

# Care of the Patient with Aneurysmal Subarachnoid Hemorrhage

AANN Clinical Practice Guideline Series



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## Preface

To meet its members' needs for educational tools, the American Association of Neuroscience Nurses (AANN) has created a series of guides to patient care called the AANN Clinical Practice Guidelines. Each guide has been developed based on current literature and is built upon evidence-based practice.

The purpose of this document is to assist registered nurses, patient care units, and institutions in providing safe and effective care to patients recovering from aneurysmal subarachnoid hemorrhage (aSAH).

The personal and societal impact of aSAH is significant with some 30,000 Americans suffering aSAH each year. Aneurysmal SAH occurs across the lifespan with risk increasing with increased age. The mean age of individuals suffering aSAH is 55 years old. Individuals of all races and ethnic backgrounds suffer aSAH equally. Approximately 50% of individuals suffering aSAH do not survive the initial injury. Of those who do survive, an additional 30%–50% will suffer a secondary injury from one or more of the following: rebleed, cerebral edema, increased intracranial

pressure, or cerebral vasospasm (the most common complication of aSAH). The end result of primary and secondary injury from aSAH is a high rate of mortality and disability.

When a patient suffers aSAH, neuroscience nurses play a pivotal role in patient monitoring and management of care to prevent secondary injury thereby improving outcomes. Resources and recommendations for practice will provide neuroscience nurses with a tool to maximize outcome of individuals suffering aSAH and secondary sequelae.

This reference is an essential resource for neuroscience nurses responsible for the care of this patient population with a multitude of biopsychosocial needs. This guide is not intended to replace formal learning, but rather to augment the knowledge base of clinicians and provide a readily available reference tool.

Neuroscience nursing and AANN are indebted to the volunteers who have devoted their time and expertise to this valuable resource, created for those who are committed to neuroscience patient care.

## I. Introduction

### A. Purpose

The purpose of this document is to assist registered nurses, patient care units, and institutions in providing safe and effective care to adults recovering from aneurysmal subarachnoid hemorrhage (aSAH). The goal of the guideline is to provide background on the biological processes occurring during and after rupture of a cerebral aneurysm and provide evidence-based guidelines for providing nursing care to this population.

### B. Rationale for Guideline

The impact of aSAH is significant, affecting people of all ages, races, and genders. Recovery from aSAH is complicated by secondary injuries, some specific to individuals recovering from this disease process. The mortality and disability rates for the aSAH population are high. Nurses providing quality care based on empirical evidence with a focus on preventing secondary injury will maximize recovery for this population.

### C. Goals of Clinical Practice Guidelines

When presented with a patient with a possible aSAH, it is imperative that nurses and other healthcare professionals are able to recognize the underlying clinical components, understand the severity of the situation, initiate early treatment, and act judiciously in order to prevent secondary complications and further deterioration in this relatively infrequent and often misdiagnosed clinical encounter. The goals for caring for a patient with aSAH are as follows:

- early recognition and accurate diagnosis
- stabilization of the aneurysm
- prevention of complications
- early recognition of complications
- treatment
- rehabilitation.

### D. Assessment of Scientific Evidence

A review of the published literature from January 1982 to November 2006 was conducted using Medline/PubMed, CINAHL, and Evidence-Based Medicine Reviews using the following search terms: *subarachnoid hemorrhage, cerebral vasospasm, management, and outcomes*. Monographs, textbooks, and review articles were also consulted. Studies not directly pertaining to aSAH or not written in English were excluded from further evaluation.

For the AANN Clinical Practice Guidelines, data quality is classified as follows:

- Class I: Randomized control trial without significant limitations or metaanalysis
- Class II: Randomized control trial with important limitations (e.g., methodological flaws or inconsistent results), observational studies (e.g., cohort or case-control)

- Class III: Qualitative studies, case study, or series
- Class IV: Evidence from reports of expert committees and/or expert opinion of the guideline panel, standards of care, and clinical protocols

The Clinical Practice Guidelines and recommendations for practice are established based upon the evaluation of the available evidence (AANN, 2006, adapted from Guyatt & Rennie, 2002; Melnyk, 2004):

- Level 1 recommendations are supported by class I evidence.
- Level 2 recommendations are supported by class II evidence.
- Level 3 recommendations are supported by class III and IV evidence.

## II. Statement of the Problem

Aneurysmal subarachnoid hemorrhage (aSAH) is hemorrhagic stroke whereby blood from the vasculature enters the subarachnoid space. Saccular or berry aneurysms, the most common type of cerebral aneurysms, are acquired lesions that develop at vessel bifurcations or branching points in the cerebral vasculature that resemble small, thin-walled blisters. Other types of aneurysms include fusiform aneurysms (also called atherosclerotic aneurysms) or dissecting aneurysms (because of a tear in the vessel wall). Aneurysms typically form in the bifurcations of the large vessels that make up the circle of Willis. When one of these vascular lesions ruptures, blood leaks into the subarachnoid space and is known as an aSAH. Cerebral aneurysms are thought to arise from defective layers of arterial lamina and tunica media from which an outpouching or ballooning of the vessel develops into what is known as the dome of the aneurysm. It is this dome that usually ruptures, leading to blood extravasation into the subarachnoid space. An aSAH is a catastrophic, emergent event and is the leading cause of nontraumatic SAH and the fourth most frequently occurring cerebrovascular disorder. Immediate attention is warranted at the time of rupture as a delay in treatment will adversely affect outcome (Level 2; Kowalski et al., 2004; Lorenzi, Kerr, Yonas, Alexander, & Crago, 2003).

### A. Incidence of Aneurysm Formation and aSAH

The prevalence of unruptured aneurysm is probably underestimated with up to 5% of the population having undiagnosed aneurysms found on autopsy. Saccular aneurysms can range in size from <10 mm in diameter (78%) to >24 mm (2%). There are few known risk factors for aneurysm formation, including familial history (more than two immediate relatives with history of intracranial aneurysm) and select inherited connective tissue disorders (e.g., fibromuscular dysplasia, Marfan syndrome, sickle cell disease, polycystic kidney disease, and other

connective tissue diseases), anomalous vessels (e.g., coarctation of the aorta) and high-flow states (e.g., vascular malformations, fistulae). Aneurysms that have not ruptured but have manifested with other symptoms, such as a new-onset third nerve palsy (an emergency that requires urgent treatment of the aneurysm), brain stem compression, or visual loss (caused by an ophthalmic artery aneurysm), should be treated because the risk of rupture is believed to be significantly higher than that of incidentally discovered lesions.

Multiple intracranial aneurysms occur in 10%–30% of all cases with a stronger predilection in females. About 75% of patients with multiple intracranial aneurysms have two aneurysms, 15% have three, and 10% have more than three intracranial aneurysms.

Intracranial aneurysms are uncommon in children, accounting for less than 2% of all cases. Aneurysms in children are more commonly post-traumatic or mycotic, have a slight male predilection, and tend to be larger than those found in adults (average diameter is 17 mm).

Aneurysm rupture can occur with any size aneurysm, but is more typical in those >3–5 mm. Aneurysmal SAH accounts for 6%–8% of all strokes, yet unlike other types of stroke, the incidence of aSAH has not declined in the last 30 years. Incidence of aSAH in the general U.S. population is approximately 8–10 cases per 100,000 annually, resulting in approximately 24,000–27,000 new cases each year.

Risk of aneurysm rupture and aSAH is positively correlated with aneurysm size, hypertension, and smoking (Level 2; Juvela, Hillbom, Numminen, & Koskinen, 1993; Wiebers et al., 2003). The risk of aSAH increases linearly with age from 25 to 64 years when data is corrected for the age distribution within the population and peaks between 50 and 60 years old depending on the population or study referenced (Level 2; Wermer, van der Schaaf, Algra, & Rinkel, 2007). Aneurysmal SAH occurs more commonly in women than men (Level 2; Wermer et al.). Reports regarding racial differences also vary from no difference in the rate or prevalence of SAH to a two-fold increase in black versus white Americans (Level 2; Broderick, Brott, Tomsick, Huster, & Miller, 1992). Certain hypertensive states such as those induced by use of stimulants (e.g., cocaine, amphetamines) have been shown to promote aneurysm growth and rupture (Level 2; Brisman, Song, & Newell, 2006; Levine et al., 1990; Mayberg et al., 1994). Reports of oral contraceptive use, heavy alcohol consumption, illicit drug use, hormone replacement therapy, hypercholesterolemia,

**Table 1. Hunt and Hess Classification Scale**

Grade I	Asymptomatic, mild headache, slight nuchal rigidity
Grade II	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
Grade III	Drowsiness, confusion, mild focal neurological deficit
Grade IV	Stupor, moderate to severe hemiparesis
Grade V	Coma, decerebrate posturing

*Note.* From "Surgical Risks as Related to Time of Intervention in the Repair of Intracranial Aneurysms," by W. E. Hunt and R. M. Hess, 1968, *Journal of Neurosurgery*, 28, pp. 14–20.

**Table 2. Fisher Grading Scale**

0	Unruptured
I	No subarachnoid blood detected
II	Diffuse or vertical layer <1 mm thick
III	Localized and vertical layers ≥1 mm thick
IV	Intracerebral or intraventricular clot with diffuse or no subarachnoid blood

*Note.* From "Relation of Cerebral Vasospasm to Subarachnoid Hemorrhage Visualized by CT Scanning," by C. M. Fisher, J. P. Kistler, and J. M. Davis, 1980, *Neurosurgery*, 6, pp. 1–9.

and vigorous physical activity do not appear to be robust independent risk factors (Level 2; Brisman et al.; Mayberg et al.). Although there are many postulated risk factors for aSAH, there is little conclusive evidence to support most of them, other than female gender, increasing age, hypertension, and cigarette smoking.

### B. Mortality and Morbidity

Most saccular aneurysms are asymptomatic until they rupture, at which time they are associated with extreme morbidity and mortality despite improvements in care during the last 3 decades. Approximately 10%–15% (and in some references up to 30%) of patients with aSAH die before obtaining medical attention (Level 2; Broderick, Brott, Duldner, Tomsick, & Leach, 1994; Olafsson, Hauser, & Gudmundsson, 1997). For those who survive until hospital arrival, another 30%–60% will die because of the initial hemorrhage or secondary sequelae (Ingall, Asplund, Mähönen, & Bonita, 2000). Thirty-day mortality is approximately 50% with the highest number of deaths occurring within the first 14 days (Level 2; Broderick et al., 1994; Ingall et al.; Olafsson et al.). Survival is inversely proportional to aSAH grade upon presentation (**Table 1** and **Table 2**) as well as age and overall health. Even in patients who present in good clinical condition, only 55% have good outcomes at 90 days. Outcomes are better for patients admitted to major medical centers, especially those with interventional neuroradiology, within 7 hours of hemorrhage (Level 2; Lorenzi et al., 2003).

Patients that survive aSAH are most often left with cranial nerve palsies, paralysis, aphasia, cognitive impairments, behavioral disorders, and psychiatric disturbances (Level 2; Bellebaum et al., 2004; Hutter, Kreitschmann-Andermahr, & Gilsbach, 1998, 2001; Mavaddat, Sahakian, Hutchinson, & Kirkpatrick, 1999).

### C. Secondary Injury After aSAH

Functional sequelae after initial aSAH are significant. Secondary injury from aSAH is a major concern and typically results from three sources: (1) increased volume within the cranial vault from hemorrhage into the subarachnoid space leading to compressive force, injury to local tissues, mass effect, and increase in intracranial pressure (ICP); (2) meningeal irritation from contact with blood; and (3) compromise of cerebral blood flow because of cerebral vasospasm.

