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INSIDE

High-Dose Epinephrine in Children: No Benefit, Maybe Harmful

ABSTRACT & COMMENTARY

Source: Perondi MB, et al. A comparison of high-dose and standard-dose epinephrine in children with cardiac arrest. *N Engl J Med* 2004; 350:1722-1730.

THE AUTHORS OF THIS BRAZILIAN STUDY ENROLLED CHILDREN sustaining witnessed, in-hospital cardiac arrests from any cause. Patients who remained in cardiac arrest despite cardiopulmonary resuscitation and an initial standard dose of epinephrine (0.01 mg/kg) received either standard dose epinephrine (0.01 mg/kg) or high-dose epinephrine (0.1 mg/kg) for the second and any subsequent doses of drug. The resuscitation otherwise proceeded in the usual fashion. Study group assignment was determined in a randomized, double-blind fashion. The main study endpoint was survival at 24 hours after the arrest.

Sixty-eight patients were enrolled during a 23-month period. Protocol violations–usually incorrect doses of epinephrine–were noted in 18 patients, and analyses were performed on an intent-to-treat basis. While seven of 34 patients in the standard-dose epinephrine group survived at 24 hours, only one of 34 in the high-dose group met this endpoint (odds ratio for death in the high-dose group, 8.6). This effect persisted after adjustment for inter-group differences. Among patients sustaining cardiac arrest as a result of asphyxia, 24-hour survival was noted in seven of 18 in the standard-dose epinephrine group, compared with none of 12 in the high-dose group. Four children who had received standard-dose epinephrine survived to hospital discharge—two of whom had normal neurologic function at six months, and two of whom returned to their abnormal baseline functional status—while no patient receiving high-dose epinephrine survived to discharge. No children with shockrelated cardiac arrest survived to discharge.

COMMENTARY BY DAVID J. KARRAS, MD, FAAEM, FACEP

High-dose epinephrine currently is considered a treatment option for pediatric victims of cardiac arrest. Data supporting the use of high-dose epinephrine is limited, and largely consists of a non-randomized study using historical controls.[1] Recent studies have failed

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to show a benefit over standard-dose therapy in adult resuscitations. This study provides powerful evidence that high-dose epinephrine offers no advantage in the resuscitation of children. While stronger conclusions are limited by the relatively small sample size, it appears that patients in the high-dose epinephrine group actually fared worse than those receiving standard-dose therapy. Further analyses showed that the protocol violations were unlikely to have biased the results—if anything, more patients received high-dose therapy—and the authors caution that their results may not apply to children sustaining unwitnessed, out-of-hospital arrests. ❖

Reference

1. Goetting MG, Paradis NA. High-dose epinephrine improves outcome from pediatric cardiac arrests. *Ann Emerg Med* 1991;20:22-26.

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Editorial Group Head: Valerie Loner.
Managing Editor: Martha Jo Dendinger.
Marketing Manager: Schandale Kornegay.

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Editorial E-Mail Address: martha.dendinger@thomson.com

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Prehospital Hypertonic Saline Resuscitation Not Helpful for Hypotensive Patients with Severe Traumatic Brain Injury

ABSTRACT & COMMENTARY

Source: Cooper DJ, et al for the HTS study investigators. Prehospital hypertonic saline resuscitation of patients with hypotension and severe traumatic brain injury – a randomized controlled trial. *JAMA* 2004;291:1350-1357.

PATIENTS WITH HYPOTENSION FOLLOWING SEVERE traumatic brain injury (TBI) have a higher mortality rate and worse neurological outcome for survivors. Resuscitation with intravenous hypertonic saline (HTS) may have theoretical benefits in this setting by increasing blood pressure and decreasing intracranial pressure compared with isotonic resuscitation fluids.

In this well-designed, double-blind, controlled study, investigators in Australia randomized 229 patients with severe TBI (i.e., Glasgow Coma Scale score less than 9) and hypotension (i.e., systolic blood pressure less than 100 mmHg) to receive either HTS or isotonic saline fluid administered in the pre-hospital setting. One hundred-fourteen patients in the HTS group received 250 mL of 7.5% saline, while 115 control patients received 250 mL of Ringer's lactate solution. All patients then received standard resuscitation fluids and other care in the prehospital setting. Patients with penetrating trauma or traumatic arrest were excluded. Investigators followed patients through their hospital course and at three and six months to assess survival and neurologic outcome.

In both groups, prehospital hypotension had resolved on arrival to the emergency department. However, the HTS group did have a higher mean serum sodium level (149 vs 141 mEq/L). Despite this difference, there was no survival difference between the HTS and control group at hospital discharge (55% vs. 57%, p = 0.32), at three-month follow-up (55% vs. 48%, p = 0.26), and at six-month follow-up (55% vs. 47%, p = 0.23), respectively.

