Aneurysmal subarachnoid hemorrhage: medical management and role of PT in the acute setting

Mary Beth Holmes PT, DPT, NCS: Boston University
Stacey Maguire PT, DPT, NCS: Beth Israel Deaconess Medical Center
Jon Robinson PT, DPT, NCS: University of Vermont Medical Center

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Disclosures

• No disclosures
Objectives

• Understand pathophysiology, epidemiology, risk factors and clinical presentation of aneurysmal subarachnoid hemorrhage (aSAH)
• Discuss current medical management for aSAH
• Explore the relationship between vasospasm, delayed cerebral ischemia, early brain injury and prognosis in aSAH
• Discuss the role and safety of PT in early mobilization
• Evaluate and discuss examination and treatment strategies
Epidemiology

• SAH accounts for 5% of strokes
  • Most caused by ruptured saccular aneurysms
• SAH affects approximately 30,000 individuals annually in the US
  • African American > Caucasian
  • Women > Men
  • Mean age at aneurysmal rupture is 55 yrs
• Mortality rates are reported between 30-40%
• Risk of permanent disability is reported as approximately 50%

## Risk Factors

<table>
<thead>
<tr>
<th>Risk factors for Formation of Aneurysms</th>
<th>Risk Factors for Rupture of Aneurysms</th>
</tr>
</thead>
<tbody>
<tr>
<td>HTN</td>
<td>Race</td>
</tr>
<tr>
<td>Smoking</td>
<td>Hispanic ethnic group</td>
</tr>
<tr>
<td>Chronic alcohol consumptions</td>
<td>HTN</td>
</tr>
<tr>
<td>Female sex</td>
<td>Current smoking</td>
</tr>
<tr>
<td>20% of individuals with have multiple</td>
<td>Alcohol abuse</td>
</tr>
<tr>
<td>Genetic conditions</td>
<td>Use of sympathomimetic drugs</td>
</tr>
<tr>
<td>Family history</td>
<td>Aneurysms larger than 7mm</td>
</tr>
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<td></td>
<td>Location of aneurysm</td>
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Mr. T

• 45m admitted to OSH 7/26/13: “worst headache of life”, nausea

• Rapid decline in mental status -> intubated

• Imaging at OSH: SAH R frontal lobe – med-flighted to BIDMC

• CTA at BIDMC: extensive R SAH with IVH, ACOM aneurysm

• 7/26 CTA: diffuse SAH with IVH; focal IPH R frontal lobe; ventricular enlargement; suggestion of inferior tonsillar herniation; low density of the brainstem concerning for edema; multilobed 8X6 ACOM aneurysm
Clinical presentation

• Thunderclap headache (approx. 95% of cases)
• In 10-40% of patients there is a preceding sentinel headache typically 2-8wks before SAH
• Nausea vomiting
• Photophobia
• Stiff neck
• Focal neurological deficits
Diagnosis

- Noncontrast head CT is initial choice in most cases
- CTA or cerebral angiography.
Clinical classification

Hunt and Hess Classification of subarachnoid hemorrhage

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
Mr. T – hospital course

- **EVD** placed and transferred to ICU
  - Initial ICP 3-17 mm HG
- HD 1: underwent cerebral angiography with 4 coils in ACOM- started on hypothermia protocol
- HD 4: re-warmed; patient with seizure- increased Keppra dosage; EVD dislodge and replaced
- Course also significant for UTI, VAP, vasospasm
- HD 6 – repeat angio with intra-arterial verapamil injections
- HD 11 – Trach and PEG
- HD 12 – successful wean from ventilator
Surgical Management

https://www.youtube.com/watch?v=MjOc3Zpv2K8
Vasospasm

- Occurs in up to 70% of patients
- Starts 3-10 days following SAH
- Can result in ischemic stroke
- Presence of vasospasm is associated with unfavorable outcome
- Detected by cerebral angiography or transcranial doppler (TCD)
- TCD
  - Mean flow velocity > 120 suggestive of vasospasm > 200 severe
  - Lindegaard ratio > 3 indicative of moderate > 6 indicative of severe vasospasm

Budohoski et al 2014, Frontera 2009
Delayed Brain Injury

• Delayed Cerebral Ischemia
  • Occurrence of focal neurological impairments or decrease of at least 2 points on GCS that last for at least 1hr and is not apparent immediately after aneurysm occlusion, and cannot be attributed to other causes by means of clinical assessment, CT, MRI and other lab studies.

