Treatment of Subarachnoid Hemorrhage

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KEYWORDS
- Subarachnoid hemorrhage
- Vasospasm
- Delayed cerebral ischemia
- Aneurysm
- Transcranial Doppler ultrasonography

KEY POINTS
- Subarachnoid hemorrhage is typically caused by a ruptured intracranial aneurysm and presents with a sudden severe headache often accompanied by syncope, nausea, and vomiting.
- Initial management includes airway assessment, blood pressure control, treatment of pain, and noncontrast computed tomography (CT), followed by urgent catheter or CT angiography.
- To reduce the risk of delayed cerebral ischemia (DCI), all patients should be treated with nimodipine and be maintained in a euvoicmic state in the days after hemorrhage.
- The development of narrowing of large cerebral vessels (vasospasm) can be detected with transcranial Doppler ultrasonography, CT, or conventional angiography. Vasospasm is closely correlated with DCI, but each can occur independently.
- DCI can be treated with combinations of blood pressure or cardiac output augmentation, angioplasty of proximal vasospastic vessels, and selective intra-arterial infusions of vasodilators.

INTRODUCTION

Nontraumatic subarachnoid hemorrhage (SAH) typically presents as a sudden severe headache, often described as “the worst headache of my life.” Consciousness may be impaired, but focal neurologic deficits are uncommon. The incidence of SAH ranges from 10 to 18 per 100,000 people.1 It often occurs in middle-aged patients and has a female predominance.2 Of all spontaneous SAHs, 80% are the result of the rupture of an intracranial aneurysm, 15% do not have a bleeding source identified, and the remainder are owing to a myriad of other causes, mostly vascular malformations, but also vasculitis or posterior reversible vasoconstriction syndrome. Genetic factors seem to play a role in some families.
Intracranial berry (ie, saccular) aneurysms are typically found near the circle of Willis at the branching points of large cerebral arteries. Hemodynamic stress at arterial branching sites and inflammation seem to contribute to aneurysm formation. About 2% to 5% of the population harbor intracranial aneurysms. Although aneurysms are thought to develop over many years, cases of rapid growth in size do occur. They frequently arise off the internal carotid artery at the take-off of the anterior and posterior communicating arteries and middle cerebral artery. Relatively few occur in the posterior circulation. Risk factors for aneurysmal rupture include smoking, hypertension, alcohol use, and having first-degree relatives with SAH. Autosomal-dominant polycystic kidney disease is the most common heritable disorder to increase the risk for SAH; others include connective tissue disorders.

PATIENT EVALUATION

The classical clinical triad for presentation of SAH includes sudden severe headache, syncope, and vomiting. Other common symptoms include nausea, photophobia, and altered consciousness. Focal neurologic deficits occur in about 10% of patients. They can be owing to aneurysmal compression of a cranial nerve (typically a posterior communicating artery aneurysm compressing the third nerve producing ptosis, a dilated pupil, and limited medial and vertical gaze). More ominous are focal deficits owing to thick subarachnoid clots or parenchymal hematoma. Blood released under high pressure may directly cause damage to local tissues. Additionally, the vessel rupture produces an increase in intracranial pressure (ICP) that approaches arterial pressure and cerebral perfusion falls to nil. If hemorrhage stops and the acute rise in ICP is transient, it can result in nausea, vomiting, and syncope; if the high pressure is sustained, it is uniformly fatal. Exposure of the meninges to blood causes irritation resulting in photophobia, neck stiffness, and eventually back pain. Blood pressure is frequently elevated, which may increase the risk of re-rupture.

Initial Stabilization

Initial management should focus on airway management in comatose patients and blood pressure control to stabilize the patient, with the goal of obtaining a computed tomography (CT) scan as soon as possible (Box 1).

Patients may be unable to protect their airway and require intubation for multiple reasons, including hydrocephalus, seizure, or sedation. In addition, elective intubation may be necessary in agitated patients to safely and expeditiously perform cerebral angiography.

