Intraoperative Neuroanesthesia

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Abstract

This chapter broadly describes considerations and objectives that are included in the formulation of a neuroanesthetic management plan. There are unique intraoperative issues confronting the anesthesiologist for all neurosurgical procedures as anesthetic agents and technique may significantly affect cerebral oxygen consumption ($CMRO_2$), cerebral oxygen delivery (CDO_2), cerebral blood flow (CBF), intracranial tissue volume, intracranial pressure (ICP), arterial oxygen content (CaO_2), and the autoregulation of CBF. Recognition and treatment of concurrent comorbidities, optimization of the surgical exposure, and avoidance and management of surgery-related events in the operating room and during the perioperative period broadly define secondary objectives of the anesthetic intervention.

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A selection of representative clinical scenarios including surgeries for mass lesions, lesions in the posterior fossa, transphenoidal hypophysectomies, open and endovascular surgeries for aneurysms and other vascular malformations, surgical treatment of stroke, spine surgeries, and functional operations are presented. Unique considerations and complications such as positioning, postoperative visual loss, venous air embolism, neuroprotection, and neuromonitoring are reviewed in detail.

Keywords

Neural autoregulation • Postoperative visual loss • Venous air embolism • Trigeminal-cardiac reflex • Neuroprotection • Neuromonitoring • Cerebral aneurysmal clipping • Carotid endarterectomy • Functional surgeries • Pituitary adenoma • Posterior fossa

Introduction

Surgical intervention on the central nervous system (CNS) requires an in-depth appreciation of intracranial and spinal cord physiology and pathophysiology. The dynamic course of a surgical intervention can be quite challenging and requires an actively involved and informed anesthesiologist to assure the most favorable outcome.

This chapter will broadly describe considerations and objectives that are included in the formulation of a neuroanesthetic management plan. We will discuss the intraoperative issues confronting the anesthesiologist for a selection of representative clinical scenarios. Upon completion of surgery, the outcome of the intraoperative anesthetic and surgical management often does not become fully manifest until the patient is in the intensive or postanesthesia care unit. Familiarity with both the anesthetic and surgical characters of postoperative recovery can help one distinguish findings that are related to the anesthetic and are ephemeral from more ominous ones that may require aggressive intervention.

Anesthesia and Basic Neuropathophysiology

The balance of cerebral and spinal cord oxygen delivery with its consequences (e.g., induced systemic hypertension, lactate acidosis, free radical formation, vasodilation) is the foundation of every neuroanesthetic plan. These tasks are interrelated because agents used to accomplish surgical anesthesia also influence cerebral and spinal cord hemodynamics. Anesthetic agents and technique may significantly affect cerebral oxygen consumption ($CMRO_2$), cerebral oxygen delivery (CDO_2), cerebral blood flow (CBF), intracranial tissue volume, intracranial pressure (ICP), arterial oxygen content (CaO_2), and the autoregulation of CBF. Recognition and treatment of concurrent comorbidities, optimization of the surgical exposure, and avoidance and management of surgery-related events in the operating room and during the perioperative period broadly define secondary objectives of the anesthetic intervention.

Cerebral Oxygen Consumption

Approximately 40 % of oxygen consumed by the brain is used to maintain the cellular integrity of its neural tissue. The remaining 60 % of oxygen is consumed accomplishing cellular electrophysiologic functions [1]. Inhalational agents (IA), narcotics, and commonly used hypnotic agents all lower cellular metabolism (i.e., $CMRO_2$) by decreasing cortical neuron electrical activity. Isoflurane is perhaps the most studied IA for neurosurgical procedures. In addition to decreasing $CMRO_2$, isoflurane uniquely and substantially lowers the critical CBF at which the EEG begins to demonstrate cerebral ischemia from a baseline lower CBF flow threshold of 20 ml/100 g brain tissue/min to nearer 10 ml/100 g brain tissue/min [2, 3]. For example, the incidence of ischemia suggested by electroencephalography (EEG) during carotid endarterectomy (CEA) is lower with isoflurane than with the other IAs [4]. These observations have led to a body of work generally supportive of the use of isoflurane as a neuroprotective agent in low cerebral blood flow states. However, it is not clear if these effects are temporary or permanent [4]. Sodium thiopental and propofol, along with all other intravenous hypnotic agents (except ketamine), reduce $CMRO_2$ along with CBF and will in sufficient dose produce an isoelectric EEG pattern.

Cerebral oxygen consumption increases with cortical seizure activity and during hyperthermia. Conversely, cooling a patient slows all enzymatic and chemical reactions and lowers $CMRO_2$ by slowing processes responsible for maintaining cellular integrity and those responsible for electrical function [5]. Hence, cooling is one modality used to protect the brain from transient ischemic events. When autoregulation is intact, cooling the brain produces an approximate 5–7 % decrease in $CMRO_2$ per degree Celsius cooled [6]. Any decrease in CBF reduces cerebral blood volume (CBV) and decreases intracranial pressure (ICP). Induced hypothermia is not completely benign as it has associated risks of impaired coagulation, cardiac arrhythmias, leftward shift of the oxygen-hemoglobin dissociation curve, increased infection, and slowed metabolism of many medications.

While the effectiveness of hypothermia for neuroprotection in "global" ischemia continues to accumulate, evidence suggests that there may be a difference for "focal" ischemia [7–13]. In 2002, two studies showed improved neurological outcomes and mortality for patients who had medically

induced mild hypothermia after cardiac arrest [8, 9]. Since then, therapeutic mild hypothermia has been incorporated into the American Heart Association cardiac arrest guidelines [10]. For patients with traumatic brain injury, there are conflicting results regarding whether induced hypothermia is effective for neuroprotection [11, 12]. Studies are underway to further investigate the effectiveness of hypothermia in stroke patients [13].

For the management of temporary ischemia during aneurysm clipping, the Intraoperative Hypothermia for Aneurysm Surgery Trial (IHAST) published in 2005 did not find any outcome differences from mild hypothermia (33.5 °C) in this setting [7]. The majority of patients enrolled into the study had low-grade aneurysms, World Federation of Neurological Surgeons score of I, II, or III, where little difference in neurological outcome between any treatment groups would be expected. Many neuroanesthesiologists would employ normothermia in patients with these lower-grade diseases to avoid the associated risks (arrhythmias, prolonged intubation, increased infection, increased coagulation), while others maintain the potential benefit of decreased $CMRO_2$ outweigh these risks.

Intracranial Pressure

The intracranial space is a fixed volume with three major components: brain tissue (80–85 %), cerebrospinal fluid (7–10 %), and blood (5–8 %). In 1783, Alexander Monro described the cranium as a "rigid box" filled where the total volume tends to remain constant and that any increase in the volume of the cranial contents (e.g., brain, blood, or cerebrospinal fluid) will elevate intracranial pressure. Additionally, if any of the three volumes increase, it must occur at the expense of volume of the other two elements. George Kellie confirmed and published these observations in the early nineteenth century [14]. Now known as the Monro-Kellie doctrine, it describes a hyperbolic compliance curve relating ICP changes to intracranial volume. Normal ICP is approximately 10 mmHg. If the volume of one component increases, the volume of another must decrease or else the ICP rises. Brain tissue volume comprises both neural cells and extracellular fluid which can expand by tumor growth, intracranial bleed, and/or edema. There are two types of edema: vasogenic and cytotoxic. Vasogenic edema occurs in brain tissue surrounding tumors and is a consequence of the breakdown of the blood–brain barrier. This type of edema is amenable to steroid therapy. Cytotoxic edema, on the other hand, is a result of tissue ischemia and trauma and does not respond to steroid therapy. It will, however, respond to some degree to osmotic agents, although not to the same degree as normal brain tissue. Hydrocephalus occurs when there is excess cerebral spinal fluid (CSF). This can be due to impaired CSF absorption and is also referred to as communicating hydrocephalus, or impaired CSF circulation also referred to as obstructive hydrocephalus. Finally, cerebral blood volume can expand by either vasodilation with increased cerebral blood flow or obstruction to cerebral venous drainage.

Cerebral Blood Flow and Its Influences

Cerebral blood flow (CBF) is normally under autoregulatory control and dependent on cerebral oxygen demand ($CMRO_2$), cerebral perfusion pressure (CPP), arterial CO_2 ($PaCO_2$), and arterial O_2 (PaO_2). CBF in adults is normally approximately 50 ml/100 g/min. Gray matter has blood flow of approximately 75 ml/100 g/min and white matter 25 ml/100 g/min. The higher blood flow to gray matter is mostly secondary to the higher gray matter metabolic rate and related oxygen consumption. $CMRO_2$ is a major determinate of CBF. The exact mechanism of this regulation is not precisely known. Temperature, as previously mentioned, also has a great effect on $CMRO_2$, with a 5–7 % decrease per degree Celsius cooling (in the temperature range from 27 to 37 °C), and therefore also cerebral blood flow [6].

