Cerebral Aneurysms & Subarachnoid Hemorrhage

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Epidemiology

- 5% of the population with intracranial aneurysm
  - 20-30% of this group with multiple aneurysms
- Annual incidence of SAH 2-25 per 100,000
- ~30,000 SAH in US yearly

Stehbens WE. Arch Pathol
WFNS. JNS
# Risk Factors for Formation

<table>
<thead>
<tr>
<th>Modifiable</th>
<th>Non-modifiable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cigarette smoking</td>
<td>Previous SAH</td>
</tr>
<tr>
<td>HTN</td>
<td>PCKD</td>
</tr>
<tr>
<td>Moderate to Heavy ETOH</td>
<td>CT disease</td>
</tr>
<tr>
<td>Cocaine</td>
<td>Aortic coarctation</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>Pseudoxanthoma elasticum, Moyamoya, AVM, FMD, Vasculitis, NF1, FH</td>
</tr>
</tbody>
</table>
Unruptured intracranial aneurysms: natural history, clinical outcome, and risks of surgical and endovascular treatment

International Study of Unruptured Intracranial Aneurysms Investigators

<table>
<thead>
<tr>
<th>Size (mm)</th>
<th>Group 1</th>
<th>Group 2</th>
<th>7-12 mm</th>
<th>13-24 mm</th>
<th>&gt;25 mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3-0%</td>
<td>6-4%</td>
</tr>
<tr>
<td>7-12</td>
<td>2-5%</td>
<td>3-4%</td>
<td>14-5%</td>
<td>18-4%</td>
<td>50%</td>
</tr>
</tbody>
</table>

AC=anterior communicating or anterior cerebral artery. IC=internal carotid artery (not cavernous carotid artery). MC=middle cerebral artery. Post-P comm=vertebrobasilar, posterior cerebral arterial system, or the posterior communicating artery.

Table 4: 5-year cumulative rupture rates according to size and location of unruptured aneurysm

Lancet 2003
Risk Factors for Rupture

• Previous ruptured aneurysm
History

- SUDDEN SEVERE HA, WORST HA OF MY LIFE
Physical Exam

- General Exam
- Kernig’s sign
- Brudinisk sign

http://what-when-how.com
Neurologic Exam

- Full neurologic exam
- CN exam may demonstrate III nerve compression or VI palsy
- Retinal examination due to risk of subhyloid hemorrhage
Differential DX

- SUDDEN SEVERE HEADACHE
  - Ruptured saccular cerebral aneurysm
  - Traumatic SAH
  - Vascular malformations
    - AVM
    - Cavernous malformation
  - Dissection w/ pseudoaneurysm
  - Oncotic aneurysm
  - Endocarditis with mycotic aneurysm rupture
  - Meningitis/Encephalitis
  - Thunderclap HA
Evaluation

- Non contrast CT
- CT angiogram/DSA
- MRA
- LP
- LABS
Lumbar Puncture
SAH

- 6-8/100,000 annual incidence
- Peak age 55-60 years
- SAH with IPH: 20-40%
- SAH with IVH: 13-28%
- SAH with SDH: 2-5%
Management Goals

• Reduce re-rupture
• Address HCP
• Treat elevated ICP
• Prevent SZ
• Monitor and treat hyponatremia
• Monitor and treat cerebrovasospasm
Rebleeding

- 3-4% risk during first 24 hours
- 2% risk second day
- 20% risk in 2 weeks
- 50% risk during first 6 months
Prevention of re-bleeding

• Secure the aneurysm

• Antifibrinolytics
  • Epsilon-aminocaproic acid
    • 4mg IV
    • 1g/hr
  • Tranexamic acid
    • 1 g IV load, 1 g/hour infusion
Hydrocephalus

- Worse grade SAH increased risk
- CSF diversion via EVD
- Initially 15-20 cm above EAC
- ICP goal <20 mm Hg
- VPS needed in 30% of cases
Seizure

- Occur in 10% of patients at ictus
- May consider treatment for 7 days from SAH
Hyponatremia

- CSW mostly
- Common in SAH
- Thought to be related to neural humoral control of atrial natriuretic factor (ANF) and brain natriuretic peptide (BNP)
Vasospasm
• Vasospasm after aSAH occurs most frequently days 7-10 after aneurysm rupture and resolves after 21 days.

