# Aneurysmal subarachnoid hemorrhage: Treatment and prognosis

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## INTRODUCTION

Nontraumatic subarachnoid hemorrhage (SAH) is most commonly caused by ruptured saccular aneurysms. SAH is often a devastating event with substantial mortality and high morbidity among survivors.

The treatment of aneurysmal SAH and its complications are reviewed here. Other aspects of this illness are discussed separately. (See "Treatment of cerebral aneurysms" and "Aneurysmal subarachnoid hemorrhage: Epidemiology, risk factors, and pathogenesis" and "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis".)

The management of nonaneurysmal SAH, including traumatic SAH, is discussed elsewhere. (See "Nonaneurysmal subarachnoid hemorrhage", section on 'Management and prognosis' and "Management of acute moderate and severe traumatic brain injury".)

## CRITICAL CARE MANAGEMENT

**Stabilization** — Initial care of patients with SAH is directed at reversing or stabilizing life-threatening conditions, particularly for comatose patients. Important steps include ensuring a secure airway, normalizing cardiovascular function, and treating seizures [1,2].

Indications for endotracheal intubation include a Glasgow Coma Scale (GCS) score ≤8 ( table 1), elevated intracranial pressure (ICP), poor oxygenation or hypoventilation, hemodynamic instability, and requirement for heavy sedation or paralysis.

**Grading severity of SAH** — The degree of neurologic impairment and the extent of subarachnoid bleeding at the time of admission are the most important predictors of neurologic complications and outcome [1,3]. Therefore, it is imperative to grade the severity of SAH as soon as feasible after presentation and stabilization of patients with SAH.

A number of grading systems are used in practice to standardize the clinical classification of patients with SAH based upon the initial evaluation. The grading system proposed by Hunt and Hess ( table 2) and that of the World Federation of Neurological Surgeons (WFNS) ( table 3) are among the most widely used. The WFNS system incorporates the Glasgow Coma Scale ( table 1) combined with the presence of motor deficit.

The Fisher grade is an index of vasospasm risk based upon a computed tomography (CT)-defined hemorrhage pattern ( table 4), and the Claassen grading system (also known as modified Fisher scale) is a similar index of the risk of delayed cerebral ischemia due to vasospasm ( table 5).

A system proposed by Ogilvy and Carter stratifies patients based upon age, Hunt and Hess grade, Fisher grade, and aneurysm size ( table 6). In addition to predicting outcome, this scale more accurately substratifies patients for therapy.

> Grading scales for SAH are discussed in greater detail separately. (See "Subarachnoid hemorrhage grading scales".)

**Admit or transfer to expert center** — Patients with aneurysmal SAH are most appropriately cared for in high-volume centers with dedicated neurocritical care units and experienced staff that includes neurovascular surgeons, endovascular specialists, and neurologic intensive care specialists [2-4]. Observational data suggest that such centers have improved outcomes with lower mortality [5-9].

**Acute care** — The most important goal of SAH management is the prevention of rebleeding by early repair of the unsecured aneurysm with surgical clipping or endovascular coiling. (See 'Aneurysm treatment' below.)

Additional measures to reduce the risk of neurologic and systemic complications, particularly vasospasm and delayed cerebral ischemia, involve blood pressure control, maintenance of euvolemia, treatment with nimodipine, and continuous monitoring for hemodynamic and

neurologic complications. Additional acute measures include bedrest, analgesia, venous thromboembolism prophylaxis, and discontinuation of antithrombotics (plus reversal of anticoagulation when present). Hypoxemia, hyperglycemia, fever, and cardiovascular instability are common complications associated with poor outcome and should be prevented and promptly treated.

**Blood pressure control** — The optimal therapy of hypertension in SAH is not clear. For patients with acute SAH and an unsecured ruptured aneurysm, we suggest a gradual reduction of elevated systolic blood pressure (SBP; eg, >180 mmHg or mean arterial pressure [MAP] >120 mmHg), in agreement with guidelines [3,10]. The specific target should be individualized based on severity of initial blood pressure elevation, presence of brain swelling, and risk of kidney impairment. For most patients with acute SAH, we use a target SBP <160 mmHg or MAP <110 mmHg. It is reasonable to use premorbid baseline blood pressure to refine these targets [4]. Hypotension should be avoided.

When blood pressure control is necessary, intravenous labetalol, nicardipine, clevidipine, or enalapril are preferred. The use of vasodilators such as nitroprusside or nitroglycerin should be avoided because of their propensity to increase cerebral blood volume and therefore ICP.

While lowering blood pressure may decrease the risk of rebleeding in a patient with an unsecured aneurysm, this benefit may be offset by an increased risk of infarction. Cerebral perfusion pressure (CPP) equals the MAP minus the ICP. Thus, with increased ICP, cerebral perfusion may be impaired, and increases in MAP may be the only means to maintain CPP at a level necessary to prevent infarction. Results from one study suggest that this CPP threshold may be 70 mmHg [11]. In another report of 134 patients with SAH, 80 received antihypertensive therapy to lower the diastolic pressure below 100 mmHg [12]. The patients given antihypertensive therapy had a lower incidence of rebleeding (15 versus 33 percent) that was offset by a higher incidence of infarction (43 versus 22 percent).

In the absence of ICP measurement, antihypertensive therapy is often withheld unless there is a severe elevation in blood pressure [13]. The patient's cognitive status may be a useful guide. If the patient is alert, CPP is adequate and lowering the blood pressure may decrease the risk of rerupture; we typically keep the systolic blood pressure in such patients below 140 mmHg. In contrast, antihypertensive therapy is generally withheld in those with a severely impaired level of consciousness since the impairment may be due to a reduced CPP.

**Antithrombotic reversal** — We recommend discontinuation of all antithrombotic agents and reversal of all anticoagulation for acute SAH until the aneurysm is definitively repaired by surgery or coiling, in agreement with guidelines [3,14].

- **Antiplatelets** – We pretreat SAH patients with platelet transfusion (1 apheresis unit) prior to surgery if they had received antiplatelet agents or are thrombocytopenic (platelet count <100,000/microL) [14]. Otherwise, the role of platelet transfusion in intracranial hemorrhage is not established, even for patients on concomitant antiplatelet therapy. (See "Reversal of anticoagulation in intracranial hemorrhage", section on 'Limited role of platelet transfusions'.)

  In addition, a single intravenous dose of desmopressin (DDAVP) at 0.4 mcg/kg over 30 minutes can be given to patients who have received antiplatelet agents, but this is based on weak evidence and is not part of our routine practice.

- Warfarin – Any vitamin K antagonist effect should be reversed immediately with 4-factor prothrombin complex concentrate (PCC) and intravenous vitamin K; if a PCC is not available, a plasma product such as fresh frozen plasma (FFP) or thawed plasma may be used. Warfarin reversal is discussed in detail elsewhere. (See "Reversal of anticoagulation in intracranial hemorrhage", section on 'Warfarin'.)

- **Direct oral anticoagulants (DOACs)** – Available strategies for reversing the anticoagulant effect of DOACs include the following:

  - A specific reversal agent/antidote (for dabigatran, idarucizumab; for the oral direct factor Xa inhibitors, andexanet alfa) ( table 7)
  - Nonspecific agents such as prothrombin complex concentrates
  - Desmopressin
  - Drug removal from the circulation and/or gastrointestinal tract

  These options are discussed in detail separately. (See "Reversal of anticoagulation in intracranial hemorrhage", section on 'Dabigatran' and "Reversal of anticoagulation in intracranial hemorrhage", section on 'Apixaban, edoxaban, and rivaroxaban'.)

