Avoiding Collateral Damage in Aneurysmal SAH

Mary Kay Bader RN, MSN, CCNS
CCRN, CNRN, SCRN, FNCS, FAHA
Neuro/Critical Care CNS
Mission Hospital
Badermk@aol.com

Disclosures

- Honorarium
  - Bard
- Medical/Scientific Advisory Board
  - Neuroptics
- Board of Directors: President
  - Neurocritical Care Society
- Stock/Stock options
  - Neuroptics
  - Ceribell

Introduction

- Stroke Definition
  - Abrupt and dramatic development of a focal neurologic deficit caused by an occlusion or hemorrhage of a vessel feeding the brain

Hemorrhagic Stroke

- 10-15%
  - Intracerebral
  - Subarachnoid Hemorrhage
    - Aneurysm -5% of all strokes
      - 10.5 cases per 100,000 population
        - 1-5% of population
        - 30,000 annually
      - 50-80% don't rupture over lifetime
    - Vascular Malformations

Aneurysmal Subarachnoid Hemorrhage

Aneurysmal Subarachnoid Hemorrhage

- Sacular outpouching of a cerebral artery which ruptures into the subarachnoid space
- 27,000 cases each year
- Mortality
  - 12% prior to tx
  - 25% within 24 hours
  - 30 day 40-60%
  - Overall 32-67%
- Age 55-60
- 10-20% of survivors have major disability

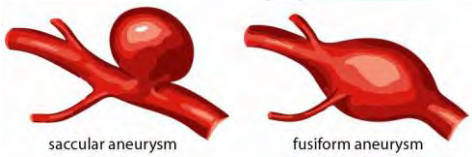
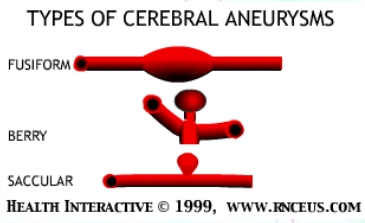
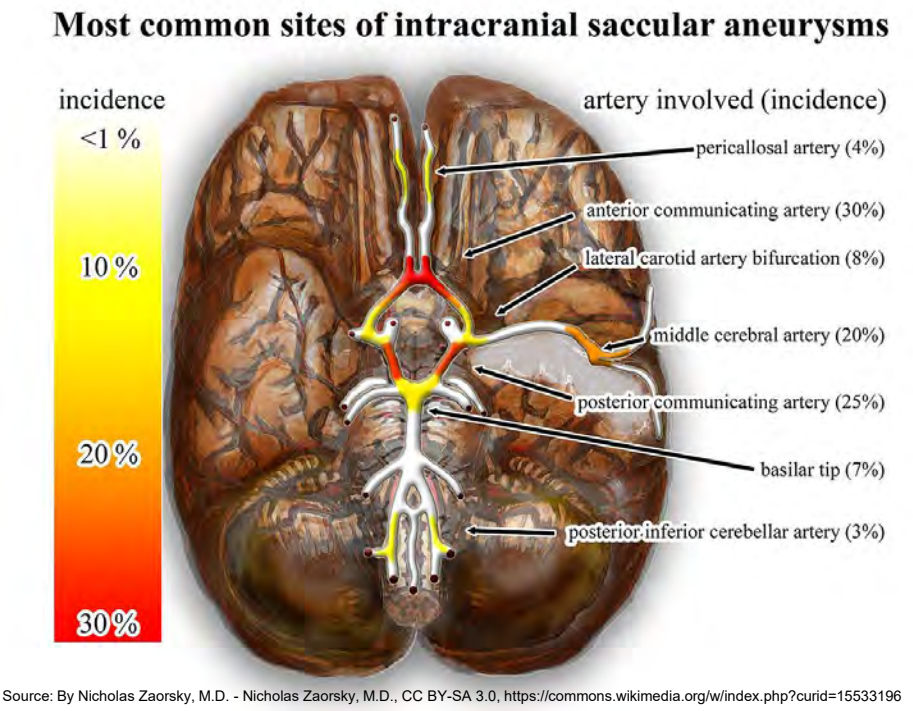


Figure Source: "Transition to Turbulence in Physiological Flows: Direct Numerical Simulation of Hemodynamics in Intracranial Aneurysms and Cerebrospinal Fluid Hydrodynamics in the Spinal Canal". Jain K, 2016 Dissertation.

Types of Aneurysms

- Berry or sacular – acquired, hemodynamically induced injury to vessel wall
- Fusiform
- Mycotic
- Traumatic
- Giant > 25 mm





Source: By Nicholas Zaorsky, M.D. - Nicholas Zaorsky, M.D., CC BY-SA 3.0, https://commons.wikimedia.org/w/index.php?curid=15533196

Aneurysmal Subarachnoid Hemorrhage

- SAH:
  - Rupture of cerebral aneurysm
  - Vasculitis, infectious vascular abnormalities such as mycotic aneurysms, neoplastic lesion hemorrhage, and stimulant drug use
- Risk Factors: Smoking, Hypertension, Women 1.6 higher than Men, African American greater risk, Family History, Older age, Excess alcohol intake/drug use
  - 7-20% have 1st or 2nd degree relative
- Diseases:
  - Polycystic kidney disease
  - Connective tissue disease
  - Ehler Danlos Syndrome type IV & neurofibromatosis type I
  - Marfan Syndrome

Aneurysmal Subarachnoid Hemorrhage

- SAH Signs/Symptoms
  - Worst headache of my life"
    - Sudden, thunderclap, immediate, maximum intensity
    - Warning or sentinel headache precedes in 10-40% of patients
      - Days or weeks prior
  - Loss of consciousness at onset related to initial rise in ICP and reduction of CBF

Aneurysmal Subarachnoid Hemorrhage

- SAH Signs/Symptoms
  - May have increased ICP S/S: decreased LOC, transient or prolonged LOC, nausea, vomiting, syncope
  - Focal neuro signs
    - Hemiparesis, aphasia, abulia (absence of willpower) (ACA/MCA)
    - Occipital or posterior cervical pain: AICA or PICA
  - Photophobia/Nuchal rigidity
  - Brudzinski's sign (passive flex neck involuntary flexion of knees/hips) or Kernig's sign (flex hip @90 – straighten leg pain in hamstring)
  - CN abnormalities
    - III CN: pupil dilation, ptosis, pain behind eye, loss of pupil light reflex
    - VI CN: if in cavernous sinus
    - III / VI with ataxia: Basilar aneurysm

Aneurysmal Subarachnoid Hemorrhage

- Non-aneurysmal SAH
  - 15% patients with spontaneous or non-traumatic SAH no obvious signs of aneurysm, AVM, or other
  - Most common location cisterns around midbrain show bloody CSF
  - Rarely have vasospasm
  - Cause/mechanism unclear

