**Aneurysmal SAH**

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**Epidemiology of aSAH**

- Incidence 9.7 to 14.5 per 100,000 in the United States.
- About 30,000 yearly in North America.
- Mean age of onset is 55 years.
- Most aneurysmal SAH occurs between 40 and 60 years of age.
- African Americans appear to be at higher risk - 2 times the incidence than Caucasian Americans.
- Female preponderance 1.3 - 1.6:1 female to male ratio - hormonal component.

*(Connolly et al., 2012)*

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**Etiology of SAH**

- Trauma
- Aneurysm rupture 75 - 80%
- AVM rupture 4- 5%
- Vasculitis
- Unknown 14 - 22%

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**Risk Factors of aSAH**

- **Modifiable:**
  - Cigarette smoking
  - HTN
  - Alcohol
  - Drug Use - sympathomimetic drugs
  - Cocaine

- **Non-modifiable:**
  - Age
  - Connective tissue disorder
  - Polycystic kidney disease
  - Ehlers-Danlos syndrome (deficient collagen type III)
  - Marfan's syndrome
  - Fibromuscular dysplasia
  - Family History
Cerebral Aneurysm

- Localized dilation of arterial lumen caused by a weakness of vessel wall.
- Result from degenerative vascular diseases, hypertension, and atherosclerosis.
- Formation is influenced by hemodynamic forces.
- 95% are acquired from hemodynamically induced degeneration of cerebral wall at the level of the bifurcation - point of maximum hemodynamic stress.

Types of Aneurysm

- Berry/Saccular
- Fusiform (atherosclerosis)
- Mycotic (septic emboli)
- Traumatic

Location

- Anterior Circulation (85%)
  - Anterior cerebral artery
  - Anterior communicating artery
  - Middle cerebral artery
  - Posterior communicating artery
- Posterior Circulation (15%)
  - Vertebral artery
  - Basilar artery
  - Posterior cerebral artery
- Most often occur at the bifurcations of the Circle of Willis
- 10 - 30% of people have multiple aneurysms

Events Surrounding aSAH

- Turbulent blood flow enters the aneurysm through its narrow neck and contributes to enlargement.
- Atherosclerosis and inflammation may weaken arterial walls leading to aneurysm rupture.
- Following rupture of an aneurysm, the blood vessel immediately clamps down to prevent further bleeding.
- This vasospasm may lead to ischemia and infarction.
**Events Surrounding aSAH Cont.**

- Rupture of an aneurysm releases blood directly into the subarachnoid space under arterial pressure.
- Rapidly increasing intracranial pressure (ICP)
- Blood acts as a chemical irritant
  - Inflammatory reaction
- The blood spreads quickly within the cerebral spinal fluid (CSF), interferes with its circulation, and prevents CSF absorption via the arachnoid villi.
- Hydrocephalus
- Enlargement of ventricles
- The bleeding usually lasts only a few seconds, but re-bleeding is common and occurs more often within the first day.
- A clot begins to form around the aneurysm.

**Clinical Manifestations**

- Sudden headache
  - “The worst headache of my life”
- Nuchal rigidity
- Nausea/Vomiting
- Loss of consciousness
- Confusion
- Photophobia
- Motor abnormalities
  - Hemiparesis
- 25% have seizures
- Cranial nerve palsy
- Dilated pupil
- Diplopia
- Pain above or behind eye
- Papilledema and/or retinal bleed

**“Warning Leak”**

- 30 - 50% of patients have a minor hemorrhage or “warning leak” that precedes a major SAH by 6 to 20 days.
- Manifested by a sudden and severe headache (sentinel headache).
- May be accompanied by nausea and vomiting. Meningismus is uncommon.

**Morbidity and Mortality of aSAH**

- High mortality rate - 51% fatality.
- 10% of patients with aneurysm SAH die prior to reaching the hospital.
- 25% die within 24 hours of SAH onset.
- 45% die within 30 days
- 25% will have some functional impairment.
- 25% will recover fully.

(Bader and Littlejohns, 2010)
Differential Diagnosis
- Migraine
- Ischemic Stroke
- Intraparenchymal hemorrhage
- Meningitis
- Neoplasm
- Encephalitis
- Cerebral Abscess
- Bell’s Palsy

Diagnosis
- Non-contrast CT of brain STAT
  - Detects blood in cisterns, fissures, and ventricles, edema, hydrocephalus, and mass effect.
  - Sensitivity of detecting SAH is nearly 100 percent in first 6 to 12 hours after SAH.
  - Sensitivity is reduced with minor bleeds.
- MRI
  - SAH may be identified on proton density and FLAIR sequences.
  - Limited studies to demonstrate sensitivity.

