Intracranial Hypertension

MANAGEMENT OPTIONS

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Raised intracranial pressure (ICP) after severe brain trauma is a common cause of secondary cerebral injury. ICP frequently precedes brain death. Therefore, vigilance to its presence and monitoring its extent by clinical and invasive modalities are required for the logical treatment of this common clinical entity.

For all practical purposes, the intracranial space is an incompressible constant. This space is filled with brain tissue, which comprises 80% of the volume of the intracranial space; cerebral blood volume and cerebrospinal fluid (CSF) occupy 10% each. The CSF is produced by the choroid plexuses at the rate of 20 ml/hr and is absorbed by the arachnoid villi; only 150 ml of CSF is contained within the cranium at any time.

These percentages may change under pathologic circumstances or can be manipulated by therapeutic maneuvers. For example, the brain tissue may swell when ischemia or infection is present. CSF production is decreased when furosemide or acetazolamide is administered and when the patient has hypothermia or alkalosis. CSF absorption by the arachnoid villi can be depressed by hemorrhage or infection. Absorption increases in the presence of ICP. Cerebral blood volume can increase when vascular resistance is altered; for example, vasodilating drugs such as sodium nitroprusside and nitroglycerin may increase the cerebral blood volume, and hypoxia, hypercapnia, or hypertension may aggravate and contribute to the problem. On the other hand, treatment of hypertension and hypoxia and decreasing arterial CO_2 to subnormal levels by mechanical ventilation will increase the vascular resistance and decrease cerebral blood volume.

Cerebral blood flow is normally constant over a wide range of mean systemic arterial pressures. This autoregulatory mechanism maintains a constant flow of 50 to 150 mm Hg and contributes much to the maintenance of normal ICP. In addition, metabolic autoregulation occurs. For example, when seizure activity precipitates increased metabolic demand within the brain, vasodilation occurs with resulting increased cerebral blood flow.

In conclusion, although increasing volume within the cranium will increase the ICP, slow increases in volume such as tumorous growth will not increase the ICP to the same extent as rapid volume increases that occur during hemorrhage or brain swelling.

Cerebral perfusion pressure (CPP) is defined as the mean arterial pressure (MAP) minus the ICP. If the central venous pressure (CVP) is greater than the ICP, then the CPP = MAP − CVP. CPP is normal between 60 and 90 mm Hg.

Causes

Intracranial hypertension is defined as a pressure greater than 15 mm Hg. Many institutions do not initiate treatment unless the ICP is greater than 20 mm Hg. At the Shock Trauma Center of the Maryland Institute for Emergency Medical Services Systems (MIEMSS) we have found treatment worthwhile when the pressure is greater than 15 mm Hg (see box, page 390).

ICP may also rise in the presence of intracranial mass lesions (i.e., hematoma), cerebral edema, or elevated intrathoracic pressure. The latter is seen frequently in association with rigid posturing, seizures, positive pressure ventilation, positive end-expiratory pressure (PEEP), tension pneumothorax, tracheal suctioning, and abdominal distension. In addition, jugular vein compression can contribute to raised ICP.

One should have a high index of suspicion for the presence of raised ICP when confronted with certain clinical situations. These include the patient with head injury and altered level of consciousness or loss of consciousness, the patient who has had evacuation of an intracranial hematoma, the patient with anoxic encephalopathy, and the patient with hydrocephalus—in other words, any patient with brain injury of any degree.

Symptoms and Signs

The patient may complain of headache or nausea and may experience episodic blindness.

Vomiting is occasionally a sign that intracranial hypertension is present. The most important sign of raised ICP is deterioration of the level of consciousness. Cranial nerve paralysis may occur. Paralysis of the third nerve produces ipsilateral dilation of the pupil and sluggish reaction to light stimulus. Paralysis of the sixth cranial nerve causes dysconjugate gaze. Other signs include systemic arterial hypertension, bradycardia, and bra-

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dypnea. However, these are relatively late signs of raised ICP. More subtle signs such as agitation or any alteration in mental status may be early indications of intracranial injury or hypertension. Any focal abnormality of the neurologic examination is a reason for rapid investigation or intervention.

Diagnosis

Computed tomographic (CT) scan is the initial diagnosis modality of choice in the investigation of the brain-injured patient (Figs. 1 and 2). CT scanning usually elucidates the brain pathologic conditions necessary to make appropriate clinical decisions and often obviates invasive diagnostic procedures such as angiography. Effacement of the ventricles or sulci or midline shift indicates cerebral edema and highly suggests raised ICP (Fig. 3). Intraaxial or extraaxial hemorrhage also is a strong indication of intracranial hypertension. A normal CT scan, however, does not rule out the possibility of raised ICP. Kishore et al. indicated that 17% of patients with an initially normal CT scan went on to have elevated ICP.

