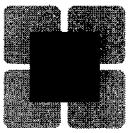

Management of subarachnoid hemorrhage in the critical care unit

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■ Subarachnoid hemorrhage from a ruptured intracranial saccular aneurysm is associated with a high rate of morbidity and mortality. Most complications occur two to three weeks after the initial hemorrhage. The key to minimizing morbidity and mortality is early and aggressive intensive-care management. The Cleveland Clinic Neurosurgical Intensive Care Unit approach is reviewed.

□INDEX TERMS: CRITICAL CARE; SUBARACHNOID HEMORRHAGE □CLEVE CLIN J MED 1989; 56:775–785

UNLIKE most other forms of stroke, which have declined in frequency over the past 40 years, the incidence of subarachnoid hemorrhage has not changed significantly.[1–3] Since a saccular aneurysm often presents initially as a subarachnoid hemorrhage, most work in the field has been directed toward improvement of posthemorrhagic medical and surgical management (*Figure 1*).

The introduction of microsurgical techniques has substantially reduced the operative morbidity and mortality of aneurysmal surgery.[4,5] However, nonoperative complications are common and may contribute to poor outcome in many patients.[1,4,6] Early aggressive management of subarachnoid hemorrhage can substantially reduce morbidity and mortality for survivors of the initial hemorrhage.[4]

Stabilizing the patient's condition and preventing rehemorrhage are the initial foci of management (*Table 1*). After several days, the risk of cerebral vasospasm becomes a major concern and efforts to prevent or treat this disorder are undertaken. Throughout the posthemorrhagic period, hydrocephalus and seizures may also occur and must promptly be recognized and treated.

The treatment guidelines for subarachnoid hemorrhage and its complications shown in *Tables 2* and *3* are used at the Cleveland Clinic's Neurosurgical Intensive Care Unit. The rationale for these guidelines is discussed below.

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CARDIAC COMPLICATIONS

Subarachnoid hemorrhage is often accompanied by a massive rise in circulating levels of catecholamines.[7–10] Cardiac disturbances, including subendocardial ischemia,[10–12] infarction,[13] and arrhythmias,[10–12,14,15] have been attributed to the sudden rise in sympathetic tone and subsequent autonomic imbalances.[10,16] A rise in circulating myocardial enzymes or electrocardiographic changes should be regarded as evidence of true cardiac damage[8,11,12,17] and are associated with a poorer clinical outcome.[17] Appropriate measures to prevent cardiac complications should be taken early after subarachnoid hemorrhage. These include afterload reduction, antiarrhythmia therapy, careful maintenance of intravascular volume, and use of beta-adrenergic blockers.[7,8]

