheal intubation, and undergo prompt transport by highly skilled paramedics to a definitive care facility.^{7E 8E 9E 10E}

Extrication and Initial Evaluation

When resuscitative efforts will be attempted, the victim should be rapidly extricated, with protection of the cervical spine. Immediate BLS and ALS interventions will ensure adequate airway, oxygenation, ventilation, and circulation. As soon as the victim is stabilized (and even during stabilization), prepare the victim for rapid evacuation to a facility that provides definitive trauma care. Use lateral neck supports, strapping, and backboards throughout transport to minimize exacerbation of an occult neck and spinal cord injury.^{5E}

When multiple patients receive serious injuries, emergency personnel must establish priorities for care. When the number of patients with critical injuries exceeds the capability of the EMS system, those without a pulse should be considered the lowest priority for care and triage. Most EMS systems have developed guidelines that permit the out-of-hospital pronouncement of death or withholding of cardiac resuscitative efforts when there are multiple patients with critical injuries or when there are injuries incompatible with life. EMS personnel therefore should work within such guidelines when available.

BLS for Cardiac Arrest Associated With Trauma

Provision of BLS requires assessment of responsiveness, establishment of a patent airway, assessment of breathing, support of oxygenation and ventilation if indicated, and assessment and support of circulation.

Establish Unresponsiveness

Head trauma or shock may produce loss of consciousness. If spinal cord injury is present the victim may be conscious but unable to move. Throughout initial assessment and stabilization, the rescuer should monitor patient responsiveness; deterioration could indicate either neurological compromise or cardiorespiratory failure.

Airway

When multisystem trauma is present, or trauma isolated to the head and neck, the spine must be immobilized throughout BLS maneuvers. A jaw thrust is used instead of a head tilt–chin lift to open the airway. If at all possible, a second rescuer should be responsible for immobilizing the head and neck during BLS and until spinal immobilization equipment is applied.

Once the airway is anatomically open, the mouth should be cleared of blood, vomitus, and other secretions. Remove this material with a (gloved) finger sweep or use gauze or a towel to wipe out the mouth. It may also be cleared with suction.

Breathing/Ventilation

Once a patent airway is established, the rescuer should assess for breathing. If breathing is absent or grossly inadequate (eg, agonal or slow and extremely shallow), ventilation is needed. When ventilation is provided with a barrier device, a pocket mask, or a bag-mask system, the cervical spine should be immobilized. If the chest does not expand during ventilation despite repeated attempts to open the airway with a jaw thrust, a tension pneumothorax or hemothorax may be present and should be ruled out or treated by ACLS personnel.

Deliver breaths slowly to reduce the development of gastric inflation and possible regurgitation.

Circulation

If the victim has no signs of circulation (no breathing, coughing, or movement) in response to the rescue breaths and if the healthcare provider detects no carotid pulse, chest compressions should be provided. If an AED is available, it is applied when absence of circulation is detected. The purpose is to check whether VF/VT occurred *first*, causing loss of consciousness, then the trauma.

The AED will evaluate the victim's cardiac rhythm and advise shock delivery if appropriate.

Apply external compression to stop external hemorrhage.

Disability

Throughout all interventions, assess the victim's response. The Glasgow Coma Scale is useful and can be assessed in seconds. Monitor closely for signs of deterioration.

Exposure

The victim may lose heat to the environment through evaporation. Such heat loss will be exacerbated if the victim's clothes are removed or the victim is covered in blood. When possible, keep the victim warm.

ACLS for Cardiac Arrest Associated With Trauma

ALS includes continued assessment and support of airway, oxygenation and ventilation (breathing), and circulation.