#### 1. Aneurysmal rebleeding

One of the most feared and earliest complications in patients who survive the initial aSAH is rebleeding of the aneurysm. A second hemorrhage is a significant contributor of morbidity and mortality following aSAH and is of immediate concern. There is a 2%–4% risk of aneurysmal rebleed within the first 24 hours of ictus and that risk increases to 15%–20% during the next 2 weeks (Brisman et al., 2006). Untreated ruptured aneurysms have a very high rebleeding risk (20%–50%) after the initial hemorrhage, especially in the first 24 hours (Mayer, Bernardini, Solomon, & Brust, 2005). The mortality rate after a rehemorrhage is extremely high (50%–80%; Suarez, Tarr, & Selman, 2006). In addition to increased mortality related to aneurysm rebleeding, 30% of these patients suffer other serious complications (Suarez et al., 2006). Symptoms of aneurysm rebleed are typically related to increased ICP and include increase in headache, decrease in level of consciousness, and new onset of focal symptoms.

In one study a reduction in the rebleeding rate from 10.8% to 2% was achieved when antifibrinolytic therapy was administered for fewer than 72 hours (Level 1; Hillman, Fridriksson, Nilsson, & Jakobsson, 2002). Prolonged antifibrinolytic administration (e.g., aminocaproic acid tablets [Amicar]) is complicated by ischemia and thromboembolic events and no overall improvement in outcome (Level 2; Suarez et al., 2006; van Gijn & Rinkel, 2001). For these reasons, antifibrinolytic therapy has been abandoned (or is typically avoided) as a standard therapy.

#### 2. Acute hydrocephalus

Acute hydrocephalus, indicated by an enlargement

of the ventricles, occurs in up to 65% of SAH patients depending on diagnostic criteria used and can be life threatening (Level 2; Hasan, Vermeulen, Wijdicks, Hijdra, & van Gijn, 1989; Mehta, Holness, Connolly, Walling, & Hall, 1996; Milhorat, 1987). It usually presents within the first 24 hours and is characterized by abrupt mental status change with or without sixth nerve palsy or gaze deviation and progresses to an obtunded state if left untreated. Late or chronic hydrocephalus, occurring in 10%–15% of patients, is typically because of a blood clot within the ventricular system (Level 2; Demirgil et al., 2003). Late or chronic hydrocephalus generally occurs 10 or more days after SAH and is characterized by incontinence, gait instability, and cognitive deterioration (Level 2; Demirgil et al.).

#### 3. Cerebral vasospasm

Secondary injury because of cerebral vasospasm may occur in as many as 70% of patients with up to 40% demonstrating clinical symptoms (Level 2; Adams, Kassell, Torner, & Haley, 1987; Al-Yamany & Wallace, 1999; Dehdashti, Mermillod, Rufenacht, Reverdin, & de Tribolet, 2004; Dorsch, 2002). The cause of cerebral vasospasm appears to be due to the direct effect of blood and metabolites on the adventitia of the artery. Prolonged smooth muscle contraction is mediated by oxyhemoglobin and release of vasoactive substances from the vessel wall causing inflammatory changes (Level 2; Arai, Takeyama, & Tanaka, 1999; Fujii & Fujitsu, 1988; Macdonald et al., 2001; Takenaka et al., 1991). Cellular response from prolonged smooth muscle contraction causes intimal hyperplasia and subendothelial fibrosis of the vessel. Subsequent leukocyte infiltration and platelet aggregation leads to further reduction in the caliber of the vessel (Level 2; Janjua & Mayer, 2003; Treggiari-Venzi, Suter, & Romand, 2001).

Ultimately, cerebral vasospasm results in the focal narrowing of large arteries and can lead to impaired cerebral autoregulation, cerebral ischemia, and infarction. The most commonly involved arteries are the internal carotid and proximal portions of the anterior and middle cerebral arteries. Vessels undergoing vasospasm are typically unrelated to the initial aneurysm location. Cerebral vasospasm typically occurs within 4–14 days following hemorrhage in the case of virgin bleeds and earlier with recurrent hemorrhage. Risk of vasospasm is positively correlated with subarachnoid blood volume, clinical severity of the initial bleed, female gender, younger age, and smoking. Symptomatic vasospasm can be manifested by one or more of

the following: severe headache, change in mental status from acute confusion and lethargy to obtunded state, or appearance or exacerbation of a focal deficit (Mayer et al., 2005; Treggiari-Venzi et al., 2001). Symptoms vary, but patients typically present with a new onset of a general decrease in level of consciousness or with new focal neurological deficit. Angiographic cerebral vasospasm occurs in up to 70% of individuals recovering from aSAH, and up to 40% will suffer devastating neurological sequelae from ischemia or infarcts (Level 2; Kassell, Sasaki, Colohan, & Nazar, 1985; Treggiari-Venzi et al.).

#### 4. Seizures

Seizures occur in as many as 25% of patients and are most common after middle cerebral artery (MCA) ruptures. Seizures can lead to increased cerebral blood flow, hypertension, and elevated ICP, thus escalating the risk of aneurysm rebleed and neurologic deterioration. Seizures at onset have been shown to be an independent risk factor for late seizures and a predictor of poor outcome (Butzkueven & Hart, 2000).

#### 5. Cardiac abnormalities

Electrocardiogram (EKG) abnormalities frequently occur (Jain, Deveikis, & Thompson, 2004; Zaroff, Rordorf, Newell, Ogilvy, & Levinson, 1999). Most are benign and reversible; however, differentiating myocardial ischemia and left ventricular dysfunction from the benign changes is important (Khush et al., 2005; Zaroff et al., 1999; Zaroff, Rordorf, Ogilvy, & Picard, 2000). Changes resembling acute myocardial ischemia are noted in 25%–80% of patients. In approximately 20% of cases the arrhythmias can be severe or life threatening. The current theory is that EKG changes after aSAH are due to release of excess catecholamines and increased sympathetic tone. There is some thought that they may also be related to vascular vasospasm in the coronary system. Other researchers have postulated that contraction band necrosis or myofibrillar degeneration may be the underlying pathology driving this phenomenon. Typical EKG changes seen after aSAH include prolonged QT and T wave changes (Jain et al., 2004; Zaroff et al., 1999). Cardiac isoenzymes such as troponin and creatine kinase MB fraction are often increased (Zaroff et al., 1999). Myocardial injury after aSAH may increase the risk of cerebral ischemia because of inadequate cardiac output leading to inadequate cerebral perfusion.

#### 6. Cerebral hyponatremia

Cerebral hyponatremia occurs in up to 50% of cases and is correlated with poor outcomes (Level 2;

Doczi, Bende, Huszka, & Kiss, 1981; Qureshi et al., 2002; Revilla-Pacheco, Herrada-Pineda, Loyo-Varela, & Modiano-Esquenazi, 2005; Wijdicks, Vermeulen, Hijdra, & van Gijn, 1985). This is thought to be due to excessive renal secretion of sodium leading to a syndrome known as cerebral salt wasting (CSW) rather than a dilutional effect from inappropriate antidiuretic hormone secretion (Doczi et al.; Revilla-Pacheco et al.; Wijdicks et al.). Besides the direct neural effects on renal function, CSW is associated with disturbances in levels of atrial natriuretic, brain natriuretic, and C-type natriuretic peptides (Level 2; McGirt et al., 2004). Lower serum sodium concentration results in hypoosmolality; this tonicity gradient across the blood-brain barrier can lead to cerebral edema. In addition, these patients are at particular risk of developing cerebral ischemic deficits as a result of increased blood viscosity.

#### 7. Fever

Patients with aSAH are at risk for developing both infectious and noninfectious fever (Commichau, Scarneas, & Mayer, 2003) and are often not responsive to treatment. Fever occurs in as many as 54% of patients recovering from aSAH and is a predictor of poor prognosis (Wartenberg et al., 2006). Fever increases cerebral metabolic rate and is thought to cause release of excitatory neurotransmitters, increased production of oxygen free radicals, and cellular cytoskeletal degradation, as well as break down the blood brain barrier (Badjatia et al., 2004), all resulting in an increased risk for ischemia.

### III. Background

#### A. Cerebral Vasculature Anatomy and Physiology

Arterial blood flow to the brain occurs through four major arteries: two large internal carotids providing blood to the anterior portion of the brain and two smaller vertebral arteries providing blood to the posterior portion of the brain, brainstem, and spinal cord. The two internal carotid arteries branch off the aortic arch and extend to the level of midbrain where they enter the circle of Willis. The MCA and anterior cerebral arteries (ACA) branch off the internal carotid arteries at this junction. The MCA provides blood to lateral portions of the brain in the frontal (including the primary motor strip), parietal (including the primary sensory strip), and occipital lobes. The ACAs provide blood to the medial portion of the brain, optic tract, and subcortical structures of the brain. The anterior communicating artery (ACOMM) connects the two ACAs, allowing for bilateral blood flow in the presence of lesions to one ACA before the ACA–ACOMM junction. The two vertebral arteries

unite at the level of the brainstem to form the basilar artery. The basilar artery continues up the brain stem before branching into two posterior communicating arteries (PCOMM), which form the posterior portion of the circle of Willis. The PCOMM arteries connect to the internal carotid arteries on either side, closing the circle of Willis. PCOMM arteries provide blood to the anterior vessels of the circle of Willis in the face of lesions to the internal carotid arteries. The posterior cerebral arteries (PCA) branch off the basilar artery at the same junction as the PCOMMs and supply blood flow to the occipital lobe and portions of the temporal lobe. See **Figure 1** for the vessels of the circle of Willis.

Unlike other areas in the body, the venous system does not mimic arterial system design. Deep veins and the dural sinuses are responsible for the majority of venous drainage; both empty into the internal jugular veins. The exception is a small amount of venous blood that drains through the ophthalmic and pterygoid venous plexuses into the emissary veins to the scalp and down the system of paravertebral veins in the spinal canal.

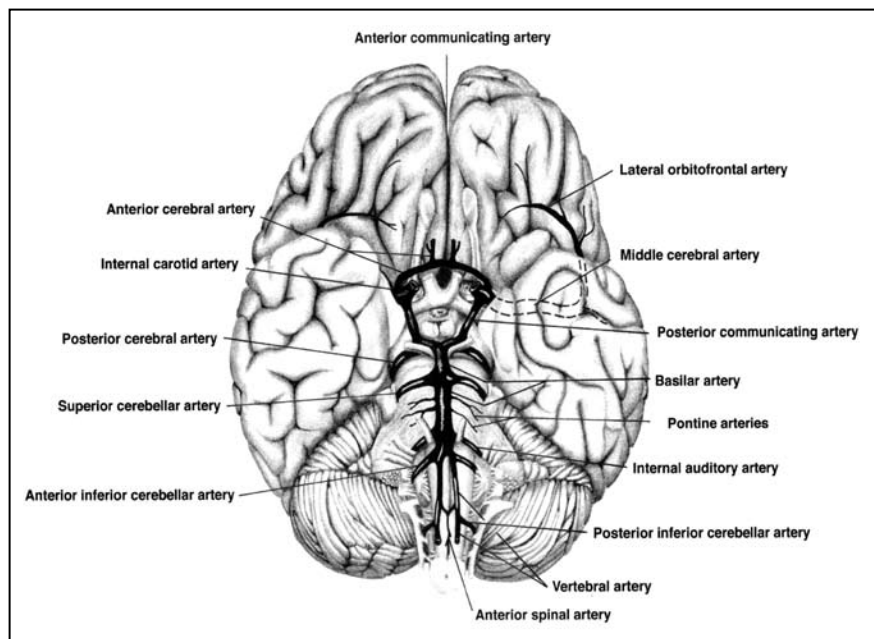
A normal arterial wall consists of three layers: the intima, which is the innermost endothelial layer; the media, which consists of smooth muscle; and the adventitia, the outermost layer, which consists of connective tissue (**Figure 2**).

