Lesson S46: Management of the Patient for Intracranial Aneurysm Ablation

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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
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TERMINATION DATE: December 31, 2016

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Professional Gaps

With the increasing use of aneurysm coiling in interventional radiology, anesthesiologists are required to function in locations other than an operating room. Familiarity with these areas is essential for the future of safe anesthetic care. Moreover, controversy exists over appropriate management of intracranial aneurysm ablation. Anesthesiologists should understand the pros and cons of the techniques involved.

Learning Objectives

At the completion of the activity, the reader will be able to:

1. List the intracranial effects of different anesthetic agents
2. Explain the importance of understanding the neurosurgical procedure
3. Describe appropriate monitoring for aneurysm ablation
4. Discuss the Hunt and Hess classification
5. Differentiate between clipping and coiling
6. Explain the precautions that are required in interventional radiology
7. Present an appropriate anesthetic plan
8. Identify ways to improve brain survival
9. Discuss how anesthetic techniques should be modified when evoked potential monitoring is required
10. Prepare a scheme for fluid replacement
Case

A 35 year old woman presented at the emergency room complaining of a very severe headache. She was alert and awake but mildly confused as to time and location. She had marked neck rigidity. Initial lab studies were normal. Blood pressure was 160/80, pulse rate 88 bpm. EKG showed occasional ventricular premature contractions and nonspecific ST/T wave segment abnormalities. Apart from a long standing history of smoking, past medical and surgical history was non-contributory. A ruptured posterior cerebral artery aneurysm was diagnosed. The interventional radiologist determined that the aneurysm could be coiled although the neurosurgeon favored open craniotomy.

Introduction

Surgical ablation of aneurysm was first described by Dandy in 1937. Endovascular coiling was developed in 1991 by Guido Guglielmi at the University of California in Los Angeles. While the use of electrically induced thrombosis for intracranial aneurysms was described for the first time in 1965, the microcatheters necessary for this procedure only became available in the late eighties. In recent years, coiling has gradually become an important alternative to surgical clipping.

Endovascular coiling aims to prevent rupture in intact aneurysms and prevent rebleeding in ruptured aneurysms. If ruptured, coiling is performed quickly after onset of symptoms because of the high risk of rapid-onset rebleeding. Patients most suitable for coiling are those that have aneurysms with a small neck size (preferably < 4 mm), luminal diameter < 25 mm; and those that have aneurysms that are distinct from the parent vessel. Technological advances have also made possible the coiling of many other types of aneurysms. Coiling promotes blood clotting around the coils, eventually sealing the aneurysm and reducing pressure on its outer wall. Clipping obliterates the aneurysm immediately.

Coiling is performed by an interventional neuroradiologist using fluoroscopic imaging guidance and typically occurs in an offsite location with the patient under general anesthesia. Such procedure rooms are usually distant from the central core and present challenges with communication. Equipment and supplies may be different from those that are readily available in an operating room setting. Also, the staff are typically not trained in operating room procedures.

The procedure usually takes about two hours and requires standard ASA monitors in addition to an arterial line. Use of entropy monitoring may indicate early signs of bleeding or vasospasm. A catheter is inserted through the femoral artery and advanced to a site close to the aneurysm. By angiography the aneurysm is localized and assessed. A microcatheter is then threaded into the aneurysm. (Figure 1)

Coils consist of platinum (e.g. Guglielmi detachable coils); matrix coils that are coated with a biopolymer and hydrogel-coated coils. Detachable coils are inserted into the aneurysm via the microcatheter. A series of progressively smaller coils are inserted into the aneurysm until it is completely filled. In the case of wide-necked aneurysms a stent may also be required.

Several studies have questioned the efficacy of endovascular coiling over the more traditional surgical clipping. Concerns center mainly on the chance of later bleeds or other recanalization. However, coiling is less invasive and allows faster recovery times.
A 2007 study indicated that 28.6% of aneurysms recurred within one year of coiling, and that the recurrence rate increased with time. Clipping allows immediate obliteration with less chance of recurrence.