Hunt and Hess Classification System
- Grade 1
  - Asymptomatic or minimal headache or stiff neck
- Grade 2
  - Severe headache, stiff neck, photophobia
- Grade 3
  - Drowsy or confused, or mild hemiparesis
- Grade 4
  - Stuporous with hemiparesis or posturing
- Grade 5
  - Deeply comatose

Fischer Grade of Cerebral Vasospasm Risk in SAH
- 1 - No blood detected.
- 2 - Diffuse, deposition of thin layer with all ventricle layers less than 1mm thick.
- 3 - Localized clot and/or vertical layers 1mm or more thick.
- 4 - Intracerebral or intraventricular clot with diffuse or no subarachnoid blood.
Diagnosis Continued

- Lumbar puncture
  - If CT negative for hemorrhage.

- Findings:
  - Elevated opening pressure
  - Elevated RBC count that does not diminish from CSF tube 1 to 4
  - Xanthochromia - represents Hgb degradation products.
  - Indicates that blood has been in CSF for at least 2 hours.
  - False positives

Diagnosis Continued

- CTA/MRA
  - May identify aneurysms ≥ 3-5mm or larger.

Diagnosis Continued

- DSA - digital subtraction angiography.
  - Gold Standard
  - Highest resolution to detect intracranial aneurysms.
  - Definitive study for identifying aneurysm/vascular malformation.
  - Detects location of vasospasm.

Complications of aSAH

- Re-bleeding
- Hydrocephalus
- Increased ICP
- Seizures
- Hyponatremia r/t hypothalamic dysfunction and pituitary insufficiency
- Cardiac Abnormalities
- Vasospasm and delayed cerebral ischemia
Initial Management of SAH

- ABCs
- ECG, BP, RR, O2 sat monitoring
- Frequent Neuro Checks
- Maintain SBP 120-150mmHg
  - Labetalol prn
  - Hydralazine prn
  - Nicardipine gtt
  - Do not drastically lower the blood pressure. Moderate hypotension can cause cerebral ischemia.
  - Nitroprusside and Nitroglycerine should be avoided because they increase cerebral blood volume which increases ICP.
- Reverse coagulopathy
  - Vitamin K, FFP, platelets, Prothrombin Complex Concentrate

Initial Management of SAH Cont.

- Reduce cerebral edema
  - Hyperosmolar agents (Mannitol)
  - Loop diuretics
  - Corticosteroids - controversial, limited evidence from clinical trials to support its use as beneficial.
- Control ICP
  - May require ventriculostomy
  - HOB elevated to 30 degrees with head midline
  - Hyperventilation results in cerebral vasoconstriction - should only be used for brief periods of time.
  - Treat fever with cooling blanket
  - Pain control
  - Hyperosmolar agents

Initial Management of SAH Cont.

- Analgesics prn
- Daily Transcranial Doppler's (TCDs)
  - Monitors cerebral vasospasm.
- Prevent vasospasm
  - Calcium channel blocker (Nimodipine)
  - Pravastatin
- Control seizure activity
  - Anticonvulsants
  - Seizure prophylaxis not recommended
- Glucose control - avoid hypoglycemia
- Stool softeners - to avoid straining
- Fluid intake
  - 2,500 - 3,000cc/day
- DVT prophylaxis
  - SCDs
  - G1 prophylaxis
  - PN, HD blocker
  - Correct electrolyte abnormalities
  - Magnesium
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Treatment Options

- Surgical Clipping
- Endovascular Coiling

Type of treatment depends on
- Size of aneurysm
- Location
- Type of aneurysm
- Neck-to-dome ratio
- Patient’s age and co-morbidities.
Surgical Clipping

- Surgical procedure
  - Craniotomy
  - Placement of a clip across the neck of the aneurysm to exclude it from circulation without occluding the normal vessel.
- Operative risks:
  - Intraoperative hemorrhage
  - Infection
  - New or worsened neurologic deficits caused by brain retraction or arterial occlusion.
  - Seizure

