Perioperative Implications

Preoperative Preparation

- Control BP with pharmacologic agents, and institute anticonvulsant therapy for seizure prophylaxis as needed.
- Examine the airway and evaluate coagulation status.
- Careful intravascular repletion could minimize the severe hypotension seen with regional or general anesthesia. If in doubt, consider TTE to quantify volume status.

Monitoring

- Basic monitoring should include measuring BP by noninvasive method, ECG, pulse rate, pulse oximetry, and UO measurements.
- Invasive BP monitoring with the use of an intra-arterial cath may be necessary in unstable pts with severe PEC or EC; pts needing frequent ABG measurements; pts requiring potent pharmacologic treatment for malignantly increasing BPs; or pts with significant body habitus (obesity) without the availability of an appropriate BP cuff size.
- Repetitive physical exam (e.g., DTRs) and measurement of Mg^{2+} blood levels for pts on magnesium sulfate for antiseizure prophylaxis.

Induction

- Early neuraxial analgesia (epidural) is often the preferred anesthetic technique for delivery (unless contraindicated or unstable); it may relieve vasospasm, decrease circulating catecholamines, improve uteroplacental perfusion, and decrease airway catastrophe risk.
- With the need for general anesthesia in the setting of severe gestational Htn, PEC, and/or EC:
  - Pharmacologic agents should be used to minimize the sympathetic response from direct laryngoscopy and endotracheal intubation.
  - Avoid the use of ketamine for induction due to its sympathetic effects.
  - Airway edema may complicate optimal mask ventilation and rapid-sequence intubation. Various sizes of endotracheal tubes should be available.
  - With the anticipation of a difficult intubation, consider the use of airway adjuncts (e.g., videolaryngoscopy) or an awake/anesthetized fiberoptic endotracheal tube placement. A difficult airway cart should always be in close proximity.

Maintenance

- Use of magnesium sulfate in the management of PEC-EC can contribute to the loss of uterine muscle tone, hence increasing the risk of uterine atony and postpartum hemorrhage. Consider the use of additional uterotonics after delivery. Avoid the use of methylergonovine maleate if possible.
- Reduce the dosage of muscular relaxants when used in conjunction with magnesium sulfate due to the potentiation of neuromuscular relaxant effects. Continuous monitoring with a peripheral nerve stimulator should be utilized.

Extubation

- If evidence of thrombocytopenia prior to placement of epidural, consider rechecking platelet count prior to removal of cath.
- Ensure that pt fulfills all "extubation criteria" prior to extubation.

Postoperative Period

- Pts with PIH are still at risk for progression to PEC and EC after delivery. Seizure prophylaxis with magnesium sulfate may be continued after delivery (48 h postpartum).
- Triage: Consider the need for ICU monitoring post delivery if concerned or if pt is unstable.

Preterm Infant

Meredith Anne Kato

Risk

- Births at less than 37 post conceptual wk (PCW) rose sharply from 1990 to 2006 in USA due to the increase in assisted reproductive technology and multiple gestation. It has been slowly declining in the ensuing years.
- In 2013, 11.4% of all live births were <37 PCW; 3.4% were <34 PCW.
- 67% of all infant deaths occur among the premature. Babies born at less than 32 wk are 88 times more likely to perish compared to full-term babies.
- Neonates have the highest periop morbidity and mortality among pediatric pts, with premature infants carrying the highest risk.

Perioperative Risks

- Laryngospasm
- Hypothermia
- Hypoglycemia
- Massive blood loss
- Rapid onset hypoxia
- Bradycardia, poor cardiac output
- Apnea

Worry About

- Occult congenital abnormalities
- Difficult intubation, vascular access
- Cardiac decompensation
- Persistent or reversion to fetal circulation and high pressures in the pulmonary vascular tree
- High airway pressures
- Oxygen toxicity
- Medication or dilution errors
- Pain control
- Transport disaster (extubation or hemodynamic compromise)

