

# Subarachnoid Hemorrhage

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A 66-year-old woman with a medical history of hypertension and coronary artery disease and a prosthetic mitral valve for which she was receiving anticoagulation therapy presented to the emergency department with the sudden onset of headache, nausea, vomiting, and subsequent loss of consciousness with seizure activity. The patient was treated with fosphenytoin and a computed tomography scan of the head without contrast was obtained, which showed a subarachnoid hemorrhage (**Figure 1**). She was admitted to the neurology critical care unit and a ventriculostomy was performed. Cerebral angiography revealed a basilar tip aneurysm (**Figure 2**) with ongoing bleeding. Endovascular coiling of the aneurysm was successful (**Figure 2**); however, the patient subsequently developed an episode of rebleeding. She had increasing pressor requirements with worsening renal function and absent corneal, gag, and oculocephalic reflexes. With the family's approval, the patient was placed on comfort care.

**S**ubarachnoid hemorrhage (SAH) is a devastating acute neurologic emergency that follows rupture of a blood vessel into the subarachnoid space.<sup>1</sup> SAH affects 21,000 to 33,000 persons per year in the United States,<sup>2</sup> leading to significant morbidity and mortality. The 30-day mortality rate approaches 50%, and those who survive often suffer major neurologic morbidity.<sup>3,4</sup> It is the most common type of stroke until the fifth or sixth decade of life, with peak incidence around age 55 years for men and age 60 years for women. SAH is more common in females than in males at a ratio of approximately 3:2.<sup>1</sup> This article reviews the diagnosis and medical management of SAH.

## ETIOLOGY

Cerebral aneurysms and arteriovenous malformations (AVMs) cause approximately 70% to 80% and 5% to 10% of nontraumatic cases of SAH, respectively (**Table 1**).<sup>1</sup> The autopsy prevalence of intracranial aneurysms in the adult population is between 1% and 6%.<sup>5-7</sup> The incidence of SAH from a ruptured aneurysm in the United States is estimated to be 1 per 10,000 persons per year.<sup>8</sup> Saccular aneurysms are typically formed at branch points of arteries, where arterial pulsation stress is maximal. The risk of rupture depends on the location, size, and wall thickness of the aneurysm.<sup>9</sup> Aneurysms less than 7 mm in diameter in the anterior cerebral circulation have the lowest risk of rupture, whereas risk is higher for aneurysms in the posterior circulation and increases with size.<sup>10</sup> In contrast to sporadic aneurysms, familial aneurysms are more likely to

be greater than 10 mm, and patients with familial SAH are more likely to have multiple aneurysms.<sup>11</sup>

AVMs are vascular anomalies that consist of a plexiform network of abnormal arteries and veins linked by one or more fistulae. They lack the typical capillary bed interposed between the arteriole and venule and have arterioles with a thinner than normal muscularis.<sup>12</sup> They are classified into 2 main groups: congenital and acquired. Acquired AVMs occur as a result of sinus thrombosis, trauma, or craniotomy.<sup>13</sup> The annual rate of hemorrhage is approximately 2% among patients with no history of bleeding; the annual rate of rehemorrhage is 18%.<sup>12</sup> The predominant forms of presentation are intracranial hemorrhage (65% of cases) and seizure (20%), and the remaining 15% are asymptomatic.<sup>14,15</sup>

