Increased pressure within the cranial cavity is a common and important problem in the care of patients with serious injuries and diseases of the central nervous system. Elevated intracranial pressure (ICP) is most commonly associated with severe head injury; some degree of increased ICP was found in 80 percent of all patients with a major head injury and in over one half of these patients the pressure was significantly elevated.\(^1\) Increased ICP accounts for 50 percent of all head injury deaths.\(^2\) While the majority of cases of increased ICP are due to head injury, elevated pressure is seen as a secondary effect of a variety of other conditions including brain tumors, subarachnoid hemorrhage, spontaneous intracerebral hemorrhage, and Reye's syndrome. A number of toxic and viral encephalopathies are also accompanied by intracranial hypertension.

The mortality rate in head injury is roughly proportional to the ICP. Normal ICP is 0 to 10 mm Hg. If ICP rises to 40 mm Hg, the mortality rate is 65 percent; when ICP reaches 60 mm Hg, the mortality rate is 100 percent. Approximately 15 percent of severe head injury patients develop elevated pressure which cannot be controlled; all of these patients eventually succumb.\(^3\) The relationship of ICP and morbidity in head injuries is not as clear. Patients with moderately elevated (25 to 40 mm Hg) ICP which is successfully lowered are often left with severe neurologic residuals. The ability to normalize ICP is not a guarantee of a good clinical result.

The direct measurement of ICP is a relatively new technique. Until Lundberg\(^4\) demonstrated the feasibility and clinical usefulness of direct long-term measurement in 1960, the measurement of ICP was obtained through lumbar puncture. Spinal subarachnoid space pressure thus obtained did not necessarily represent the pressure in the cranial cavity. The technique was dangerous in the presence of focal intracranial masses and was not applicable to prolonged monitoring. Lundberg's monitoring technique used a catheter placed in the lateral ventricle, a common neurosurgical procedure. The catheter was connected by a fluid-filled tube to a pressure transducer and a chart recorder. Reliable ICP measurement was now possible for days at a time.

Techniques of ICP monitoring and control were first applied to head injuries, and the great majority of clinical information has been developed from this source. A number of reports have demonstrated a significant decrease in head injury deaths, from approximately 52 percent to nearly 40 percent, when aggressive monitoring and control of ICP has been applied.\(^1,4-7\) Difficulties with patient classification and differences in treatment protocol make scrupulous comparison between series difficult. The incidence and severity of ICP elevation is greater if an intracranial focal mass is present. The severity of the immediate parenchymal injury is also a powerful force determining outcome, irrespective of the level of ICP elevation. Despite these problems, it is generally agreed that monitoring and control of intracranial pressure favorably affects the outcome of severe head injuries.

Similar aggressive treatment has become widely used in Reye's syndrome. Here its value can be better appreciated since there is no appreciable underlying brain damage to influence results. The mortality of severe (grade 4) cases has been decreased from 80 percent to near 10 percent by stringent control of ICP.\(^8,9\) It is clear that intracranial hypertension is a secondary effect of a number of pathologic processes and not a disease of itself. The most careful control of ICP will not reverse the brain damage caused by the primary disease or injury. The goal of ICP control is to permit the recovery of damaged tissue and to prevent secondary injury caused by intracranial hypertension.

**PATHOPHYSIOLOGY OF INCREASED ICP**

The deleterious effects of raised intracranial pressure are a result, not of the raised pressure itself, but of its effects on cerebral blood flow. The ultimate effect of increased ICP is cerebral ischemia, either focal or...
global, resulting from generalized increased pressure or from focal brain compression. Increased pressure in the cranial cavity is caused by the expansion of an abnormal mass in the intracranial space. This mass may be an increase in one of the normal components of the intracranial space, as in hydrocephalus, or the expansion of a new mass, eg, hematoma or tumor. The normal intracranial components are the brain (80 percent of volume), cerebral spinal fluid (10 percent), and blood in the cerebral vessels and dural venous sinuses (10 percent). The relationship of these three compartments is described in the Monro-Kellie doctrine developed in the 18th and 19th centuries and modified by Cushing in the 20th.\(^\text{10}\) The doctrine as understood today defines the volume of the intracranial cavity as nearly constant and the intracranial contents as incompressible. Thus, any increase in the volume of one of the compartments must be offset by a decrease in the volume of the others. This relationship is best defined in terms of the pressure-volume diagram (Fig 1).\(^\text{11,12}\)

