Chapter 189: Anesthetic Considerations for Cranial Base Surgery

Mahesh P. Mehta, Martin D. Sokoll

The anesthetic management of patients with base of skull tumors extending intracranially concerns both the surgeon and the anesthesiologist. Probably in no other field can the skills of an anesthesiologist so profoundly affect the work of the surgeon. The anesthesiologist must fully understand the fundamental principles of neuroanesthesia, preoperative neurologic status of the patient, and procedure and goals of the surgeon. Knowledge of the cerebral and systemic effects of various anesthetics and special techniques can aid the surgeon in planning the procedure, resulting in better operating conditions that lead to an optimal patient outcome. Thus, a team approach should be adopted to properly apply these concepts to the anesthetic and surgical care of patients with intracranial extensions.

Physiology and Pharmacology

Intracranial pressure

The intracranial space may be considered as a rigid container of almost constant volume connected to the remainder of the body via the circulatory system. Blood, brain, and cerebrospinal fluid (CSF) are the normal intracranial components. To maintain normal intracranial pressure (ICP) a change in one component must be offset by a similar change in volume of another. Physiologic changes in ICP are compensated by displacement of CSF from cranium to spinal subarachnoid space, increase in CSF reabsorption, and probably displacement of blood out of the venous sinuses. If a small increase in volume of one compartment occurs, pressure volume equilibrium will be rapidly established, and no long-standing increase in ICP will occur.

The pressure-volume relationship within the cranium follows an exponential curve with flat and steep portions (FIg. 189-1). As one compartment increases in volume, the volume in the other compartments decrease and little or no increase in pressure is seen (Fig. 189-1). At the inflection point of the curve, the ability to compensate is impaired so that relatively small changes in volume lead to greater increases in pressure (Fig. 189-1). This biphasic response can be understood by considering the flat portion of curve as a result of CSF or venous blood displacement in response to an increase in cranial content. As the buffering capacity of the CSF and venous sinuses is exhausted, the cerebral substance plays a major role in the volume-pressure relationship and the pressure rises steeply. This volume-pressure relationship or compliance has received much experimental and clinical verification (Miller et al, 1973).

Cerebral blood flow

Normal cerebral blood flow (CBF) in the adult is about 45 mL/100 g/min or 750 mL/min (Lassen and Hoedt-Rasmussen, 1966). In the gray matter the flow is about 80 mL/100 g/min, while in white matter it is 20 mL/100 g/min (INhvar et al, 1965).

Autoregulation

CBF is remarkably stable over a wide range of mean arterial blood pressure (MABP). This phenomenon, termed *autoregulation*, refers to the ability of normal brain to maintain a constant CBF despite variations in MABP and body position (Lassen, 1974). The normal range of autoregulation is between 60 and 150 torr (Fig. 189-2). In the hypertensive patient, this range is shifted to the right (Strandgaard et al, 1973). At the lower limits of autoregulation, 60 torr perfusion pressure in normal brain, the CBF begins to decrease, and when the perfusion pressure reaches 25 to 30 torr, the CBF will have fallen to about 50% of control. At the other extreme, autoregulation begins to fail when MABP exceeds 150 torr. As this limit is passed, there is disruption of blood-brain barrier and increased blood flow causing progressive cerebral edema and hemorrhage (Haggendal et al, 1970). Above and below the range of autoregulation, the flow is passively dependent on MABP.

The mechanism of autoregulation of CBF is unclear; metabolic, chemical, myogenic, and neurogenic mechanisms have been postulated. A probable explanation is that CBF autoregulation is a result of an intrinsic response in arteriolar smooth muscle to distension or relaxation caused by changes in intraluminal pressure (Folkow, 1964). Autoregulation is altered by volatile anesthetics. This effect is dose dependent and at deep levels of anesthesia CBF is pressure dependent. Autoregulation may also be lost in the presence of brain tumor, infarct, subdural hematoma, hypoxia, hypercarbia, and other drug therapies.

Carbon dioxide and cerebral blood flow

Hypercapnia produces vasodilatation and increased CBF, whereas hypocapnia causes vasoconstriction (Phelps et al, 1973; Reivich, 1964). This effect is rapid and linear (Fig. 189-2). When the $PaCO_2$ is between 20 and 80 torr, each increase or decrease of 1 torr causes a corresponding increase or decrease in the CBF of 2 mL/100 g. However, when $PaCO_2$ is below 20 or above 100 torr, there is very little further change in CBF. If intracranial compliance is normal, a small increase in $PaCO_2$ will have very little effect on the ICP. If intracranial compliance is reduced, however, small changes will have marked effect on ICP as a result of increase in cerebral blood volume. The goal of therapeutic hyperventilation is to maintain $PaCO_2$ of 25 to 30 torr (Marsh et al, 1977).