Airway

Indications for intubation in the injured patient include

- Respiratory arrest or apnea
- Respiratory failure, including severe hypoventilation, hypoxemia despite oxygen therapy, or respiratory acidosis
- Shock
- Severe head injury
- Inability to protect upper airway (eg, loss of gag reflex, depressed level of consciousness, coma)
- Thoracic injuries (eg, flail chest, pulmonary contusion, penetrating trauma)
- Signs of airway obstruction
- Injuries associated with potential airway obstruction (eg, crushing facial or neck injuries)
- Anticipation of the need for mechanical ventilatory support

Endotracheal intubation is performed with cervical spine outside the trachea. Generally orotracheal intubation is performed. Avoid nasotracheal intubation in the presence of severe maxillofacial injuries, because the tube may migrate outside the trachea during placement. Proper tube placement should be confirmed by clinical examination and use of oximetry and exhaled CO₂ monitor immediately after intubation, during transport, and after any transfer of the patient (eg, from ambulance to hospital gurney).

Unsuccessful tracheal intubation for the patient with massive facial injury and edema is an indication for cricothyrotomy. Cricothyrotomy will provide an emergent, secure airway that supports oxygenation, although ventilation will be suboptimal.

Once a tracheal tube is inserted, simultaneous ventilations and compressions may result in a

tension pneumothorax in an already damaged lung, especially if fractured ribs or a fractured sternum is present. Synchronized ventilations and compressions in a ratio of 1 to 5 may be required in the presence of a damaged thoracic cage.

Unless severe maxillofacial injuries are present, a gastric tube can be inserted to decompress the stomach. In the presence of severe maxillofacial injuries, inserted gastric tubes can migrate intracranially. They should be placed with caution under these conditions, with confirmation of placement into the stomach.

Ventilation

High concentrations of oxygen should be provided even if the victim's oxygenation appears adequate. Once a patent airway is ensured, assess breath sounds and chest expansion. A unilateral decrease in breath sounds associated with inadequate chest expansion during positive-pressure ventilation should be presumed to be caused by *tension pneumothorax* until that complication can be ruled out. Perform needle decompression of the pneumothorax immediately, followed by chest tube insertion. In the absence of an immediate hemodynamic response to thoracic decompression or alternatively in the presence of a penetrating thoracic wound, surgical exploration is warranted.^{9E}

Rescuers should look for and seal any significant open pneumothorax. Tension pneumothorax may develop after sealing of an open pneumothorax, so decompression may be needed.^{5E}

Hemothorax may also interfere with ventilation and chest expansion; treat hemothorax with blood replacement and chest tube insertion. If hemorrhage is severe and continues, surgical exploration may be required.

If the victim has a significant *flail chest*, spontaneous ventilation likely will be inadequate to maintain oxygenation. Treat flail chest with positive-pressure ventilation.

Circulation

Once airway, oxygenation, and ventilation are adequate, circulation is evaluated and supported. As noted above, if pulseless arrest develops, outcome is poor unless a reversible cause can be immediately identified and treated (eg, tension pneumothorax). Successful trauma resuscitation is often dependent on restoration of an adequate circulating blood volume.

The most common terminal cardiac rhythms observed in trauma victims are PEA, bradysystolic rhythms, and occasionally VT/VF. Treatment of PEA requires identification and treatment of reversible causes, such as severe hypovolemia, hypothermia, cardiac tamponade, or tension pneumothorax.^{11E} Development of bradysystolic rhythms often indicates the presence of severe hypovolemia, severe hypoxemia, or cardiorespiratory failure. VF/VT is of course treated with defibrillation. Although epinephrine is typically administered during the ACLS treatment of these arrhythmias, it may be ineffective in the presence of uncorrected severe hypovolemia.

Open thoracotomy does not improve outcome from out-of-hospital blunt trauma arrest, but open

thoracotomy can be lifesaving for patients with penetrating chest trauma, particularly penetrating wounds of the heart.^{6E 8E} During concurrent volume resuscitation for penetrating trauma, prompt emergency thoracotomy will permit direct massage of the heart and indicated surgical procedures. Such procedures may involve relief of cardiac tamponade, control of thoracic and extrathoracic hemorrhage, and aortic cross-clamping.^{6E 8E}

Penetrating cardiac injury should be suspected whenever penetrating trauma to the left chest occurs and whenever penetrating injury is associated with low cardiac output or signs of tamponade (distended neck veins, hypotension, and decreased heart tones). Although pericardiocentesis theoretically is useful, efforts to relieve pericardial tamponade due to penetrating injury should be undertaken only in the hospital.