There also was no difference in neurologic outcome for survivors as measured by a number of parameters including the extended Glasgow Outcome Scale (GOSE), which measures neurologic recovery on an eight-step scale (one = dead; eight = upper good recovery). In addition, there was no significant difference in the proportion of patients with a good recovery

(i.e., GOSE greater than 4), and no difference in the rates of return to work between the two groups. Based upon their findings, the authors conclude that prehospital HTS does not improve mortality rates or long-term neurologic function in patients with severe TBI and hypotension compared with conventional resuscitation fluids.

COMMENTARY BY THEODORE C. CHAN, MD, FACEP

This is an excellent study from a number of standpoints. First, it is one of the few examples of a blinded, randomized controlled study of therapies in critically ill patients conducted in the prehospital setting with excellent long-term follow-up. In fact, patient loss to follow-up at six months was only 1%. Second, while there were only 229 subjects, this study had 80% power to detect a one-grade change in the neurologic outcome score (GOSE).

A number of points should be kept in mind regarding this study. First, patients also received standard therapies in the prehospital setting, which included fluid resuscitation. Thus, both groups received other resuscitation fluids (median 1250 mL)—including both isotonic fluid and colloid—which may have had a diluting effect on the 250 mL of HTS. Second, more than 90% of patients had multi-system trauma, as opposed to isolated head injury, which may have affected mortality and neurologic outcomes in both groups. Third, prehospital transport times were relatively long (median time 60 minutes), which may have affected their results, though a subgroup analysis suggested no difference between HTS and control groups when looking at time of transport. Fourth, recent studies have suggested that a combination of HTS and colloid (HTS-dextran) may have additive benefits that this study did not investigate. Finally, it should be noted that while not statistically significant, there was a trend toward improved survival in the HTS group, indicating the need for further study in the future. ❖

Do All Human Bite Wounds Need Antibiotics?

Source: Broder J, et al. Low risk of infection in selected human bites treated without antibiotics. *Am J Emerg Med* 2004;22:10-13.

ABSTRACT & COMMENTARY

Most emergency medicine textbooks agree that human bite wounds, as well as dog and cat bite wounds, require antibiotic prophylaxis in addition to usual wound care practices. This study from the University of Maryland challenges this belief, and attempts to define a group of human bites at low risk of infection that do not require any antibiotic prophylaxis.

This prospective, double-blind, placebo-controlled study randomized patients with certain superficial human bite wounds to antibiotic or placebo treatment in an attempt to determine whether these wounds had similar rates of infection. Patients were eligible for enrollment if the bite wound was superficial (i.e., penetrating only the epidermis) and did not involve hands, feet, or skin overlying joints or cartilaginous structures. Exclusion criteria included immune-compromised status, age younger than 18 years, bites older than 24 hours, and allergy to penicillin or related compounds. No patients with puncture-type bite wounds were enrolled.

Patients meeting entry criteria were randomized into the placebo or antibiotic arm of the study. Wounds were debrided as necessary and irrigated with 500 cc of normal saline. Tetanus prophylaxis was given as necessary. No wounds required sutures. Patients then were discharged from the emergency department with a five-day course of either cephalexin plus penicillin or placebo, and were instructed to return at 48 and 96 hours to be checked for signs of infection. All wounds were rechecked by the same examiner (blinded to treatment group) to eliminate the possibility of inter-rater variation in the assessment of whether wound infection was present.

One hundred-twenty-seven (127) patients were enrolled. One patient in each group was lost to follow-up, leaving 125 patients completing the protocol. The two groups were similar with respect to age and weight. None of the 63 patients in the antibiotic group developed wound infections (0%, 95% CI 0-4.6%), and one of 62 patients in the placebo group developed wound infection (1.6%, 95% CI 0-7.3%). The groups were similar in terms of medication compliance, and no patient developed an allergic reaction.

The authors conclude that this study supports changing the practice of routinely giving antibiotic prophylaxis to patients with these specific types of human bite wounds.

COMMENTARY BY JACOB W. UFBERG, MD

This study supports a more common-sense approach to human bite wounds: Very superficial wounds in low-risk body areas that are given meticulous wound care are unlikely to become infected. While the study is limited by small numbers, even the wide confidence intervals do not exceed wound infection rates commonly seen among the wounds we treat in the emergency department.

Caution should be exercised, however, in putting these guidelines into practice. We must be sure that the wounds meet the authors' criteria if we are to withhold antibiotics. Puncture wounds, deeper lacerations, and bites to the hand all have been shown to have high infection rates, which may be lowered by antibiotic prophylaxis. Also, we should not extrapolate these results to include dog and cat bites, which are more likely to cause deeper lacerations and puncture-type wounds due to the differences in shape between human and animal teeth. ❖

Comparing Methods of Loading Phenytoin in Seizure Patients

ABSTRACT & COMMENTARY

Source: Swadron SP, et al. A comparison of phenytoin-loading techniques in the emergency department. *Acad Emerg Med* 2004;11:244-252.

