CHAPTER 27

Anesthesia for Neurosurgery

KEY CONCEPTS

1. Regardless of the cause, intracranial masses present according to growth rate, location, and intracranial pressure. Slowly growing masses are frequently asymptomatic for long periods (despite relatively large size), whereas rapidly growing ones may present when the mass remains relatively small.
2. Computed tomographic and magnetic resonance imaging scans should be reviewed for evidence of brain edema, a midline shift greater than 0.5 cm, and ventricular displacement or compression.
3. Operations in the posterior fossa can injure vital circulatory and respiratory brainstem centers, as well as cranial nerves or their nuclei.
4. Venous air embolism can occur when the pressure within an open vein is subatmospheric. These conditions may exist in any position (and during any procedure) whenever the wound is above the level of the heart.
5. Optimal recovery of air following venous air embolism is provided by a multiorificed catheter positioned at the junction between the right atrium and the superior vena cava. Confirmation of correct catheter positioning can be accomplished by intravascular electrocardiography, radiography, or transesophageal echocardiography.
6. In a patient with head trauma, correction of hypotension and control of any bleeding take precedence over radiographic studies and definitive neurosurgical treatment because systolic arterial blood pressures of less than 80 mm Hg predict a poor outcome.
7. Massive blood loss from injuries to the great vessels can occur intraoperatively with thoracic or lumbar spine procedures.

Anesthetic techniques must be modified in the presence of intracranial hypertension and marginal cerebral perfusion. In addition, many neurosurgical procedures require patient positions (eg, sitting, prone) that further complicate management. This chapter applies the principles developed in Chapter 26 to the anesthetic care of neurosurgical patients.

Intracranial Hypertension

Intracranial hypertension is defined as a sustained increase in intracranial pressure (ICP) above 15 mm Hg. Intracranial hypertension may result from an expanding tissue or fluid mass, a depressed skull fracture, interference with normal absorption

of cerebrospinal fluid (CSF), excessive cerebral blood volume (CBV), or systemic disturbances promoting brain edema (see below). Multiple factors are often simultaneously present. For example, tumors in the posterior fossa usually are not only associated with some degree of brain edema and mass effect, but they also readily obstruct CSF outflow by compressing the fourth ventricle (obstructive hydrocephalus).

Although many patients with increased ICP are initially asymptomatic, they typically develop characteristic symptoms and signs, including headache, nausea, vomiting, papilledema, focal neurological deficits, and altered consciousness. When ICP exceeds 30 mm Hg, cerebral blood flow (CBF) progressively decreases, and a vicious circle is established: ischemia causes brain edema, which in turn, increases ICP, resulting in more ischemia. If left unchecked, this cycle continues until the patient dies of progressive neurological damage or catastrophic herniation. **Periodic increases in arterial blood pressure with reflex slowing of the heart rate (Cushing response) can be correlated with abrupt increases in ICP (plateau or A waves) lasting 1–15 min**. This phenomenon is the result of autoregulatory mechanisms periodically decreasing cerebral vascular resistance and increasing arterial blood pressure in response to cerebral ischemia; unfortunately, the latter further increases ICP as CBV increases. Eventually, severe ischemia and acidosis completely abolish autoregulation (vasomotor paralysis).

CEREBRAL EDEMA

An increase in brain water content can be produced by several mechanisms. Disruption of the blood–brain barrier (vasogenic edema) is most common and allows the entry of plasma-like fluid into the brain. Increases in blood pressure enhance the formation of this type of edema. Common causes of vasogenic edema include mechanical trauma, high altitudes, inflammatory lesions, brain tumors, hypertension, and infarction. Cerebral edema following metabolic insults (cytotoxic edema), such as hypoxemia or ischemia, results from failure of brain cells to actively extrude sodium causing progressive cellular swelling. Interstitial cerebral edema is the result of obstructive hydrocephalus and entry of CSF into brain interstitium. Cerebral edema can also be the result of intracellular movement of water secondary to acute decreases in serum osmolality (water intoxication).

TREATMENT

Treatment of intracranial hypertension and cerebral edema is ideally directed at the underlying cause. Metabolic disturbances are corrected, and operative intervention is undertaken whenever appropriate. Vasogenic edema—particularly that associated with tumors—often responds to corticosteroids (dexamethasone). Vasogenic edema from trauma typically does not respond to corticosteroids. Blood glucose should be monitored frequently and controlled with insulin infusions (if indicated) when steroids are used. Regardless of the cause, fluid restriction, osmotic agents, and loop diuretics are usually effective in temporarily decreasing brain edema and ICP until more definitive measures can be undertaken. Diuresis lowers ICP chiefly by removing intracellular water from normal brain tissue. Moderate hyperventilation ($Paco_2$ of 30–33 mm Hg) is often very helpful in reducing CBF, CBV, and ICP acutely, but may aggravate ischemia in patients with focal ischemia.

Mannitol, in doses of 0.25–0.5 g/kg, is particularly effective in rapidly decreasing intracranial fluid volume and ICP. Its efficacy is primarily related to its effect on serum osmolality. A serum osmolality of 300–315 mOsm/L is generally considered desirable. Mannitol can transiently decrease blood pressure by virtue of its weak vasodilating properties, but its principal disadvantage is a transient increase in intravascular volume, which can precipitate pulmonary edema in patients with borderline cardiac or renal function. Mannitol should generally not be used in patients with intracranial aneurysms, arteriovenous malformations (AVMs), or intracranial hemorrhage until the cranium is opened. Osmotic diuresis in such instances can expand a hematoma as the volume of the normal brain tissue around it decreases. Rapid osmotic diuresis in elderly patients can also occasionally cause a subdural hematoma due to rupture of fragile bridging veins entering the sagittal sinus. Rebound edema may follow the use of mannitol; thus, it is ideally used in procedures (such

as a craniotomy for tumor resection) in which intracranial volume will be reduced.

Use of a loop diuretic (furosemide), although having a lesser maximal effect than mannitol and requiring up to 30 min, may have the additional advantage of directly decreasing formation of CSF. The combined use of mannitol and furosemide may be synergistic, but requires close monitoring of the serum potassium concentration.

Anesthesia & Craniotomy for Patients with Mass Lesions

Intracranial masses may be congenital, neoplastic (benign or malignant), infectious (abscess or cyst), or vascular (hematoma or arteriovenous malformation). Craniotomy is commonly undertaken for neoplasms of the brain. Primary tumors usually arise from glial cells (astrocytoma, oligodendroglioma, or glioblastoma), ependymal cells (ependymoma), or supporting tissues (meningioma, schwannoma, or choroidal papilloma). Childhood tumors include medulloblastoma, neuroblastoma, and astrocytoma.

1 Regardless of the cause, intracranial masses present according to growth rate, location, and ICP. Slowly growing masses are frequently asymptomatic for long periods (despite relatively large size), whereas rapidly growing ones may present when the mass remains relatively small. Common presentations include headache, seizures, a general decline in cognitive or specific neurological functions, and focal neurological deficits. Symptoms typical to supratentorial masses include seizures, hemiplegia, or aphasia, whereas symptoms typical of infratentorial may include cerebellar dysfunction (ataxia, nystagmus, and dysarthria) or brainstem compression (cranial nerve palsies, altered consciousness, or abnormal respiration). As ICP increases, signs of intracranial hypertension also develop (see above).

PREOPERATIVE MANAGEMENT

The preoperative evaluation for patients undergoing craniotomy should attempt to establish the presence

2 or absence of intracranial hypertension. Computed tomography (CT) and magnetic resonance imaging (MRI) scans should be reviewed for evidence of brain edema, a midline shift greater than 0.5 cm, and ventricular displacement or compression. The neurological examination should document mental status and any sensory or motor deficits. Medications should be reviewed with special reference to corticosteroid, diuretic, and anticonvulsant therapy. Laboratory evaluation should rule out corticosteroid-induced hyperglycemia, electrolyte disturbances due to diuretics, or abnormal secretion of antidiuretic hormone. Anticonvulsant blood concentrations may be measured, particularly when seizures are not well controlled.

