



**Carbon Monoxide  
Poisoning**  
Bryan E. Bledsoe, DO, FACEP



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



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### Review Board

- Roy Alson, MD, PhD, FACEP
- James Augustine, MD, FACEP
- Edward Dickinson, MD, FACEP
- Marc Eckstein, MD, FACEP
- Steven Katz, MD, FACEP
- Mike McEvoy, PhD, RN, EMT-P
- Joe A. Nelson, DO, MS, FACOEP, FACEP
- Ed Racht, MD
- Mike Richards, MD, FACEP
- Keith Wesley, MD, FACEP
- Paula Willoughby-DeJesus, DO, MHPE, FACOEP



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



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## CHEMISTRY



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### Chemistry of Carbon Monoxide

- ☛ **Gas:**
  - ☛ Colorless
  - ☛ Odorless
  - ☛ Tasteless
  - ☛ Nonirritating
- ☛ Results from the incomplete combustion of carbon-containing fuels.
- ☛ Abbreviated "CO"



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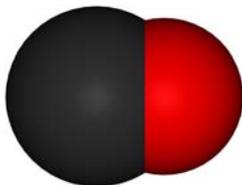
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## Chemistry of Carbon Monoxide

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



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## SOURCES



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## Sources of Carbon Monoxide

- Endogenous
- Exogenous
- Methylene chloride



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## Sources of Carbon Monoxide

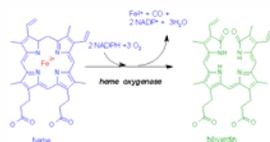
### Endogenous:

Normal heme catabolism (breakdown):

Only biochemical reaction in the body known to produce CO.

Levels increased in:

- Hemolytic anemia.
- Sepsis



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## Sources of Carbon Monoxide

### Exogenous:

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



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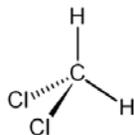
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## Sources of Carbon Monoxide

### Methylene chloride:

- Paint and adhesive remover.
- Converted to CO in the liver after inhalation.



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# INCIDENCE



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## 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.



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## Incidence

**Accidental CO poisoning deaths declining:**

- ✿ Improved motor vehicle emission policies.
- ✿ Use of catalytic converters.



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## 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 exhausts.



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## Incidence

### Increased accidental CO deaths:

- Patient > 65 years of age.
- Male
- Ethanol intoxication.

### Accidental deaths peak in winter:

- Use of heating systems.
- Closed windows.



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## Incidence

### Significant increase in CO poisoning seen following disasters.

- Primarily related to loss of utilities and reliance on gasoline-powered generators and use of fuel-powered heaters.



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## 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.



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## EXPOSURE



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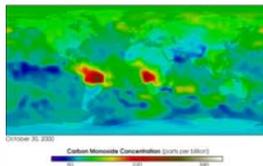
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## Environmental CO Exposure

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



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## CO Exposure

Source	Exposure (ppm)
Fresh Air	0.06-0.5
Urban Air	1-30
Smoke-filled Room	2-16
Cooking on Gas Stove	100
Actively Smoking a Cigarette	400-500
Automobile Exhaust	100,000




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## CO Exposure

### CO absorption by the body is dependent upon:

- ✿ Minute ventilation ( $V_{min}$ ).
- ✿ Duration of exposure.
- ✿ Concentration of CO in the environment.
- ✿ Concentration of  $O_2$  in the environment.




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## Exposure Limits

### OSHA:

- ✿ 50 ppm (as an 8-hour time-weighted average).



### NIOSH:

- ✿ 35 ppm (as an 8-hour time-weighted average).




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## 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 slightly lighter than dry air (Vapor Density = 0.97).



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## Firefighter Risks

☛ SCBA extremely important in CO prevention.

☛ CO often encountered during overhaul operations.



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## CO POISONING PATHOPHYSIOLOGY



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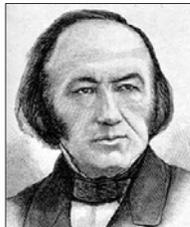
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## Pathophysiology

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



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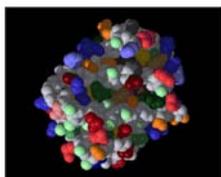
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## Pathophysiology

- 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).



