CHAPTER 30

Cerebrovascular Disorders

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Learning Objectives

Upon completion of this chapter, the reader will be able to:

1. Explain the pathophysiology of arteriovenous malformations.
2. Describe the pathophysiology of cerebral aneurysms.
3. Compare and contrast the diagnostic modalities of arteriovenous malformations and cerebral aneurysms.
4. Develop a plan of care for patients with arteriovenous malformations and cerebral aneurysms.
5. List optimal patient outcomes that may be achieved through evidence-based management of cerebrovascular disorders.

Arteriovenous malformations (AVM) and cerebral aneurysms can cause subarachnoid and intracranial hemorrhage with devastating results. Prompt diagnosis and treatment by practitioners educated in cerebral vascular care are pivotal to providing appropriate interventions to optimize outcomes. This chapter will describe the incidence, pathophysiology, diagnosis, and treatment of AVMs and cerebral aneurysms.

ARTERIOVENOUS MALFORMATIONS

AVM Incidence

Since only 12% of AVMs cause symptoms, the incidence of AVM in the United States is not fully known. The incidence is thought to be around 1 per 100,000, equaling 300,000 cases. As technology advances and early detection increases, these numbers may rise. The average age of AVM diagnosis is 33 years, with 64% being identified before age 40 (Greenburg, 2001).

Untreated AVMs represent a threat to patients, because they have an annual major hemorrhage rate of anywhere from 2% to 17% (Bollet et al., 2004). AVMs account for 8.6% of all subarachnoid hemorrhages (SAHs) (Hickey & Buckley, 2003). The average rate of hemorrhage increases by 3% annually in unruptured vessels. This risk increases to 6% to 18% the first year after hemorrhage but has been shown to decrease to 4% annually thereafter (Greenburg, 2001; Greene et al., 1995). Although ruptured AVMs cause only 2% of all hemorrhagic strokes, the results can be devastating (Choi & Mohr, 2005). Lethal results from intracerebral AVM hemorrhages have been reported in as many as 29% of cases (Bollet et al.). Fortunately, thanks to advances in technology, twice as many AVMs are being identified before rupture than in years past (Choi & Mohr).

Pathophysiology

Normal cerebral vasculature includes arteries connecting to capillary systems that diminish the intravascular pressure before reconnecting to the veins. With AVMs, high-flow arterial blood shunts directly into low-resistance venous vessels. This tangled bundle of abnormal vessels possesses characteristics of thin or irregular muscularis and elastica, endothelial thickening, and islands of sclerotic tissue (Choi & Mohr, 2005). An AVM has three morphologic components: the feeding arteries, the nidus, and the draining veins. The feeding arteries supply blood flow to the AVM. The nidus is the main

tangle of connecting arterial and venous vessels. Dilated veins drain blood flow away from the AVM. Due to the vascular change from a high-flow system to a low-flow system, intravascular pressure is increased, predisposing the vessels to rupture.

The second effect of impaired perfusion is shunting of blood away from the surrounding brain tissue. Little to no functioning brain tissue within the lesion has been found, which leads to the assumption that functional displacement is pushed to the margins of the malformation (Choi & Mohr, 2005). The diversion of vascular blood to the AVM is called the "steal phenomenon." Theoretically, when blood flow into the AVM shunts blood away from surrounding brain tissue, it results in underperfusion and possibly ischemic brain in tissue beneath and around the AVM (Choi & Mohr; Iwama, Hayashida, Takahashi, Nagata, & Hashimoto, 2002).

AVMs are assumed to arise during fetal development. Vessels noted in utero suggest that their course may span over several decades, with some progressing, others remaining static, and a few regressing. AVMs are rarely familial (Choi & Mohr, 2005). Ninety percent of AVMs are supratentorially located, with 15% affecting deep locations (basal ganglia, brain stem, and corpus collosum).

Presentation

Eighty percent of AVM patients who present with symptoms do so between 20 and 40 years of age. The remaining 20% develop symptoms before age 20 (Hickey & Buckley, 2003). The most common clinical presentation for AVMs is intracerebral hemorrhage, which occurs in 50% to 60% of cases (Cockroft, Hwang, & Rosenwasser, 2005). Depending on the lesion's location and its angioarchitecture, the hemorrhage can be parenchymal, subarachnoid, intraventricular, or a combination of these. In patients presenting with hemorrhage, 30% are subarachnoid, 23% are intraparenchymal, 16% are intraventricular, and 31% are combined (Choi & Mohr, 2005). Seizure activity is seen in 30% of symptomatic patients (Cockroft et al., 2005), with headache reported in 11% to 14% (Choi & Mohr). In rare cases, evolving focal neurological deficits are seen as presenting symptoms. The onset and progression of symptoms has been proposed to be the result of "steal phenomenon" effects or local compression of tissue from the growing lesion. Direct compression of brain matter from the expanding AVM is also theorized to cause areas of localized ischemia (Choi & Mohr).

Diagnosis

Evaluation of these symptoms usually begins with neuroimaging studies. A computerized tomography (CT) scan of the head with and without contrast can reveal bleeding sites and brain tissue abnormalities, often with calcifications. More comprehensive analysis of the tangled blood vessels can be obtained via the injection of radioactive reagents into the bloodstream, followed by a magnetic resonance imaging (MRI) technique. This study can be used to further identify AVM location in comparison to surrounding brain structures. The gold standard for AVM imaging is four-vessel angiography. This invasive procedure involves threading a wire through a femoral artery catheter into the origin of the cranial vessels. A contrast reagent is then delivered close to the AVM site and examined under fluoroscopy imaging. Flow into and out of the vessels can be observed. Three-dimensional angiography is the latest technology in AVM diagnosis, which provides a 360-degree view of the feeder arteries, nidus, and venous outflow vessels. At present, no international standards or diagnostic algorithms for AVM detection exist (Choi & Mohr, 2005).

Treatment

The decision regarding whether and how to treat an evolving AVM depends on a variety of factors. These factors include the patient's age, medical condition, symptoms, AVM size, AVM location, and type of venous drainage (Nakaji & Spetzler, 2005). Additionally, the natural history of AVMs in general should be considered. Research data suggest that the hemorrhage rate of unruptured AVMs is approximately 3% per year (Nakaji & Spetzler). After hemorrhage, rebleed rates have been noted to increase (Cockroft et al., 2005). Mortality rates associated with episodes of bleeding are 10%, with an average neurologic morbidity of 20% (Nakaji & Spetzler). Given the relatively high morbidity and mortality associated with hemorrhage, elimination of AVMs is usually considered desirable.

Options for treatment currently fall into three categories: surgical resection, endovascular embolization, and radiosurgery. While surgical resection is a mainstay, AVM management generally requires multiple modalities and a team approach. Long-term risk versus immediate risk of various treatment options should be considered. Collaborative discussions with the patient among the neurosurgeon, interventional radiologist, and radiation oncologist, coupled with the underlying knowledge of practitioner skill and experience with lesions, will further guide treatment choices.

