A review of cerebral aneurysms and their anesthetic management

SHEREE L. HELMS, CRNA, BSN
JOE R. WILLIAMS, CRNA, MS
Nashville, Tennessee

The authors present a summary of the pathophysiology of intracranial aneurysms, with special emphasis on the anesthetic management of ruptured and unruptured cerebral aneurysms. The selection of hypotensive techniques is also discussed.

The cerebrovascular lesion most commonly responsible for death in young adults is the intracranial aneurysm. Routine autopsies have reported an occurrence of unruptured aneurysms of anywhere from 4 to 17%. Intracranial aneurysms are second to aortic aneurysms as the cause of vascular dilation resulting from defects in the vascular network elasticity. They are more frequent in men and the peak age-related incidence for rupture is the fifth decade of life.

Aneurysms have been classified into three types: congenital, arteriosclerotic and mycotic. Congenital or berry aneurysms are responsible for 50% of all subarachnoid hemorrhages. Major characteristics of the congenital type are that they are usually multiple, occur at vascular bifurcations and are thought to arise from a muscular defect in the vessel wall.

Arteriosclerotic aneurysms cause about 15% of subarachnoid hemorrhages and rarely rupture. The mycotic type represents 5% of diagnosed aneurysms and is the most common in distal cerebral circulation. These aneurysms usually rupture. Arteriovenous malformations constitute the remaining causes of subarachnoid hemorrhages.

Etiology

Intracranial aneurysms are caused by congenital or acquired factors. Congenital defects affect the medial lining of cerebral arteries, especially the vessels composing the Circle of Willis and their bifurcations. This defect permits local distention of the vessel wall whenever blood pressure is elevated. Medial lining defects occur in 80% of the population. Acquired aneurysms account for the remaining causes of aneurysm formation. This type occurs secondary to fragmentation of the elastic lamina due to arteriosclerosis and hypertension.

Using carotid angiography, McKissock in 1956 summarized the location of cerebral aneurysms in a group of 206 patients. About 50-60% of patients admitted with spontaneous subarachnoid hemorrhage will have an intracranial aneurysm either in the Circle of Willis or in one of its major branches. Approximately 10% will have an intracranial angioma, and in the remaining patients, no vascular lesion will be present. The distribution and incidence of major intracranial aneurysms is shown in Figure 1.

Pathophysiology

An intracranial aneurysm should be suspected if a patient complains of unilateral headache of sudden onset. If the oculomotor nerve is involved, ptosis and pupillary dilation may be present due to nerve root compression. In about 70% of patients with subarachnoid hemorrhage, a history of sudden headache, meningismus or transient collapse will be elicited.
After an aneurysm ruptures, the clinical presentation depends on whether or not a significant subdural or intraparenchymal hematoma develops or whether intraventricular rupture occurs. The patient may also have signs of meningeal initiation caused by the presence of blood in the subarachnoid space. Neurological deficits are common and include hemiparesis, speech and visual alterations and cranial nerve palsies. Five to 10% of patients will have a seizure at the time of rupture.¹

An important cerebral complication during the prerupture and postrupture periods is the development of vasospasm. Vasospasm is defined as radiographically demonstrated obstruction of cerebral arteries. Vasospasm occurs in 21 to 62% of patients with subarachnoid hemorrhage, and is the most common cause of neurological deterioration after subarachnoid hemorrhage. Vasospasm can occur after closed head injury, intracranial surgery, central nervous system infections, penetrating cerebral injuries or drug therapy.²

The cause of vasospasm is unknown; however, various factors implicated are angiotensin, serotonin, prostaglandins, norepinephrine, iron and hemoglobin fragments. The autonomic nervous system is probably closely involved, as subarachnoid hemorrhage causes a depletion of catecholamine stores in the cerebral perivascular sympathetic plexus. The vessel wall spastically constricts in the presence of circulating catecholamines.¹

There are two approaches to treatment of vasospasm. These are: (1) the use of cerebral vasodilators to overcome spasm; and (2) attempting to increase cerebral perfusion beyond the area of vasospasm.