Monitoring

In general, a Glasgow Coma Scale (GCS) score of 8 or less suggests that the patient is likely to have intracranial hypertension. At the Shock Trauma Center, our protocol demands that any patient with a GCS score of 8 or less must have a monitoring device inserted (box, page 391). Patients with scores between 9 and 12 may simply undergo close observation. However, if a patient with a score in that range must undergo general anesthesia, where close neurologic examination cannot be performed, then a monitoring device must be inserted. The subset of patients whose GCS score is 13 to 15 may be observed clinically. However, those whose CT scan of the brain shows a traumatic lesion must have a monitor inserted if they must undergo general anesthesia.

The use of the intracranial monitor will guide the physician in managing the patient in whom intracranial hypertension develops or who has decreased intracranial compliance. The patient with brain injury who requires positive pressure ventilation with high levels of PEEP, the patient who requires medications such as nitroglycerin or nitroprusside, and the patient who requires tracheal suctioning may all have an elevated ICP.

The neurosurgical department at the Shock Trauma Center uses three devices: the subarachnoid bolt, the intraventricular catheter, and the Camino catheter (Camino Laboratories, San Diego, Calif.). The subarachnoid bolt is easy to insert and has a low infection rate; however, CSF cannot be drained

Management of elevated ICP

ICP <15 mm Hg
- Observe patient.

ICP >15 mm Hg (Proceed until pressure is controlled to levels <15 mm Hg or, at a minimum, CPP >55 mm Hg.)

▪ Position	Straight, unobstructed neck Elevate head of bed
▪ Manage pain	Opiates (fentanyl, morphine)
▪ Control agitation	Minor tranquilizers (midazolam, lorazepam) Major tranquilizers (haloperidol) Short-acting barbiturates
▪ Minimize intrathoracic pressure	Treat cough (lidocaine) Adjust ventilator to reduce peak airway pressure Paralysis*
▪ Hyperventilation	Reduce $Paco_2$ to 30-32 mm Hg
▪ Diuresis	Increase serum osmolarity to 305-310 mOsm/kg (first 48-72 hr, use mannitol); (after 48-72 hr, use furosemide)
▪ Increase hyperventilation	$Paco_2$ 25 mm Hg
▪ Barbiturate sedation	
▪ Barbiturate coma or surgical resection	

Neurologic reexamination with or without CT scan at each step.
CPP, Cerebral perfusion pressure.
*Paralysis may follow hyperventilation if necessary.

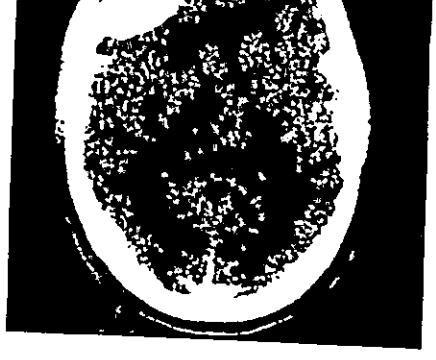

Fig. 1. *CT scan of intracranial injury. Axial CT section obtained in victim of closed-head injury demonstrates large epidural hematoma occupying right frontal area. A significant associated mass effect is seen with compression of frontal horns of ventricles. Convex margin toward brain and sharply defined edges is typical of epidural hematoma.*

as a therapeutic maneuver for elevated ICP. The intraventricular catheter provides accurate ICP measurements and allows incremental drainage of CSF, producing a dramatic fall in ICP. However because of the risk of ventriculitis, a sample of CSF must be sent daily for culture and sensitivity, Gram's stain, protein and sugar level, and cell count. The Camino catheter, which is placed directly into the frontal lobe, records the ICP directly from the brain tissue. This mode of pressure monitoring may be the most accurate of all. However, the catheters are expensive and easily dislodged and require highly trained personnel for proper insertion and maintenance. We usually do not insert a pressure monitor if coagulopathy is present because this maneuver may cause serious intracranial hemorrhage.