Endovascular Coiling

- Minimally invasive procedure.
- Detachable platinum coil is inserted into the lumen of the aneurysm. A thrombus then forms around the coils, obliterating the aneurysmal sac.
- Risks
  - Intraprocedural rupture
  - Failure to obliterate aneurysm
  - Recurrence
**Complication: Re-bleeding**
- Highest risk is in the first 24 hours.
- Incidence 6.9 to 8.6 percent.
- 70% mortality rate for patients who re-bleed.
- Diagnosed - acute deterioration of neurologic status accompanied by appearance of new hemorrhage on CT scan.
- In patients:
  - Toddlers, severe HA
  - Elevated BP
  - Decreased LOC
  - Seizure
- Prevention of re-bleeding:
  - Intervention to secure aneurysm
  - Endovascular coiling
  - Surgical intervention
  - Blood pressure control - keep SBP <150mmHg (Per guidelines < 160mmHg is reasonable)
  - Aminocarproic acid is reasonable if delay in securing aneurysm

**Complication: Hydrocephalus**
- Secondary to blockage of CSF reabsorption by platelets, erythrocytes, and breakdown products.
- Acute: 15% of patients
  - May require EVD
- Chronic: 8 - 40% of patients
  - Intraventricular hemorrhage
  - Posterior circulation aneurysms
  - High Hunt and Hess Score
  - Older age
- Signs/symptoms:
  - Loss of consciousness
  - Drowsiness
  - Bilateral motor changes
  - Downward deviation of eyes

**Complication: Increased ICP**
- Monroe-Kelli Doctrine
  - Small changes in intracranial volume may ultimately cause intracranial pressure to increase due to the rigid and inelastic properties of the skull.
- Skull contains:
  - 80% brain
  - 10% CSF
  - 10% circulating blood

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Complication: Increased ICP

- Control/Monitor ICP
  - Ventriculostomy
  - Hyperosmolar agents
  - Loop diuretics
  - Drainage of CSF hourly
  - Pain control
  - Sedation

With increased ICP, cerebral perfusion may be impaired. CPP = MAP - ICP.

Complication: Seizures

- Seizures occur in 6-18% of patients with aSAH.

Risk factors for development of seizures:
- Aneurysm in MCA
- Associated intracerebral hematoma
- Re-bleeding
- Infarction
- High Hunt and Hess score
- History of HTN

Use of antiepileptic drugs to prevent seizures with SAH has been widely debated. Studies have been done with phenytoin. The impact of other anticonvulsant medications is less clear.

Argument for AED - seizure may lead to re-bleeding in unsecured aneurysms.

Potential side effects of anticonvulsants - fever, hypotension, vasospasm, DCI.

Guidelines/Recommendations -
- Routine use of anticonvulsant prophylaxis with phenytoin is not recommended after SAH.
- The use of other prophylactic anticonvulsants may be considered in the immediate post-hemorrhagic period.

Complication: Hyponatremia

- Incidence 10 - 30%

 related to hypothalamic injury causing inappropriate secretion of antidiuretic hormone.
- Central Nervous System
  - Characterized by renal deperfusion
  - Treated with isotonic saline - restoration of euvolemia will suppress release of ADH.

SIADH

- Fluid volume is normal or elevated
- Treated with isotonic saline or hypertonic saline - avoid fluid restriction as it may increase risk of vasopressor-related ischemic injury.

Treatment
- Isotonic saline
- 3% saline
- Salt tabs
- Fludrocortisone acetate

Complication: Cardiac Abnormalities

- ECG changes d/t ischemic changes in subendocardium of left ventricle
- ST segment depression
- QT interval prolongation
- Torsades de Pointes

Regional left ventricular wall motion abnormalities - may be reversible.
- Takotsubo - apical LV dysfunction that mimics MI in absence of significant CAD.

Myocardial injury/dysfunction is most likely the result of a centrally mediated release of catecholamines d/t hypoperfusion of posterior hypothalamus.
Complications: Vasospasm

- Delayed narrowing of the cerebral arteries which may be associated with clinical signs/symptoms that correspond to the region perfused by the involved artery.

(Koenig, 2012)

Complication: Vasospasm Cont.

- Pathogenesis
  - Poorly understood
  - Spasmogenic substances generated during the lysis of subarachnoid blood clots can cause endothelial damage and smooth muscle contraction.
  - Vascular endothelium produces nitric oxide (vasodilator). Endothelial damage may interfere with nitric oxide production leading to vasoconstriction.
  - Increased release of endothelin (potent vasoconstrictor) may play a role in cerebral vasospasm post aSAH.
  - Lumenal narrowing - decreased flow - cerebral ischemia - cerebral infarction.

