Head injuries are the most common cause of disability and death among trauma patients, particularly those under 50 years of age. Head injuries also impose a large social burden, accounting for over five million days of hospitalization and over 30 million days of work lost annually in the United States. Although much of the morbidity and mortality of head-injured patients is due to their initial insult, further preventable damage may occur during the resuscitative period. The following article has been prepared to help clinicians understand and manage patients with head trauma in an acute hospital setting. It reviews the pathophysiology, treatment, and prognosis of head injuries from a critical care rather than a neurosurgical point of view.

PATHOPHYSIOLOGY

Primary Brain Injury

Two kinds of damage result from head injury: primary damage attributable to the trauma itself, and secondary damage related to expanding mass lesions and brain swelling, displacement of brain tissue caused by increased intracranial pressure (ICP), and concomitant or late ischemic, hypoxic, metabolic, and infectious abnormalities. The distinction between primary and secondary damage is important in determining the effects of therapeutic intervention.

Despite its common occurrence, the exact mechanism of primary brain damage due to blunt head trauma is unknown. Ommaya and Gennarelli believe that the basic phenomenon is cerebral concussion, in which rotational shear forces following abrupt acceleration or deceleration disrupt axons and myelin sheaths. The shear forces are maximal at the brain surface, minimal at its center, and intensified where the brain is exposed to bony or dural protrusions such as those at the frontal and temporal tips. The forces therefore cause a centripetal pattern of nerve fiber injury that affects subcortical structures only after the cortex is involved.

In keeping with this model, patients often have suffered diffuse brain damage following blunt trauma even though they manifest autonomic dysfunction, impaired pupillary reflexes, and other apparent manifestations of injury to the brainstem. This is supported by several studies that document widespread degenerative changes in short term survivors of head trauma who did not develop intracranial hypertension. These studies also suggest that the extent of damage following head trauma is graded depending on the extent of the shear forces. Thus, the effects of cerebral concussion may range from temporary paralysis and retrograde amnesia to a persistent vegetative state or death.

Although the Ommaya and Gennarelli model of diffuse brain damage applies to most patients, focal injuries may occur alone or in concert with concussion. Such injuries include cerebral contusion, which is characterized by subpial and intracerebral extravasation of blood, and cerebral laceration, in which the pia mater is torn. Contusion and laceration may occur under areas of extreme impact and most often involve the frontal and temporal poles. However, they also may accompany penetrating injuries due to stab, puncture, or missile wounds that damage deeper brain structures.