The International Subarachnoid Aneurysm Trial, or ISAT, also tested the efficacy of endovascular coiling against traditional micro-surgical clipping. The study initially found favorable results for coiling. Since the study's release in 2002, and again in 2005, some researchers have reported higher recurrence rates with coiling, while others have concluded that there is no clear consensus over which procedure is preferred. The most recent trial found coiling to be effective and safe with a lower, but not statistically significant decreased death rate.

Risks of endovascular coiling include stroke, aneurysm rupture during the procedure and aneurysm recurrence and rupture after the procedure. In some patients, coiling may not be successful. In general, coiling is only performed when the risk of aneurysm rupture is higher than the risks of the procedure itself.

The Pathogenesis of Intracranial Aneurysms

Aneurysms are not infrequently found at autopsy, occurring in 0.2 to almost 10%. The occurrence increases with age. The most common type is saccular (80-90%) with sessile, pedunculated, multilobulated and fusiform types seen much less often. About 90% of aneurysms are found in the anterior circulation, that is, on vessels around the circle of Willis. Thus the operative approach is a fronto-temporal incision in the supine position. Much less common are lesions arising in the posterior circulation. A sitting, park bench or prone position is required to gain access.

Following rupture, changes caused by subarachnoid hemorrhage include impaired autoregulation and
carbon dioxide reactivity, vasospasm, increased intracranial pressure and several systemic complications including cardiac irregularities, altered pulmonary shunt, cerebral salt wasting syndrome and coagulopathies. (Figure 2)

**Figure 2. Pathophysiology of Subarachnoid hemorrhage.** *Following rupture of an intracranial aneurysm, pathologic changes increase intracranial pressure (ICP).*

![Pathophysiology of Subarachnoid hemorrhage](image)

The Hunt and Hess classification has been used as a prognostic indicator of outcome following aneurysm rupture. A Grade = 1 represents minimal risk of mortality and Grade = 5 is maximal. (Table 1)

**Table 1. Hunt and Hess Classification.** *A simple classification is used to assess mortality after subarachnoid hemorrhage.*

<table>
<thead>
<tr>
<th>GRADE</th>
<th>SIGNS AND SYMPTOMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mild headache, alert and oriented, minimal (if any) nuchal rigidity</td>
</tr>
<tr>
<td>2</td>
<td>Full nuchal rigidity, moderate – severe headache, alert and oriented, no neurological deficit besides CN palsy</td>
</tr>
<tr>
<td>3</td>
<td>Lethargy or confusion, mid focal neurological deficits</td>
</tr>
<tr>
<td>4</td>
<td>Stuporous, more severe focal deficit</td>
</tr>
<tr>
<td>5</td>
<td>Comatose, showing signs of severe neurological impairment (e.g., posturing)</td>
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</table>

Choice of Anesthesia

Many factors enter into the choice of anesthesia for the neurosurgical patient including the pathology, the needs of the patient and surgeon and the location where interventions will occur. An anesthetic plan may range from moderate sedation to general anesthesia. There are also situations where both awake and unconscious states are desired during the same procedure. Choice of monitoring depends on the surgical procedure and condition and morbidities of the patient and, as such, flexibility of anesthetic care is important. Research has centered on how anesthetic drugs and techniques may alter intracranial dynamics and thus outcome.

General Requirements

Although the choice of anesthesia must accommodate many types of neurosurgical situations, there are several general aims in the care of patients undergoing aneurysm ablation. For example, intracranial pressure and cerebral blood flow should be maintained as close to normal as possible. Systemic hemodynamic stability is essential and the anesthesiologist should usually aim for prompt awakening. Normoglycemia is important throughout the perioperative period, especially if dexamethasone is administered. Blood sugar also tends to rise due to stressful situations. If evoked potential monitoring is used, the choice of anesthetic may have to be modified. If motor evoked potentials are to be monitored, muscle relaxation should be kept to a minimum with a goal of maintaining at least 2:4 twitches. Also, inhalation concentration should be minimal, combining it with low dose propofol, fentanyl, remifentanil or dexmedetomidine infusions. Consultation with a neurophysiologist should be continued to establish the baseline anesthetic response.

