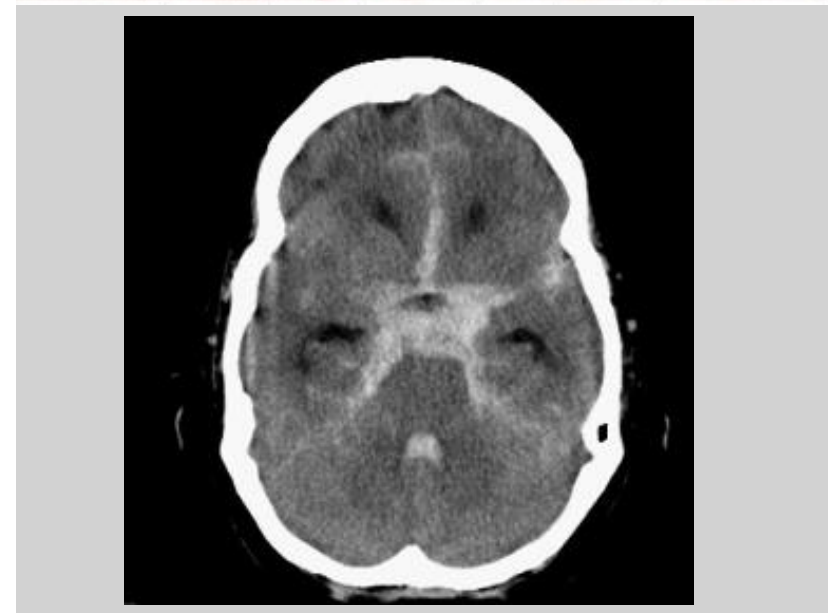
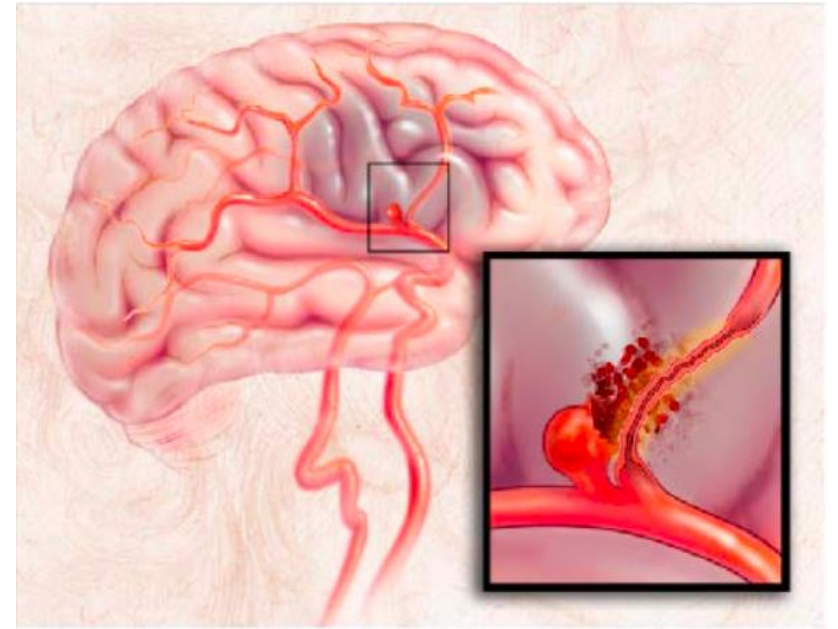


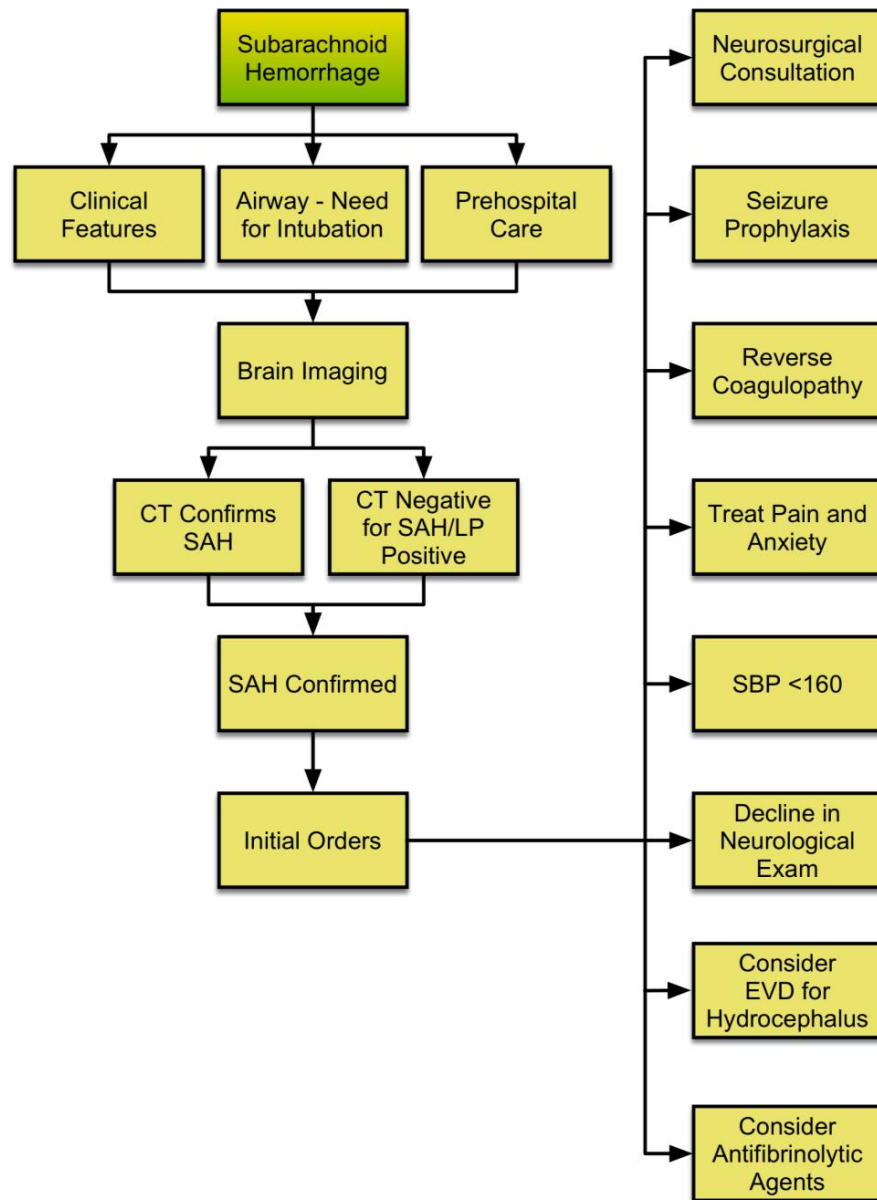
Thunderclap Headache and Subarachnoid Hemorrhage

