

Assessment Points				
System	Effect	Assessment by Hx	PE	Test
RESP	Tachypnea	Tachypnea that persists during sleep and is unpleasant to the conscious patient	Resp rate Normal inspiratory and expiratory excursion	ABG (all must be present to diagnose): PCO ₂ (low) pH (alkalotic) PaO ₂ (increased for age) Decreased bicarbonate Alveolar-to-arterial gradient not larger than normal
CNS		Pt cannot volitionally inhibit hyperventilation	Focal or nonfocal CNS findings	CSF pH may be normal CT/MRI

Key Reference: Tarulli AW, Lim C, Bui JD, et al.: Central neurogenic hyperventilation. A case report and discussion of pathophysiology, *Arch Neurol* 62:1632–1634, 2005; Kramer CL, Wijidicks EF: Central neurogenic hyperventilation, *Neurology* 83:376, 2014.

Differential Diagnosis for Hyperventilation

- Metabolic acidosis
- Bowel ischemia with acidosis
- Pulm pathology with hypoxemia (pneumonia, pulm embolus, pulm edema, and restrictive or obstructive lung disease)
- Drug toxicity (salicylates, theophylline, cyanide, and topiramate)
- Sepsis
- Encephalopathy/CNS lesions (glioblastoma, encephalitis, MS, brainstem lymphoma, brainstem glioma, brainstem infarction, and liver dysfunction)
- Anxiety
- Psychogenic
- Cardiac (CHF and valvular disease)
- High altitudes
- Hyperthyroidism

- Pregnancy
- Must exclude other etiologies for respiratory alkalosis with appropriate lab/Dx testing

Adverse Effects

- Respiratory alkalosis shifts oxyhemoglobin curve to the left.
- Hypocapnia is a potent cerebral vasoconstrictor, subsequently decreasing cerebral blood flow and volume, which may result in ischemic insults.
- Effect of severe hypocapnia in normal brains is less clear and may produce ischemia when combined with Bohr effect. (Hemoglobin's oxygen affinity is inversely related to the concentration of CO₂.)

Treatment

- No completely effective or consistent treatment currently known

- Narcotics may attenuate resp rate and improve blood gases but will not correct rate or alkalosis.
- Mechanical ventilation with neuromuscular blockade and sedation during treatment of tumor has been attempted.
- Treatment of tumor with steroids, chemotherapy, or radiation therapy is sometimes effective.
- Increasing dead space ventilation and administration of supplemental oxygen and benzodiazepines are not effective.

Outcome

- Death from progressive neurologic deterioration or other complications (aspiration, pneumonia) is likely.
- Pt may have improvement with treatment of tumor or long-term narcotics.

Cephalopelvic Disproportion

John Kissko III

Risk

- Occurs in 1–3% of the pregnant population

Perioperative Risks

- Increased maternal and fetal morbidity and mortality
- Protracted labor
- Arrested labor
- Uterine rupture
- Increased rate of cesarean section
- Increased rate of forceps- or vacuum-assisted delivery

Worry About

- Increased need for surgical delivery
- Increased incidence of fetal distress and need for emergency intervention

Overview

- Subset of fetopelvic disproportion.
- Methods of Dx include various clinical and radiographic estimations of fetal head size and pelvic capacity, none of which alone is an accurate predictor of increased risk of failed labor.
- Leads to abnormal labor pattern with increased likelihood of operative delivery.
- Operative delivery associated with higher incidence of morbidity and mortality in mother and fetus.
- Anesthesia necessities: Complete preop evaluation for possible emergency C-section, including airway exam and neuraxial anesthesia landmarks.

Etiology

- Maternal causes include contracted pelvis (inlet, midpelvis, or outlet), Hx of scoliosis, previous pelvic trauma (especially fracture with callous formation), and Hx of poliomyelitis.
- Fetal causes include macrosomia (often secondary to gestational diabetes), abnormal presentation, and hydrocephalus.

