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Ultrasound (US) continues to evolve as a valuable adjunct to the clinical care of patients in the emergency department (ED). US has emerged as a safe, portable modality for the diagnosis or exclusion of many significant disease processes in children. The ability to avoid unnecessary exposure to ionizing radiation has become a priority, and the emergency physician (EP) plays a critical role in determining the ideal test for each patient.

Understanding the strengths, limitations, and clinical implications of each focused US examination can help the EP to decide the best and most specific test for each patient.

The first part of this two-part series addressed the use of US for complications of early pregnancy in adolescent females and for testicular complaints in pediatric and adolescent males. This issue focuses on the use, role, and limitations of US in the evaluation of cardiac, abdominal, and renal disease processes.

—The Editor

Advances in Pediatric Ultrasound

Part 2: Focused Applications for Cardiac, Abdominal, and Renal Complaints

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Pediatric Cardiac Sonography

Pediatric echocardiography is a rapidly developing field. Fetal echocardiography, especially the transesophageal approach, can identify congenital cardiac abnormalities with improved resolution and accuracy. Three-dimensional (3-D) echocardiography adds a powerful tool to the pediatric cardiology and cardiothoracic specialties. Transthoracic echocardiography remains the most readily available, non-invasive, and least anxiety-provoking cardiac imaging modality that can be brought to the bedside of the pediatric patient. The use of bedside cardiac sonography in the ED by EPs is well documented in the adult population. In contrast, research regarding the use of cardiac US in the pediatric emergency medicine literature is very sparse. The purpose of pediatric cardiac sonography in the ED is not identification and specification of complex, congenital heart defects, but instead the identification of cardiac pathology that immediately affects clinical care. Potential clinical applications include: the evaluation of the presence or absence of a pericardial effusion, cardiac tamponade, or global wall motion /function, including the differentiation of subcategories of hypotensive states. Although adult cardiac emergencies are a routine part of most EPs' practice, pediatric cardiac emergencies are uncommon.[1] The focus of cardiac ultrasound in the ED includes: 1) identification of pericardial effusions and cardiac tamponade, and 2) assessing global cardiac wall function. In addition, the ultrasound screening evaluation of the older child/adolescent, especially the athlete, who presents with symptoms that may be sentinel events for sudden cardiac death is included.[2]

A pediatric transducer equipped with a small footprint will enable transmission between ribs. A frequency of 5.0 MHZ is suitable for the child or infant and 3.5 MHz for the average-sized adolescent. The main cardiac views are the parasternal and subxiphoid (subcostal) approaches in a supine infant or child.

The Parasternal View. The parasternal view can be a long axis view (PSLA) or a short axis view (PSSA).

The PSLA view is obtained by placing the transducer near to, but not on, the left sternal edge. The marker should be pointing toward the patient's right shoulder. Make slight rotations of the transducer until an elongated image appears of the heart. (*See Figure 1.*) The US image depicts the apex toward the left of the screen. The fibrous pericardium is echogenic (white) and surrounds the heart. Blood in the chambers is echolucent (black). The myocardium is of medium echogenicity. Structures closest to the transducer surface appear at the top of the monitor. During this part of the evaluation, observe the movement of the walls — they thicken during systole— toward the center of the chamber. Assess the global wall motion. Next, assess ventricular cavity size and change during systole as global assessment of cardiac ejection. On the PSLA view, the descending aorta is seen in cross section. The descending aorta is a very important landmark. It appears as an echolucent circle, posterior to the heart at the atrioventricular sulcus and the mitral valve.

Convert from the PSLA to the PSSA view by slowly rotating the probe marker clockwise so that it points in the direction of the patient's left shoulder. (*See Figure 2.*) Maintain full contact of the probe with the patient. PSSA views have multiple image options created by slight angulations of the transducer. Angling the transmitted US beam slowly between the patient's right shoulder and the left hip provides cross-sectional cuts. The left ventricle normally has thicker walls and is draped anteriorly by the right ventricle. The right ventricle is thin-walled and has a semi-lunar appearance. The mitral valve traverses the left ventricular cavity. Its movement resembles that of a fish's mouth. The aortic valve is seen in PSSA cuts. The aortic valve is centrally located in this view between the anterior right ventricular outflow tract and the posterior left-sided chambers.

The Subcostal View. Place the transducer at the subxiphoid area. Apply gentle pressure to the abdomen, then direct the beam just under the left ribcage and to the child's left shoulder. Rotate the probe to get a four-chamber view of the heart. The apex is seen clearly in this view.

A modified subcostal view provides information on the longitudinal axis of the proximal inferior vena cava (IVC) as it enters the heart. The beam is directed to the patient's back with the marker to the head. Slight angling of the transducer to the right of the midline reveals the IVC. In a normovolemic patient with normal central venous pressures, the IVC can be observed to collapse to approximately half of its width with a patient's deliberate sniff or with inspiration (a decrease in intrathoracic pressure improves venous return). Distended hepatic veins and a distended IVC support the concern of an increased central venous pressure.

Neonatal Cardiac Disease (Shock). Pediatric cardiac sonography may be very valuable in the first 30 days of life. Early recognition of significant cardiac lesions and differentiation of

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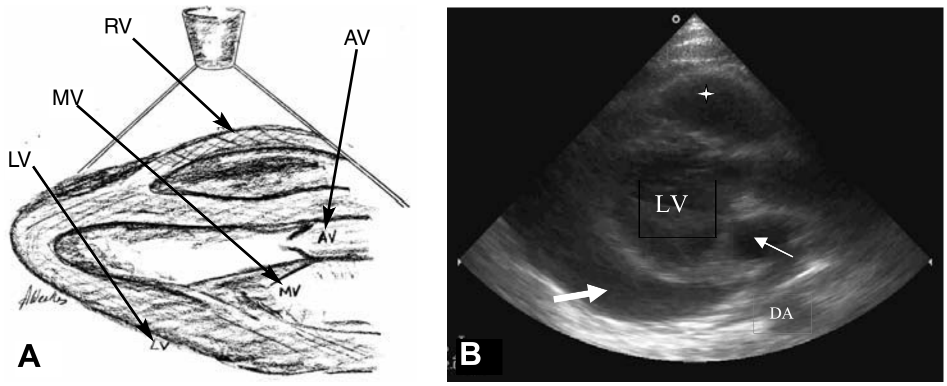
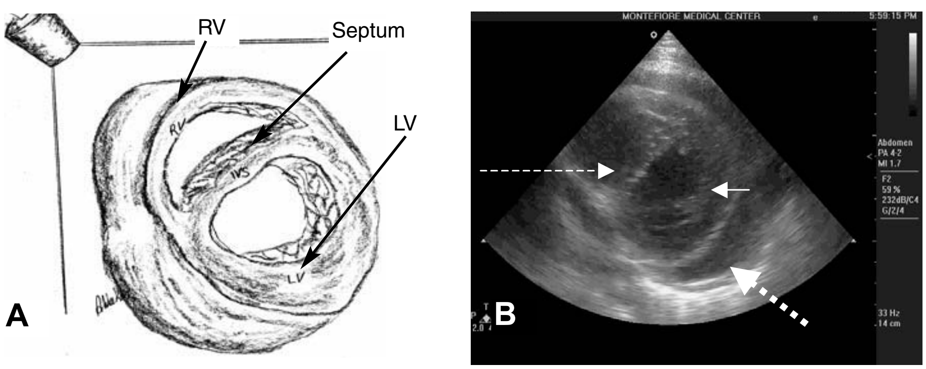
Figure 1. Parasternal View - Long Axis



Parasternal long axis view of the heart. A: Diagram of the heart's appearance on the screen. B: Notes right ventricle (cross), left ventricle (LV), left atrium (thin short arrow), pericardial effusion (wide short arrow). Note the descending aorta in cross section (DA).

Figure 2. Parasternal View - Short Axis



Parasternal short axis view of the heart. **A**: Diagram of the heart's appearance on the screen. **B**: Ultrasound image: Right ventricle (arrowhead), left ventricle (thin, short arrow), pericardial effusion (wide, dotted arrow) and septum (thin, dotted arrow).

cardiac lesions from sepsis or pulmonary disease, allows the EP to accurately direct the medical care of these critical infants and expedite referral to the appropriate sub-specialist.

In the first few days of life, a patent ductus arteriosus (PDA) may maintain pulmonary and systemic vascular perfusion, despite the presence of obstructive left-sided cardiac lesions such as critical aortic stenosis and preductal coarctation of the aorta. Upon closure of the ductus arteriosus, the neonate will have signs of systemic hypoperfusion – shock. The left atrium is under increased pressure and dilates. The foramen ovale directs left atrial blood flow to right-sided chambers, thus increasing the pulmonary blood flow and congestion.

The hypoplastic left heart syndrome also may present with congestive heart failure or signs of hypoperfusion. In these cases, echocardiography detects hypoplastic left ventricular walls and hypertrophied right-sided chambers.

Initial medical management is focused on reestablishing and maintaining the patency of the ductus arteriosus. This is accomplished in all cases with Prostaglandin E1 infusions. Additional required therapy may include: ventilator support and management, inotropic agents, and systemic vascular resistance reduction agents. Further definitive interventions may include surgical valvular repair and cardiac catheterization to the valve or area of coarctation.

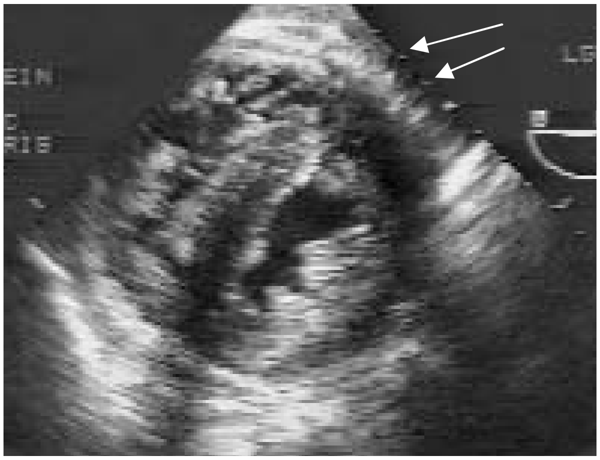
Neonatal Cardiac Disease (Cyanosis). Newborns also may present with cyanosis and tachypnea, and differentiating between pulmonary and cardiac causes of hypoxia is extremely challenging. Pulmonary congestion on chest auscultation and cyanosis may exist in both scenarios. Accurate classification and delineation of clinically significant cardiac murmurs are difficult in a distressed tachypneic infant.

