Intracranial Aneurysms

Intracranial aneurysms are abnormally shaped cerebral arteries with the potential for devastating neurological consequences secondary to rupture and associated complications. Ruptured aneurysms are associated with 25% mortality and significant morbidity in 50% of survivors. Risk factors for rupture include previous rupture, size > 10 mm, hypertension, smoking, EtOH, cocaine / amphetamine use, & OCP use.

ANESTHETIC CONSIDERATIONS:

- Unruptured aneurysm
  - Potential for rupture with massive blood loss
  - Need for tight hemodynamic control
- Specific considerations for **ruptured aneurysm**:
  - Potential emergency surgery with unstable patient, full stomach, inadequate history
  - Prevent secondary brain injury – maintain CPP and O2 delivery in setting of ↑ICP +/- vasospasm
  - Avoid rebleed – maintain TMPG (avoid rapid swings in ICP and MAP)
  - Potential massive blood loss
- Potential increased ICP – maintain CPP
- Complications associated with **SAH**:
  - Neurologic
- Rebleeding, vasospasm, hydrocephalus, ICH, IVH – cerebral protection strategies
  - Systemic
- Neurogenic pulmonary edema, myocardial stunning, arrhythmias, hypoNa (SIADH, CSW)
- Comorbidities (HTN, smoking, CAD)
- Surgical considerations of surgical access and patient positioning including:
  - Potential need for DHCA, prone / sitting / supine position +/- head rotation, decreased access to patient including airway
  - Brain relaxation
  - Rapid, smooth emergence for postop neurological assessment

ANESTHETIC GOALS:

- Ruptured:
  - ABCs, seizure control (if present), maintain adequate CPP, manage raised ICP, address complications of SAH
- Precise control of systemic and cerebral hemodynamics to prevent rupture or rebleeding
  - Avoid increases in transmural pressure gradient (TMPG) and acute hypertension
  - Precisely manipulate MAP during application of surgical clips and attempts to control bleeding
- Prevent secondary brain injury
  - Maintain high-normal MAP to prevent critical reductions of CBF; balance with risk of aneurysm rupture and raised ICP management
- Facilitate surgical access to aneurysm
  - Intraoperative brain relaxation to facilitate surgical access to aneurysm
- Hyperventilation, osmotic diuresis, barbiturates, CSF drainage
  - Prevent patient movement
- Assess and manage associated metabolic abnormalities
- Rapid return to a level of consciousness that permits neurologic assessment post-operatively

HISTORY

- Standard anesthetic history
- Has rupture occurred?
  - Severe HA ("worst HA of my life"), neck stiffness, N/V, photophobia, lethargy, transient LOC, cranial nerve deficits
  - Small herald bleed in 50%; non-specific mild symptoms including dizziness, retroorbital pain, mild neurological deficits, often misdiagnosed
- Is there elevated ICP? (HA, vision changes, decreased LOC, CN III palsies)
- Risk factors for SAH
  - Smoking, HTN, EtOH, OCP, FmHx, hypercholesterolemia, congenital weakness of vessel wall
- Coexisting disease
  - CAD
  - Connective tissue disease (Ehlers Danlos type IV, Marfan's, fibrous dysplasia, neurofibromatosis type I, adult PCKD)
- Assign a HH or WFNS-SAH grade (see Pathophysiology section)

PHYSICAL

- **GENERAL**
  - Vitals including evidence of Cushing reflex (bradycardia, HTN, irregular respiratory pattern)
- **CNS**
  - GCS, pupils
  - Focused neurologic exam noting any deficits, neck stiffness
  - Signs of raised ICP (decreased LOC, papilledema, visual field deficits)
- **CVS**
  - Volume status, heart rhythm, heart sounds
- **RESP**
  - Respiratory distress, air entry, crackles (pulmonary edema)

INVESTIGATIONS

- **Labs**
  - CBC/D, coags, x-match
  - Electrolytes (hypoNa, hypoMg), Posm, urea, Cr
  - Troponin if EKG changes
- **Imaging**

- EKG: “canyon T-waves”, long QT (risk Torsades w/ > 550 ms), ST depression, or U-waves
- CXR: pulmonary edema
- CT head: non-contrast
- Angiography – CTA, MRA, intraarterial catheterization

