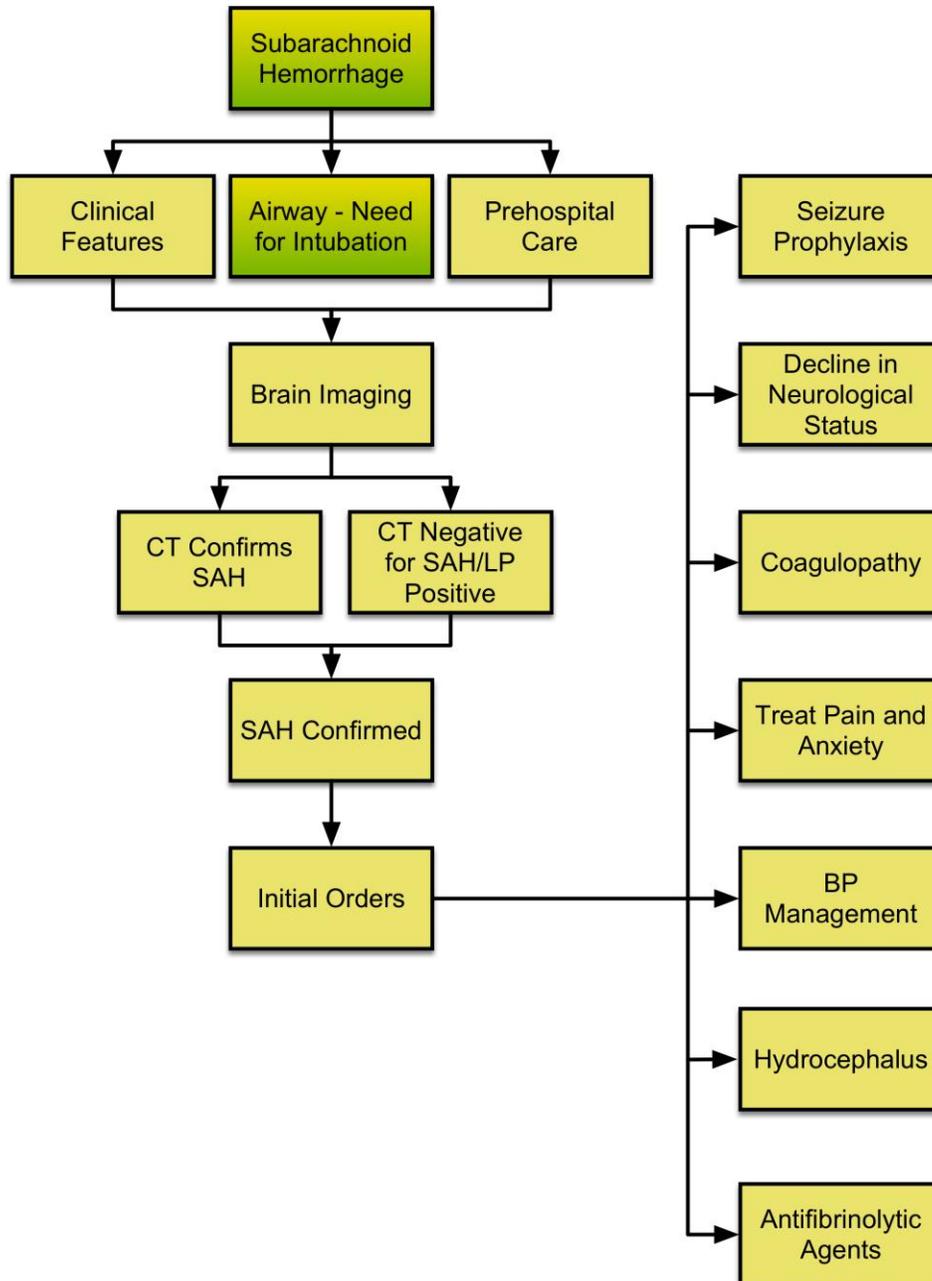


# Emergency Neurological Life Support

## Subarachnoid Hemorrhage

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[Checklist & Communication](#)

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## Checklist

- Brain Imaging
- Labs: PT/PTT, CBC, platelets, electrolytes, creatinine, troponin, toxicology screen
- 12 lead ECG
- Blood pressure goal established
- Address hydrocephalus

## Communication

- Airway status
- Clinical presentation (level of consciousness, motor exam, pupil exam)
- WFNS score and Hunt-Hess Grade
- Imaging/LP results
- Coagulopathy present?
- Hydrocephalus present?
- Medications given (dose and time administered), including sedative, analgesics, seizure prophylaxis, anti-hypertensives and nimodipine
- Coordination of other vascular imaging



## Antifibrinolytic Agents

### Preventing re-rupture

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

- Antifibrinolytic agents such as amicar 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 strategy appears safe (Hillman et al, J Neurosurg (2002) 97:771).



## Blood Pressure Management

### Avoid hypertension to prevent re-rupture

#### General principles:

- Precise guidelines for BP management do not exist (see Bederson *et al*, Guidelines for the management of aneurysmal SAH; Stroke. 2009 40:994)
- Many specialists recommend SBP < 140 mmHg in a patient with no history of hypertension. SBP > 160 mmHg has been associated with aneurysmal re-rupture, and over treatment of BP can lead to brain ischemia (especially if hydrocephalus is present).
- Use short acting, titratable medications such as labetalol or nicardipine
- Avoid long-term nitroprusside due to concern of raising ICP



## 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 physicians advocate a 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 a SAH is shown below





## 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.

N 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 Examination Features:

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

Determine the clinical severity of the subarachnoid hemorrhage using one of the scales below:

World Federation Neurological Scale (WFNS):

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

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

## Coagulopathy

### Elevated INR or low platelets?

For platelet count  $< 50 \times 10^9/l$ , administer 6-pack of platelets.