Secondary Brain Injury

Severe head trauma usually produces a loss of autoregulation and an initial increase in cerebral blood flow (CBF). Although this increase may not be observed clinically, Brown and Brown demonstrated in monkeys that head blows or electroconvulsions cause a transient decrease in mean arterial pressure (MAP) and CBF that is followed within 30 seconds by an increase in MAP of 75 percent and in CBF of 250 percent above control levels. The increase in MAP is similar to that originally described by Cushing in patients with brainstem ischemia due to intracranial hypertension and transtentorial herniation. It presumably results from profound peripheral vasoconstriction that redistributes blood volume preferentially to the cerebral vessels. Cerebral blood volume can triple as a
result of these cardiovascular events and can occupy a substantial amount of the intracranial cavity. Mean arterial pressure and CBF return to control levels after five to ten minutes in experimental animals, and probably in humans, following primary brain injury. Autoregulation remains impaired, however, and a phase of intracranial arterial spasm supersedes the previous vasodilatory phase. Presumably as a result of the vasospasm, CBF is reduced to 15 to 20 percent below control levels in adults, unless severe intracranial hypertension is present. The fall in CBF generally is paralleled by a reduction in cerebral oxygen consumption (CMRO₂) but is not dependent upon it, in contrast to the normal relationship between oxygen uptake and supply. Adults who improve after head trauma usually manifest a gradual return of CBF and CMRO₂ to control levels. On the other hand, patients with fatal injuries develop a very low CBF and CMRO₂ preterminal. The initial increase in CBF resulting from head trauma probably accounts for the cerebral swelling frequently observed during this period. Another possible explanation for the swelling is vasogenic edema. Such edema requires an increase in permeability of the cerebral microvasculature that could be caused by the initial surge of blood to the brain. In support of this hypothesis, punctate superficial hemorrhage was observed by Brown and Brown in the brains of experimental animals following electroconvulsive shock and concussive trauma. Children and adolescents also may manifest cerebral swelling following head injury. This swelling also has been attributed to brain edema, but Bruce and associates have demonstrated on computed tomography (CT) that it most likely represents hyperemia. The increase in CBF results most often from trauma associated with acceleration and deceleration, but its occurrence after other insults suggests that it is a general response to central nervous system injury in young patients. The hyperemia also occurs in the absence of intracranial hypertension, although it may be more marked with an elevated ICP. Secondary brain injury may involve expanding mass lesions in addition to generalized swelling of the brain. Epidural hematomas most often arise from temporal skull fractures that are associated with lacerations of the posterior branch of the middle meningeal artery. By contrast, subdural bleeding usually emanates from small cerebral veins bridging the cortex and the superior sagittal sinus, especially in the frontal region. Intracerebral hematomas may result from penetrating injuries or other conditions in which great force is applied to a small area of the brain. Abscesses and empyema also may follow penetrating injuries or depressed skull fractures. As is the case with cerebral swelling, expanding mass lesions injure the brain largely by increasing ICP. Intracranial pressure is increased above the normal level of 10 mm Hg in more than 80 percent of patients with severe head injury shortly after hospital admission, and is almost invariably elevated in those with rapidly expanding mass lesions. Lundberg, who introduced the technique of continuous ICP monitoring in head injured patients, observed that ICP exceeded 115 mm Hg in some individuals. Pitts and Martin noted that the ICP generally reaches its zenith some two to three days after head trauma, a point at which cerebral swelling is most severe. Intracranial hypertension damages the brain by compressing, distorting, and displacing tissue. The latter effect is seen most dramatically during transtentorial herniation, when ICP rises on the cephalad side of the tentorial notch and forces brain structures caudad because pressure is lower on that side. During the descent, compression of the brainstem and the third nerve by the temporal lobe is responsible for a combination of ipsilateral oculomotor nerve paresis, contralateral hemiparesis progressing to decerebrate rigidity, and altered level of consciousness. Respiratory irregularities that culminate in apnea, and the hypertensive response result from increased pressure in the posterior fossa with medullary dysfunction. A less dramatic but equally important consequence of intracranial hypertension is a global reduction in CBF. Cerebral blood flow is directly proportional to the cerebral perfusion pressure (CPP) and inversely proportional to the cerebral vascular resistance (CVR). When autoregulation is impaired due to brain injury and CVR does not vary, CBF is dependent entirely upon CPP. The cerebral perfusion pressure is equal to MAP, the inflow pressure, minus ICP, which resembles pressure in the cerebral veins and therefore can be used as the effective outflow pressure. Kety and associates and Greenfield and Tindall have demonstrated that CBF decreases significantly when ICP exceeds 40 mm Hg under experimental circumstances; assuming a normal MAP of 90 mm Hg, this ICP rise would produce a CPP of 50 mm Hg. Recent evidence has correlated a poor outcome after head injury with a CPP of 60 mm Hg or less. Before leaving this subject, it is important to stress that a CPP of 50 to 60 mm Hg can be reached by decreasing MAP as readily as by increasing ICP. Hypotension generally is uncommon among patients with head injury unless there is failure of the medulla oblongata, but it may be seen with concurrent spinal cord trauma or other insults. In fact, hypotension due to multiple injuries was observed in 13 of 100 consecutive head-injured patients transferred to a major trauma center. Hypoxemia occurred in 30 percent of the patients and often complicated their resuscitation. The same was true of anemia, hypoglycemia, and...
infection.

