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Neurologic Evaluation and Support in the Child with an Acute Brain Insult

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A deteriorating neurologic state in any child with an acute brain insult should be considered to be a life threatening condition; the decisions made during the initial minutes of management often will dictate the outcome. The definition of the management often will dictate the outcome. The definition of the various levels in disturbance of consciousness are not uniform. For the purposes of this discussion, lethargy implies that the patient is sleepy but arousable and capable of responding to verbal commands and tactile stimuli; stupor means that the child falls asleep when left alone but can be aroused by verbal or painful stimuli; obtundation describes the patient who is unconscious but responds to extreme stimuli by crying out or moaning; coma means that regardless of stimulation the patient cannot open eyes, give verbal responses, or obey commands.

The Glasgow Coma Scale (GCS) has been found to be predictive of the outcome of head injured patients and is a useful system for categorizing the severity of central nervous system (CNS) impairment.[1] Furthermore, the GCS implies that the best indicators of outcome in the patient with an acute brain insult are the ability to open one's eye or to speak in response to verbal or tactile stimulation (Table 1). In Jennett's initial studies of head injured patients who were unable to spontaneously open their eyes within 24 hours of illness, 64% were dead or in a vegetative state within 6 months; on the other hand, all those patients who were responsive within 24 hours, 65% had a good recovery. It should be emphasized that these data are reflective of a patient population with a mean age of 33 years; therefore a modified GCS for infants is presented in Table 2.

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DIFFERENTIAL DIAGNOSIS AND MANAGEMENT

There are multiple causes of coma or other alterations in the level of consciousness. However, because initial management often is instituted prior to identification of the cause and must be rigorous regardless of etiology, a therapy directed diagnostic protocol will be presented prior to the discussion of the specific entities.

General Measures

Resuscitation of the patient with CNS disease begins with airway control and ventilation. Children with impaired consciousness often develop upper airway obstruction due either to pharyngeal hypotonia or the inability to clear pharyngeal secretions. Obstruction to airflow with resultant hypoxemia and respiratory acidosis will: aggravate or promote intracranial hypertension[2]; may potentiate pulmonary hypertension particularly in infants with a reactive pulmonary vascular bed[3]; and will interfere with oxygen delivery to vital organ systems.[4] Since assisted ventilation and oxygenation is most easily controlled with the use of an artificial airway, we recommend endotracheal intubation of patients with 1) evidence of intracranial hypertension (see below), 2) difficult to control seizures, 3) any abnormality of respiratory rate or rhythm that interferes with gas exchange, or 4) a GCS of eight or less.

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TABLE 1
GLASGOW COMA SCORE

Activity	Best Response	Score
Eye Opening	Spontaneous	4
	To verbal stimuli	3
	To pain	2
	None	1
Verbal	Oriented	5
	Confused	4
	Inappropriate words	3
	Nonspecific sounds	2
	None	1
Motor	Follows Commands	6
	Localizes pain	5
	Withdraws in response to pain	4
	Flexion in response to pain	3
	Extension in response to pain	2
	None	1

TABLE 2
MODIFIED COMA SCORE FOR INFANTS

Activity	Best Response	Score
Eye Opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Verbal	Coos, Babbles	5
	Irritable cries	4
	Cries to pain	3
	Moans to pain	2
	None	1
Motor	Normal spontaneous movements	6
	Withdraws to touch	5
	Withdraws to pain	4
	Abnormal flexion	3
	Abnormal extension	2
	None	1

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Assessment of the patient's hemodynamic status is discussed elsewhere in this symposium. In terms of brain resuscitation, maintenance of an "adequate" cardiac output to assure appropriate cerebral blood flow cannot be over emphasized. Fluid support of the circulation is critical but excessive free water may augment existing brain edema, and therefore hypotonic fluids should be avoided. Hypotension may result in inadequate cerebral perfusion and therefore must be treated, utilizing appropriate fluid infusion and inotropic agents if necessary. Placement of a central venous or pulmonary artery catheter for determination of cardiac function may guide management in the hemodynamically unstable child. It should be stated that hypovolemia in a head injured patient most commonly is due to blood loss related to associated abdominal, pelvic, or long bone trauma and not the result of intracranial hemorrhage.