Intracranial pressure

Intracranial pressure - normally around 10-15mmHg - becomes elevated whenever any increase in brain bulk, cerebrospinal fluid or intracranial blood occurs. Treatment is dependent on the cause. For example, a subdural or epidural hematoma requires surgical drainage as soon as possible whereas steroids and/or diuretic therapy reduce the edema surrounding a tumor. Moreover, as the weight of the brain has been shown to increase by about 82 grams after traumatic brain injury and the specific gravity of contused brain tissue increases. Cytotoxic edema contributes to intracranial hypertension rather than simply breaking down the blood brain barrier, indicating multifactorial causes of intracranial hypertension. Even trivial increases in intracranial pressure may cause cerebral ischemia, herniation and neurogenic pulmonary edema, and all attempts to maintain stability should be made.

Anesthetic techniques can critically alter intracranial dynamics by changing cerebral blood flow, either by choice of anesthetic and ancillary agents; ventilatory maneuvers; and by close attention to appropriate fluid management. Cerebral blood flow is altered by manipulations of arterial carbon dioxide (paCO2) levels in a linear manner. Previously, hyperventilation and hypocapnia were advocated as means to decrease intracranial pressure but any 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. The IMPACT database (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 by 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.9

**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 with the later halogenated agents. (Table 2) Nitrous oxide increases both the metabolic rate of oxygen utilization and blood flow and has limited use in intracranial surgery, although it may still have a place in uncomplicated spinal surgery. Sevoflurane has been shown to be less vasoactive than other inhaled agents10 and to cause less cerebral vasodilation than isoflurane or desflurane at the same anesthetic depth.11 Little or no changes in rCBF and electrocorticography were observed.12 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.13 Emergence times were similar.14

**Table 2. Inhaled Anesthetics.** *The effects of the inhaled agents on intracranial dynamics.*

<table>
<thead>
<tr>
<th>Drugs</th>
<th>CMRO₂</th>
<th>CBF</th>
<th>vasodilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Halothane</td>
<td>↓</td>
<td>↑↑↑</td>
<td>++</td>
</tr>
<tr>
<td>Enflurane</td>
<td>↓</td>
<td>↑</td>
<td>+</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>↓↓</td>
<td>↑</td>
<td>+</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>↓↓</td>
<td>↑</td>
<td>+</td>
</tr>
<tr>
<td>Desflurane</td>
<td>↓↓</td>
<td>↑</td>
<td>+</td>
</tr>
<tr>
<td>N₂O</td>
<td>↑</td>
<td>↑</td>
<td>-</td>
</tr>
</tbody>
</table>

By comparison 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. Intravenous Anesthetics.** *Effects of the commonest intravenous agents on intracranial dynamics.*

<table>
<thead>
<tr>
<th>Drugs</th>
<th>CMRO₂</th>
<th>CBF</th>
<th>Direct cerebral vasodilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiopentone</td>
<td>↓↓↓↓</td>
<td>↓↓↓</td>
<td>-</td>
</tr>
<tr>
<td>Etomidate</td>
<td>↓↓↓</td>
<td>↓↓</td>
<td>-</td>
</tr>
<tr>
<td>Propofol</td>
<td>↓↓</td>
<td>↓↓</td>
<td>-</td>
</tr>
<tr>
<td>Midazolam</td>
<td>↓</td>
<td>↓↓</td>
<td>-</td>
</tr>
<tr>
<td>Ketamine</td>
<td>↑</td>
<td>↑↑</td>
<td>+</td>
</tr>
</tbody>
</table>
Both autoregulation and CO$_2$ 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 adults and children.\textsuperscript{15-17} Also, responsiveness to changing CO$_2$ levels is little altered by 1.0 MAC of sevoflurane. Thus, in response to hyperventilation, cerebral blood flow will decrease in response to an emergent event.\textsuperscript{18} The amount of anesthesia required for intracranial intervention is low as there are no pain endings within the brain. Cardiovascular effects of sevoflurane are minimal at 1.0 MAC and thus this agent seems to be better able to preserve cerebral perfusion pressure than propofol.\textsuperscript{19} At the time of temporary clipping of an aneurysm, some brain protection may be afforded by mild hypertension with phenylephrine, propofol and/or lidocaine. Although the Intraoperative Hypothermia Aneurysm Study Trial (IHAST) indicated that mild hypothermia conferred no benefit in aneurysm surgery, a more recent review suggests long-term functional outcome may be improved with therapeutic hypothermia at 35 degrees C for 10 days.\textsuperscript{20-21}

