BMJ Best Practice

Subarachnoid hemorrhage

Straight to the point of care

Last updated: Dec 20, 2024

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Summary

Subarachnoid hemorrhage (SAH) presents as a sudden severe headache, often described as "the worst headache of life," with nausea, vomiting, and photophobia.

This topic focuses on the diagnosis and management of subarachnoid hemorrhage caused by aneurysm (i.e., aneurysmal SAH).

Examination can be normal or may reveal altered consciousness, meningismus, intraocular hemorrhages, or focal findings.

Computed tomography (CT) indicated if subarachnoid hemorrhage is clinically suspected. Lumbar puncture is indicated if CT is unrevealing. Cerebral angiography confirms the presence of aneurysms.

Initial stabilization followed by surgical clipping or endovascular coil embolization is standard therapy. Enteral nimodipine should be started early to prevent vasospasm, delayed cerebral ischemia and improve functional outcomes.

Complications are common and include rebleeding, acute hydrocephalus, and delayed cerebral ischemia.

Definition

SAH is bleeding into the subarachnoid space and is an emergency. The most common cause of nontraumatic SAH is rupture of an intracranial aneurysm, which is the focus of this topic.[1] Aneurysmal SAH causes substantial morbidity and mortality. When a cerebral aneurysm ruptures, blood flows into the subarachnoid space, sometimes seeping into brain parenchyma and/or ventricles. The sudden increase in intracranial pressure, as well as the destructive and toxic effects of blood on brain parenchyma and cerebral vessels, accounts for most complications.

Epidemiology

Worldwide, almost 500,000 individuals develop a SAH caused by an aneurysm each year, with almost two-thirds of these in low- and middle-income countries.[6] The global incidence of SAH declined between 1980 and 2010 which may parallel the global decreases in blood pressure and smoking prevalence.[7] However, a large variation in SAH incidence exists according to region, age, and sex.

The incidence in the US is between 6 and 8 cases out of 100,000 per year.[6][8] A higher incidence in Hispanic populations compared with in non-Hispanic populations has also been noted in some areas of the US.[9] Some countries have seen increases in the incidence of SAH. In Japan, the incidence of SAH increased between 1980 and 2010, especially in women older than 55 years.[10] Incidence also increases with age. The average age at onset is between 50 and 55 years.[1] [11] [12] SAH is 1.6 times more common in women than in men, and 2.1 times more common in black people than in white people.[8] [13]

SAH accounts for about 5% of all strokes.[14]

Etiology

Rupture of an intracranial saccular aneurysm is the leading cause of nontraumatic SAH, accounting for approximately 80% of cases. The remaining 20% are attributed to nonaneurysmal perimesencephalic SAH, arteriovenous malformations, arterial dissections, use of anticoagulants, and other rare conditions.[15] This distinction is crucial, as aneurysmal SAH has a different spectrum of complications and outcome, requiring more specific treatment and management. This topic focuses on the diagnosis and management of subarachnoid hemorrhage caused by aneurysm (i.e., aneurysmal SAH)

Cerebral saccular aneurysm formation is an acquired process. Very little is known about this process, but evidence suggests structural abnormalities are acquired in the intimal and medial layers of cerebral vessels, and could result from an inflammatory process occurring within these layers.[16] [17] [18] Structural abnormalities may be influenced by smoking, hypertension, and alcohol abuse.[15] Patients with previous SAH are at substantial risk for new aneurysm formation and enlargement of previously diagnosed and untreated aneurysms. This suggests that aneurysm formation is a dynamic, continuous process.[19] [20] [21] Hereditary and genetic factors may also contribute. Patients with Ehlers-Danlos syndrome, Marfan syndrome, pseudoxanthoma elasticum, adult polycystic kidney disease, and neurofibromatosis type I are at increased risk of aneurysm formation and SAH.

Pathophysiology

Cerebral aneurysms arise at the bifurcation of major arteries that form the circle of Willis. The majority are located at the anterior communicating/anterior cerebral artery junction (Acom/ACA), distal internal carotid artery/posterior communicating artery junction (ICA/Pcom), and middle cerebral artery bifurcation (MCA). Less than 10% arise from the vertebral or basilar arteries. Up to 19% of patients are found to have multiple aneurysms.[11] [12] Greater pressures at the apexes of arterial bifurcation, pulsatile flow patterns, and turbulence have been suggested as explanations for the predilection of aneurysm growth at these sites.[17] [22]

The risk of aneurysm rupture depends on its size, location, the presence of symptoms, the presence of multiple aneurysms, and whether previous aneurysms have ruptured.[17] [23] [24] [25] [26] [27] Patient-related predictors of rupture are age and smoking. Small, asymptomatic aneurysms (<7 mm) are less prone

to rupture than bigger ones that exert mass effect on surrounding structures. Aneurysms located at the basilar tip, in the vertebrobasilar, posterior cerebral distribution, or posterior part of the circle of Willis are more likely to rupture compared with aneurysms in other locations.[24] [28] The 5-year cumulative rupture rate of an aneurysm <7 mm in diameter is 0% when located on ICA, Acom, or MCA and 2.5% when located on Pcom or posterior cerebral, vertebral, or basilar arteries.[28] An unruptured aneurysm discovered during workup for SAH (caused by a different aneurysm) has a higher annual incidence of rupture than a single unruptured aneurysm.[17] [25][26] In this case, the 5-year cumulative rupture rate ranges between 1.5% and 3.4% for aneurysms <7 mm and between 2.6% and 18.4% for aneurysms between 7 mm and 24 mm.

Case history

Case history #1

A 53-year-old black woman presents with a sudden, excruciating headache which started while sitting at work. The headache is diffuse, intense, and accompanied by nausea and vomiting. She describes the headache as the worst headache of her life. She loses consciousness following the onset of the headache and is on the floor for less than 1 minute. She is being treated for hypertension and is a smoker. On examination she has a normal mental state, meningismus, bilateral subhyaloid hemorrhages, and right third cranial nerve palsy. There are no sensory deficits or weakness. Brain computed tomography (CT) reveals diffuse subarachnoid blood in basal cisterns and sulci.

Other presentations

An atypical history of SAH includes less severe headaches, headaches accompanied by vomiting and low-grade fever, and prominent neck pain. Around 10% to 43% of patients experience a sentinel headache during the 3 months prior to SAH.[2] Some of these headaches are caused by minor leaks from the aneurysm, which CT is unreliable in detecting.[3] Patients who experience sentinel headache might have an increased risk of rebleeding.[4] [5]

Approach

Occurrence of a sudden, severe headache is characteristic of SAH.[37] It is the most important clue to diagnosis and is often described as "the worst ever headache." Prompt diagnostic workup and evaluation are recommended to diagnose/exclude aneurysmal SAH (aSAH) and to minimize morbidity and mortality.[37]

History and exam

The first priority should be an urgent assessment of level of consciousness and need for cardiopulmonary resuscitation and/or ventilatory support.[38] History-taking (from the patient and/or relatives) may reveal risk factors of smoking, cocaine use, hypertension, family history of SAH, connective tissue disorders, or autosomal dominant polycystic kidney diseases. Consciousness level should be assessed using the Glasgow Coma Scale (GCS).[39] On admission, up to two-thirds of people with SAH have a depressed level of consciousness, half of whom are in a coma.[40] Physical exam can be normal, or there can be altered level of consciousness, agitation, altered mental state, meningismus, and focal findings. A poor level of awareness and seizures on presentation are risk factors for aspiration. A large hemorrhage burden and the presence of a subdural hematoma are associated with the occurrence of seizures after aneurysm rupture.[41] Photophobia, nausea, and vomiting are common symptoms. A full neurologic exam should be performed with special attention to pupillary reaction. Fixed and dilated pupils in comatose patients are associated with a poor prognosis, especially when present bilaterally.[42] Intraocular hemorrhages (secondary to increased intracranial pressure) are seen in 10% to 40% of patients with SAH.[43] They cause visual loss in the affected eye. This is associated with worse prognosis and increased mortality.[43] Cranial nerve palsies may be present. Isolated dilation of one pupil and loss of the pupillary light reflex may indicate brain herniation as a result of rising intracranial pressure, caused by an intraparenchymal component to the hemorrhage or hydrocephalus. A poor neurologic status on admission seems to predict cardiac abnormalities thought to be secondary to overwhelming sympathetic activation.[44] [45] [46] [47] Close monitoring of vital signs should be instituted, including blood pressure, heart rate and rhythm, and respiratory rate.[48]

Serum tests and ECG

Complete blood count, serum electrolytes, and clotting profile should be ordered in the initial workup in addition to serum troponin I. Half of patients have an abnormal ECG on admission.[49] Abnormalities include arrhythmias, prolonged QTc, and ST segment/T wave abnormalities.[44] [49][50][51]

Computed tomography (CT) and lumbar puncture (LP)

Suspicion of SAH based on a history of sudden, severe headaches is sufficient to order an emergency noncontrast brain CT as the first test.[37] [52] However, the specific workup required depends on the time of presentation from symptom onset and the patient's neurologic status.[37] Some, but not all patients will require additional investigation:[37]

- In patients who present <6 hours from symptom onset of acute onset severe headache, a noncontrast head CT performed on a high-quality scanner and interpreted by a board-certified neuroradiologist is reasonable to diagnose/exclude aneurysmal SAH (aSAH).
- In patients with acute onset of severe headache who present >6 hours from symptom onset or who have a new neurologic deficit, a negative noncontrast head CT should prompt a lumbar puncture (LP) to diagnose/exclude aSAH.

- In patients with acute onset of severe headache without a new neurologic deficit, application of the Ottawa SAH Rule may be reasonable to identify those at high risk for aSAH.

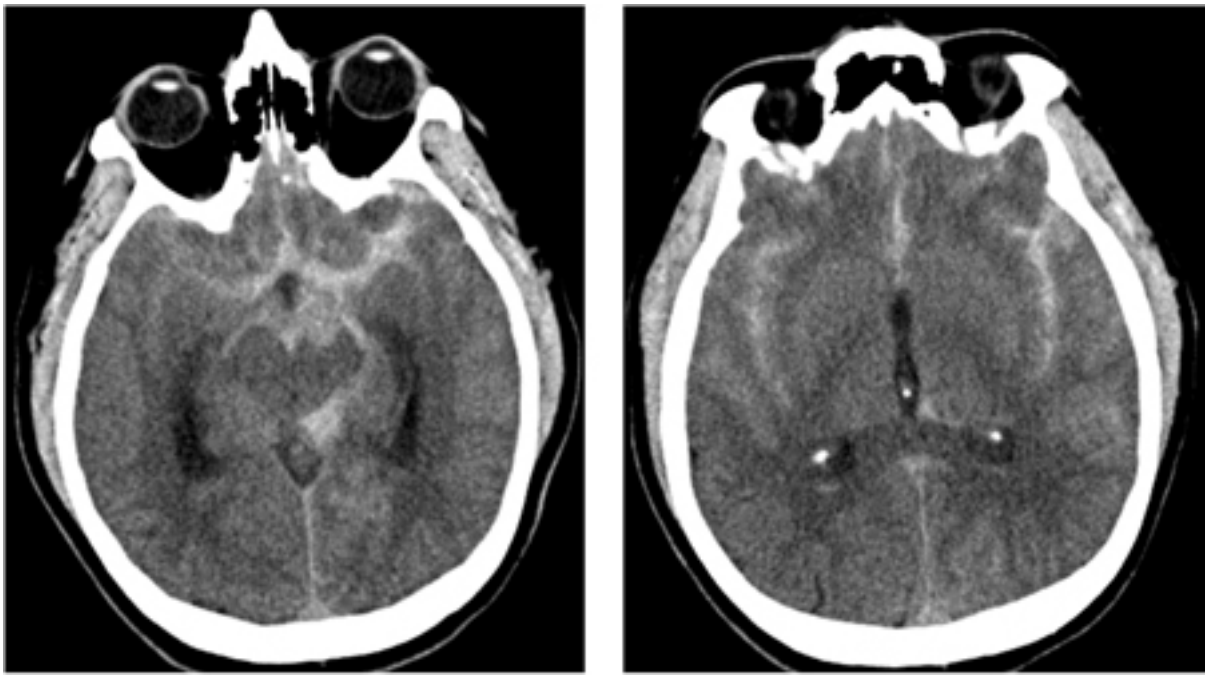
In atypical presentations, such as primary neck pain, syncope, seizure, or new focal neurologic deficit where there is a high suspicion of SAH, appropriate imaging and workup should still be considered.[37]

Ottawa SAH rule[53]

For alert patients >15 years of age with new severe nontraumatic headache reaching maximum intensity within 1 hour. Patients require additional investigation for SAH if they meet any of the following criteria:

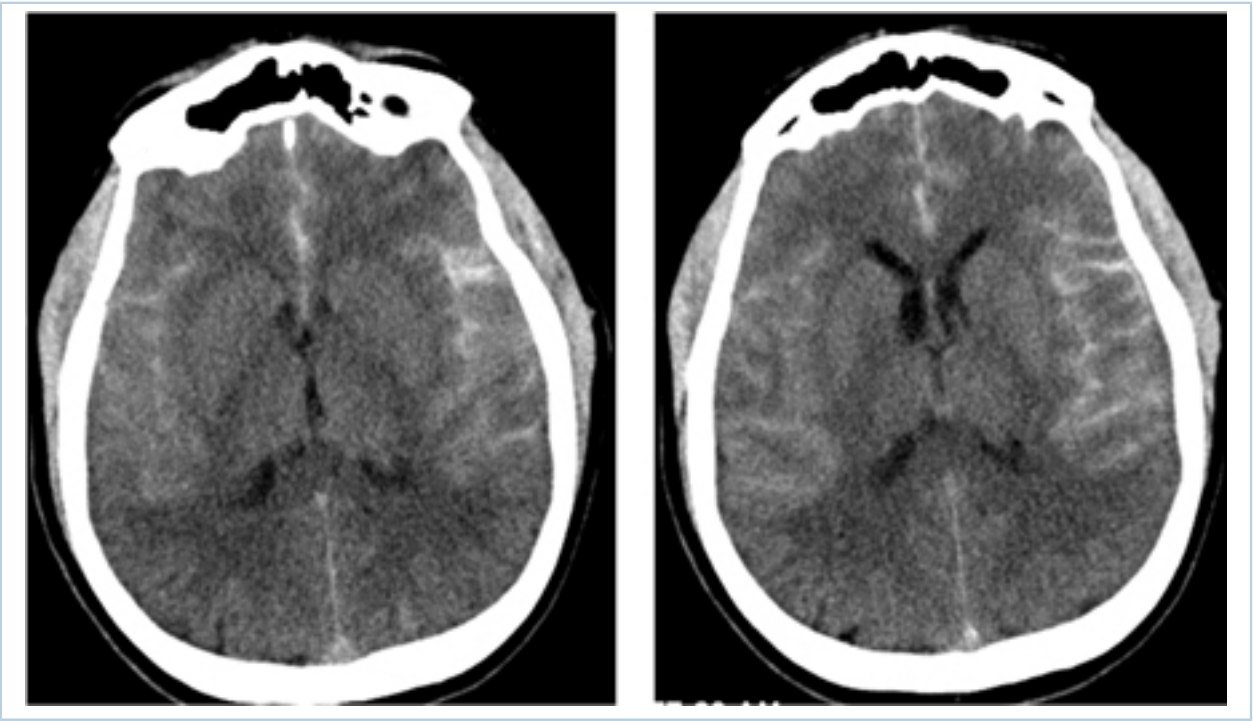
1. Ages ≥40 years
2. Neck pain or stiffness
3. Witnessed loss of consciousness
4. Onset during exertion
5. Thunderclap headache (instantly peaking pain)
6. Limited neck flexion on exam