Changes in mean arterial pressure (MAP) and therefore cerebral perfusion pressure (CPP) normally cause changes in cerebral blood flow only when outside the limits of autoregulation which have been conventionally characterized between mean arterial pressure of 50 and 150 mmHg in normotensive patients and higher in chronically hypertensive patients (Fig. 41.1). When the MAP falls below the lower autoregulation limit, the brain is considered at risk for hypoperfusion and ischemia, and when the MAP is above the upper autoregulatory limit, hyperperfusion, hyperemia, and cerebral edema become risks. Cerebral blood flow autoregulatory response may not function properly at MAPs below 60–70 mmHg in

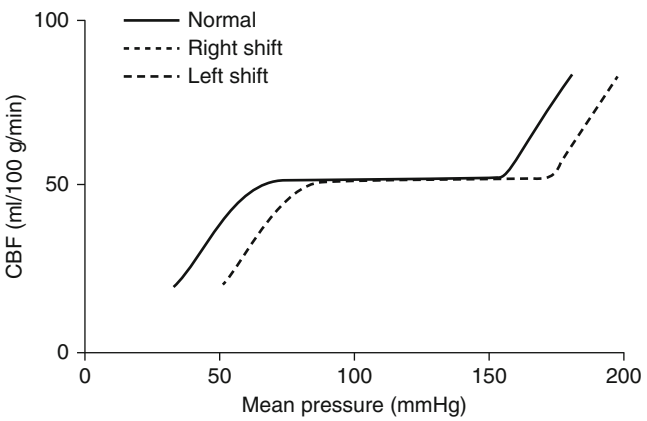

Fig. 41.1 Normal autoregulatory curve with right shift and left shift depicted (Reproduced with permission from Gravenstein and Gravenstein [75])

normotensive adults or at even higher levels in the uncontrolled chronically hypertensive patient and is slower in adolescents [15–17]. When autoregulation of regional CBF is compromised by acute injury, tumor-associated factors, vascular malformation, ischemia, or deeper planes of general anesthesia, CBF becomes increasingly pressure dependent. Proper intra- and perioperative blood pressure management are thus even more critical in these cases.

The effect of MAP on CBF, cerebral blood volume, and ICP is dependent on the volume of brain tissue under autoregulatory control. In situations where ICP is elevated but a substantial part of the brain remains under normal autoregulatory control, elevation of blood pressure will augment CPP by both increasing mean arterial pressure and through autoregulatory arteriolar constriction causing a slightly lowered CBV in the autoregulated part of the brain that exceeds the CBV increase experienced in the poorly autoregulated part of the brain. The combined effect causes a decrease of CBV followed by decreased ICP. In the scenario where a more extensive cerebral insult has occurred and autoregulation is fully compromised, ICP changes will follow MAP and CBV changes in the same direction. Because similar treatment can produce very different effects on patients with variable level of cerebral blood flow autoregulation, multimodality monitoring (jugular venous bulb oximetry [$S_{jv}O_2$], transcranial Doppler, cerebral oximetry, EEG, tissue pO_2, and invasive ICP monitoring) has all been used to guide therapy in selective cases [16, 18].

The primary influence of tissue pH on CBF is via the arterial partial pressure of CO_2 ($PaCO_2$). As $PaCO_2$ decreases, CBF is reduced and consequently CBV and ICP are also lowered. For $PaCO_2$ levels between 20 and 70 mmHg for each 1 mmHg change, there is an approximately 4 % change in cerebral blood flow (increase for CBF for increased $PaCO_2$ and decreased CBF for decreased $PaCO_2$). Outside of these levels, there is a plateau and no further cerebral vascular tone effects are seen. Aggressive hyperventilation poses the risk of inducing cerebral ischemia through the powerful vasoconstrictive effect of hypocapnia [19]. Deep hyperventilation therapy to control ICP has been associated with less favorable outcomes compared to normocapneic management. Intraoperatively, a mild hypocapneic state ($PaCO_2$, 35–40 mmHg) is the currently preferred goal.

Oxygenation (PaO_2) also exerts an effect on CBF. When administered in sufficient quantity to elevate the PaO_2 above 300 mmHg, it produces a mild (10 %) reduction of CBF [20]. Profound hypoxemia ($PaO_2 < 50$ mmHg) causes cerebral vasodilation and an exponential rise in cerebral blood flow, blood volume, and ICP. This is thought to be due to a decrease in pH secondary to lactic acidosis.

Ventilation parameters affect $PaCO_2$ and PaO_2. Anesthetic agents simultaneously influence $CMRO_2$, CPP [CPP = MAP – (the greater of CVP or ICP)], as well as diminishing in a dose-dependent fashion the CBF autoregulatory response [21]. All IAs (halothane, enflurane, isoflurane, sevoflurane, desflurane) attenuate cerebral autoregulation. Additionally, $CMRO_2$ is disproportionately decreased (uncoupled) compared to its effect on cerebrovascular resistance (CVR) and CBF. Halothane, the most potent cerebral vasodilator among the IAs, was therefore seldom used during neurosurgery because of this attribute. When IAs are used as part of a balanced neuroanesthetic, they are limited to being used in low concentrations and in conjunction with mild hypocapnia when there is concern about limited intracranial or intraspinal elastance (such as with elevated ICP or spinal cord injury).

Both general anesthesia and surgery have effects on cerebral physiology which may continue into the postoperative period. Cerebral hyperemia may persist for up to 30 min following extubation from an anesthetic combination of isoflurane /N_2O/O_2/fentanyl/atracurium or propofol/O_2/air/fentanyl/atracurium [22]. Whether these more prolonged effects result from trace anesthetic-induced impairment of autoregulation, hemodilution, recovery from prolonged hyperventilation, or a nonspecific response to stress is not known [23]. The intraoperative and postoperative management of blood pressure is also potentially related to the incidence of postoperative intracranial hemorrhage (ICH). When intraoperative or postoperative blood pressure (within the first 12 h of operation) remained above 160/90 mmHg for two or more consecutive measurements made 5 min apart, the incidence of postoperative ICH after craniotomy was higher in one study [24]. At the time of this writing, it is still not clear whether blood pressure control can prevent hyperemia.

The brain can recover from brief episodes of ischemia but has little chance of return to normal following traumatic herniation. Therefore, when the potential for mechanical brain injury via herniation exists, aggressive acute hyperventilation ($PaCO_2$, 20–25 mmHg) can in conjunction with other measures decrease cerebral volume even though it may risk cerebral ischemia. A $PaCO_2$ as low as 20 mmHg will decrease CBF to near 10 ml/100 g/min. Although this blood flow normally produces profound cerebral ischemia, in the presence of isoflurane anesthesia, it represents, at least in normal brain, the lowered threshold of where the EEG first demonstrates a pattern of ischemia [2, 3].

The anesthesiologist can perform several clinically simple maneuvers that can further improve CPP in the perioperative period. A non-neutral head position, a tight tracheostomy tie, or ECG lead stretched tightly across the neck can all cause obstruction to jugular venous drainage [25, 26]. Placing the patient into a head-elevated position to augment venous drainage, short-term judicious hyperventilation to produce mild hypocapnia with decreased CBV and lowered ICP, hyperosmolar or hypertonic therapy based on intravascular volume, ventriculostomy drainage, infusion of a vasopressor to maintain cerebral inflow, and institution of muscle

relaxation to increase chest wall compliance (and decrease intrathoracic and consequently CVP) can all improve CPP.

The reference (zero) all blood pressure transducers to the brain is by convention to the ear tragus at the Willis circle level.

Arterial Oxygen Content

Principal determinants of CDO_2 include hemoglobin concentration, oxygen saturation, and cardiac output. The Fick equation describes that oxygen delivery is equal to $1.36 \times CO \times SaO_2 + 0.003 \times PaO_2$. Coexisting disease states, such as pulmonary or cardiac contusion, heart failure, and aspiration, may substantially influence transfusion, ventilation, and cardiovascular support limits. The need for and benefit of transfusion is based on an estimate of probable cardiovascular reserve that is balanced against the current hemoglobin concentration, pulmonary function, and an estimate of the risk and severity of any additional bleeding. Currently, the optimum hemoglobin level for neurocritically ill patients is unknown. Very few studies have looked at the specific population of patients with neurological disease and optimum transfusion goals [27].

Fluid and Electrolyte Management

Fluid replacement during neurosurgical procedures differs from that used during non-CNS surgery. The tight-intercellular junctions found in the CNS and collectively referred to as the blood–brain barrier, with an effective pore size of only 8 Å, are essentially impermeable to sodium, other ions, or proteins. They are, however, freely permeable to water [28]. Hence, to avoid cerebral or spinal cord edema, intravenous solutions that are at least isotonic, i.e., 285 mOsm/kg, should be used. Thus, normal saline, i.e., 0.9 % NaCl, or hypertonic saline (without glucose) is preferred over solutions like lactated Ringer's (LR) that are relatively hypotonic. Clinicians will observe that 0.9 % NaCl solution has a calculated osmolality of 308 mOsm/kg water, suggesting that it is hypertonic, while LR solution has a calculated osmolality of 273 mOsm/kg water. These calculations that are printed on the solution bags represent the simple summation of the component electrolytes. These solutions are actually isotonic and hypotonic, respectively, because the *calculated* osmolalities do not account for solute ion interactions that are responsible for a *measured* osmolality that is actually considerably (approximately 20 mOsm/kg/water) less than the calculated value (Table 41.1). The labeling on the solution bags is always of the calculated and never the measured osmolality.

Solutions with glucose are generally avoided in neurosurgical patients for two reasons. First, although the glucose-containing solution may be iso- or hypertonic (Table 41.1), the glucose is rapidly metabolized. Thus its osmotic contribution of 252 mOsm/kg water is lost and a net free water gain results. This resulting free water will aggravate edema. Secondly, uncontrolled hyperglycemia has been associated with worsened neurologic outcome in patients with focal ischemic injuries. One theory for the mechanism of injury suggests that the presence of glucose increases neuronal metabolism and thereby decreases cellular viability during ischemia. Infants below 5 kg are at some risk for intraoperative hypoglycemia as they have very limited glycogen stores and in anticipation of potential hypoglycemia may therefore receive slightly hypertonic (from the transient glucose contribution intravenous maintenance therapy with D2.5 % normal saline) at our institution. This regimen addresses neuronal dependence on glucose as a metabolic substrate, the limitations of gluconeogenesis, and the high metabolic rate observed in this population. It also yields an isotonic fluid after the glucose is consumed. However, when fluid boluses are needed, 0.9 % NaCl solution, rather than the D2.5 % maintenance normal saline, is used. While the relative risk of hyper- vs hypoglycemia has been well addressed in ICU patient, clear guidelines on blood sugar control in the perioperative period are still unclear but in general maintained below 150 mg/ml in patients with poor glucose tolerance.

