Lesson S22: Preanesthetic Assessment of the Patient with an Intracranial Aneurysm

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REVIEW DATE: December, 2011

Read this article, reflect on the information presented, then go online and complete the lesson post-test and course evaluation before the termination date below. (CME credit is not valid past this date.) You must achieve a score of 80% or better to earn CME credit.

TIME TO COMPLETE ACTIVITY: 2 hours
RELEASE DATE: January 1, 2012
TERMINATION DATE: January 31, 2013

TARGET AUDIENCE: Anesthesiologists

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Needs statement

Choice of anesthesia for neurosurgical procedures under varying circumstances remains controversial. The subject was recently addressed by expert panels at national meetings including the annual meeting of the American Society of Anesthesiologists and the New York State Society of Anesthesiologists in 2011. Clinical anesthesiologists must be knowledgeable of current practices and will benefit from a review of current literature referable to this topic with specific attention to addressing the anesthetic needs of the patient with an intracranial aneurysm.
Learning Objectives

At the end of this activity, the participant should be able to:

1. Discuss the intracranial effects of different anesthetic agents.
2. Explain the importance of understanding the neurosurgical procedure.
3. Describe how newer monitoring strategies are used intraoperatively.
5. Specify criteria for anesthetic care in off-site procedures.
6. Anticipate anesthetic complications associated with neurosurgical procedures.
7. List common neurosurgical procedures.
8. Differentiate the intracranial effects of hypo- and hyper-ventilation.
9. Interpret the findings of the IHAST study.
10. Identify methods of improving brain survival.

Case History

A 36 year old woman reports the sudden onset of severe headache without any associated neurologic abnormalities or loss of consciousness. CT scan findings reveal a posterior cerebral artery aneurysm. She has a family history of hypertension and has been a smoker for about 20 years. Her blood pressure on admission was 160/95 with a marked tachycardia. Electrolytes were normal except for a blood glucose level of 150mg/dl. The interventional radiologist proposed coiling the aneurysm and the patient was emergently prepared for surgery.

Introduction

The anesthetic plan for the neurosurgical patient must consider the specific pathology, the needs of both the patient and the surgeon, and the site where interventions will be performed. An anesthetic plan can range from moderate sedation to general anesthesia and can potentially include awake and unconscious states during the same procedure. The choice of monitoring procedures depends on the surgical procedure and the condition of the patient. Flexibility of anesthetic care is very important as interventions are performed outside of the traditional setting of the operating room and more pathologies become amenable to treatment. In addition, anesthetic drugs and techniques may alter intracranial dynamics and thus outcome.

General Requirements

There are several goals for anesthetic care of patients undergoing procedures involving the neurological system. (Table 1) Intracranial pressure and cerebral blood flow should be maintained as close to normal as possible. Systemic hemodynamic stability is essential and the anesthesiologist should aim for prompt awakening. Normoglycemia is very important throughout the perioperative period.
Table 1: Neurologic pathologies requiring accommodation in anesthetic techniques

<table>
<thead>
<tr>
<th>Aneurysm</th>
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<tbody>
<tr>
<td>Spinal column pathology</td>
</tr>
<tr>
<td>Tumor</td>
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<tr>
<td>Trauma Congenital malformation Stroke</td>
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<tr>
<td>Deep brain stimulation</td>
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Intracranial pressure

Intracranial pressure is normally around 10-15 mmHg and becomes elevated whenever any increase in brain bulk, cerebrospinal fluid or intracranial blood occurs. Most times, the choice of treatment depends on the cause of the change in pressure. For example, a subdural or epidural hematoma requires immediate surgical drainage while steroids and/or diuretic therapy are appropriate to reduce the edema surrounding a tumor. The weight of the brain has been shown to increase by about 82g after traumatic brain injury. The specific gravity of a contused brain tissue increases. Intracranial hypertension is a result of cytotoxic edema rather than a simple breakdown of the blood brain barrier, indicating multifactorial causes of intracranial hypertension. It is essential to maintain stability since small increases in intracranial pressure can cause cerebral ischemia, herniation and neurogenic pulmonary edema.