Premedication

Sedative or opioid premedication is best avoided if intracranial hypertension is suspected. Hypercapnia secondary to respiratory depression increases ICP. Corticosteroids and anticonvulsant therapy should be continued until the time of surgery.

INTRAOPERATIVE MANAGEMENT

Monitoring

In addition to standard monitors, direct intraarterial pressure monitoring and bladder catheterization are used for most patients undergoing craniotomy. Rapid changes in blood pressure during anesthetic procedures, positioning, and surgical manipulation are best managed with guidance from continuous invasive monitoring of blood pressure. Moreover, arterial blood gas analyses are necessary to closely regulate $Paco_2$. Many neuroanesthesiologists zero the arterial pressure transducer at the level of the head (external auditory meatus)—instead of the right atrium—to facilitate calculation of cerebral perfusion pressure (CPP). End-tidal CO_2 measurements alone cannot be relied upon for precise regulation of ventilation; the arterial to end-tidal CO_2 gradient must be determined. Central venous access and pressure monitoring should be considered for patients requiring vasoactive drugs. Use of the internal jugular vein for access is theoretically problematic because of concern that the catheter might interfere with venous drainage from the brain. Some clinicians avoid this issue by passing a long catheter into the central circulation from the median basilic

vein. The external jugular, subclavian, and femoral veins may be suitable alternatives for intraoperative use. A bladder catheter is necessary because of the use of diuretics, the long duration of most neurosurgical procedures, and its utility in guiding fluid therapy. Neuromuscular function should be monitored on the unaffected side in patients with hemiparesis because the twitch response is often abnormally resistant on the affected side. Monitoring visual evoked potentials may be useful in preventing optic nerve damage during resections of large pituitary tumors. Additional monitors for surgery in the posterior fossa are described below.

Management of patients with intracranial hypertension may be guided by monitoring ICP perioperatively. Various ventricular, intraparenchymal, and subdural devices can be placed by neurosurgeons to provide measurements of ICP. The transducer should be zeroed to the same reference level as the arterial pressure transducer (usually the external auditory meatus; see above). A ventriculostomy catheter provides the added advantage of allowing removal of CSF to decrease ICP.

Induction

Induction of anesthesia and tracheal intubation are critical periods for patients with compromised intracranial pressure to volume relationships, particularly if there is an elevated ICP. Intracranial elastance can be improved by osmotic diuresis, dexamethasone, or removal of small volumes of CSF via a ventriculostomy drain. The goal of any technique should be to induce anesthesia and intubate the trachea without increasing ICP or compromising CBF. Arterial hypertension during induction increases CBV and promotes cerebral edema. Sustained hypertension can lead to marked increases in ICP, decreasing CPP and risking herniation. Excessive decreases in arterial blood pressure can be equally detrimental by compromising CPP.

The most common induction technique employs propofol together with modest hyperventilation to reduce ICP and blunt the noxious effects of laryngoscopy and intubation. Cooperative patients can be asked to hyperventilate during preoxygenation. All patients receive controlled ventilation once the propofol has been injected. A neuromuscular blocker (NMB) is given to facilitate ventilation and prevent straining or coughing, both of which can abruptly increase ICP. An intravenous opioid given with propofol blunts the sympathetic response, particularly in young patients. Esmolol, 0.5–1.0 mcg/kg, is effective in preventing tachycardia associated with intubation in lightly anesthetized patients.

The actual induction technique can be varied according to individual patient responses and coexisting diseases). Succinylcholine may theoretically increase ICP, particularly if intubation is attempted prior to the establishment of deep anesthesia. Succinylcholine, however, remains the agent of choice for rapid sequence induction or when there are concerns about a potentially difficult airway, as hypoxemia and hypercarbia are much more detrimental than any effect of succinylcholine to the patient with intracranial hypertension.

Hypertension during induction can be treated with β_1-blockers or by deepening the anesthetic with additional propofol. Modest concentrations of volatile agents (eg, sevoflurane) may also be used, provided that hyperventilation is also used. Sevoflurane best preserves autoregulation of CBF and produces limited vasodilatation; it may be the preferred volatile agent in patients with elevated ICP. Because of their potentially deleterious effect on CBV and ICP, vasodilators (eg, nicardipine, nitroprusside, nitroglycerin, and hydralazine) are avoided until the dura is opened. Hypotension is generally treated with incremental doses of vasopressors (eg, phenylephrine).

Positioning

Frontal, temporal, and parietooccipital craniotomies are performed in the supine position. The head is elevated 15–30° to facilitate venous and CSF drainage of CSF. The head may also be turned to the side to facilitate exposure. Excessive flexion or rotation of the neck impedes jugular venous drainage and can increase ICP. Before and after positioning, the tracheal tube should be secured, and all breathing circuit connections checked. The risk of unrecognized disconnections may be increased because the patient's airway will not be easily assessed after surgical draping; moreover, the operating table is usually turned 90° or 180° away from the anesthesiologist.

Maintenance of Anesthesia

Anesthesia can be maintained with inhalation anesthesia, total intravenous anesthesia techniques (TIVA), or a combination of an opioid and intravenous hypnotic (most often propofol) and a low-dose inhalation agent. Even though periods of stimulation are few, neuromuscular blockade is recommended—unless neurophysiological monitoring contradicts its use—to prevent straining, bucking, or movement. Increased anesthetic requirements can be expected during the most stimulating periods: laryngoscopy–intubation, skin incision, dural opening, periosteal manipulations, including Mayfied pin placement and closure. TIVA with remifentanil and propofol facilitates rapid emergence and immediate neurological assessment. Likewise, the α_2-agonist dexmedetomidine can be employed during both asleep and awake craniotomies to similar effect.

Hyperventilation should be continued intraoperatively to maintain $Paco_2$ at roughly 30–35 mm Hg. Lower $Paco_2$ tensions provide little additional benefit and may be associated with cerebral ischemia and impaired oxygen dissociation from hemoglobin. Positive end-expiratory pressure (PEEP) and ventilatory patterns resulting in high mean airway pressures (a low rate with large tidal volumes) should be avoided because of a potentially adverse effect on ICP by increasing central venous pressure and the potential for lung injury. Hypoxic patients may require PEEP and increased mean airway pressures; in such patients, the effect of PEEP on ICP is variable.

Intravenous fluid replacement should be limited to glucose-free isotonic crystalloid or colloid solutions. Hyperglycemia is common in neurosurgical patients (corticosteroid effect) and has been implicated in increasing ischemic brain injury. Colloid solutions can be used to restore intravascular volume deficits, whereas isotonic crystalloid solutions are used for maintenance fluid requirements. Neurosurgical procedures are often associated with "occult" blood loss (underneath surgical drapes or on the floor).

Emergence

Most patients undergoing elective craniotomy can be extubated at the end of the procedure, provided that neurological function is intact. Patients who will remain intubated should be sedated to prevent agitation. Extubation in the operating room requires special handling during emergence. Straining or bucking on the tracheal tube may precipitate intracranial hemorrhage or worsen cerebral edema. As the skin is being closed, the patient should resume breathing spontaneously. Should the patient's head be secured in a Mayfield pin apparatus, care must be taken to avoid any patient motions (eg, "bucking on the tube"), which could promote neck or cranial injuries. After the head dressing is applied and full access to the patient is regained (the table is turned back to its original position as at induction), any anesthetic gases are completely discontinued, and the neuromuscular blockade is reversed. Rapid awakening facilitates immediate neurological assessment and can generally be expected following an appropriate anesthetic. Delayed awakening may be seen following opioid or sedative overdose, when the end-tidal concentration of the volatile agent remains >.2 minimum alveolar concentration (MAC), because of various metabolic derangements, or when there is a perioperative neurological injury. Patients may need to be transported to the CT scanner directly from the operating room for evaluation when they do not respond as predicted. Immediate reexploration may be required. Most patients are taken to the intensive care unit postoperatively for close monitoring of neurological function.

