Chapter 10
ANESTHESIA FOR NEUROSURGICAL PROCEDURES IN CHILDREN

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Introduction

Recent advances in neurosurgery, neuromonitoring, and neurointensive care have dramatically improved outcomes of patients with surgical lesions of the central nervous system (CNS). To reduce morbidity and mortality, anesthetists caring for children requiring neurosurgical procedures should have a thorough knowledge of neuroanatomy, neurophysiology, normal cognitive development, and the effects of anesthetics on the developing central nervous system, the fundamental differences between children and adults, and the implications of the surgical approaches. The objective of this chapter is to review fundamental principles related to the perioperative anesthesia management of pediatric patients undergoing surgery to treat neurological pathology.1,2

Anesthetists in developing countries may not see some of the patients and procedures described in this chapter or be able to provide some of the care discussed, but by understanding the principles discussed in this chapter, they will be better able to provide better care for all of their patients, neurosurgical and others.

Neurophysiology

At birth, the central nervous system (CNS) is incompletely developed. There are significant differences in the physiology of the cerebrovascular system and in cranial bones development during different stages of development. The CNS undergoes many structural and physiological changes over the first two years of life, and these changes have major implications for the anesthetist’s preoperative evaluation and perioperative management of neurosurgical patients.3

The skull is an enclosed space that is occupied by brain tissue (80%), cerebrospinal fluid (10%), and blood (10%) in normal children. These percentages may change significantly with the presence of space-occupying lesions, such as edema, tumors, hematomas, or abscesses. According to the Monro-Kellie doctrine an increase in the volume of one intracranial compartment must be accompanied by a decrease in the volume of one or both of the other compartments to maintain normal intracranial pressure. The skulls of infants and small children can expand to accommodate changes in intracranial...
volume because their cranial sutures are open. However, this accommodation occurs slowly and does not allow immediate compensation for acute changes in volume.

*Intracranial compliance* is defined as a change in intracranial pressure (ICP) with a change in intracranial volume. When the intracranial volume is normal, the ICP is low and the brain can easily accommodate changes in intracranial volume. However, rapid increases in intracranial volume overwhelm the brain’s ability to compensate, which leads to intracranial hypertension and deterioration of the patient’s condition.\(^4\)

The fontanels and open cranial plates or open sutures of neonates and infants provide space for the cranium to expand. The mass effect of slow-growing intracranial lesions, e.g., tumors and diseases related to increased production or decreased absorption of cerebrospinal fluid (CSF), is often masked by this head enlargement. Slow increases in ICP and intracranial volume are compensated for by expansion of the cranial plates. When an infant presents with signs and symptoms of intracranial hypertension, he/she has significantly advanced disease. The true consequence of not being able to further expand the skull (volume) is a potentially lethal rise in ICP. The volume change necessary to accommodate an acute change in ICP is very limited even in presence of opened fontanels and mobile cranial plates. Furthermore, the non-elastic nature of the duramater prevents further a rapid expansion of the volume to limit the change in pressure. Once the sutures and fontanels are closed, the intracranial compliance of children is less than that of adults. The posterior fontanel usually closes at two months of age; the anterior fontanel closes a few months later.\(^5\)

The ICP of normal neonates and infants is 2-to-6mmHg. In older children, it is <15 mmHg, which is lower than that of adults. In children the choroid plexus produces 0.35 ml/min, or about 500 ml/day, of CSF, which is resorbed through arachnoid villi and ependymal lines in the ventricles. Reabsorption of CSF increases with a slow increase in ICP. However, some pathological processes may obstruct the arachnoid villi or alter CSF flow within the brain and spinal cord, and this decreases absorption of CSF. Pathological processes, such as intracranial hemorrhage, inflammation, infection, tumors or congenital malformations, may decrease CSF reabsorption. If this occurs, the increase in intracranial volume increases ICP.

The cerebral circulation is tightly regulated by a number of homeostatic mechanisms. The main factors influencing CBF are the systemic blood pressure, \(\text{CO}_2\), \(\text{O}_2\), blood viscosity, and cerebral autoregulation. Adenosine and nitric oxide also affect CBF by altering coupling between metabolism and cerebral blood flow. Cerebral blood flow is directly coupled to metabolic oxygen demand, and both increase immediately after birth. A full-term newborn will autoregulate her/his mean arterial blood pressure between 20-60 mmHg. This narrow range of autoregulation makes infants vulnerable to ischemia and intraventricular hemorrhage. Thus, it is important to monitor arterial blood pressure to detect and treat high or low pressures that could increase the risk for intraventricular hemorrhage or cerebral ischemia and increase perioperative morbidity.\(^6,7\)
In normal adults CBF is approximately 55ml/100g of tissue per minute. Almost 15% of the cardiac output goes to an organ (brain) that is only 2% of body weight. CBF in children is approximately 100ml/100g/min, which is 25% of their cardiac output. CBF in both preterm and term infants is lower, 40-50ml/100g/min. The cerebral metabolic rate for oxygen (CMRO2) is one determinant of cerebral blood flow. The CMRO2 and glucose requirements are higher in children than in adults (5.8 versus 3.5ml of oxygen/100g of brain tissue/min). The child’s metabolic rate for glucose is 6.8mg/100g of brain tissue/min and the adult’s is 5.5mg/100g of brain tissue/min.

The coupling of CMRO2 to CBF is probably mediated by the local hydrogen ion concentration in cerebral vessels. Conditions that cause acidosis and hypoxemia dilate cerebral vessels and increase CBF and cerebral volume. When autoregulation is impaired, CBF is determined by factors other than metabolic demands. If CBF exceeds metabolic requirements, hyperemia occurs. Finally, infants and children have larger heads in relation to body surface and a larger percentage of their cardiac output directed to their brains. The increased head size and increased amount of blood directed to the brain can contribute to hemodynamic instability during neurosurgery in children.

Neuropharmacology

A major goal of neurosurgical anesthesia is to ensure adequate brain and spinal cord perfusion pressures while providing adequate surgical conditions (e.g., a relaxed brain). If drugs or the anesthetic technique used during surgery are not administered appropriately, pre-existing abnormal intracranial conditions can worsen and the risk of further CNS injury will be increased. Some anesthetics and anesthetic techniques protect brains exposed to metabolic and surgical stress. Thus, knowledge of anesthetic effects on the cerebral circulation, metabolism, and intracranial pressures in normal and pathological conditions is necessary. It is also very important to know the effects of drugs and techniques on patients undergoing functional neurosurgery and minimally invasive procedures (e.g., awake craniotomy, stereotactic surgery, identification of epileptic foci, and interventional neuroradiological procedures).

Some studies suggest that the lethal dose for some medications (LD50) is significantly lower in human neonates and children than in adults. The sensitivity of infants to sedatives, hypnotics, and narcotics is significantly higher, probably due to immaturity of the central nervous system (incomplete myelination, immature blood-brain barrier) and increased permeability of the brain to some medications. The effect of inhaled anesthetics also varies with age. The minimum alveolar concentration [(MAC), i.e., the concentration of inhaled anesthetic at which 50% of patients respond to a skin incision] is much lower for neonates (0-31 days of age) than for 1-6 months old infants. Although infants have increased anesthetic requirements, their margin of safety (i.e., the difference between adequate anesthesia and profound cardiovascular depression)
is much less than in adults. Therefore, drug doses should be carefully calculated and their pharmacologic effects carefully monitored to avoid adverse effects.  

**Intravenous Anesthetics**

All intravenous anesthetics decrease CBF and CMRO\textsubscript{2}. These decreases are caused by depression of neuronal function that lessen brain metabolism. Ketamine is the only intravenous anesthetic that increases CBF, CMRO\textsubscript{2}, and ICP.

**Barbiturates**

*Barbiturates* bind to the alpha subunit of the GABA receptors, which causes sedation and amnesia. They also reduce epileptiform activity. Sodium Thiopental is a neuroprotective agent that causes a dose dependent decreases in CBF, cerebral blood volume (CBV), and CMRO\textsubscript{2}. Sodium thiopental also reduces ICP while maintaining cerebral autoregulation and reactivity of cerebral blood vessels to CO\textsubscript{2}. It also attenuates ischemia-induced release of glutamate and inhibits intracellular release of calcium, which protects the brain during hypoxia/ischemia events.

*Sulfhydryl molecules* provide additional cerebral protection by scavenging free radicals, and this reduces the extent of brain damage during focal cerebral ischemia. High, nonclinical doses (10-to-55 mg/kg) of thiopentone decreased CMRO\textsubscript{2} by 50% when used to produce an isoelectric EEG. It may not be necessary to cause complete EEG suppression to achieve neuroprotection. Barbiturates are also used to prevent increases in ICP associated with laryngoscopy and tracheal intubation. Production and absorption of CSF is unaffected by barbiturates. A significant problem when using barbiturates for cerebral protection is that they depress myocardial contractility and systemic arterial blood pressure, which decreases cerebral perfusion pressures. Clinical doses of phenobarbital decrease the size of an infarct zone in rats following focal cerebral ischemia. Barbiturates have been used to reduce ICP and provide neuroprotection in patients undergoing neurosurgical procedures if cardiovascular stability can be maintained. The slow metabolism of barbiturates causes them to accumulate in the body.

**Propofol**

*Propofol* has similar properties to barbiturates, i.e., they decrease ICP, CBF, and CMRO\textsubscript{2}. Cerebrovascular autoregulation and cerebrovascular responses to alterations in arterial blood pressure and PaCO\textsubscript{2} are preserved. The decrease in brain metabolism leads to decreases in CBF. However, several studies have shown that CBF decreases more than CMRO\textsubscript{2}, suggesting that propofol has direct vasoconstrictor effects on cerebral vessels. In some patients (e.g., Moyamoya disease), large doses of propofol could cause cerebral ischemia. In animals, propofol has antioxidant activity, activates GABA receptors, attenuates excitotoxicity-mediated glutamate release, prevents mitochondrial swelling, and has endo-cannabinoid interactions. All of these
features protect the brain. Even low doses of propofol were shown to provide brain protection.\textsuperscript{13} Patients at risk for intracranial hypertension and decreased cerebral perfusion had better brain protection with propofol than with inhaled anesthetics, at least until the dura matter was opened. Some studies suggest that prolonged administration of propofol (usually days) causes metabolic acidosis, hyperlipidemia, progressive heart failure, and death in pediatric patients (Propofol Infusion Syndrome). Therefore, prolonged use of this drug is contraindicated in children. If for some reason propofol must be used for days, the patient’s condition must be closely monitored in an intensive care unit.\textsuperscript{14}

**Benzodiazepines**

*Benzodiazepines* bind to GABA receptors to produce amnesia and anxiolysis. These drugs are said to decrease CBF by 25% and to decrease CMRO\textsubscript{2} and ICP, while elevating the seizure threshold. Flumazenil\textsuperscript{TM}, a benzodiazepine antagonist, reverses the beneficial effects of benzodiazepines on CBF, CMRO\textsubscript{2} and ICP. Consequently Flumazenil should not be given at all or should be given cautiously to patients with intracranial pathology that could increase ICP and to patients who are predisposed to seizures.\textsuperscript{15,16}

**Etomidate**

*Etomidate*, a barbiturate-like drug, progressively decreases CMRO\textsubscript{2} until an isoelectric electroencephalogram (EEG) is produced. The marked decrease in CBF suggests a direct vasoconstrictor effect on cerebral vessels. Clinical doses of Etomidate reduce CBF and CMRO\textsubscript{2} by 35-50%. By decreasing cerebral blood volume, Etomidate effectively reduces ICP. It either has no effect on cerebral perfusion pressure or increases it. CO\textsubscript{2} vascular reactivity is maintained. Despite Etomidate’s advantages, particularly its lack of cardiovascular depression, its use is limited by its capacity to suppress the adreno-cortical axis and by its ability to induce severe myoclonic activity, involuntary muscle movements, and myoclonic activity.\textsuperscript{17,18}

**Ketamine**

*Ketamine* increases CBF (60%) and CMRO\textsubscript{2}. The increased CBF may elevate ICP, especially in patients with intracranial pathology. The vasodilator effects of ketamine are due in part to its metabolic stimulant effects, direct vasodilator effects, and to cholinergic mechanisms. Although it has been suggested that ketamine has cerebral protective effects, recent studies in young animals showed significant neuronal apoptosis (cell death), even in the absence of brain injury. However, the ketamine-induced apoptosis may have been related to the high doses of drug used and the prolong duration of exposure to it in young animals. Under these experimental conditions, ketamine, other sedatives, and inhaled anesthetics are neurotoxic, but the extent of the neurotoxicity in humans is unknown.\textsuperscript{19,24}
Dexmedetomidine

*Dexmedetomidine* is a highly selective alpha-2 adrenergic agonist with good sedating, anxiolytic, and analgesic properties. Evidence suggests that it protects the heart, brain, and kidneys from ischemic and hypoxic injury.\(^{25}\) Dexmedetomidine decreases sympathetic transmission in the locus ceruleus, located in the brain stem, which produces sedation. The analgesic effects of this drug are due to activation of alpha\(_2\) adrenergic receptors located within the dorsal horn of the spinal cord. This action prevents the release of substance P.\(^{26}\) Dexmedetomidine has been used for premedication, as an anesthetic adjunct, and as treatment for postoperative delirium in children. Because of its moderate effects on the cardiovascular system, it is widely used for analgesia in pediatric intensive care units. There is evidence that it protects the developing brain.\(^{27,28}\)

The cardiovascular effects of dexmedetomidine are mediated by central and peripheral nervous system adrenergic receptors. Small doses of this drug suppress the sympathetic nervous system, which decreases arterial blood pressure and heart rate. Hypotension and bradycardia have been reported when a loading dose of dexmedetomidine is given to children. Infusing the drug at 0.3-to-0.7 mcg/kg/h reduces the unwanted side effects. A great advantage of dexmedetomidine is its ability to maintain spontaneous ventilation and airway reflexes, even when high doses are used.

Dexmedetomidine decreases CBF and CMRO\(_2\) by similar amounts in adults. It has no effect on ICP, CSF pressure, CPP, or on the reactivity of cerebral vessels to CO\(_2\). Data suggest that dexmedetomidine provides brain protection during cerebral ischemia\(^{29}\) in the developing brain of animals by blocking activation of the pro-apoptosis caspase-3 and by expressing tyrosine kinase, which is important for cellular plasticity.\(^{30}\)

Dexmedetomidine preserves motor and sensory evoked potentials, making it a good drug for surgeries requiring motor and sensory monitoring. When used in combination with opioids and/or propofol, dexmedetomidine facilitates neurophysiological monitoring for scoliosis surgery and for placement of deep brain electrodes in pediatric patients.\(^{31,32}\) Because sedation with dexmedetomidine acts through adrenoreceptors in the locus ceruleus, effects that mimic normal sleep, the drug is particularly useful for sedating children requiring EEG studies.

Localized tumor or seizure focus resection near areas important for language or movement (motor cortex) requires patient cooperation during surgery. Many drugs besides dexmedetomidine have been used to allow proper intraoperative monitoring, however, most cause respiratory depression, airway obstruction, hemodynamic instability, vomiting, dysinhibition of the CNS, and pain. Because it produces sedation, anxiolysis and analgesia without causing respiratory depression, dexmedetomidine is commonly used for awake craniotomy in older children and adolescents.\(^{33,37}\)
**Inhalational Anesthetics**

*Volatile anesthetics* dilate cerebral vessels and increases CBF. Their effect on the ratio of CBF/CMRO₂ could increase brain volume and ICP. Volatile anesthetics cause more cerebral vasodilation in children than adults. They increase CBF in the following order: halothane > desflurane > isoflurane > sevoflurane.³⁸

In children, halothane-induced increases in CBF persist despite reducing or discontinuing the drug. This phenomenon (i.e., cerebrovascular hysteresis) does not occur with isoflurane. At equipotent doses, isoflurane and sevoflurane decrease CMRO₂ less than halothane. Desflurane is the most powerful cerebral vasodilator of the modern inhalational anesthetics, while sevoflurane has the least effects on CBF and CBV in both adults and children.