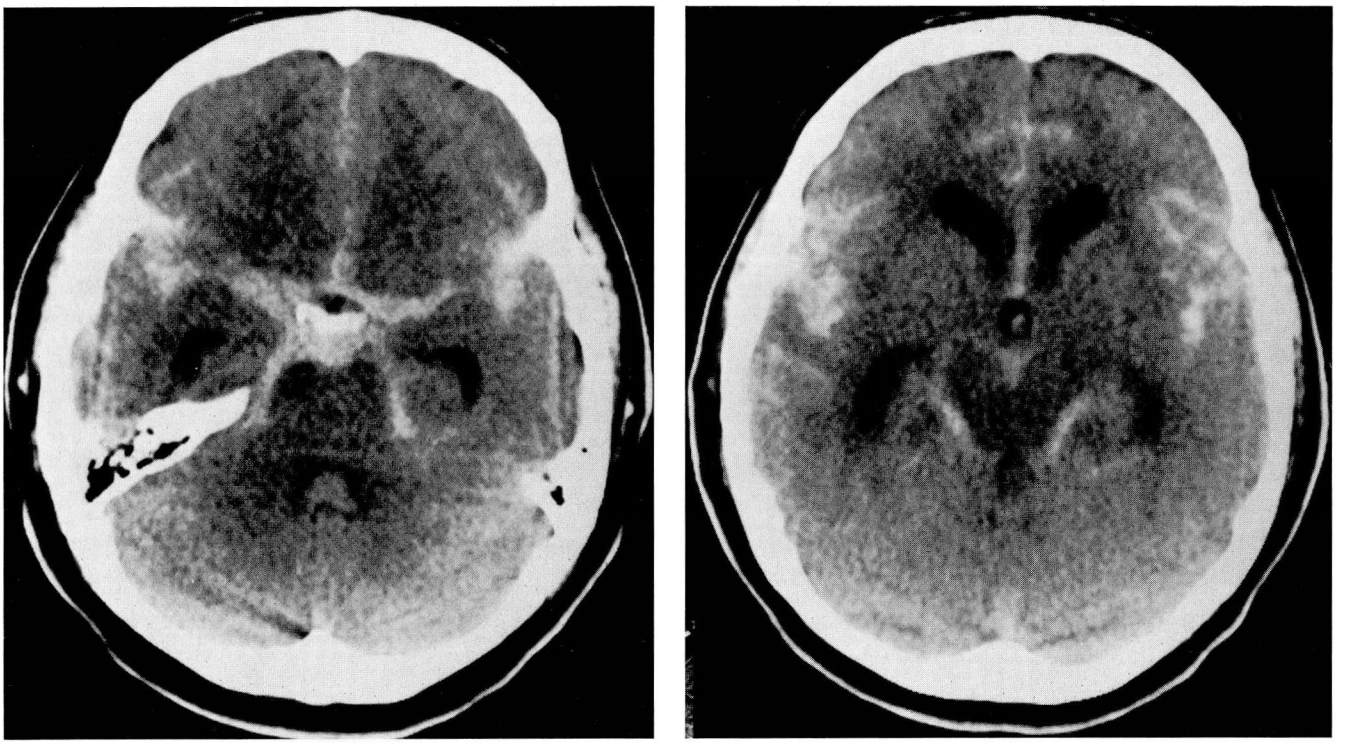

FIGURE 1. Computed tomographic images of the brain show a large amount of blood in the subarachnoid space and ventricular system following subarachnoid hemorrhage.

TABLE 1
MANAGEMENT OF SUBARACHNOID HEMORRHAGE

Patient stabilization
Neurological
Cardiac
Pulmonary
Prevention of rehemorrhage
Early surgery
Minimize transmural pressure
Antifibrinolytic agents
Prevention/therapy of vasospasm
Hydration, volume expansion, phlebotomy
Blood pressure control
Cerebral vasodilators
Calcium channel blockers
Treatment of hydrocephalus
Treatment of seizures

Arrhythmias occur in up to 90%–100% of patients in some series[11,13,15,18] and are most commonly premature ventricular complexes, bradyrhythmias, or supraventricular tachycardia. Potentially malignant arrhythmias, such as arteriovenous dissociation, idioventricular rhythms, and ventricular tachycardia, occur in 20%–40% of patients.[11,13,15] These are most common in the first 48 hours after subarachnoid hemorrhage,[11,13] are often associated with QT_c prolongation[15] and hypokalemia,[11,13,15] and may lead to sudden death.[11,16] Consequently, continuous electrocardiographic monitoring after subarachnoid hemorrhage is essential.

We do not favor the treatment of bradycardia or withholding beta-adrenergic blocking agents unless the heart rate is <45 beats per minute, heart block is present, or the patient is hypotensive. In such situations, atropine (0.5–1.0 mg, intravenous push) may be used to restore normal sinus rhythm.[12]

PULMONARY COMPLICATIONS

Common pulmonary complications of subarachnoid hemorrhage include aspiration pneumonia and pulmonary edema.[19] An arterial blood gas measurement should be obtained upon admission, and detection of hypoxia should suggest the presence of either of these disorders. Evaluation includes careful pulmonary examination, chest radiograph, and at times, assessment of cardiac filling pressures. It is important to consider that obtaining radiographic evidence of either disorder may take 12 hours or more.

Ideally, aspiration pneumonia should be confirmed by sputum culture; however, if aspiration is certain, the risk