Normal cerebral circulation requires a constant, total cerebral blood flow under varying conditions. Factors affecting cerebral blood flow include arterial pressure, venous pressure, intracranial pressure, blood viscosity, and the degree of active constriction or dilation of the cerebral arterioles. Because the skull is not pliable and brain tissue and spinal fluid are essentially incompressible, the volume of blood, spinal fluid, and brain in the cranium at any one time must be relatively constant (Monro-Kellie doctrine). Normal cranial capacity for blood and spinal fluid is 125–150 ml.

## B. Pathophysiology and Etiology of aSAH

The occurrence, growth, thrombosis, and rupture of intracranial saccular aneurysms can best be explained by abnormal hemodynamic shear stress on the walls of large cerebral arteries, particularly at bifurcation points, although other factors such as congenital weakness in the arterial or degenerative changes from conditions such as atherosclerosis may act as triggers or cofactors in the disease process. Most

**Figure 1. Circle of Willis**

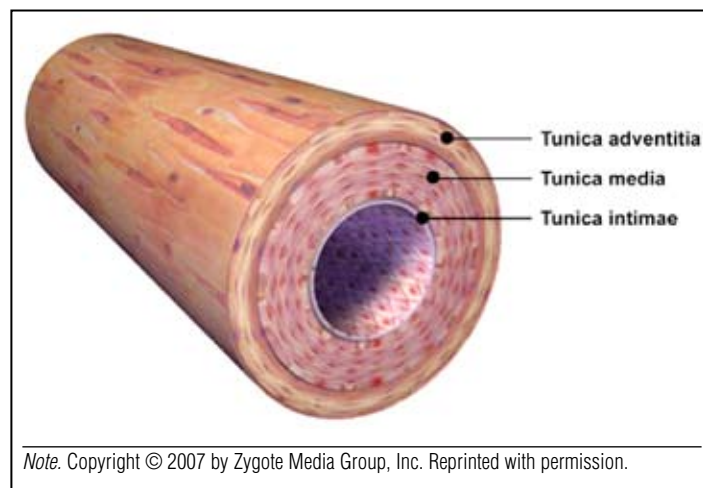


saccular intracranial aneurysms (86.5%) occur in the anterior (carotid) circulation within or near the circle of Willis (Brisman et al., 2006). Approximately 60% of these aneurysms occur at the MCA bifurcation and along the ACA. Other common vessels in the anterior circulation include the bifurcation of the PCA and ophthalmic artery.

Approximately 10% of cerebral aneurysms arise from the vertebral and basilar arteries in the posterior circulation with the tip of the basilar artery being the most common location followed by the origin of the posterior inferior cerebellar arteries. The remaining 3.5% of aneurysms occur in sites such as where the superior cerebellar and the anterior inferior cerebellar arteries branch from the basilar artery. See **Figure 3** for common aneurysm locations.

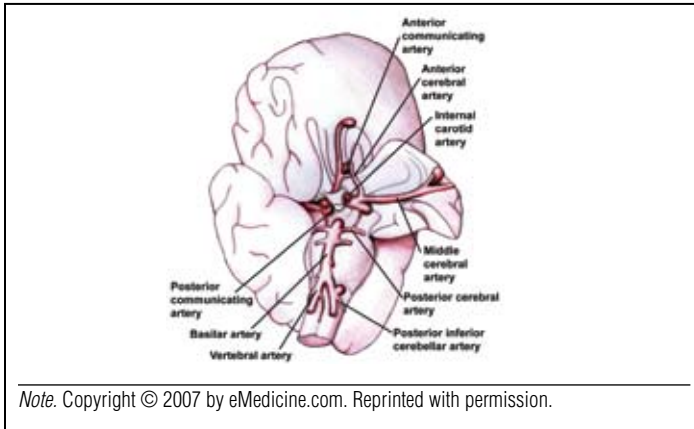
The aneurysmal sac itself is usually composed of

**Figure 2. Layers of a Normal Arterial Wall**



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**Figure 3. Common Aneurysm Locations Within the Circle of Willis**



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only the intima and adventitia vessel layers. The intima is typically normal, although subintimal cellular proliferation may be present. The internal elastic membrane is reduced or absent, and the media ends at the junction of the aneurysm neck with the parent vessel. Lymphocytes and phagocytes may infiltrate the adventitia and fill the lumen of the aneurysmal sac with thrombotic debris. Crucial to this model is the impact vascular and internal flow hemodynamics has on the origin, growth, and configuration of the aneurysms. One of the most important relationships on flow pattern is the geometric relationship between the aneurysm and its parent artery. Understanding the flow patterns not only helps understand the pathogenesis of the aneurysm but is important in selecting the type and placement of a treatment device. In lateral aneurysms, such as ones arising from the internal carotid artery (ICA), blood typically moves into the aneurysm at the distal aspect of its ostium and exits at the proximal aspect. This causes a slow-flow vortex in the aneurysm center. Opacification of the lumen occurs in a cranial-to-caudal fashion leading to flow stagnation. In contrast, intraaneurysmal circulation associated with vessels, arising at the origin or branching vessels or a terminal bifurcation, is rapid. Vortex formation with blood stasis is rare.

### C. Signs and Symptoms of aSAH

Patients with aSAH typically present with a characteristic intense, unrelenting, and overwhelming headache of sudden onset (occurring within seconds). It is often referred to as a “thunderclap headache,” although no sound is heard. A patient often describes the headache as “the worst headache of his life” or “as if the top of his head is being blown off.” In patients with a history of headaches, including migraines, aSAH headache is typically different, being more severe and associated with a feeling of doom. Patients with less severe hemorrhage may

present only with headache or with a headache of moderate intensity that may or may not be associated with nonspecific symptoms, or with neck pain. An aSAH headache can be difficult to assess in patients with decreased levels of consciousness.

Symptoms of meningeal irritation, such as neck stiffness, photophobia, and low back pain, are fairly common, as is nausea, vomiting and double vision from an increase in ICP or meningeal irritation. Depending on the vessel involved, aneurysm size, aneurysm location, and resultant changes in blood flow to brain parenchyma, focal neurological deficits including hemiparesis may also be present. Approximately 10%–25% of patients may present with seizure because of a sudden increase in ICP or cortical irritation from blood, or both. An altered level of consciousness, ranging from mild confusion to coma, is frequently present.

Approximately 10%–15% of patients with ruptured aSAH report having prodromal symptoms in the days or weeks prior to rupture. Prodromal signs present 10–20 days prior to rupture and are present in up to 50% of cases. The most common of these signs are headache (48%), dizziness (10%), orbital pain (7%), diplopia (4%), and vision loss (4%). Other less common prodromal signs include sensory or motor disturbance (6%), seizures (4%), ptosis (3%), bruits (3%), and dysphasia (2%). Jallo and Becske (2007) suggest that these premonitory signs and symptoms either represent small sentinel leaks or aneurysm expansion.

Neurologic examination may demonstrate nuchal rigidity, meningismus, retinal hemorrhage, and to a lesser extent cranial neuropathy (most commonly third [oculomotor] or sixth cranial [abducens] nerve involvement), or other localized neurologic deficit such as aphasia or hemiparesis. Ocular hemorrhage, papilledema, and hypertension may also be present. Many of these findings are clues to the underlying area of brain involved.

There are three prognostic scales widely used as adjuncts for treatment decision making in the SAH population: (1) the Hunt and Hess classification scale, (2) the World Federation of Neurological Surgeons subarachnoid hemorrhage grading scale, and (3) the Fisher grading scale. The patient’s level of consciousness is a cardinal determinant in outcome in the first two scales. The Hunt and Hess classification scale classifies patients based on initial presentation (see Table 1). The World Federation of Neurological Surgeons subarachnoid hemorrhage grading scale has better outcome predictive power, especially in high-grade patients (Table 3). The Fisher grading scale is based on

initial computed tomography (CT) scan findings and specifically predicts risk of cerebral vasospasm (Table 2). These grading systems—in addition to information such as age and medical condition of the patient, aneurysm size and location, accessibility of the aneurysm, presence of a clot, patient wishes, and institutional experience—are used in making clinical decisions regarding treatment (Class I, Level 2; Bederson, et. al, 2009).

#### D. Diagnostic Studies

There are three categories for common diagnostic studies for aSAH: (1) tests to identify subarachnoid blood; (2) tests to identify aneurysm presence, size, and location; and (3) tests that monitor for cerebral edema and cerebral vasospasm and for further bleeding and tissue damage (i.e., stroke). The following section describes tests used to identify subarachnoid blood and identify aneurysm presence, size, and location; however, the same tests may be used later in the patient’s stay to monitor for further bleeding, cerebral edema, cerebral vasospasm, and stroke.

##### 1. CT scan

Nonenhanced brain CT scan is considered the first study of choice in the initial evaluation of patients presenting with suspected SAH (aneurysmal, traumatic, or other cause) with sensitivity approaching 98% with modern CT scanners when performed within 24 hours of symptom onset. Films should be read by a neuro expert (e.g., neuroradiologist, neurosurgeon, or neurologist experienced in diagnosing SAH; Class I, Level 2; Bederson, et al., 2009) for subtle findings such as subarachnoid blood in the posterior horns, Sylvian fissure, and sulci. Failure to undergo an initial head CT in suspect patients is one of the risk factors in misdiagnosis of aSAH (Kowalski et al., 2004).

CT scans use X-ray technology to characterize density within the cranial vault. Substances with increased density appear lighter on the CT scan, and less dense substances appear darker. Therefore, bone and blood appear white and cerebrospinal fluid (CSF) appears black on the CT scan. SAH blood on the CT scan appears as a high-attenuating and formless matter in the subarachnoid space around the brain, thus making what would normally be dark appear white. This effect typically appears as a white star shape in the center of the brain (Figure 4).

The location of blood within the subarachnoid space correlates with the location of the aneurysm in 70% of cases. Generally, blood that is localized to the basal cisterns, the Sylvian fissure, or the interhemispheric fissure indicates rupture of a sacular aneurysm. Blood found over the convexities

**Table 3. World Federation of Neurological Surgeons Subarachnoid Hemorrhage Grading Scale**

World Federation of Neurological Surgeons Subarachnoid Hemorrhage Grading Scale	Glasgow Coma Scale Score	Motor Deficit
I	15	Absent
II	14–13	Absent
III	14–13	Present
IV	12–7	Present or absent
V	6–3	Present or absent

*Note.* From “Report of the World Federation of Neurological Surgeons Committee on a Universal Subarachnoid Hemorrhage Grading Scale,” by C. G. Drake, 1988, *Journal of Neurosurgery*, 68, pp. 985–986.

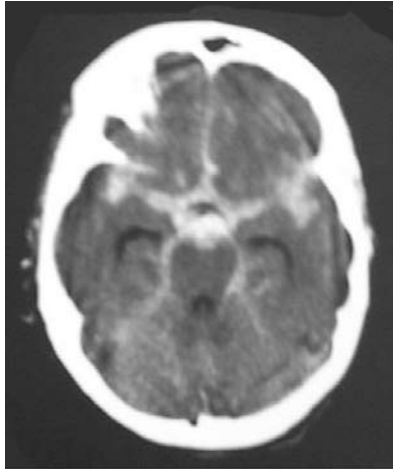
or within the superficial parenchyma of the brain often is indicative of arteriovenous malformation (AVM) or mycotic (from an infectious process) aneurysm rupture.

