Three intracranial vascular lesions which may present for surgery are aneurysms, arteriovenous malformations (AVM) and occlusive cerebrovascular disease. As aneurysms are the most frequent, the majority of this presentation will focus on them.

Who has a cerebral aneurysm and what happens to them? Intracranial aneurysms are saccular dilatations that occur most commonly at bifurcation points of the major intracranial cerebral vessels. The prevalence in North America is estimated to be 2,000 per 100,000 (1:50!!) with an annual incidence of subarachnoid hemorrhage (SAH) from rupture of 12 per 100,000, although the recently published International Study of Unruptured Intracranial Aneurysms (ISUIA) found a much lower incidence of rupture, especially for small lesions. SAH occurs most commonly between the ages of 40-60 with a female preponderance of 1.6:1.

What happens when an aneurysm ruptures? At the moment of rupture there is free communication between the artery and the subarachnoid space resulting in (local) intracranial pressure (ICP) equal to blood pressure which manifests as sudden severe headache and (temporary) loss of consciousness. The blood spreads through the subarachnoid space resulting in meningism, headache and hydrocephalus, either from obstruction of CSF resorption or clot in the ventricle. Neurological injury manifests as depressed consciousness and focal neurological signs. Isolated cranial nerve palsy may reflect neuropraxia from “jet impact” injury. Clinical diagnosis is confirmed by CT scan, MRI and four-vessel angiography.

Approximately one third die or are severely disabled at the time of the initial bleed and of the remaining patients only one third are "functional" survivors. This occurs in spite of an operative mortality of less than 10% in experienced hands and reflects the severity of the non-operative complications, primarily re-bleeding and vasospasm.

What are the neurological complications? Re-bleeding: The risk of re-bleeding without surgery is 30-50% in the first two weeks with a mortality > 50%. During the initial bleed, blood is able to spread throughout the subarachnoid space. With subsequent bleeds, blood clots and adhesions prevent such spread so that intracerebral hematomas are more likely. The major impetus for early surgery is the prevention of re-bleeding. In addition, it may reduce the likelihood of developing vasospasm by removing blood in the subarachnoid space, enable safer administration of more aggressive therapy for vasospasm, reduce the risk of developing medical complications and reduce costs by reducing hospital stay.

Hydrocephalus: Occurs in 15-20% of patients from communicating or obstructive hydrocephalus.

Vasospasm: Vasospasm remains a major cause of morbidity and mortality. Angiographic evidence of vasospasm occurs in up to 60% of patients, but is manifest clinically in only half. The clinical features begin insidiously with depressed consciousness (a reflection of global cerebral hypoperfusion) and later, focal neurological signs. The occurrence is related to the volume and location of subarachnoid blood and the clinical grade of the patient. The exact etiology remains uncertain but is related to oxyhemoglobin and its breakdown products. Transcranial Doppler (TCD) is a useful bedside adjunct in the diagnosis of vasospasm. With the onset of vasospasm there is an increase in the blood flow velocity to >120cm/s which subsequently diminishes as the spasm further progresses. Nimodipine is the standard (prophylactic) drug in the management of vasospasm. There is no evidence that nimodipine angiographically relieves vasospasm and its mechanism of action may reflect a “brain protective” mechanism. Nimodipine or similar drugs will make patients prone to hypotension if they are hypovolemic and especially at the induction of anesthesia.

Currently, the most effective treatment available is hypervolemic-hypertensive-hemodilution (“Triple H” therapy). The goals of this treatment are to augment cardiac output, improve the rheological characteristics of the blood and to increase cerebral perfusion pressure (CPP). Triple H therapy has been shown to reverse ischemic neurological deficits associated with vasospasm in up to 70% of patients. Other treatments include angioplasty and papaverine.

Are there any systemic effects from SAH? Cardiovascular: SAH causes a massive sympathetic discharge at the time of bleeding which results in hypertension and may cause myocardial dysfunction, ST segment changes, rhythm disturbances and neurogenic pulmonary edema. These cardio-pulmonary effects are due to intense intra-myocardial release of catecholamines resulting in focal calcium over-load, which may result in cell necrosis. EKG changes occur in >50% of patients and rhythm disturbances can occur in up to 90%. The type of EKG change, other than Q
waves or elevated ST segments, do not usually indicate underlying myocardial dysfunction which correlates more with the severity of the neurological state than the type of EKG change, i.e. there is a relationship between the extent of the neurological injury and the cardiac injury. The appropriate management of cardiac injury in SAH is poorly defined and does not usually influence management other than occasionally requiring more invasive cardiac monitoring and potentially making endovascular rather than open surgery a preferred treatment option.