**Antifibrinolytic therapy** — Antifibrinolytic agents (eg, tranexamic acid, aminocaproic acid) should not be used routinely in the management of aneurysmal SAH [3,10]. A 2022 meta-analysis of 11 trials (2717 patients) found that antifibrinolytic therapy did not reduce the risk of poor outcome, defined as death, vegetative state, or severe disability [15]. In this analysis, antifibrinolytic treatment was associated with a reduced risk of rebleeding (odds ratio [OR] 0.65, 95% CI 0.47-0.91), but substantial heterogeneity between trials limits the certainty of these results. In addition, one large trial of 955 patients included in the analysis that assessed the effect of early administration of tranexamic acid (median 185 minutes) when risk of rebleeding may be highest found similar rates of rebleeding, functional improvement, and mortality for tranexamic acid or placebo [16].

**DVT prophylaxis** — Deep venous thrombosis (DVT) prophylaxis with intermittent pneumatic compression devices is started prior to aneurysm treatment [17]. Chemoprophylaxis with subcutaneous unfractionated heparin or low molecular weight heparin should be added once the aneurysm is secured [3]. (See "Prevention and treatment of venous thromboembolism in patients with acute stroke", section on 'Approach in subarachnoid hemorrhage'.)

**Seizure prophylaxis** — The prophylactic use of antiseizure medications in patients with SAH is controversial [3,18,19]. Many experts believe that seizure prophylaxis in the setting of an unsecured aneurysm is reasonable, given the relatively low risk associated with antiseizure medication administration versus the potential deleterious effects of seizures on an already dysautoregulated brain [20,21]. However, evidence from a large case series suggests that antiseizure medication exposure to phenytoin may be associated with worse neurologic and cognitive outcome after SAH [22]. Therefore, the use of antiseizure medications for seizure prophylaxis after SAH should probably be minimized whenever possible, and phenytoin is generally avoided.

The decision to treat with antiseizure medications may be based in part upon the distribution of blood on axial imaging studies. A higher threshold to start antiseizure medications is warranted in the setting of perimesencephalic blood without cortical layering, since this pattern of hemorrhage is associated with a particularly good prognosis. In contrast, initiation of antiseizure medications in higher risk patients with poor neurologic grade, cortical infarction, or hydrocephalus may be reasonable [3].

In the absence of acute seizures, antiseizure medication therapy should be discontinued in most patients after the aneurysm is secured [23].

**Euvolemia maintenance** — Hypovolemia is a risk factor for ischemic complications and should be avoided [24,25]. Euvolemia is the goal of intravenous fluid administration, usually with normal saline, which should be monitored by documentation of fluid input and output [3,10]. Fluid administration should also target normal electrolyte balance. Hyponatremia, in particular, is common, and sodium levels should be checked at least daily. (See 'Hyponatremia' below.)

There is no clear consensus about the optimal methods to determine euvolemia [1]. Combinations of methods are often used, including replacing urine output and strict monitoring of fluid balance, central venous pressure, and echocardiography. Some experts treat patients with significant diuresis using fludrocortisone or hydrocortisone [1].

**Monitoring** — A patient presenting with aneurysmal SAH is admitted to an intensive care setting for constant hemodynamic, cardiac, and neurologic monitoring [26,27].

- **Transcranial Doppler** – Transcranial Doppler (TCD) sonography is useful for detecting and monitoring vasospasm in SAH [3,4,28-30]. Velocity changes detected by TCD typically precede the clinical sequelae of vasospasm. Daily recordings offer a window of opportunity to treat patients prior to clinical decline. However, it is an operator-dependent technology that has imperfect sensitivity and specificity. In general, digital subtraction angiography is required to diagnose vasospasm and institute treatment. (See 'Vasospasm and delayed cerebral ischemia' below.)

- **Brain and vascular imaging** – Accumulating data suggest that arterial narrowing on CT angiography (CTA) and brain perfusion asymmetry demonstrated on CT perfusion (CTP) scanning in the acute stage of SAH may be useful and sensitive methods for predicting delayed cerebral ischemia [31-34]. The use of this technique as a monitoring tool may be limited by risks of recurrent dye loads and radiation exposure [3]. A finding of perfusion-diffusion mismatch on magnetic resonance imaging may be another method of detecting brain areas at risk of infarction in this setting [35]. The clinical utility of either of these methods remains to be established.

- **Continuous electroencephalography (EEG)** – Continuous EEG can be useful to detect subclinical seizures or nonconvulsive status epilepticus, particularly for patients with poor-grade SAH who develop unexplained neurologic deterioration or fail to improve [3,4].

- **Frequency of neuro checks and monitoring studies** – Patients with acute SAH should be carefully examined every one to two hours, especially during the high-risk period for delayed cerebral ischemia [1,27]. Symptomatic vasospasm and delayed cerebral ischemia are manifested clinically by neurologic decline, including the onset of focal neurologic abnormalities. (See 'Vasospasm and delayed cerebral ischemia' below.)

  A change in neurologic status (eg, development or worsening of inattention and confusion, new focal neurologic deficit, reduced level of consciousness, seizure) should be evaluated with an urgent head CT scan (to identify rebleeding, cerebral infarction, hydrocephalus), angiography (to identify symptomatic vasospasm), and/or EEG (to detect subclinical seizures). Medical complications can also contribute to a change in neurologic status.

  Even in the absence of clinical change, it may be important to identify cerebral vasospasm and decreased cerebral perfusion. Therefore, some centers monitor all patients with aneurysmal SAH with TCD sonography daily and head CT, CTA, and CT perfusion on admission and between days 3 to 5 and days 7 to 10 to screen for evidence of decreased cerebral perfusion or vasospasm [1]. Digital subtraction angiography can be used in place

of CTA/CTP. Additional monitoring may be employed for high-risk patients with poor neurologic status, including EEG, and invasive monitoring of brain tissue oxygenation and cerebral blood flow [36].

- **Cardiac studies** – Troponin levels and electrocardiogram should be checked on admission [4]. Echocardiography is also recommended, particularly for patients with suspected myocardial dysfunction.

- **Intracranial pressure monitoring** – We generally place a ventriculostomy in patients with enlarged ventricles on CT or with WFNS scale score ≥3 ( table 3); this allows direct measurement of ICP and also allows treatment by drainage of cerebrospinal fluid (CSF) when appropriate. While some concern has been raised about precipitating rebleeding with abrupt lowering of ICP, this has not been substantiated in clinical studies [37]. (See "Intraventricular hemorrhage", section on 'External ventricular drain' and "Evaluation and management of elevated intracranial pressure in adults", section on 'ICP monitoring'.)

**Nimodipine** — Nimodipine 60 mg every four hours is administered to all patients with aneurysmal SAH starting within 48 hours of symptom onset, or sooner once the patient is stabilized. Nimodipine must be given orally or by nasogastric tube because inadvertent intravenous administration has been associated with serious adverse events, including death. Treatment is continued for 21 days.

The calcium channel blocker nimodipine was initially studied in patients with SAH as a means to prevent vasospasm. However, despite the vasodilatory effects of nimodipine on cerebral vessels, there is no convincing evidence that nimodipine affects the incidence of either angiographic or symptomatic vasospasm [38-43]. Nevertheless, nimodipine has been demonstrated to improve outcomes in SAH and is the standard of care in these patients [3,10,38-41,44-47].

Meta-analyses of randomized trials of prophylactic nimodipine administered for SAH have consistently noted a benefit [42,43,48]:

- Nimodipine treatment compared with placebo improved the odds of a good outcome after SAH by 1.86 (99% CI 1.07-3.25) [42].

- Nimodipine reduced the odds of deficit, mortality, or both attributed to vasospasm by 0.46, and it reduced the rate of delayed cerebral ischemia by 0.53 (95% CI 0.39-0.72) [42,47].

- Overall mortality was lower with nimodipine than placebo (OR 0.73, 95% CI 0.53-1.00) [47].

- The number needed to treat (NNT) with nimodipine to prevent one poor outcome was 13 (95% CI 8-30) [43].