**Ventilatory Regulation**

The effects of head trauma on ventilatory regulation have been evaluated by Plum and Posner, who described six respiratory patterns resulting from lesions in the central nervous system: 1) eupneic or normal respiration, which may occur even in the presence of small lesions; 2) Cheyne-Stokes or periodic respiration, which is associated with bilateral hemispheric disease; 3) central neurogenic hyperventilation, which may be due to lesions at the pontine level; 4) apneustic respiration, which also may reflect damage to the pons; 5) ataxic respiration related to medullary injury in the medulla; and 6) apnea caused by overwhelming damage to the medullary respiratory control center. However, North and Jennett were unable to predict such a precise lesion localization based on respiratory pattern. Therefore, it must be stressed that any of these six patterns may be observed immediately after head trauma or may develop later, especially if transtentorial or transtemporal herniation occurs.

**Pulmonary Gas Exchange**

Although many respiratory patterns are possible, hyperventilation or eupneic respiration with a normal arterial carbon dioxide tension (PaCO₂) usually are seen in patients who survive head injury. In fact, the most common arterial blood gas abnormality in these individuals is hypoxemia with a decreased arterial oxygen tension (PaO₂). Frequently, hypoxemia can be explained by bacterial pneumonia, gastric aspiration, lobar atelectasis, pulmonary edema, or other processes that alter the chest on roentgenographic examination. Yet the roentgenogram also may appear normal.

Moss and co-workers described hypoxemia (mean PaO₂ breathing room air = 57 mm Hg) in 11 spontaneously breathing patients with head injuries who had not received anesthetics and whose chest roentgenographic films appeared normal. These patients had a normal PaCO₂ level and a normal ventilatory responsiveness to breathing high CO₂ or low O₂ gas mixtures, indicating that their ventilatory regulation was unimpaired. However, the patient's venous admixture was significantly increased. Schumacker and colleagues subsequently demonstrated that the increased venous admixture of a similar group of head-injured patients was due to areas of low ventilation-perfusion matching and right-to-left shunt in the lungs.

Several explanations have been offered for the increased venous admixture of head-injured patients normal on chest roentgenographic examinations. One is that the patients have microatelectasis related to the reductions in their functional residual capacity (FRC). Although FRC was normal in the patients studied by Schumacker and associates, Cooper and Boswell recently described 24 persons who manifested an increase in venous admixture associated with a significant reduction in FRC following head trauma. In an editorial accompanying this article, deTroyer and Martin speculated that head injury might depress involuntary activity in the intercostals and other respiratory muscles and thereby reduce FRC by relaxation of the chest wall.

The third possible explanation for both the increased venous admixture and the decreased FRC of head-injured patients is that extravascular water accumulates in their lungs. The patients therefore might be thought to manifest clinical or subclinical neurogenic pulmonary edema (NPE). Frost and associates concluded that neurologic factors accounted for an increased venous admixture in over two-thirds of head-injured patients many of whom had normal chest roentgenographic films. Mackersie et al demonstrated increased extravascular lung water in nine of 18 patients with severe head trauma or spontaneous subarachnoid hemorrhage. None of the patients had evidence of pulmonary contusion, pneumonia, gastric aspiration, or cardiac failure. Although several of the patients did not exhibit intracranial hypertension, overall there was a significant correlation between increases in extravascular lung water and ICP.

Theodore and Robin have argued that NPE results from the hypertensive response that is provoked by intracranial hypertension and brainstem ischemia. This response produces a transient vasoconstriction that shifts blood from the systemic circulation into the pulmonary vascular bed. The simultaneous increase in pulmonary microvascular pressure causes water to leak from the blood vessels into the lung interstitium. The vessels then leak proteins as well as water because they have been injured in the hydrostatic phase.