Anesthesia for Surgery in the Posterior Fossa

Craniotomy for a mass in the posterior fossa presents a unique set of potential problems: obstructive hydrocephalus, possible injury to vital brainstem centers, pneumocephalus, and, with unusual positioning, postural hypotension and **venous air embolism**.

Obstructive Hydrocephalus

Infratentorial masses can obstruct CSF flow through the fourth ventricle or the cerebral aqueduct of Sylvius. Small but critically located lesions can markedly increase ICP. In such cases, a ventriculostomy is

often performed under local anesthesia to decrease ICP prior to induction of general anesthesia.

Brain Stem Injury

3 Operations in the posterior fossa can injure vital circulatory and respiratory brainstem centers and cranial nerves or their nuclei. Such injuries may occur as a result of direct surgical trauma or ischemia from retraction or other interruptions of the blood supply. Damage to respiratory centers is said to nearly always produce circulatory changes; therefore, abrupt changes in blood pressure, heart rate, or cardiac rhythm should alert the anesthesiologist to the possibility of such an injury. Such changes should be communicated to the surgeon. Isolated damage to respiratory centers may rarely occur without premonitory circulatory signs during operations in the floor of the fourth ventricle. Historically, some clinicians have employed spontaneous ventilation during these procedures as an additional monitor of brain function. At completion of the surgery, brainstem injuries may present as an abnormal respiratory pattern or an inability to maintain a patent airway following extubation. Monitoring brainstem auditory evoked potentials may be useful in preventing eighth nerve damage during resections of acoustic neuromas. Electromyography is also used to avoid injury to the facial nerve, but requires incomplete neuromuscular blockade intraoperatively.

Positioning

Although most explorations of the posterior fossa can be performed with the patient in either a modified lateral or prone position, the sitting position may be preferred by some surgeons.

The patient is actually semirecumbent in the standard sitting position (Figure 27–1); the back is elevated to 60°, and the legs are elevated with the knees flexed. The head is fixed in a three-point holder with the neck flexed; the arms remain at the sides with the hands resting on the lap.

Careful positioning and padding helps avoid injuries. Pressure points, such as the elbows, ischial spines, heels, and forehead, must be protected. Excessive neck flexion has been associated with swelling of the upper airway (due to venous obstruction), and, rarely, quadriplegia (due to compression of the cervical spinal cord). Preexisting cervical spinal stenosis probably predisposes patients to the latter injury.

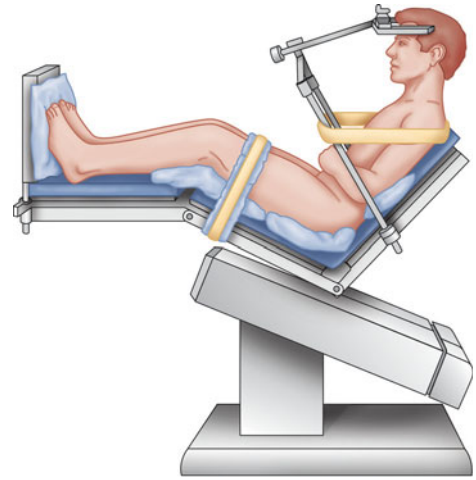

FIGURE 27–1 The sitting position for craniotomy.

Pneumocephalus

The sitting position increases the likelihood of pneumocephalus. In this position, air readily enters the subarachnoid space, as CSF is lost during surgery. In patients with cerebral atrophy, drainage of CSF is marked; air can replace CSF on the surface of the brain and in the lateral ventricles. Expansion of a pneumocephalus following dural closure can compress the brain. Postoperative pneumocephalus can cause delayed awakening and continued impairment of neurological function. Because of these concerns, nitrous oxide is rarely used for sitting craniotomies. (see also below).

Venous Air Embolism

4 Venous air embolism can occur when the pressure within an open vein is subatmospheric. These conditions may exist in any position (and during any procedure) whenever the wound is above the level of the heart. The incidence of venous air embolism is greater during sitting craniotomies (20% to 40%) than in craniotomies in any other

position. Entry into large cerebral venous sinuses increases the risk.

The physiological consequences of venous air embolism depend on the volume and the rate of air entry and whether the patient has a right-to-left intracardiac shunt (eg, patent foramen ovale [10% to 25% incidence]). The latter are important because they can facilitate passage of air into the arterial circulation (**paradoxical air embolism**). Modest quantities of air bubbles entering the venous system ordinarily lodge in the pulmonary circulation, where they are eventually absorbed. Small quantities of embolized air are well tolerated by most patients. When the amount entrained exceeds the rate of pulmonary clearance, pulmonary artery pressure rises progressively. Eventually, cardiac output decreases in response to increases in right ventricular afterload. Preexisting cardiac or pulmonary disease enhances the effects of venous air embolism; relatively small amounts of air may produce marked hemodynamic changes. Nitrous oxide, by diffusing into the bubbles and increasing their volume, can markedly accentuate the effects of even small amounts of entrained air. The dose for lethal venous air embolism in animals receiving nitrous oxide anesthesia is one-third to one-half that of control animals not receiving nitrous oxide.

Definitive signs of venous air embolism are often not apparent until large volumes of air have been entrained. A decrease in end-tidal CO_2 or arterial oxygen saturation might be noticed prior to hemodynamic changes. Arterial blood gas values may show only slight increases in $Paco_2$ as a result of increased pulmonary dead space (areas with normal ventilation but decreased perfusion). Conversely, major hemodynamic manifestations, such as sudden hypotension, can occur well before hypoxemia is noted. Moreover, large amounts of intracardiac air impair tricuspid and pulmonic valve function and can produce sudden circulatory arrest by obstructing right ventricular outflow.

Paradoxic air embolism can result in a stroke or coronary occlusion, which may be apparent only postoperatively. Paradoxic air emboli are more likely to occur in patients with right-to-left intracardiac shunts, particularly when the normal transatrial (left > right) pressure gradient is reversed. Some studies suggest that a right > left pressure gradient can develop at some time during the cardiac cycle, even when the overall mean gradient remains left > right.

A. Central Venous Catheterization

A properly positioned central venous catheter can be used to aspirate entrained air, but there is only limited evidence that this influences outcomes after venous air embolism. Some clinicians have considered right atrial catheterization mandatory for sitting craniotomies, but this is a minority viewpoint.

5 Optimal recovery of air following venous air embolism is provided by a multiorificed catheter positioned at the junction between the right atrium and the superior vena cava. Confirmation of correct catheter positioning can be accomplished by intravascular electrocardiography, radiography, or transesophageal echocardiography (TEE). Intravascular electrocardiography is accomplished by using the saline-filled catheter as a "V" lead. Correct high atrial position is indicated by the appearance of a biphasic P wave. If the catheter is advanced farther into the heart, the P wave changes from a biphasic to a undirectional deflection. A right ventricular or pulmonary artery waveform may also be observed when the catheter is connected to a pressure transducer and advanced too far.

B. Monitoring for Venous Air Embolism

The most sensitive monitors available should be used. Detecting even small amounts of venous air embolism is important because it allows surgical control of the entry site before additional air is entrained. Currently, the most sensitive intraoperative monitors are TEE and precordial Doppler sonography. These monitors can detect air bubbles as small as 0.25 mL. TEE has the added benefit of detecting the volume of the bubbles and any transatrial passage through a patent foramen ovale, as well as evaluating any effect venous air embolism may have on cardiac function. Doppler methods employ a probe over the right atrium (usually to the right of the sternum and between the third and sixth ribs). Interruption of the regular swishing of the Doppler signal by sporadic roaring sounds indicates venous air embolism. Changes in end-tidal respiratory gas concentrations are less sensitive but are important monitors that can

also detect venous air embolism before overt clinical signs are present. Venous air embolism causes a sudden decrease in end-tidal CO_2 tension in proportion to the increase in pulmonary dead space; however, decreases can also be seen with hemodynamic changes unrelated to venous air embolism, such as decreased cardiac output. A reappearance (or increase) of nitrogen in expired gases may also be seen with venous air embolism. Changes in blood pressure and heart sounds ("mill wheel" murmur) are late manifestations of venous air embolism.