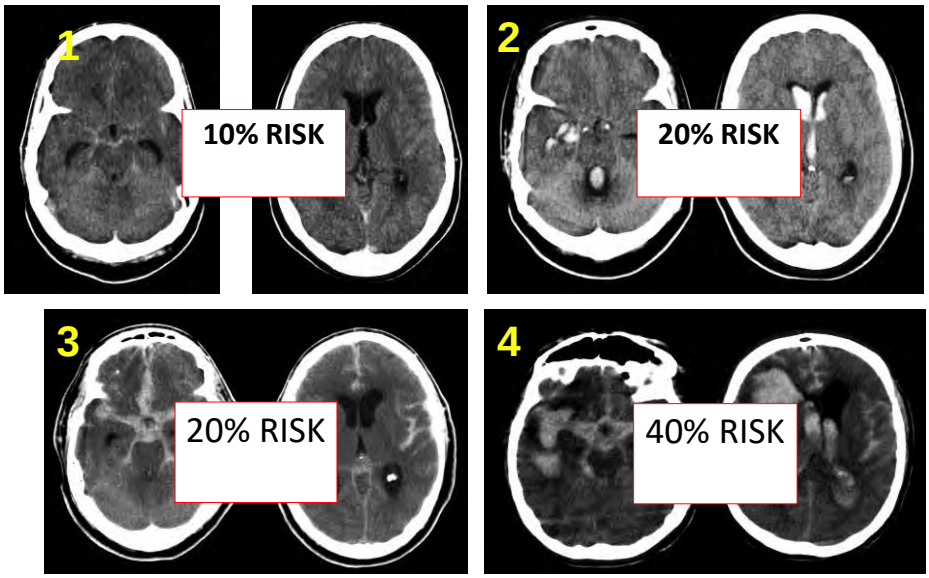
Hunt and Hess

Grade	Description
0	Unruptured aneurysm
1	Asymptomatic or mild headache and slight nuchal rigidity
1a	No acute meningeal or brain reaction, but with fixed neurologic deficit
2	Cranial nerve palsy, moderate to severe headache, nuchal rigidity
3	Mild focal deficit, lethargy or confusion
4	Stupor, moderate to severe hemiparesis, early decerebrate rigidity
5	Deep coma, decerebrate rigidity, moribund appearance

Fisher Score –

Grade	Definition
I	No subarachnoid blood seen on CT
II	Diffuse or vertical layers of SAH < 1mm thick
III	Diffuse clot and/or vertical layer > 1 mm thick
IV	Intracerebral or intraventricular clot with diffuse or no subarachnoid blood

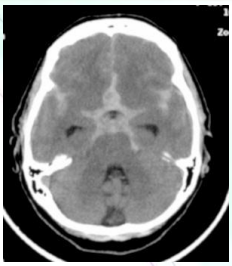
Columbia (Modified Fisher) CT Rating Scale

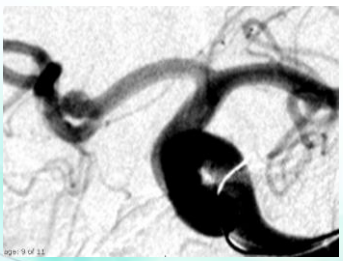


Claassen J et al *Stroke.* 2001;32:2012; Frontera J et al. *Neurosurgery.* 2006;59;21

Diagnostic Tests

- CT scans & CT angio
- MRI & MRA
- Cerebral Angiograms
- Cerebral Blood Flow
  - Xe CT scans
  - SPECT
  - PET





Pathophysiology of Intracranial Chaos

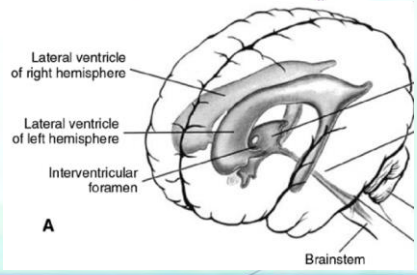
Potential Complication: Rebleeding

- Greatest risk is first 24 hours (8%)
  - Risks – Hypertension, Larger aneurysm size, presence of ICH, Higher mFisher Grade 3-4, & external CSF drainage
  - Results in 50% mortality
- Lethal complication – 8-23% risk
  - Fatal in up to 60% of patients
- 2nd leading cause of increased morbidity and mortality
- Increases with conservative therapy

Rebleeding Risk: van Donkelaar et al 2015: Stroke 46 (8) P 2100-2106. https://doi.org/10.1161/STROKEAHA.115.010037

Immediate Hydrocephalus

- Hydrocephalus with or without intraventricular hemorrhage
  - Often present in ED awake…then…
    - Decompensate within 30-60 minutes
    - Close observation and neuro checks
  - Ventriculostomy
  - If blood in ventricles, clearing clot with tPA



AANN Core Curriculum 2016. Page 23

Increased Intracranial Pressure

- Indications
  - SAH: Low GCS, IVH, Hydrocephalus, signs of herniation
- ICP
  - Types: Parenchymal vs Ventriculostomy
  - Normal
    - 0-20 mm Hg
    - Treat elevations greater than 20 mmHg

Pathophysiology: Hyponatremia

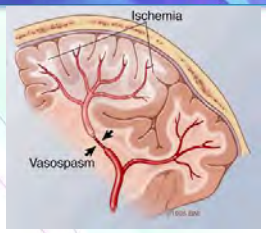
- Most common electrolyte imbalance-34%
- Higher grade SAH
  - Grade III- V
  - Theories

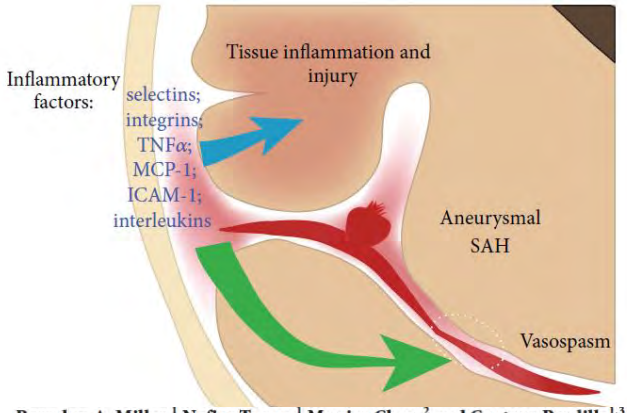
Hyponatremia

- Salt wasting theory
  - Brain Natriuretic Peptide (BNP) Hormone
    - suppresses renin-angiotensin-aldosterone system
    - natriuresis & diuresis
    - rises sharply after initial hemorrhage and just prior to onset of vasospasm
  - Treatment related to volume replacement and salt replacement
  - Fluid restriction could be detrimental to patients

Potential Complication: Vasospasm

- Narrowing of cerebral arteries around the Circle of Willis
  - causes an increase in velocities of arteries
  - decreases blood delivery to cerebral tissue





Brandon A. Miller,[1] Nefize Turan,[1] Monica Chau,[2] and Gustavo Pradilla[1,3]

Review Article
Inflammation, Vasospasm, and Brain Injury after Subarachnoid Hemorrhage

BioMed Research International
Volume 2014, Article ID 384342, 16 pages
http://dx.doi.org/10.1155/2014/384342

Selectins, Integrins, TNFalpha, Macrophage chemoattracant, Leukocyte adhesion, endothelin-1 (potent vasoconstrictor)

Besides Lots of Blood, What Else Can Increase Risk of Delayed Cerebral Ischemia After SAH?

- Poor clinical grade
- Volume depletion (dehydration)
- Sentinel bleeding (double bleed model)
- Low cardiac output (LV dysfunction)
- Smoking
- Chronic hypertension
- Fever

Potential Complication: Vasospasm

- Signs/symptoms
  - In the awake pt.
    - headache
    - increasing lethargy
    - hemiparesis
    - aphasia in dominant hemisphere
  - In the comatose pt. - check PbtO2/TCD
- If untreated--cerebral infarction
  - Delayed Ischemic Neurologic Deficit

Potential Complication: Vasospasm Countermeasures

- Vasospasm
  - Nimodipine
  - Elevate BP
  - CT scan to r/o infarct or bleed
  - Angiogram
    - Intraarterial Verapamil
    - Cerebral angioplasty

Potential Complication: Cardiac Dysfunction

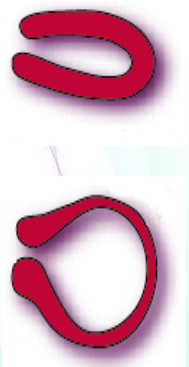
- Myocardial Stunning
- Neurogenic Pulmonary Edema

Myocardial Stunning

When the Worst Headache Becomes the Worst Heartache!

