Topical Review

Head Injury in Children

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ABSTRACT

Trauma in the United States is the leading cause of death and disability in the pediatric population. Differences of age and development affect recovery and outcome following head injury. Mechanisms and pathophysiology of head injury are varied in both the pediatric and adult populations. Assessments of injury are varied and some measurements are more sensitive than others as well as more specific with regard to certain types of injury. Treatment and management should be tailored to each case in order to effect a positive outcome with respect to brain functioning. Aggressive intervention for prevention of primary and secondary injury must be continued and understanding of the impact of these injuries should provide for a brighter future for these patients. (*J Child Neurol* 1998;13:2–15).

Trauma in the United States despite multiple preventative measures, remains a significant societal and public health problem. It is the leading cause of death and disability accounting for over 50% of the deaths in the pediatric population.[1] The majority of these cases also involve head injury, which is a major factor affecting mortality and outcome.[2] Even in children who suffer only mild or moderate head injuries, there is often long-term cognitive and motor dysfunction, particularly in severely injured children.[3] It has been estimated that the annual economic burden resulting from head injury in children in the United States is between $10 and 15 billion dollars, largely related to medical expenses and rehabilitation.[4] This is expected to continue to increase as more and more children with acquired disabilities require extended services throughout their lifetimes.

Because of the uniqueness of the immature brain and its response to injury, it is important to understand the role that the overall differences of age and development play both in the acute setting following a head injury and during the chronic period of recovery. As well, it is important to differentiate the mechanism of injury and physiologic responses not only in children as a group compared to adults, but the differing responses in children at different stages of maturity. This review of head injury in children discusses the different mechanisms and types of injury seen in children, the management issues and their implications in children, and last, the unique relationship between injury and recovery in the pediatric population.

EPIDEMIOLOGY AND MECHANISMS OF INJURY

There are approximately 100,000 to 200,000 new head injuries in children each year with an estimated population incidence of 193 to 367 per 100,000.[5–7] Patients under the age of 18 years constitute the majority of victims of traumatic brain injury[8] and in children there are two peak periods of incidence: early childhood (less than 5 years of age) and mid-to-late adolescence.[2] Up to the age of 5 years, the incidence of head injury between males and females is relatively equal; however, after the age of 5 years, the incidence of head injury among males is higher, with a ratio ranging from approximately 2–4:1.[2] Socioeconomic factors can also affect the rate of head injuries in all ages with the highest rate of injuries reported among the lower socioeconomic classes. Each year, 10% to 15% of children hospitalized with head trauma suffer a severe head injury[7]; of these children, the mortality rate is approximately 33% to 50%, although the survivors of injury often have permanent deficits.[8]

Traumatic brain injury can be grouped into multiple categories depending on the mechanism of injury: focal versus diffuse injuries, closed head versus penetrating injuries, and primary versus secondary injuries. Diffuse injuries are more common in children than focal injuries, but closed head injuries account for the vast majority of traumatic brain

Received Jan 31, 1997. Accepted for publication July 30, 1997.

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injury (95%).[9] Focal, diffuse, closed head, and penetrating injuries are all considered primary mechanisms of injury and occur at the time of impact. Each of these primary mechanisms potentially elicit a secondary response from the brain as a reaction to that injury. This secondary response is a cascade of biochemical and physiologic events within the brain that is believed to contribute to the diffuse cerebral swelling and further tissue damage and loss frequently seen following pediatric traumatic brain injury.

It is important to differentiate between the secondary injury from this cascade of events and secondary injury caused by secondary insults. The secondary response normally observed after traumatic brain injury includes the loss of cerebral autoregulation, breakdown of the blood-brain barrier, intracellular and extracellular edema (cytotoxic versus vasogenic), diffuse and focal edema, and ischemic brain injury. Multiple factors are believed to contribute to the evolution of the injury including intracranial hypertension from diffuse swelling, ischemia, and vasospasm. In addition to the evolution of the secondary damage in the injured brain, injury can be greatly augmented by secondary extracerebral insults such as hypoxemia or hypotension. Aldrich et al[1] reported a strong association between diffuse brain swelling and either hypoxemia or early hypotension, and commented that these factors play a particular role in the pathogenesis of diffuse swelling frequently seen in children. Children can be more prone to suffer a hypoxemic or hypotensive episode post trauma and as a result can be more likely to develop diffuse swelling.

Age-dependent factors influence the severity of the insult, the inherent susceptibility of the brain, and the regenerative mechanisms that all can impact on mortality and the extent of tissue damage. The very young developing brain can be particularly susceptible to extensive damage and, as a result, have a higher likelihood of worse outcome. It is during this developmental period that the immature brain can be anatomically more vulnerable to shearing, or to disturbances of autoregulation of cerebral perfusion after the injury.[10–12]

Mechanisms of Injury

In adults, the majority of traumatic brain injuries occur secondary to passenger motor vehicle accidents.[2,6] Though similar to adults, in that head trauma in children (more than 4 years old) is often motor vehicle related,[9] the percentage of motor vehicle and motor vehicle–related accidents also increases with increasing age: 20% in children 0 to 4 years of age, up to 66% in adolescents.[9] Younger children more commonly suffer pedestrian or bicycle-related injuries whereas adolescents are more often injured in motor vehicle accidents as passengers similar to adults. Motor vehicle injuries also account for the highest proportion of fatal injuries in all groups. Among infants, toddlers, and young children, the major causes of head trauma are assaults/child abuse and falls accounting for nearly 50% of cases. In older children, falls and assaults/abuse result in less than 20% of traumatic brain injury. In older children, other etiologies of traumatic brain injury include: sports- or recreation-related injuries and penetrating injuries. In later childhood, including adolescence, the leading causes of trauma are similar to adults and include motor vehicle accidents (as passenger), sports, and recreational activities.[2,6,9]

Child Abuse

Child abuse tends to occur more often in the very young child (less than 4 years of age) and may even be the major cause of severe brain injury in this group, representing almost ⅔ of head injuries in the 0- to 4-year-old range in some series.[13] The uniqueness of child abuse compared to other injuries is that in "shaken baby syndrome," the injuries tend to be multiple and diffuse and differ from motor vehicle- or sports-related injuries, which more likely result from a single impact. As well, these children often have a secondary injury related to hypoxia and hypotension, possibly from the delay of reporting the injury.[14] Child abuse reports along with mortality reports of assaults of children have increased over the past few years[13] and these trends probably have multiple reasons. Physicians are much more aware of this mechanism of injury in young children, and as a result, are better able to identify suspicious "falls" and report them to investigatory services. Previously, many reports noted a significant number of fatalities as a result of falls from less than 3 feet.[15] It is now clear that fatalities from falls from a short distance are rare and are most likely attributable to inaccurate reporting. Second, there are now many state laws that require the reporting of suspected child abuse and, as a result, physicians who were uncomfortable in breaching the doctor-patient and family relationship and confidentiality now are duty bound to report concerns. This legal support of the child relieves the physician of the responsibility of being the major factor in the legal decision making, and keeps the interaction on a patient-physician level. Last, hospitals and supportive services are now much more of an advocate of the child and services are available to help families of abused children to counsel and treat rather than remove the child permanently from the home. In this way, further injury and possibly death can be avoided.