To reduce the risk of rebleeding before the aneurysm is secured, blood pressure should be maintained at the patients baseline levels or, if unknown, a mean arterial blood pressure of less than about 110 mm Hg. Effective pain control may be sufficient to manage blood pressure; otherwise short-acting intravenous medications (eg,
labetalol and hydralazine) are preferred (Table 1). Alternatively, continuous infusions of calcium channel blockers (eg, nicardipine) can be effective, especially in patients with refractory hypertension. Cerebral vasodilators such as nitrates should be avoided because they can raise the ICP. If acute hydrocephalus is present, then management of blood pressure should be mitigated until a ventricular drain has been placed. After the aneurysm is secured, the blood pressure goals should be liberalized. This permissive hypertension may help to augment cerebral perfusion should delayed cerebral ischemia (DCI) develop.

Pain control is best achieved with judicious use of short-acting intravenous medications to avoid oversedation. Often, opiates are needed to be effective. Long-acting agents should be used with caution because their sedatives effects are difficult to distinguish from the development of hydrocephalus.

**DIAGNOSIS**

**SAH**

The preferred diagnostic procedure to identify the presence of SAH is noncontrast CT. In the first 12 hours, blood will be apparent on CT scan in nearly 100% of cases; over the first 24 hours, this value drops to 95% to 99%. Although magnetic resonance imaging (MRI) may be as sensitive as CT within the first 1 or 2 days, it is usually logistically much more difficult to obtain. Two or more days after the ictus, MRI with fluid-attenuated inversion recovery or susceptibility weighted imaging sequences may be more sensitive. If a diagnostic CT and/or MRI are negative and clinical suspicion is high, a lumbar puncture should be performed. Although the presence of a large number of red blood cells in the fluid is suggestive of SAH, it is frequently owing to a traumatic tap. A more definitive test is to evaluate the fluid for xanthochromia using spectrophotometric analysis. If the results are equivocal, then vascular imaging to look for an aneurysm may be considered.

**Identification of Bleeding Source**

Until recently, conventional digital subtraction angiography was considered the best test to elucidate an aneurysmal source of hemorrhage. More recently, CT angiography (CTA) has come into regular use but may miss small aneurysms. The sensitivity and specificity for CTA depends on the modality and experience of the reader and compared with conventional angiography is respectively, 90% to 97% and 93% to 100%. Most of the aneurysms missed are less than 4 mm. MRI, magnetic resonance angiography, or CTA may also help with operative planning. If cerebral angiography fails to find an aneurysm, it should be repeated in a few days, because an aneurysm missed on the initial angiogram may be identified.

In 10% to 15% of SAH cases, an aneurysm or other cause of hemorrhage is not identified. The majority of these patients fit into the syndrome of perimesencephalic nonaneurysmal SAH. In this case, the blood is only present in the basal cisterns and is thought to arise from venous rather than arterial bleeding. Their course is usually

<table>
<thead>
<tr>
<th>Table 1 Medications for blood pressure control on presentation</th>
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<tr>
<td><strong>Dose</strong></td>
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<tr>
<td>Labetalol</td>
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<td>Hydralazine</td>
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<td>Nicardipine</td>
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benign, without risk of rebleeding. DCI, vasospasm, and hydrocephalus can occur in these patients, although it is rare.

**Clinical Grading Scales**

There are 3 SAH grading scales that are well known and typically assigned at presentation. Each of the 3 scales was created for a different reason and none are strongly correlated with outcome. The Hunt and Hess scale was created in 1986 to help stratify patients based on surgical risk. It is easy to administer, well known, and has some predictive value for outcome. However, the categories may overlap and patients with poor grades can still have good outcomes. The World Federation of Neurosurgeons scale was created in 1988 based on the Glasgow Coma Scale to help standardize assessment. It is easy to administer but reviews are mixed on how well each stage correlates with outcome. The modified Fisher scale was created from the Fisher scale to better predict cerebral vasospasm based on CT characteristics in 2006. It does show an increase in vasospasm risk in the worst grade and therefore is the most used for predicting DCI.