When a small volume is added to the intracranial space by subarachnoid infusion of fluid or the inflation of a subdural balloon, there is relatively little increase in ICP. This initial phase of compensation, the horizontal portion of the curve, is accounted for by the displacement from the cranial cavity of a volume of cerebrospinal fluid (CSF) equal to the volume introduced. Although the blood volume may be decreased by narrowing of the venous caliber, most of the compensation appears to be due to migration of cranial CSF into the relatively distensible spinal subarachnoid space via the subarachnoid cisterns.\(^\text{12}\) As the added volume is increased, the compensatory mechanisms are exhausted, and a further increase results in a rapid rise in ICP; each increment in volume causing successively greater increases in pressure. The vertical portion of the curve—the phase of decompensation—represents the relatively incompressible brain tissue. The pressure developed by additional volume is a measure of stiffness, or elastance, of the brain. The elastance can be measured by injecting or removing 1 ml of saline solution from the ventricular system and noting the change in ICP.\(^\text{13}\) The elastance depends on the physical state of the brain whose stiffness is increased by arterial hypertension and decreased by osmotic diuretics.\(^\text{14,15}\) These differences will alter the shape of the pressure-volume curve (Fig 2).

The importance of a generalized increase in ICP rests on its effect on cerebral blood flow (CBF) throughout the brain. The total cerebral blood flow is approximately 50 ml/100 g minute, but flow is not uniform throughout the brain, being four times greater in gray matter than white. In spite of these regional variances, CBF is maintained within narrow limits in

\[\text{Cerebral Protection (John E. McGillicuddy)}\]
The arterial mechanism of autoregulation. If arterial pressure falls, arterioles dilate to lower resistance and preserve flow. When arterial pressure rises, the arterioles constrict. The CBF is maintained stable in a range of mean arterial pressures from 50 mm Hg to 160 mm Hg; if the pressure falls below 50 mm Hg, CBF passively follows the decrease in arterial pressure (Fig 3).

The true driving force of CBF is the perfusion pressure, or the difference between mean arterial pressure and venous pressure, which in the intracranial space is essentially the same as ICP. When ICP is low, perfusion pressure may be considered the same as mean arterial pressure. When ICP increased, perfusion pressure falls. Autoregulation maintains a stable CBF over a wide range of perfusion pressures down to 50 mm Hg (Fig 4). If ICP rises high enough to lower perfusion pressures below 50 mm Hg, CBF decreases passively with perfusion pressure and thus marked increases in ICP can produce cerebral ischemia.

Of even greater importance is the loss or impairment of autoregulation which is associated with many of the causes of increased ICP such as trauma and subarachnoid hemorrhage. In areas of disturbed autoregulation, CBF tends to change proportionally with perfusion pressure (Fig 4). Here, a modest decrease in perfusion pressure, insufficient to cause a decrease in CBF in a normally reacting area, may cause a significant fall in flow. The combination of defective autoregulation and increased ICP predisposes to cerebral ischemia. In severe head injuries, where both factors are often present, more than 90 percent of patients dying of head injuries show areas of ischemic brain damage.

The arterial carbon dioxide level has an important influence on both blood flow and ICP. Cerebral blood flow is very sensitive to the level of PCO2, increasing with hypercarbia and decreasing with hypocarbia. Cerebral blood flow changes by 2 percent to 3 percent for each change of 1 mm Hg in Pco2 from the normal level of 40 mm Hg. These alterations in CBF are caused by appropriate changes in arteriolar diameter, hence the level of Pco2 directly affects cerebral blood volume. An increase in blood volume has little effect on ICP if the pressure is low; if ICP is already high from some other cause, the increase in volume due to hypercarbia can bring about a major rise in pressure. Conversely, hypocarbia constricts arterioles, decreases cerebral blood volume and can reduce an elevated ICP.

Intracranial pressure can also produce cerebral ischemia via the complex effects of the growth of a localized mass. As the mass expands, local pressure on the brain causes a decrease in CBF in the area, especially if autoregulation has been impaired. Since the brain is relatively rigid and can be only slightly compressed, continued growth of a mass forces the brain to shift within the intracranial cavity which is partially divided by the planes of the dural folds, the falx and the tentorium (Fig 5). Brain tissue can be forced into the openings surrounded by these folds, principally into the tentorial hiatus, obstructing the openings and thereby dividing the intracranial space into separate compartments. This herniation of brain tissue into the openings has two effects—obstruction of blood flow to the herniated brain tissue and obliteration of the subarachnoid fluid spaces within the openings. These spaces normally are the conduits for the
redistribution of CSF that maintains an equal pressure throughout the intracranial space. Hence, their obstruction allows the development of pressure differences between the compartment containing the mass and the remaining compartments of the intracranial space. A pressure difference across the tentorium is frequently seen and a disparity between pressures over the two cerebral hemispheres is also not rare.

Protrusion of the medial portion of the temporal lobe into the tentorial opening compresses the oculomotor nerve and the midbrain, causing the syndrome of transtentorial herniation (ie, an ipsilateral fixed dilated pupil and a contralateral hemiparesis) and eventually brain stem failure and death. The syndrome of transtentorial herniation is widely accepted as a sign of increased ICP; it is more correctly a sign of brain shift.

The relationships between ICP and CBF and the effects of brain shifts, loss of autoregulation, and variations in brain compliance are quite complex and have only been outlined above. Two recent reviews are recommended for a more detailed discussion.

**MONITORING OF ICP: RATIONALE AND METHODS**

Unfortunately, there are no reliable clinical signs of rising ICP, and therein lies the principal reason for direct ICP monitoring. Lundberg's early work showed that ICP could increase to very high levels before the appearance of any change in neurologic or vital signs. Pressures of 70 mm Hg occurred without even evidence of headache. Comatose patients may show few warning signs of increasing pressure and then deteriorate profoundly and suddenly. Hence, monitoring of ICP in these patients provides an early warning of impending neurologic catastrophe. Studies in head injury patients clearly show that the higher the ICP rises, the more difficult it becomes to lower it again. If therapy is to be successful, it is critical to identify increasing ICP early, and this is only possible if the pressure is being monitored.

The continuous recording of ICP also allows the clinician to follow the effects of therapy. The response to the chosen regimen can be clearly seen, and further treatment decisions can be made on the basis of this response.

Controlled hyperventilation is one of the mainstays of the treatment of increased ICP, and its use often requires neuromuscular paralysis. Barbiturate coma is also coming into increasing use for the same purpose. In both of these states, neurologic evaluation of the patient is fruitless. If these techniques are used in treatment, ICP monitoring is essential as it is the only means of following the state of the intracranial contents.

The intracranial pressure can be continuously recorded by a variety of methods. The intraventricular catheter system previously described was the first to be developed and remains the most widely used. Reliable, reproducible recording with minimum damping is provided and the catheter rarely becomes occluded. The intraventricular method remains the standard against which newer techniques must be compared. A major advantage of the system is that ventricular fluid can be withdrawn to abruptly lower ICP. The principal deficiency of intraventricular monitoring is the fluid pathway between the exterior transducer and the ventricular system, which increases the risk of ventricular infection. The incidence of ventriculitis is about 5 percent in long-term catheter placements and is directly related to the duration of monitoring. Eighty five percent of these infections occurred in systems which were in place for more than five days. Frequent cultures of ventricular fluid should be taken and the catheter changed to a new location every five days.

The catheter is inserted through a burr hole over the convexity just anterior to the coronal suture and 2 cm lateral to the midline (Fig 6). The ease of placement within a ventricle is proportional to its size. Very small or distorted ventricles may make placement impossible.

The subdural plug or bolt was developed as an alternative to ventricular puncture. The plug is screwed into a small twist-drill hole in the convexity behind the hairline after opening the underlying dura. The internal opening of the hollow bolt is placed flush with the inner table of the skull and opens into the subdural space (Fig 6). The plug is connected to a pressure transducer by fluid-filling tubing. Penetra-

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**Figure 6.** Schematic representation of a subdural pressure bolt (left) and an intraventricular catheter (right). Both systems are connected by nonexpansile tubing to a pressure transducer.
tion of the brain is avoided, ventriculitis does not occur, and the system can be used when the ventricles have been collapsed by swollen brain. The principal disadvantages of this method are the occasional occlusion of the plug by swollen brain resulting in a spuriously low pressure reading and the impossibility of withdrawing CSF to lower elevated pressure. The incidence of local and subdural infection is 5 percent.

Subdural and intraventricular monitoring are used with about equal frequency. The choice of method may often be made on the basis of CT scan findings; if the ventricles are normal or enlarged, the catheter is placed; if they are narrow slits, the subdural plug is used.

Other techniques have been developed, but are less widely used. Miniature transducers have been developed for placement between the dura and the skull. Theoretically attractive since the dura need not be opened and there is no fluid coupling, these electronic transducers have many problems with baseline drift and stability which have precluded their wide use. An implantable transducer based on fiberoptics and, unlike electronic transducers, capable of recalibration after insertion, has been developed and appears to measure ICP reliably.

**TREATMENT OF INCREASED ICP**

The methods used in treating increased ICP will depend largely on its etiology. Surgical treatment is indicated if the pressure is due to hydrocephalus or to a focal abnormal mass (eg, tumor, hematoma) which can be removed. Quite often, however, the pathologic factor is an increase in the volume of one of the normal components of the intracranial space such as cerebral edema, contused brain, or diffuse cerebral swelling secondary to pathologic vasodilatation. In these instances, no removable mass is present, and medical treatment of increased ICP is directed at decreasing the volume of the intracranial contents. Ideally, a decrease in the size of the enlarged component is desired but this is not often possible. Reducing the volume of any of the components will decrease the pressure. The immediate goal of treatment is to reduce the total volume of the cranial contents, and thus, any therapy which effectively decreases the volume of any component is of value.

The therapeutic modalities available include ventricular drainage, controlled hyperventilation, osmotic diuretics, steroids such as dexamethasone, and barbiturate coma. All of these methods are temporizing in nature; none of them, alone or in combination, will permanently decrease ICP. The immediate aim of treatment is to keep the ICP under control until the underlying pathologic condition abates. These means can also be used to keep ICP decreased while preparing for the surgical removal of a focal mass.

Treatment of increased ICP should not be undertaken without a diagnosis of its cause. This is best determined with CT scanning which will show the size, location, and the nature of the offending mass or masses and determine whether medical or surgical therapy is more appropriate. The appearance of the scan may also give some guidance as to the form of medical therapy most likely to be effective. The best way to monitor therapeutic effect, however, is to measure ICP during the treatment. Changes in clinical findings cannot be relied upon to demonstrate the response to therapy.

Arterial blood pressure should be monitored closely as well. Hypotension is especially dangerous when autoregulation is impaired and low arterial pressure can lead to cerebral ischemia. Excessive hypertension may be just as dangerous in a pressure-passive arterial tree. If the cerebral arterioles are maximally dilated to adapt to a low perfusion pressure, a sudden increase in arterial pressure will further dilate the vessels, increasing cerebral blood volume. Increased arterial pressure will be transmitted to the capillary level, disrupting the blood-brain barrier and causing cerebral edema. Both of these effects cause further increases in ICP.

Frequent arterial blood gas determinations are also indicated during therapy. It is obviously important to avoid high PCO₂ values, but a PO₂ of 80 or more must be maintained as well. In conditions of low CBF such as can occur with intracranial hypertension, very modest decreases in PO₂ can cause major decreases in brain function.

The relationship between the height of ICP and the level of neurologic function is not clear cut. At what level should treatment of ICP be started? Certainly patients with pseudotumor cerebri may have transient pressures as high as 80 mm Hg without any neurologic deterioration. This level of ICP can be tolerated if the brain and its blood vessels are normal and autoregulation is intact. Injured brain will not tolerate such pressures; clinical and electrical evidence of neurologic deterioration appears at ICP levels of 40 mm Hg in brain-injured patients. In order to maintain a margin of safety, treatment of ICP is begun in most centers when the level rises above 20 to 25 mm Hg.

**Ventricular Drainage**

The most rapid reduction of ICP is accomplished by the removal of fluid from a lateral ventricle. Often, it is only necessary to remove 1 or 2 ml to effect a marked drop in pressure. An indwelling ventricular catheter can be used both for fluid removal and pressure monitoring. Repeated withdrawal of small amounts of CSF may serve to control ICP. If excessive fluid is removed, the ventricular volume may be collapsed by the pressure of swollen brain, leading to occlusion of the catheter. Although a quick and effective method of
lowering ICP, ventricular drainage is not useful as an initial emergency procedure because of the time required to visualize the ventricles by CT scan and to place the catheter. Repeated opening of the system to withdraw fluid also increases the possibility of ventricular infection.

Hyperventilation

Hyperventilation lowers ICP by reducing the PCO₂ which, in turn, causes cerebral vasoconstriction and decreases the cerebral blood volume. This effect of PCO₂ on vessel diameter probably occurs both through direct action on vascular smooth muscle and stimulation of peripheral chemoreceptors. Hyperventilation can reduce ICP by an average of 50 percent in most patients with intracranial hypertension. It takes an average of 7.5 minutes to effect this decrease, and if hyperventilation is continued, the reduction can be maintained for many hours. If treatment is stopped abruptly, intracranial pressure rapidly returns to its former level and may even overshoot it. When hyperventilation is discontinued, it should be tapered, allowing PCO₂ to slowly rise to normal levels.

In clinical practice, PCO₂ is decreased to between 20 and 25 mm Hg. Below this level, PCO₂ has proportionally less effect upon blood flow and volume and very little further reduction of ICP occurs. Furthermore, the intense vasoconstriction caused by PCO₂ of less than 20 is associated with CSF lactic acidosis in normal subjects, implying the development of ischemic hypoxia. Although this may not occur when using hyperventilation to treat marked cerebral vasodilation, it appears advisable to avoid profound hypocapnia.

Prolonged hyperventilation requires endotracheal intubation and neuromuscular paralysis if the carbon dioxide level is to be controlled. Continuous hyperventilation under this control appears effective at preventing a rise of ICP during treatment. There is some dispute over the long-term effectiveness of this modality; in some studies, up to one half of patients have shown an eventual return of intracranial hypertension during therapy.

Although hyperventilation does quickly reduce ICP, there is no convincing evidence that it is associated with a decrease in mortality. Since most comatose patients have an endotracheal tube in place early to protect their airway and since the technique is safe and effective, it has become widely used, especially as an initial emergency treatment.

Osmotic Diuretics

The two most commonly used osmotic diuretics are urea and mannitol. These agents act by reducing the water content of normal brain tissue and depend upon the presence of an intact blood-brain barrier. The tight junctions between endothelial cells of the cerebral capillaries prevent the movement of water-soluble substances into the brain tissue and are the anatomic source of the blood-brain barrier. If the osmolality of the blood is increased by adding a substance that will not cross the blood-brain barrier, there will be a net movement of water from brain to blood down the osmotic gradient.

Artificial osmotic gradients were exploited by neurosurgeons 50 years ago, but the only hypertonic solution without serious side effects was 50 percent glucose. Although glucose could rapidly extract brain water and lower ICP, it eventually crossed the blood-brain barrier reversing the osmotic gradient and negating its initial effect. The first reliable hypertonic agent was urea, introduced by Javid in 1956. Urea produced a rapid sustained fall in ICP, but a large number of clinicians noted a rebound, or increase in pressure above the original level, after a few hours. The distribution of urea after intravenous infusion was investigated, and it was found to cross the blood-brain barrier, although very slowly. Up to 12 hours are required for equilibrium between brain and blood concentrations. The second osmotic agent, mannitol, was introduced into clinical use by Wise and Chater in 1962. This agent does not appear to cross the blood-brain barrier. A number of investigators have claimed that there is a lesser incidence and severity of rebound with mannitol, although there is still considerable controversy over this point. Nonetheless, this apparent advantage over urea has led to mannitol becoming the most widely used osmotic diuretic. Most clinical and investigative work has been centered on this agent.

Both agents appear to be equally effective at reducing ICP when given at equivalent doses. The usual dosage for urea is 1 g/kg; that for mannitol is 0.5 to 1.0 g/kg. The duration of action of both agents is approximately four hours, and both have their maximum effect on ICP about 20 minutes after infusion. The diuretic effect of both agents is profound and a Foley catheter should be placed before using them. Their effectiveness is not dependent upon their diuretic action which, in fact, limits their effect by decreasing their serum concentration (and the serum osmolality) as they are lost in the urine. As a consequence of the falling concentration, urea and mannitol need to be repeated in four to six hours if ICP begins to increase again. The dosage schedule of these agents should be guided by the level of ICP and not by a fixed time schedule.

The dependence on an intact blood-brain barrier indicates that neither agent will decrease the water content of brain in areas where this barrier is defective. Pappius and Dayes have shown that the water content of edematous brain is not decreased by urea. Mannitol has had a similar lack of effect. The blood-brain barrier
appears to be disrupted in areas of clinical edema; in fact, this breakdown of the barrier is likely the cause of cerebral edema in nearly all cases. The effect of osmotic agents, then, is to decrease the volume of normal brain and thus “make room” for the edema mass. Mannitol has been shown to reduce the elasticity, or stiffness, of brain tissue to a greater degree than it lowers the intracranial pressure. Urea might be expected to have a similar effect in blunting the volume-pressure response and shifting the volume-pressure curve to the right (Fig 2).

The principal danger in osmotic therapy is the induction of severe serum hyperosmolality when large, repeated doses of the agents have been used in a desperate attempt to control rapidly rising ICP. Severe hyperosmolality can cause massive kidney damage, and if osmolarity must be increased above 320 mOsm to control ICP, mortality from either uncontrollable ICP or renal failure approaches 100 percent. Clearly, the smallest effective dose of osmotic diuretics must be used, and continuous ICP monitoring is vital to assess the response to the agent and the necessity for repeated doses. Serum electrolyte levels and osmolality must be frequently checked. If ICP cannot be controlled at a serum osmolality of 320, barbiturate coma should be considered.

**Steroids**

Steroids, especially dexamethasone, have long been used in the treatment of increased ICP. This agent is particularly effective when the excess pressure is due to cerebral edema around a brain tumor. A dose of 4 mg every six hours is usually adequate in this setting. When dexamethasone is used to treat other causes of increased ICP, the results are much less favorable. They are of little value in the ischemic edema of stroke or in intracerebral hematomas. A large volume of literature has accumulated regarding their value in head injury. In general, steroids do not improve the outcome in head injury. Initial enthusiasm for “megadose” steroids up to 150 mg of dexamethasone per day in head injury has not been borne out by recent studies of their effect on clinical outcome.

**Barbiturate Coma**

A small group of patients with increased ICP and no removable mass lesion develop persistent elevated pressure which cannot be controlled by the usual means. In patients with severe head injury, about 15 to 25 percent fall in this category and all go on to die. These patients are candidates for barbiturate therapy. Marshall et al have shown that 75 percent of such patients can be controlled with barbiturate coma.

Interest in barbiturate therapy was stimulated by the report of Shapiro et al that the rise in ICP caused by the induction of anesthesia could be negated by thiopentone. The following year, this therapy was reported to be successful in controlling increased ICP in patients with head injury who failed to respond to treatment with hyperventilation, osmotic diuretics, and steroids. Since that time, barbiturate coma has been increasingly used in this desperate situation. Although ICP can be reduced effectively in a high proportion of cases, the effect of barbiturate therapy on the ultimate clinical outcome is still not clear.

Barbiturates may lower ICP by decreasing the cerebral metabolic rate and increasing cerebral vascular resistance. Both of these effects lead to a decrease in cerebral blood flow and cerebral blood volume. In addition to the volume-reducing effect that barbiturates share with other modalities for treating increased ICP, they also decrease the oxygen demand of brain tissue which may allow these cells to better withstand the effects of decreased perfusion pressure. The significance of this facet of its action is as yet unknown.