TABLE 2
TREATMENT GUIDELINES: SUBARACHNOID HEMORRHAGE

1. Keep the patient recumbent in a darkened, quiet room. Limit the number of visitors.
2. Begin administering stool softeners (casanthranol and docusate sodium [Peri-Colace]), twice daily.
3. Administer propranolol (10 mg, orally, four times daily or 1 mg, intravenously, four times daily). The dosage can be increased if necessary to reduce systolic arterial blood pressure to <120 mmHg. It should not be discontinued unless the patient is profoundly bradycardic (<45 beats per minute), asthmatic, or hypotensive (systolic, <90 mmHg).
4. Administer phenobarbital (30–60 mg, intravenously or orally, three times daily) as a sedative.
5. Administer aminocaproic acid (1.5 g/h, continuous intravenous infusion) until surgery. Discontine if thromboembolic complications develop or if there is evidence of vasospasm.
6. To maintain systolic blood pressure <120 mmHg, administer sodium nitroprusside (50–200 mg/250 mL normal saline). Consider raising the blood pressure limit to 150 mmHg in geriatric patients.
7. Use phenytoin load (18 mg/kg) for seizure control only.
8. Sedate the patient as required to prevent large blood pressure swings. Provide adequate analgesia and start dexamethasone (4 mg every six hours).
9. Administer intravenous lidocaine or methohexital (Brevital) for any painful or uncomfortable procedures, such as suctioning.
10. If the patient's level of consciousness changes, obtain a computed-tomographic scan to check for hydrocephalus. Measure arterial blood gas to check for hypoxia and serum drug level to check for toxicity.
11. After the patient has been adequately sedated, insert a Swan-Ganz catheter. Attempt a pulmonary artery wedge pressure (PAWP) between 14–18 mmHg, using 5% or 20% albumin as appropriate. Determine the patient's optimum wedge pressure. Maintain an intravenous crystalloid infusion (75 mL/h) of normal saline. Adjust the sodium dosage as necessary.
12. Administer nimodipine (60 mg, orally or nasogastrically, every four hours) for calcium blockade, in an attempt to prevent vasospasm.
13. Maintain hematocrit level at 35% by (in the following order) volume expansion, transfusion, or phlebotomy.
14. Maintain arterial blood oxygen saturation at 95% or greater.
15. Maintain blood glucose between 100–150 mg/dL. Check at least every four hours or more frequently if required.
16. In the event of hydrocephalus, insert a ventricular catheter (or lumbar drain for clearly communicating hydrocephalus). Drain if intracranial pressure is ≥15 mmHg. If frequent drainage is required, drain continuously at this level. Do not overdrain.
17. Begin feeding two to three days after hemorrhage. Use parenteral feeding, started gradually, if the patient is obtunded.

TABLE 3
TREATMENT GUIDELINES: VASOSPASM AFTER SUBARACHNOID HEMORRHAGE

1. The patient should have a pulmonary artery catheter. Attempt to achieve a PAWP of 14–18 mmHg using albumin, blood, or fresh frozen plasma as required. Determine the optimal PAWP by maximal cardiac output and revise with each change in positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP).
2. Optimize the systemic vascular resistance index (SVRI) using sodium nitroprusside to reduce an elevated SVRI. To normalize a low SVRI, use dopamine (200 mg/250 mL), l-norepinephrine (Levophed) with a renal dose of dopamine (1 μg/kg/min), or phenylephrine (Neo-Synephrine) (10 mg/250 mL).
3. Discontinue administration of antifibrinolytic agents.
4. Continue administration of sedating agents, especially phenobarbital (at approximately 0.5–1.0 mg/kg, three times daily).
5. Strictly maintain blood glucose level at less than 150 mg/dL.
6. Allow the systolic blood pressure to rise to 140–160 mmHg, maximum. However, continue propranolol and sedation, which may prevent wide swings in pressure.

of rehemorrhage due to cough probably outweighs the benefit of obtaining a culture. A sputum sample obtained by nasotracheal suction or through an endotracheal tube must only be obtained after the patient's cough reflex has been arrested by administration of methohexital (50–120 mg, intravenous push)[20] or lidocaine (1.0–1.5 mg/kg, intravenous push).[21,22] If no culture material is obtained, empiric therapy with a third-generation cephalosporin, in addition to an aminoglycoside, is usually effective.

Pulmonary edema after subarachnoid hemorrhage may be due to elevated cardiac filling pressures or may be neurogenic when pulmonary capillary tight-junction disruption is postulated as an important contributor.[19] In either case, only severe symptomatic pulmonary edema should require therapy. Treatment of this disorder warrants the placement of a pulmonary artery catheter (which is routinely done in patients with subarachnoid hemorrhage in our unit) and measurement of PAWP. Raised PAWP should be reduced to between 12–15 torr by appropriate administration of either diuretics, such as furosemide or bumetanide, or afterload reducing agents, such as nitroprusside.[23] Extreme caution should be exercised with this treatment because reduction of PAWP to <12 torr may potentiate clinical vasospasm.[24] The key point with regard to PAWP is that the optimal pressure for each patient is different depending on the medical and neurological status. Overall clinical status often dictates the degree of intervention.

The development of pulmonary edema may also ne-

cessitate the use of CPAP or PEEP. If the patient is breathing satisfactorily, then CPAP and increasing the fraction of inspired oxygen (F_iO_2) may suffice. However, agitation may necessitate sedation sufficient to cause respiratory suppression that requires treatment with mechanical ventilation and PEEP. PEEP and CPAP must be used cautiously for patients with subarachnoid hemorrhage because the associated limitation of venous return can adversely affect intracranial pressure.[25,26] Hyperventilation is of no clear benefit after subarachnoid hemorrhage and should be reserved for cases when raised intracranial pressure is a problem.

HYPONATREMIA

Hyponatremia is detected in up to 33% of patients following subarachnoid hemorrhage. This imbalance can be associated with neurologic deterioration; therefore, early detection and treatment are essential.