W David Freeman, MD

No conflicts of interest or disclosures



Subarachnoid Hemorrhage



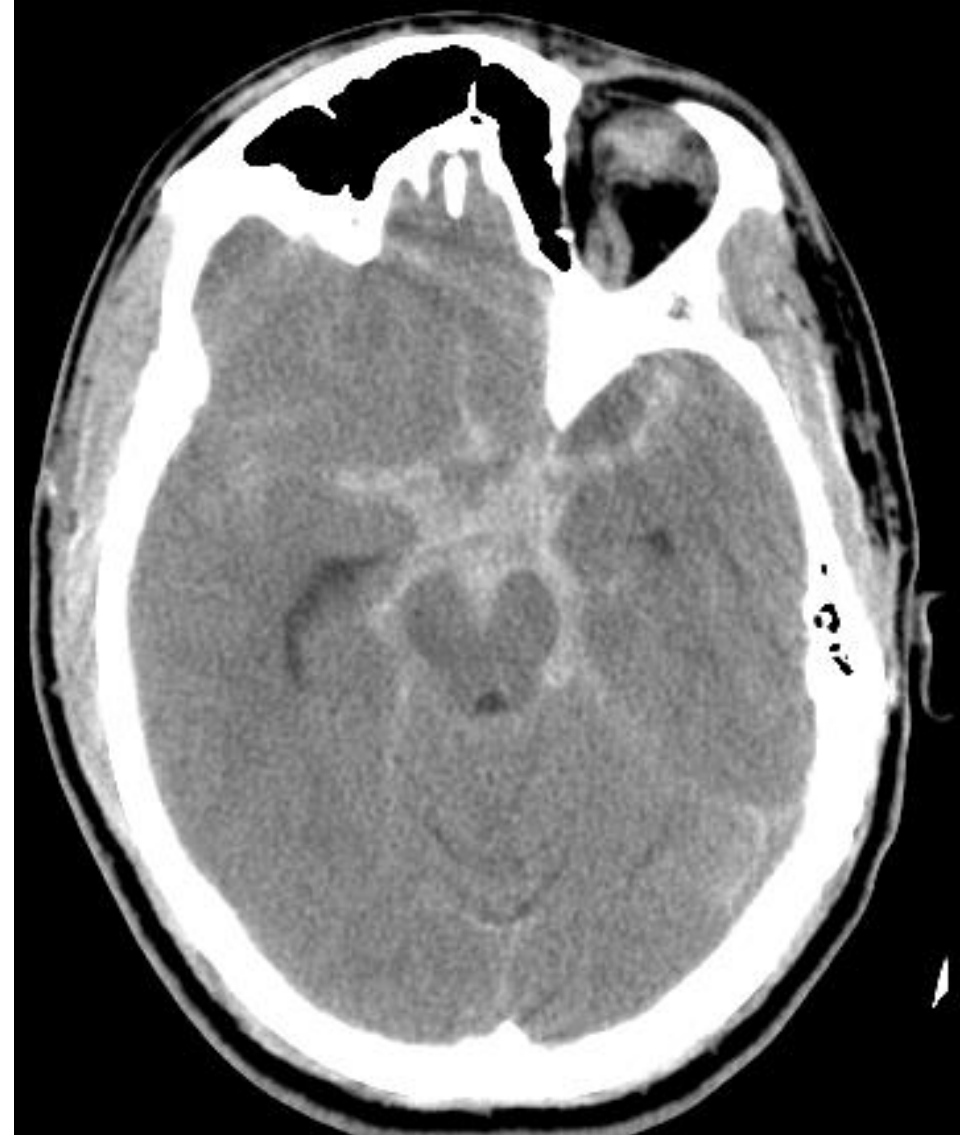
Subarachnoid Hemorrhage

Objectives

- Recognize the symptoms (thunderclap headache) and signs of SAH
- Initiate workup for suspected SAH
- Initial management of SAH patient

Case

- 39 year old woman presents with severe headache, uncertain time of onset, associated with nausea
- PMH: long history of migraine headaches
- Meds:
 - Reports taking ibuprofen prior to ED, with moderate improvement of headache
 - Taking warfarin for a pelvic DVT - year prior during pregnancy
- Exam: sleepy, yet follows commands, left eye droop and dilated pupil, moving all extremities
- BP 160/95 mmHg, RR 24/min, HR 98/min
- Breathing shallow and rapid, airway protection uncertain



Initial Checklist in Emergency Dept.

☐ Brain Imaging

☐ Labs: PT/PTT, CBC, electrolytes, BUN, Cr, troponin, toxicology screen

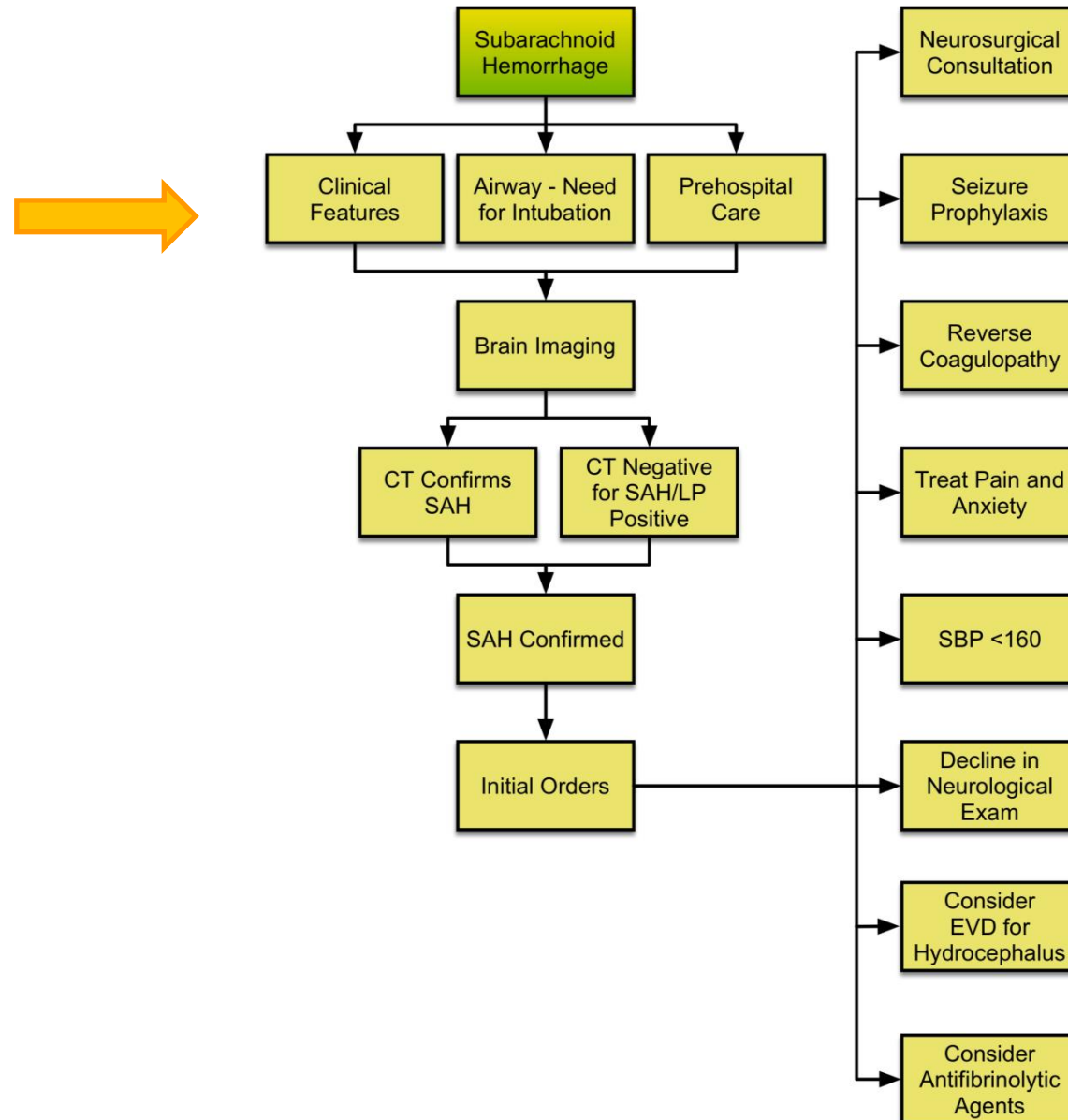
☐ 12 lead ECG

☐ Blood pressure goal established

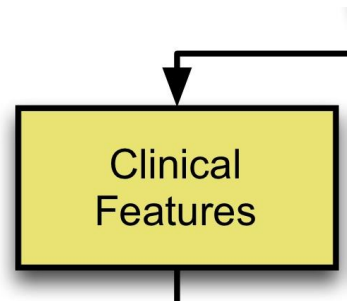
☐ Consult neurosurgery

☐ Address hydrocephalus

SAH Algorithm

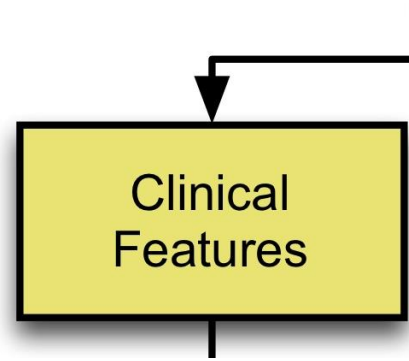


SAH Symptoms and Signs



CLASSIC	NOT-SO-CLASSIC
Abrupt onset of severe headache (HA), i.e. thunderclap	HA is not reported as abrupt (patient may not remember event well)
“Worst or first” headache of one’s life that is instantaneously maximal at onset (“thunderclap” after lightening strike)	HA responds well to non-narcotic analgesics
May have nausea, vomiting and neck pain	HA resolves on its own in few hours
May transiently lose consciousness, present in coma, or have focal deficits	40% patients with aneurysmal SAH will have normal neuro exam without meningismus
Nature of HA onset distinguishes from other forms of stroke	Do not necessarily appear acutely ill

