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Hypoglycemia in Infants and Children

Hypoglycemia is the most common metabolic disorder in children. The causes for hypoglycemia are many and diverse.[1] One of the most frequent causes of hypoglycemia is insulin/glucose imbalance in diabetic children; since the management of diabetes and its complications is a subject on its own, this topic will not be included in this issue. The authors focus on issues important to the emergency physician, with emphasis on the need to diagnose and treat hypoglycemia quickly. In the very young infant, failure to recognize and treat hypoglycemia may lead to permanent neurological sequelae. It is also important to remember that hypoglycemia is a symptom and not a diagnosis or a disease entity and always has an underlying etiology that must be ascertained. Sometimes the emergency department (ED) physician may determine the underlying cause, and sometimes the symptom must be treated without knowing the underlying cause. Because hypoglycemia can be rapidly and easily determined at the bedside, there is little reason not to obtain this important piece of clinical information. Recognition of hypoglycemia in the seriously ill child is so significant that it may be seen as important as a critical vital sign.

— The Editor

Hypoglycemia and Glucose Metabolism

Hypoglycemia is defined generally as a serum glucose < 50 mg/dL with neuroglycopenic symptoms, or < 40 mg/dL in the absence of symptoms. In the developing neonate, however, there is no agreed-upon absolute minimum, with the suggested range 30–45 mg/dL. Using < 30 mg/dL as a minimum in neonates, the estimated incidence of hypoglycemia varies from 0.4% to 11.4%.[1]

Glucose homeostasis in infants and children results from a complex interplay of glucose ingestion and storage as glycogen as well as synthesis of glucose from metabolic intermediates and the use of alternative metabolic fuels such as fats and amino acids, all under hormonal control and subject to exogenous factors such as stress and drugs. Beginning during gestation, glucose is transported across the placenta from the mother to meet the metabolic needs of the fetus. The developing brain utilizes glucose 20 times faster than any other organ and development is critically dependent on glucose availability. As a result, persistent hypoglycemia may result in permanent neurologic damage in the newborn. Near the end of gestation, however, the appearance of hepatic enzymes for glycogen synthesis and glycogenolysis as well as gluconeogenesis prepare the fetus and newborn to utilize and generate the high glucose levels needed in the perinatal and neonatal period. With the interruption of placental blood flow, a cascade of events enables glucose homeostasis in the newborn. Hormonal release results in mobilization of hepatic glycogen and adequate gluconeogenesis which must be coupled with ingestion of glucose, amino acid, and lipid-containing breast milk or formula. Due to the

Executive Summary

- Hypoglycemia is common and a potential source of morbidity and mortality for children if not recognized early and treated aggressively.
- Hypoglycemia should be considered, and appropriate testing performed, in any child that presents to the ED, especially infants or children with tachypnea, tachycardia, vomiting, weakness, altered mental status, seizures, or coma.
- Hypoglycemia may be a symptom of an underlying disease entity, and ED physicians should obtain critical blood samples during the acute episode to facilitate a timely definitive diagnosis and impress subspecialists.
- Even after the initial hypoglycemia is treated, it is critical to continue to monitor and correct any further episodes of hypoglycemia.

rapid utilization of glucose in the neonatal period, full-term, healthy infants have exuberant gluconeogenesis to compensate for rapid fluctuations. Such compensatory mechanisms are often less than adequate in the preterm infant, increasing the likelihood of hypoglycemia between feeds and during stress.

Glucose mobilization and storage is regulated by the hormones insulin, glucagon, catecholamines, cortisol, and growth hormone, as well as local cellular mediators. Insulin plays a primary role in serum glucose homeostasis beginning in the fetus. To summarize, when glucose levels increase, it is transported to the pancreas and results in insulin release, while decreasing serum glucose concentration results in decreased insulin concentration.

Decreased blood sugar also results in counterregulatory hormone release (cortisol, epinephrine, and glucagon), which stimulates gluconeogenesis and synthesis of glucose from precursors, utilization of fats and amino acids for energy sources, and the release of substrate through glycogenolysis in the liver. Metabolism of alternative fuels leads to ketotic waste products in the blood. In the absence of sufficient glycogen stores, as in the preterm infant, counter-regulatory hormones may not be able to maintain serum glucose in this way.

Symptoms

Symptoms of hypoglycemia are multiple and varied. They may be very subtle, or part of a life-threatening presentation. Classic clinical manifestations of hypoglycemia include the triad of altered mental status, serum glucose < 40 mg/dL, and relief of symptoms with glucose administration. The clinical picture of a hypoglycemic patient may, however, be significantly clouded or exaggerated by age, recent diet, previous hypoglycemic episodes, current medications (including sedatives), and pre-existing illness. In general, hypoglycemic symptoms can be divided into two broad categories: adrenergic, which are those associated with activation of the autonomic nervous system; and neuroglycopenic, resulting from low brain glucose.[2] *(See Table 1.)* Adrenergic symptoms usually occur early in the process and may be seen prior to arrival in the ED or be the reason for the ED visit. Typically, a rapid decline in the glucose level leads to these nonspecific symptoms that include tachycardia, tachypnea, vomiting, weakness, nervousness, and diaphoresis. All of these are common findings in infants and children in the ED.[3]

Neuroglycopenic symptoms occur late or with a more gradual decrease in the glucose level. Initially, symptoms include headache, irritability, psychotic behavior, poor feeding, lethargy, altered mental status, seizures, and coma. These symptoms may be obvious in the older child; however, newborns and infants may present with more subtle presentations, including hypotonia, hypothermia, feeding difficulties, jitteriness, and exaggerated reflexes.

If the hypoglycemic state is not corrected, transient or permanent cerebral dysfunction may occur. The mechanism of neurologic injury is incompletely understood and not solely due to an absence of substrate but may involve cytotoxic intermediate byproducts such as aspartic acid. The most common neurological damage caused by persistent hypoglycemia includes ischemia and hemorrhage in the posterior white matter areas, middle cerebral artery infarction, and basal ganglia/thalamic abnormalities.[2] These injuries may lead to permanent cognitive and motor manifestations.

Hypoglycemia may be the underlying etiology for a diversity of critical illnesses such as acute respiratory failure, which can mimic pneumonia; sepsis; acute congestive heart failure; and status epilepticus.[4-6] Altered level of consciousness in children is a clinical challenge, and of the many etiologies, hypoglycemia must be sought and treated immediately.[7]

It is essential to consider hypoglycemia as a potential cause of seizures in the newborn.[8] Neonatal seizures may be very subtle. Clinical findings may include:

- Ocular signs (tonic horizontal deviation of eyes or sustained eye opening with ocular fixation or cycled fluttering);
- Oral-facial-lingual movements (chewing, tongue-thrusting, lip-smacking);
- Limb movements (cycling,

Table 1. Clinical Presentation of Hypoglycemia

Whipple's Triad
• Altered mental status
• Serum glucose less than 40 mg/dL
• Relief with glucose administration
Adrenergic Symptoms
• Tachypnea
• Tachycardia
• Vomiting
• Weakness
• Nervousness
• Diaphoresis
Neuroglycopenic Symptoms
• Headache
• Irritability
• Psychotic behavior
• Lethargy
• Altered mental status
• Seizures
• Coma
Neonatal Hypoglycemia
• Hypotonia
• Hypothermia
• Feeding difficulties
• Jitteriness
• Hyperreflexia

paddling, boxing jabs);
- Autonomic phenomena (tachycardia or bradycardia);
- Apnea.

Causes of Hypoglycemia

The myriad of causes for hypoglycemia can be best discussed along physiologic lines and are summarized in **Tables 2 and 3.**

Decreased production/availability of glucose. *Small for gestational age (SGA) and premature infants.* As soon as placental flow is interrupted, the newborn must depend on its own supply of glucose. Infants who are premature or SGA will have less stores of glucose, an immature glucose production system, or both.[9] Add this to the fact that the brains of these infants are significantly larger per body mass than at any other time in life, and it is obvious why these children may be susceptible to hypoglycemia. Obtaining the history that the child presenting to the ED was premature or SGA will alert the ED physician to the possibility of hypoglycemia. These patients should be considered for bedside glucose testing immediately upon arrival. Correction of hypoglycemia should be a priority.

Malnutrition/fasting. Ketotic hypoglycemia is the most common cause of hypoglycemia in children from 1 year to 5 years of age.[3] In normal fasted individuals, the maintenance of plasma glucose concentrations in the normal range is dependent upon:

1. A normal endocrine system;
2. Functionally intact hepatic glycogenolytic and gluconeogenic systems; and
3. An adequate supply of endogenous metabolic fuels.

Adults are capable of maintaining a normal glucose concentration even when totally deprived of calories for weeks or more. Children and neonates, in contrast, are unable to supply sufficient glucose to meet obligatory demands and exhibit progressive fall in plasma glucose concentration to hypoglycemic levels when fasted for even short periods of time (12 to 24 hours). For unknown reasons, children with ketotic hypoglycemia are less able to tolerate brief periods of fasting.

Classically, this disorder presents between 18 months and 5 years of age, coinciding with the time children usually sleep through the night. Fortunately, there is usually spontaneous resolution by 8–9 years of age due to a relative decrease in glucose requirement per body mass, maturity of the autonomic nervous system, and maturation of the gluconeogenic pathways. Although the cause of the hypoglycemia remains undefined, it is often seen in children who are small for their age. Increased glucose requirements on a per kilogram body weight basis in the young child, when compared with adults, may result in a modest compromise in the supply of endogenous gluconeogenic substrates (e.g., amino acid from decreased muscle mass, independent of a specific enzyme or hormone defect), predisposing to the development of hypoglycemia and ketosis.

Ketotic hypoglycemia may constitute one end of a spectrum representing the normal distribution pattern of tolerance to fasting.[8,10] As the child has longer sleep time and longer time between feedings, this problem may become evident and result in the clinical presentation of a previously healthy child who is brought to the ED in the morning for vomiting, weakness, headache, or jitteriness. Evaluation will reveal hypoglycemia, ketosis, and an elevated anion gap. Children normally develop ketosis faster than adults, and ketosis is associated with a drop in plasma concentrations of glucose and insulin. This tendency of children is accentuated in children with ketotic hypoglycemia. In children with a typical presentation of ketotic hypoglycemia, it is useful to obtain additional blood testing to exclude other causes and definitively make the diagnosis. Children with ketotic hypoglycemia have normal venous lactate and pyruvate concentrations, but have hypoalaninemia prior to and during fasting or ketogenic diet challenge. The low plasma insulin and high free-fatty acid and ketone body concentration virtually exclude hyperinsulinemia as the underlying cause.[8] Children with ketogenic hypoglycemia are more prone to hypoglycemic episodes with intercurrent illness, especially if there is prolonged fasting. Parents are typically instructed to maintain frequent feedings of a high-protein, high-carbohydrate diet, and may check the child's urine for ketones.

Malabsorption/diarrhea. Hypoglycemia is a recognized complication of severe malnutrition and diarrhea.[11] Although morbidity and mortality from diarrhea is low in the United States, it remains a significant problem in less-developed countries. Hypoglycemia has been

Table 2. Causes of Hypoglycemia (Decreased Production / Availability of Glucose)

Low glycogen stores
- Small for gestational age, prematurity

Malnutrition / fasting
- Ketotic hypoglycemia

Malabsorption / diarrhea

Hormone abnormalities
- Growth hormone deficiency
- Cortisol deficiency
- Hypothyroidism

Inborn errors of metabolism
- Carbohydrate
  - *Glycogen storage diseases*
  - *Galactosemia*
  - *Hereditary fructose intolerance*
- Amino acid
  - *Tyrosinemia*
- Fatty acid
  - *Carnitine deficiency*
  - *Fatty acid transport deficiency*
  - *Beta oxidation deficiency*

Increased use of insulin
- Hyperinsulinism
  - *Beta cell hyperplasia*
  - *Insulin dysregulation*
- Infant of diabetic mother
- Beckwith-Weidemann Syndrome

Stressors associated with hypoglycemia
- Specific infections
  - *Shigella*
  - *Malaria*
  - *Meningococcus*
- Sepsis syndrome
- Miscellaneous
  - *Congenital heart disease*
  - *Burns*
  - *Reye Syndrome*
  - *Hepatitis*
  - *Shock*
  - *Tumors*
  - *Surgery*
  - *Alpha-1-antitrypsin deficiency*

Pharmacologic causes of hypoglycemia

Methanol, ethanol	Salicylates
Beta blockers	Oral hypoglycemics
Pentamidine	Insulin therapy

Adapted from: Kwon K, Tsai V. Metabolic emergencies. *Emerg Med Clin North Am* 2007;4:1041-1060.

shown to be a potentially fatal complication of infectious diarrhea in both well nourished and poorly nourished children.[12] A recent study has shown that hypoglycemia is not just a problem in developing countries but must be considered even in patients in the United States with diarrhea.[13] Approximately 10% of the children in this series with diarrhea had hypoglycemia. Although none of the children had significant clinical symptoms such as altered mental status or hypotension, it was noted that because of the signs and symptoms of dehydration, the signs and symptoms of hypoglycemia were masked. Also of clinical importance, children with hypoglycemia generally had a longer clinical course of vomiting and may remain symptomatic even after the vomiting has subsided. These children may remain in a fasting state, refusing feedings. Correction of the hypoglycemia often is associated with the child regaining the interest in oral feedings.