CT: Thin cuts should be ordered (3-5 mm); otherwise, small, thin collections of blood might be missed. Subarachnoid blood will appear hyperdense (white) in the basal cisterns, major fissures, and sulci.[54] Detection of SAH on CT depends on density of blood, quantity of SAH, and timing of CT from ictus. A small quantity of blood in the subarachnoid space may be missed, and blood with hemoglobin below 10 g/dL may not be visible.[54] Nonetheless, the advent of third-generation CT scanners has dramatically improved the sensitivity of detecting subarachnoid blood, reaching 100% when performed within 6 hours of headache onset and read by experienced neuroradiologists.[55] [56] [57] The aneurysm rupture site can be predicted, though inconsistently, from patterns of blood accumulation on CT (thick collection in fissures) or parenchymal hematoma.[58]



CT brain showing subarachnoid hemorrhage from a ruptured posterior cerebral artery aneurysm (1 of 2)

Courtesy of Dr Salah Keyrouz; used with permission



CT brain showing subarachnoid hemorrhage from a ruptured posterior cerebral artery aneurysm (2 of 2)

Courtesy of Dr Salah Keyrouz; used with permission

Lumbar puncture: Four tubes of cerebrospinal fluid (CSF) should be collected and examined for gross blood. A serial count of red blood cells (RBCs) in tubes 1 to 4 is not sufficiently accurate to distinguish SAH from a traumatic LP. Visual inspection for xanthochromia is unreliable. Spectrophotometric analysis of hemoglobin degradation products is most reliable.[59] RBCs in the subarachnoid space start lysing approximately 12 hours after the bleed. Lysed RBCs will impart a xanthochromic (faint, yellow tinge) appearance to the CSF. However, high protein content in CSF or contamination with iodine used for disinfection can cause CSF to look xanthochromic.[60]

Further imaging

After SAH is confirmed by CT or LP, further imaging tests should be ordered.[67] Digital subtraction angiography (DSA) is the most accurate imaging technique used to diagnose aneurysms. CT angiography (CTA) and magnetic resonance angiography (MRA) are noninvasive imaging methods that have been compared with DSA.[67] [68] [69] [70] [71] CTA is widely available and often is the next diagnostic test performed when SAH is diagnosed with noncontrast CT.[37] A meta-analysis reported CTA to have a sensitivity of 92.7% and specificity of 77.2%, though another reported sensitivity and specificity surpassing 95%, especially when newer-generation multidetector scanners were used.[68] [69] [72][73] CTA head sensitivity for detecting aneurysm decreases for aneurysms <3 mm in size in the setting of diffuse SAH, and for aneurysms occurring adjacent to an osseous structure.[67] CTA may be sufficient to rule out a vascular cause of SAH when the location of hemorrhage is isolated to the perimesencephalic region with follow-up catheter-directed angiography indicated in CTA negative diffuse or peripheral SAHs.[67] [74] However, there remains equipoise concerning the appropriate diagnostic pathway for a perimesencephalic distribution of SAH with CTA alone versus catheter-based DSA.[37] Similarly, meta-analysis has shown that MRA has a sensitivity of 95% and specificity of 89%.[75] Limitations of MRA head include required safety screening and relatively long acquisition time in urgent clinical scenarios.[67] DSA is considered the gold-standard modality for the evaluation of cerebrovascular anatomy and aneurysm geometry and can aid in decision-making on the choice of optimal treatment modality.[37]

AHA/ASA recommends that in patients with spontaneous SAH with high level of concern for aneurysmal source and a negative or inconclusive CT angiography (CTA), digital subtraction angiography (DSA) should be performed to diagnose/exclude cerebral aneurysm(s).[37] For diffuse SAH, DSA is indicated for evaluation regardless of CTA results because small aneurysms or other vascular lesions may not be fully appreciated or defined on CTA imaging owing to limitations in spatial resolution.[74] [76] [77] [78] [79]

History and exam

Key diagnostic factors

headache (common)

- Most important clue to diagnosis when described as sudden, severe, or "worst ever."[37] Around 10% to 43% of patients experience a sentinel headache in the 3 months prior to SAH.[2]

photophobia (common)

- Eye pain with exposure to light.

loss of consciousness (common)

- On admission, up to two-thirds of people with SAH have a depressed level of consciousness, half of whom are in a coma.[40]

third cranial nerve palsy (uncommon)

- The presence of third cranial nerve palsy can be very useful and specific, as it typically signals the presence of a posterior communicating artery aneurysm compressing the ipsilateral third cranial nerve. Given their proximity to the third cranial nerve, aneurysms arising from the superior cerebellar artery or posterior cerebral artery can result in the same.

Other diagnostic factors

age >50 years (common)

- Average age between 50 and 55 years.[1] [11] [12]

female sex (common)

- After the sixth decade, women are affected 1.6 times more than men.[80]

black people (common)

- When adjusted for age and sex, in the US the incidence of aSAH is greater in black patients (15.4) compared with that in non-Hispanic white patients (9.9) and other races and ethnicities.[81]

nausea/vomiting (common)

- Seen in majority of patients with SAH but nonspecific.[82] [83]

altered mental status (common)

- Common but nonspecific.[82]

meningismus (uncommon)

- A clue to diagnosis only when associated with sudden, severe headache.

unilateral or bilateral sixth cranial nerve palsies (uncommon)

- This indicates increased intracranial pressure. Nonspecific.[84]

intraocular hemorrhage (uncommon)

- Intraocular hemorrhages are seen in 10% to 40% of patients with SAH.[43] Intraocular hemorrhages cause visual loss in the affected eye. This is associated with worse prognosis and increased mortality.[43]

focal neurologic deficits (uncommon)

- Focal neurologic deficits reflect presence of mass effect from subdural or parenchymal hematomas.[82] Independently associated with higher in-hospital mortality rates and severe disability at discharge.[85]

seizures (uncommon)

- Seizures occur in around 1% to 10% of patients with SAH.[86] [87] [88]

Risk factors

Strong

hypertension

- Hypertension is an important risk factor (relative risk is 2.8) and is potentially modifiable.[26] [29] [30] [31] [32] [33] [34]

smoking

- Smoking is one of the most important potentially modifiable risk factors.[26] [30] [31] [32] [33] [34] [35] Relative risk is 1.9.[10] [11]

family history

- First-degree relatives of patients with SAH have a 4% prevalence of harboring cerebral aneurysms and a three-fold to seven-fold increased risk of having SAH than the general population.[15] [34] The risk is highest when the affected relative is a sibling.[29] Population studies of aneurysmal SAH have demonstrated that 9% to 14% of patients with a SAH have a family history of SAH in a first-degree relative.[25]
- Having two or more first-degree relatives with SAH has a relative risk of SAH of 6.6.[15] Patients who have two or more first-degree relatives with SAH are potential candidates for aneurysm screening.[25] [29]

autosomal dominant polycystic kidney disease (ADPKD)

- ADPKD is an important risk factor (relative risk is 4.4).[29] One-quarter of patients with ADPKD have aneurysms at autopsy, and 2% to 8% of patients with aneurysms have ADPKD.[36]
- Individuals with ADPKD are potential candidates for aneurysm screening.[25] [29]

Weak

alcohol use

- The relationship of SAH to excessive alcohol use is less robust than that of hypertension or smoking.[30] [31] [34] [35]

cocaine use

- The relationship of SAH to cocaine use is less robust than that of hypertension or smoking.

Marfan syndrome

- Connective tissue disorder with an increased risk for aneurysmal formation and SAH.[36]

Ehlers-Danlos syndrome

- Connective tissue disorder with an increased risk for aneurysmal formation and SAH.[36]

pseudoxanthoma elasticum

- Connective tissue disorder with an increased risk for aneurysmal formation and SAH.[36]

neurofibromatosis type I

- Connective tissue disorder with an increased risk for aneurysmal formation and SAH.[36]

Tests

1st test to order

Test	Result
CT head • This is the standard diagnostic test for SAH and should be ordered if SAH is suspected.[52] • Modern, third-generation scanners have dramatically improved the sensitivity of detecting subarachnoid blood, reaching 100% when performed within 6 hours of headache onset and read by experienced neuroradiologists.[55] [56] [57] In patients who present <6 hours from symptom onset of acute onset severe headache, a noncontrast head CT performed on a high-quality scanner and interpreted by a board-certified neuroradiologist is reasonable to diagnose/exclude aneurysmal SAH.[37] In patients with acute onset of severe headache who present >6 hours from symptom onset or who have a new neurologic deficit, a negative noncontrast head CT should prompt a lumbar puncture to diagnose/exclude aneurysmal SAH.[37] CT may also show subdural or parenchymal hematoma, hypodensities, hydrocephalus, and sometimes the aneurysm(s) if large or thrombosed.[54]	**hyperdense areas in the basal cisterns, major fissures, and sulci**
CBC • This is a nonspecific test. Leukocytosis following SAH is an independent risk factor for cerebral vasospasm.[89]	**may show leukocytosis**
clotting profile • Coagulopathy may be present.	**may show coagulopathy: elevated INR, prolonged PTT**
serum electrolytes • Hyponatremia is usually associated with syndrome of inappropriate antidiuretic hormone secretion (SIADH).[7] It may also occur due to salt wasting.[7] [48] Hyponatremia occurs in up to 50% of patients.[90] It is associated with increased morbidity including vasospasm.[91]	**may show electrolyte abnormalities**
troponin I • Elevated in 20% to 28% of cases during the first 24 hours, in the absence of coronary artery disease.[44] [50] This elevation in troponin I is an order of magnitude less than what is usually seen in the setting of myocardial infarction, and it seems to be associated with an increased risk of delayed cerebral ischemia, poor outcome, and death following SAH.[51] [92]	**may be elevated**
serum glucose • Hyperglycemia develops in one third of SAH patients and is a feature of any acute brain injury. It is associated with poor clinical condition on admission and independently associated with poor outcome.[39]	**may be elevated**
ECG • Fifty percent of patients with SAH have an abnormal ECG on admission.[49] Abnormalities include arrhythmias, prolonged QTc, and ST segment/T wave abnormalities.[44] [49] [50] [51]	**may be abnormal: arrhythmias, prolonged QT, ST segment, or T wave abnormalities**

Other tests to consider

Test	Result
lumbar puncture (LP) • In patients with acute onset of severe headache who present >6 hours from symptom onset or who have a new neurologic deficit, a negative noncontrast head CT should prompt a lumbar puncture to diagnose/exclude aneurysmal SAH. • Cerebrospinal fluid (CSF) opening pressure should be measured. Four tubes of CSF should be collected and examined for gross blood. A serial count of red blood cells in tubes 1 to 4 is not sufficiently accurate to distinguish SAH from a traumatic LP. Visual inspection for xanthochromia is unreliable. Spectrophotometric analysis of hemoglobin degradation products is most reliable.[59] • Xanthochromia is an indicator of the presence of blood in the subarachnoid space.	**bloody CSF (xanthochromia)**
digital subtraction angiography (DSA) • DSA is considered the gold-standard modality for the evaluation of cerebrovascular anatomy and aneurysm geometry and can aid in decision-making on the choice of optimal treatment modality.[37] AHA/ASA recommends that in patients with spontaneous SAH with high level of concern for aneurysmal source and a negative or inconclusive CT angiography (CTA), digital subtraction angiography (DSA) should be performed to diagnose/exclude cerebral aneurysm(s).[37] For diffuse SAH, DSA is indicated for evaluation regardless of CTA results because small aneurysms or other vascular lesions may not be fully appreciated or defined on CTA imaging owing to limitations in spatial resolution.[74] [76] [77] [78] [79]	**aneurysm**
computed tomography angiography (CTA) • CTA is widely available and often is the next diagnostic test performed when SAH is diagnosed with noncontrast CT.[37] A meta-analysis reported CTA to have a sensitivity of 92.7% and specificity of 77.2%, though another reported sensitivity and specificity surpassing 95%, especially when newer-generation multidetector scanners were used.[68] [69] [72] [73] • CTA head sensitivity for detecting aneurysm decreases for aneurysms <3 mm in size in the setting of diffuse SAH, and for aneurysms occurring adjacent to an osseous structure.[67] CTA head may be sufficient to rule out a vascular cause of SAH when the location of hemorrhage is isolated to the perimesencephalic region with follow-up catheter-directed angiography indicated in CTA negative diffuse or peripheral SAHs.[67] [74] However, there remains equipoise concerning the appropriate diagnostic pathway for a perimesencephalic distribution of SAH with CTA alone versus catheter-based DSA.[37]	**aneurysm**
magnetic resonance angiography (MRA) • Noninvasive. Meta-analysis has shown that MRA has a sensitivity of 95% and specificity of 89%.[75] Limitations of MRA head include required safety screening and relatively long acquisition time in urgent clinical scenarios.[67]	**aneurysm**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Nonaneurysmal perimesencephalic SAH	• There are no features in the history or on examination that differentiate this condition from aneurysmal SAH.	• No aneurysms are found on angiography. CT usually reveals subarachnoid blood in front and around the pons (perimesencephalic or pontine cistern). Caution is required when this blood distribution pattern is seen, as it may also be seen with a ruptured aneurysm located in the posterior circulation.[60] Overall, it has a better outcome than aneurysmal SAH.
Arterial dissection	• Pain is less severe and is frequently felt behind the eye or localized to anterior or posterior neck region. Dull neck pain might precede a more severe pain, occurring at the time of SAH. Examination findings might include a Horner sign and/or neurologic deficits related to stroke secondary to dissection.	• Dissected arteries are visualized on cerebral angiography, magnetic resonance angiography, or computed tomography angiography. Axial T1 and T2 fat-suppressed neck MRI images might visualize the characteristic intramural hemorrhage associated with dissection.
Cerebral and cervical arteriovenous malformation (AVM)	• Symptoms and signs are similar to aneurysmal SAH. Subarachnoid hemorrhage could be preceded or accompanied by findings related to mass effect caused by the AVM.	• Arteriovenous malformations visualized on cerebral angiography, magnetic resonance angiography, or computed tomography angiography.
Dural arteriovenous fistulae (AVF)	• Symptoms and signs are similar to aneurysmal SAH.	• Arteriovenous fistulae visualized on cerebral angiography, magnetic resonance angiography, or computed tomography angiography.
Vasculitis	• A subacute to chronic history of recurrent neurologic deficits, with corresponding abnormalities on exam. Headache is usually less severe.	• Cerebrospinal fluid pleocytosis may be present. Angiography might disclose beading of medium and small intracranial arteries. Brain and meningeal biopsy is, however, the diagnostic standard for this condition.