Intraparenchymal hemorrhage may occur with middle-communicating artery and posterior-communicating artery aneurysms, whereas inter-hemispheric and intraventricular hemorrhages are often seen with anterior communicating artery aneurysms. The outcome is worse for patients with extensive clots in basal cisterns than for those with a thin, diffuse hemorrhage.

Over the cerebral hemispheres, SAH blood is most conspicuous the first 24 hours after hemorrhage. Decreased visualization of the normally hypoattenuating fluid within the sulci and basal cisterns and enlargement of the ventricles may be signs of a communicating hydrocephalus. The amount of SAH is evaluated by the Fisher grading scale, which was initially formulated to predict the risk of cerebral vasospasm but also has prognostic value in predicting overall patient outcome (Table 2). A Fisher grade  $\geq 3$  is robustly associated with the likelihood of developing vasospasm.

A false-negative CT scan can result from severe anemia or small-volume SAH. If the CT scan is positive for possible SAH, further imaging such as cerebral angiography, CT angiography (CTA), or magnetic resonance angiography (MRA) will be required to characterize the hemorrhage source (see pages 12 & 13). Extremely large aneurysms may be visible on CT scans, but further testing to obtain more detailed information about size and angle of the aneurysm as well as vessel involvement is usually required for treatment.

**Figure 4. CT Scan Showing Subarachnoid Blood**



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## 2. Lumbar puncture

If imaging studies such as noncontrast CT are negative in the presence of strong clinical suspicion of an aSAH, a lumbar puncture (LP) should be performed to confirm the diagnosis (Class I, Level 2; Bederson, et al., 2009). A CT scan should always be performed prior to the LP to rule out any significant intracranial mass effect or obvious intracranial bleed. LP is contraindicated in the presence of mass effect, obvious intracranial bleed, and in cases where there is an increase in ICP because of the risk of potential herniation.

A lumbar puncture involves the insertion of a large bore needle into the subarachnoid space between the lumbar vertebrae. CSF is drained from the spinal column and analyzed for blood cells. Presence of xanthochromia (yellow-tinged CSF caused by the breakdown of hemoglobin) is very suggestive of a diagnosis of SAH (sensitivity greater than 99%). Xanthochromia may be present as early as 6 hours following SAH and remains detectable until about 2–3 weeks after hemorrhage. LP is most sensitive 6–12 hours after symptom onset. When gross blood is present, as from a traumatic spinal tap and not an SAH, there should be a successive decrease in blood in successive specimen tubes. It is important if relying on visual inspection for xanthochromia, instead of spectrography, that the correct light and a white background be used to fully appreciate any discoloration. The increase in CSF red blood cells (RBCs) related to a traumatic LP and pain to the patient during the procedure make LP a less commonly used method for diagnosing

SAH. If the CSF reveals evidence of SAH, either overt hemorrhage or xanthochromia, a cerebral angiography, CTA, or MRA should be performed.

## 3. Cerebral angiogram

After the diagnosis of SAH is confirmed, a cerebral angiography is performed to visualize the cerebrovascular anatomy; identify the location, size, and shape of the aneurysm; establish the orientation of the aneurysm dome and neck; determine the relationship of the aneurysm to the parent artery and perforating arteries; and to establish the presence of multiple aneurysms (Class I, Level 2; Bederson, et al., 2009). Newer three-dimensional rotational angiography, which allows for 360° imaging that can be rotated in three-dimensional space, is particularly helpful in providing a more accurate depiction of the aneurysm than two-dimensional films.

Despite development of diagnostic testing, cerebral angiography—with its high degree of accuracy—remains the gold standard in determining the presence and location of an intracranial aneurysm. Cerebral angiography is an invasive procedure with a small but significant risk of complications, including perforation of the vasculature and hemorrhage from the catheter insertion site. A cerebral angiogram is a procedure where a catheter is inserted into the femoral artery in the groin and guided up into the cerebral vasculature. After the catheter is in the cerebral vasculature, a radiographic, iodine-based dye is injected into the catheter. The dye is held in the vasculature, and X rays are taken that permit visualization of the vasculature. An unsecured aneurysm fills with dye-infused blood and appears as an opaque, dark bulb on the X ray (Figure 5). Cerebral angiography has a small, false-negative rate, so another cerebral angiogram must be repeated within 10–14 days if the initial angiogram is negative.

## 4. CTA

Many hospitals now have the capabilities to perform computed tomography angiogram (CTA). Because of the risk associated with cerebral angiography, CTA was developed as a noninvasive test to visualize the cerebral vasculature and identify size and location of a cerebral aneurysm. A baseline CT scan is obtained, and a dye is injected. An additional CT scan is obtained as the dye is filling the cerebral blood vessels. The strength of the signal is stronger in the blood vessels where the dye-filled blood exists. Computer processing by either a neurosurgeon or neuro-radiologist removes static from bone and other structures leaving a clear, three-dimensional

**Figure 5. Angiographic Film Showing Cerebral Aneurysm Before Treatment**

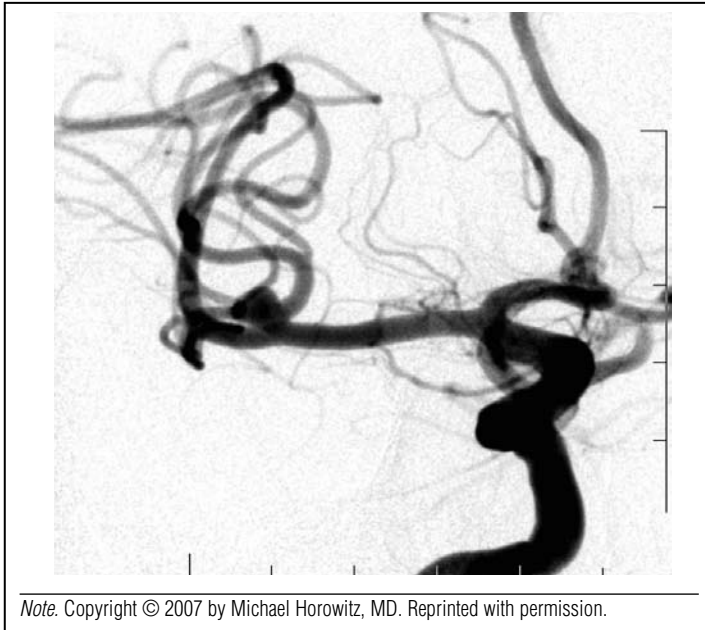


figure of the blood vessels. The dye-filled aneurysm is easily identified in the three-dimensional figure. CTA can be easily performed immediately after a noncontrast CT scan and is becoming a routine test in the work-up of patients with suspected SAH or aneurysm. CTA has the advantages of being noninvasive with the sensitivity and specificity approaching that of cerebral angiography (Jayaraman et al., 2004), especially in lesions greater than 3 mm; however, the computer processing required when obtaining images introduces potential error. CTA can be useful in planning interventional procedures such as coiling or surgery.

#### 5. MRI and MRA

Use of magnetic resonance imaging (MRI) is gaining popularity in identification of aneurysms after aSAH. However, because blood can be more difficult to distinguish on MRI and because of the lack of sensitivity, availability, and increased cost of MRI compared to CT, it is rarely performed as a first-line test, but exceptions to this rule are growing. MRI is similar to CT; both use radiant energy that is directed at the patient. MRI differs in that it uses radio frequency pulsing rather than an X ray. The radio frequency pulse excites the hydrogen ions and then can be measured as changes in the corresponding emanating radio frequency pulses. A patient is placed in a magnet to align the protons of the hydrogen atoms, and a radio frequency (RF) is administered. Signal intensity is measured at a time interval, known

as time to echo (TE), following RF administration. The RF pulse is administered many times in generating an image. The time to repetition (TR) is the time between these RF pulses. Signals characteristic of intracerebral hemorrhage depend on hemoglobin degradation. Deoxyhemoglobin is the MRI substrate for demonstration of blood because of its paramagnetic properties causing signal loss on susceptibility-weighted sequences.

The two basic MRI sequences in common usage are T1- (short TE and TR) and T2- (long TE and long TR) weighted images. Other MRI sequences in common usage include fluid-attenuated inversion recovery (FLAIR) and susceptibility- and diffusion-weighted imaging. Diffusion-weighted imaging is valued for its ease of interpretation because ischemia appears as a bright, white light against a dark gray or black background.

MRI can be helpful when angiography findings are negative, in patients with multiple aneurysms, in bleeds that are several days old, and for identifying small infarcts. In some cases, MRI may provide greater sensitivity than CT in detecting small areas of subarachnoid clot and in helping to determine the particular lesion responsible. FLAIR imaging is particularly useful for demonstrating early or subtle T2 signal changes such as changes associated with edema. Diffusion-weighted MRI is extremely helpful in detecting early ischemia and stroke.

MRA provides a noninvasive means of examining blood flow in the intra- and extracranial vasculature and may be performed in cases where the angiogram failed to show the etiology of the aneurysm (e.g., in dissection, AVM, delayed imaging, or when a patient cannot undergo CT or conventional angiography; Level 2; Bederson, et al., 2009). In general, MRA is still considered less sensitive than catheter angiography, especially in its ability to detect posterior inferior communicating artery and anterior communicating artery aneurysms, but this technology is rapidly evolving.

Gadolinium is the contrast agent used in MRA. Gadolinium-enhanced images are usually acquired with a T1-weighted sequence. There is no cross-reactivity between contrast used for CT and gadolinium. Gadolinium does not have the nephrotoxicity of iodinated contrast used in CTA and conventional angiography. MRI or MRA may only be safely used in the absence of metal objects (foreign bodies, plates, and screws) and pacemaker and defibrillator devices. Some people with claustrophobia cannot tolerate MRI.

## E. Treatment of Aneurysm

Initially, treatment of the aSAH patient is focused on preventing rebleeding of the aneurysm. Although there are many nursing interventions designed to prevent rebleed of the aneurysm (see pages 14–17), securement of the aneurysm is paramount. Newer surgical and endovascular therapeutic options have significantly changed the approach to aSAH management. Definitive treatment is recommended as soon as possible, especially for good-grade patients (i.e., patients with low Hunt and Hess scores or low Fisher grade on admission). Use of an accepted grading system, such as the Hunt and Hess or Fisher Scale, to determine the degree of neurological impairment can be useful for prognosis and triage (Class IIa, Level 2; Bederson, et al., 2009). The two primary options for aneurysm treatment include (1) craniotomy and aneurysm neck clipping and, less commonly, wrapping or ligation and (2) endovascular coiling.

Surgery requires an incision and removal of bone. After the bone has been removed, the temporal lobe can be separated from the parietal and frontal lobe along the Sylvian fissure. Separation of the lobes provides a window through which the aneurysm is visualized. When the aneurysm can be clearly seen, a surgical clip is attached at the base of the aneurysm (where it bulges away from the blood vessel). Application of the surgical clip prevents blood from entering the aneurysm and rebleeding. When the surgical clip is in place, the dome of the aneurysm is punctured or excised, and the aneurysm is monitored shortly to assure no more blood is entering the aneurysm. Many aneurysms are either in a position that is difficult to reach via craniotomy, as in aneurysms in the posterior circulation, or have a very broad base (or neck) that is not amenable to clip placement. Surgical clipping of an aneurysm is still a surgical procedure and, as such, has inherent risks. Risks from surgical aneurysm clipping are similar to risks associated with any other brain surgery and include infection, cerebral edema, pneumocephalus, and risks associated with administration of general anesthesia.

