Toxins

Carbon Monoxide

What is the likely SPO2 level on an obtunded victim suspected of CO poisoning with a Hgb-CO of 30 and placed on a non-rebreather mask?

a) 70  
b) 80  
c) 90  
d) 100
Learning Objectives

Upon the completion of this program participants will be able to:

1. Understand the pathophysiology of carbon monoxide (CO) poisoning
2. Describe the signs and symptoms of CO poisoning
3. Understand the methods of CO detection, and its sometimes elusive diagnosis
4. Describe the treatment and disposition of patients with CO poisoning

Chemistry of Carbon Monoxide

- **Gas:**
  - Colorless
  - Odorless
  - Tasteless
  - Nonirritating
- Results from the incomplete combustion of carbon-containing fuels.
- Stable Molecule: Triple Bond
- Some ambient

CO Pathophysiology

- Affinity for heme 2-300x O₂
  - displaces O₂ from binding sites.
  - prevents O₂ from binding.
- Shifts oxy-Hgb curve to left preventing release of O₂
- Essentially induces relative (functional) anemia
Pathophysiology

• CO also binds to other iron-containing proteins:
  – Myoglobin (dysrhythmias and cardiac dysfunction)
  – Cytochrome oxidase (met acidosis)

• Direct injury to endothelium releases nitric oxide
  – Peripheral vasodilation and hypotension
  – Inflammatory response
  – Increased free radical injury

Epidemiology

• CO is leading cause of poisoning deaths in industrialized countries.
• CO may be responsible for half of all poisonings worldwide.
• ~5,000–6,000 people die annually in the United States as a result of CO poisoning.
• ~40,000–50,000 emergency department visits annually result from CO poisoning.

Incidence

• Significant increase in CO poisoning seen following disasters.
• Primarily relates to loss of utilities and reliance on gasoline-powered generators and use of fuel-powered heaters.
Incidence and Epidemiology

• Most accidental deaths are due to:
  – House fires.
  – Automobile exhaust.
  – Indoor-heating systems.
  – Stoves and other appliances.
  – Charcoal grills.
  – Camp stoves.
  – Water heaters.
  – Boat exhausts.

Seasonal and Demographic Variation

• Increased accidental CO deaths:
  – Patient > 65 years of age.
  – Male
  – Ethanol intoxication.

• Accidental deaths peak in winter:
  – Use of heating systems.
  – Closed windows.

Clinical Features of CO Poisoning

• Presentation resembles other diseases
  – Often dubbed the great imitator

• Often misdiagnosed as:
  – Viral illness (e.g., the “flu”)
  – Acute coronary syndrome
  – Headache/Migraine
  – Other Toxic Ingestions

• Misdiagnosis may occur in 30-50% of CO-patients presenting to the ED, often due to failure to consider

• Key is to suspect the diagnosis in first place.
Signs and Symptoms

• CNS
  – Confusion
  – Altered mental status
  – Drowsiness
  – Visual disturbances
  – Gait disturbance
  – Agitation
  – Seizures
  – Coma
  – Death

• Cardio-pulmonary
  – Chest pain
  – Palpitations/Dysrhythmias
  – Non-cardiogenic pulmonary edema
  – Hypotension
  – DOE/SOB
  – Syncope

• Other
  – Abdominal pain
  – Nausea/vomiting
  – Renal failure
  – Rhabdomyolysis

Signs and Symptoms (Chronic)

• Same as with acute CO EXCEPT that onset and severity may be extremely varied.

• Cherry red skin color is unreliable, not always present
  – May be a late finding- typically at post mortem

• CO-Hb may not correlate with symptoms/sequelae.

