Stroke

AHA/ASA GUIDELINE

# 2023 Guideline for the Management of Patients With Aneurysmal Subarachnoid Hemorrhage: A Guideline From the American Heart Association/American Stroke Association

*The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists.*

*The American Association of Neurological Surgeons/Congress of Neurological Surgeons Cerebrovascular Section affirms the educational benefit of this document.*

*Endorsed by the Neurocritical Care Society, Society of Neurointerventional Surgery, and Society of Vascular and Interventional Neurology*

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subarachnoid hemorrhage.

**METHODS:** A comprehensive search for literat
involving human subjects, published in English

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invasive monitoring may also be useful in patients with high-grade aSAH with limited neurological examination.

7. Early initiation of enteral nimodipine is beneficial in preventing delayed cerebral ischemia and improving functional outcomes after aSAH. Routine use of statin therapy and intravenous magnesium is not recommended.
8. Elevating blood pressure and maintaining euvolemia in patients with symptomatic delayed cerebral ischemia can be beneficial in reducing the progression and severity of delayed cerebral ischemia. However, prophylactic hemodynamic augmentation and hypervolemia should not be performed to minimize iatrogenic patient risks.
9. Cerebrovascular imaging after treatment and subsequent imaging monitoring are important in treatment planning for remnants, recurrence, or regrowth of the treated aneurysm and to identify changes in other known aneurysms. Although the risk of rerupture is low, the use of imaging to guide treatment decisions that may reduce the risk of future aSAH among survivors is recommended, especially in patients with residual aneurysm. Imaging monitoring for the development of de novo aneurysms is also important in younger patients with multiple aneurysms or with ≥2 first-degree relatives with aSAH.
10. A multidisciplinary team approach to identify discharge needs and design rehabilitation treatment is recommended. Among aSAH survivors, physical, cognitive, behavioral, and quality of life deficits are common and can persist. Early identification with validated screening tools can identify deficits, especially in behavioral and cognitive domains. Interventions for mood disorders can improve long-term outcomes, and counseling on the higher risk for long-term cognitive dysfunction may be beneficial.

## PREAMBLE

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Surgeons, American Academy of Neurology, Neurocritical Care Society, Society of Neurointerventional Surgery, Society of Vascular and Interventional Neurology, and 39 individual content reviewers. Appendix 2 lists the reviewers' comprehensive disclosure information.

## 1.4. Scope of the Guideline

This guideline addresses the diagnosis and treatment of aSAH in adults and is intended to update and replace the AHA/ASA 2012 aSAH guideline.[12] This 2023 guideline is limited explicitly to aSAH and does not address other types of SAH such as those caused by trauma, vascular malformation, or hemorrhage-prone neoplasm. Furthermore, this guideline does not overlap with AHA/ASA guidelines or scientific statements on the treatment of intracerebral hemorrhage (ICH),[13] arteriovenous malformations,[14] and unruptured intracranial aneurysms.[15]

This guideline aims to cover the full course of aSAH (Figure 1), from initial diagnosis (Section 4), systems of care (Section 5), and acute interventions (Sections 6, 7, and 7.1) to further inpatient care of post-aSAH complications (Sections 8–8.5). New sections in this 2023 aSAH guideline include nursing care (Section 8.1) and recovery (Section 9). Risk factors for recurrent aSAH are also addressed (Section 10); however, risk factors for aneurysm development and rupture and management of unruptured aneurysms are not included in this guideline because these topics are addressed in a separate guideline for management of unruptured intracranial aneurysms.[15] The new, important emphases in this guideline are shared decision-making, health equity, and systems of care.

Some aspects of inpatient aSAH medical care and post-aSAH rehabilitation and recovery are likely to be similar between patients with aSAH and patients with

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mechanisms in the first 72 hours after aSAH that drive early brain injury also influence secondary complications and overall outcomes.[23] DCI is now hypothesized to be caused by the combined effects of large-vessel cerebral vasospasm and multiple brain injury processes triggered by aneurysm rupture and early brain injury. Mechanisms involving arteriolar constriction and cerebral microthrombosis, cortical spreading depolarization/ischemia, blood-brain barrier breakdown, cerebral autoregulation impairment, and capillary transit time heterogeneity are hypothesized to play a role in the pathophay

favorable outcome in 12 months. Similar results were reported in a meta-analysis by Zhao et al,[40] which included 85 studies with 4506 patients with poor-grade aSAH. Good outcomes were observed in 39% of treated patients.