Abdul Hakeem, MD; Adam D. Marks, MPH; Sabha Bhatti, MD; Su Min Chang, MD

Tako-tsubo Cardiomyopathy or Syndrome is also known as:
neurogenic myocardial stunning,
stress cardiomyopathy
stress-induced cardiomyopathy,
transient left ventricular apical ballooning, "ampulla" cardiomyopathy" or "broken heart syndrome".

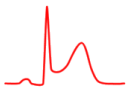


Google Image Result for http://www.takotsubo.com/summary%202.jpg

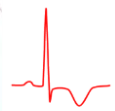
Tako-tsubo Syndrome

- Severe tako-tsubo syndrome can be lethal…leading to
  - Cardiogenic shock
  - Diagnosis
    - Cardiac bio markers of heart damage (troponin, creatine kinase) are only very slightly elevated
    - EKG - non-specific ST-T abnormalities, ST elevation, and/or QT prolongation with large negative T waves

ECG Changes in *Tako-Tsubo* Cardiomyopathy



Stage 1: *acute stage*,
This stage lasts only a few hours. Stage of ST elevation and fairly short QT interval. The R wave might be preserved.



Stage 2: *sub acute stage*.
This stage can last days. QT segment prolongation and large and deep negative T waves.

Stage 3: *recovery stage*. Flipped T wave persists for days to weeks, but QT interval is again normal.

Treatment of Myocardial Stunning

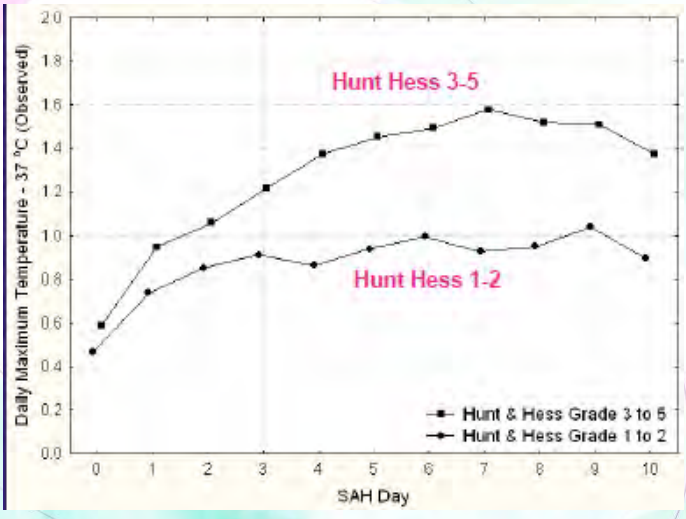
- Supportive
  - Unload the left ventricle
  - Reduce vasopressors
    - Usually initiated due to low BP…result is increased SVR and afterload
    - Makes it difficult for left ventricle to contract
  - Use contractility agents if possible
    - Dobutamine
  - Address pulmonary edema
    - Lasix

Potential Complication
Fever

Fever: Danger of Hyperthermia SAH

- Fernandez et al 2007)
  - 353 pts with mean temperature > 37.5 degrees C during the first week
  - Fever predicted by poor clinical grade, increased age, male
  - Fever independently associated with
    - Death & severe disability
    - Poor cognitive outcome
- Ramer et al 2017 (NCC 26:41-47) 584 patients
  - Early onset of fever, number of hours of fever, & days of fever associated with poor functional outcome

Time Course of Fever Burden: SAH



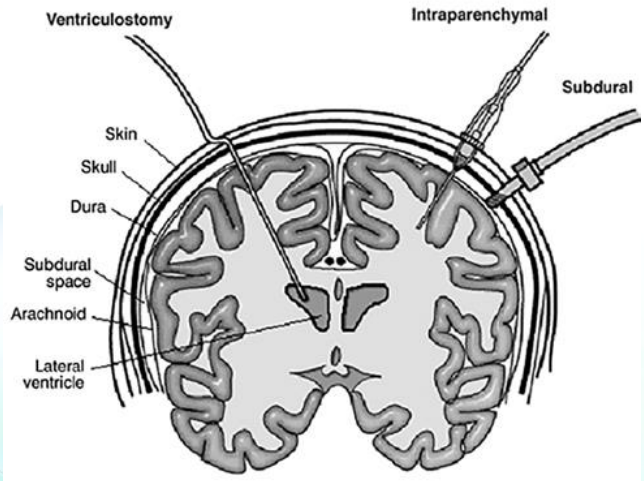
Source: Fernandez et al 2007: DOI: https://doi.org/10.1212/01.wnl.0000258543.45879.f5 Neurology

Monitoring Technologies with SAH

- ICP monitoring
- Brain Tissue Oxygen Monitoring
- Transcranial Dopplers (TCDs)

Intracranial Pressure Monitoring

- ICP Monitor



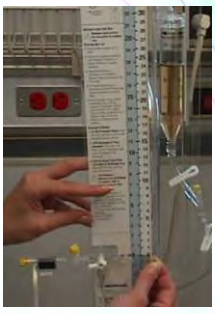
Source: "Treatment of Intracranial Hypertension," by K. M. Giugno, T. R. Maia, C. L. Kunrath, & J. J. Bizzi, 2003, *Jornal de Pediatria (Rio J), 79*(4), pp. 287–296. Copyright 2003 by Elsevier.

ICP

- Gold standard – venticulostomy
  - Monitors ICP
    - Normal 0-15 mm Hg
    - Abnormal > 20 mm Hg
  - Provides CSF drainage

ICP: Nursing Implications CSF Drainage

- Level CSF drainage system with zero reference point
- Drain CSF
  - Produce ≈ 20 cc/hour
  - 125-150cc circulating at any given time
    - 20% in the lateral ventricles
- Do not over drain
  - Over-drainage
    - Sagging cerebrum
    - Pulling of bridging veins
    - Hematoma development
- Assure aseptic technique at all times when changing bag/sampling



Manage Increased ICP

- HOB at 30 degrees
- Drain CSF
- Sedation/Analgesia
  - Short acting desirable
  - Sedation vacations generally not indicated if ICP ⬆
- Ventilated: Do not hyperventilate
  - Maintain PaCO2≥35 mm Hg
- Mannitol/Hypertonic Saline
- Manage MAP/BP
- Normothermia

Continuous Drain: How long do you wait after [illegible] document the ICP?

Journal of Neuroscience Nursing

Intracranial pressure (ICP) is often obtained via external ventricular drain (EVD) placement and is discussed as a key vital sign in neuroscience. Nurses are most often delegated the task of observing, adjudicating, and documenting ICP. Cerebrospinal fluid drainage requires that the transducer connected to the EVD is open to drain, prohibiting ICP monitoring. There are no recent data to support an evidence-based standard for the period an ICP waveform should be observed, after the EVD is clamped, to be able to adjudicate a value that represents the patient's status. Therefore, the purpose of this study is to determine the optimal period for which an EVD should be closed to obtain an accurate ICP value. In a sample of 30 subjects who received continuous ICP monitoring for 15 minutes, there was no universal pattern to ICP after clamping an EVD. The conditional probability of observing a patient's highest ICP if ICP is observed for 5 minutes, is 0.0181. The conditional probability increased to 0.0402 if ICP is observed for 10 minutes. There were no instances of ICP elevation requiring intervention. The results suggest that at least 5 minutes of ICP monitoring is safe and is required to provide an ICP value that reflects true ICP.