Condition	Differentiating signs / symptoms	Differentiating tests
Saccular aneurysms of spinal arteries	• Pain localized to posterior neck/occipital area. Meningismus might be more prominent. A sciatica-like picture due to blood in the lumbar thecal sac can be seen.	• Spinal angiography visualizes the aneurysm(s).
Cardiac myxoma	• Age between 30 and 60 years. Cardiac, obstructive, or constitutional symptoms precede SAH.	• Echocardiography is method of choice for diagnosis.
Septic (mycotic) aneurysm	• On physical exam there may be a fever, heart murmur, skin petechiae, Osler nodes, Janeway lesions, splinter hemorrhages under the nails, and Roth spots in optic fundi. Ischemia may occur in the bowel and spleen.	• Subarachnoid blood is usually focal, not widely distributed in cisterns, fissures, and sulci as in aneurysmal SAH. Blood cultures may be positive. Blood tests may show an elevated erythrocyte sedimentation rate and peripheral leukocytosis. Cerebral angiography reveals aneurysms located distally, typically in the distribution of the middle cerebral artery. Intracerebral hematomas are likely to be seen on CT. Echocardiography might reveal valvular vegetations.
Pituitary apoplexy	• Patients have a known history of pituitary adenoma. Visual loss is seen in up to half of patients with pituitary apoplexy (not a feature of aneurysmal SAH). Acute adrenal insufficiency develops in two-thirds of patients.	• MRI with contrast shows pituitary hemorrhage or infarction. Subarachnoid blood is minimal and confined to the region around the pituitary gland.
Cocaine abuse	• There is a history of drug abuse preceding the event. The headache is usually less severe.	• Urine drug screen is positive for cocaine. Subarachnoid blood is usually minimal and focal in sulci. CT might also reveal intracerebral hematomas.
Anticoagulant-associated intracranial hemorrhage	• There is a history of anticoagulant use. The headache is less severe.	• A CT shows minimal subarachnoid blood and possible intracerebral hematomas. Coagulation studies are abnormal

Condition	Differentiating signs / symptoms	Differentiating tests
		(prolonged PTT and/or elevated INR).
Sickle cell disease	• There is a history of sickle cell disease, previous strokes, or sickling episodes.	• Computed angiography might reveal intracerebral hematomas associated with subarachnoid blood. Hemoglobin S is identified upon testing.

Criteria

Hunt and Hess Grading Scale[93]

Grade I: asymptomatic or minimal headache and slight nuchal rigidity (survival 70%).

Grade II: moderate to severe headache, nuchal rigidity, and no neurologic deficits other than cranial nerve palsy (survival 60%).

Grade III: drowsiness, confusion, or mild focal deficits (survival 50%).

Grade IV: stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity, and vegetative disturbances (survival 20%).

Grade V: deep coma, decerebrate rigidity, and moribund appearance (survival 10%).

World Federation of Neurological Surgeons Grading Scale (adapted from Suarez et al)[1]

Grade I: Glasgow Coma Scale (GCS) score 15. Motor deficit absent.

Grade II: GCS score 14-13. Motor deficit absent.

Grade III: GCS score 14-13. Motor deficit present.

Grade IV: GCS score 12-7. Motor deficit present or absent.

Grade V: GCS score 6-3. Motor deficit present or absent.

Modified Fisher Grading Scale[94]

The Modified Fisher and the Fisher grading scales predict the risk of vasospasm and delayed cerebral ischemia in SAH based on the amount and type of blood on computed tomography.[94][95] The modified version improves the original by incorporating the presence of intraventricular hemorrhage (IVH).[94]

Grade I: focal or diffuse thin SAH, no IVH.

Grade II: focal or diffuse thin SAH, with IVH.

Grade III: thick SAH present, no IVH.

Grade IV: thick SAH present, with IVH.

Screening

SAH is associated with high mortality and morbidity. Screening provides a major opportunity to treat intracranial aneurysms before catastrophic rupture. However, it is uncertain that widely applied screening programs are cost-effective given the low prevalence of cerebral aneurysms in the general population and risk of rupture. In addition, there are controversies about when and how to treat unruptured intracranial aneurysms. Patients who have two or more first-degree relatives with SAH or those with autosomal dominant polycystic kidney disease are potential candidates for aneurysm screening with computed tomography angiography (CTA) or magnetic resonance angiography (MRA).[25] In addition, patients who have survived SAH are at higher risk for another, due to a newly formed aneurysm. However, whether this subgroup will benefit from screening and how it should be performed is undecided.[29] [72]

Approach

SAH requires emergency treatment and early referral to a dedicated neurocritical care unit.[37][96] When patients are evaluated in rural or community settings, strong consideration should be made for expedited referral to high-volume tertiary care centers with multidisciplinary neurointensive care services, comprehensive stroke center capabilities, and experienced cerebrovascular surgeons/neuroendovascular interventionalists.[37] [97] Surgical or endovascular treatment of the ruptured aneurysm should be performed as early as feasible after presentation, preferably within 24 hours of onset, to improve outcome.[37]

Stabilization and cardiopulmonary support

Stabilization of patients simultaneously with workup is vital to prevent unwanted early complications. It is essential to establish the need for endotracheal intubation and mechanical ventilation as the first priority.[38] Consciousness level should be assessed using the Glasgow Coma Scale, in addition to airway adequacy and cardiovascular function.[39] A poor level of awareness and seizures on presentation are risk factors for aspiration. A large hemorrhage burden and the presence of a subdural hematoma are associated with the occurrence of seizures after aneurysm rupture.[41] A full neurologic exam should be performed with special attention to pupillary reaction. Fixed and dilated pupils in comatose patients are associated with a poor prognosis, especially when present bilaterally.[42] Intraocular hemorrhages (secondary to increased intracranial pressure) are seen in 10% to 40% of patients with SAH.[43] They cause visual loss in the affected eye. This is associated with worse prognosis and increased mortality.[43] Isolated dilation of one pupil and loss of the pupillary light reflex may indicate brain herniation as a result of rising intracranial pressure. A poor neurologic status on admission seems to predict cardiac abnormalities thought to be secondary to overwhelming sympathetic activation.[44] [45] [46] [47] Close monitoring of vital signs should be instituted (e.g., blood pressure, heart rate and rhythm, and respiratory rate).[37] [48]

In patients with aneurysmal SAH (aSAH) and unsecured aneurysm, frequent blood pressure (BP) monitoring and BP control with short acting medication(s) is recommended to avoid severe hypotension, hypertension, and BP variability.[37] There is insufficient evidence to recommend a particular BP target.[37] Sudden, profound reduction of BP should be avoided.[37] [101] In patients who are receiving anticoagulants, emergency reversal with appropriate agents should be performed to prevent rebleeding.[37] Reversal strategies should follow current published standards for life-threatening bleeding.[37] [102] If present, coagulopathy should be treated aggressively using prothrombin complex concentrate (PCC) or fresh frozen plasma (FPP), and vitamin K. See Anticoagulation management principles .

Electrolytes and fluids

Close monitoring and goal-directed treatment of volume status are reasonable to maintain euvolemia.[37] [103] [104] [105] Induction of hypertension and hypervolemia is potentially harmful because of the association with excess morbidity including cerebral edema, hemorrhagic transformation in areas of infarction, reversible leukoencephalopathy, myocardial infarction, and congestive heart failure.[37] [39] [106] [107] [108] [109] Therefore, prophylactic hemodynamic augmentation should not be performed to reduce iatrogenic patient harm.[37]

Electrolyte imbalances (e.g., hyponatremia) are common and should be corrected.[37] If present, coagulopathy should be treated aggressively using prothrombin complex concentrate or fresh frozen plasma, and vitamin K. American Heart Association states that use of mineralocorticoids such as

fludrocortisone is reasonable to treat natriuresis and hyponatremia, as supported by several RCTs.[37] However, although fludrocortisone use reduces excess sodium excretion, urine volume, and intravenous fluid use in patients with SAH, it has not been found to consistently affect outcome.[37] [110] [111] [112] [113] Some guidelines state there is insufficient evidence to support its use in maintaining normal serum sodium concentrations or improving functional outcome.[113] See Hyponatremia .

Analgesia

Analgesia should be provided to conscious patients. Acetaminophen can be used as a first-line option.[39] For severe pain, opioids such as codeine or tramadol should be given.[39] If a patient is still in pain, morphine or oxycodone may be required.[39] Aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided before aneurysm occlusion.[39] Mental status also needs to be closely monitored, especially in patients at risk of acute hydrocephalus or vasospasm. Judicious use of analgesia is therefore recommended.

Anticonvulsants

Prophylactic use of anticonvulsants following SAH is controversial.[114] [115]

US guidelines suggest that prophylactic anticonvulsants may be considered in patients with aSAH and high-seizure-risk features (i.e., ruptured middle cerebral artery aneurysm, high-grade SAH, intracranial hemorrhage, hydrocephalus, and cortical infarction).[37] These guidelines recommend against the routine use of anticonvulsants in patients with aSAH without high-seizure-risk features since phenytoin for seizure prophylaxis is associated with excess morbidity and mortality.[37] [88] [116] The risk of seizures is significantly lower after coil embolization than following surgical clipping of aneurysm.[37] Short-course treatment may be adequate prophylaxis, and some evidence suggests that it is better tolerated than a longer course.[87] [117] Routine long-term use of anticonvulsants is not recommended, but may be considered for patients with known risk factors for delayed seizure disorder.[37] In patients with aSAH who present with seizures, treatment with anticonvulsants for ≤7 days is reasonable to reduce seizure-related complications in the perioperative period.[37] In patients with aSAH without prior epilepsy who present with seizures, treatment with anticonvulsants beyond 7 days is not effective for reducing future aSAH-associated seizure risk.[37] [39] [114]

There is some evidence that in the US levetiracetam may be the most commonly prescribed agent for the prevention of seizures following SAH.[118] One small prospective study comparing levetiracetam with phenytoin for seizure prophylaxis after neurologic injury (including SAH) found the same outcomes with respect to mortality and seizure control, but levetiracetam-treated patients experienced better long-term functional outcomes than those treated with phenytoin (as as evaluated by the Glasgow Outcome Scale-Extended and Disability Rating Scale).[119]

Nimodipine

Early initiation of enteral nimodipine (a calcium-channel blocker) is beneficial in preventing vasospasm, delayed cerebral ischemia and improving functional outcomes in patients with aneurysmal SAH.[37]

Post stabilization

Once stabilized, the patient should be admitted to a dedicated neurocritical care unit.[97] These units significantly reduce in-hospital mortality and length of stay.[120] On admission to ICU, neurologic status should be graded using scales such as the Hunt and Hess Scale or the World Federation of Neurological Surgeons (WFNS) Scale.[37] These scales are recommended to determine initial clinical severity and

predict outcome.[37] The higher the grade, the poorer the outcome.[121] The WFNS scale is preferred over the Hunt and Hess scale; it is more reliable because it uses the GCS score in defining the mental status.[35] In contrast, the Hunt and Hess Scale grades level of consciousness into drowsiness, stupor, and deep coma.[35] The modified Fisher Scale can be used to document and grade the quantity and distribution of subarachnoid blood on admission CT.[94] Although not definitive, it helps to predict the potential risk of vasospasm, which is a serious complication.

Antitussives and stool softeners

Cough may be suppressed with antitussives to prevent potential rebleeding. Cough and cold medications that include opioids, such as codeine or hydrocodone, should not be used in children ages 18 years or younger as the risks (slowed or difficult breathing, misuse, abuse, addiction, overdose, and death) outweigh the benefits when used for cough in these patients.[122] Stool softeners are used routinely, as straining to defecate can potentially cause rebleeding.

Surgery and coil embolization

A neurosurgeon and interventional neuroradiologist should be involved in the decision about how to treat an aneurysm. Complete obliteration of the ruptured aneurysm is indicated whenever feasible to reduce the risk of rebleeding and retreatment.[37] [123] [124] [125] Most surgeons operate on patients with good neurologic status during the first 72 hours to prevent rebleeding, a practice that also seems to be associated with improved outcome.[126] Treatment should be individualized according to patient-specific factors such as medical comorbidities and pre-hemorrhage functional status, and should incorporate shared decision-making with the family or surrogate decision makers.[37]

AHA/ASA recommends:[37]

- In patients with high-grade aSAH, aneurysm treatment is reasonable, after careful discussion of likely prognosis with family members, to optimize patient outcome
- In patients with aSAH and advanced age, aneurysm treatment is reasonable, after careful discussion of prognosis with family members, to improve survival and outcome.
- In patients with aSAH who do not improve after correction of modifiable conditions and are deemed unsalvageable because of evidence of irreversible neurologic injury, treatment of the aneurysm is not beneficial.

Controversy exists over the choice between surgical clipping and endovascular coil embolization. The ruptured aneurysm should be evaluated by specialist(s) with endovascular and surgical expertise to determine the relative risks and benefits of surgical or endovascular treatment according to patient (e.g., age, neurologic status on admission, comorbid conditions) and aneurysm characteristics (e.g., size and location).[37] The results of a major international prospective and randomized trial have sparked major controversies.[127] [128] [129] The International Subarachnoid Aneurysm Trial (ISAT) included over 1000 patients in each treatment group. At 1 year, 23.7% of patients were dead or dependent following coiling compared with 30.6% in the clipping group.[128] Criticisms of the study included uneven distribution of enrolled patients (almost all came from Europe), differing levels of expertise among the interventionists and surgeons, and enrollment criteria that aneurysms be considered suitable for either surgical or endovascular repair.[127] Long-term follow-up of patients enrolled in ISAT has revealed that despite an increased risk of recurrent bleeding in the coiling group, the 5-year death risk remained significantly lower compared with the clipping group.[130] Another publication assessing long-term follow-up (10 years and beyond) after ISAT concluded that despite a higher risk of rebleeding, the probability of disability-free survival was significantly greater in the endovascular group than in the neurosurgical group.[131]

In surgical clipping, a craniotomy is performed to expose the aneurysm, and a clip is placed on its neck to exclude it from the circulation. Complications of clipping include aneurysm rupture, injury to vascular structures, postoperative stroke, and clipping of arterial perforators. A craniotomy is not needed for endovascular coil embolization. An arterial catheter is advanced to the aneurysm lumen where titanium coils are deposited. A thrombus forms in the lumen, excluding the aneurysm from the circulation. Ongoing technological advances have refined coil embolization of aneurysms, making it a therapeutic option for complex aneurysms that were only amenable to surgical clipping in the past.[132] Yet there remain drawbacks to coil embolization, mainly incomplete embolization and recurrences requiring reintervention.[133] [134] [135] [136] [137] Potential adverse events of the procedure itself are stroke, vessel rupture, and dissection.

For patients with good-grade SAH from ruptured aneurysms of the anterior circulation equally suitable for both primary coiling and clipping, AHA/ASA recommends primary coiling in preference to clipping to improve 1-year functional outcome.[37] [128] [138] However, the guideline notes both treatment options are reasonable in this patient group to achieve a favorable long-term outcome.[37]

AHA/ASA recommends coiling in preference to clipping in patients with aSAH from ruptured aneurysms of the posterior circulation that are amenable to coiling, to improve both short- and long-term outcomes.[37] [138] [139] [140]

Patient age may inform the modality of treatment. For aSAH patients <40 years of age, clipping of the ruptured aneurysm might be considered the preferred mode of treatment to improve durability of the treatment and outcome. Longer life expectancy and better long-term protection from re-rupture favor consideration of clipping in younger patients.[37] [128][141] However, for patients >70 years of age, the superiority of coiling or clipping to improve outcome is not well established.[37] [128] [142]

For patients with aSAH deemed salvageable and with depressed level of consciousness due to large intraparenchymal hematoma, emergency clot evacuation should be performed to reduce mortality.[37]

Venous thromboembolism (VTE) prophylaxis

In patients with aSAH whose ruptured aneurysm has been secured, pharmacologic or mechanical venous (intermittent pneumatic compression) VTE prophylaxis is recommended to reduce the risk for VTE.[37] See Venous thromboembolism (VTE) prophylaxis .