3. When controlling for degree of neurological injury, older patients compared with younger patients with aSAH have less favorable outcomes.[28,29,31–33] In a post hoc analysis of the 405 patients included in BRAT (Barrow Ruptured Aneurysm Trial), 42% of patients >65 years of age reached functional independence at the 6-year follow-up. Although this number was significantly smaller than in the younger cohort (82%), it demonstrates that aneurysm treatment in this age group is reasonable and should be considered after discussion with the family and surrogates.[31]

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**Table 3. Ottawa SAH Rule**

| For alert patients >15 y of age with new severe nontraumatic headache reaching maximum intensity within 1 h. Patients require additional investigation for SAH if they meet any of the following criteria: | |
|---|---|
| 1 | Age ≥40 y |
| 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 examination |

SAH indicates subarachnoid hemorrhage.

diagnosis, these small differences are critical. LP for xanthochromia evaluation should be performed in patients presenting >6 hours from ictus in whom there is high suspicion for SAH.

3. High-quality CT scanners can detect SAH with a high sensitivity, especially when the images are interpreted by fellowship-trained, board-certified neuroradiologists. (Equipment specifications for a high-quality CT scanner have been published by the American College of Radiology.[65]) For patients presenting within 6 hours of headache onset who have no new neurological deficits, the lack of SAH on a noncontrast head CT is likely sufficient to exclude aSAH.[50–53] This question was evaluated in a 2016 meta-analysis in which 8907 patients were studied. Thirteen patients had a missed SAH on head CT performed within 6 hours, leading to a sensitivity of 98.7% and specificity of 99.9%. Therefore, when performed within 6 hours of symptom onset, a negative head CT was likely to miss <1.5 in 1000 SAHs.[51] It is important to note that many of these analyses do not apply to patients with atypical presentations such as primary neck pain, syncope, seizure, or new focal neurological deficit. Therefore, the lack of a classic presentation should still prompt appropriate imaging and workup.
4. The Ottawa SAH Rule serves as a method to screen out individuals with a low likelihood of aSAH.[45] Application of the rule requires that patients who present with a severe headache and meet any of the criteria outlined in Table 3 may need to undergo additional testing, as directed by the treating physician. The initial study by Perry et al[45] enrolled 2131 patients, of whom 132 (6.2%) had SAH. Application of the rule was 100% sensitive but only 15.3% specific. The rule was later validated by the study authors at 6 medical centers in a prospective manner, with 1153 patients enrolled and 67 SAHs, and was found to be 100% sensitive and 13.6% specific.[55] The rule was externally validated by Bellolio et al[54] in 454 patients, of whom 9 had SAH, and it was 100% sensitive but only 7.6% specific. Use of the Ottawa SAH Rule can therefore identify a subset of patients (albeit small) who are unlikely to have aSAH and thereby avoid additional imaging and workup that use resources and expose patients to unnecessary risk.
5. CTA is widely available and often is the next diagnostic test performed when SAH is diagnosed with noncontrast CT. Certain hemorrhage patterns likely reflect a greater risk for the presence of an underlying aneurysm than others (eg, diffuse basal cistern and sylvian fissure SAH versus small-volume focal cortical SAH). 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.[56–59,66]
6. 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. CTA alone may, in certain clinical settings, be used for treatment decision-making.[60,61]

### *Knowledge Gaps and Future Research*

- *Utility of magnetic resonance imaging:* Diagnostic accuracy studies of various established and emerging magnetic resonance imaging sequences for the detection and characterization of aSAH are needed.
- *Perimesencephalic SAH:* There is currently equipoise concerning the appropriate diagnostic pathway for a perimesencephalic distribution of SAH with CTA alone versus catheter-based DSA.
- *Emerging technologies:* Dual-energy CT and single-photon counting CT represent novel imaging techniques that may be helpful for SAH and aneurysm detection.