## EVALUATION

### History

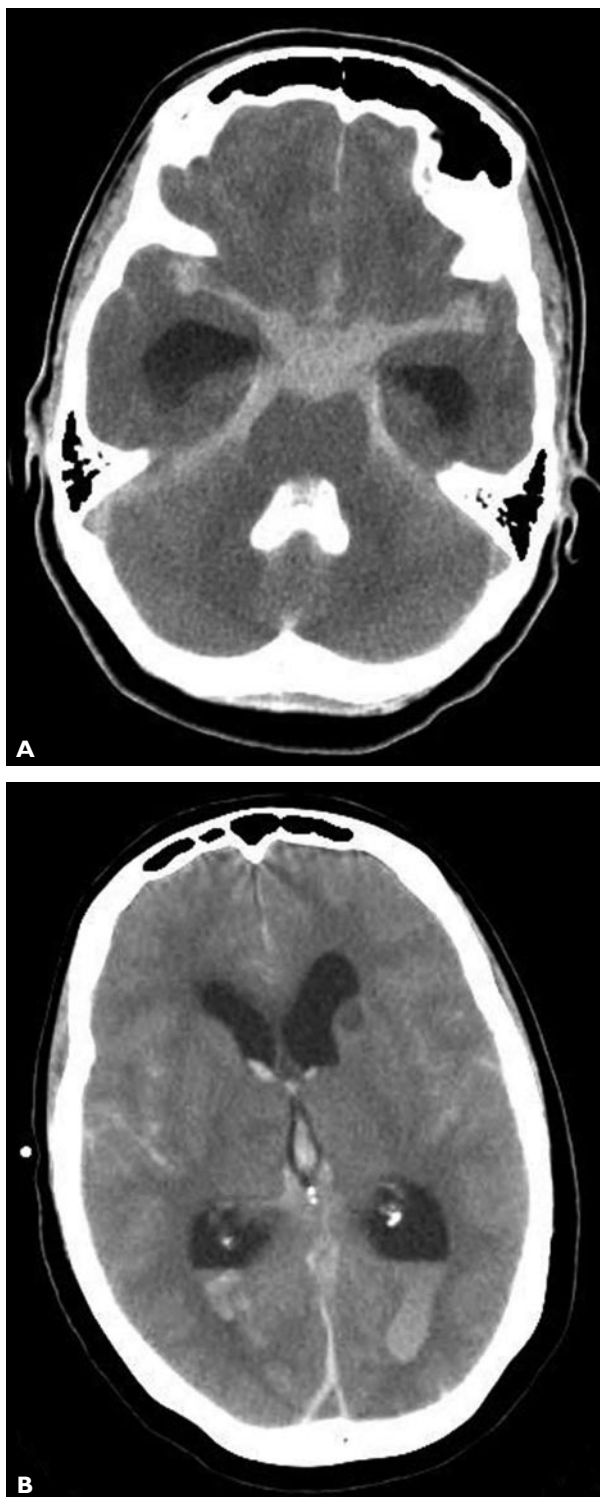
The classic presentation for SAH is the sudden onset of severe headache, often described by patients as "the worst headache of my life." Frequent associated symptoms include nausea, vomiting, photophobia, and focal or generalized acute neurologic symptoms, including seizures, changes in memory or the ability to focus, and meningismus. The patient may lose consciousness after the event, either transiently due to mildly increased

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### TAKE HOME POINTS

- Subarachnoid hemorrhage (SAH) is the most common stroke prior to the sixth decade of life.
- Consider SAH in a patient with new-onset or severe headache.
- Misdiagnosis of SAH at first physician contact is associated with normal mental status, small SAH volume, and right-sided aneurysm.
- Diagnostic evaluation includes careful history and physical examination, head computed tomography scan without contrast, lumbar puncture (which must be performed before SAH can be ruled out), and cerebral angiography.
- Obtain early neurosurgical consultation.
- Management includes procedural intervention as well as prevention and management of hospital complications, which may include vasospasm, re-bleeding, hydrocephalus, hyponatremia, hyperglycemia, and seizures.
- Prophylaxis for vasospasm is nimodipine within 12 hours after SAH diagnosis. For patients with an aneurysm secured by endovascular or surgical means, medical treatment of vasospasm is hyperdynamic therapy (hypervolemia, induced arterial hypertension, and hemodilution) with aggressive hemodynamic monitoring.

intracranial pressure or irreversibly in severe cases. Approximately one third of patients who present with SAH have a history of acute and unusual headache over the preceding weeks before the acute presentation, which is thought to be due to minor leaking of blood from the aneurysm into the subarachnoid space.<sup>5,16,17</sup> This warning headache may initially be misdiagnosed as migraine headache, sinusitis, influenza, or malingering.<sup>5,18</sup> Misdiagnosis occurs in 23% to 53% of patients with SAH on their first physician visit, so care must be exercised when evaluating patients with severe or new-onset headache.<sup>19,20</sup> Misdiagnosis is associated with normal mental status, small SAH volume, and right-sided aneurysm location at first medical contact. Of patients with normal mental status at first evaluation, misdiagnosis was associated with worse quality of life at 3 months and an increased risk of death or severe disability at 12 months.<sup>21</sup> Other epidemiologic data may help in deciding who should undergo work-up for SAH; **Table 2** lists risk factors for SAH.<sup>22-29</sup>



**Figure 1.** Computed tomography scan demonstrating subarachnoid hemorrhage with blood filling (A) the basal cisterns and fourth ventricle and (B) sylvian fissures, interhemispheric fissure, and third and lateral ventricles, as well as effacement of sulci secondary to hydrocephalus.