THE AUTHORS HAVE TACKLED A COMMON PROBLEM IN the emergency department (ED): finding the most effective phenytoin-loading technique. They enrolled patients with sub-therapeutic phenytoin concentrations who presented within 48 hours of a seizure. They received either 20 mg/kg of oral phenytoin (PO), divided in maximum doses of 400 mg every two hours, 18 mg/kg of intravenous phenytoin (IVP) at an initial infusion rate of 50 mg/min, or 18 mg/kg (phenytoin equivalents) of intravenous fosphenytoin (IVF) at an initial infusion rate of 150 mg/min. Forty-five patients were included: 16 in the PO group, 14 in the IVP group, and 15 in the IVF group. Therapeutic drug concentrations were achieved in (mean ± standard deviation [SD]) 5.62 ± 0.28 hours (PO), 0.24 ± 0.3 hours (IVP), and 0.21 ± 0.28 hours (IVF). A total of 17, 27, and 32 adverse drug events were observed in the PO, IVP, and IVF groups, respectively; this rate was significantly lower in the PO group. No significant difference was found between the numbers of necessary adjustments to the infusions in the two intravenous groups. Time to safe ED discharge was significantly faster for the intravenous groups compared with the PO group ($p < 0.001$). Interestingly, four patients experienced a seizure after phenytoin loading (three in the fosphenytoin group, one in the oral phenytoin group, and none in the intravenous phenytoin group). All of these patients had therapeutic phenytoin levels. The main differences between the oral route and the intravenous routes were directly related to two side effects: irritation at the infusion site (called phlebitis) for the IVP group and pruritis (generalized and perineal) in the IVF group. There were two episodes of hypotension in the IVP and one episode in the IVF. The authors conclude that the oral loading leads to less frequent adverse drug events than either intravenous loading technique, but when therapeutic concentrations are required quickly, oral loading may be disadvantageous. Although IVF loading is faster, from an adverse-drug event perspective, no advantage of IVF over IVP was apparent.

COMMENTARY BY RICHARD J. HAMILTON, MD, FAAEM, ABMT

There are a great many strongly held opinions on loading phenytoin in patients who have sub-therapeutic concentrations. I have used all of the techniques studied in this paper, plus unique combinations of the two—for example, loading half of the dose intravenously and the rest by mouth. In my experience, there are two key determinants in rapid disposition of patients: rapid determination of a phenytoin level and intravenous loading of phenytoin or fosphenytoin. However, while this approach may result in the rapid restitution of therapeutic phenytoin levels, there is little evidence that it is of any value to patients or protects them from subsequent seizures. Furthermore, since side effects from intravenous loading are not trivial and include side effects that are potentially life-threatening, oral phenytoin may have the greatest risk benefit/ratio for the patient. My approach is to establish intravenous access on a seizure patient, obtain a phenytoin level, use an intravenous benzodiazepine if seizures reoccur, and consider intravenous loading with phenytoin when a reliable intravenous line can be established in a large forearm vein. If such a line is not available, or the intravenous line is in a tenuous wrist or hand location, the oral loading technique should be employed. Fosphenytoin offers its greatest advantage via the intramuscular route. ❖

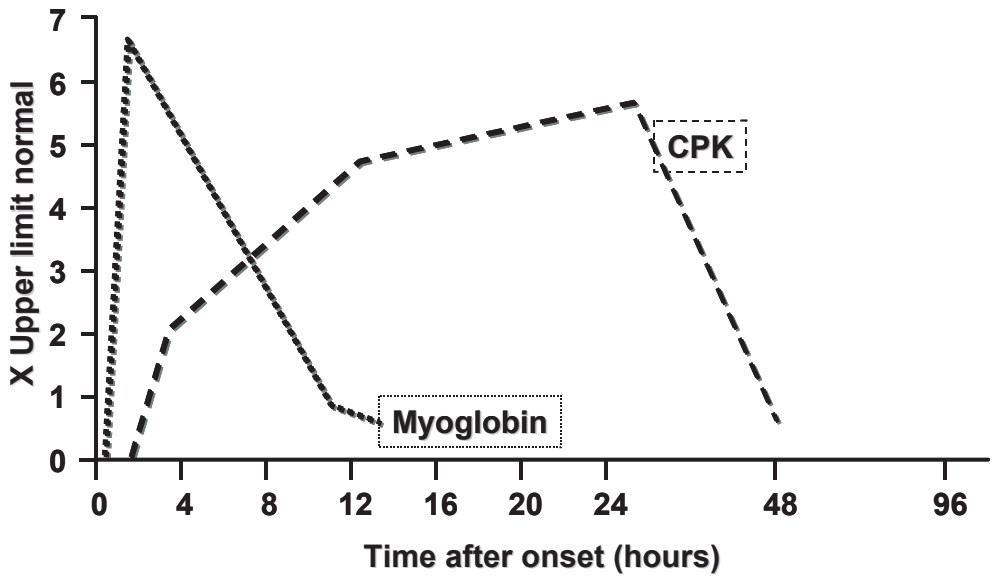
Special Feature

Rhabdomyolysis

By William J. Brady, MD

RHABDOMYOLYSIS IS DEFINED AS INJURY TO skeletal muscle resulting in breakdown of muscle tissue with subsequent leakage of intracellular contents, including myoglobin, potassium and other electrolytes,