Role of oxygen and cerebral blood flow

Oxygen in clinical concentrates has little effect on CBF (Fig. 189-2). A measurable change occurs if PaO_2 falls below 50 torr, after which CBF increases (Cohen et al, 1967), reaching twice normal values at PaO_2 of 20 torr. Hyperbaric oxygenation at 1 or 2 atmosphere will result in cerebral vasoconstriction and decreased blood flow.

Effect of Anesthetics on Cerebral Blood Flow and Intracranial Pressure

Inhalation anesthetics

All inhalation anesthetics modify the autoregulation and increase CBF through vasodilation in a dose-dependent manner. With progressive increases in dose of a volatile agent, the plateau portion of the autoregulatory curve diminishes and higher CBF occurs at the same or lower MABP. In humans and animals, at equivalent minimum alveolar concentration, halothane is a more potent cerebral vasodilator than enflurane or isoflurane.

Halothane has been shown to be a vasodilator in many studies. It produces a small increase in ICP in patients with normal CSF pathways; however, marked increase in ICP may occur in patients with space-occupying lesions (Jennett et al, 1969). Enflurane (Stullken and Sokoll, 1975) and isoflurane (Adams et al, 1981) also have been shown to produce large increases in ICP in patients with space-occupying lesions. This increase in ICP may be attenuated either by prior hyperventilation (Adams et al, 1972) or by barbiturates (Shapiro et al, 1973). Isoflurane, in humans, produces isoelectricity or burst suppression of the EEG at 2.0 MAC concentration (Newberg et al, 1983). This unique feature of isoflurane has prompted the suggestion that it offers brain protection similar to barbiturates. In experimental animals, isoflurane appears to offer brain protection in incomplete global ischemia and in incomplete regional ischemia, but it appears not to be as protective as thiopental. This property of isoflurane has led to the suggestion that it is the inhalation anesthetic of choice in neurosurgical procedures, although there is no clinical evidence of brain protection.

The effect of nitrous oxide on CBF is controversial, but in patients with intracranial space-occupying lesions it has been shown to increase ICP. This response can also be attenuated by hyperventilation (Henriksen and Jorgensen, 1973).

Barbiturates

Barbiturates produce dose-dependent cerebral vasoconstriction in patients with space-occupying lesions. During normocapnia, thiopental causes a decrease in cerebral oxygen consumption ($CMRO_2$) and ICP. If hypocapnia is instituted, CBF decreases even more (Pierce et al, 1962). Recent animal studies indicate that barbiturate therapy may prolong the brain tolerance to induced ischemia. The magnitude of protection is directly related to the magnitude of metabolic depression induced by the barbiturate (Michenfelder and Theye, 1973) or the decrease in CBF and ICP (Smith et al, 1974).

Etomidate

Etomidate, a rapidly acting intravenous anesthetic, has also been shown to lower $CMRO_2$, improve CBF (Renou et al, 1978), and decrease ICP (Schulte am Esch et al, 1978). Also, etomidate does not depress the cardiovascular or respiratory system and may be an alternative thiopental when hypnosis is desirable. Unfortunately, use of etomidate has been limited as it has been reported to inhibit adrenal steroid genesis. The clinical implications of this effect are not clear at this time (Mehta et al, 1985; Wagner et al, 1984).

Propofol

Propofol is a new intravenous anesthetic that has been shown to produce dose-dependent reduction in both CBF and ICP (Ravussin et al, 1988; Vandesteene et al, 1988). Its use, at present, has been limited yo short outpatient surgical procedures because of the expense. Its major advantage in neurosurgical patients may be its rapid recovery, enabling early postoperative evaluation, and its low incidence of postoperative nausea and vomiting.

Narcotics and other drugs

Morphine (Weitzner et al, 1963) and meperidine (Messick and Theye, 1969) both cause a progressive and parallel decrease in $CMRO_2$ and CBF with minimal changes in ICP when ventilation is controlled. In dogs, fentanyl caused a marked and short-lasting decrease in $CMRO_2$ and CBF, whereas droperidol was found to be a more potent and longer-acting cerebral vasoconstrictor than fentanyl (Michenfelder and Theye, 1971). Neurolept anesthesia, that is, a combination of droperidol and fentanyl, has been shown in patients with normal and abnormal CSF pathways to decrease ICP, probably due to decrease in CBF (Fitch et al, 1969). Alfentanil and sufentanil, derivatives of fentanyl, appear pharmacodynamically similar to the parent drug and are alternative drugs for use in neurosurgical procedures.