Overview

- Immature organ systems present very specific challenges to the anesthesiologist.
- Cardiac physiology is different in the premature and early neonate. The heart has fewer and disorganized contractile elements. Muscle cells contain fewer mitochondria. With low compliance, cardiac output is dependent upon heart rate. Yet, parasympathetics dominate predisposing to bradycardia. The premature heart is exquisitely sensitive to drops in serum calcium levels.
- Fetal lungs have inadequate surfactant production up until about 34–36 wk. The lungs are stiffer, harder to ventilate, and prone to atelectasis. They are vulnerable to volutrauma and barotrauma, which can lead to chronic pulmonary compromise.
- High pressure caused by an ill-fitting endotracheal tube against the trachea can lead to post extubation stridor or potentially subglottic stenosis.
- Babies have a higher oxygen demand and a lower oxygen reserve (FRC), but it is important not to over-oxygenate. Premature infants produce fewer antioxidants against the oxygen free radicals produced during oxygen therapy. Oxygen therapy is associated with retinopathy of prematurity and bronchopulmonary dysplasia.
- Hepatic metabolism of drugs is immature in the premature infant, which alters pharmacokinetics.
- Glucose homeostasis is immature. Glycogen stores are low, predisposing to hypoglycemia. These babies also frequently have dextrose or total parenteral nutrition infusions, which puts them at risk of iatrogenic hyperglycemia. These pts are also relatively insulin resistant.
- Hypothermia is common and can occur rapidly. Premature babies have immature mechanisms for heat homeostasis; they burn brown fat in "nonshivering thermogenesis."
- Premature kidneys have a lower glomerular filtration rate and a decreased ability to concentrate urine. Renal clearance of drugs is lower.
- The coagulation cascade of healthy neonates is immature but "functionally balanced." In sick babies, however, this immaturity may predispose them to coagulopathies leading to bleeding (intraventricular hemorrhage) or thrombotic events.
- Compared to an adult, the larynx is more cephalad, the epiglottis is omega shaped, the glottis lies at an angle, and the narrowest part of the airway is subglottic.
- The risk of postop apnea is high, especially with concomitant anemia (HCT <30%). It is usually mixed central and obstructive and made worse by anesthetics. The risk of apnea is greater than 1% in babies born before 35 wk who have not yet reached 54 PCW and in babies born before 32 wk who have not yet reached 56 PCW. It is important to monitor these babies postop. There is some evidence that spinal anesthesia without additional sedatives is somewhat protective against postop apnea.
- Common problems in critically ill premature infants include congenital abnormalities causing cardiac, respiratory, gastrointestinal, renal or hepatic insufficiency, intraventricular hemorrhage, necrotizing enterocolitis, hernias, or retinopathy of prematurity. These can be the reasons these babies come to the OR, or can complicate surgery done for another reason.

Etiology

- Etiology for prematurity is multifactorial and incompletely understood. Risk factors include:
  - Maternal factors including previous preterm birth, race, extremes of age, substance abuse, including smoking, multiple gestation, obesity, infection, and anemia
  - Fetal factors including congenital anomalies, intrauterine growth restriction, and male sex

Usual Treatment

- Optimization in a NICU setting, including respiratory care, nutrition, hemodynamic support, treatment of infection, and surgical correction or palliation of congenital abnormalities

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Difficult or lost airway	Previous intubation, presence of syndrome (e.g., Pierre Robin, Treacher Collins, Klippel-Feil, Goldenhar)	Dysmorphic, micrognathia	Imaging or genetic testing as needed
CV	Cardiac decompensation	Feeding difficulties, cyanotic spells, vital signs, volume status	Cyanosis, poor saturation, murmur, edema	ECHO, ECG, cardiac cath, vital signs including preductal and postductal oxygen saturation, skin turgor
RESP	Respiratory failure	Feeding difficulties, cyanotic spells, vital signs	Air hunger, use of accessory muscles or instability on vent	Pulse oximetry, ABG
RENAL	Hypovolemia or volume overload, renal insufficiency	Review of intake and output and UO	Skin turgor, fontanel	Basic metabolic labs
HEME	Postop apnea, poor tolerance of blood loss, arrest	Recent blood loss or phlebotomy, previous episodes of apnea, Hx of treatment with caffeine	Pallor, air hunger	CBC

Key References: Spaeth JP, Kurth CD: The extremely premature infant (micropremie). In Coté CJ, Anderson BJ, Lerman J, editors: *Cote and Lerman's a practice of anesthesia for infants and children,* ed 5, Philadelphia, 2013, Elsevier; Jones LJ, Craven PD, Lakkundi A, et al.: Regional (spinal, epidural, caudal) versus general anaesthesia in preterm infants undergoing inguinal herniorrhaphy in early infancy. *Cochrane Database Syst Rev* 6:CD003669, 2015.

Perioperative Implications

Preoperative Preparation

- Look for dysmorphic features, micrognathia, and obvious congenital abnormalities.
- Listen for a heart murmur and have a low threshold for obtaining an ECHO. Obtain a feeding history; breast or bottle feeding is the "stress test" of the neonate.
- Make sure pt is metabolically optimized; check basic labs, especially potassium, calcium, and glucose. Consider getting an ABG.
- Warm the room.
- Calculate estimated blood volume (approximately 95 mL/kg) and maximum allowable blood loss (EBV × [HCT initial – HCT minimum]/HCT initial).
- Calculate doses of all medications and consider unit syringes; avoid drug errors.
- Prepare emergency drugs (atropine, succinylcholine, calcium, epinephrine). Consider drips of epinephrine, dopamine, or milrinone.
- De-air all lines; check and recheck.
- For all but the simplest cases, have an active type and cross. For high-risk procedures, have blood in the room. There is generally very little lead time between unexpected surgical bleeding and the need to transfuse.
- If coming from the ICU, transport with backup emergency airway supplies, warmer, and drugs. Check and recheck endotracheal tube position and IV patency before leaving and upon arrival to the OR.

