CHAPTER 53 – NEUROSURGICAL ANESTHESIA

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This chapter provides guidelines for the management of common situations in neurosurgical anesthesia. The first section begins with a review of issues that arise in connection with a wide variety of neurosurgical procedures. These issues constitute a checklist that the practitioner should review before undertaking anesthesia for any neurosurgical procedure. Procedure-specific discussions follow. This chapter assumes familiarity with the cerebral physiology and effects of anesthetics as described in Chapter 21. Neurologic monitoring is described in Chapter 38, and carotid endarterectomy is discussed in Chapter 52.
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RECURRENT ISSUES IN NEUROANESTHESIA

Several basic elements of neurosurgical/neuroanesthetic management are recurrent and should be discussed and agreed on with the surgical team at the outset of every neurosurgical procedure. The list will vary with the procedure and may include the intended surgical position and requisite positioning aids; intentions with respect to the use of steroids, diuretics, anticonvulsants, and antibiotics; the surgeon's perception of the "tightness" of the intracranial space and the remaining intracranial compliance reserve; appropriate objectives for the management of blood pressure, carbon dioxide tension, and body temperature; anticipated blood loss; the intended use of neurophysiologic monitoring (which may impose constraints on the use of anesthetics or muscle relaxants, or both); and occasionally, the perceived risk of air embolization. The considerations driving the decisions made about these issues are presented in this section. One additional recurrent issue, brain protection, is discussed briefly in the section "Aneurysms and Arteriovenous Malformations" and in detail in Chapter 21.

CONTROL OF INTRACRANIAL PRESSURE/brain relaxation

The necessity of preventing increases in intracranial pressure (ICP) or reducing an ICP that is already elevated is recurrent in neuroanesthesia. When the cranium is closed, the objective is to maintain adequate cerebral perfusion pressure (CPP) (CPP = mean arterial pressure [MAP] – ICP) and prevent the herniation of brain tissue between intracranial compartments or through the foramen magnum (Fig. 53-1). When the cranium is open, the issue may be one of providing relaxation of the intracranial contents to facilitate surgical access or, in extreme circumstances, reverse the process of brain herniation through a craniotomy (see Fig. 53-1). The principles that apply are similar whether the cranium is open or closed.

Figure 53-1 Schematic representation of various herniation pathways: 1, subfalcine; 2, uncal (transient); 3, cerebellar, and 4, transcaval.

The various clinical indicators of increased ICP include headache (particularly a postural headache that awakens the patient at night), nausea and vomiting, blurred vision, somnolence, and papilledema. Suggestive findings on computed tomography (CT) include midline shift, obliteration of the basal cisterns, loss of sulci, ventricular effacement (or enlarged ventricles in the event of hydrocephalus), and edema. Edema appears on a CT scan as a region of hypodensity.

Figure 53-2 Computed tomography scan depicting normal (left) and compressed (right) basal cisterns. The basal, or perimesencephalic, cerebrospinal fluid space consists of the interpudendular cistern (anterior), the ambient cisterns (lateral), and the quadrigeminal cisterns (posterior). In the right panel, in a patient with diffuse cerebral swelling (as a result of sagittal sinus thrombosis), the cisterns have been obliterated.
(Courtesy of Ivan Petruchiv, M.D.)
The basal cisterns appear on CT as a black (fluid) halo around the upper end of the brainstem (Fig. 53-2). They include the interpeduncular cistern, which lies between the two cerebral peduncles, the quadrigeminal cistern, which overlies the four colliculi, and the ambient cisterns, which lie lateral to the cerebral peduncles. Figure 53-3 presents the pressure-volume relationship of the intracranial space. The plateau phase occurring at low volumes reveals that the intracranial space is not a completely closed one and that there is some compensatory latitude. Compensation is accomplished principally by the translocation of cerebrospinal fluid (CSF) and venous blood to the spinal CSF space and the extracranial veins, respectively. Ultimately, when the compensatory potential is exhausted, even tiny increments in volume of the intracranial contents can result in substantial increases in ICP. These increases have the potential to result in either herniation of brain tissue from one compartment to another (or into the surgical field) (see Fig. 53-1), with resultant mechanical injury to brain tissue, or reduction in perfusion pressure with a concomitant ischemic injury. Figure 53-3 Intracranial pressure-volume relationship. The horizontal portion of the curve indicates that initially there is some latitude for compensation in the face of an expanding intracranial lesion. This compensation is accomplished largely by displacement of cerebrospinal fluid (CSF) and venous blood from the intracranial to the extracranial spaces. Once the compensatory latitudes are exhausted, small increments in volume result in large increases in intracranial pressure with the associated hazards of herniation or decreased cerebral perfusion pressure (CPP) resulting in ischemia.

Several variables can interact to cause or aggravate intracranial hypertension (Fig. 53-4). For clinicians faced with the problem of managing increased ICP, the objective is, broadly speaking, to reduce the volume of the intracranial contents. For mnemonic purposes, when developing a clinical approach, the clinician can divide the intracranial space into four subcompartments (Table 53-1): cells (including neurons, glia, tumors, and extravasated collections of blood), fluid (intracellular and extracellular), CSF, and blood. Again for mnemonic purposes, the blood compartment can be subdivided into venous and arterial components. It is this last compartment, the blood compartment, that is most amenable to rapid manipulation by the clinician, and accordingly, it is the compartment to which the greatest level of attention is ultimately directed.

1. The cellular compartment. This compartment is largely the province of the surgeon. However, it may be the anesthesiologist’s responsibility to pose a well-placed diagnostic question. When the brain is bulging into the surgical field at the conclusion of evacuation of an extradural hematoma, the clinician should ask whether a subdural or extradural hematoma is present on the contralateral side that warrants either immediate bur holes or immediate postprocedure radiologic evaluation.

2. The CSF compartment. There is no pharmacologic manipulation of the size of the CSF space whose time course and magnitude are relevant to the neurosurgical operating room. The only relevant means for manipulating the size of this compartment is by drainage. A tight surgical field can sometimes be improved by passage of a brain needle by the surgeon into a lateral ventricle to drain CSF. This maneuver may be relevant in both supratentorial and infratentorial procedures when poor conditions in the posterior fossa are thought to be the result of downward pressure by the contents of the supratentorial space. Lumbar CSF drainage can be used
to improve surgical exposure in situations with no substantial hazard of uncal or transforamen magnum herniation.

3. The fluid compartment. This compartment can be addressed with steroids and diuretics. The use of these agents is discussed in the sections "Management of Blood Pressure" and "Steroids," respectively.

4. The blood compartment. This is the compartment that receives the anesthesiologist's greatest attention because it is the most amenable to rapid alteration. The blood compartment should be considered two separate components: venous and arterial.

![Diagram](image)

Figure 53-4 Pathophysiology of intracranial hypertension. The figure depicts the manner in which increases in the volumes of any or all of the four intracranial compartments, blood, cerebrospinal fluid (CSF), fluid (interstitial or intracellular), and cells (four-part rectangle) result in increases in intracranial pressure and eventual neurologic damage. Elements that are potentially under control of the anesthesiologist are indicated by asterisks. (Control of CSF volume requires the presence of a ventriculostomy catheter.) The herniation pathways are depicted in Figure 53-1.

| Table 53-1 - Intracranial compartments and techniques for manipulation of their volume |
|---------------------------------------------|---------------------------------|
| **Compartment**                            | **Volume Control Methods**      |
| 1. Cells (including neurons, glia, tumors, and extravasated blood) | Surgical removal |
| 2. Fluid (intracellular and extracellular)  | Diuretics                      |
|                                               | Steroids (principally tumors)   |
| 3. Cerebrospinal fluid                       | Drainage                       |
| 4. Blood                                     | Decrease cerebral blood flow    |
| Arterial side                                | Improve cerebral venous drainage |
| Venous side                                  |                                 |
We suggest giving first consideration to the venous side of the circulation. It is largely a passive compartment that is frequently overlooked. Passive though it is, engorgement of this compartment is a common cause of increased ICP or poor conditions in the surgical field (Fig. 53-5). A head-up posture to ensure good venous drainage is the norm in neurosurgical anesthesia and critical care. Obstruction of cerebral venous drainage by extremes of head position or circumferential pressure (Philadelphia collars, endotracheal tube ties) should be avoided. Phenomena occurring downstream from the venous structures of the neck may also be relevant. Anything that causes increased intrathoracic pressure can result in obstruction of cerebral venous drainage. A variety of commonplace events can lead to obstruction, including kinked or partially obstructed endotracheal tubes, tension pneumothorax, coughing/straining against the endotracheal tube, or gas trapping as a result of bronchospasm. These too should be sought and remedied. Most practitioners carefully maintain paralysis during craniotomies unless a contraindication is present because a sudden cough can result in dramatic herniation of cerebral structures through the craniotomy.

![Graph](image)

Figure 53-5 Effect of obstruction of cerebral venous outflow on intracranial pressure (ICP) in a patient with intracerebral hematoma. Bilateral jugular compression was applied briefly to verify the function of a newly placed ventriculostomy. The ICP response illustrates the importance of maintaining unobstructed cerebral venous drainage.

Thereafter, the anesthesiologist should consider the arterial side of the circulation. Attention to the effect of anesthetic drugs and techniques on cerebral blood flow (CBF) (see Chapter 21) is an established part of neuroanesthesia. Such attention is relevant because in general, increases in CBF are associated with increases in cerebral blood volume (CBV). The notable exception to this rule occurs in the context of cerebral ischemia caused by hypotension or vessel occlusion, when CBV may increase as the cerebral vasculature dilates in response to a sudden reduction in CBF. However, the relationship generally applies, and attention to the control of CBF is relevant in situations in which volume compensation mechanisms are exhausted or ICP is already increased. The general approach is to select anesthetics and control the physiologic parameters in a manner that avoids unnecessary increases in CBF. The parameters that influence CBF are listed in Table 53-2 and are discussed in Chapter 21.
Table 53-2 — Factors that influence cerebral blood flow*

<table>
<thead>
<tr>
<th>Factor</th>
<th>Influence</th>
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<tr>
<td>PaO₂</td>
<td>Blood pressure/status of autoregulation</td>
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<tr>
<td>PaCO₂</td>
<td>Vasoactive agents</td>
</tr>
<tr>
<td>Cerebral metabolic rate</td>
<td>Anesthetics</td>
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<tr>
<td>Arousal/pain</td>
<td>Pressors</td>
</tr>
<tr>
<td>Seizures</td>
<td>Inotropes</td>
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<tr>
<td>Temperature</td>
<td>Vasodilators</td>
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<tr>
<td>Anesthetics</td>
<td>Blood viscosity</td>
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<td></td>
<td>Neurogenic pathways (intra- and extra-axial)</td>
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**SELECTION OF ANESTHETICS**

The question of which anesthetics are appropriate, especially in the context of unstable ICP, arises often. Chapter 21 provides relevant information in detail, and only broad generalizations are made here.

In general, intravenous anesthetic, analgesic, and sedative drugs are associated with parallel reductions in CBF and cerebral metabolic rate (CMR) and will not have adverse effects on ICP. Ketamine, given in large doses to patients with a generally normal level of consciousness before anesthesia, may be the exception. It appears that for the most part, autoregulation and CO₂ responsiveness are preserved during the administration of all intravenous drugs (Chapter 21).

By contrast, all of the volatile anesthetics cause dose-dependent cerebral vasodilation. The order of vasodilating potency is approximately halothane > enflurane > isoflurane > desflurane > sevoflurane. As noted in Chapter 21, the CBF differences among isoflurane, desflurane, and sevoflurane are probably not significant to the clinician. The net CBF effect of introducing a volatile anesthetic will depend on the interaction of several factors: the concentration of the anesthetic, the extent of previous CMR depression, simultaneous blood pressure changes acting in conjunction with previous or anesthetic-induced autoregulation abnormalities, and simultaneous changes in PaCO₂ acting in conjunction with any disease-related impairment in CO₂ responsiveness.

Nitrous oxide is also a cerebral vasodilator, the CBF effect of which is greatest when it is administered as a sole anesthetic, least when it is administered against a background of narcotics, propofol, or benzodiazepines, and intermediate when administered in conjunction with volatile anesthetics (Chapter 21). Nonetheless, experience dictates that both N₂O and volatile anesthetics, the latter usually in concentrations less than the minimum alveolar concentration (MAC), when administered as components of a balanced anesthetic technique in combination with narcotics, can be used in most elective and many emergency neurosurgical procedures. Exceptions will be rare. When they occur (a somnolent, vomiting patient with papilledema, a large mass, and compressed basal cisterns; a head injury victim with an expanding mass lesion or obliterated cisterns and sulci on CT), the clinician may be well advised to use a predominantly intravenous technique until the cranium and dura are open and the effect of the anesthetic technique can be assessed by direct observation of the surgical field. Inhaled anesthetics will be entirely acceptable components of most anesthetics for neurosurgery. However, in circumstances in which ICP is persistently elevated (in a closed-cranium procedure) or the surgical field is persistently "tight," N₂O and volatile anesthetics should be viewed as potential contributing factors and be eliminated from the anesthetic in favor of intravenous drugs.
Muscle relaxants (Chapter 13 and Chapter 21) that have the potential to release histamine (curare, metocurine, mivacurium, atracurium) should be given in small, divided doses. Although succinylcholine has been associated with increases in ICP, these increases are small and transient. Moreover, the increases can be blocked by a preceding dose of metocurine, 0.03 mg/kg,\(^5\) and in at least some instances, are not evident in patients with common emergency neurosurgical conditions (head injury, subarachnoid hemorrhage [SAH]).\(^6\) Accordingly, in a clinical situation that calls for rapid relaxation for the purpose of controlling or protecting the airway, succinylcholine in conjunction with proper management of the airway and MAP is reasonable. From the material just presented and the preceding discussion of cerebral physiology in Chapter 21, a systematic clinical approach should follow readily. A schema for approaching the problem of an acute increase in ICP or acute deterioration in conditions in the surgical field is presented in Table 53-3.

**Table 53-3 -- The high—I intracranial pressure/"tight-brain" checklist**

1. Are the relevant pressures controlled?
   - Jugular venous pressure
   - Extreme head rotation or neck flexion?
   - Direct jugular compression?
   - Head-up posture?
   - Airway pressure
   - Airway obstruction?
   - Bronchospasm?
   - Straining, coughing, adequately relaxed?
   - Pneumothorax?
   - Partial pressure of CO\(_2\) and O\(_2\) (PacO\(_2\), PacO\(_2\))
   - Arterial pressure

2. Is the metabolic rate controlled?
   - Pain/arousal?
   - Seizures?

3. Are any potential vasodilators in use?
   - N\(_2\)O, volatile anesthetics, nitroprusside, calcium channel blockers

4. Are there any unrecognized mass lesions?
   - Blood, air ± N\(_2\)O

If the problem has not resolved satisfactorily after following the approach in Table 53-3, what then? Table 53-4 presents the options.
Table 53-4 — Methods for rapid reduction of intracranial pressure/brain volume (after review of the "checklist" in Table 53-3)

<table>
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<td>Drainage of cerebrospinal fluid (ventriculostomy, brain needle)</td>
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<td>Diuresis (usually mannitol)</td>
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<td>Suppression of the cerebral metabolic rate (usually barbiturates)</td>
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<td>Reduction in mean arterial pressure (if dysautoregulation is present)</td>
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CSF drainage was discussed earlier. The use of additional osmotic diuretics is theoretically limited by an upper acceptable osmolarity limit of approximately 320 mOsm/L. However, in extremis, the use is frequently empirical, and repeated doses (e.g., 12.5 g) are administered until a clinical response is no longer observed. Barbiturates have long been used most to induce a reduction in CMR, with the objective of causing a coupled reduction in CBF and thereby CBV. Propofol is gaining popularity for this application. Note, however, that although the use of barbiturates is supported by intensive care unit (ICU) experience demonstrating efficacy in control of ICP, no such experience has been accumulated for propofol. Furthermore, a frequently fatal syndrome of metabolic acidosis and rhabdomyolysis has recently been recognized in patients who have received prolonged propofol infusions in the ICU setting. The MAP reduction will occasionally reduce vascular engorgement and thereby reduce total brain bulk. This approach is most likely to be relevant in the event of dysautoregulation occurring in the context of resection of arteriovenous malformations (AVMs) (see the later section "Aneurysms and Arteriovenous Malformations").

MANAGEMENT OF PACO₂

At the outset, the anesthesiologist and the surgeon should agree on the objectives with respect to PaCO₂.

Induction of hypocapnia is a time-honored part of the management of intracranial neurosurgical procedures. The rationale is principally that the concomitant reduction in CBF (see Fig. 21-4) and CBV will result in a reduction in ICP, or "brain relaxation." The rationale is valid. However, two considerations should influence the clinician's use of hyperventilation. First, the vasoconstrictive effect of hypocapnia has the potential to cause ischemia in certain situations. Second, the CBF-lowering effect is not sustained.

HYPOCAPNIA-INDUCED CEREBRAL ISCHEMIA

At first, clinicians were skeptical that hyperventilation could actually result in ischemia, and it does in fact appear that normal brain is unlikely to be damaged by the typical clinical use of hyperventilation. However, such may not be the case in certain pathologic conditions.

NORMAL BRAIN.

The available data indicate that in normal subjects, ischemia will not occur at a PaCO₂ over 20 mm Hg. This generalization appears to also apply during induced hypotension. However, physiologic alterations, as evidenced by both metabolic and electroencephalograph (EEG) abnormalities, have been observed in human volunteers and in normal animals at severe hypocapnia (PaCO₂ <15 mm Hg) and in dogs subjected to the combination of extreme hypocapnia (PaCO₂ of 10 mm Hg) and severe anemia (hemoglobin content of 5 g/dL). In one of these studies, EEG abnormalities and paresthesias occurred in volunteers hyperventilating to PaCO₂ values less than 20 mm Hg, and these effects were reversed by hyperbaric oxygenation, thus suggesting that they may truly have been caused by ischemia. In two separate
investigations in cats at PaCO₂ levels of 10 to 12 mm Hg, modest reductions in brain phosphocreatine levels with increased brain lactate but normal adenosine triphosphate levels were observed. It has been suggested that the changes observed may in part reflect pH-related alterations in enzyme function (specifically, an increase in the activity of phosphofructokinase causing increased lactate formation) rather than ischemia. Accordingly, given that a PaCO₂ of less than 20 to 25 mm Hg offers very little additional benefit in terms of improvement in intracranial compliance, it seems prudent to limit acute PaCO₂ reduction to 25 mm Hg in previously normocapnic individuals. Normal brain will not be injured by this degree of hypocapnia.

INJURED BRAIN.

Although preventing herniation, maintaining ICP under 20 mm Hg, minimizing retractor pressure, and facilitating surgical access remain priorities that may justify hypocapnia, evidence is also accumulating that hyperventilation is potentially deleterious and should not be overused. In the setting of head injury, there is evidence that hyperventilation can result in ischemia, especially when baseline CBF is low, as is commonly the case in the first 24 hours after injury. An increased frequency of brain regions with very low CBF has been demonstrated in head-injured patients who were acutely hyperventilated. In addition, from centers that monitor jugular venous oxygen saturation (SjvO₂), there have been numerous observations that low SjvO₂ values can be increased and that lactate levels in the jugular venous effluent can be decreased by reducing the degree of hyperventilation although the inability of SjvO₂ monitoring to detect ischemia consistently has also been reported. However, at present, there is little information to confirm a deleterious effect of hyperventilation. The closest thing to "proof" resides in a study of patients with moderate head injuries by Muizelaar and colleagues. These authors divided patients into a near-normocapnic group in which PaCO₂ was maintained at approximately 35 mm Hg, a hypocapnic group in which PaCO₂ was maintained in the vicinity of 25 mm Hg, and a third group in which carbon dioxide tension was maintained at 25 mm Hg and the buffer tromethamine was administered. Tromethamine is a buffer that can cross the blood-brain barrier, and it has been theorized that tromethamine can attenuate the adverse effect of the reduction in bicarbonate levels in CSF and brain extracellular fluid that occurs with chronic hyperventilation. They examined outcomes 3 and 6 months after injury and observed a poorer status in a post hoc subgroup of the hyperventilation group. That subgroup included patients with the best initial motor scores, specifically, a subgroup in which the severity of injury was such that they merited intubation by conventional criteria but whose clinical condition may have been such that hyperventilation was not necessarily required for control of ICP and who therefore had little to gain from hyperventilation.

Accordingly, hyperventilation should not be an automatic component of every "neuroanesthetic." It should be treated like any other therapeutic intervention. There should be an indication for instituting it (usually elevated or uncertain ICP or the need to improve conditions in the surgical field, or both). Hyperventilation should be used with the knowledge that it has the potential for causing an adverse effect, and as is the case with any other therapeutic intervention, it should be withdrawn as the indication for it subsides. The concern regarding the hazards of hypocapnia, which evolved in the context of head injury, has influenced all of neurosurgery. In particular, it is now widely avoided in the management of SAH because of the postictal low-CBF state that is known to occur. In addition, brain tissue beneath retractors can have a similarly reduced CBF.