The benzodiazepines, midazolam and diazepam, have been shown to decrease CBF and $CMRO_2$ (Nugent et al, 1982), midazolam having a greater effect than diazepam. Due to its short duration of action and lesser cardiovascular effects, midazolam may be a useful alternative to thiopental.

Ketamine causes marked increase in CBF and ICP in both normal and abnormal brains (Shapiro et al, 1972). It appears to have no use in neuroanesthesia.

Muscle relaxants

Curare administration is associated with a rise in ICP accompanied by a decrease in MABP and cerebral perfusion pressure. It is believed that histamine release is the underlying cause (Tarkkanen et al, 1974). Succinylcholine has been shown to alter intracranial hemodynamics, and its use is controversial (Cottrell et al, 1983). Atracurium and vecuronium, depolarizing muscle relaxants of intermediate duration and minimal cardiovascular side effects, are the drugs of choice. Pancuronium may be used as an alternative.

Anesthetic Management of Patients with Elevated Intracranial Pressure

Preoperative evaluation and premedication

When intracranial extension from a cranial base tumor occurs, it is usually into the posterior or middle cranial fossa. The problems posed in the anesthetic management of these cases are related to compression of brainstem and elevation of ICP due to obstructive hydrocephalus. The preoperative anesthetic visit should include determining the patient's level of consciousness, evaluating the presence or absence of elevated ICP, ascertaining the extent of neurologic deficits, and performing the routine evaluation.

Preoperatively, aggressive therapy to reduce ICP with diuretics and steroids may be necessary in these patients. This therapy is often accompanied by intravascular volume depletion and electrolyte abnormalities, which must be corrected before surgery. Patients with a decreased level of consciousness frequently have an associated aspiration pneumonitis, and a chest radiograph and blood gas measurements should be performed to aid in clinical assessment of this condition.

Premedicants capable of causing respiratory depression or alteration of state of consciousness in patients with increased ICP should be avoided. Anticholinergic drugs are useful in most patients, as they reduce tracheobronchial secretions and, in sufficient dosage, will minimize vagal effects on the heart secondary to anesthetic drugs or operative manipulation.

Induction and maintenance of anesthesia

During induction of anesthesia, minimal disturbances in intracranial dynamics is desirable. Achieving this goal requires maintenance of a stable hemodynamic state and a clear airway. A smooth, rapid induction may be established with an intravenous barbiturate. Thiopental produces a slight to moderate blood pressure fall, is a potent cerebral vasoconstrictor, and reduces ICP by decreasing CBV. Controlled hyperventilation to maintain $PaCO_2$ of 25 to 30 torr should immediately follow loss of consciousness; this goal may be facilitated by using a long-acting, nondepolarizing muscle relaxant. Hyperventilation usually increases cerebrovascular resistance and decreases CBV and ICP. Neurolept-anesthesia or volatile anesthetics may then be introduced safely in low concentrations after hypocarbia has been established.

Because autoregulation may be lost or impaired by the anesthetic agents or disease process, sudden large changes in blood pressure should be avoided. Laryngoscopy and intubation can produce a marhed hypertensive resonse. Gentleness and a well-anesthetized patient are essential. Good muscular relaxation to avoid coughing and straining is a useful adjuvant. In addition, small doses of thiopental, a short-acting hypotensive agent (nitroglycerin or nitroprusside), intravenous lidocaine, or spraying the trachea with local anesthetic may attenuate this response. An armored endotracheal tube should be used to avoid kinking of the tube with positioning of the head. Because positioning may also displace the tube, its proper location should be verified after placement and again after the final operative position is achieved. Also, while securing the airway, care must be taken to prevent extensive movement of the head and neck, especially in patients with large posterior fossa masses, as medullary compression can occur with neck movement.

Anesthesia can be maintained with intermittent doses of narcotics or barbiturates or low concentration of a volatile anesthetic, nitrous oxide, and muscle relaxant. The degree of neuromuscular blockade should be assessed with a "block-aid" monitor, and paralysis is maintained so that only a faint twitch response is present. This technique ensures that the patient will not cough, strain, or move during surgery - all of which can result in brain engorgement, bulging, and increased bleeding. The other advantage of maintaining a faint twitch is that during the surgery it helps to identify the facial nerve, which may be involved in extensive tumors. If no paralysis is desirable during surgery, such as when the facial nerve is in jeopardy, the anesthesiologist may maintain a deeper plane of anesthesia with

intravenous or inhalation anesthetics.

