



Emergency Neurological Life Support Subarachnoid Hemorrhage Protocol Version 4.0

Authors

Sayona John, MD
William J. Meurer, MD, MS
Stephanie Qualls, RN, BSN, CNRN
Brian L. Edlow, MD

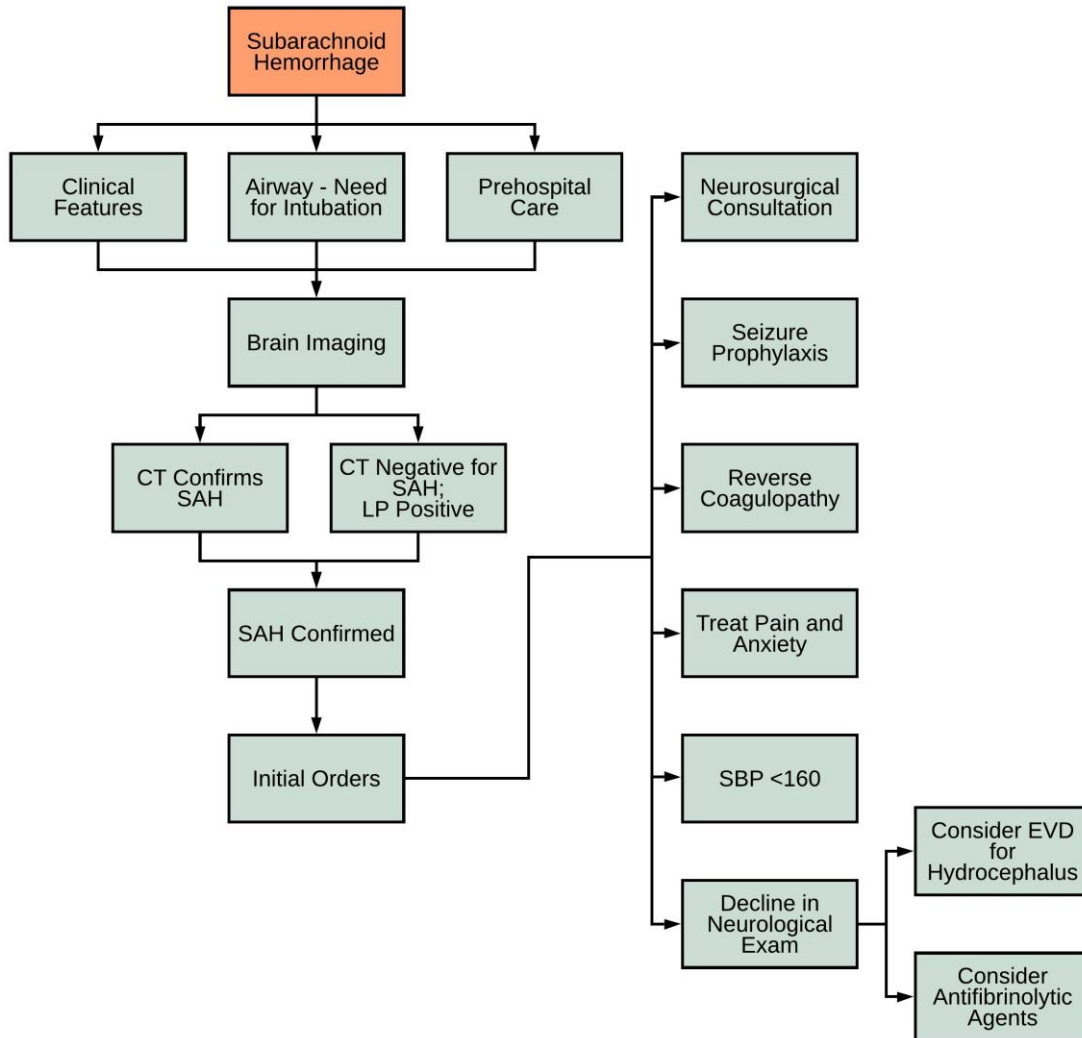
Last updated:

October 2019

Protocol developed by:

Sayona John, MD
Brian L. Edlow, MD

Subarachnoid Hemorrhage Algorithm (click each box for details)