DURATION OF HYPOCAPNIA-INDUCED REDUCTION IN CBF

The effect of hypocapnia on CBF is not sustained. Figure 53-6 is a nonquantitative representation of changes in CBF and CSF pH occurring in association with a sustained period of hyperventilation. With the onset of
hyperventilation, the pH of both CSF and the brain's extracellular fluid space increases, and CBF decreases abruptly. However, the cerebral alkalosis is not sustained. By alterations in function of the enzyme carbonic anhydrase, the concentration of bicarbonate in CSF and the brain's extracellular fluid space is reduced, and in a time course of 6 to 18 hours, the pH of these compartments returns to normal. The implications are twofold. First, the clinician should ideally hyperventilate patients for only as long as a reduction in brain volume is required. Prolonged, but unnecessary hyperventilation may lead to a circumstance wherein subsequent clinical events call for additional maneuvers to reduce the volume of the intracranial contents. However, if carbon dioxide tension is already in the 23- to 25-mm Hg range, it would be difficult to impose sufficient additional hyperventilation to once again accomplish the original reduction in CBF without the hazard of pulmonary barotrauma. Second, in a patient who has been hyperventilated for a sustained period (e.g., 2 days in an ICU setting), rapid restoration of carbon dioxide tension from values in the vicinity of 25 to typical normal values (e.g., 40 mm Hg) should ideally be accomplished slowly. A sudden increase in carbon dioxide tension from 25 to 40 mm Hg in an individual who has been chronically hyperventilated will have the same physiologic effect that a rapid change from 40 to 55 mm Hg would have in a previously normocapnic subject.

Figure 53-6 Changes in PaCO₂, cerebral blood flow (CBF), and cerebrospinal fluid (CSF) pH with prolonged hyperventilation. Whereas the decreased arterial PaCO₂ (and the systemic alkalosis) persist for the duration of the period of hyperventilation, the pH of the brain and CBF return toward normal over a period of 8 to 12 hours.

If hypocapnia has been required as an adjunct to brain relaxation during craniotomy, PaCO₂ should also be allowed to rise once the retractors are removed (if dural closure requirements permit) to minimize the residual intracranial pneumatocele (see the later section "Pneumocephalus").

MANAGEMENT OF ARTERIAL BLOOD PRESSURE

Acceptable blood pressure limits should similarly be agreed on at the beginning of a neurosurgical procedure. One of the prominent themes of contemporary neurosurgery is that CPP should be maintained at normal or even high-normal levels after acute central nervous system insults and during most intracranial neurosurgical procedures. This concept has evolved from the growing appreciation that CBF is frequently perilously low in some brain regions after acute neurologic insults, in particular, head injury (see additional discussion in the later section "Head Injury") and SAH. Two additional factors should be considered. The first is that the autoregulatory response to decreasing blood pressure may not be intact throughout the brain. Figure 53-7 depicts the ischemic hazard that attends the circumstance of a low resting CBF and absent autoregulation even at blood pressure levels considered safe when autoregulation is intact. In addition, there is the appreciation that maintenance of arterial pressure is also relevant to brain compressed under retractors because the effective perfusion pressure there is lowered by increased local tissue pressure.
who have sustained a recent spinal cord injury, to patients whose spinal cord is under compression or at risk for compression or vascular compromise because of a disease process (most commonly cervical spinal stenosis with or without ossification of the posterior longitudinal ligament) or an intended surgical procedure, and to those undergoing surgery involving retraction of the spinal cord. Were we empowered to "legislate" the standard of care, we would mandate that blood pressure during anesthesia in these patients be maintained as close as possible and certainly within 10% of average awake values.

STERIODS

The administration of steroids for the purpose of reducing or limiting the formation of edema is another time-honored practice in neurosurgery. The efficacy of steroids in reducing the edema associated with tumors is well confirmed. The time course of this effect is relatively rapid, though not so rapid that it is relevant to the management of intraoperative events. However, administration beginning 48 hours before an elective surgical procedure has the potential to reduce edema formation and improve the clinical condition by the time of craniotomy. Although clinical improvement, specifically, a decreased frequency of ICP plateau waves and an improvement in the pressure-volume response (the increment in ICP in response to a standardized intracranial volume challenge), occurs within 24 hours, a reduction in ICP may not occur for 48 to 72 hours after the initiation of therapy. This has been interpreted to indicate that steroids in some way improve the "viscoelastic properties" of the intracranial space before a reduction in edema occurs, although the mechanism is undefined. Steroids are usually given intraoperatively and postoperatively to maintain the effects achieved by preoperative treatment.

The practice of administering steroids to adult head injury patients has largely been abandoned as a result of controlled trials that demonstrated either no benefit or deleterious effects. No such studies have been performed in pediatric trauma victims, and practices vary among pediatric neurosurgeons with respect to the administration of steroids after head injury in this population. A recent European trial reported a benefit of the administration of large doses of triamcinolone in adult head injury victims with focal intracranial lesions. The potent fluorinated steroids had not been studied previously in this population. The results of this study have prompted a reconsideration of the use of steroids in head-injured patients, and a trial of high-dose methylprednisolone is under way.
DIURETICS

Diuretics are used widely in neurosurgery to reduce the volume of the brain's intracellular and extracellular fluid compartments. It is probably largely the extracellular compartment that is influenced because neurons and glia have quick and efficient cell volume regulation mechanisms. Both osmotic and loop diuretics have been used. Although data suggest that loop diuretics can be effective, osmotic diuretics, principally mannitol, are preferred clinically because of their speed and efficacy. The only osmotic diuretic available in most formularies is mannitol, although urea once had its proponents. However, urea is a smaller molecule that clearly has greater potential to enter brain parenchyma. That is not to say that mannitol does not enter brain parenchyma. Data indicate that it enters brain tissue and, over a reasonably short time course, appears in the CSF space. The possibility that the mannitol that gains access to the parenchyma can aggravate swelling has resulted in varying degrees of reluctance among clinicians to administer mannitol. Most, nonetheless, find it a mainstay of ICP management. There is the concern that it will be effective only when some degree of blood-brain barrier integrity is preserved in a significant portion of the brain. Most clinicians respond to this concern by making empirical use of mannitol; that is, if it is effective in reducing ICP or improving conditions in the surgical field, repeated doses can be administered or will be administered. If it is ineffective (or if serum osmolarity reaches the traditional limit of 320 mOsm/L), its administration is withheld.

The dosages of mannitol used vary from 0.25 g/kg to 100 g "for all comers." One gram per kilogram appears to be the most common dose. However, a systematic study in head-injured patients demonstrated that an equivalent initial ICP-reducing effect can be achieved with 0.25 g/kg, although that effect may not be as sustained as with larger doses.

Some clinicians advocate the combined administration of a loop diuretic (usually furosemide) and an osmotic diuretic. The superficial rationale is that mannitol establishes an osmotic gradient that draws fluid out of brain parenchyma and that the furosemide, by hastening excretion of water from the intravascular space, facilitates the maintenance of that gradient. A second mechanism may add additional justification for the practice of combining the two diuretics. Neurons and glia, as mentioned earlier, appear to have powerful homeostatic mechanisms to ensure regulation of cell volume. Neurons and glia that shrink in response to increased osmolarity in the external environment recover their volume rapidly as a consequence of the accumulation of so-called idiogenic osmoles that serve to minimize the gradient between the internal and external environment. One of these idiogenic osmoles is chloride. It has been demonstrated in the laboratory that loop diuretics inhibit the chloride channel through which this ion must pass and thereby retard the normal volume-restoring mechanism.

The normal volume regulatory mechanisms of neurons and glia may also be relevant to the phenomenon of rebound swelling. Rebound is commonly attributed to the previous use of mannitol and assumed to be a function of the accumulation of mannitol in cerebral tissue. Although this may be part of the story, the rebound may in fact be "hypertonic rebound" rather than "mannitol rebound." It seems reasonable to be concerned that after a sustained period of hyperosmolarity of any cause, rebound swelling of neurons and glia (which have accumulated idiogenic osmoles) may occur in the event that systemic osmolarity decreases rapidly toward normal levels. It is certainly well known that rebound cerebral swelling can occur after an episode of extreme blood glucose elevation. Accordingly, it should not be assumed that the use of, for instance, hypertonic saline rather than mannitol will obviate this phenomenon.
ANTICONVULSANTS

The general principle is that any acute irritation of the cortical surface, including acute neurologic events such as head injury and SAH, has the potential to result in seizures.\textsuperscript{[63][64]} Cortical incisions and irritation of the brain surface by retractors may similarly be potential foci. Given the relatively benign nature of diphenylhydantoin, provided that it is given judiciously, routine administration to patients undergoing most supratentorial craniotomies, as well as those who have sustained a significant head injury or SAH, seems appropriate in the absence of a contraindication. It is generally said that diphenylhydantoin should be administered at rates not greater than 50 mg/min. However, there is no necessity for rapid administration in a situation in which the intention is to prevent postoperative seizures, and it should be administered at a significantly slower rate.

POSITIONING

The intended surgical position and the necessary position aids should be agreed on at the outset. The commonly used positions and positioning aids/supports are listed in Table 53-5.

Table 53-5 -- Common neurosurgical positions and positioning aids

<table>
<thead>
<tr>
<th>Positions</th>
<th>Positioning Aids/Supports</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine</td>
<td>Pin (&quot;Mayfield&quot;) head holder</td>
</tr>
<tr>
<td>Lateral (park bench)</td>
<td>Radiolucent pin head holder</td>
</tr>
<tr>
<td>Semilateral (Janetta)</td>
<td>Horseshoe head rest</td>
</tr>
<tr>
<td>Prone</td>
<td>Foam head support (e.g., Voss, O.S.I., Prone-View)</td>
</tr>
<tr>
<td>Sitting</td>
<td>Vacuum mattress (&quot;beanbag&quot;)</td>
</tr>
<tr>
<td>Prone</td>
<td>Wilson-type frame</td>
</tr>
<tr>
<td>Prone</td>
<td>Andrews (&quot;hinder binder&quot;)-type frame</td>
</tr>
<tr>
<td>Prone</td>
<td>Relton-Hall (four-poster) frame</td>
</tr>
</tbody>
</table>

GENERAL CONSIDERATIONS

The prolonged duration of many neurosurgical procedures should be taken into account in all positions. Pressure points should be identified and padded carefully, and pressure and traction on nerves must be avoided. Thromboembolic precautions, including support hose and sequential pneumatic compression devices, are often appropriate. For cranial procedures, almost invariably some component of head-up posturing (e.g., 15 to 20
degrees) will be appropriate to ensure optimal venous drainage. The conspicuous exception occurs with evacuation of a chronic subdural hemorrhage, after which patients are usually nursed flat to discourage reaccumulation. Patients are also often maintained flat after CSF shunting to avoid overly rapid collapse of the ventricles.

SUPINE

The supine position is used with the head neutral or rotated for frontal, temporal, or parietal access. Extremes of head rotation can obstruct jugular venous drainage, and a shoulder roll can attenuate this problem. The head is usually in a neutral position for bifrontal craniotomies and transphenoidal approaches to the pituitary. The head-up posture is best accomplished by adjusting the operating table to a chaise lounge (lawn chair) position (flexion, pillows under the knees, slight reverse Trendelenburg). This orientation, in addition to promoting cerebral venous drainage, decreases back strain.

SEMILATERAL

The semilateral position, also known as the Janetta position after the neurosurgeon who popularized its use for microvascular decompression of the fifth cranial nerve, is used for retromastoid access. It is achieved by lateral tilting of the table 10 to 20 degrees combined with a generous shoulder roll. Again, extreme head rotation sufficient to cause compression of the contralateral jugular by the chin should be avoided.

LATERAL

The lateral position can be used for access to the posterior parietal and occipital lobes and the lateral posterior fossa, including tumors at the cerebellopontine angle and aneurysms of the vertebral and basilar arteries. A vacuum mattress that can be molded to the patient's anatomy greatly facilitates maintenance of a stable lateral position. An axillary roll is important for preventing brachial plexus injury.

PRONE

The prone position is used for spinal cord, occipital lobe, craniosynostosis, and posterior fossa procedures. The prone position has also been referred to, aptly, as the Concorde position because for cervical spine and posterior fossa procedures, the final position commonly entails neck flexion, reverse Trendelenburg, and elevation of the legs, usually with pillows. This orientation serves to bring the surgical field to a horizontal position. Before turning to the prone position, the anesthesiologist should ensure that the intravenous catheter and endotracheal tube are secure and that appropriate personnel are available to prevent injury during the turn. The anesthesiologist should have a plan for detaching and reattaching monitors in an orderly manner to prevent an excessive monitoring "window." Awake tracheal intubation and prone positioning can be used in patients with an unstable cervical spine, and it should be confirmed that neurologic status is unchanged before induction of anesthesia in the final surgical position. It is also occasionally performed in very obese patients. The head can be positioned in a pin head holder (applied before the turn), a horseshoe headrest, or a disposable foam headrest. A complication of the prone position to which there must be constant attention is retinal ischemia and blindness caused by occlusion of the central retinal vessel as a result of orbital compression. It must be intermittently confirmed (e.g., every 15 minutes) and after any surgery-related head/neck movement that pressure has not come to bear on the eye. However, it should be understood that not all postoperative visual loss (POVL) is a result of direct orbital compression. Ischemic optic neuropathy (ION) actually appears to be a
more frequent cause of POVL than pressure-induced occlusion of the central retinal vessels does. The causeand-effect relationships associated with ION are uncertain, but low arterial pressure, low hematocrit, and lengthy surgical procedures are statistically associated with the phenomenon. Ophthalmologists have long recognized the phenomenon of ION in nonanesthetized patients and have identified as risk factors many causes of vascular disease (hypertension, diabetes, smoking, hyperlipidemia) and hypotension, especially when it occurs in hypertensive individuals. It is also suspected that certain normal anatomic and physiologic variations, including poor collateralization or absence of autoregulation of the vasculature of the optic nerve head (or both), a small and therefore anatomically "crowded" optic nerve head (the "disk at risk"), an increase in intraocular pressure that has been observed to occur during prolonged spine surgery in the prone position, and impaired cerebral venous drainage, may contribute to the intraoperative occurrence of ION. Note that the notion that ION is merely a function of arterial hypotension is probably an inaccurate oversimplification of a phenomenon that almost certainly involves the interplay of numerous preoperative and intraoperative factors.

Direct pressure can also result in various degrees of pressure necrosis of the forehead, maxillae, and chin, especially during prolonged spinal procedures. The clinician should attempt to ensure that pressure is as evenly distributed as possible over facial structures. Other pressure points to be checked include the axillae, breasts, iliac crests, femoral canals, genitalia, knees, and heels. When the arms are placed in the "stick-em up" position, traction on the brachial plexus must be avoided. This can usually be accomplished by not exceeding a "90-90" position (arms abducted not >90 degrees; elbows flexed not >90 degrees), with care taken to ensure that the elbow is anterior to the shoulder to prevent wrapping of the brachial plexus around the head of the humerus. An antisialagogue such as glycopyrrolate may help reduce loosening of the tape used to secure the endotracheal tube.

An objective during prone positioning, especially for lumbar spine surgery, is avoidance of compression of the inferior vena cava. Impairment of vena caval return diverts blood to the epidural plexus and increases the potential for bleeding during laminectomy. Avoidance of inferior vena cava compression is an objective of all of the spinal surgery frames and is accomplished very effectively by the Relton-Hall, Wilson, and Andrews variants. It does, however, introduce a risk of air embolism, although clinical occurrences have been very infrequent.

Attention should be directed at preventing injury to the tongue in the prone position. With both cervical and posterior fossa procedures, it is frequently necessary to flex the neck substantially to facilitate surgical access. Such flexion reduces the anterior-posterior dimension of the hypopharynx, and compression ischemia of the base of the tongue (as well as the soft palate and posterior wall of the pharynx) can occur in the presence of foreign bodies (endotracheal tube, esophageal stethoscope, oral airway). The consequence can be postextubation airway obstruction of rapid onset as a result of "macroglossia" caused by the accumulation of edema after reperfusion of the ischemic tissue (see later). Accordingly, unnecessary paraphernalia in the pharynx should be avoided. Omitting the oral airway entirely is unwise because the tongue may then protrude between and be trapped by the teeth as progressive swelling of facial structures occurs during a prolonged prone procedure. A bite block akin to those used with laryngeal masks will prevent this problem without adding bulk to the hypopharynx.
SITTING

Several reviews of large experiences with procedures performed in the sitting position have been published. All concluded that the sitting position can be used with acceptable rates of morbidity and mortality. However, these reports were prepared by groups that perform 50 to 100 or more of these procedures per year, and the hazards of the sitting position may be greater for teams that have less frequent occasion to use it. With increasing frequency, the sitting position is being avoided through the use of one of its alternatives (prone, semilateral, lateral). However, we are likely to continue encountering it because even surgeons who are inclined to use alternative positions may opt for the sitting position when access to midline structures (the floor of the fourth ventricle, the pontomedullary junction, and the vermis) is required. Nonetheless, alternative positions for posterior fossa surgery exist and should be considered when contraindications to the sitting position exist.

ACHIEVING THE SITTING POSITION.

A properly positioned patient is more commonly in a modified recumbent position as shown in Figure 53-8 rather than truly sitting. The legs should be kept as high as possible (usually with pillows under the knees) to promote venous return and thereby enhance circulatory stability. Ideally, the head holder should be attached to the back portion of the table (see Fig. 53-8A) rather than to the portions under the thighs or legs (see Fig. 53-8B). Such attachment will permit lowering of the head and closed-chest massage if necessary without the necessity of taking the patient out of the head holder.

Figure 53-8 The sitting position. The patient is typically semirecumbent rather than sitting. In A, the head holder support is correctly positioned such that the head can be lowered without the necessity of first detaching the head holder. The configuration in B, with the support attached to the thigh portion of the table, should be avoided.

(From Martin JT. Positioning in Anesthesia and Surgery. Philadelphia, WB Saunders, 1988.)
When procedures are performed in the sitting position, the clinician should think in terms of measuring and maintaining perfusion pressure at the level of the surgical field. This objective is best accomplished by referencing transducers to the interaural plane. If a manual blood pressure cuff on the arm is used, a correction** to allow for the hydrostatic difference between the arm and the operative field should be applied. A series of hazards are associated with the sitting position. Circulatory instability, macroglossia, and quadriplegia are discussed in this section. Pneumocephalus is discussed in the section "Pneumocephalus." Venous air embolism and paradoxical air embolism are discussed in the section "Venous Air Embolism." Several of these hazards are also relevant when cervical spine and posterior fossa procedures are performed in nonsitting positions, but they occur with greater frequency in the sitting position.

**CARDIOVASCULAR EFFECTS OF THE SITTING POSITION.**

Placing an anesthetized patient in the sitting position conveys some risk of impaired cardiovascular function, in particular, hypotension. Pressor administration will be required in some patients. Measures to avoid hypotension include prepositioning hydration, wrapping of the legs with elastic bandages to counteract gravitational shifts of blood, and slow, incremental adjustment of table position. Both aggressive volume loading and the G-suit (a.k.a. pneumatic antishock trousers, MAST suit) have been shown to attenuate the effects of assuming the sitting position. However, neither of these measures has been widely applied. In most healthy subjects, the hemodynamic changes are of nonthreatening magnitude. In a study of healthy (American Society of Anesthesiologists [ASA] grade 1 and 2) anesthetized adult subjects aged 22 to 64 years, Marshall and coworkers observed relatively modest changes. MAP was relatively unaffected, whereas wedge pressure, stroke volume, and the cardiac index decreased, the latter by approximately 15%, although some variation was noted with the anesthetics used. The combination of an unchanged MAP (which in general requires the use of a "light," high-sympathetic tone anesthetic) and a reduced cardiac index implies that systemic vascular resistance (SVR) rose. Their calculations and the observations of other investigators in fact reveal significant elevations in SVR. Accordingly, for patients in whom an abrupt increase in SVR may be poorly tolerated, the sitting position may represent a physiologic threat and alternative positions should be considered. A pulmonary artery catheter may be warranted in those with clinical or historical evidence of antecedent coronary artery or valvular heart disease and arbitrarily in patients older than 60 to 65 years.

During procedures performed in the sitting position, MAP should be transduced at or corrected to head level to provide a meaningful index of CPP. Specifically, CPP (MAP minus estimated ICP) should be maintained at a minimum value of 60 mm Hg in healthy patients, in whom it is reasonable to assume normal cerebral vasculature. The safe lower limit should be raised for elderly patients, for those with hypertension or known cerebral vascular disease (or both), for those with degenerative disease of the cervical spine or cervical spinal stenosis because they may be at risk for decreased spinal cord perfusion, and in the event that substantial or sustained retractor pressure must be applied to brain or spinal cord tissue.

**MACROGLOSSIA.**

Sporadic reports have described upper airway obstruction after posterior fossa procedures in which swelling of pharyngeal structures, including the soft palate, posterior wall, pharynx, and base of the tongue, has been observed. These episodes have been attributed to edema occurring at the time of reperfusion after trauma/prolonged ischemia as a result of foreign bodies (usually oral airways) causing pressure on these structures during lengthy procedures with sustained neck flexion (which is usually required to improve access to posterior structures). It is customary (and we think ideal, although there is no science to support the practice) to
maintain at least two fingerbreadths' distance between the chin and the sternum to prevent excessive reduction of the anterior-posterior diameter of the oropharynx. In addition, it is our practice to position patients with the oral airway in place and then, once the final head position is achieved, withdraw it until its tip functions as a bite block between the teeth. Consideration of the macroglossia phenomenon may also be relevant as clinicians contemplate the use of transesophageal echocardiography (TEE) in the neurosurgery suite. For the most part, centers that routinely use TEE in neurosurgery use either pediatric or custom-made small-diameter probes to avoid trauma to the pharyngeal and perilaryngeal structures.

**QUADRIPLÉGIA.**

The sitting position per se has been implicated as a cause of rare instances of unexplained postoperative paraplegia. It has been hypothesized that neck flexion, which is a common concomitant of the seated position, may result in stretching or compression of the cervical spinal cord. This possibility may represent a relative contraindication to the use of this position in patients with significant degenerative disease of the cervical spine, especially those with evidence of associated cerebral vascular disease. The blood pressure management implications were mentioned in the preceding section on cardiovascular effects. It may also represent a justification for monitoring of somatosensory evoked responses during the positioning phase of a sitting procedure for patients perceived to be at high risk.