Figure. Release Kinetics of Serum Markers in Rhabdomyolysis



creatine phosphokinase (CPK), and other muscle enzymes. These same intracellular elements serve as diagnostic markers for the syndrome. Rhabdomyolysis classically involves the triad of muscle weakness, myalgias, and darkened urine. As is true with most such traditional constellations of symptoms and signs, this classic triad is noted on presentation in few patients. More commonly, the patient complains of muscular aches and weakness, prompting the diagnosis of a benign viral syndrome. The term myoglobinuria—incorrectly used as a synonym for rhabdomyolysis—indicates only the presence of myoglobin in the urine and, therefore, is a less descriptive term for the clinical syndrome; it is best avoided in clinical practice.

Etiology and Pathophysiology

The potential causes of rhabdomyolysis are numerous, including exposure to ethanol, various medications and other toxins, trauma (e.g., crush injuries and orthopedic fractures), prolonged compression of limbs, excessive seizure activity, electrolyte disorders, strenuous activities (e.g., military basic training, weight lifting, and marathon running), and muscle cellular enzyme deficiency states. Ethanol abuse and its sequelae, limb compression, and prolonged seizure activity were the three most commonly encountered etiologic factors of rhabdomyolysis in a series of hospitalized patients.[1] A significant number of cases, however, still are classified as "idiopathic" despite detailed histologic, enzymatic, and biochemical studies.

The pathophysiology of rhabdomyolysis must be understood on cellular (microscopic) and muscle compartment (macroscopic) levels. The final common pathway in the cellular pathophysiology of rhabdomyolysis, regardless of etiology, is believed to be an impairment of either production or utilization of adenosine triphosphate (ATP). The reduction in ATP, whether relative or absolute, causes energy-requiring reactions and homeostatic mechanisms to fail. The macroscopic pathophysiology of rhabdomyolysis involves local hydrostatic and osmotic pressure conditions within the myofascial compartment. When the intracompartmental pressure in-creases above a specific level due to such factors as hemorrhage, inflammatory fluid, or external compression, perfusion to the compartment is impaired, resulting in disruption of the skeletal muscle metabolic processes. The net effect is increased intracompartmental pressure and further disruption of perfusion to the closed space of the myofascial compartment as well as to distal structures of that vascular distribution.

Clinical Presentation

The presentation of rhabdomyolysis ranges from very straightforward to very elusive. Patients may complain only of myalgias and weakness. Alternatively, the patient may present as a multiple trauma victim with a compartment syndrome. Examination may reveal localized or diffuse muscular tenderness, focal muscular weakness, edema, skin changes consistent with pressure necrosis, or findings of a compartment syndrome.

The laboratory diagnosis of rhabdomyolysis includes both urine and serum examinations. A urine specimen with a dipstick positive for blood and simultaneously demonstrating zero-to-few red blood cells is consistent with rhabdomyolysis, due to the presence of myoglobin. While serum and urine myoglobin levels can confirm these findings, they are not reliable indicators of rhabdomyolysis due to rapid plasma clearance; the serum half-life of myoglobin is only 1-3 hours and there is poor correlation of myoglobinuria with myoglobinemia. (*See Figure.*)[2,3] CPK levels have been shown to be the most sensitive marker of myocyte injury and rhabdomyolysis.[4] The use of CPK measurement is the most appropriate laboratory method for confirming the diagnosis of rhab-

domyolysis due to a number of factors, including the ease of test performance in most clinical settings, near-immediate appearance of measurable levels of CPK in the serum after muscle injury, and lack of rapid clearance of CPK from the blood. (*See Figure.*) CPK levels (usually 100% MM fraction) at least five times above the upper limit of normal are required to fulfill the criteria for rhabdomyolysis.[5] The absolute height of the CPK elevation, however, does not translate to disease severity and risk of complication. The CPK level peaks at 24-36 hours after the skeletal muscle "insult" and declines at a rate of approximately 40% per 24 hours. Failure of CPK levels to decline at the appropriate rate suggests an ongoing process of skeletal muscle injury.