Below 1.5 MAC, sevoflurane has little effect on the CBF of children and adults. Cerebral autoregulation is maintained at sevoflurane concentrations below 1 MAC. Cerebrovascular reactivity to CO₂ is also maintained. The cerebrovascular response to CO₂ is lost in children when their PaCO₂ exceeds 45-50mmHg. This does not occur in adults. Despite lower blood gas partition coefficient and more rapid recovery from general anesthesia, the effects of desflurane on the cerebral vasculature make it a less desirable agent for neurosurgery than either sevoflurane or isoflurane, especially for patients who are neurologically compromised.³⁹,⁴⁰

**Nitrous Oxide**

*Nitrous oxide* (N₂O) dilates the cerebral vessels of both adults and children when used alone or in combination with halogenated anesthetic agents or propofol. It increases cerebral blood flow in gray matter, especially in the supratentorial region. The exact mechanism for this increase is unknown; however, there is evidence that it is mediated by activation of ischemic mitochondria and by adrenal sympathetic stimulation. Cerebral autoregulation is affected when N₂O is used alone or in combination with sevoflurane. In conclusion, N₂O alters cerebral autoregulation, cerebrovascular reactivity to CO₂, increases CBF and CMRO₂, and increases ICP while decreasing the threshold for ischemia in infants and children. It is recommended that N₂O not be used for patients at risk for having abnormal cerebral perfusion pressures and neuronal ischemia.⁴¹,⁴²

**Opioids**

*Opioids* are thought to have few or no effects on CBF, CMRO₂, and ICP. Cerebral vascular reactivity to CO₂ and cerebral autoregulation are preserved. By blocking pain-induced release of catecholamines, opioids may indirectly decrease CBF. Opioids prevent hemodynamic responses during direct laryngoscopy, especially in patients with increased ICP or cerebrovascular disease. Cerebrovascular CO₂ reactivity and cerebral autoregulation are normal in patients given fentanyl. Fentanyl has no effect on CSF production, but it reduces reabsorption of by 50%.
Remifentanyl is an ultra short acting synthetic opioid that shares some similarities with fentanyl and alfentanyl, however its analgesic effect is about 65 times that of alfentanyl. The extremely short half-life (3-5 min) and rapid recovery after remifentanyl makes it ideal for use in neurosurgical patients. Its metabolism by plasma and tissue esterases is independent of renal and liver function. Remifentanyl decreases the CMRO$_2$ of both animals and humans. Even very high doses (over 3 mcg/kg/min; clinical dose 0.1 to 0.5mcg/kg/min) of remifentanyl have no effect on CMRO$_2$, making remifentanyl useful in neurosurgical patients.

In humans, infusion of remifentanil increases regional blood flow in a dose-dependent fashion in areas of the brain that process pain. Low-dose remifentanil (0.05mcg/kg/min) significantly increases regional CBF. Several animals and human studies found no effect of remifentanil on CSF production or its reabsorption. Cerebrovascular reactivity to CO$_2$ is preserved. Morphine and fentanyl, but not remifentanil, disrupt cholinergic neurotransmission. This causes postoperative delirium and impaired memory. Both mean arterial pressure and heart rate are reduced by propofol-remifentanil anesthesia without causing any effects on CBF, implying that cerebral blood volume (CBV) and autoregulation are preserved.

Minimally invasive neurosurgical techniques and functional procedures are now common. Anesthetics that maintain adequate brain relaxation, reduce interference with electrophysiological monitoring, ensure rapid neurological recovery after surgery, and provide neuroprotection are needed. Remifentanyl meets all these requirements. Remifentanil plus an intravenous hypnotic or volatile anesthetic are commonly used. Quick recovery from anesthesia is one of the advantages of using remifentanil during neurosurgery. This allows rapid neurologic evaluation of the patient following surgery, more rapid tracheal extubation, and a better state of consciousness.

Although no single anesthetic agent meets all neurosurgical anesthesia objectives, attempts have been made to identify an anesthetic or anesthetic technique that maintains coupling between CBF and oxygen metabolism, maintains cerebrovascular autoregulation, and does not increase CBV and ICP. Propofol and remifentanil are commonly used for these reasons, especially in patients who have intracranial hypertension. However, as with all patients with intracranial hypertension, a normal or slightly low PaCO$_2$ must be maintained. In patients without evidence of intracranial hypertension, sevoflurane-remifentanil is a good alternative to the use of propofol. Patients with space-occupying lesions, increased ICP, and decreased intracranial compliance do well with a propofol-based anesthetic.
Depolarizing Neuromuscular Relaxants

Succinylcholine

*Succinylcholine* is the only easily available nondepolarizing muscle relaxant. Whether there is an intracranial space-occupying lesion or not, it increases the ICP of both animals and humans. This increase has been associated with muscle fasciculation, increased activity of afferent fibers on muscles, and increased cerebral blood flow. Neck muscles contractions compress the jugular veins and this is thought to increase ICP. The increase in ICP can be prevented or diminished by prior administration of small doses of a non-depolarizing muscle relaxant. Succinylcholine has caused life-threatening hyperkalemia, especially in patients with subarachnoid hemorrhage, traumatic brain injury, cerebral hypoxia, stroke, and paraplegia.

Non-depolarizing Neuromuscular Relaxants

Some muscle relaxants and their metabolites affect cerebral circulation through histamine release. Clinical doses for atracurium appear to have significant effects on CBF, CMRO₂, and ICP. Laudanosine, a metabolite of atracurium, easily crosses the blood-brain barrier and may induce seizures in seizure-prone patients. Seizures seldom occur in other patients. Cisatracurium releases less laudanosine and histamine than atracurium and has weaker CNS effects than atracurium.⁵³ Pancuronium, vecuronium and rocuronium have minimal effects on CBF, CMRO₂, and ICP. The increase in blood pressure and heart rate produced by these drugs might further increase the ICP of patients who have intracranial hypertension, especially if they have disordered cerebral autoregulation. Vecuronium does not induce histamine release or change heart rate and blood pressure. Because rocuronium has a rapid onset of action and none of succinylcholine’s adverse effects, it is often used for rapid sequence induction of anesthesia and tracheal intubation. Like other neuromuscular relaxants, rocuronium has no effect on CBF.⁵⁴,⁵⁵

Preoperative Evaluation

Complete assessment and preparation of the pediatric patient for anesthesia and neurosurgery are essential to minimize perioperative morbidity and mortality (See Chapter 1). Preoperative evaluation of neurosurgical patients should include a complete review of the patient’s medical conditions and the conditions for which the procedure is being done.⁵⁶ *(Table 10-1). Table 10-2* gives some of the special concerns for pediatric patients with neurologic problems. The cornerstone for evaluation of brain function remains clinical history and physical examination.
### TABLE 10-1: General Perioperative Concerns in Infants and Children

<table>
<thead>
<tr>
<th>Condition</th>
<th>Anesthetic Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prematurity</td>
<td>Postoperative apnea</td>
</tr>
<tr>
<td>Congenital heart Disease</td>
<td>Hypoxia, arrhythmia, cardiovascular</td>
</tr>
<tr>
<td>instability, paradoxical air embolism</td>
<td></td>
</tr>
<tr>
<td>Gastrointestinal reflux</td>
<td>Aspiration pneumonia</td>
</tr>
<tr>
<td>Upper respiratory tract infection</td>
<td>Laryngospasm, bronchospasm,</td>
</tr>
<tr>
<td>hypoxia, pneumonia</td>
<td></td>
</tr>
<tr>
<td>Craniofacial abnormality</td>
<td>Difficulty with airway management</td>
</tr>
</tbody>
</table>
### TABLE 10-2: Common Perioperative Concerns for Infants and Children with Neurological Lesions

<table>
<thead>
<tr>
<th>Condition</th>
<th>Anesthetic Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Denervation Injuries</td>
<td>Hyperkalemia after succinylcholine</td>
</tr>
<tr>
<td></td>
<td>Resistance: nondepolarizing muscle relaxants</td>
</tr>
<tr>
<td></td>
<td>Abnormal: response to nerve stimulation</td>
</tr>
<tr>
<td>Chronic anticonvulsant therapy</td>
<td>Hepatic and hematologic abnormalities</td>
</tr>
<tr>
<td>Arteriovenous malformation</td>
<td>Potential congestive heart failure</td>
</tr>
<tr>
<td>Neuromuscular disease</td>
<td>Malignant hyperthermia, respiratory failure</td>
</tr>
<tr>
<td></td>
<td>Sudden cardiac death</td>
</tr>
<tr>
<td>Arnold-Chiari malformation</td>
<td>Apnea, Aspiration pneumonia</td>
</tr>
<tr>
<td>Hypothalamic / pituitary lesions</td>
<td>Diabetes insipidus, Hypothyroidism</td>
</tr>
<tr>
<td></td>
<td>Adrenal insufficiency</td>
</tr>
</tbody>
</table>
Preoperative Evaluation of the Neurosurgical Patient Includes:

1) Evaluation of the patient’s neurological status includes looking for the presence of intracranial hypertension and abnormal vital signs, assessing the respiratory and cardiovascular systems and looking for abnormalities in these systems that can affect neurological processes in the brain and spinal cord, assessing the level of consciousness, and evaluating motor and sensory neurological deficit and cranial nerve involvement.

2) Determination of the presence of raised ICP and associated neurological deficits. (Table 10-3) The presentation of patients with increased ICP varies, depending on the duration of ICP elevation. A sudden increase in ICP frequently leads to coma, whereas in less acute raises does not. Patients who awaken with a headache each morning may have hypercapnia during sleep, increased CBV, and decreased intracranial compliance. Neonates and infants who have increased ICP usually have a history of irritability, decreased appetite, and lethargy. Bulging anterior fontanels, dilated skull veins, and an enlarged deformed skull are common signs of increased ICP in neonates and infants. Other signs include double vision (diplopia) caused by oculomotor palsy (rising sun sign) and strabismus (cranial nerve VI palsy). Older children who have increased ICP often vomit in the morning. Papilledema and absent venous pulsation are seen in the eyes when the ICP is elevated.

3) Detection of electrolyte disturbances. Electrolyte disturbances can occur with decreased consciousness, vomiting, bulbar dysfunction, use of osmotic or loop diuretics to lower ICP, and prolonged fasting. Inappropriate secretion of antidiuretic hormone, the brain-induced salt loosing syndrome, and diabetes insipidus may cause disordered sodium and water balance.

4) Detection of hyperglycemia. Hyperglycemia is common in patients with neurologic diseases, especially those who are treated with steroids. Hyperglycemia often causes osmotic diuresis, hypovolemia, and electrolyte disturbances. Patients with a craniopharyngioma frequently have pituitary dysfunction. Proper pre-surgical evaluation of their endocrine system is mandatory.

5) Detection of allergies to foods, drugs, or radiological contrast material. The presence of a latex allergy must be sought, particularly in patients who have a myelomeningocele or others who require regular bladder catheterization.

6) Detection of toxicity to chronic anticonvulsant use. Patients with seizures are often on high doses of anticonvulsants that may cause hematologic disorders, liver dysfunction, or both. Children on chronic seizure therapy often require larger doses of sedatives, nondepolarizing muscle relaxants, and opioids because their anti-seizure medications have increased the enzymes that metabolize these drugs.57

7) Evaluation of X-rays, MRIs, and brain computed tomography (CT) studies. All of these studies must be reviewed before surgery to confirm the location of the primary lesion, the
presence or absence of hydrocephalus, compression, cerebral edema, and the anticipated patient position required for surgery. Laboratory tests should be based on the patient's condition and the proposed surgical procedure. Due to the risk for massive bleeding during neurosurgery, the anesthetist should evaluate the patient's hemoglobin (Hb.) concentration, hematocrit (Hct), platelet count, partial thromboplastin time (PTT), prothrombin time (PT), and fibrinogen concentration and should make sure there will be sufficient blood available when needed.

8) Assessment of the airway. It is of great importance that the airway be evaluated thoroughly because some patients with neurological diseases, mainly those with craniofacial malformations, have difficult airways (See Chapter 7). It is vitally important that these abnormalities are recognized and planned for preoperatively.

<table>
<thead>
<tr>
<th>TABLE 10-3: Signs of Intracranial Hypertension in Infants and Children</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Infants</strong></td>
</tr>
<tr>
<td>Irritability</td>
</tr>
<tr>
<td>Full Fontanels</td>
</tr>
<tr>
<td>Widely separated cranial sutures</td>
</tr>
<tr>
<td>Cranial enlargement</td>
</tr>
<tr>
<td>Pupillary changes</td>
</tr>
</tbody>
</table>

**Premedication**

Premedication of patients undergoing neurosurgery can cause respiratory depression, hypercapnia, loss of protective airway reflexes, and sudden increases in ICP that can alter the patient's state of consciousness, hemodynamic stability, and can increase her/his risk for death. Therefore, preoperative sedatives and narcotics should be avoided in these patients unless the drugs are given in a monitored environment under the supervision of an anesthetist.

Patients with normal ICPs scheduled for endovascular procedures are often sedated to reduce anxiety, prevent systemic hypertension, and prevent rupture of intracranial vascular lesions. Oral midazolam (0.5mg/kg) is particularly useful for relieving anxiety, producing amnesia, and reducing the risk for seizures in some neurosurgical patients. The taste of this bitter drug must be hidden in a sweet solution (e.g., 5ml of Coca Cola). Emotional preparation of the patient, when properly done, is often the only “premedication” needed for children who are older than six years of age.

This type of “premedication” helps reduce preoperative anxiety and helps avoid potentially deleterious intracranial cerebrovascular changes that raise ICP.
Perioperative Considerations

Knowledge of potential complications of neurosurgical procedures allows the anesthetist to develop strategies to prevent these complications and to initiate prompt treatment for them if they occur. Both early recognition and early treatment of complications reduces the morbidity and mortality related to these procedures. Factors such as emergency surgery, severe co-morbidities, age (prematurity), complications with sedation, increased difficulty in placing vascular accesses, massive bleeding, and massive transfusion are potential complications that increase the morbidity and mortality of infants and children.58-61

The goal of neuroanesthesia is to ensure that the child has adequate cerebral perfusion pressure and that the ICP does not increase during induction of anesthesia by preventing hypoxemia, hypercapnia, and hypertension. An understanding of the patient’s preoperative status, coexisting conditions, and the presence of an elevated ICP allow the anesthetist to choose appropriate drugs for induction and maintenance of anesthesia. IV induction with thiopental 5-8 mg/kg or propofol 2-4 mg/kg, an opioid (fentanyl and remifentanil), and a short acting neuromuscular relaxant (succinylcholine, rocuronium), facilitates rapid tracheal intubation of children who have intracranial hypertension and are at risk for pulmonary aspiration. If the use of succinylcholine is contraindicated by its effects on CBF and ICP or the patient has a spinal cord injury, prolonged standing, burns, and subacute paretic limbs, rocuronium is often used rather than succinylcholine. Patients with the problems listed above can develop sudden hyperkalemia and die when given succinylcholine.

Children without an elevation of ICP and no intravenous access or those in whom obtaining IV access will be difficult can have anesthesia induced with sevoflurane and oxygen while ventilation is controlled to prevent unwanted increases in CO₂ and ICP. After placement of an IV, sodium thiopental or propofol can be given to prevent the increases in ICP associated with laryngoscopy and tracheal intubation. During the induction of anesthesia, moderate hyperventilation (PCO₂ 30) via facemask reduces the increases of ICP produced by inhaled anesthetics, the administration of opioids and hypnotic agents, and by laryngoscopy and tracheal intubation. However, sevoflurane and hyperventilation can induce seizures, particularly in children with know epilepsy. At times sevoflurane can cause seizures in children who have no history of seizures.62 Ketamine should not be used to induce anesthesia because of its detrimental effects on CBF, CMRO₂ and ICP.