PATHOPHYSIOLOGY OF PEDIATRIC TRAUMATIC BRAIN INJURY

Though similar pathologies can be seen in both the adult and child following head injury, each group has different incidences of findings. Focal injuries such as subdural, epidural, and intracerebral hematomas occur with a higher incidence in adults (30%–42%) vs children (15%–20%).[16–18] Younger children (≤ 4 years of age) tend to have a higher incidence of hematomas compared to older children where the incidence is quite uncommon. Focal injuries are most often caused by a single impact in a linear vector. Skull fractures occur in approximately 5% to 25% of children and are less commonly associated with epidural hematomas (40%) vs adults (61%). Frontal epidural hematomas are often associated with parenchymal injuries in both adults and

children.[2,19,20] Hematomas are more often attributable to falls in children and to motor vehicle accidents in adults.[2,19,20]

Children more frequently present with diffuse injury and cerebral swelling with resultant intracranial hypertension compared to adults,[1,21,22] and up to 44% of children exhibit diffuse cerebral swelling after severe traumatic brain injury.[21] Although it has been hypothesized that the diffuse swelling phenomenon in children may be neuroprotective, since they often have better outcomes than adults following severe traumatic brain injury,[1,12] other clinical studies have reported a 50% mortality in children when diffuse swelling is noted on computed tomographic (CT) scans.[1] This is comparable to the observed mortality rates in similarly injured adults.[1,2,15] As well, increased mortality following severe traumatic brain injury has been reported in the younger children (< 4 years old) who more commonly develop diffuse swelling compared to adolescents.[2]

The histopathologic findings of a diffuse, primary injury include: diffuse axonal injury or shearing, or vascular injury. These injuries can result from linear acceleration injury, but are most often due to rotational acceleration and deceleration forces. Axonal injury, which can be focal or diffuse, represents the stretching or transection of axons by shearing forces that at times lead to widespread disconnection. The pathologic findings in these patients include shear injury in the corpus callosum, subcortical white matter, cerebellar peduncles, and the brain stem. Diffuse axonal injury is possibly a key limiting factor in the recovery and rehabilitation of these children. Children are more susceptible to this type of injury because of their unique anatomical relationships of the head to body ratio, weak neck musculature, and the lack of myelination.

The typical findings at autopsy in the brains of children with diffuse cerebral swelling are vascular congestion with absent cerebrospinal fluid spaces.[10,11,23–25] Diffuse cerebral swelling has been defined radiologically, using CT scans, as compressed or obliterated mesencephalic cisterns and other cerebrospinal fluid spaces and small to normal size ventricles in the absence of other intracranial pathology.[1,12,22] Up to 90% of these children also have findings of ischemic neuronal damage or infarction.[26] This entity may soon be better defined in children prior to autopsy with the development of novel imaging techniques to differentiate vasogenic and cytotoxic edema. Apparent diffusion coefficient maps from diffusion-weighted images using magnetic resonance imaging (MRI) have shown in experimental traumatic brain injury studies significant cytotoxic edema formation and ischemia, and suggested that this secondary response plays a major role in the development of diffuse swelling after traumatic brain injury.[27] Perfusion and functional magnetic resonance imaging have also been used to identify changes in cerebral blood flow, disturbances in the blood-brain barrier, and the development of vasogenic edema in experimental traumatic brain injury studies.[28] These imaging modalities will soon be available for clinical application, and could be particularly helpful in defining the secondary responses to injury contributing to intracranial hypertension.

Cerebral Blood Flow

Early outcome studies of severe traumatic brain injury in children suggested that the diffuse brain swelling was from vasomotor paralysis, cerebrovascular dilation, and increased cerebral blood volume, but *not* edema.[29] On CT scans, the brains in these patients had increased density due to hyperemia, which was believed to result in the "malignant" brain swelling observed. More recently, this concept has come into question. Early after traumatic brain injury in adults, cerebral blood flow was found to be low with an increase in the arterial-jugular venous difference in oxygen content, suggesting early hypoperfusion or ischemia. It is during this early period following the injury with early flow reduction that oxygen delivery can be marginal, and can be contributory to outcome in the acute period in both children[30–32] and adults.[33] One third of patients with severe traumatic brain injury had "ischemic" cerebral blood flow (≤ 18 mL/100 g/min). In the more delayed phases (after 24 hr), cerebral blood flow increases with an apparent uncoupling of cerebral blood flow and oxidative cerebral metabolism (cerebral metabolic rate of O_2 [$CMRO_2$]). There is a low arteriovenous difference of O_2 ($AVDO_2$) which suggests hyperemia.[34] In this study, a more severe hyperemia and uncoupling of cerebral blood flow and $CMRO_2$ were noted at deeper levels of coma, and occurred more often in younger patients. Post-traumatic hyperemia is more common in children (> 70%) than in adults (46%).

Clinically, the concern has been that increased cerebral blood flow and cerebral blood volume may cause intracranial hypertension that needs to be carefully managed to avoid herniation or focal ischemia. It remains unclear, though, whether or not the mechanisms of hyperemia, edema, or ischemia are major factors leading to the resultant cerebral swelling seen in children.[1,12] As well, the importance of glucose or nonoxidative metabolism in the early period after traumatic brain injury remains unclear. Experimental studies have shown early hyperglycolysis with delayed recovery of oxidative metabolism.[35,36] Recent work using positron emission tomography (PET) to measure the cerebral metabolic rate of glucose (CMRglc) has shown an early hyperglycolytic period shortly after injury.[37] This novel approach has yet to be tried in children, but may help to elucidate whether or not this early hyper-nonoxidative metabolic state in combination with low early cerebral blood flow is an important mechanism in the evolution of secondary injury.