Right-sided heart failure signs such as cyanosis and tachypnea in the neonatal period can be secondary to pulmonary valve stenosis with an intact ventricular septum. Right ventricular hypertrophy with pulmonic valvular stenosis and an intact septal wall are the main echocardiographic features. This congenital cardiac condition may be treated successfully with Prostaglandin E1 infusions and balloon pulmonary valvulotomy. Atrial and ventricular defects can present weeks after birth depending upon the severity and location of the lesions.

Hypertrophic Cardiomyopathy. Cardiac sonography also may be a valuable screening tool for athletes who present with symptoms of chest discomfort (e.g., pain or tightness), lightheadedness, syncope, or palpitations. In patients with any of these clinical presentations, EPs should consider the possibility of an arrhythmia or a cardiac structural abnormality. In particular, a previous history of recurrent syncopal events, a family history of hypertrophic cardiomyopathy or sudden death, symptoms exacerbated by dehydration or exertion, or a loud systolic ejection murmur on physical examination should raise suspicion for hypertrophic cardiomyopathy (HCM). HCM occurs in 1 in 500 of the general population and accounts for approximately 40% of the cardiac disease in young athletes.

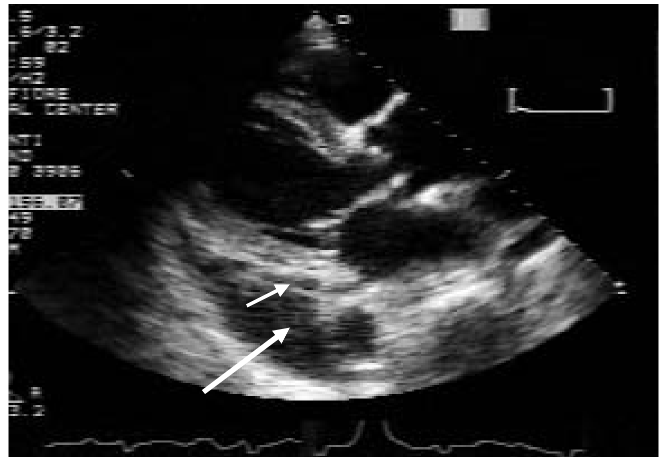
Left ventricular hypertrophy (LVH) is seen normally in the serious athlete, and septal and posterior wall hypertrophy occurs in male, but not female athletes. In most athletes, a wall thickness greater than 13 mm is a reliable indicator of hypertrophic

Figure 3. Hypertrophic Cardiomyopathy



Patient with exertional syncope due to hypertrophic cardiomyopathy. The heart's walls are markedly thickened greater than 18 mm. See centimeter depth markers (arrows).

Figure 4. Pericardial and Pleural Fluid Collections



Parasternal long axis view of the heart shows thin rim of pericardial fluid (short arrow). Pleural fluid (long arrow) extends posterior to the landmark descending aorta.

cardiomyopathy. Unfortunately, idiopathic LVH accounts for 10% of sudden cardiac death cases.[2] Patients found to have this condition need immediate referral to a cardiologist for a definitive diagnostic evaluation and determination of activity restrictions. (*See Figure 3.*)

Pericardial Effusion. Cardiac sonography may be used to exclude or confirm the diagnosis of pericardial effusion. Clinical scenarios where a pericardial effusion may be suspected include: suspected or known pericarditis, recent cardiac surgery, medical resuscitations of hypotensive patients, respiratory distress, pulseless electrical activity, or in the setting of penetrating thoracic trauma. Patients with malignancies, end stage renal disease, AIDS, and rheumatologic and autoimmune diseases also may build up large volumes of pericardial fluid.

Pericardial fluid accumulation may be clinically silent, or the symptoms may develop slowly and be subtle. The most common presenting symptom is chest discomfort. The enlarging pericardial sac also may affect adjacent structures and result in a variety of symptoms including dysphagia, cough, dyspnea, prolonged hiccups, hoarseness, nausea, and abdominal fullness. Subacute presentations may be as vague as anxiety, dyspnea, fatigue, and altered mental status.

The clinical findings associated with a pericardial effusion are difficult to appreciate even in optimal settings. Muffled heart sounds may be difficult to hear because of rapid, loud breath sounds. Heart sounds also may be transmitted clearly through the thin chest wall of the neonate or small child. Pericardial rubs and pulsus paradoxus are not specific physical examination findings of pericardial effusion. Beck's triad of hypotension, muffled heart, and distended neck veins is considered a very late and non-specific finding. Distended neck veins may not ever occur in the hypovolemic patient.

Electrocardiogram findings of decreased QRS voltage and electrical alternans are not found commonly in patients with cardiac tamponade.

The sonographic appearance of a pericardial effusion is that of an echolucent area surrounding the heart. The pericardial space envelops the heart except at the AV sulcus. As mentioned above, the descending aorta on the PSLA view is an important landmark to the sonographer. It helps distinguish the echolucent pericardial fluid from the pleural fluid. Fluid in the pericardial space does not extend posterior to the descending aorta or left atrium. Any fluid seen posterior to the descending aorta is considered as pleural fluid. (*See Figure 4.*) The subcostal cardiac view offers the advantage of showing only the heart with the liver as the acoustic window. With the subcostal view there is no pleural reflection between the liver and the right-sided heart structures. Any fluid near the heart is pericardial or free abdominal fluid. Pericardial fluid will conform to the outline of the heart.

The presence of a pericardial effusion is a necessary, but not a sufficient condition for making the diagnosis of cardiac tamponade. The pericardial space normally holds up to 50 mL in the physically mature healthy adolescent. The pericardial sac can accommodate a gradual increase of pericardial fluid volume without significant alterations in intrapericardial pressure. Neonates can generate rapid heart rates to compensate for low, chamber-filling pressures. They may be able to maintain cardiac output even in the presence of cardiac tamponade. Rapid increases in pericardial volume, as in the setting of penetrating cardiac trauma, rapidly exceed the thinner-walled right atrial and ventricular end-diastolic wall pressures, and in this clinical scenario, small volumes of pericardial fluid can result in cardiac tamponade. Collapse of either right-sided chamber wall at end-diastole is diagnostic of cardiac tamponade.

Emergent pericardiocentesis or pericardial window placement is the definitive treatment.

Abdominal Sonography

Abdominal sonography in the pediatric ED is focused on the identification of three main intestinal abnormalities. Normally, the bowel is not the focus in abdominal sonography and may be a hindrance to other scanning goals such as aortic scanning. During the sonographic evaluation, a normal bowel displays peristaltic activity, and its walls are barely discernible. The internal substances, mixed with gas, can cast shadows that are not demarcated sharply. These are called "dirty shadows," which are in contrast to the "clean" shadows cast by stones and bone. The normal bowel is compressible with gentle transducer pressure on the abdomen.

Appendicitis. Appendicitis is the most common reason for abdominal surgery in children and presents a diagnostic challenge to even the most experienced clinician. Of children presenting to the pediatric ED with abdominal pain, 1-8% are diagnosed as having appendicitis.[3] The younger the child, the more challenging the diagnosis, with significantly higher appendiceal rupture rates reported in pediatric patients younger than 3 years when compared with the adolescent age group.[3,5] Fortunately, fewer than 5% of pediatric appendicitis cases occur in children younger than 5 years.

As the child grows, the appendix grows from an average length of 4.5 cm in infants to 9.5 cm in the adult. The shape also matures from a funnel shape to a more conical appearance with aging. Obstruction is less likely with the early, funnel shape.

The clinical presentation of children with appendicitis may lack specificity. Almost one-third of pediatric patients may not have a history of migration of pain from the periumbilical abdominal region to the right, lower quadrant, making the diagnosis more challenging. One out of every four patients with an eventual diagnosis of appendicitis was misdiagnosed initially due to misleading symptoms such as vomiting prior to abdominal pain, dysuria, upper respiratory symptoms, or diarrhea /constipation symptoms.[5] The morbidity and mortality of a ruptured appendix is much higher than for surgical removal of an unruptured appendix.

Plain radiographs of the abdomen are rarely diagnostic, (an appendicolith in the right lower quadrant and/or a localized air fluid level suggesting a localized ileus may be helpful) and are insensitive and non-specific for appendicitis.

Computed axial tomography (CAT) scanning of the abdomen has been shown to be highly sensitive and specific for the diagnosis of appendicitis, although there continues to be concern about radiation doses from abdominal/pelvic CTs in children. Currently, centers vary in their use of oral, intravenous, or rectal contrast. The use of oral contrast may result in diagnostic delays and staff challenges, especially with an ill child. Centers that use non-enhanced or non-contrast CT scans have shown that the interpretation by attending radiologists produces sensitivity, specificity, and accuracy results of 95-100%, 98-100%, and 97-99%, respectively. The residents' non-enhanced CT interpretation of appendicitis showed a sensitivity of 63%, specificity of 96%, and an accuracy of 88%.[6-9] In addition, use of the non-enhanced CT reduces time delays, and the test takes only several minutes to perform.

Graded compression US showed a sensitivity of 100%, specificity of 88%, and an accuracy of 91% in one series.[8] Other studies have demonstrated greater variability in accuracy, with some studies supporting these results, and a single study showing less favorable sensitivities of 79%, specificities of 82%, negative predictive value of 88%, and positive predictive values of 65%.[10-12]

Graded compression US is performed using a linear transducer, although appendicitis has been visualized during the right adnexal views of pelvic transvaginal sonography.

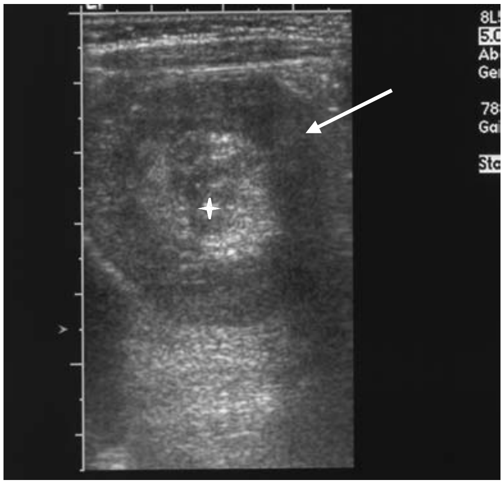
The normal appendix is visualized uncommonly by sonography, and when visualized, is compressible with manual or tranducer pressure on the abdomen, and is usually fewer than 6 mm in thickness.[13] Procedurally, the scan should be conducted with slow, but firm, pressure applied a few centimeters to the right of the umbilicus (displace underlying bowel), and then move down the abdomen at one-half centimeter increments until the right lower quadrant of the abdomen is scanned thoroughly. The other option is to allow the patient to place the transducer over the area of most pain/tenderness.