Airway Management

Developmental changes in the pediatric airway have significant impact on anesthetic management. Because the trachea is relatively short, the tracheal tube can easily migrate into the right bronchus when the head is flexed, especially when the child is placed in the prone position with her/his neck flexed. Extension of the head may cause accidental tracheal extubation. Care should be taken to secure the tracheal tube to the patient’s face. Nasotracheal
Intubation may provide greater stability if the tube is fixed in place by placing a suture through the columella and the edge of the tracheal tube and then tying the suture around the tube. Oral tubes can be sutured to the teeth. Nasal tubes may be more comfortable after surgery for patients requiring postoperative tracheal intubation and mechanical ventilation. Nasotracheal intubation is used for surgery done in the prone position, when the airway will be inaccessible during surgery, and when the patient is very young. Contraindications for nasal intubation include choanal stenosis, procedures to repair basal skull fractures, and sinusitis. Tracheal tubes can bend and obstruct when they curve around the base of the tongue. Use of a pre-formed tracheal tube (e.g., RAE) prevents this kinking.

**Patient Position During Surgery**

Part of the preparation for anesthesia includes preparing the operating table and having appropriate equipment to prevent injury to the patient induced by the positioning during surgery. Artificial tears/ointment should be placed in the eyes before sealing the eyelids closed with a suture or tape. Pressure on the globe during surgery can cause retinal ischemia and postoperative blindness. Furthermore, in prone position the face and other sensitive areas must be padded to prevent injury from excessive pressure. To avoid compromising ventilation in the prone position, a “U-shaped” bolster is placed under the child to allow free movement of the abdomen and diaphragm. In smaller patients, small rolls can be placed under the upper chest and the pelvis. Placing a roll under the patient’s abdomen would increase intra-abdominal pressure, compress the inferior vena cava and epidural veins, and increase bleeding during spinal surgery. (Figure 10-1)

**Figure 10-1: Prone Position for Spinal Tumor Resection.**

Elevating the head 10 degrees improves cerebral venous return and reduces venous congestion. This reduces sagittal sinus pressure, which may increase the risk for venous air embolism after the
skull is opened. Rotating the head to the side compress the jugular veins, reduces venous return, and increases ICP. If the head must be rotated, rotating the patient’s trunk to maintain axial alignment can prevent obstruction of venous return.

During any surgical procedure, it is important for the anesthetist to maintain access to the tracheal tube, connections to the tube, and the anesthetic circuit so he/she can be easily inspect them. Furthermore, it is desirable to have a hand or foot visible during surgery to assess peripheral perfusion, skin color, and neuromuscular relaxation. Significant facial and airway edema occur in patients who are placed in the prone position for long periods of time and/or are given large volumes of IV fluids. If significant facial edema occurs, it is advisable to leave the child’s trachea intubated during the immediate postoperative period and wait for the edema to subside. Postoperative blindness has occurred during spinal surgery done in the prone position. The usual causes are blood loss (hypovolemia) or pressure on the eye. Pressure of the eyeball, anemia, and systemic hypotension must be avoided if blindness is to be prevented.63,64

Vascular Access and Hemodynamic Monitoring

Because of the risk for massive intraoperative bleeding and because it may be difficult obtain additional vascular access during neurosurgical procedures, two large peripheral venous catheters should be inserted before the patient is positioned for surgery. In small infants, blood transfusion should be initiated sooner rather than later because it is so easy to fall behind on volume. If possible, blood should be brought to the operating room and stored in an ice chest before the procedure begins. Blood transfusion should be performed via a peripheral IV when possible and not via a central venous catheter to reduce the risk of potassium induced cardiac arrhythmias.

Monitoring during anesthesia includes a precordial or esophageal stethoscope, ECG, noninvasive blood pressure, pulse oximetry, capnography and temperature. Urine output should be monitored during lengthy procedures, especially when osmotic diuretics are used.65 Patients scheduled for major craniotomies and spine surgery are at risk for hemorrhage and hemodynamic instability cause by air embolism, manipulation of cranial nerves, and herniation of the brain. The high risk for cerebral injury during neurosurgery justifies inserting an arterial line (See Chapter 2) to monitor the patient’s hemodynamic status, intravascular volume, acid-base status, electrolytes, blood sugar and lactic acid concentrations, and hematocrit. Increase variability of the arterial pressure waveform with positive pressure ventilation is an excellent indicator of intravascular volume deficit.

Arterial catheters are often percutaneously placed into a radial, posterior tibial, or femoral artery.66,67 The utility of central venous access is controversial in pediatric patients. However, its use should be considered when intravenous access is difficult, when assessment of right ventricular filling pressures might be helpful, and when it may be necessary to infuse inotropes or vasopressors during surgery. Central venous catheters are most often inserted into the
subclavian and femoral veins (See Chapter 2). Femoral vein catheters are usually more easily available to the anesthetist during the surgery, but they should be removed as soon after surgery as possible to avoid complications from their use. The internal jugular route is seldom for insertion of central lines in patients undergoing neurosurgery because they compromise cerebral venous return, especially in young children. In patients undergoing neurosurgery, a precordial Doppler should be used routinely, along with capnography and an intra-arterial catheter, to detect air embolism before hemodynamic instability develops.68,69

Neurophysiological Monitoring

Recent advances in neurophysiological monitoring have improved the safety of brain and spinal cord surgery. In developed countries, intraoperative neurophysiological monitoring (IONM) is used for most spinal and cranial surgeries. IONM provides the surgeon with valuable information about the integrity of the spinal cord, nerve roots, and peripheral nerves during critical moments in the surgery.70

Preoperative evaluation should include deciding which anesthetics are appropriate for the type of neurophysiological monitoring that will be used. Some intravenous and inhaled anesthetics have effects on intraoperative neurophysiologic recordings. Neurosurgical procedures that benefit the most from IONM are procedures involving the corticospinal tracts, dorsal columns, and the cranial nerves and nerve roots. These include surgery for anterior and posterior spinal fusion, release of a tethered spinal cord, dorsal rhizotomy, craniotomies for tumor resection, and posterior fossa decompression. (Figures 10-2A-2B)
Figure 10-2 A: (Above) Preparation for Neurophysiological Monitoring in Spinal Tumor Resection. Figure 10-2 B: (Below) Spinal Tumor Resection
Monitors used during neurosurgery include electromyography (EMG), somatosensory evoked potentials (SSEP), motor evoked potentials (MEP), electroencephalography, electrocortigraphy (EEG-EcoG), auditory evoked potentials of the brain stem (PEA), and EMG for monitoring cranial nerves VII, IX, X, XI, XII. Cerebral oximetry (NIRS), EEG, jugular bulb catheters, and transcranial Doppler have been used to monitor cerebral oxygenation during surgery.\textsuperscript{71-73}
Electromyography (EMG)

During surgery, the EMG is often used to monitor activity of muscles. This type of monitoring provides real-time information on the state of conduction through nerve roots and peripheral nerves. Peripheral nerves can be irritated by surgical manipulation, inadvertent retraction or compression of nerves or spinal cord, stretching of a nerve, or by ischemia from inappropriate positioning of the patient for surgery. This can be detected by EMG monitoring. Muscle relaxants should not be used during EMG monitoring because they prevent muscles from responding to normal signals from nerves. Facial muscles innervated by the seventh nerve are especially sensitive to neuromuscular relaxants.

Somatosensory Evoked Potential (SSEP)

SSEPs are generated by action potentials in the nuclei of the central nervous system and by primary sensory cortical neurons in response to peripheral nerve stimulation. The nerves most often stimulated include the median or ulnar for the upper limbs and the posterior tibial nerve for the lower extremities. The signal generated travels via the stimulated ipsilateral peripheral nerve and ascends to the cuneiform nuclei (upper limb) or graciles nuclei (lower extremities and trunk) of the dorsal spine. From there the signal crosses to the contralateral side and travels to the sensory cortex via the thalamus.

SEPs provide valuable information about sensory tract integrity from the peripheral nerve to the sensory cortex. The amplitude and latency of the waves produced by the electrical stimulus must be evaluated before surgery to determine whether the signal is normal or abnormal. During surgery SSEPs are compared with the signals obtained prior to surgery to detect acute intraoperative changes. The surgeon and anesthetist should be notified immediately of a greater than 50% decrease in signal amplitude or of a 10% increase in conduction latency from pre-incision values.

Blunt trauma of the spinal cord typically causes immediate changes in SSEPs, whereas with spinal cord ischemia the changes to occur later. Acute loss of SSEPs indicates loss of neurological function and requires immediate action by the surgical team. Blood loss that is sufficient to reduce blood flow to the cuneiform and gracilis nuclei, thalamus, or primary sensory cortex alters SSEPs. SSEPs disappear when the cortical blood flow is below 15-18ml/100g /min. It is important to monitor the effects of patient position on SEPs prior to surgery because nerve compression or tissue ischemia affects the signals. Decreased ulnar SSEPs are often seen during traction on the brachial plexus. Changes in SSEPs should never be ignored because they may indicate nerve injury and a potential for increased postoperative morbidity. Over time, prolonged infusion of propofol can lead to gradual decreases in SSEPs as propofol accumulates in the tissues. The effects of anesthetics should always be distinguished from pathological effects. If the anesthetist is familiar with the pharmacokinetics of drugs used during neurosurgery, he/she can adjust the
infusion rates (doses) of the drugs during long procedures to reduce unwanted complications and to shorten wake up times. Neuromuscular blocking drugs do not directly influence the SSEP. In fact, they may improve the quality of SSEPs by eliminating muscle artifacts. Inhalation anesthetics reduce the amplitude of SSEPs.

**Motorsensory Evoked Potentials (MEP)**

The major drawback to SSEP monitoring is that it only evaluates the integrity of the ventral motor portion of the spinal cord cortical tracts. MEPs are acquired during surgery by applying a high voltage, short duration stimulus to the motor cortex via the scalp. The amplitude, latency, and morphology of the signal are used to assess the integrity of motor conduction. In general, all inhalational anesthetic agents, except desflurane, have significant negative impact on MEPs, even at low doses. Total intravenous anesthesia (TIVA) with propofol and remifentanil reduces or prevents alterations of MEPs. Muscle relaxants block MEPs and should not be used when MEPs are being monitored. A history of seizures is a relative contraindication to the use of MEPs because the electrical stimulation used to test for MEPs can induce intraoperative seizures that might go unnoticed. If this happened, detrimental effects on the brain might occur. Ventriculoperitoneal shunts, skull fractures, and implanted metallic devices can affect placement of scalp needles and interfere with the electrical stimulation and monitoring of the MEPs. The presence of a cochlear implant is an absolute contraindication to the use of MEPs.

**Electroencephalography (EEG)**

EEGs are used intraoperatively to evaluate depth of anesthesia, adequacy of the cerebral regional and global perfusion, assess electrical patterns following cortical stimulation, and determine the presence of seizure activity. Continuous EEG monitoring can detect cerebral ischemia. The EEG pattern deteriorates when CBF is <18ml/100g of brain/min or approximately 40% of normal. During surgery for MoyaMoya disease, EEG monitoring is used to determine which PaCO₂ provides the best CBF. It is also used to determine the adequacy of blood volume and the effects of temperature on the balance oxygen of supply and demand. EEG monitoring is also used in these patients to determine the onset of ischemia.

EEG Monitoring is also helpful during repair of cerebral aneurysms and arteriovenous malformations. A decrease in CBF is accompanied by a decrease in EEG amplitude. In the absence of medications that potentially produce an isoelectric signal, the presence of an isoelectric EEG suggests brain death. Asymmetry in the amplitude of the EEG between the right and left hemispheres indicates an imbalance in the CBF between the two hemispheres, possibly due to an hematoma or ipsilateral vascular insufficiency.
Auditory Evoked Potentials (AEP)

AEPs are divided into short latency, average latency, and long latency signals. During surgery short-latency responses are resistant to the effects of anesthetics; the others are not. AEPs are used to assess the integrity of the eighth cranial nerve or the ascending auditory pathway up to the level of the inferior colliculus. AEPs are also useful during resection of acoustic neuromas, meningiomas, or pontomedullary tumors.75

Spinal fusion surgery to correct scoliosis is perhaps the most common use of IONM in children. Before IONM was available, the wake-up test was used intraoperatively to determine if the motor system was intact. This test has been abandoned where IONM is available because many patients woke up with nerve deficits despite a normal wake up test deficits during surgery. Other complications occurred, including inadvertent tracheal extubation in prone position, accidental removal of the venous catheters and arterial line, and an increased risk for neurological injury. The problem with this test was that it only provided valuable information during the short period when the patient was awake. No information is provided when the patient was anesthetized.

Acute complete loss of SSEPs or MEPs during spinal surgery is a major concern for the surgeons because it indicates trauma to the spinal cord. This injury may be the result of direct nerve injury, vascular insufficiency, or vascular ischemia from systemic or local hypotension. The loss of MEPs or SSEPs should trigger an immediate response by the anesthetist and surgeon to restore spinal cord perfusion as quickly as possible by raising the perfusion pressure.

The anesthetist’s response should include increasing the inspired oxygen concentration when possible, decreasing the amount of anesthetic being administered, and increasing intravascular volume with boluses of crystalloid fluids or RBCs. Vasopressor or inotropic drugs are used to increase the perfusion pressure when the above measures fail to correct the problem. The surgeon must look for surgical causes for the altered MEPs and/or SSEPs. If the MEPs or SSEPs cannot be restored within 15 minutes, it is essential to undo the surgical correction and prepare to awaken the patient to determine if spinal cord damage has occurred. If awakening the patient confirms the findings of the MEPs and SSEPs, a spinal cord injury protocol should be initiated immediately to prevent further cord damage and restore function to the spinal cord. Several institutions have recommended quickly giving a bolus of methylprednisolone 30mg/kg followed by a continuous infusion of 5.4mg/kg/hour of methylprednisolone for the next 24 hours. Methylprednisolone stabilizes cell membranes by decreasing the release of free radicals during ischemia, which decreases the risk of neuronal injury during spinal cord reperfusion. Hypothermia, hypotension, hypoxemia and hypocapnia also affect neuromonitoring.76
Maintenance of Anesthesia

General anesthesia is maintained with inhaled or intravenous anesthetics or with both. The ideal medications for this purpose would decrease CMRO$_2$ and ICP while maintaining normal CBF. Remember, all anesthesia agents are vasodilators and have the potential for increasing CBF, CBV and ICP in patients with decreased intracranial compliance. Low concentrations of isoflurane or sevoflurane (less than 1 MAC), when combined with a continuous infusion of fentanyl or remifentanil and sufficient positive pressure ventilation to maintain normocapnia have minimal effects on CBF and ICP. N$_2$O is usually not used for maintenance anesthesia during neurosurgery because of its detrimental effects on CMRO$_2$, CBF, ICP and its increased risk of causing postoperative nausea and vomiting. Moreover, N$_2$O has unwanted effects on SSEPs and MEPs. Neuromuscular blocking agents should be used (when appropriate) during the perioperative period to prevent inadvertent movement and occasionally fatal neurological consequences. Chronic preoperative use of anticonvulsants increases the doses of muscle relaxants and opioids required because anticonvulsants increase activity of the liver enzymes that metabolize these drugs. When muscle relaxants cannot be used during surgery, dexmedetomidine provides effective sedation and analgesia without affecting ventilation, calm awakening from anesthesia, and neuroprotection. Consequently, dexmedetomidine is widely used during electrophysiological monitoring and awake craniotomy in children.