Intracranial Pressure Values Are Highly Variable After Cerebral Spinal Fluid Drainage

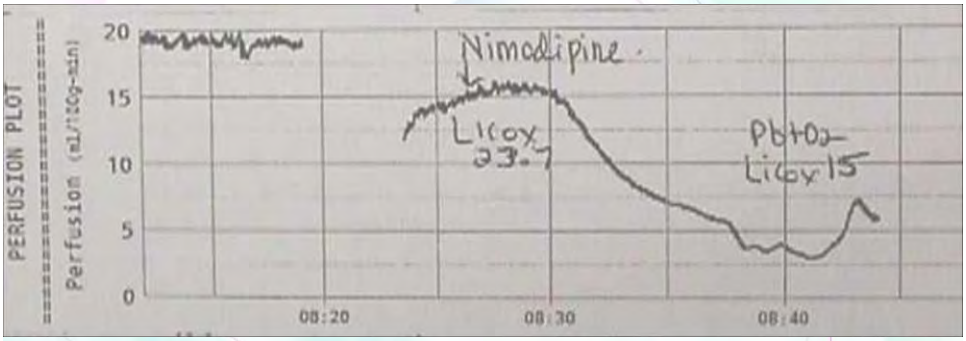
Michael Rogers, Sonja E. Stutzman, Folefac D. Atem, Samanpita Sengupta, Babu Welch, DaiWai M. Olson

Volume 49 • Number 2 • April 2017

Brain Tissue Oxygen

- Evaluate the possible causes of decline
  - Increased Intracranial Pressure
  - Vasospasm
  - Pulmonary
  - Cardiac/Hemodynamic

Impact of Nimodipine on PbtO2 and CBF



Transcranial Dopplers

- Increased velocities correlate with vasospasm
  - Mean velocities: Norm MCA mean 55-70 ± 10cm/s
    - >200 = critical velocities
  - Lindegaard Ratio = $\frac{\text{Mean MCA}}{\text{Mean ExICA}}$
    - Normal: < 1.7
    - Moderate spasm: > 3
    - Severe spasm: > 6

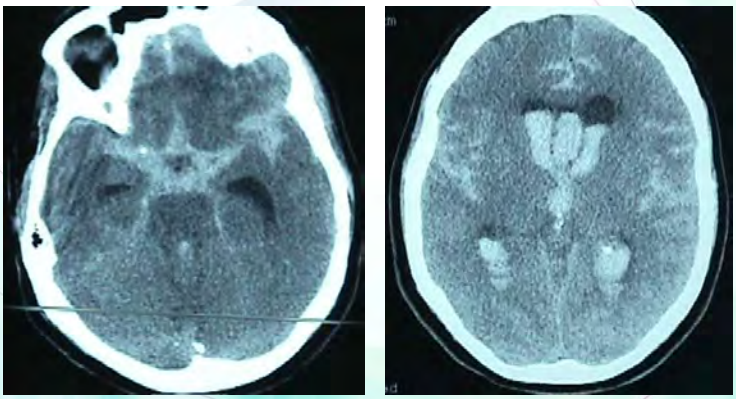
Recognition of SAH

- "Worst headache of my life"
  - Sudden, thunderclap, immediate, maximum intensity
    - Warning or sentinel headache precedes in 10-40% of patients
- Associated signs and symptoms:
  - Nausea and/or vomiting
  - Meningeal irritation from blood in Subarachnoid Space
    - Nuchal rigidity
    - Photophobia
  - Loss of consciousness: Brief vs Sustained
  - Focal cranial nerve palsy; 3rd nerve in PCA aneurysms
  - Seizure

Patient Arrival: Initial Interventions

- Stroke Alert – Stroke Team in Emergency Department
- Initial interventions:
  - Airway/Breathing: Continually reassess for changes in condition necessitating airway control
    - Intubation
  - Circulation – HR and BP evaluation and IV insertion
  - Deficit- Neuro exam and ICP evaluation
  - Laboratory and Radiographic Evaluation
- CT or LP: SAH is confirmed
  - The goal is to reduce the chance of aneurysm re-rupture and expedite treatment of the aneurysm while preventing any medical complications
- BP after confirmation
  - Keep < 140 – 160 mm Hg

Management Priorities after confirmation of SAH



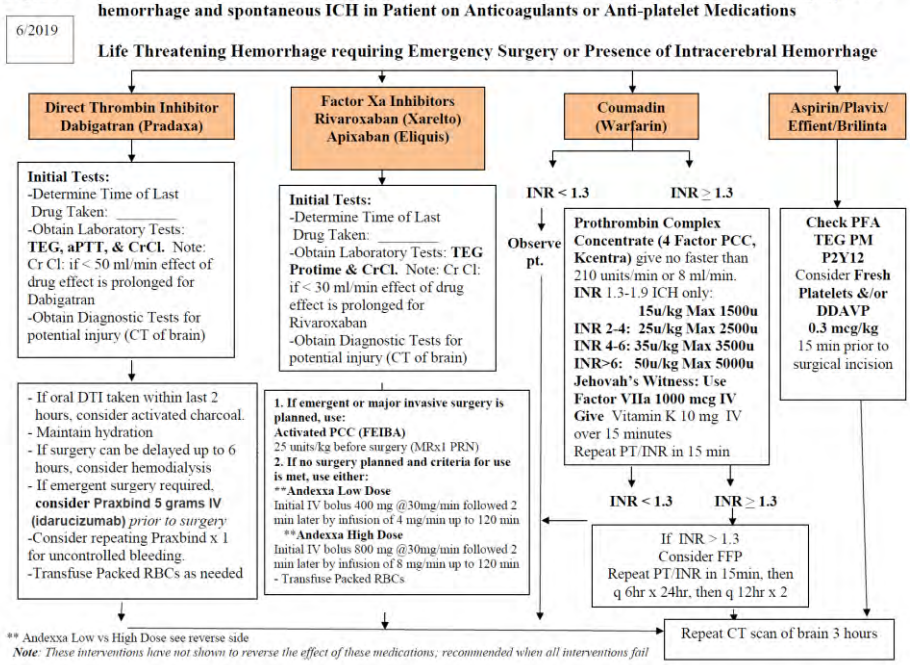
Aneurysmal SAH

- Phase I: Hemorrhage Ictus – Securement
  - Control acute hypertension: SBP Control < 140-160 mmHg
    - Nicardipine and labatelol
  - Keep environment quiet
  - Manage Pain/Headache
  - Reassessment of Neuro Status
    - Look for decline related to hydrocephalus
    - Management of ICP
  - Anti-fibrinolytic if delay in securement
    - Decreased rebleeding, associated with clot formation (DVT)
      - Amniocaproic acid, tranexamic acid

Correct coagulopathies

- Thrombocytopenia: Give platelets if < 100,000
- If patient taking anticoagulation medications
  - Warfarin
    - PCC (Kcentra)
    - FFP (less efficient)
    - Vitamin K 10 mg
  - Other anticoagulants
    - Factor XA inhibitors: Rivaroxaban/Apixaban/Edoxaban
      - Andexxa
    - Direct Thrombin Inhibitors: Dabigatran
      - Antidote: Idarucizumab