C. Treatment of Venous Air Embolism

1. The surgeon should be notified so that he or she can flood the surgical field with saline or pack it with wet gauzes and apply bone wax to the skull edges until the entry site is identified and occluded.
2. Nitrous oxide (if used) should be discontinued, and the inhalation anesthetic should be delivered in 100% oxygen.
3. If a central venous catheter is present, it should be aspirated in an attempt to retrieve the entrained air.
4. Intravascular volume infusion should be given to increase central venous pressure.
5. Vasopressors should be given to treat hypotension.
6. Bilateral jugular vein compression, by increasing cranial venous pressure, may slow air entrainment and cause back bleeding, which might help the surgeon identify the entry point of the embolus.
7. Some clinicians advocate PEEP to increase cerebral venous pressure; however, reversal of the normal transatrial pressure gradient may promote paradoxic embolism in a patient with incomplete closure of the foramen ovale.
8. If the above measures fail, the patient should be placed in a head-down position, and the wound should be closed quickly.
9. Persistent circulatory arrest necessitates the supine position and institution of resuscitation efforts using advanced cardiac life support algorithms.

Anesthesia for Stereotactic Surgery

Stereotaxis can be employed in treating involuntary movement disorders, intractable pain, and epilepsy and can also be used when diagnosing and treating tumors that are located deep within the brain.

These procedures are often performed under local anesthesia to allow evaluation of the patient. Propofol or dexmedetomidine infusions are routinely used for sedation and amnesia. Sedation should be omitted, however, if the patient already has increased ICP. The ability to rapidly provide controlled ventilation and general anesthesia for emergency craniotomy is mandatory, but is complicated by the platform and localizing frame that is attached to the patient's head for the procedure. Although mask ventilation or ventilation through a laryngeal mask airway (LMA) or orotracheal intubation might be readily accomplished in an emergency, awake intubation with a fiberoptic bronchoscope prior to positioning and surgery may be the safest approach when intubation is necessary for a patient whose head is already in a stereotactic head frame.

Functional neurosurgery is increasingly performed for removal of lesions adjacent to speech and other vital brain centers. Sometimes patients are managed with an asleep–awake–asleep technique, with or without instrumentation of the airway. Such operations require the patient to be awake to participate in cortical mapping to identify key speech centers, such as Broca's area. Patients sleep during the painful periods of surgery (ie, during opening and closure). LMAs are often employed to assist airway management during the asleep portions of these surgeries.

Patients undergo deep brain stimulator insertion for control of movement and other disorders. A stimulator electrode is placed via a burr hole using radiologic guidance to establish coordinates for electrode placement. A microelectrode recording (MER) is obtained to determine the correct placement of the stimulator in brain structures. The effect of stimulation upon the patient is noted. Sedative medications can adversely affect MER potentials, complicating the location of the correct depth of

TABLE 27–1 Advantages and disadvantages of drugs used for conscious sedation.

Agents	Advantages	Disadvantages
GABA receptor agonists		
Benzodiazepines	Anxiolysis	Large dose abolishes MER Alters the threshold for stimulation Induces dyskinesia
Propofol	Widely used Short acting Predictable emergence profile	Abolish tremors Attenuation of MER Unpredictable dosing in patients with Parkinson disease Induces dyskinesia Tendency to cause sneezing
Opioids		
Fentanyl	? Minimal effect on MER	Rigidity
Remifentanil	Short acting	Suppression of tremors
Alpha-2 agonist		
Dexmedetomidine	Non-GABA-mediated action Less effect on MER Anxiolysis and analgesic effects Sedation—easily arousable Does not ameliorate clinical signs of Parkinsonism Maintains hemodynamic stability Preserves respiration	High doses can abolish MER Hypotension, bradycardia

MER, microelectrode recording; GABA, γ-aminobutyric acid.

Modified, with permission, from Venkatraghavan L, Luciano M, Manninen P: Anesthetic management of patients undergoing deep brain stimulator insertion. Anesth Analg 2010;110:1138.

stimulator placement. Dexmedetomidine has been used to provide sedation to these patients; however, during MER and stimulation testing, sedative infusions should be discontinued to facilitate patient participation in determining correct electrode placement (Table 27–1).

Anesthesia for Head Trauma

Head injuries are a contributory factor in up to 50% of deaths due to trauma. Most patients with head trauma are young, and many (10% to 40%) have associated intraabdominal or intrathoracic injuries, long bone fractures, and/or spinal injuries. The outcome from a head injury is dependent not only on the extent of the neuronal damage at the time of injury, but also on the occurrence of any secondary insults. These additional insults include: (1) systemic factors such as hypoxemia, hypercapnia, or hypotension; (2) formation and expansion of an epidural, subdural, or intracerebral hematoma; and (3) sustained intracranial hypertension. Surgical and anesthetic management of these patients is directed at preventing these secondary insults. The **Glasgow Coma Scale (GCS) score** (Table 27–2) generally correlates well with the severity of injury and outcome. A GCS score of 8 or less on admission is associated with approximately 35% mortality. Evidence of greater than a 5-mm midline shift (on imaging) and ventricular compression on imaging are associated with substantially increased morbidity.

Specific lesions include skull fractures, subdural and epidural hematomas, brain contusions (including intracerebral hemorrhages), penetrating head injuries, and traumatic vascular occlusions and dissections. The presence of a skull fracture greatly increases the likelihood of an intracranial lesion. Linear skull fractures are commonly associated

TABLE 27–2 Glasgow coma scale.

Category	Score
Eye opening	
Spontaneous	4
To speech	3
To pain	2
Nil	1
Best motor response	
To verbal command	
Obeys	6
To pain	
Localizes	5
Withdraws	4
Decorticate flexion	3
Extensor response	2
Nil	1
Best verbal response	
Oriented	5
Confused conversation	4
Inappropriate words	3
Incomprehensible sounds	2
Nil	1

with subdural or epidural hematomas. Basilar skull fractures may be associated with CSF rhinorrhea, pneumocephalus, cranial nerve palsies, or even a cavernous sinus–carotid artery fistula. Depressed skull fractures often present with an underlying brain contusion. Contusions may be limited to the surface of the brain or may involve hemorrhage in deeper hemispheric structures or the brainstem. Deceleration injuries often produce both coup (frontal) and contrecoup (occipital) lesions. Epidural and subdural hematomas can occur as isolated lesions, as well as in association with cerebral contusions (more commonly with subdural than epidural lesions).

Operative treatment is usually elected for depressed skull fractures; evacuation of epidural, subdural, and some intracerebral hematomas; and debridement of penetrating injuries. Decompressive craniectomy is used to provide room for cerebral swelling. The cranium is subsequently reconstructed following resolution of cerebral edema.

ICP monitoring is usually indicated in patients with lesions associated with intracranial hypertension: large contusions, mass lesions, intracerebral hemorrhage, or evidence of edema on imaging studies. ICP monitoring should also be considered in patients with signs of intracranial hypertension who are undergoing nonneurological procedures. Intracranial hypertension should be treated with moderate hyperventilation, mannitol, pentobarbital, or propofol Studies suggest that sustained increases in ICP of greater than 60 mm Hg result in severe disability or death. Unlike treatment following spinal cord trauma, multiple randomized trials have failed to detect the efficacy of early use of large doses of glucocorticoids in patients with head trauma.