Hormone abnormalities. Hormone abnormalities that may cause hypoglycemia include growth hormone deficiency, cortisol deficiency, and hypothyroidism. The mechanism of hypoglycemia in these patients is incompletely understood but is characterized by blunted response to glucagon induced glycogenolysis as well as gluconeogenesis stimulated by hypoglycemia.

Inborn errors of metabolism. Inborn errors of metabolism associated with a specific enzyme deficiency are rare disorders but frequently associated with hypoglycemia. Disorders of carbohydrate metabolism leading to hypoglycemia involve deficiencies in hepatic enzymes used in the metabolism of glycogen, galactose, and fructose. The time course of hypoglycemia onset after the last meal may give a clue to the enzyme deficiency. For example, patients with galactosemia or heredity fructose intolerance may become hypoglycemic several hours after the ingestion of galactose or fructose. Galactosemia is the most common disorder of carbohydrate metabolism

and is identified through neonatal screening programs. Galactosemia results in the inability to utilize lactose found in human breast milk and cow's-milk-based formulas as an energy source and results in hypoglycemia. Conversely, patients with deficiencies of glycogenolysis or gluconeogenesis may become symptomatic between two hours and 20 hours after the last meal. In addition, glycogenolysis-deficient patients may have mild to moderate ketosis. Finally, infants and children who have disorders of gluconeogenesis develop ketotic hypoglycemia and lactic acidosis after fasting long enough to deplete glycogen stores and do not respond to glucagon administration.

Disorders of amino acid metabolism causing hypoglycemia result in organic acidemias with an anion gap and typically become clinically apparent during the neonatal period after a short well-appearing period. These children may be mistaken for having sepsis and may eventually develop failure to thrive and developmental delay. The hypoglycemia associated with amino acid disorders may be due to associated liver disease (tyrosemia) or carnitine deficiency. These disorders become most evident during stress episodes with metabolic decompensation including illness, trauma, or surgery.

Disorders of fatty acid metabolism and ketone body formation are rare causes of hypoglycemia. The most common disorders are carnitine deficiency, fatty acid transportation defects, and beta oxidation enzyme defects. During fasting or periods of stress, free fatty acids are mobilized from adipose tissue and utilized for energy by various organs. Free fatty acids undergo beta oxidation in the liver to produce ketone bodies such as beta-hydroxybutyric acid and acetoacetic acid. The stimulus for this process is decreased serum glucose and insulin suppression. The cause of hypoglycemia in these disorders has been postulated to involve decreased hepatic glucose production or the rapid utilization of glucose in the absence of ketone body availability. Classically, these disorders are not ketotic. Symptoms of these disorders include profound hypoglycemia during prolonged fasting or exercise or a low-carbohydrate, high-fat diet. Clinically, these patients also have persisting CNS disturbance and hypotonia in the face of corrected blood glucose.

Increased use of glucose. *Hyperinsulinism.* Hyperinsulinism (HI) is the most common cause of both transient and persistent hypoglycemia in infants and is usually due to beta cell hyperplasia or nesidioblastosis.[14] HI was first called "idiopathic hypoglycemia of infancy" and has had several other names. It is caused by dysregulated secretion of insulin. It consists of a group of clinically, genetically, and morphologically heterogeneous disorders. It occurs in 1 of 30,000 to 50,000 live births. It usually presents during the first few days of life but may occur later in infancy and childhood.[14] Premature infants with the disorder may present with severe hypoglycemia that is difficult to control. For unclear reasons, some of these children will have hypertrophic cardiomyopathy.[14] Newborns presenting with persistent and severe hypoglycemia, often with seizures, lethargy, and apnea, should have extra blood drawn at the time of treatment to evaluate for the critical diagnostic markers of disease entities. The ED physician may not be able to make the definitive diagnosis, and treatment should not be delayed, but obtaining critical blood samples during the acute phase is very useful in assisting with a timely, definitive diagnosis. Treatment is giving enough glucose to maintain euglycemia.

Infant of diabetic mother. While in utero, the infant of a diabetic mother is exposed to frequent episodes of hyperglycemia. Glucose crosses the placenta, but insulin does not. This stimulates the fetus to produce insulin. When the maternal blood supply and glucose is stopped at delivery, these infants, in addition to facing the regular problems of maintaining glucose levels, are faced with high levels of insulin. The persistence of elevated levels of insulin can cause prolonged hypoglycemia.[15]

Beckwith-Weidemann Syndrome. Patients with Beckwith-Weidemann syndrome have macrosomia, macroglossia, hemi hypertrophy, transverse creases of the ear lobes, hypoglycemia, and predisposition to childhood tumors. Up to 50% of the patients will have hypoglycemia that can be transient and mild to persistent and severe. The cause is unclear and spontaneous remission often occurs.[13] Otherwise, they may be controlled by medical therapy or partial pancreatectomy.

Stressors. Children, especially toddlers and infants, have limited glycogen stores and rapidly develop hypoglycemia during periods of stress. Severe hypoglycemia with subsequent central nervous system damage may occur if hypoglycemia is not identified and treated.

Specific infections. Although not well studied, there are reports of the association of hypoglycemia with severe infection and sepsis. The association of hypoglycemia in patients with malaria and shigellosis was reported early on, but the association with other infections was not reported until more recently, when Halamek and colleagues described the presence of hypoglycemia in three children with severe meningococcemia and adrenal hemorrhage.[16] Other authors have emphasized the importance of glucose testing in septic patients.[17] A very recent study showed a prevalence of hypoglycemia in 8.6% of patients with bacteremic pneumococcal infection.[18] A higher morbidity has also been substantiated in those patients presenting with hypoglycemia. The mechanism of hypoglycemia and severe sepsis is not well understood. In humans, endotoxin plays a crucial role in the pathogenesis of gram-negative shock, which leads to impairment of glucose homeostasis and lethal septic shock. A recently published study showed a greater frequency of hypoglycemia in previously malnourished children, hypothermic patients, and those with *Shigella*

Table 3. Etiology of Hypoglycemia in Neonates

Transient neonatal hypoglycemia

- *Day 1 of life:* Developmental immaturity of fasting adaptation (mechanism: impaired ketogenesis and gluconeogenesis)
- *First two days of life:* Transient hypoglycemia due to maternal factors
  - Maternal diabetes (mechanism: hyperinsulinism)
  - Intravenous glucose administration during labor and delivery (mechanism: hyperinsulinism)
  - Medications: oral hypoglycemics, terbutaline, propranolol (mechanism: hyperinsulinism)

Prolonged neonatal hypoglycemia

- Perinatal stress-induced hyperinsulinism (low birth weight, birth asphyxia, maternal toxemia or pre eclampsia, prematurity)
- Beckwith-Weidemann syndrome
- Hypopituitarism

Permanent neonatal hypoglycemia (caused by congenital endocrine or metabolic disorders)

- Congenital hyperinsulinism
  - ATP-sensitive potassium channel hyperinsulinism
  - Glutamate dehydrogenase hyperinsulinism
  - Glucokinase hyperinsulinism
  - Short-chain 3-hydroxyacyl-CoA dehydrogenase hyperinsulinism
  - Congenital disorders of glycosylation
- Counter regulatory hormone deficiency
  - Hypopituitarism
  - Adrenal insufficiency
- Gluconeogenesis or glycogenolysis enzyme defects
- Fatty acid oxidation disorders

Adapted from Jan I, et al. Hypoglycemia associated with bacteremic pneumococcal infections. *Int J Infect Dis* 2009:13:570-576

as a cause of their diarrhea.[11] Also, hypoglycemia was more common in more severe infections. Morbidity was increased in patients with bacteremia and hypoglycemia. In their analysis, bacteremic children with clinical sepsis were four times more likely to be hypoglycemic. The authors recommended that a rapid bedside glucose test should be considered as an inexpensive way to help in management decisions for ill children with diarrheal illness and bacteremia.

Certain more unusual infections have been associated with hypoglycemia. Although not common in the United States, malaria is the most important parasitic disease in the world. It kills more people annually than any other disease except tuberculosis and HIV/AIDS. Annually, there are more than 515 million clinical cases of malaria worldwide. Many pediatric cases present with impaired consciousness, repeated seizures, severe anemia, respiratory distress, or shock. Deaths are particularly common with children who present with impaired consciousness, shock, or hypoglycemia.[19] While most cases will occur in endemic areas, patients may return to the United States after a visit to one of these areas. Children with severe malaria present with hypoglycemia in 10% of cases. In patients with cerebral malaria, hypoglycemia is more common and occurs in up to 30% of cases.[19] The clinical presentation of cerebral malaria may be mild with a one- to four-day history of fever, listlessness, anorexia, irritability, vomiting, and cough before seizures and coma appear.[20] As with several other illnesses presenting with hypoglycemia, the treatment of severe malaria requires attention to multiple other problems. Hypoglycemia is associated with increased mortality and brain injury.

Sepsis. Sepsis is an uncommon but very serious condition that can occur at any time from the newborn period to adulthood. The incidence of sepsis is estimated to be 0.56/1,000 children. The highest incidence is in infancy at 5.6/1,000 children. The overall mortality is estimated at over 10%. The systemic inflammatory response syndrome (SIRS) for children requires presentation with hypo- or hyperthermia or elevated white blood cell count in the presence of one of the following findings: abnormal temperature, leukocytosis, tachypnea, or tachycardia. Sepsis is defined as proven or suspected infection with the diagnosis of SIRS. Patients most at risk include those not immunized and those with any form of immunocompromise. The presentation of sepsis varies by age and underlying etiology. The clinical presentation of sepsis is especially variable in the newborn period. The realities of current medical practice make it very

clear that the treatment of sepsis needs to be aggressively initiated as soon as suspected. Hypoglycemia must be anticipated in the septicemic child. Children may present with sepsis in community hospitals and subsequently require transfer, or a bed in the intensive care unit (ICU) may be delayed by hours or longer. Consequently, ED physicians may need to provide ongoing critical care while awaiting transfer to an inpatient service. ED physicians must aggressively manage early sepsis and recognize concomitant issues such as hypoglycemia. Early, aggressive treatment may result in a significant decrease in mortality and morbidity.

Regardless of the age or the cause, immediate attention to many details is required to optimize the treatment of patients with sepsis. Early use of "goal-directed therapy" (EGDT) is recommended to organize an approach to sepsis. EGDT was formulated in 2001 at Henry Ford Hospital, and much like the algorithms in PALS, ACLS, and ATLS, the standardized approach to sepsis is useful. Of the many variables included in the management of sepsis are early antibiotics, corticosteroids, glucose control, and consideration of recombinant human activated protein C lung protective strategies. Much as the use of pulse oximetry in ED patients as a vital sign has increased the diagnosis of hypoxia, blood glucose levels obtained routinely on all sick children will alert the physician to elevated or low glucose levels. A recently published study used the monitoring of glucose levels as one of the parameters to evaluate the success of treatment. The goal in this study was to keep the glucose level above the lower limit of normal but below 150 mg/dL.[21] Hyperglycemia is considered to be an early sign of sepsis, and management with insulin has been recommended.[21] With aggressive insulin management of hyperglycemia, hypoglycemia can result.[22] Hypoglycemia may also be a late sign of sepsis. Whether the glucose level is elevated or low, the management and monitoring of the glucose level aids in the management of sepsis and the response to interventions.

Miscellaneous. Children with heart disease are predisposed to hypoglycemia, thought to be due to under-perfusion of the liver. Similarly, numerous disease states increase the body glucose requirement (shock, burns, and tumors) or affect the liver's ability to produce glucose (Reye's syndrome, hepatitis, alpha-1-antitrypsin deficiency). Finally, surgery may result in hypoglycemia due to preoperative fasting without carbohydrate loading, intra-operative stress, and intra-operative medications.

Acute metabolic encephalopathy is a relatively common problem and may be the initial presentation or a complication of a metabolic disorder. A detailed history, examination and investigations performed during the acute illness (blood sugar, blood gases, plasma ammonia, blood lactate, plasma ketones, plasma amino acids, liver function tests, and urinary organic acids) should identify those patients in whom a metabolic disorder is likely. The differential diagnosis of acute metabolic encephalopathy can be divided into three broad categories: endocrine, metabolic, and hepatic.[23] *(See Table 4.)*

Reye's syndrome was first described in 1963. An association with aspirin use was determined and since aspirin use has been discouraged the prevalence of Reye's syndrome has reduced greatly. Despite this, there are still a few cases of Reye's syndrome.[24,25] It is an acute encephalopathy and hepatopathy. There is often an antecedent viral infection, frequently with influenza and varicella. With the recent pandemic of H1N1 influenza, the ED physician must be watchful for any cases of Reye's syndrome. To make the diagnosis of Reye's syndrome, it is necessary to exclude metabolic disorders that may also present with similar symptoms.[25] The syndrome had a 42% mortality and another 11% residual neurologic damage. One of the most challenging aspects in the management of the child with Reye's syndrome is maintenance of normal glucose levels. Hypoglycemia is profound and persistent, often requiring a continuous infusion of 10% dextrose in addition to the other supportive care.