Usual Treatment

- Obstetric: Proper evaluation and planning before and during labor
- Anesthesia: Neuraxial anesthesia for pain relief during labor or operative delivery

Assessment Points

System	Effect	Assessment by Hx	PE	Test
GYN	CPD	Failure to progress adequately in labor	Pelvic exam	Radiographic cephalopelvimetry

Key References: Cunningham F, Leveno KJ, Bloom SL, et al: Abnormal labor. In Cunningham F, Leveno KJ, Bloom SL eds: *Williams obstetrics*, ed 24, New York, NY, 2013, McGraw-Hill; Hillyard SG, Bate TE, Corcoran TB, et al: Extending epidural analgesia for emergency Caesarean section: a meta-analysis. *Br J Anaesth*. 107(5):668–678, 2011.

Perioperative Implications

- Labor usually more prolonged and painful in pts with CPD
- Epidural or CSE usually adequate to cover pain without significantly prolonging the course of labor
- Increased need for surgical delivery or C-section, many times in emergent fashion

Anesthetic Technique

- Labor epidural (PCEA): A low concentration of bupivacaine 0.125% or ropivacaine 0.10% is supplemented with an opioid (e.g., fentanyl) to help reduce motor block. Continuous infusion at 8–10 mL per h with pt-controlled boluses of 6–8 mL every 10 min can be used as needed. Programmed intermittent bolus with additional patient-controlled boluses is also an option.

- Labor CSE: In early first-stage labor (<4 cm cervical dilation), consider injection of 15 mcg fentanyl combined with 1 mL sterile saline followed by continuous epidural as with a PCEA. In late first-stage labor (>4 cm cervical dilation), intrathecal injection of local anesthetic (e.g., 1 mL of 0.25% bupivacaine) and opioid (e.g., 15 mcg fentanyl) is sometimes sufficient for the remainder of the first and second stages of labor, although a PCEA is usually started.

- Nonemergent C-section: For all C-sections regardless of level of immediacy, standard OR and anesthesia machine check, left uterine displacement to maximize uterine blood flow, and application of ASA standard monitors. For *elective C-section*, usually spinal anesthesia is used to achieve a T4 level to pain sensation. This can be accomplished by 1.8–2 mL of 0.75% bupivacaine injected at L3-L4 interspace, often combined with opioid (fentanyl and/or preservative-free morphine) and epinephrine. Maintain BP within normal limits using adequate IV hydration, and phenylephrine or ephedrine when necessary.
- Emergency C-section: Following labor *without* fetal distress (e.g., failure to progress) or labor *with* fetal distress (e.g., Category III fetal heart tracing), if pt has a reliable epidural block, epidural anesthesia is extended, using a higher-concentration local anesthetic (e.g., 3% chloroprocaine, 2% lidocaine, or 0.5% ropivacaine) if time permits. If pt does not have a functioning epidural catheter, then a spinal technique may be used (again, if time allows). Otherwise, general anesthesia with rapid sequence induction may be required.

Anticipated Problems/Concerns

- If there is no epidural catheter in place and a difficult airway is suspected, then consider spinal anesthesia or performing an awake intubation for general anesthesia. Emergency airway equipment (e.g., LMA, video laryngoscope, fiberoptic scope, jet ventilator) should be readily available.
- Neuraxial anesthesia should only be attempted while the fetus is not in imminent danger. The clinical situation must be discussed fully, effectively, and efficiently with the managing obstetricians to develop the safest plan for both the pt and fetus.

Cerebral Arteriovenous Malformations

L. Jane Easdown

Risk

- Cerebral AVMs are rare: 1–2% cause of CVA in a younger population; mean age of diagnosis is 31 y.
- 55% of pts are men.
- Symptomatic cases: 1:100,000 per y.
- Found in 4.3% of population at autopsy.
- Of those affected, 45–70% present with hemorrhage, 30% with seizures, 12% with persistent neurologic deficits, and 1% with headaches.
- Yearly risk of hemorrhage is 1–3%.

Perioperative Risks

- Risk of hemorrhage at embolization is 2–4%.
- Intraop blood pressure management is critical.
- Postop NPPB occurs as blood is diverted from the AVM to the surrounding brain, presenting risk of cerebral edema or hemorrhage.

