Carbon Monoxide Poisoning

Carbon monoxide poisoning is almost always associated with burns and smoke inhalation. CO produces tissue hypoxia by it's 200-fold greater affinity for hemoglobin than oxygen and by its ability to shift the hemoglobin dissociation curve to the left, thereby impairing unloading of O2 to the tissues and producing tissue hypoxia. CO can also cause metabolic acidosis by interfering with mitochondrial function, uncoupling oxidative phosphorylation and ATP production.

ANESTHETIC CONSIDERATIONS:

- Considerations of the burn patient and concomitant injury including inhalational injury
- Unreliability of routine SpO2 monitoring for O2 saturation
  - Co-oximitry should be used to measure O2 saturation
  - Classic 'cherry red' color may not be present because it occurs only at COHb concentration >40% and may be obscured by coexistent hypoxia and cyanosis
- Complications of systemic hypoxemia
  - CNS – headache, dizziness, confusion, agitation, combativeness, coma, hypoxic brain injury
  - CVS – myocardial ischemia, arrhythmias, myocardial infarction
  - RESP – hypoxemic respiratory failure, failure to unload O2 at tissue level
  - GI – nausea and vomiting
  - GU/metabolic – metabolic acidosis, lactic acidosis
- Provision of 100% O2 to improve oxygenation and promote elimination of CO
- Consider hyperbaric O2 therapy
  - Comatose or neurologically abnormal at presentation
  - CO >40%
  - Pregnant with CO >15%

ANESTHETIC GOALS:

- Maintain oxygenation by providing 100% O2 and or hyperbaric oxygen therapy
- Encourage CO elimination by providing 100% O2 and/or hyperbaric oxygen therapy

PATHOPHYSIOLOGY

- CO is a colorless, odorless, nonirritating gas that is easily absorbed through the lungs. IT is a common cause of mortality and the leading cause of poisoning mortality in the USA.
- Poisoning may be accidental (fire related smoke inhalation, motor vehicle exhaust, poorly functioning heating system) or intentional
- Amount of CO absorbed is dependent on:
  1. Minute ventilation
  2. Duration of exposure
  3. Ambient CO and oxygen concentrations
- CO toxicity results from tissue hypoxia and direct CO mediated cellular damage
  1. Impaired release of O2 to tissues secondary to left shift of oxyhemoglobin dissociation curve causing tissue hypoxia
  2. Disruption of oxidative metabolism, increased NO concentrations, brain lipid peroxidation, generation of oxygen free radicals, and produces other metabolic changes that may result in neurologic and cardiac toxicity
- CO competes with O2 for binding to hemoglobin
  1. CO affinity for Hgb is 200x > O2
  2. CO affinity for HbF is even greater than adult Hgb
  3. CO readily crosses placenta and fetal COHgb may exceed maternal COHgb concentration and fetal eliminiation of CO is slower than mom
- Signs and symptoms of CO exposure are initially nonspecific
  1. Headache, nausea, vomiting, weakness, difficulty concentrating, confusion
  2. Brain and heart highly O2 dependent – show major signs of injury
     1. Tachycardia and tachypnea in response to cellular hypoxia
     2. Angina, dysrhythmias, pulmonary edema from increased CO secondary to hypoxia
     3. Syncope and seizures (cerebral hypoxia and cerebral vasodilation)
  3. Systemic hypotension in CO poisoning is correlated with severity of CNS structural damage
  4. Classic 'cherry red lips' is rarely seen
- Long term sequelae:
  1. Persistent or delayed neurologic effects
  2. Delayed neuropsychiatric syndrome
     1. Cognitive dysfunction
     2. Memory loss
     3. Seizures
     4. Personality changes
     5. Parkinsonism
     6. Dementia
     7. Mutism
     8. Blindness
     9. Psychosis
- Treatment:
  1. Remove the patient from the source of the CO production
  2. Immediate supplemental O2 administration
     1. Decreases elimination half time of CO by competing at the binding sites for Hgb and improves tissue oxygenation
        a. CO half life
           i. FiO2 0.21: 4-6 h
           ii. FiO2 1.0: 40-80min
           iii. FiO2 hyperbaric O2 (100% O2 in pressurized chamber resulting in increased dissolved O2 in blood): 15-30min

iii. Supportive care
    1. Airway management
    2. Blood pressure support
    3. Cardiovascular stabilization
iv. Hyperbaric O2 therapy
    1. Indications:
        a. Coma or impaired neurological status at presentation
        b. COHb >40% (>30% in barash; >40% coexisting)
        c. Pregnant and COHb >15%
    2. Accelerates CO elimination
    3. May decrease frequency of neurologic sequelae in severe CO exposure

HISTORY

- History of smoke inhalation?
    - Associated airway edema?
    - Associated airway obstruction or impending airway obstruction?
    - Associated pulmonary inhalational injury?
- History of motor vehicle exhaust exposure?
- History of CO exposure in the home? (faulty heater)
- Symptoms:
    - <15-20% COHb: Headache, dizziness, confusion
    - 20-40% COHb: nausea, vomiting, disorientation, visual impairment
    - 40-60% COHb: agitation, combativeness, hallucinations, coma, shock
    - >60% COHb: Death
- PMHx
- Meds
- Allergies
- Past surgical history/Past anesthetic history

PHYSICAL

- Vitals
    - SpO2 inaccurate and will overestimate SaO2
    - Co-oximetry by ABG analysis is the most accurate way of determining oxygenation and COHb concentration
- Routine preoperative examination including:
    - Focused airway exam
    - Focused cardiorespiratory exam
    - Focused neurological exam

LABS

- ABG with co-oximeter (spectrophotometry)

INVESTIGATIONS

- Preoperative tests as indicated
- ABG

OPTIMIZATION

- Aggressive oxygenation with 100% FiO2 +/- hyperbaric O2 therapy as indicated

ANESTHETIC OPTIONS

- None
- Regional
- Neuraxial
- General

ANESTHETIC SETUP

- Standard emergency drugs
- Equipment
    - cooximeter

MANAGEMENT OF ANESTHESIA

INDUCTION

- Preoxygenation
- Avoid succinylcholine in burns after 24h – 1 year

MAINTENANCE

- Nothing specific

EMERGENCE

- Nothing specific

POSTOPERATIVE DISPOSITION

COMPLICATIONS

- Concurrent cyanide poisoning

REFERENCES:

Stoelting's Anesthesia and Coexisting Disease Page 552-553
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