Surgical Resection

Research regarding optimal treatment for AVMs is ongoing. Currently, it is thought that the best candidates of surgical resection are patients with a good life expectancy, angiographic or clinical risk factors, small to medium-size AVMs (see **Table 30-1**) (Cockroft et al., 2005), good medical condition, positive symptoms, and AVMs anatomically located in surgically ac-

TABLE 30-1 Spetzler–Martin Surgical Grading Scale for Cerebral Arteriovenous Malformations

Category		Point Value
Size (maximal dimension)		
	< 3 cm	1
	3–6 cm	2
	> 6 cm	3
Location		
	Noneloquent brain	0
	Eloquent brain	1
Venous drainage		
	Superficial only	0
	Deep	1

Source: Greenburg, 2001.

cessible parts of the brain. Additional reasons to choose surgery are the AVM's association with aneurysms or venous outflow obstruction and a patient who has failed endovascular therapy or radiotherapy (Nakaji & Spetzler, 2005). An advantage of surgical treatment is the possible complete removal of the malformation in one operation. Surgical risks include perioperative hemorrhage, infection, brain edema, stroke, and death (Choi & Mohr, 2005).

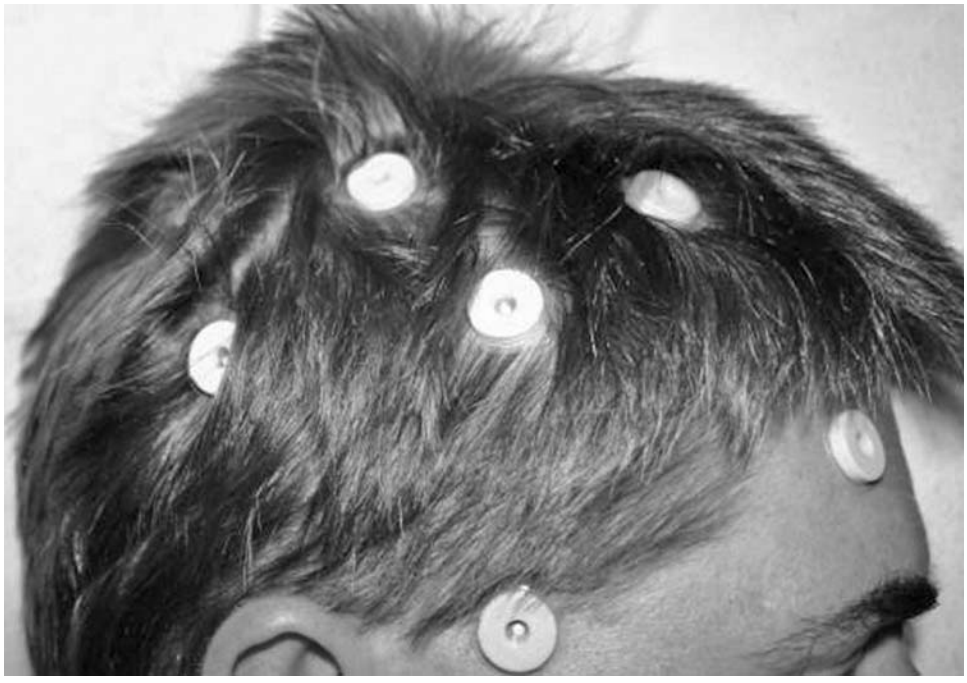
If chosen, surgical treatment may begin with an MRI with fiducial placement. Fiducials are circular discs that are placed on the patient's scalp prior to the MRI (see **Figure 30-1**). The location of the fiducials is processed by a stealth navigator computer, which calculates the three-dimensional location of the AVM. This image is then used at the time of surgery to help locate the malformation precisely, thus minimizing injury to the surrounding brain and maximizing lesion removal. Access to the AVM occurs via craniotomy bone removal. Once visualization occurs, excision of lesions using standard microsurgical techniques generally begins with the arterial feeders. Arterial feeder removal is then followed by excision of the nidus and resection of the draining veins. Intraoperative and/or postoperative angiogram is used to determine the presence of residual lesions. If present, residual lesions should be immediately resected or treated, utilizing alternative therapy to prevent vessel rupture.

Endovascular Treatment

The goal of endovascular therapy is to obliterate the feeding arteries and the vessels at the site of the nidus (Choi & Mohr, 2005). The first endovascular treatment of a cerebral arteriovenous malformation was performed in 1960 by injecting silastic spheres through surgically exposing the cervical carotid artery (Howington, Kerber, & Nelson, 2005). Due to the inadvertent occlusion of normal vessels and neurologic injury with this agent, assessment of various occlusion strategies to advance techniques continued. In 1974, Serbinenko succeeded in accessing cerebral arteries by using a detachable balloon mounted on a floating catheter (Hoelper, Hofmann, Sporleder, Soldner, & Behr, 2003; Serbinenko, 1974). While it offered improved results, this technique was not vessel-specific because the balloon was carried distally within the vessel with the most flow, and the balloon size precluded its entrance into the nidus (Howington et al.).

The use of particles as embolic agents for AVM treatment began in the 1970s. Since that time, embolic endovascular

FIGURE 30-1 Fiducial Placement



therapy has continued to evolve. Current agents include N-butylcyanoacrylate (NBCA), detachable coils, or Onyx® liquid polymer (Choi & Mohr, 2005). NBCA (Trufill®) is the most popular liquid agent and is the only "glue" approved by the U.S. Food and Drug Administration for use in cerebral AVMs. NBCA is a clear, colorless, radiolucent liquid that begins polymerization upon contact with blood saline and ionic contrast media (Cockroft et al., 2005). Onyx, a nonthrombogenic, liquid alcohol polymer, is another embolic agent currently being evaluated for efficacy in obliteration of AVMs. Coil therapy will be discussed under aneurysm treatment but also may be used in endovascular occlusion.

To achieve the goal of endovascular therapy, staged procedures over several days or weeks may be necessary to facilitate the gradual adjustment of vessels to pressure changes. Total embolization of AVMs occurs in 13% to 40% of patients (Choi & Mohr, 2005; Hartmann et al., 2002). Morphological characteristics of the AVM may cause embolization to be done as an adjunct to surgery or radiosurgery with the focus not being obliteration but rather reduction in the AVM size and bleeding risk (Choi & Mohr). Preoperative embolization should be done 24 to 48 hours prior to surgical intervention, because development of collateral flow into the nidus can occur within two days (Buckmiller, 2004).

Complications of intravascular AVM treatment can be characterized as ischemic, hemorrhagic, or groin related. Ischemic events occur due to glue emboli or catheter-induced dissection or vascular occlusion. Hemorrhagic complications can occur due to vessel rupture or inadvertent occlusion of the draining veins, which may result in too rapid an alteration in nidus hemodynamics and ultimately bleeding. Potential groin complications include infection and pseudoaneurysms. Mortality and morbidity rates of patients endovascularly treated for AVMs since 1990 are 1% and 8%, respectively (Cockroft et al., 2005).

Radiosurgery

The principle underlying radiosurgery is the use of focused radiation beams into selective tissue for ionization. Radiosurgery began in 1949 with the use of proton particles to irradiate brain tumor lesions. The "gamma knife" followed in 1968 and used cobalt-60 within a helmet device to direct gamma radiation to a specific area. Another type of radiosurgery called the LinAc was introduced in the mid-1980s. This device differs from the gamma knife in that radiation is emitted by a single source that rotates slowly around the patient's head. Ionization produces inorganic ions, which are deleterious to cells, secondary to the formation of free radicals that are harmful to cell and nuclear membranes. Irreparable damage ensues, resulting in permanent thickening of vascular channels, thrombosis, and cell death (Hickey & Buckley, 2003).