Fluid Management

Intraoperative fluid management has many implications for anesthetists during pediatric neurosurgical procedures. These patients experience rapid changes in intravascular volume from bleeding, administration of osmotic diuretics, or diabetes insipidus. The goals of fluid management include maintaining CPP, preserving intravascular volume, and preventing cerebral edema by maintaining an isovolemic, isotonic, and isooncotic state.

The Starling equation describes the factors that govern movement of fluids between intravascular and extracellular spaces. Unlike other tissues, the brain and spinal cord respond to changes in intravascular volume differently than most other tissues because the brain and spinal cord are isolated from the intravascular compartment by the blood brain barrier (BBB). The BBB is composed of astrocytic foot processes and endothelial cells that form tight junctions. These tight junctions limit movement of molecules between the intravascular space and the CNS. The very small pore size at these junctions (7-to-9 Å) limits free movement of many molecules, including electrolytes and proteins, into and out of the brain. The BBB acts as a semipermeable membrane. Water moves freely through this membrane in response to the relative concentrations of impermeable solutes on each side of the membrane. Glucose and amino acids require energy and transporters to cross the membrane. In muscle, lung, and other tissues, the pore size of capillary endothelium is about 65 Å, which allows many small molecules and ions (Na$^+$, Cl$^-$) to
move freely across the membrane. Large molecules, such as proteins, do not move freely. Electrolytes easily pass from the capillary lumen to the extracellular space of muscle etc. Movement of water between the intravascular and extravascular spaces of peripheral tissues is governed by the oncotic gradient created by the plasma concentration of large macromolecules.

Conversely, fluid moves in and out of the CNS through osmolar gradients that exist between plasma and extracellular fluid. These gradients are created by the relative concentrations of all osmotically active particles, including most electrolytes. These differences between the brain and other tissues explain why administering large volumes of iso-osmolar crystalloid causes peripheral edema (by dilution of plasma proteins) without increasing the amount of water in the brain or increasing ICP. Giving hypo-osmolar fluids allows more water to enter the brain. This causes cerebral edema and increases ICP. On the other hand, giving hyperosmotic fluids, such as mannitol or hypertonic saline (3%), removes water from the brain and reduces the cerebral edema, and ICP. If the BBB is intact, plasma osmolality is the major determinant of water movement between the CNS and the intravascular space.77,78

Fluid therapy for neurosurgical patients requires knowledge of three properties of blood, osmolarity, oncotic pressure, and hematocrit. Anesthetists have a variety of commercial solutions available for infusion during surgery that are best categorized by their osmolarity, dextrose content, and oncotic pressure. Normal saline is commonly used as maintenance fluid during neurosurgery because it is slightly hyperosmolar (308mOsm/L) and minimizes cerebral edema. However, hyperchloremic acidosis occurs when more than 60 ml/kg of normal saline are administered. Saline-induced hyperchloremic acidosis does not increase morbidity and mortality. In general hypo-osmolar solutions, such as Ringer's lactate solution plus dextrose, are not used during neurosurgery because they increase the amount of free water available for transport into the brain. Metabolism of the dextrose also produces free water and will directly contribute to enlarge the interstitial volume and edema.

To maintain adequate intravascular volume, cardiac output and tissue perfusion, ongoing blood loss, insensible water loss (e.g., through ventilation), and urine output should be replaced with an iso-osmolar crystalloid solution. Excessive fluid resuscitation should be avoided. There is strong evidence showing that monitoring of DYNAMIC cardiovascular variables (changes in arterial pulse pressure, changes in systolic blood pressure during positive pressure ventilation) makes it easier to maintain a stable blood volume than does monitoring STATIC hemodynamic parameters (right atrial pressure, central venous pressure).79

It is very difficult to quantify blood loss during neurosurgical procedures because much of the blood is confined to the surgical field and hidden. Furthermore, the surgeons use of large volumes of irrigating solution throughout surgery that mix in the suction with blood, making it difficult to quantitate true blood loss.
A large percentage of blood loss in pediatric patients occurs during dissection of the scalp. Infiltration of the skin and scalp with local anesthetic with epinephrine just prior to surgery reduces blood loss. The risk of significant blood loss during craniotomy and the resulting decrease in blood volume should lead anesthetists to routinely calculate each child’s predicted blood volume and determine how much blood he/she will allow the patient to lose before initiating blood transfusion. The decision to transfuse or not transfuse a patient should not only depend on the patient’s Hb. concentration and HCT but also on the patient’s hemodynamic status and the adequacy of tissue perfusion. During acute bleeding, the recommendation is to maintain a Hb. concentration of 8g/dl. More than four-month-old hemodynamically stable infants who are not actively bleeding can have an Hb concentration of 7g/dl before they are transfused. Infants <4 months of age and those with cyanotic or other congenital heart disease, chronic lung disease, and hemoglobinopathies should have their Hb. concentrations maintained at 10g/dl. When massive bleeding is anticipated, the blood bank should be consulted prior to surgery to ensure that there will be a reserve of less than two-week old blood available for the procedure. Blood older than this duration of storage time has high potassium concentrations, and rapid transfusion of this blood has caused cardiac arrests. The volume of blood to be transfused should be calculated based on the formulas given above. Each 5cc/kg of packed red blood cells (Hct 70%) raises the hemoglobin concentration by 1g. Whether to transfuse a patient or not depends on the expected blood loss during the remainder of the surgery and on the expected postoperative blood loss from drains, frequent laboratory examinations, surgical re-intervention, etc. It is safer to transfuse blood to a patient from a single donor than from multiple donors. Table 10-4 provides the steps and the objectives for management of perioperative bleeding in neurosurgical patients.

<table>
<thead>
<tr>
<th>TABLE 10-4: Steps and Goals for Perioperative Bleeding Management in Neurosurgical Patients</th>
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<tr>
<td><strong>Steps</strong></td>
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<tr>
<td><strong>Preoperative</strong></td>
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<tr>
<td>Calculate child’s blood volume in function of the age</td>
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<tr>
<td>Calculate allowable blood loss</td>
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<tr>
<td>Reserve blood products in accordance to the patient needs and the surgery</td>
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<tr>
<td>Order fresh red blood cells (maximum one or two weeks of storage)</td>
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<tr>
<td>Keep available the red blood cells in OR when risk of massive blood loss is present</td>
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TABLE 10-4: (Continued)

**Intraoperative**
- Install the hemodynamic monitoring according to the hemodynamic changes and predicted blood loss.
- Install a central vascular access according to expected blood loss.
- Secure two large bore peripheral venous access.
- Control regularly blood loss.
- Monitoring acid base, electrolyte, lactic acid, glycemia, platelets, TP, TPT, Hb, Hct.

**Goals**
- Maintain cerebral perfusion pressure and tissue perfusion within normal values.
- Maintain diuresis at 0.5-1 ml/kg/h.
- Maintain blood pH between 7, 35 and 7,45.
- Maintain calcium around 1.15 mmol/l.
- Maintain normothermia and normocapnia at all time.

Glucose-containing solutions should not be used in patients with CNS or spinal cord pathology. As stated above, metabolism of the glucose releases free water, which reduces the serum osmolality and increases the brain water content and ICP. Many studies in humans and animals have reported that glucose administration increased CNS damage and worsened focal ischemic insults by causing tissue acidosis. The mechanism by which hyperglycemia does this is believed to be through increased lactic acid production. The increased intracellular lactate and acid have neurotoxic effects that cause neuronal death and cellular necrosis. Hyperglycemia also increases glutamate release, which increases neuronal injury and worsens outcomes.85 Pediatric patients, particularly young infants, are at risk for developing hypoglycemia if they have low glycogen stores. The potential for developing hypoglycemia is increased by the fact that they have limited ability to make more glucose via gluconeogenesis. Therefore, it is often necessary to continuously infuse sufficient glucose to maintain normoglycemia. The current recommendations is to give 120mg/kg/hour or 2mg/kg/min of glucose to preterm and to term babies who had glucose infused preoperatively, to children with liver disease or metabolic disorders, to children receiving total parenteral nutrition, to malnourished children whose weights are below the 3rd percentile for age, and to children treated with beta blockers. The only way to know if a patient is normoglycemic, hypoglycemic, or hyperglycemic is to frequently measure her/his blood glucose concentration.86-90

**Management of the Intracranial Pressure**

During neurosurgical procedures cerebral edema can have devastating consequences. Causes for cerebral edema include increased permeability of the blood-brain barrier with accumulation of osmolytes in the interstitium of the brain; this causes lysis and necrosis of cells and cerebral ischemia. Hyperventilation (ETCO2 25-30mmHg) is sometimes used to prevent eminent herniation of the brain, but hyperventilation itself can also injure the brain by reducing CBF and cerebral
perfusion pressure. For every mmHg decrease in PaCO₂, brain blood flow decreases by two percent. Elevating the head 10° and using hyperosmolar therapy should be instituted early.⁹¹

Administration of hypertonic saline (3%) (3-5ml/kg bolus over 30-60 min, continuous infusion 0.1-1.0ml/kg/h) produces an osmotic gradient, which allows transfer of brain interstitial water to the intravascular space. This significantly decreases cerebral edema and ICP. Hypertonic saline is thought to also decrease production of CSF.⁹²

The beneficial effects of hypertonic saline include reduction of ICP and reduction of cerebral edema without compromising intravascular volume, arterial blood pressure, and CPP. Hypertonic saline probably reduces ICP by restoring cell membrane potentials, causing the release of atrial natriuretic peptide, by anti-inflammatory effects, and by stimulating cardiac output. Unwanted side effects include rebound increases in ICP, pontine myelinolysis, renal failure, subarachnoid hemorrhage, and hyperchloremic acidosis.⁹³, ⁹⁴ Children who received 10ml/kg of 3% saline had significant reductions in their ICP, whereas those who received the same volume of normal saline 0.9% did not.⁹⁵

Mannitol 20% (osmolality 1,098) is the osmotic diuretic most commonly used during neurosurgery. Small doses 0.25-to-0.5g/kg increase osmolarity by 10 mOsm, which is enough to reduce brain edema and ICP within 10-to-15 min. The effects of Mannitol last about two hours. Mannitol should not be given faster than 0.25-to-0.5g/kg over a period of 20-to-30 minutes to avoid hemodynamic instability. Mannitol administration has a biphasic effect on the ICP. It temporarily increases ICP by initially increasing the intravascular volume and cerebral blood flow. Water is then rapidly withdrawn form the intracellular and interstitial spaces of the brain, which reduces ICP. Administering repeated doses of mannitol has led to hyperosmolality, renal failure, and cerebral edema. Acute tubular necrosis and renal failure from high plasma osmolarity (320 mOsm) and excretion of unmetabolized mannitol in the urine have been observed in adults.⁹⁶,⁹⁷

Furosemide and ethacrynic acid (loop diuretics) cause diuresis, decreased CSF production, less cerebral edema, and improved intracellular water transport. The recommended dose of these diuretics is 0.2-to-0.3mg/kg when administered with mannitol or 0.5-to-1mg/kg when administered alone. Furosemide prevents mannitol-induced rebound cerebral edema.⁹⁸

Steroids

Corticosteroids are used in a variety of diseases in children, particularly neurological conditions, such as brain tumors. Steroids inhibit tumor-induced angiogenesis, cerebral edema, and CSF production. They also decrease the release of free radicals.
Temperature Homeostasis

Anesthesia-induced alterations of skin blood flow and exposure to a cold operating room, cause perioperative hypothermia in pediatric patients. Neonates and infants are especially prone to hypothermia due to their large surface-to-volume ratio. Hypothermia has effects on the pharmacokinetics and pharmacodynamics of many anesthetic agents. It prolongs the effects of volatile agents, intravenous anesthetics, and neuromuscular relaxants. Hypothermia increases perioperative blood loss, inhibits activation and aggregation of platelets, increases release of heparin-like anticoagulant substances, inhibits the synthesis of coagulation factors, and alters fibrinolytic activity. All these changes increase the need for blood transfusion. Other effects of hypothermia include depression of myocardial function, impaired hypoxic pulmonary vasoconstriction, increases risk of hypoglycemia, and increased infections. Hypothermia also increases oxygen consumption and pain. The patient’s temperature should be monitored during surgery and measures should be taken to keep the temperature within normal limits. During the induction of anesthesia and placement of intravenous and invasive monitoring lines, children should be covered with cotton blankets, lie on a thermal mattress (if available), have a convective air system over them when possible, be placed under a radiant heat lamp that is no closer than three feet from the surface of the patient’s skin (to avoid burns), and have warm intravenous fluids administered.99-103

Air Embolism

Air embolism occurs commonly during intracranial procedures. This is because air enters the central circulation through open veins in the scalp and skull during spontaneous breathing when the pressure inside the open veins is lower than atmospheric pressure. Placing the surgical site above the level of the heart (e.g., during a semi-sitting craniotomy for posterior fossa surgery) or having a low CVP increases the pressure gradient between the surgical site and the heart. This facilitates venous air embolism (VAE). The problem is made worse by the fact that the bony venous sinuses in the skull are held open by their attachment to bone and cannot collapse. Other possible sites for air entry include the bridging veins and epidural venous sinuses. When air enters the central circulation, blood flow in the right ventricle and pulmonary artery is blocked. This causes sudden pulmonary hypertension, decreased pulmonary artery blood flow, decreased left ventricular preload, cardiovascular collapse, and in some cases cardiac arrest. The severity of the patient’s symptoms depends on the speed with which air enters the central circulation and on the volume of gas in the heart and vessels. Intracardiac shunts (e.g., a patent foramen ovale) often allow passage of air from the venous to the arterial circulation, causing paradoxical air embolism. As little as 0.1cc of air in a coronary artery can be fatal to a young child. The incidence of air embolism during craniosynostosis surgery is high (>80%), even when the operating table is kept completely flat.104 An air embolus is more likely if there is hemorrhaging and the CVP is low. A low CVP increases the pressure gradient between veins in the head and the heart.
Echocardiography (either transthoracic or transesophageal), precordial Doppler, a pulmonary artery catheter, and capnography can be used to detect air emboli. When used, the precordial Doppler is placed in the right 4th or 5th interspace near the sternum. This device is easy to use, non-invasive, and inexpensive. Proper positioning of a Doppler is confirmed by listening for the characteristic sound of air in the venous circulation with rapid injection of a few milliliters of normal saline, which always contains micro bubbles of air. Although transesophageal or transthoracic echocardiography detects small emboli more effectively, these devices are not easy to use, the data from them is not easy to interpret during neurosurgical procedures in children, and they are very expensive. Capnography can detect air emboli, but its sensitivity is low. ECG changes, alteration in the heart rate, sudden decreases in blood pressure, and sudden increases in CVP may also suggest the presence of air embolism.

Measures to prevent air embolism include minimizing pressure gradients between the surgical site and the heart, preventing hypovolemia, and using positive pressure ventilation throughout surgery. Treatment of air emboli includes: 1) informing the surgeon immediately of the problem and having her/him flood the surgical field with saline to prevent further air entry. All exposed bony surfaces are covered with bone wax; 2) administering 100% oxygen, 3) discontinuing inhalational anesthetics to limit further cardiovascular depression; 4) placing the child in Trendelenburg position (i.e., put the surgical site below the heart), and 5) placing the child in the left lateral position to increase venous return of blood; Doing these things favors pulmonary perfusion and mobilization of air in the apex of the right ventricle, 6) withdrawing as much air as possible through the central venous catheter (if there is one), and 7) preventing further air entry by administering bolus of fluid and vasopressors to increase intravascular volume and blood pressure. If a child has a cardiac arrest in the prone position, the anesthetist should immediately begin cardiac compressions from the back and continue doing so until the patient can be turned to the supine position.