**TREATMENT OF THE ANEURYSM**

Aneurysms can be treated with surgical clipping or endovascular coiling. The choice of which treatment to perform is based on the location and shape of the aneurysm (neck to dome ratio), as well as the type of patient. Aneurysms in the middle cerebral artery region or in tortuous vessels are typically surgically clipped because of the difficulty in reaching the aneurysm through endovascular methods. Aneurysms that are deep in the brain or in the posterior circulation are better suited for endovascular coiling. Aneurysms in patients with multiple comorbidities are often treated with endovascular coiling. The International Subarachnoid Trial was a landmark study that randomly assigned aneurysms with clinical equipoise to clipping or coiling. Patients who had their aneurysms coiled had lower mortality and disability (7% absolute risk reduction of poor outcome) but a greater risk of rebleeding. Typically, patients who have their aneurysm coiled require follow-up vascular imaging after 6 to 12 months to identify any inadequate occlusion and provide retreatment if coil compaction and recannulation of the aneurysm occurred.

**COMPLICATIONS**

**Seizures**

It is not uncommon for seizure-like activity to occur at the time of hemorrhage. Whether these represent true seizures or motor posturing owing to elevated ICP is uncertain. Several studies have shown that there is no correlation between seizure activity at the time of aneurysm rupture and long-term epilepsy.

The risk of clinical seizures during hospitalization is low, at about 2%. However, in the minority of patients who have impaired consciousness, nonconvulsive status epilepticus is increasingly being found. This has led to frequent use of continuous electroencephalographic monitoring in comatose patients. Based on data from the International Subarachnoid Trial, the risk of seizures after discharge is greater among patients who had their aneurysm treated surgically and can occur in up to 10% of patients. In patients who will have their aneurysms surgically repaired and have not had a seizure, most practitioners administer a short (3- to 7-day) course of prophylactic anticonvulsants. Patients who undergo coiling are not given prophylactic anticonvulsants in many centers. After a retrospective review that found that use of phenytoin
after SAH was associated with worse long-term outcomes, leviracetem has become the preferred agent for prophylaxis.19 Patients who have had a seizure tend to be kept on anticonvulsants for one to several months.

**Rebleeding**

Rebleeding can be a devastating complication of SAH. In the first 24 hours, 4% to 15% of patients will rebleed; the risk of rebleeding decreases over the next 2 weeks. Administration of antifibrinolytics, early aneurysm repair, and blood pressure control are employed to reduce the chances of rebleeding.

Prolonged use of antifibrinolytics reduces the risk of rebleeding, but is also associated with increased ischemic events, negating any beneficial effects.20 More recent studies of short-term use of antifibrinolytics (≤3 days) suggest that rebleeding can be reduced without more risk of ischemia; thus they may be administered if aneurysm repair will be delayed. An ongoing multicenter, prospective, randomized, open-label trial is underway to determine whether a short-term antifibrinolytic leads to better functional outcome.21

Diagnostic CTA or catheter angiography should be performed as soon as possible, so that surgery or coiling can be undertaken and the aneurysm secured. The goal is to have the aneurysm repaired in less than 24 hours from presentation. Again, patients with unsecured aneurysms should have their blood pressure maintained in their normal range, or, if unknown, below a mean blood pressure of about 110 mm Hg.