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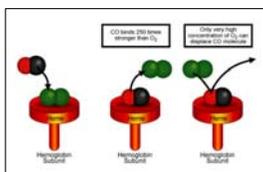
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## Pathophysiology

- CO displaces  $O_2$  from the hemoglobin binding sites.
- CO prevents  $O_2$  from binding.
- COHb does not carry  $O_2$ .
- COHb causes premature release of remaining  $O_2$  into the tissues.



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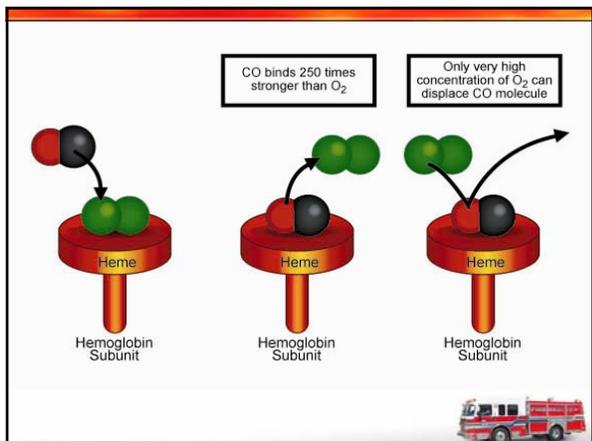
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### Pathophysiology

- ☛ COHb ultimately removed from the circulation and destroyed.
- ☛ Half-life:
  - ☛ Room air: 240-360 minutes
  - ☛ O<sub>2</sub> (100%): 80 minutes
  - ☛ Hyperbaric O<sub>2</sub>: 22 minutes



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### Pathophysiology

- ☛ CO also binds to other iron-containing proteins:
  - ☛ Myoglobin
  - ☛ Cytochrome
  - ☛ Neuroglobin
- ☛ Binding to myoglobin reduces O<sub>2</sub> available in the heart:
  - ☛ Ischemia
  - ☛ Dysrhythmias
  - ☛ Cardiac dysfunction



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### Normal COHb Levels

Source	COHb (%)
Endogenous	0.4-0.7
Tobacco Smokers:	
1 pack/day	5-6
2-3 packs/day	7-9
cigars	Up to 20
Urban Commuter	5
Methylene chloride (100 ppm for 8 hours)	3-5




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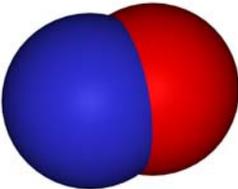
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### Pathophysiology

**Nitric oxide (NO):**

- ☛ Highly-reactive gas that participates in numerous biochemical reactions.
- ☛ Oxygen free-radical
- ☛ Levels increased with CO exposure.





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### Pathophysiology

**Nitric Oxide (NO):**

- ☛ Causes cerebral vasodilation:
  - ☛ Syncope
  - ☛ Headache
- ☛ May lead to oxidative damage to the brain:
  - ☛ Probable cause of syndrome of delayed neurologic sequelae (DNS).
  - ☛ Associated with reperfusion injury.





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## 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



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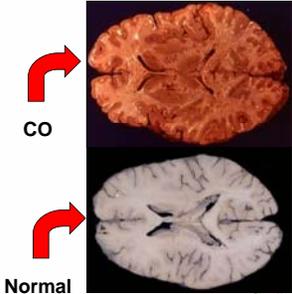
## Pathophysiology

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.



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## 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.



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## 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.



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## 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.



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## Pathophysiology Summary

- ☛ **Limits O<sub>2</sub> transport:**
  - ☛ CO more readily binds to Hb forming COHb.
- ☛ **Inhibits O<sub>2</sub> transfer:**
  - ☛ CO changes structure of Hb causing premature release of O<sub>2</sub> into the tissues.
- ☛ **Tissue inflammation:**
  - ☛ Poor perfusion initiates an inflammatory response.