PREOPERATIVE MANAGEMENT

Anesthetic care of patients with severe head trauma begins in the emergency department. Measures to ensure patency of the airway, adequacy of ventilation and oxygenation, and correction of systemic hypotension should go forward simultaneously with neurological and trauma surgical evaluation. Airway obstruction and hypoventilation are common. Up to 70% of such patients have hypoxemia, which may be complicated by pulmonary contusion, fat emboli, or neurogenic pulmonary edema. The latter is attributed to marked systemic and pulmonary hypertension secondary to intense sympathetic nervous system activity. Supplemental oxygen should be given to all patients while the airway and ventilation are evaluated. All patients must be assumed to have a cervical spine injury (up to 10% incidence) until the contrary is proven radiographically. Patients with obvious hypoventilation, an absent gag reflex, or a persistent score below 8 on the GCS (Table 27–2) require tracheal intubation and hyperventilation. All other patients should be carefully observed for deterioration.

Intubation

All patients should be regarded as having a full stomach and should have cricoid pressure applied during ventilation and tracheal intubation. In-line stabilization should be used during airway manipulation to maintain the head in a neutral position, unless radiographs confirm that there is no cervical

spine injury. Following preoxygenation and hyperventilation by mask, the adverse effects of intubation on ICP are blunted by prior administration of propofol, 1.5–3.0 mg/kg, and a rapid-onset NMB. Succinylcholine may produce mild and transient increases in ICP in patients with closed head injury; however, the necessity for expeditious airway management trumps these concerns. Rocuronium is often used to facilitate intubation. Video laryngoscopy performed with in-line stabilization generally permits neutral position intubation of the trauma patient. An intubating bougie should be available to facilitate tube placement. If a difficult intubation is encountered with video laryngoscopy, fiberoptic or other techniques (eg, intubating LMA) can be attempted. If airway attempts are unsuccessful, a surgical airway should be obtained. Blind nasal intubation is contraindicated in the presence of a basilar skull fracture, which is suggested by CSF rhinorrhea or otorrhea, hemotympanum, or ecchymosis into periorbital tissues (raccoon sign) or behind the ear (Battle's sign).

Hypotension

Hypotension in the setting of head trauma is nearly always related to other associated injuries (often intraabdominal). Bleeding from scalp lacerations may be responsible in children. Hypotension may be seen with spinal cord injuries because of the sympathectomy associated with spinal shock. In a patient with head trauma, correction of hypotension and control of any bleeding take precedence over radiographic studies and definitive neurosurgical treatment because systolic arterial blood pressures of less than 80 mm Hg predict a poor outcome. Glucose-containing or hypotonic solutions should not be used (see above). Otherwise, a mix of colloid, crystalloid, and blood products can be administered as necessary. Massive blood loss in the patient with multiple injuries should result in activation of a massive transfusion protocol to provide a steady supply of platelets, fresh frozen plasma, and packed red blood cells. Invasive monitoring of arterial pressure, central venous pressure, and ICP are valuable, but should not delay diagnosis and treatment. Arrhythmias and electrocardiographic abnormalities in the T wave, U wave, ST segment, and QT interval are common following head injuries, but are not necessarily associated with cardiac injury; they likely represent altered autonomic function.

Diagnostic Studies

The choice between operative and medical management of head trauma is based on radiographic and clinical findings. Patients should be stabilized prior to any CT or other imaging studies. Critically ill patients should be closely monitored during such studies. Restless or uncooperative patients may additionally require general anesthesia. Sedation without control of the airway should generally be avoided because of the risk of further increases in ICP from hypercapnia or hypoxemia.

INTRAOPERATIVE MANAGEMENT

Anesthetic management is generally similar to that for other mass lesions associated with intracranial hypertension. Management of the airway is discussed above. Invasive monitoring should be established, if not already present, but should not delay surgical decompression in a rapidly deteriorating patient.

Anesthetic technique and agents are designed to preserve cerebral perfusion and mitigate increases in intracranial pressure. Hypotension may occur after induction of anesthesia as a result of the combined effects of vasodilation and hypovolemia and should be treated with an α-adrenergic agonist and volume infusion if necessary. Subsequent hypertension is common with surgical stimulation, but may also occur with acute elevations in ICP. The latter may be associated with bradycardia (Cushing reflex).

Hypertension can be treated with additional doses of the induction agent, with increased concentrations of an inhalation anesthetic or vasodilators. β-Adrenergic blockade is usually effective in controlling hypertension associated with tachycardia. CPP should be maintained between 70 and 110 mm Hg. Vasodilators should be avoided until the dura

is opened. Hyperventilation to a $Paco_2$ <30 should be avoided in trauma patients to avoid excessive decreases in oxygen delivery.

Disseminated intravascular coagulation occasionally may be seen with severe head injuries. Such injuries cause the release of large amounts of brain thromboplastin and may also be associated with the acute respiratory distress syndrome. Pulmonary aspiration and neurogenic pulmonary edema may also be responsible for deteriorating lung function. PEEP can be applied on the ventilator. When PEEP is used, ICP monitoring can be useful to confirm an adequate CPP. Diabetes insipidus, characterized by excessive dilute urine, is frequently seen following injuries to the pituitary stalk. Other likely causes of polyuria should be excluded and the diagnosis confirmed by measurement of urine and serum osmolality prior to treatment with fluid restriction and vasopressin Gastrointestinal bleeding is common in patients not receiving prophylaxis; it is usually due to stress ulceration.

The decision whether to extubate the trachea at the conclusion of the surgical procedure depends on the severity of the injury, the presence of concomitant abdominal or thoracic injuries, preexisting illnesses, and the preoperative level of consciousness. Young patients who were conscious preoperatively may be extubated following the removal of a localized lesion, whereas patients with diffuse brain injury should remain intubated. Moreover, persistent intracranial hypertension requires continued paralysis, sedation, and hyperventilation.

Anesthesia & Craniotomy for Intracranial Aneurysms & Arteriovenous Malformations

Saccular aneurysms and AVMs are common causes of nontraumatic intracranial hemorrhages. Surgical or interventional neuroradiologic treatment may be undertaken either electively to prevent hemorrhage or emergently to prevent further complications once hemorrhage has taken place. Other nontraumatic hemorrhages (eg, from hypertension, sickle cell disease, or vasculitis) are usually treated medically.

CEREBRAL ANEURYSMS

Preoperative Considerations

Cerebral aneurysms typically occur at the bifurcation of the large arteries at the base of the brain; most are located in the anterior circle of Willis. Approximately 10% to 30% of patients have more than one aneurysm. The general incidence of saccular aneurysms in some estimates is reported to be 5%, but only a minority of those with aneurysms will have complications. Rupture of a saccular aneurysm is the most common cause of subarachnoid hemorrhage. The acute mortality following rupture is approximately 10%. Of those that survive the initial hemorrhage, about 25% die within 3 months from delayed complications. Moreover, up to 50% of survivors are left with neurological deficits. As a result, the emphasis in management is on prevention of rupture. Unfortunately, most patients present only after rupture has already occurred.

Unruptured Aneurysms

Patients may present with prodromal symptoms and signs suggesting progressive enlargement. The most common symptom is headache, and the most common physical sign is a third-nerve palsy. Other manifestations could include brainstem dysfunction, visual field defects, trigeminal nerve dysfunction, cavernous sinus syndrome, seizures, and hypothalamic–pituitary dysfunction. The most commonly used techniques to diagnose an aneurysm are MRI angiography, angiography, and helical CT angiography. Following diagnosis, patients are brought to the operating room, or more likely the radiology suite, for elective clipping or obliteration of the aneurysm. Most patients are in the 40- to 60-year-old age group and in otherwise good health.

Ruptured Aneurysms

Ruptured aneurysms usually present acutely as subarachnoid hemorrhage. Patients typically complain of a sudden severe headache without focal neurological deficits, but often associated with nausea and vomiting. Transient loss of consciousness may

TABLE 27–3 Hunt and Hess grading scale for SAH.