SAH : Signs (Exam)



Key Exam Features:

GCS

Pupil exam

Fundoscopic exam for vitreous/retinal hemorrhages (Terson's syndrome)

Neck exam for meningismus (versus neck pain)

Hunt and Hess or WFNS score

SAH: Clinical Severity Scales

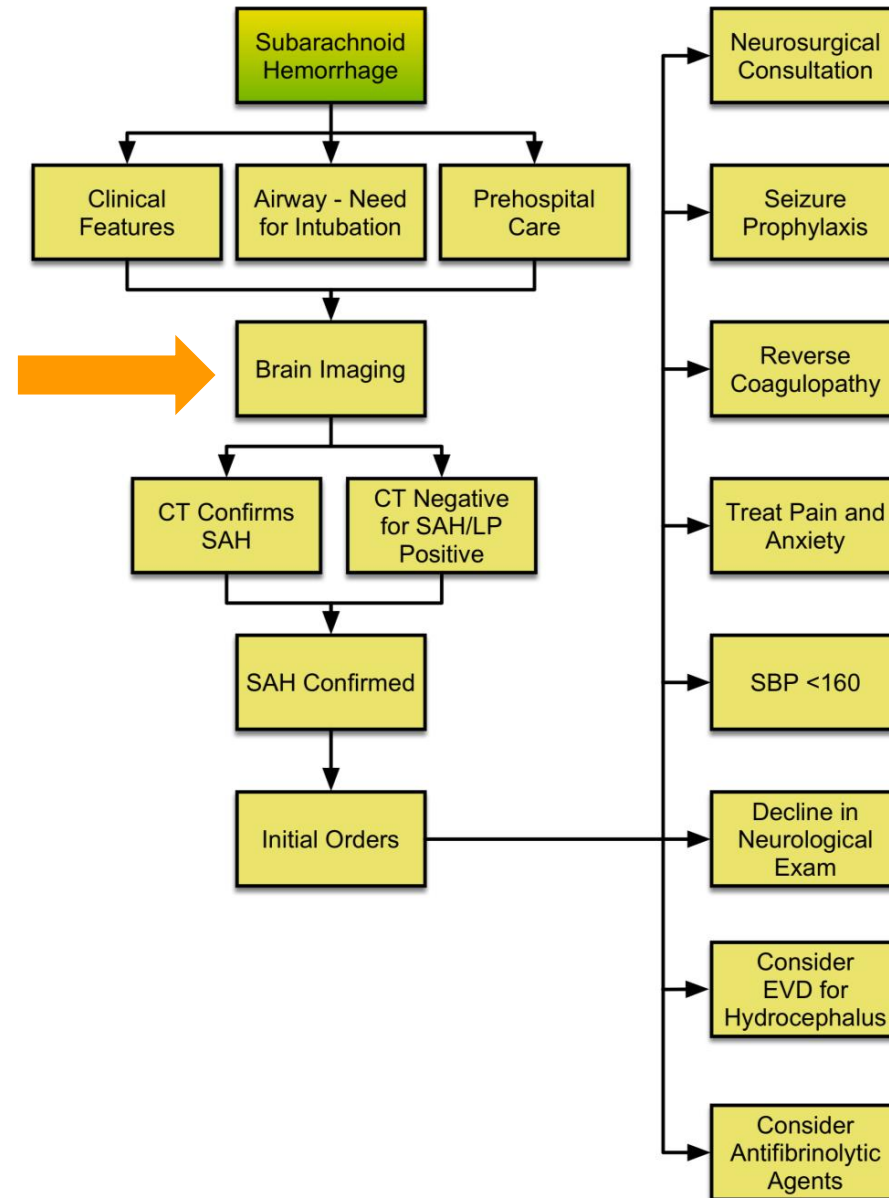
World Federation Neurological Scale

Grade	Criteria
1	GCS 15
2	GCS 13-14, without neurological deficit
3	GCS 13-14, with neurological deficit
4	GCS 7-12
5	GCS 3-6

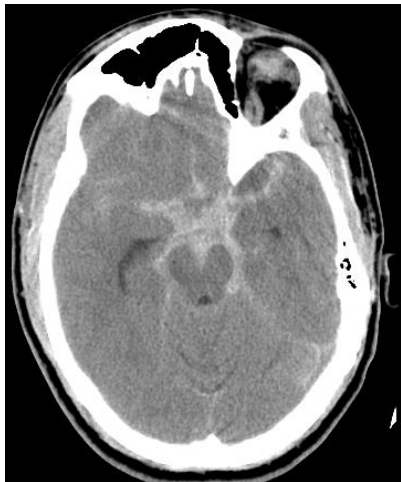
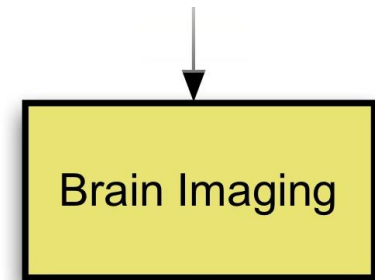
Hunt & Hess Clinical Grading Scale

Grade	Criteria
1	Asymptomatic, mild headache, slight nuchal rigidity
2	Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy
3	Drowsiness / confusion, mild focal neurologic deficit
4	Stupor, moderate-severe hemiparesis
5	Coma, decerebrate posturing

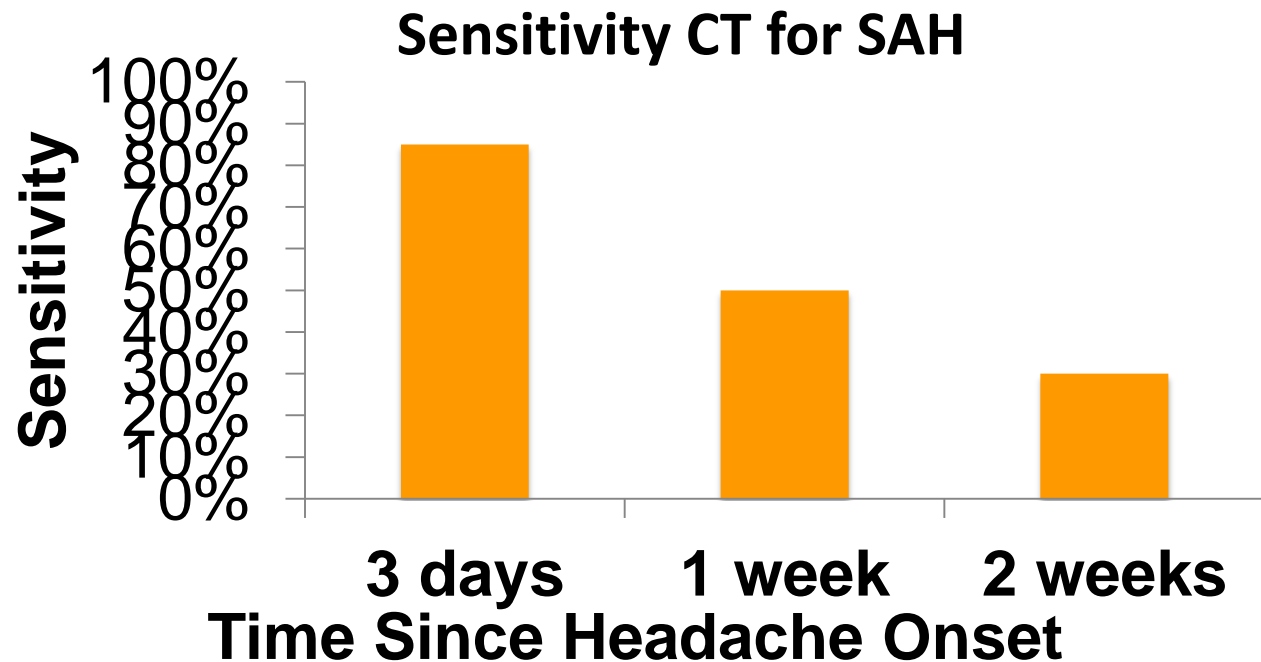
SAH-What's Next?