Blood pressure fluctuations and hypotension are common after administration of nimodipine. Thus, blood pressure monitoring is essential to avoid hypotension and decreased cerebral perfusion pressure.

The mechanism of benefit of nimodipine in SAH is unknown. Putative mechanisms include neuroprotection via dilation of small arteries not visible on angiograms, reduction of calcium-dependent excitotoxicity, diminished platelet aggregation, inhibition of ischemia triggered by red blood cell products, or some combination of these actions.

**Pain control** — For pain control of headache, neck pain, and other sources of pain, short-acting opiates (eg, morphine sulfate) are typically used [23]. European guidelines recommend starting with acetaminophen (paracetamol) 500 mg every three to four hours, avoiding aspirin before aneurysm is secured [4]. For more severe pain, the guidelines suggest opiates (eg, codeine, tramadol by suppository or intravenous administration) or piritramide (a mu opioid receptor agonist marketed in certain European countries) as a last resort.

**General care** — Patients with acute SAH are given stool softeners and kept at bedrest to diminish hemodynamic fluctuations and lower the risk of rebleeding prior to securing the aneurysm. The administration of stress ulcer prophylaxis is an area of controversy, as discussed separately. (See "Stress ulcers in the intensive care unit: Diagnosis, management, and prevention", section on 'Prophylaxis'.)

## ANEURYSM TREATMENT

After aneurysmal SAH, the patient is at substantial risk of rebleeding (see 'Rebleeding' below). Aneurysm repair with surgical clipping or endovascular coiling is the only effective treatment to prevent this occurrence and should be performed as early as feasible, preferably within 24 hours [3]; some expert centers report a median time to aneurysm repair of 7 hours from admission [1].

Patients in whom aneurysm treatment is not possible or must be delayed may be candidates for antifibrinolytic therapy (tranexamic acid or aminocaproic acid), but these agents should not be used for more than 72 hours. (See 'Antifibrinolytic therapy' above.)

Aneurysm treatment is discussed in more detail separately. (See "Treatment of cerebral aneurysms".)

## EARLY COMPLICATIONS

Medical and neurologic complications are common after SAH and contribute substantially to the overall morbidity and mortality. Patients with SAH are at risk for hemodynamic instability and neurologic deterioration. In one study, neurologic worsening occurred in 35 percent of patients within the first 24 hours of admission and heralded the onset of complications and poor outcomes [49].

**Rebleeding** — After aneurysmal SAH, the patient is at substantial risk of early rebleeding (4 to 14 percent in the first 24 hours, with maximal risk in the first 2 to 12 hours) [50-53]. Rerupture is associated with a high mortality.

Factors identified as predictors of rebleeding include [3,51,52,54-56]:

- Longer time to aneurysm treatment
- Worse neurologic status on admission
- Larger aneurysm size
- High systolic blood pressure
- Presence of intracerebral or intraventricular blood
- Acute hydrocephalus
- Incomplete obliteration of aneurysm with treatment

While most rebleeding is into the subarachnoid space, bleeding can also occur into the intraparenchymal, intraventricular, or subdural compartments. Rebleeding is usually diagnosed based on an acute deterioration of neurologic status accompanied by appearance of new hemorrhage on head computed tomography (CT) scan. Lumbar puncture is harder to evaluate because xanthochromia from the initial bleeding can persist for two weeks or more. (See "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis", section on 'Lumbar puncture'.)

Aneurysm treatment is the only effective treatment for the prevention of rebleeding. Therefore, patients with rebleeding should have emergency aneurysm repair. (See 'Aneurysm treatment' above.)

Rebleeding is associated with a higher rate of other complications and worse outcomes [57]. The mortality associated with rebleeding is reported to be as high as 70 percent.

**Vasospasm and delayed cerebral ischemia** — Delayed cerebral ischemia is a frequent complication of SAH; it contributes substantially to morbidity and mortality after SAH [58-60].

Delayed cerebral ischemia occurs in approximately 30 percent of patients with aneurysmal SAH, typically between 4 and 14 days after symptom onset [1,61].

The definition of delayed cerebral ischemia requires the occurrence of focal neurologic impairment (such as hemiparesis, aphasia, apraxia, hemianopia, or neglect) or a decrease of at least two points on the Glasgow Coma Scale that lasts for at least one hour, was not apparent immediately after aneurysm occlusion, and cannot be attributed to other causes after appropriate clinical assessment, brain imaging, and laboratory studies [62]. However, in patients with poor clinical grade (ie, already stuporous or comatose), delayed cerebral ischemia may not be clinically discernable [63].

In one case series of 56 patients with delayed cerebral ischemia, the two most common patterns of infarction were single cortical infarcts, typically located near the site of the ruptured aneurysm, and multiple widespread infarcts, often involving bilateral and subcortical regions and frequently located distal to the ruptured aneurysm, seen in 40 and 50 percent of patients, respectively [64]. Hypodense lesions on CT consistent with infarction appear to be more likely with larger volume SAH and poor initial clinical condition [65-71].

**Mechanisms and risk factors** — The most common cause of delayed cerebral ischemia after SAH is assumed to be vasospasm [72]. The severity of symptoms depends upon the artery affected and the degree of collateral circulation. Vasospasm typically begins no earlier than day 3 after hemorrhage, reaching a peak at days 7 to 8. However, vasospasm can occur earlier, even at the time of hospital admission [73-76]. Vasospasm is believed to be produced by spasmogenic substances generated during the lysis of subarachnoid blood. (See "Aneurysmal subarachnoid hemorrhage: Epidemiology, risk factors, and pathogenesis", section on 'Pathogenesis'.)

Risk factors for vasospasm include the severity of bleeding and its proximity to the major intracerebral blood vessels. The location and extent of blood on CT scan can help predict the likelihood of complicating cerebral vasospasm [77-79]. Radiologic grading scales including those of Fisher ( table 4) and Claassen ( table 5) are often used to predict the likelihood of vasospasm and cerebral ischemia. (See "Subarachnoid hemorrhage grading scales".)

Other factors that may increase the risk of vasospasm include age less than 50 years and hyperglycemia [80,81]. Most [82-84] but not all [80] studies have found that poor clinical grade (eg, Hunt and Hess grade 4 or 5, or Glasgow Coma Scale score <14) is associated with an increased risk of vasospasm. It is unclear whether the type of aneurysm treatment (surgical clipping versus endovascular coiling) influences the risk of vasospasm. Observational data from several studies suggest that endovascular coiling is associated with a reduced risk of vasospasm

[85-88], while other studies suggest that clipping and coiling have a similar risk [89,90], and still others suggest that coiling is associated with more severe vasospasm [91].

Mechanisms other than vasospasm may contribute to delayed cerebral ischemia. These include microcirculatory dysfunction with loss of autoregulation, microthrombosis, cortical spreading depression, and delayed cellular apoptosis [92,93]. There is also experimental evidence implicating early brain injury beginning at the time of aneurysm rupture and triggered by increased intracranial pressure (ICP) and global hypoperfusion that leads to glial activation, endothelial dysfunction, diffuse neuroinflammation, and subsequent ischemia [94].

**Management**

- **Prevention** – To reduce the risk of poor outcomes from delayed cerebral ischemia, all patients should receive nimodipine, and euvolemia should be maintained. (See 'Euvolemia maintenance' above and 'Nimodipine' above.)

  We do **not** use prophylactic hemodynamic augmentation as a preventive strategy for patients at risk for vasospasm and delayed cerebral ischemia, in agreement with guidelines [3]. Hemodynamic augmentation is generally reserved for treatment of radiographic or symptomatic vasospasm to minimize the risk of adverse cardiovascular effects associated with treatment.

