Arterio-Venous Malformations

A cerebral AVM is an abnormal vascular connection between the arterial and venous circulation consisting of a complex tangle of abnormal thin-walled vessels; the resulting high flow / low resistance shunt can result in mass effect, circulatory disturbances, seizures, and cerebral metabolic disturbances.

ANESTHETIC CONSIDERATIONS:

- Unruptured
  - Tight BP control: Minimize perioperative hemodynamic alterations to prevent inadvertent rupture and maintain stable transmural gradient
  - Potential for perioperative rupture: blood available
  - Mass effect: increased ICP, seizures, neurological deficit
  - Loss of CBF autoregulation → Maintain CPP
  - Pharmacologic interactions (i.e. phenytoin)
  - Use of short acting anesthetic agents to facilitate postoperative neurological evaluation
- Ruptured
  - Emergency surgery with no time for preoperative optimization
  - Full stomach precautions and RSI
  - Loss of cerebral autoregulation → Maintain adequate CPP
  - Potential for hemodynamic instability
  - Increased ICP: minimize iatrogenic increases in ICP
    - If herniation imminent: hyperventilate for ETCO2 30, HOB elevated, head in neutral, mannitol/3% saline, lasix, CSF drainage
  - Cerebral protection: normocapnia, normotension, normoglycemia, volatiles vs barbiturates, minimize shivering
  - Postoperative ICU
- Procedural
  - Remote airway
  - Remote site (if neurovascular coiling)
  - Tight BP control requiring invasive BP monitoring
  - Facilitate surgical field
  - Increased risk of VAE in prone/sitting position

ANESTHETIC GOALS:

- Tight hemodynamic control within 10% of preoperative value
  - Maintain CPP 60-70
  - Avoid increased ICP
- Minimize secondary neurological injury
- Provide a quiet, relaxed (brain relaxation) surgical field
- Facilitate rapid postoperative neurological assessment

HISTORY

- Standard anesthetic history
- Evidence of raised ICP:
  - HA, N/V, visual changes, altered LOC, fatigue and drowsiness, new onset seizures, focal neuro deficits
    - Neurological deficits as a result of ischemia (AVM-induced steal phenomenon), raised ICP (venous engorgement, mass effects of AVM, hemorrhage), side effects or complications of pre-op embolization
- Presenting symptoms and previous treatment
- Spetzler-Maring Grading Scale

PHYSICAL

- Standard anesthetic Px
- **CNS**
  - GCS, pupils (reactivity, mydriasis), fundoscopy for papilledema, focal neuro deficits
- **CVS**
  - HTN (reflex in attempt to maintain CPP in presence of raised ICP → reflex ↓HR)
  - High output heart failure (displaced apex, hyperdynamic precordium, S3)
- **RESP**
  - Irregular respiratory pattern, respiratory insufficiency

INVESTIGATIONS

- Labs
  - CBC, x-match (massive bleeding potential)
  - Lytes (SIADH/CSW), renal function
  - Glucose (steroid use and concern re: hyperglycemia and secondary brain injury)
- Imaging
  - MRI
  - Angiography gold standard for Dx and evaluation of anatomy

OPTIMIZATION

- Usually elective → optimize co-morbidities
- Endovascular embolization (or radiotherapy) can reduce bleeding risk and shorten resection time
- Minimal preoperative sedation with benzodiazepines (may impair postop neuro exam)
- Continue antibiotics, steroids, and antiepileptic meds

ANESTHETIC OPTIONS

- Depends on treatment modality

- Endovascular embolization
    - GA advantages:
        - Improves visualization for interventionalist
        - Allows for temporary apnea for angiography and embolization
        - Facilitates profound temporary hypotension required during injection of embolizing agent
    - MAC / TIVA advantages:
        - Allows continuous neuro assessment
    - Disadvantages: out of OR
- Microsurgical excision
    - GA required

ANESTHETIC SETUP

- **Drugs**
    - Emergency drugs (vasopressors, vasodilators)
- **Equipment**
    - Standard CAS monitors, OR setup
    - Large bore IV's with blood set
    - Arterial line preinduction
    - +/- CVL post-induction for CVP monitoring, VAE treatment and drug infusions
    - Nerve stimulator on limb NOT affected by neurological deficits
    - Foley
    - Doppler for VAE monitoring
- **Other**
    - 4U PRBCs in OR fridge
    - Blood bank alerted for possibility of massive transfusion