Sarnoff and Sarnoff demonstrated that experimental cerebral lesions produced an elevation of systemic and pulmonary vascular pressures sufficient to cause both the hydrostatic and increased microvascular permeability phases of NPE. Brashear and Ross found that the hypertensive response to increases in ICP and the NPE that followed it could be blocked by therapy with alpha-adrenergic antagonists. Ducker and co-workers observed that cervical spinal cord section also prevented NPE in dogs and monkeys with intracerebral hypertension, as did Simon and associates in sheep with status epilepticus induced by bicuculline.

Although these investigations stress that the hypertensive response is required for NPE to develop, other studies suggest that it is not essential. For example, Bowers et al noted that increasing ICP to a level near MAP produced a marked increase in the flow of
protein-rich lymph in sheep without a marked increase in pulmonary microvascular pressure. VanDerZee and colleagues\textsuperscript{40} reported that similar increases in protein and water conductance could be prevented by alpha-adrenergic blocking agents. Millen and Glauser\textsuperscript{a} found that the increases in pulmonary microvascular permeability associated with intracranial hypertension in cats could occur without an elevation of systemic or pulmonary vascular pressure.

Whatever its underlying mechanism, NPE should be suspected in patients who manifest hypoxemia with or without chest roentgenographic abnormalities immediately following head injury. Neurogenic pulmonary edema also should be thought of several days after the initial insult if ICP becomes elevated. Nevertheless, NPE usually should be considered a diagnosis of exclusion, and other causes of hypoxemia such as drug-induced pulmonary edema, gastric aspiration, and overly vigorous fluid resuscitation should be ruled out after injury.

Respiratory Defense Mechanisms

Head trauma and the coma it causes may alter respiratory defense mechanisms in several ways. The best known alteration is depression of the gag reflex that ordinarily limits bacterial contamination of the lower airways. Profound coma also may depress the cough reflex and cause upper airway obstruction through relaxation of the tongue musculature. Impairment in mucociliary clearance has not been evaluated in head-injured patients but remains a theoretic possibility. So do abnormalities in surfactant metabolism in areas of microatelectasis and alteration in alveolar macrophage function in the presence of pulmonary edema fluid.\textsuperscript{42}

Presumably these changes in host defenses, coupled with iatrogenic insults such as endotracheal intubation and residence in the intensive care unit (ICU), are responsible for respiratory complications during the resuscitation phase. Bacterial pneumonia is the most common of these complications, occurring in over 50 percent of head-injured patients at San Francisco General Hospital who are intubated for longer than five days (LH Pitts, personal communication, 1985). Lobar atelectasis also may occur after injury, along with pulmonary edema caused by fluid overload, sepsis, and other conditions. If the chest roentgenographic film appears normal but hypoxemia exists, bronchospasm and pulmonary embolism should be considered.

Medical Management

Initial Evaluation

The initial evaluation of head-injured patients is essential in determining the extent of damage to the brain and other vital organs, directing treatment, and estimating prognosis. However, thorough history-taking and physical examination should not be performed until the cervical spine is immobilized, the airway is protected, bleeding is controlled, seizures are treated, arterial blood gases are drawn, a nasogastric tube and bladder catheter are passed, and ventilation is supported, if necessary. Conscious patients or onlookers may be questioned about the type and circumstances of injury, if and when the patients lost consciousness, and whether they have concurrent medical or surgical problems.

Bruce and co-workers\textsuperscript{5} stress that the examination of head-injured patients should focus on their level of consciousness, pupillary size and reaction to light, corneal reflexes, oculomotor function (usually determined by oculovestibular rather than oculocephalic testing due to the possibility of spinal cord damage), retinal vessels, gag reflex, cough reflex, rectal tone, and pattern of ventilation. In addition to noting these variables, it may be helpful to categorize patients by means of the Glasgow Coma Scale (GCS),\textsuperscript{54,44} which is based on their best level of eye opening and verbal and motor response (table 1).

Patients with signs of tentorial herniation may be candidates for exploratory burr holes at this stage in their evaluation. However, if time permits and proper facilities are available, diagnostic roentgenographic studies should be performed. These usually should include cervical spinal views and a chest roentgenographic examination. Plain skull roentgenograms once were taken routinely in trauma patients, but CT can visualize brain structures in addition to the skull.\textsuperscript{45,46} Angiography may be used if CT is not available, or if additional information is required.