A recent discussion of three cases of infant botulism pointed out the difficulty in making this diagnosis. One patient presented with severe acidosis and hypoglycemia, suggesting an underlying metabolic disorder.[26]

Table 4. Causes of Hypoglycemia and Acute Encephalopathy

Endocrine

- Hypopituitary coma

Metabolic

- Organic acidemias
  - Maple syrup urine disease
  - Methylmalonic acidemia
  - Acetoacetyl-CoA thiolase deficiency
  - Propionic and isovaleric acidemias
- Fat oxidation defects
  - Medium, long-chain, and multiple acyl-CoA dehydrogenase deficiencies
  - 3-Hydroxy-3-methylglutaryl-CoA lyase deficiency
  - Others not fully characterized

Drugs and toxins

- Alcohol
- Oral hypoglycemic agents
- Salicylates

Hepatic

- Fulminant liver failure
- Reye's syndrome

Adapted from: Surtees R, et al. Acute metabolic encephalopathy: A review of causes, mechanisms and treatment. *J Inher Metab Dis* 1989:12:42-54

Exogenous Pharmacologic Causes of Hypoglycemia

Ingestions associated with hypoglycemia include methanol, salicylates, beta blockers, oral hypoglycemics, and pentamidine.

Ethanol is found in alcoholic beverages and many other products (e.g., mouthwash, cold preparations) to which a child may have access. Ethanol ingestion results in hypoglycemia by inhibiting gluconeogenesis and decreasing uptake of gluconeogenesis substrates by the liver. The resulting hypoglycemia may be severe leading to seizures and death. The younger the child, the more at risk for hypoglycemia. The classic triad of acute ethanol overdose in children is hypothermia, hypoglycemia, and coma. These symptoms may occur with ethanol levels of 50–100 mg/dL. Development of hypoglycemia does not seem to be directly related to the seriousness of the ingestion, and may be accompanied by metabolic acidosis. Occasionally, these patients may present with seizures.[28] Ethanol ingestion should be suspected in the presence of metabolic acidosis with an anion gap, elevated lactate, and blunted response to glucagon.

Salicylates. Similarly, salicylates should be considered in the patient with hypoglycemia, metabolic acidosis, increased anion gap, delirium, and hyperventilation. Although there has been a great reduction in the number of salicylate poisonings, a timely diagnosis requires consideration of the disease process. Nausea, vomiting, fever, tinnitus, and hyperventilation occur with significant ingestions. Most patients will have respiratory alkalosis and metabolic acidosis. Salicylate affects both central and peripheral glucose homeostasis. Animal studies have shown that toxic doses of salicylate produce a profound decrease in brain glucose concentration despite normal serum glucose levels.[29]

Methyl salicylate ingestion can result in serious toxicity in the child. It can be found in numerous products, but the most potent form is oil of wintergreen, which is 98% methyl salicylate. Methyl salicylate can be found in some Asian herbal remedies. Deaths have been reported with as little as a teaspoon ingestion.[30]

Beta blockers cause hypoglycemia by blunting the typical autonomic response to hypoglycemia, including glycogenolysis, gluconeogenesis, inhibition of glucose uptake by tissues, and inhibition of insulin receptor secretion. Although there has been an increase in the number of beta blocker ingestions over the last few years, there have been no fatalities in toddlers.[17] Still, there is reason to be cautious with the ingestion of a large amount of lipophilic beta blocker (propanolol) and consider monitoring of blood glucose levels. Earlier concerns that one or two tablets may be fatal have not been substantiated.

Sulfonylureas are a class of hypoglycemic drugs used in the management of adult-onset diabetes. Hypoglycemia is seen quickly after ingestion and the duration is usually less than 24 hours. For sulfonylureas, the onset can be delayed and the drug effects can be greatly prolonged. Cases have been reported in which symptoms have persisted for 3-4 days.[31] Even a single dose of a sulfonylurea in a child can result in life-threatening hypoglycemia.

Other. Pentamidine is an antimicrobial agent that is used in the treatment and prevention of *Pneumocystis pneumonia,* usually in patients with HIV. It is also used as a prophylactic antibiotic for children undergoing chemotherapy for leukemia. The drug is cytotoxic to the pancreatic islet cells, resulting in inappropriately high levels of insulin. Accidental ingestion can cause hypoglycemia.

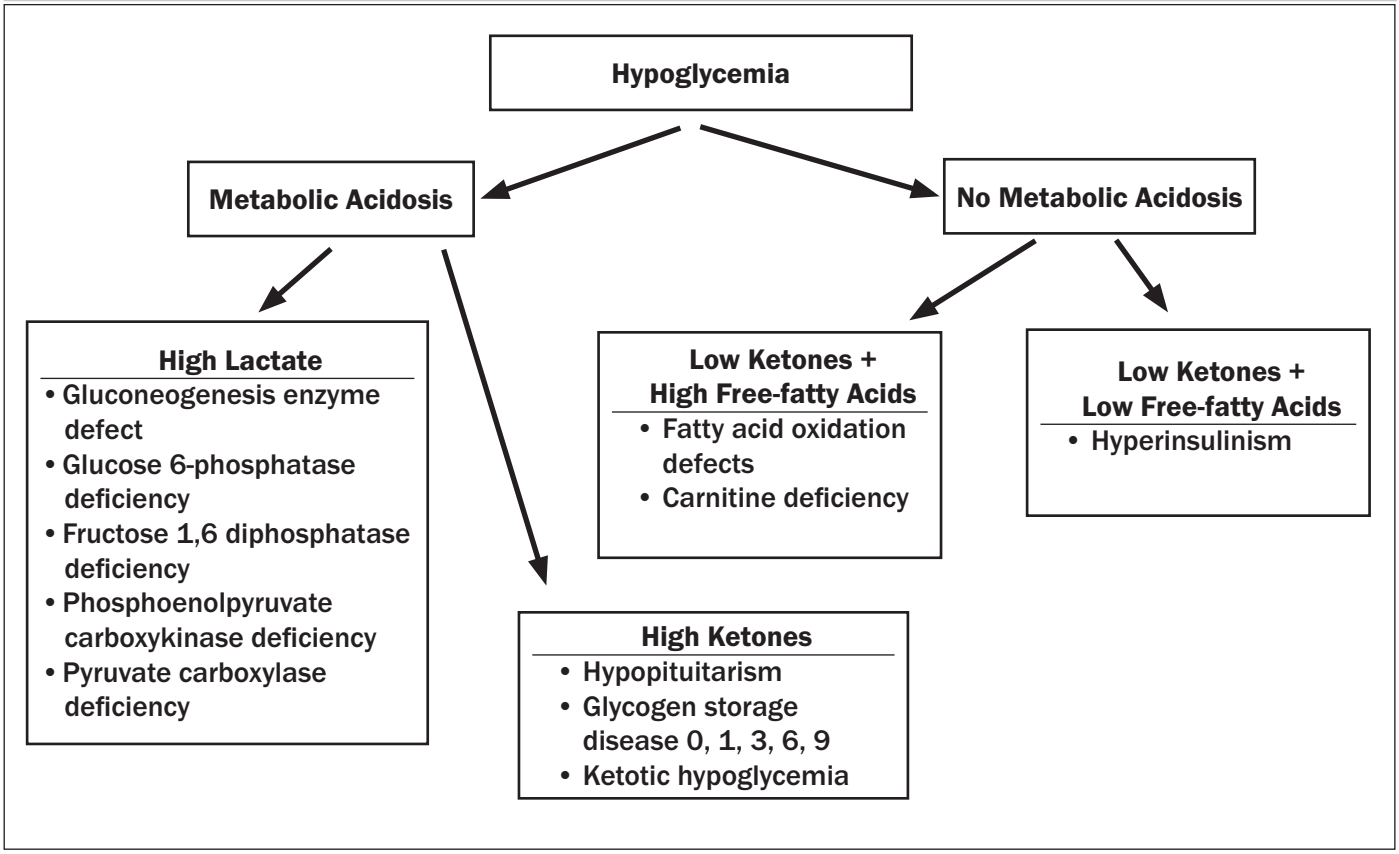
A frequent cause of hypoglycemia is a relative or absolute excess of insulin in the type 1 diabetic child.

Children with diabetes mellitus type 1 may develop hypoglycemia due to error in administration or preparation of insulin, failure to ingest proper quantities of carbohydrates, and late-evening exercise — which may be impossible to predict in the young child. Type I diabetic children are at high risk of hypoglycemia due to the possibility of undetected nocturnal hypoglycemia and the blunted gluconeogenesis response to glucagon and epinephrine in recurrent hypoglycemics. Typically, these disorders are nonketotic. As diabetes is a complete subject unto itself, it will not be discussed further. However, one must always be aware that diabetics can suffer from some of the other disease conditions that cause hypoglycemia, and these causes should also be considered.

Hypoglycemia in the Newborn

Hypoglycemia is one of the most commonly encountered metabolic problems in the newborn period. At no other time in life is management of hypoglycemia more important than in the newborn period. Hypoglycemia can cause neonatal encephalopathy, resulting in permanent brain damage. Several of the disorders may be present at birth or in the perinatal period. Typically these are diagnosed in the newborn nursery, but with early discharges and home deliveries, these patients may also be seen in the ED. Hyperinsulinemic hypoglycemia (HH) is a major cause of recurrent and persistent hypoglycemia.[4] Delay in initial diagnosis and subsequent treatment may lead to severe brain injury and mental retardation. Congenital forms may be unresponsive to medical therapy. HH may be secondary to other risk factors, including intrauterine growth retardation, infants with perinatal asphyxia, infants of diabetic mothers (both gestational and insulin-dependent), and in some infants with Beckwith-Weidemann syndrome. In most of these conditions, HH is transitory and resolves spontaneously.[4] Thus with appropriate management of their hypoglycemia these patients can have normal growth and development. Hypoglycemia should be suspected

Figure 1. Approach to Hypoglycemia



when any newborn with any potential problem comes to the ED.

Diagnosis of Hypoglycemia

As previously mentioned, there is no absolute acceptance of what blood glucose level defines hypoglycemia. Regardless, the rapid identification and treatment of hypoglycemia is required for management of the seriously ill child. Even the time taken to receive a laboratory value is time wasted. Thus, it is common practice to obtain bedside or point of care glucose determination. This is often done with AccuChek Inform (Roche Diagnostics, Mannheim, Germany). AccuChek measurements are done on whole blood, and laboratory values are done on serum. This in itself leads to the need for a correction factor. Whole blood glucose is measured lower than serum and should be multiplied by 1.11 to estimate serum values. The accuracy of AccuChek has been shown in various studies.[9] Use in the ICU has been questioned because of the need for very tight glucose control in septic patients, but for the ED, it is accurate.[32]

At the time of ED presentation during the acute hypoglycemic episode, it is beneficial to obtain critical blood samples to determine a definitive etiology of the episode. Since the blood is drawn during the period of acute hypoglycemia, the critical sample reflects the physiologic response—or lack of response—of the child. *(See Figure 1.)* Critical blood sampling tubes include: red top tube for insulin, C-peptide, cortisol, and carnitine; grey top tube for beta hydroxybutyrate, free fatty acids, lactate; green top tube for growth hormone, ammonia, aminoacids; lavender top tube for glucagon; separate containers for quantitative urine sampling of organic acids, aminoacids, and reducing substances.

Treatment

Regardless of the underlying cause of hypoglycemia, the serum glucose must be restored to normal range. This is especially true in the symptomatic child and the very young child. Ideally, an IV has been established and 0.5–1.0 grams per kilogram (2–4 mL D25W or 5–10 mL per kilogram D10W). This can be given by an intraosseous line, as well. The more concentrated dextrose solutions must be used with caution, because infiltration can cause significant tissue destruction. In the child with a small-gauge needle, D10W may be used. Monitoring of the response to the dextrose infusion should be done with bedside testing. As hypoglycemia is rarely an isolated problem, the underlying cause must be determined and additional therapies instituted as indicated. Some disorders may require additional dextrose or even continuous dextrose infusion.

Summary

The skills of the ED physician are tested when presented with a severely ill child. Treatment and stabilization are the first priority, but thought must also be given to the reason for the critical presentation. Sometimes the cause is easy to determine, as with trauma. Many times the presentation is very confusing. It is then that the ED physician must use a systematic approach to the patient's management. We are all familiar with the ABCs of resuscitation. This remains the best initial approach to these patients. The patient's vital signs are also extremely important and must be addressed as soon as any abnormality is identified. Hypoglycemia by itself can present as a child in dire distress. It can also be a symptom in association with many other abnormalities, all of which must be addressed. The ABCs are designed to deal with the most serious problems the patient has in order of severity, and the likelihood that if untreated, the patient may die or have a poor outcome. Because hypoglycemia is very prevalent in seriously ill children and because if untreated can lead to death or neurological impairment, in critical patients, blood glucose levels should be considered as important as the vital signs.