MANAGEMENT OF ANESTHESIA

- **Induction**
    - Any induction agent which produces rapid, reliable onset of unconsciousness with minimal effects on CBF is acceptable
    - Ensure adequate depth of anesthesia prior to stimulation with laryngoscope (avoid acute HTN)
    - Elevated ICP *rarely* an issue in AVM resection
- **Maintenance**
    - TIVA vs. volatile agent + remifentanil infusion to ↓$CMRO_2$ and prevent stimulation (associated ↑CBF and ICP)
        - Avoid N_2O (risk of VAE and tension pneumocephalus)
        - Ketamine controversial (traditionally thought to ↑ICP)
    - Ventilation
        - Titrate to $PaCO_2$ = 32-25 mmHg, no PEEP
    - Brain relaxation and protection:
        - Positioning to ensure adequate venous drainage from head
        - NMB to prevent spontaneous movement with associated ↑ICP, bleeding, and herniation
        - Replace fluid deficits with colloid or slightly hypertonic solutions
            - Avoid hypotonic solutions (cerebral edema)
        - Caution with peripheral vasodilators (SNP / NTG may ↑CBF and ICP despite decreasing systemic BP)
        - Consider seizure prophylaxis (phenytoin)
        - Maintain euglycemia
    - Induced hypotension may be required to facilitate hemostasis in setting of small, deep feeding vessels
- **Emergence**
    - Rapid emergence desired to facilitate post-op neurological assessment
        - Avoid coughing, straining, bucking, vomiting, hypercarbia (target ETCO2 < 50), HTN
            - Prophylactic lidocaine or remifentanil, antiemetics
        - Avoid excess sedatives/analgesics
    - Aggressive BP management to prevent emergence HTN (→ cerebral edema and hemorrhage)
        - Prophylactic hydralazine / labetalol
    - Adequate analgesia

DISPOSITION

- ICU for postop monitoring of neurologic and hemodynamic status

COMPLICATIONS

- Microsurgical resection:
    - Massive bleed with raised ICP necessitating induced hypotension for surgical control
    - Acute hypotension in retracted areas → ischemia
    - VAE / paradoxical AE
    - Emergence or post-op HTN
    - Delayed awakening – consider urgent CT head with ETT in situ
        - dDx:
        - Anesthetic drugs
        - Structural – hematoma, edema, obstructive hydrocephalus
    - Intraoperative normal perfusion pressure breakthrough (NPPB) or cerebral dysautoregulation:
        - Can result in massive intraoperative brain swelling

        - Diagnosis of exclusion (must R/O more common causes)
        - Management:
            - Tight BP control – maintain CPP with lowest possible CBF, a-adrenergic blockers
- Endovascular embolization:
    - AVM perforation with massive bleed → reverse heparin, other routine hemorrhage treatment
    - Inadvertent vascular occlusion → induced HTN +/- thrombolytics

OBSTETRIC

- See 'Anesthesia considerations for intracranial aneurysms' document
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PATHOPHYSIOLOGY

- Complex tangle of thin-walled abnormal vessels (the "nidus") without an intervening capillary bed:
    - Acts as a high flow / low resistance AV shunt for blood flow
- Epidemiology:
    - 4% population (autopsy data)
    - 12% symptomatic
    - 10% also have intranidal aneurysms
- Presentation:
    - Spontaneous ICH / IVH (50%) – vs SAH with ruptured aneurysm
    - New onset seizure (25%)
    - HA (15%)
    - Focal neurologic deficit or pulsatile tinnitus (< 5%)
    - < 80% present before 40 y.o.
        - If no Sx by age 50, will likely remain stable and asymptomatic
- Spetzler-Martin AVM Grading Scale:
    - Size:
        - 0-3 cm = 1; 3.1-6.0 cm = 2; > 6.0 cm = 3
    - Location:
        - Non-eloquent = 0; eloquent = 1
    - Deep venous drainage:
        - Not present = 0; present = 1
    - Sum Total = Grade:
        - Grades 1-2 = really good outcome
        - Grade 3 = moderate outcome
        - Grades 4-5 = poor outcome (30% and 50% morbidity, respectively)
- Pathophysiologic effects on the brain:
    - **Mass effect**
        - Expanding nidus or intranidal aneurysm, edema, hematoma
    - **Metabolic depression** (diaschisis)
    - **Seizure activity**
    - **Cerebral ischemia**
        - High flow / low resistance system allows large amount of bulk blood to pass through AVM resulting in capillary hypoperfusion → cerebral hypotension along path of shunt
        - Historically, "cerebral steal" thought to cause neuro deficits; hypotension in normal brain adjacent to AVM → maximal vasodilation and therefore ↓CBF → AVM "steals" blood from normal surrounding brain
        - Evidence now suggests neuro deficits actually result from local mass effects
    - **Diffuse bleeding and edema** intra/post-operatively
        - Traditional theory – normal perfusion pressure breakthrough (NPPB) aka cerebral dysautoregulation
            - Despite normal CBF, normal vessels surrounding AVM are perfused at lower than normal pressures → vasodilate to preserve perfusion and prevent ischemia → loss of autoregulation
            - Following AVM resection adjacent vessels exposed to higher pressures than accustomed to and are unable to autoregulate within normal BP range → regional hyperemia despite normal systemic BP → vasogenic edema and hemorrhage
        - Current theory – occlusive hyperemia
            - Hemorrhage and edema caused by arterial stagnation and obstruction or venous outflow obstruction that result directly from AVM resection
- Treatment options:
    - Endovascular embolization
        - Often pre-op adjuvant to surgery (↓surgical time and bleeding during excision)
        - Usually done in several stages to ↓risk of NPPB (NO evidence this is true)
    - Microsurgical excision
    - Radiation

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