Common Conditions Requiring Emergency Life Support

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Education Gaps

Providers should be up to date on the evidence-based current guidelines for pediatric resuscitation and be able to identify and manage life-threatening illness and injury.

Objectives

After completing this article, readers should be able to:

1. Understand the major changes in the 2015 American Heart Association guidelines for pediatric basic life support, advanced life support, and postresuscitation care.
2. Initiate the management and identify prognostic factors associated with near drowning.
3. Identify the signs and symptoms of life-threatening thoracic injuries, including pneumothorax, hemothorax, flail chest, and cardiac tamponade.
4. Identify and manage cerebral edema in the asphyxiated patient.

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ABBREVIATIONS

AHA	American Heart Association
CPR	cardiopulmonary resuscitation
CT	computed tomography
ED	emergency department
ICP	intracranial pressure
ROSC	return of spontaneous circulation

OVERVIEW

There are common emergencies in pediatrics that any pediatrician needs to be equipped to handle. Common office emergencies include respiratory distress, dehydration, seizures, and anaphylaxis. However, pediatricians also need to be prepared for common conditions that require emergency life support. These include, but are not limited to, cardiac arrest, near drowning, thoracic injuries, and cerebral edema. This article focuses on these common conditions that require emergency life support.

PEDIATRIC RESUSCITATION

Introduction

Cardiac arrest is rare in the pediatric population. In 2015, the American Heart Association (AHA) released updated recommendations for pediatric advanced life support. (1) These updated guidelines, incorporating the available

evidence, can guide resuscitation efforts to continue to improve overall survival after pediatric cardiac arrest and, in particular, improve survival with a favorable neurologic outcome.

Basic Life Support

An estimated 6,000 children experience out-of-hospital cardiac arrest per year in the United States. (2) The survival to hospital discharge is 11.3%, and survival with a neurologically favorable outcome is 9.1%. (3) The promotion of bystander cardiopulmonary resuscitation (CPR) remains important. Bystander CPR is performed in out-of-hospital pediatric arrest at an abysmally low rate of 46.5%, with the lowest rates in African American and Hispanic children. (3) Bystander CPR in children increases survival to hospital discharge by 3.7% and increases neurologically favorable outcomes by 2.7%. (4)

The sequence of interventions in cardiopulmonary arrest are different in pediatrics. In 2010, the general AHA guidelines changed from an airway-breathing-circulation (A-B-C) intervention sequence to a circulation-airway-breathing (C-A-B) sequence in an attempt to reduce the time to the first compression. (5) This recommendation was reaffirmed in 2015. However, evidence suggests that the focus on compression-only CPR may not be beneficial in the pediatric population. In infants (<1 year of age), there was no improvement in survival with compression-only CPR compared with no CPR. (3) For older children, the provision of CPR, including rescue breathing and chest compressions, resulted in improved neurologically favorable survival. (3) Given the high rates of asphyxial arrest in the pediatric population, conventional CPR with rescue breathing is recommended. However, starting resuscitation with compressions will not delay the initiation of ventilations by more than 20 seconds. In addition, the sequence of interventions is of less importance in the health-care setting, where more than 1 provider is available and tasks can be accomplished concurrently.

Advanced Life Support

The 2015 updates have been changed to reflect the lack of clear evidence favoring either amiodarone or lidocaine, allowing for the use of either during pediatric cardiac arrest due to ventricular fibrillation and ventricular tachycardia with a pulse. (1) A recent pediatric study demonstrated improvement in the rate of return of spontaneous circulation (ROSC) and 24-hour survival with lidocaine use but no difference in survival to hospital discharge compared with amiodarone use. (6) In 2016, an adult study compared lidocaine with amiodarone and placebo in out-of-hospital cardiac arrest and demonstrated no benefit to either medication over placebo. (7)

There have also been minor modifications to the recommendations for both depth and rate of compressions, with maximums now being added. For depth, the recommendation is that compressions be between 5 and 6 cm due to adult studies showing harm with deeper compressions. (8) There have been no changes to the depth recommendation of 4 cm in infants and 5 cm in children. For rate, the recommendation is now 100 to 120/min based on increased survival with compression rates in that range. (9)

There also continues to be conflicting evidence surrounding the benefits of extracorporeal CPR (extracorporeal membrane oxygenation) in the noncardiac patient. Extracorporeal CPR involves attaching the patient to a machine that externally oxygenates and circulates the blood. There does seem to be clear evidence of improved survival for cardiac patients, both in the postoperative period and otherwise. (10)(11)(12) However, a recent article suggests that it may have general benefits, with the caveat that more patients in the extracorporeal CPR group were patients with a cardiac history. (13)

Postresuscitation Care

Once ROSC is achieved there are evolving recommendations on how to maximize survival to discharge and improve neurologic outcomes. The newest literature focuses on the manipulation of temperature after arrest. There is a clear benefit to avoiding hyperthermia. (14) However, the 2 largest trials on therapeutic hypothermia after in-hospital (15) and out-of-hospital (16) pediatric cardiac arrest did not demonstrate a benefit of hypothermia over therapeutic normothermia.