Intravascular volume replacement therapy does not differ between neurosurgical and non-CNS procedures. Suggested replacement of blood loss is a 3:1 (isotonic crystalloid to blood loss) volume ratio for 0.9 % saline solutions and 1:1 for colloid, 3 % NaCl, and blood products. Deficits calculated from fasting, insensible losses, urine output, and third-space losses are replaced 1:1. When blood or crystalloid replacement therapy exceeds a few liters (caused by hemorrhage, diabetes insipidus, or a pharmacologically induced

Table 41.1 Calculated osmolarity and osmolality of common intravenous fluids

Fluid	Osmolarity	Osmolality
Water	0	0
D5W	252	259
D5 .2NS	325	321
NS	308	282
LR	273	250
D5LR	525	524
3 % saline	1,027	921
6 % hetastarch	310	307
20 % mannitol	1,098	1,280
Plasma protein fraction	–	261

Reproduced with permission from Gravenstein and Gravenstein [75]
Abbreviations: D5W 5 % dextrose in water, *D5 .2NS* 5 % dextrose in 0.2 normal saline, *D5NS* 5 % dextrose in normal saline, *D5LR* 5 % dextrose in lactated Ringer's solution

diuresis), electrolytes, especially calcium, potassium, and sodium, are followed serially and corrected.

Blood transfusion during neurosurgical procedures may be viewed as somewhat more aggressive compared to other surgery, but the management objective remains to keep the hematocrit in the 24–30 % range. Anemia provokes increased cardiac output and cerebral vasodilatation.

Getting Started

Surgical intervention dictates that the anesthesiologist prepares an anesthetic management plan. This requires that pathophysiology be involved and implications of the surgical approach or procedure be considered.

Stable, controlled cerebral and cardiovascular hemodynamics are the goals prior to surgery. Induction of general anesthesia, laryngoscopy, intubation, positioning, application of the head pinioning apparatus, and eventually skin incision are all profound yet transient insults that make attainment of this goal challenging.

Careful timing and communication to allow coordination of drug effects with stimulation are essential to smoothly navigate the assorted surgical stimuli. Laryngeal or intravenous administration of lidocaine (1–1.5 mg/kg) prior to intubation attenuates endotracheal stimulation. Pinioning of the head may be accomplished in most adults with only a 5–10 % variation of the vital signs. One approach is just prior to pinion placement administer 1–1.5 mg/kg esmolol and 0.5 mg/kg propofol. Thiopental, widely used in the past, is currently not available in the US market.

If the heart rate is lower than preoperative values, the dose of esmolol is reduced. Similarly, if the blood pressure has not recovered from induction, the propofol or STP dose is reduced. Once the drugs have been administered, allowing sufficient time for the heart rate or the blood pressure shows a drug effect before proceeding to pinion the head is recommended. This coordination accomplishes matching the peak surgical stimulus with the peak drug effect, regardless of circulation time. Other regimens, including preemptive local anesthetic infiltration of the pinion sites and a titrated remifentanil infusion, have also been advocated for pinioning [29].

Positioning

Following induction, the task of positioning is undertaken. Both the presence of anesthesia and muscle relaxation during patient transfer and positioning present conditions where the patient is unable to protect himself and is more susceptible to nerve and spinal injury with the removal of muscular tone.

The different positions into which a patient may be placed for surgery are associated with various known risks [30]. The supine position places pressure on the heels and occiput, reduces lumbar lordosis, and may cause flexion of the neck or pressure on peripheral nerves. The ulnar nerve at the elbow is the most frequent place for position-related nerve injury. Injury of the brachial plexus can be caused by aggressive abduction and in the lateral position by a chest roll that slips into the axilla from its intended upper thoracic location. The lateral position further risks lateral flexion of the cervical spine, pressure from the surgical table on the "down arm" and the fibula, threatening the brachial plexus and superficial peroneal nerve. Regardless of body position, one must be cautious with rotation or extension of the head as it may cause venous congestion and is associated with elevated ICP [26, 27]. The prone position requires the abdomen to be properly suspended and knees, testicles, and breasts to be free from pressure.

Special Perioperative Clinical Considerations

Postoperative Visual Loss

Postoperative blindness is a catastrophic complication, the etiology of which is not fully understood. In the prone position the risk of ischemic optic neuropathy (ION) and central retinal artery occlusion (CRAO) is increased. It has been described both when the head has been rested on a foam cushion with a cutout for the eyes, nose, and lips and when the head has been suspended by a pinioning system. Young and old patients are at risk as are patients of any physical status. Pediatric patients (<18 years of age) and the elderly (>84 years) may be more susceptible to visual loss following spine surgery [31]. Speculation is that the pathogenesis involves length of surgery, low arterial perfusion pressure, elevated episcleral venous and intraocular pressures, anemia, embolic events, Wilson frame, and the use of pressor agents [32, 33]. According to the ASA's 2012 Practice Advisory for Perioperative Visual Loss Associated with Spine Surgery, the length of the procedure is also an important risk factor with the vast majority (94 %) of ION occurring in cases lasting longer than 6 h [34]. Therefore patients considered at high risk are ones undergoing prolonged procedures (greater than 6.5 h), in the prone position, with substantial blood loss. Staging procedures is an option to consider but must be balanced against the risks of a staged procedure: possible increase in infection rates, neurological injury, thromboembolism, and cost [34]. Deliberate hypotension has not been shown to definitely increase risk but is not commonly employed anymore. Central (or peripheral) [35] venous pressure monitoring may be considered in high-risk patients and colloid plus crystalloid can be employed to help minimize periorbital edema. There is no set low level of hemoglobin, but commonly a level of 9 is used as a trigger for transfusion [34]. In January 2012, risk factors for ION were assessed in

a large, multicenter, case-controlled study [36]. Eighty patients were from the American Society of Anesthesiologist Postoperative Visual Loss Registry with ION and 315 control subjects without ION after prone spinal surgery [36]. Using multivariate analysis, risk factors found for ION after spinal fusion surgery were male sex (OR 2.53), obesity (OR 2.83), Wilson frame use (OR 4.3), anesthesia duration (OR per 1 h = 1.39), estimated blood loss (OR per 1 l = 1.34), and colloid as percent of non-blood replacement (OR per 5 % = 0.67) [36]. Direct pressure on the eye obviously places patients at increased risk for CRAO (not ION) and should be avoided. In the postoperative period a visual exam should be performed as soon as possible. If any visual disturbance is identified, an ophthalmology consult should be placed and consideration given to optimization of the patient's hemoglobin and hematocrit [35].

Venous Air Embolism

Venous air embolism (VAE) is best known as a danger associated with the sitting position but also is well described in association with the lateral, prone, and supine positions [37, 38]. The incidence of air detectable in neurosurgical cases (craniotomy, cervical laminectomy) using the sitting position is near 45 % but can exceed 70 % [38, 39]. About 20 % of VAE cases in adults produce clinically significant effects, while in children the significant effects are twice that of adults [39]. Because the morbidity of VAE is significant, prevention and monitoring that allow early detection of intravascular gas are key elements of a successful management plan.

Continuous positive end-expiratory pressure (PEEP) in the airway and adequate intravascular volume help to reduce VAE occurrence. Early detection is readily accomplished with continuous precordial Doppler monitoring. Transesophageal echocardiography (TEE) is more sensitive and specific but is used less frequently due to the patient's position and the need for direct observation to make the diagnosis of VAE. Additionally, if fluoroscopy is being used, the probe is not radiolucent. The observance of acute changes, during an otherwise stable anesthetic, of decreased exhaled CO_2, increased central venous, pulmonary artery or decreased systemic blood pressure, or SpO_2 are also suggestive of VAE. Successful management of VAE includes several maneuvers. For a head-elevated operation, the surgeon is notified of the suspicion of a VAE, the wound is flooded with irrigating fluid if possible, the surgical site is lowered to a dependent position relative to the heart, and light manual jugular venous pressure is applied to arrest further entrainment of air and increase cerebral venous pressure. The elevated cerebral venous pressure from manual external jugular compression will in many cases demonstrate a new site of bleeding, i.e., the site of air ingress prior to the artifactual elevation of venous pressure. All myocardial depressants (e.g., any potent IA) are discontinued and ventilation switched to 100 % oxygen. Nitrous oxide poses a special risk because it has low blood solubility and can aggravate a serious condition through its rapid diffusion into an intravascular gas collection, doubling its size in less than 15 min. The effectiveness of repositioning the patient head down with left side down to sequester gas away from the right ventricular outflow track has been challenged and may interfere with other resuscitation maneuvers [40]. In the face of an air-filled right ventricle, pulmonary hypertension, and diminishing systemic pressure, aggressive administration of volume to support preload and intravenous inotropic support and CPR are begun. Aspiration of air or foam from a multi-orifice central venous catheter if present may also be attempted but is unlikely to be therapeutic. As many interventions may be necessary, early calls for assistance are advised.