Other investigated therapies

Statins and magnesium sulfate have been investigated for their presumed neuroprotective effect in the treatment of SAH. However, neither has been shown to be beneficial in terms of mortality and clinical outcomes in SAH patients.[113] [143] [144] [145] [146] AHA/ASA recommends against routine use of statin therapy or intravenous magnesium sulfate to improve outcomes.[37]

Endothelin receptor antagonists have not been shown to improve functional outcomes or mortality in meta-analysis of randomized controlled trials.[145] [147] Several organizations strongly recommend against endothelin receptor antagonists based on this lack of benefit and an increased risk of adverse events.[113] The most common adverse effects in phase 3 trials included pulmonary complications related to fluid retention, hypotension, and anemia.[113] However, based on two Japanese phase 3 trials of endovascular coiling and surgical clipping clazosentan has been approved in Japan for the prevention of vasospasm, vasospasm-related cerebral infarction, and ischemic symptoms after aneurysmal SAH.[148] In contrast to most other trials, the investigator found clazosentan resulted in a decrease in

morbidity and mortality with no unexpected safety findings.[148] Endothelin receptor antagonists have not been approved for this indication in the US and Europe.

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)		
all patients		
	1st	stabilization and cardiopulmonary support
	plus	surgical clipping or coil embolization
	plus	nimodipine
	plus	venous thromboembolism (VTE) prophylaxis
	adjunct	anticonvulsants
	plus	stool softeners
■ cough	adjunct	antitussives
■ headache	adjunct	analgesia
■ hyponatremia	adjunct	sodium replacement
	adjunct	fludrocortisone

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

all patients

1st **stabilization and cardiopulmonary support**

» Patients should be admitted to dedicated neurocritical care unit.[37]

» Consciousness level should be assessed using the Glasgow Coma Scale, and need for endotracheal intubation and mechanical ventilation should be established. Blood pressure, heart rate, and respiratory function should be closely monitored. In patients with aSAH and unsecured aneurysm, frequent blood pressure (BP) monitoring and BP control with short-acting medication(s) is recommended to avoid severe hypotension, hypertension, and BP variability.[37] There is insufficient evidence to recommend a particular BP target.[37] Sudden, profound reduction of BP should be avoided.[37] [101]

» In patients who are receiving anticoagulants, emergency reversal with appropriate agents should be performed to prevent rebleeding.[37] Reversal strategies should follow current published standards for life-threatening bleeding.[37] [102] See Anticoagulation management principles .

» Close monitoring and goal-directed treatment of volume status are reasonable to maintain euvolemia.[37] [103] [104] [105] Induction of hypertension and hypervolemia is potentially harmful because of the association with excess morbidity including cerebral edema, hemorrhagic transformation in areas of infarction, reversible leukoencephalopathy, myocardial infarction, and congestive heart failure.[37] [106] [107] [108] [109] [39] Therefore, prophylactic hemodynamic augmentation should not be performed to reduce iatrogenic patient harm.[37] Electrolyte imbalances (e.g., hyponatremia) are common and should be corrected.[37]

plus **surgical clipping or coil embolization**

Treatment recommended for ALL patients in selected patient group

» Surgical or endovascular treatment of the ruptured aneurysm should be performed

Acute

as early as feasible after presentation, preferably within 24 hours of onset, to improve outcome.[37] A neurosurgeon and interventional neuroradiologist should be involved. Most surgeons operate on patients with good neurologic status during the first 72 hours to prevent rebleeding, a practice that also seems to be associated with improved outcome.[126]

» Complete obliteration of the ruptured aneurysm is indicated whenever feasible to reduce the risk of rebleeding and retreatment.[37] [123] [124] [125] Treatment should be individualized according to patient-specific factors such as medical comorbidities and prehhemorrhage functional status and should incorporate shared decision-making with the family or surrogate decision makers.[37]

» AHA/ASA recommends:[37]

- In patients with high-grade aSAH, aneurysm treatment is reasonable, after careful discussion of likely prognosis with family members, to optimize patient outcome
- In patients with aSAH and advanced age, aneurysm treatment is reasonable, after careful discussion of prognosis with family members, to improve survival and outcome.
- In patients with aSAH who do not improve after correction of modifiable conditions and are deemed unsalvageable because of evidence of irreversible neurologic injury, treatment of the aneurysm is not beneficial.

» Controversy exists over the choice between clipping and coil embolization. For patients with SAH, the ruptured aneurysm should be evaluated by specialist(s) with endovascular and surgical expertise to determine the relative risks and benefits of surgical or endovascular treatment according to patient (e.g., age, neurologic status on admission, comorbid conditions) and aneurysm characteristics (e.g., size and location).[37]For patients with good-grade SAH from ruptured aneurysms of the anterior circulation equally suitable for both primary coiling and clipping, AHA/ASA recommends primary coiling in preference to clipping to improve 1-year functional outcome.[37] [128] [138] However, the guideline

Acute

notes both treatment options are reasonable in this patient group to achieve a favorable long-term outcome.[37]

» AHA/ASA recommends coiling in preference to clipping in patients with aSAH from ruptured aneurysms of the posterior circulation that are amenable to coiling, to improve both short- and long-term outcomes.[37] [138] [139] [140] Patient age may inform the modality of treatment. For aSAH patients <40 years of age, clipping of the ruptured aneurysm might be considered the preferred mode of treatment to improve durability of the treatment and outcome. Longer life expectancy and better long-term protection from re-rupture favor consideration of clipping in younger patients.[37] [128] [141] However, for patients >70 years of age, the superiority of coiling or clipping to improve outcome is not well established.[37] [128] [142] For patients with aSAH deemed salvageable and with depressed level of consciousness due to large intraparenchymal hematoma, emergency clot evacuation should be performed to reduce mortality.[37]

» Controversy exists over the choice between clipping and coil embolization. Patient factors that should be taken into account include age, neurologic status on admission, comorbid conditions, and size and location of the aneurysm.

» Complications of clipping include aneurysm rupture, injury to vascular structures, postoperative stroke, and clipping of arterial perforators.

» Complications of coiling include stroke, vessel rupture, and dissection, and incomplete embolization, and recurrences requiring reintervention.[133] [134] [135] [136] [137]

plus **nimodipine**

Treatment recommended for ALL patients in selected patient group

Primary options

» nimodipine: 60 mg orally/nasogastrically every 4 hours for 21 days

» Early initiation of enteral nimodipine (a calcium-channel blocker) is beneficial in preventing vasospasm, delayed cerebral ischemia and improving functional outcomes in patients with aneurysmal SAH.[37]

Acute

plus **venous thromboembolism (VTE) prophylaxis**

Treatment recommended for ALL patients in selected patient group

» In patients with aneurysmal SAH whose ruptured aneurysm has been secured, pharmacologic or mechanical venous (intermittent pneumatic compression) VTE prophylaxis is recommended to reduce the risk for VTE.[37] Follow your local anticoagulation protocols. See Venous thromboembolism (VTE) prophylaxis

adjunct **anticonvulsants**

Treatment recommended for SOME patients in selected patient group

» Prophylactic use of anticonvulsants following SAH is controversial.[114] [115] US guidelines suggest that prophylactic anticonvulsants may be considered in patients with SAH and high-seizure-risk features (i.e., ruptured middle cerebral artery aneurysm, high-grade SAH, intracranial hemorrhage, hydrocephalus, and cortical infarction).[37] These guidelines recommend against the routine use of anticonvulsants in patients with SAH without high-seizure-risk features since phenytoin for seizure prophylaxis is associated with excess morbidity and mortality.[37] [88] [116] The risk of seizures is significantly lower after coil embolization than following surgical clipping of aneurysm.[37] Short-course treatment may be adequate prophylaxis, and some evidence suggests that it is better tolerated than a longer course.[87] [117] Routine long-term use of anticonvulsants is not recommended, but may be considered for patients with known risk factors for delayed seizure disorder.[37] In patients with SAH who present with seizures, treatment with anticonvulsants for ≤7 days is reasonable to reduce seizure-related complications in the perioperative period.[37] In patients with SAH without prior epilepsy who present with seizures, treatment with anticonvulsants beyond 7 days is not effective for reducing future SAH-associated seizure risk.[37] [39][114]

» There is some evidence that in the US levetiracetam may be the most commonly prescribed agent for the prevention of seizures following SAH.[118] One small prospective study comparing levetiracetam with phenytoin for seizure prophylaxis after neurologic injury (including SAH) found the same outcomes with respect to mortality and seizure control,

Acute

but levetiracetam-treated patients experienced better long-term functional outcomes than those treated with phenytoin (as evaluated by the Glasgow Outcome Scale-Extended and Disability Rating Scale).[119]

plus **stool softeners**

Treatment recommended for ALL patients in selected patient group

» Stool softeners to prevent straining can reduce the risk of rebleeding. There are many available, including docusate and senna.

cough **adjunct** **antitussives**

Treatment recommended for SOME patients in selected patient group

Primary options

» codeine sulfate: 15-30 mg orally every 4-6 hours when required, maximum 120 mg/day

» Cough suppression can help prevent rebleeding. An antitussive agent such as codeine may be given. Cough and cold medications that include opioids, such as codeine or hydrocodone, should not be used in children aged 18 years or younger as the risks (slowed or difficult breathing, misuse, abuse, addiction, overdose, and death) outweigh the benefits when used for cough in these patients.[122]

» Opioids may cause respiratory depression and have the potential for addiction, abuse, and misuse. Opioids may be used for headaches in patients with SAH so it is important to take this into account when considering whether to prescribe opioids for cough suppression.

headache **adjunct** **analgesia**

Treatment recommended for SOME patients in selected patient group

Primary options

» acetaminophen: oral: 325-1000 mg orally every 4-6 hours when required, maximum 4000 mg/day; intravenous (<50 kg body weight): 15 mg/kg intravenously every 6 hours when required, maximum 75 mg/kg/day; intravenous (≥50 kg body weight): 1000 mg intravenously every 6 hours when required, maximum 4000 mg/day

Secondary options

Acute

» codeine sulfate: 15-60 mg orally every 4-6 hours when required, maximum 360 mg/day

OR

» tramadol: 50-100 mg orally (immediate-release) every 4-6 hours when required, maximum 400 mg/day

Tertiary options

» morphine sulfate: 10-30 mg orally (immediate-release) every 4 hours when required initially, adjust dose according to response; 2.5 to 10 mg subcutaneously/intramuscularly/intravenously every 2-6 hours when required

OR

» oxycodone: 5-15 mg orally (immediate-release) every 4-6 hours when required initially, adjust dose according to response

» Analgesia should be provided to conscious patients.

» Acetaminophen can be used as a first-line option.[39] For severe pain, opioids such as codeine or tramadol should be given.[39] If a patient is still in pain, morphine or oxycodone may be required.[39] Aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided before aneurysm occlusion.[39]

» Opioids may cause respiratory depression and have the potential for addiction, abuse, and misuse. Opioids may be used for cough suppression in patients with SAH so it is important to take this into account when considering whether to prescribe opioids.

» Mental status also needs to be closely monitored, especially in patients at risk of acute hydrocephalus or vasospasm. Judicious use of analgesia is therefore recommended.

hyponatremia **adjunct** **sodium replacement**

Treatment recommended for SOME patients in selected patient group

» Electrolyte imbalances (e.g., hyponatremia) are common and should be corrected.[37] See Hyponatremia

»

adjunct **fludrocortisone**

Acute

Treatment recommended for SOME patients in selected patient group

Primary options

» fludrocortisone: consult specialist for guidance on dose

» The American Heart Association states that use of fludrocortisone is reasonable to treat natriuresis and hyponatremia, as supported by several RCTs.[37] However, although fludrocortisone use reduces excess sodium excretion, urine volume, and intravenous fluid use in patients with SAH, it has not been found to consistently affect outcome.[37] [110] [111] [112][113] Some guidelines state there is insufficient evidence to support its use in maintaining normal serum sodium concentrations or improving functional outcome.[113]

Primary prevention

One-quarter of patients with autosomal dominant polycystic kidney disease (ADPKD) have aneurysms at autopsy, and 2% to 8% of patients with aneurysms have ADPKD.[36] Individuals with ADPKD are potential candidates for aneurysm screening.[29]

Because of the possible hereditary aspects of SAH, patients who have two or more first-degree relatives with SAH are potential candidates for aneurysm screening.[25] [29] [35]

Hypertension should be corrected, as this is one of the most important modifiable risk factors.[26] [29] [30] [31] [32] [33] [34]

Smoking is another important modifiable risk factor.[26] [30] [31] [32] [33] [34] All patients should be encouraged not to smoke.

Secondary prevention

Advise patients on lifestyle measures including recommendations to:[102]

- Exercise regularly
- Maintain a healthy diet
- Manage weight
- Reduce alcohol consumption
- Quit smoking

Patient discussions

The patient is usually instructed to take it easy for 2-4 weeks after discharge (in particular, no lifting) and slowly return to normal life pace (based on expert opinion).

The patient is told to seek urgent medical attention if he or she experiences sudden, severe headache or weakness, numbness on one side of the body, slurred speech, double vision, vision loss, or difficulty swallowing. Counseling patients and caregivers on the high long-term risk of cognitive dysfunction can be beneficial to identify long-term needs.[37]

Monitoring

Monitoring

A plan for follow up care and multidisciplinary rehabilitation after SAH should be developed and shared with the patient and their caregiver.

Follow-up neuroimaging should be considered for patients who have had an aneurysmal subarachnoid hemorrhage.[67] The choice of imaging modality and the frequency and duration of follow-up should be tailored to the individual patient and will usually be based on the type and outcome of any neurointervention or surgery on the initial aneurysm, the presence of any non-culprit aneurysm, estimated risk of further bleeding, risks of planned investigations and any subsequent interventions, and patient preference.[67]

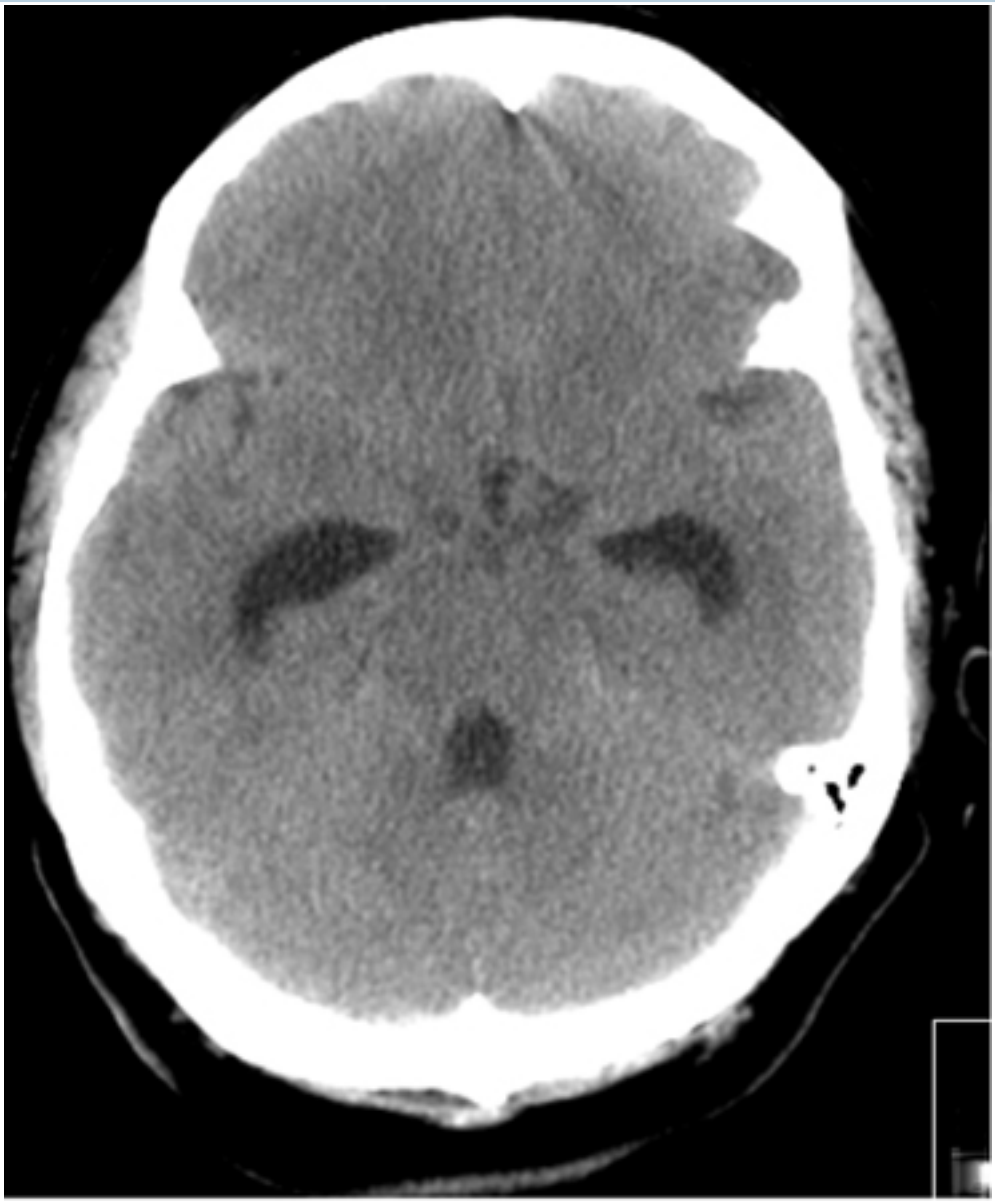
Patients who have undergone coiling or clipping of a ruptured aneurysm should always have delayed follow-up vascular imaging to screen for the recurrence or regrowth of the treated aneurysm, or the development of new aneurysms.[37] Retreatment may be considered if there is a clinically significant (e.g., growing) remnant.[37] In patients with untreated, unruptured intracranial aneurysms, follow-up with a neurosurgeon/endovascular specialist is warranted to gauge the possible growth of aneurysm(s).