**Hydrocephalus**

Hydrocephalus can complicate the SAH shortly after hemorrhage or it can develop in a delayed fashion. Acute hydrocephalus is evident on presentation or manifested by a progressive worsening of mental status within the first 1 to 3 days. On the initial CT scan, many patients exhibit radiographic hydrocephalus (which is based on the bicau-date index), but may be asymptomatic. About 20% develop symptomatic hydrocephalus and require placement of an external ventricular drain through a burr hole in the skull.22 This fluid is allowed to drain until the patient’s aneurysm is secured and patient is more stable. In about 40%, of cases hydrocephalus resolves and placement of a permanent shunt is not required. About 14% to 23% of all SAH patients, or 60% of those patients with an external ventricular drain, develop chronic hydrocephalus and require permanent cerebrospinal fluid (CSF) diversion.23–27 Patients without acute hydrocephalus can develop delayed hydrocephalus weeks after hemorrhage, but this is rare. Older age, thicker blood clot in the subarachnoid space, increased ventricular size, and worse clinical grade are predictive of the need for placement of a permanent shunt.24,28

**Cardiac**

Cardiovascular disturbances after SAH range from minor disturbances in cardiac rate and rhythm to congestive heart failure and cardiogenic shock. Sinus arrhythmia, peaked T waves, T wave inversions, ST segment depressions or elevations, and prolongation of the QT interval are commonly seen on an electrocardiogram.29 More significant rhythm disturbances are infrequent. Cardiac enzymes are elevated in 20% to 30% of patients, reflecting a catecholamine-related myocardial injury and a hypercontractile state rather than a lack of coronary blood flow.30,31 Still, in patients with coronary artery disease, cardiac ischemia must be considered. Although the mean troponin value is typically low, some patients can have elevations of greater than 10.92 In general, beta-blocking agents are administered to patients with elevated cardiac enzymes.
Echocardiography may be useful to help distinguish cardiac ischemia from catecholamine-induced cardiac injury and helps to assess cardiac function. Patients with changes on an electrocardiogram or elevated enzymes are more likely to have echocardiographic changes. Left ventricular dysfunction with wall motion abnormalities or a classic Takotsubo cardiomyopathy with apical ballooning are common echocardiographic findings. Takotsubo cardiomyopathy, so called owing to its similar shape on an echocardiogram to a Japanese “octopus pot,” is more commonly found in those with a poor grade of SAH and elevated cardiac enzymes and is associated with an increased risk of cerebral vasospasm. Acute cardiomyopathies may require aggressive management using standard approaches to management of congestive heart failure, including judicious diuresis, inotropic agents, and, in extreme cases, intra-aortic balloon pumps. Cardiac dysfunction, even if severe, typically begins to resolve within several days.

Cardiac dysfunction is frequently complicated by pulmonary edema attributed to both cardiac and neurogenic causes. Pulmonary edema owing to a neurologic injury is typically managed the same way as other causes of pulmonary edema. When mechanical ventilation is needed, hypercarbia should be avoided owing to its detrimental effect on ICP. Similarly, nitroglycerin and nitroprusside should be avoided because their venovasodilatory effects can markedly elevate ICP. Because cardiomyopathy occurs early after hemorrhage before the period of vasospasm, lower blood pressures may be tolerated.

**Fever**

Fever is associated with worse outcomes, including increased disability and cognitive impairment outcomes from SAH. It is likely a component of the systemic inflammatory response caused by SAH. Initially, all fevers should be investigated as a possible infection. Antipyretics (typically acetaminophen and ibuprofen) may be effective, but in some cases additional measures are needed including ice packs, cooling blankets, and other surface or intravascular cooling devices. As fever is treated, shivering can occur, increasing metabolic demand and heat production. Shivering should be treated; options include surface counterwarming, meperidine, buspirone, dexmedetomidine, and potentially intravenous magnesium infusion.

**Hyponatremia**

Hyponatremia complicates the course of about one third of SAH patients and has been associated in past and contemporary studies with poorer outcomes. A number of studies indicate that it is likely owing to a combination of cerebral salt wasting (excessive renal sodium and water excretion) and syndrome of inappropriate antidiuretic hormone (inappropriate renal water retention). In an attempt to avoid hyponatremia, SAH patients are administered isotonic fluids. Because of its potential for exacerbating cerebral edema, hyponatremia is usually treated at very mild levels. Because hypovolemia associated with fluid restriction may increase the risk for DCI, hyponatremia after SAH is not treated with fluid restriction, but with oral free water restriction and administration of mildly hypertonic fluids such as 1.5% or 2% sodium chloride. Use of fludrocortisone or hydrocortisone may help with diuresis and help to reduce hyponatremia. Vasopressin receptor agonists are effective in correcting hyponatremia, but they must be used with caution because they can cause a brisk diuresis and hypovolemia.