Electrolyte abnormalities also are seen in rhabdomyolysis. Hyperkalemia, resulting from excessive release of potassium from damaged muscle, renal failure, and metabolic acidosis, can appear within the first few days after the onset of the illness. Hypocalcemia is thought to occur from the deposition of calcium salts in damaged skeletal muscle. Hypercalcemia also has been noted during the diuretic phase of myoglobin-induced renal failure, when serum phosphate concentrations fall and the calcium salts are reabsorbed. Hyperphosphatemia results from phosphate release during muscle injury. Hyperuricemia, which tends to be much higher in patients with exertional rhabdomyolysis, results from purines released from injured muscle cells. Concentrations of blood urea nitrogen and creatinine in the serum may increase, resulting from both acute renal failure and release from skeletal muscle.

Complications

Complications of rhabdomyolysis include various electrolyte abnormalities, acute renal failure (ARF), compartment syndrome, disseminated intravascular coagulation, acute respiratory failure (rare), and cardiomyopathy (very rare). The most feared complication of rhabdomyolysis is renal failure due to acute tubular necrosis. Renal injury results from myoglobin casts obstructing renal tubules, a decreased glomerular filtration rate, and direct nephrotoxicity of ferrihemate, a breakdown component of myoglobin. Furthermore, hypovolemia and any process producing urinary acidification predispose to acute renal failure. With urine pH less than 5.6, myoglobin dissociates into ferrihemate and a globin moiety. Ferrihemate is a dose-related nephrotoxin. The toxic effect of ferrihemate on a cellular level has been shown to be a result of ferrihemate-related production of free hydroxy radicals. Researchers at Denver General Hospital developed the discriminant function equation to predict the risk of ARF in such patients. The equation is as follows: R = 0.7 (potassium) + 1.1 (creatinine) + 0.6 (albumin) – 6.6. The R values and the associated risk of ARF are noted: $R < 0.1$ (0% chance of ARF) and $R > 0.1$ (41% chance of ARF).[1]

Treatment

Treatment of patients with rhabdomyolysis includes not only an attempt at identification of the triggering event but also management of metabolic complications and organ dysfunction. The clinician must make a diligent search for any reversible cause of rhabdomyolysis (e.g., compartment syndrome) and halt ongoing muscle damage. Saline (0.9%) loading by intravenous route is the mainstay of therapy in that it restores intravascular volume and induces a solute diuresis. All patients should have a urinary catheter to adequately monitor fluid output. Diuresis can be accomplished using an osmotic agent (mannitol) or a loop diuretic (furosemide). Sodium bicarbonate may protect the kidneys from the effects of myoglobinuria by rapidly increasing urinary pH, provided that the development of frank metabolic alkalosis is avoided.[6] The use of sodium bicarbonate is based upon the theoretical advantage of inhibiting the formation of the nephrotoxin ferrihemate. Hyperkalemia is treated in the usual manner with infusion of insulin, glucose, and calcium gluconate, inhalation of a nebulized beta-agonist agent, administration of oral and rectal exchange resins, electrocardiographic monitoring, and elimination of potassium intake. Dialysis may be required to correct electrolyte abnormalities or to treat oliguric renal failure. Finally, hospital admission is required to treat the syndrome with the aim of halting progression to renal failure, monitoring for other associated complications, and ensuring that the process of skeletal muscle death is not ongoing or does not recur. ❖

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Physician CME Questions

1. **Which of the following is the most reliable method of diagnosing rhabdomyolysis?**
   a. A patient history of weakness and muscular aches
   b. Analysis of the urine demonstrating blood
   c. An elevated CPK value
   d. The absence of myoglobin in the urine

2. **Causes of rhabdomyolysis include:**
   a. ethanol abuse.
   b. ethylene glycol toxicity.
   c. methanol toxicity.
   d. isopropyl alcohol toxicity.

3. **Treatment of rhabdomyolysis might reasonably include:**
   a. acidification of the urine.
   b. intravenous saline infusion.
   c. fomepizole.
   d. hydroxyurea.

4. **Which of the following wounds is at the lowest risk for infection, and possibly may be treated without antibiotic prophylaxis?**
   a. Puncture wound due to a cat bite
   b. Human bite to the hand
   c. Human bite over the elbow
   d. Human bite to the mid-thigh area, epidermis penetration only

5. **In the study of the use of prehospital hypertonic saline for patients with traumatic brain injury and hypotension, the investigators reported that hypertonic saline:**
   a. improved survival to ED arrival, but not hospital discharge.
   b. improved neurologic outcome at three-month, but not six-month follow-up.
   c. did not improve survival at three-month or six-month follow-up.
   d. improved survival to hospital discharge for patients with long transport times.

6. **In children sustaining in-hospital cardiac arrest, administration of high-dose epinephrine (0.1 mg/kg) is associated with:**
   a. lower 24-hour survival rate.
   b. lower rate of survival to discharge.
   c. poorer outcome among those with asphyxiation-related arrest.
   d. All of the above

7. **Oral loading of phenytoin potentially offers which of the following advantages over intravenous loading of either fosphenytoin or phenytoin?**
   a. Faster time to therapeutic drug level
   b. Less akathisia
   c. Lower rate of adverse drug events
   d. Less nausea and vomiting

Answer Key

1. c;
2. a;
3. b;
4. d;
5. c
6. d
7. c

CME Objectives

To help physicians:

- Summarize the most recent significant emergency medicine-related studies;
- Discuss up-to-date information on all aspects of emergency medicine, including new drugs, techniques, equipment, trials, studies, books, teaching aids, and other information pertinent to emergency department care; and
- Evaluate the credibility of published data and recommendations.