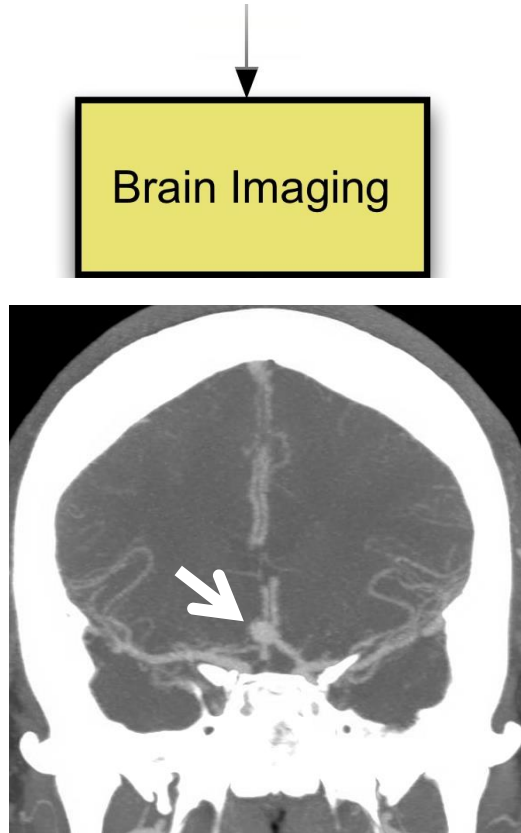
SAH Brain Imaging



- Non-contrast CT imaging of the brain is the gold-standard for identifying SAH with sensitivity of 95-100% if:
 - Classic presentation with thunderclap HA
 - CT completed within six hours of HA onset
 - The patient is completely neurologically intact
 - The CT is read by an attending radiologist
- Sensitivity of CT decreases with time
- Falsely negative CT: time, anemia (HCT <30) low volume SAH, and a technically poor scan



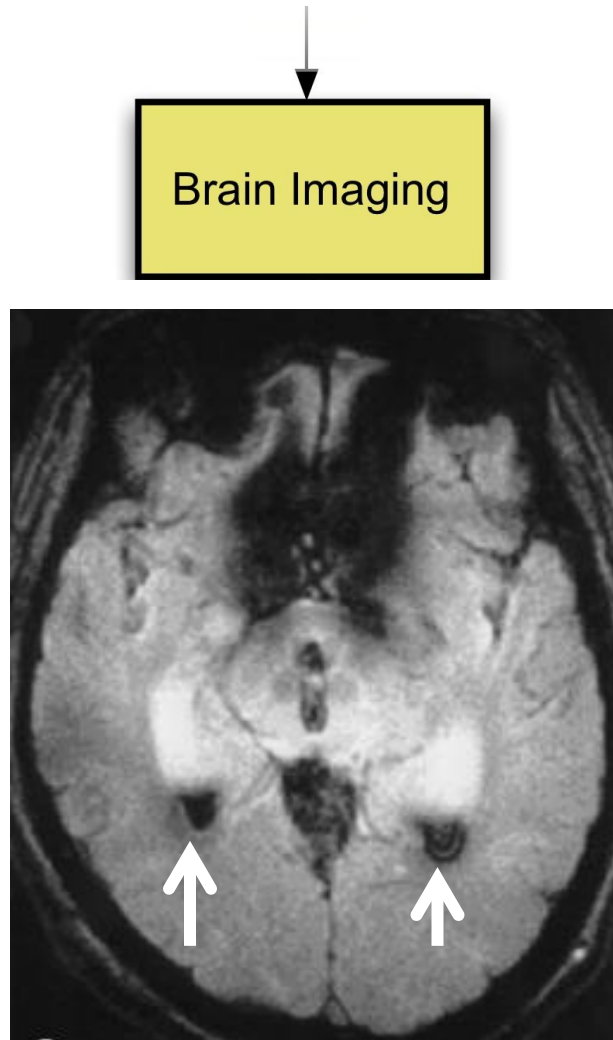
SAH: CTA Brain Imaging



CTA → ACOM aneurysm

- Some physicians advocate a CTA at the time of the CT scan to look for an intracranial aneurysm
- Caution regarding renal function and excessive contrast administration
- Although CTA 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 aneurysmal SAH

SAH Other Brain Imaging

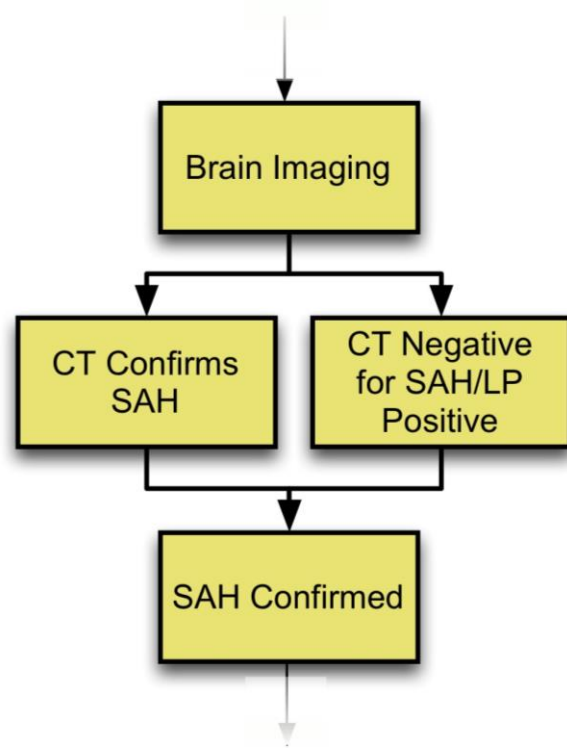


MR Hemosiderin Sequence

- MRI can be useful in patients who are imaged a few days or week or longer following the SAH
- Specific sequences (GRE, SWI, FLAIR) can be used to image subarachnoid blood even several days later

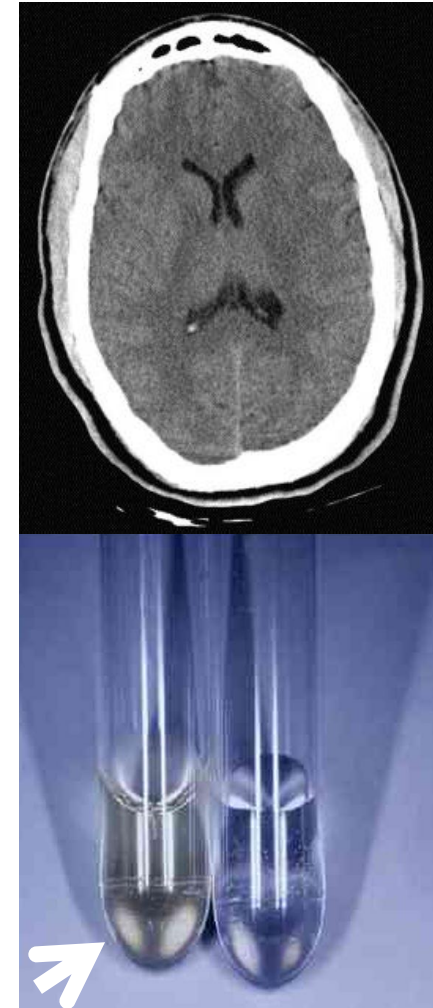
ENLS: SAH

Lumbar Puncture



- Must perform LP if CT is negative and history suggests SAH
- Rationale for LP is to confirm xanthochromia-staining of CSF by heme breakdown products
- Presence of xanthochromia is time dependent- takes several hours to develop

