Lesson 299: PreAnesthetic Assessment of the Patient With Acute Ischemic Stroke

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REVIEW DATE: May, 2012

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: August 1, 2012
TERMINATION DATE: August 31, 2013

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Professional Gaps

Evidence-based medicine has changed the management of acute stroke in recent years. Several major trials have been completed. Knowledge of the findings and recommendations is important to providing appropriate anesthetic care.

Learning Objectives

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

1. Summarize anesthetic problems presented by patients with acute ischemic stroke.
2. Describe the clinical presentation of a patient with acute ischemic stroke.
3. Apply appropriate preoperative testing and evaluation.
4. List proper monitoring of the patient.
5. Plan methods to maintain hemodynamic control perioperatively.
6. Outline the different treatment modalities available for stroke.
7. Present an anesthetic and analgesic plan for a patient undergoing a carotid endarterectomy (CEA).
8. List associated complications of acute ischemic stroke.
9. Anticipate, recognize, and manage likely postoperative complications following CEA.
10. Understand standard anesthetic techniques for CEA.
Case History

A 68-year-old man presented to the emergency room complaining of sudden onset of right face and arm weakness and dysphasia for the past 3 hours. Three months previously, he experienced temporary weakness in the right arm that resolved spontaneously after about 30 minutes. The patient called his physician at that time and was told to make an appointment for medical evaluation. Family events prevented him from following through and, moreover, he felt well. The patient denied any loss of consciousness, headache, falls, or trauma to the head. Further questioning revealed a 30-pack per year history of smoking and long-standing hypertension for which he took amlodipine and diltiazem, although he admitted to occasionally forgetting to take the medications. Physical examination revealed a normal cephalic head; a brisk bruit was heard over the left carotid artery and a systolic crescendo-decrescendo murmur identified best at the right sternal border. Atrial fibrillation with a ventricular response rate of 90 was identified. Strength against resistance was decreased in the right arm and leg compared with the left along with hyperreflexia. The patient was admitted and neurologic consultation sought. Orders were placed for bilateral ultrasound examination of the carotid arteries and non-contrast computed tomography (CT) scan of the head. Significant plaque occluding more than 90% of the left internal carotid artery was demonstrated. Vital signs included blood pressure (BP) of 165/90 mm Hg. Blood sugar was 167 mg/dL. The patient was started on an aspirin regimen and oral antihyperglycemic agents. His BP medications were reviewed and metoprolol and simvastatin were added. The deficit improved significantly over the next 7 days. After options and risks were discussed, the patient was scheduled for a carotid endarterectomy (CEA) 2 weeks after the stroke.

Preanesthetic Considerations for Stroke

Cerebrovascular disorders may be classified as either ischemic (80%) or hemorrhagic (20%) and are typically characterized by a sudden loss of neurologic function. Ischemic stroke can be due to thrombosis, embolism, or either systemic hypo- or hypertension, whereas hemorrhagic stroke is due to subarachnoid or intracerebral hemorrhage. A transient ischemic attack (TIA) is a brief neurologic dysfunction that persists for less than 24 hours.

Stroke is the leading cause of disability and the third leading cause of death in the United States behind heart disease and cancer. Approximately 795,000 people suffer a stroke annually; 600,000 of these are first attacks. Approximately 75% of all strokes occur in people over the age of 65 years. The incidence of stroke in men is greater than in women until age 85. The male–female incidence ratio is 1.25 at ages 55 to 64; 1.50 for ages 65 to 74; 1.07 at 75 to 84; and 0.76 at 85 and older. The data may reflect smoking incidence, hormonal changes, and/or sex-associated life span. Strokes in the elderly are more severe and debilitating, necessitating long-term care and institutionalization. Because most victims survive, the number disabled secondary to a cerebrovascular accident is expected to increase substantially as people live longer.

