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occur in ~30% of cases. One-half of AVMs become evident as ICHs. In most, the hemorrhage is mainly intraparenchymal with extension into the subarachnoid space in some cases. Unlike primary SAHs (Chap. 440), blood from a ruptured AVM is usually not deposited in the basal cisterns, and symptomatic cerebral vasospasm is rare. The risk of AVM rupture is strongly influenced by a history of prior rup ture. Although unruptured AVMs have a hemorrhage rate of ~2–4% per year, previously ruptured AVMs may have a rate as high as 17% a year, at least for the first year. Hemorrhages may be massive, leading to death, or may be as small as 1 cm in diameter, leading to minor focal symptoms or no deficit. The AVM may be large enough to steal blood away from adjacent normal brain tissue or to increase venous pressure significantly to produce venous ischemia locally and in remote areas of the brain. This is seen most often with large AVMs in the territory of the middle cerebral artery. Large AVMs of the anterior circulation may be associated with a sys tolic and diastolic bruit (sometimes self-audible) over the eye, forehead, or neck and a bounding carotid pulse. Headache at the onset of AVM rupture is generally not as explosive as with aneurysmal rupture. MRI is better than CT for diagnosis, although noncontrast CT scanning sometimes detects calcification of the AVM and contrast may demon strate the abnormal blood vessels. Once identified, conventional x-ray angiography is the gold standard for evaluating the precise anatomy of the AVM. Surgical treatment of AVMs presenting with hemorrhage, often done in conjunction with preoperative embolization to reduce opera tive bleeding, is usually indicated for accessible lesions. Stereotactic radiosurgery, an alternative to conventional surgery, can produce a slow sclerosis of the AVM over 2–3 years. Several angiographic features can be used to help predict future bleeding risk. Paradoxically, smaller lesions seem to have a higher hem orrhage rate. The presence of deep venous drainage, venous outflow stenosis, and intranidal aneurysms may increase rupture risk. Because of the relatively low annual rate of hemorrhage and the risk of com plications due to surgical or endovascular treatment, the indications for surgery in asymptomatic AVMs are uncertain. The ARUBA (A Randomized Trial of Unruptured Brain Arteriovenous Malformations) trial randomized patients to medical management versus interven tion (surgery, endovascular embolization, combination embolization and surgery, or gamma-knife). The trial was stopped prematurely for harm, with the medical arm achieving the combined endpoint of death or symptomatic stroke in 10% of patients compared to 31% in the intervention group at a mean follow-up time of 33 months. This highly significant finding argues against routine intervention for patients presenting without hemorrhage, although debate ensues regarding the generalizability of these results. ■ ■CAVERNOUS ANGIOMAS Cavernous angiomas (cavernous malformations) are

tufts of capil lary sinusoids that form within the deep hemispheric white matter and brainstem with no normal intervening neural structures. The pathogenesis is unclear. Most cavernous angiomas are congenital, but they may occur during life as well. Familial cavernous angiomas have been mapped to several different genes: KRIT1, CCM2, and PDCD10. Both KRIT1 and CCM2 have roles in blood vessel formation, whereas PDCD10 is an apoptotic gene. Cavernous angiomas are typically <1 cm in diameter and are often associated with a venous anomaly. Bleeding is usually of small volume, causing slight mass effect only. The bleeding risk for single cavernous malformations is 0.7–1.5% per year and may be higher for patients with prior clinical hemorrhage or multiple malforma tions. Seizures may occur if the malformation is located near the cerebral cortex. Surgical resection eliminates bleeding risk and may reduce sei zure risk but is usually reserved for those malformations that form near the brain surface in patients with prior clinical episodes of bleeding or with medically refractory seizures. Stereotactic radiosurgery has been considered as a secondary treatment, but risks may outweigh benefits. Retrospective data show that intracranial hemorrhage from cavernous malformations is likely not increased with administration of antiplatelet and anticoagulant medications prescribed for other medical conditions.