*A column of water 32 cm high exerts a pressure of 25 mm Hg.

**PNEUMOCEPHALUS**

The issue of pneumocephalus arises most often in connection with posterior fossa craniotomies performed with a head-up posture because these operations entail the probability that air will be retained within the cranium. During these procedures, air may enter the supratentorial space, much as air enters an inverted pop bottle. Depending on the relationship of the brainstem and temporal lobes to the incisura, the pressure in the air collection may or may not be able to equilibrate with atmospheric pressure. This phenomenon has relevance to the use of N₂O because any N₂O that enters a trapped gas space will augment the volume of that space. In those (probably uncommon) intraoperative circumstances where there is in fact a completely closed intracranial gas space, the use of N₂O may result in an effect comparable to that of an expanding mass lesion. We do not view N₂O as absolutely contraindicated because before dural closure, intracranial gas is probably only rarely trapped. Nonetheless, attention to this possibility is important when one is presented with the problem of an increasingly "tight" brain during a posterior fossa craniotomy.

During a posterior fossa procedure performed in a head-up posture, when surgical closure has reached a stage such that the intracranial space has been completely sealed from the atmosphere, it is probably appropriate to then omit N₂O because of the possibility of contributing to tension pneumocephalus. Note that the use of N₂O up to the point of dural closure may actually represent a clinical advantage in that the gas pocket can be expected to shrink more rapidly as a result of the presence of N₂O (because N₂O will diffuse out much more quickly than nitrogen). Tension pneumocephalus is often naïvely viewed as being exclusively a function of the use of N₂O. However, it can most certainly occur as a complication of intracranial neurosurgery entirely unrelated to the use of N₂O. Tension pneumocephalus is one of the causes of delayed awakening or nonawakening after both posterior fossa and supratentorial procedures (Fig. 53-9). It occurs because air enters the cranium with the patient in a head-up position at a time when the volume of the intracranial contents has been reduced as a result of some combination of hypocapnia, good venous drainage, osmotic diuresis, and loss of CSF from the operative field. When the cranium is closed and the patient is returned to the near-supine
position, CSF, venous blood volume, and extracellular fluid return or reaccumulate and the air pocket becomes an unyielding mass lesion (because of the very slow diffusion of nitrogen). It may cause delayed recovery of consciousness or severe headache. Among supratentorial craniotomies, the largest residual air spaces occur after frontal skull base procedures in which energetic brain relaxation measures are used to facilitate subfrontal access (see Fig. 53-9). At the end of these procedures, which are typically performed in a supine/brow-up position, it is not feasible to fill the intracranial dead space with normal saline as is commonly done with smaller craniotomy defects, and a large residual pneumocele may be left. Once again, we doubt that the possible occurrence of this phenomenon represents a contraindication to the use of N₂O. However, withdrawal of N₂O may be appropriate at the time of cranial closure. The diagnosis of pneumocephalus is confirmed by a brow-up lateral radiograph or CT scan. The treatment is a twist drill hole followed by needle puncture of the dura.

Figure 53-9 Postoperative computed tomographic scan demonstrating a large pneumocephalus after a subfrontal approach to a suprasellar glioma. Immediately postoperatively, the patient was confused and agitated and complained of severe headache.

Residual intracranial air should be considered at the time of repeat anesthesia, neurosurgical or non-neurosurgical. Air frequently remains evident on CT for more than 7 days after a craniotomy. Pneumocephalus can also develop de novo in the postoperative period in patients who have a residual dural defect and communication between the nasal sinuses and the intracranial space.

VENOUS AIR EMBOLISM

The rate of occurrence of venous air embolism (VAE) varies according to the procedure, the intraoperative position, and the method of detection used. During posterior fossa procedures performed in the sitting position, VAE is detectable by precordial Doppler in approximately 40% of patients and by TEE in up to 76%. The incidence of VAE during posterior fossa procedures performed in nonsitting positions is much less (12% with the use of precordial Doppler in the report of Black and associates), and it is probable, but unproven that the average volume of air entrained per event is also smaller. The rate of VAE is apparently lower with cervical laminectomy (25% using TEE in the sitting position versus 76% for posterior fossa procedures). Although VAE is principally a hazard of posterior fossa and upper cervical spine procedures, especially when performed in the sitting position, it can occur with supratentorial procedures. The most common situations will involve tumors, most often parasagittal or falx meningiomas that encroach on the posterior half of the sagittal sinus (Fig. 53-10), craniosynostosis procedures, which are typically performed in children. Pin sites and trapped gas under pressure can also lead to VAE, although clinically relevant events have been very rare.

Figure 53-10 Horizontal (top) and coronal magnetic resonance images of a parasagittal meningioma. Resection of meningiomas arising from the dural reflection overlying the sagittal sinus or from the dura of the adjacent convexity or falx entails a risk of venous air embolism because of the proximity of the sagittal sinus (the triangular structure at the superior end of the interhemispheric fissure in the bottom panel).
Common sources of critical VAE are the major cerebral venous sinuses, in particular, the transverse, the sigmoid, and the posterior half of the sagittal sinus, all of which may be noncollapsible because of their dural attachments. Air may also enter through emissary veins, particularly from the suboccipital musculature, the diploic space of the skull (which can be violated by both the cranietomy and pin fixation), and the cervical epidural veins. It is our belief (not confirmed by systematic study) that the risk of VAE associated with cervical laminectomy is greatest when the exposure requires dissection of suboccipital muscle with the potential to open emissary veins to the atmosphere at their point of entry into occipital bone. There is also anecdotal evidence (see the review by Matjasko[103] that air under pressure in the ventricles or subdural space can occasionally enter the venous system, perhaps following CSF’s normal route of egress.

DETECTION OF VENOUS AIR EMBOLISM

Monitors used for detection of VAE should provide (1) a high level of sensitivity, (2) good specificity, (3) a rapid response, (4) a quantitative measure of the VAE event, and (5) an indication of the course of recovery from the VAE event. The combination of precordial Doppler and expired CO₂ monitoring meets these criteria and is the current standard of care. Doppler placement in a left or right parasternal location in the second to fourth intercostal space has a very high detection rate for gas embolization,[105] and when good heart tone is obtained, maneuvers to confirm adequate placement appear to be unnecessary. TEE is more sensitive to VAE than precordial Doppler is[106] (Fig. 53-11) and offers the advantage of identifying right-to-left shunting of air.[105][106][107][108][109] However, its safety during prolonged use (especially with pronounced neck flexion) is not well established. Expired nitrogen analysis is theoretically attractive. However, the expired N₂ concentrations involved in anything less than catastrophic VAE are very small and push the available instrumentation to the limits of its sensitivity.[110] Furthermore, effective application requires absolute freedom from air contamination of the ventilator and anesthetic circuit.

![Diagram](Figure 53-11 Relative sensitivity of various monitoring techniques to the occurrence of venous air embolism. BP, blood pressure; C.O., cardiac output; CVP, central venous pressure; ECG, electrocardiogram; ET-CO₂, end-tidal CO₂; PAP, pulmonary artery pressure; T-ECHO, transesophageal echocardiography.)
Figure 53-12 presents the physiologic and monitor response to an air embolic event. Table 53-6 offers an appropriate management response to such an event.

Figure 53-12 Responses of the electrocardiogram (ECG), arterial blood pressure (BP), pulmonary artery pressure (PAP), pan-ideal CO₂ concentration, precordial Doppler, and central venous pressure (CVP) to the intravenous administration of 10 cc of air over a 30-second period to an 11-kg dog.

Table 53-6 — Management of acute air embolic events

<table>
<thead>
<tr>
<th>1. Prevent further air entry</th>
</tr>
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<tbody>
<tr>
<td>Notify surgeon (flood or pack surgical field)</td>
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<tr>
<td>Jugular compression</td>
</tr>
<tr>
<td>Lower the head</td>
</tr>
</tbody>
</table>

<table>
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<tr>
<th>2. Treat the intravascular air</th>
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<tbody>
<tr>
<td>Aspirate via a right heart catheter</td>
</tr>
<tr>
<td>Discontinue N₂O</td>
</tr>
<tr>
<td>F₁₀₂: 1.0</td>
</tr>
<tr>
<td>(Pressors/inotropes)</td>
</tr>
<tr>
<td>(Chest compression)</td>
</tr>
</tbody>
</table>
WHICH PATIENTS SHOULD HAVE A RIGHT HEART CATHETER?

Essentially all patients who undergo sitting posterior fossa procedures should have a right heart catheter. Although catastrophic, life-threatening VAE is relatively uncommon, a catheter that permits immediate evacuation of an air-filled heart will occasionally be the sine qua non for resuscitation. Latitudes are much wider with the nonsitting positions, and it is frequently appropriate, after a documented discussion with the surgeon, to omit the right heart catheter. The perceived risks of VAE associated with the intended procedure and the patient's physiologic reserve are the variables that will contribute to the decision. Microvascular decompression of the fifth or seventh cranial nerves for tic douloureux and hemifacial spasm, respectively, is an example of procedures for which the right heart catheter is usually omitted. The essentially horizontal semilateral position and the very limited retromastoid craniectomy that these procedures require have resulted (at our institution) in a very low incidence of Doppler-detectable VAE. However, one should come to know the local surgical practices, particularly with respect to the degree of head-up posture, before becoming casual about omitting the right atrial line. For instance, with regard to the Janetta procedure, recall that the necessary retromastoid craniectomy is performed in the angle between the transverse and sigmoid sinuses and that venous sinuoids and emissary veins in the suboccipital bone are common. If this procedure is performed with any degree of head-up posturing, the risk of VAE may still be substantial.

WHICH VEIN SHOULD BE USED FOR RIGHT HEART ACCESS?

Although some surgeons may ask that neck veins not be used, a skillfully placed jugular catheter is often acceptable. In a very limited number of patients, high ICP may make the head-down posture undesirable. In others, unfavorable anatomy with an increased likelihood of difficult cannulation and formation of hematomas may also encourage the use of alternative access sites. Access to the brachial veins has been greatly facilitated by the commercial availability of multiorifice catheters that use a Seldinger cannulation technique and a lengthy J-tipped guidewire to negotiate the axilla or deltopectoral groove.

POSITIONING THE RIGHT HEART CATHETER

The investigation of Bunegin and colleagues suggests that a multiorifice catheter should be located with the tip 2 cm below the superior vena cava (SVC)-atrial junction and a single-orifice catheter with the tip 3 cm above the SVC-atrial junction.\textsuperscript{11,12} Although these small distinctions in location may be relevant for optimal recovery of small volumes of air when cardiac output is well maintained, for the recovery of massive volumes of air in the face of cardiovascular collapse, anywhere in the right atrium should suffice. Confirmation of right heart placement can be accomplished by (1) radiographs, (2) pull back from the right ventricle while monitoring intravascular pressure, or (3) intravascular electrocardiography (ECG).\textsuperscript{11,12} Although no literature in support of the practice has been published, with catheter access through the right internal jugular vein, measured placement to the level of the second right intercostal space should suffice when the catheter passes readily. The intravascular ECG technique makes use of the fact that an ECG "electrode" placed in the middle of the right atrium will initially "see" an increasing positivity as the developing P-wave vector approaches it (Fig. 53-13) and then an increasing negativity as the wave of atrial depolarization passes and moves away from it. The resultant biphasic P wave is characteristic of a mid-atrial "electrode" position. The technique requires that the central venous pressure (CVP) catheter become an exploring ECG electrode, which is accomplished by filling the catheter with an electrolyte solution (bicarbonate is best) and attaching an ECG lead (the leg lead if lead II is selected) to the hub of the CVP catheter. Commercial CVP kits with an ECG adapter are available. The ECG configurations that will be observed at various intravascular locations are shown in Figure 53-13. To minimize
the microshock hazard, a battery-operated ECG unit is preferable, and unnecessary electrical apparatus should be detached from the patient during placement of the catheter.

**Figure 53-13** Electrocardiographic (ECG) configurations observed at various locations when a central venous catheter is used as an intravascular ECG electrode. The configurations in the figure will be observed when "lead II" is monitored and the positive electrode (the leg electrode) is connected to the catheter. P indicates the sinoatrial node. The heavy black arrow indicates the P-wave vector. Note the equi-biphasic P wave when the catheter tip is in the mid-right atrial position.  

**PARADOXICAL AIR EMBOLISM**

Much concern has been raised about the possibility of the passage of air across the interatrial septum through a patent foramen ovale (PFO) (known to be present in approximately 25% of adults).  

The concern is that this phenomenon carries the risk of major cerebral or coronary morbidity, or both, although the morbidity that can realistically be attributed to a paradoxical air embolism (PAE) has not been precisely defined. Even though the precise pressure required to open a PFO is not known with certainty, it is thought that the gradient necessary may be as much as 5 mm Hg. In a clinical investigation, Mammo and coworkers observed that PAE occurred only in the context of major air embolic events, thus suggesting that significant increases in pressure in the right side of the heart are an important predisposing factor for the occurrence of PAE.  

Several clinical investigations have examined factors that influence the right atrial-to-left atrial pressure gradient. The use of positive end-expiratory pressure (PEEP) was shown to increase the incidence of a positive right atrial pressure (RAP)-pulmonary capillary wedge pressure (PCWP) gradient, and generous fluid administration (e.g., 2800 mL per patient versus 1220 mL per control patient) was shown to reduce it. As a result, the use of PEEP, which had previously been advocated as a means of preventing air entrapment, diminished and the practice of more generous fluid administration to patients undergoing posterior fossa procedures evolved. However, subsequent data indicated that even when mean left atrial pressure (LAP) exceeds mean RAP, PAE can still develop because transient reversal of the interatrial pressure gradient can occur during each cardiac cycle.  

Some centers have advocated the use of preoperative precordial echocardiography or prepositioning intraoperative TEE examination to identify patients with a PFO with a view to using alternatives to the sitting position in this subpopulation. However, this practice is not universal and at the time of writing is not a community-wide standard of care. Furthermore, because the morbid events attributable to PAE have been quite infrequent, surgeons who are convinced that the
sitting position is optimal for a given procedure are loath to be dissuaded from using it on the basis of what may seem like a very remote possibility of an injury occurring to the patient by this mechanism.

For those undertaking prepositioning detection of a PFO, it should be understood that TEE-based methods are more efficient in detecting a PFO than transthoracic echocardiographic techniques are. The greatest sensitivity is achieved with the combination of TEE contrast imaging and color Doppler imaging, in part because the former may fail to identify shunting if LAP is persistently higher than RAP at the time of study.\textsuperscript{123}

**TRANSPIRATORY PASSAGE OF AIR**

It appears likely that air can occasionally traverse the pulmonary vascular bed to reach the systemic circulation.\textsuperscript{124,125} Transpulmonary passage is more likely to occur when large volumes of air are presented to the pulmonary vascular "filter."\textsuperscript{125} Evidence also suggests that pulmonary vasodilators,\textsuperscript{126} including volatile anesthetics, may lower the threshold for transpulmonary passage.\textsuperscript{127} The magnitude of differences among anesthetics does not appear, to these reviewers, to mandate any related "tailoring" of anesthetic techniques. However, it does reinforce the notion that N\textsubscript{2}O should be discontinued promptly after even apparently minor VAE events because of the possibility that air may reach the left-sided circulation through either a PFO or the pulmonary vascular bed.

**TECHNIQUES FOR REDUCING THE INCIDENCE OF VENOUS AIR EMBOLISM**

As noted earlier, PEEP has been advocated in the past as a means of both reducing the incidence of VAE and responding to an acute VAE event to prevent further air entry. However, a study by Perkins and Bedford\textsuperscript{114} presented data suggesting that PEEP increases the risk of PAE and that these data therefore argue against the use of PEEP in patients undergoing seated neurosurgical procedures. Furthermore, as the authors point out, even 10 cm H\textsubscript{2}O of PEEP would be unlikely to result in positive venous pressure in cerebral venous structures, which may be as much as 25 cm above the heart. The ineffectiveness of PEEP\textsuperscript{128} and the relative superior of jugular venous compression\textsuperscript{129,130} in raising CVP have been confirmed by other investigations. An inflatable neck tourniquet available for rapid inflation in the event of VAE has been studied in animals and used in humans by Pfitzner and McLean.\textsuperscript{131} The G-suit has also been reported to be more effective in producing increases in RAP than 10 cm H\textsubscript{2}O of PEEP is and can do so without increasing the RAP-PCWP gradient.\textsuperscript{132} This latter report is available only in a non-peer-reviewed (abstract) form. There are additional arguments against the acute use of PEEP in the event of VAE. The various investigations of the identification of PFOs have confirmed the efficacy of a Valsalva maneuver, in particular, its release, as a means of promoting paradoxical embolism.\textsuperscript{117,132,133} In addition, the impairment in systemic venous return caused by the sudden application of substantial PEEP may be undesirable in the face of the cardiovascular dysfunction already caused by the VAE event.

It has been recommended that a patient who has sustained a hemodynamically significant VAE be placed in a lateral position with the right side up. The rationale is that air will remain in the right atrium where it will not contribute to air lock in the right ventricle and where it will remain amenable to recovery with a right atrial catheter. The first difficulty is that such repositioning is all but impossible with a patient in a pin head holder. In addition, the only systematic attempt to examine the efficacy of this maneuver, albeit performed in dogs, failed to identify any hemodynamic benefit.\textsuperscript{133}
**NITROUS OXIDE**

$N_2O$ will diffuse into air bubbles trapped in the vascular tree, and accordingly, $N_2O$ should be eliminated after a clinical VAE event to avoid aggravating the cardiovascular impact. As noted earlier, the PAE phenomenon adds an additional reason for eliminating $N_2O$ after the occurrence of VAE. When a major VAE occurs, no matter how the RAP-LAP gradient was manipulated before the event, RAP will rise abruptly with respect to LAP, and a major VAE will result in an acutely increased risk of PAE in patients with a PFO. Should $N_2O$ be used at all in patients at risk for VAE? Some will decide that it is the "path of least resistance" to simply avoid it and thereby avoid having to worry about the considerations that it creates. However, $N_2O$ can be used with the knowledge that it neither increases the incidence of VAE, nor aggravates the hemodynamic response to VAE, provided that it is eliminated when VAE occurs.

**MONITORING**

Neurologic monitoring techniques are discussed in Chapter 38. Invasive monitoring is frequently appropriate in neurosurgery. Some of the numerous indications for an arterial catheter are listed in Table 53-7.

<table>
<thead>
<tr>
<th>Table 53-7 -- Relative indications for intra-arterial pressure monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated intracranial pressure</td>
</tr>
<tr>
<td>Ischemia or incipient ischemia of neurologic tissue</td>
</tr>
<tr>
<td>Recent subarachnoid hemorrhage</td>
</tr>
<tr>
<td>Recent head injury</td>
</tr>
<tr>
<td>Recent spinal cord injury</td>
</tr>
<tr>
<td>Intended or potential temporary vessel occlusion</td>
</tr>
<tr>
<td>Circulatory instability</td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Spinal cord injury (spinal shock)</td>
</tr>
<tr>
<td>Sitting position</td>
</tr>
<tr>
<td>Possible barbiturate coma</td>
</tr>
<tr>
<td>Possibility of induced hypotension</td>
</tr>
<tr>
<td>Possibility of induced hypertension</td>
</tr>
<tr>
<td>Anticipated/potential major blood loss</td>
</tr>
</tbody>
</table>

Patients with increased ICP may be intolerant of the vascular engorgement associated with sudden hypertension occurring as a consequence of light anesthesia. Surgical relief of increased ICP may be associated with sudden hypotension as brainstem compression is relieved. Beat-by-beat arterial pressure monitoring also serves as an important depth-of-anesthesia monitor and as an early warning system for neurologic injury. Much of the brain is insensate. As a consequence, the intracranial portion of many neurosurgical procedures is not very
stimulating, and to achieve circulatory stability, relatively light anesthesia is often necessary. There should be
constant attention to the possibility of sudden arousal (most often associated with cranial nerve traction or
irritation), especially when paralysis is precluded by the use of electromyographic recording of facial muscles to
monitor cranial nerve integrity. Blood pressure responses may reveal imminent arousal. They may also serve to
warn a surgeon of excessive or unrecognized irritation, traction, or compression of neurologic tissue. These
problems occur most often with posterior fossa procedures involving the brainstem or cranial nerves, and abrupt
changes should be reported to the surgeon immediately.

The use of right heart catheters for air retrieval is discussed in the section "Venous Air Embolism." Thereafter,
anticipated blood loss and fluid flux (including aggressive mannitol use) and an evaluation of the patient's
physiologic reserve should determine the necessity for CVP or pulmonary artery catheters. The use of
precordial Doppler is also described in the section "Venous Air Embolism."

**INTRANOVUS FLUID MANAGEMENT**

The general principles of fluid management for neurosurgical anesthesia are (1) maintenance of normovolemia
and (2) avoidance of a reduction in serum osmolarity. The first principle is a derivative of the concept presented
in the section "Management of Blood Pressure" that it is in general ideal to maintain normal MAP in patients
undergoing most neurosurgical procedures and neurosurgical critical care. Maintaining normovolemia is
simply one element of maintaining normal MAP. The second principle is a derivative of the oft-repeated
observation that lowering serum osmolarity results in edema of both normal and abnormal brain. Administering
fluids that provide free water (i.e., fluids that do not have sufficient non-glucose-containing solutes to render
them iso-osmolar with respect to blood) will lower serum osmolarity if the amount of free water administered is
in excess of that required to maintain ongoing free water loss. Half-normal saline is probably a reasonable
choice for the traditional maintenance fluid allowance. However, fluids administered to replace blood and third-
space loss (i.e., iso-osmolar losses) should be more nearly iso-osmolar with respect to plasma (295 mOsm/L).
Normal saline and lactated Ringer's solution (LR) are often used. Normal saline is in fact slightly hyperosmolar,
308 mOsm/L. It has the disadvantage that in large volumes it can cause hyperchloremic metabolic acidosis.
The physiologic significance of this acidosis, which involves the extracellular but not the intracellular fluid
space, is unclear. At a minimum, it has the potential to confuse the diagnostic picture when acidosis is present.
As a result, many clinicians use predominately LR. Although LR (273 mOsm/L) is in theory not ideal for either
maintenance fluids or replacement of losses individually, it serves as an entirely reasonable compromise for
meeting both needs simultaneously and is very suitable in most instances. However, it is a hypoosmolar fluid,
and in healthy experimental animals, it is possible to reduce serum osmolarity and produce cerebral edema with
a large volume of LR. Therefore, in the setting of large-volume fluid administration, such as significant
blood loss and multiple trauma, it is our practice to alternate, liter by liter, LR and normal saline.