FOLLOW UP

Complications

Complications	Timeframe	Likelihood
rebleeding	**short term**	**high**
Rebleeding is an important complication. The incidence is 5.7% during the first 72 hours, and cumulative risk approaches 22% at 1 month after SAH.[11] [12] Patients with modified Fisher grade bleeding of III and IV are more likely to rebleed.[94] [95] [175] Rebleeding is responsible for 8% to 22% of mortality and is significantly associated with poor outcome.[11] [12] [49] Routine use of antifibrinolytic therapy does not improve functional outcome.[37] [113] Antifibrinolytic therapy is associated with increased risk of cerebral ischemia.[176][177] Conversely, ultra-early administration of antifibrinolytic therapy might reduce the risk of rebleeding, but this effect has not been consistent across trials.[37] [177] [178] [179] Prompt obliteration of the ruptured aneurysm is the only treatment proven to be effective to reduce the likelihood of rebleeding and thus early mortality.[37]		
acute hydrocephalus	**short term**	**high**
Acute hydrocephalus (HCP) occurs in 15% to 20% of patients during the first 72 hours and is an obstructive HCP.[11] [12] [180] Its occurrence is related to presence of intraventricular blood and, to a lesser extent, thick cisternal blood collection.[180] [181] The mortality rate in SAH patients with HCP is higher than in those without it.[180] In patients with aSAH and acute symptomatic hydrocephalus, urgent cerebrospinal fluid (CSF) diversion (external ventricular drain [EVD] and/or lumbar drainage) should be performed.[37] The use of prophylactic antibiotics with EVD is not established but is commonly adopted.[60] The optimum modes of CSF drainage (intermittent vs. continuous), EVD weaning (rapid vs. gradual), and EVD wean timing (following aneurysm securement vs. time of vasospasm risk) are uncertain.[7] [113][182] It is unclear whether microsurgical fenestration of the lamina terminalis during surgical clipping of the aneurysm protects against the need for a permanent shunt in patients with hydrocephalus.[183]		

Complications	Timeframe	Likelihood



Communicating hydrocephalus in the setting of subarachnoid hemorrhage; note dilation of fourth and temporal horns of lateral ventricles
Courtesy of Dr Salah Keyrouz; used with permission

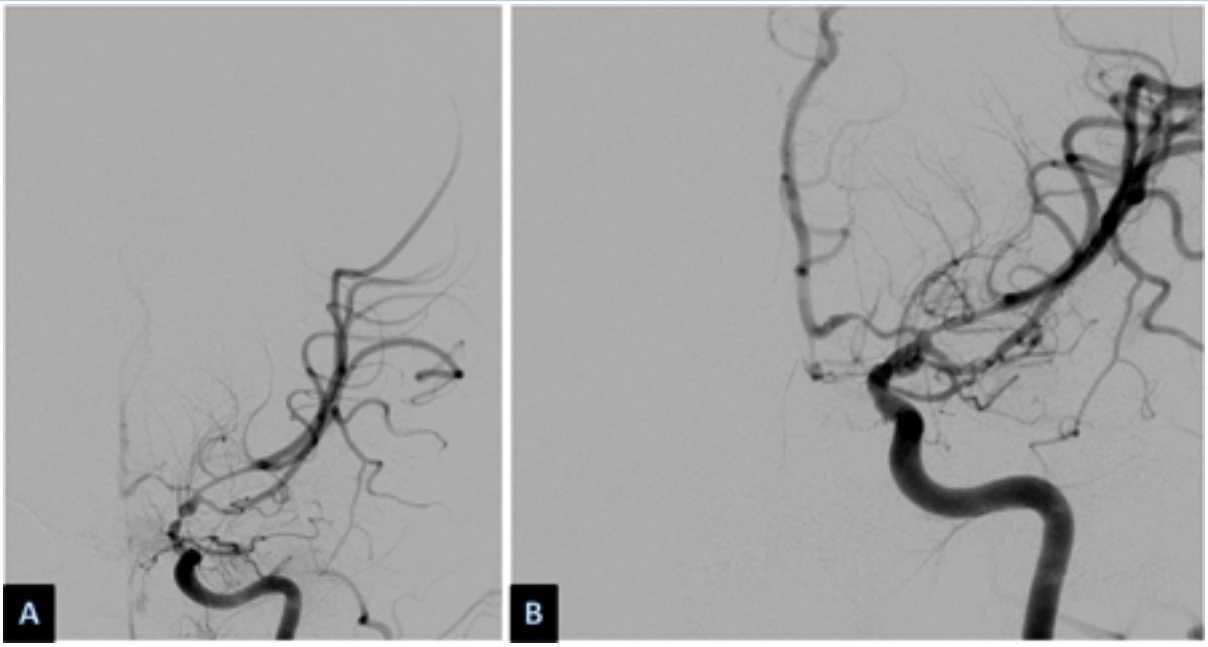
vasospasm	**short term**	**high**

Vasospasm is a delayed, focal, or diffuse narrowing of large capacitance vessels of the circle of Willis. It accounts for 23% of deaths related to SAH.[49] The pathophysiology of vasospasm is poorly understood, a fact compounded by the use of varying nomenclature and definitions; however, it is believed to result from a delayed and reversible vasculopathy, impaired autoregulatory function, and hypovolemia, culminating in a global or regional reduction of cerebral perfusion, which when below a certain threshold causes ischemia.[185] In addition, low hemoglobin may impair oxygen delivery to brain regions with precarious perfusion, further compounding this problem.[186] [187] [188] [189] [190] Presence of oxyhemoglobin in the subarachnoid space seems to be necessary, and an inflammatory response is implicated in the pathogenesis.[191] [192] [193]

Vasospasm develops between days 4 and 14 after SAH and is seen on angiography in 50% to 70% of cases. Half of these patients develop delayed cerebral ischemia (DCI) secondary to reduced regional or

Complications	Timeframe	Likelihood

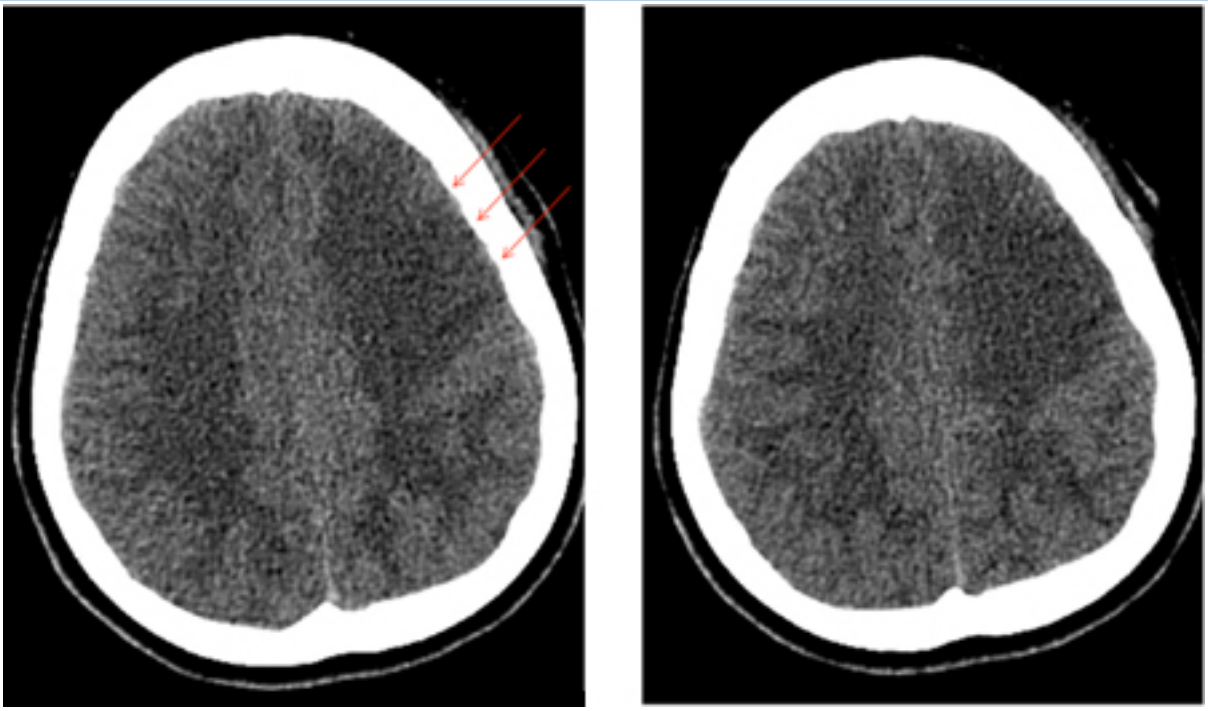
overall cerebral blood flow (CBF).[194] If untreated, DCI progresses to permanent cerebral infarction in 50% of cases. Ischemic deficits may also be seen in the absence of discrete angiographic vasospasm. This is believed to be due, in part, to altered autoregulation of distal cerebral vessels, microthrombi in such vessels, and/or cortical spreading depolarization.[195] Risk factors for DCI are a poor clinical condition on admission, quantity and duration of exposure to subarachnoid blood, thick blood collections in cisterns and fissures, intraventricular blood, and duration of unconsciousness.[176] [181] [196] [197] [198] Although the presence of blood in the subarachnoid space is necessary to the development of vasospasm, surgical clipping, during which most of the subarachnoid blood is washed out, does not seem to carry a lesser risk of vasospasm than endovascular coiling.[199] [200]



Severe vasospasm of distal left internal carotid artery and proximal middle and anterior cerebral arteries before (A) and after (B) intra-arterial infusion of nicardipine and transluminal balloon angioplasty
Courtesy of Dr Salah Keyrouz; used with permission

Complications

Timeframe Likelihood



Left frontal infarct (arrows) in a patient with subarachnoid hemorrhage-related vasospasm
Courtesy of Dr Salah Keyrouz; used with permission

The diagnosis of DCI is a clinical one made after excluding rebleeding, hydrocephalus, seizures, electrolyte imbalances, and other metabolic disturbances. Clinically, patients develop alteration of consciousness or acute to subacute fluctuating focal neurologic deficits.[11] [12] [192]

However, the diagnosis can be challenging, and although serial neurologic exams are important, they are of limited value in patients with high-grade aSAH.[37] In patients with suspected vasospasm or limited neurologic exam, CTA or CT perfusion (CTP) can be useful to detect vasospasm and predict DCI.[37] CTP is noninvasive and, most importantly, measure perfusion, not merely arterial diameter or flow velocities.[201] CT angiography (CTA) and CTP have shown excellent accuracy in diagnosing vasospasm. CTA vasospasm scores are direct predictors of DCI and poor neurologic outcome, and CTP allows early prediction of perfusion abnormalities.[37] However, these CT imaging techniques do not allow for therapeutic intervention (e.g., transluminal balloon angioplasty and intra-arterial vasodilators) in the same way that angiography permits.[202] CTA is highly correlated to conventional angiography for larger proximal intracranial vessels with decreasing correlation in the smaller more distal arteries.[67] [203] CTA shows high sensitivity (91%) for detecting central vasospasm when symptoms develop.[37] [204] Transcranial Doppler (TCD) ultrasound monitoring is a noninvasive, safe bedside neuromonitoring technique that allows repetitive and dynamic assessment of vasospasm after SAH.[37] It is reasonable to use TCD to detect vasospasm and predict DCI but it is less reliable than angiography.[37] [205] [206] TCD is operator dependent, can be limited by patient anatomy (poor temporal bone window), and can be affected by other physiologic measures (such as heart rate and BP).[37] [207] [208] In patients with high-grade aSAH, continuous EEG (cEEG) monitoring can be useful to predict DCI.[37] [209]

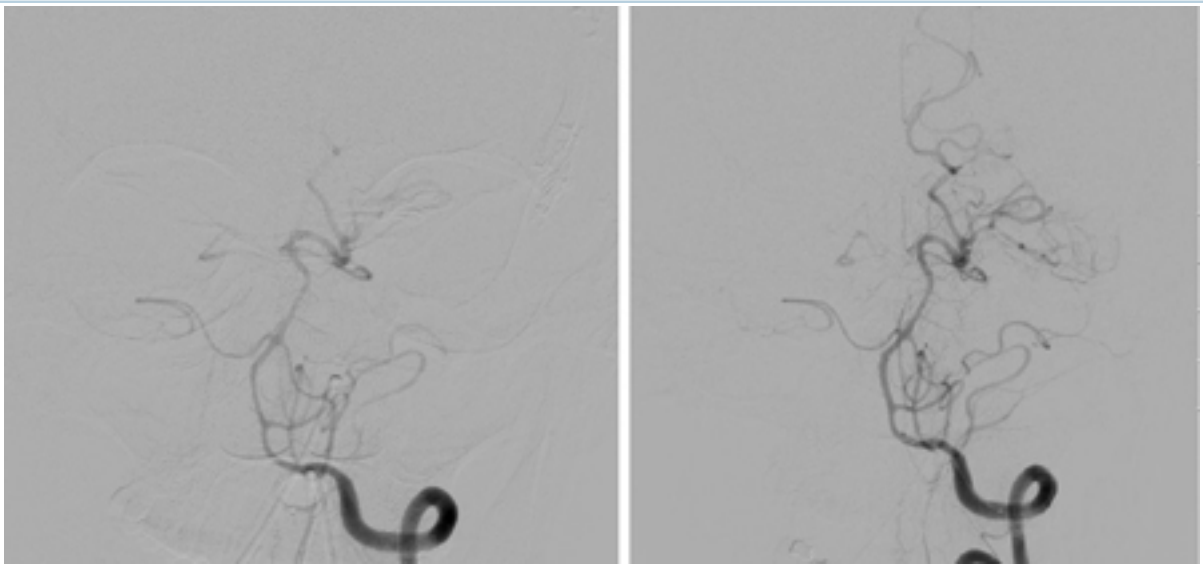
In patients without symptomatic vasospasm, induction of hypertension and hypervolemia is potentially harmful because of the association with excess morbidity including cerebral edema, hemorrhagic transformation in areas of infarction, reversible leukoencephalopathy, myocardial infarction, and congestive heart failure.[37] [39][106] [107] [108] [109] Instead, maintaining euvolemia can be beneficial in preventing DCI and improving functional outcomes.[37] However, in patients with SAH and symptomatic vasospasm, elevating systolic BP values may be reasonable to reduce the progression and severity of DCI.[37] [210] [211] Symptomatic patients should be kept hypervolemic (central venous pressure ≥8 cm H_2O) and hypertension induced using vasopressors. Isotonic fluids are used. Hypertension should be titrated to a mean arterial pressure (MAP) of at least 15% higher than the patient's average MAP, until clinical

Complications | Timeframe | Likelihood

improvement or adverse effects occur. Clinicians should be aware of the possibility of posterior reversible encephalopathy syndrome development in the context of increased blood pressure. The diagnosis is usually heralded by clinical deterioration and confirmed by magnetic resonance imaging.[212]

Triple H (hypertension, hypervolemia, and hemodilution), also known as hyperdynamic or hypervolemic hypertensive therapy (HHT), is safe, even in patients with prior cardiac disease.[194] Clinical improvement can be dramatic, but large prospective outcome studies of HHT are lacking.[108] A systematic review of uncontrolled studies has suggested that hypertension seems to be more effective in increasing CBF than hemodilution or hypervolemia.[213] Randomized controlled studies are difficult to conduct, given the multiplicity of factors that affect outcome in SAH.