Coil embolization, developed in 1991 as a minimally invasive, nonsurgical method of securing aneurysms, was approved in 1995 by the U.S. Food and Drug Administration and represents a significant and rapidly evolving advancement in the care of the aSAH patient. Coil embolization involves cerebral angiographic techniques to guide a catheter to the location of the aneurysm. Platinum coils are attached to the end of a guide wire and advanced through a microcatheter into the dome of the aneurysm, where they are detached. Coils are packed

into the aneurysm until it is filled. After the aneurysm is filled with coils, blood can no longer enter the aneurysm, and it is considered secure. The blood in the aneurysm where the coils are placed will clot and solidify, but there is no additional blood entering the aneurysm, and there is no further risk of rebleed. Newer techniques include adjuvant use of stents as well as balloons for assisting with broad-neck aneurysms. Although this is a minimally invasive procedure and does not have the risks related to craniotomy, coil embolization has the same risks as cerebral angiography—primarily perforation of vasculature and bleeding from the catheter insertion site. Currently, both methods are safe and effective when performed by experienced, qualified personnel; however, endovascular coiling is associated with improved outcome and is the preferred method for post-circulation, cavernous segment, and internal carotid artery aneurysm (Bederson, et al., 2009). In cases where both surgical clipping and endovascular coiling are potential therapeutic options, endovascular coiling is the preferred method of aneurysm securement (Level 2; Molyneux et al., 2002); however, there is still controversy regarding this subject. Early treatment reduces the risk of rebleeding and is probably indicated in the majority of cases (Level 2; Bederson, et al., 2009). See **Figure 6** for an angiogram showing an aneurysm pre- and postcoiling.

Beginning 24–48 hours after hemorrhage, cerebral edema often develops increasing risk of poor outcome if a surgical intervention is attempted. In addition, risk of cerebral vasospasm dramatically increases 48–96 hours after hemorrhage. Surgical intervention on a patient experiencing even mild cerebral vasospasm greatly increases risk of tissue damage and stroke after surgery. For these reasons, a patient whose aneurysm is not secured in the first few days after aSAH may not be eligible for surgical securement for several days. Nursing care of the patient with an unsecured aneurysm is common in the first 1–2 days after hemorrhage; however, specific portions of this care may be required for longer periods of time in patients who have delayed securement of the aneurysm.

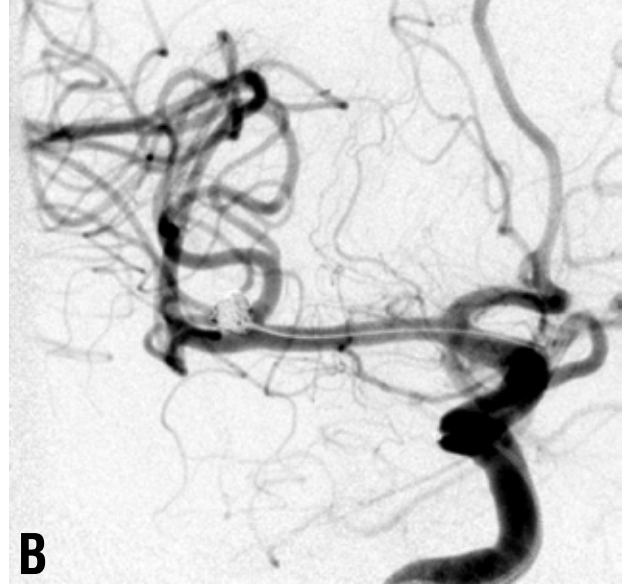
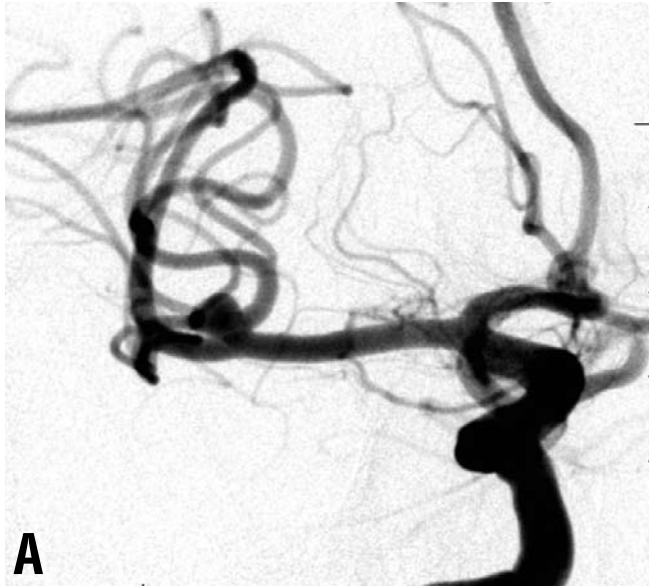
## IV. Patient Care

### A. Preaneurysm Securement

#### 1. Assessment

Upon admission of the patient to the intensive care unit (ICU), hourly neurologic exam checks (including a complete neurologic exam, National Institutes of Health Stroke Scale, Glasgow Coma Scale, and hemodynamic monitoring) are performed and compared to baseline to detect early deterioration because of aneurysmal rebleed,

**Figure 6. Cerebral Angiogram Showing an Aneurysm (A) and the Same Aneurysm Postcoiling (B)**



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acute hydrocephalus, ischemia related to inadequate cerebral perfusion (from early cerebral vasospasm or other causes), or other medical complications.

#### 2. Airway and oxygenation

Intubation and mechanical ventilation may be indicated for patients with decreased mental status, compromised airways, or acute lung injuries from subarachnoid hemorrhage (SAH; e.g., neurogenic pulmonary edema), aspiration, or a Glasgow Coma Scale motor score of *withdrawal*. Modes of ventilation vary, especially in patients who have pulmonary complications following SAH. The goal is to maintain adequate oxygenation and ventilation without compromising both intracranial and cerebral perfusion pressures. Positive end-expiratory pressure of 5 cm H<sub>2</sub>O may be used cautiously in the aSAH patient; however, it does decrease blood pressure (BP) and may lead to cerebral ischemia (Level 2; Meunch et al., 2005). Pressure-controlled ventilation should be considered if the patient has significant aspiration or early acute respiratory distress syndrome.

Patients recovering from aSAH are critically ill patients at risk for many common secondary injuries such as atelectasis and pneumonia. Hourly monitoring of breath sounds and frequent deep breathing should be encouraged. Coughing is discouraged in the SAH patient before aneurysm securement because of the increased risk of aneurysm rupture with the increased ICP and BP that occurs during coughing.

#### 3. BP management

The exact relationship between aneurysmal rebleed and BP remains to be identified; however, most clinicians agree that to prevent rebleed, BP control is achieved before aneurysm securement. Systolic BP is kept between 90 and 140 mm Hg before aneurysm securement (Level 3; Suarez et al., 2006). There are a variety of vasoactive agents used to maintain BP within an acceptable range. Choice of vasoactive agent and BP target range varies depending upon institutional policy (i.e., policy and procedures) and managing clinician preference. Some institutions require clinicians to follow systolic BP, and other institutions follow mean arterial pressure. Typically, BP is maintained within the target range using an initial bolus followed by commencement of an intravenous (IV) drip that is titrated to maintain BP within the target range (Level 2; Kraus, Metzler, & Coplin, 2002). Use of sublingual agents that may cause a rapid drop in BP is not recommended. BP should be lowered in a controlled manner as a sudden drop in BP increases the risk of cerebral ischemia.

Hypotension occurring before aneurysm securement places the patient recovering from aSAH at risk for ischemia. Hypotension should be treated with rapid IV fluid replacement beginning with isotonic saline (0.9%) and colloids as necessary. For persistent hypotension, IV vasopressors should be instituted.

#### 4. Intracranial pressure monitoring

When a patient shows symptoms of increasing ICP, or is at increased risk of increased ICP

because of large blood load, an external ventricular catheter or subarachnoid bolt is inserted. This can be done in the operating room (during surgical clipping or as a separate surgical procedure) or emergently at the bedside to decrease ICP. Poor clinical grade on admission, acute neurologic deterioration, or progressive enlargement of ventricles on CT scan are clear indications for the use of an external ventricular device (Level 2; Mayberg et al., 1994; Rordorf, Ogilvy, Gress, Crowell, & Choi, 1997; Suzuki, Otawara, Doi, Ogasawara, & Ogawa, 2000). Newer data suggest that external ventricular drainage does not include likelihood of aneurysm rehemorrhage when drainage is performed at moderate pressures (<10 cm H<sub>2</sub>O; Level 2; Fountas et al., 2006). Aseptic technique is essential during external ventricular drain or subarachnoid bolt insertion because an infection can occur, especially if the drain is left in for an extended period of time. Cultures are to be routinely performed, and antibiotics are initiated if any signs of infection are present. Some clinicians and institutions use prophylactic antibiotics for aSAH patients with an external ventricular drain, although there is no literature supporting this practice.

Although all of these catheters allow monitoring of ICP, the external ventricular catheter permits CSF drainage to control ICP and clear blood from the CSF. The external ventricular catheter is associated with a higher infection rate than other catheters (Level 2; Lozier, Sciacca, Romagnoli, & Connolly, 2002). Care related to CSF management varies by institution and clinician preference. Continuous drainage of CSF from an external ventricular drain (EVD) at a specified level (above the external auditory meatus or foramen of Monroe as per institutional policy) prevents ICP from rising above that level and allows for continuous clearance of bloody CSF from the ventricles and subarachnoid space (see *Guide to the Care of the Patient with Intracranial Pressure Monitoring: AANN Reference Series for Clinical Practice*).

#### 5. Fever management

In febrile patients (temperature >38.3 °C or as per institutional policy), fever reduction should be achieved with administration of acetaminophen every 4–6 hours to achieve normothermia (Level 3; Suarez et al., 2006). Surface or intravascular cooling is instituted to maintain temperature <38.3 °C if medications are not effective (Level 3; Suarez et al., 2006). It is important to control fever in this population as it is associated with poorer recovery from aSAH (Level 2;

Commichau et al., 2003; Fernandez et al., 2007). Surveillance cultures may be obtained daily in patients receiving cooling therapy, otherwise cultures should be obtained per Society of Critical Care Medicine guidelines (Level 3; O'Grady et al., 1998). In patients receiving surface cooling, monitor and treat shivering with warm compresses to the hands and sedation or paralytics as needed. Induced hypothermia is not routinely recommended (Level 2; Bederson, 2009).

#### 6. Laboratory data

Initial laboratory data provides clinicians with additional baseline data regarding the patient's medical condition and may help in identification of comorbid conditions. Because complications, including cardiac, pulmonary, and fluid and electrolyte imbalances, are known to arise from the moment of aneurysmal rupture, it is imperative to monitor the overall status of the patient. Initial laboratory data include the following:

- basic metabolic chemistry and electrolytes
- cardiac troponin, creatine phosphokinase (CPK) isoenzymes
- coagulation studies
- complete blood count
- type and screen
- urine toxicology and chemistry.

Arterial blood gases are ordered upon admission and as necessary for intubated patients or those in respiratory distress. Admission testing also includes a 12-lead electrocardiogram and a chest X ray.