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CME Questions

41. The classic triad of clinical manifestations of hypoglycemia does NOT include:

    A. Altered mental status
    B. Serum glucose < 40mg/dL
    C. Sepsis
    D. Relief of symptoms with glucose administration

42. Which of the following statement(s) is/are true regarding hypoglycemia in young children?

    A. Neurological damage may include hemorrhage in the posterior white matter areas.
    B. In the very young infant, failure to recognize and treat hypoglycemia may lead to permanent neurological sequelae.
    C. Common findings in children presenting to the ED — e.g., tachycardia, tachypnea, vomiting, weakness, nervousness, and diaphoresis — may be traced to a rapid decline in glucose level.
    D. Adrenergic symptoms usually are seen earlier in the process than neuroglycopenic symptoms.
    E. All of the above are true

43. Ketotic hypoglycemia is a rare cause of hypoglycemia in children from 1 year to 5 years of age.

    A. True
    B. False

44. Hyperinsulism:

    A. occurs in 1 in 30,000 to 50,000 live births.
    B. is rarely the cause of transient and persistent hypoglycemia in infants.
    C. exclusively presents during the first few days of life.
    D. is always treated by pancreatectomy.

45. The classic triad of acute ethanol overdose in children includes:

    A. Hypothermia, hypoglycemia, hyperammonemia
    B. Hypothermia, hypoglycemia, coma
    C. Hypothermia, hypoglycemia, respiratory acidosis
    D. Hypothermia, hypoglycemia, fever

46. A child who presents to the ED with nausea, vomiting, fever, tinnitus, and hyperventilation, and whose history indicates exposure to Asian herbal remedies, should raise suspicion of:

    A. Methyl salicylate (oil of wintergreen) ingestion
    B. Ethanol ingestion
    C. Beta-blocker ingestion
    D. Pentamidine ingestion

47. A frequent cause of hypoglycemia in the type 1 diabetic child is a relative or absolute excess of insulin.

    A. True
    B. False

48. In the developing neonate, the agreed-upon minimum serum glucose level indicating hypoglycemia is:

    A. < 50 mg/dL
    B. < 60 mg/dL
    C. 65 mg/dL
    D. There is no universally agreed-upon minimum

49. Which of the following is NOT an adrenergic symptom of hypoglycemia?

    A. Tachypnea
    B. Weakness
    C. Hypotonia
    D. Nervousness

50. A non-immunized, immunocompromised, hypoglycemic child should raise suspicion for:

    A. Reye's syndrome
    B. Sepsis
    C. Beckwith-Weidemann Syndrome
    D. Hyperinsulism

Answers: 41. C; 42. E, 43. B, 44. A, 45. B, 46. A, 47. A, 48. D, 49. C, 50. B

Pediatric Emergency Medicine Reports

CME Objectives

Upon completion of this educational activity, participants should be able to:

1. recognize specific conditions in pediatric patients presenting to the emergency department;
2. describe the epidemiology, etiology, pathophysiology, historical and examination findings associated with conditions in pediatric patients presenting to the emergency department;
3. formulate a differential diagnosis and perform necessary diagnostic tests;
4. apply up-to-date therapeutic techniques to address conditions discussed in the publication;
5. discuss any discharge or follow-up instructions with patients.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a credit letter. When your evaluation is received, a credit letter will be mailed to you.

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Pediatric Emergency Medicine Reports

2010 Reader Survey

In an effort to learn more about the professionals who read *Pediatric Emergency Medicine Reports* (PEMR), we are conducting this reader survey. The results will be used to enhance the content and format of *PEMR*.

Instructions: Fill in the appropriate answers. Please write in answers to the open-ended questions in the space provided. Return the questionnaire in the enclosed postage-paid envelope by **July 1, 2010.**

1. Are the articles in *Pediatric Emergency Medicine Reports* written about issues of importance and concern to you?

- O A. Always
- O B. Most of the time
- O C. Some of the time
- O D. Rarely
- O E. Never

2. How would you describe your satisfaction with your subscription to *PEMR*?

- O A. Very satisfied
- O B. Somewhat satisfied
- O C. Somewhat dissatisfied
- O D. Very dissatisfied

Questions 3-14 ask about articles appearing in *Pediatric Emergency Medicine Reports*. Please mark your answers in the following manner:

A. very useful B. fairly useful C. not very useful D. not useful at all

3. Asthma (June 2009)	O A	O B	O C	O D
4. Methicillin-resistant staph (July 2009)	O A	O B	O C	O D
5. Foreign body removal/Tools of the trade (Aug. 2009)	O A	O B	O C	O D
6. Hand injuries, part I (Sept. 2009)	O A	O B	O C	O D
7. Hand injuries, part II (Oct. 2009)	O A	O B	O C	O D
8. Eye injuries (Nov. 2009)	O A	O B	O C	O D
9. Sickle cell complications (Dec. 2009)	O A	O B	O C	O D
10. Petechial rashes (Jan. 2010)	O A	O B	O C	O D
11. Syncope (Feb. 2010)	O A	O B	O C	O D
12. H1N1 update (March 2010)	O A	O B	O C	O D
13. Pediatric abdominal emergencies (April 2010)	O A	O B	O C	O D
14. Pediatric thoracic injuries (May 2010)	O A	O B	O C	O D

15. Do you plan to renew your subscription to *PEMR*? O A. yes O B. no

If no, why? _____

16. What is your title?

- O A. Practicing emergency medicine physician
- O B. Physician assistant
- O C. Professor/academician
- O D. Emergency medicine manager/director
- O E. Other _____

17. On average, how much time do you spend reading each issue of *PEMR*?

- O A. 21-30 minutes
- O B. 31-59 minutes
- O C. 1-2 hours
- O D. 2-3 hours
- O E. More than 3 hours

18. On average, how many people read your copy of *PEMR*?

- O A. 1
- O B. 2-3
- O C. 4-6
- O D. 7-9
- O E. 10 or more

19. How large is your hospital?

- O A. fewer than 100 beds
- O B. 100-200 beds
- O C. 201-300 beds
- O D. 301-500 beds
- O E. more than 500 beds

20. How would you rate the overall coverage of topics in *PEMR*?

- O A. very useful
- O B. fairly useful
- O C. not very useful
- O D. not useful at all

21. Would you be interested in earning continuing education credits with this publication?

- O A. Yes
- O B. No

Please rate your level of satisfaction with the following items.
A. excellent B. good C. fair D. poor

22. Quality of newsletter	○ A	○ B	○ C	○ D
23. Article selections	○ A	○ B	○ C	○ D
24. Timeliness	○ A	○ B	○ C	○ D
25. Quality of *Trauma Reports*	○ A	○ B	○ C	○ D
26. Length of newsletter	○ A	○ B	○ C	○ D
27. Overall value	○ A	○ B	○ C	○ D
28. Customer service	○ A	○ B	○ C	○ D

29. To which other publications or information sources about pediatric emergency medicine do you subscribe?

_____

_____

_____

30. Which publication or information source do you find most useful, and why? _____

_____

31. Please list the top three challenges you face in your job today.

_____

_____

_____

32. What do you like *most* about *Pediatric Emergency Medicine Reports*?

_____

_____

_____

33. What do you like *least* about *Pediatric Emergency Medicine Reports*?

_____

_____

_____

34. What specific topics would you like to see addressed in *Pediatric Emergency Medicine Reports*?

_____

_____

_____

35. Has reading *Pediatric Emergency Medicine Reports* changed your clinical practice? If yes, how? _____

_____

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Health Care Reform Update

What Health Care Reform Means to You — A supplement to *Pediatric Emergency Medicine Reports*

Increased provider access tops list of what clinicians will like about HC bill

Changes will take a few years

HEALTH CARE CLINICIANS AND ORGANIZATIONS LIKELY will find that the new health care reform bill's positive features outweigh its drawbacks, experts say.

The Patient Protection and Affordable Health Care Act, signed into law on March 23, 2010, by President Barack Obama, provides a series of changes to take place to health care insurance coverage, Medicare, Medicaid, prescription drugs, quality improvement initiatives, medical malpractice, and other items. These are to be implemented from 2010 to 2014.

"The thing that is so big is the coverage for tens of millions of people who don't have health insurance now," says **Cecil Wilson**, MD, an internist in Winter Park, FL, and the president-elect of the American Medical Association in Chicago, IL.

People no longer will have to worry about losing health care coverage for existing diseases if they lose their jobs, and increasing numbers of people will have access to preventive care, primary care, and disease management, Wilson adds.

"Those are the big things that make this such a sea change in my opinion," he says. "For physicians, this is good because they won't have to worry about their patients' insurance being cut off, and thus putting their patients at risk."

Hospitals will find that significantly more patients will have health care coverage, resulting in a decline in uncompensated care, says **Caroline Steinberg**, vice president for trends analysis for the American Hospital Association of Washington, DC.

"We also would expect that demand for care from formerly uninsured patients will increase," Steinberg says. "Hopefully, we'll see some increases in primary care so by the time they hit the hospital they won't have some of the same kinds of problems they've had before."

The new bill provides billions of dollars in funding for clinics that provide primary care to uninsured, indigent, and immigrant patients. In 2014, it also expands Medicaid to all non-Medicare eligible individuals who have incomes up to 133% of the federal poverty level. These initiatives could help send more people to primary care services and keep them from using the emergency room for non-emergency care, Steinberg adds.

"We may [identify] more people with conditions that require specialty care because once people have access to coverage they tend to use more health care across all levels of the system," Steinberg says. "So that could go either way."

Plus, hospitals should expect the next few years to continue to be rough fiscally since most of the more significant provisions in the bill will not be fully implemented until 2014.

"Our hospitals are telling us that uncompensated care is going up because of job losses and loss of insurance, and these people show up in hospitals," Steinberg says.

There won't be much improvement in the immediate future until the economy recovers and the government provides more funding for Medicaid, she notes.

More oncology patients will have access to care, as a result of the bill's prohibition of lifetime limits on the dollar value of coverage, which begins Jan. 1, 2014. There is a temporary national high-risk pool to provide health care coverage to people with pre-existing medical conditions, which will be in place between June 2010 and 2014.

"Many cancer patients who need repeated courses of treatment can easily exceed their caps and find themselves unable to afford needed treatment and medication," says **Allen S. Lichter**, MD, chief executive officer of the American Society of Clinical Oncology (ASCO), in a statement issued after the bill was signed.

By this fall, insurers will not be able to exclude children with pre-existing conditions from being covered by their family policy, and this also is a positive move, Lichter says.

The bill's focus on prevention and wellness will benefit infectious disease and public health initiatives.

"There are a few things in the bill that we're pleased to see stay in the final version," says **Michael Ochs**, government relations associate with the Infectious Diseases Society of America (IDSA) in Arlington, VA.

The bill's emphasis on wellness and disease prevention with billions of additional federal dollars for these is one example, Ochs says.

The bill's impact on physician and other provider payments is a more mixed bag, however. *(See story on*

physician payments, below.)

"There's a 10% incentive pay for primary care and general surgery," says **Jason A. Scull**, program officer for clinical affairs at IDSA.

"They're focusing on primary care in a lot of these new innovative payment models, but I think primary care does need to be incentivized," Scull says.

But the drawback is that cognitive specialists, like infectious disease specialists, cardiologists, and neurologists, could be shortchanged as the pie is cut differently, but not expanded.

"There will be unintended consequences," Scull notes. "Already last year the Centers for Medicare & Medicaid Services [CMS] eliminated payments for consultation codes that cognitive specialties use to give them money to distribute elsewhere in the fee schedule and to send more to primary care physicians."

This redistribution of payments might result in fewer medical students choosing to spend extra years of training beyond their general internal medicine residency, he adds.

While the sweeping health care reform provides some specifics on how changes will occur in the industry, no one knows precisely how things will change until the regulatory details emerge, the experts say.

"There are a lot of moving pieces to this," Scull says. "I think it's anybody's guess to where all of this ends up." ■

Doctors will be more closely scrutinized with bill's provisions

Experts talk about bill's negatives

PAY ATTENTION TO THE NEW HEALTH CARE BILL'S REGulatory details, experts warn providers.

There are some items in the sweeping legislation that could result in more documentation, work, and risk for physicians and other providers.

For instance, the new bill makes it clear that the government wants doctors to be doctors and not own hospitals, says **LaDale K. George**, JD, a partner with Neal, Gerber, Eisenberg in Chicago, IL.

The bill puts a moratorium on any physician-owned hospitals in non-rural settings that were not Medicare providers as of December 2010.

"The new law says that the practice of physicians owning hospitals no longer is allowed," he explains. "If a physician owns or has a financial interest in a hospital and refers patients to that hospital then every service the patient receives at the hospital is a Stark violation of $25,000 per incident."

Also, the anti-kickback law has been changed by the new bill.

"The way the new act changes it is that it appears to eliminate the need to have actual knowledge or specific intent to violate the statute," George says. "It moves in the direction of where the Stark law is where if you do not meet the safe harbors in which providers can refer to one another and engage in commercial practices together then you will be viewed as being guilty."

From physicians' perspectives, some of the other requirements will be more onerous, particularly as far as documentation and accounting are concerned.

For instance, the bill's Physician Payment Sunshine Provision requires physicians to disclose every payment they receive from pharmaceutical and biotech companies in excess of $100, and this includes drug samples. This could prove to be an accounting problem for physician investigators and others.

This likely will be a headache to physicians, who will have to keep track of every sample they receive and every payment that flows through to them for research, George says.

The new health care bill also appears to give physicians incentives and/or penalties depending on their compliance with reporting data as part of the physician quality reporting initiative (PQRI), which was established with the 2006 Tax Relief and Health Care Act.

"What's clear is that Congress is moving into the direction of mandating physicians to participate in PQRI and also moving in the direction of mandating physician resource use reporting," says **Jason A. Scull**, program officer for clinical affairs at the Infectious Diseases Society of America.