Definitions of Vasospasm

- Symptomatic vasospasm:
  - New focal neurological sign, deterioration in level of consciousness, or both when other possible causes of neurological deterioration have been eliminated.
  - Subtle.

- Delayed contrast ischemia (DCI)
  - Symptomatic vasospasm with deficits attributable to vasospasm.
  - Infarct may be distinguished from AV shunting, embolization, or other cause.

- Instrumental Doppler vasospasm
  - Mean flow velocity in any vessel >150 cm/sec.

- Angiographic vasospasm
  - Non-flow-related narrowing of digital subtraction angiography not attributable to dissection, catheter-induced vasospasm, or coil impalpation.

Complication: Vasospasm

- Leading cause of death and disability after aSAH.

- 15 - 20% of patients who experience vasospasm die despite treatment to resolve vasospasm.

- Symptomatic vasospasm occurs in 20 - 30% of patients with aSAH.

- Seen angiographically in 75% of aneurysmal SAH.

- Occurrence during 3-21 days post SAH.
  - Typical onset 3-5 days
  - Peak days 7-8 days
  - Resolution over 14-28 days.
Complication: Vasospasm Cont.

- Risk factors:
  - Location of blood on CT scan and its extent can predict the likelihood.
  - Age less than 50
  - Hyperglycemia upon admission
  - MAP >110mmHg upon admission
  - Poor clinical grade (Hunt and Hess >3)
  - Type of intervention (surgical vs. endovascular) does not influence risk.

(Carron et al., 2009; Fortuna et al., 2009; Nakae et al., 2009)

Correlation of DCI with Hunt and Hess Grade

<table>
<thead>
<tr>
<th>H/H Grade</th>
<th>% Clinical Vasospasm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22%</td>
</tr>
<tr>
<td>2</td>
<td>33%</td>
</tr>
<tr>
<td>4</td>
<td>53%</td>
</tr>
<tr>
<td>5</td>
<td>74%</td>
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Diagnosis of Vasospasm

- Clinical examination
- Neuro checks Q2 hours
- Rule out other etiology for change in neurologic status – hyponatremia, infection, seizure, hypoxia, hydrocephalus, cerebral edema, oversedation.

- Transcranial Doppler - ultrasound measurement of intracranial arterial velocities. works on Bernoulli effect
- Decreased diameter - increased velocity

- CT Angiography/Perfusion - detection of cerebral blood flow, blood volume, and mean transit time.

- Digital Subtraction Angiography - gold standard for diagnosis of cerebral vasospasm.

Complication: Vasospasm Cont.

- Non-localizing findings
  - Non or decreasing intracranial pressure
  - Alterations in level of consciousness
  - Disorientation
  - Nystagmus

- Focal Neurological signs
  - Arterial cerebral artery stenosis - frontal lobe/occipital (partial, acute)
    - Hypertension, dizziness, vertigo, dysarthria, vomiting, seizures, headache
  - Subarachnoid hemorrhage - hemiparesis, hemiparesis, aphasia

- Unexpected rise in blood pressure
  - Concomitant medication to restrict cerebral blood flow.
Transcranial Doppler's

- Uses Doppler effect to indirectly measure vessel diameter as a function of blood flow velocity.
- MCA velocities:
  - Normal: < 120
  - Mild: 120 – 200
  - Severe: > 200
- Lindegaard ratio (evaluation for vasospasm in light of hypoxemia):
  - MCA:ICA ratio
- Lindegaard ratio:
  - Normal: <3
  - Mild: 3-6
  - Severe: >6

**How well does the use of TCD predict DCI?**

- Transcranial Doppler’s sensitivity and specificity 95%, specificity of measuring vasospasm when MCA velocities > 120cm/s.
- Carrera et al. - Retrospective study - 441 patients
- 21% of patients developed DCI.

**Results**

- Sensitivity of any mBFV more than 120cm/s in identifying DCI was 61.5%
- 85% of patients who developed DCI failed to reach velocity of 120cm/s before symptom onset.
- 55% of patients who developed DCI never had mBFV > 120cm/s.

TCDs may falsely reassure clinicians that vasospasm risk is low.