The crystalloid versus colloid discussion is a recurrent one that usually arises in the context of a head injury
victim. Despite numerous fervent beliefs regarding this issue, there has in fact been only a single demonstration
that a reduction in colloid oncotic pressure in the absence of a change in osmolarity can actually contribute to
augmentation of cerebral edema in the setting of experimental head injury. The transcapillary membrane
pressure gradients that can be produced by a reduction in colloid oncotic pressure are in fact very small by
comparison to those created by changes in serum osmolarity. Nonetheless, it appears that these small gradients
do, probably in the setting of a blood-brain barrier injury of intermediate severity, have the potential to augment
edema. It seems reasonable, then, to select a fluid administration pattern that in addition to maintaining normal
serum osmolarity, will prevent substantial reductions in colloid oncotic pressure. For the large majority of
elective craniotomies, administration of colloid solutions will not be required. However, in situations requiring substantial volume administration (multiple trauma, aneurysm rupture, cerebral venous sinus laceration, fluid administration to support filling pressure during barbiturate coma), a combination of isotonic crystalloid and colloid may be appropriate.

Which colloidal solutions should be used? Albumin is a reasonable choice. Dextran-containing solutions are generally avoided because of their effects on platelet function. The various starch-containing solutions should be used cautiously in neurosurgery because in addition to a dilutional reduction of coagulation factors, they interfere directly with both platelets and the factor VII complex.\textsuperscript{143,144,145,146} Note that the literature addressing the coagulation effects of starches should be read with the understanding that the effects on coagulation are proportional to the average molecular weight of the starch preparation. Unfortunately, the preparations in use in North America are those with the greatest molecular weights. Accordingly, although hetastarch solutions will be used in neuroanesthesia, the clinician should respect the manufacturer's recommended dose restrictions and use additional restraint in situations with other reasons for impairment of the coagulation mechanism. Several reported instances of bleeding in neurosurgical patients have been attributed to hetastarch administration. However, all of them have involved circumstances in which the manufacturer's recommended limit of 20 mL/kg/day was exceeded\textsuperscript{147} or in which hetastarch was administered up to the recommended limit on successive days, probably resulting in an accumulation effect.\textsuperscript{148} The decision to use or not use these products will frequently be a matter of local bias. If used, the manufacturer's recommended limit (20 mL/kg/24 hr) should be observed.

Substantial current interest has been generated in the use of hypertonic fluids, in particular, in the resuscitation of a multiple-trauma victim.\textsuperscript{149} It appears unlikely that the ICP effect of these fluids on the brain is substantially different from an equiosmolar exposure to mannitol.\textsuperscript{150,151} Accordingly, when decisions about the appropriateness and relevance of these fluids in the resuscitation process are eventually made, it seems unlikely that these decisions should be made on the basis of specific cerebral effects. It seems more likely that the final judgment regarding this class of fluids will be made on the basis of their effects on the systemic circulation.\textsuperscript{150} In general, an intervention that has the advantage of more effectively restoring systemic hemodynamics is likely to be advantageous to the injured brain. That assertion is made with the proviso that sustained hyperosmolarity (e.g., >320 mOsm/L) caused by any fluid has the potential to result in rebound swelling of the brain.

**HYPOTHERMIA**

The effects of hypothermia on cerebral physiology and its potential cerebral protective mechanisms have been presented in Chapter 21 (see Table 21-8 and text). There have been numerous laboratory demonstrations of the efficacy of mild hypothermia (32°C to 34°C) in reducing the neurologic injury occurring after standardized cerebral and spinal cord ischemic insults. These investigations have resulted, in spite of the absence of any demonstration of efficacy in humans, in the relatively widespread use of induced hypothermia in the management of cerebral vascular procedures, in particular, aneurysms\textsuperscript{152,153} and occasionally AVMs. An international multicenter trial of mild hypothermia in aneurysm surgery has been undertaken. Enrollment of the intended 1000 patients was completed in April 2003 (M.M. Todd, personal communication), but the results were unavailable at the time of this writing.

Despite the fact that mild hypothermia is perceived to convey certain hazards, including coagulation dysfunction and an increased postoperative wound infection rate, neither of these problems has been evident in the context of aneurysm surgery, although once again no systematic study has been conducted. Anecdotally,
hypertension on emergence has been noted to occur in patients who are not adequately rewarmed, and a modest overshoot in temperature has been observed to occur in patients who were cooled intraoperatively. The issue of where body temperature should be recorded to best reflect brain temperature during craniotomy has been addressed. It appears that esophageal, tympanic membrane, pulmonary artery, and jugular bulb temperatures are all very similar and provide a reasonable reflection of deep brain temperature whereas bladder temperature does not. Superficial layers of cortex may be substantially cooler than deep brain and central temperatures.

Because ischemia is recognized to make a postsinult contribution to neuronal injury after head injury, hypothermia was also studied in the laboratory in the context of traumatic brain injury. Its efficacy resulted in at least four single-institution trials of 24 to 48 hours of mild hypothermia after head injury. These trials demonstrated either significant improvement in outcome or favorable trends. The investigations revealed some physiologic dysfunction associated with more prolonged mild hypothermia, all of which was reversible with restoration of normal temperature. Physiologic dysfunction included decreased creatinine clearance, elevation of pancreatic enzymes, and a suggestion of an increased infection rate. A decreased incidence of seizures was an apparent adjunctive benefit in one study. As a result, a multicenter, prospective trial of hypothermia was performed but did not show a benefit of hypothermia (33°C) induced within 8 hours of injury and maintained for 48 hours. Post hoc subgroup analysis indicated that patients younger than 45 years who arrived at the tertiary care facility with a temperature lower than 35°C and were randomized to the cooling limb of the trial did, in fact, have an improved outcome. A follow-up trial in which the protocol seeks to achieve cooling more rapidly is under way.

On the basis of lack of demonstrated efficacy in humans, routine use of hypothermia in neurosurgery cannot be advocated in a standard text. The decision to use it, generally in the context of aneurysm surgery, will be a local one. If used, attention should be paid to the possibility of cardiac dysrhythmias and coagulation dysfunction if temperatures become too low, as well as the necessity of adequate rewarming before emergence to avoid shivering and hypertension.

Yet another application of mild hypothermia is in evolution. Very recently, two multicenter trials have demonstrated improved neurologic outcome in survivors of witnessed cardiac arrest who were cooled to 32°C to 34°C within 4 hours and maintained at that temperature for 12 to 24 hours. Consideration of bringing this therapy to bear on victims of intraoperative cardiac arrest or other neurologically threatening events seems inevitable, but to our knowledge, it has not yet been attempted.

EMERGENCE FROM ANESTHESIA

Most practitioners of neuroanesthesia believe that a premium should be placed on "smooth" emergence, that is, one free of coughing/straining and arterial hypertension. Avoidance of arterial hypertension is seen as desirable because of the belief that arterial hypertension can contribute to intracranial bleeding and increased edema formation. In the face of a poorly autoregulating cerebral vasculature, hypertension also has the potential, through vascular engorgement, to contribute to elevation of ICP. Much of the concern with coughing/straining has a similar basis. The sudden increases in intrathoracic pressure are transmitted to both arteries and veins, and the transient increases produced in both cerebral arterial and venous pressure have the same potential consequences: edema formation, bleeding, and elevation of ICP. Coughing is a specific concern with certain individual procedures. In the circumstances of transphenoidal pituitary surgery in which the surgeon has opened and subsequently taken pains to close the arachnoid membrane to prevent leakage of CSF, it is believed that coughing has the potential to disrupt this closure because of sudden and substantial increases
in CSF pressure. Opening a pathway from the intracranial space to the nasal cavity conveys a substantial risk of postoperative meningitis. In other procedures, notably those that have violated the floor of the anterior fossa, there is also the potential for air to be driven into the cranium and, in the event of a flap valve mechanism, cause a tension pneumocephalus. This latter event can take place only when coughing occurs after the endotracheal tube has been removed.

It should be acknowledged that there is a paucity of systematically obtained clinical data to give a perspective to the actual magnitude of the risks associated with an emergence that is not "smooth." One clinical investigation confirmed the association of increased CBF velocity and hypertension occurring during emergence.\textsuperscript{170} A second retrospective study revealed that elevated postoperative blood pressure was a correlate of intracerebral bleeding after craniotomy.\textsuperscript{171} However, there is in fact no proof to show that it is specifically hypertension occurring at emergence that is correlated with postoperative intracerebral bleeding. The same acknowledgment must be made with the matter of edema formation. It has been demonstrated in anesthetized animals that sudden substantial increases in arterial pressure can result in breach of the blood-brain barrier with extravasation of tracers such as Evan's blue.\textsuperscript{167} However, no data have confirmed that the hypertensive pressure transients associated with the typical coughing episode or with emergence are in fact associated with increased edema formation. Nonetheless, it seems reasonable to take measures, to the extent that these measures do not themselves add potential patient morbidity, to prevent these occurrences.

A common fomr for the management of systemic hypertension during the last stages of a craniotomy is the expectant or reactive administration (or both) of vasoactive drugs, most commonly labetalol and esmolol.\textsuperscript{172} Other drugs, including enalapril and diltiazem, have been used to good effect. Administration of dexmedetomidine during the procedure and up to 30 to 60 minutes before conclusion of the procedure has also been reported to lessen the requirement for antihypertensives during emergence.\textsuperscript{173} There are also many approaches to the prevention of coughing and straining. We have several biases, however. We encourage trainees to include in their anesthetic technique "as much narcotic as is consistent with spontaneous ventilation at the conclusion of the procedure." That practice is based on the same physiologic effect that justifies the administration of codeine and related compounds as antitussive medication, specifically, the depression of airway reflexes by narcotics. We also have the bias that patients emerge more rapidly and smoothly when the last inhaled anesthetic to be withdrawn is nitrous oxide and that clinicians should seek to avoid the "neither here nor there" phase of anesthesia that occurs in patients who are stimulated in the face of residual exhaled concentrations of volatile anesthetic on the order of 0.2 to 0.3 MAC. A common practice among neuroanesthetists near the conclusion of a craniotomy is the relatively early discontinuation of the volatile anesthetic and supplementation of residual nitrous oxide with propofol by either bolus increments or infusion at rates in the range of 25 to 100 μg/kg/min.

An additional principle relevant to emergence from neurosurgical procedures that practitioners will learn either from a book or by bad experience is that emergence should be timed to coincide, not with the final suture, but rather with the conclusion of the application of the head dressing. Many a good anesthetic for neurosurgery has been spoiled by severe coughing and straining that occur in association with endotracheal tube motion during application of the head dressing. Another nuance of our practice has been to withhold the administration of neuromuscular antagonists as long as possible as a hedge against misjudgment while lightening anesthesia in a patient in the later stages of the procedure. An additional popular and apparently effective technique for reducing airway responsiveness and the likelihood of coughing/straining while reducing the depth of anesthesia is the administration of lidocaine. Bolus doses on the order of 1.5 mg/kg, often given as application of the head dressing begins, are appropriate for this purpose.
The premium placed on minimizing coughing/straining and hypertension will result, in most instances, in patients being extubated very expeditiously once extubation is appropriate. In some instances, one may be tempted to extubate patients before complete recovery of consciousness. This practice may be acceptable in some circumstances. However, it should be undertaken with caution when the circumstances of the surgical procedure make it possible that neurologic events may have occurred that will delay recovery of consciousness or when lower cranial nerve dysfunction may be present. In these circumstances, it will generally be best to wait until the likelihood of the patient's recovery of consciousness is confirmed or until patient cooperation and airway reflexes are likely to have recovered (or until both).

**SPECIFIC PROCEDURES**

Many of the considerations relevant to individual neurosurgical procedures are generic ones that have already been presented in the section on recurrent issues. The descriptions that follow will highlight only procedure-specific issues.

**SUPRATENTORIAL TUMORS**

Craniotomies for excision or biopsy of supratentorial tumors are among the most common neurosurgical procedures performed. Gliomas and meningiomas are among the most frequent tumors. Relevant preoperative considerations include the patient's ICP status (see the earlier section "Control of Intracranial Pressure/Brain Relaxation") and the location and size of the tumor. Location and size will give the anesthesiologist an indication of the surgical position and the potential for blood loss and, occasionally, will reveal a risk for air embolism. The risk of VAE developing is quite low for most other supratentorial tumors. However, lesions (usually convexity meningiomas) that encroach on the sagittal sinus may convey a substantial risk of VAE. Accordingly, full VAE precautions, including an atrially placed CVP catheter, are usually reserved for only supratentorial tumors that lie near the posterior half of the sagittal sinus.

Patients with craniopharyngiomas and pituitary tumors with suprasellar extension may undergo procedures that involve dissection in and around the hypothalamus. Irritation of the hypothalamus can elicit sympathetic responses, including hypertension. Damage to the hypothalamus can result in a spectrum of disturbances in consciousness varying from lethargy to obtundation. Disturbances in water balance may also occur. Diabetes insipidus is the most likely, although the cerebral salt-wasting syndrome can potentially occur. The latter has been very infrequent. The various disturbances in water balance typically have a delayed onset and begin 12 to 24 hours postoperatively rather than in the operating room. Postoperative temperature homeostasis may also be disturbed.

Patients who undergo a craniotomy involving a subfrontal approach may, on occasion, manifest a disturbance of consciousness in the immediate postoperative period. Retraction/irritation of the inferior surfaces of the frontal lobes can result in a patient who is lethargic and will not awaken "cleanly." Patients exhibiting this phenomenon are sometimes referred to "frontal lobey." The phenomenon is more likely to be evident in the event of bilateral subfrontal retraction than when it occurs only unilaterally. The anesthetic implication is that the clinician should be more inclined to confirm the return of consciousness before extubating the patient than to extubate expectantly. A further implication taken by us (though not confirmed by any systematic study) is that less liberal use of fixed agents (narcotics, benzodiazepines) may be appropriate when a bilateral, subfrontal retraction is to be performed. This recommendation is based on the rationale that the low residual concentrations of these drugs that would be compatible with reasonable recovery of consciousness in most
patients may be less well tolerated in this population. Subfrontal approaches will be most commonly used in patients with olfactory groove meningiomas and those with suprasellar tumors (craniopharyngiomas and pituitary tumors with suprasellar extension).

PREOPERATIVE PREPARATION

Patients with a significant tumor-related mass effect, especially if tumor-related edema is present, should receive preoperative steroids. If the patient is not receiving steroids, it is the anesthesiologist's responsibility to ask why. A 48-hour course is ideal (see the earlier section "Steroids"), although 24 hours is sufficient for a clinical effect to be evident. Dexamethasone is the most commonly used steroid. A regimen such as 10 mg intravenously or orally followed by 10 mg every 6 hours is typical. Because of concern about producing carbon dioxide retention in a patient whose intracranial compliance is already abnormal, patients with any substantial mass effect are not usually premedicated with anything beyond full-dose reassurance.

MONITORING

Frequently, the nature of the procedure does not require anything more than routine monitoring. However, some situations do argue for invasive monitors (see the section "Monitoring"). Preinduction placement of an arterial line may be appropriate in patients with a severe mass effect and little residual compensatory latitude. It is the period of induction during which hypertension, with its attendant risks in a patient with impaired compliance and autoregulation, is most likely to occur. Procedures with a potential for substantial blood loss (tumors encroaching on the sagittal sinus, large vascular tumors) may also justify arterial or CVP catheters, or both. Is ICP monitoring ever warranted for intraoperative management? In our opinion the answer is no. We have sufficient understanding of the potential impact of anesthetic drugs and techniques that we should be able to manage induction of anesthesia "blind." Then, once the cranium is open, observation of conditions in the surgical field provides equivalent information.

MANAGEMENT OF ANESTHESIA

The principles governing the choice of anesthetics are presented under the subheading "Selection of Anesthetics" in "Control of Intracranial Pressure/Brain Relaxation."

ANEURYSMS AND ARTERIOVENOUS MALFORMATIONS

Contemporary management of intracranial aneurysms calls for early surgical intervention after SAH. The definition of "early" varies up to and including the first 72 hours after bleeding. This approach was originally applied only to patients in the better neurologic grades, that is, grades I to III and perhaps IV of the World Federation of Neurosurgeons (WFNS) classification (Table 53-8) or grades I to III of the Hunt-Hess classification (Table 53-9), but more recently it has been extended to patients with higher grades. If early intervention is not feasible, surgery is usually delayed for at least 2 weeks to be safely beyond the period of maximal risk of vasospasm (i.e., days 4 to 12 after SAH). Currently, some clinicians are advocates of the so-called ultra-early intervention, which entails clipping within 18 hours of the initial SAH.
Table 53-8 -- World Federation of Neurosurgeons (WFNS) SAH Scale

<table>
<thead>
<tr>
<th>WFNS Grade</th>
<th>GCS Score</th>
<th>Motor Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>15</td>
<td>Absent</td>
</tr>
<tr>
<td>II</td>
<td>14–13</td>
<td>Absent</td>
</tr>
<tr>
<td>III</td>
<td>14–13</td>
<td>Present</td>
</tr>
<tr>
<td>IV</td>
<td>12–7</td>
<td>Present or absent</td>
</tr>
<tr>
<td>V</td>
<td>6–3</td>
<td>Present or absent</td>
</tr>
</tbody>
</table>

GCS, Glasgow Coma Scale; SAH, subarachnoid hemorrhage.


Table 53-9 -- Hunt-Hess classification of neurologic status after subarachnoid hemorrhage

<table>
<thead>
<tr>
<th>Category</th>
<th>Criteria*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>Asymptomatic or minimal headache and slight nuchal rigidity</td>
</tr>
<tr>
<td>Grade II</td>
<td>Moderate to severe headache, nuchal rigidity, no deficit other than cranial nerve palsy</td>
</tr>
<tr>
<td>Grade III</td>
<td>Drowsiness, confusion, or mild focal deficit</td>
</tr>
<tr>
<td>Grade IV</td>
<td>Stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity and vegetative disturbances</td>
</tr>
<tr>
<td>Grade V</td>
<td>Deep coma, decerebrate rigidity, moribund appearance</td>
</tr>
</tbody>
</table>

*Serious systemic diseases such as hypertension, diabetes, severe arteriosclerosis, chronic pulmonary disease, and severe vasospasm seen on arteriography result in placement of the patient in the next less favorable category.


The rationale for early clipping is several-fold. The sooner the aneurysm is clipped, the less the likelihood of rebleeding (and rebleeding is the principal cause of death in patients hospitalized after SAH[170]). Second, management of the ischemia caused by vasospasm involves volume loading and induced hypertension. Early clipping of the aneurysm eliminates the risk of rebleeding associated with this therapy. Vasospasm appears to be related to the presence of blood in the basal cisterns in the vicinity of the circle of Willis. Some of this blood can be removed at the time of aneurysm clipping, and accordingly, early clipping not only makes therapy for vasospasm safer but may also reduce the incidence and severity of the problem. In addition, early access to the circle of Willis allows direct instillation of tissue plasminogen activator into the basal cisterns to further aid in clearing clot from the circle of Willis[178][179]. The results of the initial evaluations of tissue plasminogen activator therapy are suggestive of a substantial benefit, although this therapy remains experimental. Previous surgical practices entailed maintaining the patient at bed rest until approximately day 14, when the period of risk for spasm had passed. Early aneurysm clipping reduces the period of hospitalization and decreases the incidence of medical complications (deep venous thrombosis, atelectasis, pneumonia) associated with a lengthy period of enforced bed rest.

Early intervention can make the intraoperative course more difficult. Brain tissue in the early post-SAH period is likely to be more edematous than after a 2-week delay. A mild degree of hydrocephalus is common after
blood contaminates the subarachnoid space. In fact, 10% to 20% of SAH victims may actually require CSF shunting at some point in their course. Early intervention may also enhance the risk of intraoperative aneurysm rupture somewhat because of the lesser time for clot to organize over the site of the initial bleeding. All of these issues place a substantial premium on techniques designed to reduce the volume of the intracranial contents (see the section "Control of Intracranial Pressure/Brain Relaxation") to facilitate exposure and minimize retraction pressure.

PREOPERATIVE EVALUATION

Many patients scheduled for intracranial aneurysm clipping will come directly from the ICU, and elements of their management there may influence their immediate preoperative status.

Fluid Management.

In some patients, the syndrome of inappropriate antidiuretic hormone secretion (SIADH) develops after SAH, and it will be appropriately managed with fluid restriction. However, hyponatremia after SAH is more likely to be a consequence of the cerebral salt-wasting syndrome that probably occurs as a result of the release of a natriuretic peptide (similar to what occurs in the heart). Cerebral salt-wasting syndrome is characterized by the triad of hyponatremia, volume contraction, and high urine sodium concentrations (>50 mmol/L). Distinction between this syndrome and SIADH is important. SIADH, which is characterized by normovolemia or mild hypervolemia, is treated by volume restriction. Cerebral salt wasting is associated with a contracted intravascular volume. Fluid restriction and further volume contraction may be especially deleterious in a post-SAH patient and should be avoided. Although clinical distinction between these two causes of hyponatremia (SIADH and cerebral salt wasting) may be difficult, management of both is relatively simple: administration of isotonic fluids with intravascular normovolemia used as the end point.