Maintenance of anesthesia is mainly concerned with recognition and management of problems presented by prolonged cases complicated by blood loss and hypothermia. Temperature monitoring, heating pad, and the use of blood warmers help to offset patient cooling. A Foley catheter is also useful to guide fluid therapy and is mandatory if diuretics or hypotensive drugs are used. A central venous catheter or pulmonary artery catheter is also helpful in assessing blood and fluid loss and replacement.

Monitoring during anesthesia should consist of electrocardiogram (ECG), intraarterial blood pressure, temperature, urine output, neuromuscular blockade, and a precordial or esophageal stethoscope. A central venous catheter or a pulmonary artery catheter is helpful in patients with cardiovascular diseases; these devices are indicated if the patient is in a sitting position. In addition, when the operative site is above the level of the right atrium, a Doppler ultrasound and end-tidal carbon dioxide monitor should be used to detect air embolism.

Emergence and recovery from anesthesia

The period of recovery should be as smooth as the induction period. Straining and coughing must be avoided, as these can lead to postoperative hemorrhage and cerebral edema due to arterial hypertension and elevated cerebral venous pressure. Therefore, anesthetic depth must be maintained until the operating is finished and the anesthesiologist has access to the head. Intermittent small doses of thiopental or lidocaine may also help in controlling emergence from anesthesia.

The preoperative level of consciousness may indicate that it may be impossible for the patient to be alert with a self-protected airway. In addition, intraoperative trauma to respiratory centers or cranial nerves may impair ventilation postoperatively and require airway control or ventilatory support.

Most intracranial procedures are associated with minimal postoperative pain. For this reason and to avoid the possibility of drug-induced respiratory depression, narcotics are usually reversed in extubated patients. With newer narcotics, reversal is not essential. In patients who remain intubated with ventilatory supprt, narcotics and muscle relaxants may not be reversed. Vital signs and neurologic status are carefully observed during the recovery period. It must be remembered that abnormal neurologic reflexes, characterized by hyperactive stretch reflexes, ankle clonus, the Babinski reflex, and decerebrate posturing, are common during emergence from anesthesia and may last as long as 1 hour after anesthesia (Rosenberg et al, 1981).

Immediate postoperative problems

Edema, hematoma, and infarction of the brainstem and cerebellum are extremely serious due to the close proximity of respiratory and cardiovascular control centers. Patients severely compromised, who had previously been awake and talking, will suddenly become unresponsive with systemic hypertension, bradycardia, and irregular or absent respiration (Cushing's triad). This triad, secondary to brainstem compression, is an indication for reintubation and prompt evaluation of the patient looking for causes of the increased ICP.

Patients are frequently hypertensive in the recovery room even without a history of high blood pressure before surgery. Because of the danger of hematoma or hemorrhagic infarction, blood pressure must be well controlled. If analgesics are not effective, then hypotensive agents, such as nitroglycerine, nitroprusside, or hydralazine, are indicated. Dysrhythmias are also common in the first 24 hours, and monitoring of the heart rate and rhythm is essential for at least 24 hours after surgery (Finkelstein and Nigaglioni, 1961). For these reasons, it is essential to admit patients to the intensive care unit for 24 hours.

Serum electrolytes, osmolarity, urinary volume, and specific gravity should be measured, as diuretics may decrease serum potassium and increase osmolarity. It is also common to develop an increased temperature after an exploration at the base of the cranium. This febrile response is usually due to the presence of blood in the subarachnoid space and should be controlled, as temperature reduction decreases the metabolic rate and adds to the comfort of the patient. Severe hyperpyrexia may also affect the level of consciousness and decrease the seizure threshold. Acetaminophen (Tylenol), cooling blankes, and steroids have been useful in controlling the temperature. Other causes for rise in temperature should be sought and treated accordingly.

Special Techniques and Problems

Positioning

The base of the skull is either approached in the prone or lateral position with head elevated or, rarely, in the sitting position. Proper positioning entails coordination of optimal surgical access to the lesion when concern for any adverse effect of position on the surgical field or the patient.