- **Special**
  - LP if suspected SAH and CT normal (xanthochromia)

OPTIMIZATION

- If ruptured
  - Tight hemodynamic control to maintain CPP
  - Manage raised ICP (elevate HOB, 100% O2, mild hyperventilation, mannitol)
  - Manage fluid and electrolyte abnormalities
  - Aspiration prophylaxis if emergency surgery
- Unruptured
  - Optimize comorbidities
  - Continue preop meds – seizure prophylaxis, nimodipine for vasospasm prophylaxis
  - Sedation to prevent stress response with HTN (caution: hypercarbia will ↑ICP)

ANESTHETIC OPTIONS

- Microsurgical clipping requires GA with ETT
- Coiling of aneurysm by interventional neuroradiology
  - GA advantages:
    - Improved visualization for interventionalist
    - Ability to provide temporary apnea for angiography and coil insertion
    - May require asystole for coil insertion to prevent downstream dislodgement (defibrillation pads pre-op, adenosine 1 mg/kg)
  - MAC / TIVA advantages - continuous evaluation of neurological function

ANESTHETIC SETUP

- **Drugs**
  - Hypotensive agents: remifentanil, STP, propofol, hydralazine, labetalol +/- SNP
  - Brain relaxation agents: mannitol, furosemide
  - Cerebral protection agents: STP
  - Local anesthetic for airway topicalization and head pinning
- **Equipment**
  - CAS monitors including 5 lead EKG
  - Large bore IVs with blood set
  - Pre-induction arterial line for continuous assessment of transmural pressure gradient
  - Consider CVL for volume management with large doses mannitol, hemodynamic manipulation
    - Trendelenberg position will further increase ICP; consider femoral access
  - Foley
  - Twitch monitor
  - Surgeon may use intraoperative angiography
  - Consider ICP monitor for calculating CPP
  - Consider SSEPs or BAEPs for vertebrobasilar aneurysm clipping
  - Consider EEG and jugular venous bulb monitoring if temporary clipping anticipated

MANAGEMENT OF ANESTHESIA

- **Induction**
  - Actual technique unimportant provided that exaggerated hemodynamic response to intubation is avoided; titrate to deep plane of anesthesia
    - Barbiturates protect against ischemia
    - Propofol not protective against ischemic insults
    - Etomidate not protective against ischemia; may worsen cerebral oxygenation?
    - Inhalational induction relatively contraindicated (high concentration volatile will ↑CBF and ICP)
  - Blunt hemodynamic response to intubation... HTN will disrupt poorly organized clot → re-bleeding at induction often fatal
  - Hypertension or sudden ↓ICP may ↑TMPG (= MAP-ICP) and lead to rupture
  - Emergency ruptured aneurysm
    - Consider conflict of aspiration and RSI vs. titrated deep induction
- **Maintenance**
  - Low concentration volatile + remifentanil infusion or propofol-remifentanil TIVA
    - Avoid N_2O (risk of VAE and tension pneumocephalus)
    - No difference among various volatile agents on outcome after focal or global ischemia
    - No greater protective effect with high concentration of volatile agent (ie: sufficient to cause EEG suppression) compared to more modest levels (eg: 1 MAC)
    - Ischemic insult better tolerated with volatile agents relative to awake state
    - Ensure deeper level of anesthesia for positioning, head pinning, and raising of bone flap
  - NMBA to prevent patient movement during case
  - Positioning
    - Most commonly supine with head rotated slightly away from operative side
  - Maintain normovolemia
  - Induced hypotension
    - Theory: ↓TMPG → ↓risk of rupture / re-bleed