Hypotension, defined as "a systolic blood pressure less than the fifth percentile for age," in patients with ROSC should be avoided because it is associated with an increased odds of in-hospital mortality. (17) The recommendations for oxygenation and ventilation goals are less clear. In the pediatric literature, it is uncertain whether hyperoxia (18)(19)(20)(21) or hypercarbia (19)(20) after ROSC have detrimental effects on outcomes. Current recommendations are to avoid extremes and target an oxygen saturation greater than or equal to 94%, but they do not include specific ventilation targets. (1)

The question of when to initiate, or terminate, CPR can be difficult. The AHA defines the goal of resuscitation as "to preserve life; restore health; relieve suffering; limit disability, and respect individuals' decisions, rights and privacy." (22) There is currently no single factor or combination of factors that reliably predict outcomes sufficiently to guide recommendations for initiation or termination of CPR. In addition, after ROSC, there are few well-studied

factors that reliably predict neurologically favorable outcomes. Given that children's brains have a higher potential for recovery, the decisions surrounding starting and stopping CPR continue to be challenging and should be made in a discussion between the treating physicians and family members. Several studies have shown that it is beneficial to provide families the opportunity to be physically present at the resuscitation because this has been shown to decrease posttraumatic stress disorder–related symptoms (23) and lessen the pain of death (24) without being disruptive to the resuscitative efforts. (25) Early consultation with a palliative care team, even in cases of prognostic uncertainty, can be beneficial because many physicians have difficulty initiating and leading timely discussions on end-of-life care in pediatric patients. (26)(27)

For cardiac arrest in pediatric patients, based on strong research evidence, pediatricians can:

- Continue to encourage conventional CPR with rescue breathing in pediatric patients (3)
- Promote bystander CPR (4)
- Know the role of extracorporeal membrane oxygenation, especially for cardiac patients (10)(11)(12)
- Pay close attention to blood pressure and temperature in the post-ROSC period (16)
- Promote initiatives for the physical presence of families during resuscitation (23)

DROWNING

Introduction

Drowning accounts for 6,000 to 8,000 deaths in the United States (28) and at least 450,000 deaths worldwide annually. (29) Drowning rates are higher in low-income countries and, predictably, in countries with large accessible bodies of water. (30) In the United States, drowning and near drowning commonly occur in homes with pools and near bodies of fresh water. (28) Drowning is more frequent during the summer months and more common in males and non-Hispanic minorities. (31) There is a bimodal distribution in age, with peaks in the toddler and adolescent age groups. (31)

There are multiple individual risk factors for drowning and near drowning. For younger children, the main factor in drowning events is inadequate supervision by adults. (28) Alcohol use is a frequent trigger for these events and is involved in most adolescent drowning events. (28) Overestimating swimming abilities, risk-taking behaviors such as hyperventilating before submerging, and injuries such as cervical spine injuries also contribute to increased drowning rates. (28)

Several studies indicate an increased risk of drowning in children with epilepsy. (32)(33) A meta-analysis of patients with epilepsy demonstrated a 15-fold greater rate of drowning in this population. (32) Adequate anticonvulsant levels may not be protective, and these patients should be specifically counseled regarding water safety.

Apnea and aspiration lead to pulmonary complications and hypoxemia. (34)(35) Systemic hypoxia involves every organ, with poor outcomes attributed primarily to cerebral hypoxia. (36)(37) The primary systems affected are the pulmonary, neurologic, and cardiovascular.

Near Drowning

Near drowning is distinct from drowning in that the victim survives the drowning episode for at least some period. (31) Near drowning rates are uncertain, with an approximate estimate of being 3 to 4 times as common as drowning events. (38) Loss of consciousness is usually included in the definition of near drowning. (28) There are various subcategories of near drowning, including wet near drowning with pulmonary aspiration of fluids as well as dry near drowning with laryngospasm leading to hypoxia without significant aspiration. (39)(40) The clinical importance of these distinctions has not been established. Children who survive drowning may be left with long-term adverse health outcomes, which can increase health-care costs and create difficulty for families. (30)