Prone position

Access to the midline and lateral structures is adequate in the prone position, although the superior parts of the posterior fossa may be difficult to reach. The major disadvantage is venous congestion, although the likelihood of air embolism is less. The patient's chest may be supported on a frame to prevent respiratory embarrassment and increased venous pressure. Pressure on the eyes should be avoided, as retinal ischemia and blindness have been reported due to orbital compression. This position may be better maintained with the use of skull pins and head clamps.

Lateral position

The lateral position is modified to the "park-bench" position by elevating, flexing, and rotating the head toward the floor to allow a wide exposure of the back of the head as in operations for acoustic neuroma. Another modification is the semiprone position with the face turned down to obtain more exposure of the back of the head for cerebellopontine angle tumors. In this position, care must be taken to avoid obstruction of the internal jugular vein.

Sitting position

The sitting position allows access to all parts of the posterior fossa and cervical spine. It provides excellent surgical exposure and gravitational drainage, thus reducing venous oozing and congestion at the site of surgery; however, it also introduces the hazard of sudden cardiovascular decompensation and air embolism and is less comfortable for the surgeon.

Anesthetics, muscle relaxants causing ganglionic blockade, increasing age, intravascular volume depletion, and hypocarbia reduce the ability of the cardiovascular system to compensate when the patient is moved from supine to the sitting position. When the sitting position is to be used, the anesthetic management should be designed to interfere minimally with circulatory reflex adjustments to position changes. The legs and thigs should be wrapped to increase venous return. A level of anesthesia that maintains high sympathetic tones, such as nitrous oxide, narcotic, and relaxant or low-dose volatile anesthetics, may be used. Positioning of the patient should proceed slowly with constant monitoring of blood pressure. In all three positions due to the long duration of some of these surgical procedures, detailed attention to positioning is needed to prevent peripheral nerve injuries and pressure necrosis.

Air embolism

Air can enter via the exposed venous system, which is at subatmospheric pressure, when the head is above the heart level. The rate and volume of air entering and the condition of the patient will determine the severity of cardiovascular insult. Air entering the veins rapidly reaches the right side of the heart and pulmonary circulation. Massive amounts of air that flood the right ventricle of the heart and pulmonary circulation can lead to death from acute cor pulmonale and anoxia. Small amounts of air are eliminated through the lungs, although some obstruction of pulmonary arterial circulation is produced. In the presence of an anatomic right-to-left shunt, air entering the right atrium can reach the systemic circulation. If nitrous oxide has been used as a part of the anesthetic technique, the volume of intravascular air will be augmented (Mehta et al, 1984) because nitrous oxide is more soluble in blood than is nitrogen; therefore, it can be transported rapidly into the trapped intravascular air.

Diagnosis is easier and more accurate if appropriate monitoring devices are used, and if a high degree of vigilance is maintained throughout the operation. The Doppler ultrasound monitor can detect less than 1 mL of intravascular air. Air embolism should be suspected when changes in heart sounds ("wheel-mill" murmur), cardiac dysrhythmias, fluctuation of blood pressure, low end-expired carbon dioxide, or elevated central venous pressure readings are noticed. Other signs may be gasping respiration in previously adequately controlled respiration. The surgeon may also notice tiny air bubbles in the exposed vain or hear a hissing sound from the site of air entry.

Treatment consists of preventing additional air entry at the surgical site by packing the wound and applying bilateral jugular vein compression. Simultaneously, an attempt should be made to remove air from the right heart through a previously positioned right atrial catheter, and 100% oxygen and vasopressores should be administered. If vital signs are not restored by these measures, the patient may have to be moved into the left lateral position with the head down to stop air entry, facilitate its aspiration, and increase venous return to

the heart. The use of a "G-suit" may provide transient cardiovascular support by increasing venous pressure and return, but its use is limited because of the inconvenience of placement during surgery.

Cardiovascular and respiratory problems

Cardiovascular system

Due to the close proximity of the medulla and cranial nerves, continuous observation of heart rate and rhythm can detect early signs of interference with these structures. Bradycardia, tachycardia, hypertension, hypotension, or extrasystoles are watched for; and the surgeon should be informed of their occurrence when they are not related to anesthesia. The surgeon must keep the anesthesiologist informed as to the stage of the operation and proximity to the brainstem. Tachycardia and hypertension are common with brainstem stimulation. With surgical manipulation around the fifth cranial nerve tachycardia, hypertension, and extrasystoles may be seen, whereas manipulation around the tenth cranial nerve results in bradycardia and hypotension. With the appearance of these signs, dissection should be temporarily discontinued, and dysrhythimas and blood pressure treated if necessary.