**Vasospasm and DCI**

It has long been recognized that some SAH patients deteriorate neurologically several days after hemorrhage. More than 5 years ago, these deteriorations were linked with
arterial narrowing and later with reduced cerebral blood flow (CBF). Although arterial narrowing is currently referred to as “vasospasm,” the clinical syndrome is now referred to as “delayed cerebral ischemia” (DCI). DCI is defined as the occurrence of neurologic deterioration that is not owing to other causes, or the evidence of a new infarct on CT imaging performed more than 72 hours after aneurysm rupture (Box 2, Tables 2 and 3). Infarction is a major complication of SAH because it has such a great impact on functional outcome. DCI peaks between days 3 and 14 and affects about 30% of patients. The risk of DCI is increased with a greater volume of SAH, intraventricular hemorrhage, poor clinical condition on admission, and a history of smoking.

DCI may be caused by many factors, including arterial vasospasm, cortical spreading depression, inflammation, and intravascular microthrombosis. Vasospasm of large cerebral conducting vessels can be easily identified with angiography or transcranial Doppler ultrasonography (TCD) and is common in patients who develop DCI. Approximately 70% of patients develop evidence of vasospasm after SAH and somewhat fewer than half develop DCI. The release of oxygenated hemoglobin into the CSF initiates an inflammatory cascade that results initially in smooth muscle contraction and eventually in hypertrophy and fibrosis of the vessel walls. Arterial vasospasm may start appearing 3 days after rupture and reaches a peak in incidence and severity at 7 to 10 days.

**DCI prophylaxis: euvolement**

Up to half of SAH patients treated with standard maintenance volume of fluids develop intravascular volume contraction. In older studies, fluid restriction to treat hyponatremia was associated with infarction and worse outcomes. In randomized, controlled trials, prophylactic hypervolemia did not improve CBF or clinical outcomes and was associated with a higher risk of cardiopulmonary complications. Typically euvolement is ensured by strict monitoring of fluids and replacement of urine output starting approximately 3 days after the initial SAH. Fludrocortisone or hydrocortisone may be helpful in patients with significant diuresis. The use of pulmonary artery catheters and central venous pressure monitoring to direct fluid management is not currently recommended owing to potential complications and a lack of data to support effectiveness.

<table>
<thead>
<tr>
<th>Box 2</th>
<th>Management of delayed cerebral ischemia (DCI)</th>
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<tbody>
<tr>
<td>Prophylaxis</td>
<td>Nimodipine</td>
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<tr>
<td>Prophylaxis</td>
<td>Maintenance of euvolement</td>
</tr>
<tr>
<td>Prophylaxis</td>
<td>Lumbar drainage</td>
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<tr>
<td>Symptomatic DCI</td>
<td>Medical</td>
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<tr>
<td>Symptomatic DCI</td>
<td>○ Induced hypertension</td>
</tr>
<tr>
<td>Symptomatic DCI</td>
<td>○ Cardiac output augmentation</td>
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<tr>
<td>Symptomatic DCI</td>
<td>○ Transfusion</td>
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<tr>
<td>Symptomatic DCI</td>
<td>Endovascular</td>
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<tr>
<td>Symptomatic DCI</td>
<td>○ Angioplasty</td>
</tr>
<tr>
<td>Symptomatic DCI</td>
<td>○ Intra-arterial vasodilators</td>
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DCI prophylaxis: calcium channel blockers
Nimodipine has been standard of care for SAH for almost 3 decades. In a recent meta-analysis, nimodipine had a relative risk reduction of poor outcome of 0.67 with a recommended dose of 60 mg every 4 hours for 3 weeks.\(^4\) Nimodipine seems to exert its effect owing to its action on neuronal calcium channels; it does not reduce the rate of vasospasm even though it improves outcomes.