The syndrome of inappropriate antidiuretic hormone secretion has traditionally been thought to cause hyponatremia after subarachnoid hemorrhage,[27,28] but recent evidence seems to implicate salt wasting due to atrial natriuretic factor secretion as well.[27,29,30] This, in turn, can lead to hypovolemia, which is known to be detrimental in vasospasm.

Treatment includes maintenance of normovolemia and the limitation of free water in the intravenous fluids.

RE-BLEEDING

Aneurysmal re-bleeding is a relatively frequent and often devastating complication of subarachnoid hemorrhage. A large multi-institutional study conducted in the 1960s found that the risk of re-bleeding peaks between the fourth and 10th day, then gradually decreases, plateauing in the fourth week.[31,32] It was also concluded from this study that for unclipped aneurysms, 54% of re-bleeds occur by the end of the second week, 70% occur by the end of the fourth week, and 11% occur more than one year after the initial hemorrhage.[33] Recently, several investigators have reported that the peak incidence of re-bleeding may actually be within the first 48 hours of the initial event, with the incidence being between 4% and 20%.[34–36]

The mortality rate associated with re-bleeding of an aneurysm is approximately 45%[31] and is substantially higher than the 10%–15% rate associated with the initial subarachnoid hemorrhage.[33] Reducing the incidence of aneurysmal re-bleeding can have a major impact on outcome and is one of the greatest challenges in neurosurgical critical care following subarachnoid hemorrhage.

Surgical obliteration of an intracranial aneurysm is the most effective means of preventing a re-bleed. Unfortunately, few patients can undergo an operation immediately following a subarachnoid hemorrhage, and surgery may be delayed for a week or more.

Minimizing the risk of aneurysmal re-bleed during the interim period between subarachnoid hemorrhage and the definitive procedure is the cornerstone of intensive-care management after subarachnoid hemorrhage. In general, medical therapy for the patient who has not undergone operation is directed at reducing the pressure gradient across the aneurysm wall, the so-called "transmural pressure" (i.e., the difference between the intracranial arterial pressure and intracranial pressure), and impairing lysis of the clot that seals the defect in the aneurysm wall.

Reduction of transmural pressure by rising intracranial pressure may be a physiologic mechanism by the body to stop the bleeding at the time of the initial subarachnoid hemorrhage.[37] Medical management of transmural pressure following the initial event is affected by mild intracranial-pressure augmentation[38] and by lowering the intracranial arterial pressure.[39,40]

Intracranial pressure augmentation is limited to keeping the patient recumbent and avoiding measures that might cause a sudden drop in intracranial pressure, such as overzealous drainage of cerebrospinal fluid via a lumbar puncture. Ventricular catheter placement is routinely performed in our neurosurgical intensive care unit for poorer-grade patients because it provides a way of monitoring and reducing intracranial pressure and subsequently increasing cerebral perfusion. Maintaining intracranial pressure between 10–15 torr is optimal.

Even transient episodes of hypertension may precipitate re-bleeding, consequently, strict minute-to-minute blood pressure control is essential.[23,39,40] Precautions include a combination of bed rest, analgesia, dim lighting, strict visitor limitations, and stool softeners; in short, avoidance of any stimulus that may cause agitation or excitement resulting in a rise in blood pressure.[4–6,41,42] This approach has been shown to reduce the risk of re-bleeding as compared to allowing the patient to be exposed to an uncensored hospital environment.[41,42] In keeping with this approach, patients receive adequate analgesia (codeine or morphine)[4,41] and mild sedation (phenobarbital, 30–60 mg, three times daily)[4,41] to produce drowsiness. Dexamethasone (4 mg, four times daily) may alleviate severe headaches and brain edema.[4,6,41] Extra analgesia and sedation is given prior to

performing procedures such as placement of central venous pressure or arterial lines. Extremely uncomfortable procedures, such as intubation or suctioning, should be preceded by sufficient sedation and reflex suppressants to eliminate any autonomic response to the procedures[21,22] (lidocaine, 1.0–1.5 mg/kg, intravenous push, or methohexital, 50–120 mg, intravenous push).[20] An infusion of a potent antihypertensive agent, such as nitroprusside (50 mg/250 mL of normal saline) should be mixed and ready to administer intravenously in the event that the premedication fails to prevent a substantial rise in blood pressure.[43]