Head CT (-)

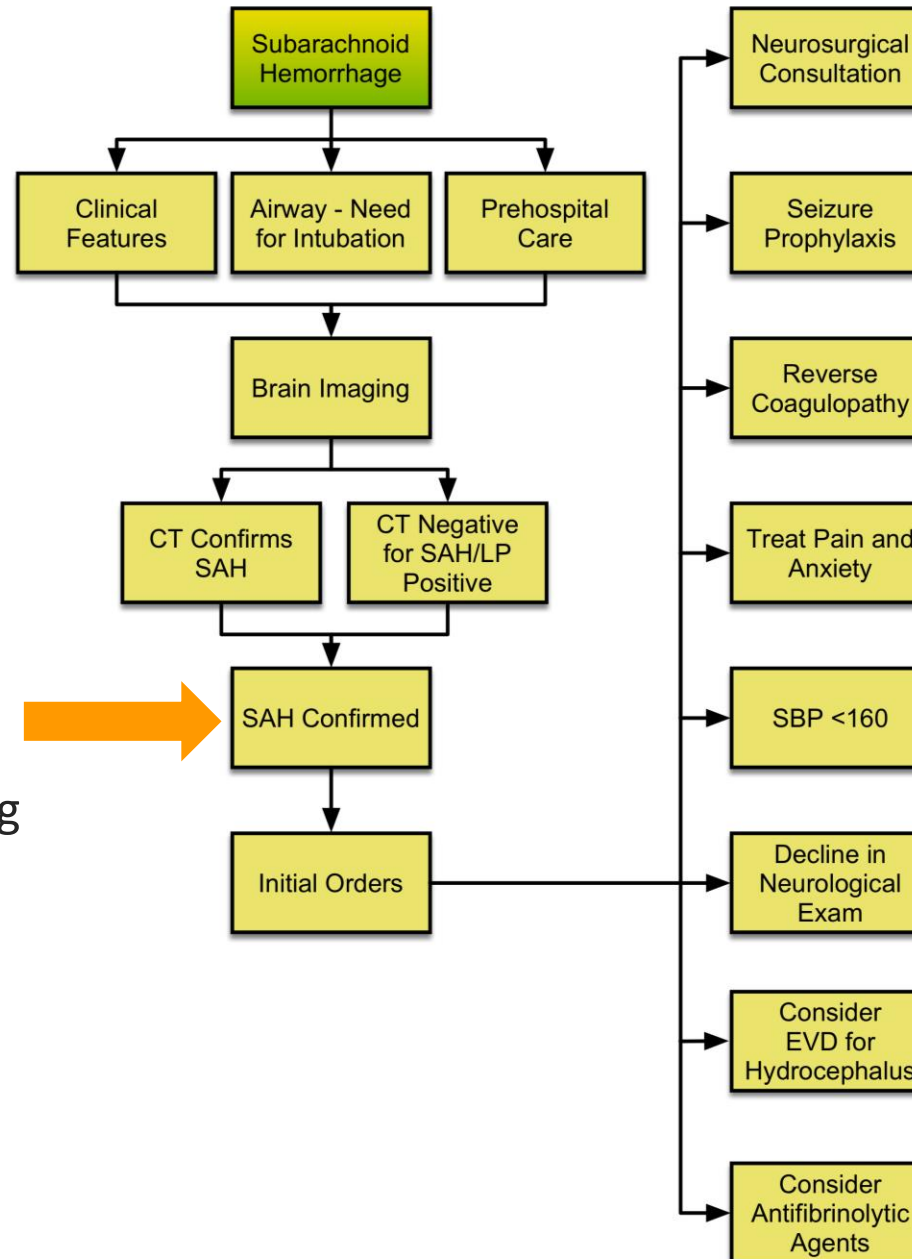


ENLS: SAH

Lumbar Puncture

Typical LP Findings	Atypical or Inconclusive	Not suggestive of SAH
↑ RBCs, No clearing from tube 1→ 4	Clearing of RBCs from tube 1→ 4	CSF clear of RBCs
< 5 WBC, WBC:RBC ratio 1:700	↑ WBC:RBC ratio suggest another process, meningitis or encephalitis	Occasionally, rapidly expanding unruptured aneurysm may present with HA, recommend urgent consultation
Xanthochromia present (However if CSF Protein >100mg/dL may be false positive)	Xanthochromia absent (Assuming LP is done more than 12 hours following headache onset).	Xanthochromia absent
Opening pressure elevated (~2/3 patients)	OP normal	OP normal

SAH

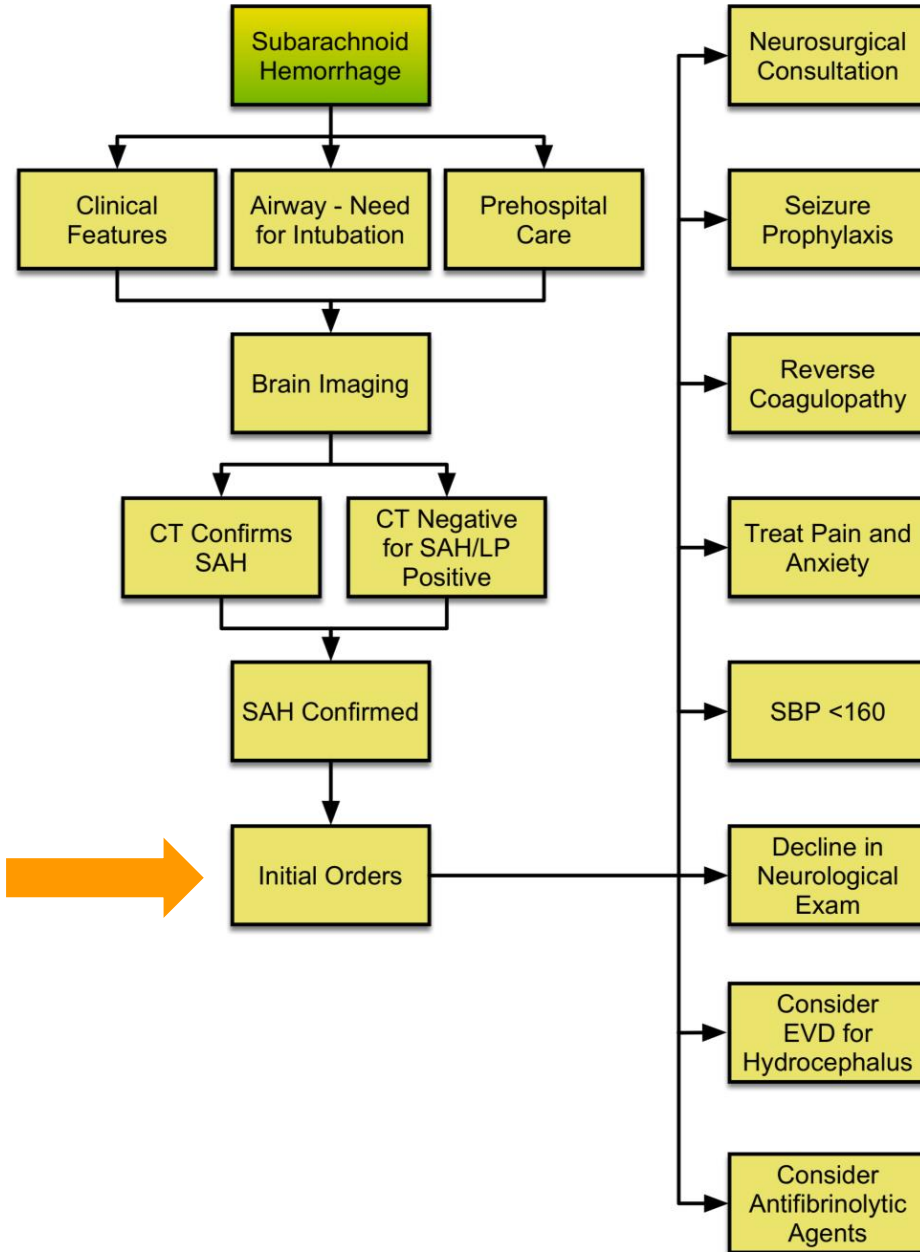


Diagnosis of SAH

confirmed. The goal is to reduce the chance of aneurysm re-rupture and expedite treatment of the aneurysm while preventing or minimizing medical or neurologic complications