Long-Term Complications

• Delayed Neurologic Syndrome (DNS):
  – Recovery seemingly apparent.
  – Behavioral and neurological deterioration 2-40 days later.
  – True prevalence uncertain (estimate range from 1-47% after CO poisoning).
  – Patients more symptomatic initially appear more apt to develop DNS.
  – More common when there is a loss of consciousness in the acute poisoning.
### Delayed Neurologic Syndrome

- **Signs and Symptoms:**
  - Memory loss
  - Confusion
  - Ataxia
  - Seizures
  - Urinary incontinence
  - Fecal incontinence
  - Emotional lability

- **Signs and Symptoms:**
  - Disorientation
  - Hallucinations
  - Parkinsonism
  - Mutism
  - Cortical blindness
  - Psychosis
  - Gait disturbances
  - Other motor disturbances

### Non-combustion Source

- Don’t forget methylene chloride *(for the test)*
  - Look for in the patient using paint thinner/stripper
- Transdermal absorption
- Is metabolized to CO
- Carboxyhemoglobin levels may continue to rise
  - Unlike inhaled - should decline once source removed
  - Also could be utilized on a test scenario

### Diagnosis

- Lab determination of carboxyhemoglobin
  - Suspicion primarily based on history
  - Treatment primarily based on symptoms in setting of CO
- Now have co-oximetry capable of detecting
  - Carboxyhemoglobin
  - Methemoglobin
- May have value as screening tool
  - Fire/EMS units often have RAD-57 (Massimo)

---
Carbon Monoxide Detection

- New generation oximeter/CO-oximeter can detect 4 different hemoglobin forms.
  - Deoxyhemoglobin (Hb)
  - Oxyhemoglobin (O2-Hb)
  - Carboxyhemoglobin (CO-Hb)
  - Methemoglobin (MET-Hb)

- Provides:
  - SpO2
  - SpCO
  - SpMET
  - Pulse rate

- Pitfalls
  - SpO2 level falsely high in CO poisoning (Test question)
  - High ambient light

Patient Groups at Higher Risk

- Children
- Pregnant women
  - Fetal hemoglobin has greater affinity for CO
  - may exhibit milder symptoms with high fetal toxicity
- Elderly and underlying disease, co-morbidity in any ages
  - Cardiopulmonary diseases
  - Anemia

Treatment

- Supportive
  - ABC
  - Cardiac monitor
  - Symptomatic management
- Early high concentration oxygen reduces T 1/2:
  - Room air: 240-360 minutes
  - O2 (100%): 80-90 minutes
  - Hyperbaric O2: 22 minutes
Treatment

- Efficacy of hyperbaric oxygen therapy (HBO) is a matter of conjecture although still commonly practiced.
- Generally reserved for severe poisonings.
- May aid in alleviating tissue hypoxia.
- Significantly decreases half-life of CO-Hb.
- Specialists believe mitigates sequelae

Indications for HBO Therapy

- Syncope
- Altered mental status
- Coma
- Focal neurologic deficits
- Acute myocardial ischemia
- Seizures
- Pregnant CO-Hb >15% or fetal distress
- CO-Hb >25%

Take-Home Points

- CO poisoning is the great imitator.
- Early high flow $O_2$ is critical to reducing half-life
- CO-Hgb may not correlate with symptoms
  - Beware young, old and pregnant
  - Non-invasive CO monitoring is available
- Use of HBO therapy is controversial
  - Use in severe/refractory symptoms
- Immediate and long term effects from CO poisoning
**CO Poisoning**

- Remember, CO poisoning is the great imitator.
- Missed CO exposure often leads to death and disability.
- CO is a particular risk for firefighters.

**Treatment**

- *Administer high-concentration oxygen.*
  - Maximizes hemoglobin oxygen saturation.
  - Can displace some CO from hemoglobin.
  - Associated with improvements in neurological and cardiac complications.