Catecholamine antagonists that have been unsuccessfully used in an attempt to treat vasospasm include phenoxybenzamine, phentolamine and ethanol. The beta adrenergic stimulator insoproterenol has been used successfully. Direct vascular smooth muscle relaxants such as lidocaine, papaverine and nifedipine have had some success. The combination of kanamycin and reserpine has recently been reported to reduce the ischemic syndrome secondary to vasospasm.³

In addition to vasospasm, recurrent bleeding is another cause affecting mortality after subarachnoid hemorrhage caused by a ruptured intracranial aneurysm. The use of bedrest and antifibrinolytic drugs has been shown to reduce the rebleeding rate from approximately 25% to 10%.⁷ Recent studies have shown that the best results are obtained with high antifibrinolytic drug doses given on the fourth to seventh post-bleed day to patients in good neurological condition.⁴

**Surgical treatment**

Successful obliteration of the aneurysm in neurologically intact patients is an accepted practice anytime after subarachnoid hemorrhage. A study by Sundt et al in 1978 concerning the surgical management of subarachnoid hemorrhage showed an operative mortality rate of 5%, ranging from 1.6% for patients with normal preoperative neurologic function to 35% for severely disabled

---

**Figure 1**

Distribution of major aneurysms in a series of 206 patients from the circle of Willis

![Diagram of major aneurysms in the circle of Willis](image)

Fig. 1 reproduced by kind permission from (Wylie and Churchill-Davidson) *A Practice of Anaesthesia* (4th ed. 1978), edited by H. C. Churchill-Davidson, M.D. London: Lloyd-Luke (Medical Books).
patients. They surmised that the results of surgical treatment were preferable than to allow the natural course of the disease.

Surgery is usually delayed two to 10 days following subarachnoid hemorrhage to prevent aggravating vasospasm. Several surgical approaches are available, depending on the location and severity of the aneurysm. Surgical approaches include:

1) A chemical reinforcement or packing of the aneurysm sac with muscle.
2) Trapping or fixing a surgically inaccessible aneurysm between a parent vessel.
3) Microscopic obliteration or clipping of the aneurysm neck.
4) Percutaneous or stereotactic stabilization of the aneurysm with medication injected into the sac.

Anesthetic management

The goals of anesthetic care include the prevention of rupture of the aneurysm prior to surgical access, the provision of adequate oxygen to the brain, the prevention of increases in intracranial pressure, and the rendering of a stable, bloodless field. These goals are especially challenging for the anesthetist, because the high cerebral perfusion pressure needed to ensure adequate blood flow is achieved at the cost of increased risk of aneurysm rerupture.

Preoperative considerations. Patients who are lethargic or obtunded due to sedation or neurologic process should not receive any preoperative medication. Those patients who are alert, or complain of headache or stiff neck may receive a light preoperative medication. Patients who have increased intracranial pressure secondary to intercerebral aneurysm rupture should not receive a narcotic preoperatively because of increased cerebral vessel dilation from CO₂ retention. At the authors’ institution, an oral benzodiazepine such as diazepam or lorazepam is ordered 90 minutes prior to surgery. Usually these patients are transported with oxygen by mask or nasal cannula to the operating room.

Intraoperative considerations

Ruptured phase. The goals of anesthetic care for a ruptured or unruptured cerebral aneurysm are basically the same. However, there are a few critical techniques that must be kept in mind. Because a ruptured aneurysm is a surgical emergency, expeditious patient preparation is required. The anesthetist should attempt to maintain the degree of hypotension already present. The use of unilateral or bilateral carotid artery compression is very effective in helping produce a bloodless field. Moderate hypotension combined with intermittent unilateral or bilateral manual carotid artery compression can reduce intracranial bleeding dramatically, and thus reduce the risk of cardiovascular collapse due to blood loss. When intravenous lines are established, the blood volume should be rapidly corrected with intravenous fluids.

Unruptured phase. The first anesthetic priority is ensuring a smooth induction and endotracheal intubation. The primary problem that can occur in the periinduction period is hypertension during laryngoscopy and intubation. This can be prevented by a satisfactory level of anesthesia, adequate topical anesthesia of the trachea, and satisfactory muscle paralysis before laryngoscopy is attempted. Lidocaine has been shown to depress the sympathetic response of intubation. A study done by Bedford et al. in 1980 found that lidocaine in a dose of 1.5 mg/kg is as effective as thiopental for rapid reduction of intracranial pressure while causing less hypotension than thiopental.