Endovascular techniques such as transluminal balloon angioplasty and intra-arterial vasodilators will reverse arterial narrowing, though clinical improvement is not consistent.[214] [215] ASA/AHA recommends that in patients with aSAH and severe vasospasm, cerebral angioplasty may be reasonable to reverse cerebral vasospasm and reduce the progression and severity of DCI.[37] There is some evidence to support early angioplasty (within 2 hours of symptom onset) in providing sustained clinical improvement.[216] Increased age and poor neurologic status at presentation are predictive of poor clinical outcome after angioplasty.[217] Enteral nimodipine, a calcium-channel antagonist, is given for vasospasm prophylaxis.[37] It reduces risk of poor outcome and secondary ischemia after aneurysmal SAH.[218] [219] Despite their wide use, studies of corticosteroids in SAH have failed to show positive effects on DCI or overall outcome.[220] Tirilazad, a nonglucocorticoid 21 amino-steroid free-radical scavenger, was studied in several controlled trials for prevention of vasospasm. It was well tolerated but had inconsistent effect on overall outcome across the different studies.[221] [222] [223] [224] [225] Studies have investigated the use of simvastatin and pravastatin in SAH with mixed results.[226] [227] [228] [229] Routine use of statin therapy to improve outcomes is not recommended.[37] Studies investigating the use of antiplatelet agents in SAH, especially following endovascular coiling, have yielded conflicting results on the risk of DCI and overall outcome. Those conflicting findings, together with an increased risk of hemorrhagic complications, have resulted in less than universal adoption of this practice.[230] [231]



Distal left vertebral and basilar arteries spasm before (left) and after (right) intra-arterial Infusion of nicardipine
Courtesy of Dr Salah Keyrouz; used with permission

The effect on vasospasm and DCI of pharmacologic compounds with controlled-release forms has drawn much interest. Nicardipine prolonged-release implants are placed in the subarachnoid space at the time of surgical clipping of aneurysm. Small case series and a randomized double-blind trial using such implants have reported a lower than expected incidence of DCI.[232] [233] [234] [235]

Other drugs used in an intracranial, controlled-release system include papaverine, fasudil, and nitric oxide donors.[236] Larger controlled trials are needed before widespread use of this technique. Papaverine, although a historically effective vasodilator, is generally avoided because of the risk of neurotoxicity.[37]

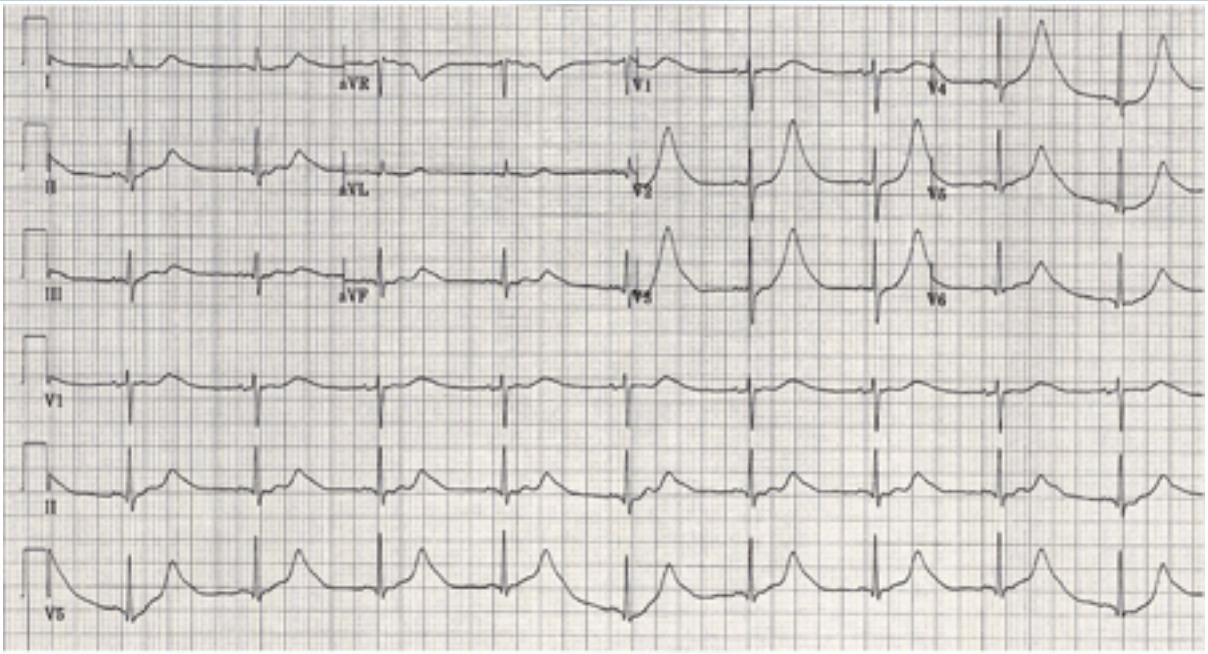
Complications	Timeframe	Likelihood

[237] The use of thrombolytic agents instilled during surgery and/or after securing the aneurysm into the intrathecal space could potentially be beneficial. They rid the subarachnoid space of blood necessary for the development of vasospasm. A meta-analysis concluded that while such compounds could improve outcome, the analyzed studies had limitations, including considerable risk of bias.[238] Another intervention that rids the subarachnoid space of blood is lumbar drainage (LD) of cerebral spinal fluid. Trials of LD show a benefit on the incidence of vasospasm and DCI, but no effect on functional outcomes or mortality at 6 months.[239] Studies of other forms of intracranial delivery of nicardipine (intrathecal, intraventricular and intracisternal) have yielded mixed results. In the absence of positive randomized controlled trials, these alternative forms of intracranial delivery are reserved for severe cases of vasospasm and DCI where other therapies have failed.[235]

It is unknown whether angiographic, asymptomatic vasospasm needs to be treated.[240] Except for one retrospective study using albumin, prophylactic volume expansion in asymptomatic patients has failed to show an impact on outcome.[107] [108][241]

cardiac abnormalities	short term	high

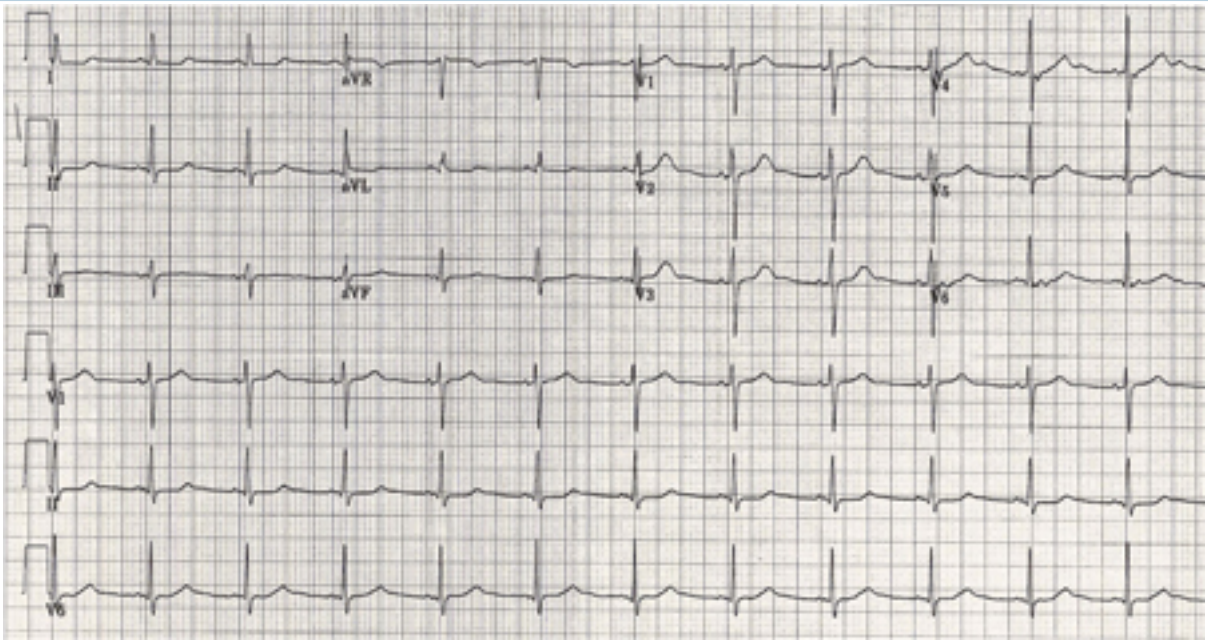
Arrhythmias and nonspecific ECG changes are common.[44] [49] [50] [51] Ventricular wall motion abnormalities occur in 27% of patients. An apex-sparing pattern is seen in half of affected patients. These abnormalities rarely cause cardiac failure and are almost always reversible.[47] [242] [243]



ECG done on admission of a patient with subarachnoid hemorrhage; note peaked, tall T waves (1 of 2)
Courtesy of Dr Salah Keyrouz; used with permission

Treatment of cardiac failure is supportive. Beta-blockers can be administered for ischemic-type ECG changes accompanied by troponin leaks; however, there is no evidence in support of this practice.

Complications	Timeframe	Likelihood



Same patient, 24 hours later; note normalization of T waves (2 of 2)
Courtesy of Dr Salah Keyrouz; used with permission

pulmonary edema	short term	high

Pulmonary edema occurs in up to 23% of patients.

Pulmonary edema is severe, requiring ventilatory support in 6% of cases.

hyperglycemia	short term	high

It is debated whether it is an independent predictor of outcome or is merely associated with severity of SAH.[244] [245] [246] [247]

Hyperglycemia on admission, during aneurysm surgery, or within 72 hours of SAH presentation has been associated with vasospasm, delayed cerebral ischemia, unfavorable short-term and long-term functional outcomes, and risk of death in both patients with diabetes and those without diabetes in multiple studies.[37] [247] [248] [249] [250] AHA/ASA recommend that in patients with SAH, effective glycemic control, strict hyperglycemia management, and avoidance of hypoglycemia are reasonable to improve outcome.[37] However, data are conflicting on what glycemic threshold should be targeted, what monitoring and treatment intensities should be used, and whether all these affect outcome.[37] It remains to be determined whether tight glycemic control could lead to systemic or cerebral hypoglycemia and metabolic crisis in the acutely injured brain and potentially worsen brain injury and outcome.[37] Until more robust evidence exists for treating hyperglycemia in SAH, one should refrain from some aggressive management strategies (i.e., continuous insulin infusion) used in critically ill patients.[251]

seizure	short term	medium

Recent studies using EEG monitoring suggest a seizure incidence of 7.8% to 15.2%.[37] [170] [171][172] The cumulative incidence of epilepsy (defined as 2 or more unprovoked seizures) is 12% at 5 years.[173] It is independently associated with poor functional recovery and quality of life.[172] [174] The risk of seizures is significantly lower after coil embolization than following surgical clipping of aneurysm.[37]

Risk factors for the development of early seizures associated with SAH include clinical grade (Hunt and Hess [HH] grade ≥3), presence of a middle cerebral artery aneurysm, and hydrocephalus.[37] Continuous

Complications	Timeframe	Likelihood
EEG (cEEG) monitoring is reasonable to detect seizures in patients with SAH and either fluctuating neurologic exam, depressed mental state, ruptured middle cerebral artery (MCA) aneurysm, high-grade SAH, intracranial hemorrhage (ICH), hydrocephalus, or cortical infarction.[37] Prophylactic use of anticonvulsants following SAH is controversial.[114] [115] US guidelines suggest that prophylactic anticonvulsants may be considered in patients with SAH and high-seizure-risk features (i.e., ruptured MCA aneurysm, high-grade SAH, ICH, hydrocephalus, and cortical infarction).[37] These guidelines recommend against the routine use of anticonvulsants in patients with SAH without high-seizure-risk features since phenytoin for seizure prevention and/or seizure prophylaxis is associated with excess morbidity and mortality.[37] [88] [116] Short-course treatment may be adequate prophylaxis, and some evidence suggests that it is better tolerated than a longer course.[87] [117] Routine long-term use of anticonvulsants is not recommended, but may be considered for patients with known risk factors for delayed seizure disorder.[37] In patients with SAH who present with seizures, treatment with anticonvulsants for ≤7 days is reasonable to reduce seizure-related complications in the perioperative period.[37] In patients with SAH without prior epilepsy who present with seizures, treatment with anticonvulsants beyond 7 days is not effective for reducing future SAH-associated seizure risk.[37] [39] [114]		
fever	**short term**	**medium**
Fever in the neurologic intensive care unit (NICU) appears to be an independent predictor of poor outcome.[37] It is associated with longer ICU stay and overall length of hospital stay.[252] Intraventricular catheterization is a risk factor for unexplained fever in the NICU. SAH increases the risk of developing infectious and unexplained fever, which could be associated with a worse outcome.[253] [254] An infectious etiology should always be sought. Blood, sputum, urine, and cerebrospinal fluid (if applicable) samples should be obtained for Gram staining and culture. If fever occurs, antibiotics should be withheld until there is clear evidence of an infection. In patients with SAH with fever refractory to antipyretic medications, the effectiveness of therapeutic temperature management (TTM) such as pharmacological treatment, surface cooling devices with or without a feedback loop, and endovascular cooling devices during the acute phase of aSAH is uncertain.[37] [255] [256] It is reasonable to consider achieving normothermia during active or ongoing brain injury, while considering the consequences of TTM. TTM can be associated with complications such as shivering requiring pharmacologic control. Shivering should be monitored and managed to limit risk of secondary injury.[37] [257]		
neuropsychiatric problems	**long term**	**high**
Over 50% of survivors report cognitive impairment (e.g., mood and memory problems), resulting in a negative impact on functional status, emotional health, and quality of life.[160] [161] Depression can occur in about one-third of aSAH survivors; anxiety and post-traumatic stress disorder can be seen in 15% to 20% of patients.[258] [259] [260] Use of validated grading scores or patient-reported outcome measures prior to hospital discharge is recommended to screen for physical, cognitive, behavioral, and quality of life deficits.[37] Use of validated screening tools in the postacute period is recommended to identify cognitive dysfunction; it is reasonable to choose the Montreal Cognitive Assessment (MoCA) over the Mini-Mental Status Examination (MMSE) to identify cognitive impairment.[37] In patients with depression, psychotherapy and pharmacotherapy are recommended to reduce symptoms.[37]		
death and disability	**long term**	**high**
In-hospital mortality is around 20%, with all-cause mortality increasing to 25% at 6 months after SAH. In one study, 35% of patients were “dead or disabled” (defined as modified Rankin Scale ≥4) at 6 months.[159]		