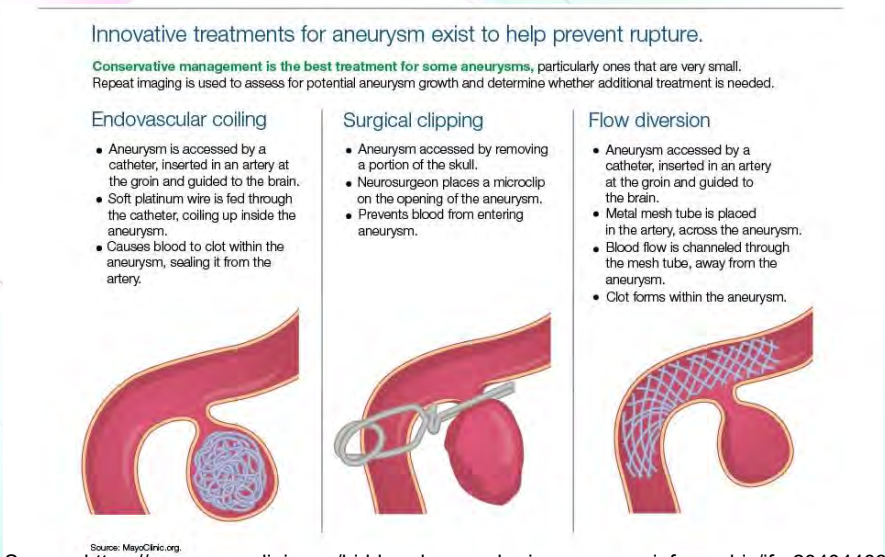
Figure 1. Decision Tree for Use of Reversal Agents in Life threatening Hemorrhage, Traumatic Brain injury with hemorrhage and spontaneous ICH in Patient on Anticoagulants or Anti-platelet Medications



Aneurysmal SAH

- Phase II: Management of Aneurysm Patient
  - Secure early rather than later
  - Decision on How?
    - Clipping (open craniotomy)
    - Coiling (interventional tx)
    - Stenting
  - Location, morphology, risks, & personnel available

Isolating the Aneurysm: Surgical Clip, Coil or Stent



Source: https://www.mayoclinic.org/hidden-dangers-brain-aneurysm-infographic/ifg-20404403

Operative Phase Interventions

- SAH
  - Ventriculostomy placement
  - Ancillary monitors: CBF/PbtO2/Microdialysis
  - Transport of patient if ventilated
- SAH
  - Monitor during cerebral angiogram and coiling procedures
  - Monitor during clipping procedure
  - Be prepared for possible rebleeding during aneurysmal securement

Aneurysmal SAH

- Phase III: Post OP / Vasospasm (Delayed Cerebral Ischemia DCI)
  - Post op Care (usually in ICU for close monitoring)
    - Airway/Breathing
      - Intubated with mechanical ventilation
        - Keep PaCO2 > 35 mm Hg
      - Sedation with goal to extubate
        - Extubated: Limit sedation if possible
    - Circulation: Maintain BP 120-140 mm Hg
    - ICP Management
      - Ventriculostomy for CSF drainage
      - Mannitol / Hypertonic Saline

Nursing Management

- Assess neuro status closely
  - Monitor LOC closely-watch for neuro changes
  - Assess motor status-check strength and note increase in tone
  - Cranial nerves-especially III, IV, VI; V & VII; and IX & X
  - Sensory status changes

Nursing Care: Prevent Complications

- Manage Blood Glucose
  - Avoid Hypoglycemia
  - Treat Hyperglycemia
    - 80-180 mg/dL
- Prevent Hyperthermia
  - Evaluate potential sources of infection
  - Normothermia should be maintained
    - Antipyretic agents
    - Ice bags
    - Iced Saline Boluses
    - Surface or Endovascular cooling devices

Nursing Management

- Assess Swallow
- Provide nutrition
- Establish bowel program
- Foley care – Remove ASAP
- Mobility: Range of motion
- DVT prevention
- Good skin care
- Family involvement
- PT/OT/ST & Rehab consult

Post-op

- Vasospasm: greatest risk 4-14 days post bleed
  - Manifested by delayed neurologic deficit
  - Prevention is the key!
    - Nimodipine
    - Avoidance of hypovolemia and hyponatremia
  - Detection and Diagnosis
    - Monitor clinical exam
    - Monitor sodium
    - Monitor TCDs
    - Comatose Pt: Multimodality monitoring

Aneurysmal SAH

- Nimodipine 60 mg
  - Oral calcium channel blocker
  - Does NOT prevent vasospasm
  - Is associated with improved outcomes at 90 days
    - Unclear mechanism
      - Neuroprotectant
      - Support of collateral circulation
  - Dosing 60mg po every 4 hours
    - Hypotension side effect
    - Dose may be split to 30mg po every 2 hours
    - When to discontinue of significant impact on BP
  - Do not take with grapefruit juice

Treatment

- Induced Hypertension
- Volume optimization – isotonic fluids euvolemia
- Rescue Therapy for Refractory DCI
  - Hemoglobin optimization *9-10 g/L
  - Endovascular Therapy
    - Dilation of vessels- PTCA
    - Intra-arterial infusion of vasodilators
  - Cardiac Output Optimization
    - Use CO monitoring system
    - Optimize fluids/inotropes (dobutamine/milrinone)
  - Tier 2: Non-evidence Measures
    - Increase sodium goals
    - Aortic flow diversion, Intrathecal Nicardipine, IABP counter-pulsation

Discharge Planning

- Transitions in care
  - Home
  - Acute Rehab
  - Skilled Nursing Facilities
- Engage Care Partners in process

Case SP: Event

- 59 year old female experiences the worst headache of her life
  - 911 called by husband
    - Pt loses consciousness and vomits several times
  - Transported by EMS
    - Deteriorates en route
  - Taken to nearest receiving center
    - Lays on gurney in hallway of ED for 45 minutes
    - Husband notes wife is posturing

Events at Hospital X

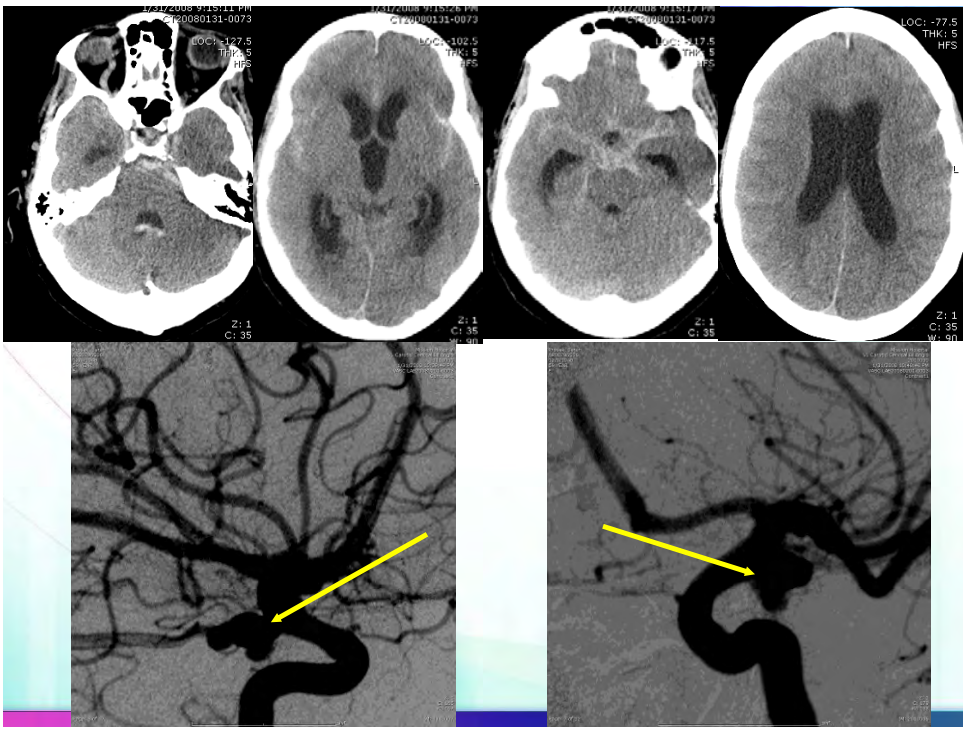
- ED
  - Intubation takes place in ED 45 minutes after arrival
  - GCS 1-1-1
  - CT scan reveals SAH
  - Admit to CCU
    - No ICP
    - No diagnosis except for SAH
- Next Day…

Enter Cousin Connie……..