The inflamed appendix, in cross-section, has an edematous wall. The thick wall appears echolucent. A wall thickness greater than 3 mm (e.g., diameter more than 6 mm) is considered one of the main criteria for the sonographic diagnosis of appendicitis. A target sign consisting of five concentric layers is found in slightly more than 50% of cases of non-perforated appendicitis. There is usually no peristaltic movement. Compare this with the adjacent bowel. View the structure in longitudinal section. It should elongate into, and end as, a blind loop. There is central echogenicitiy from apposed inner mucosal lining. There may be a central echolucency representing fluid in the lumen of the appendix. A fecalith may add a central echogenic focus with posterior shadowing.

In one study, the appendix was not seen on US in up to 10% of the cases. Non-visualization of the appendix accounted for nearly all of the false negative studies.[13] A ruptured appendix may be decompressed sufficiently to succumb to transducer pressure and identification. Peri-appendiceal fluid or purulence may be echolucent, and the central appendix can appear as an echogenic tube with a blunt end, emerging from the larger cecum. Edematous surrounding fatty tissue is echogenic. The application of color flow signal can be of some assistance. The absence of color flow signal over the appendix can be found in both the normal and abnormal appendix. Increased vascularity shows up as a ring of fire. This sign helps support the diagnosis of appendicitis. Sonographic features associated with a perforated appendix include: fluid within the non-compressible appendix; thickened bowel loops with minimal peristaltic activity; inhomogeneous pericecal/appendiceal echogenic mass; or echolucent fluid.[13] A retrocecal appendix is beyond the purview of the linear transducer.

US may be used initially to rule *in* appendicitis in equivocal and high pretest probability cases. It has no radiation exposure, no IV contrast risks, and is non-invasive. Non-visualization of

Figure 5. Intussusception



Abdominal scan of a 4-year-old boy shows a transverse view of thickened bowel of intussusception. The distal receiving bowel segment is edematous and echolucent (arrow). The mucosal and muscular layers of the infolded bowel forms the echogenic center (star).

the appendix by sonography should not be interpreted as "negative for appendicitis" or as a "normal appendix." In one study, a normal appendix was visualized by sonography in only two out of 83 pediatric patients without appendicitis, whereas CT identified a normal appendix in 62 of 74 patients without appendicitis.[14] Due to the range of sensitivities and specificities of US with appendicitis, the positive and negative likelihood ratios should be applied cautiously to the clinical pretest probability of appendicitis. The desired outcome in this clinical decision-making scenario is to move one's post-test probability below the threshold for further diagnostic imaging or above the threshold for surgical treatment or admission.[15] Regardless of the pretest probability of appendicitis, if the clinician wishes or feels compelled to rule out appendicitis, then a negative or equivocal US reading should prompt further testing. In the majority of cases this would include surgical consultation or a CT scan.

Positive sonographic findings for appendicitis should result in enough evidence in moderate to high pretest probability cases for surgical consultation for definitive management or a CT of the abdomen and pelvis. Ultrasound's negative likelihood ratio range for detecting appendicitis does not support its use in dismissing the diagnosis of appendicitis at any level of pretest probability. In a prospective study of 139 pediatric patients with equivocal clinical findings of acute appendicitis, US was the first imaging modality in the protocol. It was followed by limited CT with rectal contrast if the US was indeterminate or negative. If the US reading was positive for appendicitis, the patient went to the operating room. This protocol resulted in a sensitivity of 94%, specificity of 94%, and accuracy of 94%. US should be used to confirm the diagnosis of appendicitis, but should not be used to exclude the diagnosis.[14]

Pyloric Stenosis. Hypertrophic pyloric stenosis (HPS) is the most common pediatric surgical cause of vomiting. This form of gastric outlet obstruction occurs in one out of every 250 births with a male to female ratio of 4:1. The diagnosis of HPS must be considered in the young infant who presents within the first 10 weeks of life with frequent dramatic post-prandial emesis. The peak age for presentation is 3-6 weeks. The emesis is classically forceful, non-bilious and occurs immediately at the end of feeding to within 30 minutes after. Early in the disease course, the infant is aggressively hungry after vomiting, but after several days may become lethargic from dehydration, electrolyte disturbances, and metabolic alkalosis.

Clinically, these children may present with varying degrees of dehydration, based upon the length of the illness and the severity of the symptoms. The infant with a palpable, olive-sized mass in the epigastric area can be diagnosed confidently with HPS, but the insensitivity of this physical examination finding usually prompts further diagnostic testing. A definitive diagnosis of HPS may be achieved by a skilled sonographer.

Just lateral to the midline of the abdomen, at the subxyphoid region, is where the enlarged pylorus is palpated usually. A linear 7 MHz or higher frequency probe is used to scan this region. The walls of the hypertrophied circular muscles of the pyloric sphincter appear echolucent and thick. The central mucosa is echogenic. A wall thickness greater than 4 mm is diagnostic of pyloric stenosis. Several studies show 100% specificity and 86-92% sensitivity of the 4 mm muscle wall thickness criterion. Other parameters such as muscle length (e.g., greater than 2 mm) and channel length (e.g., greater than 17 mm) also are used.[16-21]

The immediate and crucial goals of medical management are correction of volume deficits and electrolyte abnormalities. The definitive care of the pediatric patient identified with HPS is surgical, with a pyloromyotomy providing a full correction of the condition.

Intussusception. Intussusception occurs when a proximal segment of bowel, usually the terminal ileum, invaginates into a distal segment of bowel pulling portions of mesentery with it. Incidence is estimated at 2.4 cases per 1,000 births.[22] Although this condition occurs mainly in infancy, prior to the age of two years, it also may occur in older children, adolescents, and adults. In fewer than 10% of cases, intussusception may be initiated by an anatomical lead point such as polyposis or a neoplasm, but most commonly it is idiopathic. Older children have a higher incidence of an anatomic lead point. Closely associated with viral illnesses such as upper respiratory infections and gastrointestinal illnesses, the occurrence of intussusception peaks during the viral illness peaks of summer and winter.

Patients typically present with intermittent, episodic abdominal pain, vomiting, and a guaiac positive stool. In some cases, lethargy during the pain-free intervals may be the only clinical clue to the condition. Physical examination findings vary and may include a sausage-shaped, right-sided mass and abdominal tenderness.

Abdominal radiography is not a reliable screening tool. Radiographs may be normal, show bowel obstruction, no stool in the distal colon, or very little intestinal gas. In some centers, the enema is the first option for its dual benefits of diagnostic imaging and therapeutic intervention. Others consider sonography to be an ideal non-invasive, screening diagnostic modality that limits radiation exposure to children. Using the high frequency linear probe will reveal a target or bull's eye lesion in the tranverse scan plane. The hypoechoic rim represents the edematous bowel wall of the receiving end (i.e., intussuscipiens) infolded bowel loops. The central echogenicity represents the mucosal surfaces and muscular wall of the infolded bowel (i.e., intussusceptum). A transverse scan of the proximal segment of intussusception may have a central anechoic region (bull's eye), giving it a target-like appearance. (*See Figure 5*.) On the longitudinal view, edematous bowel encasement is oval (i.e., it looks like a renal cortex), and the central echogenicity resembles that of a sinus of a kidney, hence the term "pseudo kidney" sign.

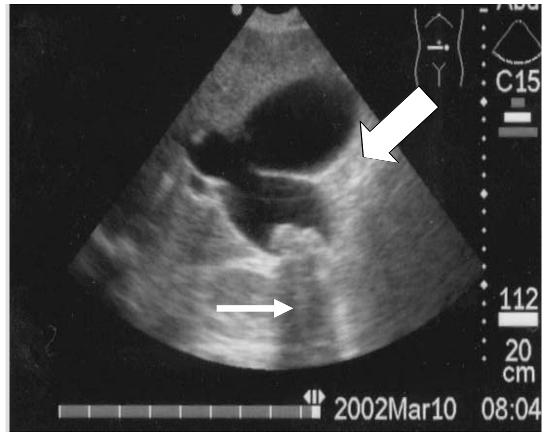
The sonographic identification of invaginated and edematous bowel wall in the pediatric patient suspected of intussusception can be treated rectally with the hydrostatic pressure of fluid or air. An enema alone may provide diagnostic and therapeutic benefits. Ultrasound guidance can be used also. Surgical involvement may be indicated if reduction attempts are unsuccessful.

Gallbladder Disease. Biliary tract disease and gallbladder abnormalities should be suspected in patients with upper abdominal pain and nausea and vomiting, or abdominal tenderness and jaundice.

A 3.5-5 MHz transabdominal probe should be used in the small child. If the patient is old enough and able to cooperate, ask the child to hold his/her breath. This will move the liver below the costal margin to provide an excellent acoustic window. Imaging also may be improved by rolling the patient onto his/her left side with the shoulders vertical. If this is not possible, then scan between the ribs, although this is a very limited scan area.

The non-contracted gallbladder (GB) is visualized with careful attention to key surrounding landmarks. It is usually inferior to the liver, anteromedial to the superior pole of the right kidney, and anterior to the echogenic walled portal vein. The main lobar fissure connects the GB to the right portal vein. The normal gallbladder has smooth echogenic walls, no internal echoes, and posterior acoustic enhancement. Normal variants include septations near the GB neck, Phrygian cap (e.g., folded fundus), Hartman's pouch (e.g., folded neck), agenesis, or duplicate GB. The GB usually is contracted for a few hours after a meal. The normal cystic duct can be 1-2 cm in length and 1.8 mm in width. It is uncommon to visualize the cystic duct by sonography. The GB measures 3.0-3.2 cm in the infant, then matures to 7-10 cm at adulthood. The GB width is usually one-third the length. The GB should be viewed in both longitudinal and transverse planes. Posterior acoustic enhancement makes it difficult to measure the GB wall thickness at the posterior GB wall. Use only the anterior GB wall to assess or measure wall thickness. The posterior wall measurement is compromised by posterior acoustic enhancement artifact. Gallbladder wall thickness more than 4 mm is considered indicative of wall inflammation from a diversity of etiologies.

Figure 6. Gallstones



A longitudinal gallbladder scan of an adolescent with sickle cell anemia and RUQ pain. The gallbladder folded on itself (thick arrow). The gallbladder wall is not thickened. Gallstones show posterior shadowing (thin arrow).

Cholelithiasis is uncommon in the preadolescent patient unless a predisposing, comorbid condition exists. Gallstones commonly are found in pediatric patients with sickle cell disease and may be found in the pediatric patient with hemolytic anemia, liver disease, Crohn's disease, cystic fibrosis, total parenteral nutrition, and as an uncommon side consequence of certain drug use.[23] If cholecystitis is suspected, the main sonographic features focus on the GB wall. The presence of gallstones is not sufficient evidence to state that gallstones cause the patient's condition. Gallstones usually are echogenic structures on the inner wall of the GB. GB polyps are also echogenic. Polyps remain attached to the GB wall, whereas the majority of gallstones move to the gravity-dependent areas when the patient's position is changed. Polyps do not produce shadows. When gallstones are 3 mm or larger, they produce posterior shadowing. The shadows produced by gallstones are "clean shadows" (i.e., sharp edges and free of internal echoes). Small, non-shadowing echogenic structures that move when the patient is repositioned are considered as stones. (*See Figure 6*.)