Intravascular fluid volume & Electrolytes: Many with SAH have depletion of their intravascular volume presumed to be from bed rest, supine diuresis, and stress. Electrolyte abnormalities especially hyponatremia, hypokalemia, and hypocalcemia are frequent and may need correction. Hyponatremia occurs in approximately 30% and may be due to the Cerebral Salt Wasting (CSW) syndrome or Syndrome of Inappropriate ADH secretion (SIADH). CSW is caused by secretion of brain and atrial natriuretic hormone and is associated with extracellular volume depletion and thus should be treated with fluid loading with normal saline and very occasionally hypertonic saline. SIADH reflects an excess of water and theoretically should be treated by fluid depletion but in SAH it is better to keep intravascular volume high, so salt containing IV solutions are usually used.

How are aneurysms treated? Small aneurysms (<7mm) which have never ruptured have a very low incidence of rupture, 0.05-1% per year, and may be managed with follow-up imaging to monitor growth. Aneurysms that have bled have a 10 fold higher annual incidence of bleeding and are thus best treated by elimination from the vasculature. Two approaches are currently used, surgical direct clipping of the aneurysm’s neck or endovascular coiling.

Surgery has been the standard treatment for the past 50 years and remains the current “gold standard”. Delayed surgery was the preferred option because it allowed brain swelling to subside, clot in the aneurysm to organize, and the period of maximal vasospasm to pass, but over the past decade early surgery has become the option of choice. Improved surgical and anesthetic techniques make brain swelling less of an issue and early surgery eliminates the risk of re-bleeding when triple H therapy is used for vasospasm.

Although endovascular approaches were attempted in Russia in the early 1970’s, it was not until a decade ago with the introduction of the Guglielmi detachable coils that this approach became a viable alternative. There has been a subsequent explosion of catheters and coils and more recently stents through which coils can be placed. The International Subarachnoid Aneurysm Trial (ISAT) has generated significant interest and controversy. The study involved patients with SAH where there was equipoise in treatment, i.e. either surgery or endovascular was deemed suitable by the care-givers and patients where one or other treatment was felt to be best were excluded. The study has published its two-year follow-up, although many of the patients have now been followed for considerably longer, up to 7 years. The number of patients dead or dependent significantly favors the endovascular group (23.5% vs. 30.9% p<0.0001), which also had less epilepsy. However complete occlusion was only achieved in 66% vs. 82% for surgery and the overall re-bleed rate was higher in the endovascular group compared to surgery, 52 vs. 41 patients. This study and other less controlled ones have already influenced clinical practice and many more patients are being treated by endovascular techniques.

Anesthetic management for surgery. There is substantial overlap in the anesthetic considerations for surgical and endovascular treatment, with obvious differences in the venue, potential blood loss and need for brain relaxation. Pre-procedural assessment should include neurological status, co-morbidities and complications (mentioned above). The clinical grade of the SAH correlates well with both the ICP and cerebrovascular reactivity. Patients with grade I or II SAH may be assumed to have normal ICP, intact cerebral autoregulation and a normal response to hyperventilation. Patients with grade III or IV SAH will have raised ICP, impaired autoregulation and reduced CO2-reactivity.

What am I trying to achieve? The goals of anesthetic management are to 1) control the aneurysm's transmural pressure gradient, whilst 2) maintaining adequate cerebral perfusion and oxygen delivery, and 3) avoiding precipitous changes in intracranial pressure. Anesthetic management should also maximize surgical exposure and reduce retraction on the brain, especially with early operation where the brain is often tense and edematous. The aneurysm's transmural pressure gradient (TMP) is equal to the pressure within the aneurysm (arterial blood pressure) minus the pressure outside/around the aneurysm (ICP). This is the same equation that describes cerebral perfusion pressure (CPP = MAP - ICP). This highlights the dilemma of balancing adequate cerebral perfusion against the risk of potential aneurysmal rupture. Until the aneurysm is clipped blood pressure should not be allowed to rise above the preoperative baseline. Patients with high grade SAH frequently have increased ICP. In addition, hematoma, hydrocephalus and a giant aneurysm may all serve to increase ICP. Management should not exacerbate this problem
but any reductions in ICP should be gradual, at least until the dura has been opened (ICP = 0) because sudden reductions in ICP produce acute increases in the aneurysm’s TMP.

**What monitoring is required?** All patients should have the following monitors – 5 lead EKG, intra-arterial blood pressure, pulse oximetry, capnography, esophageal/core temperature and neuromuscular blockade. Central venous and/or pulmonary pressures and TEE may be indicated in patients with significant heart disease. EEG and/or sensory/motor evoked potentials are monitored by some. There are no prospective human trials showing a benefit to neuromonitoring.