Cardiac contusions causing significant arrhythmias or impairing cardiac function are present in approximately 10% to 20% of victims of severe, blunt chest trauma.^{12E} Myocardial contusion should be suspected if the trauma victim demonstrates extreme tachycardia, arrhythmias, and ST-T-wave changes. Serum creatinine phosphokinase are often elevated in the patient with blunt chest trauma. An MB fraction >5% has been used historically to diagnose cardiac contusion, but this is not a sensitive indicator of myocardial contusion.^{13E} The diagnosis of myocardial contusion is confirmed by echocardiography or radionuclide angiography.

Volume resuscitation is an important but controversial part of trauma resuscitation. In the field, bolus administration of isotonic crystalloid is indicated to treat hypovolemic shock. Adequate and aggressive volume replacement may be necessary to obtain adequate perfusing pressures.

For patients with penetrating chest trauma who are located a short distance from the trauma center, aggressive fluid resuscitation in the field can increase transport time and has been associated with lower survival than rapid transport with less aggressive fluid resuscitation.^{4E} When massive penetrating trauma or severe hemorrhage is present, immediate surgical exploration is required. Aggressive volume resuscitation in the field will delay arrival at the trauma center, delay surgical interventions to close off bleeding vessels, increase the blood pressure, and consequently accelerate the rate of blood loss.^{4E 14E} Replacement of blood loss in the hospital is accomplished with a combination of packed red blood cells and isotonic crystalloids.

Bleeding must be controlled as soon as possible by whatever appropriate means to maintain adequate blood volume and oxygen-carrying capacity. If external pressure does not stop bleeding or internal bleeding continues, surgical exploration is required.

Indications for Surgical Exploration

Resuscitation may be impossible in the presence of severe, uncontrolled hemorrhage or in the presence of some cardiac, thoracic, or abdominal injuries. In these cases surgical intervention is required. Urgent surgical exploration is indicated for the following conditions:

- Hemodynamic instability despite volume resuscitation
- Excessive chest tube drainage (1.5 to 2.0 L or more total, or >300 mL/h for 3 or more hours)
- Significant hemothorax on chest x-ray
- Suspected cardiac trauma
- Gunshot wounds to the abdomen
- Penetrating torso trauma, particularly if associated with peritoneal perforation
- Positive diagnostic peritoneal lavage (particularly with evidence of ongoing hemorrhage)
- Significant solid-organ or bowel injury

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► Cardiac Arrest Associated With Pregnancy

Background

Most pregnant women have little interest in thinking about the prospect of death. Mortality related to the pregnancy itself is rare, occurring in an estimated 1 of every 30 000 deliveries.^{1F}

A cardiovascular emergency in a pregnant woman creates a special situation—the involvement of a second person, the in utero child. The child must always be considered when an adverse cardiovascular event occurs in a pregnant woman. The decision of whether to perform an emergency cesarean section must be made quickly. Emergency cesarean section has the highest chance of improving the outcome for both mother and child.^{2F}

Significant physiological changes occur in a woman during pregnancy. For example, cardiac output, blood volume, minute ventilation, and oxygen consumption all increase. Furthermore, the gravid uterus may cause significant compression of iliac and abdominal vessels when the mother is in the supine position, resulting in reduced cardiac output and hypotension.

Causes of Cardiac Arrest Associated With Pregnancy

There are many different causes of cardiac arrest in pregnant women. Cardiac arrest in the mother is most commonly related to changes and events that occur at the time of delivery, such as

- Amniotic fluid embolism
- Eclampsia
- Drug toxicity (eg, due to magnesium sulfate or epidural anesthetics)

It may also be related to the complex physiological changes associated with the pregnancy itself, such as

- Congestive cardiomyopathy
- Aortic dissection
- Pulmonary embolism
- Hemorrhage due to a pregnancy-related pathological condition

Finally, and tragically, pregnant women suffer the same problems of motor vehicle accidents,