Another possible finding within the GB is sludge. Normal bile is completely echolucent. Sludge is considered modified bile. It has midlevel echogenicity (gray). It is viscous, does not produce shadows, is gravity-dependent, and conforms to the walls of the GB. Its clinical significance is unclear but has been noted to exist in situations of bile stasis and GB infection. It also has been noted to be a precursor of sludge balls, which have the ability to cause GB obstruction.

Cholecystitis —inflammation with or without infection of the GB wall— may be found in the absence of gallstones in the young patient. In contrast, acalculous cholecystitis is a rare occurrence in the adult patient. As the GB wall thickens, the inflammation may appear as an echolucent, middle-wall layer.

(*See Figure 7.*) Advanced inflammation, extending throughout all the wall layers, can decrease the echogenicity of the entire GB wall. The GB may become distended if the cystic duct is obstructed completely. Wall thickness greater than 5 mm is considered abnormal. The ultrasonographic Murphy's sign, although reported to have poor sensitivity and specificity, may provide additional information. Use the probe to apply pressure to the right, upper quadrant as the patient takes in a deep breath. If maximal tenderness is elicited when active GB compression is displayed on the monitor, then that is considered a positive Murphy's sign. Nuclear medicine tests are more reliable than US for the diagnosis of acalculous cholecystitis. Choledochal cyst is a congenital dilation of the common bile duct. This cyst may cause intermittent pain, vomiting, and a palpable mass in the pediatric patient. The biliary obstruction may cause jaundice. US is not useful in confirming or dismissing a diagnosis of biliary atresia, an obstructive disorder that needs rapid diagnosis and surgical intervention in the neonatal patient.

Common Bile Duct. The common bile duct (CBD) is important to assess. It is formed after the union of the cystic duct and the common hepatic duct. The CBD is joined eventually by the pancreatic duct before it enters the duodenum. It runs anterior to the portal vein and, for a part of its course, runs parallel to it on the longitudinal scan view. The probe is positioned at the right costal margin at the mid-clavicular line. The patient can be supine or turned 45° onto the left side. Keeping the probe in the transverse position, slowly angle from the left hip to the right shoulder until the portal vein's echogenic walls are noted. Decreasing the depth setting of the machine provides more detailed images. Slight rotation of the probe will elongate the portal vein and its immediately anterior CBD. The normal echolucent bile duct lumen is narrower than the portal vein's lumen. Slight rotation of the probe will obtain a transverse view of the portal vein and the hepatic artery and common bile duct lumina. It has the appearance of a silhouette of a round face with two small ears. The enlarged CBD will give an asymmetry to the ears. Measurements of the bile duct are made from inner wall to the inner wall perpendicular to the long axis of the CBD. Patients with symptoms determined to be due to cholelithiasis and/or cholecystitis should be admitted for pain control, observation, or antibiotic administration.

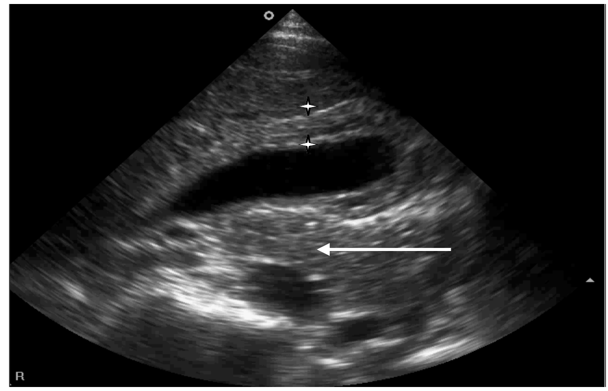
Pediatric Renal Sonography

Bedside US is useful in the pediatric patient presenting with acute urinary retention, renal colic, acute renal failure, acute pyelonephritis, possible renal abscess, or renal trauma. The focused application of emergency US in the pediatric patient presenting with flank pain is determination of the presence or absence of hydronephrosis.

The history should focus on factors that predispose to renal disease such as inborn errors of metabolism or other metabolic disorders, history of renal calculi, or urinary tract infections. Also, query patients or primary care providers regarding flank pain, trauma to the kidney, recent streptococcal infections, colicky abdominal pain, change in urinary output, urinary frequency, dysuria, retention, or hematuria in the child. A family history of renal disease, such as polycystic kidneys, calculi, or renal failure, also may help direct the diagnostic evaluation. On physical examination, pay particular attention to vital signs suggesting the distress of pain and abdominal or flank tenderness. Laboratory examination should include an examination of renal functioning, urinalysis, urine microscopy, and crystals.

Renal Sonography. Emergency renal sonography requires evaluation of both kidneys and the bladder in the longitudinal and transverse planes. Utilizing a low frequency probe (e.g., 3.5-5 MHz) and B-mode sonography, place the patient in the supine or lateral decubitus position. The kidneys are located anatomically in the retroperitoneum and are surrounded by adipose tissue. Both are bound supero-posteriorly by the dome of the diaphragm. Typically, the right kidney is imaged inferior to the costal margin in approximately the anterior axillary or mid-axillary line. The right kidney is slightly larger and more inferiorly displaced than the left. The right kidney is easier to image sonographically as the liver serves as an optimal acoustic window, while the left kidney is bound by the spleen, pancreas, stomach, and small and large intestines, creating more of a challenge. Identify the superior and inferior poles of the kidney with particular attention to the renal parenchyma (e.g., cortex and medulla) and the sinus into which the medullary pyramids/papillae drain to form the pelvis and travel with the renal artery and renal vein at the hilum. Scan in a longitudinal plan and obtain images of both the superior and inferior poles. Rotate the transducer 90° to obtain the images in the transverse plane. For the left kidney, place the transducer inferior to the costal margin at the level of the posterior axillary line. Scan the bladder in both planes with the transducer placed just superior to the pubic bone.[24]

Figure 7. Acalculous Cholecystitis



Longitudinal view of the gallbladder shows no gallstones, but the gallbladder wall is markedly thickened. Measurements are made of the anterior gallbladder wall (see markers) Posterior acoustic enhancement effect (arrow) makes it difficult to determine the outer edge of the posterior wall.

Table 1. Performing a Bedside Renal Ultrasound

PATIENT COMFORT:
- Pain control
- Supine or lateral decubitus position
- Isolate testicles propped on towels

LOW FREQUENCY PROBE

IDENTIFY STRUCTURES
- Each kidney in transverse and longitudinal planes
- Bladder in transverse and longitudinal planes
- Look for ureteral jets

Table 2. Sonographic Mimics of Hydronephrosis

- Renal cysts
- Prominent medullary pyramids
- Full bladder
- Pregnancy

A white, echogenic capsule—with less echogenic parenchyma than that of liver parenchyma— surrounds each kidney. The less echogenic and darker medullary pyramids merge to form the pelvis, which is black and hypoechoic, consistent with fluid-filled structures. The centrally located sinus appears white to gray in color, secondary to fat content. The bladder appears as an anechoic fluid-filled vessel surrounded by a thin, echogenic white line. On the transverse view, the ureteral jets marking urine flowing into the bladder from the ureters may be viewed using color flow Doppler.

Hydronephrosis appears as anechoic fluid-filled pockets prominently displayed in the central sinus. Hydronephrosis is graded as mild, moderate, or severe based upon the EP's judgment performing the study. Occasionally, a calculus will be viewed within the kidney parenchyma. If hydronephrosis is viewed at the level of the kidney, evaluate for presence or absence of ureteral jets on bladder view. Absence of a ureteral jet on the affected kidney side is highly consistent with an obstructive calculus.

Commonly confused with hydronephrosis is prominent renal pyramids. This is due to the central location within the kidney and its similar sonographic appearance. To distinguish prominent pyramids from hydronephrosis, look for a triangular shape consistent with a pyramid, and look for intervening renal cortex.

If an abnormality of the kidney such as a duplicated collecting system, horseshoe kidney, a pelvic kidney, or congenital absence of a kidney, is recognized, order a formal US performed by a radiologist. Do not lose sight of the focused, clinically applicable question.

Radiological Imaging in the Pediatric Patient. There is no gold standard for evaluating hydronephrosis in the pediatric patient. Sonography is accepted as an initial study for establishing the diagnosing hydronephrosis. A combination of plain radiographs, intravenous pyelogram (IVP), voiding cystourethrography, and nuclear scintigraphy may be utilized to evaluate the hydronephrosis further. Although sonography does not provide information on kidney functioning or stone location, the EP can decide if there is hydronephrosis and how to proceed clinically in evaluating an individual patient. Sonography also may be useful for locating cysts and differentiating renal masses, but has limited applicability in the ED.[24] US may be falsely negative if the patient is dehydrated, and falsely positive if the patient's bladder is full, causing bilateral hydronephrosis. Repeat the scan after rehydration or voiding. Unilateral hydronephrosis, especially on the right side, may be seen in pregnant patients.[24] The bedside US plus plain, abdominal radiograph (KUB) is considered as sensitive as intravenous pyelogram (IVP) in screening patients with suspected ureteral colic.[24-27] Prospective data support the use of contrast-enhanced magnetic resonance imaging (MRI) for the evaluation of hydronephrosis in children, but neither is time nor cost effective for most EPs.[28,29] Studies comparing sensitivity and specificity of US versus non-enhanced helical CT focus on the adult not pediatric population.[30] Although IVP allows detection and location of renal calculi, its relative disadvantages include: placing an IV in an already agitated pediatric patient; awaiting laboratory results of renal function; a radiologist to administer the dye and perform the examination; and contraindications of pregnancy, contrast allergies, and renal insufficiency.

Renal Calculi. Pediatric and adolescent patients do present with renal calculi although the incidence is approximately 1/50 that of adults. Ninety percent of patients have hematuria, and 50% have preceding abdominal or flank pain.[31,32] Renal calculi in the pediatric population commonly result from hypercalcuria and often are genetic.[31,33] Infection-induced stones are more common in younger children, and are 80% more prevalent in boys. A small percentage of stones are secondary to cystinuria from hereditary disorders of amino acid metabolism and also secondary to uric acid with hyperuricemia or hyperuricosuria from increased purine synthesis.[32] In North America, children and adolescents with metabolic disease account for approximately 50% of kidney stone presentations. In Europe, the majority of kidney stones are infection related, while in Southeast Asia, uric acid bladder stones are most prevalent. Unlike the male predominance in adults, kidney stones present equally in boys and girls.[32,34] After the ED assessment described above, utilize bedside sonography to assess the kidneys expediently. Scan the bladder with color Doppler and evaluate for ureteral jets. If obstruction is suggested by sonography, urgent urologic consultation is recommended.