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.

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ECG Review

A Special Wave

By Ken Grauer, MD

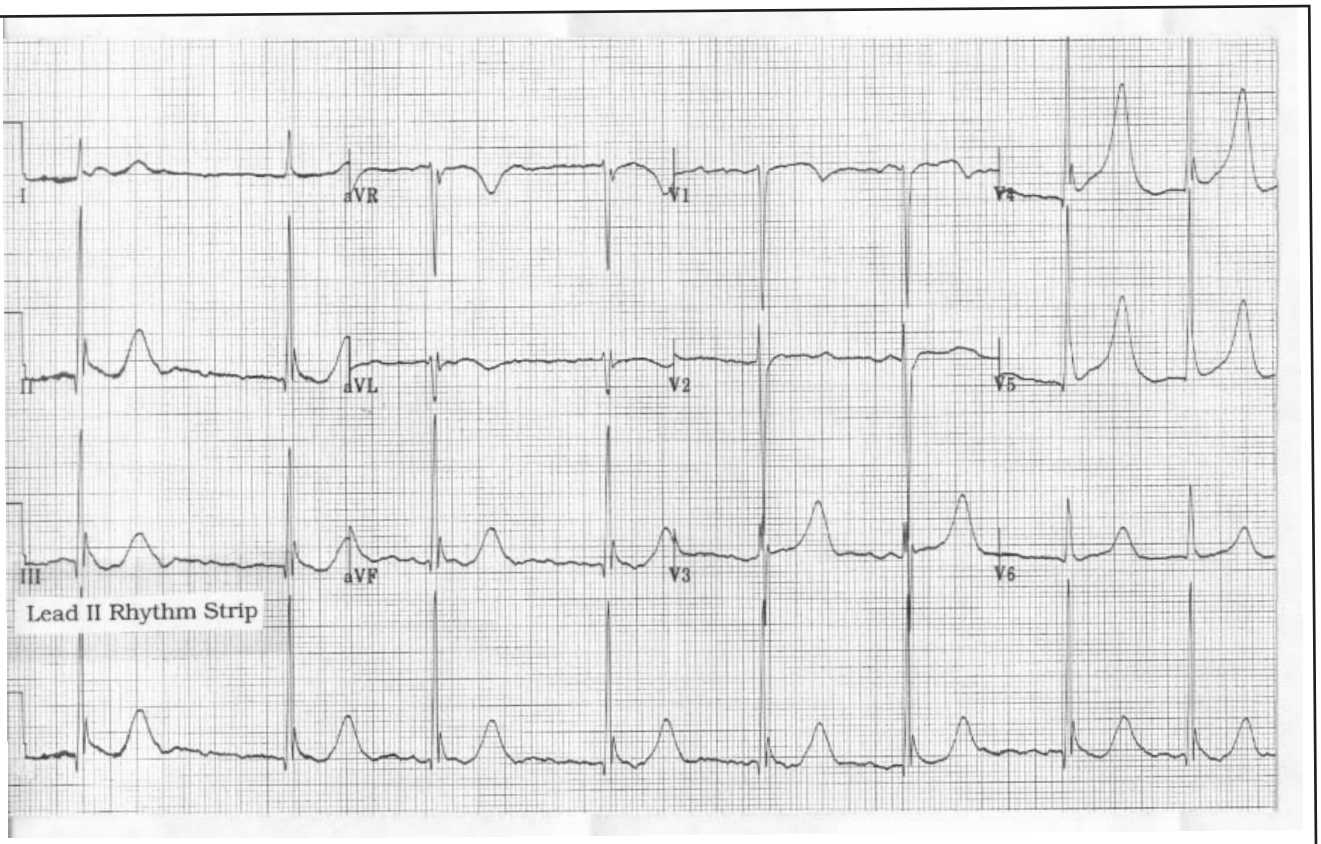

Figure. 12-lead ECG and accompanying rhythm strip obtained from a 68-year-old man admitted following a drug overdose.

Clinical Scenario: The electrocardiogram (ECG) in the Figure was obtained from a 68-year-old man admitted to the intensive care unit (ICU) for a drug overdose. In view of his ECG, what vital sign needs to be checked? How many ECG findings consistent with this patient's clinical condition can you identify?