"These are somehow merged into a value modifier that also will adjust payment based on the quality of care they provide," Scull says.

About one of six eligible physicians now makes the reports, and about half of these receive incentive payments, he adds. ■

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Rapid Access Management Guidelines

Pediatric Emergency Medicine Reports

The Practical Journal of Pediatric Emergency Medicine

Hypoglycemia in Infants and Children

Clinical Presentation of Hypoglycemia

Whipple's Triad
- Altered mental status
- Serum glucose less than 40 mg/dL
- Relief with glucose administration

Adrenergic Symptoms
- Tachypnea
- Tachycardia
- Vomiting
- Weakness
- Nervousness
- Diaphoresis

Neuroglycopenic Symptoms
- Headache
- Irritability
- Psychotic behavior
- Lethargy
- Altered mental status
- Seizures
- Coma

Neonatal Hypoglycemia
- Hypotonia
- Hypothermia
- Feeding difficulties
- Jitteriness
- Hyperreflexia

Etiology of Hypoglycemia in Neonates

Transient neonatal hypoglycemia
- *Day 1 of life:* Developmental immaturity of fasting adaptation (mechanism: impaired ketogenesis and gluconeogenesis)
- *First two days of life:* Transient hypoglycemia due to maternal factors
  - ○ Maternal diabetes (mechanism: hyperinsulinism)
  - ○ Intravenous glucose administration during labor and delivery (mechanism: hyperinsulinism)
  - ○ Medications: oral hypoglycemics, terbutaline, propranolol (mechanism: hyperinsulinism)

Prolonged neonatal hypoglycemia
- Perinatal stress-induced hyperinsulinism (low birth weight, birth asphyxia, maternal toxemia or pre eclampsia, prematurity)
- Beckwith-Weidemann syndrome
- Hypopituitarism

Permanent neonatal hypoglycemia (caused by congenital endocrine or metabolic disorders)
- Congenital hyperinsulinism
  - ○ ATP-sensitive potassium channel hyperinsulinism
  - ○ Glutamate dehydrogenase hyperinsulinism
  - ○ Glucokinase hyperinsulinism
  - ○ Short-chain 3-hydroxyacyl-CoA dehydrogenase hyperinsulinism
  - ○ Congenital disorders of glycosylation
- Counter regulatory hormone deficiency
  - ○ Hypopituitarism
  - ○ Adrenal insufficiency
- Gluconeogenesis or glycogenolysis enzyme defects
- Fatty acid oxidation disorders
-

Adapted from Jan I, et al. Hypoglycemia associated with bacteremic pneumococcal infections. *Int J Infect Dis* 2009:13:570-576

Causes of Hypoglycemia (Decreased Production / Availability of Glucose)

Low glycogen stores
- Small for gestational age, prematurity

Malnutrition / fasting
- Ketotic hypoglycemia

Malabsorption / diarrhea

Hormone abnormalities
- Growth hormone deficiency
- Cortisol deficiency
- Hypothyroidism

Inborn errors of metabolism
- Carbohydrate
  - *Glycogen storage diseases*
  - *Galactosemia*
  - *Hereditary fructose intolerance*
- Amino acid
  - *Tyrosinemia*
- Fatty acid
  - *Carnitine deficiency*
  - *Fatty acid transport deficiency*
  - *Beta oxidation deficiency*

Increased use of insulin
- Hyperinsulinism
  - *Beta cell hyperplasia*
  - *Insulin dysregulation*
- Infant of diabetic mother
- Beckwith-Weidemann Syndrome

Stressors associated with hypoglycemia
- Specific infections
  - *Shigella*
  - *Malaria*
  - *Meningococcus*
- Sepsis syndrome
- Miscellaneous
  - *Congenital heart disease*
  - *Burns*
  - *Reye Syndrome*
  - *Hepatitis*
  - *Shock*
  - *Tumors*
  - *Surgery*
  - *Alpha-1-antitrypsin deficiency*

Pharmacologic causes of hypoglycemia

Methanol, ethanol	Salicylates
Beta blockers	Oral hypoglycemics
Pentamidine	Insulin therapy

Adapted from: Kwon K, Tsai V. Metabolic emergencies. *Emerg Med Clin North Am* 2007;4:1041-1060.

Causes of Hypoglycemia and Acute Encephalopathy

Endocrine

- Hypopituitary coma

Metabolic

- Organic acidemias
  - Maple syrup urine disease
  - Methylmalonic acidemia
  - Acetoacetyl-CoA thiolase deficiency
  - Propionic and isovaleric acidemias
- Fat oxidation defects
  - Medium, long-chain, and multiple acyl-CoA dehydrogenase deficiencies
  - 3-Hydroxy-3-methylglutaryl-CoA lyase deficiency
  - Others not fully characterized

Drugs and toxins

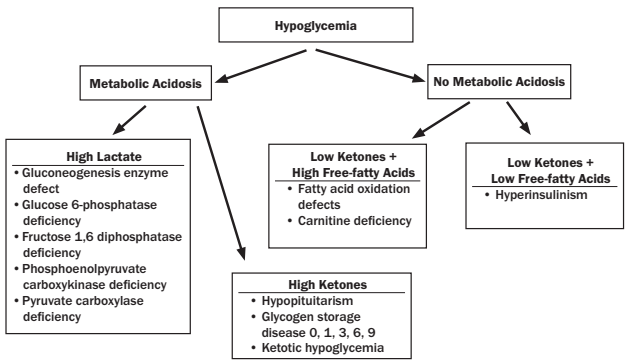
- Alcohol
- Oral hypoglycemic agents
- Salicylates

Hepatic

- Fulminant liver failure
- Reye's syndrome

Adapted from: Surtees R, et al. Acute metabolic encephalopathy: A review of causes, mechanisms and treatment. *J Inher Metab Dis* 1989:12:42-54

Approach to Hypoglycemia



Supplement to *Pediatric Emergency Medicine Reports*, May 2010: "Hypoglycemia in Infants and Children." Authors: **Robert A. Felter, MD, FAAP, CPE, FACPE**, Professor of Clinical Pediatrics, Georgetown University School of Medicine; Assistant Director, Pediatric Inpatient and Emergency Service, Inova Loudoun Hospital, Leesburg, VA. **Ron D. Waldrop, MD, FACEP, CPE, FACPE**, Assistant Professor of Clinical Pediatrics, Georgetown University School of Medicine; Director, Pediatric Inpatient and Emergency Services, Inova Loudoun Hospital, Leesburg, VA. *Peer reviewer:* **Ademola Adewale, MD, FAAEM**, Assistant Professor of Emergency Medicine, Assistant Program Director, Florida Hospital Emergency Medicine Residency Program, Orlando, FL.

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Considerations in Pediatric Thoracic and Abdominal Trauma

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Trauma is the single greatest cause of morbidity and mortality in the pediatric and adolescent populations. Management of pediatric trauma patients is highly specialized, requiring a team approach of nurses, technicians, therapists, social workers, and physicians. Special considerations must be made for pediatric trauma, as children cannot be treated as "small adults." Superior survival outcomes have been demonstrated for the most severely injured children when treated at a dedicated pediatric trauma center.[1]

The initial assessment of pediatric trauma proceeds much like that for adults as outlined in the Advanced Trauma Life Support (ATLS) course of the American College of Surgeons.[2] The primary survey, with immediate correction of life-threatening problems, is followed by a detailed secondary survey and imaging studies of the cervical spine, chest, and pelvis.[3] Next, additional imaging and laboratory testing may be ordered on a case-by-case basis, depending on the findings of the data gathered during the initial resuscitation.

There is growing concern about the use of CT scanning in the pediatric and adolescent population due to exposure to ionizing radiation and the potential development of excess cases of neoplastic disease.[4] Ideally, imaging would be tailored to each individual patient instead of being applied in an algorithmic fashion, subjecting those not likely seriously injured to the potential hazards of unnecessary testing.

In this issue, the authors focus on trauma to the pediatric chest and abdomen. Specifically reviewed are the pediatric mechanisms of injury, potential injury patterns, physical exam findings, and initial stabilization, concluding with a look at imaging and some of the controversies surrounding management of these patients.

— The Editor

Statement of Financial Disclosure

Dr. Dietrich (editor in chief); Drs. Nesbit and Iskyan (authors), and Santamaria (peer reviewer); and Ms. Behrens (nurse reviewer) report no relationships with companies related to this field of study.

Epidemiology

In children, death by injury exceeds all other causes of death combined. Injury is the leading cause of death of children older than the age of 1 year, and, in this population exceeds all other causes of death combined. Injury results in more years of life lost than sudden infant death syndrome, cancer, and infection combined together.[5] Most deaths in the youngest children are from unintentional injury, but homicide and suicide become more prevalent as the population nears young adulthood. The Centers for Disease Control and Prevention report that more than 50,000 children died in motor vehicle accidents from 1999 to 2006, the largest single cause of death in the pediatric and adolescent population.[6]

Non-fatal injuries take an even greater toll on the pediatric population. Nearly 30 million children visit an emergency department (ED) every year in the United States alone. Male children have a higher rate of visits than females, while younger children have higher visit rates than older children.[7] About 40% of the yearly ED visits are for traumatic injury. The International Classification of Diseases (ICD) codes for "unintentional fall" and "unintentional struck by/against" account for most of these visits.[6,8]

The aftermath of these injuries can be staggering, psychologically, financially, and physically. Nearly a decade ago, Miller and colleagues estimated that childhood injuries resulted in $1 billion in resource expenses, $14 billion in lifetime medical spending, and $66 billion in present and future work losses.[9] In 1996, injury left more than 150,000 children and adolescents with a permanent disability, which in many cases will require lifelong medical care. Trauma continues to be a costly and devastating disease among the youngest and most vulnerable of our population. Trauma and accidental injury claim many lives and dramatically impact on many more.

Etiology

Pediatric thoracic trauma is overwhelmingly caused by blunt mechanisms.[10] The most common causes of pediatric blunt chest trauma are motor vehicle collisions (MVCs), pedestrians struck by vehicles, and falls. The vast majority of these are deemed accidental. There are patterns that are somewhat predictable based on age. MVCs and abuse are the leading causes of chest trauma for infants and toddlers. Once children start to attend school, pedestrian accidents come into play; impulsivity can lead them to run into the paths of cars, or their inquisitive nature causes them to play or hide around cars. As they age, skateboarding and cycling start to emerge as causes of significant trauma.[10] Pulmonary contusions, rib fractures, pneumothorax, and hemothorax are the most common injuries after blunt thoracic trauma.[10-12] Aortic, esophageal, diaphragmatic, cardiac, and tracheobronchial injuries are uncommon in children.[10,11] Unfortunately thoracic trauma is rarely a child's only injury, as more than 50% will have more than one intrathoracic injury while about 70% will have additional extrathoracic injuries.[12] Peclet and colleagues report that in children with multiple injuries, death is 10 times more likely if a thoracic injury is present.[13]

Likewise, the vast majority of pediatric abdominal trauma is from blunt mechanisms. The most common causes are associated with MVCs, handlebar injuries, and intentional injury. The pattern of injury changes with age. Children younger than 2 years of age are the most likely to suffer intentional injury, while older children are typically involved in physical activities that may lead to injury. They may suffer collisions during bicycling, sledding, snowboarding, sporting activities, or aggressive play. The solid organs, namely liver and spleen, are most frequently injured.[11,14,15] Bowel, bladder, and kidney injuries also occur, but are much less frequent.[16]

Penetrating thoracic and abdominal trauma, when it does occur, is usually the result of violence. Stabbing and gunshot wounds are the most common mechanisms seen as the pediatric population approaches adulthood.[17] The majority of these types of injuries will likely require operative intervention. Simultaneous assessment and resuscitation of the patient should occur in parallel with preparation of an operating room. If necessary, arrangements to rapidly transfer the patient should be made during the initial assessment and resuscitation.