- **Surveillance** – We generally do noninvasive angiography with CT angiography (CTA) with or without cerebral CT perfusion to confirm vasospasm for patients with elevated velocities on transcranial Doppler ultrasound (see 'Monitoring' above). Isolated, asymptomatic angiographic vasospasm traditionally has not been treated, but some experts will treat if the vasospasm is severe.

  An important distinction must be made between angiographic vasospasm, which is seen in 30 to 70 percent of angiograms performed at day seven after SAH, and clinical or symptomatic vasospasm, which is seen in 20 to 30 percent of patients [95-97]. Symptomatic vasospasm is associated with a clinical decline and portends a poorer prognosis.

  Angiography is used to identify patients with symptomatic vasospasm who might benefit from treatment. In one series in which angiograms were performed systematically in 381 patients at 9±2 days after aneurysmal SAH, the presence and severity of vasospasm correlated with the presence of cerebral infarction on CT [98]. However, even symptomatic vasospasm may not be identifiable on cerebral angiography as spasm in small arteries defies the resolution of even state-of-the-art angiography.

- **Treatment** – Aggressive therapy of vasospasm can only be pursued after the aneurysm has been treated with surgical clipping or endovascular coiling. Following aneurysmal occlusion, treatment options include:

  - **Hemodynamic augmentation** – First-line therapy for the treatment of new-onset delayed cerebral ischemia consists of hemodynamic augmentation by raising the mean arterial pressure (MAP) and thereby increase cerebral perfusion [36]. The treatment involves induced hypertension with vasopressor agents such as norepinephrine or phenylephrine and maintenance of euvolemia using crystalloid or colloid solution [3,4]. Blood pressure augmentation should progress in a stepwise fashion with assessment of clinical status at each mean arterial pressure level [4].

    The addition of inotropic support with agents such as dobutamine or milrinone has been reported to be helpful in patients who do not appear to respond to pressors alone [4]. Milrinone may also have a cerebral vasodilatory effect. Patients treated with hemodynamic augmentation need to be monitored for complications such as pulmonary or cerebral edema and volume overload.

    Hypervolemia should **not** be routinely used to prevent or treat delayed cerebral ischemia [3,10]. Earlier versions of this therapy included hypervolemia along with hemodilution and hypertension, so-called "triple-H" therapy, but subsequent studies revealed that hypervolemia was not beneficial and might be harmful [36,99].

  - **Balloon angioplasty** – For patients with focal vasospasm of larger cerebral arteries refractory to hemodynamic augmentation, balloon angioplasty has become the mainstay of treatment at many centers, despite an absence of clinical trial data [100,101].

  - **Intra-arterial administration of vasodilators** – For patients with focal or diffuse vasospasm of smaller cerebral arteries refractory to hemodynamic augmentation, intraarterial instillation of vasodilators may be used. Agents reported as effective for improving vasospasm in case series include intra-arterial nicardipine [102], milrinone [103], papaverine [104-106], nimodipine [107], verapamil [108], and intrathecal nitroprusside [109]. Intra-arterial vasodilator therapy and angioplasty also may be used in combination [105,108].

**Elevated intracranial pressure** — Patients with SAH may develop elevated intracranial pressure (ICP) due to a number of factors, including hemorrhage volume, acute hydrocephalus (see 'Hydrocephalus' below), reactive hyperemia after hemorrhage and/or ischemia, and distal cerebral arteriolar vasodilation [60,110-113]. In a series of 234 patients with SAH who had ICP

monitoring, increased ICP occurred during the hospital stay in 54 percent, including 49 percent of those considered to have a good clinical grade (Hunt and Hess grades I to III) [114].

**Hydrocephalus** — Hydrocephalus affects 20 to 30 percent of patients with SAH. It usually presents within the first few minutes to hours after SAH [23]. It can also be a later complication. Hydrocephalus after SAH is thought to be caused by obstruction of cerebrospinal fluid (CSF) flow by blood products or adhesions or by a reduction of CSF absorption at the arachnoid granulations [115]. The former occurs as an acute complication; the latter tends to occur two weeks after or later and is more likely to be associated with shunt dependence.

The clinical presentation of hydrocephalus in this setting is progressive deterioration in level of consciousness, accompanied by ventricular dilation on head CT scan. Ocular signs of elevated ICP (miosis, downward eye deviation, or restricted upgaze) occur in many but not all patients [116]. Spontaneous improvement occurs in approximately 30 percent of patients with acute hydrocephalus and impaired consciousness, usually within 24 hours [23,117]. In the remainder, acute hydrocephalus is associated with increased morbidity and mortality secondary to rebleeding and cerebral infarction [118].

Factors associated with an increased risk for hydrocephalus include older age, intraventricular hemorrhage, posterior circulation aneurysms, treatment with antifibrinolytic agents, and a low Glasgow Coma Scale score on presentation [119,120]. The incidence had also been reported to be increased in patients with hyponatremia or a history of hypertension.

**Management of elevated ICP**

- **CSF diversion** – Immediate CSF diversion with an external ventricular drain (EVD) or lumbar drainage is indicated for patients who have a deteriorating level of consciousness and evidence of elevated ICP and/or hydrocephalus [3,118]. (See "Evaluation and management of elevated intracranial pressure in adults", section on 'Removal of CSF'.)

  EVD placement is always indicated if the hydrocephalus is deemed noncommunicating (ie, from obstruction of CSF flow, typically at the level of the aqueduct). EVD placement ( figure 1) can improve the clinical grade of patients presenting with SAH [3]. Lumbar drainage is an alternative for patients with communicating hydrocephalus but is contraindicated with obstructive hydrocephalus or intraparenchymal hematoma [2].

  There is controversy regarding the preferred mode of CSF drainage through an EVD [121]. Some experts recommend a rapid weaning of the EVD after the aneurysm is secured, or within 48 hours of EVD placement, for patients who are neurologically stable [23,122].

Other experts prefer to avoid weaning the EVD during the first week or longer after the onset of SAH, a time when the risk of vasospasm reaches its peak.

It is unclear whether weaning or direct clamping of the EVD is optimal [3,10]. An intermittent CSF drainage with rapid EVD wean approach has been associated with fewer ventriculoperitoneal (VP) shunt placements, fewer complications, and shorter length of stay compared with a continuous CSF drainage with gradual EVD wean approach [123]. However, premature EVD wean resulting in increased ICP could exacerbate cerebral hypoperfusion, a particularly hazardous problem if the patient is also having vasospasm [124]. In one study, the number of failed clamping trials (ie, clamping the EVD catheter and stopping external drainage of CSF to determine if EVD removal can be tolerated) increased the likelihood that the patient would require a shunt [125]. Additional research is necessary to define the optimal strategy for CSF drainage and EVD wean in patients with aneurysmal SAH.

EVD placement may be complicated by infection (eg, ventriculitis/meningitis), particularly when drainage is continued for more than three days [117,126], and bleeding (eg, hemorrhage along the catheter tract), with a risk of approximately 8 percent for each [127]. There are few high-quality data to guide optimal EVD placement and management to minimize these risks [122].

- **Osmotic therapy and diuresis** – Patients without a ventriculostomy can be managed medically with osmotic therapy and diuresis. Hyperventilation is generally avoided because it may precipitate or exacerbate vasospasm. The use of hypertonic saline has been evaluated in patients with elevated ICP related to traumatic brain injury and appears to lower ICP and may improve cerebral perfusion. A small observational study in 16 patients with poor-grade SAH suggests that it may also be useful in this setting, but further study is required [128]. (See "Evaluation and management of elevated intracranial pressure in adults", section on 'Osmotic therapy and diuresis'.)

- **Hemicraniectomy** – In some cases, decompressive craniectomy may be needed for ICP control in the setting of intracerebral hemorrhage and/or severe cerebral edema. (See "Evaluation and management of elevated intracranial pressure in adults", section on 'Decompressive craniectomy'.)