SAH

Management →

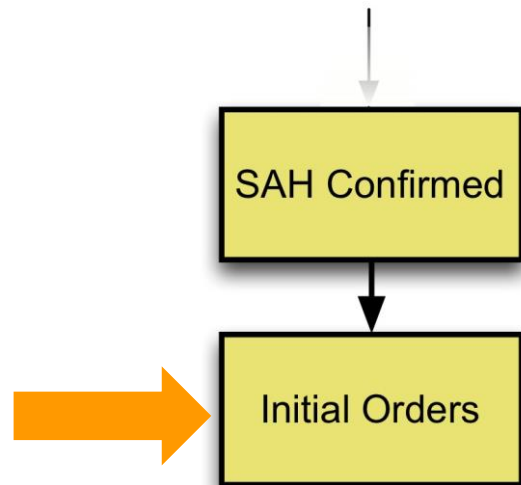


SAH

Initial Orders

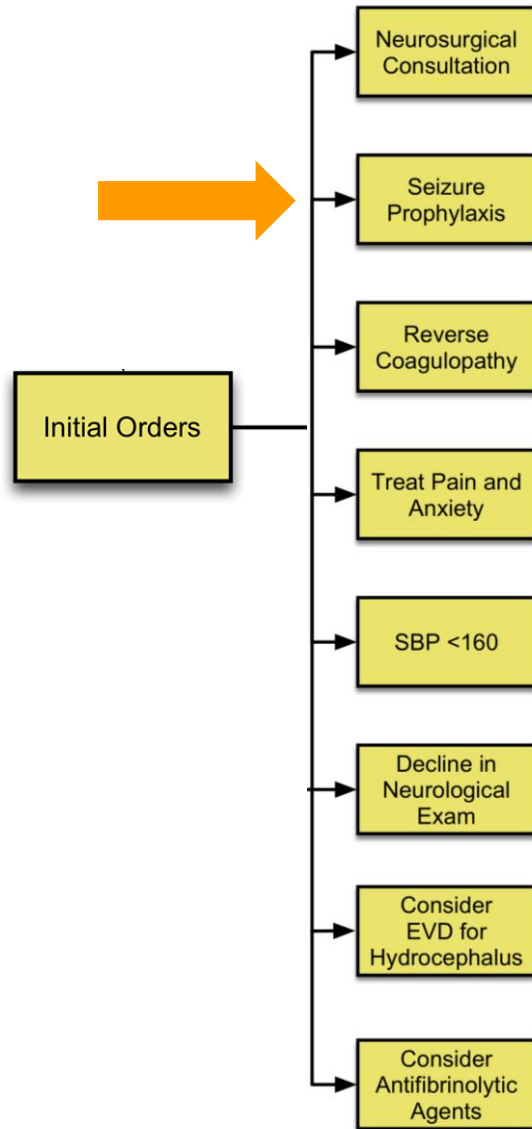
Once SAH is diagnosed, take these first steps:

- Bed rest
- Obtain pre-intervention labs: CBC, Platelets, PT/PTT, INR, electrolytes, BUN, Cr, cardiac enzymes
- 12-lead ECG
- Cardiac telemetry
- Nimodipine 60 mg po/ng (watch for hypotension)
- AED until aneurysm secured
- Consult Neurosurgery



SAH

Seizure Prophylaxis & Management



Prophylactic Anticonvulsants- Controversial

- **Pro:** seizures following SAH and prior to definitive treatment can be associated with re-rupture and can raise ICP
- **Con:** phenytoin use has been associated with worse cognitive outcomes in SAH
- 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
- Although frequently used by many centers there is limited data on use of alternative anticonvulsant agents such as levetiracetam

Active Treatment of Seizures

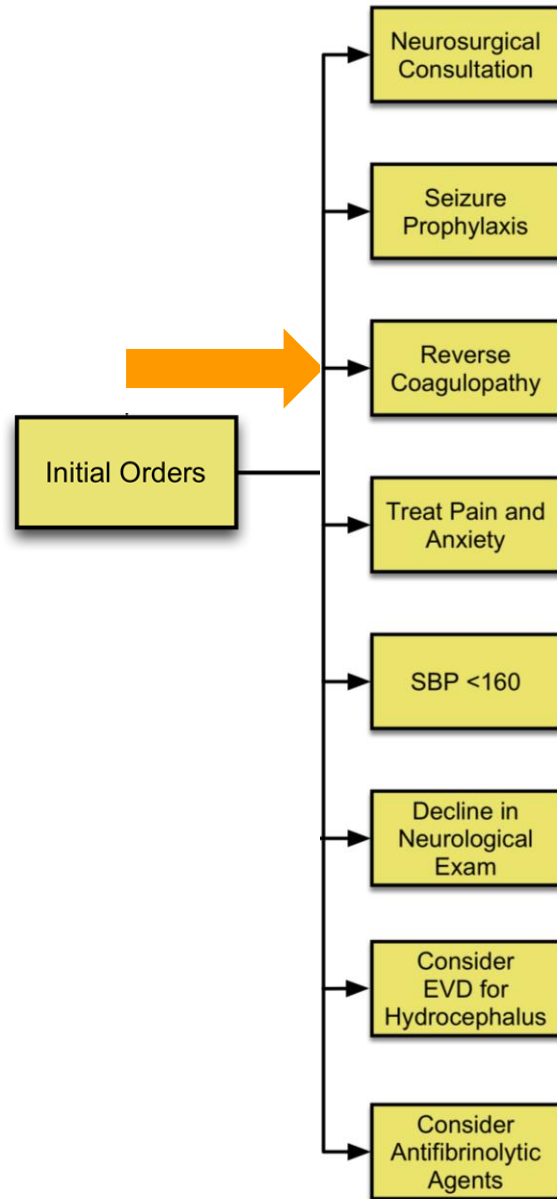
- Administer lorazepam for acute seizure management
- Administer loading dose of phenytoin
- More recently, many centers use levetiracetam

SAH

Coagulopathy

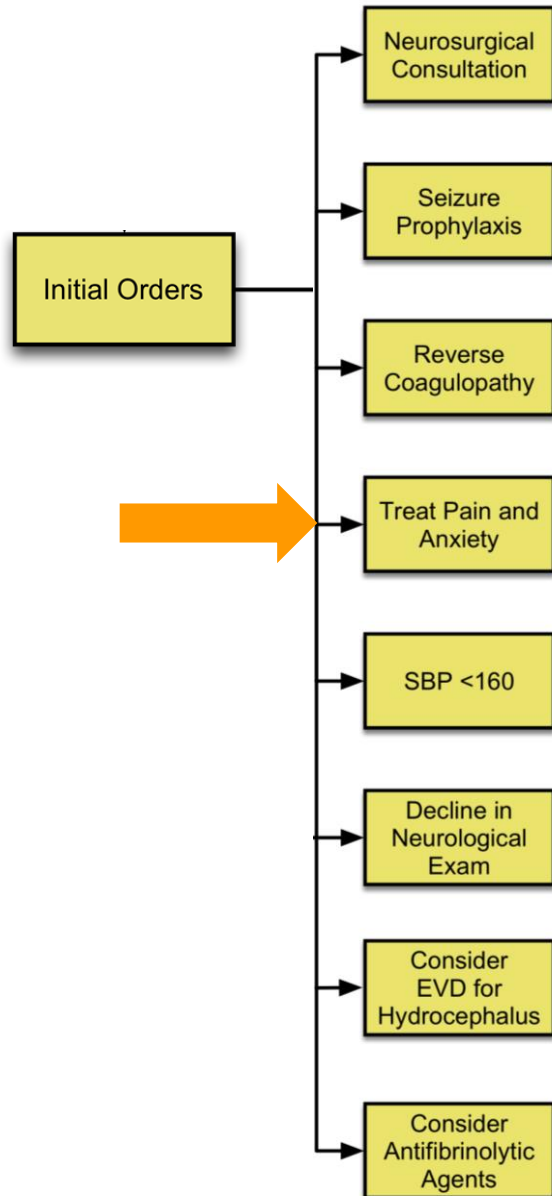
Correct underlying coagulopathies

- Goal INR < 1.4
- Goal platelets > 50,000
- Consider platelet transfusion for those on anti-platelets
- See ENLS pharmacology manuscript