Prehospital Care

The most important phase of treatment is the prehospital care phase. The most critical factor is immediate resuscitation, including CPR, by witnesses and bystanders. (37) CPR by laypersons has been demonstrated to improve survival. (37) If resuscitation is delayed until emergency medical services arrive, there is a poor chance of survival or survival with good neurologic outcome. (41) Because the main concern in drowning is hypoxia, rescue breathing alone, and compressions if necessary, can improve the rates of morbidity and mortality in near drowning. (37) If supplemental oxygen is available, it should be used to mitigate organ damage due to hypoxia. (37) Rewarming measures should be initiated immediately, especially for a core temperature less than 91.4°F (<33°C). (37) Because these patients have typically swallowed large amounts of water, the high risk of emesis and aspiration can be diminished by placing breathing patients in a lateral decubitus position if CPR is not being performed. (34)

Routine cervical spine immobilization is not recommended for drowning victims without risk factors (such as a shallow dive or signs of trauma) and may actually

increase the chances of aspiration. (37) The Heimlich maneuver is not recommended as a method of water expulsion as water in the lungs is rapidly reabsorbed and abdominal compressions increase the risk of regurgitation. (37) Arrhythmias requiring an automated external defibrillator, such as ventricular fibrillation, are rare, and management should not delay CPR. (42)

Emergency Department Care

In the emergency department (ED), early intubation and ventilation, or providing supplemental oxygen as needed, should be considered. (43) The patient should be monitored serially, with cardiac monitoring, end-tidal carbon dioxide monitoring, and oxygen saturation measurements. (34) Patients should be monitored with frequent blood gases and a baseline chest radiograph. (34) Acute respiratory distress syndrome is common; as a result, ventilation with high pressures may be necessary. (44) If there is concern for infection (ie, contaminated water), antibiotics should be administered. (45) An electrocardiogram and serum electrolyte measurements should be performed in near drowning victims, but these rarely show life-threatening or severely abnormal values, although metabolic acidosis is usually present. (46) Euglycemia should be maintained for optimal cerebral perfusion. (37)

Resuscitation and rewarming measures should continue once the patient is brought to the hospital. Multiple passive and active rewarming measures can be used in the ED, including removal of wet clothing, basic drying, protective warmed clothing or blankets, warmed intravenous fluids, inhalation of warmed oxygen, and lavage of body cavities with warmed fluids. (34)(37) Prolonged resuscitative efforts may be necessary in hypothermic patients until core temperatures are increased beyond 91.4°F (33°C). Cases of complete recovery have been observed in hypothermic patients despite cardiac arrest. (47) Therapeutic hypothermia has not proved beneficial, (41) although hyperthermia should be avoided.

Although a variety of ED interventions have been put forth, no specific intervention has demonstrated clear superiority to good medical management alone. Candidate interventions not shown to be clearly beneficial include high-dose barbiturate treatment, intracranial pressure (ICP) monitoring, and protective hypothermia. (42)

Disposition

Hospital admission decisions can be guided by the patient's clinical status. Patients who are initially asymptomatic may develop delayed pulmonary edema as a result of ongoing damage to the alveolar lining. In a retrospective study of 75 pediatric near drowning patients, 98% of patients who had delayed onset of symptoms occurred within 4.5 hours of submersion, and all developed symptoms within 7 hours. (48) Another study confirmed that pediatric near drowning patients could be safely discharged after a 4- to 6-hour observation period if their initial Glasgow Coma Scale score was greater than or equal to 13, they required no advanced life support measures, and they had normal oxygen saturations and pulmonary examination findings after the observation period. (49) A normal chest radiograph in a patient who remains asymptomatic after a period of observation can guide the disposition decision, although chest radiographs were not found to be predictive of the clinical course. (48)

Prognostic Factors

Prognosis mainly depends on prehospital factors. Submersion times play a critical role, with submersions longer than 5 minutes having a poorer prognosis. (50)(51)(52) However, there is no clear submersion timeframe during which a near drowning event has a safer outcome.

Hypothermia is typically thought to result in a better outcome than warm water drowning because hypothermia has been postulated to be neuroprotective, especially if it occurs rapidly. (34)(42)(53) However, this has been questioned in a recent study, with cold water showing no protective effect regarding neurologic outcome. (52)

As mentioned previously herein, early resuscitative efforts are the most important factor in predicting a favorable outcome. (51) Previous studies have demonstrated that resuscitation efforts started within 10 minutes of the drowning episode are more likely to be successful, (51) and resuscitation efforts for longer than 25 minutes are typically associated with poor outcomes. (54)(55) However, there have been some patients with favorable outcomes even with prolonged aggressive resuscitation efforts or prolonged submersion times because neurologic recovery can be difficult to predict in the initial period. (43) In a study of 274 pediatric drowning victims, 17% who continued to be resuscitated after arrival at the ED had functional recovery. (43) This study found 3 predictors of poor clinical outcome: a continuing need for CPR and resuscitative efforts in the ED, a pH less than 7, and coma or apnea in the ED. (43) These results have been supported by other studies. (51) Many prognostic scoring systems have been proposed, but none have been universally accepted.