DCI prophylaxis: lumbar drains
Lumbar drains may wash out the blood from the subarachnoid space and, consequently, inflammatory mediators that could cause cerebral vasospasm. Recent studies have suggested that lumbar CSF drainage can prevent DCI.\(^4\) Although it does not change the need for permanent CSF diversion, and the results for long-term outcomes are not robust, it has consistently shown a decrease in DCI and further studies are ongoing.

Diagnosing Vasospasm and DCI
Diagnosis and monitoring for clinically relevant vasospasm and DCI can be done several ways. Neurologic examinations are done multiple times per day (usually every 2 hours) to monitor for clinical signs of DCI. If a clinical examination deteriorates (a decrease of 2 points on the Glasgow Coma Scale or a new focal deficit), an evaluation for other causes (fever, metabolic disturbance, cerebral edema, or hydrocephalus) is indicated. Additional testing to corroborate a diagnosis of DCI (see below) may be undertaken or therapy initiated.

TCD
Some centers perform daily TCD to measure CBF velocity. As vessels narrow from vasospasm, CBF velocities rise (assuming CBF is unchanged). The threshold for mild vasospasm is considered greater than 120 cm/sec in the middle cerebral artery.

### Table 2
**Definitions**

<table>
<thead>
<tr>
<th>Term</th>
<th>Large Vessel Narrowing</th>
<th>Clinical Symptoms</th>
<th>Infarction</th>
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<tbody>
<tr>
<td>Vasospasm</td>
<td>Identified on TCD, angiogram or CTA</td>
<td>May or may not be present</td>
<td>May or may not be present</td>
</tr>
<tr>
<td>DCI</td>
<td>May or may not be present</td>
<td>Must be present</td>
<td>May or may not be present, if present sufficient for diagnosis</td>
</tr>
</tbody>
</table>

**Abbreviations:** CTA, computed tomography angiography; TCD, transcranial Doppler ultrasonography.

### Table 3
**Management of common complications**

<table>
<thead>
<tr>
<th>Complication</th>
<th>Management</th>
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<tbody>
<tr>
<td>Seizures</td>
<td>Prophylactic anti-epileptics for 3–7 d</td>
</tr>
<tr>
<td>Rebleeding</td>
<td>Blood pressure control, early aneurysm treatment</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>Extraventricular drainage</td>
</tr>
<tr>
<td>Cardiac dysfunction</td>
<td>Echocardiogram, euvoolemia</td>
</tr>
<tr>
<td>Fever</td>
<td>Antipyretics, surface cooling, intravascular cooling</td>
</tr>
<tr>
<td>Hyponatremia</td>
<td>Hypertonic fluids</td>
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</table>
and greater than 200 cm/sec for severe vasospasm. Additional indicators include change in serial measurements of more than 50 cm/sec, or a Lindegaard index (ratio of middle cerebral artery to internal carotid artery velocity) of greater than 6. This technique is limited by its ability to evaluate only large proximal arteries, limited utility in the posterior circulation, and poor specificity. Operator error and difficult temporal bone windows can make it unreliable. In combination with clinical examination, the majority of centers use serial TCD to judge which patients need further imaging with CT angiogram or conventional angiography.

**CTA, CT perfusion, and angiography**
Routine angiography during the critical phase of highest risk for DCI is another option for screening for vasospasm. More recently, CTA and CT perfusion have emerged as possible modalities to screen for and diagnose DCI and vasospasm. The combination of CTA findings for arterial narrowing and CBF with elevated mean transit time are the most accurate. These techniques are still evolving but recent studies publish sensitivities of 74% to 84% and specificity of 79% to 93%.