All pathophysiologic factors that are known to increase intracranial pressure or diminish cerebral perfusion are to be avoided in the initial phases of brain resuscitation.[2] The patient's head should be placed in the midline position and be elevated at a 20 to 30° angle to maximize cerebral venous drainage. Hyperthermia, seizure activity, hyponatremia, hypoglycemia, and metabolic acidemia must be treated.[5]

If intracranial hypertension is suspected, diagnostic and therapeutic maneuvers must be instituted during initial resuscitation. Intracranial hypertension should be *suspected* in any patient with a CNS insult and 1) a GCS of eight or less, 2) difficult to control seizure activity, 3) abnormalities in vital signs (systemic hypertension, bradycardia, abnormal respiratory patterns), 4) dilated, unreactive pupils, or 5) decerebrate or decorticate posturing.[6] Hyperventilation (hypocapnia) is a means of reducing intracranial pressure (ICP) by decreasing the cerebral blood volume, and we recommend a reduction of the arterial pCO2 to 20 to 25 mmHg. Osmotic agents may be administered to decrease ICP by the removal of free water from brain cells with an intact blood brain barrier; intravenous mannitol at a dose of 0.25 to 1.0 gm/kg may be safely administered once a bladder catheter has been placed.

After these initial resuscitation steps have been performed, the patient should be taken for a computerized tomography (CT) study of the brain to rule out the presence of surgically treatable lesions such as the accumulation of extracerebral blood or acute hydrocephalus. If the CT scan is consistent with brain swelling alone, then the patient should be transported to the pediatric intensive care unit for the initiation of the secondary phases of brain resuscitation,[7] including ICP monitoring.[8]

Specific Measures

There are three categories of CNS lesions which produce alterations in the level of consciousness: supratentorial mass lesions, infratentorial mass lesions, and metabolic abnormalities. A convenient way of determining the specific problem is to evaluate the neurologic findings (Table 3) and the acid base state (Table 4) at the time of presentation. Detailed discussions of the specific entities are included in the references.[7-10]

The important causes of supratentorial mass lesions producing progressive deterioration in children include: cerebral hyperemia secondary to head

TABLE 3
NEUROLOGIC FINDINGS IN THE COMATOSE CHILD

Supratentorial Lesions
1. Initial signs and symptoms suggest focal hemispheric disease.
2. Signs progress in a rostal to caudal direction.
3. Pupillary reflexes are usually depressed.
4. Motor signs are often asymmetrical.

Infratentorial Lesions
1. Brainstem signs and symptoms are common.
2. Signs are not rostal to caudal in evolution.
3. Cranial nerve palsies are common.
4. Abnormalities of the respiratory pattern are common and appear at the onset of coma.

Metabolic Lesions
1. Stupor or coma precede motor signs.
2. Motor signs are usually symmetrically depressed.
3. Pupillary reactions are preserved.
4. Acid-base imbalance is common.
5. Seizures or abnormal motor movements are common findings.

TABLE 4
ACID-BASE IMBALANCE IN THE COMATOSE CHILD

Metabolic Acidosis (increased anion gap)
1. Lactic acidosis (hypoxic-ischemic insult; septic shock)
2. Diabetic ketoacidosis
3. Renal failure
4. Organic aciduria
5. Ingestions (e.g. methanol, ethylene glycol)
6. Salicylate poisoning (late)
7. Diarrhea

Respiratory Acidosis (apnea or hypoventilation)
1. Supratentorial or infratentorial lesions
2. Ingestions (e.g. narcotics, clonidine)
3. Respiratory muscle fatigue; neuromuscular disease
4. Metabolic encephalopathies
5. Generalized seizure activity

Respiratory Alkalosis (hyperventilation)
1. Intracranial hypertension
2. Septic shock (early)
3. Hepatic failure
4. Salicylate poisoning (early)
5. Reyes syndrome
6. Brainstem dysfunction

TABLE 5
DIFFERENTIAL DIAGNOSIS OF METABOLIC COMA IN INFANCY

I. Hypoxic-Ischemic
  A. Respiratory failure
  B. Shock syndromes
  C. Severe anemia
  D. Apnea of infancy
  E. Carbon monoxide poisoning
  F. Cerebral vasculitis ("cerebritis")
II. Infections
  A. Encephalitis
  B. Meningitis
  C. Botulism
III. Posticta1 state
IV. Hypoglycemia
V. Non-Endocrine organ failure
  A. Hepatic
  B. Renal
VI. Endocrine Organ Failure
  A. Pancreas
  B. Adrenal
  C. Thyroid
  D. Pituitary
VII. Poisonings
  A. Narcotics
  B. Barbiturates
  C. Sedatives
  D. Others
VIII. Miscellaneous
  A. Reyes Syndrome
  B. Electrolyte Abnormalities
  C. Hypo or hyperthermia

trauma,[10] epidural and subdural hematomas, intracerebral hemorrhage, acute hydrocephalus, and subdural empyemas. In most instances, patients in this category will present with evidence of increased intracranial pressure; the initial management has been presented. Surgical decompression is the definitive treatment when cerebral hemorrhage results in brain shift, a herniation syndrome, or acute hydrocephalus. The most common clinical situations in which children present with acute supratentorial lesions include: accidental and non-accidental head trauma, severe systemic hypertension, obstruction of an existing ventricular-peritoneal shunt, and a bleeding arteriovenous malformation. Because of the special nature of non-accidental head trauma as a cause of coma, this will be presented in a following section.