Monitoring

- In addition to standard monitors, consider preductal and postductal pulse oximetry to watch the effect of a PDA on systemic circulation.
- Consider arterial line for continuous BP and for frequent checks of glucose, electrolytes, and hematocrit. Obtain central access if the risk of needing vasoactive drips is high.

Airway

- Always have an LMA for emergencies. Have an advanced airway device if the baby has a history of a difficult intubation or looks dysmorphic.
- Position with a shoulder roll; the large head relative to the rest of the body tends to flex the neck when supine.
- Prepare multiple sizes of tubes and oral airways.
- Preoxygenate.
- Check for an air leak at <20 cm H_2O.
- Consider a nasal tube if a prolonged intubation is anticipated.
- Check and recheck tube position throughout the case. The distance between a mainstem intubation and perfect positioning can be cm or even mm.
- Watch for kinking of the tube.

Induction

- As with any pt, the acuity of the illness will dictate the anesthetic plan.
- IV and arterial line access can be very difficult in these pts.
- Inhalational induction with sevoflurane is reasonable in a relatively healthy baby undergoing a minor procedure. IV induction is more common, however.
- Consider atropine 20 mcg/kg, fentanyl 2 mcg/kg, rocuronium 0.6 mg/kg for induction.

Maintenance

- For maintenance, consider a fentanyl infusion, low-dose inhalational agent, and nondepolarizing paralytic.
- Reduce oxygen levels and maintain saturations between 88–95% to avoid oxygen toxicity.
- Avoid hypercarbia and acidosis, as both raise pressure in the pulmonary arterial tree.
- Check and recheck the tube for dislodgement or kinks throughout the case.
- Maintain normothermia. While it is easy for pts to get cold, it is also very easy to overheat them with warming devices.
- Warm fluids and blood products if given. Consider "spinning" blood in a blood salvage device (CellSaver) to warm and reduce potassium load.
- Run dextrose containing maintenance fluids or keep TPN running. Check glucose during long cases.
- Be careful not to overtransfuse or fluid overload. Use measured aliquots of fluid and blood products.
- Monitor blood loss; communicate with the surgeons.
- Beware of "monitor failure." Loss of multiple monitors may indicate serious decompensation.

Extubation

- Consider postop location when planning extubation; keep intubated for long transports to ICU.
- If extubating, reverse paralytic.

Postoperative Period

- Consider overnight monitoring for any preterm baby who has not yet reached 56 PCA. Caffeine or theophylline can help attenuate the risk of postop apnea.
- Pain management should include opioids, acetaminophen, and nonpharmacologic adjuncts, along with nonnutritive sucking, breastfeeding, and maternal contact.
- Do not use ketorolac in premature infants.

Anticipated Problems/Concerns

- Difficult IV access
- Postop apnea
- Long transport hazards
- Under-resuscitation or over-resuscitation

Protein C Deficiency

Charles Weissman

Risk

- Congenital deficiency. Homozygote is estimated at 1:500,000-750,000 live births. Occurs when gene coding for protein C on both chromosomes #2 is affected.
- Heterozygote ~0.2–0.4% of healthy population; 2–5% in pts with DVT.
- Acquired deficiency also seen.

Perioperative Risks

- Pts with protein C deficiency are at risk for venous thrombosis and pulm embolism (immobility, endothelial damage, and decreased blood flow during periop period may be triggers).

Worry About

- Increased incidence of thrombophlebitis and pulm embolism
- Thrombosis of other vessels, such as intracerebral and coronary arteries, can occur.

Overview

- Protein C is a vitamin K–dependent protein found in blood and synthesized in the liver.
- Activated after forming a complex with thrombin on endothelial cell receptor thrombomodulin; facilitated by binding to endothelial cell protein C receptors.
- Inhibits blood coagulation by proteolytic inactivation of factors V and VIII.
- Protein S is a cofactor of protein C.
- Stimulates fibrinolysis possibly by neutralizing plasminogen activator inhibitors
- Deficiency causes hyperthrombotic state
- During SIRS and sepsis, there is decreased synthesis of protein C.

Etiology

- Inherited: Autosomal dominant with variable expressivity
- Homozygotes develop life-threatening visceral vessel thrombosis or purpura fulminans (massive cutaneous necrosis) in early neonatal period.