Checklist & Communication

Checklist

- Airway, breathing, circulation
- Head computed tomography (CT)
- Laboratories: PT/INR, PTT, CBC, chemistries, troponin, toxicology screen
- 12 lead ECG
- Target SBP goal < 160 mmHg
- Consult neurosurgery/NCC team
- Address hydrocephalus if present

Communication

- Airway status
- Hemodynamic status and blood pressure control (BP goals)
- Clinical presentation (level of consciousness, motor examination, pupils)
- WFNS score and Hunt–Hess Grade
- Imaging/LP results
- Coagulopathy present?
- Hydrocephalus present?
- Medications given (dose and time administered), including sedatives, analgesics, seizure prophylaxis, anti-hypertensives, and nimodipine
- Coordination of other vascular imaging

Sample sign off narrative:

“I am signing out a 45-year-old man with no known past medical history”

“He complained of a severe headache and was then found unresponsive at his work place at 3 PM, last seen normal by his coworker at 2:45 PM. GCS in the field was 9, and BP was 180/110”

“On arrival to the ED here, he was the same, so we drew labs and sent him for a head CT”

“CT completed at 4:30 PM showed a diffuse subarachnoid hemorrhage, minimal intraventricular hemorrhage with mild hydrocephalus. CTA shows a right anterior communicating artery measuring 7 mm”

“When he returned to the ED, he was sleepier, with a GCS of 5, so he was intubated using rocuronium and etomidate”

“His labs came back with a Troponin of 0.9 and ECG with diffuse T wave inversions. His chest x-ray reveals mild volume overload”

“Neurosurgery has been called, and they are on their way to see him. He is in ED Resuscitation Room 3, intubated and sedated now on propofol at 30

mcg/kg/min and fentanyl 50 mcg/h. His BP is 140/80 with no other treatment”

“They are ready to take him in Bed 12 in the NCCU in 5 min. Nursing is also calling report”

Subarachnoid Hemorrhage (SAH)

Blood within the subarachnoid space

Subarachnoid Hemorrhage (SAH) is most commonly produced by trauma and next most common by a ruptured intracranial aneurysm. For the latter, it is imperative that a timely diagnosis is made because the prevention of aneurysm re-rupture can be lifesaving.

Clinical Diagnosis of Subarachnoid Hemorrhage (SAH)

Clinical features

The diagnosis of **traumatic SAH** is based on history and brain imaging. The protocol for management of traumatic SAH can be found under the ENLS protocol Traumatic Brain Injury.

Aneurysmal SAH has a classic presentation though signs and symptoms may vary.

Classic presentation:

- Abrupt onset of a sudden, severe headache; onset is typically less than 1 minute.
- The headache is a NEW, QUALITATIVELY DIFFERENT headache for the patient.
- May have neck pain, nausea and vomiting.
- The patient may transiently lose consciousness, or present in coma.
- The nature and onset of the headache is the key distinguishing feature from other forms of stroke, syncope, and seizure.

Variant presentation:

- Headache is not reported as abrupt (the patient may not remember the event well).
- Headache responds well to non-narcotic analgesics or “anti-migraine” medications.
- Headache resolves on its own within hours.
- Approximately 40% of patients with SAH will have a normal neurological examination. They may or may not have meningismus (which may take time to develop). They do not necessarily appear acutely ill.

Key aspects of the neurological examination:

- Glasgow Coma Scale (GCS)
- Pupil exam
- Fundoscopic exam for retinal hemorrhages
- Neck exam for meningismus

Determine the clinical severity of the SAH using one of the scales below:

World Federation of Neurological Societies (WFNS) scale:

Grade 1: GCS 15

Grade 2: GCS 13-15 without neurological deficit

Grade 3: GCS 13-15 with neurological deficit

Grade 4: GCS 7-12

Grade 5: GCS 3-6

Subarachnoid Hemorrhage Protocol

↑ Flowchart ↑

Hunt-Hess Scale (increase by 1 grade for angiographic vasospasm or serious systemic illness):

Grade 1. Asymptomatic, mild headache, slight nuchal rigidity

Grade 2. Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy

Grade 3. Drowsiness / confusion, mild focal neurologic deficit

Grade 4. Stupor, moderate-severe hemiparesis

Grade 5. Coma, decerebrate posturing

Airway

Assess need for intubation

Factors contributing to necessity of intubation include:

- Insufficient airway protection
- Hypoventilation
- Hypoxemia
- Expected decompensation during transport within hospital or to another hospital

See ENLS protocol Airway, Ventilation and Sedation.