Control of ICP

Position

The head of the bed should be elevated to 45 to 90 degrees. This slanting increases venous drainage and thereby reduces cerebral venous pressure, which affects ICP. The neck should be maintained in a neutral position by the application of a Philadelphia collar. This device avoids torsion of the neck veins, which by itself can elevate the ICP. All constraining devices around the neck, such as tracheostomy tapes and dressings, should be loose. Sudden angulation of the neck (e.g., to insert an internal jugular vein line) can cause a sudden increase in the ICP. Despite its advantages, head elevation has some associated problems. If the patient has hypotension, for example, elevation can clearly impair cerebral perfusion.

Indications for ICP monitoring
Any patient with GCS score ≤8
Any patient undergoing general anesthesia* with
- GCS score 9-12, or
- GCS score 13-14, or loss of consciousness *and* an abnormal CT scan

Relative indications
- Obstructed fourth ventricle, or
- Diffuse edema, or
- Mass lesion

Any patient who will be unavailable for repeat neurologic examination.

Metabolism

Factors that increase the metabolic rate (e.g., seizures, sepsis, pyrexia) must be treated. Intracranial and extracranial sepsis may cause marked deterioration in neurologic function. The patient with pyrexia must have frequent blood, urine, and sputum cultures as well as a chest x-ray film to identify infection. Any patient who has had a penetrating skull injury, fractured base of the skull with CSF leak, intraventricular catheter insertion, or craniotomy is at risk for meningitis. If the patient cannot undergo lumbar puncture, a broad-spectrum antibiotic regimen may be necessary. At the Shock Trauma Center a combination of ticarcillin, amikacin, nafcillin, and chloramphenicol is usually administered to the patient with suspected central nervous system infection when CSF is unavailable for examination. The brain-injured patient commonly aspirates before tracheal intubation, and a bacterial respiratory infection may become superimposed within the first few days after injury.

Respiratory system

Hyperventilation causes a rapid fall in $Paco_2$ and decreases cerebral blood flow and cerebral blood volume; therefore, ICP is commonly reduced. CSF production is also decreased. The severely brain-injured patient is usually hyperventi-

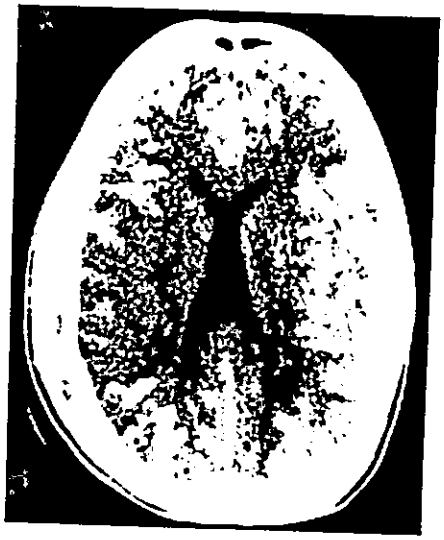

Fig. 2. *CT scan of intracranial injury. Axial CT section obtained in young man after high-speed decelerating injury in motor vehicle crash reveals numerous punctate hemorrhages predominantly at gray-white matter junction. This distribution is believed to represent "shearing" injury across gray-white interface caused by rotation acceleration-deceleration forces. Lateral ventricles are mildly compressed by diffuse cerebral edema.*

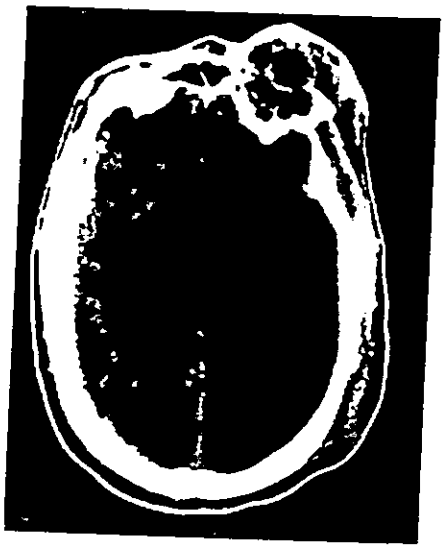

Fig. 3. *CT scan of severe cerebral edema. Axial CT image obtained after closed-head injury reveals thin subdural hematoma along right convexity (arrows). However, lateral ventricles are markedly compressed, cortical sulci are obliterated, and gray-white matter distinction is poor, indicating severe diffuse cerebral edema. Region of low attenuation in left temporal-occipital region suggests ischemia or infarction.*

lated to a $Paco_2$ between 30 and 35 mm Hg for 3 days. If the ICP increases when eucapnia is restored, then hyperventilation is reinstituted. Hyperventilation is of most benefit within the first 3 to 5 days after injury when the brain is prone to swelling; however, we have frequently seen the same effects of hyperventilation on ICP for many days beyond this period. Hyperventilation may be performed by increasing the ventilatory rate, tidal volume, or both, or in an acute situation by simply "bagging" the patient manually to achieve a dramatic fall in $Paco_2$ and ICP. Complications with this modality include respiratory alkalosis and increased cerebral venous pressure, especially when the lung is compliant.

