#### Carbon Monoxide Poisoning Dr. Simin Najafgholian Assistant Professor of Emergency Medicine

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# **Objectives**

Upon successful completion of this module, you will be able to:

- 1. Identify physical characteristics of CO
- 2. Identify sources of CO
- 3. Identify statistics of CO incidents
- 4. Identify pathophysiology of CO poisoning
- 5. Identify CO effects on hemoglobin
- 6. Identify CO effects on major body systems
- 7. Identify CO exposures and limits (ppm)
- 8. Discuss importance of being able to monitor CO levels for patients

# **Objectives cont'd**

- 9. Identify signs/symptoms of CO poisoning and smoke inhalation
- 10. Identify the treatment of CO poisoning and smoke inhalation
- 11. Identify possible long term effects of CO poisoning
- 12. Identify the difference between acute and chronic CO poisoning

# **Carbon Monoxide (CO)**

- An odorless, colorless, tasteless gas
- Results from incomplete combustion of carboncontaining fuels
  - Gasoline, wood, coal, natural gas, propane, oil, and methane
- Affects 40 50,000 Americans annually who need to seek care
- Kills an additional 6,000 persons annually in the USA
- CO is the #1 cause of poisoning in industrialized countries

# Sources of Carbon Monoxide – any combustible item

- Homes
- Cigarette smoke
- House fires
- Automobile exhaust fumes
- Worksites
  - Including fumes from propane-powered equipment like forklifts
- Commercial structures
- Smoke from charcoal-fired cook stoves & ovens

# Sources cont'd

#### Heat provided to homes

- Gas-fueled heaters
- Wood burning stoves
- Indoor stoves
- Camp stoves
- Gas-powered generators
- Recreational environments
- Recreational vehicles
- Boat exhaust fumes

# **Carbon Monoxide Incidents**

- Peak time of day 1800 2159
- Overall, 75% of non-fire CO incidents are reported between 0900 and 2259
- Peak months are December and January for non-fire CO incidents
- Almost 9/10 (89%) of non-fire CO incidents took place in the home

 Source: Non-Fire Carbon Monoxide Incidents Reported in 2005; NFPA Fact Sheet

# What Effect Does Carbon Monoxide Have on Hemoglobin?

- Hemoglobin molecules each contain four oxygen binding sites
- Carbon monoxide binds to hemoglobin
- This binding reduces the ability of blood to carry oxygen to organs
- Hemoglobin occupied by CO is called carboxyhemoglobin
- Body systems most affected are the cardiovascular and central nervous systems

# **Effects of Carbon Monoxide**

- Oxygen cannot be transported because the CO binds more readily to hemoglobin (Hgb) displacing oxygen and forming carboxyhemoglobin
- Premature release of O<sub>2</sub> prior to reaching distal tissue leads to hypoxia at the cellular level
- Inflammatory response is initiated due to poor and inadequate tissue perfusion
- Myocardial depression from CO exposure
  - Dysrhythmias, myocardial ischemia, MI
- Vasodilation from increased release of nitric oxide; worsening tissue perfusion and leading to syncope

## Half-life of Carbon Monoxide

- Half-life time required for half the quantity of a drug or other substance to be metabolized or eliminated
- CO half-life on 21% room air  $O_2 4 6$  hours
- CO half-life on 100%  $O_2 80$  minutes
- CO half-life with hyperbaric  $O_2 22$  minutes

# **CO Levels**

- Fresh air
- Urban air
- Smoke filled room
- Cooking on gas stove
- Actively smoking cigarette
- Automobile exhaust

0.06 - 0.5 ppm

- 1 300 ppm
- 2 16 ppm

100 ppm

400 – 500 ppm

100,000 ppm

# Expected Carboxyhemoglobin Levels

- Non-smokers 5%
- Smokers up to 10%
  - 5 6% for a 1 pack per day smoker
  - 7 9% for a 2-3 pack per day smoker
  - Up to 20% reported for cigar smokers
- Urban commuter 5%