After 48 hours, if neurologic recovery or improvement is not seen, it would be appropriate to initiate a discussion with the family on possible withdrawal of care. (43) In a study of 44 pediatric patients with near drowning, all of the patients who did not have recovery of spontaneous, purposeful

movements within 24 hours had very poor neurologic outcomes or died. (56) In addition, a Glasgow Coma Scale score of less than or equal to 5 and unreactive pupils have been shown to be predictors of poor neurologic outcome. (57)

Prevention

It is hypothesized that 80% of drownings can be prevented. (37) Prevention strategies include appropriate supervision, avoidance of intoxicants such as alcohol, knowledge of swimming and safety, use of appropriate flotation devices during water sports and activities, and the presence of lifeguards. (37)(45) The use of fencing and self-latching gates around pools has been shown to substantially decrease drowning incidents among children. (31)(58) Caregivers should be counseled on appropriate supervision of infants and toddlers in any area of shallow water, including bathtubs. (59)

Infant swimming programs may provide caregivers a false sense of security and should not replace close supervision. In a 2010 policy statement on drowning prevention, the American Academy of Pediatrics reevaluated the age at which swimming lessons can be considered protective based on new evidence. (60) The American Academy of Pediatrics continues to support swimming instruction for children older than 4 years. Several recent studies have suggested that children 1 to 4 years of age may also benefit from lessons, although lessons for this age group are not recommended. (61)(62)

In drowning prevention and near drowning, based on strong research evidence, it is important for pediatricians to:

- Understand the factors that influence prognosis in near drowning (50)(51)(52)
- Provide anticipatory guidance to parents of young children and those with epilepsy regarding water safety (60)

LIFE-THREATENING THORACIC INJURIES

Introduction

Although thoracic trauma occurs infrequently in pediatric patients, it is essential to identify potentially life-threatening injuries to provide prompt medical or surgical interventions. The incidence of thoracic trauma in children has been demonstrated to be between 4% and 8%, with blunt and penetrating trauma accounting for 85% and 15% of cases, respectively. (63)(64) The most common mechanisms of injury in patients sustaining blunt trauma are motor vehicle accidents, intentional injury, and falls. The most common thoracic injuries due to blunt trauma include pulmonary contusions, pneumothorax, hemothorax, and rib fractures. (65) Gunshot wounds, stabbings, and impalement are the most common etiologies of penetrating thoracic trauma resulting in pneumothorax, hemothorax, pulmonary contusion, pulmonary laceration, and blood vessel injury. (65) The mortality rate for thoracic injury is approximately 20%, with a higher rate in penetrating trauma than in blunt trauma. (65)(66) This review focuses on the identification (Table 1) and management (Table 2) of 4 acutely life-threatening thoracic injuries: pneumothorax, hemothorax, flail chest, and cardiac tamponade.

Pneumothorax

Pneumothorax is defined as the accumulation of air in the pleural cavity. Tension pneumothorax occurs when air in the pleural space causes a contralateral shift of the mediastinum (Fig 1). This causes compression of the thoracic vasculature, reducing systemic venous return to the heart and, therefore, reducing cardiac output. Compression of the contralateral lung restricts lung expansion. Children have a higher rate of tension pneumothorax due to greater compliance of the mediastinal structures. Tension pneumothorax is a diagnosis that should be made clinically, and management should not be delayed for radiologic confirmation. Common symptoms include shortness of breath and chest pain. Vital sign abnormalities may include tachypnea, tachycardia, hypotension, and hypoxia. Examination findings may include cyanosis or pallor, contralateral tracheal deviation, ipsilateral decreased breath sounds, and neck vein distention. The absence of lung sliding on a "point of care focused abdominal sonography in trauma" can provide immediate bedside confirmation. Management includes immediate needle decompression in the midclavicular line at the second intercostal space followed by chest tube insertion.

An open pneumothorax, also known as a sucking chest wound, typically results from penetrating trauma. Air preferentially enters during inspiration through the chest wall defect, compromising lung expansion. A sterile dressing that is occlusive on 3 sides (applied when the patient is in full expiration) will prevent air entry through the chest wall defect on inspiration while allowing air to escape from the nonocclusive fourth side during expiration. Definitive management incudes chest tube placement and closure of the chest wall defect.