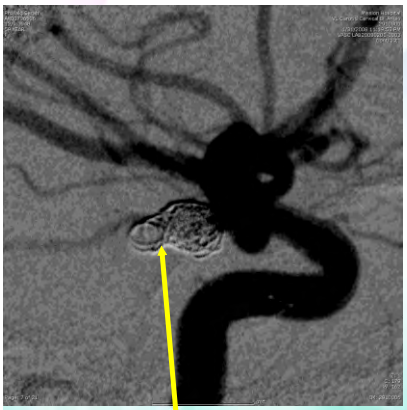
- Phone call from her mom
- Phone call to Mary Kay…
  - Words of wisdom: Get her out of there….
- Connie arrives at Hospital X
  - Phone call to Mission Neurointerventional team
  - Phone call to Mary Kay
- Attempted conversation with Neurosurgeon
  - Phone call to Mary Kay
- Attempted conversation with Neurosurgeon
  - Phone call to Mary Kay
- Conference with COO, QI Director, and CNO at hospital X
- Getting her the heck of out of there……to Mission

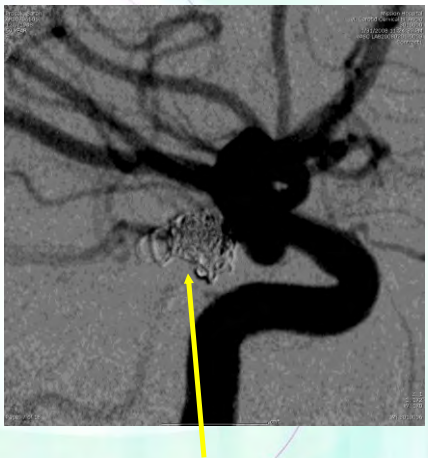
Arrives at Mission 22 hours After Event

- To CT and CT angio
  - Obstructive hydrocephalus
  - Left posterior communicating artery aneurysm
- To OR
  - Ventriculostomy and LICOX
- To Interventional
  - Coiling of aneurysm
- To SICU

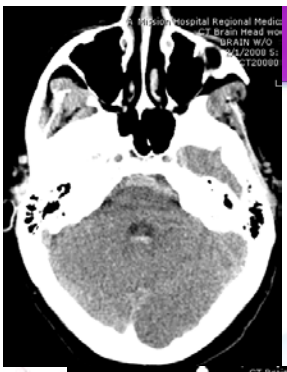


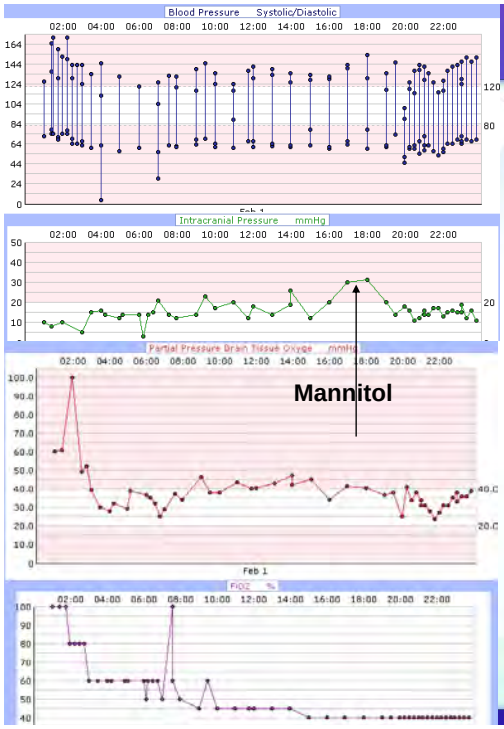
Coil Embolization





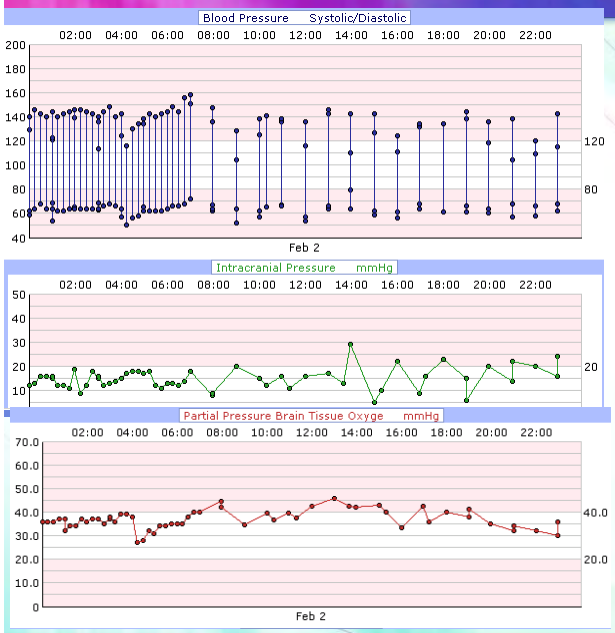
Post Op





Feb 1

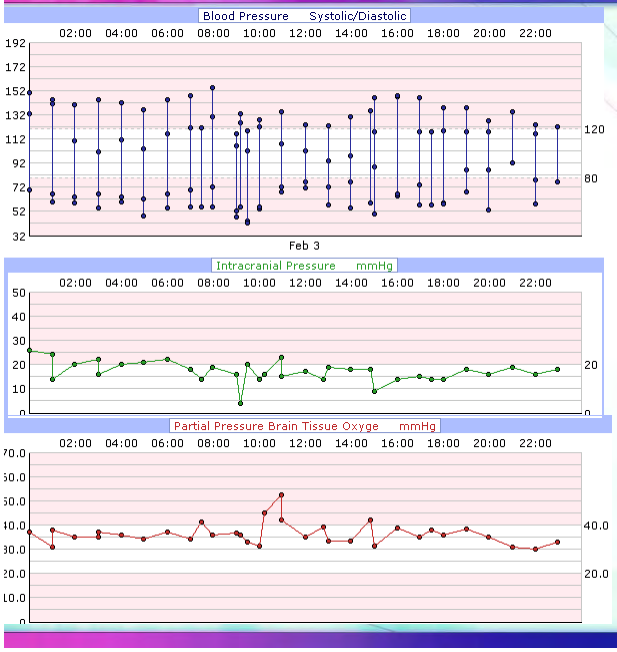
- Stabilize postop
  - BP 120-140
  - ICP
    - Initially < 20
    - 6pm: 30 mm Hg
  - PbtO2
    - Titrates down with decreasing FIO2
  - TCDs normal