- **Shunt-dependent hydrocephalus** – The need for long-term CSF diversion is assessed by attempting to wean or stop ventricular drainage in the subacute period [1,23]. Some patients who had EVD removed in the hospital after passing a clamping trial may

subsequently require placement of a ventricular shunt to treat delayed recurrence of symptomatic hydrocephalus that develops after hospital discharge [129,130].

In a systematic analysis including 23 studies and more than 22,000 patients with SAH, 22 percent developed shunt-dependent hydrocephalus [131]. Notable predictors of developing shunt dependency included acute hydrocephalus at presentation, poor clinical grade (elevated Hunt and Hess score) at admission, vasospasm, intraventricular bleeding, and female gender. Shunt rates were similar between patients undergoing surgical clipping and endovascular coiling [1,23,129,130,132,133]. Other studies have reported similar results [129,134,135].

**Hyponatremia** — Hyponatremia develops in up to 30 percent of patients with SAH [1,136] and is probably mediated by hypothalamic injury [23]. The water retention that leads to hyponatremia following SAH may result from either the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) or from cerebral salt wasting. These are physiologically distinct:

- Patients with SIADH are euvolemic. In other settings, therapy of asymptomatic hyponatremia in SIADH usually consists of water restriction, but fluid restriction is not desirable in patients with SAH as it increases the risk of intravascular volume contraction and consequently vasospasm-related ischemic injury. Thus, hyponatremia in patients with SAH is treated with hypertonic saline. (See "Treatment of hyponatremia: Syndrome of inappropriate antidiuretic hormone secretion (SIADH) and reset osmostat", section on 'Subarachnoid hemorrhage'.)

- Cerebral salt wasting is caused by excessive secretion of natriuretic peptides that diminish central sympathetic activity. It is characterized by volume depletion, which leads to the release of antidiuretic hormone (ADH). Cerebral salt wasting can be treated with fluid administration using infusions of isotonic saline if serum sodium is normal but may require infusions of hypertonic saline and fludrocortisone to counteract diuresis and natriuresis when these conditions impede maintenance of euvolemia. Restoration of euvolemia will suppress the release of ADH. (See "Cerebral salt wasting".)

SIADH and cerebral salt wasting can be difficult to differentiate since the laboratory findings are similar in both, including hyponatremia (<134 mEq/L), low serum osmolality (<274 mosmol/kg), high urine sodium (>40 mEq/L), and high urine osmolality (>100 mosmol/kg) [23]. The major distinguishing feature is intravascular volume status; patients with SIADH are euvolemic or even hypervolemic, while patients with cerebral salt wasting are hypovolemic.

A protocol for managing hyponatremia using 3 percent sodium chloride (NaCl) solutions in patients with SAH is presented in the table ( table 8).

**Seizures** — Acute seizures occur in 8 to 15 percent of patients with SAH [3]. Risk factors include thick subarachnoid clot, intracerebral hemorrhage, delayed infarction, and aneurysm in the middle cerebral artery. Seizures that occur prior to aneurysm treatment are often a sign of early rebleeding [23]. (See 'Neurologic morbidity' below.)

Patients with acute seizures after SAH are treated with antiseizure medications to prevent recurrence. Agents with favorable side effect profile, such as levetiracetam, are typically used. We **avoid** phenytoin because its use has been associated with worse cognitive outcomes in patients with aneurysmal SAH [22]. (See "Initial treatment of epilepsy in adults".)

For most patients with SAH and acute seizures, we typically wean antiseizure medication after one week, unless seizures recur, in agreement with guidelines [3]. While acute seizures are a risk factor for developing epilepsy, a longer duration of treatment does not reduce the risk of epilepsy, and most patients do not require long-term antiseizure medications. (See 'Neurologic morbidity' below.)

Generalized convulsive status epilepticus is unusual after SAH, occurring in 0.2 percent of patients in one study [137]. However, with increased use of continuous electroencephalography (EEG) monitoring, nonconvulsive status epilepticus and subclinical seizures have been recognized as a potential contributor to prolonged impairment of consciousness in patients after SAH. In one series, subclinical seizures were documented by continuous recording in 7 percent of patients after SAH [138,139]. Even when documented, the clinical correlates of nonconvulsive seizures can be subtle and nonspecific and may be limited to changes in heart rate, blood pressure, and/or ICP [140].

The management of status epilepticus is discussed separately. (See "Convulsive status epilepticus in adults: Classification, clinical features, and diagnosis".)

Nonconvulsive status epilepticus (NCSE) and subclinical seizures are increasingly recognized as a potential contributor to prolonged impairment of consciousness in patients after SAH. Diagnosis of NCSE and subclinical seizures requires a high index of suspicion as patients with NCSE are often those with poor neurologic grade and other neurologic complications of SAH, making it difficult to detect subclinical seizures; continuous EEG monitoring is often required. NCSE is associated with worse outcomes after SAH [140]. The diagnosis and management of NCSE is discussed separately. (See "Nonconvulsive status epilepticus: Classification, clinical features, and diagnosis".)

**Anemia** — Anemia is common after SAH, occurring in 18 percent of patients during their hospital stay in one report [141]. Most studies suggest that anemia is associated with worse outcomes, while higher hemoglobin levels are associated with fewer cerebral infarctions and

improved outcomes [142-145]. A goal hemoglobin for transfusion has not been defined in patients with SAH [3,10]; some experts recommend a target above 8 to 10 g/dL [4] and one randomized trial found that a higher transfusion goal (11.5 g/dL versus 10 g/dL) appeared to be safe [146]. Yet, red blood cell transfusion has been independently associated with worse outcomes in observational studies of patients with SAH, particularly among patients without delayed cerebral ischemia [147,148]. Therefore, larger randomized trials are necessary to determine the optimal transfusion strategy in SAH [149].

**Cardiopulmonary complications** — Pulmonary edema and cardiac arrhythmias complicate 23 and 35 percent of SAH cases respectively [150]. (See "Complications of stroke: An overview", section on 'Cardiac complications' and "Complications of stroke: An overview", section on 'Pulmonary complications'.)

A number of cardiac changes occur after SAH, including electrocardiography (ECG) changes, structural changes on echocardiography, and acute troponin elevations. These appear to be more common and more severe in those with more severe SAH [151,152]. Occasionally, focus on these findings can result in misdiagnosis.

- **ECG abnormalities** – The most frequent ECG abnormalities are ST segment depression, QT interval prolongation, deep symmetric T-wave inversions, and prominent U waves [153]. Life-threatening rhythm disturbances such as torsades de pointes have also been described, as well as atrial fibrillation and flutter. ST-T wave abnormalities along with bradycardia and relative tachycardia were found in one large series to be independently associated with mortality [154]. Another study reported an association between QT prolongation and angiographic vasospasm [155].

  The ECG changes are predominantly reflective of ischemic changes in the subendocardium of the left ventricle. The development of actual myocardial injury (in as many as 20 percent of patients) can be established by elevations of creatine kinase-MB (CK-MB) or serum troponin I (>0.1 mcg/L), which is a specific and more sensitive marker of myocardial necrosis [152,156]. Patients with elevated troponin I concentrations are more likely to have ECG abnormalities and clinical evidence of left ventricular dysfunction. Subendocardial ischemia is an independent predictor of poor outcome in patients with SAH and must be managed aggressively (although without the use of aspirin) [151]. (See "Troponin testing: Clinical use" and "Overview of the acute management of non-ST-elevation acute coronary syndromes".)

- **Left ventricular dysfunction** – Echocardiographic abnormalities may be found in approximately 21 percent of patients with acute SAH [157]. A wide spectrum of regional

left ventricular wall motion abnormalities can occur with SAH; these are typically but not always reversible [152,158,159]. Heart failure and pulmonary edema can result [160].