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### Pathophysiology Summary

**Poor cardiac function:**

- ↓ O<sub>2</sub> 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.



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### Pathophysiology Summary

**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.



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### Patient Groups at Risk

- Children.
- Elderly.
- Persons with heart disease.
- Pregnant women.
- Patients with increased oxygen demand.
- Patients with decreased oxygen-carrying capacity (e.g., anemias, blood cancers).
- Patients with chronic respiratory insufficiency.



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# CO POISONING SIGNS & SYMPTOMS



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## CO Poisoning

- ✿ Signs and symptoms usually vague and non-specific.

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



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## CO Poisoning

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



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## Signs and Symptoms

### **Carbon Monoxide** **The** **Great Imitator†**



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



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## 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.



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## Signs and Symptoms (Acute)

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



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### 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



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### 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.



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### Signs and Symptoms (Chronic)

☛ Signs and symptoms the same as with acute CO poisoning except that onset and severity may be extremely varied.



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Cherry red skin color is not always present and, when present, is often a late finding.

**Symptoms**

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

Fatal	> 60%	Death
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CO ppm	Duration	Symptoms
50	8 hours	OSHA minimum
200	2-3 hours	Mild headache, fatigue, nausea, dizziness
400	1-2 hours	Serious headache—other symptoms intensify. Life-threatening > 3 hours
800	45 minutes	Dizziness, nausea and convulsions. Unconscious within 2 hours. Death within 2-3 hours.
1,600	20 minutes	Headache, dizziness and nausea. Death within 1 hour.
3,200	5-10 minutes	Headache, dizziness and nausea. Death within 1 hour.
6,400	1-2 minutes	Headache, dizziness and nausea. Death within 25-30 minutes.
12,800	1-3 minutes	Death




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### 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.





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## 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.




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## Delayed Neurologic Syndrome

### Signs and Symptoms:

- |                        |                            |
|------------------------|----------------------------|
| ☛ Memory loss          | ☛ Disorientation           |
| ☛ Confusion            | ☛ Hallucinations           |
| ☛ Ataxia               | ☛ Parkinsonism             |
| ☛ Seizures             | ☛ Mutism                   |
| ☛ Urinary incontinence | ☛ Cortical blindness       |
| ☛ Fecal incontinence   | ☛ Psychosis                |
| ☛ Emotional lability   | ☛ Gait disturbances        |
|                        | ☛ Other motor disturbances |




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## Long-Term Complications

### Cardiac Complications:

☛ 230 sequential patients with moderate to severe CO poisoning treated with HBO.

CO Myocardial Injury	Patients (n)	Died (%)	5-year Survival (%)
Myocardial injury from CO	85	37.6	71.6
No Myocardial injury from CO	145	15.2	88.3

Henry CR, Satran D, Lindgren B, et al. Myocardial injury and long-term mortality following moderate to severe carbon monoxide poisoning. *JAMA*. 2006;295:398-402




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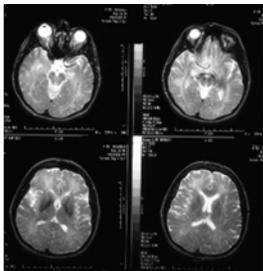
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## Long-Term Complications

- ☛ 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.



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## CO DETECTION



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## 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.



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## Carbon Monoxide Detection

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



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## Carbon Monoxide Detection

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



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## Carbon Monoxide Detection

- Hand-held devices now available to assess atmospheric levels of CO.
- Multi-gas detectors common in the fire service:
  - Combustible gasses
  - CO
  - O<sub>2</sub>
  - H<sub>2</sub>S



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## Carbon Monoxide Detection

- ✿ New generation pulse oximeter/pulse CO-oximeter can detect 4 different hemoglobin forms.
  - ✿ Deoxyhemoglobin (Hb)
  - ✿ Oxyhemoglobin (O<sub>2</sub>Hb)
  - ✿ Carboxyhemoglobin (COHb)
  - ✿ Methemoglobin (MetHb)
- ✿ Provides:
  - ✿ SpO<sub>2</sub>
  - ✿ SpCO
  - ✿ SpMet
  - ✿ Pulse rate




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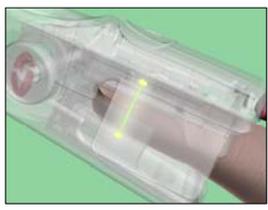
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## Pulse CO-Oximetry

- ✿ 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.