Emergence From Anesthesia

At end of surgery, tracheal extubation can be done in either the operating room or the intensive care unit, but where ever it is performed every effort should be made to prevent coughing, staining, systemic hypertension, and hypercarbia. This could potentially increase ICP, arterial blood pressure, and reduce venous return from the CNS. The tracheal tube is only removed when patients have protective airway reflexes, adequate spontaneous breathing, and are fully alert (to protect the airway). However, if these criteria are not met in the operating room or if there is a high likelihood for postoperative intracranial hypertension, the tracheal tube should be left in place and the patient taken to the intensive care unit for monitoring and support of ventilation.

The incidence of nausea and vomiting in neurosurgery patients is high, due to the irritant effect of blood on the ventricles and to the use of opioids. Antiemetic drugs should be given before the
end of surgery to prevent vomiting. The effects of muscle relaxants should be reversed at the end of surgery because their residual effect will decrease ventilation, cause hypercapnia, and interfere with the postoperative neurological assessment of the patient.

Pain after a craniotomy is usually not severe and can be managed with oral or intravenous acetaminophen in most cases. If the pain is moderate or severe, titrated doses of opioids are recommended. Anti-inflammatory drugs, such as dexmedetomidine, can be used to reduce pain if the drug chosen has no significant effects on platelet function. However, dexmedetomidine, by its sedative effects, may make it difficult to assess the patient’s neurologic status. Dexmedetomidine is often used during neurosurgery to provide sedation and analgesia because it does not cause respiratory depression. Giving dexmedetomidine before the end of surgery reduces the need for postoperative opioids.108

**Anesthetic Considerations for Specific Neurosurgical Procedures**

**Neural Tube Defects**

If the neural tube fails to close during fetal development, a broad range of malformations, from spina bifida to anencephaly, occur. Failure of the vertebral arches to fuse during fetal development causes spina bifida. When only a dural sac filled with cerebral spinal fluid bulges through the bony defect, it is called a meningocele. If the bulging sac also contains neural tissue, it is called a myelomeningocele. In children, these two defects are the two most common conditions in the lumbosacral area that require neurosurgical care. While both lesions can cause neurologic symptoms, meningoceles cause fewer and less severe neurologic symptoms. Paralysis, when present, is often mild and incomplete. Myelomeningoceles, on the other hand, often have severe bladder dysfunction requiring frequent bladder catheterization. They also may have severe bowel dysfunction and complete paralysis of their lower extremities. Meningoceles and myelomeningoceles require surgical correction within 24 hours of birth to minimize the risk of dural sac rupture and infection. Most patients who have a myelomeningocele have hydrocephalus 3-5 days after closure of the myelomeningocele. When this occurs, they require a ventriculoperitoneal shunt to relieve pressure in the brain. Patients with hydrocephalus may also have an Arnold Chiari malformation type II of the skull. Some myelomeningoceles are now being corrected in utero, which has the potential for reducing the incidence of hydrocephalus and the need for ventriculoperitoneal shunting.109

*Encephaloceles* are neural tube defects that usually occur on the head but can occur anywhere along the neural tract. These defects are sometimes associated with head enlargement, making ventilation with a facemask and tracheal intubation difficult and unpredictable. (Figures 10-3A-10-3B)
Anesthetic considerations for neural tube defects include:

1. Having a thorough knowledge of the general principles of managing children for anesthesia in the neonatal period, especially their fluid requirements, metabolic flux of glucose, temperature management, and the differences in their cardiovascular, respiratory, and renal systems is very important. A complete preoperative evaluation and understanding of coexisting congenital defects is mandatory. Infants are at high risk of perioperative morbidity and mortality if they were born prematurely or have congenital anomalies besides the neural tube defect. Their risk is also increased by the immaturity of most of their systems and by the narrow margin for error for administration and dilution of medications, difficulty with airway management, and intravascular access.110

2. **Positioning:** In most cases induction of anesthesia is accomplished in the supine position after the child is positioned on a u-bolster to prevent applying direct pressure to the myelomeningocele sac.

3. **Latex allergy:** Patients with a history of neural tube defects are at high risk for developing latex allergy, because of they require multiple surgical procedures, and frequent bladder catheterization with rubber catheters. Care should be taken to ensure a latex free environment in the operating room.111

Children presenting for repair of a myelomeningocele rarely have increased ICP because the dural sac is very compliant or ruptured. However, if the child has an Arnold-Chiari malformation, the intracranial cavity may be isolated from the spinal canal preventing drainage of CSF from the brain. When this occurs, patients have increased ICP. Deficits that occur at about the T-4 level often cause paraplegia.

In some infants with myelomeningocele, evaporation of water from the myelomeningocele sac is high. For this reason, the baby’s hydration status must be determined and abnormalities corrected before anesthesia is induced. Special care should be taken to ensure that blood is
available in the operating room before beginning surgery. With large lesions, there can be significant blood loss when the surgeons mobilize tissues to close the defect. When the patient is placed in the prone position, rolls are placed under the chest and hips to allow adequate excursions of the chest and abdomen during breathing and to reduce pressure on the abdomen that might compress the inferior vena cava and epidural veins and increase bleeding.

A Chiari II malformation is a boney abnormality of the posterior fossa and upper cervical spine that allows displacement of the cerebellar vermix, fourth ventricle, and brainstem through the foramen magnum. Children with this lesion can have vocal cord paralysis, stridor, respiratory distress, apnea, swallowing disorders, and aspiration of secretions. A tracheostomy and gastrostomy should be considered earlier rather than later.\textsuperscript{112} Chiari I malformations more commonly occur in healthy children without myelodysplasia. With this defect, there is caudal displacement of the cerebellar tonsils below the foramen magnum, but the clinical manifestations are usually mild and consist of headache and neck pain. Surgical treatment of a Chiari I malformation includes a decompressive craniectomy and a suboccipital laminectomy. Patients with a tethered spinal cord have tissue attachments that prevent the spinal cord from moving in the narrow spinal canal as the child grows. This condition is diagnosed clinically, or radiologically. The most common signs and symptoms of a tethered spinal cord are muscle weakness of lower limbs, sensory disturbance of the legs, bowel or bladder dysfunction, back pain, and gait disorders.\textsuperscript{113} Intraoperative release of a tethered cord requires neurophysiological monitoring when available. Direct nerve stimulation and an EMG aid the surgeon in differentiating neural tissue from other tissue. Observing the anal sphincter response or detrusor function of the bladder to stimulation improves the likelihood of preserving these functions. Muscle relaxants should not be used during tethered cord surgery because they block responses to nerve stimulation. This may cause the surgeon to inadvertently transect some nerves.

Hydrocephalus

Hydrocephalus (water on the brain) is caused by an imbalance between production and absorption of cerebrospinal fluid. This imbalance causes the ICP to rise. Hydrocephalus may be congenital or acquired. (Table 10-5) Although some reports suggest that incidence of hydrocephalus in children has decreased in some developed countries, others reports suggest it has increased due to survival of more premature babies. Neonatal infections and neural tube defects are common in some developing countries, which has significantly increased the number of patients with hydrocephalus worldwide. In East Africa there are more than 6000 new cases of hydrocephalus each year. In many low income countries, hydrocephalus is a major cause of morbidity and mortality.\textsuperscript{114} Hydrocephalus causes pathological changes in brain morphology and maturation, in the microstructure of the brain, in cerebral blood flow, in brain biochemistry, and in metabolism. (Figure 10-4) Although surgical treatment does not always reverse the damage, untreated hydrocephalus leads to progressive CNS damage and death.
TABLE 10-5: Causes of Hydrocephalus

<table>
<thead>
<tr>
<th>CONGENITAL CAUSES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myelomeningocele</td>
</tr>
<tr>
<td>Stenosis of the aqueduct of sylvius</td>
</tr>
<tr>
<td>Dandy-Walker syndrome</td>
</tr>
<tr>
<td>Mucopolysaccharidoses</td>
</tr>
<tr>
<td>X-linked hydrocephalus</td>
</tr>
<tr>
<td>In-utero intraventricular hemorrhage</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ACQUIRED CAUSES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infections</td>
</tr>
<tr>
<td>Intraventricular hemorrhage of prematurity</td>
</tr>
<tr>
<td>Space occupying intracerebral cysts</td>
</tr>
<tr>
<td>Tumors</td>
</tr>
</tbody>
</table>
Chapter 10: ANESTHESIA FOR NEUROSURGICAL PROCEDURES IN CHILDREN

Figure 10-4: Acute Hydrocephalus:

Axial CT showing significant lateral and third ventricles enlargement due to transependimal edema secondary to aqueductal stenosis.

The signs and symptoms of hydrocephalus are the result of increased ICP. Increase in ICP during acute hydrocephalus can be fatal unless someone quickly removes some of the excess CSF. The causes of acute hydrocephalus include sudden obstruction of the ventricular system (ventricular hemorrhage in premature infants, hemorrhage into a tumor, or displacement of a third ventricle cyst). In the absence of early treatment, ICP rapidly increases, causing brain herniation and cardiorespiratory arrest. Chronic hydrocephalus occurs with aqueductal stenosis, meningitis, and intracranial tumors. The clinical manifestations of chronic hydrocephalus develop more slowly and include irritability, headache, decreased school performance, confusion, and lethargy. In neonates, widening of the sutures and an increase in head circumference accommodate a slow
Anesthesia Care of Pediatric Patients (George A. Gregory & Dean B. Andropoulos)

progressive increase in ICP up to a point. The symptoms of chronic hydrocephalus in infants may be nonspecific and include irritability, decreased appetite, and vomiting. Treatment of hydrocephalus is surgical and consists of diverting CSF from the cerebral ventricles to the abdomen (ventriculoperitoneal shunt) or elsewhere. If a ventriculoperitoneal shunt cannot be placed, a ventricular-atrial shunt (lateral cerebral ventricle to jugular vein) or ventricular-pleural shunt (lateral cerebral ventricle to the pleural space) can be inserted.\textsuperscript{115} Although surgery for hydrocephalus is one of the most common neurosurgical procedures, it is occasionally associated with several catastrophic complications, including bleeding from accidental rupture of a venous sinus, a pneumothorax or hemothorax, injury to neck vessels, and injury to abdominal organs (intestinal perforation, liver laceration). During placement of a ventriculo-atrial shunt, air embolism, ventricular arrhythmias, and vessel injury can occur. A tension pneumothorax is more common with ventriculo-pleural shunts.

Endoscopic ventriculostomy is used in selected cases to create a shunt. This involves inserting a flexible endoscope into the brain and advancing it to the lateral or third ventricle. A hole is then made in the floor of the ventricle to allow CSF to drain into the basal cisterns and subarachnoid space. When the procedure is successful, there is no need for one of the other shunts. Complications of endoscopic ventriculostomy include basilar artery rupture and hypovolemic shock, which requires an urgent craniotomy. Severe bradycardia occasionally occurs during manipulation of the third ventricle and when cold saline is injected into the third ventricle.\textsuperscript{116}

The perioperative management of patients who have hydrocephalus depends on the presence or absence of intracranial hypertension, the cause of the hydrocephalus, and the presence of associated comorbidities. Preoperative sedation should be avoided in patients who have hydrocephalus because it increases the risk of respiratory depression, hypercapnia, and elevated ICPs. Patients with hydrocephalus and history of myelomeningocele must be considered to have latex allergy and should not be exposed to additional latex. Intraoperative monitoring should include non-invasive arterial blood pressure, \textit{SaO}\textsubscript{2}, ECG, ETCO\textsubscript{2}, body temperature, and continuous measurements of end-tidal inhaled anesthetic concentrations, if possible. If infants and young children have no evidence of increased ICP and have intravenous access, anesthesia can be induced with either inhaled or intravenous anesthetics. But positive pressure ventilation should be instituted as soon possible after giving the drugs used for induction of anesthesia to prevent hypercarbia, cerebral vasodilation, and increased ICP. Induction of anesthesia with inhaled agents should probably be avoided if the patient has intracranial hypertension, because inhaled agents may worsen ICP and increase vomiting and aspiration. Intravenous induction of anesthesia with propofol 3-4mg/kg or thiopental sodium 5-6mg/kg is probably safer for patients with intracranial hypertension. Opioids (fentanyl or remifentanyl) and a muscle relaxant should also be considered for rapid sequence induction of anesthesia and tracheal intubation. Maintenance of anesthesia is done with an opioid (fentanyl or remifentanyl) and sevoflurane or isoflurane. The patient is mechanically ventilated to control CO\textsubscript{2}. Postoperative management of
patients of patients with hydrocephalus depends on her/his neurological status and the presence of preexisting co-morbidities.\textsuperscript{113,117}

**Craniosynostosis**

*Craniosynostosis* is a developmental disorder of the skull that prematurely closes one or more cranial sutures. It occurs in approximately 1 in 2000 live births. In about 80\% of cases, it is an isolated anomaly. In the other 20\%, it is part of a syndrome or genetic disorder (Crouzon syndrome, Pfeiffer syndrome, Apert syndrome, Muenke, Acrocephalosyndactyla type III). These syndromes are sometimes associated with other craniofacial malformations and extracranial abnormalities. (Figure 10-5)

**Figure 10-5: Pfeiffer Syndrome.**

Diagnosis of craniosynostosis is commonly based on the phenotype of the skull deformation. Where available, CT and 3-D reconstruction are used to locate the specific suture that requires repair and to establish a surgical plan. Untreated craniosynostosis can sometimes lead to intracranial hypertension and impaired intellectual and neurological development. Patients with a syndrome and craniosynostosis often have multiple closed sutures and increased ICP. The best cosmetic and neurodevelopmental outcomes occur when surgery to correct these lesions is done during the first year of life.
Laboratory tests for this type of surgery usually include a complete blood count (hemoglobin, hematocrit and platelet count), coagulation tests (PT, PTT, INR), and the cross matching of sufficient blood for transfusion to treat perioperative bleeding if it occurs. The airway should be carefully evaluated preoperatively because some of these children have difficult airway. Each syndrome has its own set of potential complications that must be considered. For instance, children with Apert syndrome have midface hypoplasia and severe proptosis that can make facemask ventilation difficult.\textsuperscript{118,119}

When providing anesthesia for craniosynostosis, the anesthetist’s main concerns for surgery include the potential for the patient having a difficult airway, massive bleeding, and air embolism. These problems significantly increase the morbidity and mortality of the procedure.\textsuperscript{120} The main intraoperative challenge in these patients is hemorrhage, particularly if the patient is less than six months old. Blood loss varies with the number of sutures involved and the surgical technique used (open vs. endoscopic craniectomy).

During scalp dissection a patient can loose 30\% of her/his blood volume. Blood loss can be even greater when the peristium is elevated, when osteotomies are done, or when venous sinuses are damaged. Massive bleeding can occur within seconds and may be very difficulty to control. Some studies suggest starting to transfuse blood early during craniosynostosis surgery, since transfusion is almost always required for maintenance of intravascular volume and hemodynamics.\textsuperscript{121} (Figure 10-6)
Perioperative cardiac arrest in children is usually caused by massive hemorrhage and hypovolemia. The reasons for the hypovolemia include underestimation of intraoperative blood loss, inadequate venous access, inadequate monitoring, complications of blood transfusion, complications of massive transfusion (hyperkalemia, hypocalcemia), and coagulopathy. Venous air embolism (VAE) occurs in as many as 83% of patients undergoing craniosynostosis surgery, although most of these episodes have no hemodynamic consequences. VAE also occurs 8% of patients undergoing endoscopic craniosynostosis repairs. The possibility of raised ICP should...
be confirmed by CT scan, ophthalmologic examination, or by nonspecific clinical findings, such as headache in older children. Forty-seven percent of patients with syndromic craniosynostosis and multiple closed sutures have increased ICPs; the incidence is only 14% when a single suture is stenotic.

