Endorsements

This educational module has been endorsed by the International Association of Firefighters (IAFF).

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Carbon monoxide is the most frequent cause of poisonings in industrialized countries.

**CHEMISTRY**

**Chemistry of Carbon Monoxide**
- **Gas:** Colorless, Odorless, Tasteless, Nonirritating
- **Results from the incomplete combustion of carbon-containing fuels.**
- **Abbreviated “CO”**
Chemistry of Carbon Monoxide

- Molecule consists of one carbon atom joined to one oxygen atom by a triple bond.
- Extremely stable molecule.

\[ :C\equiv O: \]

112.8 pm

Sources of Carbon Dioxide

- Endogenous
- Exogenous
- Methylene chloride

Sources
**Sources of Carbon Monoxide**

**Endogenous:**
- Normal heme catabolism (breakdown):
  - Only biochemical reaction in the body known to produce CO.
- Levels increased in:
  - Hemolytic anemia.
  - Sepsis

**Exogenous:**
- House fires.
- Gas-powered electrical generators.
- Automobile exhaust.
- Propane-powered vehicles.
- Heaters.
- Camp stoves.
- Boat exhaust.
- Cigarette smoke.

**Methylene chloride:**
- Paint and adhesive remover.
- Converted to CO in the liver after inhalation.
INCIDENCE

Incidence
- CO is leading cause of poisoning deaths.
- 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
- Accidental CO poisoning deaths declining:
  - Improved motor vehicle emission policies.
  - Use of catalytic converters.
**Incidence**

Most accidental deaths are due to:
- House fires.
- Automobile exhaust.
- Indoor-heating systems.
- Stoves and other appliances.
- Gas-powered electrical generators.
- Charcoal grills.
- Camp stoves.
- Water heaters.
- Boat heaters.

**Incidence**

Increased accidental CO deaths:
- Patient > 65 years of age.
- Males.
- Ethanol intoxication.
- Accidental deaths peak in winter:
  - Use of heating systems.
  - Closed windows.

**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

Fetal hemoglobin has a much greater affinity for CO than adult hemoglobin.
Pregnant mothers may exhibit mild to moderate symptoms, yet the fetus may have devastating outcomes.

EXPOSURE

Environmental CO Exposure

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

<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>

### CO Exposure

- **CO absorption by the body is dependent upon:**
  - Minute ventilation ($V_{\text{min}}$).
  - Duration of exposure.
  - Concentration of CO in the environment.
  - Concentration of O2 in the environment.

### Exposure Limits

- **OSHA:**
  - 50 ppm (as an 8-hour time-weighted average).

- **NIOSH:**
  - 35 ppm (as an 8-hour time-weighted average).
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.
- CO is heavier than air and accumulates in lower regions.

Firefighter Risks

- SCBA extremely important in CO prevention.
- CO often encountered during overhaul operations.

CO POISONING PATHOPHYSIOLOGY
Pathophysiology

- Pathophysiology of CO poisoning first described by French physician Claude Bernard in 1857.

Pathophysiology

- 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 (COHb).

Pathophysiology

- CO displaces O₂ from the hemoglobin binding sites.
- CO prevents O₂ from binding.
- COHb does not carry O₂.
- COHb causes premature release of remaining O₂ into the tissues.
Pathophysiology

- COHb ultimately removed from the circulation and destroyed.
- Half-life:
  - Room air: 240-360 minutes
  - O₂ (100%): 80 minutes
  - Hyperbaric O₂: 22 minutes

- CO also binds to other iron-containing proteins:
  - Myoglobin
  - Cytochrome

- Binding to myoglobin reduces O₂ available in the heart:
  - Ischemia
  - Dysrhythmias
  - Cardiac dysfunction
**Normal COHb Levels**

<table>
<thead>
<tr>
<th>Source</th>
<th>COHb (%)</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**

**Nitric Oxide (NO):**
- Highly reactive gas that participates in numerous biochemical reactions.
- Oxygen free-radical
- Levels increased with CO exposure.