Pathophysiology

Children differ considerably from adults anatomically and physiologically. Proportionally different, children have larger heads than adults, raising their centers of gravity and contributing to different patterns of injury than seen in adults.[18] Thoracic trauma accounts for about 5% of injuries in hospitalized chil-

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dren, but is the second leading cause of death in pediatric trauma.[11,12,18] Differing injury patterns are partially due to the flexibility of pediatric thoracic structures. The chest wall of a child is elastic and pliable due to increased ligamentous laxity, less rib mineralization, and incomplete ossification of the ribs. Instead of breaking, children's ribs bend when compressed, transmitting more energy to the lungs and thoracic contents.[10,12] In addition, the mediastinum of children is more mobile. Consequently large pneumothoraces or hemothoraces can cause dramatic mediastinal shift resulting in more respiratory or vascular compromise than adults.[11] Lastly, the higher metabolic demands and decreased pulmonary function residual capacity of children results in faster development of hypoxemia.[12]

Abdominal trauma accounts for about 10% of all pediatric trauma admissions, and the abdomen ranks second in the list of most commonly injured sites.[16,19] The abdominal walls of children are thinner, with less developed musculature and fat, than those of adults. This provides less protection to the abdominal organs, allowing the transmission of greater force to the abdominal and retroperitoneal organs.[14] Proportionally, the abdominal organs of a child are also larger, providing a greater surface area over which to absorb force.[14,16] Additionally, the mesentery is less adherent in children, allowing for greater mobility of some organs, possibly contributing to greater bowel injury in deceleration type trauma such as MVCs or falls from a height. Seemingly minor injuries involving handlebar-to-abdomen impacts are associated with injuries to the small bowel and pancreas and are actually a greater risk for injury than flipping over the handlebars.[20] The bladder of very young children is partly located in the abdomen, descending into the pelvis as they age. Thus, bladder injury should also be considered in the younger child presenting with abdominal trauma.[16]

Abdominal trauma in children should also raise concern for spine injury. The spinal columns of children have significantly greater ligamentous laxity, less supporting musculature, and a higher fulcrum of flexion than those of adults.[21] Children restrained only by a lap belt may suffer the so-called "lap belt syndrome" of abdominal wall injury, intra-abdominal organ injury, and vertebral fracture.[22]

The physiological differences between children and adults can lull us into a false sense of security based on "normal" vital signs taken out of context with the overall picture of the patient. Children's vital signs vary significantly with their age and it is important to realize that normal vitals signs in one age group may be an ominous sign in another group.[23] *(See Table 1.)* A minimum systolic blood pressure can quickly be calculated by multiplying the age in years of the child by 2 and adding 70 to the result.[23] The finding of hypotension in an injured child is ominous, as children have a greater capacity to compensate for volume loss, and may occur later in children than it does in adults. Normal or nearly normal vital signs do not exclude significant hypovolemia secondary to blood loss. Children may lose 30% of their blood volume before showing the obvious signs of shock.[17] Frequent vital sign checks are imperative. Simply having a child on continuous monitoring may be insufficient, as the numbers may be deceivingly reassuring. Altered mental status, tachycardia, tachypnea, and diaphoresis may also be indicators of hypoperfusion with impending decompensation. Speaking with the child, if he or she is verbal and old enough, may better allow the additional assessment of perfusion of the brain based on mental status. Helping calm an otherwise frightened and anxious child is an additional benefit.

Table 1. Pediatric Vital Signs by Age Group

AGE	PULSE (UPPER LIMIT)	RESPIRATORY RATE (UPPER LIMIT)	SYSTOLIC BP (LOWER LIMIT)
0-1 mo.	180	60	60
2-12 mos.	160	50	70
1-2 yrs.	140	40	75
2-6 yrs.	120	30	80
6-12 yrs.	110	20	90
> 12 yrs.	100	20	90

Key: BP = blood pressure

Clinical Features

The clinical features of pediatric thoracic and abdominal trauma are very similar to those of adults. Unfortunately, the history and physical exam in pediatric patients may not be reliable and is often more difficult. Depending on the child's age, history may be provided exclusively by those around the child, or it may not be available except as reported by the emergency medical technicians (EMTs), paramedics, or flight crew. The physical exam, especially in those younger than 5 years old, is often hampered by a child's lack of verbal skills, fear, apprehension, and separation from family. Other injuries are extremely distracting and may influence the physical exam.[14,24]

Physical exam findings on children with thoracic injuries may include chest crepitance, subcutaneous emphysema, nasal flaring, diminished or absent breath sounds, tachypnea, dyspnea, or low oxygen saturation.[12] Children with significant thoracic injury may have very little in the way of external signs of trauma due to compliance of the chest wall.[17] Remember that a normal external superficial exam does not exclude significant internal injury.

Signs of abdominal injuries include abrasions, abdominal tenderness, or distention, Cullen's sign (ecchymosis in the periumbilical region), Turner's sign (lateral abdominal wall ecchymosis), and vomiting.[14] There is debate about the importance of the "seat belt sign," which is abdominal erythema, ecchymosis, or abrasions across the abdomen. While Sokolove and colleagues showed the seat belt sign is more common in those with intra-abdominal injuries than in those without injuries after MVCs, Chidester's retrospective study of 331 pediatric patients with abdominal trauma discovered that children with seat belt sign were 1.7 times more likely to sustain abdominal injury, but that it was not statistically significant.[25,26] At the very

least, signs of external abdominal injury should alert the team to the potential presence of internal injury that will necessitate further examination and possible imaging or lab studies to assess for injury.

Diagnostic Studies

The ultimate question for all trauma patients is, "Does this patient have injuries that require immediate operative intervention?" Additionally, if you are at a hospital without full surgical capabilities, transfer may be required for definitive care.

The decision to perform surgery is based mainly on clinical findings and potential deterioration, not imaging studies. Computed tomography (CT) scans, however, do affect diagnosis, management plans, and level of monitoring.[11] To this end, the use of CT scanning for pediatric trauma patients should not be a knee-jerk response, but rather a calculated decision. Imaging should be guided by a review of the mechanism of injury, vital signs, and physical examination. Many adult trauma centers employ the "pan-scan" approach, scanning the head, neck, thorax, abdomen, and pelvis of all trauma patients. There is evidence to suggest that this approach may be beneficial in adults.[27] In children, however, there is less literature on the subject. It is undisputed that the use of CT scan uncovers many injuries, but does the detection of these injuries effect management and, ultimately, outcomes of patients?[28,29]

Of all CT scans, the chest CT is the least commonly used to evaluate trauma patients. Despite this finding, Fenton and colleagues showed that CT scans of the chest are most likely to show injury in excess of a screening chest x-ray.[4] Similarly, a retrospective review of 333 pediatric trauma patients by Markel and colleagues found that conventional chest x-ray remained an acceptable screening tool to evaluate for thoracic trauma. Of the six patients that required emergent surgery for cardiac or arterial compromise, all the injuries were seen on chest x-ray or the scout view of the chest CT. Unfortunately, 5% of chest x-rays in their series falsely reported normal findings that may have ultimately altered management.[30]

There are similar findings when abdominal trauma is considered. In the past, abdominal injuries were diagnosed and managed mainly through an exploratory laparotomy. Today, however, about 95% of children with liver or spleen injuries are managed non-operatively.[31] Holmes and his group reported that 95% of 1,818 patients with solid organ injury were managed non-operatively. The median time to failure (requiring operative intervention) for the remaining 5% was only three hours.[32] The non-operative approach decreased lifetime risk of asplenic sepsis and was associated with shorter hospital stays, fewer blood transfusions, and decreased overall mortality.[31] As most abdominal injuries are managed expectantly via cautious observation, the question becomes "Is any imaging necessary initially?" The decision to operate should ultimately be based on the patient's physiologic response to the injury, not the imaging findings.

Although CT scans provide invaluable information, are there alternatives for the detection of serious thoracic and abdominal injuries? As outlined above, the routine chest x-ray, combined with physical examination, provides excellent information about the likelihood of serious thoracic injury. The use of ultrasound and diagnostic peritoneal lavage (DPL) for the evaluation of abdominal injury requires further evaluation.

The use of ultrasound assessment of the abdomen is routine in many adult trauma centers and the focused abdominal sonography for trauma (FAST) exam is an adjunct to the ATLS protocols for management of trauma patients. Intuitively, pediatric patients seem ideal for a FAST exam as they have small abdominal cavities without large abdominal fat deposits.[4] However, there is considerably less evidence of the utility of FAST in assessment of pediatric trauma.

A paper by Eppich and Zonfrillo reviews the literature regarding management of blunt abdominal trauma.[33] In this review, based on four papers, they note that FAST in children for the detection of blunt abdominal trauma demonstrates variable sensitivity (55%–92.5%) and negative predictive value (50%–97%) but consistently good specificity (83%–100%) when compared to abdominal CT scanning. While the FAST exam does miss some patients with free fluid, the clinical significance of this is not clear given that most abdominal injuries in children are managed expectantly. One of the four papers, that by Soudack and colleagues, concludes that a positive FAST exam necessitates further "definitive imaging."[24]

More recently, Holmes and colleagues conducted a meta-analysis of the use of ultrasonography in pediatric blunt abdominal trauma.[34] Their analysis included 3,838 children from 25 articles. They concluded that a negative ultrasound exam has "questionable utility as the sole diagnostic test to rule out the presence of IAI [intra-abdominal injury]" and go on to state that a positive ultrasound in the hemodynamically stable child should lead to immediate CT scanning. They additionally conclude that children with a moderate pretest probability of intra-abdominal injury should undergo abdominal CT scanning regardless of the findings on abdominal ultrasound.

One of the criticisms of the FAST exam is its inability to identify solid organ injury that may not produce hemoperitoneum. In the meta-analysis by Holmes, it was found that the additional ultrasound evaluation of solid organs only slightly increased the sensitivity of the standard FAST exam in pediatric patients, to 82% from 80%. However, the question was raised concerning the ability of non-radiologists to ultrasound solid organs.[34]

The use of DPL has fallen out of favor given the discomfort to the patient and lack of specificity of the exam. It is not recommended for the assessment of an isolated abdominal injury, but is useful to diagnose children with abdominal trauma who sustained multiple injuries and require immediate surgery for another injury, often a subdural or epidural hematoma.[14]

Can laboratory testing help in identifying children who should undergo CT scans for injuries? Capraro, Mooney, and Waltzman examined the utility of the "trauma panel" in the assessment of blunt abdominal trauma.[35] In a retrospective

review of 382 pediatric patients, they found that none of their regularly tested chemical or hematological parameters had sufficient sensitivity or negative predictive value to be helpful as a screening tool. Cotton and Beckert considered both clinical and laboratory data. They determined that 23 variables were potentially associated with intra-abdominal injury.[36] Logistic regression identified four positive predictors for injury: tenderness, abrasions, ecchymosis, and elevated ALT. Holmes and colleagues published two papers in May 2002 addressing this subject in both abdominal and thoracic trauma.[19,37] They derived clinical decision rules to identify children with thoracic or intra-abdominal injuries after blunt trauma. The prospective series for abdominal trauma enrolled 1,095 children younger than 16 years with blunt trauma. They identified 107 patients with intra-abdominal injuries. Statistical analysis identified six findings associated with abdominal injury: low systolic blood pressure, abdominal tenderness on exam, femur fracture, serum AST >200 U/L or serum ALT >125 U/L, urinalysis with >5 RBCs per high-powered field, and an initial hematocrit of less then 30%. Of the 107 children with an intra-abdominal injury, 105 had at least one of these findings, while absence of any of the six was seen in all but two children with injury. The authors acknowledged some limitations, as they did not evaluate the use of ultrasound in their decision rule and not all of the children with abdominal trauma underwent imaging due to "ethical considerations."[19]

In another series, the Holmes group applied the same type of analysis to children with thoracic injury.[37] Nine-hundred-eighty-six patients with thoracic trauma were enrolled, and 80 of them were found to have injuries. Analysis identified the following predictors of thoracic injury: low systolic blood pressure, elevated age-adjusted respiratory rate, abnormal thorax exam, abnormal chest auscultation, femur fracture, and Glasgow Coma Scale (GCS) score of < 15. Seventy-eight of the 80 injured patients had at least one of these findings, while two did not have any of these findings. The two missed cases did not require intervention for their thoracic injuries.

Holmes and colleagues have recently published a paper on validation of their derived prediction rule for blunt torso trauma.[38] In this series of 1,119 children with blunt torso trauma, they identified 149 of 157 injured children. Of the eight patients that were missed, only one underwent laparotomy for a serosal tear and mesenteric hematoma that did not require "specific surgical intervention." Application of their decision rule would have resulted in a reduction of CT scans by 33%. They conclude that further refinement of their prediction rule is needed before it is ready for widespread use.

Management

The management of pediatric abdominal and thoracic trauma is similar to that of adults. Standard ATLS protocols should be followed.[3] In the primary ATLS survey, all life-threatening injuries must be identified and addressed before progression with the detailed secondary survey. *(See Table 2.)* All pediatric patients being assessed for trauma must be continuously monitored for blood pressure, heart rate, respiratory rate, and blood oxygen saturation. In addition, every child should have a recorded temperature and be protected from hypothermia. Supplemental oxygen should be provided. Adequate intravenous (IV) access is imperative. If two peripheral IV lines cannot be rapidly secured, intraosseous access or central venous access should be considered.

Table 2. Immediately Life-threatening Thoracic Injuries

- Airway obstruction and injury
- Lung and chest wall injuries
- Open pneumothorax
- Tension pneumothorax
- Hemopneumothorax
- Flail chest
- Widened mediastinum / aortic disruption
- Cardiac tamponade

Any signs of shock should be treated aggressively with fluid resuscitation. First-line fluids should be crystalloids given in 20 mL/kg boluses. Packed red blood cells (PRCBs) should be transfused at 10 mL/kg if the blood pressure does not respond to two fluid boluses.