• Angiographic Vasospasm
  • Arterial narrowing of large cerebral vessels observed on radiological test such as CT angiography, MRA, or digital subtraction angiography.

• Symptomatic Vasospasm
  • Clinical worsening after other possible causes of deterioration have been eliminated

Geraghty and Testai 2017, Fracoeur and Mayer 2016, Vergouwen 2010
Delayed Cerebral Ischemia

• Angiographic vasospasm without DCI
  • Vasospasm occurs in up to 70% after SAH
  • DCI only develops in 20-30%
• No angiographic vasospasm WITH DCI
• Outcomes are more impacted by DCI

Geraghty 2017, D’Souza 2015, Budohoski et al 2014
Summary: Delayed Cerebral Ischemia

- DCI develops in approximately 30% of patients
  - Occurs between 3 and 14 days following rupture

- Factors that increase the likelihood of developing DCI
  - Smoking
  - ETOH use
  - Hyperglycemia
  - Hydrocephalus
  - Poor clinical condition upon initial presentation/Fisher score

- More clinically relevant than vasospasm

- Treatment: Nimodipine

Foreman et al 2017, D’Souza 2015, De Rooij et al 2013
Mr. T cont’d

- PMH: unremarkable
- Medications:
  - Acetaminophen
  - Hydralazine
  - Levetiracetam
  - Nimodipine
Prognosis

• Consistent predictors of outcome:
  ▪ Clinical grade after neurological resuscitation
  ▪ Rebleeding
  ▪ Absence of IVH on initial CT
  ▪ Radiological infarctions on brain imaging (DCI)
  ▪ Age < 50ish

Shukla DP 2017, Galea 2017, Pegoli et al 2015
Is early mobility safe following aneurysmal subarachnoid hemorrhage?
Factors to consider

• Status of aneurysm
• Intracranial pressure/ Cerebral perfusion pressure
• EVD
• Vasospasm —> DCI
• Hemodynamic Instability
• Ventilatory Support
• Unable to provide an accurate meta-analysis of early vs late ambulation in patients with SAH following aneurysm rupture due to a lack of RCT

Ma, Wang & Liu 2013
Karic et al 2017

- Prospective interventional study
- 77 patients in control group - usual care (2010)
- 94 patients in “early rehab group” (2011)
- Early rehab group mobilized based on mobilization algorithm starting first day after aneurysm repair

Karic, Roe, Haug Nordenmark, Becker, Soteberg, & Soteberg 2017
Level of Mobilization

- 0 - Bed rest, head elevation 30 degrees
- 1 - Bed rest, head elevation 60 degrees
- 2 - Bed rest, head elevation 80 degrees
- 3 - Sitting on the edge of the bed
- 4 - Sitting in a chair
- 5 - Standing at bedside, walking a few steps at bedside
- 6 - Walking to the rest room & in the hallway

Karic, Roe, Haug Nordenmark, Becker, Soteberg, & Soteberg 2017
Vasospasm

• Occurrence of vasospasm DID NOT delay mobilization unless patients had severe or clinical signs

Karic, Roe, Haug Nordenmark, Becker, Soteberg, & Soteberg 2017
Karic 2017 Results

• Early rehab group: higher levels of mobility earlier, higher levels of mobility on discharge

• Average level of mobility achieved on day 7 by the control group was bedrest with head of bed 60 degrees; early rehab: edge of bed

• No increased risk of adverse events for the early rehab group
Karic 2017 Results

• Lower rate of clinical vasospasm in the early rehab group
• Clinical vasospasm in early rehab group less severe
• Early Rehab Group demonstrated a 30% risk reduction for severe vasospasm for each step of mobilization achieved during the first 4 days after aneurysm repair
Karic 2016

• Same cohort (Control n=76, Early Rehab n=92)
• Examined outcomes at 1 year: Modified Rankin Scale (mRS), Glasgow Outcome Scale Extended (GOSE)
• Findings:
  ▪ Good clinical status prior to aneurysm repair (based on Hunt and Hess) increased probably of better outcome
  ▪ Patients with poor grade SAH (based on WFNS) had (statistically significant) more favorable outcomes in the early rehab group than the control group