## 5. HOSPITAL CHARACTERISTICS AND SYSTEMS OF CARE

| Recommendations for Hospital Characteristics and Systems of Care Referenced studies that support recommendations are summarized in online Data Supplement 3. | | |
|---|---|---|
| **COR** | **LOE** | **Recommendations** |
| 1 | B-NR | 1. For patients with aSAH, timely transfer from hospitals with low case volume to higher-volume centers with multidisciplinary neurointensive care services, comprehensive stroke center capabilities, and experienced cerebrovascular surgeons/neuroendovascular interventionalists is recommended to improve outcomes.[67–77] |
| 1 | B-NR | 2. For patients with aSAH, care should be provided in a dedicated neurocritical care unit by a multidisciplinary team.[78–80] |



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### Synopsis

Patients with aSAH should undergo repair of their aneurysm as soon as it is feasible to reduce the risk of aneurysm rerupture, an event that is frequently fatal. However, the choice of treatment modality is highly nuanced. The goal of securing the aneurysm must be balanced with the risks of the procedure. Open surgical options and endovascular techniques have different advantages and disadvantages that need to be carefully weighed for each individual patient because many patient-specific factors (including patient age, aneurysm geometry and location, and presence of intraparenchymal hemorrhage) must be considered. Sometimes complete obliteration is not feasible, either technically or because procedural risks outweigh the benefits. The best outcomes for patients with SAH will be achieved when both endovascular and open surgical options are available. The quality of the evidence supporting recommendations for treatment modality is relatively limited, with a particular paucity of data on comparison of different endovascular techniques with surgical techniques or with each other. Further studies for many of these questions are necessary, and several of these are listed under "Knowledge Gaps and Future Research."

### Recommendation-Specific Supportive Text

1. Early treatment of ruptured aneurysms reduces the risk of rebleeding and facilitates treatment of DCI. Timing of ruptured aneurysm treatment has been directly examined in only 1 small randomized prospective trial of patients with good-grade aSAH in the precoiling era; this study of 159 patients demonstrated that early surgery (0–3 days from SAH) resulted in lower death and dependence at 3 months compared with intermediate surgery (4–7 days) or late surgery (≥8 days).[103] Subsequent retrospective and prospective observational studies of both clipping and coiling, including post hoc analyses of the randomized ITj -0.r. Alermenional stbsearh onoid5 ( )]TJ 0.05 TTw 0 -1.2 Td [(weAurysm trT)3-wele.andwith g lo24, 4 -

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surgical treatment options by specialists with expertise, individually or as a team, in both modalities is necessary to optimally evaluate the relative risks and benefits of each treatment strategy.

7. Although older patients (>70 years of age) are often preferentially treated with coiling, in practice, there are insufficient data to support a clear benefit of coiling in this population. In the largest available RCT, ISAT, subgroup analysis by age demonstrated no benefit of coiling in the group >70 years of age, with an RR of death dependency of 1.15 (95% CI, 0.82–1.61) for coiling.[113] Within the cohort of patients >65 years of age in ISAT, outcome was dependent on aneurysm location, with coiling superior in those with internal carotid and posterior communicating artery aneurysms but clipping superior for those with ruptured middle cerebral artery (MCA) aneurysms.[114] Nonrandomized registry and observational data have also failed to demonstrate an effect of treatment modality on outcomes in the elderly (>75 years of age).[128,129]
8. Longer life expectancy and better long-term protection from rerupture related to clipping favor consideration of clipping in young patients. Subgroup analysis from the largest available RCT, ISAT, indicates less benefit of coiling in those <50 years of age,[113] and calculations based on ISAT data suggest that clip placement may be more advantageous for patients <40 years of age.[115]
9. The Cochrane review and meta-analysis of 4 RCTs of clipping versus coiling indicates that primary coiling provides higher odds of functional independence (mRS score 0–2) at 1 year, with an RR 0.77 (95% CI, 0.67–0.87) for death/dependency.[110] The

coiling are limited, and further studies focused on these outcomes are needed.

- *Modality of treatment in patients with high-grade aSAH:* Although patients with high-grade aSAH are often preferentially treated with coiling in practice, there are insufficient robust randomized data on coiling versus clipping specifically in patients with high-grade aSAH to support clear evidence-based guidance in this population.
- *Anterior circulation aneurysms:* Ruptured MCA aneurysms, and anterior circulation bifurcation aneurysms in general, are typically considered more favorable for clipping, which is supported by limited data; however, there are insufficient data to provide definitive guidance, especially in the setting of emerging endovascular technologies.
- *Novel flow diversion technologies:* Endosaccular flow diverters and less thrombogenic intravascular flow diverters reduce the need for dual antiplatelets and attendant higher complications. Limited noncomparative data suggest protection against rebleeding and acceptable outcome with endosaccular devices, but there are insufficient comparative data and long-term outcomes to provide guidance on the use of these devices for ruptured aneurysms.
- *Evolving endovascular technologies:* In general, evolving endovascular technologies may provide additional treatment options for ruptured aneurysms, but their comparative efficacy cannot be extrapolated from prior data on other endovascular techniques such as primary coiling. New technologies should be studied prospectively relative to existing treatment options before widespread adoption.

