Subarachnoid Hemorrhage and Management of Intracranial Aneurysms

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Skull and Intracranial Compartments

- Scalp
  - Subgaleal Space
- Bone (Calvarium)
  - Epidural Space
- Dura Mater
  - Subdural Space
- Arachnoid
  - Subarachnoid Space
- Pia Mater
- Brain Parenchyma
- Ventricles
Normal Vasculature with Aneurysm
Subarachnoid Space

- Contains larger cerebral blood vessels prior to arterial branches penetrating the brain parenchyma through Virchow Robin spaces
- Contains cerebrospinal fluid (CSF)

- WHEN BLOOD VESSELS TEAR OR RUPTURE THEY GENERALLY RELEASE BLOOD INTO THE SUBARACHNOID SPACE WHERE IT MIXES WITH CSF. OCCASIONALLY THIS BLOOD CAN EXTEND INTO THE BRAIN PARENCHYMA (20-40% INCIDENCE), INTRAVENTRICULAR SPACE (13-28% INCIDENCE, AND SUBDURAL SPACE (2-5% INCIDENCE) DEPENDING UPON THE ANEURYSM’S LOCATION
Etiology of SAH

- Trauma (most common cause of SAH)
- Spontaneous SAH
  - Aneurysms 75-80%
    - Congenital
    - Traumatic
    - Mycotic (Infectious)
    - Flow related
    - Dissecting
  - Arteriovenous Malformations (AVMS; usually secondary to rupture of an associated aneurysm or dilated vein) 4-5%
  - Vasculopathy/Vasculitis
  - Intracranial arterial dissection (hemorrhage into the wall of a blood vessel) and subsequent extension through the wall and into the SA space
  - Rupture of small intracranial vessel
  - Coagulopathy
  - Spinal AVM
  - Drugs (Cocaine, amphetamine)
  - Sickle cell disease
  - Pituitary apoplexy (hemorrhage into the pituitary gland secondary to tumor infarction)
  - Benign perimesencephalic hemorrhage
  - Unknown etiology (14-22%)
Aneurysm Definition/Description

- A cerebral aneurysm is a dilatation of an intracranial artery.
  - Saccular a. (berry) more common than fusiform a.
  - Saccular a. are generally located at a point where the artery divides into two branches
  - Fusiform a. are generally located along the long axis of the artery
- Saccular a. have a neck (opening) and a fundus (sac)
- Aneurysm size refers to the diameter of the fundus measured in mm. (usually measured as longest length across the fundus)
  - Fundus:Neck (F:N) ratio is often measured
- Giant aneurysm (>25mm)
  - Often present secondary to mass effect (pressure on the brain) and neurologic deficit
- Wide neck aneurysm defined as F:N <2 or neck greater than 4 mm in diameter
Aneurysm Images (Mycotic)
Aneurysm (Traumatic GSW)
Basilar Aneurysm (Saccular)
Aneurysm Biology

What causes aneurysms to develop?

- Intracranial arteries are relatively fragile with less muscle, elastic protein, and thinner walls than peripheral arteries of similar size.
- The intracranial arteries have less supporting tissue surrounding them than peripheral arteries.
- Aneurysms most often form at bifurcations where there is turbulent flow and stress.
- Congenital defects in the media layer of the artery may exist in some individuals.
- Infection and subsequent inflammation and weakening of the arterial wall.
- Traumatic destruction of the arterial wall.
- Spontaneous hemorrhage into the arterial wall with subsequent aneurysm formation.
Incidence and Demographics

- Based on autopsy studies approximately 5% of the Western population harbors an intracranial aneurysm
- 30,000 aneurysm ruptures per year in US
- 20% incidence of multiple aneurysms in those with aneurysms
- Estimated annual rate of aneurysmal SAH is 6-8/100,000
- Peak age of SAH is 55-60 years (20% of cases occur between the ages of 15-45 years)
- Female > Male
- Approximately 50% of aneurysms rupture
- 2% of aneurysms present during childhood. These are frequently associated with AVMs or traumatic
Risk Factors for Aneurysms