Developmental venous anomalies are the result of development of anomalous cerebral, cerebellar, or brainstem venous drainage. These structures, unlike AVMs, are functional venous channels. They are of little clinical significance and should be ignored if found incidentally on brain imaging studies. Surgical resection of these anomalies may result in venous infarction and hemorrhage. Venous anomalies may be associated with cavernous malformations, which do carry some bleeding risk.

Capillary telangiectasias are true capillary malformations that often form extensive vascular networks through an otherwise normal brain structure. The pons and deep cerebral white matter are typical locations, and these capillary malformations can be seen in patients with hereditary hemorrhagic telangiectasia (Osler-Rendu-Weber) syn drome. If bleeding does occur, it rarely produces mass effect or signifi cant symptoms. No treatment options exist. Dural arteriovenous fistulas are acquired connections usually from a dural artery to a dural sinus. Patients may complain of a pulse-synchro nous cephalic bruit (“pulsatile tinnitus”) and headache. Depending on the magnitude of the shunt, venous pressures may rise high enough to cause cortical ischemia or venous hypertension and hemorrhage, particularly SAH. Surgical and endovascular techniques are usually curative. These fistulas may form because of trauma, but most are idiopathic. There is an association between fistulas and dural sinus thrombosis. Fistulas have been observed to appear months to years fol lowing venous sinus thrombosis, suggesting that angiogenesis factors elaborated from the thrombotic process may cause these anomalous connections to form. Alternatively, dural arteriovenous fistulas can produce venous sinus occlusion over time, perhaps from the high pres sure and high flow through a venous structure. Subarachnoid Hemorrhage CHAPTER 440 ■ ■FURTHER READING Anderson CS et al: Rapid blood-pressure lowering in patients with acute intracerebral hemorrhage. N Engl J Med 368:2355, 2013. Christensen H et al: European stroke organization guideline on reversal of oral anticoagulants in acute intracerebral hemorrhage. Euro Stroke J 4:294, 2019. Greenberg SM et al: 2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage: A Guideline From the American Heart Association/American Stroke Association. Stroke 53:e282, 2022. Ma L et al: The third Intensive Care Bundle with Blood Pressure Reduction in Acute Cerebral Haemorrhage Trial (INTERACT3): An international, stepped wedge cluster randomised controlled trial. Lancet 402:27, 2023. Mohr JP et

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J. Claude Hemphill, III Subarachnoid hemorrhage (SAH) renders the brain critically ill from both primary and secondary brain insults. Excluding head trauma, the most common cause of SAH is rupture of a saccular aneurysm. Other causes include bleeding from a vascular malformation (arteriovenous malformation or dural arteriovenous fistula) and extension into the

subarachnoid space from a primary intracerebral hemorrhage. Some idiopathic SAHs are localized to the perimesencephalic cisterns and are benign; they probably have a venous or capillary source, and angi ography is unrevealing.

■ ■SACCULAR (“BERRY”) ANEURYSM Autopsy and angiography studies have found that ~2% of adults harbor intracranial aneurysms, for a prevalence of 4 million persons in the United States. The incidence of SAH from aneurysmal rupture is estimated at between 6 and 11 per 100,000 person-years, resulting in 25,000–30,000 cases annually in the United States, with a 1.3 relative risk in women. Although most affected patients are under age 55, there is also an increas ing incidence with age. The overall mortality rate for aneurysmal SAH is ~35%, with approximately one-third of patients dying immediately and prior to hospital admission. Of those who survive, more than half are left with clinically significant neurologic deficits because of the initial hemor rhage, delayed cerebral ischemia, or hydrocephalus. If the patient survives but the aneurysm is not obliterated, the rate of rebleeding is ~20% in the first 2 weeks, 30% in the first month, and ~3% per year afterward. Given these alarming figures, the major therapeutic emphasis is on preventing the predictable early complications of the SAH. PART 13 Neurologic Disorders Unruptured, asymptomatic aneurysms are much less dangerous than recently ruptured ones. A large international observational study found that the annual risk of rupture for unruptured aneurysms <7 mm in size was 0% over 5 years. However, subsequent studies from Japan and Finland found that the majority of ruptured aneurysms were <6 mm in size. Aneurysms of <3 mm in size rarely, if ever, bleed. As the size of an aneurysm increases, so does the risk for rupture, but growth is not linear and appears to occur in phases, making surveillance for growing aneurysms problematic. The location of an unruptured aneurysm is also important in assessment, as basilar bifurcation and origin poste rior communicating artery aneurysms appear to have a higher risk for rupture than other sites. Because of their longer length of exposure to risk of rupture, younger patients with aneurysms >10 mm in size may benefit from prophylactic treatment (see below). As with the treatment of asymptomatic carotid stenosis (Chap. 438), this risk-benefit ratio strongly depends on the rate of procedural complications. Giant aneurysms, those >25 mm in diameter, occur at the same sites (see below) as small aneurysms, and account for 5% of cases. The three most common locations are the terminal internal carotid artery, middle cerebral artery (MCA) bifurcation, and top of the basilar artery. Their risk of rupture is ~8–10% annually after identification and may remain high indefinitely. They often cause symptoms by compressing the adjacent brain or cranial nerves. Mycotic aneurysms are usually located distal to the first bifurca tion of major arteries of the circle of Willis. Most result from infected emboli due to bacterial endocarditis causing septic degeneration of arteries and subsequent dilation and rupture. Whether these lesions should be sought and repaired prior to rupture or left to heal spontane ously with antibiotic treatment remains