## 7.1. Anesthetic Management of Surgical and Endovascular Treatment of aSAH

**Recommendations for Anesthetic Management of Surgical and Endovascular Treatment of aSAH**
**Referenced studies that support recommendations are summarized in online Data Supplement 6.**

| COR | LOE | Recommendations |
|---|---|---|
| 2a | B-R | 1. In patients with aSAH, the intraoperative use of mannitol or hypertonic saline can be effective in reducing ICP and cerebral edema.[135,136] |
| 2a | B-NR | 2. In patients with aSAH, anesthetic goals should include minimizing postprocedural pain, nausea, and vomiting.[137–140] |
| 2a | B-NR | 3. In patients with aSAH, prevention of intraoperative hyperglycemia and hypoglycemia during aneurysm surgery is reasonable to improve outcomes.[141–147] |
| 2a | C-LD | 4. In patients with aSAH and an unsecured ruptured aneurysm, frequent intraoperative BP monitoring and BP control are reasonable to prevent ischemia and rerupture.[148–153] |
| 2b | B-NR | |
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cytotoxic edema, which greatly increase the risk of poor outcome if associated with local and global cerebral ischemia.[135] In addition, surgical exposure and the operative procedure become more difficult. Both mannitol and hypertonic saline have been used to decrease ICP and increase cerebral blood flow (CBF) and brain relaxation.[136] Mannitol is a potent diuretic and can cause hypovolemia and hypotension, whereas hypertonic saline increases blood sodium, has minimal effect on diuresis, and can increase BP. A clinical trial to evaluate the optimal intraoperative dose of mannitol in patients with aSAH is ongoing at the time of guideline publication (ClinicalTrials.gov identifier: NCT04135456). Currently, there is insufficient evidence to recommend one therapy over the other or to affirm whether outcomes are affected.

2. Postoperative nausea and vomiting can have a negative impact after aneurysm coiling and clipping by increasing the risk of aspiration of gastric contents.[137] Incidence after aSAH has not been studied, but postoperative nausea and vomiting after craniotomy occur in 22% to 70% of patients.[140] A multimodal regimen of medication targeting different chemoreceptors is recommended.[172] Although serotonin 5-HT3 receptor antagonists (eg, ondansetron), steroids (eg, dexamethasone), and their combination are the most frequently used antiemetics, the addition of propofol, reduction of narcotics, and euvolemia are generally advocated. Medications that can cause confusion or sedation such as anticholinergics (eg, scopolamine) and phenothiazines (eg, promethazine) at higher doses may impair neurological examination. The use of volatile anesthetic medications for craniotomy has been associated with a higher incidence of postoperative nausea and vomiting compared with intravenous agents such as propofol,[138,173] and dexmedetomidine may offer an advantage compared with fentanyl as an analgesic.[139] Further clinical trials comparing different regimens that significantly reduce the incidence of postoperative nausea and vomiting after aSAH would provide relevant data in this patient population.
3. Poor perioperative glycemic control in patients with aSAH has been associated with increased risk of poor clinical outcome. The management of intraoperative glucose concentrations has not been well studied; however, the prevention of intraoperative hyperglycemia and hypoglycemia during aneurysm surgery is probably indicated.[141,142,144–146] A post hoc analysis of IHAST (Intraoperative Hypothermia for Aneurysm Surgery Trial)[147] determined that commonly encountered hyperglycemia was associated with long-term changes in cognition and gross neurological function.[143]
4. Intraoperative volume status and BP goals are not well defined in intraoperative aSAH management. Numerous pathophysiological states may be present after aneurysmal rupture such as cardiovascular dysfunction, systemic inflammation, autoregulatory failure, and spreading depolarizations that can be affected by intravascular volume.[152] During the intraoperative period, frequent BP monitoring and BP control are reasonable to prevent ischemia and rerupture. Hypovolemic states often necessitate additional pressor support, especially when clinical management necessitates hypertensive therapy. There are suggestions that hypovolemia (in the perioperative period) may contribute to the incidence of DCI,[152] whereas hypervolemia lacks benefit[148,149,151,153] and the rapid reduction of BP is potentially harmful.[150] Careful BP management needs to occur throughout the perioperative period, including transportation.
5. Intraoperative neuromonitoring can be used to evaluate functional brain integrity in a timely manner during aneurysm surgery. Common intraoperative neuromonitoring modalities used are spontaneous electroencephalography (EEG) ysiologica(2 Td