- Polycystic Kidney Disease (15% have aneurysms)
- Fibromuscular Dysplasia (FMD)
- Renal FMD (7% have aneurysms)
- Aortocranial FMD (21% have aneurysms)
- AVM (may have aneurysms on feeding arteries or within the nidus/body of the AVM itself)
- Moya-moya disease (a type of vasculopathy that leads to gradual arterial occlusion)
- Connective tissue disorders (esp. Ehlers-Danlos Type 4 due to deficiency in collagen type 3)
- Marfan’s Syndrome (autosomal dominant inherited connective tissue disorder)
- Familial aneurysms (2 or more third degree or closer relatives with aneurysms)
- Coarctation of the aorta (a congenital condition whereby the aorta narrows in the area where the ductus arteriosus (ligamentum arteriosum after regression) inserts
- Osler-Weber-Rendu syndrome (a genetic disorder that leads to abnormal blood vessel formation in the skin, mucous membranes, and often in organs such as the lungs, liver and brain.
- Atherosclerosis
- Bacterial endocarditis (mycotic/infectious aneurysms)
- Smoking
Natural History of Unruptured Aneurysms

  - 6,697 aneurysms in 5,720 patients followed over 11,660 aneurysm-years
  - 91% of aneurysms discovered incidentally
  - 66% female
  - Mean age 62.5 years
  - Mean size 5.7 mm
  - Older patients had larger aneurysms
Natural History of Unruptured Aneurysms

- Overall rupture rate 0.95%/year
- Mortality from rupture itself 35%
- Major morbidity from rupture 29%
- Risk Factors Associated with Rupture
  - Female
  - Age >70
  - **Location**
    - Anterior communicating artery, posterior communicating artery (Hazard Ratio 2) using MCA bifurcation as reference
  - Hypertension
  - Hyperlipidemia
  - Daughter sac
  - **Increasing size** (risk significantly increases at \( \geq 7 \) mm)
    - 3-4 mm (reference)
    - 5-6 mm (1.13 Hazard Ratio)
    - 7-9 mm (3.35 HR)
    - 10-24 mm (9.09 HR)
    - \( \geq 25 \) mm (76.26 HR)
  - Calcification of thrombosis
  - Smoking, presence of another aneurysm causing SAH, family history, multiple aneurysms did not significantly affect risk of rupture of each aneurysm

- Risk factors for rupture include increasing aneurysm size, aneurysm location (posterior circulation), for aneurysms greater than 7 mm SAH from a separate aneurysm
Natural History of Unruptured Aneurysms

- Annual Rate of Rupture According to Size and Location

<table>
<thead>
<tr>
<th>Location</th>
<th>3-4 mm</th>
<th>5-6 mm</th>
<th>7-9 mm</th>
<th>10-24 mm</th>
<th>&gt;25mm</th>
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<td>MCA</td>
<td>0.23</td>
<td>0.31</td>
<td>1.56</td>
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<td>0.75</td>
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<td>ICA</td>
<td>0.14</td>
<td>0</td>
<td>1.19</td>
<td>1.07</td>
<td>10.61</td>
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<td>1.00</td>
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<td>BA-SCA</td>
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<td>0</td>
<td>0</td>
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<td>0</td>
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<td>Total</td>
<td>0.36</td>
<td>0.50</td>
<td>1.69</td>
<td>4.37</td>
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</table>
Possible Risk Factors for SAH

- Hypertension
- Oral contraceptive use
- Cigarette use
- Cocaine use
- Alcohol
- Pregnancy
- Advanced age
SAH Symptoms

- Sudden HA (“Thunderclap HA) 97% of cases
  - “Worst HA of my life”
- Emesis
- Loss of consciousness
- Neck pain (meningismus)
- Photophobia (aversion to light)
- Sentinel HA (warning HA that may occur in 30-60% of patients often 6-20 days before actual major SAH). May be secondary to either small hemorrhage (leak) or aneurysm change/growth
SAH Signs

- Focal neurologic deficit including cranial nerve palsy. Often 3\textsuperscript{rd} nerve palsy due to compression of the nerve by a posterior communicating artery aneurysm or a superior cerebellar artery aneurysm.
- Nuchal rigidity (especially seen during attempted neck flexion).
- Coma.
- Ocular hemorrhage (due to compression of the central retinal vein due to elevated ICP causing venous hypertension and vein rupture).
  - Pre-retinal hem.
  - Intra-retinal hem.
  - Vetreous humor hem. (Terson’s Syndrome) 4-27% incidence.
Patient Classification Based on Presenting Examination