Feb 2

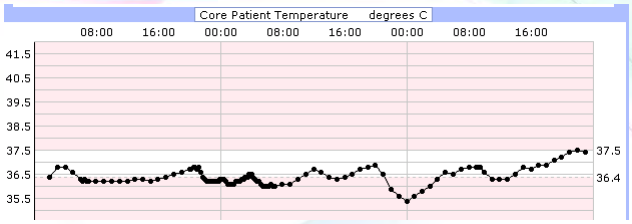
- BP 120-140
- ICP 10-30 mm Hg
  - Drain CSF
  - HS/Mannitol
- PbtO2 stable

Feb 3

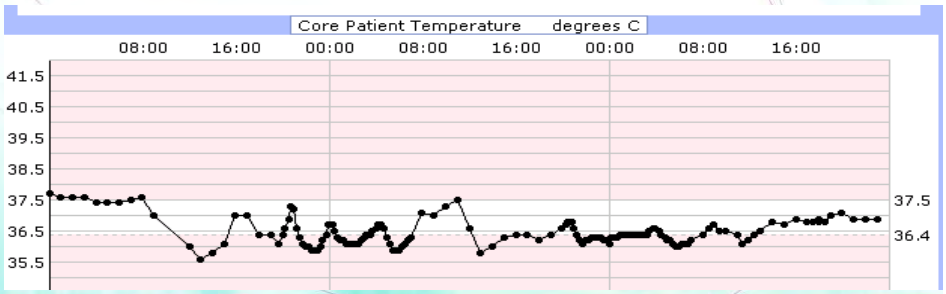


- BP 120-140
- ICP 10-28
  - Drain CSF
  - Mannitol/HS
- PbtO2 stable

Temperature Control

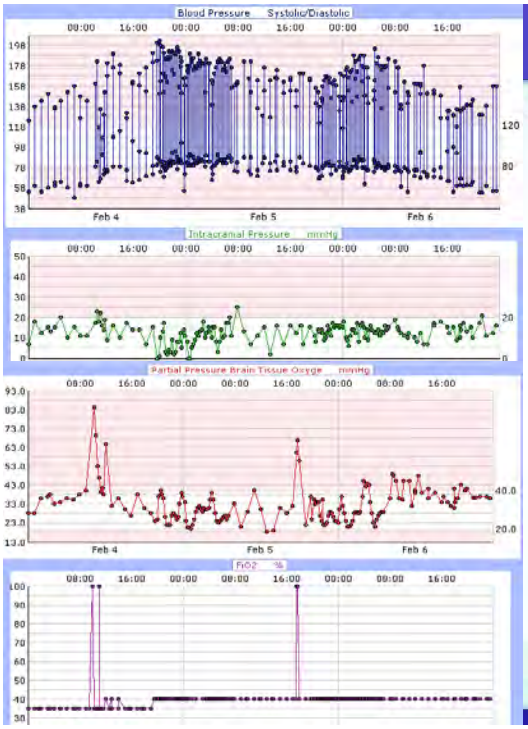


- Temp
  - 36 to 37



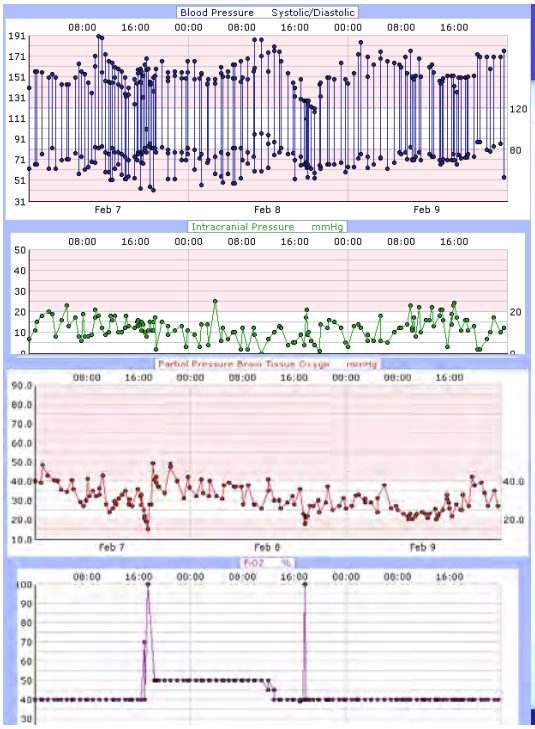
Bispectal Index Monitor

- Shivering
  - Watch for increase in EMG activity
  - 1st sign of shivering
  - Use early shivering strategies
    - Counter warming
    - Acetaminophen
    - Buspar and Demerol
    - Magnesium



Feb 5-6

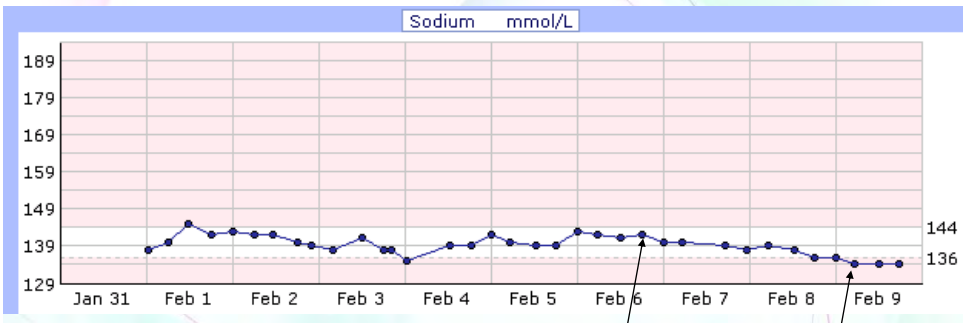
- Systolic BP
  - 160-180 mm Hg
  - Vasospasm detected on TCD
    - R2.3 L 1.7
    - R MCA 111
- ICP < 20
- PbtO2 ↑ with FIO2 ↑
- Respiratory problem
  - Bronchoscopy on 2/5
    - Mucous plugs
  - Tracheostomy on 2/6
  - Negative lower extremity venous dopplers



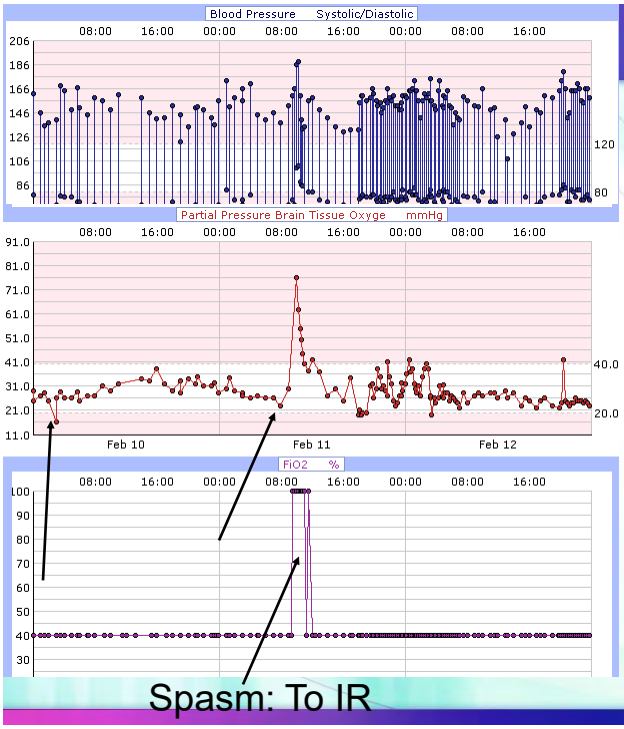
Feb 7-9

- Vasospasm
  - 2/7 Lindegaards
    - 2.7 R
    - 1.5 L
  - 2/9
    - 2.3 L
    - 2.4 R
  - Systolic BP 150-160
    - Phenylephrine
    - Norepinephrine
  - ICP controlled
  - PbtO2 drops