**Pathophysiology**

**Nitric Oxide (NO):**
- Cause cerebral vasodilation:
  - Syncope
  - Neckache
- May lead to oxidative damage to the brain:
  - Probable cause of syndrome of delayed napalm sequelae (DNS).
  - Associated with reperfusion injury.
Pathophysiology

Impact of CO on major body systems:

Neurologic:
- CNS depression resulting in impairment:
  - Headache
  - Dizziness
  - Confusion
  - Fatigue
  - Coma
- Long-term effects:
  - Cognitive and psychiatric problems

Pathophysiology

46-year-old woman with chronic exposure to CO from old car.
- COHb = 46%
- Autopsy:
  - Cherry-red tissues
  - Cerebral edema
  - Immediate cause of death: ventricular fibrillation due to CO poisoning.

Pathophysiology

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.
Pathophysiology

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 COHb.

- **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.

Vasodilation:
- Results from NO increase.
- Cerebral vasodilation and systemic hypotension causes reduced cerebral blood flow.
- NO is largely converted to methemoglobin.

Free radical formation:
- NO accelerates free radical formation.
- Endothelial and oxidative brain damage.

Patient Groups at Risk

- Children.
- Elderly:
- Persons with heart disease.
- Pregnant women.
- Patients with increased oxygen demand.
- Patients with decreased oxygen-carrying capacity (i.e., anemias, blood cancers).
- Patients with chronic respiratory insufficiency.
CO POISONING
SIGNS & SYMPTOMS

CO Poisoning

Signs and symptoms usually vague and non-specific.

You must ALWAYS maintain a high index of suspicion for CO poisoning!

CO Poisoning

Signs and symptoms closely resemble those of other diseases.
Often misdiagnosed as:
- Viral illness (e.g., the "flu")
- Acute coronary syndrome
- Migraines
Estimated that misdiagnosis may occur in up to 30-50% of CO-exposed patients presenting to the ED.
Carbon Monoxide Poisoning (IAFF) 6/12/2007

Signs and Symptoms

Carbon Monoxide
The Great Imitator†

† - So is:
• Syphilis
• Lyme disease
• Fibromyalgia
• Lupus erythematosus
• Multiple sclerosis

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
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

Firefighter Headaches
- While CO should always be considered a possible cause of headaches in working firefighters, there are more common causes:
  - Tight helmet ratchet.
  - Too heavy a helmet (especially leather).
  - Dehydration.

Signs and Symptoms (Chronic)
- Signs and symptoms the same as with acute CO poisoning except that onset and severity may be extremely varied.
Carbon Monoxide Poisoning (IAFF) 6/12/2007

**Signs and Symptoms**

<table>
<thead>
<tr>
<th>Severity CO-Hb Level</th>
<th>Signs &amp; Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild &lt; 15 - 20%</td>
<td>Headache, nausea, vomiting, dizziness, blurred vision.</td>
</tr>
<tr>
<td>Moderate 21 - 40%</td>
<td>Confusion, syncope, chest pain, dyspnea, weakness, tachycardia, tachypnea, rhabdomyolysis.</td>
</tr>
<tr>
<td>Severe 41 - 59%</td>
<td>Palpitations, dysrhythmias, hypotension, myocardial ischemia, cardiac arrest, respiratory arrest, pulmonary edema, seizures, coma.</td>
</tr>
<tr>
<td>Fatal &gt; 60%</td>
<td>Death finding.</td>
</tr>
</tbody>
</table>

**COHb levels do not always correlate with symptoms nor predict sequelae.**

**CO ppm**  | **Duration** | **Symptoms** |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></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**

CO may be the cause of the phenomena associated with haunted houses:
- Strange visions.
- Strange sounds.
- Feelings of dread.
- Hallucinations.
- Inexplicable deaths.

Cherry red skin color is not always present and, when present, is often a late finding.
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
- Disorientation
- Hallucinations
- Parkinsonism
- Mutilam
- Cortical blindness
- Psychosis
- Gait disturbances
- Other motor disturbances

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 (n)</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>

**Long-Term Complications**

- 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.

**CO DETECTION**

**Carbon Monoxide Detection**

- CO detectors have been widely-available for over a decade.
- Still vastly underutilized.
- Underwriters Laboratories (UL) revised guidelines for CO detectors in 1998.
- Units manufactured before 1998 should be replaced.
Carbon Monoxide Detection

**Biological detection of CO limited:**
- Exhaled CO measurement.
- Hospital-based carboxyhemoglobin levels (arterial or venous).