Acute Renal Failure. Renal sonography is the radiological adjunct study of choice to differentiate post-renal from intrarenal or pre-renal acute renal failure (ARF). Hydronephrosis is evident sonographically if the cause of ARF is obstructive in nature. In chronic renal failure, kidneys appear sonographically small, atrophic, and hyperechoic.

Renal Cysts. Sonographically, renal cysts are seen quite frequently. Do not mistake them for hydronephrosis. Cysts appear round or oval-shaped with a thin wall, a clear interface with the surrounding renal parenchyma, and are located eccentrally. Simple cysts contain clear fluid without internal echoes and demonstrate mild, posterior enhancement. Distinguish cysts from hydronephrosis by scanning to see that cysts do not connect centrally within the renal pelvis.[24] Suspect polycystic kidney disease when sonographically large cysts distort the normal renal architecture. In polycystic kidney disease, patients typically present with hypertension, flank pain, and hematuria.

Acute Pyelonephritis or Renal Abscess. The sonographic appearance of acute pyelonephritis is typically that of a normal kidney. If the infection has progressed to an abscess, the EP may view an oval or round, heterogeneous-appearing mass filled with fluid, septations, or debris.[24]

Renal Mass. EPs are increasingly detecting renal masses as incidental findings when performing bedside US in the ED.[35] The sonographic appearance of a renal mass is heterogeneous and varies in presentation from solid to cystic with variable echogenicity. Wilm's tumor or nephroblastoma is the most common intra-abdominal malignancy in childhood. Most patients present before age 5, and US is the radiographic modality preferred for initial localization.[36] Order a formal complete US performed by the department of radiology when a mass is viewed.

Acute Urinary Retention. The patient presenting with acute urinary retention, also may be evaluated with US. Initially, place the US probe over the bladder and obtain images in two planes. Estimate the size of the bladder in a small, medium, or large quantification. Image both kidneys to determine the presence of hydronephrosis. Based upon findings, place a urinary catheter for decompression or seek alternative causes. Most US machines have an automatic calculator setting for bladder volume measurement. Although of questionable accuracy, a rough estimate of bladder volume is obtained by multiplying length by width by height by 0.75.[37]

Renal Trauma. The use of sonography and typical findings in the pediatric patient presenting with renal trauma will be presented in a later article.

Summary and Pitfalls. Perform a limited, pediatric renal sonography when there is clinical suspicion of hydronephrosis. Order a formal, complete renal US for any incidental finding.

Conclusions

Ultrasound is a valuable diagnostic tool for the EP in the evaluation of cardiac, abdominal, and renal diseases. Its portability, safety, and accuracy suggest that this modality will become increasingly popular as a tool for screening pediatric patients for potentially serious disease processes. The EP should remain focused on the value of each test, the skills of the individual (i.e, EP or radiologist) performing the test, and associated limitations.

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Physician CME Questions

41. A 7-year-old male presents with three episodes of vomiting, abdominal pain, and fever during the past 24 hours. He has right lower quadrant tenderness. The genital exam is unremarkable. A right lower quadrant ultrasound is performed. Which of the following sonographic findings is most suggestive of appendicitis?
    A. A non-compressible target-like structure on transverse scan with no peristaltic activity
    B. A target-like structure on transverse, blind-ending loop on longitudinal section that is compressible
    C. A target-like structure on transverse view—diameter of 4 mm— with peristaltic activity
    D. Non-visualization of the appendix

42. A 14-year-old male presents to the ED with a history of 24 hours of abdominal pain. The radiologist says he can't visualize the appendix on ultrasound. The child is still very tender in the right lower quadrant. The most appropriate approach to this patient is:
    A. Send him home with follow-up in 24 hours.
    B. Repeat the ultrasound in 12 hours.
    C. Obtain plain radiographs of the abdomen.
    D. Order a CT scan of the abdomen.

43. Which of the following sonographic features is/are associated with a perforated appendix?
    A. Fluid within a non-compressible appendix
    B. Thickened bowel loops
    C. Inhomogeneous, pericecal echogenic mass
    D. Inhomogeneous, appendiceal echogenic mass
    E. All of the above

44. A 4-week-old white male presents with non-bilious projectile emesis. An ultrasound is conducted. Which of the following confirms the diagnosis of hypertrophic pyloric stenosis?
    A. A wall thickness greater than 4 mm
    B. A fluid-filled stomach
    C. A wall thickness of 1-2 mm
    D. Gastric peristalsis

45. Which of the following is true as it relates to screening cardiac sonography?
    A. The descending aorta on a parasternal long axis view is not helpful to differentiate between a pericardial and a pleural effusion.

CME Objectives

The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

a.) Quickly recognize or increase index of suspicion for specific conditions;

b.) Understand the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Be educated about how to correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed;

e.) Provide patients with any necessary discharge instructions.

B. The presence of a large, pericardial fluid collection confirms the presence of cardiac tamponade.
C. An athlete with recurrent exertional syncope and 15-mm thick ventricular walls on sonography can return to full practice drills.
D. Right ventricular hypertrophy can be a key sonographic feature in a neonate with congestive heart failure.
E. Fluid viewed between the liver and heart in the subcostal cardiac scan is pleural.

46. Which of the following statements regarding pediatric abdominal sonography is true?
A. The finding of gallstones in a patient with sickle cell disease confirms cholecystitis.
B. The anterior wall of the GB should be used to assess gallbladder wall thickness.
C. Edematous bowel walls are echogenic sonographically.
D. The sonographic features of pyloric stenosis are detected most easily using a low frequency abdominal probe.
E. The liver impedes proper visualization of the gallbladder and the right kidney.

47. The primary clinical indication for bedside renal sonography is detection of a renal calculus.
A. True
B. False

48. Which of the following describes the sonographic appearance of a renal cyst?
A. Centrally located
B. Ill-defined borders
C. Irregularly shaped
D. Fluid filled
E. Connects centrally with other cysts

49. Which of the following is/are *not* true regarding intussusception?
A. Ultrasound may be used as a screening tool.
B. Abdominal radiographs are usually diagnostic.
C. Children may present with lethargy.
D. Enemas may be diagnostic and therapeutic.
E. All of the above

50. Which of the following conditions predisposes a child to the development of gallstones?
A. Sickle cell disease
B. Inborn errors of metabolism
C. Hemolytic anemia
D. Total parenteral nutrition
E. All of the above

Answer Key

41. A	42. D	43.E	44. A	45. D
46. B	47. B	48.D	49.B	50. E

In Future Issues: **Pediatric Obesity**

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Pediatric head injuries are common occurrences with the potential for serious morbidity or mortality. Fortunately, the incidence of traumatic brain injury (TBI) has been declining, mainly because of the development of effective prevention strategies (e.g., car seats and bicycle helmets). Although it is difficult to determine the exact incidence of head trauma in children due to variations in definitions and classifications, the majority of head injuries in children are minor and result in no long-term morbidity or mortality. However, early identification of a potentially serious injury and aggressive management of a child with a head injury facilitates the optimal possible outcome. The topic of pediatric TBI is extensive, and the majority of information is very familiar to the practicing emergency department (ED) physician. The author discusses two areas of controversy — patient selection for imaging and an update on management strategies for children with TBI. Selecting patients who require imaging following head trauma is easy if the child has an abnormal mental status or a Glasgow Coma Scale (GCS) score less than 15; he or she needs a head CT scan. The challenge is identifying high-risk patients with a GCS score of 15. The author reviews the available literature and presents currently available guidelines. Since TBI is the leading cause of death and disability, aggressive management of a child with a TBI is critical. The author reviews available therapies and their current application to pediatric patients.

—The Editor

Pediatric Controversies: Diagnosis and Management of Traumatic Brain Injuries

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Reviewer: **Mary Jo A. Bowman, MD,** Associate Professor of Clinical Pediatrics, Ohio State University College of Medicine; Attending Physician, Columbus Children's Hospital, Columbus, OH.

Introduction

Trauma is the leading cause of childhood death,[1] and TBI is the leading cause of death and disability for children who sustain trauma.[2] Each year, more than 400,000 children younger than 14 years have emergent evaluations for head trauma.[3,4] Children younger than age 4 have considerable morbidity from head trauma. This age group has a prevalence of TBI that is more than twice the rate of the general population and nearly twice the rate for older children.[4] In addition, recent research indicates that even "minor" trauma may have the potential to result in life-long sequelae.[5,6] Thus, when evaluating children with head trauma, the practitioner must determine which patients are at risk, based on their history and physical exam, for significant injury requiring diagnostic imaging, careful monitoring, and aggressive intervention.

Evaluation of Children with Accidental Head Trauma

Injury patterns vary by the age of the child, with older children most likely sustaining an injury while participating in sports or when involved in motor vehicle collisions. However, children younger than 4 years most commonly sustain TBIs as a result of falls, motor vehicle collisions, or abuse. In the younger child, contact head injuries, such as linear skull fractures, hematomas, and cerebral contusions, can occur as the result of short, vertical falls.[7,8]

One study found that children who fell from a greater height were more likely to have injuries, but a number of patients had skull fractures or brain injury following falls from heights of less than three feet.[9] When there is a contact injury to the head, the point of impact causes the inner table of the skull to bend inward, which may injure blood vessels within the epidural or subdural space, as well as the parenchyma of the brain itself.[10] At the same time, there is also simultaneous outbending of the skull around the site of impact.[10] This puts the outer table of the skull under tension, and a fracture may result, either proximate to, or remote from, the site of impact. Children who sustain isolated skull fractures typically do not present with significant alterations in mental status, unless there is underlying brain injury with mass effect.[10]

Children Younger than 2 Years

Children younger than 2 years have been thought to be at high risk for significant brain injury after accidental head trauma.[11] Earlier studies often did not have enough data in the youngest age groups to recommend anything except a very cautious approach to evaluating head trauma in children younger than 2 years.[12,13] It has been estimated that the overall rate of brain injury after trauma in children younger than 2 years is approximately 5%, but infants younger than 2 months may have the highest prevalence of brain injury.[5]

Two studies in 1999 both evaluated infants younger than 1 year who presented to the ED with accidental head trauma.[5,9] The prevalence of brain injury was 12% in the 0-2 month age group, 6% in the 3-11 month age group, and 2% in infants older than 12 months.