Ventilator management requires attention to detail. Endotracheal intubation must be performed expeditiously, and coughing and straining by the patient must be avoided, because they can cause elevated ICP. Thiopental (when the systemic arterial pressure is normal or elevated) and parenteral lidocaine can be helpful. We prefer to place the ventilator in the intermittent mandatory ventilation (IMV) mode initially. However, large tidal volumes and high levels of PEEP can elevate the ICP and lower the mean arterial pressure, thereby reducing the cerebral perfusion pressure. If high airway pressures are encountered, as in patients with adult respiratory distress syndrome (ARDS), we will frequently switch to the pressure control mode. Patients who are "fighting" the ventilator should be controlled by sedation or neuromuscular blockade, in which case controlled ventilation is required. Endotracheal tube suctioning should be preceded by the administration of thiopental (Pentothal) or lidocaine (topical or parenteral) or performed when the ICP is under optimum control, for example, when the patient has recently been given mannitol.

Concern has often been raised about the use of PEEP in the brain-injured patient. These patients have commonly aspirated and are at risk of having an increased pulmonary shunt. We routinely apply 5 cm H_2O of PEEP. When the ratio of Pao_2 to fraction of inspired oxygen (Fio_2) is less than 300, we increase the PEEP as necessary. If PEEP is not increased early at this point, much higher levels may be subsequently necessary to prevent arterial desaturation. The ICP should be assessed after each change in PEEP to monitor the effect of the change.

Fluid therapy

Fluid therapy must be judicious. In general, a serum osmolality of 295 to 305 mOsm/kg should be achieved; thus only isotonic fluids are given. Although decreasing cerebral interstitial water is the ultimate goal, this result should not be achieved by inducing hypovolemia. Excessive fluid restriction may lead to tissue hypoperfusion caused by inadequate stroke volume, a situation that may jeopardize neuronal viability. Inserting a pulmonary artery catheter is extremely useful, allowing the physician to optimize cardiac filling pressures, cardiac output, and oxygen consumption. If the ICP is very difficult to control, we commonly increase the serum osmolality to 310 mOsm/kg.

Diuretics

Diuretics are commonly required for the medical treatment of elevated ICP. Furosemide and mannitol are the drugs we most frequently use. A loop diuretic, furosemide acts by decreasing CSF production from the choroid plexuses and decreasing intravascular volume and free water, which increase a favorable gradient for flow from the interstitial space to the intravascular space to decrease edema. The use of furosemide can be complicated by hypovolemia, which may cause cerebral hypoperfusion. This drug is also potentially nephrotoxic and ototoxic. We usually administer a dose of 10 to 20 mg intravenously; this dose may be repeated frequently as long as no significant hypovolemia or severe serum hyperosmolarity develops.

Mannitol is an effective osmotic diuretic that yields a rapid decrease in brain size. One can expect to see results, that is, a fall in ICP, within 15 minutes. Its effect, however, is short-lived; it must therefore be administered every 6 hours in a dose of 0.25 to 0.5 gm/kg as necessary to optimize intracranial compliance. When mannitol is discontinued, rebound increase in ICP may occur. A breakdown in the blood-brain barrier may occur 24 to 72 hours after injury, which may cause leakage of mannitol into the interstitial space and increase edema. Other adverse effects include immediate hypervolemia, eventual hypovolemia, severe hyperosmolarity, and electrolyte abnormalities. Hemodynamic monitoring with a pulmonary artery catheter becomes essential when using mannitol, inasmuch as hypovolemia may occur and urinary flow is no longer an index of tissue perfusion because of the obligatory osmotic diuresis.

Sedation

Anxiety has a considerable effect on elevated ICP and must be controlled. However, control is often achieved at the expense of decreasing the level of consciousness. We prefer to use fentanyl, lorazepam, or haloperidol or a combination thereof. Fentanyl is a short-acting synthetic narcotic; its action can be reversed by the administration of naloxone. Lorazepam is a short-acting benzodiazepine. Haloperidol is a neuroleptic that we have also found very useful. Additional information on these drugs can be found in the article by Omert (page 365).