**Technology now available to detect biological COHb levels in the prehospital and ED setting.**
Referred to as CO-oximetry

**Hand-held devices now available to assess atmospheric levels of CO.**
Multi-gas detectors common in the fire service:
- Combustible gases
- CO
- O₂
- H₂S
Carbon Monoxide Detection

New generation oximeter/CO-oximeter can detect 4 different hemoglobin forms:
- Deoxyhemoglobin (Hb)
- Oxyhemoglobin (O2Hb)
- Carboxyhemoglobin (COHb)
- Methemoglobin (METb)

Provides:
- SpO2
- SpCO
- SpMET
- Pulse rate

CO-Oximetry

Uses finger probe similar to that used in pulse oximetry.
Uses 8 different wavelengths of light (instead of 2 for pulse oximetry).
Readings very closely correlate with COHb levels measured in hospital.

CO-Oximetry

![Graph showing absorption vs. wavelength for different hemoglobin forms.](Image)

- Deoxyhemoglobin
- Oxyhemoglobin
- Carboxyhemoglobin
- Methemoglobin

Absorption (mm⁻¹) vs. Wavelength (nm)
CO-Oximetry

- CO evaluation should be routine at all levels of EMS and the fire service.
- All field personnel should be educated in use of the oximeter and CO-oximeter.

Missed CO poisoning is a significant area of legal liability for EMS and fire personnel.

CO POISONING TREATMENT

Diagnostic Criteria

- Biologic:
  - COHb > 5% in nonsmokers.
  - COHb > 10% in smokers.
- Environmental:
  - No confirmatory test.
Diagnostic Criteria

- **Suspected:**
  - Potentially-exposed person, but no credible threat exists.

- **Probable:**
  - Clinically-compatible case where credible threat exists.

- **Confirmed:**
  - Clinically-compatible case where biological tests have confirmed exposure.

Treatment

- Treatment is based on the severity of symptoms.
- Treatment generally indicated with SpCO > 10-12%.
- Be prepared to treat complications (i.e., seizures, dysrhythmias, cardiac ischemia).

The importance of early administration of high-concentration oxygen CANNOT be overemphasized!
Treatment

- Prehospital CPAP can maximally saturate hemoglobin and increase oxygen solubility.
- Strongly suggested for moderate to severe poisonings.

Treatment Algorithm

- 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 COHb.
Indications for HBO Therapy

**Strongly consider for:**
- Altered mental status.
- Coma.
- Focal neurological deficits.
- Seizures.
- Pregnancy with COHb>15%.
- History of LOC.

Indications for HBO Therapy

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

Treatment

- Continue to monitor SpO2 and SpCO levels throughout treatment.
- Obtain 12-lead ECG (if ALS) and monitor ECG.
- Document findings and plot trends.
Treatment

First-generation pulse oximeters may give falsely elevated SpO₂ levels in cases of carbon monoxide poisoning.

Cannot distinguish between O₂Hb and COHb.

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.

A simple COHb reading can save a life and possibly prevent long-term complications.

METHYLENE CHLORIDE
Methylene Chloride Exposure

- Methylene chloride slowly metabolized to CO.
- Victims do not pose contamination risks to rescuers.
- Victims with contaminated clothing or skin can secondarily contaminate response personnel by direct contact or through off-gassing vapor.
- Methylene chloride vapor may also off-gas from the toxic vomitus of victims who have ingested methylene chloride.

Methylene Chloride Exposure

- Methylene chloride can cause:
  - Acute CNS depression.
  - Respiratory depression.
  - Cardiac dysrhythmias.
  - Respiratory tract irritation (at high levels).
  - Non-cardiogenic pulmonary edema (at high levels).

Methylene Chloride Exposure

- Treatment:
  - No antidote for methylene chloride.
  - Support respiratory and cardiovascular functions.
  - Administer O₂ (O₂ is an antagonist of metabolically-produced carbon monoxide).
DOUBLE TROUBLE: CO and CYANIDE

Carbon Monoxide and Cyanide

- Cyanide more often encountered in fires than once thought.
- The effects of CO and cyanide are cumulative.
- Symptoms of cyanide toxicity often attributed to CO because of lack of a high index of suspicion.