#### 7. Intravenous fluids

The goal is to maintain euvolemia (central venous pressure [CVP] 5–8 mm Hg) in the patient recovering from aSAH (Level 3; Suarez et al., 2006). Normal saline may be infused at rates between 80 and 100 cc/hr (2–3 L of 0.9% NaCl per 24 hours; Level 3; Mayer et al., 2005). Avoid fluid restriction for patients with hyponatremia due to CSW because it has been associated with increased cerebral infarction (Level 2; Wijdicks et al., 1985).

#### 8. Nutrition

Patients should not be given any food, fluid, or medication by mouth until they have passed a bedside swallow evaluation that includes a water test or have been evaluated by a speech therapist (The Joint Commission, 2009). This includes patients immediately preoperative, stuporous, or comatose. Parenteral nutrition via continuous infusion is started on day 2 after hemorrhage (Level 3; Suarez et al., 2006) if the patient is unable to eat or tolerate enteral feedings. If the patient is not preoperative, stuporous, or comatose, advancing the diet as tolerated is ideal (Level 3; Suarez et al., 2006). A consult

to a speech pathologist to evaluate swallowing capability and aid in diet-type selection is recommended for any patient whose ability to swallow is in question.

Although there is significant ongoing research to identify ideal glycemic control in ICU populations, no specific guidelines are routinely applied to the aSAH population. Hyperglycemia has been found to be associated with increased risk of morbidity and mortality following aSAH, therefore, serum glucose should be kept within the range of 80–120 mg/dl with insulin infusion if necessary (Level 3; Suarez et al., 2006).

9. Activity

Typically, activity is limited in patients with an unsecured aneurysm. All activities that increase BP (and, therefore, ICP) are limited to prevent rebleed. The patient should be maintained in a quiet environment with limited visitors until after aneurysm securement (Level 3; Suarez et al., 2006).

10. Deep vein thrombosis prophylaxis

Because of limited mobility, patients with an unsecured aneurysm are at risk for deep vein thrombosis (DVT). In these patients, thigh-high stockings and pneumatic (sequential) compression devices should be implemented as soon as possible (Level 3; Suarez et al., 2006). Anticoagulants (e.g., heparin) should be avoided until after aneurysm securement (Level 3; Suarez et al., 2006).

11. Medications

a. Seizure prophylaxis

The administration of prophylactic anticonvulsants may be considered in the immediate post-hemorrhagic period (Level 2). The routine long-term use of anticonvulsants is not recommended (Level 2) but may be considered for patients with risk factors such as prior seizure, parenchymal hematoma, infarct, or middle cerebral artery aneurysms (Level 2; Bederson, et al., 2009).

Controversy exists on the need for and length of anticonvulsant therapy in patients without a history of seizures because some anticonvulsants have been associated with poor outcomes, and the percentage of aSAH patients developing seizures is small (Level 2; Naidech et al., 2005). If using anticonvulsants, use those that do not change the level of consciousness.

b. Stool softeners

Stool softeners are initiated. The patient with an unsecured aneurysm should not strain to have a bowel movement, and stool softeners

maintain soft stool so straining is not required (Level 3). For patients able to take oral nutrition, a high-fiber diet is instituted. For patients on parenteral nutrition, a high-fiber feeding is instituted.

c. Pain management

Headache pain is usually intense after aSAH. Analgesics are administered as needed for pain. Pain causes increased BP, heart rate, and anxiety. All of these can increase risk for aneurysmal rebleed and, therefore, must be treated (Level 3). Use short-acting and reversible medications when possible.

d. Sedatives

Agitation can lead to increases in activity, dislodging of catheters, and aneurysmal rebleed. Sedation is administered as needed to patients who are agitated. A short-acting sedative should be used to facilitate frequent neurologic exams free of sedatives. It is not always possible to obtain a neurologic exam free of sedatives, but use of short-acting sedatives increases this likelihood.

e. Antiemetics

Prevention and treatment of nausea and vomiting are also important for the aSAH patient, both before and after aneurysm securement, especially during the first 24 hours. Vomiting increases ICP and can cause aneurysmal rebleed. Patients with nausea should receive an antiemetic routinely.

f. Gastrointestinal hemorrhage prophylaxis

Histamine-receptor antagonists or proton pump inhibitors are instituted to prevent ulcer formation and gastrointestinal hemorrhage.

12. Psychosocial

Alleviate anxiety by explaining procedures and ICU routine to patients and families. Incorporate a multidisciplinary approach, including pastoral care and social work, to address the patients' needs.

**B. Postaneurysm Securement**

1. aSAH patient in the ICU

After the aneurysm has been secured, many of the previous care guidelines are maintained; however, some adjustments should be made.

a. Assessment

Typically, monitoring of neurologic exam and vital signs are performed every hour after surgery or embolization. If the patient remains stable, exam and vital-sign assessment are decreased to every 2 hours and as necessary. Serial complete neurological assessment, including level of consciousness, cranial

nerve assessment, and motor exam performed at the bedside, detects subtle changes from the patient's baseline status. Any changes in neurologic exam are reported to the attending physician, resident, or nurse practitioner immediately. Initial assessment will identify changes related to surgery or possible rebleeding of the aneurysm, cerebral edema, or increasing ICP. Continued assessment is vital to optimize outcomes in this population because cerebral vasospasm is a common secondary sequelae to aSAH and develops very suddenly. Prompt identification of changes in neurologic exam initiates further testing to determine cause of the change and intervention, thereby preventing long-term damage to the brain.

b. Airway and oxygenation

For patients who do not require intubation and mechanical ventilation, frequent assessment of airway patency and oxygenation continue. Along with hourly vital-sign assessment, breath sounds are auscultated. Any changes in breath sounds should be reported to the attending physician, resident, or nurse practitioner immediately. Proper oxygenation is necessary to prevent hypoxia and cerebral ischemia. Suctioning may be performed as needed for short intervals with appropriate hyperoxygenation provided prior to suctioning in a patient recovering from aSAH after the aneurysm has been secured.

c. BP management

When the aneurysm is secure, an increase in BP is permitted. Maintaining the systolic pressure at less than 200 mm Hg has been recommended (Level 3; Suarez et al., 2006). The target range for ideal BP after aneurysm securement has not been thoroughly defined; however, the goal of BP management is to maintain perfusion of brain tissue and prevent ischemia.

d. ICP monitoring

In many patients recovering from aSAH, ICP monitoring will continue after securement of the aneurysm. Any patient at risk for increased ICP should have continued ICP monitoring. Prolonged elevations in ICP are associated with decreased cerebral perfusion pressure and increase the risk of cerebral ischemia and poor outcome (Level 2; Mayberg et al., 1994; Rordorf et al., 1997; Suzuki et al., 2000).

e. Fever management

In febrile patients (temperature  $\geq 38.3$  °C or as per institutional policy), fever reduction is achieved with administration of acetaminophen every

4–6 hours to achieve normothermia (Level 3; Suarez et al., 2006). Surface or intravascular cooling is instituted to maintain temperature  $< 38.3$  °C if medications are not effective (Level 2; Badjatia et al., 2004). It is important to control fever in this population because it is associated with poorer recovery from aSAH (Level 2; Commichau et al., 2003; Fernandez et al., 2007). Surveillance cultures are obtained daily in patients receiving cooling therapy, otherwise cultures should be obtained per Society of Critical Care Medicine guidelines (Level 3; O'Grady et al., 1998). In patients receiving surface cooling, monitor and treat shivering with warm compresses, circulating warm air, sedation, or paralytics as needed (Level 2; Badjatia et al.).

f. Laboratory data

The following laboratory values should be obtained daily after the aneurysm has been secured:

- electrolytes (including magnesium)
- troponin, CPK isoenzymes (for the first 5 days after hemorrhage)
- echocardiogram.

Also consider arterial blood gases, chest X ray, and anticonvulsant levels as needed.

g. IV fluids

IV fluids are maintained to assure adequate hydration. In patients with symptomatic vasospasm, triple H therapy (hypervolemia, hypertension, and hemodilution) remains a frequently used regimen in the prevention of cerebral vasospasm after aSAH. Triple H therapy prevents ischemic deficits in patients with mild to moderate symptomatic cerebral vasospasm by augmenting circulating blood flow and perfusion pressures, increasing cardiac output, improving rheology of blood flow, and increasing collateral circulation (Awad, Carter, Spetzler, Medina, & Williams, 1987; Kassell et al., 1982; Muizelaar & Becker, 1986). The most common symptoms of symptomatic vasospasm are focal ischemic deficits, reflecting the region experiencing ischemia; focal ischemic deficits are often referred to as delayed ischemic deficits because of the temporal establishment. These patients require more aggressive volume expansion and hypertension. The goal of triple H therapy is to achieve CVP 8–12 mm Hg, hematocrit  $< 30$ , systolic BP  $\geq 180$  mm Hg, and urine output  $\geq 250$  ml/hr. These goals can be achieved by infusing large amounts of colloid or crystalloid or through pharmacologic interventions (Level 2; Awad et al.; Janjua & Mayer, 2003; Kassell et

al., 1982; Muizelaar & Becker). Vigilant monitoring of patients is warranted because triple H therapy includes complications such as myocardial injury, pulmonary edema, hyponatremia, cerebral edema, and bleeding of unsecured aneurysm (Awad et al.; Janjua & Mayer; Kassell et al., 1982; Mocco, Zacharia, Komotar, & Connolly, 2006; Muizelaar & Becker; Solomon, Fink, & Lennihan, 1988; Treggiari-Venzi et al., 2001). Although not yet used as a standard of care in all facilities, invasive monitoring, such as a pulmonary artery catheter, is warranted in patients with cardiac dysfunction to adequately monitor and treat the patient recovering from an aSAH (Level 3; Mayer et al., 2005; Suarez et al., 2006). See **Figure 7** for an angiogram showing cerebral vasospasm before and after treatment (see pages 18 & 21–23 for treatment of the patient with cerebral vasospasm).

h. Nutrition

Patients recovering from aSAH must be screened for ability to swallow prior to receiving any food, fluid, or medication by mouth. A validated bedside screen that includes a water test should be used. A formal swallow evaluation from a speech therapist should be obtained if there are any questions about the patient's ability to safely swallow. After it has been determined that swallowing is normal, the patients' usual diet with increased fiber may be followed. Patients with impaired swallowing should have a diet prescribed by the speech therapist to prevent aspiration.

i. Activity

After the aneurysm has been secured, patients gradually increase activity. Physical and occupational therapists are consulted postoperatively when patients are stable.

