Physiologic Anemia and the Anemia of Prematurity

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Risk

- Physiologic anemia, occurring 6–8 wk after birth, is a normal process in term infants.
- Anemia of prematurity is a pathologic anemia occurring in preterm infants. Extent of prematurity and comorbidities correlate with extent of anemia.

Perioperative Risks

- Term infants with physiologic anemia tolerate minor surgery well.
- Premature infants must be evaluated for symptoms due to anemia that may contribute to increased risk of preop events.

Worry About

- Major surgery occurring at the physiologic nadir of anemia may require blood transfusion.
- Preterm infants with anemia undergoing physiologic stress due to surgery are at risk for tachycardia, tachypnea, lactic acidosis, and periop apnea and bradycardia.

Overview

- Physiologic anemia is normal response to extrauterine life. Nadir at 9th–12th wk of life, Hgb level varies 9–11 g/dL.
- In preterm infants, nadir occurs at 4–8 wk of life and may decrease to 8 g/dL.
- Anemia of prematurity may be asymptomatic or give rise to nonspecific symptoms such as tachycardia, tachypnea, lethargy, pallor, apnea and bradycardia, poor feeding, poor growth, and lactic acidosis.

Etiology

- Transition to extrauterine life includes requirement for increased oxygen to bind to hemoglobin (HbO_2 saturation 50% in utero, 95% ex utero). Fetal hemoglobin with high oxygen affinity starts to be replaced with low-oxygen-affinity adult hemoglobin.
- Survival of neonatal erythrocytes is shorter than that of adult erythrocytes. Hemoglobin decreases until oxygen needs are greater than supply. Production of EPO is triggered and erythropoiesis increases.
- Rapid growth in infants causes a rapid increase in blood volume, resulting in hemodilution. Growth is more rapid in preterm than term infants.
- Preterm infants have more severe anemia because the less sensitive hepatic oxygen sensor triggers EPO production until 40 wk PCA. After 40 wk PCA, an extremely sensitive renal oxygen sensor takes over triggering and production of EPO.
- Iron storage occurs in the last trimester; therefore, premature infants are relatively iron deficient and have difficulty increasing iron stores by feeding.
- Extent of prematurity correlates with the amount of blood loss due to blood sampling.

Usual Treatment

- No treatment required in term infants.
- Preterm infants benefit from prevention: Reduction of blood draws, appropriate dietary supplementation, and erythropoietin therapy.
- Treatment of anemia of prematurity with blood transfusion occurs when symptoms of reduced O_2 supply are present. Symptoms include continued need for mechanical ventilation, apnea and bradycardia, tachycardia (>180 bpm for 24 h), inadequate weight gain, metabolic acidosis, or anticipation of major surgery.

Assessment Points (Apply to Preterm Infants Only)

System	Effect	Assessment by Hx	PE	Test
CV	Tachycardia	Review of VS trends	Tachycardia	± ECG
RESP	Apnea/bradycardia	Number of episodes; treatment required or spontaneous resolution		

Key References: Aher S, Malwatkar K, Kadam S: Neonatal anemia, *Semin Fetal Neonatal Med* 13(4):239–247, 2008; Bishara N, Ohls R: Current controversies in the management of the anemia of prematurity, *Semin Perinatol* 33(1):29–34, 2009.

Perioperative Implications

Preoperative Preparation

- Timing of elective blood-losing surgery depends on Hgb levels.

Monitoring

- Routine

Airway

- None

Preinduction/Induction

- Routine

Extubation

- Recent Hx of apnea and bradycardia: Consider delaying extubation to allow metabolism of anesthetic agents and sedatives.

Adjuvants

- Spinal anesthesia, when appropriate, may be beneficial in preterm infant.

Postoperative Care

- Consider monitoring preterm infant for apnea and bradycardia for 24 h.

Anticipated Problems/Concerns

- Anemia is a significant risk factor for postop apnea in preterm infant undergoing surgery and anesthesia.

Pickwickian Syndrome

Aaron M. Fields | Ryan E. Rubin

Risk

- Affects 5–10% of morbidly obese pts
- Usually associated with long-standing obesity

Perioperative Risks

- Markedly greater risk among the morbidly obese vs. pts with normal BMI.
- With intraabdominal or intrathoracic procedures lasting more than 2 h, there is approximately 40% of serious morbidity.

Worry About

- Hypoventilation
- Hypercarbia
- Hypoxemia
- Polycythemia, thrombophlebitis, and subsequent pulm embolism
- Pulm Htn
- Hypersomnolence
- Biventricular cardiac failure

Overview

- Pickwickian syndrome, or OHS, is defined as the combination of obesity (BMI above 30 kg/m^2), hypoxia during sleep, and hypercapnia.
- Morbidly obese pts who hypoventilate due to sleep apnea and severe restrictive ventilatory disorder have permanent pulm Htn, acidosis, and polycythemia because of their chronic hypoxemia and CO_2 retention.
- OHS is usually associated with systemic Htn and acompensatory increase in circulating blood volume, leading to right and left ventricular failure.
- Two subtypes are recognized, depending on the nature of the disordered breathing detected on further investigation. The first is OHS in the context of obstructive sleep apnea; this is confirmed by the occurrence of five or more episodes of apnea, hypopnea, or respiration-related arousals per h (high apnea-hypopnea index) during sleep. The second is OHS primarily due to "sleep hypoventilation syndrome;" this requires a rise of CO_2 levels by 10 mm Hg (1.3 kPa) after sleep compared to awake measurements and overnight drops in O_2 levels without simultaneous apnea or hypopnea. Overall, 90% of all people with OHS fall into the first category and 10% in the second.
- On physical exam, characteristic findings are the presence of a raised jugular venous pressure, a palpable parasternal heave, a heart murmur due to tricuspid regurgitation, hepatomegaly, ascites, and leg edema.