The risk of hypothermia during craniosynostosis surgery is high because the large heads of these patients are exposed to room temperatures, cerebral perfusion is high, cerebral vessels are dilated by the anesthetics, and the patients receive large volumes of cool intravenous fluids. Measures must to be taken to prevent hypothermia. The choice of drugs used for induction of anesthesia depends on the child’s condition and on her/his airway. Usually an inhaled induction with sevoflurane is appropriate if ICP is not elevated. Many anesthetists induce anesthesia with a volatile anesthetic and enough controlled ventilation to maintain a CO$_2$ of approximately 30mmHg. Once the anesthetist confirms that he/she can maintain CO$_2$ in desired range with bag-and-mask ventilation, intravenous medications are administered to facilitate tracheal intubation. The route of tracheal intubation (oral or nasal) is based on the child's position during surgery. However, the authors recommend nasotracheal intubation when possible because it reduces the chance of inadvertent tracheal extubation, especially when the tracheal tube is secured with wires or sutures.

Anesthesia is maintained throughout the procedure with an inhaled agent and oxygen. A dexmedetomidine infusion, when available, helps maintain a stable cardiovascular system. All children should have at least two large peripheral IVs for the surgery. Blood products should be immediately available during heavy blood loss portions of the procedure. Many centers routinely insert central venous catheters for complex reconstruction of the cranial vault. An arterial line is required. A precordial Doppler is used to detect venous air embolism. A urinary bladder catheter allows continuous drainage of urine.

One of the biggest challenges during craniofacial surgery is trying to accurately identifying the amount of blood being lost. Doing so with precision is often impossible because much of the lost blood is hidden under the surgical drapes. Throughout surgery the surgical field is irrigated with large volumes normal saline, which mixes with blood collected from the field, making it difficult to estimate blood loss. Weighing all sponges helps. Each gram increase in sponge weight equals 1ml of blood. Monitoring the morphology of the arterial and venous pressure waves and the trends in arterial blood pressure (up or down) provides useful information about the patient’s volume status (See Chapter 2). The trend of central venous pressure up or down is more useful than any particular number. As indicated above, urine output, and serial measurements of hemoglobin concentration and arterial blood gases are necessary. Constant observation of the surgical field is still the best way to estimate blood loss.

A decrease in hemoglobin concentration is usually caused by hemorrhage and/or the administration of large volumes of crystalloid, colloid or blood products (dilutional coagulopathy).
Metabolic acidosis, thrombocytopenia, hypocalcemia (from binding of calcium to citrate), and hypothermia also occur. Hyperkalemia, arrhythmias, and even death may sometimes occur when more than two-week old stored red blood cells are administered rapidly. Current recommendations are to administer RBCs that are less than one week old, to wash RBCs before administering them if they are more than two weeks old, and infuse the cells at 1.5ml/kg/min (which is not always possible if blood loss is occurring rapidly).124-126

Before beginning surgery, blood products, especially red blood cells and fresh frozen plasma (FFP), should be in the operating room. Much of the blood loss during craniosynostosis surgery occurs with the initial dissection and reflection of the scalp and periosteum. Replacing blood loss with crystalloid or RBCs without FFP causes dilution coagulopathy. Dilutional coagulopathy can be avoided by using a 1:1 mixture of RBCs and FFP (1 unit of RBC and 1 unit of FFP) for transfusion. Whenever possible, a single donor should provide all blood that will be transfused to a given child.127,128 Fibrinogen levels decreases rapidly during massive bleeding. Administering concentrated fibrinogen 30-50mg/kg or cryoprecipitate 1unit/10kg should be considered when fibrinogen levels are low.129

Reducing blood loss during craniosynostosis surgery may improve patient safety and decrease morbidity, mortality, and hospital costs. For these reasons blood saving techniques should be used when possible. Patients found to be anemic several weeks before surgery should be given erythropoietin and an iron supplement to increase their hemoglobin concentrations.130-132

Meticulous surgical technique reduces bleeding. Infiltrating the scalp with epinephrine before making the skin incision also reduces blood loss. Since acidosis and hypothermia increase bleeding, efforts should be made to keep the patient’s acid-base status and temperature normal. Using restrictive transfusion thresholds (transfusing when the hemoglobin concentration is 7g/dl) reduces the need for transfusion by 44%. However, this is not appropriate if the child is hemodynamic unstable, bleeding profusely, or has severe hypoxia and/or cyanotic congenital heart disease.133,134 Each institution must decide the hemoglobin concentration at which transfusion will be initiated during these procedures.135

Tranexamic acid administration during craniosynostosis surgery decreases perioperative bleeding and reduces the need for blood transfusion.136,138 The recommended dose of tranexamic acid is 20mg/kg as a bolus followed by an infusion of 10mg/kg/h.139 This protocol decreased the need for blood transfusion, the number of side effects, and mortality by one third.140

Use of a Cell Saver device has increased in recent years because it decreases the number and volume of allograph blood transfusions required, particularly in children given preoperative erythropoietin. However, the volume of blood recovered must be sufficient to warrant its use.
Children undergoing craniosynostosis repair require intensive care postoperatively. The decision to extubate the tracheal or provide postoperative mechanical ventilation is based on the extent of surgery, the amount of fluid replacement, and the amount of facial and airway edema (particularly in patients who were in the prone position). Postoperative mechanical ventilation may also be required. Patients with Pfiffer syndrome (50%), Crouzon syndrome, or Apert syndromes have a high risk of postoperative apnea if they had preoperative obstructive sleep apnea. Postoperative pain is usually not severe following craniosynostosis repair and can be managed with intermittent doses of opioids or with oral or intravenous acetaminophen.141-145

Cerebral Tumors

After trauma, cancer is the second most common cause of death in children under 15 years of age. Twenty percent of all pediatric tumors occur in the CNS. Improved diagnostic procedures make it easier to detect CNS tumors.146 Pediatric brain tumors are conveniently divided into supratentorial and infratentorial. About half of brain tumors occur in each of the two compartments. Depending on tumor growth rate and its anatomical location, the symptoms of supratentorial tumors include neurological deficits, seizures, and/or increased ICP. Twenty-five percent of intracranial tumors (astrocytomas, oligodendrogliomas, ependymomas, and glioblastomas) are located in the cerebral hemispheres. (Figures 10-7A-10-7B)

**Figure 10-7A: Pylocytic Infratentorial Astrocytoma.**

*Axial T1-weighted post contrast and axial T2-weighted images showing enhance cerebellar mass with cystic components compressing the fourth ventricle. Enhancement is heterogeneous and includes portion of the wall of the cyst.*
Figure 10-7B: Supratentorial Oligoastrocytoma.

Axial FLAIR, axial T1-weighted post contrast and coronal T2-weighted images showing a heterogeneous cortical and subcortical mass involving the right thalamus and medial temporal lobe.

Choroid complexes papillomas are rare and usually occur in <3 year-old children. (Figures 10-8A-10-8B) They arise from lateral ventricle choroid plexus. Early hydrocephalus is common with these tumors because they increase production and decrease reabsorption of CSF. They also obstruct flow of CSF from the brain to the spinal cord. Massive intraoperative bleeding is more likely with highly vascular tumors.147
This 2 years old girl with a large choroid plexus tumor had a gait disorder, irritability and vomiting.
General anesthetic considerations for resection of supratentorial tumors include increased ICP, electrolyte imbalances, seizures, the effects of anticonvulsants on anesthetic drugs, and the risk of acute and chronic aspiration with abnormal airway reflexes. Due to the high risk of bleeding, blood should be immediately available in the operating room prior to surgery. Two large IVs and invasive hemodynamic monitoring are mandatory. A bladder catheter should be placed when the surgery will be prolonged and when osmotic diuretics or large amounts of IV fluids will be given during surgery. Tumor resection in neurologically sensitive areas of the brain is best done with intraoperative neurological monitoring. Patients older than seven years of age can be considered for awake craniotomy if other forms of intraoperative monitoring are unavailable.
Sellar and suprasellar tumors include craniopharyngiomas, germ cell tumors, hypothalamic tumors, pituitary adenomas, and optic gliomas. Like all patients requiring neurosurgery, the patient must be completely evaluated before surgery, particularly if there are serious endocrine imbalances. Craniopharyngiomas are the commonest parasellar tumors in children and adolescents and are frequently associated with pituitary and hypothalamic dysfunction. **(Figure 10-9)** These children may have endocrine dysfunction, hydrocephalus, and visual disturbances due to compression of the optic chiasm by the tumor. The surgical approach is usually by frontal craniotomy or by the transphenoidal approach.

**Figure 10-9: Craniopharyngioma.**

*Axial T1-weighted and Sagittal T1-weighted post contrast MRI. Images show a complex partially cystic suprasellar mass with the presence of an enhancing rim and solid components.*

Anesthetic considerations for patients with sellar and suprasellar tumors are similar to those for patients with other supratentorial tumors. However, the anesthetist must also look for evidence of endocrine dysfunction (hypothyroidism, growth hormone deficiency, ACTH deficiency, and diabetes insipidus). Specific hormone deficiencies should be replaced. Teenagers and older children can have pituitary adenomas removed by transphenoidal surgery. Patients having sellar and suprasellar tumors removed require the same intraoperative monitoring and vascular access as other patients with brain tumors. Their trachea is usually intubated orally with a pre-formed RAE tube. The anesthetist should always be prepared for massive intraoperative bleeding with these operations. Optic gliomas occur in children with neurofibromatosis. Children with optic gliomas present to hospital with proptosis, increased ICP, and visual disturbances. Hypothalamic dysfunction is usually a late finding. Blood loss may be significant during transphenoidal surgery.
Diabetes Insipidus

*Diabetes insipidus (DI)* is caused by a deficiency in antidiuretic hormone (ADH), which acts directly on the distal tubules and collecting ducts of the kidney to promote water reabsorption. Brain tumors are often the cause of DI, particularly hypothalamic and pituitary tumors. DI also occurs with optic nerve injury and traumatic brain injury. Approximately 75% of patients undergoing transcranial resection of pituitary tumors and in 10-44% of those undergoing transphenoidal resection have DI. Although DI can occur intraoperatively, its onset more commonly occurs 2-to-6 hours after surgery and resolves 2-to-7 days later. Diabetes insipidus is characterized by polyuria (greater than 4ml/kg/h in children and greater than 6ml/kg/h in neonates), increased plasma osmolality (greater than 300mOsm/l) and decreased urine osmolality (less than 300mOsm/l). A urine specific gravity below 1.005 and a serum Na⁺ above 145meq/l are characteristic of the hypernatremic hypovolemia associated with DI. The treatment goal for perioperative DI is maintenance of water and electrolyte balance, urine output, and normal hemodynamics. Other causes for the high urine output must be ruled out, including the use of diuretics or mannitol and hyperglycemia. If DI is present prior to surgery, preoperative and intraoperative IV administration of vasopressin should be considered. During surgery careful evaluation of urine output is necessary at least once an hour. Two-thirds of the fluid lost, plus any blood lost, should be replaced every hour to maintain hemodynamic stability. Perioperative fluid management during DI should not include hypotonic solutions. The initial dose of vasopressin 0.5mU/kg/h by infusion is increased every 5-to-10 minutes until the urine output is about 2ml/kg/hour. Vasopressin (DDAVP) 0.5-to-4mcg is given IV as a single dose. DDAVP, a potent antidiuretic with minimal vasopressor effects, can also be used in the perioperative period. A quarter of this dose is also used to decrease bleeding in patients with von Willebrand disease. Vesopressin’s effects last 8-12 hours. Serum Na⁺ levels must be monitored when using DDAVP.¹⁴⁸,¹⁴⁹

**Figure 10-10: Medulloblastoma**

An axial CT, axial T1-weighted postcontrast MRI and sagittal T2-weighted images showing infratentorial large mass filling and expanding within the fourth ventricle.
Posterior fossa brain tumors include medulloblastomas, cerebellar astrocytomas, ependymomas, and brain stem gliomas. (Figure 10-10) They block CSF circulation between the fourth ventricle and spinal canal, which increases ICP. Common signs and symptoms of posterior fossa tumors include morning vomiting, irritability, lethargy, stiff neck, cranial nerve dysfunction, and ataxia. Surgical resection of posterior fossa tumors has important anesthetic considerations beyond those of all pediatric neurosurgical procedures. Care should be taken to fix the tracheal tube securely, since most of these surgeries are done in the prone position. The head is placed in a horseshoe headrest to allow intraoperative access to the airway. Invasive hemodynamic monitoring is indicated, due to the potential for cardiac arrhythmias and sudden change in blood pressure that occur with surgical stimulation of structures in the posterior fossa. There is some risk venous air embolism in the prone position that must be searched for at all times. Most children with posterior fossa tumors have raised ICP and obstructive hydrocephalus. Surgeons often place an external ventricular drain to remove CSF before starting the surgical resection.150

**Traumatic Brain Injury and Head Trauma**

Head trauma is the leading cause of morbidity and mortality in children and adolescents worldwide. Motor vehicle accidents, bicycle accidents, child abuse, and falls are responsible for most of these injuries. Many children with traumatic brain injury (TBI) have minimal neurologic symptoms during their initial assessment. However, symptoms of increased ICP and neurologic deficits develop over time.

Brain injuries occur in two phases. The first occurs at the moment of impact when the skull, neural tissue, and vasculature are injured. The second occurs when an endogenous cascade of biochemical and cellular events is initiated. This cascade begins within minutes of the initial injury and continues for months, leading to axonal injury and cell death. Cerebral edema, hypotension, hypoxia, hypo- or hypercapnia, intracranial hypertension, and hypo- or hyperglycemia significantly increase secondary brain injury.151,152

Physical examination of these patients must search for signs of skull fracture, CSF leak, auditory canal bleeding, periauricular hemorrhage, and foreign bodies. The presence of scalp laceration must be sought because they can cause severe bleeding, hypovolemic shock, and worsen secondary brain injury.

Skull fractures are generally linear and are associated with severe contusion or concussion. Seven-to-ten percent of children with TBI have depressed skull fractures. Surgery to elevate a depressed skull fracture is indicated if the cranial plate is depressed more than the thickness of the bone, if a dural laceration is causing a neurologic deficit, and/or if there is a CSF leak. Few children have basal skull fractures. The signs of basal skull fractures include auditory canal bleeding, nasal bleeding, mastoid ecchymosis, periorbital bruising, and blood behind the tympanic membrane. Complications from a basal skull fracture include a CSF leak, meningitis, and anosmia.
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Diffuse brain injuries are the result of rapid acceleration and deceleration of the brain. Efforts should be made to detect focal lesions early, since immediate surgery changes their outcome considerably. Cerebral contusion is common in pediatric patients, and the hairline fractures associated with them are most frequently located in the temporal, frontal, and occipital regions. Bruising occurs during the second phase of the injury. The neurologic effects of cerebral contusion are due to mass effect, edema, or hemorrhage. If there is mass effect, immediate surgery is indicated to prevent or treat increased ICP. Epidural hematomas (Figure 10-11) are usually associated with parietal or temporal skull fractures and often with injury to the middle meningeal artery. Signs and symptoms of an epidural hematoma include loss of consciousness that occurs after an initial lucid period. The occurrence of contralateral hemiparesis and ipsilateral mydriasis should lead to immediate drainage of the hematoma and reduction of ICP. Medical management of ICP should be initiated as soon as possible.

Figure 10-11: Epidural Hematoma:

Axial CT shows a hyperdense, bi-convex epidural hematoma with compression of brain. The internal hypodensity implies an active bleeding.