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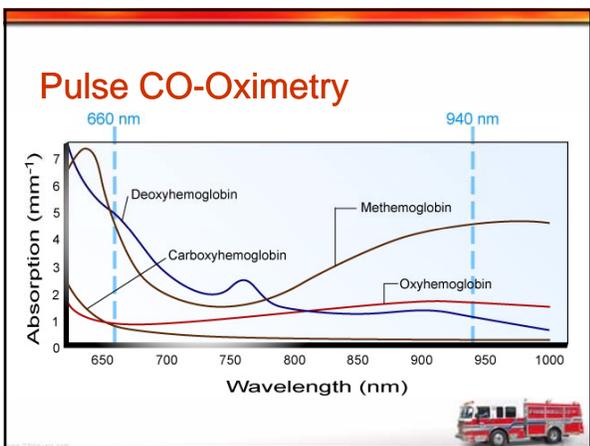
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## Pulse CO-Oximetry

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

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



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## CO POISONING TREATMENT



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## Diagnostic Criteria

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



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## 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.



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## Treatment

- ☛ Treatment is based on the severity of symptoms.

- ☛ Treatment generally indicated with COHb > 10-12%.

- ☛ Be prepared to treat complications (i.e., seizures, dysrhythmias, cardiac ischemia).



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## Treatment

- ☛ Administer high-concentration oxygen.

- ☛ Maximizes hemoglobin oxygen saturation.

- ☛ Can displace some CO from hemoglobin.

- ☛ Associated with improvements in neurological and cardiac complications.

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



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### Indications for HBO Therapy

**Strongly consider for:**

- Altered mental status.
- Coma.
- Focal neurological deficits.
- Seizures.
- Pregnancy with COHb>15%.
- History of LOC.



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### Indications for HBO Therapy

**Possibly consider for:**

- Cardiovascular compromise (e.g., ischemia, dysrhythmias).
- Metabolic acidosis.
- Extremes of age.



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### Treatment

- Continue to monitor SpO<sub>2</sub> and SpCO levels throughout treatment.
- Obtain 12-lead ECG (if ALS) and monitor ECG.
- Document findings and plot trends.



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## Treatment

- ✿ First-generation pulse oximeters may give falsely elevated SpO<sub>2</sub> levels in cases of carbon monoxide poisoning.
- ✿ Cannot distinguish between O<sub>2</sub>Hb and COHb.



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## 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.



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## METHYLENE CHLORIDE



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### 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.



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### 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).



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### Methylene Chloride Exposure

- ☼ **Treatment:**
  - ☼ No antidote for methylene chloride.
  - ☼ Support respiratory and cardiovascular functions.
  - ☼ Administer O<sub>2</sub> (O<sub>2</sub> is an antagonist of metabolically-produced carbon monoxide).



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## DOUBLE TROUBLE: CO and CYANIDE



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### 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.



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### 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.



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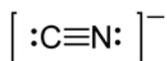
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## Chemistry of Cyanide

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




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## Cyanide

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




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## 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.




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## Pathophysiology

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




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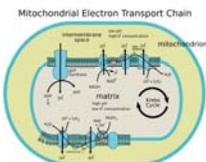
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## Pathophysiology

- ☛ Cyanide is an irreversible enzyme inhibitor:
- ☛ Cytochrome c oxidase (aa<sub>3</sub>).
- ☛ Part of the 4<sup>th</sup> complex of the electron transport chain.
- ☛ Found in the shelves (cristae) of the mitochondria in the cells.