Worry About

- Massive intraventricular or intraparenchymal hemorrhage

- Seizure
- New neurologic deficits
- Cerebral edema, hyperemia post resection, or endovascular embolization

Overview

- Localized arteriovenous shunt comprised of a tangle or “nidus” of abnormally walled vessels, which cause symptoms by rupture, ischemia, and diversion of flow or pressure on adjacent structures; many are detected on routine scans. The majority of AVMs will bleed at least once.
- 0% are supratentorial, and 4–10% are associated with aneurysms.
- Increased risk of rebleed of 6–33% within the first year.
- As the result of hemorrhage, 16% of pts are moderately or severely disabled. Hemorrhage leads to mortality in 10–30% of cases.
- Vein of Galen malformations are rare congenital lesions with connections between the intracerebral vessels and the great vein of Galen. This disorder in

neonates or infants may result in high output CHF or increased ICP from hydrocephalus.

Etiology

- Although congenital in origin, no specific genetic defect has been determined. Sometimes AVMs are associated with hereditary hemorrhagic telangiectasia.

Usual Treatment

- Evidence-based neurosurgic management is based on the Spetzler-Martin grading scale. AVMs are graded 1–5, based on their size, location (eloquent or non-eloquent brain), and pattern of venous drainage.
- Smaller and more superficial AVMs (grades 1–3) might be surgically resected for cure. Higher-grade AVMs may be treated with endovascular embolization or stereotactic radiotherapy. Multimodal therapy is common, especially embolization before surgical resection. Embolic materials include solid or liquid agents. Conservative management may improve outcomes in high-grade or unruptured AVMs.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Airway protection	Aspiration	Active gag reflex	
CV	CHF in children with vein of Galen AVM		S ₃ and CHF	CXR, ECHO, ECG
CNS	Seizures, focal deficits, CVA, and raised ICP	Headaches, seizures, changes in mentation, and focal deficits	Neurologic exam	MRI, MRA, CT, cerebral angiography

Key References: Miller C, Mirski M: Anesthesia considerations and intraoperative monitoring during surgery for arteriovenous malformations and dural arteriovenous fistulas. *Neurosurg Clin N Am* 23(1):153–164, 2012; Bendok BR, El Tecle NE, El Ahmadieh, TY, et al: Advances and innovations in brain arteriovenous malformation surgery. *Neurosurgery* 74(Suppl 1):S60–S73, 2014.

Perioperative Considerations

Preoperative Preparation

- Endovascular embolization procedures usually require GA to ensure BP control and no pt movement during microcatheter placement.
- Craniotomy for resection requires preop preparation similar to aneurysm clipping.
- Neurologic exam with attention to focal deficits and raised ICP.
- Prior embolization/radiotherapy may have been performed.
- Carefully assess size and location of AVM.

Monitoring

- Invasive BP monitoring
- Central venous access for craniotomy procedures anticipating extensive blood loss.
- O₂ saturation monitor, which might be placed on the foot on the side of the femoral introducer to monitor arterial integrity during angiography
- Precordial Doppler for detection of air

- Jugular bulb venous O₂ monitoring (has been described)
- Intraop neuro monitoring, which may include EEG and SSEPS

Airway

- ETT for craniotomy and LMA or intubation for airway management for embolization

Induction

- Careful management of BP to prevent Htn (increased ICP or hemorrhage) or hypotension (ischemia)

Maintenance

- Manage BP and ICP carefully, especially with intubation, pinning, and incision.
- Surgeons may request burst suppression with propofol for brain protection if temporary clips are used during resection.
- Surgeons may request hypotension or hypocapnia.
- Control blood glucose.
- Maintain strict isotonic/hypertonic fluid.
- Perform angiography before emergence.
- Plan for arousal and neurologic testing immediately after operation.

Extubation

- Careful BP control: Labetalol and additional opioid
- Expected request for neurologic exam
- May keep intubated to control NPPB hyperemia

Adjuvants

- Cell saver and blood products immediately available
- BP control with nicardipine, NTG, NTP, and beta-blockers
- Phenylephrine infusion
- Propofol infusion for TIVA or burst suppression
- Steroids and mannitol
- Antiepilepsy medications

Postoperative Period

- Complete obliteration of a large AVM will lead to redistribution of CBF and hyperemia or NPPB. Until autoregulation returns, pt may require lower BP and control of CO₂ by intubation and ventilation.
- Postop ICU neurologic monitoring will be required.
- After arteriogram, the femoral cath site should be monitored for bleeding.