Normal autoregulatory mechanisms regulate cerebral blood flow through the cerebral vasculature in response to the cerebral metabolic and physiologic needs. Among the variables that contribute to these relationships include: P_aCO_2, P_aO_2, $CMRO_2$, CMRglc, cerebral perfusion pressure (CPP), and blood viscosity. Following injury, these mechanisms can be disturbed, though the extent of impact to each is unclear. Some studies comment that knowing if there is impaired autoregulation helps to determine the effectiveness of certain therapeutic interventions, namely mannitol administration. Metabolic or pressure autoregulation, or

both, can be impaired after traumatic brain injury in some children,[38] whereas in others, they are intact.[39] Besides the vascular reactivity and impairment of autoregulatory mechanisms, blood-brain barrier disruption and the development of cerebral edema (both cytotoxic and vasogenic) are also important pathophysiologic processes associated with severe injury, though the extent and contribution in children is also unclear. Normally the intact blood-brain barrier is highly impermeable to polar molecules and charged species including sodium (Na^+). With damage to the barrier in conjunction with hyperemia, vasogenic edema can occur with increased osmotic as well as oncotic gradients contributing to the fluid flux into the extracellular space. This is distinct from cytotoxic edema, which results from the intracellular fluid fluxes caused by primary or secondary damage to the tissues. Edema and initial vascular permeability from blood-brain barrier compromise may contribute to coma, brain swelling, and intracranial hypertension, particularly in the early acute period. The blood-brain barrier can reconstitute within the first 24 hours, although a secondary episode of increased permeability is possible. Peak edema generally occurs between 24 and 72 hours after the injury, most likely secondary to cytotoxic edema and delayed tissue damage. It is believed that the breakdown in the cells' normal metabolic processes in response to the injury and inability to correct imbalances in the ionic species (Na^+, K^+, Ca^+) disturbances lead to further cellular damage and fluid influxes. Other contributing factors to edema include hypoxia-ischemia, inflammation, free radicals, excitotoxicity, and calcium influx. The extent that each of these processes factors into the resultant cerebral swelling is answered with the apparent diffusion coefficient maps and diffusion-weighted imaging.

Biochemical Cascade

Additionally, endogenous cascades as a result of the secondary response to traumatic brain injury play a role in the worsening of secondary brain injury. The importance of excitatory amino acids (glutamate and aspartate) causing or enhancing secondary brain injury has been reported in many experimental models,[40] and there appears to be a consistent correlation between high extracellular levels of these neurotoxic substances and the severity of brain damage.[41,42] In humans, Baker et al[43] serially measured cerebrospinal fluid levels of excitatory amino acids in patients after traumatic brain injury as well as control patients. They found a significant elevation of glutamate in the cerebrospinal fluid almost immediately after injury and found that such high levels persisted even up to 168 hours later. There was also a correlation between the level of glutamate and the extent of anatomical brain injury as determined on CT scan. Palmer et al[44] also demonstrated a progressive and sustained increase in glutamate levels in ventricular cerebrospinal fluid of adults after severe traumatic brain injury. Persson et al[45] also showed, using cerebral microdialysis, an elevation (up to 25 times) in glutamate concentrations in the extracellular fluid in the early period following injury.

With the early findings of regional and global ischemia in pediatric head injury and the association of hypoxic-ischemic episodes to outcome,[33] the effects of ischemia on the pathophysiologic cascade are believed to remain an important facet to the ongoing secondary injury. Ischemia contributes to energy failure leading to depolarization, spreading depression, calcium accumulation, lactate accumulation, and acidosis.[46] This can lead to further cellular breakdown and damage. A local inflammatory response is also initiated in the brain after traumatic brain injury. Cerebrospinal fluid levels of cytokines (IL-1, IL-6, and IL-10) are increased.[47] In addition, there is complement activation,[48] leukocyte infiltration,[49] and microglial activation.[50] IL-6 has been shown to be elevated in the cerebrospinal fluid of children early after traumatic brain injury, to levels comparable to those seen in blood during sepsis.[47] As well, levels of IL-10 were higher in those patients with a poor outcome and young age. Systemic effects of traumatic brain injury include suppression of cell-mediated immunity that occurs in both children and adults and could explain the high rate of secondary infection in the subacute phase of recovery. This biochemical "cascade" in combination with the cerebrovascular response and edema all contribute to diffuse cerebral swelling and uncontrolled intracranial hypertension. Additional studies on cerebrospinal fluid and with microdialysis will be important in the pediatric age group to understand the biochemical response to traumatic brain injury and will be essential to the development of novel treatment modalities to interrupt the ongoing secondary injury.

INITIAL ASSESSMENT AND MANAGEMENT

ABCs

In the initial stabilization of all trauma patients, including head-injured children, control of the airway and adequate ventilation ("breathing") are the first priorities. In traumatic brain injury, it is particularly important to maintain oxygenation and prevent hypoxemia, since even moderate reductions in P_aO_2 can contribute to secondary neural injury in the injured brain. The traumatically injured brain is possibly particularly susceptible to secondary insults such as hypoxia-ischemia. Whereas moderate levels in experimental models of hypoxia may not produce pathologic effects in the normal brain, hypoxia can induce permanent damage in the traumatically injured brain.[28] In addition, even moderate hypoxia (P_aO_2 < 40–50 Torr), which may not reach levels that affect cerebral viability, is a potent vasodilator and may contribute to "cerebral swelling." It is also important to maintain normocarbia, since even moderate hypercarbia can cause arteriolar vasodilation and increased cerebral blood volume, which could further contribute to increased intracranial pressure and possibly precipitate herniation.

Since adequate perfusion is the goal in the treatment of patients with severe traumatic brain injury, blood pressure is maintained at normal to slightly elevated for age. Blood pressure is continuously monitored to help ensure adequate tissue perfusion and oxygen delivery at any given

time. Often following trauma, besides the head injuries, there can be extensive extracranial injuries that produce shock or hypotension from hemorrhage. Young children (< 4 years) have a higher incidence of hypotension compared to other age groups.[2,21] Volume expanison is critical in this setting, but it is important to monitor the extent of the volume expansion to avoid the iatrogenic increase in the central venous pressure with or without exacerbation of cerebral edema.

Initial Neurologic Assessment

The initial examination and neurologic assessment are simultaneously obtained to provide a baseline anatomical and functional status, which has prognostic and therapeutic importance. This evaluation minimally consists of the Glasgow Coma Score, cranial and cranial nerve examination, motor, sensory, and reflex examinations. Lacerations and signs of skull fractures can usually be discovered during the primary survey where actual depressions or fractures can sometimes be palpated through the scalp or when probing a laceration. Otorrhea, rhinorrhea, Battle's sign, or raccoon eyes may be indicative of basilar skull fracture and other intracranial damage.

The cranial nerve or brainstem assessment often includes the pupillary response to light, eye position and movement (conjugate, disconjugate, roving, or fixed), corneal sensation, and gag response. Head injury with brainstem dysfunction is associated with a worse prognosis.[2,51] The neurologic assessment obtains the baseline evaluation of the somatosensory system that determines not only the functional aspects of strength and sensation, but also symmetry. Most major muscle groups can be tested for motor function and the sensory examination encompasses all modalities including light touch, pinprick, and proprioception (position sense). The reflex examination, particularly of the sacral reflexes, can be variable but is important in the trauma patient, particularly if there is a suspected spinal cord injury.