Karic, Roe, Haug Nordenmark, Becker, & Sorteberg 2016
Olkowski 2012

- 25 patients s/p aneurysmal SAH
- Set criteria for early mobility with gradual progression
- Findings:
  - Average days from admission to out of bed: 5.4
  - Average days from admission to ambulation: 10.7
  - Concluded safe and feasible
  - *Mostly Hunt and Hess grades II and III

Olkowski, Devine, Slotnick, Veznedaroglu, Liebman, Arcaro, & Binning 2012
Recommendations for initiating mobility

• Secured aneurysm
• Hemodynamic stability (vitals in desired range)
• No clinical signs of vasospasm (or severe vasospasm on CTA)
• ICP <20
• EVD can be be clamped
• *Throughout PT session - it is essential to be evaluating for changes in neuro status*
Mr. T: Social History

• Works full time as a computer programmer. Lives with wife. No other family in the area.
• Primary language: Vietnamese, fluent in English.
• Baseline independent without assistive device.
• Enjoys taking long walks around the city. Walks to work.
Examination

Hospital day 19
Arousal/Attention/Cognition

• Lethargic, difficult to arouse. Minimal eye opening in supine; 50% of time in sitting with continued stimulation

• Not following commands in supine; followed 4/10 commands in sitting (single-step commands with some visual cueing/demonstration)

• Oriented to self (name) only

• Very minimal verbalizations
Pulmonary

• 35% Trach mask
• Breath Sounds: diminished at bilateral bases
• Cough: moderate strength, congested. Able to somewhat clear to mouth, but therapist assisted with oral suctioning
• Breathing pattern: shallow, +accessory muscle use
Motor function/Muscle Performance

• Some purposeful movements in isolation (moving all extremities)
• Movements were slow, effortful, and demonstrated decreased accuracy
• Prox>distal weakness — Active hand movement and elbow flexion/extension against gravity but trace shoulder and hip movement
• Concern for ICU Acquired Weakness (Critical Illness Myopathy)
ICU Acquired Weakness (ICU-AW)

• Neuromuscular weakness associated with critical illness
• Most frequently associated with sepsis, shock, and multi system failure
ICU-AW

• Critical Illness Polyneuropathy:
  ▪ Distal > Proximal weakness
  ▪ May exhibit concurrent distal sensory loss

• Critical Illness Myopathy:
  ▪ Proximal > Distal weakness
  ▪ No associated sensory loss

• Critical Illness Neuromyopathy:
  ▪ Co-occurrence of both
ICU-AW Diagnosis

- Electromyography (EMG)- [gold standard]
- Medical Research Council (MRC) Scale
  - Total score <48 consistent with diagnosis of ICU-Acquired Weakness
ICU-AW: Prevention is Key

- Schweickert et al, 2009
  - 104 mechanically ventilated patients randomly assigned to usual care vs early mobility group
  - Upon hospital d/c:
    - Patients who achieved independent function: 59% early mobility group; 35% usual care
    - ICU-AW: 31% early mobility group; 49% usual care group
    - Early mobility group also had significantly shorter duration of delirium and more ventilator free days
Mr. T: EMG

• “findings consistent with a myopathy with denervating features, as seen in acute quadriplegic myopathy (i.e. due to critical illness). In addition, there is evidence for a significant central nervous system contribution to the patient’s weakness, as evidenced by the absence of activation of distal lower extremity muscles in which axonal continuity is demonstrable by normal nerve conduction studies. There is no electrophysiologic evidence for a generalized polyneuropathy. “
Balance, Functional Mobility, Endurance

• Supine to sit — total assist with high back sling (ceiling lift)
• Edge of bed balance - static - mod/maxA to maintain with use of LUE support. Loss of balance in multiple directions. Delayed balance reactions. Required assist to return to midline
• Time spent edge of bed: 5 minutes
• Bed to recliner chair - total assist with high back sling
• Left in reclined position
## Hemodynamic Response