Due to limited studies demonstrating data on survival, quality of life, and neurologic progression-free survival, the efficacy of AVM treatment utilizing radiosurgery remains controversial (Bollet et al., 2004). Some have proposed observation of inoperable AVMs rather than nonsurgical treatment. The current opinion is that stereotactic radiosurgery may be a preferred treatment for patients with an AVM located in deep structures or eloquent cortex (i.e., motor strip) lesions.

AVM treatment invariably requires a multidisciplinary approach to care and treatment, and many factors need to be considered to determine the appropriate treatment in each case. One such factor evaluated when considering radiosurgery is AVM size. Cure rates after stereotactic radiosurgery decrease as the AVM volume increases. Reduction of AVM volume to less than 10 cm has been associated in case study with higher cure rates. In these situations, endovascular embolization or surgical techniques may be used to reduce the AVM size or eliminate certain angiographic features such as intranidal aneurysms (Cockroft et al., 2005). Aside from size, common risk factors for radiosurgery complications reported in the literature include location, previous hemorrhage, and irradiated volume (Bollet et al., 2004).

Concerns associated with radiosurgery include lag time between treatment and results (AVMs take one to three years for maximal shrinkage) and effects of radiation on healthy brain tissue. The appropriateness of radiation, total radiation dosage, and type of radiation delivered are determined through collaborative discussions between the neurosurgeon and the radiation oncologist. During these discussions, consideration is given to these potential concerns related to AVM size, location, age, and general health of the patient (Kemeny, Radatz, Rowe, Walton, & Hampshire, 2004).

Nursing Care

Admission of AVM patients into the intensive care unit (ICU) begins with an accurate report of presenting events and baseline neurologic function. Ongoing monitoring of neurologic changes occurs via frequent neurologic assessments. Hemorrhage prevention and symptom management—especially blood pressure control—are the focus of AVM nursing treatment. Whether or not the lesion was detected after an initial bleed, preventing bleeding focuses on seizure control, lifestyle modifications, and prevention of hypertension.

Blood pressure control can be achieved through medication administration as well as environmental control. Antihypertensives are ordered with a target systolic or mean arterial pressure listed as the focus of therapy. The postoperative

period can cause a phenomenon called "normal-pressure perfusion breakthrough." The theory is that changes in blood pressure and flow can cause postoperative swelling or hemorrhage due to loss of autoregulation (Greenburg, 2001). Minimizing pain through administration of narcotic or alternative treatments and controlling stress-inducing situations will also assist in blood pressure reduction.

Prophylactic antiseizure medication administration can occur but may be reserved until seizure activity is noted. Lifestyle modifications include smoking cessation and limitation of exertion until the lesion is controlled.

CEREBRAL ANEURYSMS

Aneurysm Incidence

The incidence of aneurysms is difficult to estimate. However, data suggest an incidence of 5% (Greenburg, 2001). Aneurysms can be classified as ruptured or unruptured. The unruptured to ruptured ratio is 5:6 to 5:3, equivalent to an approximate 50% rupture rate (Ogilvy & Carter, 2003). The incidence of ruptured cerebral aneurysms ranges from 6 to 16 per 100,000 (Khandelwal, Kato, Sano, Yoneda, & Kanno, 2005; Manno, 2004; Linn, Rinkel, & van Gijn, 1996), or approximately 25,000 to 30,000 SAHs from aneurysms annually in the United States (Khandelwal et al.; Menghini, Brown, Sicks, O'Fallon, & Wiebers, 1998).

Morbidity and mortality from ruptured aneurysms remain significant. Mortality from ruptured aneurysms has been reported as high as 50% (Khandelwal et al., 2005; van Gijn & Rinkel, 2001). Prehospital death is thought to be related to direct neural destruction and increased intracranial pressure from exceeding reasonable limits of blood, and sudden death from ventricular arrhythmias (Khandelwal et al.). Of patients who make it to institutions to receive care, 25% die within two weeks of their admission (Khandelwal et al.; Satoh, Nakamura, Kobayashi, Miyata, & Matsutani, 2005). Of the survivors, 20% to 30% live with significant neurologic deficits (Khandelwal et al.; Rosenorn et al., 1987). Therefore, a thorough understanding of aneurysm pathology, diagnosis, and management are necessary to minimize the impact of these events on patients' lives.

Pathophysiology

The exact mechanism of aneurysm formation is controversial. Cranial vessels are known to be less elastic and have less musculature. Additionally, larger cerebral vessels are located in the subarachnoid space with little connective support, which may predispose them to the development of aneurysms (Hop, Rinkel, Algra, & van Ginj, 1998). What is known is that aneurysms tend to arise from areas of vessel bifurcation. One theory is that hemodynamic stress over time causes degeneration of the vasculature (Hickey & Buckley, 2003). Atherosclerosis or hypertension may therefore predispose individuals to develop aneurysms. Consistent risk factors cited for aneurysmal SAH include hypertension, smoking, and alcohol consumption. If two first-degree relatives have aneurysms, the incidence of additional family members having aneurysms is 15% (Ogilvy et al., 2001). Increased risk of aneurysm development is also noted in first-degree relatives of persons with known lesions. Second-degree relative risk, however, is equal to that of the general public (Greenburg, 2001; Ogilvy et al.). Gender and ethnicity also play roles: Incidence seems to increase in females, and African Americans are twice as likely as whites to develop aneurysms (Hickey & Buckley).

Location of aneurysms may vary, with 85% occurring within the anterior circulation. The three most common locations are the anterior communicating artery (Acom; 30%), the posterior communicating artery (Pcom; 25%), and the middle cerebral artery (20%). Posterior circulation aneurysm can also occur, with 10% being located on the basilar artery and 5% occurring in the posterior inferior cerebral artery or vertebral artery. Multiple aneurysms are noted in 20% to 30% of the patient population (Zipfel, Bradshaw, Bova, & Friedman, 2004). **Figure 30-2** identifies the location of aneurysms.