### Physical Examination

Physical examination is important to exclude other causes for headache, including glaucoma, sinusitis, or temporal arteritis. Nuchal rigidity occurs in approximately 70% of SAH cases. A neurologic examination must be performed to look for focal deficits. Aneurysms at the junction of the posterior communicating artery and the internal carotid artery may cause a third cranial nerve palsy<sup>30</sup> in which the affected eye has limited movement, pupillary dilatation, and/or inferolateral deviation;<sup>20</sup> this finding occurs in a small percentage of cases. An aneurysm in the cavernous sinus may cause sixth cranial nerve palsy.<sup>30</sup> Fundoscopy may reveal retinal hemorrhage or papilledema suggestive of SAH and increased intracranial pressure.<sup>20</sup> The presence of distal embolic phenomena should raise suspicion for an unruptured intracranial giant aneurysm.<sup>31</sup>

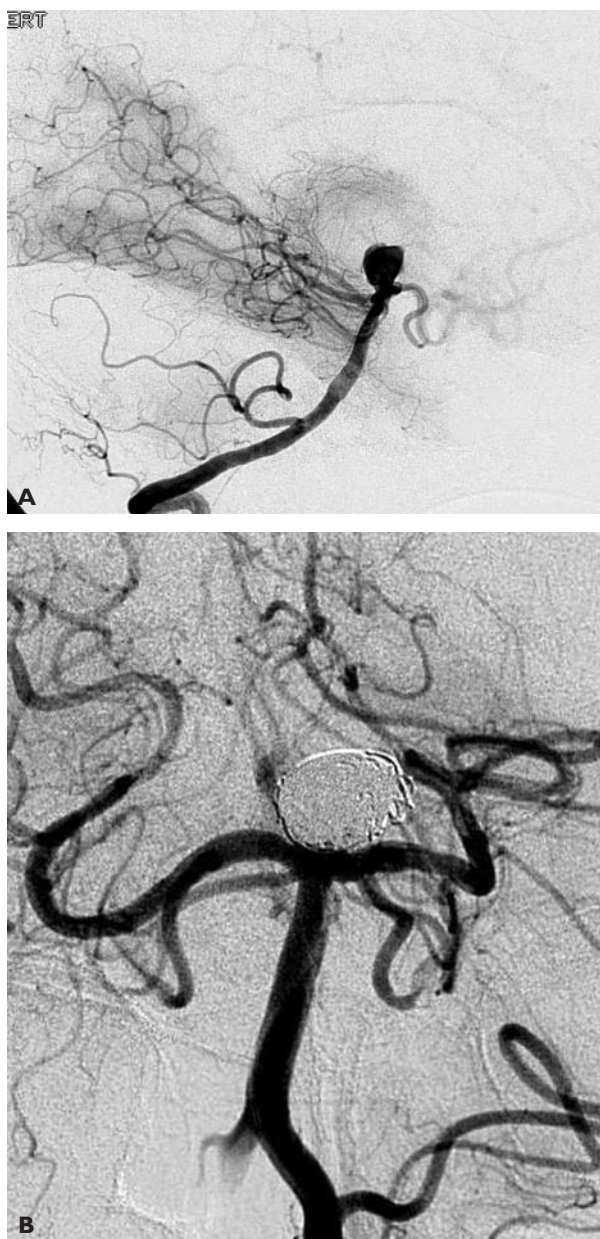
Patients who do not have a physical examination suspicious for SAH but still have no likely alternative diagnosis present the greatest challenge. Important diagnostic keys in the history that may tip the scale toward more aggressive work-up include pain quality that is severe or at least different from previous headaches, coexistent nausea and/or vomiting (particularly if vomiting is a new symptom with headache), syncope, diplopia, and seizure.<sup>20,32</sup>

### Diagnostic Imaging

Noncontrast computed tomography (CT) with thin cuts through the base of the brain is the initial diagnostic study of choice because of its high sensitivity and ability to determine the location of the hemorrhage. Sensitivity approaches 100% for scans performed in the first 12 hours after rupture<sup>33,34</sup> but dwindles to 50% at 1 week after rupture.<sup>35</sup> Thus, CT scan should be obtained as soon as possible not only in light of the critical need for early diagnosis, but also to maximize test sensitivity. While magnetic resonance imaging with fluid-attenuated inversion recovery shows promise in diagnosing SAH,<sup>19,36</sup> CT remains the first-line imaging modality because of its wider availability, lower cost, and easier access for imaging critical patients and because there is more experience with its interpretation.<sup>19,37</sup> False-negative tests may result from cases of severe anemia, cuts through the brain wider than 3 mm, or scans limited by motion artifact or other technical shortcomings.<sup>19,20</sup>