Intracranial dynamics and cerebral blood flow are critically affected by choice of anesthetic and ancillary agents; ventilatory maneuvers; or alterations in fluid management. Cerebral blood flow is affected by manipulation of arterial carbon dioxide (paCO2) levels in a linear manner. Hyperventilation and hypopcapnia were once accepted methods of decreasing intracranial pressure, but the decrease is achieved at the expense of cerebral oxygenation. One study looked at continuous monitoring of cerebral tissue oxygenation and regional blood flow (rCBF) after severe brain injury and determined that with a 20% decrease in paCO2 (40mmHg-32mmHg), rCBF decreased from 30-25ml/100gm/min (p<0.001) and the partial pressure of O2 decreased from 20-15mmHg (p<0.001). No changes in blood pressure or heart rate were observed. Thus hypocapnia may be harmful and should be restricted to emergent management of life threatening intracranial hypertension. Also, hyperventilation may cause acute lung injury. Analysis of data collected by IMPACT (International Mission for Prognosis and Clinical Trials) indicated that hypoxia results in poor neurologic outcome. While steroids may improve lung function, they may worsen neurologic outcome, possibly as a result of a hyperglycemic effect. Ventilation strategies should aim to maintain oxygenation by the judicious use of small amount of positive end expiratory pressure (PEEP) with low tidal volumes and relatively faster respiratory rates with the patient in a head up position.

Intracranial Effects of Anesthetic Agents

The intracranial effects of anesthetic agents have long been studied. Halothane was shown to cause marked cerebral vasodilation, an effect not so obvious in the later halogenated agents. (Table 2)
Nitrous oxide increases both metabolic rate of oxygen utilization and blood flow and has limited use in intracranial surgery, although it may still be used in uncomplicated spinal surgery. Sevoflurane has been shown to be less vasoactive than other inhaled agents and to cause less cerebral vasodilation than isoflurane or desflurane at the same anesthetic depth. Little or no changes in rCBF and electrocorticography were observed. In a comparison study with propofol in patients with brain tumors, no major effects on cerebral circulation were seen with sevoflurane by transcranial Doppler sonography. Emergence times were similar.

Table 2: The effects of the inhaled agents on intracranial dynamics.

<table>
<thead>
<tr>
<th>Inhaled Anesthetics</th>
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<tr>
<td>Drugs</td>
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</tr>
<tr>
<td>Halothane</td>
</tr>
<tr>
<td>Enflurane</td>
</tr>
<tr>
<td>Isoflurane</td>
</tr>
<tr>
<td>Sevoflurane</td>
</tr>
<tr>
<td>Desflurane</td>
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<td>( N_2O )</td>
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Table 3 demonstrates the effects of the intravenous agents. In general, intravenous agents maintain coupling between metabolic rate and CBF. Flow decreases as the need for oxygen decreases.

Table 3: Effects of the common intravenous agents on intracranial dynamics.

<table>
<thead>
<tr>
<th>Intravenous Anesthetics</th>
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<tbody>
<tr>
<td>Drugs</td>
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<tr>
<td>-----------</td>
</tr>
<tr>
<td>Thiopentone</td>
</tr>
<tr>
<td>Etomidate</td>
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<tr>
<td>Propofol</td>
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<tr>
<td>Midazolam</td>
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<td>Ketamine</td>
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Both autoregulation and CO2 reactivity are easily disrupted under anesthesia. Several studies have demonstrated that autoregulation is maintained up to 1.5 MAC with sevoflurane and better preserved than with isoflurane anesthesia in both adults and children (Fig 1). Also, responsiveness to changing CO2 levels is little altered by sevoflurane 1 MAC. Thus, in response to hyperventilation, CBF will decrease in the event that an emergency situation should arise. The amount of anesthesia
required for intracranial intervention is low as there is no sensation of pain in brain tissue.

**Figure 1: Recordings of dynamic autoregulation.** At changing blood pressure levels, there is no change in middle cerebral artery flow at 1.5MAC sevoflurane but there is a decrease at 1.5MAC isoflurane.

Cardiovascular effects of sevoflurane are minimal at MAC 1 and thus this agent seems to be better able to preserve cerebral perfusion pressure than propofol. At the time of temporary clipping of an aneurysm, some brain protection may be afforded by mild hypertension with phenylephrine, propofol and/or lidocaine. The Intraoperative Hypothermia for Aneurysm Surgery Trial (IHAST) study - an exceptionally well controlled trial - indicated that mild hypothermia conferred no benefit in aneurysm surgery. However, a more recent review suggests long-term functional outcome may be improved with therapeutic hypothermia at 35 degrees C for 10 days.

Risks for vasospasm are increased when there is increased intracranial pressure and hypovolemia. Preventive measures include early clipping, oral nimodipine, magnesium, sedation and a positive fluid balance while avoiding hyponatremia. No single measure has been shown effective in all situations.

Current evidence indicates that an effective technique for intracranial surgery would consist of sevoflurane 1 MAC combined with incremental doses of a narcotic such as fentanyl, judicious muscle relaxation and avoidance of nitrous oxide.

**Neuroprotection**

Several studies examined brain protection through anesthetic manipulations. Effective techniques include maintaining normothermia and avoiding substances that increase the metabolic activity of the brain such as N20 or ketamine (although ketamine is an NMDA antagonist that may also increase blood pressure in severely hypotensive situations). Agents such as magnesium, lidocaine, xenon, etc. have all been shown to have some effectiveness in improving brain survival, mostly in animal models. Volatile agents have been shown to improve ischemic outcome in animal models although to date no human outcome trials have been conducted to guide clinical practice.