Infratentorial (posterior fossa) lesions may cause coma by either destroying the ascending reticular activating system or by compression of the blood supply to this system by a tumor or mass. Coma due to a posterior fossa lesion can be easily differentiated from supratentorial lesions only if localizing brainstem signs precede the onset of coma. Important infratentorial lesions to consider are brainstem contusions associated with head trauma, basilar artery thrombosis (rare in children), cerebellar hemorrhage or tumor with secondary hydrocephalus, and brainstem encephalitis. Midbrain or pontine lesions may lead to abrupt coma, central neurogenic hyperventilation, absent oculovestibular and oculocephalic reflexes, pinpoint pupils,

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and decerebrate rigidity.

Metabolic disorders constitute the majority of non-traumatic processes responsible for acute coma in children; Table 5 presents the differential diagnosis. Changes in mental status are early signs of metabolic encephalopathy. Hypoglycemia is an important cause of coma since it is so readily treated; hyponatremia may occur because of excessive free water, inadequate salt intake, or the inappropriate secretion of antidiuretic hormone (SIADH). Hepatic or renal failure is rarely responsible for an acute change in mental status. Reye's Syndrome, however, is a cause of diffuse brain swelling, intracranial hypertension and coma.[6] Bacterial meningitis and herpes encephalitis are treatable causes of coma; if intracranial hypertension is not suspected, a lumbar puncture must be performed on all children with fever and altered mental status. Generalized seizures are another cause of prolonged unresponsiveness; if the post ictal state persists for greater than one hour, then an electroencephalogram should be performed to rule out clinically "silent" seizure activity. The ingestion of poisons should always be considered in the comatose child who has been previously well. A history of medications available in the home, a toxicology screen, and evaluation of pupillary size and reactivity (Table 6) are useful diagnostic features.

The Figure provides an algorithm for the approach to the child who presents in coma.

Child Abuse Syndrome

Child abuse is a major sociological problem and often a medical diagnostic one as well.[12-14] Unsuspected cerebral injury is the most common cause of death and disability. Factors which make the diagnosis difficult include the absence of a history of trauma, a vague clinical presentation, and, frequently, the paradox of significant intracranial bleeding without external signs of trauma to the head and face.[12,13]

Severe head injuries in the first year of life are usually the result of child abuse.[12] A shaking injury is the most frequent one encountered. Shaking injuries are particularly devastating as there is significant risk of death and a high incidence of mental retardation and neurological deficit.[12,13] Unfortunately, the signs and symptoms of this form of head trauma are non-specific. The findings may mimic infection, intoxication or metabolic abnormalities. Diagnosis depends on a high index of suspicion and the physical findings of a bulging fontanelle, head circumference greater than the 90th percentile and retinal hemorrhage.[13] The finding of bloody fluid from a lumbar or subdural tap is also highly suggestive.[13] Cranial CT findings confirm the diagnosis.[12-14]

The diagnosis must be considered when an infant arrives in the emergency room in coma with decerebrate posturing or flaccidity, dilated pupils and apnea or bradypnea with an extremely tense fontanelle. These children must be immediately intubated (with precaution to avoid further increase in intracranial pressure), hyperventilated with 100% oxygen and consideration given to tapping the lateral margins of the fontanelle with a 19 or 21 gauge spinal needle. Usually, 10 to 15 cc of bloody, non-clotting, fluid can be obtained from either side. This often produces a dramatic improvement in the neurologic state. The patient should then be transported with cerebral monitoring for a CT scan. Less severely affected infants presenting with full fontanelle, retinal hemor-

TABLE 6
PUPILLARY EXAMINATION AND COMA

MYDRIASIS	MIOSIS
Amphetamines	Barbiturates
Antihistamines	Opiates
Atropine	Propoxyphen
Botulism	Meperidine
Cocaine	Methadone
Ephedrine	Carbon Monoxide
Ethyl Alcohol	Organophosphates
Snake Venom	Clonidine

Initial Evaluation:	Secondary Evaluation:	
Stabilize neck	(1) For Increased ICP:	Hyperventilation (PaCO2-25mmHg)
Assure airway, breathing, circulation		Osmotic Agents (Mannitol 0.25-1 g/kg)
Obtain screening laboratory tests including toxicology screen		CT Scan
Administer 25% glucose (2-3cc/kg)	(2) For Normal ICP:	lumbar puncture
Administer Naloxone (0.01mg/kg)		electroencephalogram
Administer anticonvulsant medications		

Figure. Algorithm for the management of the child in coma.

rhages and altered neurologic states should also be immediately scanned. Regardless of initial presentation, these infants must be carefully observed since secondary cerebral swelling may occur. The emergency physician must be alert to make this diagnosis in order to promptly institute therapy for acute head trauma.

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