### Lumbar Puncture

If the head CT scan is negative, the next diagnostic step is lumbar puncture (LP). It is essential to perform LP prior to eliminating SAH from the differential di-



**Figure 2.** (A) Cerebral angiogram showing a basilar tip aneurysm measuring 1.3 cm. (B) Angiogram showing the aneurysm following endovascular coiling.

agnosis. Findings suggestive of SAH include the presence of red blood cells (RBCs), elevated opening pressure, and/or xanthochromia. Most LPs in known SAH have high levels of RBCs; even hemorrhage as small as 0.3 mL will lead to values as high as 10,000 RBCs/mL of cerebrospinal fluid.<sup>38,39</sup> The level in traumatic LPs is usually much lower; however, one must be mindful that any number of RBCs in the cerebrospinal fluid may indicate SAH. Opening pressure is elevated in approximately two

**Table 1.** Etiology of Subarachnoid Hemorrhage

Trauma
Cerebral aneurysms
Arteriovenous malformations
Perimesencephalic hemorrhage
Vasculitis
Hematologic causes, including disseminated intravascular coagulation, thrombotic thrombocytopenic purpura, and hemophilia
Central nervous system neoplasms
Extension of intracerebral hemorrhage
Iatrogenic injury during surgery
Arterial dissection

thirds of SAH cases,<sup>20</sup> a helpful clue that is not present in traumatic LP alone.<sup>19</sup>

Xanthochromia, a yellow discoloration, reflects the presence of RBC breakdown products, primarily to oxyhemoglobin and bilirubin, in the cerebrospinal fluid. Considered the traditional method for distinguishing the presence of SAH from traumatic LP, it is somewhat of a time-limited approach; it takes about 12 hours from SAH onset for xanthochromia to approach 100% sensitivity.<sup>38,40</sup> Although spectrophotometry has been recommended as the preferred method for detecting xanthochromia,<sup>41</sup> the majority of North American hospitals rely on visual inspection.<sup>42</sup> SAH may be excluded if experienced clinicians judge the cerebrospinal fluid to be colorless in standard conditions (in bright light, background of white paper or a white coat, and comparison with a tube of tap water). However, if the color is judged to be “yellow” or “doubtful,” spectrophotometry is recommended to determine the level of bilirubin extinction.<sup>43</sup> A positive value for bilirubin was defined at an extinction of 0.05 at wavelength 450–460 nm.<sup>43</sup> Use of spectrophotometry on all cerebrospinal fluid samples leads to a high false-positive rate and an unacceptably high number of unnecessary angiograms.<sup>42</sup>

### Angiography

For patients with positive CT scans or LP findings suggestive of SAH, neurologic or neurosurgical consultation should be obtained so that intravascular or surgical intervention may be considered. The diagnostic gold standard involves cerebral angiography, an imaging modality that is a poor screening test due to its more invasive nature, higher cost, and possibly greater morbidity; however, it allows for excellent visualization of the bleeding source and the potential for endovascular intervention to prevent rebleeding.<sup>44</sup> By the time angiography is performed, the bleeding may

**Table 2.** Risk Factors for Developing Subarachnoid Hemorrhage

Modifiable	Nonmodifiable
Hypertension	Personal history of SAH
Current or past smoking	Family history of any relative with SAH or intracranial aneurysm
Heavy alcohol use	Personal or family history of polycystic kidney disease or connective tissue disorder (ie, Ehlers-Danlos syndrome, Marfan syndrome, and pseudoxanthoma elasticum)
Lower educational level	
Low body mass index	
Cocaine and other illicit drugs	
Moderate to extreme physical exertion 2 hr prior to onset of SAH	

Data from references 22–29.

SAH = subarachnoid hemorrhage.

have stopped secondary to the cardiovascular collapse that often accompanies SAH.