Causes

Acute ischemic stroke can result from thrombosis, embolism, or systemic hypoperfusion. Thrombosis generally refers to local in situ obstruction of an artery. Thrombosis can be further divided into small and large vessel disease. Large vessel disease commonly refers to disease of the common and internal carotids, vertebral arteries, the circle of Willis, and proximal branches. The obstruction may be caused by disease in the arterial wall, such as arteriosclerosis, dissection, or fibromuscular dysplasia. In smaller
vessels, obstruction usually is the result of lipohyalinosis, which is characterized by vessel wall thickening and reduction in luminar diameter. A stroke resulting from an occlusion of a branch arising from large arteries of the circle of Willis, middle cerebral, or basilar artery is referred to as a lacunar stroke.

Embolism refers to particles, usually blood clots originating elsewhere that block arterial access to a particular brain region. Cardioembolism sources include atrial fibrillation, ventricular akinesis following myocardial infarction (MI), valvular heart disease, and dilated cardiomyopathy. Total body or systemic hypoperfusion refers to globally reduced blood flow that can be the result of cardiac pump failure, reduced cardiac output, pulmonary embolism, pericardial effusion, decreased perfusion from lactic acidosis, or bleeding. Local injury can occur from cerebral vasodilation during reperfusion.

**Risk Factors**

Identification of risk factors for ischemic stroke should facilitate stroke prevention and guide treatment. The prime risk factor, hypertension, which affects 72 million North Americans, increases stroke incidence, correlating directly with higher BP. Most patients with ischemic stroke or TIA have a history of hypertension and are treated for BP reduction. Although the exact relationship between blood lipids and stroke is evolving, elevated total cholesterol or low-density lipoprotein (LDL) cholesterol levels link directly with an increased risk for ischemic stroke and large artery atherosclerotic stroke. Autopsy examinations have revealed that atherosclerosis of carotid arteries and other arteries of the circle of Willis relates directly to blood levels of LDL. When LDL levels are elevated, lipoprotein traverses the vascular endothelium through mainly receptor-independent mechanisms and accumulates within the intima. Arteries with enlarging intimal plaques compensate initially by increasing the lumen diameter. However, the plaque may enlarge enough to cause obstruction.

Many acute stroke patients have recognized that disorders of glucose metabolism and diabetes worsen the outcome after an acute incident. An oral glucose tolerance test is recommended in all stroke patients with no prior history of diabetes. Diabetes is a proven risk factor for stroke with a 2- to 3-fold increased risk for patients with diabetes compared with those without the disease. Recently, a prospective cohort study showed an increased risk for ischemic stroke or TIA in patients with metabolic syndrome, defined as obesity and any two of the following: elevated triglycerides, reduced high-density lipoproteins (HDL), elevated BP, and/or elevated fasting plasma glucose. Another study showed insulin resistance to be independently associated with the risk for stroke even if adjusted for glycemic control. Early recognition of glucose metabolism disorders in stroke patients is crucial because hyperglycemia during the acute phase worsens outcome by reducing the salvage of penumbral tissue mediated by high lactate levels in brain tissue.

Cardiac disease is an important precursor to stroke. Coronary artery disease (CAD) can be the cause of ischemic stroke by a number of mechanisms:

1. It provides a source of embolism from the heart.
2. It is commonly associated with other risk factors.
3. Medical and surgical treatments of CAD may create a predisposition.
4. Pump failure may increase the risk.

Patients with intermittent atrial fibrillation are at the greatest risk for embolism. Hypertension causes
left ventricular hypertrophy and predisposes to stroke.

Other factors associated with a higher incidence of CAD and stroke include elevated plasma levels of homocysteine and fibrinogen. Obesity predisposes individuals to stroke by virtue of hypertension, hyperglycemia, and hyperlipidemia. Cigarette smoking and secondhand smoke are further risk factors. Nicotine, carbon monoxide, free radicals, and cytokines cause the buildup of atherosclerotic plaque, in part due to recurrent episodes of hypoxia, leading to decreased blood flow and ischemia. By altering clotting factors that favor thrombogenesis, oral contraceptives increase susceptibility to thrombotic disease.