The addition of positive end-expiratory pressure (PEEP) is not recommended for treatment of VAE even though it may lessen the volume of additional air entrained. Application of PEEP after hemodynamic consequences are observed will increase intrathoracic pressure when air is already in the right heart and pulmonary circulation. Consequently, addition of PEEP will further compromise right ventricular preload just when it is most needed. Furthermore, with an approximately 25 % incidence of patent foramen ovale in adults and a 35 % incidence in children and adolescents, PEEP may increase the likelihood of causing a paradoxical (left heart) air embolus, particularly upon release when right atrial pressure is transiently higher than the left atrial pressure [40–44]. Air traversing from the right atrium across a patent foramen ovale into the left atrium bypasses the lung that normally filters out most air bubbles. Once in the left heart circulation, these air bubbles can travel to the brain or heart, causing acute cerebral or myocardial ischemia, respectively. Clinicians must remain vigilant to this devastating complication, especially if an intraoperative VAE occurred and PEEP was being used. Hemodynamically significant air embolism after intracranial or spinal surgery may also occur upon moving the patient to a supine position [43]. Air sequestered in the originally nondependent vertebral or splanchnic veins gains access to the heart when the patient is returned to a supine position or upon release of PEEP. Spine, paraspinal, and splanchnic venous air simply floats up to the heart level in the supine position.

Mass Lesions

Tumors present unique risks to patients in the perioperative period because of features related to size, vascularity, endocrine activity, and location. Rapidly growing tumors and those causing obstructive hydrocephalus can be associated

with diminished intracranial elastance and elevated ICP. When ICP concerns exist, mild hyperventilation, reverse Trendelenburg position, hyperosmolar therapy, and decompression with CSF diversion are all ICP decreasing options to consider. CPP and oxygenation are defended throughout induction and until the dura has been opened. Once resection of the tumor is underway, bleeding becomes a primary focus of concern. The anesthesiologist will have secured intravenous access proportionate to the presumed risk of bleeding. When the arterial supply and venous drainage of a tumor are known to be high-flow vessels, analogous to those in an arteriovenous malformation, the CPP will be adjusted downward as the tumor is excised. Failure to moderate CPP once the high flow, low resistance diversion is removed risks bleeding from the tumor bed and significant postoperative cerebral hyperemia and edema. The CPP may gradually be returned to normal over 24–72 h as the cerebral vessels' muscularis layer recovers its ability to modulate the vessels' tone, i.e., local cerebral vascular autoregulation normalizes.

Posterior Fossa Surgery

Surgery in the posterior fossa, especially when near the vestibulocochlear nerves, has a greater than 50 % incidence of postoperative nausea associated with it. The nausea alone has been attributed to increasing the length of stay for some patients. Intraoperative anesthetic management can influence the incidence and the severity of postoperative nausea. We use a multifaceted pharmacological approach to reduce postoperative nausea. In addition to administration of intravenous ondansetron (4 mg) 30 min prior to the end of surgery, we typically administer dexamethasone (4 mg) and promethazine (6.25 mg) at the start of the procedure and run an infusion of propofol at 25–50 mcg/kg/min throughout the case. Anecdotally, this technique slightly slows the emergence from anesthesia, but dramatically decreases the incidence and severity of postoperative nausea. Other techniques, such as simply using higher infusion doses of propofol, may also be effective nausea prophylaxis.

Brainstem

Resection of tumors in the brainstem can result in dramatic intraoperative and postoperative findings. When the tumor is located on the floor of the fourth ventricle and areas near the ventral medulla (vasomotor center) are manipulated, hypotension or profound hypertension and bradycardia or even asystole may occur. Surgery in this vicinity may stimulate the dorsal motor nucleus of the vagus or the nucleus ambiguous, reducing cardiac contractility and blocking conduction through the atrioventricular node. To address this possibility, a transthoracic pacer or pacing pulmonary artery catheter (PAC) may be employed in stand-by mode. A transesophageal pacer would not be effective as it paces the atrium and the A-V node will not transmit atrial impulses in this scenario. In the absence of a PAC with pacing capability or transthoracic pacer, pharmacological treatment with anticholinergic medications will also terminate episodes of bradycardia, but this approach eliminates the valuable surgical feedback that the episodes of bradycardia provide.

Finally, this surgery may impair swallowing reflexes by damage of the cranial nerves VIII, IX, and X, a consideration that may affect the decision to extubate the patient early (in the OR) or late (in the ICU).

Transphenoidal Hypophysectomy

Pituitary tumors are often small tumors, microadenomas that are discovered by their unique presenting signs and symptoms. Enlargement of the sella turcica can cause visual disturbances by pressure on the optic chiasm causing bitemporal hemianopsia or extraocular muscle palsies by pressure on cranial nerves III, IV, and VI. Presenting symptoms may also be related to hypersecretion of any one of the pituitary hormones. The anterior pituitary secretes adrenocorticotropic hormone (ACTH), growth hormone (GH), prolactin (PRL), thyroid-stimulating hormone (TSH), luteinizing hormone (LH), follicle-stimulating hormone (FSH), and melanocyte-stimulating hormone (MSH). Prolactin-secreting tumors are the most common secreting tumors but usually do not pose special anesthetic risks to the patient. Patients with long-term GH-secreting tumors may present with acromegaly, placing them at risk for having a difficult airway and postoperative respiratory complications. These patients can have large, immobile mandibles and narrowing of the mandible and the airway below the level of the vocal cords. They also frequently have obstructive sleep apnea. ACTH-secreting tumors cause Cushing disease which again could cause the patient to have a difficult airway and the possibility of hyperglycemia. TSH-secreting tumors can cause hyperthyroidism that should be medically treated and stabilized with antithyroid medication and beta-adrenergic blockade prior to coming to the operating room. Patients may also present with panhypopituitarism, requiring replacement of these hormones. The posterior pituitary secretes antidiuretic hormone (ADH) and deficiency of this is manifested as diabetes insipidus (DI). Intraoperative occurrence of DI must be continually monitored for by the anesthesiologist. Suspicion of this acute syndrome should occur if urine output suddenly greatly increases disproportionately to the intravenous fluids infused and investigated with intraoperative analysis of urine specific gravity, urine osmolality, and serial plasma Na levels. A low urine osmolality (less than 200 mOsm/kg), urine specific

gravity of less than 1.005, and urine Na less than 25 are diagnostic. Unique features of this procedure are predominately in the emergence phase where hypertension and valsalva must be avoided due to the consequences of a hematoma in this region. If a transnasal surgical approach is utilized, rescue mask ventilation should include an oral airway to try to avoid pneumocephalus. A nasal airway should not be placed and nasal CPAP should not be applied postoperatively.

Vascular Neurosurgery

Cerebral Aneurysms

Cerebral aneurysms are abnormal outpouching of cerebral arteries due to weakness of the vessel wall either from congenital causes or secondary to environmental stress and exposures, i.e., hypertension, cigarette smoke, and cocaine. The majority of cerebral aneurysms are found along the circle of Willis at arterial bifurcations. About 90 % are in the anterior circulation and 10 % in the posterior circulations with these mostly occurring at the basilar tip. The rate of rupture is 0.05–6 % per year with resultant subarachnoid hemorrhage. Subarachnoid hemorrhage (SAH) is traditionally graded by multiple systems: the World Federation of Neurological Surgeons Grading System (WFNS), Hunt Hess, and Fisher Grade and modified Fisher Grade scale [45–47]. The WFNS scale (Table 41.2) and the Hunt Hess grading scale (Table 41.3) are clinical scores that assess prognosis. The WFNS is based on the Glasgow coma scale and the score is inversely related to the prognosis. Hunt Hess (HH) 0 is an unruptured aneurysm. HH-1 is asymptomatic or minor headache with no neurological deficits and carries a 2 % mortality. HH-2 is described by headache that can be severe, nuchal rigidity, and possibly a cranial nerve palsy as its only deficit. HH-2 carries a 5 % mortality. HH-3 is described by altered mental status, lethargy, and minor neurological deficits and carries a 15–20 % mortality. HH-4 entails stupor and hemiparesis with a 30–40 % mortality. HH-5 includes patients in deep coma with a motor exam no better than decerebrate and carries a 50–80 % mortality.

Following subarachnoid hemorrhage, vasospasm is a significant risk (20–40 %). The Fisher Grade scale is a radiological grading system to help predict the rate of symptomatic vasospasm. The modified Fisher scale was developed to better correlate with symptomatic vasospasm (Table 41.4). Grade 0 has no SAH or IVH. Grade 1 has thin SAH (less than 1 mm) and no IVH and carries a 24 % risk of vasospasm. Grade 2 has thin SAH with IVH and carries a 33 % risk. Grade 3 has thick SAH without IVH and has a 33 % risk. Grade 4 has thick SAH with IVH and has a 40 % risk of symptomatic vasospasm [47]. Vasospasm most frequently occurs in the first 14 days post bleed with the highest incidence between days 3 and 7. Management strategies have included nimodipine, statins, and magnesium sulfate infusions [48–51]. However, recently the MASH-2 trial demonstrated a lack of efficacy in magnesium infusion to prevent vasospasm [51].