- **Hunt and Hess Scoring** (MOST COMMONLY USED IN UNITED STATES)
  - 0  No hemorrhage
  - 1a Fixed neuro deficit (CN palsy) with no HA or nuchal rigidity
  - 1  Asymptomatic, or mild HA and slight nuchal rigidity
  - 2  Moderate to severe HA and nuchal rigidity
  - 3  Mild focal deficit, lethargy or confusion
  - 4  Stupor, moderate to severe hemiparesis, decerebrate rigidity
  - 5  Coma, decorticate rigidity (MORIBUND)

- **World Federation of Neurologic Surgeons (WFNS) Scoring**
  - 0  Unruptured
  - 1  GCS 15
  - 2  GCS 13-14
  - 3  GCS 13-14 with major neuro deficit
  - 4  GCS 7-12
  - 5  GCS 3-6
HH Score and Hydrocephalus

- The brain continuously makes approximately 0.3 cc/minute CSF. This CSF travels through the ventricular system and is eventually reabsorbed into the venous system by the arachnoid villi.
- Acute hydrocephalus develops following SAH due to blood products obstructing the normal pathways through which CSF travel thus reducing the delivery of CSF to absorption sites and/or by coating the arachnoid villi making reabsorption by the villi inefficient.
- While HH score does not take into consideration the presence of hydrocephalus many feel the HH score is not accurate, especially in higher grade patients, in the presence of hydrocephalus.
- Many patients will improve in HH score once hydrocephalus is treated with CSF diversion.
- When CSF is diverted the aim is generally to keep the ICP around 25 mm Hg until the aneurysm is obliterated.
Natural History of Aneurysmal SAH

- 10-15% die before reaching a hospital
- 10% die within first several days of reaching care
- Overall mortality is 32-67% for all comers
- 30% of survivors have significant disability
Initial Imaging and Laboratory Evaluation

- **CT Scan Head**
  - Will detect SAH in ≥93% of cases within 24 hours of SAH
  - Evaluate images for blood, enlarged ventricles, partially thrombosed aneurysm, calcifications that may indicate a calcified portion of the aneurysm
  - Blood pattern can help localize the aneurysm or determine which aneurysm bled in the case of a patient with multiple aneurysms
  - CT scan will detect hydrocephalus in 15-20% of patients with SAH
Initial Imaging and Laboratory Evaluation

- **IF CT IS POSITIVE FOR SAH THERE IS NO INDICATION FOR A LUMBAR PUNCTURE SINCE A LUMBAR PUNCTURE WITHDRAWS CSF FROM THE SUBARACHNOID SPACE, LOWERS INTRACRANIAL PRESSURE, AND AS A RESULT MAY INDUCE ANEURYSM RUPTURE BY REDUCING THE PRESSURE THAT IS EXERTED ON THE OUTSIDE OF THE ANEURYSM**

- **Lumbar Puncture** (A needle is inserted through space between the lumbar L4 and L5 spinous processes in the lower back and into the subarachnoid space surrounding the lower spinal nerve roots/cauda equina. CSF is withdrawn as a sample and is collected serially in 4 test tubes)
  - Evaluate cerebrospinal fluid (CSF) for blood products, pressure, cell counts, microbiology
  - RBC usually >100,000 RBC/cc
  - RBC count should drop between the sample in tube #1 and in tube #4 when the blood is from a traumatic tap as opposed to a SAH
  - Protein should be elevated due to RBC breakdown
  - Glucose generally normal
  - **Xanthochromia evaluation.** CSF sample is spun in a centrifuge so that cells collect at the bottom of the tube. A pink or yellow tinge to the supernatant (fluid above the cells) indicates that RBC have been present in the CSF for at least 2-4 hours prior to the lumbar puncture because the tinge is due to the pigments released by old lysed RBCs. Xanthochromia helps differentiate a traumatic tap from a positive tap due to SAH.
Initial Imaging and Laboratory Evaluation

- MRI not generally useful within first 24 hours due to limited presence of met-hemoglobin which is a breakdown product of hemoglobin that is detected by MRI
Imaging Evaluation

- If an aneurysm is suspected proceed with imaging to better visualize the intracranial vasculature

- Invasive
  - Catheter based cerebral arteriography (gold standard). Complication rate of this invasive procedure is <1%.