Chemistry of Cyanide

- Gas:
- Colorless.
- Faint bitter almond smell.
- Nearly 40% of the population cannot smell cyanide.
- Sodium cyanide (NaCN) and potassium cyanide (KCN) are both white powders.
Chemistry of Cyanide

- Molecule consists of one carbon atom joined to one nitrogen atom by a triple bond.
- Cyanide anion is extremely toxic.

\[
: \text{C}≡\text{N}:^-
\]

Cyanide

- Hydrogen cyanide is a product of combustion.
- High in:
  - Plastics.
  - Wool
  - Silk.
  - Synthetic rubber.
  - Polyurethane.
  - Asphalt.

Cyanide

- Toxicity varies with chemical form.
- Hydrogen cyanide (HCN) gas at concentrations of 130 ppm can be fatal within an hour.
- OSHA permissible exposure levels are 10 ppm as an 8-hour time-weighted average.
Pathophysiology

- Cyanide can be inhaled or ingested.
- Ingestion more common with suicide or murders.

Pathophysiology

- Cyanide is an irreversible enzyme inhibitor:
  - Cytochrome c oxidase (complex 4).
  - Part of the 4th complex of the electron transport chain.
  - Found in the shelves (cristae) of the mitochondria in the cells.

Mitochondrial Electron Transport Chain

Cyanide deactivates this enzyme
Pathophysiology

- Cyanide stops the electron transport chain and stops energy production (ATP) in the cell.
- Tissues that primarily depend on aerobic respiration are particularly affected:
  - Heart
  - Central nervous system
  - Cyanide and CO both primarily affect the heart and CNS thus multiplying the ill-effects!

Cyanide Treatment

- Antidotes available:
  - Cyanide Antidote Kit:
    - Amyl nitrite.
    - Sodium nitrite.
    - Sodium thiosulfate.
    - Hydroxocobalamin.

Cyanide Poisoning

- Amyl nitrite is administered via inhalation or ventilation.
- Sodium nitrite is administered intravenously.
- Sodium thiosulfate is administered intravenously.
Cyanide Treatment

- The nitrites promote the formation of methemoglobin.
- Cyanide has a greater affinity for methemoglobin (METHb) than the cytochrome oxidase enzyme.
- The binding of cyanide to METHb frees cytochrome oxidase so that energy production is resumed.

Cyanide Treatment

- Sodium thiosulfate binds to cyanide and forms thiocyanate.
- Thiocyanate much less toxic than cyanide anion and excreted through the kidneys.

Cyanide Treatment

- Hydroxocobalamin
  - Precursor to cyanocobalamin (Vitamin B₁₂)
  - Hydroxocobalamin combines with cyanide to form cyanocobalamin which is excreted through the kidneys.
  - FDA approval in US obtained in December 2006.
  - Marketed as Cyanokit™.
Cyanide Treatment

Problems (related to nitrites):
- METHb does not transport O₂.
- The conversion of Hb to METHb changes the state of the heme molecule where O₂ binds.
- METHb has heme in the ferric (Fe³⁺) state and not the ferrous state (Fe²⁺).
- O₂ can only bind to heme when in the Fe²⁺ state.

Cyanide Treatment

Concomitant CO and cyanide poisoning can significantly decrease the O₂-carrying capacity of the blood.

Combination of COHb and METHB can significantly reduce the O₂-carrying capacity of the blood.
Cyanide Treatment

Children are particularly at risk for hypotension and adverse effects from methemoglobinemia.

CO and Cyanide Poisoning

- Parts of cyanide antidote kit (amyl nitrite, sodium nitrite) induce methemoglobinemia.
- Cyanide antidotes and CO reduce COHb and METHb, reducing O2 capacity of blood.
- Sodium nitrite should be avoided for combination cyanide/CO poisonings when SpCO >10%.
- Hydroxocobalamin converts cyanide to cyanocobalamin (Vitamin B12) which is renally-cleared.

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