Carbon monoxide poisoning
Pediatric Emergency Care
Chang Gung Children Hospital
Jan 2010

Introduction

- Carbon monoxide (CO) intoxication is one of the most common causes of accidental and intentional poisoning
- Atmospheric composition <0.001%
- Blood carboxyhaemoglobin
  - Nonsmokers 1-3%
  - Smokers 10-15%
- Sources of CO
  - Motor vehicle exhaust fumes
  - Heating systems
  - Inhaled smoke
  - Propane-powered forklift trucks
  - Methylene chloride

Pathophysiology

- CO is colourless, odourless, nonirritant toxic gas
- CO toxicity due to
  - Cellular hypoxia
  - Direct cellular injury
- Cellular hypoxia
  - CO competes with O2 for binding to Hb
  - Affinity of Hb for CO x 200-250 > affinity for O2
  - O2-Hb dissociation curve shift to the left
  - Impaired tissue release of O2 and cellular hypoxia
Pathophysiology

- Direct cellular injury
  - CNS reoxygenation injury
  - Lipid peroxxygenation
  - Free radical formation
- CO toxicity in pregnancy
  - Risk of fetal injury

Delayed neuropsychiatric syndrome

- Incidence 10 - 30% of victims 3 - 240 days after exposure
  - Cognitive changes
  - Personality changes
  - Parkinsonism
  - Dementia
  - Psychosis
- Recovery 50 - 75% within 12 months

**Table 1. Acute Symptoms Reported by 156 Patients after Exposure to Carbon Monoxide.**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Percentage of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>91</td>
</tr>
<tr>
<td>Dizziness</td>
<td>77</td>
</tr>
<tr>
<td>Weakness</td>
<td>53</td>
</tr>
<tr>
<td>Nausea</td>
<td>47</td>
</tr>
<tr>
<td>Difficulty in concentrating or confusion</td>
<td>63</td>
</tr>
<tr>
<td>Shortness of breath</td>
<td>40</td>
</tr>
<tr>
<td>Visual changes</td>
<td>25</td>
</tr>
<tr>
<td>Chest pain</td>
<td>9</td>
</tr>
<tr>
<td>Loss of consciousness</td>
<td>6</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>5</td>
</tr>
<tr>
<td>Muscle cramping</td>
<td>5</td>
</tr>
</tbody>
</table>

(Data are from Levy et al., Myers et al., and Burany et al.)

**Figure 2. (a) Mortality in acute carbon monoxide poisoning as a function of initial carboxyhemoglobin (COHb) level. (b) Mortality in acute carbon monoxide poisoning as a function of initial arterial pH.**

**Table 1. Demographic and clinical characteristics of individuals who died of acute carbon monoxide (CO) poisoning, as compared with survivors.**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Neornicides (n = 30)</th>
<th>Survivors (n = 145)</th>
<th>p</th>
<th>Univariate Analysis</th>
<th>Logistic Regression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>55%</td>
<td>66%</td>
<td>0.12</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Male</td>
<td>55%</td>
<td>66%</td>
<td>0.12</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Intent of poisoning</td>
<td>79%</td>
<td>79%</td>
<td>3.0000</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Intentional</td>
<td>79%</td>
<td>79%</td>
<td>3.0000</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Source of CO</td>
<td>32%</td>
<td>11%</td>
<td>&lt;0.0001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Fire</td>
<td>62%</td>
<td>96%</td>
<td>0.0001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Neutres</td>
<td>84%</td>
<td>60%</td>
<td>&lt;0.0001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Loss of consciousness</td>
<td>100%</td>
<td>82%</td>
<td>&lt;0.0001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>COHb%</td>
<td>31.1 ± 13.1</td>
<td>22.4 ± 11.2</td>
<td>&lt;0.0001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Initial arterial pH</td>
<td>7.20 ± 0.12</td>
<td>7.30 ± 0.10</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>0.894</td>
</tr>
</tbody>
</table>
Apoptotic and necrotic brain lesions in a fatal case in carbon monoxide poisoning

### Diagnosis
- High level of clinical suspicion
- Serum COHb level
- Exhaled breath COHb level
- Measured by spectrophotometry
- Pulse oximetry cannot distinguish between HbO2 and COHb
- Comprehensive neurological and neuropsychological assessment
- CO Neuropsychological Screening Battery
- CT brain to exclude other conditions

### Treatment
- High-flow, FiO2 ~100%, normobaric O2
- O2 shortens the half life of COHb
  - 21% O2 = 4-6 hours
  - 100% O2 = 40-80 minutes
  - 100% O2 2.5 atm = 15-30 minutes
- Continue O2 until COHb normal
- Beware concomitant smoke inhalation and burn injury
- Normobaric vs Hyperbaric O2 therapy
  - HBO hastens resolution of acute symptoms
  - Unclear evidence for effect of HBO on late complications and mortality

### Table 2. Suggested Indications for Hyperbaric-Oxygen Therapy in Patients with Carbon Monoxide Poisoning

<table>
<thead>
<tr>
<th>Condition</th>
<th>Treatment Options</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any period of unconsciousness</td>
<td>Hyperbaric oxygen</td>
<td></td>
</tr>
<tr>
<td>Any abnormal scan on the Carboxyhemoglobin</td>
<td>Hyperbaric oxygen</td>
<td></td>
</tr>
<tr>
<td>Neuroradiologic Screening Battery</td>
<td>Hyperbaric oxygen</td>
<td></td>
</tr>
<tr>
<td>Carboxyhemoglobin level ≥40%</td>
<td>Hyperbaric oxygen</td>
<td></td>
</tr>
<tr>
<td>Pregnancy and carboxyhemoglobin level ≥15%</td>
<td>Hyperbaric oxygen</td>
<td></td>
</tr>
<tr>
<td>Signs of cardiac ischemia or arrhythmia</td>
<td>Hyperbaric oxygen</td>
<td></td>
</tr>
<tr>
<td>History of ischemic heart disease and carboxyhemoglobin level ≥20%</td>
<td>Hyperbaric oxygen</td>
<td></td>
</tr>
<tr>
<td>Recurrent symptoms for up to 1 wk</td>
<td>Hyperbaric oxygen</td>
<td></td>
</tr>
<tr>
<td>Symptoms that do not resolve with normobaric oxygen after 4-6 hr</td>
<td>Hyperbaric oxygen</td>
<td></td>
</tr>
</tbody>
</table>

*Data are from Myers and Thoms.*

### Graphs and Other Tables

**Analysis H.2:** Comparison of Hyperbaric Oxygen (HBO) vs. Normobaric Oxygen (NBO) on the Presence of Symptoms or Signs at Time of Primary Admission (48 Hours)

**Conclusions:** Three hyperbaric-oxygen treatments within a 24-hour period appeared to reduce the risk of cognitive sequelae 6 weeks and 12 months after acute carbon monoxide poisoning. (N Engl J Med 2002; 347:1057-67)

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Overview of carbon monoxide poisoning

- **History**
  - Duration and mechanism of exposure
  - Assess for major symptoms: loss of consciousness, confusion, symptoms consistent with hypoxia (chest pain)
  - Assess for minor symptoms: headache, nausea/vomiting
  - Assess pregnancy status
- **Infant and toddlers**
  - Signs may be more subtle and non-specific than adults. Fussiness and feeding difficulty

Evaluation

- **Physical examination**
  - Careful evaluation of mental status
  - Physical examination usually unremarkable
- **Diagnostic evaluation**
  - Check CO level via co-oximetry of arterial or venous blood.
  - