The authors present a review of the management of patients with intracranial injury. The purpose of the article is to provide the anesthetist with a working knowledge of the physiology of intracranial dynamics and pathophysiology of intracranial insult. Emergency techniques and perioperative management are discussed.

Physiology and pathophysiology

The management of a patient who has sustained a severe cerebral insult requires an understanding of the volume pressure relationship of the intracranial contents: cerebrospinal fluid (CSF), intravascular blood, and brain tissue.

Intracranial pressure (ICP) is the pressure relative to atmospheric pressure within the ventriculo-subarachnoid space. Pressures are given in millimeters mercury (mmHg) or centimeters water (cmH₂O). Lundberg has described a normal ICP as 10 mmHg; slightly increased as 11-20 mmHg; moderately increased as 21-40 mmHg; and severely increased above 40 mmHg.  

Earlier concepts of intracranial pressure and volume relationships were described by Monroe and Kelli between 1783 and 1835. They proposed that the intracranial contents were nearly incompressible. It was not until 1846 that Burrows described the contribution of CSF to intracranial dynamics. The modified Monroe-Kelli Doctrine states that a change in volume of one of the intracranial constituents must be accompanied by a reciprocal change in one or both of the other constituents if pressure change is to be avoided. Although CSF accounts for only 10% of the total intracranial volume, CSF translocation is the major means of buffering for expanding intracranial masses. An understanding of CSF dynamics is therefore helpful in planning a regimen for patients with intracranial hypertension.

Cerebrospinal fluid formation is accomplished primarily by the choroid plexus located in the lateral, third and fourth ventricles. Small amounts of CSF are also produced in the perivascular space and from structures in the central canal of the spinal cord. The rate and direction of CSF flow is dependent on the structure of the subarachnoid space, pressure gradients secondary to arterial and respiratory pulsations and sudden changes in position. Cerebrospinal fluid is primarily absorbed through the arachnoid villi which protrude into the dural venous sinuses. Small quantities are absorbed along the perineural and perivascular spaces.

The rate of CSF formation is constant, despite an ICP of 150 mmHg (200 cmH₂O). However, the rate of CSF absorption increases with increased ICP. Reabsorption is exclusively dependent on the hydrostatic pressure difference between the subarachnoid space and the dural sinuses. Changes in volume of the intracranial content lead to predictable changes in the CSF pressure, which de-

Journal of the American Association of Nurse Anesthetists
pend not only on the magnitude of the volume change but also on the craniospinal compliance. (Compliance is the ratio of change in volume to the corresponding change in pressure. Compliance may also be understood as a measure of the stiffness or rigidity of the craniospinal compartment).

The craniospinal compartment becomes non-compliant or "tight," when an increase in the volume of one of the intracranial constituents is not accompanied by a reciprocal decrease in the volume of another. A "slack" or compliant brain is associated with reciprocal volume changes. Intracranial volume-pressure relationships may be described by the intracranial compliance curve. (Figure 1.)

Initial compensatory mechanisms which modify increases in ICP include the shift of CSF into spinal subarachnoid space, the expansion of which is accommodated by displacement of epidural venous blood; increased reabsorption of CSF across the arachnoid villi; and reduction in intracranial blood volume due to the pressure exerted by the CSF on the thin-walled cerebral veins.

A slowly increasing mass will displace CSF from its intracranial compartment through the foramen magnum into the distensible subarachnoid space. As ICP begins to increase, CSF absorption can increase, thus providing additional ICP buffer; with time, the overall CSF content in the cerebrospinal axis is reduced. A very important factor is the time required for spatial compensation. As much as 1 ml CSF/min can be expressed from the cranial space in the presence of increased pressure. If rapid displacement of CSF were not possible, rapid expansion of even the smallest mass would be incompatible with life.

When the ability of CSF to act as the ICP buffer has been exhausted and further spatial compensation cannot be achieved by reduction in intracranial blood volume, brain stem compression occurs. Progressive enlargement of the intracranial mass distorts brain tissue and leads to herniation through the foramen magnum, collapse of the large CSF cistern and compression of the arachnoid villi.

**Cerebral blood flow and ICP**

Intrinsic regulation of cerebrovascular resistance (CVR) is the most important determinant of cerebral blood flow. Many factors influence CVR by altering the pressure gradient across the vessels or by changing resistance within them. Resistance across the vessels refers to arterial and venous pressure at brain level and ICP. Resistance within the vessels refers to the degree of active constriction or dilatation of cerebral vessels and the viscosity of blood.

Cerebral blood volume (CBV) is equal to the cerebral perfusion pressure (CPP) divided by the cerebral blood flow (CBF): 

\[
\text{CBV} = \frac{\text{CPP}}{\text{CBF}}.
\]

Factors affecting CVR and, therefore, regulating CBF, include chemical and external factors such as arterial carbon dioxide tension \((\text{PaCO}_2)\), myogenic (autoregulation) and neurogenic.

Variations in \(\text{PaCO}_2\) produce rapid and significant changes in CBF. Carbon dioxide \((\text{CO}_2)\) diffuses through vascular walls so that changes in \(\text{PaCO}_2\) are eventually reflected in the \(\text{PCO}_2\) of the CSF. Arterial smooth muscle is extremely responsive to environment pH, which is determined by a variety of factors including the concentration of \(\text{CO}_2\) and bicarbonate ions in the CSF. Carbon dioxide concentration is largely determined by \(\text{PaCO}_2\); bicarbonate concentration is controlled by cerebral metabolic processes because the blood-brain barrier is permeable to \(\text{CO}_2\) and essentially impermeable to bicarbonate. Variations of \(\text{PaCO}_2\) from 20-80 mmHg produce direct 2% changes in CBF for every 1 mmHg change in \(\text{PaCO}_2\). (Figure 2.) Hypercapnia dilates cerebral vessels and increases CBF, CBV, and ICP. Hypocapnia con-
stricts cerebral vessels, thereby reducing CBF, CBV, and ICP. Hyperventilation is therefore a useful therapeutic maneuver in patients with intracranial hypertension.

Arterial oxygen tension is inversely related to CBF. (Figure 2.) Marked hypoxia (PaO₂ < 50 mmHg) significantly increases CBF and ICP, and is capable of overriding the effects of hypocarbia. This appears to be a threshold phenomena, since increased CBF is not seen until PaO₂ decreases to a level below which hypoxia causes progressive brain tissue lactic acidosis.\(^\text{14}\)

Cerebral blood flow remains relatively constant over a wide range of arterial pressures, provided systemic blood pressure changes relatively slowly. This phenomena, called autoregulation, is a mechanism by which arteries alter their diameter in response to changes in transmural pressure so that the pressure transmitted to the microcirculation is maintained at a relatively constant level.

In normotensive patients the lower limit of autoregulation is a mean arterial pressure (MAP) of 50 mmHg, below which CBF decreases. At a MAP of 45-55 mmHg, symptoms of cerebral ischemia (nausea, syncope, dizziness, blurred vision, and so forth) appear. The upper limit of autoregulation in a normotensive patient is 150 mmHg. (Figure 2.) Above this pressure the constrictor response is lost and the blood-brain barrier is disrupted, thereby increasing the formation of edema.

In chronic arterial hypertension the autoregulatory curve is displaced to the right. Cerebral vessels adapt to higher pressure levels, and upper and lower limits of autoregulation are increased. In awake patients with hypertension (MAP's between 125-180 mmHg), the lower limits of autoregulation are 90-125 mmHg. In these patients clinical signs of cerebral ischemia occurred when the MAP ranged between 35-80 mmHg.\(^\text{5}\) Chronic hypertensives should not be subjected to pressures as low as those tolerated by patients with normal blood pressure.\(^\text{4,6,9,11}\)

Cerebral perfusion pressure (CPP) is an estimate of the pressure gradient between the internal carotid artery and the subarachnoid veins, that is, the pressure gradient across the entire brain. Cerebral perfusion pressure is equal to the MAP minus the ICP: \(\text{CPP} = \text{MAP} - \text{ICP}\).

In functioning autoregulation, CPP ranges from 85-95 mmHg. Cerebral perfusion pressure may be narrowed by either a fall in MAP or an increase in ICP.\(^\text{4,7-11}\)

Neurogenic influence on CBF is small. The larger arteries and arterioles on the surface of and within the brain are supplied with sympathetic nerves from the superior cervical ganglion and with parasympathetic nerves from the greater petrosal branch of the facial nerve. Combined experimental studies indicate that maximal stimulation of sympathetic nerves reduces CBF 5-10%; a similarly moderate vasodilator response has been observed with parasympathetic stimulation. Perhaps the major contribution of neurogenic influence on CBF is seen during hemorrhagic hypotension where sympathetic stimulation enhances the tone of the arterial vessels so that the autoregulatory response curve is shifted to the right. Acute hypertension or hypotension is better tolerated if the sympathetic nerves are stimulated simultaneously.\(^\text{10,11,16}\)

If the injury to the brain is great enough, the resultant responses include depressed neuronal activity, acidosis, edema and disturbances of all CBF regulatory mechanisms. Brain lactic acidosis is characterized by cerebral vasomotor paralysis, particularly abolished autoregulation. The formation of edema is related, in part, to vasomotor paralysis which tends to increase capillary hydrostatic pressure, and damage to the blood-brain barrier. The combination of hypercapnia, cerebral acidosis, impaired autoregulation, and systemic hypertension promote the formation and spread of edema. Edema distorts brain tissue and further increases ICP, which induces even more tissue hypoxia and acidosis.\(^\text{4,6,8,9,12}\)

The signs and symptoms associated with increased ICP are related to traction on cerebral
vessels, distortion of the pain-sensitive dura mater, impending herniation and intermittent vascular compression, or midline shifts or axial distortion of the brain stem. Headache, nausea, vomiting and papilledema are symptoms of increased ICP. Changes in vital signs have served as an accurate index of the severity of intracranial hypertension. The combination of systemic hypertension, bradycardia and respiratory irregularity (an occurrence secondary to cerebral ischemia and medullary compression), is known as the Cushing Response. Third nerve dysfunction, decerebration, alterations in muscle tone and loss of consciousness also occur with severe distortion of brain mass.5,9,10

Neurodiagnostics

Computerized axial tomography (CAT scan) is used for diagnosis of increased ICP. Other diagnostic tests which are helpful include pneumoencephalography, ventriculography, cerebral angiogram and myelogram.

General anesthesia for neuroradiologic procedures may be required in pediatric patients and with extremely apprehensive or uncooperative adults. Choice of anesthetic agent should be based on neuroanesthetic principles which will be discussed in detail. However, nitrous oxide (N2O) is not recommended after air studies have been done since up to seven days are required for air injected into the ventricles to be absorbed.17 Nitrous oxide will expand this air and may produce a further increase in ICP. Complete reabsorption of air can be confirmed by x-ray.

Management

The initial treatment of patients with acute elevation of ICP is rapid reduction of the intracranial volume to decrease ICP, improving cerebral perfusion and energy supply and demand. In addition, brain shifts and distortions and their associated systemic effects may be corrected.

Endotracheal intubation and mechanical ventilation are used to maintain PaO2 of normal values and hypocarbia. Most traumatic head-injured patients spontaneously hyperventilate as a result of severe intracerebral metabolic acidosis caused by the brain injury. Controlled hyperventilation must be guided by arterial blood gases. It is recommended that PaCO2 be 25-30 mmHg. There is evidence that a PaCO2 of less than 20 mmHg reduces CBF to near minimal values, causing tissue hypoxia. Although cerebral vascular CO2 reactivity is impaired in areas of injury, CO2 reactivity may remain in the surrounding tissue. Hyperventilation will stimulate an attempt at restoration of normal cerebral pH and, therefore, the preservation of viable brain tissue. Hyperventilation may also allow corticosteroids to begin reducing cerebral edema.8,11

Sedatives and muscle relaxants may be necessary to facilitate intubation and to maintain controlled ventilation without increasing MAP or ICP. If the blood brain barrier is intact, osmotic diuretics reduce ICP by increasing plasma osmolality greater than brain osmolality with subsequent renal excretion of brain extracellular water. Cerebrospinal fluid formation is also decreased. Mannitol is often used and is administered intravenously in a dose of .25-0.5 g/kg given within 15 minutes. Mannitol begins to reduce ICP within 10-15 minutes. The effect of the initial dose lasts at least 2 hours.

Some of the disadvantages of mannitol inherent in all osmotic dehydrating agents include: transient increases in circulating and cerebral blood volumes and ICP, changes in blood coagulation and viscosity, increased serum osmolality and decreased serum electrolytes. These effects are attenuated or prevented when the smaller dose of .25-0.5 g/kg is used instead of the standard recommended dose of 1.0 g/kg.

Recent studies have advocated the use of furosemide (Lasix®)18 or ethacrynic acid (Edecrine®)19 for reduction of intracranial hypertension. Systemic diuretics reduce ICP by (1) reducing the volume of the intracranial contents in proportion as the percentage amount of total body water decreases, and (2) by decreasing CSF formation. Both furosemide and ethacrynic acid have direct effects on brain cells; they decrease chloride and water transport into astrocytes of damaged brain tissue, thus preventing cytotoxic edema. As a primary diuretic, in doses of 1.0 mg/kg, furosemide reduced ICP without significant osmolar or electrolyte change. Furosemide may be used as an adjuvant in doses of .15-.30 mg/kg. With any diuretic, serum potassium is measured frequently and replaced if decreases below 3.5 mEq/L occur.

Steroids are commonly used to decrease vasogenic edema associated with tumor, hematoma or abscess. They may be less effective in edema associated with acute infarction or cytotoxic edema from asphyxia, hypoxia or hypo-osmolality. The mechanism by which steroids affect cerebral edema is not known, however their effectiveness is thought to be due to stabilization of the blood-brain barrier and enhancement of cerebral energy supply and metabolism. Steroids will not rapidly reduce ICP; however, within hours they may improve the neurologic status of those patients in whom
vasogenic injury is complicated by edema. Dexamethasone, 10-20 mg initially followed by 16-64 mg/day, or methylprednisolone, 100 mg followed by 100 mg/day, has been used. Reversal of impending herniation has been reported following aggressive management with hyperventilation, dehydrating agents and sedatives. These treatment modalities are utilized to some extent for patients with non-acute neurologic pathology as well as in the crisis situation.

When the acute situation has stabilized, further treatment modalities may be considered to include barbiturates or sedatives, temperature control, CSF diversion, and surgical decompression.

The use of barbiturates has been shown to be effective in reducing ICP without a simultaneous reduction in CPP. There is a dose-related depression in cerebral metabolism and a significant reduction in CMRO₂, theoretically affording the brain some protection during periods of hypoxia. Since in the doses required to lower ICP (1.0-8.0 mg/kg/hr of thiopental or pentobarbital), signs of neurologic deterioration are masked, it is recommended that barbiturates be reserved for patients in coma. Prolonged barbiturate-induced coma and improvement in neurologic outcome with treatment of resistant intracranial hypertension has been under intense investigation. Therapeutic effectiveness and improvement in neurologic outcome is under debate. Recent investigations suggest that barbiturate therapy may have no benefit in cases of global ischemia. In addition, the complication rate, and amount of time and personnel necessary to maintain a prolonged anesthetic state, is extremely costly.

Induced hypothermia is seldom used although it has been employed for a variety of clinical situations—notably clip ligation of intracranial aneurysms. Hypothermia reduces CBF and CMRO₂. For each degree decrease below 37°C, CMRO₂ is decreased 7%. Among the complications of induced hypothermia are cardiac arrhythmias, and shivering during cooling and rewarming which increases CMRO₂ by 50-200%. Moderate hypothermia in combination with drugs that have an anti-edema effect (barbiturates, steroids) may prove to be a better alternative.

Cerebrospinal fluid diversion is indicated when ventricular fluid outflow pathways are obstructed and/or intracranial CSF pathways are obliterated. Cerebrospinal fluid diversion is accomplished with the insertion of ventriculostrial shunt, ventriculoperitoneal shunt or, though rarely, ventriculolumbar shunt. The ventricular drainage shunts reduce ICP by providing a low resistance pathway for CSF drainage.

Surgical decompression, external and/or internal, may be done for uncontrollable brain swelling, especially in patients with severe craniocerebral trauma. External decompression involves removal of part of the skull, and is usually done for evacuation of epidural or subdural hematomas. Internal decompression involves removal of part of the brain. The major effect of decompressive surgery is the reduction in midline shift, brain herniation and/or brain stem displacement.

Anesthetic management

Anesthetic management is based upon the knowledge that the injured brain has regions of tissue acidosis, loss of autoregulation, loss of metabolic control of tissue perfusion and loss of CO₂ reactivity. The choice of anesthetic techniques is, therefore, determined by their effects on CBF, CMRO₂ and ICP.

The preoperative anesthetic visit includes evaluation of the state of intracranial tension and general medical condition. Traumatic head-injured patients should be carefully assessed for possible internal injuries of the chest and abdomen. Patients with decreased consciousness should be assessed for possible aspiration pneumonitis. Chest x-ray and arterial blood gases will help in the clinical assessment. Intravascular volume and electrolyte balance must be evaluated, especially in those patients treated with diuretics.

The presence of intracranial hypertension determines the use of premedications. Lethargic, obviously obtunded patients should receive no premedication. In patients with no or minimal symptoms of increased ICP, small doses of anti-emetic sedatives or diazepam may be given. Diazepam has minimal cardiorespiratory effects, reduces CBF, and has amnesic and anti-seizure activity. Hydroxyzine (Vistaril) has sedative and anti-emetic effects and little effect on ICP. Atropine or glycopyrrolate may be used to prevent excessive secretions and vagal reflexes. However, atropine is not recommended in patients presenting with cerebral aneurysms because of the increased heart rate (HR) and MAP.

Narcotics as premedicants are avoided because of their potential respiratory depression, decreased levels of consciousness, nausea, vomiting, and pupil constriction. Likewise the butyrophenones (droperidol) are avoided because of their respiratory depressant and hypotensive effects.

Monitoring

Management of the brain-injured patient is
most rationally approached by first obtaining a measurement of ICP. This can be accomplished by one of several methods: an intraventricular catheter, a subdural bolt or an epidural cup-catheter. (Figure 3.) Measurement of ICP during the perioperative period will alert the anesthetist to dangerous increases in pressure and allow for rapid treatment.

Direct blood pressure monitoring allows for careful control of blood pressure and frequent analysis of arterial blood gases. The radial, femoral, brachial, axillary and dorsalis pedis arteries are suitable for short-term cannulation.

A peripheral nerve stimulator will help to assess the extent of muscle paralysis. Muscle relaxation is necessary to prevent straining and coughing on the endotracheal tube, and any movement during dissection of vital structures.

Measurement of urinary output via a Foley™ catheter provides assessment of volume status and, during periods of hypotension, may indicate decreased renal perfusion.

An esophageal stethoscope provides continuous auscultation of heart and breath sounds. An esophageal temperature probe provides continuous observation of body temperature. Because hyperthermia increases CMRO₂ and CBF, it must be promptly treated.

**Air embolus**

During operations when the patient is in the sitting position, air entrainment is a major complication. Whenever the wound is 5 cm or more higher than the level of the right heart, a negative pressure is created in the venous system and air entrainment can occur. A right atrial catheter, placed through the subclavian, basilic, or internal or external jugular veins, provides a route for aspiration of air. Experimental evidence indicates that maximum aspiration of air is accomplished when the catheter tip is just below the junction of the superior vena cava and the right atrium. A multi-orificed catheter would withdraw more air than a single-orifice catheter. In addition, a multi-orificed catheter would eliminate the possibility of suction adhesion to the chamber wall, and efficiency of the catheter would not be as severely compromised by clot formation. Exact location of the catheter may be confirmed by chest x-ray. The right atrial catheter is also useful for infusion of fluids and drugs as well as measurement of cardiac filling pressures.

The Doppler™ is reportedly the most sensitive device for detection of air emboli. However, recent investigations have found that the Doppler™ detected air which was of no clinical significance and valuable operative time was wasted treating air emboli. Clinically significant air emboli can be detected by either the Swan-Ganz™ catheter or the end-tidal CO₂ monitor (FETCO₂) (either one in conjunction with a precordial Doppler™). Both monitoring devices were equally rapid in (1) detecting the clinical significance of air (the Doppler™ detected air no more than 30 seconds before pulmonary artery pressure (PAP) or FETCO₂ changes developed), and (2) indicating the resolution of the embolus and the safe continuation of surgery.

Venous air embolism causes intense pulmonary vasoconstriction which results in markedly increased pulmonary vascular resistance. Small volumes of air in the pulmonary circuit are reflected by increased PAP and ventilation-perfusion abnormalities which are reflected in a decreased FETCO₂ and/or increased venous admixture. End tidal CO₂ is inversely related to the PaCO₂.

**Induction of anesthesia**

The anesthetic technique is determined by the effects of drugs and maneuvers on CBF, CBV, CMRO₂ and ICP. Investigators have documented intraoperative increases in ICP during laryngoscopy, intubation, application of head clamps, and insertion of the Gigli saw guide. In those patients...
who presented with increased ICP preoperatively, the increase in pressure during perioperative maneuvers was significant. Appropriate anesthetic techniques will prevent or attenuate this response.

Sodium thiopental, a potent cerebral vasoconstrictor, increases CVR and decreases CBV and ICP. In addition, CMRO₂ is significantly decreased and parallels the decrease in CBF. Althesin® is a steroid compound with short-lasting anesthetic properties. Although not available in the United States, it is a popular drug for induction and maintenance of anesthesia in Great Britain. Althesin® has much the same effect as barbiturates; potent vasoconstriction with a decrease in CBF, CMRO₂ and ICP.

Midazolam maleate is a water soluble benzodiazepine recently introduced for induction of anesthesia. Recent investigations have found that midazolam does not increase ICP when used as an induction agent in patients with brain tumors.

Prevention of increased ICP during laryngoscopy and intubation is achieved by total muscle relaxation in a well narcotized or sedated patient. Pancuronium bromide, 0.06-0.1 mg/kg is the muscle relaxant of choice for intubation and maintenance since ICP changes are insignificant following its use. The mild tachycardia that occurs with pancuronium has been reported to result from vagolysis. For patients requiring rapid sequence induction, 0.20 mg/kg of pancuronium may be used since onset is as rapid as with succinylcholine. Gallamine has also been found to produce a mild tachycardia with no significant alteration in ICP.

Succinylcholine has been shown to moderately increase ICP even when fasciculations have been prevented. Recent reports have noted succinylcholine-induced hyperkalemia in patients with closed head injury and no evidence of peripheral paralysis. Although succinylcholine is not contraindicated in patients with intracranial pathology, pancuronium is considered to be a safe alternative.

In addition to the non-depolarizing property of d-Tubocurarine, ganglionic blockade and histamine release occurs. Cerebral blood vessels may dilate and increase CBV and ICP. Metocurarine (metubine iodide) is a non-depolarizing muscle relaxant similar to d-Tubocurarine in onset time and duration of action. Among the non-depolarizing muscle relaxants available in the United States (metocurarine, pancuronium, gallamine and d-Tubocurarine), metocurarine produces the least cardiovascular change. The effect of metocurarine on intracranial dynamics is presently being investigated.

Fentanyl (.006 mg/kg) reduces CBF with a parallel reduction in CMRO₂. Morphine and meperidine decrease CMRO₂ and CBF during hypocapnia. During normocapnia these narcotics have shown no significant effect on CBF.

Administration of intravenous lidocaine 1.5 mg/kg one minute before laryngoscopy will prevent increases in ICP associated with endotracheal stimulation. Additional doses of lidocaine may be given prior to noxious stimuli to prevent increases in ICP without cardiovascular depression. Equipotent doses of pentothal are as effective to control intracranial hypertension, however, arterial pressure would also be significantly reduced. In patients with intracranial hypertension and systemic hypotension, a further reduction in arterial pressure may compromise cerebral perfusion.

Droperidol (0.3 mg/kg) significantly decreases CBF and, to a lesser extent, CMRO₂. Persistent postoperative drowsiness may mask signs of intracranial events since central nervous system (CNS) depression may persist for 6-24 hours. The benzodiazepines, notably diazepam, may also be used as an adjuvant. Diazepam has been shown to cause a parallel reduction in CBF and CMRO₂.

Ketamine causes profound cerebral vasodilation and greatly increased CNS activity and is, therefore, not recommended.

Nitrous oxide, when used with oxygen will
cause cerebral vasodilation and thereby increase CBV and ICP. However, the cerebral vasoconstrictive effects of hyperventilation, barbiturates and narcotics will counteract this response when used concomitantly.\textsuperscript{40}

The volatile anesthetics, halothane, enflurane, and isoflurane are potent cerebral vasodilators, and cause dose-dependent increases in CBF and ICP. (Figure 4.) Impairment of autoregulation is directly proportional to the dose (1.1-1.6 MAC). The concomitant decrease in MAP and increase in ICP can severely compromise CPP.\textsuperscript{48,49} Although hyperventilation prior to the introduction of inhalational anesthetics should attenuate the increase in ICP, reports of ICP elevation, despite hypocarbia, do exist.\textsuperscript{43} Hyperventilation may fail to prevent a rise in ICP because vascular response to changes in CO\textsubscript{2} is impaired in areas of injury.

Halothane concentrations greater than 0.5 MAC reduce CPP and CVR. Furthermore, halothane has been found to alter brain water and electrolyte distribution and permeability of the blood-brain barrier, thus contributing to the formation of cerebral edema.\textsuperscript{43,44}

Convulsant activity, as noted by the electroencephalogram, occurred with enflurane administration, especially during the period of deep anesthesia combined with hypocarbia.\textsuperscript{45-47} As with halothane and enflurane, hyperventilation prior to the introduction of isoflurane may prevent an increase in ICP. During normocapnia, isoflurane produced a 25% increase in ICP, while halothane and enflurane produced pressure increases of 100%.\textsuperscript{50} Animal studies indicate that the effect of isoflurane on brain water and electrolyte distribution after cryogenic injury is comparable to that seen with halothane.\textsuperscript{51} Comparison studies in animals indicate that an increase in ICP and edema were greatest with the volatile anesthetics and least with barbiturates or neurolept anesthetics.\textsuperscript{51} (Table I reviews the effects of anesthetic agents on intracranial dynamics in patients with head injury.)

**Fluid management**

These patients may have a depleted intra-

---

**Table I**

The effects of various anesthetic agents on intracranial dynamics.

<table>
<thead>
<tr>
<th>Agent</th>
<th>CBF</th>
<th>CMRO\textsubscript{2}</th>
<th>ICP</th>
<th>CPP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Halothane</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Enflurane</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Nitrous oxide</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Nitrous oxide with Thiopental for Narcotics</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Ketamine</td>
<td>↑↑↑</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Morphine with Hypocarbia</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Meperidine with Hypocarbia</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Droperidol</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Innovar\textsuperscript{*}</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Diazepam</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Thiopental</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Althesin\textsuperscript{*}</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Succinylcholine</td>
<td>Z</td>
<td>Z</td>
<td>↑</td>
<td>Z</td>
</tr>
<tr>
<td>d-Tubocurarine</td>
<td>↑</td>
<td>NC</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Pancuronium</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
</tr>
<tr>
<td>Gallamine</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
</tr>
</tbody>
</table>

↑ Increases
↓ Decreases
sl Slightly
NC No Change
Z Effects of agent on these parameters have not been established (compiled from various sources)
vascular volume due to aggressive diuretic therapy, poor oral intake and restriction of intravenous fluids. An adequate circulating blood volume is necessary to prevent severe hypotension on induction and with position change.

Content of intraoperative fluids and the rate of fluid administration depend on the preoperative fluid deficit, maintenance fluid requirements, urine output and blood loss. Fluid loss and input should remain relatively equal. Blood replacement depends on intraoperative losses and preoperative hematocrit. It is best to replace blood loss with blood and not with crystalloids. Colloids should be avoided as they may easily pass through a disrupted blood-brain barrier and increase cerebral edema. Lactated Ringer's is the solution of choice. Administration of dextrose should be less than 100 gms/24 hours, as dextrose acts as a diuretic and its resultant water of oxidation will increase edema formation. Urine output, serum and urine osmolality and electrolytes, and hematocrit should be frequently monitored throughout the perioperative period.  