Interpretation: The ECG in the Figure manifests many of the features of hypothermia. The most commonly cited ECG finding of this condition is the elevated and very prominent J wave (also called the Osborne wave or camel-hump sign) that is especially well seen in the inferior leads and lead V4 of this tracing. The etiology of the Osborne wave is uncertain; it is most often seen when hypothermia is moderate to severe (core temperature less than 86°F or 30°C). In addition to the prominent J wave, three other features characteristic of hypothermia are also seen here: 1) bradycardia; 2) atrial fibrillation with a slow ventricular response; and 3) fine undulations in the baseline (attributable to muscle tremor). The patient in this case had a core temperature of 90°F on admission, though it was thought to be lower before his arrival to the emergency department. He had been found unresponsive in the street during cold weather after ingestion of unknown drugs the evening before. All ECG manifestations of hypothermia resolved following core rewarming.❖

In Future Issues: **Hazards of Radiation Exposure**

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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

Author: **Kirsten Bechtel, MD**, Assistant Professor of Pediatrics,Yale University School of Medicine; Attending Physician-Pediatric Emergency Department, Yale New Haven Children's Hospital, New Haven, CT.

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.

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Department of Pediatric General and Thoracic Surgery
Children's Hospital of Philadelphia, PA.

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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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Vice President/Group Publisher: Brenda Mooney
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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.

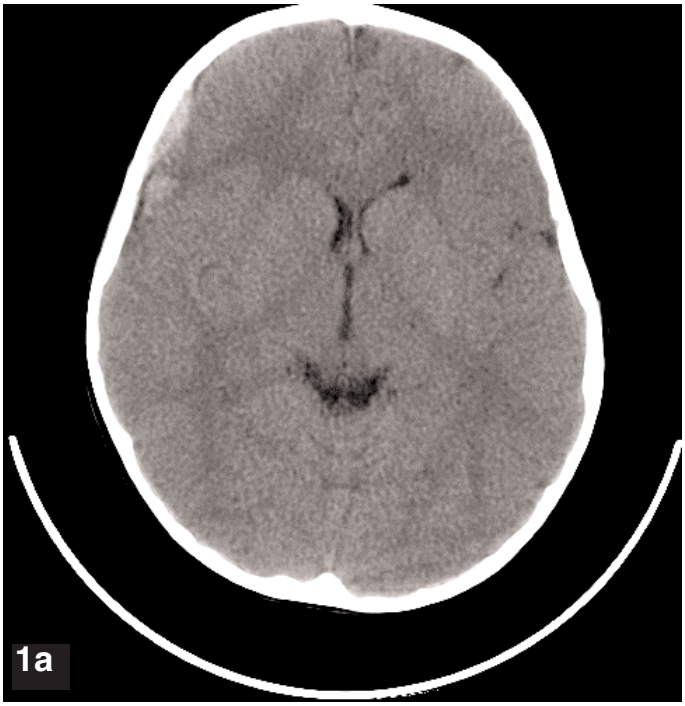
The guidelines for the acute management of severe TBI in infants, children, and adolescents made transfer to a pediatric trauma center a guideline based upon class II data (prospective and retrospective observation, cohort, and case control) and strong class III data (retrospective), and, as an option,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

Management of Intracranial Injuries

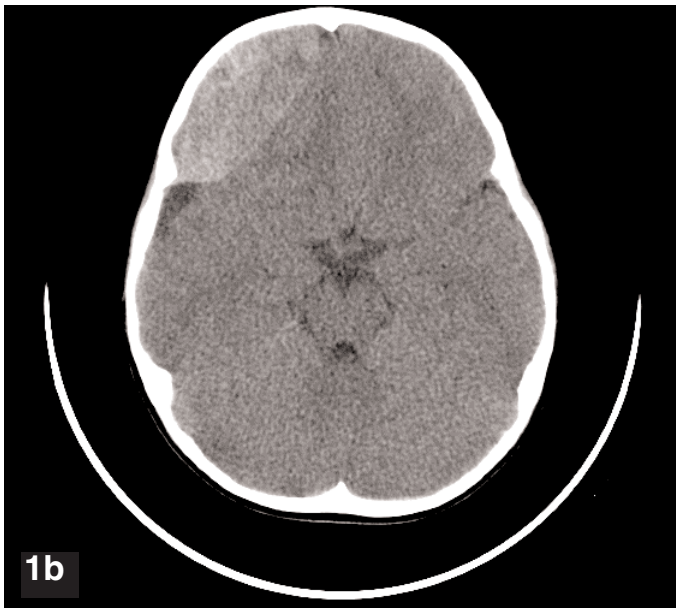
Group 1: Asymptomatic Intracranial Injuries. The optimal management and outcome of children who have intracranial injury as detected by CT scan, but who are otherwise asymptomatic, is controversial. Typically, such children are admitted to the hospital for close neurological assessment and monitoring. 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]