An important consideration during anesthetic maintenance is avoidance of rebleeding or aneurysm rupture during the operative period. This is achieved by recognizing and preventing increases in blood pressure, and avoiding hypoventilation and extreme degrees of hyperventilation. Adequate depth of anesthesia before application of the head holder and skin incision can prevent increases in intracranial pressure.
in blood pressure and intracranial pressure. It has been shown that intracranial pressure increases from a mean of 13.8 mmHg to 31 mmHg (normal intracranial pressure 5-15 mmHg) with application of a pin holder or scalp incision.12

A main concern during any neurosurgical anesthetic, including surgery for intracranial aneurysms, is the reduction of intracranial pressure. The intracranial compartment is composed of three components: (1) brain tissue (85%); 2) cerebral spinal fluid (8-12%); and (3) blood (3-7%). A reduction in any of these components will cause a decrease in intracranial volume and pressure.

The anesthetist can most easily control intracranial pressure by reducing cerebral blood flow. The most immediate and effective technique of reducing cerebral blood flow is hyperventilation. Reducing the PaCO₂ from normocarbia to 20-25 mmHg will almost halve cerebral blood flow. Reductions below 20 mmHg produce maximal vasoconstriction and can cause cerebral hypoxia. A PaCO₂ range between 25-30 mmHg is considered by many to be optimal for reducing cerebral blood flow.13

In addition to reducing cerebral blood flow, the anesthetist may reduce the volume of blood in the cranium by facilitating cerebral venous drainage. This can be done by positioning the head above the chest, which is accomplished by modest reverse Trendelenburg position. Venous drainage from the brain can also be improved by avoiding venous outflow obstruction caused by extreme flexion and twisting of the head. Such incidents as coughing or straining with an endotracheal tube in place may increase intracranial pressure by obstructing cerebral venous drainage. It is beyond the scope of this paper to discuss in detail methods of reducing intracranial pressure. Frequently, the neurosurgeon may order specific drugs or devices to reduce cerebral spinal fluid and brain tissue volume. These include the administration of corticosteroids, osmotic and loop diuretics and external cerebral spinal fluid drainage catheters.

**Hypotensive anesthesia**

There are very few cases in anesthesia where deliberate, controlled hypotension is needed. However, in neurological anesthetics pharmacologically induced hypotension is frequently requested to reduce the risk of aneurysm rupture and to provide a bloodless field should rupture occur.

Before instituting elective hypotension one must be aware of the increased risk factors. These include: (1) preoperative arterial hypertension; (2) active arterial vasospasm; (3) brain edema; and (4) intracerebral hematoma. A recent coronary infarction (that is, one that occurred less than six months prior), is a relative contraindication to elective hypotension.14

The goal of induced hypotension is to reduce cerebral perfusion pressure without significantly decreasing cerebral blood flow. Cerebral autoregulation occurs between mean arterial blood pressures of 60 to 160 torr, thus cerebral perfusion is not related to cerebral perfusion pressure. However, below 60 torr, cerebral perfusion is related to perfusion pressure.15 One must be aware that this cerebral autoregulation range refers to normotensive patients. The curve is dramatically shifted to the right in persons who have hypertension, as shown in Figure 3. A critical mean perfusion pressure greater than 40 torr, which represents a 50% reduction of the usual mean blood pressure in unanesthetized individuals, is required in order to prevent cerebral hypoxia. At this pressure cerebral blood flow is 18 ml/100 gm brain tissue/minute. At these flow rates, brain electrical activity as monitored by an EEG begins to show ischemic changes, indicating that cerebral blood flow is not supporting cerebral metabolism.16 Remember that this level represents normotensive patients. As previously stated, hypertensive patients may suffer cerebral hypoxia unless a higher critical perfusion pressure is attained.

**Methods to induce hypotension.** The elements of induced hypotension should consider alterations in body position, mechanical ventilation, anesthetic agents, and blood volume. Cerebral hypotension is facilitated by simply raising the head and lowering the legs. Blood pressure can also be reduced pharmacologically by direct arterial or venous dilators and by ganglionic blocking drugs.