controversial. Pathophysiology Saccular aneurysms occur at the bifurcations of the large- to medium-sized intracranial arteries; rupture is into the subarachnoid space in the basal cisterns and sometimes into the paren chyma of the adjacent brain. Approximately 89% of aneurysms occur in the anterior circulation, mostly on the circle of Willis (Fig. 440-1). About 20% of patients have multiple aneurysms, many at mirror sites bilaterally. As an aneurysm develops, it typically forms a neck with a dome. The length of the neck and the size of the dome vary greatly and are important factors in planning neurosurgical obliteration or endovascular embolization. The arterial internal elastic lamina disappears at the base of the neck. The media thins, and connective tissue replaces smooth-muscle cells. At the site of rupture (most often the dome), the wall thins, and the tear that allows bleeding is often ≤0.5 mm long. Fusiform aneurysms, where the locations of inflow and outflow are different, typically occur in the basilar artery and are very challenging to treat. Clinical Manifestations Most unruptured intracranial aneurysms are completely asymptomatic. Symptoms are usually due to rupture

Anterior cerebral artery Anterior communicating artery Ophthalmic artery Middle cerebral artery Anterior choroidal artery

Posterior cerebral artery Internal carotid artery

Superior cerebellar artery

Pontine arteries Posterior communicating artery Anterior inferior cerebellar artery

Basilar artery Vertebral artery Posterior inferior cerebellar artery Anterior spinal artery FIGURE 440-1 View of the major blood vessels supplying the brain and common locations of saccular aneurysms: (1) Anterior communicating (12%), (2) internal carotid (30%), (3) posterior communicating (12%), (4) middle cerebral (34%), (5) basilar terminus, (6) superior cerebellar, (7) anterior inferior cerebellar, and (8) posterior inferior cerebellar aneurysm. Locations 1–4 are considered anterior circulation aneurysms totaling 89% overall, while locations 5–8 total 11%. and resultant SAH, although some unruptured aneurysms present with mass effect on cranial nerves or brain parenchyma. At the moment of aneurysmal rupture with a major SAH, the intracranial pressure (ICP) suddenly rises. This may account for the sudden transient loss of consciousness that occurs in nearly half of patients. Sudden loss of consciousness may be preceded by a brief moment of excruciating headache, but most patients first complain of headache upon regaining consciousness. In 10% of cases, aneurysmal bleeding is severe enough to cause loss of consciousness for several days. In ~45% of cases, severe headache associated with exertion is the presenting complaint. The patient often calls the headache “the worst headache of my life”; how ever, the most important characteristic is sudden onset. Occasionally, these ruptures may present as headache of only moderate intensity or as a change in the patient’s usual headache pattern. The headache is usually generalized, often with neck stiffness, and vomiting is common. Although sudden headache in the absence of focal neurologic symp toms is the hallmark of aneurysmal rupture, focal neurologic deficits may occur. Anterior communicating artery or MCA bifurcation aneu rysms may rupture into the adjacent brain or subdural space and form a hematoma large enough to produce mass effect. The deficits that result can include hemiparesis, aphasia, and mental slowness (abulia). Occasionally, prodromal symptoms suggest the location of a pro gressively enlarging unruptured aneurysm. A third cranial nerve palsy, particularly when associated