(Carrera et al., 2009)

Transcranial Doppler’s

- Advantages for TCD evidence of MCA vasospasm:
  - Velocity greater than 200cm/sec
  - Rapid rise greater than 50cm/sec between serial TCDs
  - Lindegaard ratio greater than 4
- Disadvantages:
  - Measurements are operator dependent
  - Inter-rater variability may be high
  - Sensitivity for detecting vasospasm in Doppler cannot detect small, subtle, or secondary changes.
  - Lack of correlation between Doppler velocity and clinical worsening is unrelated.

CTA/CTP

- Sensitivity 83% and specificity 100% for identifying vasospasm.
- Cerebral ischemia can be detected on the basis of side to side differences in perfusion.
- Useful screening test for cerebral vasospasm in patients who do not have adequate temporal bone windows for TCD.
- Usefulness in screening vasospasm in asymptomatic patients with increasing TCD velocities.
- Less invasive than angiography.
- CTP requires a contrast bolus in order to calculate CBF and MTT into the cerebral hemispheres.

(Binaghi, 2007)
CTA

Angiography

CTP

CTP
Angiography

Advantages:
- Gold Standard
- 3D reconstruction images
- May proceed with endovascular treatment of vasospasm

Disadvantages:
- Invasive procedure
- Risks:
  - Infection
  - Allergic reaction to contrast media
  - RCIN – radiocontrast induced nephropathy
  - Injury to blood vessel
  - Bleeding
  - Thrombo-embolism

How should vasospasm be defined?

- Prospective trial - 580 SAH patients
- Angiographic and TCD spasm were less well-correlated with symptomatic spasm or DCI.
  - Angiographic vasospasm
    - 34% symptomatic
    - 41% had DCI
  - TCD spasm
    - 28% had symptomatic
    - 34% had DCI
- Symptomatic vasospasm and DCI were highly correlated with angiographic and TCD spasm.
  - Symptomatic vasospasm
    - 85% had angiographic vasospasm
    - 81% had TCD spasm
  - DCI
    - 84% had angiographic vasospasm
    - 76% had TCD spasm

What does this all mean?

- Advantages and disadvantages to using TCDs, CTA/CTP, and DSA when diagnosing vasospasm in patients with aSAH.
- Clinical examination is the criterion standard for diagnosing and monitoring DCI.
  - Neuro checks Q2 hours
  - Know s/s associated with clot/aneurysm location
  - Know which patients are at greatest risk of vasospasm
  - Know when vasospasm is likely to occur

Prevention is important

Prevention of Vasospasm

- Nimodipine
- Euvolemia
- Statin
- Magnesium
- Endothelin Receptor Antagonists
- Permissive hypertension
**Nimodipine**
- Calcium channel blocker - inhibits the cerebrovascular smooth muscle contraction.
- No evidence that nimodipine affects the incidence of symptomatic vasospasm or angiographic vasospasm.
- Meta-analysis of seven randomized trials:
  - Improved odds of good outcome after SAH.
  - Reduced the odds of death, mortality, or both.
- May cause hypotension
  - 60mg Q 4 hours x 21 days
  - 30mg Q 2 hours x 21 days

Meta-analysis: (Cochran review, 2005)

**Statins**
- Potential to improve cerebral vasomotor reactivity by upregulating endothelial nitric oxide synthase and increase cerebral blood flow.
- Variable results in several small, single-center randomized trials.
- Simvastatin in Aneurysmal Subarachnoid Hemorrhage (STASH) clinical trial is in progress.

((Connley, 2010))

**Magnesium Sulfate**
- Magnesium is a non-competitive calcium antagonist with vascular and neuroprotective effects.
  - Promotes vasodilation by blocking voltage-dependent calcium channels.
  - Decreases glutamate release.
  - Reduces release of reactive oxygen species.
  - Antimetabolizes the effect of potent vasoconstrictors: Endothelin 1
  - Blocks formation of reactive oxygen species.

- Results of multiple studies did not show significant difference in patient outcomes or frequency of vasospasm and DCI in patients receiving magnesium versus patients receiving placebo.
- Inducing hypermagnesemia is not recommended, but hypomagnesemia should be avoided.

((Diringer et al., 2011))

**Endothelin Receptor Antagonists**
- Endothelin 1 is an amino acid peptide that acts as a direct and potent vasoconstrictor.
- Clazosentan is a specific Endothelin receptor antagonist.
- Shown to be associated with a dose-dependent reduction in the incidence of angiographic vasospasm.