Acute subdural hematomas (Figure 10-12) are associated with cortical damage due to direct parenchymal contusion. There is disruption of the bridging veins between the dura and the cortex. Fractures occur in 30% of older patients and more frequently in those below two years of age. Mortality with subdural hematoma is high. Cerebral edema, increased ICP, and persistent neurological deficits characteristically occur during the postoperative course of patients who survive surgery for subdural hematoma.¹⁵³
Axial CT images showing a centric, non-homogeneous extra-axial collection with compression and displacement of the underlying brain structures.

**Child Abuse (Shaken Baby Syndrome)**

Child abuse accounts for 80% of deaths in children who have TBI, a chronic subdural hematoma, and who are less than two years of age. The bleeding is usually caused by acceleration and deceleration forces, direct trauma, and by traction injury to vessels of the brain stem. The child presents with changes in consciousness, lethargy, irritability, vomiting, and seizures. More than 75% of patients with the SHAKEN BABY SYNDROME have retinal hemorrhages. The presence of a subarachnoid hemorrhage on CT, particularly inter-hemispheric blood (subdural hematoma), is a specific sign of the shaken baby syndrome.¹⁵⁴
Parenchymal hematomas (Figure 10-13) are common in children with severe cortical contusions following TBI. The incidence of parenchymal hematoma is low, but when it is present the prognosis is very poor. Indications for urgent surgery in patients with TBI include an open skull fracture and a depressed fracture causing symptoms. Surgically evacuated intracerebral hematomas are often associated with severe tissue damage. They frequently occur in highly vascularized areas. Due to the poor outcomes and the impact of secondary brain injury on the outcome of TBI, treatment should be directed, not only to the evaluation, diagnosis, and stabilization of the patient, but also at reducing secondary brain injury, which ultimately is the main cause of morbidity and death.

Secondary brain injury is the result of hypotension, hypoxemia, hypo and hypercapnia, hypo- and hyperglycemia, and anemia. Neurologic outcomes with secondary brain injury may be improved by early and adequate resuscitation, \(^{155}\) triage of the patient to a facility that frequently cares for patients with TBI, and surgery when indicated. The goals of anesthetic management are to secure the airway, prevent secondary brain injury, prevent increased ICP, and to maintain adequate CPP. Positive pressure ventilation is initiated early to maintain a PaO\(_2\) >60mmHg. Positive end-expiratory pressure (PEEP) is avoided because it increases ICP and decreases cerebral venous drainage. Hypoxemia and aggressive hyperventilation must also be avoided. Therapy to prevent cerebral herniation occasionally includes inducing hypocapnia. However, hypocapnia can worsen cerebral ischemia, especially if the arterial blood pressure is low. \(^{156}\) The status of the brain should be evaluated frequently.
Because of the risk for hypovolemia and hypotension, appropriate amounts of isotonic saline must be given to maintain normovolemia. Anemia, when present, must be corrected. Although specific recommendations for transfusion therapy in pediatric patients with TBI do not exist, there is no benefit to either a liberal or restrictive transfusion protocol. Abnormal coagulation often occurs with TBI, but the exact mechanism for this is poorly understood. It is hypothesized that coagulation defects are the result of massive release of tissue factors, disseminated intravascular coagulation, fibrinolysis, hypoperfusion, activation of protein C, and platelet dysfunction (See Chapter 5).

The Society of Pediatric Critical Care published evidence-based recommendations for management of severe TBI in children and adolescents in 2012. Anesthetists should be familiar with these recommendations, as they are intended to reduce secondary injury.

Children with severe head trauma and TBI usually die from refractory increased ICP. The following is an outline for treatment of patients with TBI.

**Initial Treatment of TBI Should Focus on Management of Increased ICP and Maintenance of the CPP:**

1. ICP monitoring should be performed when possible in all children with severe TBI [Glasgow Coma Scale (GCS) <8] because they surely have intracranial hypertension and possibly high ICPs. Early ICP monitoring, when used to guide therapy, reduces morbidity and mortality.
2. ICPs above 20 mmHg should be treated if they persist more than five minutes. ICP monitoring and invasive arterial pressure monitoring are essential to calculate CPP (CPP = MAP - ICP) and guide therapy.
3. The minimum acceptable CPP in children with severe TBI is 40mmHg, the goal being 40-to-50mmHg. For infants CPP might be a lower whereas for teenagers it is higher.
4. Hypertonic saline (3%) is used to reduce intracranial hypertension in children with severe TBI. The recommended dose of 3% hypertonic saline is 6.5-to-10ml/kg/h. Hypertonic saline can be infused at 0.1-to-1ml/kg/h. It is recommended that the lowest dose of saline that will maintain the ICP below 20mmHg be used, and that plasma osmolality be kept below 360 mOsm/L. There is insufficient evidence to support or reject the use of mannitol in the treatment of intracranial hypertension in children with severe TBI.
5. There is no evidence that hypothermia (32-33°C) is beneficial during the first 24h following TBI and in fact, can negatively effect the outcome in these patients.
6. Prophylactic hyperventilation to a PaCO₂ <30 mmHg should be avoided in the first 48h after TBI. The risk on iatrogenically induced cerebral ischemia is very high. When hyperventilation is used to treat refractory intracranial hypertension, neurophysiological monitoring is required to determine the adequacy of cerebral blood flow and the presence of cerebral ischemia.
7. An external ventricular drain is sometimes used to remove CSF and lower the ICP of patients who have severe TBI.
8. Barbiturates can be used to treat hemodynamically stable children who have intracranial hypertension that has not responded to optimal medical and surgical management. The arterial blood pressure should be monitored continuously and the information gained from it used to maintain adequate CPP.
9. Decompressive craniectomy with duraplasty has been used when there are early signs of neurological deterioration, brain herniation, or refractory intracranial hypertension that are not responsive to medical management.
10. Prophylactic treatment with phenytoin may reduce the incidence of early posttraumatic seizures in pediatric patients with severe TBI.
11. Data concerning glycemic control in children with severe TBI are lacking.
12. Steroid use is not recommended for pediatric patients with severe TBI because there is no clinical evidence that it improves outcome.¹⁶⁰

The ultimate goal in the management of children with TBI is prevention of secondary brain injury. Cardiovascular resuscitation and effective ventilation are important, but other injuries must also be treated. The classic mistake is to focus only on the brain injury and ignore abdominal or thoracic injuries that often contribute significantly to the patient’s morbidity and mortality.

**Spinal Cord Injury (SCI)**

Isolated spinal cord injury is rare in children, and those who have SCI usually have TBI also. The reported incidence of SCI is 5-to-10% in both adults and children. Because pediatric patients have large, heavy heads, 42% of SCIs occurs to the cervical spine, while 31% occur in the thoracic and 27% in the lumbar spine. Automobile accidents, falls, and sports injuries are the usual causes of SCI. SCI is more common in males than females. Due to differences in growth and development of the spinal column, spinal trauma in children is significantly different from that of adults. These differences include larger and heavier heads and greater elasticity and mobility of C1-C3 vertebral bodies in children under eight years of age. Between 8-and-12 years of age, increased mobility is observed between C3-C5; in older children, it occurs at C5-C6. They have of partially ossified vertebrae, more flexible joint capsules, and horizontal, flat vertebral body surfaces.¹⁶¹,¹⁶²

Because of these differences, children <8 years of age more commonly have high cervical injuries. The younger the child the greater is the risk for high cervical injury. In children older than 8 years of age, SCI occurs at the lower portion of the cervical spine. Due to the high elasticity and mobility of the pediatric spine, radiologic criteria used to diagnose SCI in children differ from those of adults. Vertebral body dislocation of more than 4.5mm between C2-C3 or C3-C4 in <8 year old children, or subluxation of a vertebra in excess of 3.5mm at any vertebral level in patients >8 years of age is an indication of cervical spine instability. A more than seven degree angulation of one vertebra on another suggests spinal cord injury.
The signs of possible cervical spine injury most often include pain in the posterior midline, peripheral neurologic deficits, decreased level of consciousness, and evidence of cerebral cognitive dysfunctions. However, it must be remembered that the pain may be caused by other trauma and not by SCI. Spinal cord injury in children can be classified into four categories: isolated vertebral fractures, fracture with subluxation, subluxation only, and traumatic spinal cord injury without radiological evidence of injury.

Management of SCI includes: 1) securing the airway while maintaining cervical immobilization in patients who have reduced consciousness, high CSI, and/or decreased airway protection; 2) ensuring adequate oxygenation and proper lung ventilation; 3) restoring the patient’s hemodynamics to normal.

SCI has two primary causes: 1) initial injury by mechanical forces and bone fragments that cause direct trauma to the spinal cord. 2) secondary tissue damage that is caused by edema and ischemia from hypotension and hypoxemia. Inadequate resuscitation of a child with unstable SCI may exacerbate the primary lesion and favor development of a secondary injury.

Respiratory failure is the most common cause of death in patients with isolated cervical injuries. Cervical SCI-induced respiratory failure decreases tidal volume and minute ventilation, increases respiratory dead space, and causes CO₂ retention, all of which worsen secondary brain and spinal cord injury. Thus, it is vital to quickly secure the airway and ensure adequate oxygenation and ventilation. Immobilizing the neck avoids aggravating SCI. Spinal shock, loss of vasomotor tone, bradycardia, and decreased myocardial contractility are common in patients with SCI. However, the anesthetist must also look for other causes for the hypovolemic shock such as injury to the chest, abdomen, pelvis, and long bones, or obstructive shock secondary to tension pneumothorax or cardiac tamponade.

Fluid management for SCI should begin with normotonic crystalloid solutions. Vasopressors are added if the blood pressure does not respond to fluid administration.

**Spinal Cord Injury Without Evidence of Radiologic Abnormalities (SCIWORA)**

SCIWORA is a spinal cord injury that occurs without radiologic evidence of vertebral fractures or subluxation. It occurs in 13-32% of pediatric patients with SCI and most commonly affects the cervical spine. SCIWORA is the result of the relaxed spinal ligaments and severe flexion-extension of the spine with trauma. The pathophysiology of SCIWORA is related to punctate hematomas/hemorrhages, edema, and tissue infarction. A MRI helps diagnose soft tissue, ligament, and spinal cord lesions. Treatment of SCIWORA includes appropriate cardiopulmonary resuscitation to ensure proper oxygenation, ventilation, and perfusion. Systemic blood pressure is maintained within normal limits or slightly above normal. The patient’s neck is immobilized with a rigid cervical collar. Although still controversial, it is nevertheless recommended that these
patients be given a bolus of methylprednisolone 30mg/kg and a continuous infusion of 5.4mg/kg/hour of methylprednisolone for 24 hours if the patient arrives during the first three hours after the trauma. If the patient arrives after three hours, the infusion should be continued for 48 hours. Methylprednisolone improves medullary blood flow, inhibits the arachidonic acid cascade, and modulates the immune response. Patients with a transected spinal cord have a poor prognosis (3% recover). Sixty-two percent of those with an incomplete spinal cord transection recover.¹⁶³,¹⁶⁴

Vascular malformations

Arteriovenous Malformations (AVMs) and Aneurysms

Arteriovenous malformations and intracranial aneurysms are the most common causes of spontaneous intracranial hemorrhage in children. AVMs occur in 10-to-14/100,000 people, 7-10% of which are children under the age of 18 years.¹⁶⁵ (Figure 10-14)

Figure 10-14: Arteriovenous Malformation (AVM).

Axial T2-weighted, axial T1-weighted post contrast and angiographic MRI showing a tightly packed mass looking like a honeycomb distribution of flow. The lack of contrast enhancement represents an AVM nidus in the right frontal lobe.

Bleeding from an AVM frequently presents as intraventricular hemorrhage, sudden onset of seizures, or severe neurological deficits. Intracranial hemorrhage is found in 20-50% of pediatric AVMs. Eighteen-to-twenty percent of AVMs are symptomatic during childhood. Intracranial hemorrhage occurs in 75-to-80% of pediatric patients with AVMs and 50-to-65% of adults.¹⁶⁶,¹⁶⁷ Infants and children under two years of age who have AVMs often have congestive heart failure, delayed neurodevelopment (due to blood being shunted away from some parts of the brain by the AVM - so called vascular steal), headache, and a cranial murmur. The risk of rebleeding from
an AVM is high during the first twelve months after the initial bleed. Many posterior fossa and basal ganglia AVMs bleed. The incidence of rebleeding is 25% over the first five years. In children, the location and size of AVMs are risk factors for rebleeding. Deep lesions bleed more often than hemispheric lesions.

Medicosurgical management for AVMs includes endovascular embolization, stereotactic radiosurgery, or as the last recourse, surgical resection. In 70% of cases, endovascular embolization will partially obliterate the AVM, but it does not decrease the incidence of recurrent bleeding. In some cases, embolization is indicated before surgery to reduce flow in the AVM and make clipping the AVM easier. When the approach to an AVM is difficult (brain stem, basal ganglia, thalamus), the lesion is best treated with stereotactic radiosurgery, if available.

Elective embolization of vascular lesions is usually done while the patient is under general anesthesia. Moderate hyperventilation (PaCO$_2$ 30mmHg) improves the radiologist’s ability to see abnormal blood vessels because vessels in an AVM do not respond to the vasoconstriction effect (including alkalosis). Thus, hyperventilation increases flow in AVMs making it easier to see the defect. However, as it increases the amount of blood within the lesion, it can affect the ability to deposit the embolizing material and also increase tendency to bleed. Anesthetists should be aware of the material being embolized and of their side effects and any complications they can cause. Close attention must be paid to fluid management, because children with AVMs often have heart failure due to high AVM flows and to the use of contrast medium by the radiologists.

The vein of Galen, which drains directly into the straight sinus, is a short but large diameter collection of veins from the deep venous system of the brain. Most malformations of the vein of Galen occur in infants and small children, whereas AVMs are rare in newborns. The clinical presentation of patients with malformations of the vein of Galen depends on the amount of blood shunting through the malformation. Newborns (0-1 month of age) can have multiple fistulas that divert nearly 25% of their cardiac output through them, which greatly increases blood return to the right heart and causes volume-related heart failure. Most newborns with AVMs have congestive heart failure (CHF). One-to-twelve month old infants may present with hydrocephalus that is caused by compression of the aqueduct of Sylvius and also the third ventricle. This prevents normal drainage of CSF from the brain. Children with AVMs may present with compensated congestive heart failure, seizures, or focal neurological deficits. Children over six years of age may present with a subarachnoid hematoma or with intra-parenchymal cerebral bleeding.\textsuperscript{168}

Whether therapeutic intervention requires immediate or deferred attention, all children with AVMs require definitive evaluation of their AVM by CT, MRI, and cerebral angiography (if available) after complete assessment of their medical condition. Older children who are alert and oriented may be able to cooperate during these studies, but younger children or those with impaired consciousness require general anesthesia for airway protection and optimization of
conditions for the radiologist. During induction and maintenance of anesthesia, sudden changes in the arterial blood pressure, especially hypertension, can cause intracerebral hemorrhage and raise the ICP. Anesthetics used for maintenance of anesthesia are not different in patients with an AVM from those used for other intracranial procedures (See above). In the absence of congestive heart failure, controlled hypotension has been used during surgical or endovascular closure of AVMs. Because sudden massive bleeding can occur during surgical closure of an AVM, two large bore IVs and an arterial line are needed. Whether the malformation is clipped during surgery or closed endovascularly, the anesthetist must be prepared to treat sudden elevations in intracranial pressure and/or the hyperemic cerebral edema that often occur when the AVM is closed. A hypertensive crisis can be treated with nitroprusside (neonates 0.5-6mcg/kg/min; children 0.5-10mcg/kg/min) or labetalol (0.2-1.0mg/kg intravenous every 10 min, maximum dose 300mg or continuous infusion 0.4-1mg/kg/h). As in all CNS surgery, the anesthetist’s goal during closure of an AVM is to maintain hemodynamic stability, cerebral perfusion pressure, control of ICP, provide enough brain relaxation to facilitate surgical exposure of the AVM, and to base fluid administration on blood and urine losses.