Rebleeding is a feared complication that has a significant mortality associated with it. Early surgical or endovascular securing of the aneurysm within 24–48 h has improved outcome and is the only definitive way to prevent rebleeding. The International Subarachnoid Aneurysm Trial (ISAT) compared neurosurgical clipping or endovascular coiling in 2,143 patients and found low rates of rebleeding in both groups at a mean of 9 years in the long-term follow-up [52]. Before the aneurysm is secured, steps must be taken to prevent rebleeding by maintaining stable transmural vessel pressure. Prior to the operating room, this is accomplished by controlling the systemic arterial blood pressure with

Table 41.2 The World Federation of Neurological Surgeons (WFNS) scale

Grade	GCS	Motor deficit
I	15	Absent
II	13–14	Absent
III	13–14	Present
IV	7–12	Absent/present
V	3–6	Absent/present

Data from Anonymous [45]

Table 41.3 Hunt Hess score

Scale	Symptoms
0	Unruptured aneurysm
1	Asymptomatic or minor headache with no neurological deficits
2	Headache that can be severe, nuchal rigidity, and possibly a cranial nerve palsy
3	Altered mental status, lethargy, and minor neurological deficits
4	Stupor and hemiparesis
5	Deep coma with a motor exam no better than decerebrate

Data from Hunt and Hess [46]

Table 41.4 Fisher and modified Fisher Grade scale

Grade	Radiologic findings	Vasospasm risk (%)
Fisher scale		
1	No SAH or focal thin SAH	21
2	Diffuse thin SAH (<1 mm)	25
3	Thick SAH (>1 mm)	37
4	IVH	31
Modified Fisher		
0	No SAH or IVH	
1	Thin SAH (<1 mm) and no IVH	24
2	Thin SAH (<1 mm) and IVH	33
3	Thick SAH (>1 mm) and no IVH	33
4	Thick SAH (>1 mm) with IVH	40

Data from Frontera et al. [47]

a goal of the systolic blood pressure less than 140 mmHg via short-acting antihypertensives, controlling pain and anxiety, and preventing seizures.

In the operating room, induction of anesthesia must be a slow, controlled event with the goal to limit sympathetic response to laryngoscopy. The rate of rupture on induction is 0.5–2 % and carries a 75 % mortality. Ventilation parameters are initially maintained near normal to prevent altering CBF or ICP and maintaining a stable transmural pressure. For procedures requiring a craniotomy, it is common to gradually induce mild hypocapnia to decrease brain size after craniotomy, but prior to opening of the dura and also to facilitate a better surgical exposure. When the dura is taut, additional hyperosmolar or diuretic (mannitol, furosemide) may be considered, with transitory hyperventilation, and the patient may be placed into a reverse Trendelenburg position. When additional steps are taken, plasma electrolytes, arterial CO_2 end-tidal CO_2 gradient, and urine output are followed closely. Electrolytes lost with diuresis are replaced in the operating room and immediate postoperative phase. If the diuresis is brisk and sustained, and the urine dilute, the possibility of induced diabetes insipidus should be explored by checking the urine specific gravity or urine osmolality and following serial serum sodium.

Blood pressure and heart rate are kept stable during clipping of simple aneurysms that present with a favorable orientation. Not infrequently, however, clipping an aneurysm will first require placement of temporary clips to completely occlude the arterial cerebral circulation upstream of the aneurysm. In anticipation of this possibility and the implications for the regional cerebral ischemic insult, patients have been traditionally cooled via conduction and a water blanket to approximately 34.5 °C. Intraoperative prophylactic cooling, although traditionally employed, has been called into question by the Intraoperative Hypothermia for Aneurysm Surgery Trial (IHAST) [7]. IHAST did not demonstrate any improvement in 3-month neurologic outcome with the use of short-duration intraoperative hypothermia during craniotomy for good-grade patients, WFNS grades I to III, with aneurysmal SAH [7]. Preoperative discussion with the neurosurgeon on temperature management is appropriate in light of these conflicting data. Temperature can be measured from esophageal, rectal, or bladder probes, although the brain is likely to be cooler than measured core temperature because the surgical site is exposed to ambient room temperature. Blood pressure is maintained by a vasopressor (commonly phenylephrine because of its preference for peripheral over intracranial vasoconstriction), while either barbiturate or propofol is administered to near 90 % burst suppression monitored by EEG. No compelling clinical data currently support the use of propofol for neuroprotection. One study which compared the use of propofol and isoflurane in 20 patients during coronary artery bypass grafting showed no difference in neuropsychological outcomes between the two groups [53]. The hypnotic infusions are titrated to an EEG pattern showing at least 90 % burst suppression. A deeper coma with barbiturates, i.e., isoelectric EEG (100 % burst suppression), has not been demonstrated to convey any additional cerebral protection and may result in diminished hemodynamic stability, a slowed emergence and extended respiratory support. The limit to which perfusion pressure during temporary clip times can be augmented is determined by the cardiovascular reserve (i.e., onset of myocardial ischemia). Usually, a 20 % increase above baseline blood pressure is sufficient additional pressure support to protect normal cerebral function. However, if myocardial ischemia develops, efforts must be made to improve myocardial oxygen delivery/consumption balance. When a temporary or permanent clip application produces an alteration in a monitored evoked potential, the mean arterial pressure is augmented further, usually with an alpha agonist intravenous infusion. When placement of the clip occludes one or more lenticulostriate perforating vessels, the mean arterial pressure necessary to perfuse ischemic and vasodilated tissues through collateral vessels is augmented to 10–30 % above baseline [54]. Once the perfusion pressure has been adjusted, a gradual recovery of evoked potential waves is often observed (Fig. 41.2).

Vessel rupture and hemorrhage are major complications of neurovascular surgery. Ideally, the patient is rapidly cooled to a mild hypothermia goal (35 °C), and a hypnotic agent (in general propofol if hemodynamically tolerated) is administered before a temporary clip is applied and the bleeding halted. When the aneurysm rupture occurs without a temporary clip in place, acute and profound hypotension is induced in the attempt to decrease bleeding rate. Placement of the temporary clip defines the start of a regional ischemic event. The temperature of the brain and the total ischemic time will predict the likelihood of cerebral infarction. The longest ischemic time usually tolerated by the brain (approximately 60 min) is achieved in controlled situations when the patient is cooled to 18 °C and while on low-flow cardiopulmonary bypass or induced total circulatory arrest. Severe hypothermia is predictably associated with a coagulopathy and should be reversed before emerging from anesthesia.

Arteriovenous Malformations

Arteriovenous malformations (AVM) are abnormal collection of arteries and veins that lack the capillary system and therefore have shunting between the arterial and venous system. This resulting high-pressure flow to the venous system becomes at risk for rupture secondary to fibromuscular thickening and weakened elastic lamina [55]. Hemorrhage is the most common presenting symptom of AVMs. There are multiple options for treating AVMs. The Spetzler-Martin scale is used to guide therapy and scores the AVMs on the basis of surgical outcome. It looks at three characteristics: the maximum diameter, its location either within or outside of eloquent

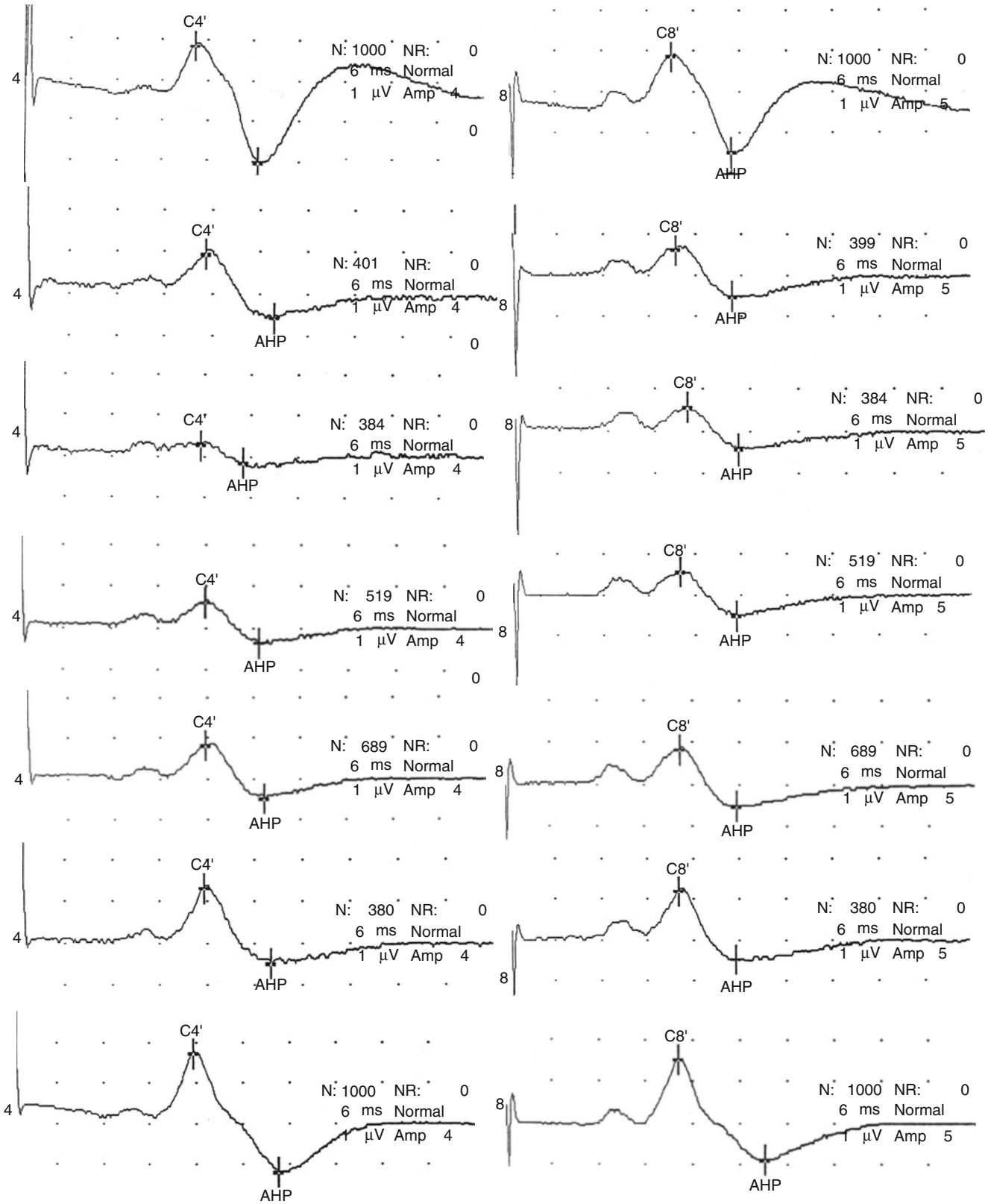

Fig. 41.2 Once the perfusion pressure has been adjusted, a gradual recovery of evoked potential waves is observed. Leads 4 and 8 follow progression of C4' and C8' (Reproduced with permission from Gravenstein and Gravenstein [75])

cortex, and the presence or absence of deep venous drainage. According to the 2001 American Stroke Association guidelines, grades 1 and 2 should be considered for surgical resection, grade 3 for endovascular treatment, and grades 4 and 5 for conservative management [56].