j. DVT prophylaxis

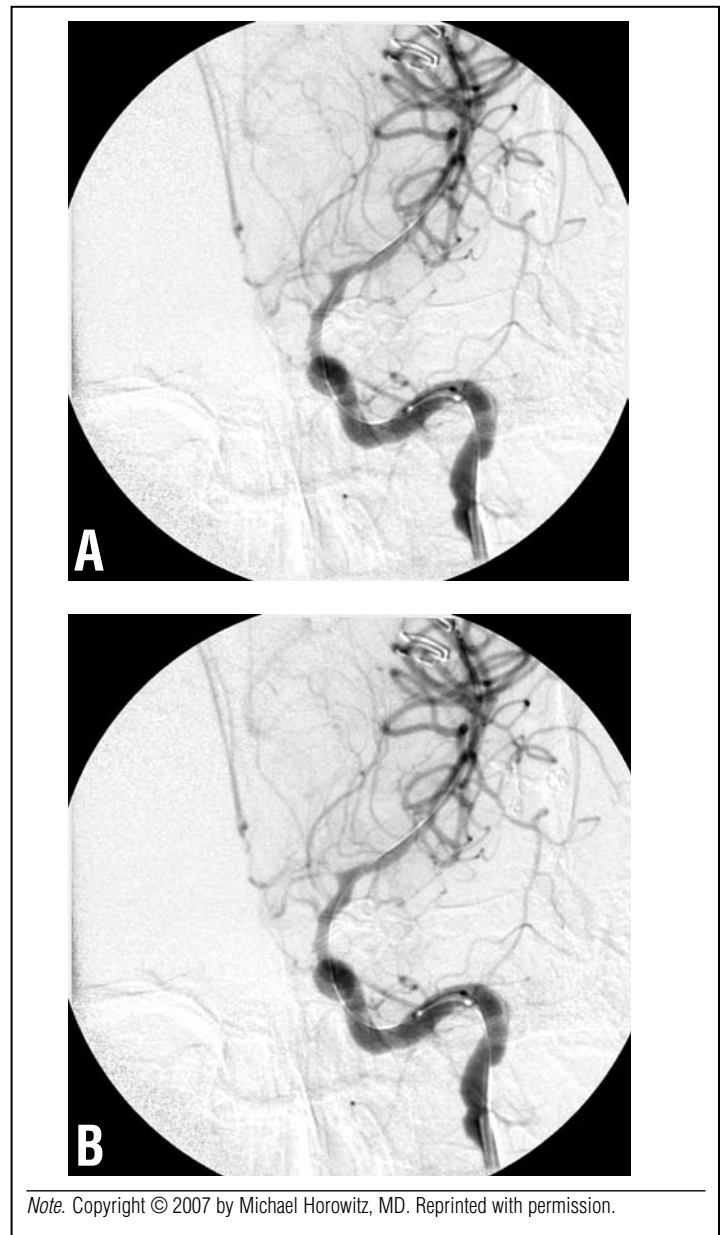
Thigh-high stockings and pneumatic (sequential) compression devices are maintained postaneurysm securement (Level 3; Suarez et al., 2006). When the aneurysm has been secured, heparin therapy for prevention of DVT may be considered. Additional factors, such as future need for surgery or angiography, are weighed into the decision to institute heparin therapy.

k. Medications

(1) Anticonvulsants—If seizures have occurred or the patient is at higher risk for seizure development, prophylaxis is maintained. If using anticonvulsants, use those that do not change the level of consciousness.

- (2) Stool softeners—Stool softeners should be continued because narcotics, other medications, and decreased physical mobility and bowel motility may cause constipation.
- (3) Sedation—Sedation may be warranted particularly in patients who are intubated, have ICP monitors and central lines, or both.
- (4) Antiemetics—Use of antiemetics may be continued as needed.
- (5) Cerebral edema treatment—In patients with cerebral edema, 2% or 3% hypertonic saline may be administered at a rate of

**Figure 7. Angiogram Showing Cerebral Vasospasm (A) and Angiogram Showing Cerebral Vessels After Being Treated for Cerebral Vasospasm (B)**



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75–150 cc/hr unless contraindicated (Level 2; Suarez et al., 1999). Frequent electrolyte monitoring is indicated at least every 6 hours. Monitor and replace potassium to maintain normal levels. Monitor serum sodium to a goal of 145–155 meq/L and serum osmolality 300–320 mOsm/L levels. Notify the provider on call if the serum sodium is >155 meq/L. Hypertonic saline therapy can be tapered slowly if no longer indicated (i.e., improving mental status or cerebral edema or the serum sodium rises to dangerous levels >155 meq/L; Level 2; Suarez et al., 1999).

- (6) BP treatment—A variety of pharmacological agents may be used to maintain BP within the target range. See page 18 for treatment of BP.
  - (7) Calcium channel blockers—Nimodipine (Nimotop), a calcium channel blocker, is the only drug currently approved by the FDA for the prevention and treatment of vasospasm following aSAH. Nimodipine crosses the blood–brain barrier and inhibits calcium entry into cells, subsequently reducing the contractile state of the vascular smooth muscle. It is indicated to reduce the incidence and severity of delayed ischemic deficits from vasospasm following aSAH and has been shown to improve outcomes following aSAH despite a lack of evidence of arteriographic efficacy (Level 1; Allen et al., 1983; Neil-Dwyer, Mee, Dorrance, & Lowe, 1987; Petruk et al., 1988; Philippon et al., 1986; Pickard et al., 1989). Solomon and colleagues (1988) proposed that the improved outcome with nimodipine was related to it inhibiting calcium entry into ischemic neurons, thereby increasing viability of these cells. Oral or enteral administration of 60 mg of nimodipine every 4 hours is instituted within 96 hours after hemorrhage and continued for up to 21 days.
- l. Other tests and treatments
- Several tests are used to monitor for presence of cerebral vasospasm. Transcranial Doppler (TCD) ultrasonography uses ultrasound waves projected through a thin spot in the skull to the cerebral blood vessels. The ultrasound waves bounce off of the RBCs as they flow through the cerebral blood vessel. A decrease in the internal lumen of the blood vessel requires the blood (and hence, the RBCs) to move at a higher velocity. Although TCD ultrasonography is not sensitive or specific enough to use to diagnose cerebral vasospasm,

it is a noninvasive diagnostic tool that can be used in conjunction with neurologic exam and other diagnostic tests to manage the aSAH patient. TCD ultrasonography has several limitations. It is only as good as the technologist performing the exam, so a neurophysiologist should be consulted whenever available. There are multiple physiologic states that will increase blood flow, thereby increasing blood velocity. Independent of neurologic exam, TCD can consistently measure MCA mean velocities and can detect increasing mean MCA velocities. MCA flow velocities <120 cm/sec and >200 cm/sec respectively have a strong negative and positive predictive power for determining which patients will develop ischemic deficits (Level 3; Aaslid, Huber, & Nornes, 1984). Some clinicians and institutions prefer to monitor patients using the Lindegaard index. The Lindegaard index was developed to predict cerebral vasospasm using TCD. It is calculated as *mean MCA velocity/mean ICA velocity*. A Lindegaard index  $\geq 3$  is indicative of MCA vasospasm and  $\geq 6$  as severe vasospasm (Level 2; Aaslid et al.; Lee et al., 1997; Lindegaard, Nornes, Bakke, Sorteberg, & Nakstad, 1988). TCD velocity associated with a decrease in neurological function, or independently in comatose patients, can be used as a preliminary screening method to identify patients requiring further intervention (i.e., CT scan or cerebral angiogram).

Cerebral angiography is the gold standard for diagnosing cerebral vasospasm. The procedure is the same as described on pages 12 & 13 for aneurysm identification. The angiogram provides a clear visualization of the cerebral blood vessels, and a decrease in lumen size is indicative of cerebral vasospasm. Variation in the decrease in lumen size also quantifies severity of cerebral vasospasm. A blood vessel with a significant decrease in lumen size requires intervention.

In patients with symptomatic vasospasm, it is often managed with triple H therapy. More severe symptomatic vasospasm requires more aggressive treatment. Endovascular therapies for refractory vasospasm include both intra-arterial vasodilators and mechanical dilatation of vessels with balloon angioplasty. The determination of which of these therapies to use is an individual decision and depends upon the patient's general health and severity of vasospasm. Papaverine is a widely used agent

(Fandino, Kaku, Schuknecht, Valavanis, & Yonekawa, 1998; Kaku, Yonekawa, Tsukahara, & Kazekawa, 1992; Polin, Hansen, German, Chaddock, & Kassell, 1998; Sawada et al., 1997), although, there is preliminary evidence that verapamil (Feng et al., 2002), nicardipine (Kasuya, Onda, Sasahara, Takeshita, & Hori, 2005; Kasuya, Onda, Takeshita, Okada, & Hori, 2002), nimodipine (Biondi et al., 2004; Hui & Lau, 2005; Tanaka et al., 1982), and fasudil hydrochloride (Tachibana et al., 1999; Tanaka, Minami, Kota, Kuwamura, & Kohmura, 2005) may be of benefit (Level 2). A review of intra-arterial treatment of cerebral vasospasm and mechanisms of action of these drugs was provided by Sayama, Liu, and Couldwell (2006).

For patients at risk for or with known cerebral vasospasm, more aggressive treatment should be used. Patients without symptoms but with elevated TCD velocities or CT evidence of diffuse cerebral vasospasm require at least a central venous catheter, repletion with crystalloids, and the above end points for volume resuscitation (CVP  $\geq 8$  and urine output  $\geq 250$  ml/hr). CVP monitoring is indicated at least every 2 hours. Treatment with fluid or albumin bolus to keep CVP  $>5$  for normovolemia or CVP  $>8$  mm Hg for hypervolemia is indicated (Level 3; Mayer et al., 2005; Suarez et al., 2006). Hypervolemia is desirable in patients without underlying cardiac disease to maintain adequate cerebral perfusion pressure (Level 3; Mayer et al.). Antihypertensive and diuretic agents should be avoided (Level 3; Mayer et al.).

For patients with a secured aneurysm and clinical evidence of cerebral vasospasm, more aggressive therapy is instituted. If not yet performed, cerebral angiography may be performed to accurately diagnose and treat cerebral vasospasm (see page 20 for angiographic treatment of cerebral vasospasm). Pulmonary pressure monitoring may be indicated in patients with cardiac dysfunction with the goal of maintaining pulmonary artery wedge pressure  $>12$  mm Hg and cardiac index  $>4.0$  L/min (Mayer et al., 2005). If desired effect is not attained, cerebral angiography for angioplasty or drug infusion may be undertaken if qualified personnel are available (see pages 12 & 20 for angiographic treatment).

## 2. Patient monitoring in the ICU

### a. Neurological

- (1) Frequent neurological assessment is indicated with a minimum of at least every hour or more frequently when patients are

actively ischemic.

- (2) For patients with external ventricular drain or subarachnoid bolt, see *Guide to the Care of the Patient with Intracranial Pressure Monitoring: AANN Reference Series for Clinical Practice*.
  - (3) Monitor TCD values including systolic velocities, mean velocities, and Lindegaard ratio and compare them to baseline and previous values. Discuss elevations (mean MCA velocity  $>120$  mm Hg or Lindegaard ratio  $\geq 3$ ) with attending physician, resident, or nurse practitioner promptly.
  - (4) Electroencephalography (EEG) is commonly used to monitor for seizure activity in many patients with neurological conditions. Continuous EEG is used to monitor patients with unexplained neurological deterioration to detect nonconvulsive seizures by providing information about global cerebral activity and cortical function (Wartenberg et al., 2002). Electrodes are placed at distinct positions around the skull, and brain activity is monitored. Typical brain activity shows much variation in the brain waves, while seizure activity is evidenced by rhythmic waves indicating neurons firing in unison. In patients with continuous EEG, collaborate with the EEG technician to ensure that leads are in place. Monitor for clinical seizures.
  - (5) Repeat CT scans and cerebral angiography are common tests used to monitor the patient recovering from aSAH. CT scans are routinely performed postoperatively and postcoiling and are warranted when the patient's clinical exam changes. Cerebral angiography should be obtained postoperatively, postcoiling (to ensure aneurysm obliteration), and when clinical exam or TCDs suggest cerebral vasospasm.
- ### b. Cardiovascular
- (1) Hemodynamic monitoring is obtained at least every hour or more frequently when titrating vasoactive agents. Monitor peripheral pulses and troponin levels during vasopressor infusion.
  - (2) CVP monitoring is indicated at least every 2 hours to keep CVP  $>5$  for normovolemia or CVP  $>8$  mm Hg for hypervolemia (Mayer et al., 2005).
  - (3) Pulmonary artery pressure monitoring is indicated in patients with cardiac dysfunction, with pulmonary artery diastolic pressure kept  $>14$  mm Hg or a cardiac