Prehospital Care

A variety of prehospital neurological examination tools, including the Cincinnati Prehospital Stroke Scale, Los Angeles Prehospital Stroke Screen, National Institutes of Health Stroke Scale, Miami Emergency Neurological Deficit Scale, and Glasgow Coma Scale, are used by emergency medical services personnel. For patients presenting with isolated headache who are neurologically intact, there are no specific prehospital interventions, apart from consideration of analgesics. For patients presenting with a headache and neurological deficits, pre-notification of the ED staff about the neurological deficits and the finger stick glucose are important first steps. Patients who are severely encephalopathic, comatose, or vomiting repeatedly may need to have their airway controlled by tracheal intubation in the field. Care at a certified comprehensive stroke center that can provide neurosurgical and endovascular consultation is recommended.

See ENLS protocol Acute Ischemic Stroke for prehospital protocol pertaining to SAH and other types of stroke.

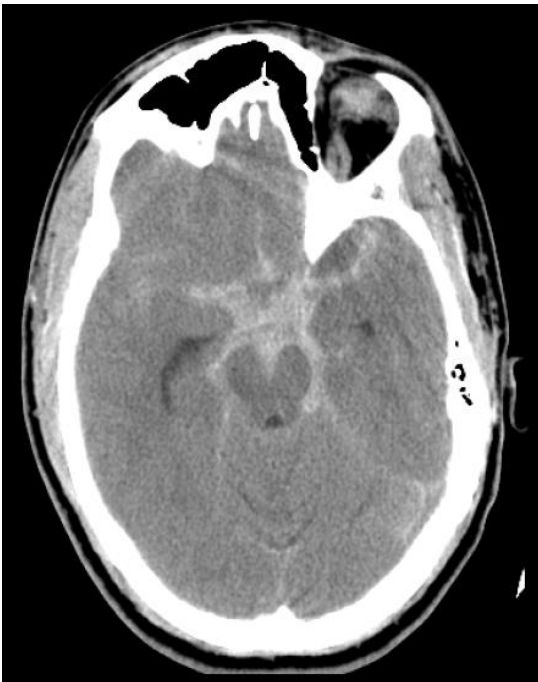
Brain Imaging for SAH

If you suspect SAH by history, head imaging is the next step

Non-contrast CT imaging of the brain is the gold-standard for identifying SAH (Class1, LOE B).

- However, CT imaging is more sensitive in the first hours following a SAH and becomes progressively less sensitive with the passage of time (so that by 3 days, it is approximately 85% sensitive). Besides time, other reasons for a false negative CT include anemia, low volume SAH and a technically poor scan.
- Some clinicians advocate for a CT angiogram (CTA) at the time of the CT scan to look for an intracranial aneurysm. Although this is helpful if an aneurysm is seen, the negative predictive value is less clear. One should not use a negative CTA alone to rule out SAH.
- MRI is useful in patients who are imaged a few days following the SAH; specific sequences can be used to image subarachnoid blood even several days later.

A CT image of an aneurysmal SAH is shown below:



CT Confirms SAH

Blood is seen on the CT scan

The diagnosis of SAH is confirmed and cerebrospinal fluid analysis is not necessary.

CT Scan is Negative - Perform Lumbar Puncture (LP) Next

Must perform an LP if the CT is negative

Recent data suggest that non-contrast CT imaging of the brain is close to 100% sensitive for SAH if all of the following are true:

- The patient has a classic presentation with a thunderclap headache,
- The CT is done within 6 hours of headache onset,
- The patient is completely neurologically intact, and
- The CT is read by an attending radiologist (or someone with equivalent experience reading brain CT scans)

If all of these criteria are met, clinicians can consider not doing an LP. The sensitivity of CT in these patients is ~ 99.5% (may miss a SAH in 1-2 patients per 1,000 who fulfill all of these criteria).

However, if these criteria are not met, one should perform an LP to determine if the patient has a radiographically occult SAH.

The LP is done to look for xanthochromia. Xanthochromia is the staining of CSF by heme breakdown products (chiefly bilirubin). It takes several hours for blood in the subarachnoid space to break down, so the presence or absence of xanthochromia is time dependent.