Monitoring

Following initial evaluation or surgery, severely head injured patients customarily are monitored in a

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Table 1—Glasgow Coma Scale
recovery area or ICU. Such monitoring should include serial physical examinations and GCS evaluation. Furthermore, because responsiveness, pupillary reflexes, and other variables frequently are altered by drugs and other therapeutic interventions, other kinds of monitoring may be required. This requirement is met most commonly by continuous recording of CPP with an indwelling systemic arterial catheter to measure MAP (and facilitate arterial blood gas analysis) and an epidural, subdural, or intraventricular catheter to measure ICP.

Given the poor correlation between CPP, CBF, and CMRO₂ in many head-injured patients, clinicians have long sought a way of directly measuring the last two variables. Cerebral blood flow can be quantitated by observing the washout curves of radionuclides introduced into the brain via carotid artery injection or inhalation. However, these techniques are either invasive or cumbersome and cannot easily be repeated in serial fashion. Some attention therefore has focused on positive emission computed tomography (PET) and single-photon emission computed tomography (SPECT). As yet experimental, PET and SPECT are potentially portable and noninvasive. Although these techniques are complicated and expensive, they may be able to provide clinical information about both CBF and CMRO₂.

Another potential monitoring tool in head-injured patients is determination of multimodality-evoked potentials (MEPS). This technique involves the recording of brain activity following auditory, visual, or peripheral stimulation and can be performed easily at the bedside. Greenberg et al have demonstrated the utility of MEPS in determining the location and extent of traumatic central nervous system lesions. Multimodality evoked potentials also are useful in predicting outcome, as will be discussed.

Other ICU monitoring techniques may be applicable in certain head-injured patients. These include on-line measurement of end-tidal CO₂ tensions, which correlate well with PaCO₂ level unless large increases in physiologic dead space limit CO₂ excretion by the lungs. A second frequently used technique is estimation of right and left ventricular end-diastolic filling pressure (and, by inference, volume) with catheters in the superior vena cava or pulmonary artery. The latter are particularly helpful in patients who are receiving therapy with barbiturates, diuretics, and other agents that reduce effective intravascular volume and in patients with coexisting respiratory or cardiovascular disease.

Ensuring Homeostasis

The medical management of head injury is intended to ensure systemic and cerebral homeostasis. From a cardiovascular standpoint, this means that MAP level should be maintained in the normal range so that CPP does not fall near the critical level of 50 to 60 mm Hg or rise to such an extent that CBF level is greatly increased. Fluids or vasopressors may be used to support CPP if necessary. Alternatively, MAP level may be lowered by vasodilator therapy in the occasional hypertensive patient. Mean arterial pressure should be lowered with extreme caution, however, because an elevated MAP may reflect chronic hypertension or the hypertensive response to brainstem ischemia.

From a respiratory standpoint, airway protection is the first priority. Mechanical ventilation also should be instituted in hypercapnic patients because the elevated PaCO₂ may dilate cerebral vessels and increase CBF. Hypoxemia should be corrected by supplemental O₂ therapy with the addition of positive end-expiratory pressure (PEEP) if necessary to avoid O₂ toxicity, despite the fact that PEEP may compromise venous return. The ideal PaO₂ level is uncertain in head-injured patients, but generally it should exceed 100 mm Hg to avoid cerebral tissue hypoxia during hyperventilation therapy.

Measures to ensure cerebral homeostasis include the administration of metabolic substrates and the normalization or reduction of cerebral metabolic needs. All patients in coma should receive treatment with 100 mg of thiamine and 0.4 mg of naloxone as soon as possible; dextrose should be added if hypoglycemia is demonstrated. Adequate nutrition is essential thereafter; caloric intake should be greater than the basal needs of 25 cal/kg per day to offset the stress of injury. Cerebral metabolic needs can be normalized by the therapeutic or prophylactic administration of phenytoin to abolish seizures, the administration of antipyretics and appropriate antibiotic therapy to treat fever, and the administration of sedatives and muscle relaxants to reduce agitation.