Respiratory system

Controlled ventilation with or without positive end-expiratory pressure is the most acceptable mode of respiration, although in the past spontaneous ventilation has been advocated for detecting interference with medullary centers. However, this is no longer thought to be necessary, as continuous monitoring of blood pressure and rhythm provides signs as valid as changes in the respiratory system.

Induced hypotension

There are few other surgical procedures in which such a strong case can be made for induced hypotension. Induced hypotension can decrease congestion caused by a tumor and reduce swelling. In addition, minimal arterial bleeding and venous oozing can improve operative conditions so that a finer, more definitive dissection is possible, thereby improving the changes for successful surgery. In addition, control of hemorrhage may make possible operations that could not otherwise be attempted. Induced hypotension may increase ICP and decrease cerebral perfusion pressure in patients with raised ICP and should be used cautiously in these circumstances. Drugs used to produce induced hypotension include sodium nitroprusside, nitroglycerine, and ganglionic blockers, for example, trimethaphan or pentolinium.

Sodium nitroprusside

Sodium nitroprusside is a potent vasodilator and is commonly used because of its rapid onset and short duration. The vasodilatory activity resides in the nitrosogroup, which reacts with sulfhydryl receptors on vascular smooth muscle, producing vasodilation. Vasodilation results in decreased peripheral resistance and increased venous capacitance. The net effect is a reduction in blood pressure with little effect on cardiac output in normal subjects.

Nitroprusside is dissolved in 5% dectrose/water immediately before its use and administered as a continuous infusion. It can be infused starting at a rate of 0.5 microg/kg/min and dose titrated until the desired clinical effect is achieved. The dose should not exceed 5 microg/kg/min (Michenfelder and Tinker, 1977) or a total of 3-3.5 microg/kg (Davies et al, 1975) because nitroprusside is metabolized to cyanide. Cyanide is probably liberated by direct combination of nitroprusside with hemoglobin and sulfhydryl groups in tissue and red cells. Circulating cyanide is converted in the liver to thiocyanate by the enzyme rhodanase. Thiocyanate toxicity appears at plasma levels of 5 to 10 mg/100 mL; fatalities have been reported at levels of 20 mg/100 mL (Palmer and Lassater, 1975). Cyanide toxicity is manifested early by development of tachyphylaxis, metabolic acidosis, and increased mixed venous oxygen content.

Nitroglycerin

Nitroglycerin is a potent vasodilator, producing relaxation of both arterial and venous smooth muscle. Its effect, however, is more on capacitance vessels, resulting in decreased venous return. It is also administered as a continuous infusion and the dose titrated to achieve clinical effect. A good hypotensive response is usually seen at 2 microg/kg/min. Nitroglycerin may be preferable for induced hypotension in neurosurgical patients, as toxic biotransformation products, tachyphylaxis, and rebound hypertension are not seen.

Diuretics

Diuretics are used frequently to facilitate surgery in patients with intracranial tumors by decreasing intracranial pressures and the size of the brain.

Osmotic diuretics

Mannitol is the most frequently used diuretic. It increases serum osmolarity and draws free water from areas of the brain with an intact blood-brain barrier. The fluid is excreted by osmotic diuresis. Maximum reduction in brain edema occurs at a serum osmolarity of 320 mosm/L (Shapiro et al, 1973). Reduction in ICP is usually seen in 20 to 30 minutes and lasts approximately 4 hours.

The dose of mannitol frequently used is 1 to 2 g/kg, but recent studies indicate that similar reductions in ICP may be achieved with doses in the range of 0.26 to 1 g/kg (Marshall et al, 1978; McGraw et al, 1978).

Mannitol may cause a transient increase in intravascular volume. This increase is especially a problem in patients with poor heart function, and the drug must be used cautiously in patients with congestive heart failure. It may also cause rebound increase in ICP when the blood-brain barrier is disrupted. Other osmotic diurectis, urea, and glycerol have largely been replaced by mannitol.

Loop diuretics

Furosemide also rapidly decreases ICP by causing diuresis, decreased CSF production, and increasing the CSF absorptive capacity (Sklar et al, 1980). When administered in doses of 1 mL/kg, reduction in ICP is seen in 30 to 60 minutes. The ability of loop diuretics to lower ICP without raising intravascular volume or blood osmolarity is an advantage over osmotic diuretics.

Summary

In conclusion, well-planned anesthesia and surgery are essential in this difficult group of patients if a good outcome is to be expected. This outcome can be achieved only by good communication and cooperation between the surgeon and the anesthesiologist.