- index >4.0 L/min (Mayer et al., 2005). Some institutions have incorporated pulmonary artery pressure monitoring as a standard of care for all aSAH patients, although the literature is not clear on the efficacy of this practice.
- c. Respiratory
    - (1) In patients requiring mechanical ventilation, frequent arterial blood gases, pulse oximetry (SaO<sub>2</sub>) and end tidal CO<sub>2</sub> (ETCO<sub>2</sub>) are indicated. Arterial blood gases should be obtained daily and with each change in ventilator settings. Continuous SaO<sub>2</sub> or ETCO<sub>2</sub> monitoring should be incorporated to maintain SaO<sub>2</sub> ≥90% or ETCO<sub>2</sub> ≥35–37 mm Hg.
    - (2) Suctioning should be performed only as necessary to maintain clear lungs and limited to 15 seconds, hyperoxygenating the patient prior to the procedure. Saline lavage prior to suctioning should be avoided.
  - d. Gastrointestinal
    - (1) Abdominal assessment is indicated at least every shift.
    - (2) Nutritional support is obtained via tube feeding if the patient is unable to take orally.
  - e. Renal
    - (1) Urine output is monitored precisely. A urinary catheter is often warranted to assure accurate monitoring.
    - (2) Urine electrolytes and specific gravity should be monitored as these patients are at risk for CSW and the syndrome of inappropriate antidiuretic hormone secretion (SIADH).
    - (3) It is important to be aware that patients receiving triple H therapy often have high urine output.
  - f. Integumentary
    - (1) In patients on complete bed rest, skin assessment is performed every shift.
    - (2) Frequent turning (at least every 2 hours) is performed for patients unable to move themselves.
    - (3) Skin-care techniques are performed every shift with the assessment.
  - g. Endocrine
 

Tight glycemic control is to be maintained, using an insulin drip if necessary. Glucose should be monitored at least daily in all patients recovering from aSAH. In patients requiring an insulin drip, glucose should be evaluated hourly until reaching the target blood glucose (100–120 mg/dl) and then every 2–4 hours.
  - h. Psychosocial
 

Social workers and pastoral personnel are consulted to assist in alleviating concerns of patients and families. Social workers should also collaborate with the critical care team to identify and facilitate appropriate after-discharge care.
  - i. Current research and future therapies
    - (1) Pharmacologic therapeutics
      - (a) Magnesium
 

Intravenous magnesium sulfate (MgSO<sub>4</sub>) is currently being researched for its potential clinical use in the prevention and reversal of cerebral vasospasm. It is especially attractive because it is readily available, inexpensive, and has been shown to be safe in humans (Veyna et al., 2002). Magnesium has many neuroprotective mechanisms of action. It has cerebral vasodilatory effects (Pyne, Cadoux-Hudson, & Clark, 2001), inhibits excitatory amino acid release, and provides N-methyl D-aspartate receptor blockade (Lin, Chung, Lin, & Cheng, 2002; Nowak, Bregestovski, Ascher, Herbet, & Prochiantz, 1984). Preliminary studies in humans have shown a significant reduction in cerebral vasospasm development (55%; Chia, Hughes, & Morgan, 2002) and delayed cerebral ischemia (35%; van den Bergh et al., 2005) in patients randomized to receive IV MgSO<sub>4</sub>. Current research is ongoing to determine therapeutic dosages for preventing cerebral vasospasm and delayed cerebral ischemia while avoiding initiating side effects.
      - (b) Statins
 

Several small randomized clinical trials, consistent with animal research, have demonstrated that the initiation of statin therapy after aSAH to significantly reduce the incidence of vasospasm and delayed ischemic deficits, and slightly reduce mortality. These studies support the routine use of statins in the care of patients with aSAH (Sillberg, 2008). However, Kramer and Fletcher (2009), in a recent review article, do not recommend statin therapy in aSAH and report no associated with improvement in neurological outcomes. More research is needed in this area.
    - (2) Advance neuromonitoring
      - (a) Brain tissue oxygen monitoring
 

PtiO<sub>2</sub> monitoring is a method to directly

monitor brain tissue oxygenation. Currently, there is only one system commercially available—the Licox system (GMS-Integra; Kiel-Mielkendorf, Germany). It contains a polarographic cell embedded in the catheter that is placed in the brain tissue of interest. When oxygen passes through the electrolyte chamber of the catheter, an electrical current is generated. The electrical current is then translated into tissue oxygenation. The sampling area of the catheter is approximately 14 mm<sup>2</sup>.

The catheter measures the tissue environment of a small portion of the brain. It is difficult to predict which area of brain tissue is at highest risk of cerebral vasospasm; hence, there is not standardization as to where the catheter should be placed in a patient recovering from aSAH. Recent research suggests that PtiO<sub>2</sub> monitoring is a safe neuromonitoring device that accurately reflects tissue oxygenation (Lang, Mulvey, Mudaliar, & Dorsch, 2007). With future research, PtiO<sub>2</sub> monitoring may be an excellent method to monitor for pending ischemia in the aSAH population. Lang and colleagues recently presented a review of literature surrounding the safety and efficacy of these catheters and their utility in neurocritical care.

(b) Neurochemistry

For cerebral application, the microdialysis technique allows clinicians to precisely monitor brain chemistry by continuously monitoring biochemical markers of energy metabolism such as glucose, lactate, pyruvate, and lactate-pyruvate ratio; cell membrane degradation such as glycerol; or excitotoxic and other metabolic pathways. A 10-mm catheter is inserted into the region at risk for vasospasm in patients with subarachnoid hemorrhage (Ungerstedt & Rostami, 2004).

The microdialysis catheter has a semipermeable membrane at the distal end which functions as a blood capillary. Standard catheters can measure molecules of 20 kDa such as glucose, lactate, and pyruvate. When perfusion fluid is pumped at a rate of 0.3 µl/min into the catheter, it flows through the

distal end of the membrane and equilibrates with the extracellular fluid. After some time, the molecules will diffuse into the perfusion fluid. Recovery of these molecules is about 70%. The molecules are extracted hourly and analyzed with the microdialysis analyzer (CMA Microdialysis, Stockholm, Sweden) at the bedside. Immediate bedside analysis alerts clinicians of perturbed energy metabolism occurring at the cellular level and, therefore, provides insight for clinicians in preventing secondary injury (Ungerstedt & Rostami, 2004). Bedside microdialysis monitoring of cerebral tissue is a useful tool but is not currently used in most facilities.

3. Care of the aSAH patient in the neurological unit

When the patient has stabilized and risk of cerebral vasospasm is low, the patient recovering from aSAH is transferred to the neurological unit.

Vital signs with complete neurologic examination should be performed every 4–8 hours. Medications should be maintained as in the ICU setting; however, patients should no longer require intravenous vasoactive medications to maintain BP; mechanical ventilation; or central venous pressure, pulmonary artery, or arterial BP monitoring. If anticonvulsants are being used, they should be continued in the unit. Antiemetics should be used as needed, although nausea and vomiting are not common in patients stable enough for transfer to the unit. Pain medications should be continued as needed. Nimodipine should be ordered at the same dose of 60 mg orally every 4 hours until 14–21 days after hemorrhage. Activity should be increased as tolerated by the patient. Physical and occupational therapy should be consulted to determine patient functioning and needs for rehabilitation during the remainder of the hospital stay and after discharge.

4. Care of the aSAH patient outside the hospital

a. Home

Most patients recovering from an aSAH will be discharged to their homes. A family member or significant other should be present when discharge instructions are given to the patient. If the patient is being discharged less than 21 days after hemorrhage, nimodipine should be continued for 14–21 days. Other medications should be continued after discharge. The patient should be instructed to take all medications as ordered. The patient should also be encouraged to drink lots of

water and other nonalcoholic liquids to ensure hydration after discharge. Although activity is not restricted after discharge, patients should be advised to monitor themselves for tiring and exhaustion and to schedule activities accordingly. Referral to outpatient physical therapy is recommended to ensure maximal recovery.

b. Rehabilitation

Some patients recovering from an aSAH will be discharged to a rehabilitation center for more intensive physical and occupational therapy. Medications should be continued after discharge. Nimodipine should be continued for 21 days after hemorrhage. In the rehabilitation setting, intake and output should be monitored closely to prevent dehydration.

**C. Patient and Family Education**

For the SAH patient, education may not be possible immediately upon admission to the hospital. In many cases, the patient is too ill or has too low of a level of consciousness to benefit from education. However, when the patient is awake enough, education by the health professionals should begin immediately.

Because of the severity of subarachnoid hemorrhage, education usually focuses on the family members. It is normal for the family to be overwhelmed and have many questions. Because of the stress that the family members experience, many times education must be repeated and reinforced until the family members can process this information.

The brain may take 6–15 months to recover to the fullest ability (Haug et al., 2007; Samra et al., 2007). It is quite common for headaches to last up to 6 months or longer. The family also must be educated on the symptoms of another stroke. These symptoms include, but are not limited to, severe headache, sudden speech difficulties, sudden vision change, inability to move one side of the body, and numbness or tingling on one side of the body. The patient should call emergency services if any of these symptoms appear.

Although there is no literature supporting the screening of family members of aSAH patients for aneurysms, some physicians refer first-degree relatives for MRI, MRA, CTA, or angiography for cerebral aneurysms. The family members considered at risk and the technique used for screening are currently based on physician preference.

The American Stroke Association ([www.strokeassociation.org](http://www.strokeassociation.org)) and the National Stroke Association ([www.stroke.org](http://www.stroke.org)) have excellent Web sites

that can be resources for nurses, patients, and family members. Another resource that is excellent for education of the family is a booklet titled *Brain Aneurysm: Understanding Care and Recovery*. This booklet is distributed by Krames and may be ordered by calling 800/333-3032. This booklet is also endorsed by the American Association of Neuroscience Nurses.

Key areas of patient, family, and caretaker education in the subarachnoid hemorrhage population are as follows:

- What is a brain aneurysm?
- What is a subarachnoid hemorrhage?
- Signs and symptoms of a ruptured aneurysm
- What is hydrocephalus?
- What is cerebral vasospasm?
- Possible medical procedures that the patient may encounter while in the hospital
  - CT scan
  - lumbar puncture
  - arteriogram/angiography
  - Transcranial Doppler ultrasonography
  - MRI
- Treatment options
  - clipping of aneurysm via craniotomy
  - endovascular procedures (coiling)
- Length of hospital stay
  - ICU stay (average 10–14 days)
  - step-down/unit stay (average 5–7 days)
- Common complications are as follows:
  - cerebral vasospasm
  - hydrocephalus
  - hyponatremia
  - loss of short-term memory
  - behavior changes
  - seizures
  - depression
  - dysphagia
  - skin breakdown
  - urinary/bowel incontinence
- After the hospital
  - inpatient rehabilitation
  - long-term nursing care
  - recovery/prognosis
- Screening of first-degree relatives

#### D. Documentation

Documentation is similar to the documentation for the ischemic stroke patient. Documentation should include the following:

- time of onset
- symptoms
- neurological assessment: level of physical functioning, cognitive level, muscle strength, and cranial nerve findings. (Some providers prefer the nurse to describe “what they saw” versus saying that a certain cranial nerve is not functioning.)
- vital signs: BP, pulse rate and rhythm, respirations, oxygen saturation, temperature, blood glucose, CVP, ICP (if patient has EVD), cardiac output (if patient has a Swan-Ganz catheter)
- input and output
- swallowing ability
- mechanism of communication
- activity level
- skin integrity
- psychosocial issues
- patient and family education
- discharge planning.

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