Consider vitamin K antagonist reversal with purified factor concentrates or FFP if warfarin or other vitamin K antagonists have been prescribed, followed by vitamin K 10 mg IV. 4-factor prothrombin complex concentrates (PCC) are preferred to FFP. To calculate the volume of plasma or IU of prothrombin complex concentrate:

1. Decide on target INR
2. Convert INR to percent (%) functional prothrombin complex:

INR Range	Percent function prothrombin complex
> 5	5%
4.0 – 4.9	10%
2.6 – 3.9	15%
2.2 – 2.5	20%
1.9 – 2.1	25%
1.7 – 1.8	30%
1.4 – 1.6	40%
1.0	100%

3. Calculate dose:  
 (Target in %PC - Current level in %PC) X weight (kg) = mL of FFP or IU of PCC needed  
 Example: a patient with INR on arrival = 7.5, target INR 1.5, body weight = 80 kg:  
 $(40-5) \times 80 = 2,800$   
 Therefore, the needed dose is 2,800 mL of FFP or 2,800 IU of PCC.

Reference: Schulman, S. Care of patients receiving long-term anticoagulant therapy. NEJM (2003) 349:675

For patients with ICH who have taken dabigatran, idarucizumab may be used to reverse the anticoagulant effects of dabigatran. The recommended dose of idarucizumab is 5 g, provided as two separate vials each containing 2.5 g/50 ml idarucizumab. If idarucizumab is not available consider rVIIa 80 mcg/kg.

While no specific reversal agents exist for direct Xa inhibitors, one could consider activated charcoal if last dose was within 8 hours; however this may lead to aspiration depending on mental status and vomiting. Consider PCC 30 IU/kg for rivaroxaban or apixaban. FFP and vitamin K are not effective.

See also ENLS reference [Pharmacotherapy](#) for complete dosing of reversal agents.



## **CT Scan Confirms SAH**

### **Blood is seen on the CT scan**

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



## CT Scan is Negative - Do LP Next

### Must do an LP if the CT is negative

Recent data suggest that non-contrast CT imaging of the brain is very 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 onset of the headache,
- 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 bullet points are present, physicians 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 a lumbar puncture (LP) to make sure the patient does not have a radiographically occult hemorrhage.

The LP is done to look for xanthochromia. Xanthochromia is the staining of CSF by heme breakdown products (chiefly bilirubin) by ependymal xanthine oxidase. 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 be 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 suggested.

Stated otherwise, the typical findings of SAH on spinal fluid analysis are:

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

Atypical or inconclusive findings:

- Clearing of RBCs from tube 1 to 4 (perhaps because the spinal needle caused venous bleeding "traumatic tap") Note: if blood is present in Tube 1, a strategy to help distinguish between a traumatic tap and true SAH is to waste several cc's of

CSF between the first and last tubes, in order to maximize the likelihood of complete clearing.

- RBCs present in similar number in Tubes 1 and 4 from a LP that was done within the first 4 hours of the headache (could be SAH or traumatic tap)
- Xanthochromia is absent, and the LP was done more than 12 hours following the onset of headache (likely traumatic tap)
- Excessive WBCs (ratio WBC:RBC > 1:700) suggesting meningitis or encephalitis

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 they 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.

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





## 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 by a neurosurgeon or neurointensivist. This both treats the hydrocephalus and provides a monitor of ICP.

- If you do not have a neurosurgeon 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.



## Initial Orders

### First steps

Once SAH is diagnosed, take these first steps:

- Bed rest (Class 2B, LOE B)
- Obtain pre-operative labs: CBC, Platelets, PT/PTT, electrolytes, BUN, Cr, cardiac enzymes
- 12-lead ECG
- Cardiac telemetry
- Nimodipine 60 mg po/ng (watch for hypotension); this is not an urgent medication and may require placement of an NG tube depending on the patient's mental status.
- AED administration until aneurysm is secured.



## Intubation

### 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](#).



## Neurological Exam has Declined

### 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.



## Prehospital Issues Regarding SAH

### Prior to hospitalization

See ENLS protocol [Acute Stroke](#) for a prehospital protocol pertaining to SAH and other forms of stroke.



## **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.



## Seizure Prophylaxis

### Should one prescribe anticonvulsants now?

Use of prophylactic anticonvulsants is controversial.

- Pro: seizures following SAH and before definitive aneurysm treatment have been associated with aneurysm re-rupture, and can raise ICP.
- Con: Phenytoin use has been associated with worse cognitive outcomes

One strategy is to administer a loading dose of phenytoin in the ED, and continue it until the aneurysm is secured, then stop the medication unless seizures have occurred (Class 2B, LOE B).



## 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 life saving.

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## Treat Pain and Anxiety

### **An uncomfortable patient can re-rupture their aneurysm**

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

- Use IV medication 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 seems to be the major issue, consider small doses of an anxiolytic such as lorazepam.