Controversy exists in the literature regarding the ability of the physician to use clinical signs and symptoms to identify children at risk for brain injuries following blunt trauma. Obtaining an accurate history and a complete neurologic exam may be challenging, especially in younger children. Children younger than 2 years have been particularly identified as having subtle clinical presentations.[5] In addition, a computerized tomography (CT) scan of the head has disadvantages, including exposure to radiation, transport of the patient out of the ED, and a frequent requirement of sedation.[14-16]

Scalp Hematomas. Greenes and Schultzman sought objective markers of the presence of TBI and identified significant scalp hematomas as strongly associated with a skull fracture and brain injury in children younger than 2 years.[5] Another study also found the presence of a scalp hematoma to be the most important predictor variable for TBI identified on CT scan (e.g., intracranial hemorrhage, hematoma, or cerebral edema), in children 2 years and younger.[17] Finally, Greenes and Schutzman (2001) evaluated children younger than 2 years who sustained accidental head trauma, but had no neurological signs or symptoms.[18] The size and location of the scalp hematoma (e.g., parietal and temporal), and age younger than 3 months were each associated with skull fractures. This study also found that a skull fracture, large hematoma, and parietal location were associated with brain injury.[18] Children without a history of neurological symptoms and with a normal scalp exam were identified as a low-risk group.[9]

Abnormal Mental Status. Other series have examined the ability of an abnormal mental status to predict an abnormality on CT. Palchak et al found that of 194 children age 2 years and younger, all 15 children with a TBI on CT were predicted by the presence of a scalp hematoma and an abnormal mental status (sensitivity 100%; 95% CI 81.9—100%).[17] Of the 60 chil-

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dren in this series age 2 years and younger who underwent CT and had a normal mental status examination and no scalp hematoma, none had a TBI identified on CT scan (negative predictive value 100%; 95% CI 95.1—100%). Lethargy, irritability, full or bulging fontanel, and vital signs suggestive of increased intracranial pressure (ICP) also have been associated with brain injury, while vomiting and loss of consciousness, at least in this age group, were not.[5]

Skull Fractures. Palchak et al found that of the 194 children age 2 years and younger who underwent CT scan, 15 (7.7%) had skull fractures on CT, and 46.7% had an associated TBI identified on CT.[17] Another study reported on 102 infants younger than 13 months with skull fractures. Fifteen of the 102 patients were found to have a brain injury. The authors found that patients with lethargy prior to presentation or in the ED and patients with parietal fractures were more likely to have sustained a brain injury.[19]

Guidelines. A multidisciplinary panel of nine experts in pediatric head trauma was convened.[20] All evidence gathered from a Medline search was reviewed, and using a modified Delphi technique, a set of guidelines for the evaluation of children younger than 2 years with minor head trauma was developed. Among the guiding principles the panel recommended were the following: One should have a lower threshold for diagnostic imaging in young children, with children younger than 12 months being at higher risk and children younger 3 months being at the highest risk for intracranial injury after head trauma; the greater the number and severity of signs and symptoms, the stronger the consideration should be for obtaining a CT. The greater the forces involved, the more pronounced the physical findings (e.g., scalp swelling), and the younger the age, the greater the risk for intracranial injury.

Specifically, the panel stratified the patients into risk categories based upon clinical features (e.g., history and physical examination), mechanism of injury, and absence/presence of a skull fracture.

High-risk patients had any of the following characteristics: depressed mental status, focal neurologic findings, signs of depressed or basilar skull fracture, seizure, irritability, acute skull fracture, bulging fontanel, vomiting greater than five episodes or for more than six hours, and loss of consciousness greater than one minute. All high-risk patients required a cranial CT scan.

Intermediate-risk patients had any of the following characteristics: vomiting three to four times; loss of consciousness less than one minute; history of lethargy or irritability, now resolved; caretakers concerned about current behavior; higher force mechanism; hematoma (especially large or nonfrontal in location); unwitnessed trauma; fall onto a hard surface; vague or no history of trauma with evidence of trauma; and nonacute skull fracture older than 24-48 hours. Patients in this category could be managed in one of two ways: a period of observation (4-6 hours recommended) and reevaluation, or a head CT scan.

Low-risk patients were defined as having low-energy mechanism (e.g., fall less than 3 feet), no signs or symptoms, and more than two hours since the injury; also, the panel found that as the patient's age increases, the risk decreases. These patients may be observed in the ED or at home with reliable caretakers.[20]

Apart from these findings and the panel recommendations, evidence exists suggesting that the youngest age group is more likely to have brain injury with no neurological findings.[6,21,22]

Children Older than 2 Years

For older children, it is easier to obtain historical information and an accurate physical examination. Many series have sought to determine historical factors and clinical features that are predictive of an intracranial injury. A recent prospective study found that in 2043 children younger than18 years with head trauma, an abnormal mental status, clinical signs of skull fracture or scalp hematoma (in patients younger than 2 years), history of headache and vomiting were predictive of intracranial injury.[17] The most important variable in this series was clinical findings of a skull fracture.

These five clinical findings identified 97 (99%; 95% CI 94.4—100%) of the 98 children with TBI on CT scan and all 105 children with TBIs that required acute intervention. Of the 304 (24%) children with CT scans who didn't have any of the five predictors, only one had a TBI on CT scan (0.3%; 95% CI 0—1.8%). Of the 825 patients who had none of the five predictors, no one had a TBI requiring acute intervention (negative predictive value 100%; 95% CI 99.6%—100%). Use of this rule would have decreased CT scan utilization by approximately 25%.[17] Similarly, another study found that children older than 2 years with closed head trauma who were neurologically normal and had no clinical signs of skull fracture could be managed safely without cranial CT.[23]

In 1999, the American Academy of Pediatrics published guidelines for the management of closed head trauma in previously healthy children 2-20 years of age.[24] This consensus statement used the historical features of loss of consciousness and the presence of symptoms as an indication for obtaining a CT scan of the head. For those children without a loss of consciousness, a thorough history and physical examination should be performed, and a competent caregiver should observe the patient for any deterioration in mental status. For those who have a history of a brief loss of consciousness, along with amnesia, headache or vomiting at the time of evaluation, the prevalence of intracranial injury may be as high as 7%.[25-27] Though many of these brain injuries may have little clinical consequence, a minority of these children may require neurosurgical intervention.[26-28] Therefore, in this group of symptomatic children with a brief loss of consciousness, CT scanning of the head may be useful. However, with a brief loss of consciousness alone in an otherwise asymptomatic patient, observation of the patient for neurological deterioration may be an acceptable alternative to obtaining a CT scan of the head.[24]

While CT scanning is usually a safe procedure, some children may require sedation to obtain the study. Therefore, one must consider the risks of sedation against the benefits of obtaining a CT scan in this group of asymptomatic patients.

Once a TBI has been detected, the type of facility where the child will be evaluated and treated is important to the recovery. Several studies have examined the impact of pediatric trauma centers on the initial management of pediatric trauma. One study evaluated the morbidity and mortality rates among pediatric trauma victims in Pennsylvania and found that morbidity and mortality from TBI was reduced significantly in patients who were treated at pediatric trauma centers.[29] More neurosurgical procedures were performed in pediatric trauma centers, and there was concomitant lower mortality from TBI.[29] Another study found that the mortality rate was significantly higher for children with TBIs who were first transported to non-pediatric hospitals and subsequently transferred to pediatric trauma centers.[30] Thus, it is important that children with brain injuries be transferred to the nearest pediatric trauma facility as soon as it is feasible.

The guidelines for the acute management of severe TBI in infants, children, and adolescents made transfer to a pediatric trauma center a guideline based upon class II data (prospective and retrospective observation, cohort, and case control) and strong class III data (retrospective), and, as an option, an adult trauma center with qualifications for pediatric treatment.

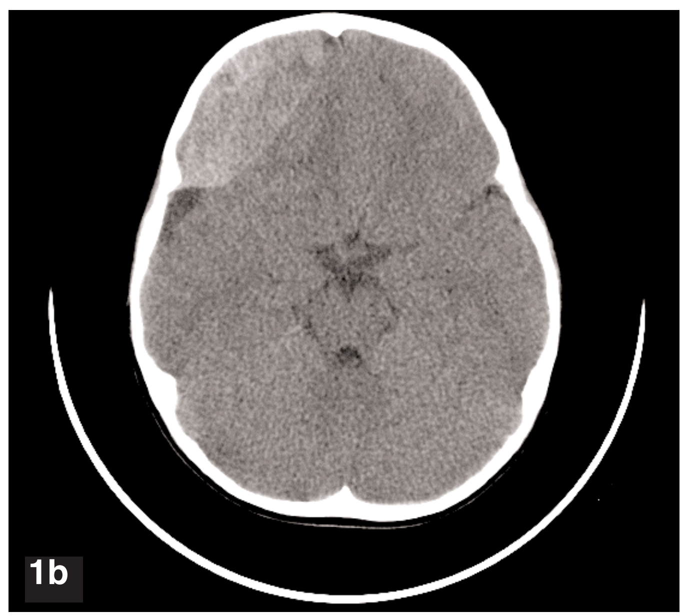
Management of Intracranial Injuries

Group 1: Asymptomatic Intracranial Injuries. The optimal management and outcome of children who have intracranial injury as detected by CT scan, but who are otherwise asymptomatic, is controversial. Typically, such children are admitted to the hospital for close neurological assessment and monitoring. Many pediatric neurosurgeons have adopted an approach of expectant management for small intracranial and extradural hematomas, taking into consideration the size of the hematoma, its propensity to increase in size, shift of midline intracranial structures and surrounding cerebral edema.[24] (*See Figure 1.*) In some cases, children with subdural hemorrhage from minor trauma may do quite well with expectant management. Four patients were reported with unilateral subdural hemorrhage, of which three occurred from minor trauma and one from a fall out of a window. In all four cases, the subdural hemorrhage resolved spontaneously within 48 hours of injury.[31]

Critical to the management of children with an acute TBI is the initial assessment of the child's neurologic status and ongoing monitoring. Standardized assessment scores are the most accurate for detecting subtle changes in a patient. The GCS is useful for repeated neurological assessments in children with TBI. (See *Table 1.*) In one study, the most important prognostic indicators for pediatric TBI were demonstrated: the presence of associated trauma, admission GCS scores, traumatic mass lesions with ICP, and the presence of diffuse axonal injury.[32] There are modifications to the GCS to accommodate children who are preverbal or who are unable to verbally communicate due to sedation or endotracheal intubation. Such modifications include the Children's Coma Scale and the Infant Face Scale.[33,34] (*See Table 2.*)

Figure 1a and 1b. Rapidly Expanding Epidural Hematoma

1a. A head CT of a child performed two hours after a fall. The child had progressive emesis and lethargy. **1b.** Same patient's head CT five hours after the head trauma done secondary to increasing lethargy. Note the rapidly expanding epidural hematoma.