- If the CSF shows xanthochromia, the diagnosis of SAH is confirmed (be careful if the CSF protein exceeds 100 mg/dl as this can lead to a false positive).
- If the CSF is clear of RBCs and xanthochromia is absent, it is highly unlikely that the patient had a subarachnoid hemorrhage. However, a rapidly expanding aneurysm without subarachnoid rupture can present with a classic thunderclap headache, so if you still suspect an aneurysm on clinical grounds, emergent neurosurgical consultation is recommended.

In summary, the typical findings of SAH on cerebrospinal fluid analysis are:

- some RBCs
- < 5 WBCs
- WBC:RBC ratio 1:700
- Xanthochromia is present
- Minimal clearing of RBCs between tubes 1 and 4.

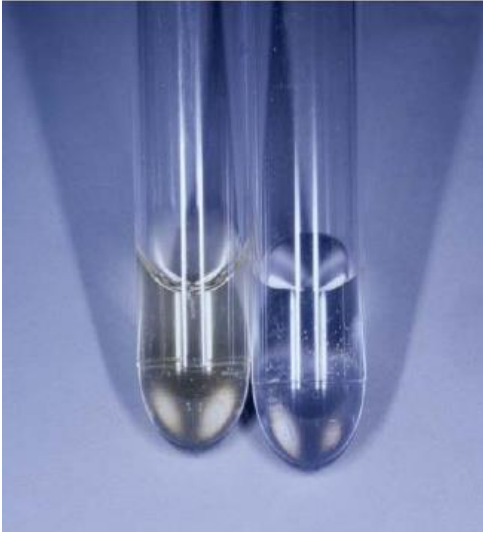
Note:

- The sensitivity of all tests for SAH are dependent upon the time from the bleed. CT is more sensitive early and less so with time. RBCs in the spinal fluid is also more likely to be seen early and will clear with time. Xanthochromia is absent early and nearly always present by 12 hours after the bleed.
- Spectrophotometry is more sensitive (but much less specific) for xanthochromia than is visual inspection (spin down CSF, compare to water in neutral light; see figure below); however visual inspection is the only test available at most hospital labs.

Subarachnoid Hemorrhage Protocol

↑ Flowchart ↑

Typical appearance of xanthochromia (left) compared to water (right). CSF is centrifuged first to take any RBCs out of solution.



SAH is Confirmed

CT or LP evidence of SAH

Diagnosis of SAH is confirmed. The goal is to reduce the chance of aneurysm re-rupture and expedite treatment of the aneurysm while preventing any medical complications.

Initial Orders

First steps

Once SAH is confirmed, *in addition to* the items outlined below (neurosurgical consultation, seizure prophylaxis, reverse coagulopathy, treatment of anxiety and pain, and blood pressure management):

- Bed rest with cardiac monitoring
- 12-lead electrocardiogram
- Labs
 - Blood: CBC, coagulation tests (prothrombin time, partial thromboplastin time, international normalized ratio), electrolytes, renal function tests, troponin, and a type and screen
 - Urine: toxicology screen

Neurosurgical Consultation

Definitive therapy is obliteration of the aneurysm, by either endovascular coiling or surgical clipping. Both of these therapies isolate the aneurysm from the cerebrovascular circulation and should be carried out as soon as feasible, ideally within the first 24 h of presentation.

Seizure Prophylaxis

Should one prescribe anticonvulsants now?

- Approximately 20% of SAH patients have seizures prior to hospital arrival, and another 5–10% experience seizures after admission.
- Early seizures may increase the risk of aneurysm re-rupture and elevated intracranial pressure (ICP).
- Acute seizures should be treated with antiseizure medications.
- In patients with persistent altered mental status, non-convulsive status epilepticus may be present, which can only be diagnosed by continuous electroencephalography (EEG).
- Both the AHA and NCS guidelines suggest consideration of antiseizure medications in the immediate post-hemorrhage period.
- A very short course of prophylactic antiseizure medications may be recommended in the period following diagnosis and before definitive aneurysm treatment because of a concern for seizure-related aneurysm re-rupture.
- As phenytoin may lead to worse long-term cognitive outcomes, the use of a different agent should be considered. Refer to ENLS Pharmacology module for more information.