Mechanisms which may offer protection of the brain from inhalation agents includes suppression of energy requirements, inhibition of excitatory neurotransmission, potentiation of inhibitory receptors, regulation of intracellular calcium responses and activation of TREK-1 two pore-domain K+ channels.

A review was performed of more than 600 abstracts from January, 1980, through April, 2010, to
determine whether neurologic outcome could be improved with anesthetic management.\textsuperscript{17}
Barbiturates, propofol, inhaled agents all protect cerebral tissues from apoptosis, degeneration, inflammation and energy failure. Isoflurane and ketamine can potentially cause degeneration in the developing brain. The studies also note that medical and surgical history are an important consideration. Most studies are in animal models.

Several studies report encouraging results related to preconditioning. Following 1 hour of preconditioning with sevoflurane 2.4%, the middle cerebral arteries (MCA) in a rat model were clamped.\textsuperscript{18,19} A marked decrease in apoptotic neuron density as compared to controls was noted in the treated animals. Another study reported marked improvement of blood brain integrity and outcome with decreased activation of astrocytes and microglia after repeated sevoflurane preconditioning over 24 hours in a rat MCA occlusion model.\textsuperscript{20} Wang et al used a similar model and exposed rats to sevoflurane for 30 min/day for 4 days. The MCA was then occluded for 1 hour. The infarct volume was decreased and neurologic outcome was better when compared to controls. The expression of inflammatory cytokines was also decreased. Other researchers have looked at postconditioning as an option in improving outcome. Rats were subjected to 90 minutes of MCA occlusion\textsuperscript{21}. Sevoflurane 1 MAC and 1.5 MAC were given at the start of reperfusion. At 24 hours there was a significant neuroprotective effect demonstrated (less infarct volume, edema; fewer deficits; improved learning and memory).

Barbiturates have long been studied as a means to improve neurologic outcome, mostly yielding disappointing results. A more recent review suggested that barbiturate coma might still be used for intractable intracranial hypertension.\textsuperscript{22,23} The authors warn that significant hypokalemia may result and abrupt discontinuation may cause severe rebound. Reversible bone marrow depression is another complication and coma can mask brain death. Complications may be reduced by using a lower dose of barbiturates (2mg/kg/h) and monitoring with entropy to keep a BIS number around 40-60.\textsuperscript{24}

**Monitoring**

Monitoring during neurosurgical procedures may be classified as systemic and cerebral. (See Table 4 and Table 5.)

**Table 4: Systemic monitoring recommended for all major neurosurgical procedures under general anesthesia**

<table>
<thead>
<tr>
<th>Oxygenation</th>
<th>Temperature</th>
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<tr>
<td>EKG</td>
<td>Coagulation profile</td>
</tr>
<tr>
<td>Arterial pressure</td>
<td>Respiratory parameters</td>
</tr>
<tr>
<td>Fluid and electrolyte balance</td>
<td>Capnography</td>
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<tr>
<td>Pulse pressure/stroke volume variation</td>
<td>Intracuff pressures</td>
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Table 5: Recommended cerebral monitoring for special circumstances

<table>
<thead>
<tr>
<th>Neurologic monitoring (and awake monitoring)</th>
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<tr>
<td>Jugular venous bulb oxygenation</td>
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<tr>
<td>Intracranial pressure EEG, entropy, spectral edge</td>
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<tr>
<td>Transcranial Doppler sonography</td>
</tr>
<tr>
<td>Cerebral blood flow/oxygenation</td>
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<tr>
<td>Stump pressure</td>
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<td>Evoked potentials</td>
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Some researchers studied the benefits of monitoring central venous pressures and pulmonary artery pressures (PAC-man trial, ESCAPE). Repeated studies indicate that blood pressure and heart rate change before central venous pressure; and a meta analysis of 13 trials of > 5,000 patients showed no improvement in mortality. Complications from monitoring were numerous and currently constitute 2% of claims in the ASA Closed Claims Analysis. The decision to administer fluid versus vasopressors may be best made by measurement of pulse pressure and stroke volume variations. Newer computerized monitors that measure both variations in the arterial wave as they occur with respiration can allow continuous recording of cardiac output as well as peripheral resistance. Also, computerized chips on endotracheal tube cuffs can determine cardiac output and other hemodynamic parameters as well as intracuff pressures.