Lidocaine

Lidocaine may be effective in ICP reduction and may be useful when the patient is hypovolemic or when cardiac depression may make the

use of barbiturates hazardous. Lidocaine's mechanism of action in treating raised ICP is uncertain, but it may act by decreasing cerebral blood flow and cerebral metabolic rate. The drug can be given before tracheal suctioning through the endotracheal tube or the intravenous route. Caution must be used, because overdosage may lead to seizure activity.

Barbiturates

The use of barbiturates is controversial. We have commonly found them to be useful in a variety of ways with gratifying results in the treatment of elevated ICP. Barbiturates decrease cerebral blood flow and cerebral metabolic rate and, of course, can be used to treat seizure activity.

We use bolus doses of thiopental sodium (100 to 150 mg) to decrease acute elevations of ICP. This drug is particularly useful if systemic arterial blood pressure is also elevated. However, the action is short-lived.

When ICP is uncontrolled by the previously mentioned modalities and particularly when >25 mm Hg, we place the patient in barbiturate coma. We believe that this modality gives a cerebral protective effect against shock, hypoxia, and ischemia, and we have had some excellent results with its use.

Barbiturate coma is initiated by giving a loading dose of pentobarbital, 10 mg/kg/hr, for 4 hours followed by a maintenance dose of 1.6 mg/kg/hr. If the ICP does not clearly fall during the first day of administration, then the infusion is stopped, inasmuch as the patient is unlikely to benefit. If a response does occur, then barbiturates are continued until the ICP is less than 15 mm Hg for 24 hours. At this point the patient is weaned from the infusion and the ICP is observed carefully. If the ICP rises once more, then the barbiturate coma is reinstituted. We do not think that continuing barbiturates for longer than 10 days is of value.

Continuous barbiturate infusion has serious side effects. Cardiac depression is to be expected and hypotension can occur very rapidly after the commencement of infusion. Therefore, a pulmonary artery catheter is essential and cardiovascular dynamics are assessed before administering the pentobarbital and at intervals thereafter. Starting an inotrope such as dobutamine before the infusion is usually beneficial. Our positive experience with barbiturate coma is likely a result of selecting patients with good pre-coma left ventricular function, that is, relatively young patients. Other side effects of barbiturates include the inability to follow the neurologic examination and the possibility of hypothermia and paralytic ileus. We monitor the barbiturate level daily, usually aiming for a range of 25 to 40 mg/L; however, we will accept a level of 60 mg/L, if necessary to control the ICP, before tapering the dose. We have not found electroencephalography of value in monitoring barbiturate coma.

The following case example illustrates our experience with barbiturate coma in a critically ill patient.

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A 48-year-old man was admitted to the Shock Trauma Center after a motor vehicle accident. He had a GCS score of 8. Injuries included multiple facial fractures, a fractured femur, and a fractured wrist. CT scan of the head revealed hemorrhagic contusions of the right temporal and left frontal regions of the brain with diffuse cerebral edema. A Richmond bolt was inserted. The initial ICP was 24 mm Hg. All surgery was deferred, and the patient received therapy for raised ICP, which included elevation of the head of the bed, fentanyl infusion, administration of mannitol and thiopental sodium, and neuromuscular blockade. Despite these measures, the ICP rose to 28 mm Hg on the third hospital day.

We decided to place the patient in barbiturate coma; a continuous pentobarbital infusion was begun. Dobutamine was added to improve cardiac contractility. On the fourth hospital day the ICP was under adequate control and the pentobarbital infusion was discontinued. However, the ICP again increased within a few hours to 20 mm Hg. The pentobarbital infusion was reinstituted with immediate resolution of intracranial hypertension. Barbiturate coma was continued until the sixteenth hospital day, when it was discontinued without recurrence of raised ICP. His neurologic status improved, and he underwent surgery for his orthopedic and facial bone injuries. His was discharged on the fifty-seventh hospital day to a rehabilitation center. He was following commands, moving all extremities purposefully, and bearing weight on his previously fractured femur.

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Surgical therapy

During the ongoing medical management of intracranial hypertension in the brain-injured patient, one must constantly be aware of and have a high index of suspicion for the possibility of a mass lesion. This problem can occur at any time after the patient's hospital admission, particularly after craniotomy for hematoma evacuation. CT scan is the modality of choice to determine the presence of a mass lesion (Fig. 4). We perform a brain CT scan on admission when indicated (admission GCS score less than 15 or when consciousness has been lost). If the CT result is abnormal, it is repeated in 24 hours. If still abnormal, the CT scan is repeated in 72 hours. If any significant neurologic deterioration occurs, the scan is repeated at any stage of hospitalization. The discovery of a space-occupying lesion warrants immediate neurosurgical consultation, and if significant, immediate craniotomy and hematoma evacuation.