Some patients develop a pattern of transient apical left ventricular dysfunction that mimics myocardial infarction (but in the absence of significant coronary artery disease), a condition known as takotsubo cardiomyopathy, or transient left ventricular apical ballooning syndrome [159,161]. (See "Clinical manifestations and diagnosis of stress (takotsubo) cardiomyopathy".)

- **Troponin release** – Acute troponin elevation after SAH appears to be associated with increased risk of cardiopulmonary and cerebrovascular complications [151,162]. Elevated peak troponin levels have been associated with an increased risk of left ventricular dysfunction, pulmonary edema, and hypotension requiring pressors as well as with increased mortality and worse functional outcome [151]. (See "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome", section on 'Acute stroke'.)

**Other complications**

- **Hyperglycemia** has been associated with a poor outcome after SAH in many studies [163-167]. One study found that the use of an aggressive hyperglycemia management protocol appeared to improve glycemic control and neurologic outcomes; however, the use of a historical control group limits the validity of this study [168]. No blood glucose targets have been specifically identified for intervention. The 2023 American Stroke Association guidelines suggest careful glucose management with strict avoidance of hypoglycemia [3].

- **Fever** of infectious and noninfectious origin is a common complication of SAH, particularly in those with a higher neurologic grade, and is associated with a poor prognosis [169,170]. Body temperature should be monitored and infection should be ruled out or treated. We treat fever with antipyretics and cooling blankets. While high-quality evidence of benefit with this approach is lacking, one nonrandomized study found that the use of external cooling devices in patients with fever after SAH was associated with improved outcomes [171].

- **Hypothalamic-pituitary dysfunction** is common after SAH, but the clinical implications and appropriate treatment is uncertain [172]. Routine administration of glucocorticoids is not recommended after SAH but may be considered in patients who are unresponsive to vasopressor therapy for vasospasm [4].

## PROGNOSIS

Outcome after treatment of aneurysmal SAH is affected by potential brain injury from the SAH and subsequent complications as well as by risks related to neurosurgery. (See "Treatment of cerebral aneurysms".)

**Mortality**

- **Early mortality** – SAH is associated with a high early mortality rate [173]. Population-based studies found that 18 to 24 percent of patients with SAH died suddenly prior to even being evaluated in a hospital [174,175]. Among patients who reach the hospital alive, much of the subsequent early mortality is caused by the common complications of aneurysmal SAH related to initial bleeding, rebleeding, vasospasm and delayed cerebral ischemia, hydrocephalus, increased intracranial pressure, seizures, and cardiac complications [176-178].

  While early mortality due to SAH remains high, it is decreasing over time [175,179-185]. Historical case-fatality rates for SAH reached 50 percent [186], but they have declined to approximately 30 percent from 1988 to 2010 [179]. In a Finnish study of patients hospitalized with SAH, the 30-day case-fatality rate declined an average of nearly 2 percent each year between 1998 and 2017 with an overall rate of 20 percent during the study period [175]. Improved diagnostic accuracy over time and therapeutic advances in neurocritical care are plausible but unproven reasons for the reduction in mortality [23,185].

- **Long-term mortality** – Survivors of aneurysmal SAH have an increased mortality rate compared with the general population (standardized mortality ratio [SMR] 1.6) [187-191]. In a Swiss national registry of 1787 patients with SAH, the one-year mortality rate was 22 percent [192]. In the International Subarachnoid Aneurysm Trial (ISAT), the risk of death at five years was lower in the endovascular therapy group than in the surgical group [187]. In one cohort study, the excess mortality risk appeared to be attributed to cerebrovascular events [190]. The risk of nonfatal vascular events (eg, stroke, myocardial infarction) was also increased (relative risk [RR] 1.5) in survivors of aneurysmal SAH [189].

**Neurologic morbidity** — Long-term complications of SAH include neurocognitive dysfunction, epilepsy, and other focal neurologic deficits. In one registry, more than 10 percent of patients with SAH remained moderately or severely disabled [192].

Several studies suggest that SAH survivors have high rates of memory and neurocognitive impairment [3,193-199]. The largest prospective evaluation of neuropsychological function evaluated 873 survivors of SAH [197]. At three months after aneurysmal clipping, global impairment was present in approximately 20 percent of all survivors and in 16 percent of those

with excellent preoperative condition. Detailed neuropsychological testing of patients after surgically treated SAH has commonly shown cognitive deficits, even among patients making an otherwise good neurologic recovery [200]. The importance of these neuropsychological deficits to long-term morbidity is controversial, but they are often permanent [59,201].

The location of the aneurysm responsible for SAH does not appear to influence cognitive outcome [193], but the occurrence of vasospasm, delayed cerebral infarction, and other complications does [59,202]. Treatment modality does not compellingly influence outcome. In the ISAT, the proportions of survivors who reached independent status were similar in those treated with endovascular therapy versus surgical clipping [187]. However, among the group that was not otherwise disabled (modified Rankin Scale [mRS] >2), the proportion of those with cognitive impairment at 12 months was higher in those treated surgically than with endovascular coiling [194]. By contrast, in a follow-up study of the ISAT, health-related quality of life was higher after endovascular coiling compared with neurosurgical clipping, which contributed to the quality-adjusted life years gained over a 10-year period [203].

Depression, anxiety, and sleep disturbances are also common and contribute to decreased quality of life; these may be more amenable to treatment [196,198,204].

The incidence of late epilepsy after SAH is unclear. In a retrospective report of 472 patients with aneurysmal SAH who had undergone surgical clipping of the aneurysm between 1994 and 2000 and were followed for at least 12 months, late epilepsy occurred in 23 (5 percent) [205]. Patients presenting with a poor grade had a higher incidence of epilepsy (9.6 and 12.5 percent of those grades 3 and 4, respectively), as did those with associated cerebral infarction or subdural hematoma. In the ISAT, 25 of 612 patients (4 percent) had a diagnosis of epilepsy at a 12-month assessment; the rate was lower in patients treated with endovascular coiling as compared with surgical clipping [194]. Patients who have had acute seizures after SAH are somewhat more likely to develop epilepsy than those who do not. The management of epilepsy is discussed separately. (See "Initial treatment of epilepsy in adults" and "Overview of the management of epilepsy in adults".)

Anosmia may complicate SAH. Studies suggest that this is a more frequent complication in patients who undergo aneurysm clipping (one in three patients) than in those who undergo endovascular coiling (one in six patients) and that recovery is more likely in patients who also have endovascular therapy [206,207]. Intraventricular hemorrhage is a risk factor for this complication. Anosmia is a less frequent complication of nonaneurysmal perimesencephalic hemorrhage [208].

**Aneurysm recurrence and late rebleeding** — Patients who survive aneurysmal SAH have a small but enduring risk of recurrent SAH, which can occur despite successful endovascular or surgical treatment of the ruptured aneurysm. Recurrent SAH may result from recurrence of the treated aneurysm, rupture of another pre-existing aneurysm in a patient with multiple aneurysms, and de novo aneurysm formation. The risk of these events and recommendations for monitoring and treatment are discussed separately. (See "Late recurrence of subarachnoid hemorrhage and intracranial aneurysms".)

**Predictive factors** — The most important predictive factors for acute prognosis after SAH include [20,27,58,79]:

- Level of consciousness and neurologic grade on admission
- Patient age (inverse correlation)
- Amount of blood on initial head computed tomography scan (inverse correlation)

Other studies have noted that hypoxemia, hyperglycemia, renal insufficiency, fever, and anemia are also common after SAH and are associated with poor outcomes [150,163,209-211]. Correction of these physiologic derangements in patients with SAH is suggested [17], although no studies have confirmed the benefit of such treatments. Seizures at the onset of SAH appear to be an independent risk factor for late seizures (epilepsy) and a predictor of poor outcome [140,212,213].