# **CO** Poisoning

- Symptoms are often vague, subtle, and non-specific; can easily be confused with other medical conditions;
  - Flu nausea, headaches
  - Food poisoning nausea
  - Cardiac and respiratory conditions shortness of breath, nausea, dizziness, lightheadedness
- CO enters the body via the respiratory system
- Poisoning by small amounts over longer periods of time or larger amounts over shorter time periods





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LOSS OF CONSCIOUSNESS

# **Carbon Monoxide Absorption**

- Dependent upon:
  - Minute ventilation
    - Amount of air exchanged in the lungs within one minute
  - Duration of exposure
    - The longer the exposure, the more the absorption
  - Concentration of CO in the environment
    - The higher the concentration, the greater the toxicity
  - Concentration of O<sub>2</sub> in the environment
    - The lower the O<sub>2</sub> concentration to begin with, the faster the symptoms will develop
      - higher altitudes
      - closed spaces

# **Assessment for CO Exposure**

- EMS may be summoned to monitor the air quality for the presence of carbon monoxide
  - Airborne CO meters are used and documentation made whether there is a patient transport or not
- A more immediate concern is the level of CO in the patient's blood
  - RAD 57 monitors are a non-invasive tool that allows results in less than 30 seconds
  - Rapid diagnosis leads to rapid and appropriate treatment

# **RAD 57 Device**

- Used like a pulse ox
- Non-invasive tool
- Readings within seconds
- Helps to quickly hone in a diagnosis
- Used in ED and in the field



## **Masimo Rad-57 Guidelines**

- The following are broad guidelines
- Treat the patient
- SpCO level readings
  - SpCO levels <5%</p>
    - Normal in non-smokers; no treatment
  - SpCO levels <u>></u>5%
    - •5-10% normal in smokers
    - In non-smokers, treat with 100% O<sub>2</sub>
    - EMS should be transported for further evaluation

# CO Levels with Related Signs and Symptoms

- <u>></u>5% mild headache
- 6-10% mild headache, SOB with exertion
- 11-20% moderate headache, SOB
- 21-30% worsening headache, nausea, dizziness, fatigue
- 31-40% severe headache, vomiting, vertigo, altered judgment
- 41-50% confusion, syncope, tachycardia
- 51 60% seizures, shock, apnea, coma

 Carboxyhemoglobin levels of <15 – 20%</li> ✓ Mild severity Headache – mild to moderate Shortness of breath Nausea and vomiting Dizziness Blurred vision

- Carboxyhemoglobin levels of 21 40%
  - Moderate severity
    - Worsening headache
    - Confusion
    - Syncope
    - Chest pain
    - Dyspnea
    - Tachycardia
    - Tachypnea
    - Weakness

- Carboxyhemoglobin levels of 41 59%
  - ✓ Severe
    - Dysrhythmias, palpitations
    - Hypotension
    - Cardiac ischemia
    - Confusion
    - Respiratory arrest
    - Pulmonary edema
    - Seizures
    - Coma
    - Cardiac arrest

Carboxyhemoglobin levels of >60%
 ✓ Fatal
 Death

Cherry red skin is not listed as a sign
 An unreliable finding

## **Increased Risks**

- Health and activity levels can increase the risk of signs and symptoms at lower concentrations of CO
  - Infants
  - Women who are pregnant
    - Fetus at greatest risk because fetal hemoglobin has a greater affinity for oxygen and CO compared to adult hemoglobin
  - Elderly
  - Physical conditions that limit the body's ability to use oxygen
    - Emphysema, asthma
    - Heart disease
  - Physical conditions with decreased O<sub>2</sub> carrying capacity
    - Anemia iron-deficiency & sickle cell

#### **Risks to Firefighters from CO Exposure**

- On the job from repeated exposures
  - Structure fires
  - Apparatus fumes
  - Portable equipment fumes
  - Gasoline powered saws
  - Generators
- Premature removal of SCBA equipment increases the risk of exposure