        - Risk: ↓O2 delivery to brain → no longer used routinely
        - May be required intraoperatively if active arterial bleeding occurs (target MAP 40-50)
    - Induced hypertension may be required
        - During periods of temporary arterial occlusion to maintain collateral perfusion
        - To 'test' aneurysm clipping (phenylephrine for target SBP 150)
    - Ventilation
        - Hypocapnia traditionally employed for brain relaxation; risk of worsening ischemia
        - Normocapnia now recommended
            - Consider hyperventilation for brain relaxation in setting of acute ↑ICP
    - Mannitol (0.5-1 g/kg) just prior to dural opening
        - Shrink brain → facilitate surgical exposure, ↓need for high retractor pressures
        - ↓Interstitial pressure around capillaries and improve rheology → ↑CBF
        - Abrupt ↓ICP may ↑TMPG → theoretical risk of aneurysm rupture; avoid rapid bolus prior to opening dura
    - Hypothermia
        - No improvement in neuro outcomes in prospective trial of *aneurysm surgery*
        - Moderate hypothermia delays emergence due to time required for sufficient rewarming
            - Awakening from anesthesia during hypothermia can result in extreme HTN
    - Brain protective strategies
        - Routine
            - Maintain MAP to ensure collateral flow and perfusion under retractors
            - Limit duration of temporary occlusion
            - Maintain euglycemia
        - Specifically applied when temporary vessel occlusion employed
            - Mild hypothermia (32-34 °C), barbiturates
    - Trapping (temporary occlusion of vessels on either side of aneurysm) to complete dissection of aneurysm neck and apply clip (usually with large aneurysm)
        - Consider EEG monitoring during clip application
            - If EEG disturbance reposition clip or administer barbiturates
        - Maintain MAP at high-normal levels to facilitate collateral CBF
        - Consider mild hypothermia
        - Occlusion times
            - < 14 min invariably tolerated
            - >20min ↑↑risk of ischemic injury
            - > 31 min ischemic injury rate of 100%
    - Lumbar CSF drainage
        - Used electively in the past to facilitate surgical exposure; inserted preoperatively
            - Contraindicated in setting of ICH (risk of herniation)
        - Avoid excessive CSF loss; abrupt ↓ICP will ↑TMPG (→ risk rebleeding) and cause sagging of brain (→ traction on bridging vv → activate CV reflexes, SDH)
            - Keep drain closed until dura opened
        - Drain opened when dura opened
        - Close drain once aneurysm clipped → allow reaccumulation of CSF (low CSF volume causes brain sagging and postop deterioration), ↓size of potential pneumocephalus
            - Drain removed postoperatively
    - External ventricular drain
        - More commonly employed than lumbar CSF drain; similar risks
    - Intraoperative angiography
        - Allow C-arm access without interfering with airway or monitoring equipment
        - Radiologist requires access to femoral vessels to insert vascular access sheath
        - Monitor rate of heparin flush via vascular access sheath
    - TXA of uncertain benefit, appears to reduce blood loss intraoperatively, but when used longer term on delayed surgery patients, increased hydrocephalus and thrombotic complications
- **Emergence**
    - WFNS grades I-II: extubated in OR for rapid neuro exam
        - Ensure adequate analgesia and no coughing / straining / vomiting / hypercarbia / HTN
    - WFNS grades III-V: maintain ETT and mechanical ventilation

DISPOSITION AND MONITORING

- All should be observed in a high acuity unit
- Regular neuro assessments and close HD monitoring
    - Avoid HTN – 20-30% increase in BP above preop baseline increases risk of ICH and edema and must be avoided; prophylactic analgesia, anti-emetic, anti-shivering, anti-HTN drugs
    - If high risk vasospasm – 10-20% increase in BP above preop baseline may be of benefit

COMPLICATIONS

- **Vasospasm**
    - Incidence post-SAH = 30% symptomatically, 70% angiographically
        - Onset after day 4-14 post-SAH, peak in 7-10d, resolve over 10-14d
    - Major cause of M+M