Chronic alcoholism and heavy drinking are risk factors for all subtypes of stroke. However, minimal alcohol consumption of 2 or fewer drinks per day may be protective. According to Reynolds, binge drinking, defined as 60 g alcohol (men) and 40 g alcohol (women) in a period of 2 hours, was associated with an increased relative risk for ischemic stroke of 1.69, whereas consumption of less than 12 g per day was associated with a reduced relative risk for ischemic stroke, 0.80. The meta-regression analysis revealed a significant nonlinear relationship between alcohol consumption and total and ischemic stroke.

**Prevention**

Based on data from numerous clinical trials, stroke can be prevented by modifying risk factors. These alterations include controlling hypertension, smoking cessation, anticoagulation in atrial fibrillation, increasing physical activity, weight reduction, controlling blood lipid levels, lowering elevated plasma homocysteine levels, and regulating blood sugar levels in diabetics.

Surgical treatment of carotid artery stenosis via CEA reduces the risk for stroke. Three studies, the ECST (European Carotid Surgery Trial), the NASCET (North American Symptomatic Carotid Endarterectomy Trial), and the Veterans Affairs CSP (Cooperative Study Program) all showed outcomes supporting CEA with moderate-term follow-up. Symptomatic patients included those with greater than 70% ipsilateral carotid stenosis and TIAs, transient monocular blindness, or nondisabling stroke. As a result of these landmark studies, most surgeons perform endarterectomy in a symptomatic patient with carotid stenosis of 70% or greater and an asymptomatic patient with 80% or greater stenosis. Duplex ultrasound and transcranial Doppler studies have shown evidence that carotid artery stenosis of 70% to 75% represents the point at which a pressure drop across the stenosis is likely to occur. Other techniques, including carotid artery angioplasty and stenting, may be therapeutic options for certain patients. However, the SAMMPRIS (Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis) trial was halted in April 2011 when a higher rate of adverse events in the angioplasty/stenting arm was identified.

**Pathophysiology of Cerebral Ischemia**

Cerebral blood flow (CBF) averages 40 mL/100 g of brain tissue per minute. When CBF falls to approximately 20 mL/100 g of brain tissue per minute, electroencephalogram (EEG) evidence of ischemia begins to appear. At a CBF level of approximately 15 mL/100 g of brain tissue per minute, cortical EEG becomes isoelectric and when it is reduced to 6 mL/100 g of brain tissue per minute, irreversible damage occurs. The core of the ischemic area receives insufficient blood flow to maintain energy metabolism and, consequently, dies within minutes without intervention. Between the area of core ischemia and region of normal perfusion, an area receiving intermediate blood flow, the ischemic
penumbra, exists. Blood supply is reduced to a level that interrupts neuronal function; however, adenosine triphosphate levels are maintained around typical levels. Nevertheless, if blood supply is not restored, an ischemic cascade results in additional, delayed damage to brain cells (Table 1).

**Diagnosis and Examination**

If stroke is suspected, prompt, accurate diagnosis and treatment are essential to minimize brain tissue damage. Diagnosis includes a medical history and a physical examination to evaluate the level of consciousness, sensation, and motor function, and to determine the cause, location, and extent of the stroke.

Symptoms of stroke depend on the type and location. Signs of hemorrhagic stroke occur gradually, whereas signs of ischemic stroke usually have a more sudden onset (Table 2). The medical history should be directed at identifying risk factors. Time of onset needs to be established from family, friends, or witnesses to determine whether or not thrombolytic therapy is an option. Documentation of vital signs is followed by basic laboratory blood testing and imaging studies (non-contrast CT scan, magnetic resonance imaging, ultrasound).

**Medical Management**

Medical management of the acute ischemic stroke patient begins with controlling respiratory function, which entails managing airway, oxygenation, and ventilation. Although in most patients oxygen supplementation is adequate, tracheal intubation may be needed if the patient is unable to protect his or her lungs from aspiration. It should be noted that vomiting is very common as intracranial pressure (ICP) increases.

Furthermore, the physician must maintain the patient’s baseline body temperature (37°C-38°C), hemodynamic stability, and blood glucose, and when appropriate, consider anticoagulant therapy (Table 3).