Increasingly, institutions are using computed tomographic angiography (CTA) with 3-dimensional reformatted images in addition to or in place of traditional digital subtraction angiography to identify cerebral aneurysms. CTA holds significant promise as the preintervention imaging technique because it is noninvasive, has a much lower risk of complications, can be performed more quickly, requires less resources, is not painful to the patient, and is suitable for critical or unstable patients. CTA is safe and effective for diagnosing ruptured and unruptured cerebral aneurysms, with a detection rate of 100% for the presenting aneurysm.<sup>45</sup>

### GRADING SYSTEMS

Before proceeding with treatment, some means of quantifying outcomes for both aggressive and conservative measures are needed to guide interventions as well as discussions with the patient and family.<sup>46</sup> Multiple grading systems are available to assist in quantifying risks and outcomes, but unfortunately there is no one ideal system. The Hunt and Hess scale is easy to administer clinically and widely used (**Table 3**). A higher grade on the scale is indicative of a poor outcome,<sup>47–49</sup> but it has several limitations. Some of the clinical identification definitions are vague, making it difficult to distinguish between grades, and the scale does not take comorbidities into consideration.<sup>46</sup> The Fisher scale, which classifies SAH based on the appearance of blood on head CT scan, is the only radiologic grading system (**Table 4**).<sup>48,50</sup> Patients with scores of 3 or 4 have an increased risk of poor clinical outcome.<sup>48</sup> This scale is limited by inter-rater variability<sup>46</sup> as well as a lack of consideration for the patient’s overall clinical condition.

**Table 3.** Hunt and Hess Scale

Grade	Clinical Condition
0	Unruptured
I	Asymptomatic or minimal headache, slight nuchal rigidity
II	Moderate/severe headache, nuchal rigidity, no neurologic deficit except cranial nerve palsy
III	Drowsy, confused, mild focal neurologic deficit
IV	Stupor, moderate/severe hemiparesis, possible early decerebrate rigidity
V	Deep coma, decerebrate rigidity, moribund appearance

Adapted from Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. *J Neurosurg* 1968; 28:14-20; and Ogilvy CS, Carter BS. A proposed comprehensive grading system to predict outcome for surgical management of intracranial aneurysms. *Neurosurgery* 1998;42:959-70.

The Ogilvy and Carter system (Table 5) combines clinical, demographic, and radiologic data and is easy to administer and comprehensive in determining prognosis in patients undergoing surgical intervention.<sup>48</sup> This more recent evaluative system combines both the Hunt and Hess grade and Fisher scale score in addition to a broader range of variables that affect clinical outcomes. Grades 0 and 1 had good or excellent outcomes in more than 95% of patients. However, outcomes significantly worsened with grades above 1, with approximately 10% mortality with grade 2, 30% with grade 3, and 50% with grade 4. No grade 5 patients underwent surgery.

The Glasgow Coma Scale (GCS) and the World Federation of Neurological Surgeons (WFNS) SAH grading scale are 2 other commonly used clinical systems. The GCS (Table 6) is not specific to SAH but is a widely used standardized system for evaluating level of consciousness. A higher preoperative GCS score (maximum score 15) predicts better outcome.<sup>51</sup> The WFNS scale (Table 7) combines the GCS scoring system with the presence of motor deficits.<sup>52</sup> Although the WFNS scale is one of the most commonly used scoring systems,<sup>5</sup> a recent systematic review found conflicting data about its prognostic value.<sup>53</sup>

Each of these systems provides helpful information to assist in gauging prognosis, but all have limitations. Outcomes depend on a wide range of clinical information. When making clinical decisions, one must not rely on one grading scale but rather must consider the full spectrum of clinical data.

### MEDICAL MANAGEMENT

In the general management of patients with SAH, 2 major objectives must be addressed: identification of

**Table 4.** Fisher Scale

Score	Description of Blood on Head Computed Tomography Scan
1	No blood detected
2	Diffuse deposition or vertical layers of blood < 1 mm thick, no clots
3	Localized clot and/or vertical layers of blood > 1 mm thick
4	Intracerebral or intraventricular clots with diffuse or no blood

Adapted from Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. *Neurosurgery* 1980;6:1-9; and Ogilvy CS, Carter BS. A proposed comprehensive grading system to predict outcome for surgical management of intracranial aneurysms. *Neurosurgery* 1998;42:959-70.