- Non-Invasive
  - CT arteriography (95% sensitivity; 83% specificity) for lesions 2-3 mm or larger
  - MR arteriography (86-95% sensitivity for lesions ≥3 mm; false positive rate 15%)
Stumbling Block

  - 1-4% of patients presenting to Ers with have SAH
  - When HA is worst of life 12% are secondary to SAH
  - When worst HA and abnormal neurologic exam is included 25% have SAH
  - 25-50% of patients with SAH are not diagnosed properly at time of first evaluation
  - Only 20-50% of patients report a thunderclap HA
  - 50% have atypical presentations
  - Modern CT scanners are 98% sensitive for scans performed within 12 hours of ictus, and 93% sensitive for scans performed within 12-24 hours
Rules to Remember

- A sudden, severe HA is aneurysmal until proven otherwise.
- A sudden, severe HA is aneurysmal even when a head CT is negative and an LP is negative since the HA could be secondary to sudden aneurysm growth or enlargement (an unstable aneurysm).
- A negative CTA or MRA in the face of a suggestive history or exam must be followed by a catheter based cerebral arteriogram to definitively rule out an aneurysm.
- A negative catheter based cerebral arteriogram is only negative when all vessels are visualized.
- If a catheter based cerebral arteriogram is negative in the presence of SAH on a CT scan (non-perimesencephalic pattern) the study should be repeated in 7-14 days.
- A catheter based cerebral arteriogram is not negative if all arteries are not seen and if cerebral vasospasm is present since this may reduce blood flow into an aneurysm making it invisible on the study.
Initial Patient Management

- Admit patient to monitored setting (ICU) for necessary life support measures (mechanical ventilation if necessary)
- Invasive arterial monitoring lines for more fragile patients
- Central venous catheters for patients grades 4 - 5
- Placement of ventriculostomy or lumbar drain for CSF diversion in most patients with HH Score of 4 or 5 and at times 3.
- Blood pressure monitoring and control (aim for normotensive in most cases)
- Nimodipine to improve outcome from vasospasm (see later slides)
- Stool softener
- IV hydration
- Pain control
- Serial neurologic examination
  - Can detect seizures, progressive hydrocephalus, aneurysm re-rupture
- Repeat CT scan if neurologic examination changes
- Prophylactic anticonvulsants (??)
Why Is Expeditious Aneurysm Obliteration Important?

- Goal of aneurysm treatment is to eliminate the aneurysm ASAP so that it cannot rerupture.
- Aneurysm rebleed rate is
  - 4% within first 24 hours and
  - 1.5% for each of the next 13 days
  - 15-20% risk of rehemorrhage in first 14 days
  - 50% risk of rehemorrhage within first 6 months
- In patients HH 1-3 generally treat within 24-48 hours.
  - Mortality of HH 1-2 patients is 20%
  - Major cause of death of HH 1-2 patients is rebleeding
- In patients HH 4-5 it is unclear whether or not early aneurysm obliteration improves long term survival
- Presence of vasospasm complicates timing of treatment due to risks of concomitant strokes with brain manipulation. Timing is often dependent upon whether the aneurysm is going to be treated by endovascular (coiling) or exovascular (clipping)
Timing of Aneurysm Surgery

  - Class 2 evidence (prospective but no randomization)
  - 3521 patients (83% had aneurysm surgery; no coiling)
  - 68 centers in 14 countries (24 in USA)
  - 6 month FU
  - Assessments by neurologists blinded to the timing of surgery

- Findings
  - Most important prognosticators for outcome
  - Level of consciousness (75% who were alert on admission had a good recovery compared to 11% who were comatose)
  - Age inversely related to outcome
  - No significant sex differences
  - Smaller aneurysms had more favorable results
  - Other good prognosticators included lower admission blood pressure, absence of pre-existing medical conditions, absence of vasospasm
  - Hunt and Hess Grade 1-2 patients had a more favorable outcome if their operation occurred between days 0-3 or after day 10
  - Hunt and Hess Grade 3 or worse did better when treated after day 10
  - Vasospasm and re-bleeding major causes of death or disability aside from the initial effects of the bleed