<table>
<thead>
<tr>
<th>Position</th>
<th>BP (MAP)</th>
<th>HR</th>
<th>RR</th>
<th>SpO2 (35% Trach Mask)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine</td>
<td>114/70 (85)</td>
<td>72</td>
<td>20</td>
<td>98% (35% Trach Mask)</td>
</tr>
<tr>
<td>Sitting</td>
<td>92/63 (73)</td>
<td>98</td>
<td>24</td>
<td>97% (35% Trach Mask)</td>
</tr>
<tr>
<td>Recovery (Reclined)</td>
<td>110/65 (80)</td>
<td>72</td>
<td>24</td>
<td>96% (35% Trach Mask)</td>
</tr>
</tbody>
</table>
What did we learn from this video?
Observational Analysis

- Arousal
- Bed mobility/ supine to sit
- Communication (yes/no reliability)
- Command following
- Strength
- Ability to use high frequency objects
- Balance
- Endurance
- Tolerance for upright
Assessment
Task Analysis

(Hedman, Rogers, & Hanke, 1996)
Task Analysis

- Temporal sequence of task
- Identify stages of task that are compromised
- Hypotheses for movement dysfunction within each stage
  - Impairments/determinants (Hedman, Rogers, & Hanke, 1996; Schenkman, Deutsch, & Gill-Body, 2006)
    - Neurologic
    - Biomechanical
    - Behavioral
      - cognitive, motivational, perceptual, and emotional aspects
- Intervention strategies targeting specific dysfunction
Early Deficits

• Possible impairments following injury
• Impaired arousal
• Impaired sustained attention
• Impaired force production
• Impaired sensation
## Stages of Learning

<table>
<thead>
<tr>
<th>Stages</th>
<th>Fitts &amp; Posner</th>
<th>Systems Theory</th>
<th>Gentile</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Early</strong></td>
<td><strong>Cognitive</strong></td>
<td><strong>Novice</strong></td>
<td><strong>Initial/Conscious</strong></td>
</tr>
<tr>
<td></td>
<td>- Attention/cognition-heavy</td>
<td>- Inefficient</td>
<td>- Goals: understand task goals &amp; dynamics</td>
</tr>
<tr>
<td></td>
<td>- Trial strategies</td>
<td>- Inflexible</td>
<td>- Start to recognize regulatory features of environment</td>
</tr>
<tr>
<td></td>
<td>- Limit DOF</td>
<td>- Limit DOF</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Large errors – minimal identification</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Variable performance</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Middle</strong></td>
<td><strong>Associative</strong></td>
<td><strong>Advanced</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Refine skills</td>
<td>- Open up DOF</td>
<td>- Optimize force production &amp; timing</td>
</tr>
<tr>
<td></td>
<td>- Less attention-dependent</td>
<td>- More adaptable to environment &amp; task changes</td>
<td>- More consistent, efficient &amp; flexible</td>
</tr>
<tr>
<td></td>
<td>- Error identification</td>
<td></td>
<td>- Fixation for ‘closed skills’, diversification for ‘open skills’</td>
</tr>
<tr>
<td></td>
<td>- Decreased error correction</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Late</strong></td>
<td><strong>Autonomous</strong></td>
<td><strong>Expert</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Increase automaticity</td>
<td>- Able to use all DOF</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Adapt to contexts</td>
<td>- More efficient, coordinated, flexible</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Dual task</td>
<td>- Take advantage of biomechanics</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Error identification and correction</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Performance, Error and Task Difficulty

**FIGURE 4.** The relation between learning curves, performance curves, and the optimal challenge point (OCP) related to 2 performers of different skill levels.
Task Analysis
Attention

• Focalization of awareness
• Capacity is limited but not fixed
• Capacity and demand change
  • Task/context requirement
  • Skill level of the performer
Selective Attention

• Bottom up
  • Directed toward novel or highly familiar/meaningful stimuli

• Top down
  • Specific intention or direction
Video Task Analysis
Task Analysis

• Impairments/Determinants
  • Neurologic?
  • Biomechanical?
  • Behavioral?
    • Attention?
    • Command following?
    • Error recognition?

• Key Interventions
Key Points

- Evaluation and intervention are intertwined
- Attention capacity and sustained attention play a big role in movement dysfunction
- Setting appropriate challenge level is key
- Movement errors are valuable for motor learning
References


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