The optimal blood pressure range after subarachnoid hemorrhage but before surgical obliteration of the aneurysm remains undefined. Generally, blood pressure is maintained strictly within approximately 5% of the premorbid range.[6,23,39,41] In practice, we maintain the young patient's systolic blood pressure at approximately 110 torr, whereas geriatric patients can reach pressures of up to 150 torr.[41] All patients receive propranolol (10–20 mg, administered orally, three times daily) as this β-adrenergic antagonist is thought to have a blood pressure "smoothing" effect that may decrease the incidence of re-bleeding.[7] It is also believed that propranolol reverses electrocardiographic changes and prevents myocardial necrosis due to the catecholamine surge following subarachnoid hemorrhage.[7,9] Propranolol may also have some cerebral protective actions.[9,17] Beta blockade should be used cautiously in the case of concomitant congestive heart failure, as it can cause sodium and water retention. It may also cause an increase in airway resistance in asthmatics.[23]

As mentioned, the risk of re-bleed is maximal within the first 48 hours after subarachnoid hemorrhage; therefore, strict blood pressure control must be assured during this period.[34,36] Usually, signs of agitation in association with hypertension are best treated by increased sedation. A rare case may require anesthesia, paralysis, and mechanical ventilation. Sedative agents that may be used after subarachnoid hemorrhage include low-dose phenobarbital[4,41] or benzodiazepines, such as lorazepam (1–2 mg, intravenous push, as needed) for agitation or haloperidol (1–2 mg, intravenously, as needed) for periods of psychosis.

Sedation may be reduced in better-grade patients to allow discussion of surgery and obtain valid consent; however, we favor resumption of sedation until surgery. The use of mild sedation and premedication is encouraged even in poorer-grade patients because autonomic responses to stimuli frequently persist despite profound reductions in consciousness, and these patients are at a higher risk for re-bleeding.[32,36]

Impaired circulation of cerebrospinal fluid is a common finding in the acute phase following subarachnoid hemorrhage and may result in elevated intracranial pressure and hydrocephalus. A rise in systemic arterial blood pressure secondary to elevated intracranial pressure may also increase the chance of rehemorrhage. The increase in systolic blood pressure is not necessarily associated with bradycardia and a widened pulse pressure (i.e., Cushing response); therefore, one must be vigilant for subtle signs of raised intracranial pressure. If the patient has a worsening level of consciousness and rising blood pressure or requires an increasing dose of antihypertensive agents, then intracranial hypertension must be excluded by measuring intracranial pressure.

Another mechanism known to reduce the risk of re-bleeding is the inhibition of dissolution of the blood clot around the tear in the aneurysm. Cerebrospinal fluid has a potent fibrinolytic activity after subarachnoid hemorrhage. Blood in the cerebrospinal fluid initiates the conversion of plasminogen to plasmin, inducing lysis of blood clots.[40,44–46] Therefore, the clot that seals the aneurysm slowly dissolves over several days. Aminocaproic acid (Amicar), a commonly used antifibrinolytic, has its main action through competitive inhibition of plasminogen activator with a minor action of competitive inhibition of plasmin.[40,47,48] Aminocaproic acid (5 g, intravenous bolus, followed by 1.5 g/h, intravenously) has been shown to decrease the risk of re-bleeding by about one half (from 20% to 10%)[40,48,49] but has been associated with increased risks of focal ischemic deficits, hydrocephalus, thrombophlebitis, and pulmonary embolus.[5,35,49–51] Some studies have shown no change in overall morbidity and mortality with the use of antifibrinolytics because of the associated increase in complications[47,49,50]; however, adverse effects may be minimized by using pneumatic antiembolus stockings and ensuring adequate hydration. We recommend antifibrinolytic therapy for virtually all patients with subarachnoid hemorrhage until the aneurysm is clipped or until 10 days after the last hemorrhage. Clinical vasospasm should prompt discontinuation of the agent. Strict adherence to these measures can significantly decrease re-bleeding prior to surgical aneurysm obliteration.

VASOSPASM

Vasospasm has become the leading cause of morbidity and mortality following subarachnoid hemorrhage. In some series,[35,52] 14%–36% of patients in neurosurgical