- Problems using this study today
  - Patients in study treated 1980-1983 (30 years ago; new treatments since then)
  - No endovascular
Aneurysm Treatment

- Endovascular Treatment (aka: coiling)
- Exovascular Treatment (aka: clipping)
Endovascular Treatment Options

- Coil
- Coil with neck remodeling device (stent)
- Coil with balloon remodeling
- Pipeline flow diversion
- Onyx 32
Coiling
Pipeline
Endovascular Therapy (Using Enterprise and Coils)
Coiling vs Clipping

  - Class 1 study (non blinded coiling vs clipping; randomized)
  - 2143 patients (1073 coiled; 1070 clipped)
  - Length of FU 1 year with ongoing evaluations
  - 43 centers (60-200 cases/year)
ISAT Findings

- **Dependence or Death 12 months after tx**
  - Coiling 23.7%
  - Clipping 30.6%
  - Relative risk reduction for endovascular 22.6%
  - Absolute risk reduction for endovascular 7.0%
  - Early survival benefits of coiling were maintained at 7 years

- **Rebleed risk at 12 months**
  - Coiling 0.16%
  - Clipping 0%

- **Late rebleed rates**
  - Coiling 0.21%
  - Clipping 0.063%

- **Risk of epilepsy at 12 months**
  - Coiling 1.3%
  - Clipping 2.2%

- **Cognitive impairment at 12 months**
  - Coiling 26.7%
  - Clipping 39%
# Coiling vs Clipping: Outcomes

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<th>Outcome</th>
<th>Stat .Significant</th>
<th>Coiling</th>
<th>Clipping</th>
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<tr>
<td>Death or Dependence</td>
<td>YES</td>
<td>22.5%</td>
<td>30.9%</td>
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<td>Incidence of rebleeding</td>
<td>NO</td>
<td>2/1276 patient-years (0.16%)</td>
<td>0/1081 patient-years</td>
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<td>Mortality at 1 yr</td>
<td>YES</td>
<td>85/1073</td>
<td>105/1070</td>
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<td>Re-Treatment Rate</td>
<td>Greater</td>
<td></td>
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<tr>
<td>Complete Occlusion</td>
<td></td>
<td>66%</td>
<td>82%</td>
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<tr>
<td>Incidence of Seizures</td>
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<td>Lower</td>
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Coiling vs Clipping

- In terms of survival free disability, outcomes from coiling are superior to outcomes from clipping.
- No subsequent studies have refuted these findings for aneurysms that are equally amenable to clipping and coiling.
For aneurysms that are equivalent candidates for clipping and coiling, clipping is the superior option. Other literature both supports and refutes ISAT however this literature is mostly retrospective and none is prospective and randomized. Hence ISAT is the only Level 1 data available on the topic.
Coiling vs Clipping (Randomized Prospective Studies)


New Developments In Endovascular Therapy

Since ISAT, technological advances have made endovascular therapy more effective while at the same time making more aneurysms amenable to minimally invasive therapies (80-85%)

- Softer coils
- Bioactive Coils
- Fibered coils
- 3 dimensional coils
- More steerable wires
- More trackable catheters
- Intracranial stents (Enterprise; Neuroform) (aka: neck remodeling device)
- Flow divertors (Pipeline; Silk)
- Medications (Plavix, Integrelin)
- Onyx liquid embolics
- Compliant balloons
What Factors Are Responsible For Up to 70% Morbidity and Mortality

- Aneurysm rerupture with and without therapy
- Unrecognized acute or chronic hydrocephalus
- Infection (meningitis, pneumonia, sepsis)
- Initial brain injury from rupture and raised ICP
- Vasospasm
- Cardiac abnormalities (ischemia, arrhythmia)
- Undefined neurometabolic changes
- Complications from treatment (stroke, direct brain injury)
Treatment Complications

- Stroke from inadvertent or intentional arterial occlusion, manipulation, stenosis
- Attempt to minimize this with meticulous technique, bypass procedures when necessary, use of intraoperative imaging
- Retraction of brain tissue
- Treatment during vasospasm
Aneurysm Rerupture

- Untreated Aneurysms
  - 20% within 2 weeks of first SAH
  - 50% within 6 months of first SAH

- Treated Aneurysms
  - Up to 2% in ISAT study
  - True number unknown since long term studies of rupture post clipping not done
  - Usually due to incomplete clipping or coiling resulting in continued aneurysm filling following treatment
Hydrocephalus (HCP)

- **Acute**
  - Occurs in 15-20% of SAH patients
  - 30-60% of patients with hydrocephalus do not demonstrate neurologic changes
  - 3% without HCP on initial CT develop it within 7 days
  - Managed with placement of an external ventricular drain (EVD; ventriculostomy) or lumbar drain and CSF diversion into a bedside collection chamber.