Grade	Clinical Description
I	Asymptomatic or minimal headache and slight nuchal rigidity
II	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
III	Drowsiness, confusion, or mild focal deficit
IV	Stupor, moderate to severe hemiparesis, and possibly early decerebrate rigidity and vegetative disturbances
V	Deep coma, decerebrate rigidity, and moribund appearance

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TABLE 27–4 World Federation of Neurological Surgeons Grading scale for aneurismal SAH.

Grade	GCS score	Motor Deficit[1]
I	15	Absent
II	13 or 14	Absent
III	13 or 14	Present
IV	7-12	Present or absent
V	3-6	Present or absent

GCS, Glasgow Coma Scale.

[1]Excludes cranial neuropathies, but includes dysphasia.

Reproduced, with permission, from Priebe H-J: Aneurysmal subarachnoid haemorrhage and the anaesthetist. Br J Anaesth 2007;99:102.

occur and may result from a sudden rise in ICP and precipitous drop in CPP. If ICP does not decrease rapidly after the initial sudden increase, death usually follows. Large blood clots can cause focal neurological signs in some patients. Minor bleeding may cause only a mild headache, vomiting, and nuchal rigidity. Unfortunately, even minor bleeding in the subarachnoid space seems to predispose to delayed complications. The severity of subarachnoid hemorrhage (SAH) is graded according to the Hunt and Hess scale (Table 27–3), as well as the World Federation of Neurological Surgeons Grading Scale of SAH (Table 27–4). The Fisher grading scale, which uses CT to assess the amount of blood detected, gives the best indication of the likelihood of the development of cerebral vasospasm and patient outcome (Table 27–5).

Delayed complications include cerebral vasospasm, rerupture, and hydrocephalus. Cerebral vasospasm occurs in 30% of patients (usually after 4–14 days) and is a major cause of morbidity and mortality. Manifestations of vasospasm are due to cerebral ischemia and infarction and depend on the severity and distribution of the involved vessels. The Ca^{2+} channel antagonist nimodipine may antagonize vasospasm. Both transcranial Doppler and brain tissue oxygen monitoring can be used to guide vasospasm therapy (Figure 27–2). Increased velocity of flow >200cm/sec is indicative of severe spasm. The Lindegaard ratio compares the blood velocity of the cervical carotid artery with that of the middle cerebral artery. A ratio >3 is likewise indicative of severe spasm. Brain tissue oxygen tension less than 20 mm Hg is also worrisome. **In patients with symptomatic vasospasm with an inadequate response to nimodipine, intravascular volume expansion and induced hypertension ("triple H" therapy: hypervolemia, hemodilution, and hypertension) are added as part of the therapeutic regimen**. Refractory vasospasm may be treated with infusion of papaverine, infusion of nicardipine, or angioplasty. However, radiologic improvement in the vessel diameter does not necessarily correlate with an improvement in clinical status.

TABLE 27–5 Fisher grading scale of cranial computerized tomography (CCT).

Grade	Findings on CCT
1	No subarachnoid blood detected
2	Diffuse or vertical layers ≤ 1mm
3	Localized clot and/or vertical layer > 1mm
4	Intracerebral or intraventricular clot with diffuse or no subarachnoid haemorrhage

Reproduced, with permission, from Priebe H-J: Aneurysmal subarachnoid haemorrhage and the anaesthetist. Br J Anaesth 2007;99:102.

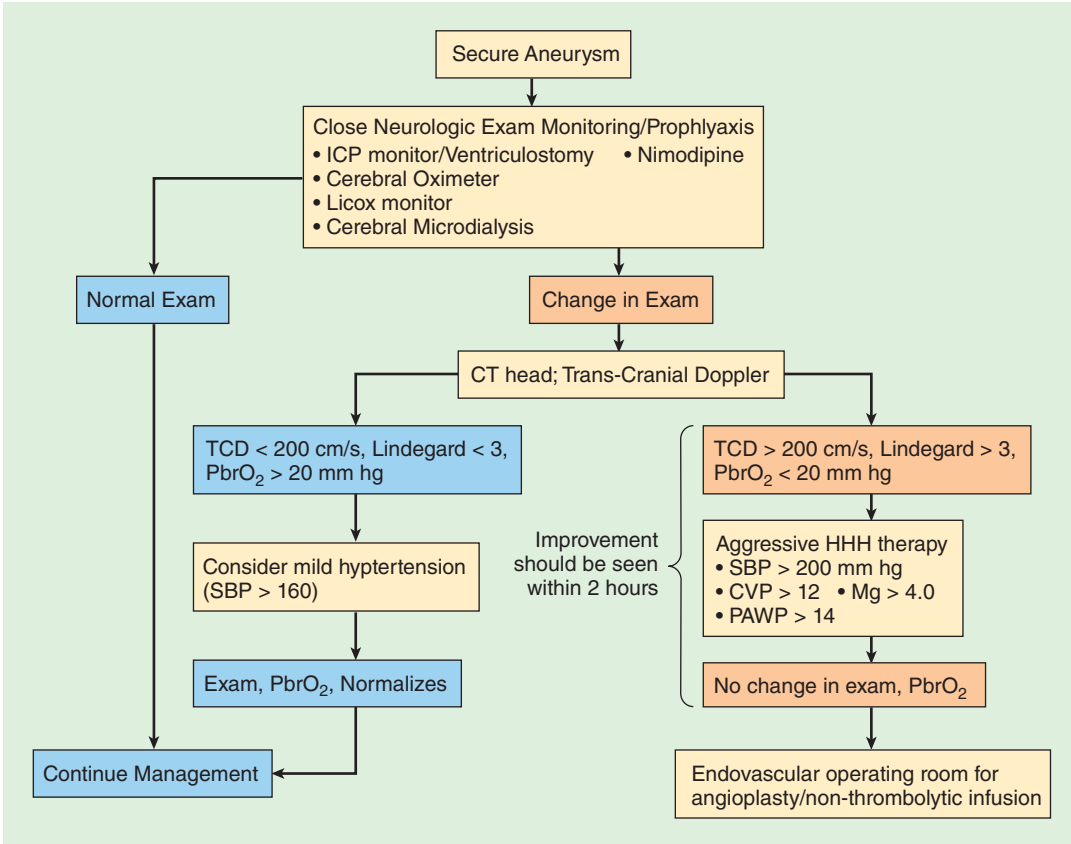

FIGURE 27–2 Schematic diagram of algorithm for management of vasospasm and delayed ischemic neurological deficit after aneurismal SAH. (Reproduced, with permission, from Bell RS, Vo AH, Veznedaroglu E, Armonda RA. The endovascular operating room as an extension of the intensive care unit: changing strategies in the management of neurovascular disease. Neurosurgery 2006;59:S3–56.)

PREOPERATIVE MANAGEMENT

In addition to assessing neurological findings, the preoperative evaluation should include a search for coexisting diseases, such as hypertension and renal, cardiac, or ischemic cerebrovascular disease. Electrocardiographic abnormalities are commonly seen in patients with subarachnoid hemorrhage, but do not necessarily reflect underlying heart disease. However, increases of cardiac troponin during SAH are associated with myocardial injury and may herald a poor outcome. Most conscious patients with normal ICP are sedated following rupture to prevent rebleeding; such sedation should be continued until induction of anesthesia. Patients with persistent elevation in ICP should receive little or no premedication to avoid hypercapnia.

INTRAOPERATIVE MANAGEMENT

Aneurysm surgery can result in exsanguinating hemorrhage as a consequence of rupture or rebleeding. Blood should be available prior to the start of these operations.

Regardless of the anesthetic technique employed, anesthetic management should focus on preventing rupture (or rebleeding) and avoiding factors that promote cerebral ischemia or vasospasm.