SAH

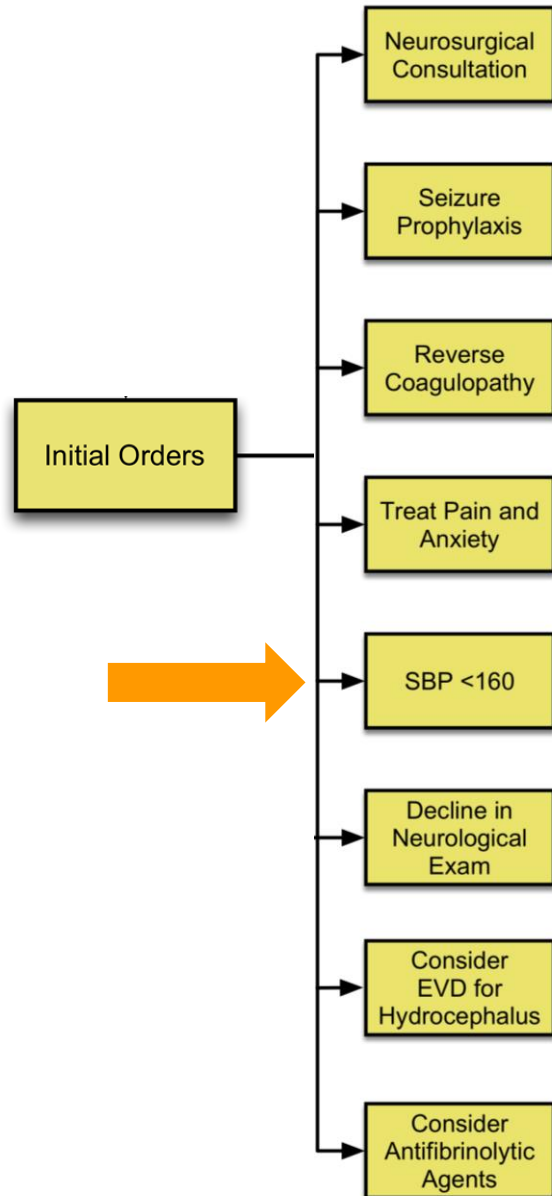
Treat Pain and Anxiety



- It is important to avoid straining, Valsalva, and writhing, as they can potentially contribute to re-rupture of a tenuous aneurysm
- One must also be careful to not over-sedate the patient as one could mask the symptoms of hydrocephalus
- Use IV medication with short half-lives (fentanyl for example)
- Liberal use of anti-emetics is justified especially if vomiting occurs
- BP control is enhanced with adequate analgesia
- If anxiety seems to be the major issue, consider small doses of an anxiolytic such as lorazepam

SAH

BP Management



- Precise guidelines for BP management in SAH unfortunately do not exist
- Retrospective data suggest higher rates of re-bleeding with SBP > 160 mmHg
- Over treatment of BP can potentially lead to brain ischemia - especially if hydrocephalus or vasospasm is present.
- Pre-morbid BP should be taken into considerations
- **Experts recommend to aim for SBP < 160 mmHg, or MAP < 110 mmHg,** keeping principles above in mind
- Use short acting, titratable intravenous medications such as beta blockers or nicardipine.
- Avoid long-term nitroprusside due to concern of raising ICP

Case

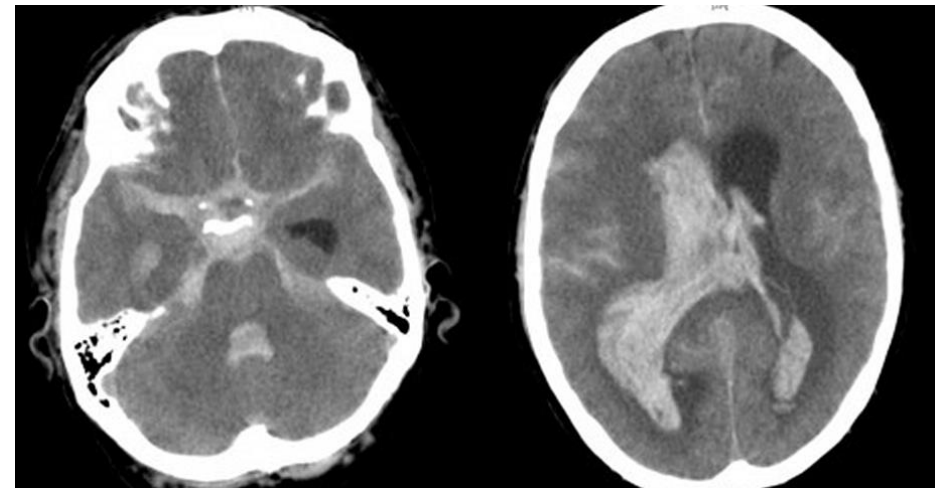
Back to our 39 y/o woman with a SAH

- Patient's level of arousal abruptly declined requiring immediate intubation
- Blood pressure spiked to 220/115 mmHg
- Both pupils transiently dilated
- Repeat Head CT demonstrated a re-bleed associated with acute hydrocephalus
- Upon return from CT, patient had a generalized tonic-clonic seizure, requiring treatment with intravenous lorazepam