• Angiographic vasospasm occurs in 30 to 70% of patients with uncertain significance.
Risk Factor

- Poor clinical grade
- Fisher grade
- Smoking
- Hyperglycemia
- History of HTN on admission
- Fever
- Hypovolemia
- Sentinel bleed

Frontera et al.  Neurosurgery 58 2006
Table 1.5  Modified Fisher and Fisher Grading Scale for Subarachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Grade</th>
<th>Modified Fisher</th>
<th>% with Vasospasm</th>
<th>Fisher</th>
<th>% with Vasospasm</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No SAH or IVH</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>1</td>
<td>Thin SAH, no IVH</td>
<td>24</td>
<td>No SAH or IVH</td>
<td>21</td>
</tr>
<tr>
<td>2</td>
<td>Thin SAH with IVH</td>
<td>33</td>
<td>Focal or diffuse, thin SAH</td>
<td>25</td>
</tr>
<tr>
<td>3</td>
<td>Thick SAH, no IVH</td>
<td>33</td>
<td>Diffuse thick or localized clot + IC or IVH</td>
<td>37</td>
</tr>
<tr>
<td>4</td>
<td>Thick SAH with IVH</td>
<td>44</td>
<td>No or diffuse thin SAH + IC or IVH</td>
<td>31</td>
</tr>
</tbody>
</table>
Modified Fisher CT

**FIGURE 1.** The Modified Fisher CT rating scale: Grade 1 (minimal or diffuse thin SAH without IVH), indicating low risk for symptomatic vasospasm; Grade 2 (minimal or thin SAH with IVH) and Grade 3 (thick cisternal clot without IVH), indicating intermediate risk for symptomatic vasospasm; and Grade 4 (cisternal clot with IVH), indicating high risk for symptomatic vasospasm. Reproduced with permission from Claassen J, Bernardini GL, Kreiter K, Bates J, Du YE, Copeland D, Connolly ES Jr, Mayer SA: Effect of cisternal and ventricular blood on risk of delayed cerebral ischemia after subarachnoid hemorrhage: the Fisher scale revisited. Stroke 32:2012–2020, 2001 (5).

**FIGURE 2.** Odds ratios for risk of symptomatic vasospasm in the original and modified Fisher CT rating scales. The risk of vasospasm progressively increases for each modified Fisher grade (A), whereas the risk for vasospasm peaks for Fisher Grade 3 and then decreases (B). OR, odds ratio. Vertical bars represent 95% confidence intervals.
Grading SAH

<table>
<thead>
<tr>
<th>Grade</th>
<th>Clinical Description</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Asymptomatic or minimal headache and slight nuchal rigidity</td>
</tr>
<tr>
<td>2</td>
<td>Moderate-to-severe headache, nuchal rigidity, and no neurologic deficit other than cranial-nerve palsy</td>
</tr>
<tr>
<td>3</td>
<td>Drowsiness, confusion, or mild focal deficit</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, moderate-to-severe hemiparesis, and possibly, early decerebrate rigidity and vegetative disturbances</td>
</tr>
<tr>
<td>5</td>
<td>Deep coma, decerebrate rigidity, and moribund appearance</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>WFNS Grades</th>
<th>CGS Score</th>
<th>Motor Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>15</td>
<td>Absent</td>
</tr>
<tr>
<td>II</td>
<td>14-13</td>
<td>Absent</td>
</tr>
<tr>
<td>III</td>
<td>14-13</td>
<td>Present</td>
</tr>
<tr>
<td>IV</td>
<td>12-7</td>
<td>Present or absent</td>
</tr>
<tr>
<td>V</td>
<td>6-3</td>
<td>Present or absent</td>
</tr>
</tbody>
</table>
Presentation Vasospasm

- Confusion
- Delirium
- Change in LOC
- Focal neurologic deficit
### Diagnostic Studies