Blood pressure control

Acute, traumatic brain injured patients frequently present with systemic hypertension. This is a necessary mechanism to maintain cerebral perfusion; hypertension at this time should not be aggressively treated. If hypertension persists after decreasing ICP to less than 20 mmHg, steps should be taken to restore normal cardiovascular hemodynamics and prevent rebleeding and increased ICP.

Systemic hypertension can occur intraoperatively as a result of light anesthesia or surgical manipulation and retraction of the brain stem or cranial nerves. If deepening of anesthesia or discontinuing surgical stimulation does not resolve the hypertension, then antihypertensives are indicated. Sodium nitroprusside, trimethaphan, hydralazine or nitroglycerine may be used. It is not within the scope of this article to elaborate on methods of antihypertensive therapy. The authors refer the reader to the references at the end of the article.

Emergence and immediate postoperative care

Emergence from anesthesia must be smooth; the patient must not cough or strain on the endotracheal tube as this will increase systemic blood pressure and ICP and threaten hemostasis. Lidocaine intravenously may be helpful in preventing coughing and straining while the head dressing is being applied. Emergence from anesthesia and reversal of muscle relaxants depends upon the patient's preoperative status, area of surgical manipulation, and intraoperative problems.

Patients who were unconscious preoperatively, not responsive postoperatively or who had lesions in the area of the respiratory center (surgical stimulation can cause significant postoperative edema and respiratory depression can ensue, requiring reintubation) are kept asleep and paralyzed.

Stable, responsive patients may be reversed with neostigmine or edrophonium and atropine or pyridostigmine and glycopyrrolate. Those patients who are asleep but breathing adequately and tolerating the endotracheal tube should remain intubated to prevent regurgitation and aspiration. Controlled ventilation and hypocarbia should be maintained during the postoperative period until the patient demonstrates an ability to maintain adequate ventilation and protective reflexes have returned. Patients who have regained protective reflexes may be extubated in the operating room following established criteria. Persistent respiratory depression after reversal of muscle relaxation and reaccumulation of CO₂ may be treated with minimal doses of naloxone (0.1-0.4 mg). Narcotic reversal is rarely necessary and should be avoided if increased MAP is undesirable.

Heart rate and rhythm and blood pressure are monitored during the patient's transport from the operating room to the recovery room. Continuous cardiovascular and ICP monitoring is resumed in the recovery room. Laboratory tests including ABG's, serum and urine osmolality and electrolytes, glucose and chest x-ray are done frequently throughout the postoperative period. Steroids, diuretic therapy and fluid restriction are continued to minimize cerebral edema.

Summary:

Patients presenting with neurologic injury represent one of the most challenging situations in clinical anesthetic practice. Most investigators and clinicians have obtained optimal results with a balanced narcotic anesthetic coupled with intensive management of cardiovascular, pulmonary and intracranial dynamics.

An understanding of intracranial dynamics and the effects of therapeutic and anesthetic intervention can make a significant difference in the prognosis of the brain injured patient.

REFERENCES


(48) Murphy FL, Jr., Kennell EM, et al. 1974. The effects of


AUTHORS

Barbara Shwiry, CRNA, graduated from Kings County Hospital School of Nursing in 1975. She received her anesthesia education from the Medical College of Pennsylvania in Philadelphia. She is presently on Staff at Kings County Hospital, Department of Anesthesiology and is a Clinical and Didactic Instructor in Neuroanesthesia at the Kings County Hospital Center School for Nurse Anesthetists. Ms. Shwiry is involved in several research projects concerning neuroanesthesia and is Clinical Research Coordinator for the School of Nurse Anesthesia.

Joseph P. Giffin, MD, is Assistant Professor, Department of Anesthesiology, State University Hospital, Downstate Medical Center, Brooklyn, New York.

James E. Cottrell, MD, is Professor and Chairman, Department of Anesthesiology, State University Hospital, Downstate Medical Center, Brooklyn, New York.