with pupillary dilation, loss of ipsilateral (but retained contralateral) light reflex, and focal pain above or behind the eye, may occur with an expanding aneurysm at the junction of the posterior communicating artery and the internal carotid artery. A sixth nerve palsy may indicate an aneurysm in the cavernous sinus, and visual field defects can occur with an expanding supraclinoid carotid or anterior cerebral artery (ACA) aneurysm. Occipital and posterior cer vical pain may signal a posterior inferior cerebellar artery or anterior inferior cerebellar artery aneurysm (Chap. 438). Pain in or behind the eye and in the low temple can occur with an expanding MCA aneu rysm. Thunderclap headache is a variant of migraine that simulates an SAH. Before concluding that a patient with sudden, severe headache has thunderclap migraine, a definitive workup for aneurysm or other intracranial pathology is required.

TABLE 440-1 Grading Scales for Subarachnoid Hemorrhage WORLD FEDERATION OF NEUROSURGICAL SOCIETIES (WFNS) SCALE GRADE HUNT-HESS SCALE

Asymptomatic, or minimal headache and slight nuchal rigidity. Normal mental status, no cranial nerve or motor findings GCSa score 15, no motor deficits

Moderate to severe headache, nuchal rigidity, normal mental status and motor function, may have cranial nerve deficit GCS score 13–14, no motor deficits

Somnolent, confused, may have cranial nerve or mild motor deficit GCS score 13–14, with motor deficits

Stupor, moderate to severe motor deficit, may have intermittent reflex posturing GCS score 7–12, with or without motor deficits

Coma, reflex posturing or flaccid GCS score 3–6, with or without motor deficits aGlasgow Coma Scale; see Table 454-1. Source: Reproduced with permission from WFNS Scale: Report of World Federation of Neurological Surgeons Committee on a Universal Subarachnoid hemorrhage Grading Scale. J Neurosurg 68:985, 1988. Aneurysms can undergo small ruptures and leaks of blood into the subarachnoid space, so-called sentinel bleeds. Sudden unexplained headache at any location should raise suspicion of SAH and be investi gated because a major hemorrhage may be imminent. The initial clinical manifestations of SAH can be graded using the Hunt-Hess or World Federation of Neurosurgical Societies classifica tion schemes (Table 440-1). For ruptured aneurysms, prognosis for a good outcome falls as the grade increases. For example, it is unusual for a Hunt-Hess grade 1 patient to die if the aneurysm is treated, but the mortality rate for grade 4 and 5 patients may be as high as 60%. Delayed Neurologic Deficits There are four major causes of delayed neurologic deficits: rerupture, hydrocephalus, delayed cerebral ischemia, and hyponatremia.

1. Rerupture. The incidence of rerupture of an untreated aneurysm in the first month following SAH is ~30%, with the peak in the first 7 days. Rerupture is associated with a 50% mortality rate and poor outcome. Early treatment eliminates this risk, and advances in endovascular and surgical techniques contribute to better outcomes.
2. Hydrocephalus. Acute hydrocephalus can cause stupor and coma and can be mitigated by placement of an external ventricular drain. More often, subacute hydrocephalus may develop over a few days or weeks and cause progressive drowsiness or slowed mentation

with incontinence. Hydrocephalus may clear spontaneously, require tem porary ventricular drainage, or in some cases require placement of a ventriculoperitoneal shunt. Chronic hydrocephalus may develop weeks to months after SAH and manifest as gait difficulty, inconti nence, or impaired mentation. Subtle signs may be a lack of initiative in conversation or a failure to recover independence.