Vasospasm.

When neurologic deterioration occurs subsequent to the patient's initial period of stabilization, vasospasm is frequently the cause. Drowsiness is a common initial clinical sign. In patients in whom there is clinical suspicion or angiographic demonstration of vasospasm, surgery is commonly deferred. If it is to proceed, CPP should be maintained intraoperatively in a high-normal range. This recommendation is contrary to the time-honored patterns of intraoperative management, which have characteristically emphasized hypotension. However, neurosurgeons recognize the potential for induced hypotension to cause or aggravate cerebral ischemia in patients with vasospasm. This concern extends even to a WFNS grade I patient who may have regions of cerebral ischemia that are subclinical when the patient is normotensive. Vasospasm is thought to be caused by the breakdown products of hemoglobin from blood that has accumulates around vessels of the circle of Willis after SAH. A specific mechanism/mediator has not been identified, but the focus of the most current research is on endothelin as a potential culprit.

In the ICU, the regimens used to treat vasospasm generally involve some combination of hypervolemia, hemodilution, and hypertension. The science behind hypervolemic-hypertensive therapy is "soft," and the efficacy of "triple H" therapy has not been proved by prospective study. The relative importance of the rheologic and pressure effects is undefined, although there is evidence for the relevance of blood pressure elevation in isolation. Phenylephrine and dopamine are the most commonly used pressors. The end point
for pressor administration varies. Most commonly, the objective is an increase in MAP of approximately 20 to 30 mm Hg above “baseline” systolic pressure. However, it has been reported that augmentation of cardiac output with dobutamine, without a simultaneous increase in MAP, augments CBF in vasospastic territories. Some believe that the hematocrit should be reduced to the low 30s. Commonly, a reduction in hematocrit occurs secondarily as a result of attempts to produce hypervolemia (usually with colloid solutions) as a part of the effort to raise blood pressure.

**Calcium Channel Blockers.**

Calcium channel blockers are now an established part of the management of SAH. Administration of nimodipine has been shown to decrease the incidence of morbidity from cerebral ischemia after SAH. However, these studies have failed to demonstrate any reduction in the incidence of vasospasm as detected by angiography, which suggests that the beneficial effect of these agents may be the result of effects on neurons rather than vascular smooth muscle. Patients coming to the operating room after SAH should be receiving nimodipine. The available information suggests that the use of nimodipine causes very little hemodynamic disturbance. Nimodipine must be administered orally, and nicardipine has been evaluated as an intravenous alternative. The multicenter nicardipine trial revealed a reduced incidence of symptomatic vasospasm but no improvement in outcome. As a consequence, nimodipine is used more commonly.

**Antifibrinolytics.**

Antifibrinolytics were administered in the past in an attempt to reduce the incidence of rebleeding. Even though they accomplish the latter, overall morbidity is not improved as a result of an increase in the incidence of ischemic symptoms and hydrocephalus. However, many of the observations that have discouraged the use of antifibrinolytics were made in the context of prolonged administration before delayed surgery. Recent results of brief administration of tranexamic acid before early surgery suggested a decreased rate of rebleeding without any apparent increase in ischemic complications, and it is possible that the role of antifibrinolytics will be reconsidered.

**Electrocardiographic Abnormalities.**

ECG abnormalities can occur in patients who have sustained SAH. In addition to the classic "canyon T waves" (Fig. 53-14), nonspecific T-wave changes, QT prolongation, ST-segment depression, and U waves have been described. It seems likely that the episode of extreme hypertension and autonomic discharge with high catecholamine levels that develops in association with the initial SAH event can result in reversible, "stunning"-like myocardial injury, presumably because of the extreme myocardial wall tension that occurs. Modest elevation in cardiac troponin occurs commonly. The relationship between ECG changes and myocardial dysfunction is not a consistent one. Myocardial dysfunction, as seen on echocardiography, correlates better with the severity of the neurologic condition than with the ECG, which is not an accurate predictor of myocardial dysfunction. The cardiac dysfunction associated with SAH does not appear to contribute to morbidity or mortality, and Brouwers and coauthors concluded that "in patients with aneurysmal SAH, ECG abnormalities do not herald impending cardiac disease." Accordingly, when ECG patterns other than those that are typical of myocardial ischemia are observed, no specific interventions or modifications in the approach to patient management are warranted. Although the nonspecific ECG changes do not appear to have important implications with respect to myocardial function, they may indicate a risk for dysrhythmias. In particular, an
increased QT interval (>550 msec) occurs quite frequently after SAH, especially in patients with more severe SAH, and it has been associated with an increased incidence of malignant ventricular rhythms, including torsades de pointes.

Figure 53-14. Electrocardiographic abnormalities associated with subarachnoid hemorrhage (SAH). The "canyon" T waves that may be seen after SAH are evident.

ANESTHETIC TECHNIQUE

Important considerations include the following:

1. Absolute avoidance of acute hypertension with its attendant risk of rerupture
2. Provision of intraoperative brain relaxation to facilitate surgical access to the aneurysm
3. Maintenance of high-normal mean arterial (perfusion) pressure to prevent critical reduction of CBF in recently insulted and now marginally perfused areas of the brain
4. Preparedness to perform precise manipulations of MAP as the surgeon attempts to clip the aneurysm or control bleeding from a ruptured aneurysm (or both)

Monitoring

An arterial line is invariably appropriate. A CVP line may be relevant if institutional practices involve large doses of mannitol to promote brain relaxation, and it may be used in older patients to guide volume replacement in the event of bleeding.

Selection of Anesthetic

Any technique that permits proper control of MAP is acceptable. However, in the face of increased ICP or a tight surgical field, an inhaled anesthetic technique may be less suitable. Prevention of paroxysmal hypertension is the only absolute requirement in patients undergoing aneurysm clipping. Rebleeding kills,177 and the poorly organized clot over the aneurysms of patients undergoing early post-SAH clipping makes them particularly prone to rebleeding. Rebleeding at induction is a frequently fatal event. The escaping arterial blood is more likely to penetrate brain substance because it cannot dissect through the CSF space (filled with clot), and the increase in ICP is extreme because of poor compliance of the intracranial space (swollen brain, hydrocephalus).

Induced Hypotension

The routine use of induced hypotension is diminishing (see the earlier section "Management of Blood Pressure"). Nevertheless, the anesthesiologist should be prepared to lower blood pressure immediately and precisely if called on to do so. Preparation must occur before the episode of bleeding. We prepare a sodium nitroprusside infusion before induction. It is placed in line at a Y-injection port at the hub of the CVP or intravenous catheter. The carrier drip should flow steadily so that any change in the nitroprusside infusion rate will be reflected as rapidly as possible in the central compartment. There are theoretical pros and cons for various hypotensive drugs. Data indicate that regimens entailing the use of a drug that is also a cerebral vasodilator (isoflurane, nitroprusside) are preferable in terms of brain oxygen delivery to approaches that do not involve cerebral vasodilation (trimethaphan, controlled hypovolemia). Deep isoflurane and nitroprusside are the most commonly used regimens, although other drugs may be suitable. The choice should ultimately be made on the basis of which regimen, in the hands of the individual practitioner, results in the most precise control of MAP. On occasion, the anesthesiologist will be asked to control MAP in the range of 30 to 50 mm Hg in the face of the active arterial bleeding. This task can be extremely difficult in a patient who is hypovolemic at the beginning of the bleeding episode. Accordingly, it is our practice to maintain normovolemia.

Induced Hypertension

Hypertension may be requested during periods of temporary arterial occlusion (see the later section "Trapping") to augment collateral CBF.152 In addition, after clipping of the aneurysm, some surgeons will puncture the dome of the aneurysm to confirm adequate clip placement and may request transient elevation of systolic pressure to 150 mm Hg. Phenylephrine is suitable in either instance.
Hypocapnia

Hypocapnia has traditionally been used as an adjunct to brain relaxation. This practice has also been questioned on the basis of concern that it will aggravate ischemia (see the earlier section "Management of PaCO₂"). The ICP/brain relaxation circumstances should probably dictate its use or avoidance.

Lumbar CSF Drainage

Some surgeons use elective drainage to facilitate exposure. It is extremely effective, effective to the point that the energetic application of other brain volume-reducing techniques is almost unnecessary. It is appropriate, when placing a lumbar CSF drain, to avoid excessive loss of CSF. A sudden reduction in the transmural pressure gradient across the dome of the aneurysm (by a sudden reduction in ICP after substantial CSF drainage) should be avoided lest such decompression encourage rebleeding.²²¹ After having verified the patency of the lumbar drainage system, common practice is to leave it closed until such time as the surgeon is opening the dura. The drain may then be opened and allowed to drain freely with the bag at floor level. Drainage should be discontinued promptly after final withdrawal of the retractors to allow CSF to reaccumulate and to thereby reduce the size of the potential pneumocephalus. The drain is usually removed immediately postoperatively.

Mannitol

Some surgeons make relatively aggressive use of mannitol (e.g., 2 g/kg). In part, it is used to shrink the brain and thereby facilitate exposure and reduce retractor pressure. There is evidence that it may have additional benefits. Specifically, data derived from both animals and humans indicate that mannitol may have a CBF-enhancing effect in regions of moderate CBF reduction.²²²,²²³,²²⁴,²²⁵ The mechanism is not defined. However, a reduction in interstitial tissue pressure around capillaries or an alteration in blood rheology (or both) has been proposed to contribute. Typically, mannitol is administered in a dose of 1 mg/kg just before dural opening. Surgeons who believe in the flow-enhancing effect may request a second 1 mg/kg approximately 15 minutes before an anticipated temporary occlusion.

Trapping

It is occasionally necessary for the surgeon to "trap" the aneurysm (i.e., to temporarily occlude the vessel on either side of the aneurysm) to complete dissection of the neck and apply the clip. This technique is more common with larger aneurysms. With giant aneurysms in the vicinity of the carotid siphon, the inferior occlusion may be performed at the level of the internal carotid artery through a separate incision in the neck. A clinical survey by Samson and colleagues of the neurologic outcome after temporary occlusion in normothermic, normotensive adults revealed that occlusion times less than 14 minutes were invariably tolerated. The likelihood of an ischemic injury increased with longer occlusion and reached 100
Brain Protection

Maintenance of MAP to ensure collateral flow and perfusion under retractors, efficient brain relaxation to facilitate surgical access and reduce retractor pressure, limitation of the duration of episodes of temporary occlusion, and perhaps mild hypothermia (see the section "Hypothermia") are the important brain protection techniques. Specific anesthetics have also been used to protect the brain (see discussion in Chapter 21). Etomidate and propofol are au courant. However, there have been no convincing laboratory demonstrations that propofol provides any greater tolerance to a standardized ischemic insult than anesthesia with a volatile anesthetic does. Attempts to demonstrate protection by etomidate in an animal model of focal ischemia actually demonstrated an adverse effect of etomidate.\[227\] Furthermore, a clinical investigation during aneurysm clipping revealed decreases in brain tissue PO2 in association with the administration of etomidate, which contrasted with the increases in brain PO2 that occurred with the introduction of desflurane. During subsequent temporary vessel occlusion, tissue pH decreased alarmingly in patients receiving etomidate and was unchanged with desflurane.\[228\] Although the available data collectively do not support an assertion that etomidate is harmful in the setting of aneurysm surgery, we think that they should dissuade any anesthesiologist from advocating the administration of etomidate as a protective substance in the context of aneurysm surgery.\[229\][230]

With respect to the volatile anesthetics, attempts in the laboratory to confirm the once-proclaimed protective efficacy of isoflurane have not demonstrated any differences among the various volatile anesthetics in terms of their influence on outcome after focal or global ischemia.\[227\][231][232][233] Nor has there been any demonstration of greater protective efficacy with concentrations of volatile anesthetics sufficient to cause EEG suppression as opposed to more modest levels (e.g., 1.0 MAC).\[233][234] Nonetheless, these animal investigations do suggest that a standardized experimental ischemic insult is better tolerated, relative to the awake state, by animals receiving a volatile anesthetic.\[232][233][235] In addition, data derived in animals also suggest that there may be a relative protective advantage to an anesthetic technique that includes a volatile anesthetic versus a strict nitrous oxide-narcotic technique.\[233][235] However, the magnitude of the differences among anesthetics and the absence of proof of relevance in patients preclude advocacy of a particular anesthetic regimen in a standard text. The important anesthetic objectives are precise hemodynamic control and timely wake-up, and these two constraints should dictate the choice of the anesthetic regimen for most aneurysm procedures. Among anesthetics, it is only the barbiturates for which additional protective efficacy has been demonstrated convincingly (see Chapter 21). Because of their potentially adverse effects on hemodynamics and wake-up, they are not ideal for routine administration. Barbiturates should probably be reserved for situations in which prolonged vessel occlusion is unavoidable, and in that circumstance it would be ideal if the ischemic hazard were first confirmed by observation of the EEG response to temporary occlusion.\[236\]

Hypothermia

As noted in the earlier section "Hypothermia," the efficacy of mild hypothermia in the context of aneurysm surgery has not been confirmed in humans, although recruitment of patients to a multicenter, prospective comparison of normothermia and hypothermia has been completed and the results are awaited. Nonetheless, neurosurgical teams in many institutions are lowering body temperature to the range of 32°C to 34°C for procedures in which vessel occlusion may occur. The institutions that use lower temperatures are those in which the team is willing to accept a delay in emergence from anesthesia. That delay results from the necessity to maintain anesthesia long enough to achieve sufficient rewarming to avoid the extreme hypertension that can occur when a patient is awakened at low body temperature. It has been confirmed by Crowder and associates that the reduction in systemic temperature does result in a concomitant decrease in brain temperature.\[159\] In that
investigation, during moderate hypothermia with the dura open, cortex temperature was approximately 0.5°C less than tympanic membrane temperature and about 1°C lower than esophageal, pulmonary artery, or jugular bulb temperature. [155]

Neurophysiologic Monitoring

Both evoked responses and the EEG have been used[236][237] though neither widely. EEG monitoring can be used as a guide to management during the period of flow interruption or to guide the administration of CMR-reducing anesthetics given before occlusion.[236] At some institutions, the surgeon places an electrode strip over the region of cortex at risk during the intended occlusion. However, the more commonly used skin surface frontal-mastoid derivation is probably sufficient to reveal a major ischemic event. The EEG can be either the conventional "raw" polygraph or a processed derivative. In most circumstances, if occlusion is deemed necessary, a temporary occlusion is performed and the EEG observed. If the EEG shows significant slowing, the usual practice is to reposition the clip or elevate MAP by some combination of phenylephrine and lightening of anesthesia (or both) to find a way to carry out temporary clipping without a major EEG disturbance. If this goal cannot be accomplished and a sustained period of occlusion seems likely, it may be appropriate to administer barbiturates (see earlier) to produce burst suppression.

Intraoperative Angiography

Intraoperative angiography is becoming an increasingly common component of the management of intracranial aneurysms. It does not have substantial implications for the anesthesiologist. However, apparatus around the patient's head must be organized to allow C-arm access without snagging of airway and monitoring equipment. In addition, the radiologist must have access to the groin vessels. The vascular access sheath, for a patient who will ultimately be in the lateral position, is easiest to use when the nondependent femoral artery is chosen. The rate of administration of heparin "flush" through the vascular access sheath should be monitored.

Special Considerations for Specific Aneurysms

The most common procedures are performed for aneurysms arising in or close to the circle of Willis. The vessel of origin may be the anterior communicating artery, the middle cerebral artery, the anterior cerebral artery, the ophthalmic artery, the tip of the basilar artery, the posterior communicating artery, and less frequently, the posterior cerebral artery. These procedures will all be relatively similar for the anesthesiologist and will typically require a supine position with the head turned slightly away from the operative side.

Ophthalmic Artery Aneurysms.

Access to the origin of the ophthalmic artery, which is the first intradural branch of the carotid artery, is made difficult by the anterior clinoid process and the optic nerve. As a result, these aneurysms frequently require temporary vascular occlusion. The surgeon will commonly first expose the carotid artery in the neck. When the stage of seeking definitive access to the neck of the aneurysm is reached, the surgeon will occlude the carotid artery in the neck first and then the intracranial portion of the carotid artery immediately proximal to the origin of the posterior communicating artery. The surgeon may also cannulate the isolated segment and place it to suction, which will entail modest ongoing blood loss as a consequence of retrograde flow from meningeal and hypophyseal vessels.
Vertebrobasilar Aneurysms.

These procedures are typically performed in the lateral position. The exposure may involve a combined middle and posterior fossa approach, with some attendant, though minor risk of VAE. Cortical or skin surface EEG monitoring is of less relevance with vertebral-basilar aneurysms. Auditory or somatosensory evoked responses, or both, have been used. As in any other procedure involving the potential for mechanical or vascular injury to the brainstem, cardiovascular responses should be monitored, and sudden changes in response to surgical manipulation should prompt immediate notification of the surgeon. Spontaneous ventilation has also been shown to have an important role to play during surgical manipulation of the vertebral arteries, the vertebrobasilar junction, and the middle portion of the basilar artery. Apnea, gasping, or other sudden changes in ventilatory pattern during manipulation of the vasculature provide important, though somewhat nonspecific warning of compromise of the vascular supply of the brainstem. Management of these aneurysms will occasionally require cardiopulmonary bypass and deep hypothermic circulatory arrest.

Vein Of Galen Aneurysms.

These "aneurysms" are more appropriately managed according to considerations relevant to AVMs. Such considerations include anticipation of the possibility of the cerebral dysautoregulation phenomenon and are considered later.

ARTERIOVENOUS MALFORMATIONS

For most intracranial AVMs, the general considerations are similar to those appropriate to aneurysm surgery: avoidance of acute hypertension and the capability of accurately manipulating blood pressure in the event of bleeding. A problem that is specific to AVMs is the phenomenon known as "perfusion pressure breakthrough," or cerebral dysautoregulation. It is characterized by an often sudden engorgement and swelling of the brain, sometimes with a relentless cauliflower-like protrusion from the cranium. It tends to occur in the advanced stages of lengthy procedures on large AVMs. The phenomenon is not entirely understood. However, it has been attributed to acute obliteration of the high-volume, low-resistance pathway (the AVM) that for many years has been stealing blood from the surrounding tissue. The result is abrupt diversion of the AVM's flow to the vasculature in adjacent and previously marginally perfused brain tissue. It has been theorized that these tissues have long been maximally vasodilated without the need to ever vasoconstrict and have become incapable of doing so. However, not all of the available information is entirely consistent with this mechanism, and some evidence indicates that the neovascularization occurring in response to chronic ischemia results in abnormal vessels that do not autoregulate even though native ones can do so.

Anesthetic Technique.

The management constraints are essentially the same as those relevant to aneurysm surgery. Institutional practices will vary. We do not use induced hypotension unless required to by bleeding. We reason that the effects of devascularizing the AVM on the surrounding brain will be best appreciated if the devascularization occurs at normal pressure. If refractory brain swelling occurs, we can then also lower MAP as part of attempts to control the swelling. The rationale is that blood flow through the involved area is pressure passive and will decrease as MAP is reduced. With severe episodes of swelling, we have used (in addition to hypotension, which we use cautiously because of the associated risk of ischemia) hypocapnia, hypothermia, and barbiturates. The latter three techniques probably serve to reduce the bulk of only normal brain tissue—hypocapnia through a
direct effect on CBF and barbiturates and hypothermia through the coupled effects of reduction of CMR on CBF. Induced hypothermia is also an adjunct to minimizing barbiturate doses. In all of neurosurgery we seek to prevent postoperative hypertension. However, it is in AVM surgery that this objective should be accomplished with greatest care because of the concern that edema or hemorrhage will develop in the "dysautoregulating" brain adjacent to the resected AVM if hypertension occurs.

HEAD INJURY

INTUBATING A HEAD-INJURED PATIENT

The anesthesiologist's first "interface" with a patient who has sustained a traumatic brain injury (TBI) may come as a result of a request for assistance with airway management. It has been determined empirically that patients with Glasgow Coma Scale (GCS) scores of 7 to 8 (Table 53-10) or less will eventually require intubation and controlled ventilation for ICP or airway control (or for both). Accordingly, these patients are almost invariably intubated promptly. Patients with less severe head injuries may also require intubation because of trauma-related cardiopulmonary dysfunction or, when uncooperative, to facilitate diagnostic procedures. The anesthesiologist, in choosing the intubation technique, may encounter a number of conflicting constraints (Table 53-11), including (1) elevated ICP, (2) a full stomach, (3) an uncertain cervical spine, (4) an uncertain airway (presence of blood, possible laryngeal-tracheal injury, possible skull base fracture), (5) an uncertain volume status, (6) an uncooperative/combative patient, and (7) hypoxemia. There is no "correct" way, and the "best" approach will be determined by the relative weight of these various factors along with the degree of urgency. However, the anesthesiologist must not get distracted by placing an excessive initial emphasis on ICP. Keep sight of the ABCs of resuscitation: securing the airway, guaranteeing gas exchange, and stabilizing the circulation are higher initial priorities than control of ICP is. Do not risk losing the airway or causing severe hypotension for the sake of preventing coughing on the tube or brief hypertension with intubation.