**Screening of family members** — First-degree relatives of patients with SAH have a two- to fivefold increased risk of SAH compared with the general population [214,215]. It may be reasonable to screen some family members for the presence of cerebral aneurysm. The risk is much higher when there are two or more first-degree family members with ruptured aneurysms [215]. This issue is discussed in detail separately. (See "Screening for intracranial aneurysm".)

## SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Stroke in adults".)

## INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5$^{th}$ to 6$^{th}$ grade reading

level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or email these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Intracerebral hemorrhage (The Basics)" and "Patient education: Subarachnoid hemorrhage (The Basics)")
- Beyond the Basics topics (see "Patient education: Stroke symptoms and diagnosis (Beyond the Basics)" and "Patient education: Hemorrhagic stroke treatment (Beyond the Basics)")

## SUMMARY AND RECOMMENDATIONS

- **Acute care** – Patients with aneurysmal subarachnoid hemorrhage (SAH) should be admitted to an intensive care setting for constant hemodynamic, cardiac, and neurologic monitoring. We suggest a regimen to reduce the risk of neurologic and systemic complications including the following initial measures (**Grade 2C**):
  - **Blood pressure management** – Elevated blood pressure should be treated while avoiding hypotension. For most patients, we use a target systolic blood pressure (SBP) <160 mmHg or mean arterial pressure (MAP) <110 mmHg. (See 'Blood pressure control' above.)
  - **Stopping antithrombotic agents** – All antithrombotic agents should be discontinued, and anticoagulation reversed, until the aneurysm is repaired by surgery or coiling. (See 'Antithrombotic reversal' above.)
  - **Selective antiseizure prophylaxis** – The use of antiseizure medications for seizure prophylaxis after SAH is controversial. Antiseizure prophylaxis prior to aneurysm repair may be reasonable for some patients with poor neurologic grade, unsecured aneurysm, and associated intracerebral hemorrhage. (See 'Seizure prophylaxis' above.)
  - **Maintaining euvolemia** – Euvolemia should be maintained and monitored by documentation of fluid input and output to decrease the risk of ischemic complications.

(See 'Euvolemia maintenance' above.)

- Nimodipine – Nimodipine 60 mg every four hours is administered for 21 days to improve outcomes for patients aneurysmal SAH. (See 'Nimodipine' above.)

- **Other supportive measures** – Other general measures for all patients with acute aneurysmal SAH include includes bedrest, analgesia for pain control, and venous thromboembolism prophylaxis. Hypoxemia, hyperglycemia, and fever should be prevented and promptly treated. (See 'Acute care' above.)

- **Treatment of aneurysm** – Aneurysm repair with surgical clipping or endovascular coiling is the only effective treatment to prevent rebleeding and should be performed as early as feasible, preferably within 24 hours, and immediately if rebleeding does occur. (See 'Aneurysm treatment' above and "Treatment of cerebral aneurysms".)

- **Early complications** – Medical and neurologic complications are common after SAH. In addition to rebleeding, major concerns include vasospasm and delayed cerebral ischemia, elevated intracranial pressure (ICP) related to hydrocephalus or other causes, hyponatremia, and seizures. (See 'Early complications' above.)

  - **Vasospasm and delayed cerebral ischemia** – Following treatment of the aneurysms, neurologic deterioration due to vasospasm may be treated with hemodynamic augmentation via induced hypertension, balloon angioplasty, and/or intra-arterial administration of vasodilators. (See 'Vasospasm and delayed cerebral ischemia' above.)

  - **Elevated intracranial pressure** – Immediate CSF diversion with an external ventricular drain (EVD) is indicated for patients who have a deteriorating level of consciousness and evidence of elevated ICP and/or hydrocephalus. Additional options for some patients include osmotic diuresis and hemicraniectomy. (See 'Elevated intracranial pressure' above.)

  - **Seizures** – Seizures should be treated promptly, and antiseizure medications are usually discontinued after one week following SAH unless seizures recur. (See 'Seizures' above and 'Seizure prophylaxis' above.)

  - **Hyponatremia** – Hyponatremia after SAH is usually due to syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and rarely due to cerebral salt wasting. A protocol for managing hyponatremia in this setting is presented in the table ( table 8). (See 'Hyponatremia' above.)

- **Prognosis** – Mortality within the first 30 days after SAH approaches 30 percent and is attributed largely to the effects of initial and recurrent bleeding. Long-term complications of SAH include neurocognitive dysfunction, epilepsy, other focal neurologic deficits, and late rebleeding from aneurysm recurrence. (See 'Prognosis' above and "Late recurrence of subarachnoid hemorrhage and intracranial aneurysms".)



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## GRAPHICS

### Glasgow Coma Scale (GCS)

| | Score |
|---|---|
| **Eye opening** | |
| Spontaneous | 4 |
| Response to verbal command | 3 |
| Response to pain | 2 |
| No eye opening | 1 |
| **Best verbal response** | |
| Oriented | 5 |
| Confused | 4 |
| Inappropriate words | 3 |
| Incomprehensible sounds | 2 |
| No verbal response | 1 |
| **Best motor response** | |
| Obeys commands | 6 |
| Localizing response to pain | 5 |
| Withdrawal response to pain | 4 |
| Flexion to pain | 3 |
| Extension to pain | 2 |
| No motor response | 1 |
| **Total** | |

The GCS is scored between 3 and 15, 3 being the worst and 15 the best. It is composed of three parameters: best eye response (E), best verbal response (V), and best motor response (M). The components of the GCS should be recorded individually; for example, E2V3M4 results in a GCS score of 9. A score of 13 or higher correlates with mild brain injury, a score of 9 to 12 correlates with moderate injury, and a score of 8 or less represents severe brain injury.

Graphic 81854 Version 9.0

## Hunt and Hess grading system for patients with subarachnoid hemorrhage

| Grade | Neurologic status |
|---|---|
| 1 | Asymptomatic or mild headache and slight nuchal rigidity |
| 2 | Severe headache, stiff neck, no neurologic deficit except cranial nerve palsy |
| 3 | Drowsy or confused, mild focal neurologic deficit |
| 4 | Stuporous, moderate or severe hemiparesis |
| 5 | Coma, decerebrate posturing |

Based upon initial neurologic examination.

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*Adapted from: Hunt W, Hess R. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg 1968; 28:14.*

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Graphic 69179 Version 5.0

## World Federation of Neurological Surgeons subarachnoid hemorrhage grading scale

| Grade | GCS score | Motor deficit |
|---|---|---|
| 1 | 15 | Absent |
| 2 | 13 to 14 | Absent |
| 3 | 13 to 14 | Present |
| 4 | 7 to 12 | Present or absent |
| 5 | 3 to 6 | Present or absent |

GCS: Glasgow Coma Scale.

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

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Graphic 65468 Version 3.0

## Fisher grade of cerebral vasospasm risk in subarachnoid hemorrhage[1]

| Group | Appearance of blood on head CT scan |
|---|---|
| 1 | No blood detected |
| 2 | Diffuse deposition or thin layer with all vertical layers (in interhemispheric fissure, insular cistern, ambient cistern) less than 1 mm thick |
| 3 | Localized clot and/or vertical layers 1 mm or more in thickness |
| 4 | Intracerebral or intraventricular clot with diffuse or no subarachnoid blood |

CT: computed tomography.

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*Reference:*

1. *Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by CT scanning. Neurosurgery 1980; 6:1.*

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Graphic 81122 Version 4.0

## Modified Fisher (Claassen) subarachnoid hemorrhage CT rating scale

| Grade | Head CT criteria |
|---|---|
| 0 | No SAH or IVH |
| 1 | Minimal SAH and no IVH |
| 2 | Minimal SAH with bilateral IVH |
| 3 | Thick SAH (completely filling one or more cistern or fissure) without bilateral IVH |
| 4 | Thick SAH (completely filling one or more cistern or fissure) with bilateral IVH |

CT: computed tomography; SAH: subarachnoid hemorrhage; IVH: intraventricular hemorrhage.