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

In reviewing the current literature, it would appear that 1.0 MAC of sevoflurane combined with incremental doses of a narcotic such as fentanyl, judicious muscle relaxation and avoidance of nitrous oxide constitutes a reasonable anesthetic technique for intracranial surgery.

**Neuroprotection**

Many studies have examined means to offer brain protection through anesthetic manipulations. Techniques that have been shown to be effective include avoidance of substances that increase the metabolic activity of the brain (e.g., nitrous oxide, ketamine - although this latter drug is an NMDA antagonist and may also increase blood pressure in severely hypotensive situations) and normothermia. Agents such as magnesium, lidocaine, and xenon, among many others, have all been shown to be more or less effective in improving brain survival, mainly 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.

The suggested mechanism of brain protection 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 of >600 abstracts published in the time period of January, 1980, through April, 2010, considered the question of whether neurologic outcome could be improved with anesthetic management.\textsuperscript{22} Barbiturates, propofol, and inhaled agents all protect cerebral tissues from apoptosis, degeneration, inflammation and energy failure. Isoflurane and ketamine may cause degeneration in the developing brain. The studies also note that medical and surgical history must be taken into consideration. Most studies are in animal models which may have limited relevance to the human brain.

Other studies have looked at preconditioning and some encouraging results have surfaced. Following one hour of preconditioning with sevoflurane 2.4%, the middle cerebral arteries (MCA) were clamped in a rat model.\textsuperscript{23-24} As compared to controls, a marked decrease in apoptosis neuron density 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. 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 better over controls. The expression of inflammatory cytokines was also decreased. As it is clearly not easy to anticipate neurologic injury, other researchers have looked at postconditioning as an option in improving outcome. Rats were subjected to 90 minutes of MCA occlusion. Sevoflurane 1.0 and 1.5 MAC were given at the start of reperfusion. At 24 hours, there was a significant neuroprotective effect demonstrated (infarct volume, edema, deficits less, learning and memory better).

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. The authors warn that significant hypokalemia may result and abrupt discontinuation may cause severe rebound. Reversible bone marrow depression is another complication and coma may 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.

**Monitoring**

Monitoring during neurosurgical procedures may be divided to systemic monitoring (Table 4) and cerebral monitoring. (Table 5).

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

<table>
<thead>
<tr>
<th>SYSTEMIC MONITORING</th>
<th>Temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygenation</td>
<td>Coagulation profile</td>
</tr>
<tr>
<td>EKG</td>
<td>Respiratory parameters</td>
</tr>
<tr>
<td>Arterial and venous pressures</td>
<td>Capnography</td>
</tr>
<tr>
<td>Fluid and electrolyte balance</td>
<td>Infracuff pressures</td>
</tr>
<tr>
<td>Pulse pressure / stroke volume variation</td>
<td></td>
</tr>
</tbody>
</table>

**Table 5. Cerebral monitoring.** *Specific cerebral monitoring recommended in special circumstances.*

<table>
<thead>
<tr>
<th>CEREBRAL MONITORING</th>
<th>Jugular venous bulb oxygenation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurologic monitoring and awake monitoring</td>
<td>Coagulation profile</td>
</tr>
<tr>
<td>EKG</td>
<td>EEG, entropy, spectral edge</td>
</tr>
<tr>
<td>Intracranial pressure</td>
<td>Cerebral blood flow / oxygenation</td>
</tr>
<tr>
<td>Transcranial Doppler sonography</td>
<td>Evoked potentials</td>
</tr>
<tr>
<td>Stump pressure</td>
<td></td>
</tr>
</tbody>
</table>