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can lead to systemic or cerebral hypoglycemia and metabolic crisis in the acutely injured brain and potentially worsen brain injury and outcome, which remains to be determined.[232,233,239]

8.

acute phase, putting them at increased risk of developing pneumonia and leading to increased LOS and poor functional outcome and mortality at 90 days.[258–261] Nurses are in a unique position to evaluate for dysphagia before administering anything by mouth. A systematic review and subgroup analysis that included 4528 patients found that nurse-initiated dysphagia screening and the use of formal guidelines had a significant positive effect in the prevention of pneumonia and decreased mortality rates.[261] Another large systematic review and single-blinded cluster RCT that included both patients with hemorrhagic stroke and patients with ischemic stroke reported various dysphagia screening tools and recommended the use of a validated tool as best practice within 24 hours of admission.[246]

5. Implementing stroke care protocols and order sets and providing specialized assessments to the patient with stroke require expert nursing care. A pre-/posttest-designed study found that nurses who participated in stroke competency training had improved knowledge of and adherence to stroke guidelines with a positive association with decreased LOS.[263]2424a (OS.)]TJ 0 Tw (-)40 0.056 Tw 0 -1.2 (pa,iated dysphagia scre,e and paocolsfamid ( (263

## 8.2. Monitoring and Detection of Cerebral Vasospasm and DCI

| Recommendations for Monitoring and Detection of Cerebral Vasospasm and DCI<br>Referenced studies that support recommendations are summarized in online Data Supplement 9. | | |
|---|---|---|
| **COR** | **LOE** | **Recommendations** |
| **2a** | **B-NR** | 1. In patients with aSAH with suspected vasospasm or limited neurological examination, CTA or CT perfusion (CTP) can be useful to detect vasospasm and predict DCI.[270–275] |
| **2a** | **B-NR** | 2. In patients with aSAH, transcranial Doppler (TCD) ultrasound monitoring is reasonable to detect vasospasm and predict DCI.[253,276–280] |
| **2a** | **B-NR** | 3. In patients with high-grade aSAH, continuous EEG (cEEG) monitoring can be useful to predict DCI.[276,280–292] |
| **2b** | **B-NR** | 4. In patients with high-grade aSAH, invasive monitoring of brain tissue oxygenation, lactate/pyruvate ratio, and glutamate may be considered to predict DCI.[293–305] |

### *Synopsis*

Narrowing of the cerebral arteries (cerebral vasospasm) occurs frequently in patients with aSAH and is associated with DCI and infarction. DCI occurs in ≈30% of patients, mostly between days 4 and 14 after aSAH. Clinical deterioration due to DCI has been defined as the occurrence of focal neurological impairment or a decrease of at least 2 points on the GCS.[306–308] This should last for at least 1 hour, is not apparent immediately after aneurysm occlusion, and cannot be attributed to other causes. New cerebral infarction has been defined as the presence of cerebral infarction on CT or magnetic resonance imaging scan of the brain within 6 weeks after aSAH not attributable to other causes.[306–308] Diagnosis of DCI can be challenging, and although serial neurological examinations are important (see Section 8.1), they are of limited value in patients with high-grade aSAH. Several diagnostic tools have been used to identify arterial narrowing and cerebral perfusion abnormalities that may help predict DCI. The most commonly available techniques include TCD ultrasound,[253,276–280]

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diagnostic accuracy of cEEG for DCI in patients with high-grade aSAH following the Standards for Reporting of Diagnostic Accuracy Studies.[315] The study protocol consisted of clinical neurophysiologists prospectively reporting prespecified EEG alarms: (1) decreasing relative alpha power variability, (2) decreasing alpha-to-delta power ratio, (3) worsening focal slowing, or (4) late-appearing epileptiform abnormalities. The diagnostic reference

techniques to target different levels of the cerebral vasculature.[373]