Hemothorax

Hemothorax is defined as the accumulation of blood in the pleural space. Hemothorax occurs 38% and 64% of the time in pediatric patients with blunt and penetrating thoracic trauma, respectively. (65) A hemothorax is most commonly

TABLE 1. **Life-threatening Thoracic Injuries: Clinical Signs**

SIGN	TENSION PNEUMOTHORAX	MASSIVE HEMOTHORAX	CARDIAC TAMPONADE
Breath sounds	Decreased (ipsilateral)	Decreased (ipsilateral)	Normal
Lung percussion	Hyperresonant (ipilateral)	Dull (ipsilateral)	Normal
Tracheal position	Deviation (contralateral)	Midline	Midline
Neck veins	Distended	Flat	Distended
Heart sounds	Normal	Normal	Muffled

The pulmonary component of the extended focused abdominal sonography in trauma examination can assist in rapidly identifying many of the injuries requiring urgent treatment at the bedside.

caused by penetrating injury through injury to systemic or hilar vessels but may also occur from blunt trauma from rib fractures lacerating the underlying lung or other vascular structures. In the adult-sized patient, a massive hemothorax is defined as more than 1,500 mL of blood in the pleural cavity. In pediatric patients, it is defined as greater than or equal to one-third of a child's blood volume (based on an estimated total blood volume of approximately 80 mL/kg).

Clinical signs and symptoms include tachypnea, hypoxemia, increased work of breathing, ipsilateral absent or decreased breath sounds, tachycardia, and hypotension. Hemothorax can be identified with point of care lung ultrasonography or chest radiography (Fig 2). Blood in the pleural cavity may tamponade bleeding, and, therefore, rapid evacuation may actually cause additional bleeding. For this reason, the patient should be adequately fluid resuscitated before thoracostomy tube placement if time permits. Ongoing bleeding may require packed red blood cell transfusion, activation of a massive transfusion protocol, tranexamic acid, or an operative thoracotomy.

Flail Chest

Flail chest is defined as the fracture of 3 or more consecutive ribs in more than 2 locations. This creates a mechanically unstable floating or flail segment. A review of the National Trauma Data Bank revealed a 1% rate of flail chest in admitted patients at level 1 and 2 trauma centers. (67) In the Israeli National Trauma Registry, 2% and 9% of the documented flail chest injuries occurred in patients aged 0 to 14 years and 15 to 24 years, respectively. (68) The same Israeli study demonstrated a mortality rate of 20%, with the risk of mortality higher with increasing age. (68)

Flail chest is typically due to a blunt mechanism, such as a motor vehicle accident, a vehicle versus pedestrian or bicycle accident, or a fall from a height. It can occur over the anterior, posterior, and lateral thoracic chest wall. The degree of force required to cause a flail chest increases the risk of associated underlying pulmonary injury.

Clinically, external signs of chest trauma are frequent. The unstable floating segment leads to paradoxical chest motion. (69) Normally, contraction of the diaphragm and chest wall expansion outward occurs during inspiration to create the negative intrathoracic pressure required for lung expansion. Paradoxical chest wall motion is observed when the flail chest segment is pulled inward from the negative intrathoracic pressure generated during inspiration. This causes a decrease in lung volumes, atelectasis, chest tightness, and dyspnea. (67)

TABLE 2. **Life-threatening Thoracic Injuries: Emergency Management**

INJURY	TREATMENT
Tension pneumothorax	Needle thoracentesis, tube thoracostomy
Open pneumothorax	3-Sided chest wall dressing, tube thoracostomy
Flail chest	Positive pressure ventilation
Massive hemothorax	Crystalloid/colloid resuscitation, tube thoracostomy
Cardiac tamponade	Pericardiocentesis

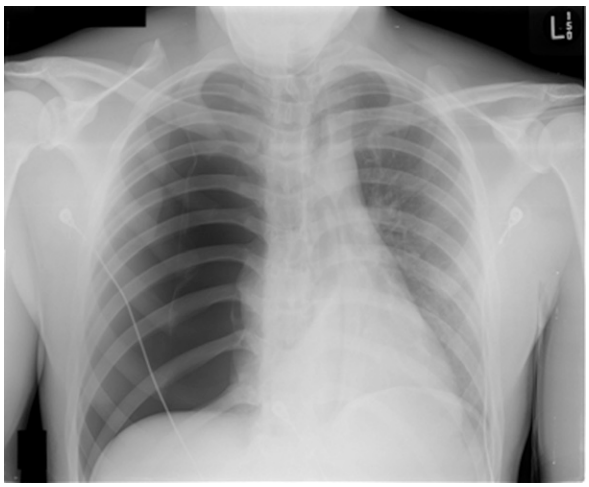

Figure 1. Chest radiograph of tension pneumothorax.

The primary management of flail chest is positive pressure ventilation and possibly surgical fixation. (69)(70)

Cardiac Tamponade

Cardiac tamponade is defined as compression of the heart due to accumulation of fluid in the pericardium; it is potentially life-threatening. (71) The pericardium is an elastic sac with a fixed intrapericardial volume, and once this volume is reached the pericardium loses its elasticity. The accumulation of fluid increases intrapericardial pressure, resulting in decreased systemic venous return, decreased stroke volume, and, subsequently, decreased cardiac output.