The benefits of monitoring of central venous pressures and pulmonary artery pressures have been questioned (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 decrease in mortality. Complications were numerous and currently constitute 2% of claims in the ASA.
Closed Claims Analysis. Fluid versus vasopressor administration may be determined better by measurement of pulse pressure and stroke volume variations. Recently introduced computerized monitors measure both variations in the arterial wave as they occur with respiration and can allow continuous recording of cardiac output and peripheral resistance. Also, computerized chips on endotracheal tube cuffs can determine cardiac output and other hemodynamic parameters as well as intracuff pressures. It is essential to avoid fluid overload to decrease pulmonary edema and extravasation of crystalloids to other, often dependent, parts of the body.

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 has its advocates although monitoring may be difficult and rather variable. Computerized analysis of TCD waveform may offer useful data on ICP. TCD can also identify obstruction to cerebral blood flow although cerebral perfusion pressure calculation is not generally available.

**Management of the Case**

Consent was obtained from the patients and the family for interventional and surgical care. It was explained that the size of the lesion might not be compatible with the less invasive procedure. The family wished to proceed with coiling as a first option. Coagulation studies yielded normal results. Nevertheless, two units of blood and one unit of fresh frozen plasma were readied. The patient was taken to interventional radiology. General anesthesia was induced after placement of standard monitors and cannulation of the left radial artery. After two hours it was determined that the aneurysm was better ablated by open craniotomy and the patient was transferred to the operating room. Anesthesia was continued with sevoflurane and low does fentanyl. During temporary clipping across the posterior cerebral artery, propofol was given and the blood pressure maintained with phenylephrine. Plasmalyte®, to a total of 1500ml was infused. No blood or blood products were required. Appropriate fluid replacement was assessed by monitoring stroke volume variation. At the end of the case, the patient was responding to commands and met all extubation criteria.

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. **Regarding ablation of intracranial aneurysms:**
   a. Endovascular ablation has been shown superior in the majority of studies
   b. The risk of recurrence is higher with coiling
   c. Coiling is performed in the operating room only
   d. General anesthesia is usually not necessary

2. **Clipping of aneurysms in the posterior cerebral artery:**
   a. Requires a sitting, park bench or prone position
   b. Requires a supine position
   c. Is the most common type of aneurysm repair
   d. Is never performed

3. **During somatosensory evoked potential monitoring:**
   a. Inhalation agents are contraindicated
   b. Only remifentanil should be used
   c. Muscle relaxation should be maximized
   d. A balanced technique with low dose inhalation agent is preferred

4. **Studies indicated that cerebral perfusion is best preserved with:**
   a. Sevoflurane up to 1.5 MAC
   b. Ketamine at any dose
   c. Propofol 100ug/kg/min
   d. Isoflurane up to 2.0 MAC

5. **Best guide to fluid and vasopressor management is made using:**
   a. A pulmonary artery catheter
   b. A central line
   c. Continuous monitoring of blood pressure and heart rate
   d. Stroke volume variation
6. **Hyperventilation to ETCO$_2$ 32mmHg**
   a. Has no effect on cerebral tissue oxygenation
   b. May decrease cerebral partial pressure of oxygen by 25%
   c. Is first line therapy for raised intracranial pressure
   d. Increases cerebral blood flow.

7. **Preconditioning using anesthetic agents:**
   a. Has been shown effective in animal studies
   b. Is difficult to incorporate in humans
   c. Appears to decrease apoptosis
   d. All of the above

8. **Difficulties encountered in interventional radiology include:**
   a. Off-site location
   b. Unfamiliar equipment
   c. Lack of cell phone usage frequently
   d. All of the above

9. **With regard to therapeutic hypothermia, studies seem to indicate that:**
   a. Hypothermia at 35 degrees C is the standard treatment after aneurysm clipping
   b. Mild hypothermia confers the most significant effect on outcome
   c. Long-term functional outcome may be improved
   d. No effect on outcome

10. **The Hunt and Hess classification:**
    a. Is of little value in determining outcome
    b. A grade of 2 indicates good neurologic recovery
    c. A grade of 5 means excellent recovery
    d. Depends on the anesthetic management