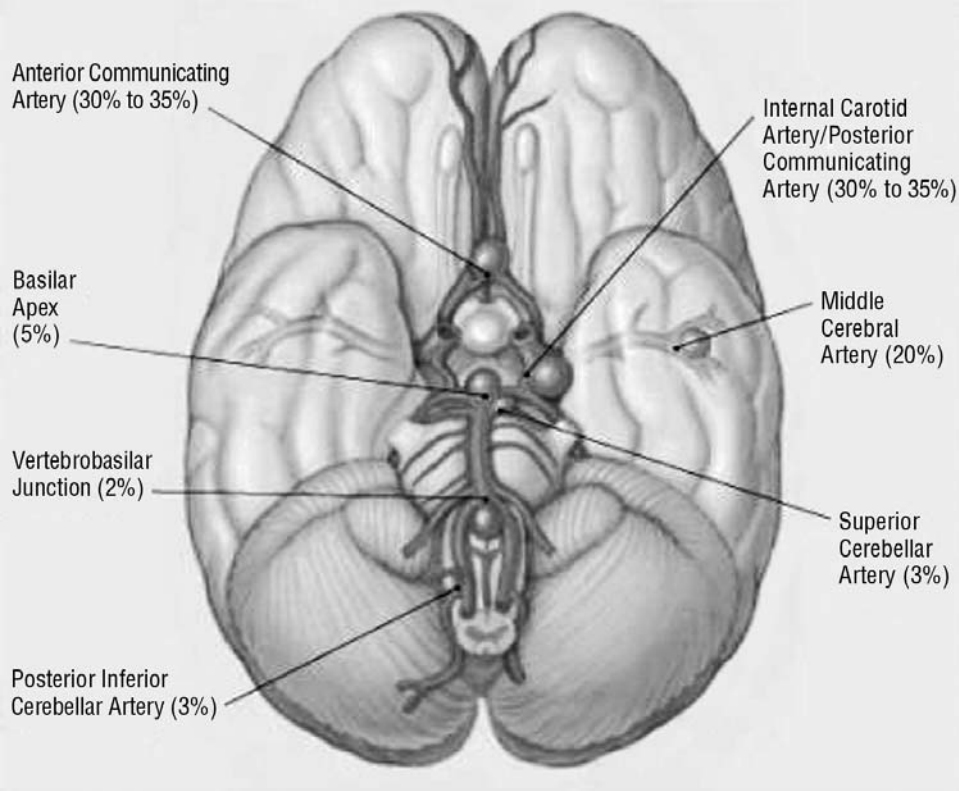
Cerebral aneurysms evolve into a variety of sizes and shapes. **Table 30-2** classifies these aneurysms by size. The most common aneurysmal shapes are berry or fusiform. Berry or saccular aneurysms are the most common type. These aneurysms have a neck or stem with a balloon-like outpouching. Berry aneurysms are most likely to be found at vessel bifurcations. Fusiform aneurysms are typically found in the vertebrobasilar system and are an outpouching without a stem.

Presentation

Presentation of patients with aneurysms can be separated into unruptured and ruptured cases. Most patients with unruptured aneurysms are completely asymptomatic. In approximately 40% of these cases, warning signs may be present. These localized symptoms may result from aneurysmal growth and compression on structures or intermittent, small leakage of blood (sentinel hemorrhage). Symptoms may include headache, third nerve palsies (i.e., dilated pupils, ptosis), extraoccular motor deficits (cranial nerves III, IV, and VI), vision changes, pain above and behind the eye, localized headaches, nuchal rigidity (neck pain with flexion), seizures, and photophobia (Greenburg, 2001).

Aneurysm patient presentation usually occurs as a result of hemorrhage. Usually this bleeding is subarachnoid, but it can also result in intracerebral hemorrhage (20–40%), intra-

FIGURE 30-2 Locations of Cerebral Aneurysms



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ventricular hemorrhage (13–28%), or subdural blood (2–5%) (Weir, Disney, & Karrison, 2002). The severity of presenting symptoms may correlate with the bleeding amount, but typical descriptions include thunderclap headache ("worst headache of my life") and nausea and vomiting with or without loss of consciousness. Additional symptoms may include cranial nerve deficits, stiff neck/neck pain, blurred vision, seizures, hypertension, bradycardia, and, depending on the area of cortex involved in the hemorrhage, localized motor weakness (Linn et al., 1996).

TABLE 30-2 Aneurysm Classification by Size

Small	≤ 10 mm
Medium	10–15 mm
Large	15–25 mm
Giant	25–50 mm
Super-giant	> 50 mm

Source: Greenburg, 2001.

Diagnosis

Evaluation of patients begins with a thorough patient history and a comprehensive neurologic exam. Once those data are obtained, diagnostic evaluation usually begins with a cerebral CT scan. For purposes of bleeding/aneurysm diagnosis, no contrast is needed. Because etiology is usually unknown upon presentation, initial evaluation will likely include CT scan with and without contrast. CT scans have been reported to have a 93% to 100% diagnostic sensitivity for identifying subarachnoid blood, but sensitivities have been found to correlate with the timing of CT obtainment relative to headache onset (Edlow & Caplan, 2000; Linn et al., 1996).

If CT findings are negative for blood and increased intracranial pressure is not suspected, lumbar puncture may be used to determine the presence of subclinical red blood cells (RBCs) and xanthrochromia (representing bile in the cerebrospinal fluid (CSF)). Discovery of RBCs in the CSF at times presents diagnostic difficulties, because "traumatic" lumbar punctures resulting in spillage of blood into the catheter and fluid occur in approximately 20% of cases (Linn et al., 1996). When this problem occurs, differentiation may be based on the presence or absence of xanthrochromia. Xanthrochromia develops in approximately 12 hours and generally takes two weeks to clear following an SAH (Linn et al., 1996). If the diagnosis is still unclear, additional studies are warranted and may include CT angiography (CTA), MRI/angiography, and angiograms.

Early CT scan techniques were insensitive to aneurysm detection. With the advances in spatial resolution and CTA, however, sensitivity to aneuryms has improved (Boesiger & Shiber, 2005). CTA utilizes a vein-injected contrast agent. An automatic injector machine is used to control the timing and rate of injection. After the injection, a rotating detector creates a fan-shaped beam of x-rays that is captured on film. With the advent of spiral CT technology, three-dimensional "casts" of the blood vessels are possible. Advantages of CTA

include being minimally invasive and offering a relatively quick turnaround time. Disadvantages include a lack of detection of smaller vessel abnormalities, potential allergic reactions, and nephrotoxicity from contrast agents.

MRI was introduced into clinical practice in the mid-1970s. MRI utilizes radio waves in a strong magnetic field. The magnetic field lines up protons, which are then spun by radiofrequency waves and produce signals. These signals are processed by the computer and ultimately result in sharp, detailed images. Contrast is generally used to highlight the vessel structures. Though it is thought to be more sensitive than CT in aneurysm detection (Mitchell, Gholkar, Vindlacheruvu, & Mendelow, 2004), MRI cannot be used in patients with implanted metal such as pacemakers or metallic ear transplants. In addition, given that MRI technology generally requires patients to tolerate confined spaces, patient size or claustrophobia may limit its use. Additionally, MRI will not detect small aneurysms (4 mm or less).

Cerebral angiography was introduced in 1927 (Boesiger & Shiber, 2005). The technology has advanced since then, such that the 360-degree angiogram represents the current gold standard for aneurysm detection. In this technique, the patient is usually sedated and may be intubated and anesthetized to minimize movement. Groin arteries are accessed utilizing a large-bore catheter. After arterial access is obtained, the neurologic radiologist threads a thin, flexible wire into the carotid-vertebral artery system. Contrast agents are then injected into the vessel, while images of contrast flow are monitored utilizing fluoroscopic techniques. The sensitivity and specificity of angiography are high and represent the standard against which other tests are judged.

Treatment

Aneurysm treatment includes measures taken both before and after definitive treatment by surgical and endovascular means. Before an aneurysm has been definitively treated, blood pressure control and symptom management are key. Target systolic and mean arterial pressure goals vary among institutions, physicians, and individual patients, with no evidence-based standard having been documented as yet. Hypertension avoidance is achieved through antihypertensive agents given intermittently or via continuous drip.

Symptom control begins with airway management, which may include mechanical intubation. Patients with a Glascow Coma Scale score less than 8 should be electively intubated to prevent aspiration pneumonia. Lidocaine may be used prior to intubation to depress the cough reflex and thus avoid increases in intracranial pressure. Circulation and hemodynamic stabilization are achieved with fluid therapy. Pain and nausea control through narcotic and antiemetic administration may be needed. In these circumstances, care should be taken to avoid oversedation to support continued neurologic assessment.