5. Angioplasty offers a mechanical option for improving perfusion in patients with severe vasospasm. Historically, angioplasty was performed with endovascular balloons and was confined to proximal vasculature. The choice between compliant and noncompliant balloons remains operator dependent.[356] A poor angiographic response to angioplasty is associated with recurrent vasospasm and risk of cerebral infarction.[337] Particular care must be taken with cerebral angioplasty given the high mortality associated with vessel rupture, although contemporary safety profiles are favorable. Limited direct comparisons exist between angioplasty and vasodilator therapy,5.

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have shown promise, treatment in patients with aSAH requires further studies.

- *Drainage via lumbar drain or EVD*: Such drainage allows expedited removal of blood products from the CSF, with computational models showing effectiveness. Available trial data suggest that drainage through a lumbar drain reduces the incidence of DCI, despite trials being population and center limited. The role of a lumbar drain in aSAH is being actively investigated.
- *Cisternal fibrinolytic and spasmolytic medication:* These medications have recently been introduced to help clear subarachnoid blood in the CSF space. An ongoing double-blinded clinical trial is underway to detect effectiveness through administration by cisternal lavage.

## 8.4. Management of Hydrocephalus Associated With aSAH

| **Recommendations for Management of Hydrocephalus Associated**<br>Tm [(s /T1_3Manag1ment of Hydroce1.531_0 1893f 0 TwORsupport 4-1.2 | | |
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key components defined in the literature to be considered and included in a bundled EVD protocol.

3. According to the literature, aSAH-associated persistent or chronic shunt-dependent hydrocephalus occurs in 8.9% to 48% of patients with aSAH.[400] Significant predictors of shunt dependency include poor admission neurological grade, increased age, acute hydrocephalus, high Fisher grades, presence of intraventricular hemorrhage, rebleeding, ruptured posterior circulation artery aneurysm, anterior communicating artery aneurysm, surgical clipping, endovascular coiling, cerebral vasospasm, meningitis, and a prolonged period of EVD.[12,397–400] According to a large observational study, clipping and coiling of ruptured and unruptured cerebral aneurysms were associated with similar incidences of ventricular shunt placement for hydrocephalus.[398] Last, permanent CSF diversion was shown to improve neurological outcome after aSAH.[12,397–400]
4.

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|---|---|---|
| **3: Harm** | **B-NR** | 4. In patients with aSAH, phenytoin for seizure prevention and/or antiseizure prophylaxis is associated with excess morbidity and mortality.[407–411,413–415] |
| Patients who present with seizures | | |
| **2a** | **B-NR** | 5. In patients with aSAH who present with seizures, treatment with antiseizure medications for ≤7 days is reasonable to reduce seizure-related complications in the perioperative period.[411,416,417] |
| **3: No benefit** | **B-NR** | 6. In patients with aSAH without prior epilepsy who present with seizures, treatment with antiseizure medications beyond 7 days is not effective for reducing future SAH-associated seizure risk.[408,410,411] |

### *Synopsis*

Although the incidence of aSAH-associated seizures is relatively common, an understanding of the management of seizures is poorly supported by randomized, controlled studies. Since the 2012 aSAH guideline,[12] meta-analyses, single-center studies, and evaluations of next-generation antiseizure medications are the best sources from which to obtain management recommendations. Although seizure-like episodes have been reported in up to 26% patients with aSAH, a better understanding of the incidence of seizures has been attained with improved EEG monitoring capability. More recent studies suggest a lower seizure incidence of 7.8% to 15.2%.[410,411,417] Early and late postoperative seizures have an incidence of 2.3% and 5.5%, respectively.[411] In patients with aSAH who present with seizures, the use of antiseizure medications for <7 days is reasonable to reduce delayed seizure or hemorrhage risk.[411,416,417] Endovascular coil embolization compared with neurosurgical clipping seems to be associated with a lower incidence of late seizures.[113,411] In addition to the surgical management of aneurysms, clinical grade (HH grade ≥