        - Cerebral infarction, persistent neuro deficits in 35%, mortality rate 31% (vs 17% without vasospasm)
    - ?Mechanism – OxyHb around cerebral vessels may stimulate endothelin secretion
    - Manifestations reflect cerebral ischemia and infarction
        - Delayed ischemic neurologic deficits – altered LOC, transient focal neuro deficits
        - May cause increase in ICP
    - Diagnosis – imaging (TCD, angiography)
    - Management
        - Target high-normal ICP to maintain perfusion
        - Nimodipine
            - Useful as prophylaxis; improved neuro outcome, ↓M+M
                - Mechanism = cytoprotective effect; does not ↓incidence of vasospasm
                - Risk of hypotension; stable BP takes priority over nimodipine
            - Usually ineffective as Tx of established vasospasm
        - Statins
            - Prophylaxis may ↓severe vasospasm, improve outcome
        - Triple H therapy (hypervolemia, hemodilution, and hypertension)
            - Used for Tx of vasospasm; not indicated for prophylaxis
                - May be contraindicated in unclipped aneurysms
            - Hypovolemia 2° to blood loss and natriuretic peptide secretion from dysfunctional hypothalamus
                - Tx: volume expansion w/ colloid vs crystalloid to target CVP 8-12 mmHg
            - Loss of autoregulation 2° to vasospasm → CBF becomes pressure-dependent
                - Tx: induced HTN, ↑SBP 20-30 mmHg above baseline, or 120-150 mmHg for unclipped and 160-200 mmHg for clipped aneurysms (phenylephrine and dopamine most commonly used)
            - Hemodilution
                - A consequence of hypervolemic Tx
                - Optimizes rheologic properties → improve microcirculatory CBF
            - Risks of 3H Tx
                - ↑Cerebral edema, ↑ICP, ↑hemorrhagic complications (eg: rebleeding of unclipped aneurysm), myocardial ischemia, pulmonary edema
        - Papaverine intracisternal after clipping aneurysm
            - Risks: mydriasis, facial nerve palsy, bradycardia, hypotension, MH mimic
        - $MgSO_4$ and TK-044 (ET antagonist) may improve outcome
        - Balloon angioplasty
            - Indicated for new neuro deficits unresponsive to medical Tx
                - May reverse or improve vasospasm-induced neuro deficits
            - More favorable outcome if performed w/in 6-12hrs of onset of ischemic Sx
            - Risks – intimal dissection, vessel rupture, thrombosis → ischemia/infarction
    - ICP monitoring often required if cerebral edema develops
- **Rebleeding**
    - Incidence post-SAH =10-30% within 2 weeks
        - Majority of rebleeds occur within first 6-12 hours after initial SAH
    - High mortality rate (up to 80%)
        - Early securing of aneurysm favored (w/in 72h)
    - Rebleed at induction frequently fatal
        - Poorly organized clot over aneurysm early after SAH easily disrupted
- **Acute hydrocephalus (25%)**
    - Blood in ventricular system obstructs ventricular drainage and CSF absorption
    - Requires emergency ventricular drainage → ↑risk of rebleeding and infection
    - Chronic hydrocephalus requires ventricular shunt insertion
- **Seizures (13%)**
    - More common in setting of neuro deficits → prophylactic anticonvulsants indicated
- **Intracerebral / intraventricular hemorrhage**
- **Hyponatremia (10-34%)**
    - Onset several days post-SAH
    - **Cerebral salt wasting**
        - Triad of hyponatremia, volume contraction, high urine Na (>50 mmol/L); often hypoMg
        - Depletion of Na and H2O 2° to cerebral ANP/BNP secretion
        - Tx: volume expansion and sodium administration (NS) to achieve euvolemia
    - **SIADH**
        - Triad of hyponatremia, normovolemia or hypervolemia, high urine Na (>50 mmol/L)
        - Excess free water 2° to ↑ADH secretion from posterior hypothalamus due to injury
        - Tx: free water restriction
- **Cardiovascular dysfunction**
    - Systemic and pulmonary HTN
    - Myocardial stunning and injury
    - Cardiogenic shock
    - Arrhythmias
    - Pulmonary edema → ALI

        - Neurogenic vs cardiogenic
- **Intraoperative complications**
    - **Massive hemorrhage /aneurysm rupture**
        - May require induced hypotension for surgical control – risk of ischemia
        - Temporary occlusion of arteries proximal and distal to aneurysm preferred
    - **Cerebral ischemia**
        - Secondary to acute hypoperfusion in retracted areas
    - **Venous air embolism / paradoxical air embolism**
        - ↑Risk with surgical site in head-up or sitting position
    - **HTN**
        - During emergence or postop
        - Pre-treat w/ hydralazine +/- labetalol, ensure adequate analgesia
    - **Delayed emergence**
        - Urgent CT → transfer to radiology with ETT in situ
        - Differential diagnosis:
            - Residual anesthetic, hematoma, edema, obstructive hydrocephalus
- **Coiling:**
    - Perforation of aneurysm → massive hemorrhage
        - Tx: heparin reversal and routine management of hemorrhage
    - Misplaced coil → cerebral ischemia
        - Tx: induce HTN to aid in retrieval and increase collateral flow
    - Vascular dissection
- **Other complications**
    - Pneumonia, sepsis, GI bleed, DVT/PE