Table 53-10 -- Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Eyes open</th>
<th>Never</th>
<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>To speech</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Spontaneously</td>
<td>4</td>
</tr>
<tr>
<td>Best verbal responses</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Garbled/incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Confused but converses</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Best motor responses</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Extension (dysreflexic rigidity)</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Abnormal flexion (decorticate rigidity)</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Withdrawal</td>
<td>4</td>
</tr>
<tr>
<td>Localizes pain</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>------------------</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>Obey commands</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>3–15</td>
<td></td>
</tr>
</tbody>
</table>

**Table 53-11 -- Factors that may be relevant during intubation of a head-injured patient**

- Full stomach
- Uncertain cervical spine
- Uncertain airway
- Blood
- Airway injury (larynx, cricoarytenoid cartilage)
- Skull base fracture
- Uncertain volume status
- Uncooperative/combative
- Hypoxemia
- Increased intracranial pressure

**THE CERVICAL SPINE**

The possibility of causing or aggravating an injury to the cervical spine is a relevant concern. Approximately 2% of patients with a closed-head injury who survive to reach a hospital will have a fracture of the cervical spine. Somewhat surprisingly, the incidence of cervical spine injury is similar (1.8% to 6.0%) for all blunt trauma victims with or without an associated TBI. This incidence suggests that a "hypnotic-relaxant-direct laryngoscopy" approach for all patients with a closed-head injury might convey a measurable risk of injuring the cervical cord. Nonetheless, although the literature contains contradictions, several published series have concluded that rapidsequence induction does not convey a significant risk of neurologic injury. However, it is possible that the incidence of intubation-related neurologic injury is underreported. An informal survey by Criswell and colleagues indicated that there have been more such events than one can infer from the published literature. Nonetheless, the literature argues that we will "get away with it" most of the time, and there are certainly units in which most patients requiring airway control are intubated with the use of a hypnotic-relaxant-direct laryngoscopy sequence. However, it is our (probably minority) opinion that the possibility of devastating spinal cord injury exists, probably most so with injuries in the atlanto-occipital region, which are also difficult to identify radiologically, and that the anesthesiologist should seek to identify circumstances in which time latitudes allow more detailed examination or radiologic evaluation. When there is any uncertainty regarding the airway or the cervical spine, direct laryngoscopy (with vigorous atlanto-occipital extension) should probably be avoided unless the exigencies of airway control demand it. The nasal route can be used in spite of concern about entering the cranial vault through a fracture in the skull base. However, use discretion (e.g., in the presence of an obvious facial smash it should be avoided) and be sensitive to unusual resistance when passing the endotracheal tube.
When a hypnotic-relaxant sequence is used, the standard approach includes the use of cricoid pressure and in-line axial stabilization. In-line traction was once favored but has been supplanted by stabilization because of the perceived risk of overdistraction and cord injury in the event of gross instability. The largest of the clinical series in which it was concluded that oral intubation with anesthesia and relaxation is reasonable\textsuperscript{[251]} used in-line stabilization with the patient's occiput held firmly on the backboard, thus limiting the amount of "sniff" that was feasible (Fig. 53-15). There is no question that in-line stabilization, properly performed, will make laryngoscopy somewhat more difficult. However, it serves to decrease the amount of atlanto-occipital extension necessary to achieve visualization of the glottis.\textsuperscript{[258]} probably because performing laryngoscopy against the assistant's counter-pressure results in greater compression of the soft tissue structures of the tongue and floor of mouth. Some recommend leaving the back half of the Philadelphia collar in place during laryngoscopy (see Fig. 53-15) because it functions as a strut between the shoulder and the occiput that serves to further limit atlanto-occipital extension.

\textbf{Figure 53-15} Intubating an acute trauma patient with an uncertain cervical spine. A hypnotic and a relaxant have been administered. One assistant maintains in-line axial stabilization with the occiput held firmly to the backboard; a second applies cricoid pressure. The posterior portion of the cervical collar remains in place to "discourage" atlanto-axial extension.


In the resuscitation situation, before initiating a hypnotic-relaxant sequence, the anesthesiologist should confirm the availability of both cricothyrotomy equipment and someone to make immediate, skilled use of it if necessary. A recently injured brain is very intolerant of hypoxia and hypotension.\textsuperscript{[259]} It is inevitable that there will be the occasional failed intubation. In the extensive experience of the Cowley Shock-Trauma Center in Baltimore, the cricothyrotomy rate is 0.3\% (personal communication, Colin Mackenzie, M.D.). As is the case in many other situations, the laryngeal mask airway (LMA) may be a very useful device for temporizing in the face of a failed intubation and may also provide access for intubation as an alternative to cricothyrotomy.

As noted in Chapter 21, although succinylcholine can cause increases in ICP, these increments are small and probably do not, in fact, occur at all in patients with serious cerebral injuries.\textsuperscript{[6]} Accordingly, succinylcholine should not be viewed as contraindicated in a TBI victim. If and when there is an urgent need to secure the airway (to control carbon dioxide tension and guarantee oxygenation) and if succinylcholine is in other respects the appropriate drug to achieve that end, it should be used.

In two clinical surveys, alert, nonintoxicated patients with a cervical spine fracture invariably had pain, tenderness, or neurologic signs.\textsuperscript{[248],[253]} Accordingly, in spite of the frequency with which you will meet patients still wearing their Philadelphia collars because the neck has not yet been "cleared," no special precautions appear to be warranted in an asymptomatic, alert patient. Note also that if the clinical situation or examination is suggestive of a cervical spine injury, a normal lateral radiograph (the anteroposterior and through-the-mouth
odontoid views are frequently not taken during the initial evaluation) cannot provide complete reassurance. The lateral view has been reported to miss between 15%\textsuperscript{[26]} and 26%\textsuperscript{[28]} of fractures.

**ANESTHETIC TECHNIQUE**

**Choice Of Anesthetics.**

Craniotomy will most commonly be performed for the evacuation of subdural, epidural, or intracerebral hematomas. The anesthetic approach is similar for all three. The guiding principles have been discussed in the section "Control of Intracranial Pressure/Brain Relaxation." In general, anesthetics that are known to be cerebral vasoconstrictors will be preferable to those that have the potential to dilate the cerebral circulation. All of the intravenous anesthetics, except perhaps ketamine, cause some cerebral vasoconstriction and are reasonable choices, provided that they are consistent with hemodynamic stability. All of the inhaled anesthetics (N\textsubscript{2}O and all of the vapors) have some cerebral vasodilatory effect. Although their administration will frequently be consistent with acceptable ICP levels or appropriate conditions in the surgical field (or both), when ICP is out of control (or unknown) or the surgical field is "tight," eliminating the inhaled anesthetics in favor of fixed anesthetics is appropriate. For patients who are likely to remain traumatically intubated postoperatively, an anesthetic based primarily on a narcotic (e.g., fentanyl) and a muscle relaxant usually serves well. Any muscle relaxant is acceptable with the proviso that those that can release histamine should be titrated in careful increments. When immediate extubation is a possibility, such as in a patient with an acute epidural hematoma who had a lucid interval before witnessed deterioration, the technique should be modified after opening of the cranium. The introduction of inhaled anesthetics or the use of shorter-acting intravenous drugs, or both, can be undertaken as guided by observation of the surgical field. If administration of N\textsubscript{2}O is contemplated at any time, remember the possibility, in the setting of missile injury or compound skull fracture, of intracranial air.

**Monitoring.**

The anesthesiologist should appreciate that the priority is to open the cranium as rapidly as possible. After achieving intravenous access, the craniotomy should never be delayed significantly by line placement. An arterial line, often placed after induction in urgent situations, is appropriate for essentially all acute trauma craniotomies. The decision to achieve central venous access can be based on the patient’s hemodynamic status. Infrequently, management of a depressed skull fracture over the sagittal sinus will justify precordial Doppler examination and, subject to the surgeon’s opinion of the risk for VAE, a right heart catheter.

**Blood Pressure Management.**

The concept that the injured brain is extremely vulnerable to what would otherwise be a minor insult, for example, modest hypotension or moderate hypoxia, has been well confirmed in the laboratory.\textsuperscript{[29][30][31]} Although as yet no completely conclusive data have been derived from humans, several clinical surveys are strongly supportive of the adverse effect of minor degrees of hypotension in the post-TBI period.\textsuperscript{[29][30][31]} The explanation for this vulnerability to hypotension probably resides in part in the observation that some patients in the postinjury period have regions of brain with precariously low CBF\textsuperscript{[29][30][31]} in which autoregulation may also be defective.\textsuperscript{[29][31]} In addition, ample evidence indicates that the low postinsult CBF values correlate with a poor eventual outcome\textsuperscript{[29][30][31]} and that a large percentage of patients who die after TBI have pathologic changes consistent with ischemia.\textsuperscript{[15]} These observations have
resulted in a much greater emphasis by many neurosurgeons and neurointensivists on aggressive support of blood pressure in TBI patients.

What constitutes an appropriate blood pressure? Systematic studies, in particular, those conducted at the University of Edinburgh, have revealed evidence that indices of the adequacy of cerebral perfusion derived from SjvO2 and transcranial Doppler data begin to deteriorate below a mean CPP of 70 mm Hg. [268][271][272] (Recall that CPP = MAP - ICP.) As a result, many neurosurgical groups adopted 70 mm Hg as the target CPP. [268][269][272] An expert panel [273] found the data insufficient to justify establishing 70 mm Hg as a "standard" CPP target, but instead identified it as a reasonable management "option," and other groups and authorities have adopted a CPP of 60 mm Hg as the management target. [274][275][276]

Added to this discussion are two alternative opinions regarding blood pressure management (Fig. 53-16). The first, promoted by the neurosurgery group at the University of Alabama, Birmingham, is that induced hypertension can be used as an adjunct to control of ICP. [268][279][280] This idea is based on the belief that autoregulation is at least partially preserved after head injury and that increased CPP will result in autoregulation-mediated vasoconstriction with a concomitant reduction in CBV and therefore ICP.

![Figure 53-16 Relationship of cerebral blood flow (CBF) to blood pressure after head injury.](image)

That CPP target has increased, in the most recent iteration to 60 to 70 mm Hg with allowance for occasional reduction to 50. [279] An approach emanating from the University of Alabama, identified as the "Birmingham" concept, entails pharmacologically induced hypertension. This approach is based on the belief that autoregulation is largely intact and that hypertension will result in cerebral vasoconstriction with concomitantly reduced CBV and ICP. [279][280]

They make aggressive use of volume expansion and vaspressors (phenylephrine, dopamine) to maintain high CPP. They report a very satisfactory local experience [280] with this approach, but it has not been applied widely, and others have reported that induced hypertension was either ineffective or deleterious as a means of reducing increased ICP. [281][282] The second alternative is the so-called "Lund concept," which is based on the premise that high blood volume and hydrostatically driven edema accumulation make a substantial contribution to the increased ICP associated with head injury. [277][278][283] As originally described, the Lund approach entailed dehydration, hyperosmolality, and the administration of metoprolol, clonidine, and dicyclopropamine to lower blood pressure to a CPP target of 50 to 55 mm Hg. At its inception, the approach was controversial because it ran counter to the perceptions of many of the importance of maintaining a CPP of 70 mm Hg [268][269][272] and because of the later demonstration that a negative fluid balance in patients with TBI is deleterious. [138] Over time (and in parallel with relaxation of the CPP targets of others from 70 toward 60 mm Hg [274][275][276]), the Lund proponents have modified their approach, and now "a CPP of 60–70 is considered optimal" and
normovolemia is the clinical objective. The essence of the once-controversial Lund approach is no longer the aggressive reduction in CPP that was the originally proclaimed cornerstone, although sedatives, clonidine, and metoprolol are administered to prevent hypertension. The Lund concept's distinguishing features are now (1) the routine infusion of low-dose thiopental (to reduce the "stress response" and to decrease CMR and thereby CBV), (2) maintenance of colloid osmotic pressure at unspecified levels by the generous administration of albumin, and (3) cautious administration of dihydroergotamine, also to reduce CBV (by a putative selective cerebral venoconstrictive effect). Despite the fact that each of the components has a theoretical basis, the existing literature provides no compelling support. Although proponents of the Lund approach assert improvements in outcome, these reports invariably entail either no control group or comparisons with nonconcurrent controls. The approach has not been adopted in North America.

What is an anesthesiologist managing a TBI patient to do in the face of these various approaches? Fortunately, perfusion pressure support of varying degrees is a theme that is common to all of them. The characteristic behavior of CBF after head injury is an initially low CBF followed by a gradual increase over a period of 48 to 72 hours to normal or sometimes even slightly hyperemic levels. Accordingly, aggressive support of CPP in the 60- to 70-mm Hg range during that period will be the most reasonable general approach. Note that in patients with subarachnoid blood, a second period of low CBF may occur from days 4 to 10 after injury, apparently on a vasospasm-related basis. It should also be understood that there are exceptions to the foregoing generalizations about CBF after TBI. Although an initially low postinjury CBF is probably the most common clinical occurrence, hyperemia does occur. It tends to develop in patients with mass lesions rather than contusions, although even these patients have an immediate postinjury period of low CBF, with delayed hyperemia peaking at 24 hours or later. Hyperemia may also be common in children. Nonetheless, in the absence of measures of CBF or brain tissue well-being (both of which are uncommonly available), careful maintenance of a CPP of 60 to 70 mm Hg in the first 72 hours after TBI will be appropriate and is common practice in a head-injured adult. A CPP target of 45 mm Hg has been recommended for children. With these firm recommendations offered, it must be acknowledged that convictions will vary and anesthesiologists should come to an understanding with the local traumatologists and neurosurgeons regarding blood pressure targets.

In the ideal situation, management of CPP is "targeted" to the pathophysiology that prevails in the individual patient. However, techniques to discriminate the various flow states (CBF measurement, transcranial Doppler, SjVo2, brain tissue Po2, see later) are not universally available or applied (nor yet proven to lead to improvement in outcome).

Hyperventilation.

The use of hypocapnia has been reviewed in detail in the section "Management of PaCO2." Hyperventilation has long been a standard component of the management of TBI patients perceived to be at risk for increased ICP. However, evidence is increasing that hyperventilation is potentially deleterious and should not be overused. That evidence suggests that hyperventilation and the concomitant vasoconstriction can result in ischemia, especially when baseline CBF is low, as is likely to be the case in the first 48 to 72 hours after head injury. The expert panel mentioned earlier, convened by the Brain Trauma Foundation, specified that "chronic prophylactic hyperventilation should be avoided during the first 5 days after severe TBI and particularly during the first 24 h." The available information argues that hyperventilation should be used selectively rather than routinely in the management of TBI patients. Maintaining ICP at less than 20 mm Hg,
preventing or reversing herniation, minimizing retractor pressure, and facilitating surgical access are still important objectives in the management of TBI patients, and to the extent that hyperventilation contributes to these objectives it is still appropriate. Once again, the anesthesiologist should agree on management parameters with the surgical team at the outset of a procedure.

**Fluid Management.**

Fluid management of a head-injured patient was addressed in the section "Intravenous Fluid Management." The important principles are that fluids should invariably be chosen to prevent a reduction in serum osmolarity and should probably be chosen to prevent a profound reduction in colloid oncotic pressure; specifically, in the circumstances of large-volume resuscitation (arbitrarily, greater than half a circulating volume), a mix of colloids and crystalloids is probably appropriate. The clinical objective should be maintenance of intravascular normovolemia, in part as an adjunct to MAP and CPP support. A chronic negative fluid balance, as can occur with the combination of modest fluid restriction and liberal use of osmotic diuretics, has been shown to be deleterious and should be avoided.[19] Remember also that a severely injured brain can liberate sufficient thromboplastin into the circulation to result in consumptive coagulopathy. Appropriate laboratory tests and replacement should be performed.[20] The clinician may also find that determination of serum osmolality early in the course of anesthetic management is useful in appreciating the cumulative effects of previous administration of mannitol. The use of hypertonic solutions and the relevant attributes of colloid solutions were also discussed in the section "Intravenous Fluid Management."

**JUGULAR VENOUS OXYGEN SATURATION**

Numerous centers have studied or made use of SjV02 monitoring as a guide to the clinical management of head-injured patients.[15][36][37][45][46][56][272][278][299] The underlying concept is that marginal or inadequate CBF will result in increasing oxygen extraction, a widening arteriovenous content difference, and decreasing jugular venous PVO2 or SjV02. There have been numerous reports of improvement in SjV02 as a consequence of reducing hyperventilation, increasing MAP, or inducing hypervolemia.[200] The availability of intravascular catheters that permit continuous monitoring of SjV02 has made the technique more practical.[301] SjV02 measurement makes an assessment of global oxygen extraction. Accordingly, it might be expected to have limited sensitivity in highly focal events, and instances in which focal inadequacy of perfusion was not reflected by a low SjV02 have been reported.[35][36]

Some technical limitations are inherent in SjV02 monitoring.[302] Catheter placement must be very precise to avoid contamination by noncerebral venous blood or attenuation of light return (with optical catheters) because of vessel wall abutment. Even in experienced hands, the false-positive rate can be significant.[37] An additional limitation inherent in unilateral placement of the catheter is the observation by Stocchetti and coworkers that there was an average side-to-side difference between simultaneous jugular bulb saturations of 5.3% ± 5% and that side-to-side differences in hemoglobin saturation of up to 15% were common.[303] These and other authors have questioned the reliability of data obtained from a unilateral SjV02 catheter.[304] The next clinical problem is the matter of what constitutes an abnormal value. Normal subjects at rest may have SjV02 values between 50% and 75%. A currently used definition of abnormal is less than 50% for 5 minutes.[165] However, there has been limited opportunity to correlate SjV02 threshold values with outcome.

In spite of the reported success with SjV02 monitoring,[44][306] we do not believe that the method is sufficiently well defined to justify advocating widespread intraoperative application. Further experience and
clinical investigation are required to define the correct role and mode of application of SjvO₂ monitoring. At present, it appears potentially useful as a trend monitor that may serve to identify the level of CPP below which cerebral perfusion begins to be compromised. However, the SjvO₂ level at which that compromise is critical has not been identified. The technique also has a potential use beyond identifying patients with low SjvO₂ and inadequate CBF. High SjvO₂ values may serve to identify a patient with elevated ICP in whom hyperemia is an important contributing factor and in whom aggressive attempts to decrease CBF (e.g., hyperventilation, barbiturates) may be beneficial.

BRAIN TISSUE PO₂ MONITORING

Small-diameter intraparenchymal electrodes are available that allow measurement of brain tissue PtO₂ (and sometimes pH and PtcO₂). They have been used to monitor cerebral well-being during the ICU management of both TBI and SAH patients and to assess the effect of operative interventions. A PtO₂ of 20 mm Hg is viewed as normal, and values less than 10 mm Hg are assumed to convey a risk of hypoxic injury. They suffer from the inverse of the problem that prevails with SjvO₂ monitoring in that they are very focal monitors that assess the oxygenation status of only small regions of brain surrounding the tip. If they are placed remote from focal injuries in a traumatized brain, they may not "see" adverse events in salvageable perilesional tissue. They may similarly fail to be a useful therapeutic guide if they are within irredeemably injured brain. Thus far, their use is neither standardized nor widespread.

HYPOTHERMIA

Mild induced hypothermia has already crept into the management of neurosurgical procedures in which there is a perceived risk of ischemic injury. To date, these procedures have encompassed principally aneurysm surgery, although proof of a favorable effect on outcome in this context has not yet been established (see the earlier section "Hypothermia"). However, its efficacy in reducing damage when induced after experimental head injury has also been demonstrated and its relevance to the management of a head injury patient has been the subject of small, prospective controlled trials in at least four centers. Because these single-center trials appeared to indicate good patient tolerance of sustained mild hypothermia (32°C to 34°C), as well as improvement in ICP, cerebral oxygen supply/demand, and outcome, a multicenter trial was performed. That trial revealed no overall benefit of hypothermia. However, post hoc analysis demonstrated that patients who were hypothermic (<35°C) on admission to the hospital benefited from hypothermia whereas those who were normothermic on admission did not. Patients older than 45 years derived no benefit at any admission temperature. As a result, hypothermia has no established role in management of head injury as of this writing. However, a follow-up trial is under way. Patients 16 to 45 years of age with GCS scores less than 8 who arrive at the hospital with a bladder temperature less than 35°C are to be randomized to cooling to 32.5°C to 33.5°C (within 4 hours of injury) for 48 hours or passive rewarming to normothermia. Outcome will be assessed at 6 months.

ICP MONITORING FOR NON-NEUROLOGIC SURGERY IN HEAD-INJURED PATIENTS

In ideal circumstances, neurosurgical consultation will be readily available, and appropriately, the anesthesiologist will rarely have to make this decision. It may, however, be necessary for the anesthesiologist to participate in this decision. Relevant variables include the following:
1. Level of consciousness. If loss of consciousness has occurred at any time or if the GCS score is less than 15, a CT scan should be obtained. If the CT scan reveals compressed basal cisterns (indicative of exhaustion of supratentorial compensatory latitudes), midline shift, or effaced ventricles and probably any intracranial lesion (contusion, small subdural), an ICP monitor should be placed. Excessive comfort should not be taken from a good GCS score. Patients with good GCS scores can "talk and deteriorate" or "talk and die" after a head injury associated with loss of consciousness. Delayed deterioration has been observed up to as much as 48 hours after the initial injury (average of 17 hours).[310] Patients with lesions, usually contusions, in the frontotemporal region and especially those with medial temporal lesions are most at risk for this phenomenon. Modest expansion of lesions in this location (i.e., close to the uncus and the incisura where herniation occurs) can result in herniation even at relatively low ICP levels (e.g., ≈20 mm Hg). At our institution, neurosurgeons would recommend avoiding an anesthetic in these patients and would certainly advise ICP monitoring if general anesthesia were unavoidable.