Reverse Coagulopathy

Coagulopathy should be urgently treated.

- Patients taking Vitamin K antagonists including warfarin with an INR ≥ 1.4 should be treated with some combination of IV vitamin K (10 mg IV), and prothrombin complex concentrates.
- Fresh Frozen Plasma (FFP) is an alternative for reversal if PCC is unavailable.
- Thrombocytopenia (platelets $< 100,000$) can be treated with platelet transfusions.

See the ENLS Pharmacology protocol regarding reversal of Factor Xa and thrombin inhibitors.

For patients with SAH taking oral anti-platelet agents, such as aspirin, clopidogrel or prasugrel, NCS and SCCM management recommendations include:

- Anti-platelet agents can potentially increase the risk and severity of aneurysm re-rupture, as well as neurosurgical complications.
- Platelet transfusion is recommended for patients with aspirin- or adenosine diphosphate (ADP) inhibitor-associated SAH who will undergo a neurosurgical procedure.
- Platelet transfusion is not recommended if no neurosurgical procedure is planned.
- The risk–benefit ratio of anti-platelet therapy reversal using other hemostatic agents such as desmopressin (DDAVP) should be considered for individual patients.

Treat Pain and Anxiety

An uncomfortable patient can re-rupture his or her aneurysm

It is important to avoid straining, Valsalva maneuver, and writhing, as this can cause re-rupture of an unsecured aneurysm. One must also be careful to not over-sedate the patient as this could mask the symptoms of hydrocephalus (obtundation).

- Use IV medications with short half-lives (fentanyl for example).
- Liberal use of anti-emetics is justified especially if vomiting occurs.
- Blood pressure control is enhanced with adequate analgesia.
- If anxiety is present, consider small doses of an anxiolytic such as lorazepam.

Blood Pressure Management: SPB <160

Avoid hypertension to prevent re-rupture

General principles:

- Current guidelines suggest treating severe hypertension in patients with an unsecured ruptured aneurysm.
- Modest hypertension [mean arterial pressure (MAP) < 110 mmHg] may not require treatment.
- Premorbid BPs should be considered and used to inform the risks and benefits of treatment.
- Antihypertensive medications that are short acting, titratable, and can be administered as a continuous infusion, such as nicardipine or clevidipine, to reduce the systolic pressure < 160 mmHg, or the MAP < 110 mmHg, should be used, keeping in mind the principles mentioned above.
- Nitroprusside and nitroglycerine should be avoided because these agents may cause cerebrovascular dilation and thereby increase ICP.

See the ENLS Pharmacology protocol.

Decline in Neurological Exam

Worsening neurological examination?

There are several immediate causes of early (within the first hour) neurological decompensation.

- Re-rupture of the aneurysm: repeat head CT is diagnostic
- Worsening hydrocephalus: repeat head CT is diagnostic; need for external ventricular drain (EVD) is now paramount; give mannitol while arranging for EVD placement
- Seizure: treat with phenytoin or levetiracetam load
- Cardiopulmonary cause: neurogenic pulmonary edema, catecholamine cardiomyopathy manifesting with worsening hypoxia or hypotension. Echocardiography is diagnostic of cardiomyopathy.

Hydrocephalus

Are the ventricles dilated?

Hydrocephalus is caused by blockage of CSF absorption and is diagnosed by interpreting the head CT scan. If the patient is obtunded or comatose, it is important to provide ventricular drainage by having an external ventricular drain placed. This both treats the hydrocephalus and provides a monitor of ICP.

- If a neurosurgeon is not available at your hospital and hydrocephalus is present, consider treating the patient with mannitol 1 gm/kg and expediting transfer to a facility with neurosurgical capability within the next hour.

Consider Antifibrinolytic Agents

Preventing re-rupture

Preventing re-rupture of the aneurysm is a major goal of initial therapy.

- Antifibrinolytic agents such as aminocaproic acid and tranexamic acid can reduce aneurysmal re-rupture. However, these agents also raise the risk of deep venous thrombosis (DVT), pulmonary embolus (PE), and ischemic stroke if they are continued. If the patient is free of recent myocardial infarction, DVT/PE or any known hypercoagulable state, many centers administer antifibrinolytic agents until the aneurysm can be secured; this may be an appropriate strategy but should be discussed with the consultant.