Routine use of electrophysiologic monitoring to detect ischemia remains controversial although marked decrease in activity in the postoperative period may indicate worsening of cerebral condition or the recollection of a hematoma. Monitoring of evoked potentials should be helpful but there are many reports of false negatives and positives. Close communication with the electrophysiologist is essential. This is especially true during deep brain stimulation and surgery for epileptic foci identification and excision when constant readjustments to sedative and anxiolytic therapy are often necessary. Transcranial Doppler (TCD) monitoring is sometimes advocated although monitoring may be difficult and variable. Computerized analysis of TCD waveform may offer useful data on ICP. TCD can also identify obstruction to CBF. Cerebral perfusion pressure calculation is not generally available yet.

Constant evaluation of clotting parameters is essential as the brain is the richest source of thromboplastin in the body. Raised intracranial pressure can release a coagulation cascade that can develop into disseminated intravascular coagulopathy.

Management of the Case

The patient was transferred to the interventional radiology suite. All arrangements were made for offsite anesthesia including mechanisms for immediate access to all drugs, equipment, and the main operating room suite. Blood was also available. After all monitors had been placed, the patient was induced into anesthesia with midazolam, fentanyl, propofol and vecuronium. A cannula was placed in the radial artery. Anesthesia was continued with sevoflurane. After approximately 3 hours, the interventionalist determined that he could not adequately coil the aneurysm and the patient was transferred to the operating room using full monitoring. All attempts were made to maintain the
temperature at 36.5 degrees C. Blood glucose measurements were made hourly and following infusion of dexamethasone 10 mg, it was found to be 200 mg/dl. An insulin infusion was started. A frontotemporal craniotomy was performed and the aneurysm located. Temporary clips were placed and at that time the anesthesiologist gave a small dose of phenylephrine to increase the blood pressure by approximately 20%. A propofol infusion was also started. Following successful clipping, anesthesia was reversed. The patient awoke promptly and was extubated. She was transferred to the ICU and to the ward 24 hours later.

Dr. Elizabeth A.M. Frost, who is the editor of this continuing medical education series, is clinical professor of anesthesiology at The Mount Sinai School of Medicine in New York City. She is the author of Clinical Anesthesia in Neurosurgery (Butterworth-Heinemann, Boston) and numerous articles. Dr. Frost is past president of the Anesthesia History Association and former editor of the journal of the New York State Society of Anesthesiologists, Sphere. She is also editor of the book series based on this CME program, Preanesthetic Assessment, Volumes 1 through 3 (Birkhäuser, Boston) and 4 through 6 (McMahon Publishing, New York City).
REFERENCES

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Post-test

1. Factors that enter into the choice of anesthesia for neurosurgery are least likely to include:
   - The pathology involved
   - The needs of the surgeon
   - The availability of standard ASA monitors
   - An off site area

2. General requirements of anesthesia for neurosurgery include:
   - Maintenance of stable intracranial dynamics
   - 20% hypertension to maintain cerebral blood flow
   - Hyperventilation to prevent atelectasis
   - Blood sugar 150mg/dl to ensure adequate cerebral nutrition

3. A true statement regarding inhalation agents:
   - Nitrous oxide increases cerebral blood flow and decreases cerebral metabolic rate
   - Halothane causes minimal cerebral vasodilatation
   - Sevoflurane is less vasoactive than other inhalation agents
   - Desflurane causes least changes in intracranial dynamics

4. Regarding the intracranial effects of intravenous agents:
   - Most agents decrease both flow and metabolism
   - Ketamine increases both flow and metabolism
   - Midazolam has no direct vasodilatory effect
   - All of the above

5. The IHAST study:
   - Proved the beneficial effect of hypotension in aneurysm clipping
   - Failed to show improved outcome of mild hypothermia during aneurysm surgery
   - Was poorly controlled
   - Has been validated by several later studies
6. **A true statement regarding intracranial pressure:**
   
a. The normal range is 15-20 mmHg  
b. An increase in blood volume or cerebrospinal fluid will not increase intracranial pressure  
c. Hyperventilation has little or no effect on intracranial pressure  
d. Cytotoxic edema contributes to intracranial hypertension in traumatic brain injury  

7. **Regarding brain protection by anesthetic manipulation:**
   
a. Propofol increases cerebral apoptosis  
b. Most studies have been done on animal models  
c. Isoflurane and ketamine protect the developing brain  
d. Sevoflurane preconditioning is ineffective  

8. **The type of systemic monitoring least likely to add important information is:**
   
a. Arterial pressure  
b. Fluid and electrolyte balance  
c. Pulmonary artery pressure  
d. Coagulation profile  

9. **Mechanisms which may offer protection of the brain include:**
   
a. Inhibition of excitatory neurotransmission  
b. Potentiation of inhibitory receptors  
c. Regulation of intracellular calcium responses  
d. All of the above  

10. **The risk of vasospasm after rupture of an intracranial aneurysm is increased by:**
    
a. Hypovolemia  
b. Hyponatremia  
c. Early clipping  
d. Oral nimodipine