Initial CT

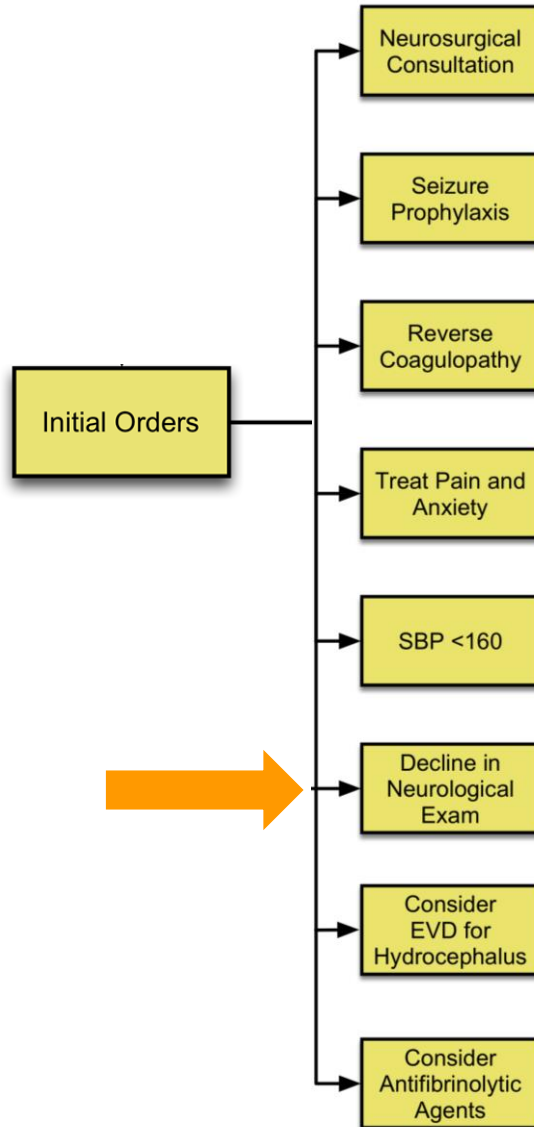


CT Following Neurologic Deterioration



SAH

Decline in Neurological Status



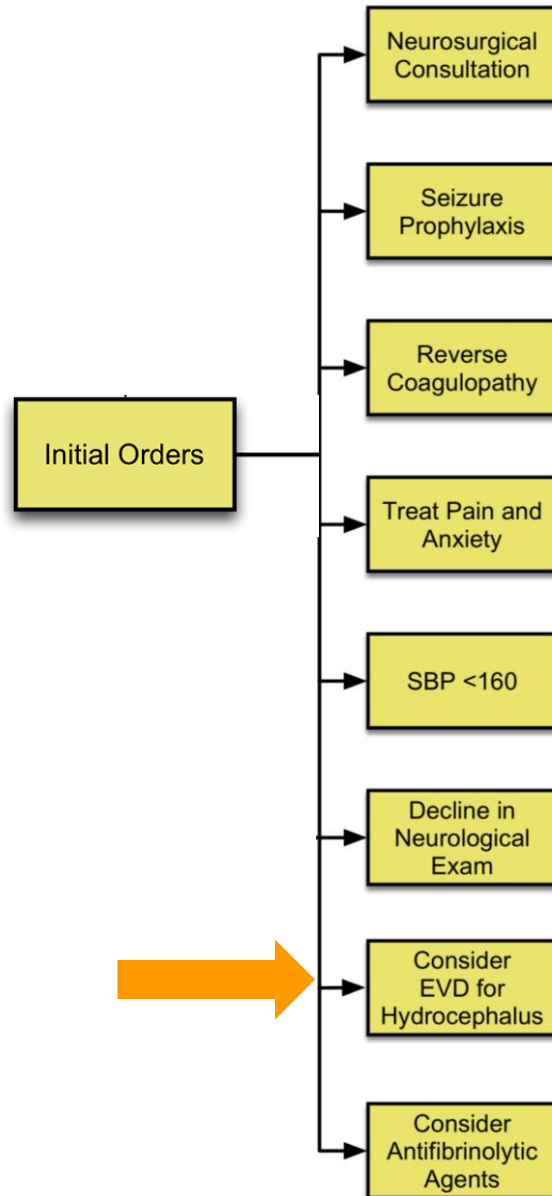
Causes of Decline in Neurological Status

- Acute Re-rupture of the aneurysm

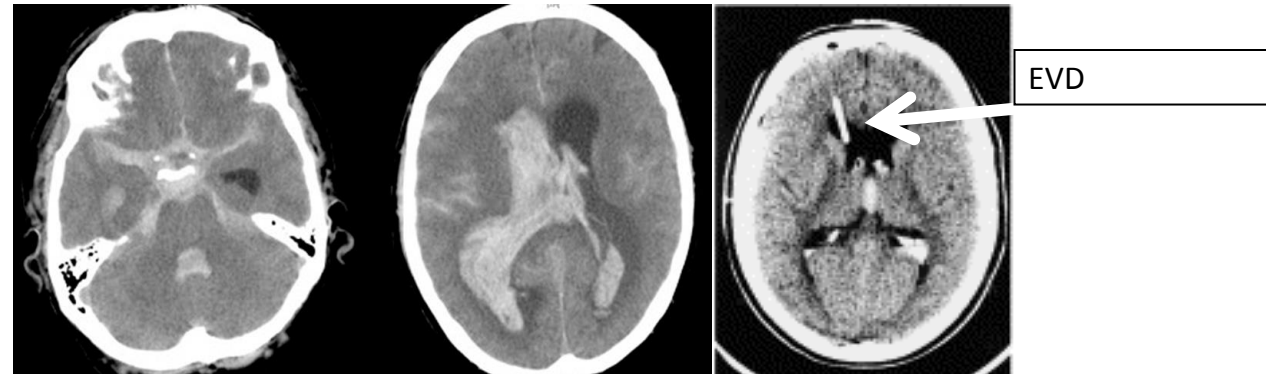
Re-rupture estimate 12-15% in the initial 24 hours

- Acute Hydrocephalus
- Seizure
- Cardiopulmonary complications
 - Neurogenic pulmonary edema
 - Neurogenic stress cardiomyopathy
 - Note cardiovascular collapse may be a sign of cerebral herniation

SAH Hydrocephalus

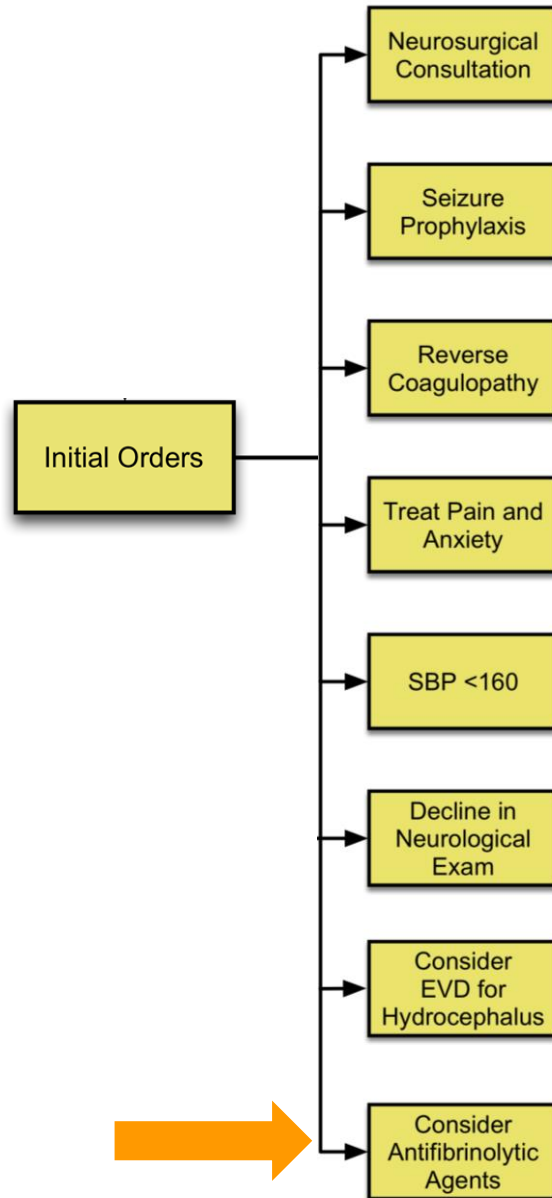


- Hydrocephalus is caused by blockage of CSF circulation and absorption within the ventricular system +/- increased CSF production and is readily diagnosed by the head CT scan
- If a patient is obtunded or comatose, a ventricular drain (EVD) can be placed. This treats the hydrocephalus and provides a monitor of ICP
- If a neurosurgeon is not available:
 - Transfer to facility with NS capabilities
 - Consider mannitol 1 gm/kg or a bolus of hypertonic saline in interim



SAH

Anti-fibrinolytic Agents



- Preventing re-rupture of the aneurysm is a goal of initial SAH management
- Antifibrinolytic agents such as ϵ -aminocaproic acid and tranexamic acid can reduce aneurysmal re-rupture
- These agents also raise the risk of thrombosis such as DVT, PE, and ischemic stroke, if they are continued beyond the acute SAH period
- If there is an unavoidable delay in obliteration of the aneurysm and the patient is free of recent MI, DVT/PE, or any known hypercoagulable state, many centers administer a time-limited course (< 72 hours) of antifibrinolytic agents until the aneurysm can be secured
- Early and LIMITED use (<72 hours) of these agents appear to be safe

Checklist for SAH

- ☐ Airway status
 - ☐ Hemodynamic Status and Blood Pressure Control
 - ☐ Clinical presentation (level of consciousness, motor exam, pupil exam)
 - ☐ WFNS 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
-



Questions?