3. Delayed cerebral ischemia. Vasospasm is the narrowing of the arter ies at the base of the brain following SAH and has been associated with delayed cerebral ischemia and infarction. Delayed cerebral ischemia occurs in ~30% of patients and is the major cause of delayed morbidity and death. Signs first appear 4–14 days after the hemorrhage, most often at 7 days. While therapies can be targeted for vasospasm, there are other complex mechanisms associated with delayed cerebral ischemia and progression to infarction indepen dent of vasospasm alone.

Narrowing of the arteries causing vasospasm and thickening of the vessel wall is believed to result from direct effects of clot ted blood and its breakdown products on the arteries within the subarachnoid space. In general, the more blood that surrounds the arteries, the greater is the chance of symptomatic vasospasm. Focal narrowing of major arteries produces symptoms referable to the

appropriate vascular territory (Chap. 437). All of these focal symp toms may present abruptly, fluctuate, or develop over a few days. The clinical syndrome often manifests as a decline in mental status and worsening headache.

Vasospasm of the large arteries can be detected reliably with conventional x-ray angiography, but this procedure is invasive and carries the risk of stroke and other complications. Transcutaneous Doppler ultrasound is based on the principle that the velocity of blood flow within an artery will rise as the lumen diameter is nar rowed. By directing the probe along the MCA and proximal ACA, carotid terminus, and vertebral and basilar arteries on a daily or every-other-day basis, vasospasm can be reliably detected and treat ments initiated to prevent cerebral ischemia (see below). Computed tomography (CT) angiography is another method that can detect vasospasm. The addition of CT perfusion imaging may help identify reversible ischemic deficits. In high-grade patients, invasive neuro monitoring techniques can also be considered. 4. Hyponatremia. Hyponatremia may be profound and can develop Subarachnoid Hemorrhage CHAPTER 440 quickly in the first 2 weeks following SAH. There is both natriure sis and volume depletion with SAH, so that patients become both hyponatremic and hypovolemic. Both atrial natriuretic peptide and brain natriuretic peptide have a role in producing this “cerebral saltwasting syndrome.” Typically, it clears over the course of 1–2 weeks and, in the setting of SAH, should not be treated with free-water restriction as this may increase the risk of stroke (see below). Laboratory Evaluation and Imaging (Fig. 440-2) The hall mark of aneurysmal rupture is blood in the CSF. More than 95% of cases have enough blood to be visualized on a high-quality noncontrast CT scan obtained within 72 h. If the scan fails to establish the diagnosis of SAH and no mass lesion or obstructive hydrocephalus is found, a lumbar puncture should be performed to establish the presence of sub arachnoid blood. Lysis of the red blood cells and subsequent conversion of hemoglobin to bilirubin stains the spinal fluid yellow within 6–12 h. This xanthochromic spinal fluid peaks in intensity at 48 h and lasts for 1–4 weeks, depending on the amount of subarachnoid blood present. The extent and location of subarachnoid blood on a noncontrast CT scan help locate the underlying aneurysm,

identify the cause of any neurologic deficit, and predict the occurrence of vasospasm. The likelihood of symptomatic vasospasm in the MCA and ACA can be predicted based on the size and location of clotted blood (Table 440-2). CT scans less reliably predict vasospasm in the vertebral, basilar, or posterior cerebral arteries. Lumbar puncture prior to an imaging procedure is indicated only if a CT scan is not available at the time of the suspected SAH. Once the diagnosis of hemorrhage from a ruptured saccular aneurysm is confirmed, four-vessel conventional x-ray angiography (both carotids and both vertebrals) is generally performed to localize and define the anatomic details of the aneurysm and to determine if other unruptured aneurysms exist (Fig. 440-2C). The ruptured aneurysm can be treated using endovascular techniques at the time of the initial angiogram to expedite treatment and minimize the number of invasive procedures. CT angiography is an alternative method for locating the aneurysm and may be sufficient for planning definitive therapy. Close monitoring (daily or twice daily) of electrolytes is important because hyponatremia can occur precipitously during the first 2 weeks following SAH (see above). The electrocardiogram (ECG) frequently shows ST-segment and T-wave changes similar to those associated with cardiac ischemia. A prolonged QRS complex, increased QT interval, and prominent “peaked” or deeply inverted symmetric T waves are usually second ary to the intracranial hemorrhage. Structural myocardial lesions produced by circulating catecholamines and excessive discharge of sympathetic neurons may occur after SAH, causing these ECG changes and a reversible cardiomyopathy sufficient to cause shock. Echocar diography often reveals a pattern of regional wall motion abnormalities that follow the distribution of sympathetic nerves rather than the major coronary arteries, with relative sparing of the ventricular wall apex. The sympathetic nerves themselves appear to be injured by direct toxicity