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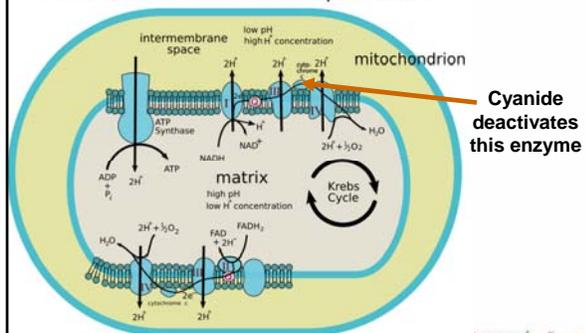
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## Mitochondrial Electron Transport Chain




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## Pathophysiology

- ✿ Cyanide stops the electron transport chain and stops energy production in the cell.
- ✿ Tissues that primarily rely 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!



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## Cyanide Treatment

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



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## Cyanide Poisoning

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



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## 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.



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## Cyanide Treatment

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



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## Cyanide Treatment

- ☼ **Hydroxocobalamin**
  - ☼ Precursor to cyanocobalamin (Vitamin B<sub>12</sub>).
  - ☼ Hydroxocobalamin combines with cyanide to form cyanocobalamin which is excreted through the kidneys.
  - ☼ FDA approval in US obtained in December 2006.
  - ☼ Marketed as Cyanokit™.



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## Cyanide Treatment

### Problems (related to nitrites):

- ☛ MetHb does not transport O<sub>2</sub>.
- ☛ The conversion of HB to MetHb changes the state of the heme molecule where O<sub>2</sub> binds.
- ☛ MetHb has heme in the ferric (Fe<sup>3+</sup>) state and not the ferrous state (Fe<sup>2+</sup>).
- ☛ O<sub>2</sub> can only bind to heme when in the Fe<sup>2+</sup> state.




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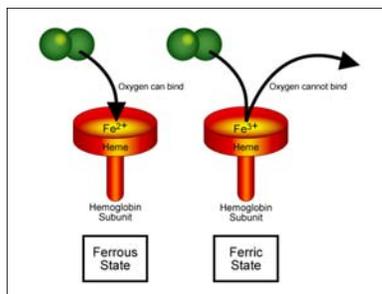
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## Cyanide Treatment




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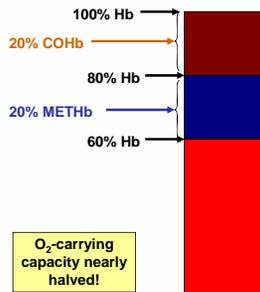
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## Cyanide Treatment

- ☛ Concomitant CO and cyanide poisoning can significantly decrease the O<sub>2</sub>-carrying capacity of the blood.
- ☛ Combination of COHb and MetHB can significantly reduce the O<sub>2</sub>-carrying capacity of the blood.




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## Cyanide Treatment

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



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## CO and Cyanide

- Parts of cyanide antidote (sodium nitrite) induce methemoglobinemia.
- Cyanide antidotes and sodium nitrite increase elevated COHb and MCHC and O<sub>2</sub> capacity of blood.
- Sodium nitrite should be used in combination with cyanide/CO antidotes if COHb >10%.
- Hydroxocobalamin converts cyanide to cyanocobalamin (Vitamin B<sub>12</sub>) which is renally cleared.

Hydroxocobalamin is the cyanide antidote of choice for mixed cyanide and CO poisonings.



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## Financial Disclosure



This program was prepared with an unrestricted grant from Masimo. Masimo did not control content.



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## Credits

- \* **Content:** Bryan Bledsoe, DO, FACEP
- \* **Art:** Robyn Dickson (Wolfblue Design)
- \* **Power Point Template:** Code 3 Visual Designs
- \* **The following companies allowed use of their images for this presentation:**
  - \* Brady/Pearson Education
  - \* Scripps/University of California/San Diego
  - \* JEMS/Brook Wainwright
  - \* Glen Ellman
  - \* Bryan Bledsoe, DO, FACEP
  - \* Masimo.



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