- **Chronic**
  - Up to 50% of patients with SAH develop chronic HCP
  - Managed with ventriculoperioperative, Vpleural, or Vatrial permanent shunt so that CSF is diverted from the ventricles to a different body compartment where it is reabsorbed into the venous system.

- When HCP is unrecognized it can lead to detrimental cognitive changes, HA, nausea, vomiting, visual loss, difficulty with ambulation, incontinence, and death
Vasospasm

- Second major cause of morbidity/mortality
- Post SAH vasospasm is a reduction in the diameter of intracranial arteries following SAH. Clinical vasospasm develops when this stenosis results in a reduction in blood supply (reduction in cerebral blood flow) and neurologic function changes secondary to cerebral ischemia.
- Vasospasm is felt to be secondary to the effects of blood breakdown products on cerebral arteries. Peak period days 4-12. The etiology is unknown, however, it may be due to changes in nitric oxide mediated mechanisms that control arterial wall smooth muscle relaxation.
- Thickness of subarachnoid blood (Fisher Grade) correlates with the development of clinical vasospasm
  - Fisher 1  No blood  No clinical spasm
  - Fisher 2  <1 mm thick  No clinical spasm
  - Fisher 3  >1 mm thick  96% incidence spasm
  - Fisher 4  Ventric/Parench blood  No clinical spasm
Vasospasm

- In autopsy and experimental studies vasospasm is associated with changes in the arterial wall adventia, media, and intima such as inflammation, muscle necrosis, cell swelling, smooth muscle proliferation, and intimal thickening.
Vasospasm

- Clinical Findings
- HA
- Altered LOC
- Neck Pain
- Fever
- Focal neurologic deficit
Vasospasm Diagnosis

- Clinical changes correlating with:
  - Angiographic confirmation of arterial stenosis
  - CT/MR confirmation of reduction in CBF
  - Elevations in transcranial doppler measured velocities in major arteries (as diameter of a tube narrows the velocity of the fluid moving through it elevates). Velocity $<120$ cm/s normal while velocity $>200$ cm/s indicates severe spasm
Vasospasm Prevention and Treatment

- Nimodipine prior to vasospasm development
  - Class 1 evidence supporting benefits
  - 60 mg PO q4h initiated within 96 hours of SAH (usually stopped before day 21 despite studies)
  - Calcium channel blocker
  - No difference in radiographic vasospasm but improvement in outcome
    - Incidence of cerebral infarction reduced by 33%; (22% with Nimodipine and 33% with placebo)
    - Poor outcome reduced by 40% (20% with Nimodipine and 33% with placebo)

- Triple H therapy
  - Retrospective study
  - Hypervolemia (fluid infusion titrated to central venous pressure and PCWP)
  - Hypertension with pharmacologic manipulation
  - Hemodilution therapy (maximize CBF and oxygen delivery by altering rheology)

- Statin therapy for prevention of vasospasm
  - Class 1 evidence supporting benefits of Pravastatin given within 72 hours of SAH and continued for 14 days
  - 32% reduction in vasospasm with duration shortened by 0.8 days
  - 75% reduction in mortality
  - Benefits present at 6 months

- Endovascular treatment of medically recalcitrant vasospasm
  - Intraarterial catheter based papavarine or verapamil infusion
  - Balloon angioplasty (no benefits of prophylactic angioplasty due to technical risks)

- Avoid pyrexia
  - ASA
  - Cooling blanckets
  - Cooling venous catheters
Cardiac Abnormalities

- Cardiac Troponin (cTnI) leak: 31%
- EKG changes: 22% (vs. 9% in patients without SAHMI)
- Echocardiographic wall motion changes: 44% with cTnI >0.3 ng/ml had depressed EF and WMA
- MI rate 31%