many instances.[422] Studies that discuss the effect of antiseizure medications on global functional outcomes are inconclusive.[407,423] Poorer cognitive outcomes have been related to phenytoin administration. Whether this increased morbidity is related to an effect on DCI through metabolic competition with nimodipine or undiagnosed transaminase elevations is unclear. Use of newer-generation antiseizure medications that may be more effective or less toxic than phenytoin remains a topic of discussion.[424] A single-blinded randomized study of levetiracetam versus phenytoin demonstrated the same outcomes with respect to mortality or seizure control as evaluated by cEEG. Therapy with levetiracetam resulted in a lower incidence of adverse effects as evaluated by the Glasgow Outcome Scale–Extended and Disability Rating Scale. The excess morbidity associated with the use of phenytoin should prompt the use of alternative antiseizure medications.[415]

5. An important distinction in the management of patients with aSAH-associated seizure is whether seizure is a component of the patient presentation. This has resulted in literature and subsequent recommendations that are based on onset, early, and late seizures. Onset seizures occur at the time of the hemorrhage; early seizures occur during the first week; and late seizures are either postoperative or occur after 1 week. Onset seizures have been found to predict poor outcome after aSAH.[417] The possibility of preventing nonconvulsive status or rerupture of an unsecured aneurysm

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|---|---|---|
| 1 | B-NR | 3. In patients with aSAH and depression, |
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with differences in the dose, duration, and type of selective serotonin reuptake inhibitors used. Among patients with stroke who were on selective serotonin reuptake inhibitors, there was a reduction in the proportion of patients with post-stroke depression (RR, 0.75; 3 studies with high-quality evidence including 5907 participants).[434] In another review (n=8 trials), pharmacological interventions (n=1025 participants) decreased depressive symptoms at the end of treatment.[433] The use of selective serotonin reuptake inhibitors is appropriate for patients with preexisting symptoms of depression. Various psychosocial interventions that have been studied for stroke survivors include music therapy, mindfulness, and motivational interviewing. Although data supporting the efficacy of these interventions are limited, they are

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epilepticus) have been treated, few interventions have been studied to promote consciousness recovery in the acute setting. Cognitive motor dissociation may be seen in 15% of patients with severe acute brain injuries, including aSAH,[459] as determined by

### Synopsis

Long-term recovery extends beyond the first 3 months in individuals with aSAH. Neurological deficits can result in an increased incidence of depression, anxiety, and cognitive impairments, resulting in changes in familial roles and a negative impact on overall QOL for months to years after the initial injury. These recommendations focus on evaluations done for the purpose of identifying treatable cognitive and behavioral sequelae after aSAH. The variable yield of testing, including the potential influence of caregiver input, means that clinicians should exercise discernment when initiating treatment plans.

### Recommendation-Specific Supportive Text

1. Identification and treatment of psychological and sexual sequelae can have a positive impact on QOL after aSAH. Screening tools to evaluate patterns of depression, anxiety, mobility, and activities of daily living detailed in the literature include the State Trait Anxiety Inventory, Hospital Anxiety and Depression Scale, Telephone Interview for Cognitive Status, and Barthel Index at 6 months, 1 year, and 2 years.[462] Use of the International Index of Erectile Function and the Female Sexual Function Index within the first 4 years[465] is recommended to evaluate sexual dysfunction in men and women, respectively. For long-term follow-up after aSAH, the Hospital Anxiety and Depression Scale can be used within the first 8.9 years[463] to examine anxiety and depression incidence, and the EuroQol-5D can be used within the first 10 years to evaluate health-related QOL.[461] Furthermore, the 36-item Short Form can be used within 4.7 years to evaluate outcomes in 8 domains: physical and social functioning, role limitations because of physical or emotional problems, bodily pain, mental and general health perception, and vitality.[464] Although the time frame for use of these screening tools is described here in accordance with the studies, their use can be considered beyond the suggested time frames for individual patients.
2. After aSAH, cognitive dysfunction is an important cause of disability. The most common cognitive complaints include mental slowness, memory, and attention difficulties. Although most deficits improve, ≈50% of patients with aSAH continue to experience cognitive difficulties for a year.[469,470]

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## ARTICLE INFORMATION

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

This guideline was approved by the American Heart Association Science Advisory and Coordinating Committee on February 20, 2023, and the American Heart Association Executive Committee on April 4, 2023. A copy of the document is available at https://professional.heart.org/statements by using either "Search for Guidelines & Statements" or the "Browse by Topic" area. To purchase additional reprints, call 215-356-2721 or email Meredith.Edelman@wolterskluwer.com.