A B PART 13 Neurologic Disorders C D FIGURE 440-2 Subarachnoid hemorrhage. A. Computed tomography (CT) angiography revealing an aneurysm of the left superior cerebellar artery. B. Noncontrast CT scan at the level of the third ventricle revealing subarachnoid blood (bright) in the left sylvian fissure and within the left lateral ventricle. C. Conventional anteroposterior x-ray angiogram of the right vertebral and basilar artery showing the large aneurysm. D. Conventional angiogram following coil embolization of the aneurysm, whereby the aneurysm body is filled with platinum coils delivered through a microcatheter navigated from the femoral artery into the aneurysm neck. from the excessive catecholamine release. An asymptomatic troponin elevation is common. While arrythmias can occur after SAH, serious ventricular dysrhythmias occurring in-hospital are unusual. TREATMENT Subarachnoid Hemorrhage Early aneurysm repair prevents rerupture and allows the safe appli cation of techniques to improve blood flow (e.g., induced hyperten sion) should vasospasm and delayed cerebral ischemia develop. At many centers, definitive repair is carried out within 24 h of the bleed in all patients who are stable enough to tolerate the procedure. TABLE 440-2 Modified Fisher Grading System for Prediction of Vasospasm Risk RISK OF SYMPTOMATIC VASOSPASM GRADE CT SCAN FINDINGS

No subarachnoid or intraventicular blood 0%

Focal or diffuse thin subarachnoid blood without intraventricular blood 24%

Focal or diffuse thin subarachnoid blood with intraventricular blood 33%

Focal or diffuse thick subarachnoid blood without intraventricular blood 33%

Focal or diffuse thick subarachnoid blood with intraventricular blood 40% Note: “Thin” is <1 mm, whereas “thick” is ≥1 mm. Abbreviation: CT, computed tomography. Source: JA Frontera et al: Prediction of symptomatic vasospasm after subarachnoid hemorrhage: The modified Fisher scale. Neurosurgery 59:21, 2006.

An aneurysm can be “clipped” by a neurosurgeon or “coiled” by an endovascular surgeon. Surgical repair involves placing a metal clip across the aneurysm neck, thereby immediately eliminating the risk of rebleeding. This approach requires craniotomy and brain retrac tion, which is associated with neurologic morbidity. Endovascular techniques involve placing coils, or other embolic material, within the aneurysm via a catheter that is passed from the femoral or radial artery. The aneurysm is packed tightly to enhance thrombosis and over time is walled off from the circulation (Fig. 440-2D). There have been two prospective randomized trials of surgery versus endovas cular treatment for ruptured aneurysms: the first was the Interna tional Subarachnoid Aneurysm Trial (ISAT), which was terminated early when 24% of patients treated with endovascular therapy were dead or dependent at 1 year compared to 31% treated with surgery, a significant 23% relative reduction. After 5 years, the risk of death was still lower in the coiling group, although the proportion of sur vivors who were independent was the same in both groups. The risk of rebleeding was generally low, but was more common in the coil ing group. These results favoring coiling at 1 year were confirmed in a second trial, although the differences in functional outcome of survivors were no longer significant at 3 years. Because some aneurysms have a morphology that is not amenable to endovascular treatment, surgery remains an important treatment option, espe cially in cases where evacuation of a parenchymal hematoma could be beneficial. Newer endovascular devices and techniques using balloon-assisted coiling or placement of flow-diverting stents are increasing the types of aneurysms amenable to endovascular inter vention. Centers that combine both endovascular and neurosurgi cal expertise likely offer the best outcomes for patients, and transfer to centers that specialize in aneurysm treatment are associated with improved outcomes and lower mortality rates. The early medical management of SAH focuses on protecting the airway, managing blood pressure before and after repair of the aneurysm, preventing rebleeding prior to the intervention, and treating hydrocephalus. Subsequent management is focused on preventing late neurologic injury, including managing vasospasm and delayed cerebral ischemia, treating hyponatremia, limiting sec ondary brain insults from medical comorbidities, and preventing pulmonary emboli. Intracranial hypertension following aneurysmal rupture occurs secondary to subarachnoid blood, parenchymal hematoma, acute hydrocephalus, and/or loss of vascular autoregulation. Patients who are stuporous should undergo emergent ventriculostomy to measure and treat high ICP in order to prevent cerebral herniation and ischemia. Medical therapies designed to combat raised ICP (e.g., osmotic therapy and sedation) can also be used as needed. High ICP refractory to treatment is a poor prognostic sign. Drain age of CSF via a lumbar route has been shown to decrease the rate of delayed cerebral injury, vasospasm, and mortality but confounds measurement of true ICP. Prior to definitive treatment of the ruptured aneurysm, care is required to maintain adequate cerebral perfusion pressure while avoiding excessive elevation of arterial pressure. If the patient is alert, it is reasonable to lower the systolic blood pressure to below 160 mmHg using agents such as nicardipine, clevidipine, or labet alol to limit blood pressure variability. If the patient has a depressed level of consciousness, ICP should be measured and the cerebral perfusion pressure targeted to 60–70 mmHg. If headache or neck pain is severe, mild sedation and analgesia are prescribed. Extreme sedation is avoided if possible because it can obscure the ability to clinically detect changes in neurologic status. Goal-directed therapy to target euvolemia is