Radiologic Assessment

Plain Films

During the initial resuscitation in the emergency department, in conjunction with the standard series of evaluative plain trauma films, a lateral cervical spine radiograph is obtained. Clearance of the cervical spine in these patients requires the completion of the standard 3 views (anterior-posterior [AP], lateral, and open-mouth) that adequately visualizes C-1 through T-1. Questions of abnormalities or instability may require further assessment with additional views or CT scan. Plain skull radiographs are probably not useful in the age of CT scans. If one suspects a skull fracture, the resultant anatomical study via CT scan is more definitive for both bone and underlying parenchymal injury. If a skull film shows a fracture, a CT scan will often be obtained to ensure that no extra-axial collections of blood are present. Where there is concern about sedating the young child for the radiologic study, especially if they are neurologically normal, a skull radiograph may be helpful if there is a scalp hematoma and the underlying skull cannot adequately be palpated.[18] The decision to obtain a CT scan in these patients should be based on the above criteria for severity even if they have a Glasgow Coma Score of 14 or 15 and have normal neurologic and radiologic examinations.

CT Scan

Rapid diagnosis and appropriate treatment of neurologic injuries is essential to optimize outcome. A CT scan of the head provides a quick reliable indicator of the extent of injury and is obtained in patients who have an altered level of consciousness (Glasgow Coma Score ≤ 13), focal neurologic signs, or preceding any surgical intervention such as laparotomy or open reduction of fracture, especially when anesthesia will preclude assessment of the mental status for any length of time. This is particularly important if the history revealed a loss of consciousness, a severe accident or mechanism of injury, a seizure or a deformation of the scalp or skull on examination. CT scanning, though, cannot be completely relied upon to define injury, since a normal CT scan does not completely rule out the presence of intracranial hypertension. O'Sullivan et al[52] demonstrated that in severely head-injured patients with a relatively normal CT scan, almost all of the patients had significant intracranial pressure elevations and decrements in cerebral perfusion pressure requiring treatment; 25% of these patients eventually died. The Traumatic Coma Data Bank noted that if the initial CT scan was normal, 10 to 15% would still have significant intracranial hypertension. Good outcome in these patients was only 71%.[9,53] If other injuries demand immediate intervention prior to CT scan, intraoperative intracranial pressure monitoring may be required during the procedure to prevent undiagnosed intracranial hypertension.

Measures of Cerebral Blood Flow and Metabolism

Several methods have been utilized to assess the changes in regional and global cerebral blood flow after head injury.[54] With the increased understanding into the unique pathophysiologic response of individual patients following their injury, it is necessary to obtain increasing information on each patient to make rational, individualized decisions regarding patient care. Stable xenon-CT (XeCT) scan uses nonradioactive xenon gas as a tracer to determine regional cerebral blood flow. Since it is directly coupled to the CT scan, the functional differences of focal and diffuse injury can be correlated to the anatomical abnormalities.[55,56] Using stable xenon-CT scan, the measure of metabolic or pressure autoregulation of cerebral blood flow (through the manipulation of P_aCO_2 and blood pressure) can be achieved in the clinical setting, which helps guide clinical care. The other more common tool for measuring cerebral blood flow is the radioactive ^{133}Xe method that also provides a regional assessment, but has limited anatomical correlates. It is a useful adjunct since it can be performed at the bedside and serial

studies can be obtained with little manipulation of the patient.

Another technique that was initally popularized by Obrist et al[57] and has been regaining popularity is the measurement of jugular venous saturation. Immediately following head injury, there is a jugular venous desaturation and increased cerebral metabolic rate of oxygen, which correlates with hypoperfusion. As well, the increase in nonoxidative metabolism early after traumatic brain injury can have particular importance with regard to adequate perfusion in this acute period of injury. Later, arterial-venous differences decrease indicating a hyperemic or "luxury perfusion" state at least relative to oxidative metabolism. Sheinberg et al[58] used an online methodology to assess the frequency of venous desaturations in head-injured patients. Increased mortality was associated with increasing numbers of desaturations that often occurred during times of systemic deterioration requiring intervention. Since cerebral desaturations in the absence of systemic arterial desaturations were uncommon, Sheinberg et al were unable to make significant correlations with these events and outcome. In children, jugular venous information has recently been utilized in a few centers to assess the metabolic changes in children,[33,59] however, the usefulness of this modality and efficacy in severe traumatic brain injury remain to be investigated fully. Similar to the monitoring and management of intracranial pressure, intervention and prognostication based on jugular bulb measures can be of value when "significant" desaturations occur and treatment interventions are used to intercede. Further study is needed, particularly in children.

TREATMENT OF PEDIATRIC TRAUMATIC BRAIN INJURY

Appropriate and aggressive management of the patient with head injury in the acute setting has led to decreased mortality and morbidity and improved functional outcomes. In the past 20 years, the mortality from head injury in children has declined, due at least in part to this aggressive approach.[34,60] Because primary brain injury following trauma is not believed to be amenable to treatment, the goal of conventional therapeutic interventions has been to interrupt the normal secondary response and avoid and treat secondary insults. Mortality following the initial traumatic brain injury is believed, in most cases, to be a result of secondary insults to an already primarily injured brain. These include: (1) systemic deterioration or hypotension, or both, (2) prolonged hypoxemia, and (3) uncontrolled intracranial hypertension; all of which can be treatable events. Present day therapy includes maintaining adequate blood pressure (mean arterial pressure [MAP]) and oxygenation as well as maintaining cerebral perfusion pressure by aggressively treating raised intracranial pressure. Since after traumatic brain injury loss of the normal autoregulatory mechanisms is possible, therapeutic modalities must be tailored to provide the optimal oxygenation and perfusion to the injured brain in the acute period after injury to achieve maximal recovery of the primarily damaged, but potentially viable, tissue. The goals of acute head injury management are: (1) prevent secondary neuronal damage to an already compromised nervous system and (2) attempt to create an environment conducive to functional recovery.

INTRACRANIAL PRESSURE MANAGEMENT

Intracranial hypertension occurs in over half the patients with a severe traumatic brain injury with the mortality in these patients of over 50%.[61] Increased intracranial pressure occurs when there is brain swelling, bleeding, or cerebrospinal fluid accumulation within the confines of the cranium. Intracranial hypertension ultimately impedes cerebral perfusion and causes secondary ischemia that ultimately leads to further brain swelling or herniation, or both. The treatment of the child with head injury is based on the understanding of the balance of components within the intracranial space, namely, brain, blood, and cerebrospinal fluid. Current therapies for the treatment of traumatic brain injury target these components through the reduction of brain water and cerebral blood volume in the uninjured areas of the brain that have normal or impaired but intact autoregulation. The maintenance of the cerebral perfusion pressure is achieved by treating increases in intracranial pressure. Cerebral perfusion pressure (defined as mean arterial pressure minus the intracranial pressure) when "adequate" does not ensure adequate cerebral perfusion in focally injured regions of brain. Both intracranial pressure and cerebral perfusion pressure should not be exclusively used as measures of adequate treatment. Some regions of the brain may require a higher level of cerebral perfusion pressure to maintain adequate cerebral blood flow, and infarcted regions of brain may not be perfused at any cerebral perfusion pressure. Optimal perfusion must therefore be assessed and tailored to individual patients.