((Shaw, 2000))
Treatment of Vasospasm

- Hemodynamic Augmentation
- Intra-arterial injections of vasodilators
- Balloon angioplasty and/or stent

Hemodynamic Augmentation

- Hypertension:
  - BP goals should be increased in a stepwise fashion with attention to improvement of neurologic deficits.
  - MAP > 120mmHg recommended
  - SBP > 160 avoid drops < 140mmHg
  - Levophed
  - Phenylephrine
  - Dopamine
  - Avoid antihypertensives
  - CI between 5 & 6
  - Dobutamine if Vasopressors fail

Hypervolemia:

- Some debate on efficacy due to complications
- Goal is now euvelemia
- Saline and Albumin for volume expansion
- Ideal CVP is 8 - 10mmHg
  - In young patients with good kidney function, higher CVPs may not be attainable
- Ideal PCWP is 12 - 18mmHg
  - Swan-Ganz placement may be considered - not recommendation.
- Complication - pulmonary edema
  - Avoid diuresis if possible

Hemodilution

- Initially used in an attempt to improve rheology
- May be detrimental
- Anemia decreases O2 carrying capacity
- Goal is Hgb 8 - 10
Complications of Hemodynamic Augmentation

- Pulmonary edema/CHF/MI
- Dilutional Hyponatremia
- Cerebral edema/ Increased ICP
- Hemorrhagic conversion of ischemia/infarction
- Infections - line sepsis
- Re-bleeding

Neurointerventional Intervention

- Mechanical:
  - Balloon Angioplasty.

- Chemical - intra-arterial injections:
  - Papaverine.
  - Calcium Channel Blockers:
    - Verapamil.
    - Nicardipine.
    - Nimodipine.

Balloon Angioplasty

- Advantages:
  - Sustained effect.

- Can treat:
  - Supraclinoid ICA, MCA trunk/ M1 segment, A1 segment (usually).
  - In some cases: M2 segments of MCA.

Balloon Angioplasty Complications

- Reported Complications:
  - Arterial dissection.
  - Thromboembolism / stroke.
  - Branch occlusion.
  - Reperfusion hemorrhage into infarct.
  - Bleeding from unsecured aneurysms.
  - Vessel Rupture.

- Complication rate of 5 %

- Vessel rupture rate of 1.1%
Balloon Angioplasty

- Treatment of distal vasospasm.
- Pre-dilatation for balloon angioplasty.
- Treatment of mild to moderate spasm.

Chemical Treatment

- Papaverine
  - Reported complications:
    - Crystalline precipitation with possible embolic events.
    - Seizures.
    - Monocular blindness.
    - Brainstem dysfunction.
    - Neurologic deficit resolving with termination of infusion.
    - Respiratory arrest.
    - Profound hypotension.
    - Aneurysm re-rupture.

- Verapamil
  - Rationale:
    - Randomized trial demonstrated improved clinical outcome with oral Nimodipine.
    - Verapamil: used to treat coronary vasospasm.
    - No significant systemic effects with IA injection of ≤7.5 mg.
    - No evidence for any effect on ICP.
Treatment approach

Asymptomatic patients will have a diagnostic angiogram around day 7.
- Mild spasm - No treatment.
- Moderate spasm - Verapamil.
- Severe spasm - Verapamil +/- angioplasty after discussion with NSG.
  Verapamil: 10 mg/vessel, over 10-20 minutes.

Symptomatic Patients:
- Mild spasm - Verapamil alone.
- Moderate spasm - Verapamil. If no angiographic improvement, consider angioplasty.
- Severe spasm - Verapamil plus angioplasty.

Contraindications to Endovascular Intervention
- Large hypodensities on CT - risk of reperfusion injury/hemorrhage.
- Patient may benefit from MR diffusion/perfusion imaging or CTP in order to identify ischemic penumbra.
- Unsecured aneurysm:
  - Usually not an issue as vasospasm may be treated chemically and/or mechanically during procedure to coil aneurysm.

Discharge Planning
- Despite technological advances, many patients who suffer aSAH will have significant neurologic deficits.
- Throughout hospitalization it is important for interdisciplinary patient management:
  - Dietary
  - PT/OT/SLP
  - Case Manager/Social Worker
  - Consult Physiatrist
  - Chaplain
- Teaching is often necessary to help patient and family make decisions and cope with the new diagnosis.
References