FOLLOW UP

Complications	Timeframe	Likelihood
chronic hydrocephalus	**long term**	**medium**
Chronic hydrocephalus is a well-known complication after aneurysmal SAH, occurring in >20% of patients.[184] In patients with aSAH and associated chronic symptomatic hydrocephalus, permanent CSF diversion is recommended to improve neurologic outcome.[37] Routine fenestration of the lamina terminalis is not indicated for reducing the rate of shunt dependency.[37] [183]		

Prognosis

Advances in operative and endovascular techniques and postoperative critical care have led to a decrease in case-fatality rate over the past 3 decades.[149] This may be related to improved diagnosis and improved surgical and medical management. The emergence of neurocritical care units may have contributed to better SAH outcomes.[150] [151] However, overall outcome in SAH is still poor.[152] [153] SAH is responsible for about one third of premature deaths related to stroke.[154] Mortality is slightly greater in black patients and women. Causes of early mortality are initial hemorrhage (19%), rebleeding (22%), vasospasm (23%), and medical complications (23%).[49] [155] Older age, level of responsiveness on admission (measured by the World Federation of Neurological Surgeons [WFNS] Scale), and volume of subarachnoid blood are powerful predictors of 30-day mortality.[1] [121] In patients undergoing clipping, perioperative and immediate postoperative neurologic injury could account for a percentage of poor outcomes in those with good admission WFNS grades.[156]

Medical complications account for one-quarter of deaths in SAH.[44] Forty percent of patients will have at least 1 medical complication during the first 3 months after SAH.[11] [12] [49] [157] Cardiac and respiratory complications are most frequent. The presence of cardiac wall motion abnormality seem to portend a worse overall outcome after SAH.[158] Some degree of hepatic dysfunction is seen in 24% (4% with severe hepatic dysfunction).[49] Renal failure (1.4%), anemia (5%), thrombophlebitis (1.4%), and pulmonary embolism (0.8%) are also complications encountered during hospitalization.[11] [12]

In-hospital mortality from SAH is around 20%, with all-cause mortality increasing to 25% at 6 months. In one study, 35% of patients were “dead or disabled” (defined as modified Rankin Scale ≥4) at 6 months.[159] Over 50% of survivors report problems with memory, mood, and other cognitive impairment, resulting in negative impact on functional status, emotional health, and quality of life.[160] [161] At 6 months, 75% of patients who were alert on admission had a good recovery, whereas only 11% of those comatose on admission survived.[11] [12] Late epilepsy (measured at 12 months after SAH) is seen in 4% to 5%.[162] [163] More than 40% of patients who die after SAH have extracerebral organ system dysfunction, which is an independent predictor of outcome.[164] [165] This identifies organ system dysfunction, other than the brain, as a potential therapeutic target that might have a positive effect on outcome. The frequency of aneurysm repairs in a hospital (more than 30 craniotomies for aneurysm repair per year) and the presence and use of endovascular therapy are independently associated with better outcome after SAH.[166] [167] [168] This led some to justify regionalization of SAH treatment.[169]

Diagnostic guidelines

International

Guideline for the management of patients with aneurysmal subarachnoid hemorrhage (https://professional.heart.org/en/guidelines-and-statements) [37]

Published by: American Heart Association; American Stroke Association **Last published:** 2023

ACR Appropriateness Criteria®: headache (https://www.acr.org/Clinical-Resources/ACR-Appropriateness-Criteria) [52]

Published by: American College of Radiology **Last published:** 2022

ACR Appropriateness Criteria®: cerebrovascular diseases - aneurysm, vascular malformation, and subarachnoid hemorrhage (https://www.acr.org/Clinical-Resources/ACR-Appropriateness-Criteria) [67]

Published by: American College of Radiology **Last published:** 2021

Guidelines for the management of patients with unruptured intracranial aneurysms: a guideline for healthcare professionals (https://professional.heart.org/en/guidelines-and-statements) [25]

Published by: American Heart Association; American Stroke Association **Last published:** 2015

Treatment guidelines

International

Guidelines for the management of aneurysmal subarachnoid hemorrhage (https://professional.heart.org/en/guidelines-and-statements) [37]

Published by: American Heart Association; American Stroke Association **Last published:** 2023

Guidelines for the management of neurocritical care management of aneurysmal subarachnoid hemorrhage (https://www.neurocriticalcare.org/Resources-Publications/Neurocritical-Care-Guidelines) [35]

Published by: Neurocritical Care Society **Last published:** 2023

Guidelines on management of unruptured intracranial aneurysm (https://eso-stroke.org/eso-guideline-directory) [35]

Published by: European Stroke Organisation **Last published:** 2023

Key articles

- Thompson BG, Brown RD Jr, Amin-Hanjani S, et al. Guidelines for the management of patients with unruptured intracranial aneurysms: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2015 Aug;46(8):2368-400. Full text (https://www.doi.org/10.1161/STR.0000000000000070) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/26089327?tool=bestpractice.bmj.com)

- Hoh BL, Ko NU, Amin-Hanjani S, et al. 2023 Guideline for the management of patients with aneurysmal subarachnoid hemorrhage: a guideline from the American Heart Association/American Stroke Association. Stroke. 2023 Jul;54(7):e314-70. Full text (https://www.ahajournals.org/doi/full/10.1161/STR.0000000000000436?rfr_dat=cr_pub++0pubmed&url_ver=Z39.88-2003&rfr_id=ori%3Arid%3Acrossref.org) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/37212182?tool=bestpractice.bmj.com)

- Steiner T, Juvela S, Unterberg A, et al. European Stroke Organization guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. Cerebrovasc Dis. 2013 Feb 7;35(2):93-112. Full text (https://www.karger.com/Article/FullText/346087) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/23406828?tool=bestpractice.bmj.com)

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Images

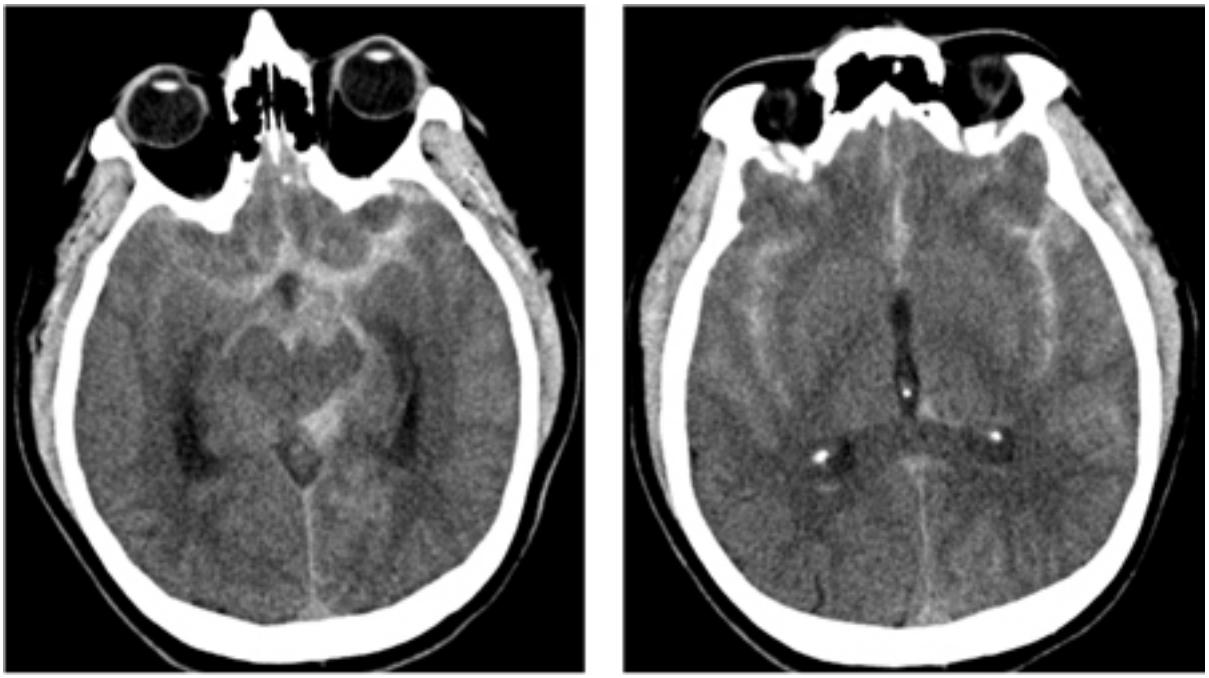

Figure 1: CT brain showing subarachnoid hemorrhage from a ruptured posterior cerebral artery aneurysm (1 of 2)

Courtesy of Dr Salah Keyrouz; used with permission

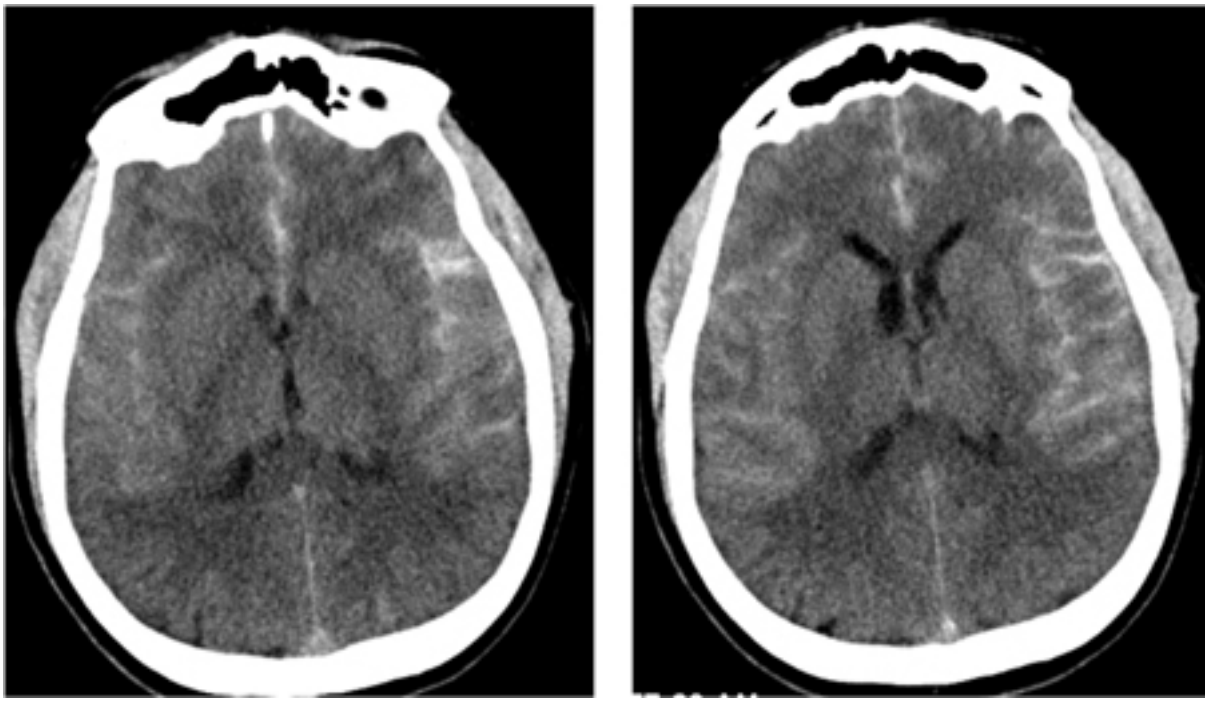

Figure 2: CT brain showing subarachnoid hemorrhage from a ruptured posterior cerebral artery aneurysm (2 of 2)

Courtesy of Dr Salah Keyrouz; used with permission

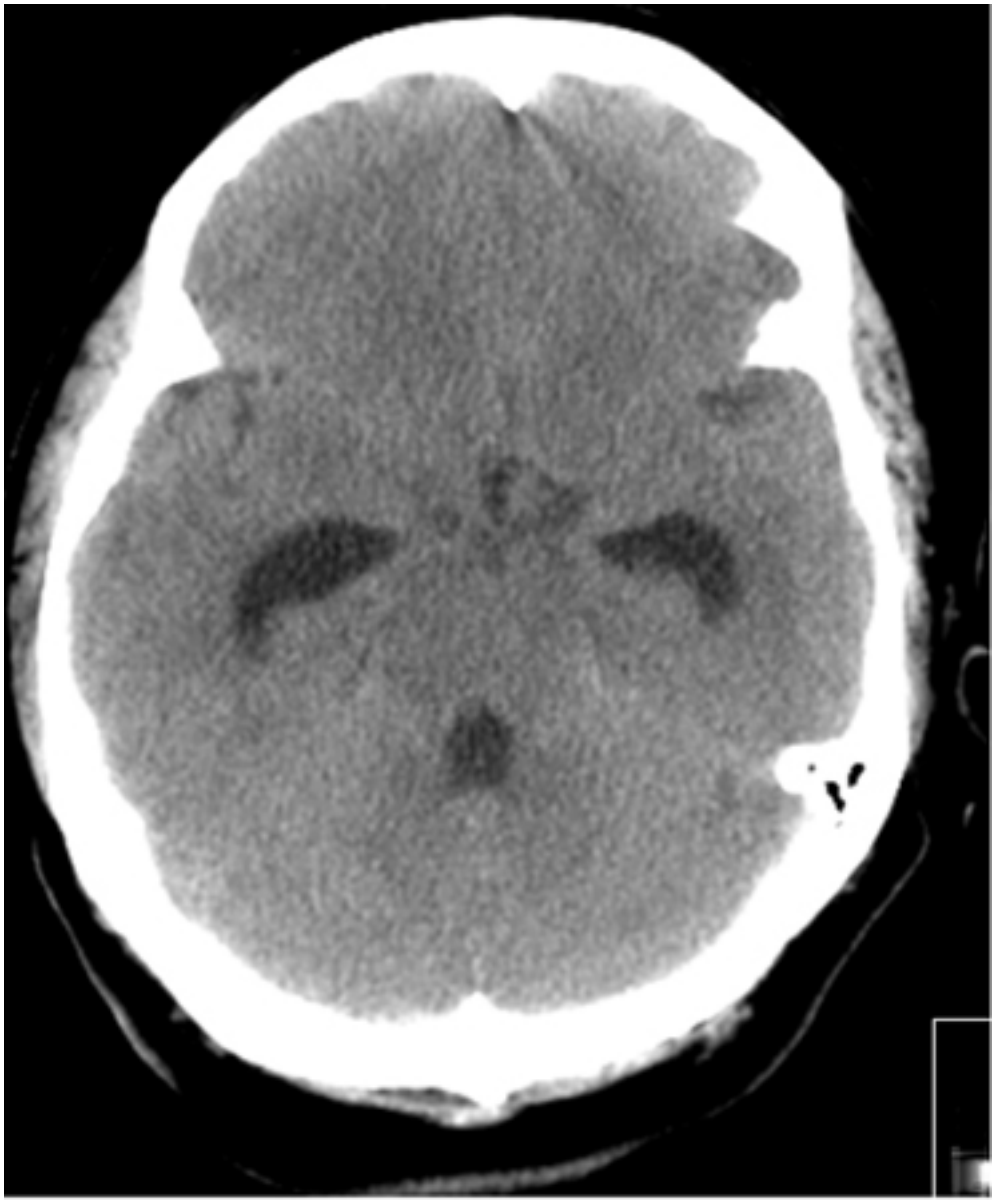

Figure 3: Communicating hydrocephalus in the setting of subarachnoid hemorrhage; note dilation of fourth and temporal horns of lateral ventricles