Intracranial Pressure Monitor/ Cerebrospinal Fluid Drainage

Conclusive data are lacking that the use of intracranial pressure monitoring, particularly in infants and young children, facilitates their aggressive management and improves outcome. Though not a standard of care in the management of severely head-injured infants and children, the use of intracranial pressure monitoring has increased and is used in most major head trauma centers to guide care. Intracranial pressure monitoring: (1) aids in the early detection of intracranial mass lesions, (2) can limit the indiscriminate use of therapies to control intracranial pressure which themselves can be potentially harmful, (3) can reduce intracranial pressure by cerebrospinal fluid drainage and thus improve cerebral perfusion pressure (provided a ventricular catheter is used), and (4) helps in determining prognosis.

The continuous monitoring of intracranial pressure is recommended for adult patients with Glasgow Coma Scores of 8 or less,[62] although the guidelines for its use in children

are still being developed (Thomas Luerrson, personal communication, 1997). Even though patients with mild or moderate head injuries are not routinely monitored, measurement of intracranial pressure may be particularly useful in these patients if they have traumatic mass lesions. Continuous intracranial pressure monitoring can be accomplished by available online systems using either an indwelling ventriculostomy (EVD) or a fiberoptic or strain gauge type intracranial pressure monitor (Camino or Codman catheters). The indwelling ventriculostomy remains the gold standard following trauma since it can be used to measure intracranial pressure and as a therapeutic modality to drain cerebrospinal fluid either continuously or intermittently during periods of increased intracranial pressure.

Head Position

Elevation of the head of the bed to 15° to 30° and maintaining the head in a neutral position have commonly been used to facilitate adequate cerebral venous drainage (and possibly cerebrospinal fluid drainage) by avoiding jugular venous compression. As a result, there is an optimization of cerebral venous pressure, cerebral blood volume, and a lower intracranial pressure. Feldman et al[63] showed in a randomized study of the effect of head position on intracranial pressure, cerebral perfusion pressure, and cerebral blood flow that though both intracranial pressure and mean arterial pressure decreased significantly by moving the head from 0° to 30°, there was no change in cerebral perfusion pressure or cerebral blood flow. When the head was elevated to 60°, there was a negative impact on cerebral blood flow. However, these studies have not been done in children.

Sedation

Sedation and paralysis are often used in the initial treatment of severely head-injured patients. They reduce the noxious stimulation of intubation, suctioning, patient agitation, and asynchronous ventilation that can increase intracranial pressure. Sedation and paralysis can be carefully titrated once intracranial pressure monitoring has been established. With short acting medications, intermittent examinations can still be obtained yet allow for patient transport or scanning in the acute setting. Narcotics and benzodiazepines along with a nondepolarizing muscle relaxant are generally recommended for intermittent or continuous use.

Osmotic and Loop Diuretics

Mannitol has been shown to attenuate intracranial hypertension by two separate mechanisms. Upon infusion, mannitol decreases blood viscosity, which can potentiate pressure or metabolic autoregulation of the intact brain and reduce intracranial pressure. Mannitol also acts by reducing the intravascular volume through increasing the serum osmolality thereby reducing brain edema. Mannitol also possibly has an effect on cerebral blood flow that contributes to its overall therapeutic effect. Mannitol can be given at 4 to 6 hour intervals or as necessary to maintain the optimal intracranial pressure, but its use is limited by the serum osmolality. Furosemide can be used acutely and in conjunction with mannitol to reduce the intravascular volume that initially follows the mannitol infusion. Bruce et al[64] dismissed the use of mannitol to treat intracranial pressure in children because of its tendency to increase cerebral blood flow in a number of patients with injury. Muizelaar et al[30] disagreed indicating that since mannitol potentiated autoregulatory mechanisms, its use was particularly useful in the acute setting.

Controlled Ventilation

The use of controlled ventilation has long been a modality for the treatment of intracranial hypertension and is based on the known cerebrovascular response to changes in P_aCO_2. With hyperventilation, cerebral vasoconstriction decreases cerebral blood volume at the arteriolar level. About a 3% change in cerebral blood flow is observed per Torr change in P_aCO_2 in adults.[57] Bruce et al[29,64] suggested that intracranial hypertension in children could be effectively treated almost exclusively with vigorous hyperventilation. Random and "blind" hyperventilation recently has come into question as a therapeutic intervention since it was found to worsen outcome in adults with severe traumatic brain injury.[34] Experimental models have demonstrated that the effect of hyperventilation on arteriolar diameter is short-lived with its therapeutic effect lasting only 20 hours. In contrast, the loss of cerebrospinal fluid bicarbonate buffer that occurs from sustained hyperventilation makes the cerebral circulation more sensitive to abrupt changes in P_aCO_2. This limited therapeutic effect in conjunction with a concern of decreasing perfusion in the early period after injury, has prompted the use of moderate hyperventilation (P_aCO_2 of 32–35 Torr) that can aid intracranial pressure management without inducing ischemia.[34] In addition, the deleterious effect of hyperventilation has been suggested in one report of 12 patients where hyperventilation was shown to induce ischemic levels of cerebral blood flow in acute brain lesions as well as affect both injured and apparently intact areas of the brain.[65] Most of these studies were done in adults and did not address age-related differences.[34,65]

Besides assessing and controlling P_aCO_2, the maintenance of good cerebral oxygenation is also necessary for the prevention of secondary injury. It has been shown that hypoxemia, defined as apnea or cyanosis in the field, or a P_aO_2 measurement of less than 60 mm Hg by arterial blood gas analysis, early after injury, contributes importantly to poor outcome.[9,66]