Sodium Trends Indicative of Spasm



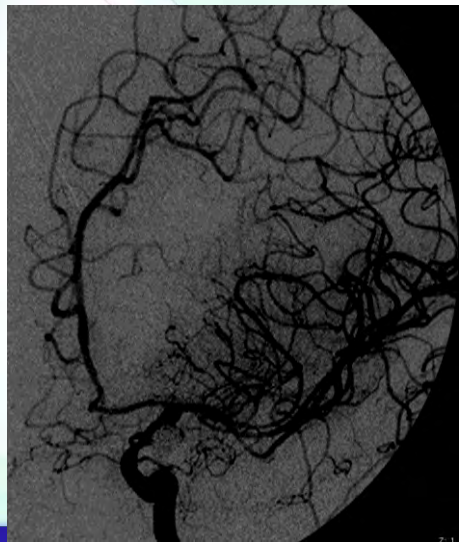
Drop in Sodium over 1-2 days

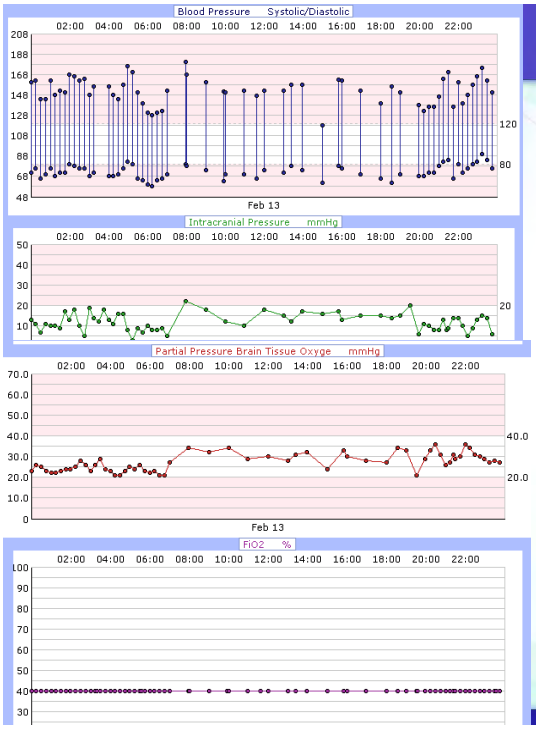


Feb 10-12

- Spasm continues
  - 2/10: Lind 3.2/2
    - RMCA 156
    - LMCA 162
  - 2/11: Lind 3.5/3.4
    - L/R MCA 115
- SBP 160-180 mm Hg
- PbtO2 indicative to spasm
- 2/10 and 2/11: IR
  - RICA/LACA moderate spasm tx with IA verapamil

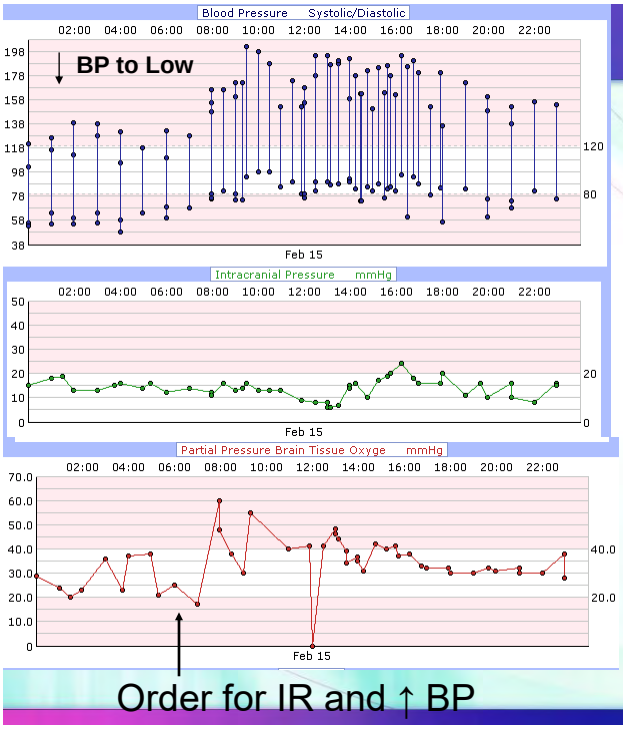
Day 11





Feb 13

- Day 13
  - TCDs better
  - ICP better controlled
  - PbtO2 up and down



Feb 15

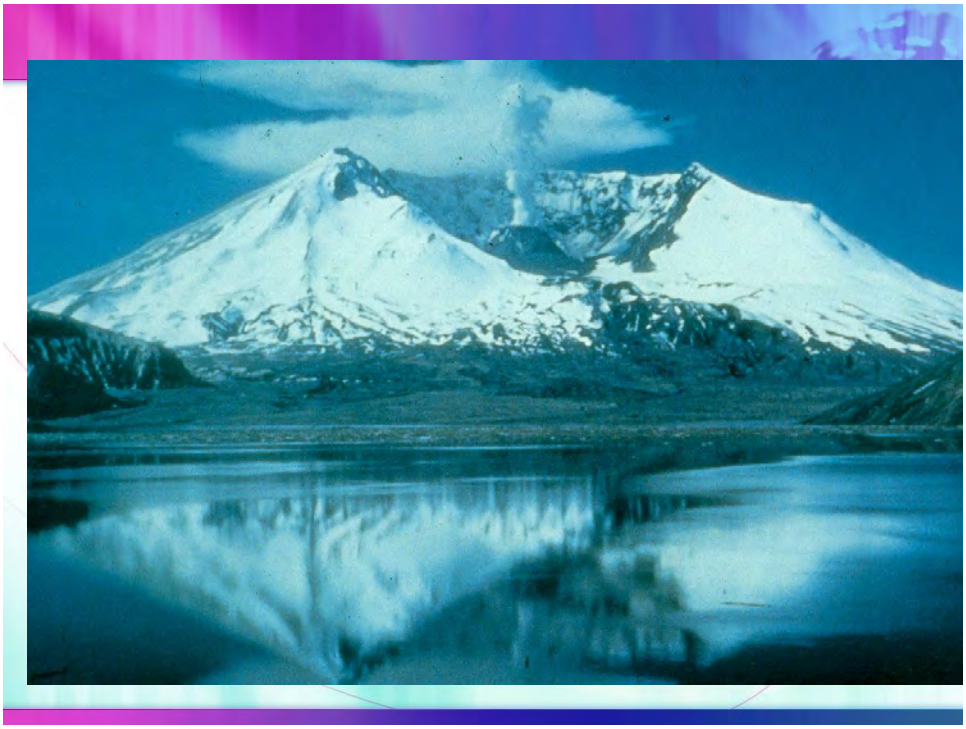
- 8am TCDS
  - Severe Spasm with ↑ velocities
  - To IR for TX
    - RICA mod
    - LICA/LACA mod to severe spasm

Days 21-30

- Day 23
  - Bilateral non-occlusive common femoral dvt
  - Placement of IVC filter
- Day 24
  - Weaning sedation
  - Opens eyes/smiles
- Days 25
  - GCS 4-6-1T
  - Mouthing words
- Day 28
  - Up in chair
  - GCS 4-6-5: Able to speak and eat with PMV
  - Transferred to PCSU

Outcome

- Transfer to ARU
- Discharged Home
  - Independent
  - Back to work by 6 months



Questions