**Table 5.** Ogilvy and Carter System

Points	Description
1	Hunt and Hess grade > III
1	Fisher scale score > 2
1	Aneurysm size > 10 mm
1	Patient age > 50 yr
1	Giant (≥ 25 mm) posterior circulation lesion

NOTE: Grade is determined by sum of points: grade 5 has worst prognosis and 0 has best prognosis.

Adapted from Ogilvy CS, Carter BS. A proposed comprehensive grading system to predict outcome for surgical management of intracranial aneurysms. *Neurosurgery* 1998;42:959-70.

the bleeding source with possible procedural intervention surgically or intravascularly, and management of subsequent complications. It is imperative to obtain early neurosurgical or cerebrovascular specialty consultation to treat intracranial aneurysms procedurally. The primary treatment modalities involve craniotomy with clip ligation (ie, "clipping") and endovascular aneurysmal occlusion with the use of detachable coils (ie, "coiling").<sup>8,9</sup>

The patient with SAH should be admitted to the intensive care unit (ICU) for continuous hemodynamic monitoring. Ideally, patients would be managed in specialized neurosurgical centers in neurology critical care units that have high SAH patient volume, as these have been shown to improve patient outcomes.<sup>9,54</sup> An airway must be secured and invasive monitoring of central venous pressure and/or pulmonary artery pressure as well as continuous arterial blood pressure must be maintained. To prevent increases in intracranial pressure, patients should be given stool softeners and

**Table 6.** Glasgow Coma Scale

<b>Eye opening</b>	
Spontaneous	4
Response to verbal command	3
Response to pain	2
No eye opening	1
<b>Best verbal response</b>	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
No verbal response	1
<b>Best motor response</b>	
Obeys commands	6
Localizing response to pain	5
Withdrawal response to pain	4
Flexion to pain	3
Extension to pain	2
No motor response	1

NOTE: Score is determined by summing scores in the 3 parameters, with a score of 3 predicting a poorer outcome and 15 predicting a better outcome.

analgesics and kept on bedrest. Early neurosurgical consultation is required in the event that surgical intervention or invasive monitoring of intracranial pressure is needed. Patients on anticoagulation therapy have significantly worse outcomes.<sup>55</sup> Therefore, it is prudent to reverse any anticoagulant effect with vitamin K and fresh frozen plasma.<sup>56,57</sup>

### Management of Complications

**Vasospasm.** Vasospasm and rebleeding are the most common causes of complications.<sup>58</sup> Vasospasm is characterized by mental status changes with focal neurologic deficit. It rarely occurs before day 3, peaks at days 6 to 8, and seldom occurs after day 17.<sup>59,60</sup> Vasospasm leads to delayed cerebral ischemia with 2 major patterns: single cortical infarction, usually near the site of a ruptured aneurysm, and multiple widespread lesions that are often not related to the site of the ruptured aneurysm.<sup>61</sup>

Cerebral angiography is the study of choice for diagnosing vasospasm, with a sensitivity of 80%. Multiple other studies have been proposed to diagnose vasospasm because of the relative inconvenience of performing angiography on a routine basis. Transcranial Doppler ultrasound is commonly used for daily monitoring because it can be performed with relative ease.<sup>62</sup> However, its use is quite controversial. Although one study showed that

**Table 7.** World Federation of Neurological Surgeons Grading Scale

Grade	GCS Score	Motor Deficit
1	15	Absent
2	13–14	Absent
3	13–14	Present
4	7–12	Present or absent
5	3–6	Present or absent

Adapted from Report of World Federation of Neurological Surgeons Committee on a Universal Subarachnoid Hemorrhage Grading Scale. *J Neurosurg* 1988;68:985–6.

GCS = Glasgow Coma Scale.

daily transcranial Doppler ultrasound monitoring had sensitivity similar to angiography in the anterior circulation (73%),<sup>62</sup> the authors of a meta-analysis found poor sensitivity and specificity for diagnosing vasospasm and recommend against its use as a screening tool.<sup>63</sup> Other developing techniques include continuous electroencephalographic monitoring, which has the potential to detect cerebral ischemia very early, before permanent neuronal injury occurs;<sup>64,65</sup> single-photon emission computed tomography, which has shown promise in the evaluation of postoperative patients susceptible to vasospasm<sup>66</sup> but has poor feasibility and requires repeated measurements for diagnosis;<sup>67</sup> and near-infrared spectroscopy, which has the potential to be a safe, noninvasive technique that could be performed at the bedside.<sup>68</sup>