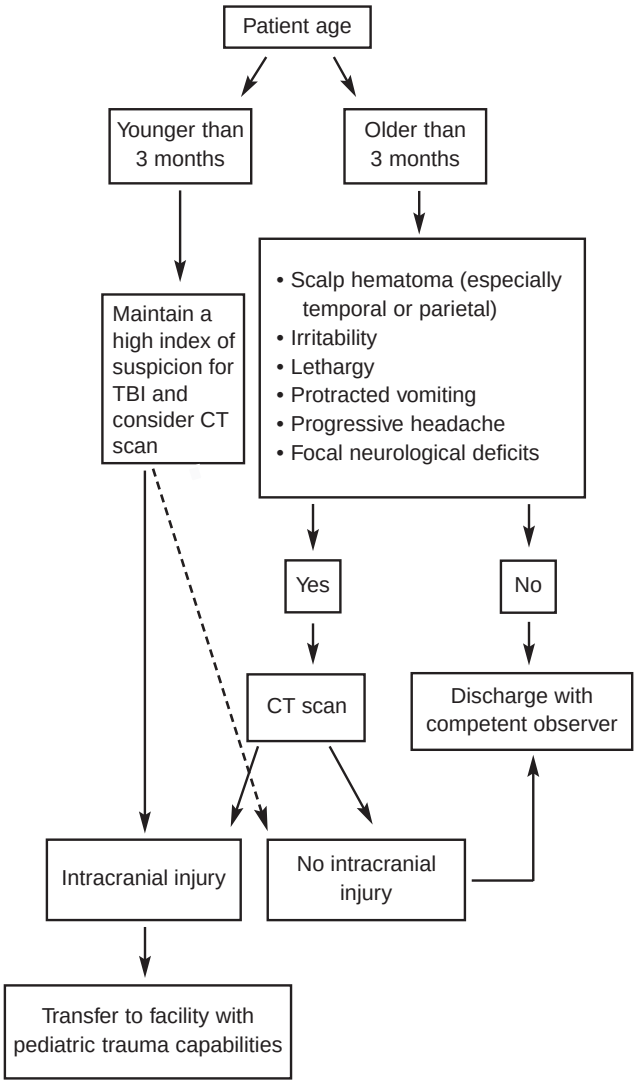
Mannitol works in a similar fashion by decreasing blood viscosity and, thereby the diameter of cerebral blood vessels. Cerebral blood flow is maintained by reflex vasoconstriction of the cerebral vasculature, but cerebral blood volume and ICP are reduced.[54] This mechanism relies on intact autoregulation 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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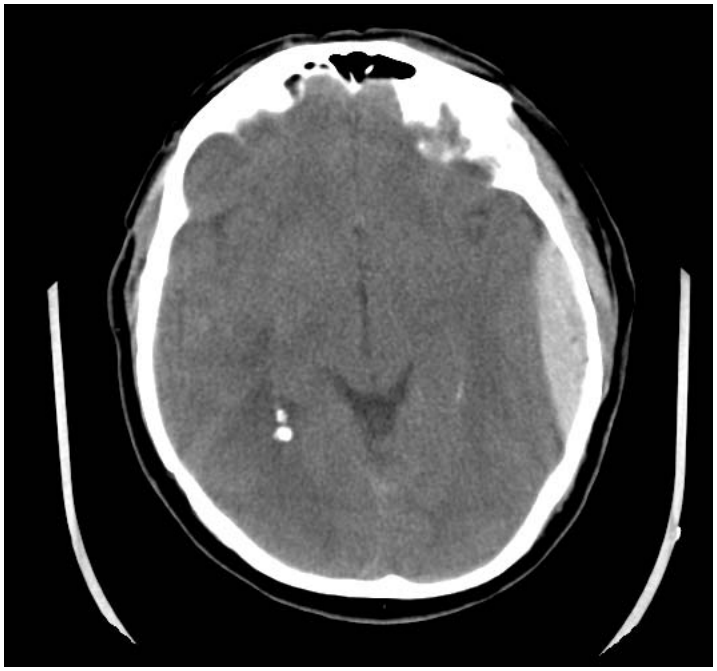
CE/CME Questions

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**

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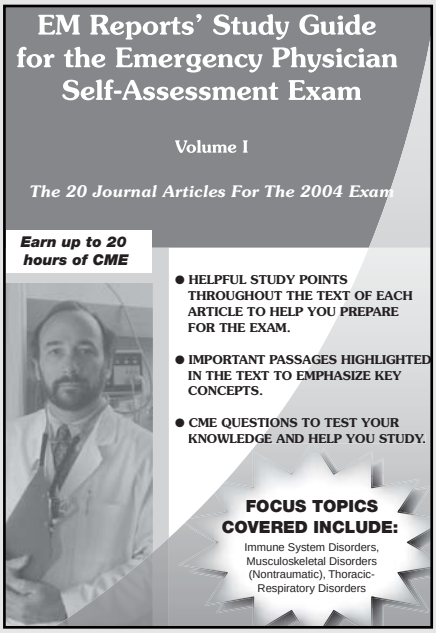

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