recommended, while avoiding hypervolemia, which has been associated with a greater risk for complications. Seizures are uncommon at the onset of aneurysmal rupture, but in patients who present with seizures, treatment with a 7-day course of an anticonvulsant such as levetiracetam is reasonable. The quiv ering, jerking, and extensor posturing that often accompany loss of consciousness with SAH are probably related to the sharp rise in ICP rather than seizures. Monitoring with electroencephalogram

(EEG) can help detect seizures in patients with poor mental sta tus or fluctuating exam findings. Anticonvulsants are sometimes administered as prophylactic therapy in high-risk patients, but if used, the treatment course should also not exceed >7 days. Phe nytoin should be avoided because of its association with increased morbidity and mortality in this setting. Glucocorticoids may reduce the headache and neckache caused by the irritative effect of the subarachnoid blood; however, there is no good evidence that steroids reduce cerebral edema, are neuro protective, or reduce vascular injury, and their routine use therefore is not recommended. Antifibrinolytic agents are not routinely prescribed but have been considered in patients in whom aneurysm repair cannot proceed immediately. They are associated with a reduced incidence of aneurysmal rerupture but may also increase the risk of delayed cerebral ischemia and deep-vein thrombosis (DVT). More recent studies showed no improvement in functional outcomes, and use of these agents is not currently recommended. Delayed cerebral ischemia remains the leading cause of mor bidity and mortality following aneurysmal SAH in patients who survive the initial hemorrhage. Treatment with the calcium channel antagonist nimodipine (60 mg PO every 4 h) has been shown to improve outcomes, perhaps by preventing ischemic injury rather than reducing the risk of vasospasm. Nimodipine can cause sig nificant hypotension in some patients, which may worsen cere bral ischemia in patients with vasospasm. Symptomatic cerebral vasospasm can also be treated by increasing the cerebral perfusion pressure by raising mean arterial pressure through plasma volume expansion and the judicious use of IV vasopressor agents, usually phenylephrine or norepinephrine. Increasing perfusion pressure has been associated with clinical improvement in many patients, but high arterial pressure may also promote rebleeding in unre paired aneurysms. Treatment with induced hypertension in symp tomatic patients requires close monitoring of arterial pressures. Prophylactic use of induced hypertension is not recommended, as it is associated with worse outcomes. Euvolemia should be targeted as significant hypervolemia may lead to cardiopulmonary complica tions. Hypovolemia should be strictly avoided. If delayed cerebral ischemia due to vasospasm persists despite optimal medical therapy, endovascular rescue therapies with intra arterial vasodilators and percutaneous transluminal angioplasty can be considered (Fig. 440-3). Vasodilatation by direct angioplasty appears to be permanent, allowing hypertensive therapy to be A B FIGURE 440-3 Vasospasm of the right middle cerebral artery. A. Catheter angiography demonstrates significant narrowing of the right middle cerebral artery (MCA). B. Because of symptomatic delayed cerebral ischemia, soft-balloon angioplasty was used to dilate the proximal portion of the main MCA stem.