Anesthetic management of the excision or embolization of AVMs is similar to that of a vascular tumor. The need for intravenous access will be substantial secondary to the risk of significant bleeding during the surgical resection. Pharmacological coma to EEG burst suppression is not induced in this disease because bleeding is not typically amenable to temporary clip placement. Patients are kept normothermic to avoid impairment of coagulation or risks of hypothermia (arrhythmias, shivering, slowed drug metabolism, left-shifted oxygen-hemoglobin dissociation curve). As the AVM is successfully removed, local CBF is expected to increase due to cerebral vasodilatation in areas that were previously chronically hypoperfused [57]. The increase in CBF can cause cerebral edema and hemorrhage. Hence, CPP will be lowered to guard against hyperemia and cerebral edema. Strict avoidance of hypertension is a continued goal for at least 24 h postoperatively. Staged procedures are another option to attempt to reduce incidence of post AVM resolution hyperemic hemorrhage from cerebral hyperperfusion.

Carotid Endarterectomy

Carotid endarterectomy (CAE) differs from intracranial vascular surgery in its association with coronary and peripheral vascular disease and proximity to the carotid sinus. If no temporary arterial shunt is applied by the surgeon, the mean arterial pressure is augmented during the time of carotid artery occlusion, just as it is for temporary clip application during cerebral aneurysm surgery. Blood pressure and heart rate can be erratic when the carotid sinus is surgically manipulated. This intraoperative and postoperative hemodynamic lability may not be improved with the use of local anesthetic field blocks of the carotid sinus [58, 59]. When the surgical procedure during carotid endarterectomy causes denervation of the carotid body, an altered chemoreceptor function and a diminished hypoxic response may result [60]. The loss of the hypoxic response is a particularly relevant postoperative consideration in the patient with a prior CEA who may now have bilateral carotid body chemoreceptor dysfunction and postoperative respiratory status should be closely monitored in the recovery room or intensive care unit before discharge to the neurosurgical ward.

Blood pressure and blood flow are reduced prior to removal of the carotid artery clamps to minimize hyperemia and the risk of postsurgical bleeding. Arterial hemorrhage into the surgical site can be provoked or worsened during emergence by perioperative coughing, bucking, and uncontrolled hypertension. Hence, the target blood pressure in this phase of the anesthetic is at the lower end of the patient's normal range. Emergence from anesthesia often utilizes narcotics for their combined analgesic, sedative, and cough suppressant effects. Intravenous lidocaine to reduce tracheal reactivity to the endotracheal tube during extubation may reduce bucking and coughing.

Neuromonitoring during neurovascular surgery aids in detection of ischemic strokes. Strokes may originate from embolic sources, as from plaque loosened during carotid endarterectomy or shunt placement and during the time of temporary clip placement or by misapplication or rotation of permanent clips during surgery for intracranial aneurysm. In recognition of these risks, CNS monitoring to alert the surgeon and anesthesia provider of such events is a priority. The mental status of awake patients who are operated for carotid endarterectomy with field block anesthesia serves as neuromonitors for cerebral ischemia. Patients are asked to verbally respond to questions or complete simple tasks such as squeezing a rubber toy, so their compliance with the request is unmistakable. When patients are under general anesthesia, CNS monitoring is accomplished with the use of electroencephalography (EEG) and/or continuous somatosensory evoked potential (SSEP) monitoring. Transcranial electrical motor- and visual evoked potential (TceMEP and VEP, respectively) and facial nerve monitoring are other modalities that have been utilized intraoperatively and described in detail elsewhere in the book. TceMEP and VEP are difficult to obtain as they are exquisitely sensitive to conventional anesthetics and can limit the surgical field because of the physical setup. When any of these monitoring modalities are used, the anesthetic technique is biased towards using reduced concentration of volatile agents and increased narcotic infusions, preferably shorter acting agents.

Endovascular

Endovascular treatment of cerebral vascular disease is minimally invasive and entails the interventional radiological access to the vessel of interest via intra-arterial catheters. This is usually accomplished by accessing the femoral artery and then the carotid or vertebral arteries, depending on the target vessel of interest. The anesthetic considerations specific to the disease state are the same for endovascular treatment as for open craniotomy. Anesthesia can be accomplished either through intravenous sedation with continual mental status monitoring or through general anesthesia. Endovascular treatment of aneurysms entails deploying of coils into the aneurysm to occlude flow. Depending on the shape of the aneurysm, additional therapeutic maneuvers such as placement of stents or temporary balloon angioplasty may be required. The manipulation of these vessels poses the possible risk of rupture of the aneurysm or stroke from embolic or occlusive complications. The anesthesiologist must be prepared for these events and have a ready armamentarium to rapidly manipulate cerebral blood flow and perfusion. Patients with subarachnoid hemorrhage may also present for angioplasty for vasospasm. If the aneurysm is already secured, hypertensive therapy will most likely be employed and should be continued throughout the angioplasty procedure.

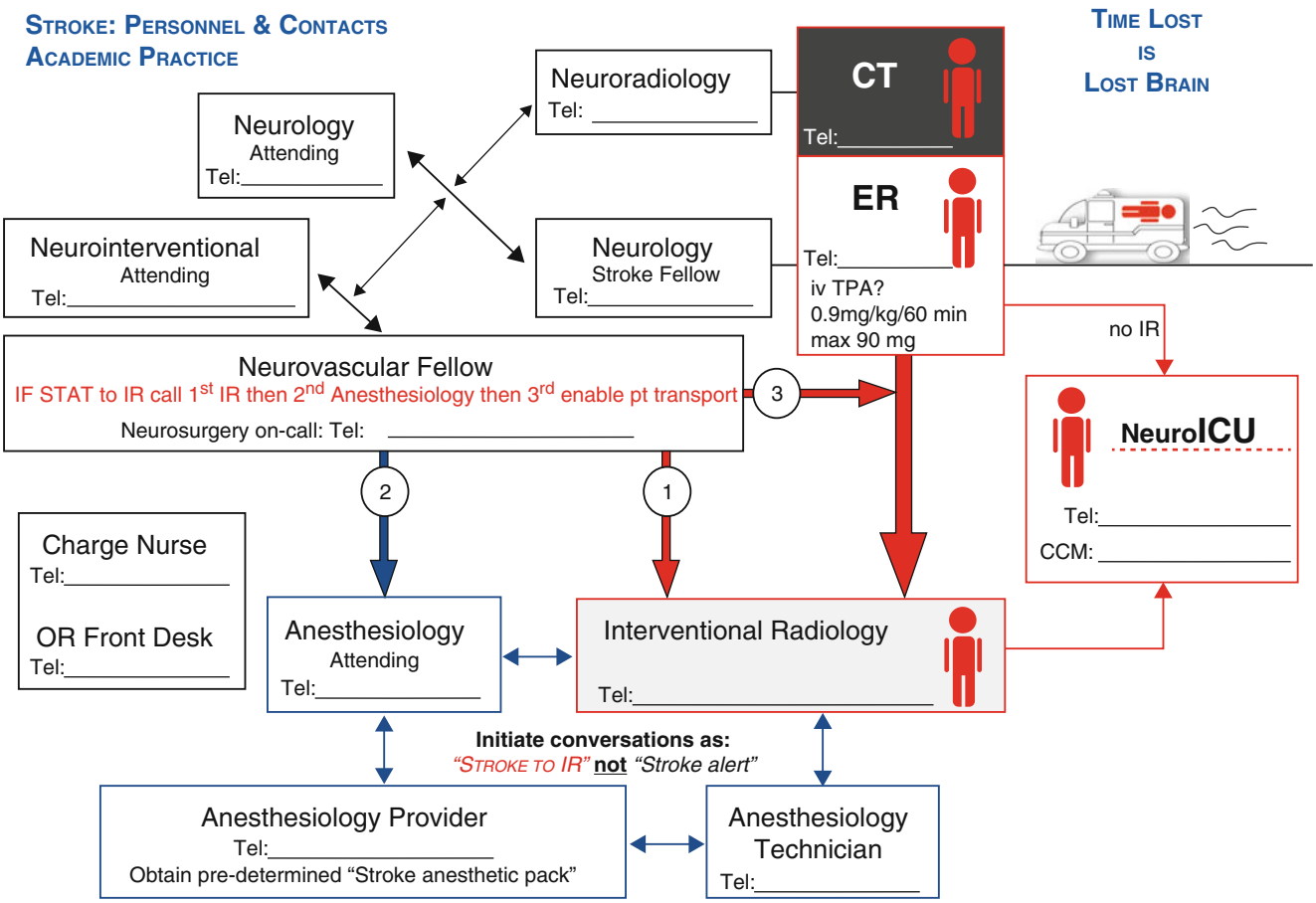

Fig. 41.3 Example protocol for activating interventional suite for stroke patient

Alleviation of the vasospasm can be attempted through balloon angioplasty or through direct intra-arterial injection of vasodilators, usually calcium channel blockers such as verapamil and nicardipine. Systemic circulation of these medications can occur with resultant profound hypotension.