After the initial resuscitation is complete, a thorough secondary survey should evaluate for other injuries. A head-to-toe examination is undertaken for signs of injury and disability. The entire surface of the child's body must be exposed for this examination. It is important to remember to examine the child's back, as well. Vital signs should be reassessed frequently throughout the resuscitation. Any deterioration in the condition of the patient should prompt immediate reassessment, starting with the primary survey. During this time, the physician should attempt to gain additional knowledge concerning past medical history, allergies, and medications. The AMPLE mnemonic is useful for this purpose.[3] *(See Table 3.)*

As previously discussed, non-operative management with close observation is now the mainstay of most pediatric thoracic and abdominal trauma. Nearly 90% of blunt pediatric chest injuries can be managed non-operatively or with a thoracostomy tube.[12] Indications for a thoracotomy include tracheobronchial injuries, esophageal injuries, diaphragmatic rupture, major vascular injury, retained hemothorax, return of 20%–30% of a child's blood volume through a chest tube, or persistent hemorrhage (defined as continued bleeding of 2–3 mL/kg per hour over a four-hour period).[12]

Greater than 90% of pediatric abdominal trauma is successfully managed non-operatively. If a patient is hemodynamically stable without peritoneal signs, non-operative management should be attempted.[39] The argument has also been made that the hemodynamically unstable child who responds to boluses

Table 3. The AMPLE History

A	Allergies
M	Medications
P	Past illness
L	Last meal
E	Events related to injury

of PRCBs may avoid the operating room.[40] Failure of non-operative management usually occurs within four hours and nearly always within 12 hours of presentation. Children with severe or multiple solid organ injuries and pancreatic injuries are nine times more likely to require operative intervention.[41] Despite previous perceptions, a cohort study by Tataria and colleagues of 2,944 children with blunt abdominal trauma showed that delayed operative management or failure of non-operative management does not change outcome in terms of mortality, ICU length of stay, hospital length of stay, or blood transfusions.[39]

Figures 1 and 2 summarize a general approach to assessing the child with thoracic or abdominal trauma. These figures are meant to serve only as a rough outline of a thought process that could be used to assess the victim of pediatric trauma, and not as specific protocols.

Current Controversies

Unnecessary Exposure to Radiation. There has been concern raised over the last two decades about the use of CT scanning in the both the adult and pediatric populations.[42] There are more than 60 million CT scans done each year in the United States. This is 20 times the number of scans performed in 1980. About four million of these are done in children.[42] Today, the medical imaging radiation dose, which is primarily from CT scans, is the largest source of radiation, besides background radiation, received by the U.S. population.[43] That CT scanning has altered diagnostic algorithms and made detection of injury more sensitive can not be argued. However, the risk of radiation is twofold: increased risk of cancer due to radiation exposure, and missing a diagnosis due to suboptimal image quality due to decreased radiation exposure settings.[44]

The authors of several papers, especially in the radiology literature, have looked at the increased risk of cancer due to radiation from CT scanning.[45-47] It has been estimated that the risk of developing a fatal cancer from a CT scan may be in the range of about 1 in 1,000.[45] In addition to their body tissues being more sensitive to radiation, children have a longer lifespan over which to develop a cancer.[46] While the risk to an individual is relatively small, this small risk multiplied by a large number of scans results in a relatively large number. Based on the above numbers, CT scans may be responsible for causing 4,000 fatal cancers a year. The number of non-fatal cancers is likely to be higher.

The Alliance for Radiation Safety in Pediatric Imaging launched the "Image Gently" campaign in 2007 to raise awareness of these important issues.[48] The "as low as reasonably achievable" (ALARA) principle is also promoted by this campaign. A recent paper by Arch and Frush finds that since a prior survey in 2001, the peak kilovoltage and tube current settings, the two principal parameters determining radiation dose from CT scanning, have decreased significantly for pediatric body multidetector CT.[49] It may be that increased awareness of the potential hazards of radiation is having an effect.

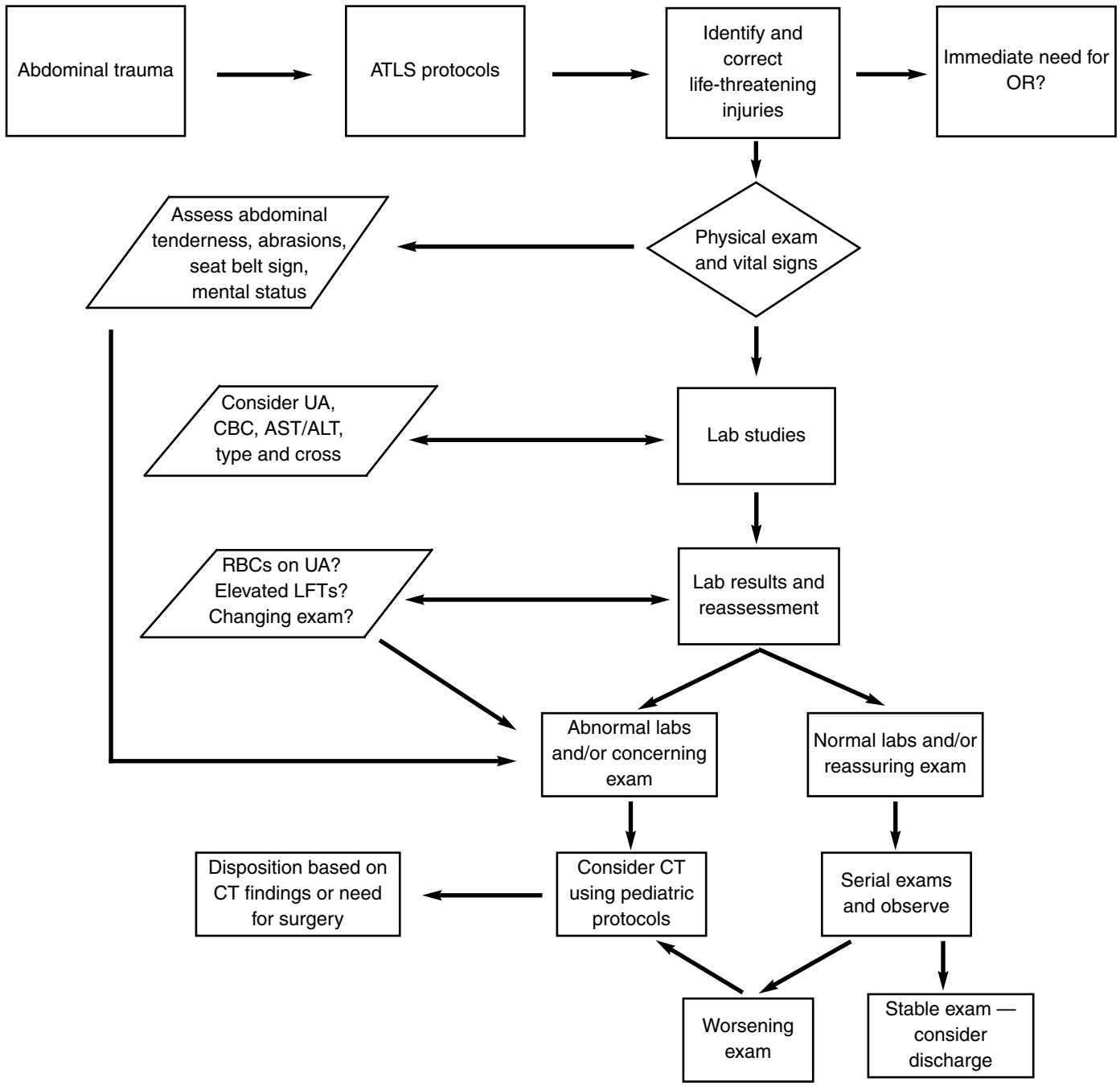
In an effort to reduce radiation exposure, Cohen raises an interesting point: How low can you reduce the dose before the radiologist becomes uncomfortable making an accurate diagnosis?[44] Lower radiation dosages equate to a lower risk of cancer, but this reduction comes with a possible reduction in diagnostic certainty. Where does the radiologist draw the line between diagnostic certainty and patient safety?

Obviously, the best way to reduce radiation exposure to pediatric trauma patients is to avoid unnecessary CT scans. While evidence based medicine and clinical judgment help determine which patients need CT scans, there are a number of other factors contributing to unneeded CT scans. Donnelly cites overcautious ordering of CT scans due to potential malpractice litigation, public pressure to use high-end technical exams, and Americans' need for immediate results as reasons for unnecessary CT scans.[50] Both physicians and the public will have to work together to decrease the number of unnecessary CT scans on children.

CT Scans and the Transfer of Pediatric Trauma Patients. When a pediatric trauma patient is being transferred to a tertiary facility for further care, should the CT scans be done at the referring facility or the receiving facility? ATLS recommends the transfer of appropriate patients without delay to a designated trauma center. Therefore, further diagnostic studies should not be undertaken, as they will not change the immediate care of the patient.[4] Additionally, just as a CT scan should not be used to determine if a patient requires the OR, a CT scan should not be used as a tool to determine if a patient should be transferred to a trauma center.[51] The decision to transfer a patient to a higher level of care is based on hemodynamic stability and the ability to provide care. The drawbacks in obtaining a CT prior to transfer include the inability to provide definitive care, the time delay in obtaining the scans, increased vulnerability of trauma patients if decompensation occurs in radiology or during transport, the lack of pediatric protocols to decrease radiation exposure at some referring facilities, and the possible need to repeat the study at the receiving facility.[4,51] Depending on location, transfer to a specialized pediatric trauma center may require a significant amount of time. Nance and colleagues recently published an interesting paper reporting that 71.5% of the pediatric population of the United States was within 60 minutes of a verified pediatric trauma center by either ground or air transport.[52] If only ground transport was considered, the percentage of the population that had access within an hour was only 43%. This percentage varies significantly from state to state and from rural to urban settings.

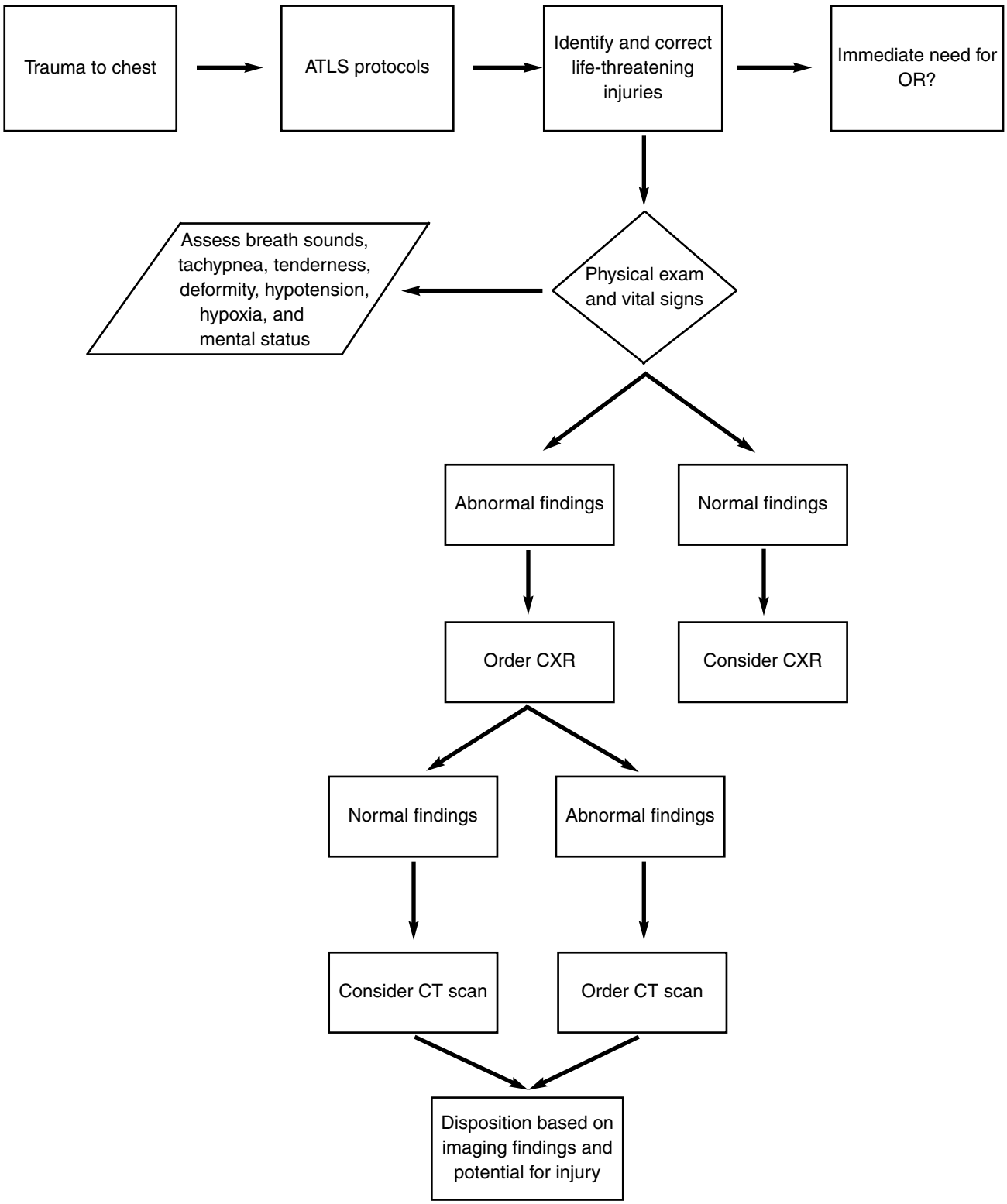
"Blush" on CT. A blush on CT indicates bleeding in or

Figure 1. Algorithm for the Evaluation of Blunt Abdominal Trauma



around an organ due to injury of a large arterial branch. While it has been described for the liver, kidney, adrenal gland, and mesentery, the spleen is the most common organ for a blush to be found.[53] In adults, the finding of a blush of a solid organ, specifically the spleen, on CT scan indicates a higher chance of non-operative failure and warrants early embolization or surgery.[53,54] In children, however, there has been controversy about whether there is a relationship between splenic blush and need for operative intervention. First, splenic blush is often missed on CT scan. In a retrospective study of 216 pediatric abdominal trauma patients, 27 whom had a splenic blush, the contrast blush was "frequently not identified" by the radiology resident and mentioned in "only a few" of the dictations by the attending radiologist.[54] Next, contrast blush can look identical on CT scan to areas of damaged splenic parenchyma or stable hematoma, making the diagnosis of a blush questionable.[54] Most importantly, studies surrounding splenic blush and the failure of non-operative management in children have been

Figure 2. Algorithm for the Evaluation of Blunt Thoracic Trauma



inconsistent. The largest study by Nwomeh and colleagues of 27 patients with splenic blush found a statistically significant correlation between contrast blush of the spleen and operative management as 46% of the children with blush went to the OR.[54] Two other smaller case series by Cox and Lutz had even higher rates of children with contrast blush requiring operative intervention.[40,53] In a case series of blunt splenic injuries, five pediatric patients with splenic blush were identified.[55] Only one

of these required operative management due to hemodynamic instability. Despite conflicting results, the conclusion of all the authors is essentially the same: CT scan can accurately define the anatomic grade of an intra-abdominal organ injury, but cannot predict the failure of non-operative management of splenic injuries. The decision for operative intervention should be based on physiologic response to the injury rather than radiographic findings.