Courtesy of Dr Salah Keyrouz; used with permission

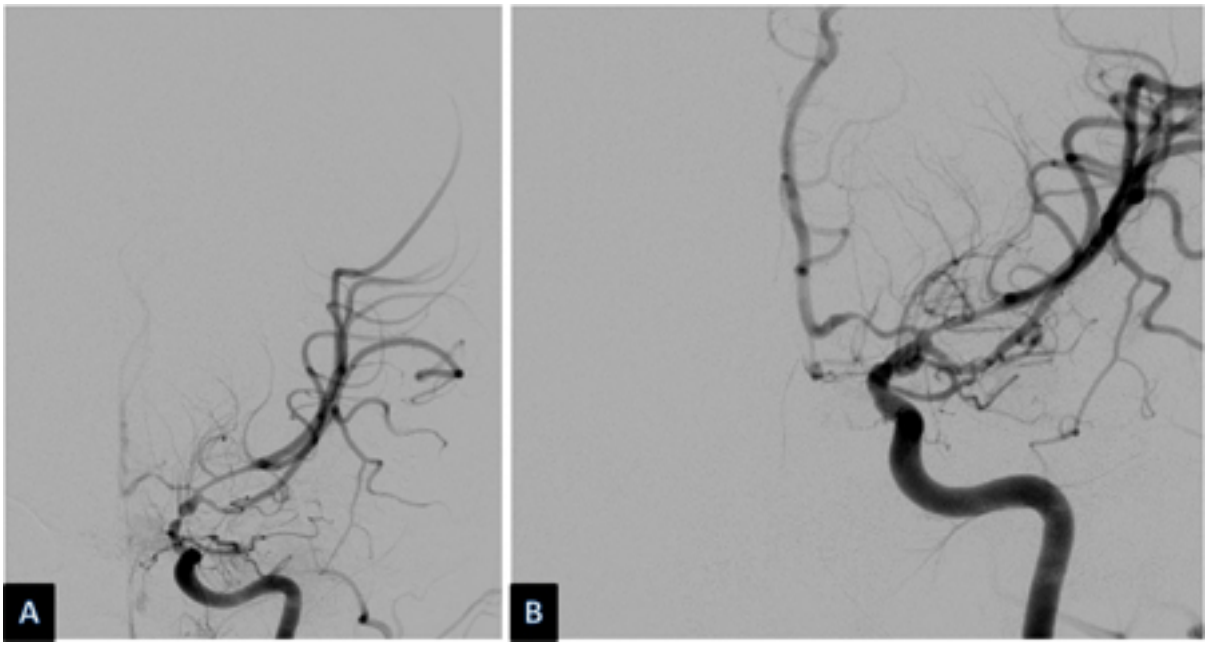

Figure 4: Severe vasospasm of distal left internal carotid artery and proximal middle and anterior cerebral arteries before (A) and after (B) intra-arterial infusion of nicardipine and transluminal balloon angioplasty

Courtesy of Dr Salah Keyrouz; used with permission

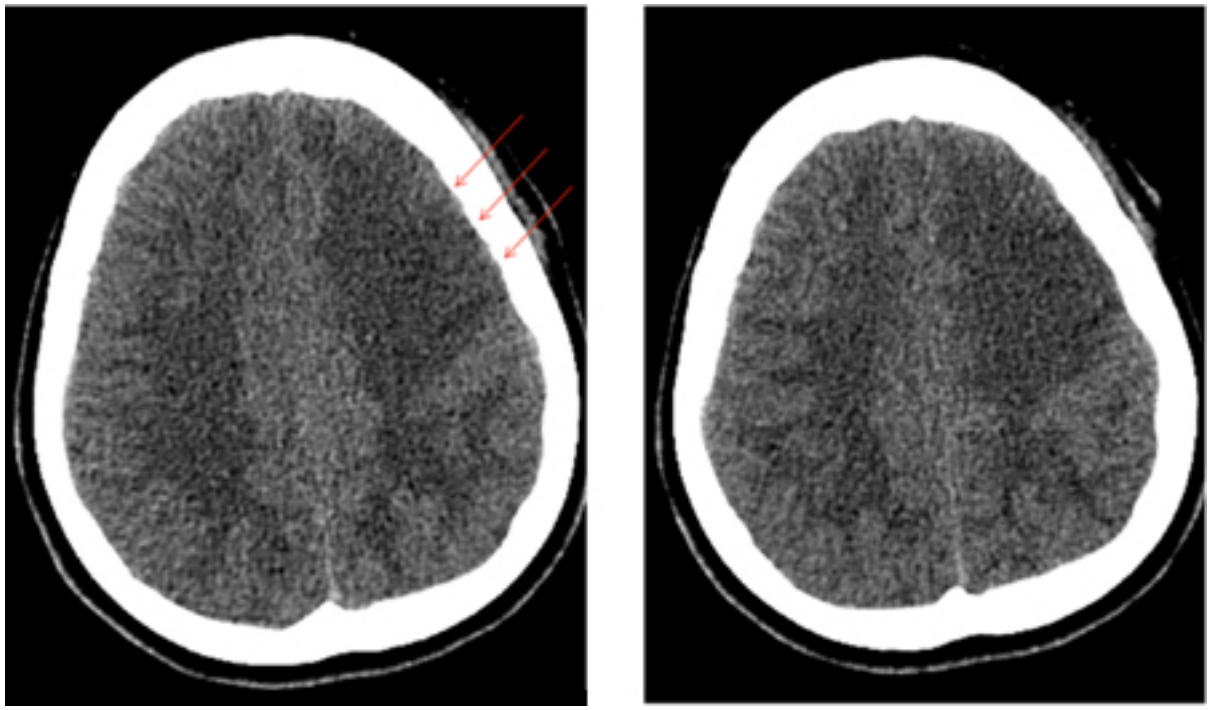

Figure 5: Left frontal infarct (arrows) in a patient with subarachnoid hemorrhage-related vasospasm

Courtesy of Dr Salah Keyrouz; used with permission

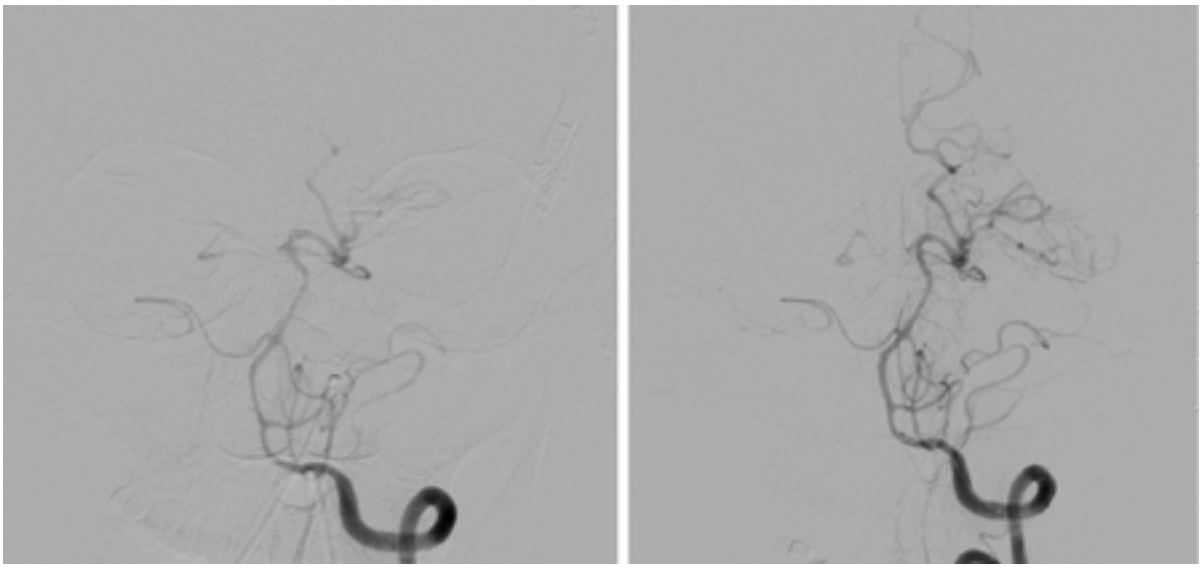

Figure 6: Distal left vertebral and basilar arteries spasm before (left) and after (right) intra-arterial Infusion of nicardipine

Courtesy of Dr Salah Keyrouz; used with permission

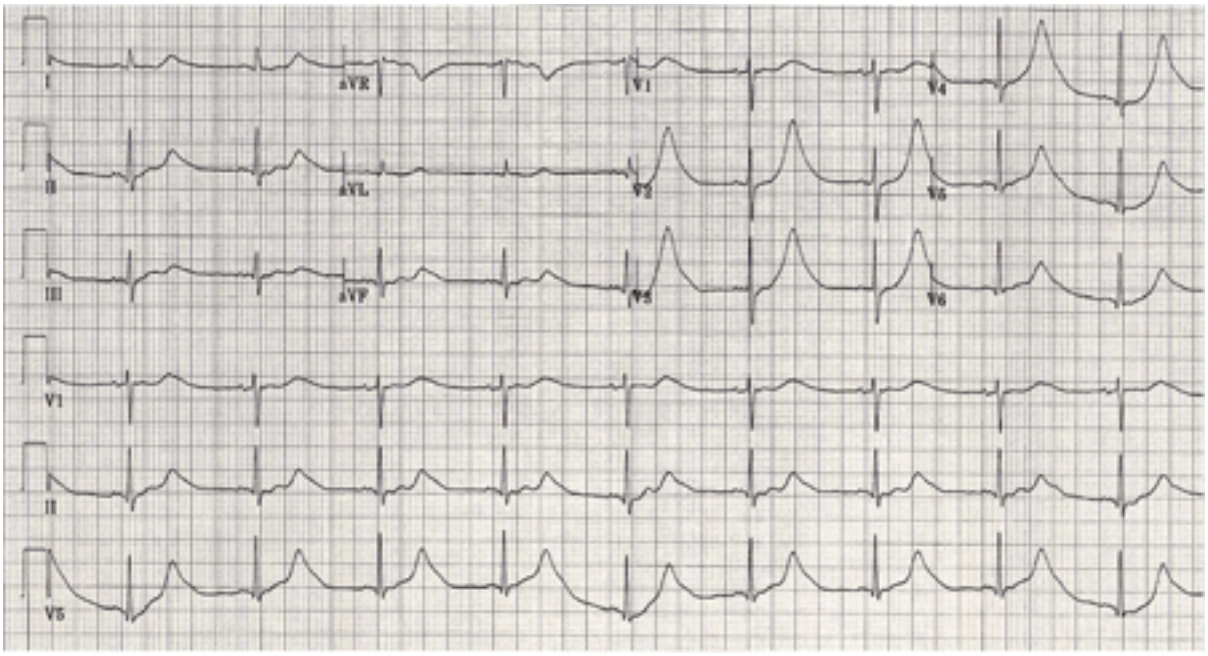

Figure 7: ECG done on admission of a patient with subarachnoid hemorrhage; note peaked, tall T waves (1 of 2)

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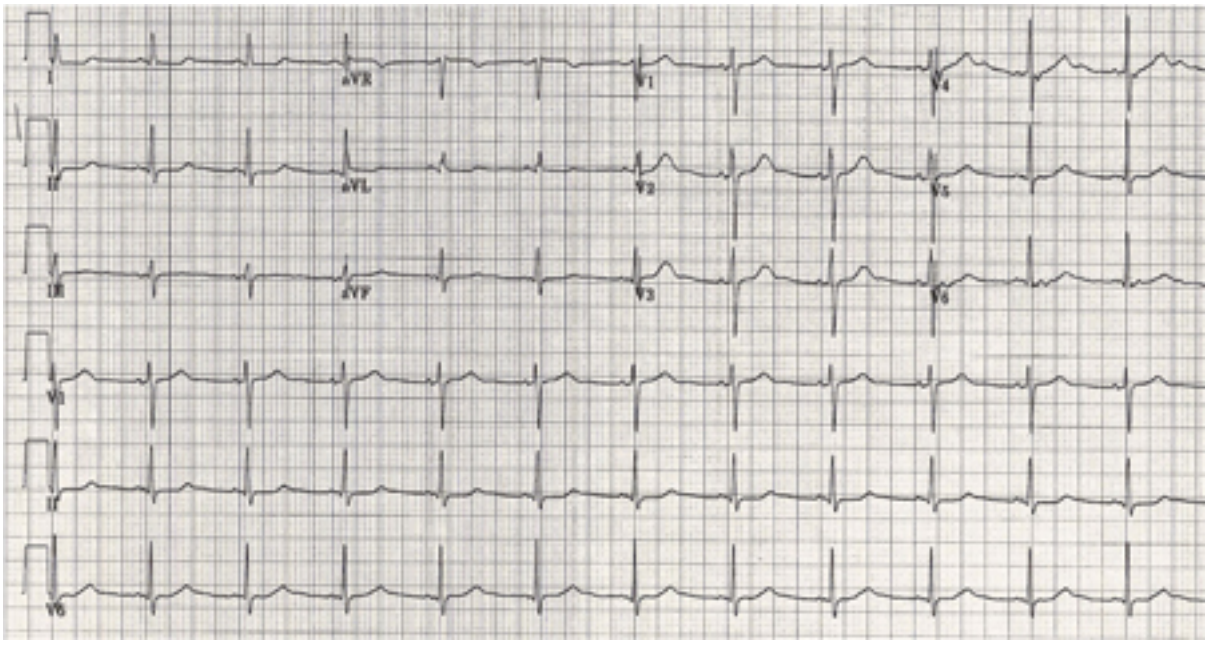

Figure 8: Same patient, 24 hours later; note normalization of T waves (2 of 2)

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This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

4-digit numerals: 1000

numerals < 1: 0.25

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Contact us

+ 44 (0) 207 111 1105
support@bmj.com

BMJ
BMA House
Tavistock Square
London
WC1H 9JR
UK

Contributors:

// Authors:

Salah Keyrouz, MD, FAHA, FCCM

Professor
Neurology and Neurosurgery, Washington University School of Medicine, St. Louis, MO
DISCLOSURES: SK is an author of a reference cited in this topic.

// Acknowledgements:

Dr Salah Keyrouz would like to gratefully acknowledge Dr Michael N. Diringer, a previous contributor to this topic.
DISCLOSURES: MND is an author of a number of references cited in this topic.

// Peer Reviewers:

Venkatesh Aiyagari, MD

Associate Professor
Department of Neurology and Rehabilitation, University of Illinois at Chicago, Chicago, IL
DISCLOSURES: VA declares that he has no competing interests.

Peter Martin, MA, BM BCh, MD, FRCP

Consultant Neurologist
Addenbrookes Hospital, Cambridge, UK
DISCLOSURES: PM declares that he has no competing interests.

Giovanni Grasso, M.D., PhD

Aggregate Professor of Neurosurgery
Neurosurgical Clinic, Department of Clinical Neuroscience, University of Palermo, Palermo, Italy
DISCLOSURES: GG declares that he has no competing interests.