OBSTETRICS

- **Epidemiology**
    - SAH in pregnancy – 77% aneurysms (33% AVMs)
    - Rupture most likely in 3rd trimester up to 6wks postpartum
- **Management of ruptured aneurysm**
    - Surgical clipping vs endovascular coiling recommended due to risk of rebleeding
    - Considerations for nonobstetric surgery in pregnancy
        - Anatomic/physiologic changes of pregnancy – airway/breathing/circulation/etc
            - RSI, L uterine displacement
        - 2 patients
            - Intraop FHR monitoring
    - Management for pregnant women undergoing aneurysm clipping
        - Premedication: anxiolytic to reduce stress response
        - Arterial line pre-induction
        - Maintain stable hemodynamics to preserve cerebral and uteroplacental perfusion
            - Prophylactic anti-hypertensive agent, adequate depth of anesthesia
        - Induced hypotension may compromise both cerebral and uterine blood flow
            - Prolonged high dose nitroprusside causes fetal cyanide toxicity
        - Avoid aggressive hyperventilation (will reduce uterine blood flow)
            - Target PaCO2 28-30 (lower end of normal range for pregnancy)
        - Avoid excessive mannitol – slowly accumulates in fetus → fetal hyperosmolality → ↓fetal lung fluid and urine producetion, fetal hypoNa
        -
- **Management of unruptured aneurysm**
    - No definite advantage of c-section over vaginal delivery
        - Decision based on individual patient
    - Minimize hemodynamic stress during labor and delivery, avoid HTN
        - Epidural to attenuate BP fluctuations, forceps to shorten 2nd stage
        - Neuraxial technique preferred for c-section

PATHOPHYSIOLOGY

- **Epidemiology**
    - ~10-30% have > 1 aneurysm
    - Rupture of aneurysm is most common cause of SAH
        - Peak incidence of rupture 5th – 6th decades of life, F >M
    - Rupture rate of aneurysms
        - 0.05% per year for small aneurysms (< 10 mm diameter) and no prior SAH
        - 0.5% per year for large (>10 mm diameter) aneurysms and for all aneurysms with previous SAH
        - Surgery does not reduce rate of disability and death from unruptured aneurysms < 10 mm in diameter and no prior SAH
    - **Outcomes** – 10% mortality rate acutely; of those who survive 25% die within 3 months, and up to 50% of survivors are left with significant neurological deficits
- **Physiology**
    - Aneurysms develop mostly at vascular bifurcations (turbulent flow)
        - Majority (80-90%) located in anterior (carotid) circulation (ACmA, PCmA, MCA)

        - 10-20% located in posterior (vertebrobasilar) circulation
    - Law of LaPlace: Tension = transmural P x radius
- **Presentation**
    - **Unruptured aneurysms**: often present with prodromal symptoms and signs suggesting enlargement (mass Sx – HA, CN III palsy)
    - **Ruptured aneurysms**: present acutely as SAH, the 'worst HA of my life,' photophobia, altered LOC, N/V; usually do *not* have focal neurological deficits
        - ↑ICP 2° to mass effect of hemorrhage, cerebral edema, hydrocephalus
        - ↓CBF and CMR
        - Impaired autoregulation with R-shift of cerebral autoregulation curve → require ↑er MAP to maintain CPP
        - Cerebrovascular CO2 reactivity usually preserved
    - After SAH, injury to posterior hypothalamus may stimulate release of NEpi from adrenal medulla and sympathetic cardiac efferent nerves can result in:
        - Myocardial stunning / injury
            - Reversible ischemic changes in subendocardium 2° to extreme ↑s in myocardial wall tension; usually resolve spontaneously
            - Severity of myocardial dysfunction correlates with severity of neurologic injury but not with severity of EKG abnormalities
            - Modest elevation in cardiac troponin common; peak levels correlate w/ severity of neuro injury and myocardial dysfxn on echo
            - Distribution of hypokinesis on echo may be inconsistent with anatomic vascular territory
            - Unclear whether EKG/echo/biochemical signs of myocardial injury associated with SAH require same Tx and carry same prognosis as those associated with primary myocardial injury
            - Triple H therapy for cerebral vasospasm may worsen myocardial injury
        - Neurogenic pulmonary edema
        - Arrhythmias
            - Classic "canyon T waves," nonspecific T-wave changes, QT prolongation, ST-segment depression, and U waves have been described
            - ECG changes do not appear to have important implications with respect to myocardial function, although may indicate risk of dysrhythmias
            - **Prolonged QT interval** (>550 ms) common after SAH, especially if severe → risk of malignant ventricular arrhythmias, including **Torsades de pointes**
        - Cardiogenic shock