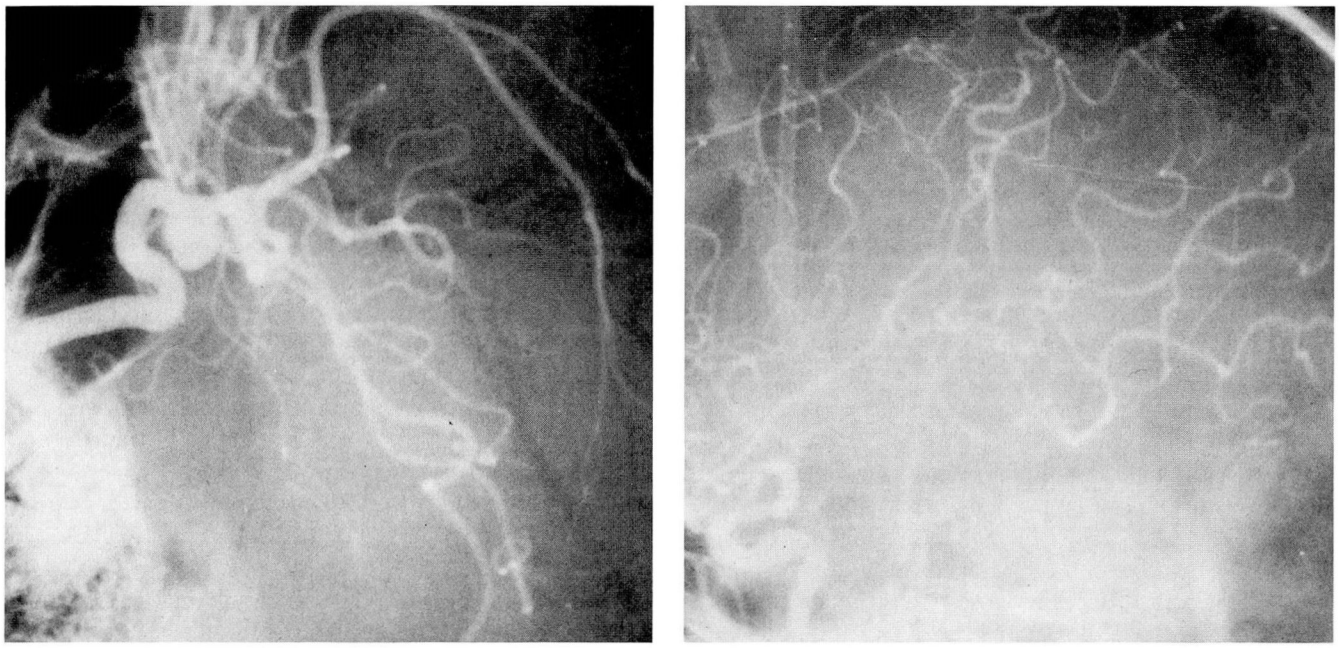

FIGURE 2A. Cerebral angiogram of the anterior cerebral circulation shows an aneurysm and normal distal vessel size. FIGURE 2B. Cerebral angiogram shows vasospasm of the supraclinoid internal carotid artery, the proximal middle and anterior carotid arteries, and the distal circulation diffusely.

centers suffered permanent neurological deficit or death due to vasospasm,[6,52] as compared with 7%–26% due to re-bleeding.[6,34,36,52] Clinical vasospasm affects, at least transiently, about 30% of patients with subarachnoid hemorrhage, although angiographic evidence of vasospasm is present in about 70% of patients (*Figure 2*). Clinical vasospasm is defined as the onset of confusion, decreasing mental status or focal deficit in the absence of hematoma, re-bleeding, hydrocephalus, metabolic imbalance, or other cause of deterioration. It usually begins between the fourth and ninth day after subarachnoid hemorrhage, with a peak incidence on the seventh day, and resolves over a period of weeks.[52,53]

Various theories about the pathogenesis of vasospasm after subarachnoid hemorrhage have been advanced, but none has been proven.[52,54–59] The pathophysiology of vasospasm is thought to be related to the increase in cerebral vascular resistance associated with arterial narrowing, resulting in local loss of autoregulation and subsequent decrease in cerebral blood flow.[52,60–62] Kassell et al[52] noted that ischemic deficits due to vasospasm can be reversed by improvement of blood flow, by changing the rheological characteristics of the blood, by increasing oxygen delivery to the tissues, and by hemodynamic augmentation measures. The various modes of therapy used in clinical practice and in research are derived from one or more of these categories.[63]

Total blood volume and red blood cell mass are decreased significantly after subarachnoid hemorrhage.[64] Hypovolemia potentiates clinical vasospasm,[24,62,65] and restitution of normovolemia can help prevent vasospasm.[24,62,66] This is accomplished by providing a crystalloid infusion of 0.9% sodium chloride at 1–1.5 mL/kg/h, supplemented with colloid infusions (5% albumin).[24,65–67] Red blood cell transfusions are used as needed to maintain the hematocrit between 33% and 37%, since optimal viscosity is an important determinant of cerebral blood flow.[62,68,69] Either central venous pressure or Swan-Ganz monitoring are recommended for optimal control of the hemodynamic state, although Swan-Ganz monitoring is preferred, especially in older patients where left ventricle pressure monitoring is crucial.[36,70,71] Central venous pressure between 8–12 mmHg or PAWP between 14–18 mmHg is maintained to achieve maximum cardiac output. This results in an increase in mean arterial pressure with a subsequent increase in cerebral blood flow.[24,72,73] Up to 70% of clinical deficits due to vasospasm can be reversed in this manner.[24,52] Brisk diuresis in response to fluid infusions will rarely require the use of low-dose desmopressin acetate

or aqueous vasopressin (Pitressin)[24] to achieve these intravascular pressures and maintain a urine output of <200 mL/h. There is little evidence to suggest that increasing blood volume such that PAWP >18 mmHg (in the absence of PEEP or CPAP) is beneficial. The consequences of overaggressive hydration include pulmonary edema, electrolyte disturbances, and cerebral edema, among others.[24]