Cardiac tamponade can be due to infectious, autoimmune, oncologic, cardiac, metabolic, and traumatic causes. Traumatic cardiac tamponade can be due to blunt or penetrating trauma.

The clinical findings of cardiac tamponade are described by the Beck triad of hypotension, distended neck veins, and distant or muffled heart sounds. Additional findings include tachycardia and pulsus paradoxus. (72) The extent of symptoms is related to the rapidity of fluid accumulation with gradual accumulation having fewer symptoms. Tachycardia compensates to maintain cardiac output in response to decreased stroke volume. Tachycardia may not be present in patients with underlying bradycardia or in patients who present with early tamponade.

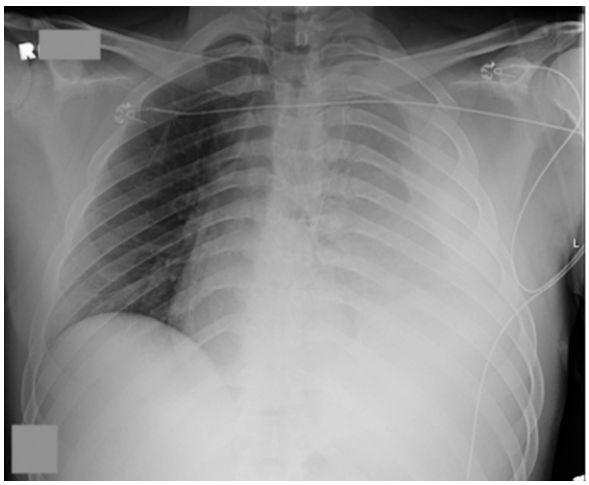

Figure 2. Chest radiograph of massive left hemothorax.

Pulsus paradoxus is defined as "an inspiratory decrease in systolic blood pressure of greater than 10 mm Hg." (72) Pericardial tamponade results in ventricular interdependence, which indicates that the total volume of blood in the heart chambers is constant. An increase in blood volume in one chamber produces a decrease in blood volume in the other chambers. During tamponade physiology, inspiration results in negative intrathoracic pressure. This increases right heart venous return and decreases left heart blood volume, ultimately decreasing systolic blood pressure during inspiration.

Electrocardiography may reveal low voltages, sinus tachycardia, signs of pericarditis such as diffuse ST-segment elevations, or electrical alternans. Low voltage is thought to be due to the pericardial fluid having a dampening effect. Electrical alternans is the alternation of the height of the QRS complex from beat to beat. Electrical alternans is thought to be due to movement of the heart in the pericardial fluid. (71)(72)

Chest radiographs may show an enlarged cardiothymic silhouette. The "point of care focused abdominal sonography in trauma" can directly visualize fluid in the pericardial space and can be used to guide pericardiocentesis.

Management of cardiac tamponade requires removal of the pericardial fluid. This can be accomplished by either pericardiocentesis or surgical drainage. Catheter drainage of pericardial fluid is the preferred method of pericardiocentesis in urgent situations. (72) Surgical drainage with or without a pericardial window is the preferred method for traumatic hemopericardium or purulent pericarditis. (72)

In cases of thoracic injury, it is important for the pediatrician to:

- Use clinical signs and symptoms to rapidly identify life-threatening thoracic injuries (based on some research evidence as well as consensus) (63)
- Understand that point of care ultrasonography, such as the extended focused abdominal sonography in trauma, performed at the bedside can confirm the clinical diagnosis of life-threatening thoracic injury and guide procedures such as pericardiocentesis (based primarily on consensus due to lack of relevant clinical studies)

- Review the rapid interventions that can prevent further deterioration; both supportive care, such as oxygen, ventilation, and fluid resuscitation, and specific interventions, such as needle thoracentesis, tube thoracostomy, and pericardiocentesis, may be required (based on some research evidence as well as consensus) (70)

CEREBRAL EDEMA

Introduction

Cerebral edema is a dreaded and devastating complication of an asphyxial or traumatic event. Asphyxiation can occur by way of several mechanisms in infants and children, including suffocation, choking, and cardiac arrest. Asphyxial cardiac arrest and traumatic brain injury are the most common reasons for children to require some form of cerebral resuscitation. (73) Both asphyxia and traumatic brain injury can result in secondary cerebral injury, including ischemic injury, stimulation of cascades of excitotoxicity, neuronal death, and cerebral edema. (73)

Cerebral edema is defined as excess accumulation of water in the intracellular or extracellular space of the brain. (74) Cerebral edema, if untreated, leads to increased ICP, ischemia, and herniation. Signs and symptoms of cerebral edema in infants and children can be subtle, and early recognition and early intervention are imperative to prevent further neurologic injury and death.