Intraarterial and central venous pressure monitoring are useful. Sudden increases in blood pressure with tracheal intubation or surgical stimulation should be avoided. Judicious intravascular volume loading permits surgical levels of anesthesia without excessive decreases in blood pressure. Because calcium channel blockers, angiotensin receptor blockers, and ACE inhibitors cause systemic vasodilation and reduce systemic vascular resistance, patients receiving these agents preoperatively may be particularly prone to hypotension. Hyperventilation is unlikely to overcome ischemia-induced vasodilation. Once the dura is opened, mannitol is often given to facilitate surgical exposure and reduce the need for surgical retraction. Rapid decreases in ICP prior to dural opening may promote rebleeding by removing a tamponading effect on the aneurysm.

Elective (controlled) hypotension has been used in aneurysm surgery. Decreasing mean arterial blood pressure reduces the transmural tension across the aneurysm, making rupture (or rebleeding) less likely and facilitating surgical clipping. Controlled hypotension can also decrease blood loss and improve surgical visualization in the event of bleeding. The combination of a slightly head-up position with a volatile anesthetic enhances the effects of any of the commonly used hypotensive agents. Should accidental rupture of the aneurysm occur, the surgeon may request transient hypotension to facilitate control of the bleeding aneurysm.

Technical improvements in temporary vascular clips have enabled surgeons to use them more often to interrupt blood flow during aneurysm surgery; induced hypertension is often requested when temporary clips are applied. Neurophysiologic monitoring may be employed during aneurysm surgery to identify potential ischemia during temporary clip application.

Mild hypothermia has been used to protect the brain during periods of prolonged or excessive hypotension or vascular occlusion; however, its efficacy has been questioned. Rarely, hypothermic circulatory arrest is used for large basilar artery aneurysms.

Depending on neurological condition, most patients should be extubated at the end of surgery. Extubation should be handled similarly to other craniotomies (see above). A rapid awakening allows neurological evaluation in the operating room, prior to transfer to the intensive care unit.

The anesthetic concerns of patients taken for aneursymal coiling in the neurointerventional suite are similar to those of surgical interventions. General anesthesia is employed. Patients require heparin anticoagulation and radiologic contrast. Communication with the surgeon or neuroradiologist as to the desired activated clotting time and need for protamine reversal is essential. Moreover, anesthesia staff in the neuroradiology suite must be prepared to manipulate and monitor the blood pressure, as with an open surgical procedure.

ARTERIOVENOUS MALFORMATIONS

AVMs cause intracerebral hemorrhage more often than subarachnoid hemorrhage. These lesions are developmental abnormalities that result in arteriovenous fistulas; they typically grow in size with time. AVMs may present at any age, but bleeding is most common between 10 and 30 years of age. Other common presentations include headache and seizures. The combination of high blood flow with low vascular resistance can rarely result in high-output cardiac failure. Acutely, neuroradiologists try to embolize AVMs. When neuroradiological interventions are not successful or available, surgical excision may be undertaken. Neuroradiological embolization employs various coils, glues, and balloons to obliterate the AVM. Risks include embolization into cerebral arteries feeding the normal brain, as well as systemic or pulmonary embolism.

Anesthetic management of patients undergoing surgical treatment of AVMs may be complicated by extensive blood loss. Venous access with multiple large-bore cannulas is necessary. Embolization may be carried out prior to surgery to reduce operative blood loss. Hyperventilation and mannitol may be used to facilitate surgical access. Hyperemia and swelling can develop following resection, possibly because of altered autoregulation in the remaining normal brain. Emergence hypertension is typically controlled using β_1-blockers to avoid any vasodilator induced increase in CBF.

Anesthesia for Surgery on the Spine

Spinal surgery is most often performed for symptomatic nerve root or cord compression secondary to trauma or degenerative disorders. Compression may occur from protrusion of an intervertebral disk or osteophytic bone (spondylosis) into the spinal canal or an intervertebral foramen. Prolapse of an intervertebral disk usually occurs at either the fourth or fifth lumbar or the fifth or sixth cervical levels in patients 30–50 years old. Spondylosis tends to affect the lower cervical spine more than the lumbar spine and typically afflicts older patients. Operations on the spinal column can help correct deformities (eg, scoliosis), decompress the cord, and fuse the spine if disrupted by trauma. Spinal surgery may also be performed to resect a tumor or vascular malformation or to drain an abscess or hematoma.

PREOPERATIVE MANAGEMENT

Preoperative evaluation should focus on any existing anatomic abnormalities and limited neck movements due to disease, traction, or braces that might complicate airway management and necessitate special techniques. Neurological deficits should be documented. Neck mobility should be assessed in all patients presenting for spine surgery at any level. Patients with unstable cervical spines can be managed with either awake fiberoptic intubation or asleep intubation with in-line stabilization.

INTRAOPERATIVE MANAGEMENT

For many of these procedures, anesthetic management is complicated by the use of the prone position. Spinal operations involving multiple levels, fusion, and instrumentation are also complicated by the potential for large intraoperative blood losses; a red cell salvage device is often used. Excessive distraction during spinal instrumentation (Harrington rod or pedicle screw fixation) can additionally injure the spinal cord. Transthoracic approaches to the spine require one-lung ventilation. Anterior/posterior approaches require the patient to be repositioned in the middle of surgery.

Positioning

Most spine surgical procedures are carried out in the prone position. The supine position may be used for an anterior approach to the cervical spine, making anesthetic management easier, but increasing the risk of injury to the trachea, esophagus, recurrent laryngeal nerve, sympathetic chain, carotid artery, or jugular vein. A sitting (for cervical spine procedures) or lateral decubitus (most commonly for lumbar spine procedures) position may occasionally be used.

Following induction of anesthesia and tracheal intubation in the supine position, the patient is turned to the prone position. Care must be taken to maintain the neck in a neutral position. Once in the prone position, the head may be turned to the side (not exceeding the patient's normal range of motion) or (more commonly) can remain face down on a cushioned holder. Caution is necessary to avoid corneal abrasions or retinal ischemia from pressure on either globe, or pressure injuries of the nose, ears, forehead, chin, breasts (females), or genitalia (males). The chest should rest on parallel rolls (of foam, gel, or other padding) or special supports—if a frame is used—to facilitate ventilation. The arms may be tucked by the sides in a comfortable position or extended with the elbows flexed (avoiding excessive abduction at the shoulder).

Turning the patient prone is a critical maneuver, sometimes complicated by hypotension. Abdominal compression, particularly in obese patients, may impede venous return and contribute to excessive intraoperative blood loss from engorgement of epidural veins. Prone positioning that permits the abdomen to hang freely can mitigate this increase in venous pressure. Deliberate hypotension has been advocated in the past to reduce bleeding associated with spine surgery. However, this should only be undertaken with a full understanding that controlled hypotension may increase the risk of perioperative vision loss (POVL).

POVL occurs secondary to:

- Ischemic optic neuropathy
- Perioperative glaucoma
- Cortical hypotension/embolism

Prolonged surgery in a head-down position, major blood loss, relative hypotension, diabetes, obesity, and smoking all put patients at greater risk of POVL following spine surgery.

Airway and facial edema can likewise develop after prolonged "head-down" positioning. Reintubation, if required, will likely present more difficulty than the intubation at the start of surgery.

When patients are placed in the prone position, the face must be checked periodically to determine that the eyes, nose, and ears are free of pressure. Even foam cushions can exert pressure over time on the chin, orbit, and maxilla. Turning the head is not easily accomplished when the head is positioned on a cushion; therefore, if prolonged procedures are planned, the head can be secured with pins keeping the face free from any pressure.

Monitoring

7 When major blood loss is anticipated or the patient has preexisting cardiac disease, intra-arterial and possibly central venous pressure monitors should be considered prior to "positioning" or "turning." Massive blood loss from injuries to the great vessels can occur intraoperatively with thoracic or lumbar spine procedures.