Manipulation of fluid must be considered in conjunction with the patient's blood pressure.[23] In patients with clinical findings of vasospasm (i.e., focal ischemia) who have not undergone operation, intravascular volume is optimized and absolute pressure limits are allowed to rise by 20-torr increments during this period while following the patient's clinical status.[24] The risk of re-bleeding is always present, and elevation of blood pressure must be carried out cautiously. Transient elevations in blood pressure must be scrupulously avoided by continued administration of propranolol, sedation, and premedication before painful manipulations. In the patient who had early aneurysm clipping followed by vasospasm, the systolic blood pressure can be allowed to rise to >200 mmHg.[24,74,75] Despite adequate hydration, pressor agents such as dobutamine, phenylephrine, or dopamine may be required to increase mean arterial pressure, and subsequently cerebral perfusion pressure, to overcome spasm-induced ischemia.[24,57,74,75]

Many agents have claimed to reverse or prevent vasospasm,[19] but few have proven clinically useful. Among the most promising are the calcium-channel antagonists. Traditional drugs in this category, including verapamil, diltiazem, and nifedipine, are not useful in this setting due to relatively large systemic vasodilating effects and minimal effect on cerebral vessels. Nimodipine and nicardipine are calcium antagonists that selectively inhibit contractions of cerebral arteries.[59] These dihydropyridine calcium-channel blockers act by dilating the leptomeningeal collateral arteries,[76,77] improving red blood cell deformability, and exerting an antiplatelet aggregation effect.[71] Experimental studies of these drugs had been promising, and a clinical trial of nimodipine revealed some beneficial effects.

Recently, nimodipine was approved by the Food and Drug Administration for routine use. A dose of 60 mg orally or by nasogastric tube every four hours has been added to the standard protocol for subarachnoid hemorrhage. Further studies are in progress with nicardipine, an intravenously administered calcium-channel blocker with better dosage regulation. This may be an option for the future treatment of vasospasm after subarachnoid hemorrhage.[35,53,59,64,77–82]

Treatment of vasospasm with other agents has been largely unsuccessful. Nitroglycerin, nitroprusside, phenoxybenzamine, and hydralazine are potent cerebrovasodilators, but intracerebral steal of blood from ischemic areas and systemic hypotension limit their use.[83–85] Aminophylline and isoproterenol were thought at one time to cause cerebral vasodilation that may redirect blood to ischemic areas, but this has not been clinically applicable.[86–88] Reserpine and kanamycin have been tried in an effort to inhibit the concentration of catecholamines and serotonin in platelets, but this treatment was only effective in early nonclinical vasospasm.[89,90] Naloxone has been found to increase cerebral blood flow, probably by inhibiting endogenous opiates, but the effective dose in humans is prohibitively high.[91,92] Studies have also evaluated thromboxane A_2 inhibitor,[93] vitamin E,[94] nonsteroidal anti-inflammatory drugs,[95] steroids,[96] and many other agents without demonstrable benefits.

Anecdotal reports abound of cerebral angiography causing deterioration in the condition of a patient with subarachnoid hemorrhage. Most often, this deterioration is attributed to vasospasm, although re-bleeding occurs frequently in undersedated patients. Many contrast agents used in angiography can cause substantial osmotic diuresis. The ensuing hypovolemia can potentiate clinical vasospasm, and symptoms are often reversed by restitution of normovolemia. For this reason, early angiography with prehydration and careful attention to replace any fluid lost is recommended.

Zubkov et al[97] recommend balloon angioplasty for the treatment of vasospasm after subarachnoid hemorrhage, although this technique is not widely used at this time.