Etiology

- Work of breathing is increased as adipose tissue restricts the normal movement of the chest muscles and makes the chest wall less compliant, causing the diaphragm to move less effectively. Respiratory muscles are fatigued more easily, and airflow is impaired by excessive tissue in the head and neck area.

- Under normal circumstances, central chemoreceptors in the brain stem detect decreased pH and respond by increasing the respiratory rate; in OHS, the ventilatory response is blunted.
- Episodes of nighttime acidosis due to sleep apnea lead to renal compensation with retention of bicarbonate.
- Nighttime apnea leads to hypoxia, causing hypoxic pulm vasoconstriction. This vasoconstriction, in turn, leads to pulm Htn as well as right ventricular failure and remodeling.
- The chronically low O_2 levels in the blood also lead to increased release of erythropoietin, causing polycythemia.

Usual Treatment

- Weight loss through diet and exercise (which is rarely successful) or bariatric surgery
- NIPPV
- Uvulopalatopharyngoplasty
- Tracheostomy

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Difficult airway access	Snoring	Poor visualization	X-ray of neck may be helpful
CV	Biventricular failure CAD	Dyspnea, poor exercise tolerance Angina, poor exercise tolerance	Venous engorgement, S_3 and S_4, dyspnea	ECG, ECHO, CXR ECG, stress ECHO, angio
RESP	Hypoventilation	Dyspnea, sleeping upright Poor exercise tolerance	Rapid shallow breathing, cyanosis	ABGs, Hct, CXR

Key References: Olson A, Zwillich C: The obesity hypoventilation syndrome, *Am J Med* 118:948–956, 2005; Chau EH, Lam D, Wong J: Obesity hypoventilation syndrome: a review of epidemiology, pathophysiology, and perioperative considerations, *Anesthesiology* 117(1):188–205, 2012.

Perioperative Implications

Preoperative Preparation

- Consider pulm function tests with bronchodilator to determine whether a reversible restrictive component exists.
- Assess for bronchitis/pneumonia, which can be improved with pulm toilet and antibiotic therapy.
- Assess myocardial and volume status using a central venous catheter or PA cath.
- Consider maintaining pt in a semisitting position to avoid sudden shifts of volume to central circulation and pulm edema.

Monitoring

- Consider an arterial line for frequent monitoring of ABGs.
- Maintain adequate respiratory volumes and pressures.
- Consider PAC or transesophageal ECHO to monitor filling volumes and wall motion.

Airway

- Awake intubation frequently required.
- Laryngoscopy can sometimes be facilitated by elevating the shoulders and head on a bolster.

Induction

- Do not expect to ventilate pt adequately by mask. Establish airway first.

Maintenance

- Pts may have to remain in reverse Trendelenburg position to allow adequate ventilation.

Extubation

- Perform with pt in sitting position without residual sedation.
- Ensure adequate tidal volume and consider preop levels of CO_2 retention in making decision to extubate, as a normal CO_2 level may not be attainable.

Adjuvants

- Regional anesthesia only if pt is able to maintain ventilation
- Residual sedation or narcosis may preclude early extubation.

Postoperative Period

- Consider prophylaxis for thromboembolism; early ambulation may minimize pulm and thromboembolic complications.
- Pts may be extremely sensitive to the respiratory depressant effects of benzodiazepines and narcotics.

Anticipated Problems/Concerns

- All those problems associated with morbid obesity apply to Pickwickian pts.
- Early ambulation may minimize pulm and thromboembolic complications.
- Prepare the pt for a possibly prolonged course of postop mechanical ventilation, especially after upper abdominal procedures.

Pierre Robin Sequence

Charles B. Cauldwell

Risk

- 1:8500-14,000 live births; PRS nonsyndromic in about 40% of cases.
- Syndromic PRS most commonly associated with Stickler, velocardiofacial, and Treacher-Collins syndromes.

Perioperative Risks

- Chronic airway obstruction, respiratory distress, hypoxia.
- Malnutrition due to feeding difficulties, GE reflux.
- Congenital heart defects with syndromic PRS.

Worry About

- Airway obstruction
- Difficult intubation

Overview

- An anomaly consisting of micrognathia (or retrognathia), glossoptosis (posterior displacement of the tongue), and varying degrees of airway obstruction as well as feeding difficulties; cleft palate may be present but is not diagnostic.
- Airway obstruction, which may be multilevel, can lead to hypoxia and /or cyanosis.
- Feeding problems associated with malnutrition, reflux, and aspiration.
- In nonsyndromic PRS, if hypoxia and malnutrition are overcome, obstruction may improve secondary to mandibular growth by the time the pt reaches several mo of age.

Etiology

- Nonsyndromic PRS associated with a defect in gene SOX9, a chondrogenic regulator.
- Syndromic PRS associated with multiple genetic syndromes involving multiple defective genes.

Usual Treatment

- Prone positioning, lavage feeding may treat 70% successfully.
- Nasopharyngeal airway is the next level of intervention,
- Glossopexy or mandibular distraction osteogenesis to relieve airway obstruction and allow growth.
- Tracheostomy for multilevel airway obstruction or failure of previous surgery.