**DCI Management: Hemodynamic Augmentation**

**Induced hypertension**
Treating DCI by elevating blood pressure has now become standard treatment for symptomatic patients. Its use in asymptomatic patients with evidence of vasospasm is controversial and is not recommended. There are case reports and case series that have shown that induced hypertension improves CBF and leads to neurologic improvement. Norepinephrine and phenylephrine are the most common agents used. Blood pressure targets can be based on a percent increase above baseline blood pressure (typically beginning with a 10%–15% rise) or a target can be arbitrarily chosen. If there is no clinical response, vasopressors should be increased further. Once the patient improves, or further increases in blood pressure are considered imprudent, the target pressure is maintained for 1 to 3 days. CTA or cerebral angiography may be repeated to assess whether the vasospasm has abated and a trial of weaning therapy may be initiated. The blood pressure target is gradually lowered while closely monitoring with clinical examinations. Any deterioration should lead to a prompt increase in pressure. After another day or two, weaning should again be attempted. The use of induced hypertension is currently being tested in a multicenter, single-blinded, randomized, controlled trial.

**Augmenting cardiac output**
There have been isolated reports of the use of inotropic agents to treat DCI. It is generally reserved for patients who fail to respond to induced hypertension or have poor cardiac function. Dobutamine and milrinone are the most common inotropic agents employed for this indication.

**Hemoglobin**
Hemodilution for patients with DCI has been abandoned with the appreciation that the fall in hematocrit and thus arterial oxygen content lowers cerebral oxygen delivery. Additionally, anemia has been identified as a risk factor for DCI and poor outcome after SAH. Although retrospective studies point to risk associated with transfusion, a prospective study found that transfusion in anemic SAH patients was associated with improved brain oxygen delivery. The current guidelines recommend transfusions to maintain hemoglobin concentration of greater than 8 to 10 g/dL and suggest that higher hemoglobin concentrations may be appropriate for patients with DCI.
**DCI Management: Intra-arterial Treatment**

Interventional therapies for DCI include transluminal angioplasty and infusion of intra-arterial vasodilators. Although some patients respond well to hemodynamic interventions, others do not respond to medical measures or cannot tolerate them owing to comorbidities. How long to wait until declaring hemodynamic interventions a failure is a matter of much discussion and few data exist, with recommendations ranging from 2 to 12 hours. Intra-arterial nimodipine, verapamil, nicardipine, and milrinone have all been shown to dilate blood vessels and augment CBF.56,58,59 The duration of this response, however, seems to be a few hours. The effects of balloon angioplasty, on the other hand, are sustained. The use of angioplasty has been limited by its inability to reach distal vessels and risk of rupture associated with early balloon designs.60 A prospective, randomized trial of prophylactic angioplasty in high-risk patients found it reduced DCI, but this was offset by complications owing to vessel rupture. Still, with improved balloon design, angioplasty is now routinely used in symptomatic patients.

**PROGNOSIS**

The overall mortality from SAH is 30% to 40%; about one half of patients die before they reach the hospital. Of the remainder, 25% die in the first 2 weeks.61,62 Of the survivors, one half have a good recovery, yet studies show that survivors with good outcome still typically experience cognitive deficits in memory, executive function, and attention that impact their day-to-day living.63 Among other factors, poor prognosis is associated with advancing age, worsening neurologic grade, ruptured posterior circulation aneurysm, greater aneurysm size, more SAH, and most recently the Apo lipoprotein E ε4 allele.64,65 Prognostication may be aided by electroencephalographic, although no specific pattern has been validated.66 Repeat CT or MRI can be helpful to identify silent infarctions.

**SUMMARY**

Because one half of patients with aneurysmal SAH survive to reach the hospital, practitioners of neurocritical care continue to search for modalities to prevent, detect, and ameliorate the secondary complications of vasospasm and DCI that worsens diffuse brain injury and cognitive deficits that impact the long-term functioning of these patients.

**REFERENCES**