Stroke

Patients suffering acute stroke are now undergoing more and more interventional procedures in attempts to reestablish cerebral blood flow. Stroke affects more than 795,000 people annually in the USA with 85 % of these being ischemic in origin and 15 % hemorrhagic. Over the past decade, there have been vast improvements in treatment of acute stroke. In 1995, IV tissue plasminogen activator (tPA) was first shown to improve outcomes if given within 3 h of symptom onset [61]. In 1999, the PROACT II study showed that intra-arterial administration of pro-urokinase within 6 h of onset improved clinical outcomes at 90 days but with an increased risk of intracranial hemorrhage (10 % vs. 2 %) [62]. In 2005, the use of the MERCI device, a clot retrieval catheter to allow recanalization of the cerebral arterial vessels, was approved for up to 8 h post onset of stroke symptoms [63]. In 2007, the Interventional Management of Stroke (IMS) II study showed decreased mortality at 3 months with a combined intravenous and intra-arterial tPA approach [64]. In 2008, Hacke and colleagues reported that alteplase could be used from 3 to 4.5 h with improved clinical outcomes at 90 days and no increase in mortality [65]. At the time of this writing, ongoing trials are looking at the safety and efficacy of new and improved devices to mechanically disrupt and remove the clot.

Currently, the use of intravenous tPA may be considered within 4.5 h from the onset of symptoms of an ischemic stroke. At our institution, an intravascular recanalization approach is performed if the patient does not improve clinically with intravenous tPA but shows a CT angiography and perfusion scans with less than one third of the vessel's territory compromised. In this case, the presence of a large amount of salvageable brain tissue penumbra, a brain territory that is ischemic but not yet infarcted, is assumed. When cerebral blood flow falls below 20 ml/100 g/min, neuronal function declines. CBF from 15–20 ml/100 g/min causes decreased synaptic activity but can still be salvaged. When CBF falls below 15 ml/100 g/min, irreversible ischemic injury occurs and below 6 ml/100 g/min neuronal cell membrane failure occurs. Because "Time is Brain," well-organized protocols need to be in place at each institution to activate all the necessary personnel if a patient is to go to the interventional suite (example protocol in Fig. 41.3).

Anesthetic management of the acute stroke patient requires fast preparation. If the patient has received IV tPA, strict blood pressure control (systolic pressures less than 185 mmHg and diastolic pressure less than 110 mmHg) needs to occur. Permissive hypertension to this point is allowed in order to improve perfusion through collaterals.

Anesthetizing a patient who has recovered function from a prior stroke either partially or wholly is accompanied by the risk of the patient and surgeon potentially experiencing the phenomenon of the patient awakening from anesthesia with some or all of the deficits that occurred at the time of the original CVA. This recapitulation phenomenon has been termed "differential awakening" of the brain and related to patients as a "reliving" of their stroke [66]. It is postulated that the patient with a remote stroke has regained function by learning to utilize new pathways that require vastly more synapses than the original pathway. The normal brain recovers function with a certain low partial pressure of anesthetic agent along neurons and at receptors. Another possible mechanism explaining the phenomenon of differential awakening is that anesthetic agents are eliminated from injured or ischemic brain more slowly than normal brain, perhaps because of differential blood flow. Finally, it has been suggested that the cumulative amounts of the same low partial pressure anesthetic along the much longer neuronal pathways and vastly larger number of receptors used to overcome the CVA deficit are sufficient to keep those pathways nonfunctional. In general these residual anesthetic effects dissipate within hours of discontinuation of anesthesia as the partial pressures and concentrations of the agents used continue to diminish. However, if memory within the pathways has also been disturbed, full recovery to preoperative baseline may require days, weeks, and possibly an even longer period of rehabilitation, but full recovery is eventually expected.

The patient with preoperative paralysis of more than several days, whether from central or peripheral neural injury, poses other anesthetic and postoperative concerns. The paralyzed patient may have up-regulated neuromuscular junctions to a degree that administration of a depolarizing muscle relaxant, i.e., succinylcholine can precipitate an acute hyperkalemic event. Monitoring of neuromuscular blockade is also complicated when attempted on an affected limb of a patient with a prior CVA. Electrically stimulating the ulnar or posterior tibial nerve in an affected limb, where there is an increased neuromuscular junction density, produces exaggerated responses compared with the normal limb. Thus, well-intentioned titration of neuromuscular blocking agent to monitored twitches on a paretic or plegic limb can lead to significant overdosage of neuromuscular blocker and subsequently an inability to reverse the patient for extubation. Simply monitoring on a non-affected side or following the activity of the levator palpebrae muscle with facial nerve stimulation or performance of a head lift for greater than 5 s will confirm suitable strength is recovered for extubation.

Spine Surgery

Spinal cord dynamics are similar to those of the brain. The cord responds to anesthetic agents, ventilation parameters, temperature, perfusion pressure, and ischemia in essentially the same fashion as the brain. Perioperative edema of the spinal cord presents with similar concerns of ischemia and infarction. The decompression, instrumentation, distraction, and fusion of vertebral elements are unique to surgery of the spine. Bleeding or the entrainment of air results most commonly from violation of the epidural veins. Intraoperative neurological injury, unless catastrophic, is not hemodynamically apparent but can usually be detected with ideally continuous neurological monitoring (e.g., SEP or MEP) or less sensitively with an intraoperative wake-up test. In at-risk patients, these monitoring and exam modalities do not, unfortunately, guarantee functional outcomes. They demonstrate pathway integrity only of the monitored tracts and only during the period they are interrogated. The monitors are usually removed at the conclusion of the case, often prior to turning the patient from a prone position to a supine position or upon emergence from anesthesia. During these events and this unmonitored time, hardware or fusion grafts can dislocate and ischemia or edema of the spinal cord may still develop.

Prolonged surgery in the prone position may produce generalized edema of the face and neck. This edema will be increased by large volume fluid administration, trauma (from a difficult intubation), and the head-down position as might be used for cervical spine surgery or as a result of positioning on a Wilson frame. Patients demonstrating significant facial and airway swelling must be carefully evaluated to establish their appropriateness for extubation, and a combined plan with the neurointensivist should be formulated if necessary. Generally, once the patient is supine and the head elevated, edema will resolve gradually over the same time course as it developed. Therefore, patients might require a planned period of postoperative mechanical ventilation.

In addition to operative management of acute spinal cord injury, medical management with methylprednisolone has been a common but controversial occurrence. This is based off of the National Acute Spinal Cord Injury Studies: NASCIS-2 and NASCIS-3, which recommended giving methylprednisolone 30 mg/kg bolus followed by an infusion of 5.4 mg/kg/h for 24 h if initial bolus was given within 3 h or for 48 h if initial bolus was given between 3 and 8 h post injury [67, 68]. While these studies showed an improvement in motor function at 6 weeks and 6 months, they lost statistical significance at 1 year and a higher trend to postoperative complication rates in the methylprednisolone arm. Specifically in the NASCIS-3 trial, the 48 h methylprednisolone group had statistically significant greater incidence of severe sepsis and severe pneumonia [68]. Evidence for complications, pneumonia, urinary tract infections, wound infections, and GI bleeding from the steroids has been more consistently

shown [69, 70]. These trials have been greatly criticized since their publications and their results have not been duplicated. Currently, methylprednisolone therapy is not recommended for routine use in acute spinal cord injury, and its use for reasons different than spinal cord ischemia should be weighed against the possibilities of complications [70].

A significant complication after spine surgery is a surgical site infection. In addition to complying with SCIP measures of antibiotic timing and normothermia, there is compelling evidence that using an FIO_2 of at least 50 % during surgery reduces the likelihood of postoperative infection in spine surgery patients [71].

Functional Operations

Functional or awake craniotomies are undertaken when the area being operated on is in the vicinity of eloquent cerebral cortex such as language, motor, sensory, and vision. It was first used for surgical resection of epileptic foci then extended to the placement of deep brain stimulators.

Awake procedures require special planning and vigilance on the part of the anesthesiologist to ensure patient safety and comfort. Preoperative evaluation and discussion with the patient is key. The patient's airway should be assessed with consideration that a patient with a difficult airway, obstructive sleep apnea, or features that put him at risk for acute obstruction under sedation can be considered relative contraindications. However, the only true contraindication is an uncooperative patient. This means that all patients need to be preemptively assessed for their level of maturity and ability to handle stressful situations. Extensive counseling and explanation of the operation is necessary to help preparing the patient. Positioning of the patient is also very important. The surgeon, anesthesiologist, and neurologist must all be able to have access to full verbal and visual communication with the patient. The patient is placed in pinions and his face visible and free of drapes not only for his comfort, to assess the level of consciousness and for neurologic and speech evaluation [72]. The patient's head should be straight with minimization of jugular compression that could raise the venous pressure. Reverse Trendelenburg positioning may also be necessary to aid in venous drainage. Airway equipment should be immediately available for possible need of emergent intubation if the patient becomes uncooperative and unresponsive and has intractable seizures or other reasons resulting in loss of airway protection. Intraoperative airway complications have been documented to occur in 1.6–2.2 % of patients [72]. An asleep-awake-asleep technique has also been described during which the patient is fully anesthetized for initial skin incision and craniotomy and is allowed to awaken and then anesthetized for closure [73]. Other potential complications or problems also may occur that need to have advanced preparation. Seizure is an obvious possible occurrence with an incidence of 0.8–4.5 % [72]. This may be due to the patient's underlying epilepsy or more commonly cortical stimulation. In the patient in pinions, seizure could pose a serious threat of facial injury. In general the seizure will terminate when the neurosurgeon stops manipulation of the field [73]. If it does not, propofol can be used in small increasing increments. Nausea is common and can be reduced with dexamethasone 4–8 mg iv, ondansetron, and low-dose propofol.