Treating Intracranial Hypertension

Intracranial hypertension may occur if cerebral blood volume increases due to decreased venous drainage. Because this may be caused by kinking of the jugular veins and lowering the brain below the level of the heart, the patient's head and neck should be kept near the midline position and elevated 30 degrees. These simple measures often serve to blunt the diminution in venous drainage that is caused by coughing, straining, and other activities that increase intrathoracic pressure. If they do not, sedatives and muscle relaxants may be given to limit muscle activity.

Positive pressure ventilation with or without PEEP also may increase ICP level by decreasing cerebral venous return. Cotev et al and Burchiel and associates have demonstrated in dogs and humans respectively that PEEP is more likely to increase ICP level if
intracranial compliance is low or respiratory system compliance is high. This is because PEEP-induced increases in intrathoracic pressure are applied more profoundly to intrathoracic vessels in the latter circumstance. Because PEEP is indicated primarily to treat pulmonary edema, which decreases respiratory system compliance, it should not adversely affect ICP in most circumstances. Nevertheless, both MAP and ICP levels should be followed closely in head-injured patients receiving this therapy.

Hyperventilation may be expected to decrease CBF by causing cerebral vasoconstriction via an increase in the pH level of brain extracellular fluid. The reduction in CBF probably is maximal at PaCO₂ levels of 20 mm Hg because the vasoconstriction is offset by the vasodilatory effect of cerebral tissue hypoxia. Although Harper has shown that the cerebrovascular responsiveness to hyperventilation varies even in health, most head-injured patients manifest at least some reactivity to changes in PaCO₂. However, after eight hours or more of hyperventilation, the pH level of brain extracellular fluid is restored to normal by active transport processes. The PaCO₂ level, therefore, should be normalized as soon as possible providing ICP does not increase.

Hyperosmolar agents reduce ICP first by decreasing brain water through the creation of an osmolar gradient between the bloodstream and brain cells and second through a reduction in total body water. Most clinicians administer treatment with a 20 percent mannitol solution either in high intermittent doses (0.25 to 0.5 g/kg every one to two hours) or continuous infusion (0.05 to 0.1 g/kg). A serum osmolality of approximately 325 mOsm should not be exceeded because excessive hyperosmolarity may injure neurons. Mannitol depletes the body of sodium and potassium as well as water, and must be used cautiously. It also should be given cautiously to head-injured children because it may transiently increase intravascular volume, CBF, and ICP levels.

Diuretics are given to decrease ICP by reducing intravascular volume. Furosemide in doses of 40 to 80 mg most commonly is employed for this purpose; it has the additional advantage of reducing CSF production by unknown mechanisms. Although water intake usually is restricted in patients receiving diuretics, the efficacy of this approach has yet to be demonstrated. Indeed, Morse and associates recently reported that water restriction did not reduce brain water in rats subjected to triethyltin poisoning or anoxic ischemia.

Acetazolamide is a carbonic anhydrase inhibitor that limits ion exchange across the choroid plexus. This agent is used to reduce cerebrospinal fluid (CSF) production in head-injured patients, particularly those with communicating hydrocephalus. The usual therapeutic dose of acetazolamide is 250 mg, two to four times a day. Treatment with furosemide also may be used to reduce CSF production, as noted earlier. Although acetazolamide therapy occasionally may be helpful, CSF drainage via a ventriculostomy usually is a more effective method of reducing the contribution of CSF to intracranial hypertension.