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**Table 1. The Ischemic Cascade**

<table>
<thead>
<tr>
<th>Event</th>
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<tbody>
<tr>
<td>Blood flow ceases</td>
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<tr>
<td>Cellular production of excessive glutamate</td>
</tr>
<tr>
<td>Anaerobic respiration produces lactic acid</td>
</tr>
<tr>
<td>Damaging free radicals formed</td>
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<tr>
<td>Cellular filling with electrolytes and swelling</td>
</tr>
<tr>
<td>Cellular death</td>
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</tbody>
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**Table 2. Signs and Symptoms of Stroke**

<table>
<thead>
<tr>
<th>Symptom</th>
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<tbody>
<tr>
<td>Difficulty speaking or understanding speech (aphasia)</td>
</tr>
<tr>
<td>Difficulty walking</td>
</tr>
<tr>
<td>Dizziness or lightheadedness (vertigo)</td>
</tr>
<tr>
<td>Numbness, paralysis, or weakness, usually on one side of the body</td>
</tr>
<tr>
<td>Seizure (relatively rare)</td>
</tr>
<tr>
<td>Severe headache with no known cause</td>
</tr>
<tr>
<td>Sudden contusion</td>
</tr>
<tr>
<td>Sudden difficulty walking, dizziness, loss of balance or coordination</td>
</tr>
<tr>
<td>Vomiting</td>
</tr>
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**Table 3. Modalities of Cerebral Protection**

<table>
<thead>
<tr>
<th>Physiologic</th>
<th>Recommendation</th>
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<tbody>
<tr>
<td>Hypothermia</td>
<td>Routine hypothermia is not recommended due to the possibility of increased myocardial oxygen consumption if patient shivers.</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>Treat by eliminating glucose-containing IV solutions and giving small doses of insulin.</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Maintain normal to high arterial pressure in most situations.</td>
</tr>
<tr>
<td>Hemodilution</td>
<td>Optimal hematocrit during cerebral ischemia is approximately 30%.</td>
</tr>
<tr>
<td>Carbon dioxide</td>
<td>Normocarbia</td>
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Based on animal data, hypothermia decreases both basal and electrical metabolic requirements throughout the brain. However, because there are few human studies evaluating the effectiveness of hypothermia in an acute stroke, it continues to be controversial. Systemic hypertension is common at initial stroke presentation. Rapid lowering of BP can decrease CBF and worsen the ischemic injury. Following stroke, patients may become very sensitive to hypotensive medication. Monitoring with direct arterial cannulation or very frequent noninvasive means is necessary. Even small doses of labetalol, for example, can cause an almost catastrophic fall in BP. If possible, BP should be lowered slowly over a 24-hour period (15%) at which point it often will stabilize spontaneously. Hyperglycemia appears to be associated with a poor outcome in ischemic stroke patients because glucose is metabolized to lactic acid resulting in acidosis and increased neuronal injury. Prophylaxis against deep vein thrombosis is most commonly attained by giving 5,000 units of heparin subcutaneously every 12 hours. Signs of increased ICP (vomiting, cardiac irregularities, neurologic, and papillary) as cerebral edema worsen the clinical course. 

Aspirin is recommended for both initial therapy of acute stroke and for prevention of recurrent strokes. IV recombinant tissue plasminogen activator may be used if treatment can be started within 4.5 hours of symptom onset. Direct infusion of thrombolytic agents (prourokinase) through micro catheters directly at the site of occlusion offers several benefits. First, there is less risk for systemic bleeding because the use of intra-arterial thrombolysis allows physicians to deliver the thrombolytic directly to the affected vessel in a lower dose than with IV therapy. Furthermore, this approach allows a large dose of concentrated agent to be directly delivered to the thrombus. Finally, intra-arterial thrombolysis can be combined with methods of mechanical disruption. Clinical trials have demonstrated that the time window may be longer than previously thought for intra-arterial thrombolysis. Many patients, unfortunately, have lingering neurologic deficiencies despite these treatments. The initial amount of neurologic dysfunction and early evidence of recovery in acute stroke are 2 powerful predictors of outcome. Thrombolytic agents are contraindicated in patients with severe sustained hypertension.