Prior to the onset of vasospasm, all patients should receive prophylaxis with nimodipine within 12 hours after SAH is diagnosed. The typical dose is 60 mg every 4 hours by mouth or nasogastric tube, and it should be continued for 21 days. Despite a lack of statistically significant improvement in overall mortality, a meta-analysis demonstrated a significant decrease in vasospasm-associated mortality with nimodipine prophylaxis.<sup>69</sup> The addition of simvastatin before or after SAH may also prove to be a potential treatment for reducing cerebral vasospasm.<sup>70,71</sup> Finally, antiplatelet therapy may play a role in reducing delayed cerebral ischemia, although prospective studies are necessary to determine safety and effect on overall outcome.<sup>72</sup>

If the aneurysm has been secured by endovascular or surgical means and vasospasm has occurred, the core of medical treatment is hyperdynamic therapy, also called “triple-H therapy.” Triple-H therapy consists of hypervolemia, induced arterial hypertension, and hemodilution with aggressive hemodynamic monitoring of central venous pressures or pulmonary artery pressures with a Swan-Ganz catheter. It is contraindicated

and should not be used if the aneurysm has not been secured. The treatment involves aggressive fluid hydration and hemodilution with colloid or crystalloid fluids and the addition of vasopressor support in the case of neurologic changes refractory to hypervolemia. In a study of 113 patients with aneurysmal SAH treated with a management protocol that consisted of early surgery and triple-H therapy, hyperdynamic therapy reversed deficits in 60% of patients, while 24% remained stable and 16% worsened.<sup>59,73</sup>

Triple-H therapy is not without risk. Complications occur in 10% to 20% of patients,<sup>59,60,74</sup> including pulmonary edema, dilutional hyponatremia, and myocardial infarction, as well as intrinsic complications related to central venous lines and/or Swan-Ganz catheters.<sup>60,74-76</sup> However, a recent trial showed that hypervolemia did not increase cerebral blood flow or blood volume compared with normovolemic therapy,<sup>77</sup> indicating that aiming for normovolemia may be a better goal that could provide benefit with reduced risk of adverse events.

In cases refractory to triple-H therapy, intra-arterial infusions of antispasmodics such as nicardipine, verapamil, or papaverine<sup>78</sup> as well as angioplasty have been used. Early (< 24 hr after onset) transluminal balloon angioplasty is preferred to the antispasmodic infusions<sup>78</sup> and leads to significant clinical improvement, with moderate to dramatic improvement in approximately 70% of cases and few adverse outcomes.<sup>79</sup>

**Rebleeding.** Rebleeding has a mortality rate of 70%, 4% during the first 24 hours and 1% to 2% per day during the next 4 weeks.<sup>79,80</sup> It is associated with a marked reduction in chance of survival with functional independence at 3 months. Aneurysm repair, the primary treatment measure, significantly reduces risk of rebleeding.<sup>81,82</sup> To reduce the risk of rebleeding prior to aneurysm repair, blood pressure must be carefully managed.<sup>80</sup> Management involves a delicate balance between maintaining adequate cerebral perfusion and decreasing the risk of rebleeding. Systolic blood pressure (SBP) should be maintained above 100 mm Hg in all patients for 21 days.<sup>83,84</sup> Prior to repair, SBP should be kept below 160 mm Hg. During symptomatic vasospasm, SBP should be increased to 200 to 220 mm Hg. Pharmacologic agents used for blood pressure management in patients with SAH are shown in **Table 8**.

**Hydrocephalus.** If a patient has acute deterioration in mental status, a repeat CT scan should be obtained to look for reversible causes of coma. One such cause, acute hydrocephalus, occurs in approximately 25% of patients after initial SAH.<sup>80</sup> The amount of SAH detected on CT is a predictor of the presence of hydrocephalus.

**Table 8.** Agents Used for Blood Pressure Management in Subarachnoid Hemorrhage

Hypotension	Hypertension
Phenylephrine	Labetalol
Norepinephrine	Esmolol
Dopamine	Nicardipine
Sliding-scale nimodipine:	
SBP 120–140 mm Hg: 30 mg nimodipine	
SBP < 120 mm Hg: hold nimodipine	

SBP = systolic blood pressure.