Location and amount of blood in the subarachnoid space have been correlated with onset and severity of vasospasm.[98–100] Early surgery with evacuation of subarachnoid clot has been advocated to prevent the development of vasospasm,[100–105] although views to the contrary have been expressed.[106]

TIMING OF ANEURYSM SURGERY

Even though prevention and treatment of vasospasm along with careful management of transmural pressure can decrease the risk of re-bleeding, the definitive treatment for a ruptured aneurysm is its surgical obliteration. The advantages of early surgery (within three days of subarachnoid hemorrhage) include virtual elimination of the risk of re-bleeding,[68,107] ability to remove subarachnoid hemorrhage clot and therefore decrease the incidence and severity of vasospasm,[33,104,105,107–110] and in-

TABLE 4
COMMONLY USED CLINICAL GRADING SCALES FOR SUBARACHNOID HEMORRHAGE

Hunt-Hess scale	
Grade	
I	Asymptomatic or mild headache
II	Moderate to severe headache, nuchal rigidity, can have oculomotor palsy
III	Confusion, drowsiness, or mild focal signs
IV	Stupor or hemiparesis
V	Coma, moribund, and/or extensor posturing
Cooperative Aneurysm Study Scale	
Grade	
I	Free of symptoms
II	Mildly ill, alert and responsive, headache present
III	Moderately ill (lethargic, headache, no focal signs, or alert, focal signs present)
IV	Severely ill (stuporous, no focal signs, or drowsy, focal signs present)
World Federation of Neurological Surgeons	
Grade	
I	No headache or focal signs (Glasgow Coma Scale Score: 15)
II	Headache, nuchal rigidity, no focal signs (Glasgow Coma Scale Score: 15)
III	Headache, nuchal rigidity, no focal signs (Glasgow Coma Scale Score: 13–14)
IV	Headache, nuchal rigidity, or focal signs (Glasgow Coma Scale Score: 13–14 or 9–12)
V	Headache, nuchal rigidity, or focal signs (Glasgow Coma Scale Score: 8 or less)

crease in the latitude for blood pressure parameters when treating clinical vasospasm.

The introduction of the operating microscope and microinstrumentation together with advancements in neuroanesthesia have substantially enhanced the ability to deal with the aneurysm shortly after hemorrhage.

Delayed surgery (longer than nine days after subarachnoid hemorrhage) allows brain swelling and subarachnoid hemorrhage clot to resolve and gives time for the patient's condition to be stabilized medically. A major disadvantage associated with delayed surgery is the increased incidence of morbidity and mortality (i.e., vasospasm, re-bleeding, hydrocephalus) due to restrictions on medical management while the aneurysm remains unclipped.[4,107,111]

Timing of aneurysm surgery has been an intensely debated topic. Many factors enter into the decision, including the patient's neurological status (as categorized in a standard grading system [*Table 4*]), the location of the aneurysm, and the patient's general medical status. In the past 10 to 20 years, the trend has been toward early operation, although there are some exceptions.

Early surgery is now thought to be the optimal surgical option for grade I and II patients (Hunt-Hess scale)[4,5,33,70,105–109,112–116] and in patients with aneurysmal subarachnoid hemorrhage associated with intracerebral hematoma whose condition is clinically deteriorating.[117] There are also those who support early surgery in all grade III and IV patients as well.[118] Delayed surgery is often reserved for most grade III–V patients and those whose condition is medically unstable.[1,70,119,120]

TREATMENT OF OTHER COMPLICATIONS OF SUBARACHNOID HEMORRHAGE

Hydrocephalus, when acute and untreated, can be a fatal complication.[121] More often, hydrocephalus develops insidiously, resulting in impaired consciousness, lower limb dysfunction, and raised intracranial pressure.[6,41,122] Ventricular drainage, as mentioned previously, allows decompression and monitoring of intracranial pressure. Overdrainage of cerebrospinal fluid must be avoided. This goal can be achieved by continuous drainage open to the atmosphere at 15–20 cm above the midventricular level or by intermittent drainage whenever intracranial pressure exceeds 15 torr. The carbonic anhydrase inhibitor, acetazolamide, may substantially reduce production of cerebrospinal fluid, thereby decreasing intracranial pressure[123] when hydrocephalus is the primary cause of intracranial hypertension. This therapy can be useful for several weeks but may also result in elevated cerebral blood flow and blood volume that could affect intracranial pressure deleteriously.

Seizures are not uncommon following subarachnoid hemorrhage[5,122] and can precipitate hypertension and mechanical intracranial stresses that may promote rehemorrhage. The phenobarbital dosage used to induce mild sedation is insufficient to have significant anticonvulsant effects for two to three weeks. The use of prophylactic anticonvulsants is controversial. Phenytoin is associated with complications,[124] such as idiosyncratic desquamation and altered cardiac conduction. For these reasons, we reserve phenytoin administration (loading

dose, 18 mg/kg; maintenance, 4–5 mg/kg/24 h) for patients who have had one or more focal or generalized seizures.[41]

CONCLUSION

The comprehensive management of the patient with subarachnoid hemorrhage requires the critical-care practitioner to strike a balance between therapies with opposing goals. The cornerstone of treatment is careful fluid balance, blood pressure control, inhibition of clot lysis, and decompression of hydrocephalus. Careful attention to medical and surgical detail can result in improved outcome from a potentially devastating disorder.

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