Other therapies include early evacuation of the clot, decompressive craniectomy, neuroprotective agents such as calcium channel blockers, citicoline, γ-aminobutyric acid agonists, glycine antagonists, lubeluzole, and N-methyl-D-aspartate antagonists. Systemic anticoagulation includes heparinoids, specific thrombin inhibitors, low-molecular-weight heparin, oral anticoagulants, unfractionated heparin, as well as thrombolysis mentioned above.

Magnesium has been studied as a neuroprotective agent in many settings. Although magnesium has been shown to be protective in animal studies, results of the IMAGES (Intravenous Magnesium Efficacy in Stroke) trial in humans following acute ischemic stroke were largely disappointing, perhaps because the therapy was not combined with post-ischemic hypothermia.

Preoperative Anesthesia Evaluation

The preoperative anesthesia evaluation should involve assessment of neurologic function and baseline vital signs. Decline of neurologic function after an acute ischemic stroke may indicate advancing neuronal injuries, thrombus propagation, early recurrent embolism, or collateral vessel failure. Hemorrhagic transformation, hydrocephalus, intracranial edema, and herniation also can be responsible for decreasing neuronal function and in these instances, decompressive hemicraniectomy becomes an emergency.
The anesthesiologist also should be concerned with common comorbid conditions, many of which are risk factors for the ischemic event. Current medications should be assessed with focus directed toward cardiovascular medications, anticoagulants, and antiplatelet medications. Evolving literature suggests a role for statin therapy in reducing postoperative morbidity and mortality. In the DECREASE III (Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echocardiography) trial, patients started on fluvastatin at 80 mg demonstrated a 47% reduction in MI.\textsuperscript{16}

The effect of modifications in head position on neurologic function has to be identified preoperatively as patients with vertebral artery disease are susceptible to angulation and compression of this artery with even minimal rotation, flexion, or extension of the head. Direct laryngoscopy for intubation predisposes the patient to unsafe head positions that can result in neurologic deficits. Carotid artery palpation should be avoided as this could induce fragmenting of the occlusion and embolism to the brain.

**Intraoperative Monitoring**

Central nervous system monitoring devices include EEG, stump pressure measurement, transcranial Doppler, and cerebral oximetry. EEG remains a sensitive indicator of inadequate cerebral perfusion. Intraoperative neurologic complications have been shown to correlate well with EEG changes indicative of ischemia. Stump pressure measurement, which measures perfusion pressure, can be an important determinant of CBF. Although neither sensitive nor specific, it involves direct measurement of the retrograde internal carotid artery pressure following occlusion of the more proximal common and external carotid arteries. Only extreme values (<25 mm Hg) are useful indicators of the state of cerebral circulation. Trans-cranial Doppler has been used to measure blood flow velocity in the middle cerebral artery during CEA. Ischemia is severe if the mean velocity after clamping is 0 to 15% of pre-clamped value, mild if 16% to 40%, and absent if greater than 40%. Cerebral oximetry, or near-infrared spectroscopy, is a technique that relates neurologic outcome to cerebrovascular hemoglobin oxygen saturation. However, results suggest that there is no significant difference between cerebrovascular hemoglobin oxygen saturation during preclamp or postclamp periods.\textsuperscript{17} In this regard, in vivo spectroscopy is used in many places as standard monitoring.

Cerebral steal is defined as the decrease in blood flow to an ischemic area secondary to blood vessel dilation in non-ischemic areas. Given that the blood vessels in the ischemic areas have a fixed resistance, the reduction in pressure caused by vasodilation of adjacent vasculature diverts blood flow away from the ischemic area. However, vasoconstriction in non-ischemic areas of the brain may increase blood flow to the ischemic areas. This condition is known as either Robin Hood phenomenon or inverse steal. The clinical significance of both of these phenomena is not well understood.