Corticosteroids have been assumed to prevent vasogenic brain edema through their stabilizing effects on vascular membranes. This assumption is supported by studies such as that of Rorit and Hagan, who reported a reduction in the size of experimental lesions in animals pretreated with dexamethasone. Nevertheless, neither low (16 mg/d) or high (96 mg/d) dose dexamethasone therapy sufficiently affected ICP or outcome in severely head-injured patients studied by Cooper and colleagues. Corticosteroid therapy is not routinely used in patients with head trauma at San Francisco General Hospital.

Treatment with barbiturates may reduce CBF and ICP by decreasing CMRO₂ or by lowering the effective circulating intravascular volume through peripheral venodilation and a mild depression of the cardiac output. Marshall and co-workers reported that high-dose pentobarbital therapy (3 to 5 mg/kg) normalized ICP level and reduced daily mannitol requirements in a majority of severely head-injured patients, although subsequent controlled trials failed to demonstrate any benefit from barbiturate therapy. A recent study suggested that treatment with lidocaine was as effective as thiopental for rapid reduction of ICP level and caused less cardiovascular depression.

Other therapies to reduce ICP include hypothermia, which reduces CBF as barbiturates do but is logistically difficult to administer; and hyperbaric oxygenation, which causes cerebral vasoconstriction similar to that achieved by hyperventilation but is much more expensive and cumbersome. Dimethylsulfoxide (DMSO) treatment also reduces ICP level in some patients but has not been shown to improve outcome.

**Prognosis**

Two important questions relate to the issue of outcome from head trauma: first, how can outcome be predicted? and second, is it altered by medical intervention? Regarding the first question, Overgaard and associates have demonstrated that the initial clinical evaluation focusing on level of consciousness, motor behavior, and pupillary reflexes is most predictive of recovery from blunt head trauma. Jennett and Teasdale introduced the GCS, in part to facilitate prognostication on the basis of early physical examination, and their approach has been validated by subsequent investigators.

Other clinical aspects, such as decerebrate rigidity, are also important indicators of outcome. Age appears to be most important, however, in that children and
adolescents frequently recover from severe head trauma despite decerebrate rigidity. Indeed, the death rate from head trauma in an unselected pediatric population should be as low as 33 percent or less, according to Berger and associates. As Bruce et al suggest, the relative infrequency of mass lesions and the high incidence of reversible cerebral swelling in children and adolescents may play a role in their improved outcome when compared to adults following severe head injury.

Narayan and co-workers analyzed the predictive utility of clinical signs, CT, ICP data, and MEPs prospectively in 133 severely traumatized patients. The combination of age, GCS, pupillary response, presence of mass lesions, extracranial motility, and motor posturing predicted outcome with an 82 percent accuracy in these patients. GCS alone was accurate in 80 percent of predictions, reaffirming its useful ness. Computed tomography and ICP data were poor prognostic indicators, but added to the accuracy of the clinical evaluation. Data from MEPs had a 91 percent accuracy, stressing the value of this study. Supplementation of the clinical evaluation with MEPs yielded the optimal prognostic power.

That initial clinical evaluation alone is highly predictive of outcome following head trauma suggests that morbidity and mortality cannot be greatly influenced by therapy. This conclusion is supported by the uniform death rate of 50 percent among 700 severely head-injured patients, most of them adults, in three countries with different care systems and management details. In marked contrast, Becker and colleagues have claimed that aggressive head injury management has lowered mortality to 30 percent. Studies such as these have engendered attitudes ranging from therapeutic pessimism to enthusiastic optimism among various clinicians and have not settled the issue of whether or not therapy is beneficial.

An intermediate viewpoint is that clinical outcome depends on both the degree of initial damage and the recognition and management of treatable injuries. The value of ICP measurement and manipulation has been questioned but has not been subjected to a prospective, randomized trial. Nevertheless, they have been shown to be helpful in detecting deterioration in patients with mass lesions, and their use is supported by extensive clinical experience. Furthermore, early identification and intervention have greatly influenced the outcome from intracerebral and subdural hematoma. Future improvements in outcome after traumatic brain insults will require innovative new therapies. Until these treatments are available, the medical management of head injury should involve the skillful use of the techniques described in this article.

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