Mean Arterial Pressure

Early after traumatic brain injury, hypotension (systolic blood pressure < 90 mm Hg) is associated with increased mortality and morbidity.[67–69] Contemporary management of the infant or child with severe traumatic brain injury includes maintenance of an adequate perfusion that can usually be achieved with normotension or mild systemic hypertension. Although induced hypertension could eventually be used as an intervention to increase cerebral perfusion

pressure, it has not yet been proven efficacious in traumatic brain injury. Since cerebral injury can be complicated by either partial or complete loss of pressure autoregulation, significant systemic hypertension would contribute to further hyperemia and cerebral swelling. In some cases, however, a higher systemic pressure is necessary to maintain adequate perfusion to compromised brain regions. Careful monitoring of intracranial pressure and cerebral blood flow is necessary to determine the impact of blood pressure manipulation. Induced hypertension could have particular effectiveness in patients in whom pressure autoregulation of cerebral blood flow is intact. If the autoregulatory mechanisms for blood pressure are intact, and cerebral perfusion pressure is reduced with hypotension, cerebral vasodilation occurs to maintain cerebral blood flow, increasing cerebral blood volume and intracranial pressure.[70] In contrast, increasing cerebral perfusion pressure by increasing mean arterial pressure would then lead to cerebrovascular constriction, decreased cerebral blood volume and intracranial pressure, but preserved cerebral blood flow. This is somewhat of a simplification, but it is possible that mean arterial pressure could be optimized to maximize adequate cerebral blood flow and cerebral blood volume and reduce intracranial pressure.[71] Because of the uniqueness of children and age and size variances for the optimal cerebral perfusion pressure, the issue of optimal mean arterial pressure in children is even that much more complex. It is becoming clear that, in the future, management of the patient with traumatic brain injury will need to be tailored and titrated individually so as not to iatrogenically worsen an already compromised brain. It is clear that hypotension (systolic blood pressure $\leq$ 50 percentile for age, ranging from 65 to 70 mm Hg for infants and 85 mm Hg for adolescents) should be aggressively managed with fluids and, if necessary, using pressor support with dopamine, neosynephrine, or other agents as indicated to optimize and maintain adequate cerebral perfusion pressure.

Barbiturates

Barbiturates, particularly pentobarbital, have long been known to be neuroprotective by reducing cerebral metabolic demands.[72] By reducing the metabolic requirements of the brain, ischemic levels of cerebral blood flow can be tolerated for longer periods. In addition, by reducing cerebral metabolic demand, cerebral blood flow is decreased with subsequent decreases in cerebral blood volume and potentially intracranial pressure. This has remained a somewhat controversial therapy in head injury and has not been used extensively except as a last resort. Early use, however, could have beneficial effects prior to the actual development of malignant intracranial hypertension. The standard level of "barbiturate coma" is achieved when there is 10 to 20 seconds of burst suppression of cortical activity monitored at bedside by electroencephalography (EEG). This therapeutic goal is limited by systemic hypotension and as a result, the patient may require pressor and fluid support to attain the desired level of cerebral suppression. Recent data suggests that "coma" levels of barbiturates may not be necessary to achieve the therapeutic effect on decreased metabolism and intracranial pressure.[72] Because barbiturates cause myocardial depression, they require cautious use in patients with pre-existing hemodynamic instability. Therapy is often continued for 48 to 96 hours or longer depending on the cerebral hemodynamics.

Anticonvulsants

Anticonvulsants are often administered to patients with severe head injury to prevent early post-traumatic seizures particularly if there is a penetrating or parenchymal injury noted on the initial CT scan. The incidence of seizures within the first week after traumatic brain injury is 4% to 25% and the incidence of seizures occurring later is 9% to 42%.[73] The incidence of post-traumatic seizure in penetrating injuries is approximately 50%.[73] The concern in the acute period is that seizures can increase intracranial pressure through multiple mechanisms: by increasing metabolic demand, valsalva effects, release of excitotoxic neurotransmitters or other metabolites, and passively via systemic effects such as hypoxemia or hypertension. The prophylactic and aggressive treatment of seizures is therefore recommended. It also has generally been believed that the prevention of seizures in the early acute period decreases the chance of the development of chronic epilepsy.[74] Therapy often continues for 10 days to 2 weeks[75] and can be extended depending on the cerebral injury, the extent and frequency of post-traumatic seizures, or EEG findings.

Corticosteroids

There is extensive literature on the use of corticosteroids and head injury, but corticosteroids have not been shown to improve outcome in severely head-injured adults. No study specifically addresses the use of corticosteroids in children, but many of the studies included children and there did not seem to be any overall improvement in outcome in the pediatric population. At this time, corticosteroids have not been shown to be effective for the treatment of traumatic brain injury and their routine use in children is therefore not recommended.

Temperature Manipulation

The use of hypothermia to treat traumatic brain injury in humans was first reported during the 1950s.[76–78] Numerous reports have shown that outcome could be improved by cooling patients at various times up to 10 days after injury even with different levels of hypothermia.[77] Hendrick[76] reported on the specific benefit of hypothermia in children following traumatic brain injury and noted that there was improved outcome following treatment. Interestingly, because of the lack of scientific methodology in these studies, the widespread use of this modality has been limited.

Recently, multiple authors have reported on prospective randomized trials in adults with severe traumatic brain injury of therapeutic moderate hypothermia.[79–81] Shiozaki et al[81] used therapeutic moderate hypothermia (32–34°C) in a

group of severely head-injured patients who had intracranial hypertension refractory to barbiturate therapy, and found a statistically significant reduction of intracranial pressure and an improvement in cerebral perfusion pressure that was sustained even after rewarming. In addition, there was a trend toward improved clinical outcome at 3 months. Marion et al[80] reported on the use of therapeutic moderate hypothermia and showed a significant reduction in intracranial pressure as well as a trend toward improved outcome at both 3 and 6 months after injury. In another study, Clifton et al[79] again documented a trend toward improved outcome with moderate hypothermia. Clinical trials are underway in children to determine the safety and efficacy in this group.

OUTCOME: MORTALITY

The overall mortality in children is less than in adults.[2,82] There are multiple factors that contribute to overall mortality following traumatic brain injury, particularly in children. These include the mechanism of injury, the age of the child at time of injury, the severity of the injury, and the extent of the secondary injury.[2] Mortality, though, is most often associated with both age and severity of injury as measured by the initial Glasgow Coma Score.