Barbiturate therapy is usually begun with a loading dose of pentobarbital of 3 to 5 mg/kg given over several minutes. A fall in ICP, if it is to occur, will be evident within 10 to 15 minutes. If there is a good response to the loading dose, treatment is continued at doses of 1.5 to 2.0 mg/kg every one to two hours. The continued dosage schedule is usually adjusted to maintain a blood level of pentobarbital which keeps ICP within normal limits (less than 20 mm Hg) and which does not exceed 4 mg/dl. Hypotension is common when levels are greater than 4 mg/dl, and these levels do not cause further appreciable decrease in ICP. Shapiro et al have suggested the use of a test dose of thiopental (1.5 to 3.0 mg/kg) to check the response of ICP to barbiturates before giving the longer-acting pentobarbital. If there is no response to this agent or to the loading dose of pentobarbital, brain death from increased ICP is inevitable.

Before starting barbiturate therapy, a CT scan should be performed to rule out the possibility that the intractable pressure is due to a developing focal mass which could be removed. Barbiturate therapy should only be considered when ICP remains persistently above 25 to 30 mm Hg in the face of the most vigorous use of all other modalities. Barbiturates depress brain stem reflexes and make useful neurologic examination impossible. Barbiturate levels of 5 mg/dl or more cause an isoelectric EEG. In addition, barbiturate levels in the upper therapeutic range are associated with hypotension and respiratory depression. Hypothermia is another common side effect.

The dangers of barbiturate therapy are obvious from the above. One serious disease—barbiturate overdose—is being substituted for another. The patient is essentially anesthetized with an unstable blood pressure and deficient respiratory drive. It is clear that the
The highest level of care and monitoring is absolutely necessary if life threatening problems are to be avoided. Monitoring of ICP gives the only indication of the state of the intracranial contents, and reliable measurement must be constantly displayed. An arterial pressure line is equally critical to maintain control of blood pressure and avoid hypotension. If mean arterial pressure falls below 80 mm Hg, cerebral perfusion pressure may be compromised, and pentobarbital must be stopped.

Respiratory depression makes the patient totally dependent on the respirator and constant attention to this machinery is required. Arterial blood gas levels must be closely followed as must electrolytes and pentobarbital levels. Adequate intravascular volume must be maintained, and barbiturate therapy cannot be considered until the volume is adequate. A Swan-Ganz catheter is essential to monitor volume status. Finally, constant monitoring of core temperature and the use of warming blankets as necessary are required to keep body temperature above 33°C. Barbiturate therapy is an instance of “desperate measures for desperate times.” The decision to begin it cannot be taken lightly. Its effectiveness in lowering ICP must be regarded in the light of its inherent dangers. Barbiturate therapy should only be used within a well-equipped, fully staffed, sophisticated intensive care unit. Until the ultimate value of this technique is clarified, its use should be carefully restricted.

CONCLUSION

Increased ICP is a frequent secondary effect of a variety of intracranial pathologic processes. It is an important determinant, although not the only one, in the final clinical outcome of these conditions. The value of lowering an elevated ICP has not yet been precisely determined; it appears great in Reye’s syndrome and other “benign” toxic or viral encephalopathies, but it is not as clear in severe head trauma and stroke. Nonetheless, raised ICP has been shown to have deleterious effects on injured and diseased brain; careful monitoring and control of ICP can prevent or limit further secondary brain damage. If elevated pressure is due to a focal intracranial mass, surgical removal is virtually always indicated. When no surgical therapy is possible, ICP can be lowered by a number of effective measures such as hyperventilation, osmotic diuretics, and barbiturate coma. If these therapies are used in conjunction with continuous direct ICP monitoring and with a clear understanding of their modes of action and their dangers, they are very valuable adjuncts in the treatment of critical intracranial problems. Although control of ICP is no panacea, lives will be saved if intracranial hypertension is prevented. The possibility of increased ICP should be considered in the evaluation of neurologic deterioration, and if present, may be successfully treated by the methods outlined above.

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