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### Disclosures

**Appendix 1. Writing Group Relationships With Industry and Other Entities (Relevant)–2023 Guideline for the Management of Patients With Aneurysmal Subarachnoid Hemorrhage**

| Writing | | | | | | | | |
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**Appendix 1. Continued**

| Writing group member | Employment | Research grant/other research support | Speakers' bureau/ honoraria | Expert witness | Ownership interest | Consultant/ advisory board | Other | Voting recusals by section‡ |
|---|---|---|---|---|---|---|---|---|
| Steven W. Hetts | University of California San Francisco | Stryker†; Siemens Medical Solutions† | None | None | None | Kaneka Pharma America LLC* | None | 7. Surgical and Endovascular Methods for Treatment of Ruptured Cerebral Aneurysms |
| Nneka L. Ifejika | University of Texas Southwestern Medical Center | None | None | None | None | None | None | |
| Regina Johnson | NA | None | None | None | None | None | None | |
| Kiffon M. Keigher | Advocate Aurora Health System and Rush University College of Nursing | None | None | None | None | None | None | |
| Thabele M. Leslie-Mazwi | University of Washington | None | None | None | None | None | None | |
| Brandon P. Lucke-Wold | University of Florida | None | None | None | None | None | None | |
| Alejandro A. Rabinstein | Mayo Clinic | None | None | None | None | None | None | |
| Steven A. Robicsek | University of Florida | None | None | None | None | None | Heineman-Robicsek Foundation* (fiduciary officer) | |
| Christopher J. Stapleton | Massachusetts General Hospital | Genentech†; Penumbra†; Route 92† | None | None | None | None | None | 7. Surgical and Endovascular Methods for Treatment of Ruptured Cerebral Aneurysms |
| Jose I. Suarez | Johns Hopkins University School of Medicine | None | None | None | None | None | None | |
| Stavropoula I. Tjoumakaris | Thomas Jefferson University Hospital at Sidney Kimmel Medical College | None | None | None | None | MicroVention*; Medtronic† | None | 7. Surgical and Endovascular Methods for Treatment of Ruptured Cerebral Aneurysms |
| Babu G. Welch | University of Texas Southwestern Medical Center | Stryker*; MicroVention* | None | None | None | MicroVention*; Stryker Corp†; Medtronic* | None | 7. Surgical and Endovascular Methods for Treatment of Ruptured Cerebral Aneurysms |

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $5000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity or owns $5000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest (<$5000).

†Significant (≥$5000).

‡Writing committee members are required to recuse themselves from voting on sections to which their specific relationships with industry and other entities may apply.

**Appendix 2. Peer Reviewer Relationships With Industry and Other Entities (Comprehensive)–2023 Guideline for the Management of Patients With Aneurysmal Subarachnoid Hemorrhage**

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**Appendix 2.** **Continued**

| Peer reviewer | | | | | | | | |
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(*Continued*)

| Peer reviewer | Employment | Research grant | Other research support | Speakers' bureau/ honoraria | Expert witness | Ownership interest | Consultant/ advisory board | Other |
|---|---|---|---|---|---|---|---|---|
| Aarti Sarwal | Wake Forest Baptist Health; Wake Forest School of Medicine | Butterfly Network, Inc.†; C. R. Bard, Inc. & Subsidiaries† | University of Technology, Sydney (Intellectual Property - Other Intellectual Property: Compensation for reviewing Thesis for a Master's project)*; Society of Critical Care Medicine (Intellectual Property - Other Intellectual Property: Social Media Editor for Critical Care Medicine)* | None | None | None | CVR Global (Independent Contractor - Site Investigator for multicenter clinical trial conducted by CVR Global)*; C. R. Bard, Inc. & Subsidiaries (Independent Contractor - Site investigator for multicenter trial sponsored by Bard)*; Biogen, Inc. (Independent Contractor - Site investigator for multicenter trial conducted by Biogen. Monies paid to Department for costs associated with multicenter clinical trial. No direct monies or support paid to me.)* | Intensive Care Society (Travel; Location: Belfast, Ireland. Reimbursement for travel to speak at ICS)*; Travelkt7S0 -0 -Fi 0 0 y OfTj 02 Tw -1.28ducted |
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