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*From: Claassen J, Bernardini GL, Kreiter K, et al. Effect of cisternal and ventricular blood on risk of delayed cerebral ischemia after subarachnoid hemorrhage: the Fisher scale revisited. Stroke 2001; 32:2012.*

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Graphic 57558 Version 5.0

## Ogilvy and Carter grading system to predict outcome for surgical management of intracranial aneurysms

| Criteria | Points |
|---|---|
| Age 50 or less | 0 |
| Age greater than 50 | 1 |
| Hunt and Hess grade 0 to 3 (no coma) | 0 |
| Hunt and Hess grade 4 and 5 (in coma) | 1 |
| Fisher scale score 0 to 2 | 0 |
| Fisher scale score 3 and 4 | 1 |
| Aneurysm size 10 mm or less | 0 |
| Aneurysm size greater than 10 mm | 1 |
| Giant posterior circulation aneurysm size 25 mm or more | 1 |
| The total score ranges from 0 to 5, corresponding to grades 0 to 5 | |

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

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Graphic 70705 Version 4.0

## Direct oral anticoagulant reversal agents for life-threatening bleeding (imminent risk of death from bleeding)

| Anticoagulant | Reversal agent (all are given intravenously) |
|---|---|
| Dabigatran (Pradaxa; oral thrombin inhibitor) | ▪ Idarucizumab (Praxbind). Dose: 5 grams* |
| Oral factor Xa inhibitors:<br>▪ Apixaban (Eliquis)<br>▪ Edoxaban (Lixiana, Savaysa)<br>▪ Rivaroxaban (Xarelto) | ▪ Andexanet alfa (AndexXa). Dosing for the initial bolus and subsequent infusion depend on the dose level of the factor Xa inhibitor and the interval since it was last taken.<br>**or**<br>▪ 4-factor PCC (Kcentra, Beriplex P/N, Octaplex). Dosing can be done with a fixed dose of 2000 units **or** a weight-based dose of 25 to 50 units per kg. |

Reversal agents carry a risk of life-threatening thrombosis and should only be used under the direction of a specialist with expertise in their use and/or in a patient at imminent risk of death from bleeding. In general, a single dose is given; dosing may be repeated in rare situations in which the oral anticoagulant persists for longer in the circulation, such as severe kidney dysfunction.

Andexanet dosing is as follows:

- If the patient took rivaroxaban >10 mg, apixaban >5 mg, or dose unknown within the previous 8 hours: Andexanet 800 mg bolus at 30 mg/minute followed by 960 mg infusion at 8 mg/minute for up to 120 minutes.
  **or**
- If the patient took rivaroxaban ≤10 mg or apixaban ≤5 mg, or if ≥8 hours have elapsed since the last dose of a factor Xa inhibitor: Andexanet 400 mg bolus at 30 mg/minute followed by 480 mg infusion at 4 mg/minute for up to 120 minutes.

Refer to UpToDate topics on treatment of bleeding in patients receiving a DOAC or perioperative management of patients receiving a DOAC for additional information on administration, risks, and alternative therapies.

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DOAC: direct oral anticoagulant; FEIBA: factor eight inhibitor bypassing activity; PCC: prothrombin complex concentrate.

* If idarucizumab is unavailable, an activated PCC (FEIBA, 50 to 80 units per kg intravenously) may be a reasonable alternative.

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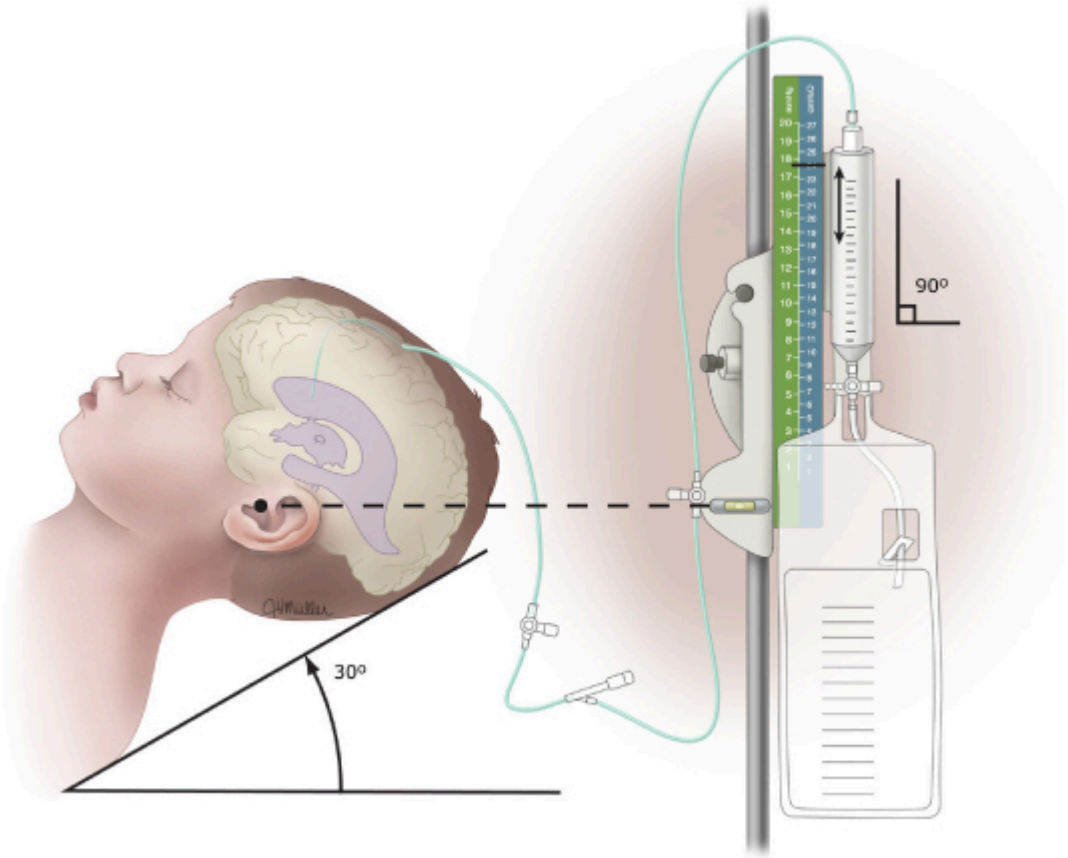
Graphic 112299 Version 10.0

# External ventricular drain




An external ventricular drain (EVD) is a small catheter inserted through the skull usually into the lateral ventricle, which is typically connected to a closed collecting device to allow for drainage of cerebrospinal fluid. The EVD can also be connected to a transducer that records intracranial pressure.

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Graphic 56391 Version 2.0

## Protocol for management of hyponatremia in patients with subarachnoid hemorrage

| For Na level <133 mEq/L or a decrease of 6 mEq/L in 24 to 48 hours: |
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| 1. NaCl tabs 3 g PO/NGT every 6 hours |
| 2. Initiate 3 percent NaCl infusion at 20 mL/hour IV |
| 3. Check serum Na every 6 hours |
| a. If Na <130 mEq/L: |
| Increase rate by 20 mL/hour (max rate = 80 mL/hour) |
| If on hold at present, initiate 3 percent NaCl infusion at 20 mL/hour IV |
| b. If Na = 130 to 135 mEq/L: |
| Increase rate by 10 mL/hour (max rate = 80 mL/hour) |
| If on hold at present, initiate 3 percent NaCl infusion at 10 mL/hour IV |
| c. If Na = 136 to 140 mEq/L: |
| No change |
| d. If Na ≥140 mEq/L: |
| Hold infusion |

NaCl: sodium chloride; PO: by mouth; NGT: nasogastric tube; IV: intravenously.

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Graphic 65772 Version 15.0