Seizure management is controversial but is usually recommended after a known seizure. Proponents of prophylactic management suggest that seizure onset may result in increased intracranial pressure and possible rebleeding.

Hydrocephalus occurs in approximately 20% of ruptured aneurysms as subarachnoid, intraventricular, or intracranial blood prevents CSF flow through the ventricular system (White, Teasdale, Wardlaw, & Easton, 2001). This complication may occur upon presentation or evolve within hours to days. Treatment requires placement of an intraventricular catheter for both CSF drainage and intracranial pressure monitoring. Lumbar drains may serve the same purpose but require further clinical evaluation before they can be recommended for all patients.

Admission electrocardiogram (ECG), chest radiograph, serum electrolytes, hematology panel, coagulation parameters, and type and cross matching are also included in the admission workup and preparation for potential diagnostic intervention and definitive treatment. Definitive treatment can be separated into surgical and endovascular modalities.

Surgical Techniques

Direct surgical clipping of intracranial aneurysms was first attempted in the 1930s, but mortality at that time was high (Boesiger & Shiber, 2005; White, Wardlaw, & Easton, 2000). Surgical clipping of aneurysms did not gain favor until the mid-1970s. Based on the scientific foundation from an international, randomized trial, surgically treated patients had a 6.5-year total mortality of 37% compared to 55% for the then standardized regulated bedrest group and 39.6% for the regulated bedrest with hypotension group (Boesiger & Shiber, 2005). Incremental reductions in surgical risk for ruptured intracranial aneurysms have since been achieved through enhanced microsurgical instruments and techniques, advances in intensive and anesthesia care, improved diagnostics, and the development of neurosurgery as a subspecialty (Molyneux et al., 2005).

Intracranial clipping is achieved through a craniotomy. Microdissection down to the aneurysmal lesion may be aided by the placement of a lumbar drain or a ventricular catheter. The drain may be placed to evacuate CSF to aid in microdissection. Once visualized, the neurosurgeon places a surgical clip at the neck of the aneurysm or feeding artery if the aneurysm itself is unclipable and the risk of permanent neurologic impairment is absent or considered to be less than the risk of re-rupture. When clipping is not possible due to

aneurysmal anatomy, surgical wrapping using fibrin glue, Teflon®, or other polymers may occur (Greenburg, 2001).

The benefits of surgical techniques include direct access in case of aneurysm rupture and definitive resolution of the aneurysm. Permanent aneurysm eradication utilizing surgical techniques occurs in more than 90% of patients, with morbidity and mortality of surgical treatment estimated at 5% to 15% (Wijdicks, Kallmes, Manno, Fulgham, & Piepgras, 2005). **Figure 30-3** displays the surgical clipping of an aneurysm. Due to its relatively low complication rate and its ability to promote clot evacuation, microsurgery has been established as the gold standard for aneurysm treatment.

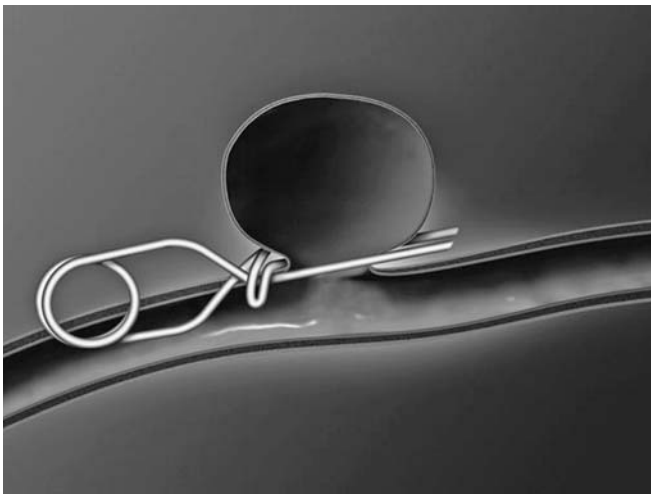
Timing of surgery remains controversial, however. In patients with a Hunt–Hess Grade of 4 to 5 (**Table 30-3**), a period of stabilization (usually more than 10 to 14 days post SAH) is recommended. The argument for delay in such cases revolves around the presence of a solid clot (which is more difficult to remove), brain edema (which would require more brain manipulation to obtain aneurysm access), potentially increased risk of aneurysm rupture, and possibly increased vasospasm risk following surgery secondary to vessel manipulation (Greenburg, 2001). Factors supporting delayed surgery include poor medical condition, poor neurologic condition (Hunt–Hess Grade 4 or greater), significant cerebral edema, active vasospasm, and difficult-to-clip aneurysms (Greenburg, 2001). Proponents of early treatment for patients who present with SAH believe that this approach eliminates subsequent bleeding, facilitates vasospasm treatment, and enables cerebral lavage to enhance elimination of potential vasospasmotic agents (Le Roux et al., 1995). Factors that favor choosing early intervention include good medical and neurologic condition, large amounts of SAH that increase the likelihood of subsequent vasospasm development, and large amount of clot with effacement of tissue (Greenburg, 2001).

TABLE 30-3 Hunt–Hess Subarachnoid Hemorrhage Classification

Grade	Description
0	Unruptured aneurysm
1a	No acute meningeal/brain reaction but fixed neurologic deficit
1	Asymptomatic, or mild headache or slight nuchal rigidity
2	Cranial nerve palsy (i.e., III, VI), moderate to severe headache, nuchal rigidity
3	Mild focal deficit, lethargy, or confusion
4	Stupor, moderate to severe hemiparesis, early decerebrate rigidity
5	Deep coma, decerebrate rigidity, moribund appearance

Source: Greenburg, 2001.

FIGURE 30-3 Surgical Clipping of an Aneurysm



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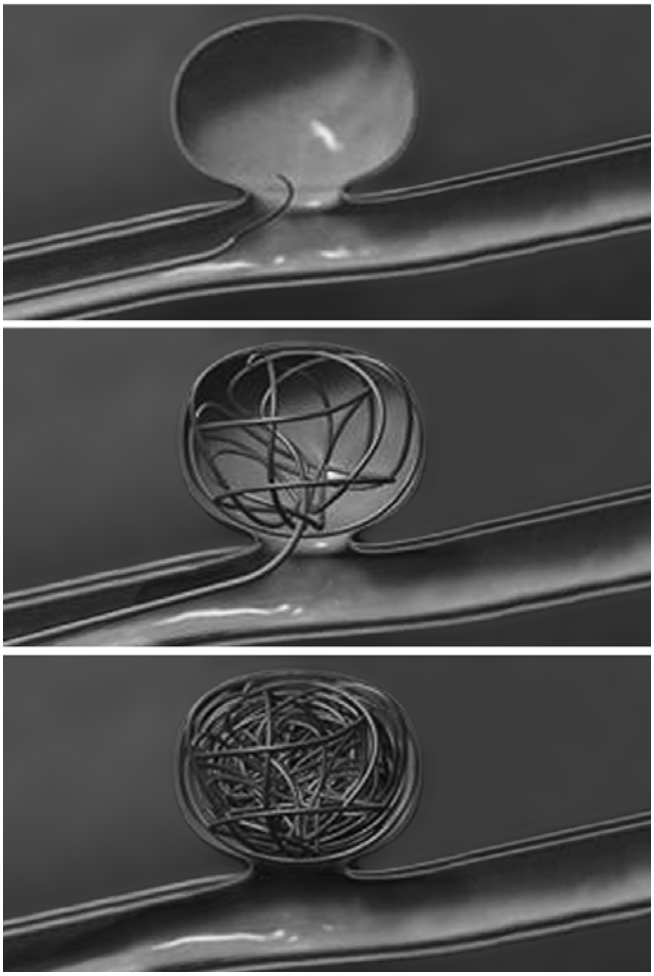
Endovascular Techniques

Due to the invasive nature of cranial surgery and advancement of endovascular technology, debate regarding the optimal aneurysm treatment continues. The results of the International Subarachnoid Aneurysm Trial added more fuel to this debate. This study demonstrated that 23.7% of endovascularly treated patients were dependent or dead at one year versus 30.6% of surgical patients (Molyneux et al., 2005).