**Indications for HBO Therapy**

- **Possibly consider for:**
  - Cardiovascular compromise (i.e., ischemia, dysrhythmias).
  - Metabolic acidosis
  - Extremes of age
Sources of Carbon Monoxide

- House fires
- Automobile (gasoline engine) exhaust
- Space heaters
- Indoor grills/barbeques
- Camp stoves
- Cigarette smoke

Environmental CO Exposure

- Environmental exposure typically <0.001% (10 ppm).
- Higher in urban areas.
- Sources:
  - Volcanic gasses
  - Bush fires
  - Human pollution

<table>
<thead>
<tr>
<th>Source</th>
<th>Exposure (ppm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fresh Air</td>
<td>0.06-0.5</td>
</tr>
<tr>
<td>Urban Air</td>
<td>1-30</td>
</tr>
<tr>
<td>Smoke-filled Room</td>
<td>2-16</td>
</tr>
<tr>
<td>Cooking on Gas Stove</td>
<td>100</td>
</tr>
<tr>
<td>Actively Smoking a Cigarette</td>
<td>400-500</td>
</tr>
<tr>
<td>Automobile Exhaust</td>
<td>100,000</td>
</tr>
</tbody>
</table>
Firefighter Risks

- CO is a significant and deadly occupational risk factor for firefighters.
- Sources:
  - Structure fires
  - Apparatus fumes
  - Portable equipment fumes
  - Underground utility fires
  - Closed-space rescue situations

Pathophysiology

- Pathophysiology of CO poisoning first described by French physician Claude Bernard in 1857.
- CO poisoning actually very complex.
- CO binds to hemoglobin with an affinity ~ 250 times that of oxygen.
- The combination of CO and hemoglobin is called carboxyhemoglobin (CO-Hb).

<table>
<thead>
<tr>
<th>Source</th>
<th>CO-Hb (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endogenous</td>
<td>0.4-0.7</td>
</tr>
<tr>
<td>Tobacco Smokers:</td>
<td></td>
</tr>
<tr>
<td>1 pack/day</td>
<td>5-6</td>
</tr>
<tr>
<td>2-3 packs/day</td>
<td>7-9</td>
</tr>
<tr>
<td>cigars</td>
<td>Up to 20</td>
</tr>
<tr>
<td>Urban Commuter</td>
<td>5</td>
</tr>
<tr>
<td>Methylene chloride (100 ppm for 8 hours)</td>
<td>3-5</td>
</tr>
</tbody>
</table>
Pathophysiology

• Impact of CO on major body systems:
  — Neurologic:
    • CNS depression resulting in impairment:
      — Headache
      — Dizziness
      — Confusion
      — Seizures
      — Coma
    • Long-term effects:
      — Cognitive and psychiatric problems

• Impact of CO on major body systems:
  — Cardiac:
    • Decreased myocardial function:
      — Hypotension with tachycardia
      — Chest pain
      — Dysrhythmias
      — Myocardial ischemia
      — Most CO deaths are from ventricular fibrillation.
    • Long-term effects:
      — Increased risk of premature cardiac death

• Impact of CO on major body systems:
  — Metabolic:
    • Respiratory alkalosis (from hyperventilation)
    • Metabolic acidosis with severe exposures
  — Respiratory:
    • Pulmonary edema (10-30%)
      — Direct effect on alveolar membrane
      — Left-ventricular failure
      — Aspiration
      — Neurogenic pulmonary edema
Pathophysiology

• Impact of CO on major body systems:
  – Multiple Organ Dysfunction Syndrome (MODS):
    • Occurs at high-levels of exposure
    • Associated with a high mortality rate.

Pathophysiology Summary

• Limits O₂ transport:
  – CO more readily binds to Hb forming CO-Hb.

• Inhibits O₂ transfer:
  – CO changes structure of Hb causing premature release of O₂ into the tissues.

• Tissue inflammation:
  – Poor perfusion initiates an inflammatory response.

Pathophysiology Summary

• Poor cardiac function:
  – ↓ O₂ delivery can cause dysrhythmias and myocardial dysfunction.
  – Long-term cardiac damage reported after single CO exposure.