Pathophysiology

The Monro-Kellie hypothesis is that the total volume of the contents of the skull—the brain, cerebrospinal fluid, and blood volume—should be constant at all times. (75) Consequentially, if the volume of one of the contents is increased, the volume of other contents must decrease to compensate. These compensatory mechanisms fail if the volume of brain, blood, or cerebrospinal fluid continues to increase, resulting in a rise in ICP. As ICP rises, cerebral blood flow is decreased, leading to diffuse ischemia and increased cerebral edema with an increased risk for herniation. (74)

After an asphyxia event, edema and increased arterial cerebral blood volume result in cerebral swelling. It is believed that an increase in cerebral blood volume in children may have a greater contribution to cerebral swelling than it does in adults. (76) An open fontanel in infants does not prevent cerebral edema and its complications. (73)

The injury cascade begins with the release of glutamate into the extracellular space, which causes calcium and sodium channels to open. Sodium builds up intracellularly by means of a membrane ATPase pump, thus creating an osmotic gradient for water to flow into the cell. Because of hypoxia, the ATPase pump is eventually inactivated, which reduces calcium and sodium exchange. Because both sodium and calcium increase intracellularly, water continues to move from the extracellular to the intracellular space, and the increase in intracellular calcium stimulates an inflammatory response. This inflammatory response results in the release of free radicals and proteases, which attack cell membranes and blood vessels, causing irreversible cell damage. (75)

Clinical Features

The presentation of cerebral edema varies by both the age of the patient and whether the increase in pressure is acute or chronic. A high index of suspicion is required to quickly identify and treat the underlying process. Failure to act quickly may have devastating consequences.

Symptoms of cerebral edema and subsequent increased ICP include headache, nausea, and vomiting. In nonverbal children and infants, the signs can be subtle and include fussiness, irritability, and somnolence. Papilledema may not be present if the rise in ICP was sudden because it may take days to develop. Occasionally, extraocular movement abnormalities and unequal pupil size are found on physical examination. Less frequently, findings of increased ICP may also include seizures or a preference for the knee-chest position. The Cushing triad is the presence of bradycardia, hypertension, and abnormal respirations. (75) These are late findings and a warning of impending herniation. (75)

Imaging

Neuroimaging should not delay treatment if cerebral edema is suspected. In patients with an acute elevation in ICP secondary to cerebral edema, neuroimaging findings may not be apparent in the first 24 hours. When imaging is obtained, a noncontrast brain computed tomographic (CT) scan is preferred owing to its rapid availability and speed of imaging compared with magnetic resonance imaging. (73) On a CT scan, areas of edema appear as low density, which is caused by the dilution of the components of the white matter. (73) The CT scan can reveal both the presence of edema and the specific type of edema. (75) Magnetic resonance imaging findings on diffusion-weighted imaging windows can show watershed pattern injury with edema.

Nonpharmacologic Management

The assessment of a patient with suspected cerebral edema follows the sequence of airway, breathing, and circulation

and the immediate management of conditions identified before assessing neurologic status. If impending respiratory failure, herniation, or loss of airway protection is suspected, the airway should be secured by means of intubation using a cerebroprotective approach to sedation and paralysis. Accumulation of carbon dioxide results in vasodilation of cerebral vasculature and, thus, it is recommended to maintain a $Paco_2$ of 30 to 35 mm Hg to help prevent the development of intracranial hypertension. (73) A $Paco_2$ less than 25 mm Hg, an end point that at one time was used to guide ventilator settings, has been found to instead cause cerebral vasoconstriction, leading to worsening hypoxia and cerebral ischemia. (75) Continuous end-tidal carbon dioxide monitoring can be used to assess the degree of hyperventilation.

Elevation of the head of the bed to no more than 15° to 30° allows for venous drainage by gravity. In addition, the patient's head should be kept midline to limit neck vein compression and impaired venous return. (74)

In a patient with cerebral edema, close blood pressure monitoring is imperative. (74) Fluid restriction has a small effect on cerebral edema and, if practiced too aggressively, can lead to hypotension, which may conversely increase ICP. (75) Hypertension can be a compensatory mechanism to maintain cerebral blood flow in the setting of elevated ICP. Rapidly decreasing an elevated blood pressure or failing to address hypotension in a patient with cerebral edema can result in cerebral ischemia and worse neurologic outcomes. (75)

Tight temperature and glucose control in a patient with cerebral edema can have a neuroprotective effect. Previous studies have demonstrated that body temperature higher than 99.5°F (37.5°C) and blood glucose level greater than 150 mg/dL (8.3 mmol/L) are associated with worsening cerebral edema. (75) Therefore, hyperthermia and glucose-containing substances should be avoided in the patient with cerebral edema. Trials of therapeutic hypothermia after in-hospital (15) and out-of-hospital (16) cardiac arrest failed to show a benefit to hypothermia over therapeutic normothermia in pediatric patients.