**What anesthetic agents should be used?** In general, the precise choice of anesthetic drugs used is less important than adherence to the principles outlined above. Induction should be smooth and faultless. Patients face a real risk of aneurysm rupture at the time of laryngoscopy and intubation and the hypertensive response to these stimuli must be attenuated. Other stimulating procedures include patient positioning and head-pin placement. All of these procedures should only be performed once adequate anesthetic depth, full muscle paralysis and control of the blood pressure are achieved. Propofol or thiopental together with an opioid and non-depolarizing muscle relaxant are most commonly used for induction. Scalp infiltration at the pin sites prior to their application is an easy way to attenuate the hemodynamic response. A TIVA technique may be preferred if ICP is markedly elevated and/or sensory evoked potentials are to be recorded. Inhalational agents at <1 MAC may also be suitable.

**How do I reduce brain bulk?** Reducing brain bulk improves surgical exposure, reduces brain retraction and facilitates clipping of the aneurysm. This is usually achieved by intravenous mannitol 0.5 - 1gm/kg sometimes with furosemide (10-20 mg IV). Peak mannitol effect occurs 30-45 minutes after infusion and the adequacy should be judged by the state of the brain and not the volume of urine. Mannitol increases CBF and reduces tissue water. The early reduction in ICP in autoregulating brain probably reflects a compensatory vasoconstriction to return CBF back to normal. Some also use CSF drainage via lumbar catheter. Drainage should be done slowly to avoid brain shift and hemodynamic changes and the volume should be limited as “brain sag” with worsening of neurological status occurs in up to 10%.

**Is induced hypotension still necessary?** A reduction in perfusion pressure of the feeding artery of the aneurysm decreases aneurysm wall stress and may facilitate surgical clipping. In addition, should rupture occur control of bleeding is easier. However, most neurosurgeons now use “local hypotension” through temporary clipping of the proximal feeding artery. Some surgeons like to use frequent short periods e.g. 3-5 minutes while others are concerned about vessel injury from frequent clip application and use 5-10 minutes. To maximize collateral blood flow, blood pressure should be kept in the normal range or slightly above. However when the temporary clip is removed one should avoid exposing an unsecured aneurysm to excessively high blood pressures.

**What should I use for brain protection?** The only randomized prospective trial in this type of surgery, the International Hypothermia Aneurysm Trial (IHAST), did not demonstrate any benefit to mild (33˚C) intraoperative hypothermia. No other putative protective strategies have been submitted to prospective randomized trials. Despite this some centers use a variety of anesthetic-based techniques. The most common is barbiturates or propofol given to achieve burst suppression. A technically skilled experienced neurosurgeon is without doubt the most important factor in achieving excellent outcomes.

**Recovery issues.** Hypertension is a frequent occurrence with emergence from anesthesia. Mild hypertension may be of benefit in augmenting cerebral perfusion, especially in patients where there is a concern about vasospasm. Blood pressure >20% above the patients normal level may be controlled with labetalol, esmolol or hydralazine as excessive rises in blood pressure may be associated with postoperative hemorrhage and edema. The acceptable blood pressure range should be established by discussion with the neurosurgeon. Patients should be awake and responsive as soon as is feasible to facilitate early neurological assessment and decisions about the need for CT scanning, angiography or the initiation of Triple H therapy, although some centers have now integrated the radiological assessment into their routine (intraoperative) practices. Patients who have had intra-operative complications or who were grade III or IV SAH preoperatively should be returned to the ICU intubated and ventilated.

**What do I need to know about AVM’s?** These occur one tenth as frequently as aneurysms. Presentation is usually at a younger age and the features are commonly intracranial hemorrhage or seizures. Current treatment is by endovascular occlusion, surgical resection or gamma radiation, often with a combination being used. As there is a
blood pressure drop from the artery to the nidus, they are much less prone to bleed with increases in blood pressure. Intraoperative care may be focused on replacing blood loss, depending on the adequacy of presurgical embolization. Post resection hyperperfusion of the resected region may occur and blood pressure should be kept normal or low for the first few post operative days.

**Is there a surgical treatment for intracranial occlusive cerebrovascular disease?** Anastomosing a branch of the external carotid artery, most commonly the superficial temporal artery, to a cortical branch of the middle cerebral artery is sometimes used in patients with atherosclerotic disease or with Moya-Moya. The Extracranial-Intracranial (EC-IC) By-pass Study examined intracranial atherosclerotic occlusions in a large randomized prospective study published in 1985. The operation was not found to reduce the frequency of death, stroke or transient ischemic attacks. However, controversy has continued and many vascular surgeons believe that there are subgroups of patients not adequately included in the original study who would benefit and also that the addition of best current medical therapy would also alter the conclusions. The ongoing Carotid Occlusion Surgery Study examines this operation in patients who have complete occlusion of one carotid.

**References:**