The introduction of the Guglielmi detachable coil in 1991 revolutionized cranial intravascular treatment. Continued enhancements such as the advent of soft and three-dimensional (3-D) coil technology and cranial stents have made coiling possible in lesions previously considered beyond the realm of intervention (Wiebers et al., 2003). **Figure 30-4** shows the use of coiling in an aneurysm.

When treating the aneurysm, patients are anesthetized to minimize motion during the delicate portions of coil placement or vessel sacrifice via balloon occlusion. If continuous neurologic evaluation is needed, a patient may be awakened and given sedative and analgesic agents. Access of the groin occurs, utilizing a femoral sheath. A guide catheter is then placed

FIGURE 30-4 Staged Endovascular Coiling Utilizing 3-D Coils



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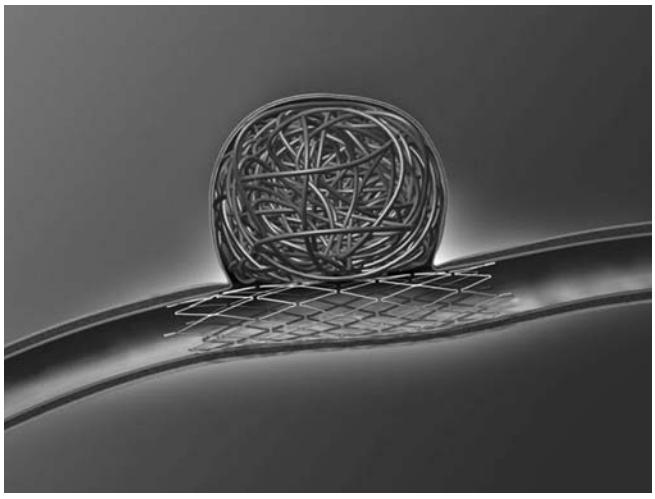
in the target vessel, with care being taken to avoid contact with the aneurysm wall. After matching the aneurysm diameter and coil properties, device selection occurs. Coil systems generally consist of a thin, spiral-woven, platinum, helix-shaped wire soldered to a stainless steel delivery system. Inside the delivery system, coils are straight; due to circular memory, however, they will resume the helix shape once deployed into the aneurysm. More elaborate coils include two-diameter, complex 3-D configuration, Dacron fibers, and bioactive technologies. The purpose of each coil is to enhance placement and promote thrombus occlusion of the aneurysm. Multiple coils are needed to pack the aneurysm and achieve occlusion.

Stent-assisted coiling is a relatively new technique that serves as a buttress to the coil. The balloon-expanded or self-expandable stent is placed outside the aneurysm neck and supports the implanted coils from slipping into the vessel. (See **Figure 30-5**.) Concerns regarding stent usage include induction of intimal hyperplasia or occlusion of small side branches. To minimize vessel occlusion, patients who are coiled with or without stenting are generally placed on aspirin with or without clopidogrel (Plavix®) once the aneurysm has been occluded. This is true even if the patient orginally presented with bleeding. Liquid embolic agents (i.e., Onyx®) may also be used for aneurysm occlusion, though coiling remains the gold standard.

Benefits of coiling include its less invasive nature, decreased system stress, and decreased length of stay. Ongoing analysis of the permanency of coiled aneurysm is needed, however. Complications of coiling include ischemic events secondary to coil herniation with thrombus formation or with distal embolization, aneurysm rupture or perforation, and groin complications. To prevent or minimize catastrophic consequences of intervention, care should be delivered in centers that focus on neurologic intervention to enable prompt management by skilled practitioners (Wiebers et al., 2003).

Patient selection for intervention versus surgical treatment requires a collaborative discussion between the neurosurgeon and the interventional radiologist. Characteristics considered in the decision-making process include aneurysm size, dome-to-neck ratio, Hunt–Hess grading, patient age, comorbidities, surgical accessibility, and practitioner skill.

FIGURE 30-5 Endovascular Stenting Prior to Aneurysm Coiling



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Nursing Care

Once treatment is provided, patient care focuses on early identification and prevention of neurologic sequelae. In nonhemorrhagic aneurysm patients, care consists of frequent cranial nerve and motor strength evaluations in the neurologic ICU. Post-procedure angiography may occur the following morning or during a follow-up visit several months to weeks after the procedure. Timing of subsequent angiograms is not standardized, but intraoperative angiograms are a current trend (Wiebers et al., 2003). If no neurologic changes occur, patient activity can progress with patient discharge occurring within several days of admission. Education related to wound care (groin or cranial), signs of neurologic dysfunction, lifestyle changes, and activity limitation should occur. Blood pressure management may also need to be monitored or treated in the outpatient arena.

Hemorrhagic aneurysmal patients are at risk for a variety of complications, including vasospasm, hyponatremia, neurogenic pulmonary edema, cardiac dysfunction, and chronic hydrocephalus. Each of these sequelae is associated with its own set of treatment strategies.

Vasospasm

Cerebral vasospasm has been described as sustained arterial contraction that is unresponsive to vasodilator medications (Oyama & Criddle, 2004). Vasospasm-induced narrowing has been estimated to occur in 70% to 90% of SAH patients. Symptomatic vasospasm occurs in only 30% of cases (Brislstra, Algra, & Rinkel, 2002; Hanel, Demetrius, & Wehman, 2005) and has an associated mortality of 7% (Levati, Solaini, & Boselli, 1998; Rosen, Sekhar, & Duong, 2000); severe deficits are noted in an additional 7% of cases (Sen et al., 2003). Vessel narrowing is defined as radiographic or clinical (symptomatic). Radiographic vasospasm occurs when visible narrowing utilizing contrast injection under angiographic observation is noted. Clinical or symptomatic narrowing develops accompanied by functional manifestations dependent upon the cerebral area affected and the degree of ischemia. Symptomatic vasospasm assessment findings range from headache, lethargy, and intermittent disorientation to hemiparesis and permanent disability (Rosen et al., 2000).

Vasospasm pathology is poorly understood. The process is self-limited. It generally begins no sooner than 3 days after SAH and resolves within 21 days. Despite our currently limited understanding of its pathology, vasospasm development can be predicted based on a variety of factors—namely, the amount and location of blood, with a higher incidence seen in Fisher Group 3 (see **Table 30-4**), increasing patient age, and history of tobacco use (Greenburg, 2001).