Age

Annegers[3] reported that 10 per 100,000 children die each year from head injury. Kraus et al[6] evaluated the difference between the overall mortality in children and those with severe injury, and reported that the overall mortality rate was 5% in children following head injury, but 59% if they had suffered a severe traumatic brain injury. In his series, 17% of all head injuries were considered severe or moderately severe. He also commented that this did not include patients who died at the scene. Jennett et al[83] reported a mortality rate in the under 20-year-old age group of 29%. Gross et al[84] had a case fatality rate in children of 33% compared with 48% in patients who were older than 15 years. Walker et al[51] reported an overall 25% mortality rate in his series of pediatric traumatic brain injury. Mortality was twice as common in those patients who had multiple trauma versus those who had an isolated head injury (32% vs 17%). In the patients with an isolated head injury, intracranial hypertension, Glasgow Coma Score of 3 to 4, and apnea were cited as the major factors involved with mortality. In the patients who died with head injury in conjunction with multiple trauma, close to 90% had associated hypoxia, hypercarbia, or hypotension.[51] The development of diffuse brain swelling has a variable effect on outcome in the literature with mortality rates of 12% to 53% when it develops in children.[1,12,82,85] The mortality rate from epidural hematomas is lower (3–10%) compared to diffuse type injury.[2,19] Focal mass lesions overall have a variable effect on mortality and range from 6% to 33%.[85,86] Most agree that the majority of the mortality occurs during the first hospital week.[1,84,87,88]

Though children survive severe traumatic brain injury more often than adults, if one stratifies the pediatric population by age, there is a clear distinction in mortality comparing young, middle, and older children.[2] Younger children (less than 4 years of age) along with mid-to-late adolescents have a higher mortality rate compared to school-aged children.[2] The reason for this difference could be the differing mechanisms of injury (described earlier) and different resultant injuries that occur in the different age subgroups. Children in the younger age groups more likely suffer from diffuse injuries and from multiple insults. During the middle childhood school age years, there are more focal injuries and a lesser severity of injury compared to other age groups. Adolescents and older children in contrast tend to have high impact injuries secondary to motor vehicle accidents. The true mortality rate as a result of a particular mechanism of injury at each age range has not been accurately assessed.

Severity

In adults, the Glasgow Coma Score has historically been an indicator of severity of injury and prognosis of outcome.[89] For middle and older children, the Glasgow Coma Score assessment remains valid since the expected elicited responses are age appropriate. Although the Glasgow Coma Score has been modified for younger children to make it more age appropriate, its validity remains in question since the verbal and eye scores are not easily assessed in this age group. As a result, the motor scores upon presentation and following resuscitation alone have been considered more indicative of the severity of injury and prognostic of final functional recovery. Even though good outcomes and recovery are occasionally seen following initially depressed motor scores in young children, this occurs much more commonly than in adults. For this reason, accurate comparisons have not easily been made. In one series of children and adults with epidural hematomas and a low Glasgow Coma Score, poor outcome was seen in only 16% of children compared to 31% of adults.[19] Others have seen similar outcomes for all types of injuries,[2] and in a study by Walker et al,[51] a low Glasgow Coma Score was a major factor in determining mortality in children with isolated head injuries.

Other aspects of the initial examination and assessment that are also helpful for determining prognosis include pupillary and brainstem function. Dysfunction of the brain stem can be indicative of severe central injury and often results in a poor outcome. Patients with pupillary dysfunction or altered oculovestibular reflexes have been shown to have a worse prognosis both in the adult[90] and pediatric populations.[2,51]

OUTCOME: MORBIDITY

Children as a group more likely have a good functional recovery than adults,[84] but the final functional outcome of children at different ages depends not only on the recovery potential of the brain at the time of injury, but also the stage of neural development. In children, not only is the mechanism of injury and severity of injury important to outcome,

but so is the developmental maturity or critical period of development of the brain at the time of injury. Since many of the functions of the developing brain are specifically directed at acquiring new information and interpreting incoming stimuli,[91] disruption or damage to these centers during development has an impact on the child's ability to acquire new and higher functions by impeding the normal processing of new information. As a result, the injury could affect the ability to retain what has already been learned as well as the ability to process new information in the future.

Similar to the effect of traumatic brain injury on mortality, injury early in childhood (infancy) can produce greater morbidity than in older children.[92,93] Koskiniemi et al[94] evaluated preschool-aged children (7 years or less) after suffering a severe traumatic brain injury to determine their long-term outcome with regard to school and work performance. Although over half (59%) were able to return to school and over 75% were able to perform normally in school, only 21% of these children were eventually able to work full-time outside the home as adults, even though they had been able to attend and graduate from a normal school. None of the children who suffered their injuries before the age of 4 years was able to work independently outside a structured environment, whereas those older than 4 years were more likely (29%) to work outside the home. Some have reported that 2 years of age is a critical age[87,95] and others have used 4 years of age as the critical age for predicting long-term outcome.[85,94]

Severity

Multiple studies have shown that the extent and length of coma and post-traumatic amnesia as measures of severity directly correlated with cognitive and functional outcome.[92,95–97] Coma less than 24 hours was rarely associated with permanent neurologic or neuropsychological sequelae,[87,95] and recovery of motor and cognitive function was proportional to the duration of coma.[92] In this study, the average length of coma for children returning to baseline intelligence was 1.7 weeks, for low intelligence, 3 weeks, for moderately impaired, 8 weeks, and severely impaired, 11 weeks. Only 24% of the brain-injured children were able to return to normal school classes. In another study, 56% of patients who spent less than 1 week in coma were able to work, while only 13% of those patients who were unconscious longer than 1 week were able to work full time.[94] It is uncommon for children to remain in a vegetative state after traumatic brain injury (less than 10%), and of those children that are in a vegetative state or severely disabled, it is rare that they will recover to a "good" outcome. Pagni et al[98] showed that only 10% of children who had a mean coma duration of approximately 6 weeks had good neurologic function at follow-up.

Deficits in Recovery

Often following traumatic brain injury, and particularly severe injury, numerous aspects of neurologic and neuropsychological function can be disrupted. These include intellect (Wechsler Full Scale and Performance IQ), attention/concentration, memory (spatial and verbal), language (syntax, location, and linguistic ability), visuomotor skills, achievement and academic performance, and motor function.[91,99–103] Emotional behavioral changes and even exaggeration of pre-existing behavioral problems can also occur following severe traumatic brain injury in children, and be magnified in the post-traumatic recovery period.[104] Other types of morbidity include post-traumatic seizures, speech and gait abnormalities, hearing and visual changes, and cranial nerve dysfunction.[91,94,101] Only about 20% attain a good outcome after severe injury.[91] The majority have a dramatic recovery in cognitive function in the first 12 months following their head injury. For this reason, patients are often followed at 6 months and 1 year after injury to ensure that they have maintained their academic abilities and performance.[41,91]

Cognitive Function

Deficits in intellectual function following traumatic brain injury have been substantiated by a multitude of authors.[91,99–101] There are significant differences, however, between the different studies with regard to the particular aspects of IQ that are affected the most. Deficits in Wechsler Verbal IQ have been reported to be less than that observed in Full Scale and Performance IQ with Performance Scale IQ scores worse than Full Performance Scale IQ.[91,101,103] The Verbal IQ score returns to within normal ranges in some studies,[102] whereas others have reported persistent deficits in Verbal IQ.[101] Although most of these studies were performed in children with severe traumatic brain injury, Ewings-Cobb et al[91] observed that there was a 20% difference in IQ between severely injured children and those with mild head injuries at time of recovery. Suffice it to say that following severe head injury, intellectual impairment may be significant and persistent, and may affect many or all modalities of intelligence function.