Although intracranial aneurysms are rare in children, they are usually large and are located in the posterior cerebral circulation. There are two types of aneurysms: “saccular” (caused by weakness of the muscle wall of the artery) and “mycotic” (caused by damage to arteries by septic emboli). Congenital aneurysms are associated with polycystic kidney diseases, fibromuscular dysplasia, connective tissue disorders (such as Ehlers- Danlos syndrome), and coarctation of the aorta. Congenital aneurysms are usually treated by endovascular embolization.169-172

**Moyamoya Disease**

*Moyamoya* disease is a chronic vascular condition of unknown etiology that is associated with a severe occlusive cerebral arterial disease. Angiography shows progressive stenosis or occlusion of the terminal portion of the internal carotid and the anterior and middle cerebral and the posterior cerebral arteries. Moyamoya means ‘Puff of Smoke’, which refers to the angiographic appearance of a puff of ‘cigarette smoke’ seen beyond the non-vascularized area.173 Symptoms of Moyamoya disease include transient cerebral ischemia, intracranial hemorrhage, seizures, and headache. Patients with Moyamoya disease have progressive cerebral ischemia from ongoing reduction in cerebral perfusion. Treatment of MoyaMoya disease is surgical revascularization of the affected areas. The goal of revascularization is to promote neo-angiogenesis and induce formation of collateral vessels. In preparation for surgery, the patient should not be fasted for long periods to avoid dehydration. Premedication is indicated to limit anxiety and crying, hyperventilation, decreased PaCO2, cerebral vasoconstriction, decreased CBF, and worsening of cerebral ischemia. There is no evidence that the volatile or intravenous drugs used for induction of anesthesia have effects on the cerebral perfusion of patients with MoyaMoya disease. During surgery, the anesthetist must maintain a balance between the oxygen supply and demand of the
brain. Hypotension, hypoxemia, and changes in CO₂ during laryngoscopy and tracheal intubation are to be avoided. Normocapnia should be maintained during all stages of the procedure. During surgery, EEG monitoring can be used to detect early episodes of cerebral ischemia.¹⁷⁴

Care should be taken to ensure normal hydration. Hyperosmolar solutions are avoided because they produce diuresis, dehydration, and hypotension. The ideal hematocrit for these patients is 30% because at this hematocrit oxygenation is adequate and tissue blood flow is increased. The increased blood viscosity associated with polycythemia decreases cerebral perfusion and this may lead to cerebral infarction. During surgery, the systemic arterial blood pressure should be maintained slightly above the patient’s preoperative pressure. A decrease in mean arterial blood pressure decreases CBF and increases the risk of ischemia or stroke. Cerebral autoregulation is substantially diminished in adults with this disease when compared to children. For this reason, a higher frequency of ischemic symptoms is observed in adults. Asymptomatic, hemodynamically stable patients undergoing a radiological procedure can usually be sent home after the procedure. Those undergoing revascularization procedures require intensive care to ensure proper hemodynamic monitoring and treatment of any abnormalities found. Arterial blood pressure, CVP, urine output, hematocrit, and SaO₂ should be continuously monitored postoperatively. Use of opioids and intravenous acetaminophen for postoperative analgesia reduces the risk of cerebral ischemia.¹⁷⁵

**Surgery for Epilepsy**

*Epilepsy* is a chronic condition that affects 0.5-to-1% of the world's population and is considered to be a public health problem.¹⁷⁶ Despite adequate anticonvulsant therapy and new medications for the treatment of epilepsy; the failure rate of therapy is still 30-40%. Inadequately treated epilepsy decreases the neurocognitive development of children. It increases neuroplasticity of the developing brain. Aggressive surgical management of epilepsy is appropriate for many patients when medical therapy fails to control epileptic discharge.¹⁷⁷

Epilepsy refractory to medical therapy is defined as inadequate control of seizures despite giving two anticonvulsant medications at their maximum tolerated doses for 1.5-to-2 years. Patients who have adequate seizure control, but suffer unacceptable side effects from the medications, are also considered to have refractory epilepsy. Patients with cortical malformations, abnormal neuronal migration syndromes (Rasmussen, West, Sturge-Weber) frequently have refractory epilepsy. Epilepsy surgery provides anesthetists with two major challenges that are not usually found in other neuroanesthesia procedures: 1) the possibility of the patient’s craniotomy being done under local anesthesia and 2) the need for prolonged narcosis. Doing an awake craniotomy in pediatric patients may be difficult. Most of them require general anesthesia. Some anesthetic are avoided during surgery for epilepsy because they affect electrical activity and intraoperative electroencephalographic monitoring.
Chapter 10: ANESTHESIA FOR NEUROSURGICAL PROCEDURES IN CHILDREN

The Anesthetist’s Management of Patients with Epilepsy Includes:

1. A thorough pre-anesthesia assessment of coexisting medical conditions, assessment of side effects of the patient’s antiepileptic drugs, and knowledge and understanding of the neurophysiological tests to be performed during surgery.
2. Providing an adequately relaxed brain to facilitate surgery.
3. Avoiding anesthetic drugs that interfere with electrophysiological monitoring.
4. Being aware that it may be necessary to induce seizures during intraoperative electrocorticography.
5. Ensuring adequate CPP and maintaining hemodynamic, respiratory, and metabolic stability.
6. Assuring the absence of awareness throughout the procedure, except when patient cooperation is required to preserve the integrity of vital areas of the brain (speech, motor function).
7. Allowing rapid awakening to allow early neurological assessment of the patient.\(^{178,179}\)

If neuropsychological testing is available, it can be used to assess the patient’s preoperative cognitive function. The results of these tests can be used to predict the impact of the surgery on outcome. EEGs are used to detect seizures, to confirm the presence of abnormal electrical activity, and to guide the surgeon to the area he/she will remove. Intraoperative EEG monitoring helps define the exact location of the seizure focus and its relationship to vital functional areas. The use of EEG monitoring is especially important when the seizure focus is outside the temporal area of the brain. MRI, SPECT and PET scanning have greatly improved localization of the epileptic focus and helped define the relationship of functional areas of the brain to the epileptic focus. Most tests for preoperative evaluation of epilepsy require that the child not move for long periods of time. Children who cannot do so can be sedated with propofol for placement of the EEG wires and recording needles. The effects of propofol on the EEG are short lived once the drug is discontinued. Once the drug is withdrawn, neurologists can obtain baseline EEG recordings. Dexmedetomidine is also be used for this purpose because it causes minimal effects on the EEG.\(^{180}\)

Longer procedures, such as MRI and SPECT, can be performed with a continuous infusion of propofol. The most common surgical procedures for treatment of childhood epilepsy are resection of the epileptic focus and insertion of a vagal nerve stimulator. Hemispheric resection is indicated for patients with a clear epileptic focus, with abnormal cortical development, hemiplegia, and for Sturge-Weber Syndrome.

Implantation of a vagus nerve stimulator (VNS) is commonly done for intractable epilepsy when it is not possible or desirable to surgically resect the epileptic focus. How VNS works is not well understood, but it appears that it activates the tractus solitaius and other nuclei in the brain stem. This apparently modulates brain excitability by activating both the limbic system and noradrenergic neurotransmitter systems. During surgery the left vagus nerve is exposed, and a
stimulating electrode is placed into the nerve. A signal generator is inserted into the fascia of the pectoral major muscle. The left vagus nerve is used because using the right vagus nerve may cause cardiac pacing. The most common side effects of this procedure are postoperative hoarseness and sore throat. Rare complications include bradycardia, paralysis of facial muscles, and worsening of obstructive sleep apnea. If patients require an MRI after implantation of a VNS, the stimulator should be turned off for the duration of the procedure and turned on once the procedure is completed.

The ideal treatment of intractable seizures is surgical resection of the seizure focus. To do this requires a seizure focus that is unifocal and one that is not close to the motor or language areas of the brain. The area containing the epileptic focus is exposed through a craniotomy. Resection of the epileptic focus is often done in two stages. During the first stage, a craniotomy is done and an array of EEG electrodes is placed directly over the area of the brain believed to contain the seizure focus. The skull is then closed and wires form the EEG electrodes are externalized to allow EEG monitoring of the focus site for approximately 48 hours. During the second surgery, the craniotomy is reopened, and the area identified by the 48 hours of monitoring is resected.181

The choice of anesthetic for induction of anesthesia depends on the age of the child, but sevoflurane or propofol are usually used. An arterial line is inserted to allow continuous monitoring of the hemodynamic changes that occur with surgical manipulation. Two large bore IVs are needed for fluid replacement, as in any invasive neurosurgical procedures. Although large blood losses are unusual, it is prudent to have blood available for transfusion. In most cases, balanced anesthesia is appropriate. When intraoperative EEG monitoring is required, volatile anesthetics are not used. Muscle relaxants are not used if EMG monitoring is required. Propofol or dexmedetomidine provide anesthesia during neurophysiological monitoring. At the end of the surgery, the trachea is usually extubated.

Resection of the corpus callosum is done when it is not possible to resect the epileptic focus. This palliative procedure is done to prevent spread of seizure activity to the other hemisphere of the brain. The anesthetic considerations for this procedure are similar to those described above. Patients undergoing corpus callosum resection usually have significant postoperative cognitive impairment. Electrophysiological monitoring is usually not indicated for this procedure.

Hemispheric resection is the most aggressive surgical procedure for epilepsy and is reserved for patients who have intractable seizures that are caused by diffuse hemispheric malformations of cortical development, stroke, childhood hemiplegia syndrome, and Sturge-Weber and Rasmussen syndromes. Anesthetic considerations for this surgery are similar to those for focal resection of an epileptic focus, except there is a greater probability of hemorrhage and blood transfusion. Anesthesia preparation should include two large bore IVs, invasive hemodynamic monitoring, and inserting an urine catheter. Patients usually require several days of intensive care and postoperative mechanical ventilation after this surgery.182,183
Deep brain stimulation is rarely done in children, but there are data that suggest stimulation of the thalamic nucleus reduces the frequency of seizures. This procedure is only done in children who have intractable seizures and are not candidates for other surgical procedures, including VNS.\textsuperscript{184,185}

**Awake and Functional Neurosurgical Craniotomy**

*Awake craniotomy* has limited utility in pediatric patients and young children but is done in older children and adolescents for epilepsy, implantation of deep brain stimulators for management of movement disorders, deep resection of tumors, and for resection of vascular lesions located in vital brain areas (language, memory, motor and sensory). One of the key requirements for using this technique is the need for patient co-operation. The anesthetist must have knowledge of specific problems that can occur during surgery and the need for continuous monitoring of the awake patient throughout surgery.

The two most important considerations for patients undergoing awake craniotomy are mental maturity and evaluation of the airway. Anxiety disorders, low pain tolerance, mental disorders, movement disorders, claustrophobia, obesity, and gastro-esophageal reflux are contraindications to awake craniotomy. Other factors to consider are tumor size and its effects on cardiovascular stability, risk of bleeding, and hemodynamic instability.\textsuperscript{186}

Positioning of patients for awake craniotomy surgery has some important considerations. The anesthetist must have access to the patients face to evaluate her/his facial expressions and speech throughout the procedure. This is vital to success of the procedure. Both asleep-awake-asleep anesthesia and monitored anesthesia have been used for this procedure. Dexmedtomidine, propofol, and remifentanil are ideal for this purpose because their effects rapidly dissipate when they are discontinued, allowing the patient to “awaken” quickly for intraoperative neurocognitive assessment. Ensuring that the patient has sufficient analgesia to prevent pain, especially when pins are inserted into the skull, when the scalp is reflected, and when the dura mater is incised and manipulated is very important.

The sleep-awake-asleep technique involves administering a general anesthetic, commonly propofol and remifentanil. During the surgery, when electrocorticography (ECoG) is required, the propofol infusion is discontinued (usually 15 minutes before ECoG monitoring begins). This drug is discontinued to prevent it from interfering with interpretation of the ECoG signals. Remifentanyl 0.05mcg/kg/min is continued during the awake phase of anesthesia because it has little effect on the ECoG signals. Dexmedtomidine 0.2mcg/kg/h can also be used, since it has also no affects on ECoG.\textsuperscript{187,188} Monitored conscious sedation is appropriate when the skull is being opened and closed. Bolus or continuous infusions of propofol or dexmedtomidine plus remifentanil or fentanyl are also used. High concentrations of oxygen must not be allowed to accumulate near the surgical field because a spark from the electrocautery could start a fire. The
number of complications occurring during awake craniotomy is low because patients undergoing this procedure are carefully selected and thoroughly prepared. However, airway problems, nausea, vomiting, cerebral edema, seizures, bleeding, agitation, air embolism, and hemodynamic instability have been reported in some patients. 189-191

**Interventional Neuroradiological Procedures**

It is increasingly common for children with intracranial disease to require anesthesia for diagnostic or therapeutic neuroradiology procedures. Procedures such as angiography and embolization of vessels are common. Anesthesia is required for these procedures because the procedures are long, technically challenging, and uncomfortable for the patient, and often require manipulation of blood pressure and ventilation during the procedure. The images obtained are also of better quality in anesthetized, immobile patients. Any complications that occur during these procedures can be taken care of more quickly in anesthetized children. 192

General anesthesia and tracheal intubation are required for these procedures because 10-to-15 second periods of apnea are required to obtain the best quality images. Enough IV fluid is given during these procedures to maintain normovolemia, adequate renal perfusion, and good urine output. Failure to do so may result in renal failure, which is caused by the large volumes of hypertonic contrast medium used. While less than four percent of patients undergoing cerebral angiography have complications, bleeding occasionally occurs. When bleeding does occur, it usually does so at the site where the radiologist obtained vascular access (femoral vessels). Adverse reactions to contrast medium occur, but do so in less than 1% of children undergoing neurointerventional procedures. These reactions consist of nausea, rash, hemodynamic instability, bronchospasm, and cardiovascular collapse. They usually occur within one hour of administering the drug. Mild reactions are self-limited and are managed symptomatically. Severe reactions should be treated like anaphylactic shock. Patients at high risk for reactions to contrast material are those who had previous reactions to these drug, asthma, or atopic dermatitis. When a patient previously has any one of these problems, he/she should be premedicated with steroids and antihistamines before the procedure begins. The recommended dose for the administration of intravenous methylprednisolone is 1-2mg/kg whereas it is 1mg/kg of diphenhidramine.

Therapeutic interventions, such as embolization of arteriovenous malformations, arteriovenous fistulas (AVF), or aneurysms, and the administration of intra-arterial chemotherapy for tumors, usually require general anesthesia. The procedures are typically long and muscle relaxation and paralysis are needed to prevent patient movement that could prove to be catastrophic. The anesthetist must keep track of how much heparinized saline the radiologist gives during these procedures and compensate by reducing the amount of IV fluid the anesthetist gives. Deliberate hypotension is sometimes used to facilitate placement of micro particles or coils in high-flow lesions. Micro particles, such as ONYX, can cause severe bradycardia, especially in infants. The risk
of rupturing an intracranial vessel is 0.5%. When this occurs, the patient must be immediately transferred the patient to an operating room for surgery. After embolization procedures, patients should go to a critical care unit for strict monitoring and control of blood pressure to reduce the risk of postoperative bleeding.²

Conclusions

The anesthetist’s main goal for patients with neurologic pathology is to assure that comprehensive care is provided. This requires knowledge of normal and abnormal brain anatomy, differences in physiology at the different stages of development and the pathophysiological consequences imposed by the medicsurgical condition. Knowing the effects of anesthetics on the brain physiology of pediatric patients significantly improves outcomes and contributes to a significant reduction in morbidity and mortality.

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