TABLE 30-4 Fisher Subarachnoid Hemorrhage Classification

Group	Description
Group 1	No detectable blood on CT
Group 2	Diffuse or vertical blood layers < 1 mm thick that do not appear dense enough to represent a large, thick homogeneous clot
Group 3	Localized clot greater than 1 mm thick in vertical plane or greater than 5×3 mm in longitudinal and transverse dimensions in the horizontal plane
Group 4	Intracerebral or intraventricular clots, but with only diffuse blood or no blood in basal cisterns

Source: Greenburg, 2001.

Diagnosis of vasospasm begins by ruling out other potential causes, such as hydrocephalus, cerebral edema, seizure activity, hyponatremia, hypoxia, and sepsis. Onset generally occurs between 4 and 14 days post SAH. Although the gold standard of testing is cerebral angiogram, large-vessel spasm may also be detected utilizing transcranial Doppler (TCD). TCD is a noninvasive cerebral artery velocity evaluation. Utilization of hand-held Doppler technology through temporal bone windows enables monitoring of large cerebral vessels. Because major vessels are the only arteries assessible with this technology, TCD should be used as a screening tool and angiograms employed as the definitive form of evaluation.

Once diagnosed, or if increased risk is suspected, several treatment options can be implemented to prevent or minimize sequelae from vasospasm. An initial prevention strategy is the use of nimodipine (Nimotop®). This calcium channel blocker is the only pharmacologic agent found useful in vasospasm treatment (Kassell et al., 1990). The dose is 60 mg orally every four hours; if hypotension occurs, 30 mg every two hours may be given for 21 days.

Hyperdynamic or triple-H therapy is another vasospasm treatment option. The use of hypertensive therapy as a treatment against vessel narrowing was first noted in 1951. Further evaluation of this concept was not achieved until the late 1960s, when the use of volume expanders and vasopressors to raise blood pressure were noted to reverse or minimize neurologic symptoms (Molyneux et al., 2005). More widespread use of triple-H therapy began with the "early treatment of aneurysm" trend. In the late 1970s, a small cohort of patients with symptomatic vasospasm was treated with colloids and phenylephrine (Neosynephrine®) to induce hypertension, and their

neurologic deficits were successfully reversed. In 1982, the concept of hemodilution as a vasospasm treatment was introduced (Rosen et al., 2000). This theory proposed that by utilizing colloids, blood viscosity could be lowered and cerebrovascular resistance thereby decreased, with resultant blood flow increase. Balancing the oxygen carrying capacity with improved flow, a hematocrit of 30% was proposed as ideal. Evidence to support this theory has yet to be obtained, making this treatment controversial.

After definitive aneurysm treatment, benefit has been demonstrated with systemic blood pressure elevation using volume expansion and ongoing blood pressure support (Greenburg, 2001). Target blood pressures are controversial, because the patient's baseline pressure needs to be taken into account (Rosen et al., 2000).

Several risk factors from triple-H therapy warrant consideration when initiating care. Approximately 10% to 20% of patients with SAH will develop pulmonary edema, especially when they are given crystalloid volume expansion (Rosen et al., 2000). Dilutional hyponatremia of less than 135 mEq/L is seen in 3% of patients and myocardial infarction in 2%; catheter-related complications from pulmonary artery catheters (sepsis, 13%; subclavian vein thrombosis, 1.3%; and pneumothorax, 1%) are also seen (Rosen et al., 2000). Therefore, care should be taken when initiating therapy, although no specific standards related to timing or appropriateness of interventions currently exist.

Once vasospasm is detected, additional pharmacologic and mechanical treatment options are available. Intra-arterial papaverine (Para-Time® SR) or verapamil hydrochloride (Calan®) may be given during an angiogram to provide short-term vasospasm relief. Because effects last for only a few hours, the patient may require multiple interventions over several days despite the risk of the invasive procedure. In addition to pharmacologic treatment, mechanical options are available.

Percutaneous balloon angioplasty may be needed in severe vasospasm. Similar to cardiac angioplasty, this technology involves threading a flexible catheter through the arterial system into the position of spasm. Once placed, the pressure-controlled balloon can be inflated with resultant displacement of previously narrowed vessel walls. Procedural risks include arterial occlusion, rupture, or dissection. Use of this technology requires large cerebral vessels and the services of an interventional radiologist trained in cerebral procedures (Greenburg, 2001).

The current strategies of calcium channel blockers and triple-H therapy have reduced mortality and morbidity rates of vasospasm from 20% in the 1980s to the current rate of 5% to 10% (Corsten et al., 2001). Advances in technology and pharmacology continue to be explored in an effort to further decrease the incidence of clinically significant vasospasm. Additional therapies requiring more study include the use of microdialysis, mild hypothermia (32–34°C) (Rosen et al., 2000), high-dose (4–5.5 mg/dL) magnesium sulfate therapy, transcranial cerebral oximetry, and molecular biology. All have demonstrated promise for vasospasm diagnosis or treatment (Nagao, Irie, & Kawai, 2003).

Hyponatremia

Hyponatremia affects 10% to 40% of patients with SAH. This condition is defined as a sodium level of less than 135 mEq/L for at least a day. Signs of hyponatremia include fever, headache, nausea and vomiting, muscle cramps, weakness, and confusion. As values drop below 110 mEq/L, stupor, seizures, and coma may occur. Several theories have been suggested to explain the link between SAH and hyponatremia. One proposes a transient release of antidiuretic hormone, which results in Syndrome of Inappropriate Antidiuretic Hormone secretion, and a dilutional drop in sodium. Another theory, which is more widely accepted, is based on the fact that atrial natriuretic factor rises and stimulates urinary loss of sodium (cerebral salt wasting). Neurologic dysfunction may occur, with hyponatremic patients having three times the incidence of delayed cerebral infarction after SAH than normonatremic patients (Gasser, Khan, & Yonekawa, 2003). Factors that increase the likelihood of hyponatremia include congestive heart failure, cirrhosis, adrenal insufficiency, diabetes, and the use of nonsteroidal inflammatory drugs, acetaminophen, narcotics, and thiazide diuretics (Veyna, Seyfried, & Burke, 2002).

Treatment of SAH-related hyponatremia differs from that provided to the general population. Fluid restriction (a usual treatment) in this population may result in increased blood viscosity and may result in ischemia from vasospasm (Gasser et al., 2003). Instead, treatment with normal or hypertonic saline, sodium tablets, or fludrocortisone acetate should be used.

Regardless of the cause, hyponatremia should be corrected slowly. If done too rapidly, the patient can be placed at risk for rebound cerebral edema. To prevent this complication, correction should not exceed a rate of 1.3 mEq/L/hr or more than 10 mEq/L in 24 hours. Frequent monitoring of chemistry values is also necessary to react to complications in a timely manner.

Neurogenic Pulmonary Edema

Massive sympathetic outflow may mediate the development of extravasation of plasma proteins across the pulmonary parenchyma (Linn et al., 1996). This results in an acute form of pulmonary edema, which may occur at the moment of SAH or within several days of injury. Reversal of this phenomenon

occurs by itself, but ventilatory support is generally needed in the short term (Linn et al., 1996).

Cardiac Dysfunction

Cardiac abnormalities with acute ECG changes are noted in almost half of patients with SAH. Presentation can occur at the time of SAH or as long as two weeks into the clinical course (Linn et al., 1996). Abnormalities can present as inverted T waves, any variety of dysrhythmias, or lethal variations that may result in sudden death. Cardiac enzyme elevation may occur and is frequently associated with myocardial dysfunction and subendocardial ischemia.