**Anesthetic Management**

The patient, the anesthesiologist, and the surgeon should agree as to whether regional or general anesthesia is preferred for the procedure. Surgeons vary in how they approach the procedure; therefore, the surgeon should be consulted prior to the surgery so that the anesthetic can be tailored to the surgeon and the patient. Finally, the anesthesiologist should proceed with the proposed anesthetic technique only if the anesthetic provider is comfortable with the technique and assesses that the technique is an acceptable one for the patient.\textsuperscript{18,19}
General Anesthesia

Currently, no anesthetic regimen has been proven to provide better cerebral perfusion and homeostasis, whether for carotid endarterectomy or stenting, although medications with rapid onset and short duration should be agents of first choice because they tend to decrease the postoperative influence on mental status and level of consciousness. Many anesthesiologists prefer general to regional anesthesia because not only are they more comfortable with this technique, but there also are some advantages associated with general anesthesia. General anesthesia is preferred in patients with anatomy/pathology that may make surgical conditions difficult. General anesthesia decreases cerebral metabolic demand and hence oxygen consumption, which is particularly important in a procedure where there are periods of decreased cerebral perfusion even if a shunt is placed. Another advantage is the ability to increase cerebral perfusion and oxygenation through manipulation of BP along with the ability to control levels of oxygen and carbon dioxide. Many also argue that general anesthesia enhances the patient’s comfort during the procedure. General anesthesia has been reported to be associated with increased use of vasoactive medications postoperatively along with extended anesthesia recovery time and recovery room stay.20

Several theories have been proposed to explain the efficacy of barbiturates to prevent or treat cerebral ischemia, yet conclusive studies are lacking. Etomidate has been used in neurovascular procedures because of its short duration of action, cardiac stability, and metabolic properties, but there is evidence in animals that etomidate worsens ischemic injury and therefore is not recommended for use as a cerebral protectant. Each of the volatile anesthetic agents produces a decrease in the cerebral metabolic rate of oxygen consumption and an increase in CBF. Desflurane affords the quickest recovery profile, which may offer an advantage for neurocognitive assessment postoperatively. When appropriate, esmolol, labetalol, hydralazine, phenylephrine, ephedrine, nitroglycerin, and in rare circumstances, sodium nitroprusside can be employed to modulate BP and heart rate.

When choosing fluids, less is probably better, especially if the patient is awake and no urinary catheter is placed.

Regional Anesthesia

The most common form of regional anesthesia used for CEA is a combination of a superficial and deep cervical plexus block with blocking of nerves originating from C2 to C4. It was previously thought that regional anesthesia was associated with a decrease in deaths and/or stroke, MIs, local hemorrhage, and pulmonary complications.18 However, Lewis et al published the results of the GALA (General Anaesthesia versus Local Anaesthesia for carotid endarterectomy) trial in which 3,526 randomized patients underwent general or regional anesthesia for CEA and there were no observed differences in the incidence of stroke, MI, or death.18 Other reports suggest that a regional anesthetic technique is associated with more selective shunt usage and may produce better anesthetic outcomes because the physicians are able to monitor cognitive function in a conscious patient providing confirmation of adequate cerebral perfusion during arterial clamping.20 Relative contraindications to the use of regional anesthesia include patient refusal, clotting abnormalities, airway compromise, prolonged surgery, local infection where the block is to be performed, chronic coughing, and an anxious patient.
Postoperative Period