Cardiovascular system

Systemic arterial hypertension and hypotension and cerebral vaso-

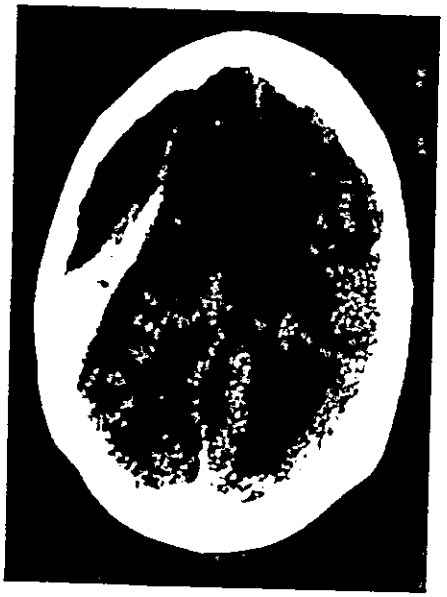

Fig. 4. *CT scan of intracranial injury. Axial CT section obtained in elderly patient after fall reveals mixed-density subdural hematoma over right convexity. Higher density component represents clotted blood. Note severe mass effect producing transfalcine herniation and entrapment of contralateral ventricle. A component of subdural collection extends into posterior interhemispheric fissure* (arrow).

dilation must be avoided. Hypertension may cause hyperemia in the injured brain (where autoregulation is altered) and aggravate edema. Hypotension will lead to low cerebral perfusion pressure, and vasodilation will increase flow and cerebral blood volume. To aid in treating these situations, which are often complex, inserting a pulmonary artery catheter and an arterial line is essential when systemic pressures are increased or decreased or routinely when brain injury is severe (GCS score of 8 or less).

Cardiac output is often depressed as a result of decreased preload or decreased contractility caused by drugs such as barbiturates. In this situation, inotropes such as dobutamine may be used to augment cardiac contractility and cardiac output. Although these drugs may be essential, one must use them with caution because they may cause vasodilation, increased cerebral blood flow, and increased ICP. We generally prefer dobutamine over dopamine because it is usually a better inotrope and has fewer cerebral vascular dilatory properties.

Systemic hypertension must be controlled, and the choice of medication may be crucial. Sodium nitroprusside and nitroglycerin are commonly used antihypertensives in the critical care setting. Both have the advantage of being easily "turned off," because they have a short half-life. Both are potent vasodilators and may elevate the ICP. Hydralazine is another direct vasodilator that may also raise ICP and has the added disadvantage of a long half-life. Labetalol, which has both alpha and beta blockade capabilities, reportedly does not raise the ICP and may be the best choice at present for treating systemic hypertension in the patient with impaired intracranial compliance. Trimethaphan camsylate is an alternative drug that has less potential to increase ICP; however, we have not used this compound in the brain-injured patient (see Simon and Reynolds, page 415).

Coagulopathy

The admission prothrombin time and partial thromboplastin time must be checked carefully, because some patients are taking drugs, such as warfarin (Coumadin), that may have anticoagulant properties. Severely brain-injured patients frequently have coagulopathy resulting from their injury. Elderly patients commonly take acetylsalicylic acid compounds, which may induce thrombasthenia. Platelet administration should be considered for these patients with hemorrhagic brain injury. All patients who have suffered brain injury or who have had craniectomy for evacuation of hematoma should have serial prothrombin time, partial thromboplastin time, and platelet count determinations. Significant deviations from normal should be treated aggressively with fresh frozen plasma, vitamin K, and platelet transfusion.

Nutrition support

We believe that early use of nutritional support is warranted in the brain-injured patient. However, judicious fluid administration is a priority until the initial insult has been overcome and ICP is well controlled—usually after the first 5 days after injury.

Conclusion

Aggressive treatment of ICP greater than 15 mm Hg is important for the successful outcome of the patient with severe brain injury. Attention to patient position and careful management of fluid therapy and the cardiovascular and respiratory systems are extremely important. A thorough knowledge of diuretics, inotropes, antihypertensives, and sedatives is required. Aggressive use of CT scans is necessary to detect intracranial mass lesions promptly. Barbiturate coma is very useful in selected patients with intracranial hypertension.

Suggested Reading

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