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falls, assault, attempted suicide, and penetrating trauma (eg, stabbings and gunshot wounds) as the rest of modern society.^{3F} Regrettably, this daily stream of violence and trauma causes many dramatic events that require heroic interventions. Our response has been to craft harsh phrases to guide emergency care, such as "postmortem C-section," "perimortem delivery," "sacrifice mother or child or save mother or child," "harvest the fetus," and "empty the uterus." We walk a thin line between aiding our memory and demeaning our patients. These guidelines will not repeat such phrases.^{4F}

Key Interventions to Prevent Arrest

In an emergency the simplest action may be the most often ignored action. Many cardiovascular problems associated with pregnancy are due to nothing more than anatomy interacting with gravity. The pregnant woman's uterus, great with child, may press down against the inferior vena cava, reducing or blocking blood flow. The ensuing failure of venous blood return can produce hypotension and even shock.^{5F 6F}

To treat a distressed or compromised pregnant patient:

- Place the patient in the left lateral position or *manually and gently* displace the uterus to the left.
- Give 100% oxygen.
- Give a fluid bolus.
- Immediately reevaluate the need for any drugs being administered.

Modifications to BLS Guidelines for Arrest

Do not forget the simple BLS actions you can take:

- Relieve aortocaval compression by manually displacing the gravid uterus.
- You can also use wedge-shaped cushions, multiple pillows, overturned chairs, a rescuer's thighs, or commercially available foam-cushion wedges (eg, the Cardiff wedge) to displace the uterus.^{7F}
- Generally perform chest compressions higher on the sternum to adjust for the shifting of pelvic and abdominal contents toward the head. We lack clear guidelines on how far the compression point should be shifted. Use the pulse check during chest compressions to adjust the sternal compression point.
- Address the need for left-lateral tilt of the torso to prevent compression or blockage of the vena cava. Foam-cushion wedges work best because they provide a wide, firm, angled surface to support the tilted torso during chest compressions. In the usual circumstances surrounding a suddenly pulseless, gravid woman, however, such single-purpose equipment will not be available.
- Two alternative means of support are the angled backs of several chairs and the angled thighs of several rescuers. Overturn a 4-legged chair so that the top of the chair back touches the floor. Align 1 or 2 more overturned chairs on either side of the first so that all are tilted in the same manner. Place the woman on her left side, align her torso parallel with the chair backs, and begin chest compressions (see Figure⁸).

Modifications to ACLS Guidelines for Arrest

There are *no* changes to the standard ACLS algorithms for medications, intubation, and defibrillation. Assess and treat the pregnant woman who has a sudden cardiac arrest by using the Primary and Secondary ABCD Surveys of ACLS as modified for the pregnant woman (Table 1 [☐](#)).

Consider a wide variety of possible causes of arrest such as amniotic fluid embolism, magnesium sulfate toxicity, mishap in patients who received spinal anesthesia, drug overdose, drug abuse, medication toxicity, and iatrogenic events.

Should You Perform an Emergency Cesarean Section to Reduce the Size and Weight of the Gravid Uterus? When Standard BLS and ACLS Fail

If standard application of BLS and ACLS fail and there is some chance that the fetus is viable, consider immediate perimortem cesarean section. The goal is to deliver the fetus within 4 to 5 minutes after the onset of arrest. If at all possible involve obstetric and neonatal personnel. [8F](#)

Why Reduce the Size and Weight of the Uterus?

With the mother in cardiac arrest, the blood supply to the fetus rapidly becomes hypoxic and acidotic, causing adverse effects in the fetus. Return of blood to the mother's heart, blocked by the uterus pressing against the inferior vena cava, must be restored. Consequently *the key to resuscitation of the child is resuscitation of the mother*. The mother cannot be resuscitated until blood flow to her right ventricle is restored.

This results in the familiar admonition to immediately begin cesarean section and remove the baby and placenta when arrest occurs in a near-term pregnant woman. That single act allows access to the infant so that newborn resuscitation can be started. Cesarean section also immediately corrects much of the abnormal physiology of the full-term mother. The critical point to remember is that *you will lose both mother and infant if you cannot restore blood flow to the mother's heart*. [9F](#)

Advance Preparation

Table 2 [☐](#) lists the multiple factors that must be considered in a very short time during an emotionally dramatic event. All Emergency Departments should rehearse their plan of action for this type of event, including location of supplies, sources of extra equipment, and best methods for obtaining subspecialty assistance.