Group 2: Symptomatic Intracranial Injuries. The primary injury is the injury that occurs to the brain as a direct result of the trauma. Once an intracranial injury has occurred, management is directed at preventing secondary insults, which can exacerbate the primary brain injury and make the patient susceptible to progressive brain injury. The major, avoidable secondary insults include hypoxia and hypotension, which may

Table 1. Glasgow Coma Scale

EYE OPENING	
Spontaneous	4
Verbal stimulation	3
Painful stimulation	2
None	1
MOTOR	
Obeys commands	6
Localizes	5
Withdraws	4
Flexion	3
Extension	2
None	1
VERBAL	
Oriented	5
Confused	4
Inappropriate	3
Incoherent	2
None	1

Table 2. Glasgow Coma Scale — Modifications for Children

CHILDREN'S COMA SCALE (HAHN ET AL 1988) BEST SCORE = 15	
• Modification to best verbal response	
Smiles, orients to sound, follows objects, interacts	5
Consolable	4
Inconsistently consolable	3
Inconsolable	2
No response	1
INFANT FACE SCALE (DURHAM ET AL 2000) BEST SCORE = 15	
• Modification to best motor response	
Spontaneous normal movements	6
Hypoactive movements	5
Nonspecific movement to deep pain	5
Abnormal, rhythmic, spontaneous movements	3
Extension, either spontaneous or to pain	2
Flaccid	1
• Modification to best verbal response	
Cries spontaneously to handling or pain, alternating with quiet wakefulness	5
Cries spontaneously to handling or minor pain, alternating with sleep	4
Cries to deep pain only	3
Grimaces only to pain	2
No facial expression to pain	1

occur in the patient with multiple trauma; and intracranial hypertension, which may occur after the primary brain injury. Secondary brain injury causes a loss of cerebrovascular autoregulation and may result in cerebral edema, thereby reducing cerebral blood flow. Secondary brain injury also may be due to release of excitatory neurotransmitters, which can alter intracellular ion concentrations; and to the formation of inflammatory mediators, which can disrupt the blood-brain barrier and exacerbate neuronal damage.[35-37] Therefore, the goals of treatment of children with significant brain injury are to lower ICP and maximize cerebral perfusion pressure and oxygen delivery to the brain.

Monitoring of the ICP is appropriate in patients who have GCS score of 8 or less; have an abnormal initial CT scan of the head that demonstrates hematomas, contusions, or cerebral edema; or in whom serial neurological examinations are not possible due to other injuries, sedation, or neuromuscular blockade. There have been several studies in children that demonstrate an association between intracranial hypertension and poor neurological status at hospital discharge.[38,39]

ICP Monitoring. Recently published guidelines for the management of severe TBI in children recommend that a ventricular catheter connected to an external strain gauge is the most accurate and reliable manner in which to monitor ICP. [41] Such a device also allows for therapeutic diversion and analysis of cerebrospinal fluid.[40] These guidelines also recommend that the ventricular ICP be used as the reference standard in comparing the accuracy of ICP monitors placed in other cranial compartments.[41] Intracranial hypertension is defined as an ICP greater than 20 mmHg. The guidelines recommend that therapy be instituted when the ICP is consistently between 20-25 mmHg.[41] Other authors have suggested that the treatment of elevated ICP should be age dependent. In the young infant, treatment should begin when the ICP is greater than 15 mmHg; for children younger than 8 years, when the ICP is greater than 18 mmHg; and for older children and adolescents, when the ICP is greater than 20 mmHg.[35]

ICP Reduction. There are several methods to reduce ICP. Hyperventilation to reduce the pCO_2 below 35 mmHg may be useful in the setting of an acute rise in ICP or when signs of impending herniation are present. While hyperventilation may temporarily reduce intracranial hypertension, it also increases the volume of hypoperfused tissue in the injured brain; thus long periods of hypocarbia should be avoided.[41] The child's head should be maintained in a neutral position, and the head of the bed elevated to 30°. These maneuvers may decrease ICP without significantly changing cerebral perfusion pressure.[35] Jugular venous obstruction, which can elevate ICP, should be avoided by ensuring that cervical collars and endotracheal tube ties are not constrictive around the neck.[35]

Cerebral perfusion pressure (CPP) is defined as the difference between the mean arterial pressure and the ICP. The CPP is the gradient that promotes cerebral blood flow and substrate delivery to the brain. A CPP of 40-65 mmHg represents a spectrum to guide the efficacy of therapeutic interventions. Children with a CPP of 40-50 mmHg tend to have better survival after TBI.[42-45] Some authors have recommended that in young children, the CPP be maintained above 40-45 mmHg and above 50 mmHg in older children and adolescents.[35]

Therapeutic Interventions

Airway Management. *Hypoxia.* Patients should be well oxygenated throughout their ED course. Sedation and neuromuscular blockade may be useful to reduce the untoward effects of painful and noxious stimuli in patients with TBIs. Such stimuli include endotracheal intubation and mechanical ventilation, endotracheal suctioning, placement and maintenance of intravascular or intracranial catheters and monitoring devices, and transport for diagnostic procedures. Painful or stressful stimuli may increase the brain's oxygen consumption and increase sympathetic tone, leading to systemic hypertension and bleeding from operative sites.[46-48] There has been no systematic study of the efficacy of sedative and paralytic agents in children with TBI, and thus, there is no consensus as to what constitutes the ideal agents for sedation and neuromuscular blockade in this group of patients. There are case reports of the systematic, but limited, use of benzodiazepines, barbiturates, propofol, and non-depolarizing paralytic agents in children with TBI.[48] Prolonged use of propofol should be avoided in children because of reports of metabolic acidosis associated with its use. When using such agents, one must be aware of potential age-related differences in the response to pain and stress and in the level of sedation that patients may have.

Hypotension. Hypotension, which may occur in a pediatric multi-trauma patient, should be managed aggressively. Patients should be monitored carefully for the early signs of shock, including tachycardia, prolonged capillary refill, and loss of peripheral pulses. All volume deficits should be corrected and transfusions, when indicated, should not be delayed, to maintain hemoglobin and hematocrit at 10 mg/dL and 30%, respectively.[49]

Osmolar Agents. Osmolar agents, such as hypertonic saline and mannitol, have long been used in the treatment of children with TBI. Hypertonic saline works by increasing serum sodium concentration and serum osmolarity, creating an osmotic gradient by which water is pulled from the intracellular and interstitial compartments into the intravascular compartment. This increases intravascular volume and cerebral perfusion pressure, and reduces cerebral edema and ICP. One study reported results of a double-blind, crossover study comparing 3% saline and 0.9% saline boluses in 18 children with TBI.[50] During the initial trial boluses with hypertonic saline, the ICP decreased and there were reduced requirements for additional interventions. The guidelines for the acute management of severe traumatic brain injury in infants, children, and adolescents lists hypertonic saline as an option. The guidelines point out that hypertonic saline has evidentiary support, but mannitol has clinical acceptance and safety. Though mannitol works in a similar fashion, the blood brain barrier is able to exclude sodium chloride from the intracranial compartment, making it less likely to accumulate in the interstitial space.[51] Hypertonic saline also causes a reduction in vascular resistance by decreasing edema in the vascular endothelium of injured tissues.[52] Hypertonic saline also may normalize resting membrane potentials and cell volumes by restoring normal intracellular electrolyte balance in injured brain cells.[53] Rapid lowering of the serum sodium concentration should be avoided. Rebound cerebral edema can occur due to intracellular fluid shifts when the serum sodium concentration falls rapidly in the face of a residual hyperosmolar intracellular environment.[52]

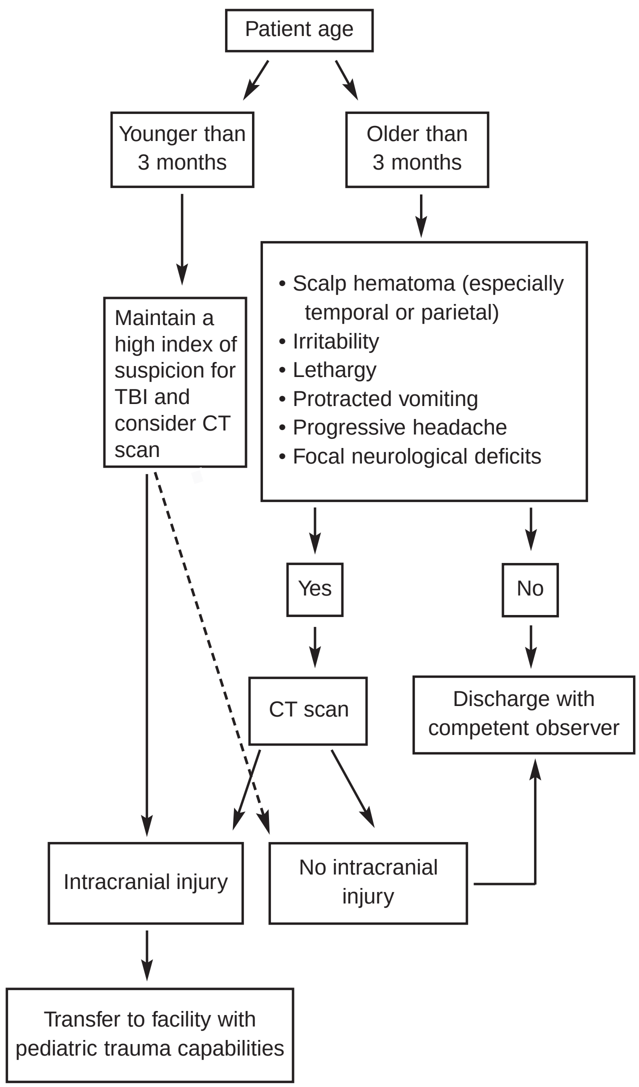
Mannitol works in a similar fashion by decreasing blood viscosity and, thereby the diameter of cerebral blood vessels. Cerebral blood flow is maintained by reflex vasoconstriction of the cerebral vasculature, but cerebral blood volume and ICP are reduced.[54] This mechanism relies on intact autoregulation of cerebral blood flow by the brain. Mannitol also reduces ICP by changing the osmotic gradient within the cerebral vasculature, causing water to move from injured tissues into cerebral blood vessels.[54] Mannitol should be administered as intermittent bolus doses. Prolonged administration of mannitol can result in its accumulation within injured tissues, reversing the osmotic gradient with the cerebral vasculature and worsening cerebral edema.[55]

Cerebral Metabolism Reduction. Reducing cerebral metabolism may be helpful in reducing ICP. Early initiation of barbiturate coma may reduce the risk of secondary brain injury. Barbiturates can lower ICP by reducing cerebral metabolism, altering cerebrovascular tone and reducing neuronal, free-radical injury.[35] Lower doses of pentobarbital initially may be given to prevent myocardial depression and systemic hypotension. It may not be necessary to use higher doses of pentobarbital to obtain burst suppression on the electroencephalogram (EEG), as lower doses still may have significant neuroprotective effects.[35]