Approximately one third of patients admitted for aneurysmal SAH require diversion of cerebrospinal fluid with a temporary external ventricular drain or permanent ventricular shunt.<sup>85</sup> Draining too much cerebrospinal fluid increases the risk of rebleeding and cerebral vasospasm.<sup>80,86</sup> Factors that increase the risk of shunt-dependent hydrocephalus include increasing age, female sex, poor admission Hunt and Hess grade, thick SAH on admission CT scans, intraventricular hemorrhage, radiologic hydrocephalus at the time of admission, distal posterior circulation location of the ruptured aneurysm, vasospasm, and endovascular treatment.<sup>87</sup>

**Hyponatremia.** Hyponatremia occurs in 30% to 35% of patients after SAH.<sup>88,89</sup> It is classically attributed to cerebral salt wasting and treated with fluid replacement and is often found in association with cerebral vasospasm.<sup>90,91</sup> However, a recent study showed hyponatremia to be primarily due to the syndrome of inappropriate antidiuretic hormone secretion (SIADH) in 69% of cases or hypovolemic hyponatremia in 21% of cases.<sup>92</sup> SIADH is typically managed with fluid restriction, which may be indicated in SAH. However, adequate hydration must be maintained to prevent cerebral vasospasm and, in light of this, fluid restriction is generally not recommended in patients with SAH. Additionally, hydrocortisone appears to be effective in minimizing excessive natriuresis and the resulting hypovolemia and hyponatremia.<sup>90</sup>

**Hyperglycemia.** Hyperglycemia is common after SAH and is likely related to the stress response following such a devastating illness. While no prospective randomized studies have been performed, a preliminary study showed insulin therapy aimed to keep blood glucose levels in a range of 90 to 126 mg/dL to be feasible and safe.<sup>93</sup> Intensive insulin therapy in general medical and surgical ICU patients reduces the incidence of morbidity and mortality;<sup>94-97</sup> therefore, intensive blood glucose control with insulin therapy also should be considered for patients with SAH.

**Epilepsy.** Epilepsy occurs in 7%<sup>98</sup> to 35%<sup>99</sup> of cases of SAH. Seizures during the early stages after SAH can lead to rebleeding, as they increase cerebral blood flow, although in multivariate analysis there was no such association.<sup>82</sup> The American Heart Association recommends the routine use of seizure prophylaxis for all patients with SAH in the immediate posthemorrhage period. However, a large case series demonstrated that prophylaxis with phenytoin was associated with poor neurologic and cognitive outcome following SAH.<sup>100</sup> Thus, antiepileptic drug therapy should be used with caution and may be more appropriately reserved for patients who have seizures while in the hospital or delayed onset of epilepsy after discharge.

**Other complications.** Multiple other medical complications are common in SAH, including pneumonia, sepsis, cardiac arrhythmias, and cardiac enzyme elevations. Physicians are reminded to keep the head of the bed elevated to 30 degrees and to treat bacterial pneumonia with antibiotics. Prophylaxis with pneumatic compression devices should be used to decrease risk of deep venous thrombosis (DVT) and pulmonary embolism (PE).<sup>101</sup> DVT occurs in 1% to 5% of patients, and PE has an incidence of 0.8%; however, anticoagulation is contraindicated in the acute stage due to the recent hemorrhage and often recent intracranial surgery. An inferior vena cava filter may be placed in the case of DVT or PE.<sup>80</sup>

### SAH RECURRENCE

Following surgical clipping, the cumulative recurrence rate of SAH was 2.2% at 10 years and 9.0% at 20 years after original treatment. Patients with ruptured cerebral aneurysms have higher risks for recurrent SAH, even after complete surgical obliteration.<sup>102</sup> In a later study, the recurrence rate of SAH after surgical clipping was about 22 times higher than expected in populations of comparable age and sex.<sup>103</sup>

### CONCLUSION

SAH presents acutely and has the potential to cause significant morbidity and mortality. Because early intervention may lead to better outcomes, physicians must consider it in the differential diagnosis of patients presenting with new-onset severe headache and/or loss of consciousness. Once the diagnosis has been made, it is important to manage the patient in the ICU setting with continuous hemodynamic monitoring and frequent neurologic evaluations and to obtain early neurosurgical consultation to assist in management. Because of poor outcomes in many patients following SAH, physicians must have daily conversations with the

patient's family to keep them current on the patient's status and to assist with making realistic clinical decisions. Despite the potentially devastating results of SAH, the outcomes after SAH have significantly improved over the past 2 decades, primarily due to aggressive treatment of hydrocephalus and vasospasm, improved surgical techniques, and better ICU care.<sup>9</sup> **HP**

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