2. Time since injury. The longer patients have had to establish their clinical course, the less pressing is the need for ICP monitoring. However, delayed deterioration, as noted earlier, has been observed for up to 48 hours.[310] and a patient with a demonstrable CT lesion is a candidate for a monitor for at least this period.

3. Intended aortic occlusion (i.e., repair of ruptured aorta). Dramatic increases in ICP have been associated with aortic occlusion. These increases may in large part be a result of the abrupt rise in blood pressure or the agents used to control it (or both). In addition, the increased airway and venous pressure associated with the lateral position and one-lung ventilation plus occasional difficulties maintaining hypocapnia during one-lung ventilation should result in a low threshold for ICP monitoring in this situation. Note, however, that the intent to heparinize systemically essentially precludes placement of a monitor.

4. Nature and duration of the intended procedure. The risk of an untoward ICP event developing is inevitably greater for a 6-hour spine instrumentation in the prone position than for a 20-minute débridement and suturing of an arm laceration.

**POSTERIOR FOSSA PROCEDURES**

Most of the topics relevant to posterior fossa procedures (Table 53-12) have been discussed in the first half of the chapter and include the sitting position and its cardiovascular effects and complications (quadriplegia, macroglossia) (see the section "Positioning"), pneumocephalus (see "Pneumocephalus"), and VAE and PAE (see "Venous Air Embolism"). These sections should be read in conjunction with this segment. Use of the sitting position to facilitate surgery on the posterior fossa increases the likelihood of all of these phenomena, although they are relevant to nonsitting positions as well. This section will review the cardiovascular events associated with direct stimulation of the brainstem and their possible implications for postoperative management.
Table 53-12 -- Considerations relevant to posterior fossa procedures and the location of the related discussion in this chapter

<table>
<thead>
<tr>
<th>Consideration</th>
<th>Section</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemodynamic effects of the sitting position</td>
<td>Positioning</td>
</tr>
<tr>
<td>Venous air embolism</td>
<td>Venous Air Embolism</td>
</tr>
<tr>
<td>Paradoxical air embolism</td>
<td>Venous Air Embolism</td>
</tr>
<tr>
<td>Hemodynamic effects of brainstem or cranial nerve manipulation</td>
<td>Posterior Fossa Procedures</td>
</tr>
<tr>
<td>Quadriplegia</td>
<td>Positioning</td>
</tr>
<tr>
<td>Macroglossia</td>
<td>Positioning</td>
</tr>
<tr>
<td>Pneumocephalus</td>
<td>Pneumocephalus</td>
</tr>
</tbody>
</table>

**BRAINSTEM STIMULATION**

Irritation of the lower portion of the pons, the upper part of the medulla, and the extra-axial portion of the fifth cranial nerve can result in a number of cardiovascular perturbations. The former two areas are most often stimulated during procedures on the floor of the fourth ventricle and the last area during surgery at or near the cerebellopontine angle, for example, acoustic neuromas and microvascular decompression of the fifth (tic douloureux), seventh (hemifacial spasm), or ninth (glossopharyngeal neuralgia) nerves. The cardiovascular responses may include bradycardia and hypotension, tachycardia and hypertension, bradycardia and hypertension, or ventricular dysrhythmias. Meticulous attention to the ECG and a directly transduced arterial pressure during manipulation in this region are necessary to provide the surgeon with immediate warning of the risk of damage to the adjacent cranial nerve nuclei and respiratory centers. Pharmacologic treatment of the dysrhythmias that occur may serve to attenuate the very warning signs that should be sought.

Balloon compression of the trigeminal ganglion is another situation in which dysrhythmias may occur. The procedure attempts to produce neurapraxia of the fifth cranial nerve by the rapid inflation of a Fogarty-type balloon within Meckel's cave. The balloon is introduced percutaneously through the cheek and beneath the maxilla. The procedure is best accomplished with general anesthesia because both the entry of the needle into Meckel's cave and the balloon compression (lasting several minutes) are intensely stimulating. A relatively profound, though transient bradycardia will occur and is, in fact, sought as confirmation of adequate compression. External pacemaker pads have been advocated but are, in our experience, unnecessary.

Any irritation and injury of posterior fossa structures that may have occurred during surgery should be taken into account when planning extubation and postoperative care. In particular, procedures involving dissection on the floor of the fourth ventricle entail the possibility of injury to cranial nerve nuclei or postoperative swelling in that region, or both. Attention should be paid to the fact that cranial nerve dysfunction, particularly nerves IX, X, and XII, can result in loss of control/patency of the upper airway and that swelling of the brainstem can result in impairment in both cranial nerve function and respiratory drive. The posterior fossa is a relatively small space, and its compensatory latitudes are even more limited than those of the supratentorial space. Relatively little swelling can result in disorders of consciousness, respiratory drive, and cardiomyotor function. The anesthesiologist and surgeon should interact to make decisions regarding whether extubation is appropriate and the location where postoperative observation should take place (i.e., ICU or non-ICU).
Spontaneous ventilation was once advocated for procedures that entailed a risk of damage to the respiratory centers. Spontaneous ventilation is now rarely used because the proximity of the cardiomotor areas to the respiratory centers should permit cardiovascular signs to serve as an indicator of impending injury to the latter. It is our opinion that the respiratory pattern is more likely to be a relevant monitor when the "threat" to the brainstem is the result of vessel occlusion (as might occur with accidental interruption of perforating vessels during vertebrobasilar aneurysm surgery) than when it is due to direct mechanical damage caused by retraction or dissection in the brainstem.

Various electrophysiologic monitoring techniques may be used during posterior fossa surgery, including somatosensory evoked responses, brainstem auditory evoked responses, and electromyographic monitoring of the facial nerve. The latter requires that the patient not be paralyzed or have a constant state of incomplete paralysis. Somatosensory evoked response monitoring imposes some constraints with respect to the selection of anesthetics, as discussed in Chapter 38.

**TRANSSPHENOIDAL HYPOPHYSECTOMY**

The transsphenoidal approach to the pituitary is used for the excision of tumors that lie within the sella or that have extension to the immediate suprasellar area. The most common lesions are prolactin-secreting microadenomas. These patients are usually women with secondary amenorrhea. Three other less common pituitary tumors are growth hormone-secreting lesions, which result in acromegaly; adrenocorticotropic hormone (ACTH)-secreting tumors, which cause Cushing’s disease; and a very rare thyroid-stimulating hormone (TSH)-secreting lesion that results in hyperthyroidism (Table 53-13).

**Table 53-13 — Tumors of the pituitary region**

<table>
<thead>
<tr>
<th>Location</th>
<th>Hormone Secreted</th>
<th>Clinical Findings</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior pituitary</td>
<td>Prolactin</td>
<td>Galactorrhea, amenorrhea, hypogonadism, infertility</td>
<td>Bromocriptine sensitive</td>
</tr>
<tr>
<td></td>
<td>Adrenocorticotropin</td>
<td>Cushing’s disease (hypercortisolism)</td>
<td>Basophilic adenoma</td>
</tr>
<tr>
<td></td>
<td>Growth hormone</td>
<td>Acromegaly/gigantism, glucose intolerance</td>
<td>Eosinophilic adenoma</td>
</tr>
<tr>
<td></td>
<td>Nonsecretory</td>
<td>Mass effect, panhypopituitarism</td>
<td>Chromophobe adenoma</td>
</tr>
<tr>
<td></td>
<td>Suprasellar</td>
<td>Panhypopituitarism, SIADH, visual (optic chiasm) symptoms</td>
<td>Craniopharyngioma or suprasellar extension of pituitary lesion</td>
</tr>
<tr>
<td></td>
<td>Nonsecretory</td>
<td>Hydrocephalus</td>
<td></td>
</tr>
<tr>
<td>Location</td>
<td>Hormone Secreted</td>
<td>Clinical Findings</td>
<td>Comment</td>
</tr>
<tr>
<td>----------</td>
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<td>-------------------</td>
<td>---------</td>
</tr>
<tr>
<td>SIADH, syndrome of inappropriate antidiuretic hormone secretion.</td>
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<td></td>
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</tbody>
</table>

**PREOPERATIVE EVALUATION**

The important preoperative considerations relate to the patient's endocrine status. In general, as a pituitary lesion expands and compresses the pituitary tissue, the sequence in which hormonal function is lost is first gonadotrophins; second, growth hormone; third, ACTH; and fourth and last, TSH. The precise definition of the adrenal status of these patients is often not critical because the patients will commonly receive adrenal hormone supplementation at least temporarily. However, profound hypocortisolism with associated hyponatremia should be corrected preoperatively. It is, in fact, uncommon for thyroid deficiency to occur. However, hypothyroidism should be sought and corrected preoperatively because hypothyroid patients have a diminished tolerance to the cardiovascular depressant effects of anesthetics. An enlarged tongue can develop in patients with advanced acromegaly, and the airway should be evaluated accordingly.

**MONITORING**

Many practitioners place an arterial catheter, but it is not absolutely necessary. Access for blood sampling is a valuable adjunct to postoperative care if diabetes insipidus develops. Blood loss is usually modest. However, the cavernous sinus is in an immediate lateral relationship to the pituitary and may be entered during the resection of large tumors. In addition, some patients have a venous sinusoid that lies in front of the pituitary gland and connects the two cavernous sinuses. This sinusoid can be the origin of substantial blood loss. It has, on occasion, actually precluded this approach to the pituitary gland.

**ANESTHETIC TECHNIQUE**

Latitudes are broad with respect to the choice of anesthetics, although tumors with suprasellar extension can cause hydrocephalus and thereby add increased ICP constraints to the anesthetic technique. The procedure is performed in a supine position, usually with some degree of head-up posture to avoid venous engorgement. A pharyngeal pack will prevent an accumulation of blood in the stomach (which causes vomiting) or in the glottis (which contributes to coughing at extubation). A RAE-type tube secured to the lower jaw at the corner of the mouth opposite the surgeon's dominant hand (e.g., the left corner of the mouth for a right-handed surgeon) is suitable. A small esophageal stethoscope and temperature probe can lie with the endotracheal tube. Covering the entire bundle with a towel drape (a plastic sheet with an adhesive edge) placed just below the lower lip so that it hangs from the lower jaw like a veil will protect it from the preparation solutions.

The procedure requires a C-arm image intensifier (lateral views), and the head and arms are relatively inaccessible once the patient is draped. It is appropriate to establish the nerve stimulator at a lower extremity site. The surgical approach is through the nasal cavity by means of an incision made under the upper lip. During the approach, the mucosal surfaces within the nose are infiltrated with a local anesthetic and epinephrine solution, and the patient should be observed for the occurrence of dysrhythmias.

Surgical preferences for CO₂ management will vary. In some instances, hypocapnia will be requested to reduce brain volume and thereby minimize the degree to which the arachnoid bulges into the sella. One of the important surgical considerations is the avoidance, when possible, of opening the arachnoid membrane. Postoperative CSF leaks can be persistent and are associated with a considerable risk of meningitis. By contrast,
in tumors with suprasellar extension, normal or increased CO₂ will help deliver the lesion into the sella for excision. As an alternative method, some surgeons have resorted to "pumping" saline or air into the lumbar CSF space.

Diabetes insipidus (DI) is a potential complication of this procedure. Antidiuretic hormone (ADH) is synthesized in the supraoptic nuclei of the hypothalamus and is transported down the supraoptic-hypophyseal tract to the posterior lobe of the pituitary. This portion of the pituitary gland is frequently spared. Even when it is excised, water homeostasis commonly normalizes, presumably because ADH is released from the cut end of the tract. However, even when the posterior lobe of the pituitary is left intact, transient DI may occur. DI usually develops 4 to 12 hours postoperatively and very rarely arises intraoperatively. The clinical picture is one of polyuria in association with a rising serum osmolality. The diagnosis is made by comparison of the osmolality of urine and serum. Hypo-osmolar urine in the face of an elevated and rising serum osmolality strongly supports the diagnosis. Urine specific gravity is a useful bedside test. In the presence of bona fide DI, specific gravity will be low, less than 1.002.

When the diagnosis of DI is established, an appropriate fluid management regimen is hourly maintenance fluids plus two thirds of the previous hour's urine output. (An acceptable alternative is the previous hour's urine output minus 30 mL plus maintenance.) The choice of fluid will be dictated by the patient's electrolyte picture. In general, the patient is losing fluid that is hypoosmolar and relatively low in sodium. Half-normal saline and 5% dextrose in water (D5W) are commonly used as replacement fluids. Beware of hyperglycemia when large volumes of D5W are used. An unacceptable fluid regimen that has been used calls for maintenance fluids plus the previous hour's urine output. This regimen has the potential to put you and the patient in a "chase your tail" situation. Should the patient become iatrogenically fluid overloaded, this regimen precludes a return to isovolemia, and in fact, when the maintenance fluid allowance is generous, it guarantees that the patient will become increasingly hypervolemic. If the hourly requirement exceeds 350 to 400 mL, desmopressin (DDAVP) is usually administered.

A smooth emergence (see the section "Emergence from Anesthesia") is desirable, especially if the CSF space has been opened (and resealed with fibrin glue or by packing the sphenoid sinus with fat or muscle). Repeated, intense Valsalva maneuvers, as with coughing or vomiting, may contribute to reopening of a CSF leak and worsen the risk for subsequent meningitis. The airway should be cleared of debris, including formed clot. Some clinicians routinely inspect the pharynx with a laryngoscope. Such inspection also permits assessment of whether active bleeding is still present, and this allows one to more confidently extubate promptly at the first signs of reactivity to the endotracheal tube.

In situations in which one is concerned that a persistent CSF leak may occur, some surgeons will place a lumbar CSF drain to maintain CSF decompression in the early postoperative period.

SEIZURE SURGERY/AWAKE CRANIOTOMY

Awake craniotomies are performed when tumors or epileptic foci lie close to the cortical areas required for either speech or motor function or close to the mesial temporal structures critical to short-term memory. Most patients will have so-called temporal lobe epilepsy. A structural lesion is commonly visible on magnetic resonance imaging (MRI). Sometimes there is a history of trauma. More commonly, the cause is presumed to be an asphyxial birth injury.
PRESURGICAL EVALUATION

Before resection, most patients will have undergone either or both a Wada test and videotelemetry. More recently, functional testing using MRI or positron emission tomography or both has also been introduced to the presurgical evaluation. The Wada test involves selectively anesthetizing the cerebral hemispheres by injection of amobarbital (Amytal) into the carotid artery to localize the hemisphere that controls speech or confirm that there is bilateral representation for short-term memory (or both). Speech is an issue when the lateral portions of the temporal lobe are involved, and memory is the concern when the involvement is medial.

ANESTHESIA FOR EEG ELECTRODE PLACEMENT

Videotelemetry is performed to permit localization of the seizure focus that is responsible for the clinically problematic events. Previous placement of either subdural strip electrodes (through bur holes) or a subdural electrode grid (requiring a craniotomy) is generally required. Occasionally, electrodes are placed deep into parenchyma, usually within the temporal lobe (placed stereotactically through bur holes), or they are positioned so that they "look at" the inferior surfaces of the temporal lobe. The latter is commonly accomplished with so-called foramen ovale electrodes. These electrodes are placed by using a needle similar to an epidural needle. The point of entry is about 2 cm lateral to the angle of the mouth. The needle is passed through soft tissue, under the temporal process of the zygomatic bone and medial to the ramus of the mandible, up to the base of the skull in the vicinity of the foramen ovale. Typically, this procedure is performed as a "MAC," although small doses of induction anesthetic, most often propofol, are usually required at the time of stimulation of the periosteum at the base of the skull by the needle. After placement of the relevant electrodes, the patient's seizure medication is discontinued and the patient remains in an observation unit with EEG and patient behavior recorded continuously. In this manner, the EEG events associated with the clinically significant seizure events and their anatomic origin can be identified.

PREANESTHETIC EVALUATION/PREPARATION

At the preoperative interview, the patient should be educated about the nature and duration of the procedure and the limitations on movement. One should obtain a description of both the aura and the seizures to facilitate recognition of them. One should ascertain whether the patient is subject to grand mal convulsions. If intraoperative electrocorticography to identify seizure foci is intended, it is common to discontinue or reduce the anticonvulsant dose by half according to the perceived risk of uncontrolled seizures. Premedics with an anticonvulsant effect, such as the benzodiazepines, should not be used because they may interfere with intraoperative EEG localization.

ANESTHETIC TECHNIQUE

The objectives of the anesthetic technique are to

1. Minimize patient discomfort associated with the potentially painful portions of the procedure and with the prolonged restriction of movement

2. Ensure patient responsiveness and compliance during the phases of the procedure that require assessment of either speech or motor/sensory responses to cortical stimulation
3. Select anesthetic techniques that produce minimal inhibition of spontaneous seizure activity

There are probably many ways of providing sedation that are consistent with the aforementioned objectives, and many techniques are in active use. They range from minimal sedation approaches, through deep sedation during which intermittent unresponsiveness is achieved with spontaneous ventilation and an unprotected airway, to asleep-awake-asleep techniques with intermittent airway management with an LMA, sometimes with positive-pressure ventilation. However, at the outset, the clinician should appreciate two things. First, the essential element of an "anesthetic" for an awake craniotomy is the surgeon's local anesthetic technique. "Sedation" cannot compensate for inadequate anesthesia of the scalp, as accomplished with pin site infiltration and nerve blocks. Anesthesiologists must not get trapped into thinking that it is their responsibility to provide a general anesthetic equivalent in a spontaneously breathing patient with an unprotected and all but inaccessible airway. Second, most craniotomies for which an awake technique is relevant can be accomplished without the presence of anesthesia personnel. No anesthetist was present when Wilder Penfield made the observations during open-craniotomy procedures that defined the motor and sensory homunculi. During the anesthesiologist shortage of the nineties, awake craniotomies were performed as "straight locals" in at least one institution that we visited, again with no anesthesia personnel present. Accordingly, there can be little justification for morbidity arising from the routine administration of sedatives and analgesics.

Several centers have used a droperidol/synthetic narcotic combination \[^{317}\] (e.g., droperidol, 2.5 to 7.5 mg; alfentanil, 5- to 10-mg/kg load, 0.5- to 1.0-mg/kg/min infusion; fentanyl, 0.7-mg/kg load, 0.7-mg/kg/hr infusion).\[^{318}\] Others use principally propofol by either physician- or patient-controlled infusion.\[^{318}\] Care should be taken when administering additional sedative agents, especially narcotics, whose respiratory depressant effects might be synergistic with propofol. This consideration is especially relevant when pin fixation is used. Pin fixation severely restricts the anesthesiologist's capacity to intervene quickly in the event of excessive respiratory depression or loss of patency of the airway. Propofol, if used, should be discontinued at least 15 minutes before EEG recording. In spite of prompt awakening, propofol leaves a residual EEG "footprint" characterized by high-frequency, high-amplitude beta activity that can obscure the abnormal activity that is being sought in the cortical surface EEG.\[^{319}\] Various groups have reported use of the LMA, commonly with narcotic-propofol sedation and spontaneous ventilation during the craniotomy; administration of the sedate is discontinued and the LMA is removed once the brain surface is exposed.\[^{321}\] One of these groups reported that complications occurred least often when the asleep phases were accomplished with infusion of remifentanil and propofol and positive-pressure ventilation.\[^{322}\] More recently, the α2-agonist dexmedetomidine has been used by some, both with and without interventional use of the LMA.\[^{321}\] The authors of these series report satisfactory conditions for functional testing, including brain stimulation for speech mapping, sometimes with the dexmedetomidine infusion ongoing at low dose (0.1 to 0.3 mg/kg/hr) during the testing.\[^{321}\] However, delay in achieving satisfactory patient responsiveness has been reported.\[^{324}\]\[^{325}\] The experience with administration of dexmedetomidine during electrocorticography is very limited, and that experience, obtained during light general anesthesia achieved with the combination of sufentanil and dexmedetomidine, revealed suppression of sharp wave and spike activity relative to preoperative recordings.\[^{326}\] It is obvious that many approaches can be effective, but the clinician should note that to our knowledge, LMA techniques have not been reported in patients in pin fixation.

Routine, noninvasive monitors are almost always sufficient. Reliable capnography to provide breath-by-breath confirmation of airway patency and respiratory drive is an essential component of the technique if deep sedation is intended for any portion of the procedure. These procedures are often lengthy, and attention to the details of patient comfort (warming blankets, a sheepskin, room temperature) will improve patient tolerance.
The uncomfortable phases of the procedure are placement of the pin head holder (not all groups use a pin head holder) and the craniotomy. Many patients also find manipulation of the dura painful, in particular, traction. The actual manipulation of supratentorial brain parenchyma is painful. The volume of local anesthetic used to infiltrate pin sites and perform a field block of the scalp can be substantial. It is appropriate for the anesthesiologist, in particular, an anesthesiologist working with surgical trainees, to keep track of the doses of local anesthetics used.

The anesthesiologist should participate actively at the time of head positioning. The more "sniff" that can be achieved before the final lockdown of the head holder, the wider the latitudes will be for sedating the patient while maintaining spontaneous ventilation and a patent airway. During positioning of the patient, attention should also be paid to the need to maintain visual access to the face. A clear line of sight to the face will be necessary both to present the patient with images to name as part of speech testing and to identify the occurrence of facial motor responses during mapping of the motor strip.

In general, after the dural opening is complete, cortical surface EEG recording is performed to locate the seizure focus. If no seizure activity is observed, provocative maneuvers may be requested. Methohexital in a dose of approximately 0.3 mg/kg is in general safe and effective. Etomidate, approximately 0.05 to 0.1 mg/kg, has also been used. Localization of the seizure focus can also be accomplished during light general anesthesia, for example, N₂O/fentanyl/low-dose isoflurane. During general anesthesia, alfentanil in a bolus dose of 30 to 50 μg/kg, [33][35] etomidate in doses of 0.2 to 0.3 mg/kg, [330][331] and remifentanil as a bolus of 2.5 μg/kg [332] have been reported to be effective in activating seizure foci.