- **Diagnosis**
    - Clinical suspicion
    - Non-contrast CT head
        - High-density (white) blood clot in basal subarachnoid cisterns in 95% of patients
        - dDx SAH
            - Intracranial aneurysm rupture, trauma, vertebral and carotid artery dissection, dural and spinal AVMs, mycotic aneurysms, sickle cell disease, cocaine abuse, coagulation disorders, pituitary apoplexy
    - Angiography to define vascular anatomy

- **Classification:**
    - **Hunt-Hess Classification:**
        - Grade I = Minimal H/A, mild nuchal rigidity
        - Grade II = Moderate H/A, nuchal rigidity, +/- CN palsy
        - Grade III = Drowsiness, confusion, mild focal deficit
        - Grade IV = Stupor, hemiparesis, early decerebrate rigidity
        - Grade V = Deep coma, decerebrate rigidity, moribund
    - **WFNS Classification:**
        - Grade I = GCS 15, no motor deficit
        - Grade II = GCS 13-14, no motor deficit
        - Grade III = GCS 13-14 + motor deficit
        - Grade IV = GCS 7-12 +/- motor deficit
        - Grade V = GCS 3-6 +/- motor deficit
    - Higher grades are associated with:
        - More neurologic dysfunction, ↑er ICP, impaired cerebral autoregulation and vascular CO2 reactivity
        - More complications – cerebral vasospasm, cardiac arrhythmias and dysfunction, hypovolemia, hypoNa
        - Less favorable outcomes – increased mortality, poor prognosis

- **Risk factors for rupture:**
    - Size >10 mm
    - Previous SAH
    - HTN
    - Hypercholesterolemia (>6.3 mmol/L)
    - Genetic conditions (eg. ADPKD)
    - Pregnancy
    - OCP use
    - Smoking
    - EtOH

- Cocaine / amphetamine abuse
- Familial (first degree relatives)
- Vertebrobasilar aneurysm

- **Management – Unruptured aneurysm**
  - Interventions to reduce risk of initial SAH
    - Craniotomy and surgical clipping
    - Endovascular coiling
- **Management – Ruptured aneurysm**
  - Early intervention ideal (within 24-72h)
    - Previously only applied to HH grades I-III; has since been extended to grade IV-V
    - If early intervention not feasible, surgery usually delayed for at least 10-14d to be safely beyond period of maximal vasospasm risk
      - Surgery often deferred in setting of cerebral vasospasm
  - Benefits of early intervention
    - ↓Re-bleeding and vasospasm
    - ↓Medical complications associated with prolonged bed rest
  - Disadvantages of early intervention
    - Surgically more challenging for several reasons
      - More brain edema
      - Mild hydrocephalus common
      - Clot not yet organized → ↑risk intraoperative rupture
  - Induced hypotension contraindicated in setting of emergency aneurysm surgery
  - Techniques
    - Coiling
      - Similar considerations as for craniotomy, including hemodynamic control, avoidance of pt movement
      - Out of OR (neuroradiology suite)
      - Avoid hyperventilation (↓CBF → more difficult to access aneurysm via endovascular approach)
      - Systemic heparinization to ↓thromboembolic risk from intraarterial catheter
        - Protamine must be available
      - Prompt transfer to OR for neurosurgical intervention must be possible
    - Craniotomy with surgical application of clips

REFERENCES

- Miller 7th ed. p.2062-67.
- Barash 6th ed. p.1020-21, 1449-50.
- Chestnut 4th ed. p.1070-71.
- Bendo, A. Intracranial vascular surgery. Anesth Clin N Am. 2002; 20: 377-88.
- Priebe HJ. Subarachnoid haemorrhage and the anaesthetist. BJA. 2007; 1: 102-18.