Seizure Control. Seizures can cause a rise in ICP by increasing the brain's metabolic demands, releasing excitatory neurotransmitters, and raising systemic blood pressure. Antiepileptic drugs (e.g., phenytoin, fosphenytoin, or phenobarbital) may be helpful to prevent seizures within the first week after severe TBI, but their effectiveness in preventing late onset (i.e., longer than one week) seizures has not been demonstrated.[56] Some authors have recommended antiepileptic prophylaxis if there is significant parenchymal injury in children with severe TBI.[35] Children younger than 2 years of age are at high risk of post-traumatic seizures, with 44-70% of those with severe brain injuries having post-traumatic seizures.[35,57]

Hypothermia.The role of hypothermia in the treatment of children with TBI is unclear. While initial studies in adults demonstrated benefit in adults with TBI and intracranial hypertension, a recent randomized prospective study showed that hypothermia did not reduce morbidity and mortality in adults with severe TBI.[58-60] A similar degree of hypothermia has been shown to be efficacious in children with uncontrolled intracranial hypertension after TBI.[61] While intracranial hypertension was ameliorated after 48 hours of induced hypothermia when compared with the normothermic group, functional outcomes of survivors were similar between the two groups. A larger randomized trial is needed to definitively determine if induced hypothermia improves survival in children with TBI. Currently, the Guidelines for Acute Management of Severe Traumatic Brain Injury in Infants, Children and Adolescents recommend as an option, to avoid hyperthermia (i.e., temperature is higher than 38.5°C), and consider hypothermia (i.e., temperature is

Figure 2. Children Younger than 2 Years with a Head Injury



32-33°C) if refractory intracranial hypertension occurs.

Operative Intervention. Finally, operative intervention may be a necessary adjunct to medical therapy for severe TBI. Significantly depressed skull fractures should be elevated and intracranial and intraparenchymal mass lesions should be evacuated or debrided when ICP and CPP cannot be optimally managed by medical measures.[35] Some studies have demonstrated that decompressive craniectomy may be useful for pediatric patients with severe head injuries with uncontrolled intracranial hypertension.[62,63]

Predictors of Outcome

There has been a significant decline in the morbidity and mortality of pediatric TBI in the United States during the past two decades.[64] The overall mortality of children with TBI in the United States has been reported to be 6%, and those children with severe head injury requiring mechanical ventilation have a mortality of approximately 18%.[65,66]

There may be several reasons for such a decline in morbidity and mortality. One study analyzed consecutive admissions of children with TBIs to three different pediatric intensive care units. He found that while there was significant variation among centers with respect to the use of neuromuscular blockade, induced hypothermia and ICP monitoring, none of these modalities had an effect on mortality. Only the use of antiepileptic agents significantly reduced mortality in this study.[67] Another study found that in children with severe traumatic brain injuries, survival was associated significantly with the maintenance of supranormal systolic blood pressure (i.e., greater than 135 mmHg).[68] Mannitol was associated with a prolonged length of stay in the pediatric intensive care unit, but had no effect on survival. Similarly, Pigula found that children with severe head injuries and systemic hypotension had a much greater mortality rate.[69] Further study is needed to determine which interventions have an impact on morbidity and mortality in children with TBIs.

Several investigators have evaluated which factors may predict both survival and functional outcomes of children with TBI. In severe TBI, the GCS score and Pediatric Risk of Mortality Score (PRISM) may be predictive of survival.[66] In a retrospective study, children with GCS scores less than or equal to 5, but with lower PRISM scores, were more likely to survive and be discharged from the hospital. At hospital discharge, 40% of these patients were functioning independently; and at two years after the injury, nearly 66% were functioning independently. However, independent functioning in childhood may not persist into adulthood. In another study, 39 adults who had sustained TBI during the preschool years were evaluated.[70] While 59% of these patients attended a regular school after recovering from their TBI, only 29% eventually had full time employment as adults.[70] Most of these patients had sustained their TBI nearly 30 years ago, and it can be argued that recent advances in resuscitation of brain-injured children eventually may improve functional outcomes that persist into adulthood. Finally, serum levels of protein S-100 beta, a calcium-binding, dimeric protein found in astroglial and Schwann cells, when obtained and measured at the initial time of injury, may have predictive value in determining functional outcome in children and adults with mild to severe TBI.[71,72]

School-age children who survive TBI are at risk for having neuropsychological deficits and developing psychiatric syndromes. Children who survive severe TBI are at risk of having deficits in verbal reasoning, learning and recall, attention, executive functions, and constructional skills within 12 months of hospital discharge. Even when evaluated as long as four years after the injury, there may be little long-term recovery of such skills.[73] Children who recover from both mild and severe TBI are more likely than those who recover from orthopedic injuries to have psychiatric disturbances, such as organic personality disorder, attention deficit-hyperactivity disorder, major depression, and anxiety disorders.[74] Siblings and parents of children who survive severe TBI may also experience psychological distress during the patient's recovery and rehabilitation periods.[75,76]

Summary

TBI can cause considerable morbidity in young children. Children younger than 1 year, and particularly those younger than 3 months, are at higher risk of sustaining a TBI after head trauma than are older children. Scalp hematomas, especially those over the parietal region, altered mental status, and focal neurological signs, are the best clinical indicators of TBI in children.

Children with TBI are best managed at trauma centers, and transfer to such facilities should be expedited when TBIs are diagnosed in children. Once a primary brain injury, or trauma that results directly from impact, has occurred, the goals of management are directed at preventing secondary insults, which can exacerbate the primary brain injury and make the patient susceptible to secondary brain injury. Maximizing CPP and reducing ICP are the goals of management of children with TBIs. Sedation, neuromuscular blockade, hyperosmolar therapy, barbiturate therapy, and antiepileptic prophylaxis are management options in children with TBIs.

Finally, children and their families will require considerable support during the rehabilitation phase after a TBI. Psychological and psychiatric sequelae are common in children after a TBI, and significant family stress can occur during the patient's recovery and rehabilitation period.

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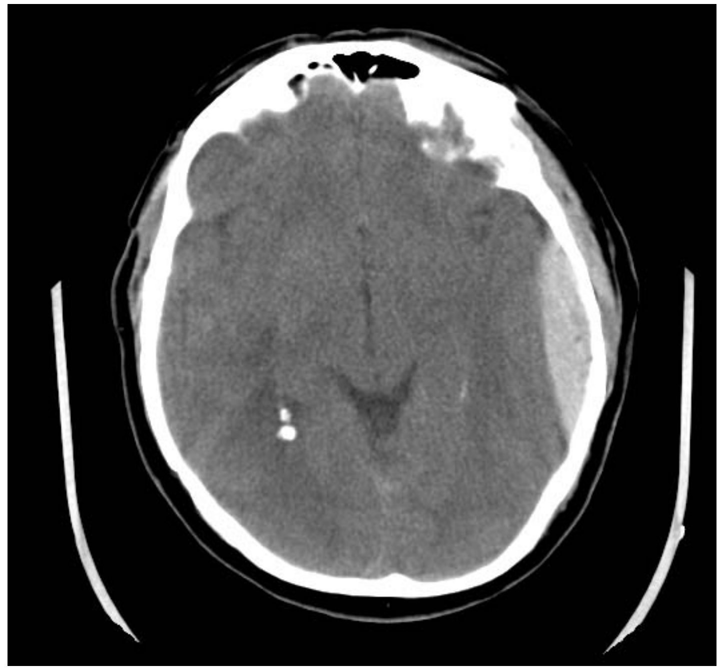
CE/CME Questions

1. Which of the following is true regarding a child younger than 2 years who sustains a head injury?
   A. The younger the child, the higher the risk for traumatic brain injury.
   B. The incidence of brain injury in a child younger than 2 years is about 5%.
   C. CT scans do have certain disadvantages, including exposure to ionizing radiation.
   D. All of the above

2. A 3-month-old male presents after his mother dropped him when she tripped. He fell approximately five feet. He is irritable, but consoles and has a large parietal hematoma. The most appropriate next test is:
   A. MRI.
   B. CT scan of the head.
   C. skull films.
   D. skeletal survey.

3. A 7-year-old male was involved in a fight at school four hours ago. He did not lose consciousness, remembers the entire event, and has had no vomiting. His neurologic examination is normal. On physical examination, he has a hematoma on his forehead. The next best test is:
   A. an MRI.
   B. a CT scan of the head.
   C. skull films.
   D. None of the above

4. Which of the following has/have been associated with an intracranial injury in a child younger than 2 years?
   A. Skull fracture
   B. Parietal scalp hematoma
   C. Large scalp hematoma
   D. All of the above



5. What is shown in the image above?
   A. Epidural hematoma
   B. Subdural hematoma
   C. Intraparenchymal hematoma
   D. None of the above

6. Which of the following is *not* considered to be high-risk criteria for TBI in a child younger than 2 years?
   A. Depressed mental status
   B. Signs of depressed or basilar skull fracture
   C. Two episodes of emesis
   D. Acute skull fracture

7. Which of the following children does *not* require a cranial CT following a fall?
   A. A 3-year-old with an occipital hematoma, no other symptoms, and a normal exam
   B. A 4-month-old who has a large scalp hematoma and is irritable
   C. A 1-year-old who has a GCS score of 13
   D. A 6-year-old with hemotypanum

8. Which of the following are critical in the initial stabilization of a child with a head injury?

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing 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 provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

CE/CME Objectives

Upon completing this program, the participants will be able to:

a.) Recognize or increase index of suspicion for pediatric head injury;
b.) Identify how to correctly and quickly stabilize and manage pediatric head trauma;
c.) Employ appropriate diagnostic modalities for pediatric head trauma; and
d.) Recognize indications and potential risks with therapeutic options for children with head trauma.

A. Avoiding hypoxia
B. Avoiding hypotension
C. Maintaining an adequate cerebral perfusion pressure
D. All of the above

9. In which of the following scenarios is ICP monitoring *not* an appropriate consideration?
A. A child with a GCS score less than 8
B. A child with a GCS score of 12 five minutes after a seizure
C. A child who was intubated at the scene, is unresponsive and has cerebral edema on CT scan
D. A child who is intubated for a multi-system trauma and must be paralyzed and sedated

10. Which of the following may be used in the management of a child with a head injury and a GCS score of 8?
A. Early intubation
B. ICP monitoring
C. Correction of hypotension
D. All of the above

Answer Key:

1. D	**6. C**
2. B	**7. A**
3. D	**8. D**
4. D	**9. B**
5. A	**10. D**

In Future Issues: **Knee Injuries**