<table>
<thead>
<tr>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catheter angiography</td>
<td>Most sensitive for large-vessel spasm&lt;br&gt;Enables measurement of cerebral circulation time&lt;br&gt;Can be combined with intra-arterial treatment</td>
</tr>
<tr>
<td>Invasive&lt;br&gt;Labour intensive&lt;br&gt;Cannot be repeated too often&lt;br&gt;Iodine use&lt;br&gt;Radiation exposure&lt;br&gt;Need for transportation</td>
<td></td>
</tr>
<tr>
<td>Transcranial doppler</td>
<td>Non-invasive&lt;br&gt;Done at bedside&lt;br&gt;Can be done daily to follow trends&lt;br&gt;Good correlation with catheter angiography&lt;br&gt;Can be combined with CO&lt;sub&gt;2&lt;/sub&gt;, challenge to test VMR</td>
</tr>
<tr>
<td>Depends on presence of bone windows&lt;br&gt;Operator-dependent&lt;br&gt;Does not assess the microcirculation well</td>
<td></td>
</tr>
<tr>
<td>CT angiography</td>
<td>Non-invasive&lt;br&gt;Can be combined with CT perfusion&lt;br&gt;Good correlation with catheter angiography</td>
</tr>
<tr>
<td>Cannot be repeated too often&lt;br&gt;Iodine use&lt;br&gt;Radiation exposure&lt;br&gt;Need for transportation</td>
<td></td>
</tr>
<tr>
<td>CT perfusion</td>
<td>Evaluates actual cerebral perfusion&lt;br&gt;Quantifiable measures&lt;br&gt;Can detect ischaemia even without detected angiographic vasospasm</td>
</tr>
<tr>
<td>Cannot be repeated too often&lt;br&gt;Iodine use&lt;br&gt;Radiation exposure&lt;br&gt;Need for transportation</td>
<td></td>
</tr>
<tr>
<td>Magnetic resonance angiography</td>
<td>Non-invasive&lt;br&gt;Can be combined with DWI/PWI</td>
</tr>
<tr>
<td>Cannot be repeated too often&lt;br&gt;Need for transportation</td>
<td></td>
</tr>
<tr>
<td>MRI with DWI/PWI</td>
<td>Measures actual cerebral perfusion&lt;br&gt;Easy identification of ischaemic penumbra</td>
</tr>
<tr>
<td>Cannot be repeated too often&lt;br&gt;Need for transportation</td>
<td></td>
</tr>
<tr>
<td>Single photon emission computed tomography</td>
<td>Measures cerebral perfusion</td>
</tr>
<tr>
<td>Cannot be combined with vessel imaging in the same session&lt;br&gt;Radioactive exposure&lt;br&gt;Need for transportation</td>
<td></td>
</tr>
<tr>
<td>Jugular oximetry</td>
<td>Regional measure of brain oxygenation&lt;br&gt;Can be measured frequently</td>
</tr>
<tr>
<td>Invasive&lt;br&gt;Susceptible to artifact</td>
<td></td>
</tr>
<tr>
<td>Brain tissue O&lt;sub&gt;2&lt;/sub&gt;</td>
<td>Local measure of brain oxygenation&lt;br&gt;Continuous measure</td>
</tr>
<tr>
<td>Invasive&lt;br&gt;Very limited spatial resolution</td>
<td></td>
</tr>
</tbody>
</table>

*Continuous transcranial doppler monitoring is also possible.*

**Table 3:** Most commonly used techniques to diagnose and monitor vasospasm and cerebral ischaemia.
PPV: >200 cm/s for MCA  
Mean Flow Velocity: 87%

NPV: <120 cm/s for MCA  
Mean Flow Velocity: 94%

Lindegaard Ratio: Corrects the MFV for hyperemia ie: from increased cardiac output, pressor use, or anemia

Table 1.6 Lindegaard Ratio

<table>
<thead>
<tr>
<th>Lindegaard Ratio</th>
<th>Angiographic Vasospasm</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;3</td>
<td>No spasm</td>
</tr>
<tr>
<td>3–4.5</td>
<td>Mild spasm</td>
</tr>
<tr>
<td>4.5–6</td>
<td>Moderate spasm</td>
</tr>
<tr>
<td>&gt;6</td>
<td>Severe spasm</td>
</tr>
</tbody>
</table>


Frontera: Decision Making in Neurocritical Care
• Large arterial narrowing results in ischemic neurologic symptoms in only 50% of cases
Effect of oral nimodipine on cerebral infarction and outcome after subarachnoid haemorrhage: British aneurysm nimodipine trial


Main results—Demographic and clinical data at entry were similar in the two groups. In patients given nimodipine the incidence of cerebral infarction was 22% (61/278) compared with 33% (92/276) in those given placebo, a significant reduction of 34% (95% confidence interval 13 to 50%). Poor outcomes were also significantly reduced by 40% (95% confidence interval 20 to 55%) with nimodipine (20%) (55/278) in patients given nimodipine v 33% (91/278) in those given placebo). Adverse reactions were reported in 14 patients given nimodipine and 10 given placebo.