Echocardiogram analysis may demonstrate significantly lowered ejection fractions with myocardial motionlessness. This dysfunction may present much like heart failure or respiratory distress syndrome. Unlike the cardiac ischemic changes seen in coronary artery disease, this "stunned myocardium" is usually reversible (Linn et al., 1996). Support for the patient experiencing this complication may include inotropic therapy, pulmonary artery monitoring, and ventilatory support as needed.

Hydrocephalus

Due to variations in definitions, the stated incidence of acute hydrocephalus ranges from 20% to 60%, with a more commonly stated range of 15% to 20% (Greenburg, 2001). Fortunately, 30% to 60% of these patients demonstrate no alteration in consciousness. Acute hydrocephalus may convert to chronic hydrocephalus when arachnoid granulations develop adhesions or permanent impairment. While not all patients convert to chronic hydrocephalus, the phenomenon does occur in 50% of SAH patients. In such a case, CSF diversion devices should be placed after post-hemorrhage protein and RBC counts decrease to avoid catheter occlusion.

General Care

Due to the generalized total body stress associated with SAH, gastric ulcer stress prophylaxis should be undertaken in all these patients. Additionally, nutrition in some form should be initiated as soon as clinically possible. Patients' relative immobility should make constipation and deep venous thrombosis prophylaxis a standard of care. Stool softeners should be given to all patients, with constant surveillance of bowel activity. A minimum of sequential compression devices should occur. Controversy exists regarding the use of unfractionated or low-molecular- weight heparin products in this population, but anticoagulation is generally avoided. Activity progression should occur when the patient is clinically able. Collaborative involvement of disciplines such as physical, occupational, and speech therapy may be required, depending on the degree of neurologic impairment.

Familial Education

Family education should be ongoing throughout the patient's hospitalization. Due to the acute nature of most patient admissions, nurses can expect to have to repeat instructions and explanations of the plan of care multiple times. Compassionate inclusion of family members will minimize stress.

PATIENT OUTCOMES

Nurses can help ensure attainment of optimal patient outcomes such as those listed in **Box 30-1** through the use of evidence-based interventions for cerebrovascular disorders.

SUMMARY

The outcome of AVM or aneurysm rupture can range from life-changing to death. Minimization of the impact of vascular malformations on patients' lives can occur with prompt diagnosis and treatment. A multidisciplinary approach to treatment includes a variety of informed clinicians, including ICU nurses, neurosurgeons, neuroradiologists, and radiation oncologists, in institutions where the latest advances in treatment can be offered. Future research will continue to focus on refinement of treatment options from surgical techniques, interventional occlusion catheters and devices, and radiosurgery techniques.

Early diagnosis and obliteration of cerebral vascular malformations in tertiary centers that focus on their treatment are needed to minimize neurologic consequences. Collaborative care between neurosurgeons, neuroradiologists, critical care nurses, and multidisciplinary team members will assist the patient and family in achieving their new level of wellness. To collaborate fully, ongoing research and awareness of AVM and aneurysm treatment are necessary.

Box 30-1

Optimal Patient Outcomes

1. Cognitive status in expected range
2. Patient and family participate in planning/providing care
3. Physical comfort in expected range
4. Decreased frequency of vasospasm
5. Remains calm and tranquil
6. Family uses stress reduction strategies

CASE STUDY

After having surgery to repair a torn knee ligament, T.F., a 32-year-old male, started experiencing global headaches. Because T.F.'s only history was asthma related to smoking, the original diagnosis was spinal headache from the spinal block he received for knee surgery.

The patient's headache persisted for several months, with an exacerbation prompting his visit to the Emergency Department. Because he lived alone, T.F. was driven to the hospital by his parents. His head CT scan was negative for blood but demonstrated calcified lesions in his left parietal region. Admission vital signs were T 98.4 °F, HR 88, BP 168/90, RR 16, and SpO_2 94% on room air. He rated his global headache as 8/10.

T.F. was admitted to the neurologic critical care (NCC) unit for hourly vital sign and neurologic observation, and for pain and blood pressure control. A cranial MRI with and without contrast demonstrated what appeared to be an AVM. A four-vessel cerebral angiogram done later in the day verified the diagnosis. T.F. was then prepped for a follow-up angiogram for occlusion of the AVM. The following day, T.F. underwent Black Onyx occlusion of his AVM with 90% occlusion. Despite being educated on the smell omitted from Black Onyx, T.F. was nervous about the potential reactions of others to the odor.

Post-procedure care included frequent neurologic and sheath/groin checks, pain control, and vital sign management. The sheath remained intact for intraoperative usage to complete the AVM occlusion.

On the morning of surgery, T.F. received a stereotactic localizing MRI with fiducials. Utilizing the stereotactic navigational system, the neurosurgeon obtained access through a cranial incision. After complete resection of the AVM confirmed by an intraoperative angiogram, T.F. returned to the NCC. Hourly vital sign and neurologic checks and groin care occurred throughout the night.

The next morning, T.F. was doing well. His postoperative cranial wrap was removed, demonstrating an incision that was clean, dry, and intact with staples. His IV was saline locked, and the urinary catheter and arterial line discontinued. T.F. moved to the floor with vital signs being taken every four hours. He was evaluated and released and returned to his home on the second postoperative day. His home instructions included smoking cessation, pain medication, incisional care, activity progression, and follow-up instructions with the neurosurgeon and neurologic interventionalist.

CRITICAL THINKING QUESTIONS

1. As the nurse caring for this patient, what information would you give the family when they state, "We have never heard of an AVM. What is this?"
2. The family asks how an AVM is treated. What would be the best response?
3. After receiving Black Onyx to partially occlude an AVM, the patient complains of a headache without focal neurologic signs. What is the probable source of his headache?
4. What postoperative problems should you be assessing for with a patient who has undergone surgery for an AVM?
5. Prior to discharge, how would you plan to transition the patient to a neuro step-down unit?
6. Which disciplines should be consulted to work with this client?
7. How would you modify your plan of care for patients of diverse backgrounds?
8. What type of issues may require you to act as an advocate or moral agent for this patient?
9. How will you implement your role as a facilitator of learning for this patient?
10. Use the form to write up a plan of care for one client in the clinical setting with a cerebral aneurysm or AVM. Rate the patient as a level 1, 3, or 5 on each characteristic. Identify the level of nurse characteristics needed in the care of this patient.
11. Take one patient outcome for a patient and list evidence-based interventions.

Using the Synergy Model to Develop a Plan of Care

SYNERGY MODEL

Patient Characteristics	Level (1, 3, 5)	Subjective and Objective Data	Evidence-based Interventions	Outcomes
Resiliency				
Vulnerability				
Stability				
Complexity				
Resource availability				
Participation in care				
Participation in decision making				
Predictability				

Online Resources

National Organization of Vascular Anomalies: www.novanews.org/vascularmalformations.htm

Brain, arteriovenous malformation: www.emedicine.com/radio/topic93.htm

Timing of surgery for aneurysmal subarachnoid haemorrhage (Cochrane Review): www.cochrane.org/cochrane/revabstr/ab001697.htm

Calcium antagonists for aneurysmal subarachnoid haemorrhage (Cochrane Review): www.cochrane.org/cochrane/revabstr/ab000277.htm

Subarachnoid hemorrhage: www.emedicine.com/neuro/topic357.htm

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