Age has a specific prognostic impact on intellectual function after severe traumatic brain injury. The majority of young children will have major, severe disabilities in intellectual function.[91,94,95] In one study with long-term follow-up of preschool-aged children with severe traumatic brain injury, 30% had a below normal IQ when tested in adulthood. The mean IQ for these patients was only 85, indicating a low normal level.[94] Brink et al[92] reported that intellectual impairment was more pronounced in the younger age groups than older children. Only 14% of the children less than 8 years old achieved an IQ greater than 84, whereas over half (52%) of children older than 8 years at the time of injury had an IQ greater than 84. The severity of the injury was related not only to IQ impairment, but also to impairment in the acquisition and retention of visuospatial memory. Even with a normal IQ, impaired retrieval of information can occur in these children.[105] Older children and adolescents consistently exhibited a higher level of performance than did children under 8 years of age on verbal learning and delayed recall tasks.[99]

Most patients in the initial post-traumatic period, including all severities of injury, often have persistent problems

in memory attention and concentration, and speed of information processing.[92,101,102,106] Memory dysfunction is one of the most common cognitive deficits following severe traumatic brain injury in children.[99,107] Verbal memory and the storage and retrieval of words is often lost after severe injury, and deficits in verbal recognition[107] as well as nonverbal or spatial memory, in particular remembering shapes, can be persistent in the younger child.[99,107,108] Klonoff et al[101] noted that nonverbal memory was worse in children who were less than 9 years of age at the time of their traumatic brain injury, and often lasted up to 3 years following the injury. In patients older than 9 years of age, persistent problems of memory function were often observed for at least 1 year following their injury. Traumatic brain injury during the early years of childhood is believed to not only directly impact on the ability to process information, but negatively impact on adaptive functioning and contribute to persistent late cognitive deficits.[108] Following the initial acute stage, the mild to moderately injured patients recover quickly from their deficits. Those children with severe injury often continue to have problems maintaining concentration both in short- and long-term follow-up studies.[100,106] Many of these patients are eventually diagnosed with attention deficit disorders, and it is this group of children that has been treated chronically with medication trials to address persistent processing dysfunction.

Speech and Language

The most common speech disorders following severe traumatic brain injury include speech motor disturbance, impaired repetition, decreased length and syntax complexity of sentences, and articulation problems,[91,92] though dysphasia and mutism can also occur. Even in children with mild or moderate head injuries, naming problems, fluency difficulty, repetition problems, and difficulty with written language have been described.[91] Again, these deficits tend to resolve and are usually found only infrequently 6 months to a year following the injury. The greater deficits occur in the severely injured[109] and these impact on communication and language in about two thirds of the patients. In one study comparing degree of language dysfunction,[91] the greatest deficit was noted in the early age groups (< 31 months of age). The younger children were noted to have deficits in expressive and receptive language, whereas those patients who were older and up to mid-adolescence had difficulty with comprehension or written expression. Levin et al[99] noted decreased verbal fluency, particularly when they correlated these findings with imaging studies using magnetic resonance. Left and right frontal abnormalities or differences in white- and grey-matter lesions tended to decrease verbal fluency more so than dorsilateral surface abnormalities on the frontal lobe.

Motor Function

Emanuelson et al[110] reported that there can be a more complete recovery of intellectual function than motor function in some severely injured children, although others have reported a minimal permanent motor impairment.[95] Motor deficits progressively recover during the first 6 months to 1 year following trauma, but deficits in fine motor movements are the most often persistent sequelae, lasting up to 1 year after injury.[92,95,103] Motor deficits are also directly related to the severity of the injury and the residua of motor deficits can involve multiple areas including limbs, face, and motor speech[91] as well as motor slowing on speed performance testing.[102] The two most common motor sequelae following severe traumatic brain injury in one series were spasticity and ataxia.[92] Even in preschool children, in whom the maximum plasticity for recovery is expected, many had incapacitating neurologic deficits including hemiparesis or tetraparesis, ataxia, dyskinesias, and cranial nerve deficits in follow-up that may persist long-term into adulthood.[91,94,101] Less than 28% of severely injured patients had deficits that were mild and not incapacitating in these series. In contrast, Mahoney et al[95] reported that only 9% of the patients had permanent motor deficits, but these patients had a mean time in coma of only 15.5 days. Many patients became independent; however, only 10% had a normal neurologic examination.[92]

The differences in motor and intellectual recovery are most likely due to the anatomical differences of the tissue subsisting these functions. Since motor function is more topographic and subcortical and intellectual function is more associative, it is hypothesized that topographic organization is less flexible or "plastic" for functional or structural reorganization. Cognitive functions, in contrast, being associative between cortical areas, possibly have increased potential for recovery due to greater synaptic or connective plasticity.[91]

Behavioral Problems

Following severe traumatic brain injury, changes in temperament, increased irritability, temper outbursts and impulsivity, aggressive behavior, hyperactivity, and problems with interpersonal social adaption are common. In the mild to moderately injured child, these changes tend to resolve, but after severe traumatic brain injury, behavioral problems often are magnified and persistent. In the younger age groups, hyperactivity, tantrums, aggressive or destructive behavior, and impulsiveness tend to predominate. Poor judgment, euphoria, and depression are more commonly observed in older children.[110] In one study, 94% of children had personality and behavioral aberrations as a result of their head injury.[111] In other studies, over 50% of children with severe traumatic brain injury continue to have impulsivity, hyperactivity, and learning problems long term.[92,101,108]

SUMMARY

Head trauma is a leading cause of death and disability and can be particularly devastating for the family of the pediatric patient. Although the primary traumatic brain injury is not amenable to treatment, conventional management is focused on lessening the effects of the secondary physiologic events

and the secondary insults that occur after the injury. Much of the treatment modalities used for the child after severe traumatic brain injury have been extrapolated from the adult data, since there is little literature that primarily involves children. Children as a group have better outcome than adults, but many factors influence prognosis in the pediatric population. The mechanism of injury, injury severity, multiple trauma, secondary insults, or the extent of secondary injury can all impact on the final outcome and are directly and indirectly related. It is clear that many of the poor outcomes observed are best prevented by preventing either the initial impact or the secondary insults that typically occur following traumatic brain injury to minimize the severity of the injury. Interestingly, very young and preschool children have worse outcomes both in mortality and long-term disability than older children and adolescents. The deficits observed are persistent and severe in the long term even with aggressive management. Continued aggressive intervention to prevent secondary injury and in the future, mechanistically targetted therapeutic modalities in the acute setting, will hopefully improve the mortality rates and functional recovery of these children.

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