Conclusions—Oral nimodipine 60 mg four hourly is well tolerated and reduces cerebral infarction and improves outcome after subarachnoid haemorrhage.


Postoperative hypertension in the management of patients with intracranial arterial aneurysms

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Elevation of systemic arterial pressure in seven patients with intracranial arterial aneurysms has been shown to be effective in alleviating ischemic symptoms attributed to cerebral vasospasm. Autoregulation is at least partially lost in patients with cerebral hemodynamic crisis. Blood volume expansion was used to augment vasopressors in maintenance of systemic hypertension. The management of these cases is discussed. Caution in the use of this technique is advised, since the regimen is not without risk.

KEY WORDS • cerebral vasospasm • hypertension, therapeutic • hypervolemia, therapeutic • intracranial arterial aneurysm • cerebral hemodynamic crisis • cerebral ischemia

J. Neurosurg. / Volume 45 / August, 1976

Summary

Postoperative hypertension, initiated by vasopressors and maintained by hypervolemia, is at times effective in the management of the hemodynamic crisis that may occur after repair of intracranial aneurysm.

Inasmuch as the clinician is usually unable to affect the vascular resistance, increasing the perfusion pressure is the most effective available treatment, provided it is instituted before vascular membranes are extensively damaged.
Endovascular therapies

- Transluminal balloon angioplasty
- Super-selective intra-arterial infusion of vasodilators
• Endovascular interventions

• Fail to respond to triple “H” therapy

• Sudden deficit referable to vascular territory at risk

• Inability to tolerate triple “H” therapy
• Oral nimodipine should be administered to all patients with aSAH (Class 1, Level A)

• Maintenance of euvolemia and normal circulating blood volume is recommended to prevent DCI (Class I, Level B)

• Prophylactic hypervolemia or balloon angioplasty before the development of angiographic spasm is not recommended (Class III, Level B)

• Transcranial Doppler is reasonable to monitor for the development of arterial vasospasm (Class IIa, Level B)

• Perfusion imaging with CT or magnetic resonance can be useful to identify regions of potential brain ischemia (Class IIa; Level B)

• Induction of hypertension is recommended for patients with DCI unless blood pressure is elevated at baseline or cardiac status precludes it (Class 1, Level B)

• Cerebral angioplasty and/or selective intra-arterial vasodilator therapy is reasonable in patients with symptomatic cerebral vasospasm, particularly those who are not rapidly responding to hypertensive therapy (Class IIa, Level B)
Treatment
Aneurysm Location

- ACOMM: 30%
- PCOMM: 25%
- MCA: 20%
- ICA TERMINUS: 8%
- Basilar 5%
ISAT

- 2143 patients randomized to open vs. endovascular treatment
- Primary outcome was death or dependence (mRS 3-6) at 1 year
- 7.4% absolute risk reduction in endovascular treatment
- Pt were good grade, ant. circ., smaller aneurysms (< 10mm) largely
- Late re-treatment 6.9 times more likely in endo group (Stroke 2007)
- Increased re-bleed rate in coiled (10/13) over one year after treatment (Lancet Neurology 2009)
- Risk of death at 5 years: 11% vs. 14% (endo vs. open) (Lancet Neurology 2009)
- Endovascular advantage cannot be assumed in those < 40 (JNS 2008)
Endovascular Aneurysm Embolization
COIL COMPACTION
Stent Assisted Coil Embolization
Flow Diversion
71 y/o female with incidental R paraclinoid aneurysm found for work up of dizziness. She does complain of retro-orbital headache on questioning otherwise asymptomatic
1st device
4mm x 18
“Corked” 1st device
2nd device
4mm x 16
Abciximab (Reopro) 5mg IA administered
PROGNOSIS SAH

- 10% DIE PRIOR TO HOSPITAL
- 65% HAVE COGNITIVE IMPAIRMENT
Interesting Cases