• Increased activation of nitric oxide (NO):
  – Peripheral vasodilation.
  – Inflammatory response.
CO Poisoning

- Signs and symptoms closely resemble those of other diseases.
- Often misdiagnosed as:
  - Viral illness (i.e., influenza)
  - Acute coronary syndrome
  - Migraine
- Estimated that misdiagnosis may occur in up to 30-50% of CO-exposed patients presenting to the ED.

CO POISONING SIGNS & SYMPTOMS

- Signs and symptoms usually vague and non-specific.
- You must ALWAYS maintain a high index of suspicion for CO poisoning!

CO Poisoning

- Classifications:
  - Acute
    - Results from short exposure to a high level of CO.
  - Chronic
    - Results from long exposure to a low level of CO.
**Signs and Symptoms (Acute)**

- Malaise
- Flu-like symptoms
- Fatigue
- Dyspnea on exertion
- Chest pain
- Palpitations
- Lethargy
- Confusion
- Depression
- Impulsiveness
- Distractibility
- Hallucination
- Confabulation
- Agitation
- Nausea
- Vomiting
- Diarrhea
- Abdominal pain

**CO ppm | Duration | Symptoms**

<table>
<thead>
<tr>
<th>CO ppm</th>
<th>Duration</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>8 hours</td>
<td>OSHA minimum</td>
</tr>
<tr>
<td>200</td>
<td>2-3 hours</td>
<td>Mild headache, fatigue, nausea, dizziness</td>
</tr>
<tr>
<td>400</td>
<td>1-2 hours</td>
<td>Serious headache—other symptoms intensify. Life-threatening &gt; 3 hours</td>
</tr>
<tr>
<td>800</td>
<td>45 minutes</td>
<td>Dizziness, nausea and convulsions. Unconscious within 2 hours. Death within 2-3 hours</td>
</tr>
<tr>
<td>1,600</td>
<td>20 minutes</td>
<td>Headache, dizziness and nausea. Death within 1 hour</td>
</tr>
<tr>
<td>3,200</td>
<td>5-10 minutes</td>
<td>Headache, dizziness and nausea. Death within 1 hour</td>
</tr>
<tr>
<td>6,400</td>
<td>1-2 minutes</td>
<td>Headache, dizziness and nausea. Death within 25-30 minutes</td>
</tr>
<tr>
<td>12,800</td>
<td>1-3 minutes</td>
<td>Death</td>
</tr>
</tbody>
</table>

**Signs and Symptoms (acute)**

- Headache
- Drowsiness
- Dizziness
- Weakness
- Confusion
- Visual disturbances
- Syncope
- Seizures
- Fecal incontinence
- Urinary incontinence
- Memory disturbances
- Gait disturbances
- Bizarre neurologic symptoms
- Coma
- Death
**Long-Term Complications**

- **Cardiac Complications:**
  - 230 sequential patients with moderate to severe CO poisoning treated with HBO.

<table>
<thead>
<tr>
<th>CO Myocardial Injury</th>
<th>Patients</th>
<th>Died (%)</th>
<th>5-year Survival (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial injury from CO</td>
<td>85</td>
<td>37.6</td>
<td>71.6</td>
</tr>
<tr>
<td>No Myocardial injury from CO</td>
<td>145</td>
<td>15.2</td>
<td>88.3</td>
</tr>
</tbody>
</table>

  *Myocardial injury and long-term mortality following moderate to severe carbon monoxide poisoning.*  

- **Depression and anxiety can exist up to 12 months following CO exposure.**
- **Higher at 6 weeks in patients who attempted suicide by CO.**
- **No differences in rates between accidental and suicide-attempt at 12 months.**

**Carbon Monoxide Detection**

- **Biological CO detection previously required hospital-based ABGs or venous sample to measure CO-Hb.**
- **Technology now available to detect biological CO-Hb levels in the prehospital and ED setting.**
- **Referred to as CO-oximetry**
**CO-Oximetry**

- Monitoring of SpO₂ and SpCO levels throughout treatment.
- Obtain 12-lead ECG (if ALS) and monitor ECG.