Patient Advocacy

Given the vulnerability of children, abuse should always be considered with pediatric trauma. Two thirds of the victims of abuse are younger than 3 years of age, and one third are younger than 6 months old.[56] In particular, child abuse should be considered with rib fractures, duodenal hematoma, pancreatitis, and pancreatic fractures.[11] Rib fractures occur in only 1%–2% of pediatric trauma; however, 82% of rib fractures in children younger than 3 years of age are related to child abuse.[10-12] There is no pathognomonic fracture indicative of abuse.[57] If there is no history of trauma, or there is a history of trauma that does not match the pattern of injury, child abuse should be suspected. The American Academy of Pediatrics 2007 guideline for the evaluation of suspected child physical abuse lists five circumstances that are concerning for intentional trauma.[58] They are:

1. No or vague explanations for significant injury.
2. Change in an important detail.
3. Explanation inconsistent with the pattern, age, or severity of the injury.
4. Explanation inconsistent with the child's physical and/or developmental capabilities.
5. Discrepancies among the stories of witnesses.

The presence of any of these circumstances should prompt further investigation and admission of the child until the details of the injury can be thoroughly investigated.

Disposition

Whether a pediatric trauma patient should be admitted is often based on clinical judgement. There have been a handful of studies evaluating whether a child who suffered from abdominal trauma with a normal abdominal CT scan can be safely discharged home. In a large, prospective, observational cohort study, Awasthi and colleagues followed 1,085 pediatric blunt trauma victims who had normal CT scans in the ED.[59] Of the 32% who were discharged to home, none returned to the hospital. Two of the 737 admitted for observation had an abdominal injury on repeat CT scan, but neither required intervention. They conclude that a child with a normal abdominal CT scan and a normal abdominal exam (no tenderness, distention, ecchymosis, or abrasions) can be discharged home, while a child with a normal abdominal CT scan and an abnormal exam should be carefully observed in or out of the hospital. To our knowledge, there are no studies that have directly evaluated the disposition of children after having a normal chest CT following trauma. If a child has undergone a CT scan to evaluate for potential thoracic trauma, he or she likely should be admitted for observation and repeat exams.

Prevention

It should not require stating that the best way to care for the pediatric trauma patient would be to prevent the trauma from ever occurring. Physicians and nurses are obligated to educate patients and their parents when given the opportunity to do so. A child sustaining a mild head injury in a fall from a bicycle should not be discharged without instructions for head injury. Likewise, if the child was unhelmeted, the importance of wearing a helmet should be addressed. If the child was wearing a helmet, reinforce the positive, and remind parents that the child needs a new helmet. Instruct parents on the proper use of car seats and seatbelts. Remind children to wear pads and helmets when skateboarding or rollerblading. There are numerous ways for all of us to educate; take a moment and do so.

Conclusions

Trauma is a significant cause of morbidity and mortality in children. A heightened awareness of potential thoracic or abdominal injuries can facilitate an appropriate diagnostic evaluation.

CT scanning is one of the most important medical advances in the last 100 years. However, improvements in imaging come with the increased risks associated with radiation, especially in the pediatric population. CT scanning undoubtedly saves lives, but imaging should be tailored to each patient after consideration of mechanism of injury, vital signs, physical examination and selected laboratory values. Further refinement of clinical decision rules may help to limit the number of questionably necessary CT scans performed for pediatric chest and abdominal trauma.

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CME Questions

1. What is the most common cause of morbidity and mortality in the pediatric population?
   A. Accidental trauma
   B. Non-accidental trauma
   C. Ingestions of toxins
   D. Congenital abnormalities

2. Which of the following injuries is immediately life-threatening, according to the ATLS Primary Survey?
   A. Chest contusion
   B. Airway obstruction

CNE/CME Objectives

Upon completing this program, the participants will be able to:
a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

CME / CNE Instructions

Physicians and nurses participate in this CME/CNE program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a letter of credit.** When your evaluation is received, a letter of credit will be mailed to you.

C. Simple pneumothorax
D. Rib fracture
E. Head laceration

3. Which of the following types of trauma is the most common cause of death in the pediatric population?
   A. Thoracic
   B. Abominal
   C. Head
   D. Musculoskeletal

4. How is most pediatric blunt trauma managed?
   A. Laparotomy
   B. Laparoscopy
   C. Celiotomy
   D. Cautious observation

5. In which of the following groups is a systolic blood pressure of 70 mmHg acceptable as a minimum?
   A. Older than 12 years old
   B. 6–12 years old
   C. 2–6 years old
   D. 2–12 months old
   E. Never acceptable

6. What percentage of the pediatric population lives within 60 minutes of a pediatric trauma center by either ground or air transport?
   A. 20%
   B. 30.5%
   C. 50%
   D. 71.5%
   E. 95.6%

7. Based on the assumption that CT scanning causes one fatal cancer in 1,000 scans, about how many cancers in the pediatric population may be attributable to CT scanning each year?
   A. 4
   B. 40
   C. 400
   D. 4,000
   E. 40,000

8. Which of the following is the most effective way to reduce radiation exposure from CT scanning in children?
   A. Specific pediatric protocols
   B. Lead shielding
   C. Limiting CT scan to area of interest
   D. Avoid CT scanning if possible

9. When compared to adults, the thoracic wall of the child is:
   A. more rigid.
   B. more protective.
   C. more compressible.
   D. more ossified.

10. What type of bony fracture is associated with the "lap belt syndrome"?
   A. Femur
   B. Rib
   C. Pelvis
   D. Vertebral

Answers: 1. A, 2. B, 3. C, 4. D, 5. D, 6. D, 7. D, 8. D, 9. C, 10. D

In Future Issues:

Adult Thoracic and Abdominal Injury

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CNE/CME Evaluation — Vol. 11, No. 3: Pediatric Thoracic and Abdominal Trauma

Please take a moment to answer the following questions to let us know your thoughts on the CNE/CME program. Fill in the appropriate space and return this page in the envelope provided. **You must return this evaluation to receive your letter of credit. ACEP members — Please see reverse side for option to mail in answers.** Thank you.

CORRECT ● **INCORRECT** ⊘ ✓ ✗ ⚠

1. In which program do you participate? ○ CNE ○ CME
2. If you are claiming physician credits, please indicate the appropriate credential: ○ MD ○ DO ○ Other _____
3. If you are claiming nursing contact hours, please indicate your highest credential: ○ RN ○ NP ○ Other _____

After participating in this program, I am able to:	Strongly Disagree	Disagree	Slightly Disagree	Slightly Agree	Agree	Strongly Agree
4. Discuss conditions that should increase suspicion for traumatic injuries.	○	○	○	○	○	○
5. Describe the various modalities used to identify different traumatic conditions.	○	○	○	○	○	○
6. Cite methods of quickly stabilizing and managing patients.	○	○	○	○	○	○
7. Identify possible complications that may occur with traumatic injuries.	○	○	○	○	○	○
8. The test questions were clear and appropriate.	○	○	○	○	○	○
9. I detected no commercial bias in this activity.	○	○	○	○	○	○
10. This activity reaffirmed my clinical practice.	○	○	○	○	○	○
11. This activity has changed my clinical practice.	○	○	○	○	○	○

If so, how? _____

12. How many minutes do you estimate it took you to complete this activity? Please include time for reading, reviewing, answering the questions, and comparing your answers with the correct ones listed. _____ minutes.
13. Do you have any general comments about the effectiveness of this CNE/CME program?

_____

I have completed the requirements for this activity.

Name (printed) _____ **Signature** _____

Nursing license number ***(required for nurses licensed by the state of California)*** _____

Optional for ACEP members: In accordance with ACEP requirements, below we provide the option for ACEP members to submit their answers for this CME activity. If you wish to submit answers for this activity, please refer to this issue (Vol. 11, No. 3) and circle the correct responses.

1. A
   B
   C
   D

2. A
   B
   C
   D
   E

3. A
   B
   C
   D

4. A
   B
   C
   D

5. A
   B
   C
   D
   E

6. A
   B
   C
   D
   E

7. A
   B
   C
   D
   E

8. A
   B
   C
   D

9. A
   B
   C
   D

10. A
    B
    C
    D

Trauma Reports

2010 Reader Survey

In an effort to learn more about the professionals who read *Trauma Reports,* we are conducting this reader survey. The results will be used to enhance the content and format of *Trauma Reports*.

Instructions: Fill in the appropriate answers. Please write in answers to the open-ended questions in the space provided. Please insert this survey in the provided envelope along with your continuing education evaluation. Return the questionnaire by **July 1, 2010.**

1. Are the articles in *Trauma Reports* written about issues of importance and concern to you?

- O A. Always
- O B. Most of the time
- O C. Some of the time
- O D. Rarely
- O E. Never

2. How would you rate your overall satisfaction with your job?

- O A. Very satisfied
- O B. Somewhat satisfied
- O C. Somewhat dissatisfied
- O D. Very dissatisfied

3. What are you most dissatisfied with in your job?

- O A. staffing
- O B. heavy workload
- O C. low morale in your department or facility
- O D. impact of cost-cutting on quality of care
- O E. other _____

Questions 4-9 ask about coverage of various topics in *Trauma Reports.* Please mark your answers in the following manner:

A. very useful B. fairly useful C. not very useful D. not at all useful

4. Imagin in Pediatric Abdominal Trauma (July/Aug. 2009)	O A	O B	O C	O D
5. ATLS Update (Sept./ Oct. 2009)	O A	O B	O C	O D
6. Traumatic Brain Injury (Nov./Dec. 2009)	O A	O B	O C	O D
7. Hand and Wrist Injuries (Jan./Feb. 2010)	O A	O B	O C	O D
8. Genitourinary Trauma (March/April 2010)	O A	O B	O C	O D
9. Pediatric Thoracic and Abdominal Trauma(May/June 2010)	O A	O B	O C	O D

10. How do you receive *Trauma Reports*?

- O A. I am a paid subscriber (proceed to question 11)
- O B. I receive it as a supplement to another publication (skip to question 12)

11. Do you plan to renew your subscription to *TR*? O A. yes O B. no

If not, why? _____

12. How would you describe your satisfaction with your subscription to *TR*?

- O A. Very satisfied
- O B. Somewhat satisfied
- O C. Somewhat dissatisfied
- O D. Very dissatisfied

13. What is your title?

- O A. Practicing emergency medicine physician
- O B. Trauma surgeon
- O C. Emergency department or surgical nurse
- O D. Physician assistant
- O E. Professor/academician
- O F. Emergency medicine manager/director
- O G. Resident

14. On average, how much time do you spend reading each issue of *TR*?

- O A. fewer than 30 minutes
- O B. 30-59 minutes
- O C. 1-2 hours
- O D. more than 2 hours

15. On average, how many people read your copy of *TR*?

- O A. 1-3
- O B. 4-6
- O C. 7-9
- O D. 10-15
- O E. 16 or more

16. On average, how many articles do you find useful in *TR* each year?

- O A. 1-2
- O B. 3-4
- O C. 5-6

17. How large is your hospital?

- O A. fewer than 100 beds
- O B. 100-200 beds
- O C. 201-300 beds
- O D. 301-500 beds
- O E. more than 2,000

Please rate your level of satisfaction with the following items.
A. excellent B. good C. fair D. poor

18. Quality of newsletter	○ A	○ B	○ C	○ D
19. Article selections	○ A	○ B	○ C	○ D
20. Timeliness	○ A	○ B	○ C	○ D
21. Length of newsletter	○ A	○ B	○ C	○ D
22. Overall value	○ A	○ B	○ C	○ D
23. Customer service	○ A	○ B	○ C	○ D

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○ B. Pediatric Emergency Medicine Reports

26. Would you subscribe to *Trauma Reports* if it were available as a 12-month subscription?

○ A. yes
○ B. no

27. To what other publications or information sources do you subscribe?

28. Please list the top three challenges you face in your job today.

29. What do you like most about *Trauma Reports*?

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