tapered sooner. In contrast, the pharmacologic vasodilators (vera pamil and nicardipine) do not last more than about 24 h, and there fore, multiple treatments may be required until the subarachnoid blood is reabsorbed. Newer therapies including milrinone, neural ganglia blocks, antithrombotic agents, and intrathecal and cisternal agents are currently under investigation.

Severe cerebral edema in patients with infarction from vaso spasm may increase the ICP to levels that reduce cerebral perfusion pressure. Treatment may include cerebrospinal fluid (CSF) drain

age, mannitol, or hypertonic saline; for intractable cases, hemicra niectomy, deep sedation, paralysis, and moderate hypothermia may be considered. Delayed cerebral ischemia may also occur in the absence of significant large-vessel vasospasm. Potential mechanisms include microthrombosis, activation of the inflammatory cascade, micro vascular dysregulation and constriction, and cortical spreading depolarization. Targeted treatments for these mechanisms are under investigation. Subarachnoid Hemorrhage CHAPTER 440 Acute hydrocephalus can cause stupor or coma. It may clear spontaneously or require temporary ventricular drainage. When chronic hydrocephalus develops, ventricular shunting is the treat ment of choice. Free-water restriction is contraindicated in patients with SAH at risk for delayed cerebral ischemia because hypovolemia and hypotension may occur and precipitate cerebral ischemia. Many patients continue to experience a decline in serum sodium due to cerebral salt wasting despite receiving parenteral fluids contain ing normal saline. Frequently, supplemental oral salt coupled with normal saline will mitigate hyponatremia, but often patients also require intravenous hypertonic (3%) saline. Care must be taken not to correct serum sodium too quickly in patients with marked hypo natremia of several days' duration, as the osmotic demyelination syndrome (Chap. 318) may occur. All patients should have pneumatic compression stockings applied to prevent pulmonary emboli. Unfractionated heparin and low-molecular-weight heparinoids administered subcutaneously for DVT prophylaxis can be initiated within 1–2 days following endovascular treatment or craniotomy with surgical clipping; this approach is more effective than use of pneumatic compression stock ings alone. Treatment of pulmonary emboli depends on whether the aneurysm has been treated and whether or not the patient has had a craniotomy. Continuous systemic anticoagulation with heparin is contraindicated in patients with ruptured and untreated aneurysms. It is a relative contraindication following craniotomy for several days, and it may delay thrombosis of a coiled aneurysm. If DVT or pulmonary emboli occur within the first days following craniotomy, use of an inferior vena cava filter may be considered to prevent additional emboli, whereas systemic anticoagulation with heparin is preferred following successful endovascular treatment. In patients who survive their acute hospitalization, follow-up care is important to address the high prevalence of cognitive and behavioral deficits that greatly impact quality of life. Efficient recognition and treatment of these disorders can improve both short-term and long-term outcomes. In addition, some patients require ongoing follow-up to manage unruptured aneurysms that may require further care. ■ ■FURTHER READING Hoh BL et al: 2023 Guideline for the management of patients with aneurysmal subarachnoid hemorrhage: A guideline from the American Heart Association/American Stroke Association. Stroke 54:e314, 2023. Molyneux AJ et al: The durability of endovascular coiling versus neu rosurgical clipping of ruptured cerebral aneurysms: 18 year follow-up of the UK cohort of the International Subarachnoid Aneurysm Trial (ISAT). Lancet 385:691, 2015. Treggiari MM et al: Guidelines for the neurocritical care management of aneurysmal subarachnoid hemorrhage. Neurocrit Care 39:1, 2023.

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