Pharmacologic Management

Hyperosmolar therapy with 3% hypertonic saline or mannitol is administered to patients with suspected cerebral edema with ICP elevations greater than 20 mm Hg or with clinical signs of impending herniation. (75) The recommended mannitol dose is 0.5 to 1.0 g/kg. This dose can be repeated every 4 hours for sustained ICP greater than 20 mm Hg. Close monitoring of urine output and blood pressure is essential because mannitol promotes diuresis. (75)

Unlike mannitol, hypertonic (3%) saline does not result in diuresis and may maintain hemodynamic stability by maintaining intravascular volume. Hypertonic saline can be administered as an initial bolus of 5 to 10 mL/kg, followed by an infusion of 0.5 to 1.5 mL/kg per hour. (75) Serum sodium levels should be monitored. Serum sodium levels greater than 160 to 165 mEq/L (>160-165 mmol/L) do not provide an additional reduction in ICP. (75)

Additional therapies to consider in patients with raised ICP include antiepileptics and corticosteroids. The role of corticosteroids in head trauma, however, is not well defined. (74)

For patients with cerebral edema, based on some research evidence as well as consensus, it is important for pediatricians to:

- Understand that signs and symptoms of cerebral edema may be subtle in young children and when readily apparent often indicate advanced disease and impending herniation. (73)(74)(75)
- Use both nonpharmacologic methods, such as elevation of the head of the bed, maintenance of normal blood pressure, and control of hyperventilation, and pharmacologic treatment with hyperosmolar therapy, such as mannitol or hypertonic saline, to rapidly reduce cerebral edema if impending herniation is suspected. (73)(74)(75)

References for this article are at http://pedsinreview.aappublications.org/content/40/6/291.

PIR Quiz

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1. A 9-month-old infant presents to the emergency department after asphyxial cardiac arrest. Which of the following is most likely to increase this child's chances of neurologically favorable survival?
    A. Bystander cardiopulmonary resuscitation (CPR).
    B. Compression depth of 2 cm.
    C. Compression rate of 80/min.
    D. Use of amiodarone.
    E. Use of compression-only CPR.
2. Return of spontaneous circulation is achieved in the emergency department in a 2-year-old child who had a witnessed cardiac arrest. Which of the following is currently recommended for postresuscitation care?
    A. Broad spectrum antibiotics.
    B. Permissive hypercapnia.
    C. Target oxygen saturations of 94% or greater.
    D. 24-hour therapeutic hypotension.
    E. 24-hour therapeutic hypothermia.
3. A 3-year-old child was found underwater in the family swimming pool in full arrest. Bystander CPR was administered. On arrival at the emergency department, the child is comatose, has a palpable pulse, and is breathing spontaneously, although respirations are labored. Which of the following is the most important next step in the management of this patient?
    A. Heimlich maneuver to empty the stomach of swallowed water.
    B. Immediate endotracheal intubation.
    C. Initiation of broad spectrum antibiotics.
    D. Maintenance of cervical spine immobilization.
    E. Measurement of serum electrolytes.
4. A 6-year-old child has hypotension, distended neck veins, and muffled heart sounds after suffering blunt chest trauma in a motor vehicle collision. Breath sounds are equal bilaterally. The nurse notes a pulsus paradoxus of 25 mm Hg. Which of the following is the most appropriate physiologic explanation for the nurse's finding?
    A. The patient has a flail chest. Outward motion of the flail chest segment during inspiration causes the pulsus paradoxus.
    B. The patient has an isolated pneumothorax. Hypotension leads directly to the pulsus paradoxus.
    C. The patient has an isolated pneumothorax. Hypoxia itself causes the pulsus paradoxus.
    D. The patient has pericardial tamponade. Tachycardia and restriction in ventilation cause the pulsus paradoxus.
    E. The patient has pericardial tamponade. Ventricular interdependence and increased right heart venous return during inspiration cause the pulsus paradoxus.

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5. A 10-year-old child has had a ventriculoperitoneal shunt in place for several years due to hydrocephalus after surgery for a central nervous system malignancy. The mother notes that the child has been increasingly sleepy during the past 24 hours, has vomited 5 times in the past 8 hours, and complains of a severe headache. On physical examination his heart rate is 50 beats/min and blood pressure is 135/75 mm Hg. He has unequal pupil sizes. His respirations are irregular. On neurologic examination, the child is quite somnolent but does respond slowly to questions. Which of the following is the most appropriate next step in the management of this patient?

   A. Administer intravenous fluids with a high concentration of glucose.
   B. Administer labetalol with a target systolic blood pressure of 110 mm Hg.
   C. Administer mannitol, 0.5 g/kg.
   D. Administer normal saline, 20 mL/kg.
   E. Lower the head of the bed.

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