# **CO Identification**

- Sooner the suspicion the sooner the appropriate treatment can be initiated
- Complications to monitor
  - Seizures
  - Cardiac dysrhythmias
  - Cardiac ischemia

# **CDC Diagnostic Criteria**

- Suspected CO exposure
  - Potentially exposed person but no credible threat exists
- Probable CO exposure
  - Clinically compatible case where credible threat exists
- Confirmed CO exposure
  - Clinically compatible case where biological tests have confirmed exposure

## **Patient Assessment**

- Continuously monitor SpO<sub>2</sub> and SpCO levels
  Demonstrate SpO<sub>2</sub> may be follooly normal
  - Remember that SpO<sub>2</sub> may be falsely normal
  - If EMS has used a CO-oximeter, findings to be reported to the ED staff

 Generally, results >3% indicate suspicion for CO exposure in non-smoker

- Cardiac monitor
- 12 lead EKG obtained and transmitted to ED

# **Pulse Oximetry**

- Device to analyze infrared signals
- Measures the percentage of oxygenated hemoglobin (saturated Hgb)
- Can mistake carboxyhemoglobin for oxyhemoglobin and give a false normal level of oxyhemoglobin
- Never rely just on the pulse oximetry reading; always correlate with clinical assessment

#### **Pulse CO-oximeter Device**



- Hand-held device
- Attaches to a finger tip similar to pulse ox device
- Most commonly measured gases in commercial devices include
  - Carbon monoxide (SpCO)
  - Oxygen (SpO<sub>2</sub>)
  - Methemoglobin (SpMet)
  - Other combustible gases
- Without the device, need to draw a venous sample of blood to test for CO levels

# **Pulse CO-oximeter Tool**

- Firefighters have an increased exposure risk
  - Active firefighting
  - Inhaled products of combustion in structure fire
  - Inhaled exhaust from vehicles and power tools

## **Treatment CO Poisoning**

- Increasing the concentration of inhaled oxygen can help minimize the binding of CO to hemoglobin
- Some CO may be displaced from hemoglobin when the patient increases their inhaled oxygen concentrations
- Treatment begins with high index of suspicion and removal to a safer environment
- Immediately begin 100% O<sub>2</sub> delivery

# **Treatment CO Poisoning**

- Guidelines from different sources may vary when to initiate treatment based on SpCO levels
  - Report levels to the ED MD
    - Remember >5% in non-smokers is abnormal
- Treatment levels vary significantly
  - If you do not have a CO-oximeter to use, maintain a heightened level of suspicion and base treatment on symptoms
- Monitor for complications
  - Seizures
  - Cardiac dysrhythmias
  - Cardiac ischemia

# Long Term Effects CO Exposure

- Hypoxemia follows CO exposure
- Effects of hypoxemia from CO exposure is dependent on presence of underlying diseases
- Hypoxemia can cause the formation of free radicals – dangerous chemicals

# Long Term Cardiovascular Effects

- Myocardial injury from hypoxia and cellular damage
  - Pump failure
  - Cardiac ischemia
  - Later development cardiovascular complications
  - Premature death especially if myocardial damage at the time of initial exposure
- Factors increasing myocardial injury risk
  - Male gender
  - History hypertension
  - GCS <14 when patient first found

# Long Term Neurological Effects

- Effects are primarily affective (mood) and cognitive (thought)
  - Increased depression and anxiety regardless if exposure accidental or suicidal attempt
  - Phenomenon called delayed neurological syndrome (1 - 47% of cases)
    - More likely if there was a loss of consciousness
    - Behavioral and neurological deterioration
    - Memory loss, confusion, ataxia, seizures, urinary & fecal incontinence, emotional lability, disorientation, hallucinations, mutism, cortical blindness, psychosis, gait disturbances, Parkinsonism