Sedation prior to mapping can be accomplished by small increments of fentanyl 25–100 μg, propofol, or remifentanil infusions. Boluses of propofol and esmolol are commonly given during pinion placement. After cortical mapping has taken place, dexmedetomidine infusion can also be used along with a remifentanil infusion [73].

Extubation Logistics in the OR

A clinical assessment of the airway and patient, taken in the context of the course of surgery, the anesthetic agents used, re-dosing intervals for drugs, and reversal or antagonizing agents given, allows for a determination of the patient's suitability for extubation. When a patient is left intubated, it is either at the surgeon's request or in situations where the clinical assessment suggests extubation would be unsafe to allow adequate airway protection. The determination for leaving a patient intubated will also be influenced by a number of intraoperative scenarios and should be communicated from the operating room to the neurointensivist for a combined plan.

Several neurosurgical procedures need special consideration on extubation of the trachea. It may be difficult to predictably emerge from anesthesia a patient following resection of tumor using a surgical approach necessitating prolonged frontal lobe retraction. These patients remain intubated until they are able to follow commands or demonstrate the ability to protect their own airway with a cough, swallow, or gag reflex. Postoperatively, patients with surgical injury or edema to structures within the brainstem may also fail to "emerge from anesthesia" and not regain consciousness. When the reticular activating system, which resides in the upper pons and mesencephalon, has been disrupted, consciousness can be affected. Similarly, spontaneous breathing can be impaired with injury to respiration-related cells located low in the medulla, parabrachial nucleus injury in the mid to caudal pons, as well as injury to suprapontine anatomy, most notably the limbic structures. Surgery in the posterior fossa may lead to dysfunction of cranial nerves VII, IX, and X. Impaired function of these cranial nerves can lead to difficulty swallowing, loss of a gag reflex, airway obstruction from vocal cord paresis, and increased risk of aspiration. When no evidence of a gag, coughing, or swallowing can be elicited by manipulation of the endotracheal tube or with oropharyngeal suctioning, the patient will be transported to the ICU intubated.

Conclusion of any operation on the cervical spine or surgery requiring a prone position always includes a reassessment of the airway for a determination of the suitability for extubation. Oropharyngeal or laryngeal edema and macroglossia are recognized complications [74]. Prolonged or vigorous lateral retraction of the trachea as can accompany anterior cervical spine approaches can provoke edema that may compromise the airway. The decision to extubate in the presence of upper airway edema, whether from local retractor-associated trauma, neck flexion during posterior cervical approach, or massive volume resuscitation, is aided by performing a few tests: the patient should be strong—with objectively verified recovery of neuromuscular function—and warm and follow commands appropriately so that the only variable is the adequacy of the airway itself. In this situation, a positive pressure leak test will give a gauge of the airway edema present. It is performed by deflating the endotracheal tube cuff and then slowly increasing the airway pressure until gas leaking around the cuff is heard at the mouth. When the threshold leak pressure is under 20 cmH_2O, the airway can typically be safely extubated and maintained by the patient. If one is contemplating extubation of a patient with difficulty placing the endotracheal tube, a more cautious approach is taken by also after oropharyngeal suctioning performing a negative pressure leak test. This test is performed by deflating the endotracheal tube cuff, disconnecting the breathing circuit from the endotracheal tube, and instructing the patient (after an exhalation) to breathe in while one occludes the endotracheal tube. With a stethoscope placed over the trachea, it is readily determined if the patient is successfully ventilating around the occluded endotracheal tube. If air movement is auscultated, this demonstrates that the patient is able to stent open his airway despite the negative pressure in the oropharynx and the space occupying endotracheal tube present. Because posttraumatic swelling may continue to increase for several hours after surgery and the level of stimulation will decrease following removal of the tracheal tube and the addition of any analgesics or sedatives, we will on occasion extubate these patients over an exchange catheter. The tube changer is taped in place just like an endotracheal tube and is generally well tolerated and can be removed several hours later or, if necessary, used to jet ventilate or re-intubate in the event worsening edema leads to evidence of obstruction.

Summary

Anesthetic agents and technique may significantly affect cerebral oxygen consumption ($CMRO_2$), cerebral oxygen delivery (CDO_2), cerebral blood flow (CBF), intracranial tissue volume, intracranial pressure (ICP), arterial oxygen content (CaO_2), and the autoregulation of CBF.

Potent inhalational agents (PIAs), narcotics, and commonly used hypnotic agents all lower cellular metabolism (i.e.,$CMRO_2$) by decreasing cortical neuron electrical activity. All PIAs attenuate cerebral autoregulation and uncouple $CMRO_2$ from cerebrovascular resistance (CVR) and CBF.

Cooling the brain or spinal cord reduces $CMRO_2$ by 5–7 % per degree Celsius decrease.

While the effectiveness of hypothermia for neuroprotection in "global" ischemia continues to accumulate, evidence suggests that there may be a difference for "focal" ischemia. The longest "safe" ischemic time (approximately 60 min) is achieved in controlled situations when the patient is slowly cooled to 18 °C and placed on low-flow cardiopulmonary bypass or circulatory arrest.

The ICP follows the net decrease of CBV downward. In the scenario where a more extensive cerebral insult has occurred and autoregulation is compromised, ICP changes will follow MAP and CBV changes.

As $PaCO_2$ decreases, CBF is reduced and consequently CBV and ICP are also lowered. For $PaCO_2$ levels between 20 and 70 mmHg for each 1 mmHg change, there is an approximately 4 % change in cerebral blood flow. Profound hypoxemia ($PaO_2 < 50$ mmHg) causes cerebral vasodilation and an exponential rise in cerebral blood flow, blood volume, and ICP. This is thought to be due to a decrease in pH secondary to lactic acidosis. Cerebral hyperemia may persist for up to 30 min following extubation.

The brain can recover from brief episodes of ischemia but has little chance of return to normal following traumatic herniation. Therefore, when the potential for mechanical brain injury via herniation exists, aggressive acute hyperventilation ($PaCO_2$: 20–25 mmHg) can in conjunction with other measures decrease cerebral volume even though it may risk cerebral ischemia.

The tight-intercellular junctions found in the CNS and collectively referred to as the blood–brain barrier, with an effective pore size of only 8 Å, are essentially impermeable to sodium, other ions, or proteins. They are however freely permeable to water. To avoid cerebral or spinal cord edema, solutions that are isotonic, i.e., 285 mOsm/kg should be used.

Suggested replacement of blood loss is a 3:1 (isotonic crystalloid to blood loss) volume ratio for 0.9 % saline solutions and 1:1 for colloid, 3 % NaCl, and blood products. Deficits calculated from fasting, insensible losses, urine output, and third-space losses are replaced 1:1.

Careful timing and communication to allow coordination of drug effects with stimulation are essential to smoothly navigate the assorted surgical stimuli.

Pediatric patients (<18 years of age) and the elderly (>84 years) may be more susceptible to visual loss following spine surgery. Speculation is that the pathogenesis involves length of surgery, low arterial perfusion pressure,

elevated episcleral venous and intraocular pressures, anemia, embolic events, Wilson frame, and the use of pressor agents.

The incidence of air detectable in neurosurgical cases (craniotomy, cervical laminectomy) using the sitting position is near 45 % but can exceed 70 %.

When ICP concerns exist, mild hyperventilation, reverse Trendelenburg position, diuretics, and decompression with CSF diversion are all ICP decreasing options to consider.

Surgery in the posterior fossa, especially when near the vestibulocochlear nerves, has a greater than 50 % incidence of postoperative nausea. When the tumor is located on the floor of the fourth ventricle and areas near the ventral medulla (vasomotor center) are manipulated, hypotension or profound hypertension and bradycardia or even asystole may occur. Patients with GH-secreting tumors will eventually develop acromegaly, placing them at risk for having a difficult airway and postoperative respiratory complications.

Before the aneurysm is secured, steps must be taken to prevent rebleeding by maintaining stable transmural vessel pressure. Prior to the operating room, this is accomplished by controlling the systemic arterial blood pressure with a goal of the systolic blood pressure less than 140 mmHg via short-acting antihypertensives, controlling pain and anxiety, and preventing seizures.

Hypothermia has been called into question by the Intraoperative Hypothermia for Aneurysm Surgery Trial (IHAST).

"Time is Brain" protocols at each institution need to be in place to activate all the necessary personnel if a patient is to go to the interventional suite.

Spinal cord dynamics are similar to those of the brain. The cord responds to anesthetic agents, ventilation parameters, temperature, perfusion pressure, and ischemia in essentially the same fashion as the brain.

Methylprednisolone therapy is now not recommended for routine use in acute spinal cord injury and its use should be weighed against the possibilities of complications.

Awake procedures require special planning and vigilance on the part of the anesthesiologist to ensure patient safety and comfort.

Conclusion

A careful review and understanding of the intraoperative anesthetic management and surgical events will assist in forming a perioperative plan and differential diagnoses for clinical findings observed in the intensive care unit. Communication among neurosurgeon, anesthesiologist, and neurointensivist is essential to better guide patients' postoperative care and the recovery expectations of family and healthcare staff, to everyone's benefit.

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