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▶ Electric Shock and Lightning Strikes

Background

Electric Shock

Most electric shock injuries to adults occur in the occupational setting.^{1G} Pediatric electric shock injuries occur most commonly in the home, when the child bites electrical wires, places an object in an electrical socket, contacts an exposed low-voltage wire or appliance, or touches a high-voltage wire outdoors.^{2G}

Electric shock injuries result from the direct effects of current on cell membranes and vascular smooth muscle and from the conversion of electric energy into heat energy as current passes through body tissues. Factors that determine the nature and severity of electric trauma include the magnitude of energy delivered, voltage, resistance to current flow, type of current, duration of contact with the current source, and current pathway. Victims of electric shock can sustain a wide variety of injuries, ranging from a transient unpleasant sensation caused by low-intensity current to instantaneous cardiac arrest caused by exposure to high voltage or high current.

High-tension current generally causes the most serious injuries, although fatal electrocutions may occur with household current (110 V in the United States and Canada, 220 V in Europe, Australia, Asia, and many other localities).^{3G} Bone and skin are most resistant to the passage of electric current; muscle, blood vessels, and nerves conduct with least resistance.^{4G} Skin resistance, the most important factor impeding current flow, can be reduced substantially by

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moisture, thereby converting what ordinarily might be a minor injury into a life-threatening shock.^{5G} Skin resistance can be overcome with increased duration of exposure to current flow. Contact with alternating current at 60 cycles per second (the frequency used in most household and commercial sources of electricity) may cause tetanic skeletal muscle contractions and prevent self-release from the source of the electricity, thereby leading to prolonged duration of exposure. The repetitive frequency of alternating current also increases the likelihood of current flow through the heart during the vulnerable recovery period of the cardiac cycle. This exposure can precipitate VF, analogous to the R-on-T phenomenon.^{6G}

Transthoracic current flow (eg, a hand-to-hand pathway) is more likely to be fatal than a vertical (hand-to-foot) or straddle (foot-to-foot) current path.^{7G} The vertical pathway, however, often causes myocardial injury, which has been attributed to the direct effects of current and coronary artery spasm.^{8G 9G 10G}

Lightning Strike

Lightning strike kills hundreds of people internationally every year and injures many times that number. Lightning injuries have a 30% mortality rate, and up to 70% of survivors sustain significant morbidity.^{11G 12G 13G}

The presentation of lightning strike injuries varies widely, even among groups of people struck at the same time.^{14G} In some victims symptoms are mild and may not require hospitalization, whereas others die from the injury.^{15G 16G}

The primary cause of death in lightning-strike victims is cardiac arrest, which may be associated with primary VF or asystole.^{15G 16G 17G 18G} Lightning acts as an instantaneous, massive direct current countershock, simultaneously depolarizing the entire myocardium.^{16G 19G} In many cases cardiac automaticity may restore organized cardiac activity, and a perfusing rhythm may return spontaneously. However, concomitant respiratory arrest due to thoracic muscle spasm and suppression of the respiratory center may continue after return of spontaneous circulation. Thus, unless ventilatory assistance is provided, a secondary hypoxic cardiac arrest may occur.^{20G}

Lightning can also produce widespread effects on the cardiovascular system, producing extensive catecholamine release or autonomic stimulation. If cardiac arrest does not occur, 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 creatine kinase-MB fraction. Right and left ventricular ejection fractions may also be depressed, but this injury appears to be reversible.^{18G}

Lightning can produce a wide spectrum of neurological injuries. Injuries may be primary, resulting from the effects on the brain. Effects may also be secondary, as a complication of cardiac arrest and hypoxia.^{12G} The current can produce brain hemorrhages, edema, and small-vessel and neuronal injury. Hypoxic encephalopathy can result from cardiac arrest. Effects of a lightning strike on the peripheral nervous system include myelin damage.^{12G}

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

For victims in cardiopulmonary arrest, BLS and ACLS should be instituted immediately. The goal is to oxygenate the heart and brain adequately until cardiac activity is restored. Victims with respiratory arrest may require only ventilation and oxygenation to avoid secondary hypoxic cardiac arrest. Resuscitative attempts may have higher success rates in lightning victims than in patients with cardiac arrest from other causes, and efforts may be effective even when the interval before the resuscitative attempt is prolonged.^{20G}

Clinical Effects

Immediately after electrocution or lightning strike, the victim's respiratory function, circulation, or both, may fail. The patient may be apneic, mottled, unconscious, and in cardiac arrest from VF or asystole.