After localization by EEG, functional testing may be performed by stimulating the cortical surface electrically and observing for motor, sensory, or speech interruption effects. During stimulation, the anesthesiologist should be prepared to treat grand mal convulsions that are not self-limited. Thiopental in 1-mg/kg increments is appropriate. However, thiopental should be withheld until it is clear that the seizure is not going to terminate spontaneously because it may interfere with subsequent EEG localization of the seizure focus for some time. A comprehensive review of the "anesthetic implications of epilepsy, status epilepticus, and epilepsy surgery" is available. [333]

**STEREOTACTIC PROCEDURES**

Stereotactically guided procedures are performed for numerous indications, including biopsy of small deep-seated lesions, placement of deep brain stimulation electrodes (for Parkinson's disease and other movement disorders), and ablation of lesions (for movement disorders and temporal lobe epilepsy). The future may also bring placement of intraparenchymal stimulators for control of epilepsy and stereotactic stem cell implantation for a variety of degenerative and ischemic disorders. The issues with which the anesthesiologist must contend include restriction of airway access by the frame, restriction of sedative latitudes when electrophysiologic recordings are to be performed as a guide to device placement, and detection and management of complications (principally intracerebral hematomas).

Preoperative evaluation should include attention to ensuring that the coagulation process is intact and that the patient has not been taking platelet-inhibiting agents (including herbal medications). Patients should be given a careful explanation of the procedure, its probable duration, and the necessary restraints on movement.
Commonly, the stereotactic frame, of which there are many variations, is placed with the use of a local anesthetic, and the patient undergoes an imaging study before being brought to the operating room. In some instances, the frame will prevent application of the mask and ventilation, laryngoscopy, or neck extension. If a general anesthetic is to be administered, awake intubation may be required. If sedation is to be used, the anesthesiologist should have certain knowledge regarding how to remove the device rapidly in an urgent situation (including knowledge of the whereabouts of the requisite "key" or spanner device).

When electrophysiologic recordings are not intended, sedative regimens similar to those used for awake craniotomy (see the preceding section, "Head Injury") may be appropriate. However, for placement of thalamic and subthalamic stimulators, part of the localization process entails identifying the typical electrophysiologic "footprint" of specific nuclei. Because the nature and duration of the effects of anesthetics on these signals are not known systematically, some surgeons may request that no sedative whatsoever be given. If sedatives are administered, they must be given in a manner that ensures the ability to perform precise intermittent neurologic examination of the patient. An issue that arises in some patients with movement disorders (e.g., Parkinson's disease) is the problem of obtaining high-quality radiologic images in the presence of a persistent tremor. Sedation immediately preceding stereotactic placement may therefore be inevitable. Propofol has been used, but the window between propofol administration and subsequent recording should be as long as possible. Dexmedetomidine has also been used and has been reported to us to be very effective in suppressing movement in patients with Parkinson's and other movement disorders in doses that maintain patient responsiveness and respiration (Mary K. Sturaitis, M.D., personal communication).

During the subsequent procedure, among the anesthesiologist's objectives is the prevention or treatment of hypertension. The concern is that in the face of multiple needle passes through the brain, hypertension will precipitate the development of an intracerebral hematoma. In the event of a substantial hematoma, an urgent craniotomy may be required, and the anesthesiologist should be prepared from the outset for this eventuality. These procedures are lengthy, and restlessness may occur, especially in unsedated patients. The nonsystematic experience, obtained in situations in which the options were to sedate the patient or abandon the procedure, has been that satisfactory recordings can be made in the presence of dexmedetomidine. That drug has the advantage that it also contributes to the control of hypertension (Mary K. Sturaitis, M.D., personal communication).

**NEURORADIOLOGIC PROCEDURES**

**MAGNETIC RESONANCE IMAGING**

The major constraints for this procedure are created by the powerful magnetic field used. It creates three conditions that have an impact on the anesthetic technique. The first is that any ferromagnetic object that approaches the magnet has the potential to become a dangerous projectile. The second is that a wide variety of electronic instruments will not function properly in the vicinity of the magnet, including most notably those that contain oscilloscopes, solenoids (e.g., some Dinamap-type blood pressure devices), or galvanometer-type gauges. The third is that when moderately large metal objects, even non-ferromagnetic objects, are brought into the vicinity of the magnet, they may degrade the image. The equipment limitations have largely been circumvented. At present, MRI-compatible ECGs, oximeters, capnographs, noninvasive blood pressure monitors, and gas machines are available. It is only temperature monitoring that cannot be readily accomplished, and with that exception, there is no longer any justification for incomplete monitoring of patients undergoing MRI.
It is most frequently children, claustrophobic adults, and patients with painful conditions who require anesthesia. Sedation with propofol and an unprotected airway and general anesthesia with either an LMA or endotracheal tube have been used successfully.

INTERVENTIONAL NEURORADIOLOGY (ALSO SEE CHAPTER 69)

A wide variety of procedures are performed for the evaluation and treatment of intracranial and extracranial disease. These procedures principally include attempts to obliterate aneurysms or devascularize tumors and AVMs. Stenting of extracranial carotid disease is occasionally performed. Vasospasm can be treated by selective intraarterial instillation of papaverine or more commonly by balloon dilatation. Most of these procedures can be accomplished without the involvement of an anesthesiologist. The duration of a procedure, individual patient factors, and occasionally, the necessity for precise physiologic control may result in requests for monitored anesthesia care or general anesthesia. A chin-tucked position (to remove the bones of the face from the line of the anteroposterior x-ray source) and absolute immobility are commonly needed by the radiologist, and as a result, when the anesthesiologist's assistance is requested, a general anesthetic will often be necessary. In addition, anesthesiologists may become involved during the resuscitation stage in the event of vascular rupture or migration of an intravascular device to an incorrect location. When detachable devices (coils, balloons) are misplaced and ischemia ensues, fluid loading and pressor administration may be requested to improve collateral CBF while the device is retrieved.

Hyperventilation may be appropriate in an attempt to divert flow away from normal brain and toward a lesion that is intended to receive the occlusive device or material. Occasionally, the anesthesiologist will be asked to lower systemic blood pressure and, it is hoped, cardiac output to facilitate the initial trapping of glue or coils within a vascular lesion by briefly reducing the flow through it. These requests have decreased in frequency as glue and balloons have been replaced by platinum coils. Adenosine has proved effective when the occasion arises.\(^{1,4}\)

The anesthesiologist may also be asked to participate in test occlusion of various cerebral vessels that are candidates for sacrifice at a subsequent procedure. In these circumstances, it is sometimes necessary to manage a patient who is restless, confused, or abruptly unconscious.

Procedures that are likely to entail requests for blood pressure manipulation are best done with an arterial catheter in place. The radiologist's arterial line cannot be dedicated to blood pressure monitoring, and accordingly, an independent arterial catheter should be established.

The relative roles of coiling and open clipping of intracranial aneurysms and stenting and open endarterectomy for extracranial carotid disease are yet to be defined. However, it seems inevitable that both coiling and stenting are here to stay. A recently reported prospective multicenter (United Kingdom, Scandinavia, northern Europe) comparison of clipping and coiling revealed significantly better 1-year disability-free survival with coiling, although some instances of rebleeding occurred in the coiling patients.\(^ {335}\) Proponents of the open operation were critical because the surgery in that trial was not consistently performed by neurosurgeons with practices dedicated to neurovascular surgery. The discussion will go on, but it appears that coiling will inevitably have a role, potentially an expanding one. The role of carotid angioplasty is similarly unclear at this time. Although it has typically been reserved for patients deemed to have the highest medical risk, morbidity and mortality similar to that associated with the open operation have been reported recently.\(^ {336}\) Note that the results of older trials may not be applicable because in many instances, distal protection devices (to protect the brain from
embolized debris from the angioplasty site) were not used. The hazards of that omission have been confirmed, and contemporary angioplasty will use them. There are at least three ongoing prospective comparisons of carotid endarterectomy versus angioplasty that should help define the optimal role of the latter.\textsuperscript{337} That role may in large part be determined by the durability of the stenting procedure.

For anesthesiologists asked to provide monitored anesthesia care for these procedures, the significant issue is the bradycardia that occurs at the time of balloon dilatation of the stenotic region. The anxiety about this physiologic event was such that at one time, transvenous pacemakers were routinely placed by some groups. This practice gave way to the availability of transthoracic pacers and finally to the realization that prophylactic glycopyrrolate and occasionally reactive administration of atropine will suffice. As the issue of profound bradycardia has become less threatening, the practice of asking for anesthesia assistance has all but ceased in many institutions (including ours).

**CSF SHUNTING PROCEDURES**

CSF shunts are inserted for the relief of a variety of hydrocephalic states and pseudotumor cerebri. Hydrocephalus can be communicating or noncommunicating. In a noncommunicating hydrocephalus, CSF egress from the ventricular system is obstructed. Such obstruction can occur as a result of blood or infection in the ventricular system or tumors in or adjacent to the ventricular system. In a communicating hydrocephalus, CSF escapes from the ventricular system but is not absorbed by the arachnoid villi. This occurs most commonly secondary to infection or blood in the CSF space. Some degree of communicating hydrocephalus is particularly common after SAH.

The ventriculoperitoneal shunt is the most commonly used device. Generally, a catheter is inserted through a bur hole into the frontal horn of the lateral ventricle on the nondominant (usually the right) side. A reservoir is placed subcutaneously adjacent to the bur hole, and the drainage limb passes through a subcutaneous tunnel to a point near the epigastrium, where it is inserted into the peritoneal space through a very small laparotomy. A moderate degree of muscle relaxation may be helpful. A distended stomach can result in an inadvertent "gastrostomy." Occasionally, most commonly in pediatric patients, there may be obstruction at more than one level in the ventricular system, and a so-called double-barreled shunt becomes appropriate. In this instance, there are two proximal ends: usually one in the lateral ventricle and one in the fourth ventricle. This latter procedure is generally performed in the prone position, whereas most ventriculoperitoneal shunts are performed in the supine position.

Occasionally, when a communicating hydrocephalus is present, a lumbarperitoneal shunt is inserted. The patient is placed in a lateral position, and a catheter is inserted into the lumbar CSF space with the use of a Tuohy-type needle. The catheter is then tunneled subcutaneously around to the anterior abdominal wall and inserted into the peritoneal space through a small laparotomy.

In the past, ventriculoatrial shunts were used, although they have largely been abandoned because of the occurrence of pulmonary embolism. For these shunts, the noncerebral end is inserted into the venous system through the jugular vein. The atrial location of the noncerebral end is confirmed by using the same ECG technique that is used to place VAE recovery catheters (see the section "Venous Air Embolism").
ANESTHETIC MANAGEMENT

Invasive monitoring is not generally required. The anesthetic technique should be chosen to avoid further increases in ICP. Moderate hyperventilation (PaCO₂ of 25 to 30) is customary. However, aggressive ICP reduction measures are not warranted because collapsing the ventricles may render them more difficult to "hit" with the ventricular catheter. The procedure is usually performed in the supine position, with the table turned 90 degrees and the head turned toward the anesthesiologist. Blood pressure may drop abruptly (as brainstem pressure is relieved) when the ventricle is first cannulated. Infrequently, brief pressor support is required. Burrowing the subcutaneous tunnel can produce a sudden painful stimulus. Postoperative discomfort is only minor.

Unlike most neurosurgical patients, shunt patients are often nursed flat after their procedures in an attempt to prevent excessively rapid collapse of the ventricular system. Empirically, there is a small incidence of subdural hematoma after shunting, and tearing of the bridging veins at the time of rapid brain shrinkage is a suspected cause.

PEDIATRIC VENTRICULOOPERITONEAL SHUNTS

Shunts are probably more commonly performed in children than adults. Common indications are hydrocephalus occurring in association with meningomyelocele, neonatal intraventricular hemorrhage, and posterior fossa tumors. Although one can never be casual about the management of these patients, open fontanelles seem to provide some margin for error in younger patients, and in addition, palpation of the fontanelles provides on-line trend monitoring of "ICP." In spite of the theoretical considerations, induction with volatile anesthetics is empirically well tolerated, even in children with closed fontanelles. However, we would avoid that induction technique in a child who was already stuporous. When an intravenous line is available, we generally use a propofol-relaxant induction sequence. For children in whom cannulation of a peripheral vein cannot be accomplished readily, induction with inhaled sevoflurane is a common approach, with initiation of controlled ventilation by bag and mask as rapidly as possible. If in the absence of sevoflurane, halothane were used for induction, immediately after loss of consciousness we would generally change to isoflurane and control the ventilation manually. After establishing controlled ventilation, an ideal course at this point is to establish an intravenous line, administer a muscle relaxant (with or without atropine according to your biases) and perhaps an induction drug, and intubate the patient in these optimal circumstances. Anesthesia is most commonly maintained thereafter with 60% to 70% nitrous oxide, mechanical hyperventilation, and isoflurane or sevoflurane as required. For children in excess of 6 months of age who were not stuporous at the outset, we commonly administer 2 to 3 µg/kg of fentanyl in the belief that this procedure is not entirely pain-free postoperatively and, in addition, that a smoother emergence can be accomplished with a narcotic background.

PEDIATRIC NEUROSURGERY (ALSO SEE CHAPTER 60)

Table 53-14 identifies common pediatric procedures and their anesthetic considerations. The most frequent procedures are probably placement and revision of CSF shunts (discussed in the preceding section "CSF Shunting Procedures"). The majority of pediatric tumors occur in the posterior fossa. Most are near the midline, and many are associated with hydrocephalus. For pediatric posterior fossa procedures, the risk of VAE, monitoring, and treatment are similar for adults and children (see the section "Venous Air Embolism"). Doppler is invariably indicated, and right heart catheters are generally placed when procedures are performed in the sitting position. Craniosynostosis procedures have the potential for substantial blood loss that is roughly
proportional to the number of sutures involved. The significant risk for VAE justifies the use of precordial Doppler.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Lesion</th>
<th>Pathogenesis</th>
<th>Anesthetic Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates</td>
<td>Intraventricular hemorrhage</td>
<td>Subependymal vascular rupture</td>
<td>Associated problems of prematurity</td>
</tr>
<tr>
<td></td>
<td>Depressed skull fracture</td>
<td>Forceps injury</td>
<td>Associated cerebral edema</td>
</tr>
<tr>
<td>Infants</td>
<td>Hydrocephalus</td>
<td>Varied</td>
<td>Increased ICP, especially dangerous in shunt-dependent revisions</td>
</tr>
<tr>
<td></td>
<td>Meningocele</td>
<td>Outpouching of meninges through skull defect</td>
<td>Large size creates airway management problems</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Prone-lateral position</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Substantial blood loss</td>
</tr>
<tr>
<td></td>
<td>Encephalocoele</td>
<td>Outpouching of meninges through skull defect with brain tissue enclosed</td>
<td>Repair may increase ICP</td>
</tr>
<tr>
<td></td>
<td>Myelomeningocele</td>
<td>Protrusion of spinal meninges and roots</td>
<td>Prone or lateral through spina bifida</td>
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<td></td>
<td></td>
<td></td>
<td>Substantial blood loss</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Respiratory restriction after covering large defects</td>
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<tr>
<td></td>
<td>Arnold-Chiari malformation</td>
<td>Impaction of posterior fossa contents into foramen magnum</td>
<td>Brainstem compression with head flexion</td>
</tr>
<tr>
<td></td>
<td>± Hydrocephalus</td>
<td></td>
<td>Increased ICP; latex allergy</td>
</tr>
<tr>
<td></td>
<td>± Myelomeningocele</td>
<td></td>
<td>Postoperative respiratory depression</td>
</tr>
<tr>
<td></td>
<td>Craniosynostosis</td>
<td>Premature fusion of cranial sutures</td>
<td>Substantial blood loss</td>
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<td></td>
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<td></td>
<td>Air embolism</td>
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<tr>
<td></td>
<td>Craniofacial dysostosis</td>
<td>Developmental abnormality</td>
<td>Lengthy procedures</td>
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<td></td>
<td></td>
<td></td>
<td>Substantial blood loss</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Brain retraction</td>
</tr>
<tr>
<td>Age Group</td>
<td>Lesion</td>
<td>Pathogenesis</td>
<td>Anesthetic Considerations</td>
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<tr>
<td></td>
<td>Vascular malformations</td>
<td>Varied</td>
<td>Air embolism</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Endotracheal tube damage</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td></td>
<td>Subdural hematoma/effusion</td>
<td>Trauma</td>
<td>Large blood loss</td>
</tr>
<tr>
<td>Older children</td>
<td>Posterior fossa tumors</td>
<td>Ependymoma</td>
<td>Elective hypotension</td>
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<td></td>
<td></td>
<td>Astrocytoma</td>
<td>Associated injuries</td>
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<td></td>
<td></td>
<td></td>
<td>Malnutrition-dehydration</td>
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<td></td>
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<td></td>
<td>Hydrocephalus</td>
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<td></td>
<td></td>
<td></td>
<td>Increased ICP</td>
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<td></td>
<td></td>
<td></td>
<td>Prone or sitting position</td>
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<td></td>
<td></td>
<td></td>
<td>Air embolism</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Brainstem compression</td>
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<td></td>
<td></td>
<td></td>
<td>Postoperative cranial nerve dysfunction or brainstem swelling or compression</td>
</tr>
</tbody>
</table>

ICP, intracranial pressure.

**SPINAL SURGERY**

Table 53-15 surveys the many issues that may arise in the context of spinal cord and column procedures undertaken by neurosurgeons. The spinal cord has been described as a "microcosm of the brain" because its physiology is, in general, similar to that of the brain: CO₂ responsiveness, blood-"brain" barrier, autoregulation, high metabolic rate and blood flow (though somewhat less than the brain), and substantial ischemic vulnerability of gray matter. Measures to reduce spinal cord swelling analogous to ICP reduction maneuvers are, however, rarely used. The relevant electrophysiologic monitoring techniques are described in Chapter 38. Prone positioning considerations and the phenomenon of POVL are addressed in the earlier section "Positioning."

**Table 53-15 -- Anesthetic considerations and position requirements associated with various spinal surgical procedures**

<table>
<thead>
<tr>
<th>Spinal Segment and Surgical Condition</th>
<th>Problems/Considerations</th>
<th>Positions Used and Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic region: degenerative disease, spinal stenosis, trauma</td>
<td>Major position change</td>
<td>Prone, lateral, or knee-chest position</td>
</tr>
<tr>
<td>Spinal Segment and Surgical Condition</td>
<td>Problems/Considerations</td>
<td>Positions Used and Comments</td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>-------------------------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td>Awake intubation and position</td>
<td>If unstable after trauma and major position change required</td>
<td></td>
</tr>
<tr>
<td>Blood loss</td>
<td>Especially with reoperations, instrumentation, and spinal stenosis; risk of occult aortoiliac or major venous tear</td>
<td></td>
</tr>
<tr>
<td>Air embolism</td>
<td>Infrequent; perhaps with knee-chest position and Relton-Hall frames</td>
<td></td>
</tr>
<tr>
<td>Postoperative visual loss</td>
<td>Etiology unclear; associated with long prone procedures, low hematocrit, large estimated blood loss, and hypotension. Patient variables may contribute (see &quot;Prone&quot; in the section &quot;Positioning&quot;)</td>
<td></td>
</tr>
<tr>
<td>Cervical region: degenerative disk disease, stenosis, trauma, rheumatoid arthritis</td>
<td>Maintain neutral neck position to avoid cord compression</td>
<td>Supine/anterior approach for most disectomies. Posterior approach (prone or sitting) for laminectomy and occasional disectomy</td>
</tr>
<tr>
<td>Maintain perfusion pressure at waking normal levels</td>
<td>If existing cord compression or recent cord injury or if cord retraction required</td>
<td></td>
</tr>
<tr>
<td>Hypotension (spinal shock)</td>
<td>Occurs with complete cervical cord injury</td>
<td></td>
</tr>
<tr>
<td>Postoperative respiratory insufficiency</td>
<td>Occurs with cervical cord injury</td>
<td></td>
</tr>
<tr>
<td>Methylprednisolone</td>
<td>30 mg/kg over 1-hr period, then 5.4 mg/kg for 23 hr</td>
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<td>Air embolism</td>
<td>With sitting laminectomies</td>
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<td>Anterior cervical disectomy</td>
<td>Traction required for anterior graft insertion?</td>
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<td>Retractor compression of airway</td>
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<tr>
<td>Postoperative swelling/airway compression</td>
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<tr>
<td>Postoperative cranial nerve dysfunction</td>
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<tr>
<td>Cervical instability</td>
<td>Awake intubation</td>
<td>Prone or supine</td>
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<tr>
<td>Awake positioning</td>
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<td>Axial stabilization for intubation</td>
<td>If awake intubation not feasible</td>
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<tr>
<td>Vertebral metastasis</td>
<td>Large blood loss</td>
<td>Prone or anterolateral/retroperitoneal</td>
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<td>Spinal Segment and Surgical Condition</td>
<td>Problems/Considerations</td>
<td>Positions Used and Comments</td>
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<td>Maintain perfusion pressure during retraction</td>
<td>Double-lumen tube for lesions above L1</td>
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<td>Methylprednisolone</td>
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<td>Wake-up test</td>
<td>Prone. Rehearse with patient</td>
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<td>Somatosensory evoked responses</td>
<td>Anesthetic restrictions</td>
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<td></td>
<td>Motor evoked responses</td>
<td>Anesthetic/relaxant restrictions</td>
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<td>Pedicle screw electromyogram</td>
<td>Relaxant restriction</td>
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ACKNOWLEDGMENT

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REFERENCES