**Ganglionic blockers.** The drug trimethaphan (Arfonad®) is the only available ganglionic blocker currently available in the United States. The drug works by sympathetic ganglion blockade, which causes dilation of resistance and capacitance vessels. There are some reports of myoneural blockade as well. Trimethaphan is excreted by the kidneys after being metabolized by serum cholinesterase. This drug is recommended for MAP reductions above 50 torr. Problems with this drug include bronchospasm, thought to be secondary to histamine release, tachycardia and tachyphylaxis. It should not be used in patients with acute angle glaucoma, because direct vessel dilators cause pupil dilation that may last for hours.16,17

**Direct vessel smooth muscle dilators.** Nitroglycerin directly dilates capacitance vessels. Its mode of action is thought to involve a specific sulphydryl...
group in the nitroglycerin receptors found in vascular smooth muscle. Perhaps nitroglycerin oxidizes these groups. The usual starting dose is 1-2 μg/kg/min. Tachycardia is a frequent problem, but propranolol can be given to attenuate this.\textsuperscript{15} Personal experience by the authors has found that extremely large doses of nitroglycerin are required to decrease blood pressure. Sodium nitroprusside (SNP) was used by the authors as a substitute for nitroglycerin when the desired decrease in blood pressure was not achieved.

SNP is another direct smooth muscle vasodilator. This drug continues to be the most widely used hypotensive agent.\textsuperscript{16} SNP is a potent rapid-acting antihypertensive agent which produces arteriolar and venous vasodilation by direct action independent of the autonomic nervous system.\textsuperscript{18}

The SNP molecule contains five cyanide groups and releases cyanide in its metabolism. Four cyanide molecules are converted to thiocyanate through the interaction of a hepatic enzyme rhodanase. The rate of conversion from cyanide to thiocyanate is dependent on vitamin B\textsubscript{12} and thiosulfate. The remaining cyanide molecule reacts with Fe\textsuperscript{+++} (methemoglobin) to form cyanmethemoglobin.\textsuperscript{15}

Cyanide poisoning is obviously a toxic side effect of SNP. This can occur when greater than 1 mg/kg of SNP is given within 24 hours. Michenfelder and Tinker have recommended that the maximum dosage of SNP not exceed 8 μg/kg/minute.\textsuperscript{15} Cyanide will eventually bind to tissue cytochrome oxidase in the absence of sufficient methemoglobin. This leads to tissue anoxia, metabolic acidosis and an increase mixed venous oxygen content.

---

**Figure 3**

Cerebral autoregulation

![Figure 3](image_url)

Fig. 3 reproduced by kind permission from (Churchill Livingstone) 1981 Anesthesia Volume 2, edited by Ronald D. Miller, M.D.
Treatment of cyanide toxicity includes stopping the infusion at the first sign of tachyphylaxis or resistance and administering 100% oxygen. Also, thiosulfate 150 mg IV and sodium bicarbonate can be given. If severe cyanide poisoning has occurred, amyl nitrate and sodium nitroprusside should be given.

When administering SNP during intracranial procedures, one must be aware of some potential problems. A study by Turner et al. in 1977 showed a statistically significant increase in intracranial pressure during the infusion of SNP in normocapnic patients. The mean increase in intracranial pressure was 6.3 mmHg to 11.7 mmHg when arterial blood pressure was reduced slightly. Additionally, it was shown that trimethaphan does not usually produce intracranial pressure increases except when intracranial compression is severe. Turner et al. surmised that if SNP is given to a neurosurgical patient before the dura is open, intracranial pressure increase may result in a brain shift.

Though SNP has potential problems, when carefully used it is still the most popular drug for elective hypotension. Those patients requiring large doses of SNP usually have increased sympathetic tone. Also, using an anesthetic technique that does not decrease sympathetic tone (such as nitrous oxide and narcotic techniques) will elevate the dose requirement of SNP.

**Postoperative management**

After intracranial aneurysm surgery, the patient must be observed for wide swings in blood pressure. Episodes of hypotension must be prevented in order to lessen the occurrence of cerebral vasospasm. After consultation with the surgeon, extubation may be performed in the early recovery period if the patient is medically stable and if no significant complications occurred intraoperatively.

**REFERENCES**


**AUTHORS**

Sheree L. Helms, CRNA, BSN is a graduate of Manley L. Cummins School of Nurse Anesthesia in Dothan, Alabama. She received her BSN from The Medical College of Georgia in Augusta, Georgia. She is currently employed as a staff anesthetist by Anesthesiology Consultants in Nashville, Tennessee. This article was written while she was a senior anesthesia student.

Joe R. Williams, CRNA, MS, graduated from Baylor University School of Nursing. He received his anesthesia education at Duke University Medical Center in Durham, North Carolina. He attained his master's degree in pharmacology from the University of North Carolina School of Medicine. Currently, Mr. Williams is director of the Manley L. Cummins School of Anesthesia in Dothan, Alabama.