Respiratory arrest may be caused by a variety of mechanisms:

- Electric current passing through the brain and causing inhibition of medullary respiratory center function
- Tetanic contraction of the diaphragm and chest wall musculature during current exposure
- Prolonged paralysis of respiratory muscles, which may continue for minutes after the electric shock has terminated

If respiratory arrest persists, hypoxic cardiac arrest may occur.

Cardiopulmonary arrest is the primary cause of immediate death due to electrical injury.^{21G} VF or ventricular asystole may occur as a direct result of electric shock. Other serious cardiac arrhythmias, including VT that may progress to VF, may result from exposure to low- or high-voltage current.^{22G}

Modifications of BLS Actions for Arrest Caused by Electric Shock or Lightning Strike

The rescuer must be certain that rescue efforts will not put him or her in danger of electric shock. After the power is turned off by authorized personnel or the energized source is safely cleared from the victim, determine the victim's cardiorespiratory status. Immediately after electrocution, respiration or circulation or both may fail. The patient may be apneic, mottled, unconscious, and in circulatory collapse with VF or asystole.

Vigorous resuscitative measures are indicated, even for those who appear dead on initial evaluation. The prognosis for recovery from electric shock or lightning strike is not readily predictable because the amplitude and duration of the charge usually are unknown. However,

because many victims are young and without preexisting cardiopulmonary disease, they have a reasonable chance for survival if immediate support of cardiopulmonary function is provided.

If spontaneous respiration or circulation is absent, initiate the ABCD techniques outlined in parts 3 and 4 of these guidelines, including EMS system activation, prompt CPR, and use of the AED. The presenting cardiac ECG rhythm may be asystole or VF. ^{23G}

As soon as possible, secure the airway and provide ventilation and supplemental oxygen. When electric shock occurs in a location not readily accessible, such as on a utility pole, rescuers must lower the victim to the ground as quickly as possible. Note: Actions that involve rescuer proximity to live current must be performed only by specially trained rescuers who know how to execute this task. If the victim remains unresponsive, rescuers should start the standard ABCD protocols, including AED use by lay responders.

If the victim has no signs of circulation, start chest compressions as soon as feasible. In addition, use the AED to identify and treat VT or VF.

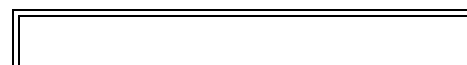
Maintain spinal protection and immobilization during extrication and treatment if there is any likelihood of head or neck trauma. ^{23G 24G} Electrical injuries often cause related trauma, including injury to the spine ^{24G} and muscular strains and fractures due to the tetanic response of skeletal muscles. Remove smoldering clothing, shoes, and belts to prevent further thermal damage.

Modification of ACLS Support for Arrest Caused by Electric Shock or Lightning Strike
Treat VF, asystole, and other serious arrhythmias with ACLS techniques outlined in these guidelines. Quickly attempt defibrillation, if needed, at the scene.

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 and complicate airway control measures, such as endotracheal intubation. For these reasons, intubation should be accomplished on an elective basis before signs of airway obstruction become severe.

For victims with hypovolemic shock or significant tissue destruction, rapid intravenous fluid administration is indicated to counteract shock and correct ongoing fluid losses. Fluid administration should be adequate to maintain diuresis to facilitate excretion of myoglobin, potassium, and other by-products of tissue destruction. ^{19G} Increased capillary permeability may develop in association with tissue injury, so local tissue edema may develop at the site of injury. Because electrothermal burns and underlying tissue injury may require surgical attention, we encourage early consultation with a physician skilled in treatment of electrical injury.

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