Instrumentation of the spine requires the ability to intraoperatively detect spinal cord injury. Intraoperative wake-up techniques employing nitrous oxide-narcotic or total intravenous anesthesia allow the testing of motor function following distraction. Once preservation of motor function is established, the patient's anesthetic can be deepened. Continuous monitoring of somatosensory evoked potentials and motor evoked potentials provides alternatives that avoid the need for intraoperative awakening. These monitoring techniques require substitution of propofol, opioid, and/or ketamine infusions for volatile anesthetics and avoidance of neuromuscular paralysis.

CASE DISCUSSION

Resection of a Pituitary Tumor

A 41-year-old woman presents to the operating room for resection of a 10-mm pituitary tumor. She had complained of amenorrhea and had started noticing some decrease in visual acuity.

What hormones does the pituitary gland normally secrete?

Functionally and anatomically, the pituitary is divided into two parts: anterior and posterior. The latter is part of the neurohypophysis, which also includes the pituitary stalk and the median eminence.

The anterior pituitary is composed of several cell types, each secreting a specific hormone. Anterior pituitary hormones include adrenocorticotropic hormone (ACTH), thyroid-stimulating hormone (TSH), growth hormone (GH), the gonadotropins (follicle-stimulating hormone [FSH] and luteinizing hormone [LH]), and prolactin (PRL). Secretion of each of these hormones is regulated by hypothalamic peptides (releasing hormones) that are transported to the adenohypophysis by a capillary portal system. The secretion of FSH, LH, ACTH, TSH, and their respective releasing hormones is also under negative feedback control by the products of their target organs. For example, an increase in circulating thyroid hormone inhibits the secretion of TSH-releasing factor and TSH.

The posterior pituitary secretes antidiuretic hormone (ADH, also called vasopressin) and oxytocin. These hormones are actually formed in supraoptic and paraventricular neurons, respectively, and are transported down axons that terminate in the posterior pituitary. Hypothalamic osmoreceptors, and, to a lesser extent, peripheral vascular stretch receptors, regulate secretion of ADH.

What is the function of these hormones?

ACTH stimulates the adrenal cortex to secrete glucocorticoids. Unlike production of mineralocorticoids, production of glucocorticoids is dependent on ACTH secretion. TSH accelerates the synthesis and release of thyroid hormone (thyroxine).

Normal thyroid function is dependent on production of TSH. The gonadotropins FSH and LH are necessary for normal production of testosterone and spermatogenesis in males and cyclic ovarian function in females. GH promotes tissue growth and increases protein synthesis as well as fatty acid mobilization. Its effects on carbohydrate metabolism are to decrease cellular glucose uptake and utilization and increase insulin secretion. PRL functions to support breast development during pregnancy. Dopamine receptor antagonists are known to increase secretion of PRL.

Through its effect on water permeability in renal collecting ducts, ADH regulates extracellular osmolarity and blood volume Oxytocin acts on areolar myoepithelial cells as part of the milk letdown reflex during suckling and enhances uterine activity during labor.

What factors determine the surgical approach in this patient?

The pituitary gland is attached to the brain by a stalk and extends downward to lie in the sella turcica of the sphenoid bone. Anteriorly, posteriorly, and inferiorly, it is bordered by bone. Laterally, it is bordered by the cavernous sinus, which contains cranial nerves III, IV, V_1, and VI, as well as the cavernous portion of the carotid artery. Superiorly, the diaphragma sella, a thick dural reflection, usually tightly encircles the stalk and forms the roof of the sella turcica. In close proximity to the stalk lie the optic nerves and chiasm. The hypothalamus lies contiguous and superior to the stalk.

Tumors less than 10 mm in diameter are usually approached via the transsphenoidal route, whereas tumors greater than 20 mm in diameter and with significant suprasellar extension are approached via a bifrontal craniotomy. With the use of prophylactic antibiotics, morbidity and mortality rates are significantly less with the transsphenoidal approach; the operation is carried out with the aid of a microscope through an incision in the gingival mucosa beneath the upper lip. The surgeon enters the nasal cavity, dissects through the nasal septum, and finally penetrates the roof of the sphenoid sinus to enter the floor of the sella turcica.

What are the major problems associated with the transsphenoidal approach?

Problems include (1) the need for mucosal injections of epinephrine-containing solution to reduce bleeding, (2) the accumulation of blood and tissue debris in the pharynx and stomach, (3) the risk of hemorrhage from inadvertent entry into the cavernous sinus or the internal carotid artery, (4) cranial nerve damage, and (5) pituitary hypofunction. Prophylactic administration of glucocorticoids is routinely used in most centers. Diabetes insipidus develops postoperatively in up to 40% of patients but is usually transient. Less commonly, the diabetes insipidus presents intraoperatively. The supine and slightly head-up position used for this procedure may also predispose to venous air embolism.

What type of tumor does this patient have?

Tumors in or around the sella turcica account for 10% to 15% of intracranial neoplasms. Pituitary adenomas are most common, followed by craniopharyngiomas and then parasellar meningiomas. Primary malignant pituitary and metastatic tumors are rare. Pituitary tumors that secrete hormones (functional tumors) usually present early, when they are still relatively small (<10 mm). Other tumors present late, with signs of increased ICP (headache, nausea, and vomiting) or compression of contiguous structures (visual disturbances or pituitary hypofunction). Compression of the optic chiasm classically results in bitemporal hemianopia. Compression of normal pituitary tissue produces progressive endocrine dysfunction. Failure of hormonal secretion usually progresses in the order of gonadotropins, GH, ACTH, and TSH. Diabetes insipidus can also be seen preoperatively. Rarely, hemorrhage into the pituitary results in acute panhypopituitarism (pituitary apoplexy) with signs of a rapidly expanding mass, hemodynamic instability, and hypoglycemia.

This patient has the most common type of secretory adenoma—that producing hyperprolactinemia. Women with this tumor typically have amenorrhea, galactorrhea, or both. Men with

prolactin-secreting adenomas may have galactorrhea or infertility, but more commonly present with symptoms of an expanding mass.

What other types of secretory hormones are seen?

Adenomas secreting ACTH (Cushing's disease) produce classic manifestations of Cushing's syndrome: truncal obesity, moon facies, abdominal striae, proximal muscle weakness, hypertension, and osteoporosis. Glucose tolerance is typically impaired, but frank diabetes is less common (<20%). Hirsutism, acne, and amenorrhea are also commonly seen in women.

Adenomas that secrete GH are often large and result in either gigantism (prepubertal patients) or acromegaly (adults). Excessive growth prior to epiphyseal fusion results in massive growth of the entire skeleton. After epiphyseal closure, the abnormal growth is limited to soft tissues and acral parts: hands, feet, nose, and mandible. Patients develop osteoarthritis, which often affects the temporomandibular joint and spine. Diabetes, myopathies, and neuropathies are common. Cardiovascular complications include hypertension, premature coronary disease, and cardiomyopathy in some patients. The most serious anesthetic problem encountered in these patients is difficulty in intubating the trachea.

Are any special monitors required for transsphenoidal surgery?

Monitoring should be carried out in somewhat the same way as for craniotomies. Visual evoked potentials may be employed with large tumors that involve the optic nerves. Precordial Doppler sonography may be used for detecting venous air embolism. Venous access with large bore catheters is desirable in the event of massive hemorrhage

What modifications, if any, are necessary in the anesthetic technique?

The same principles discussed for craniotomies apply, particularly if the patient has evidence of increased ICP. Intravenous antibiotic prophylaxis and glucocorticoid coverage (hydrocortisone, 100 mg) are usually given prior to induction. Many clinicians avoid nitrous oxide to prevent problems with a postoperative pneumocephalus (see above). Intense neuromuscular blockade is important to prevent movement while the surgeon is using the microscope. In some circumstances, the surgeon may request placement of a lumbar intrathecal catheter to drain CSF, thereby facilitating surgical exposure.

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