Patients should be monitored for BP changes, as hypertension peaks in the first 2 to 3 hours up to 24 hours postoperatively especially in patients who have predisposed risk factors such as essential hypertension. Systemic BP should be maintained at preoperative baseline to prevent myocardial ischemia and cerebral edema. If BP is not controlled, the complications that may arise include excessive bleeding at the operative site, intracerebral hemorrhage, increased ICP, and also myocardial ischemia and MI. Potent agents such as nitroprusside may be needed to treat refractory elevated BP. In case of hypotension, prompt evaluation and treatment are required. The exact mechanism for hemodynamic changes remains unknown; it may involve dysrhythmias, dehydration, decreased circulation secondary to medications, or renewed blood flow to the carotid sinus stimulating afferent nerve activity following surgery. Hypotension often is treated by ensuring first that there is no ongoing bleeding, IV crystalloid solutions, vasopressors, and/or injection of local anesthetics to the areas surrounding the carotid sinus. Finally, following CEA, patients need close observation to monitor for any type of respiratory distress. Airway obstruction secondary to overdose of anesthetics, hematoma, obesity, or obstructive sleep apnea syndrome commonly produces respiratory distress. Hyperperfusion syndrome is a potential complication following carotid endarterectomy. Clinical symptoms develop as a result of rapidly increased cerebral blood flow in excess of that required to meet metabolic demands. It is theorized that the capillary bed beyond the stenosis is prone to perfusion breakthrough bleeding after increase of the blood flow because of the presence of impaired autoregulation.

Case Management

After options were discussed, the patient was treated with a regional technique. A total of 18 mL of ropivacaine 0.5% was administered in divided doses for combined deep and superficial cervical plexus blocks. Standard ASA monitors along with a V5 lead and brain awareness monitor were placed and a total of midazolam 3 mg and propofol 240 mg were administered throughout the case. Postoperatively, the patient was given a total of labetalol 15 mg for elevated BP. He was discharged from the hospital the next day.

Summary

Acute ischemic stroke is a neurologic emergency where the risks and the mechanisms of complications are yet to be completely understood. Patients requiring CEA secondary to carotid plaques can be managed effectively by both regional and general anesthetic techniques. Due to the complexity of cerebrovascular disease, anesthesiologists must recognize the pathophysiology of acute ischemic stroke and the challenges in managing these types of patients. Effective perioperative management of these complex patients can minimize postoperative morbidity and mortality.

Acknowledgment: The authors wish to acknowledge Clinton Steffey, MD, for his contributions in an earlier version of this manuscript.

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REFERENCES

Post-test

1. Over the next few years the number of stroke victims is expected to_____.
   a. increase
   b. decrease
   c. stay the same
   d. increase slightly, then decrease

2. A primary risk factor for acute ischemic stroke is_____.
   a. alcohol abuse
   b. diabetes
   c. intermittent atrial fibrillation
   d. obesity

3. The ischemic penumbra is defined as the_____.
   a. region receiving insufficient blood flow
   b. region receiving intermediate blood flow
   c. region receiving normal blood flow
   d. region receiving no blood flow

4. Within what time period can recombinant tissue plasminogen activator be used in patients from the onset of symptoms?
   a. 20 minutes
   b. 1 hour
   c. 4-5 hours
   d. Should not be used

5. Vasoconstriction in non-ischemic areas of the brain that may increase blood flow to the ischemic area is known as_____.
   a. cerebral steal
   b. Robin Hood phenomenon
   c. hemorrhagic transformation
   d. deep vein thrombosis
6. **Medications of first choice should have which of the following qualities?**
   a. Rapid onset and short duration
   b. Rapid onset and long duration
   c. Late onset and short duration
   d. Late onset and long duration

7. **The anesthetic of choice for the patient undergoing carotid endarterectomy is____.**
   a. regional technique
   b. general anesthesia
   c. neither as local infiltration suffices
   d. neither has been proven superior

8. **What is hyperperfusion syndrome?**
   a. A synonym for hemorrhagic stroke.
   b. Inability of the brain to autoregulate secondary to prolonged decreased cerebral blood flow.
   c. Increased blood flow to one region of the brain resulting in another region becoming ischemic.
   d. Increased blood flow to the extremities.

9. **In the acute ischemic stroke patient, the anesthesiologist must____.**
   a. maintain cerebral perfusion pressure
   b. maintain systemic hemodynamic stability
   c. understand the pathophysiology of acute ischemic stroke
   d. All of the above

10. **Preferred cerebral monitoring during carotid endarterectomy includes____.**
    a. cerebral oximetry
    b. arterial pressure
    c. temperature
    d. All of the above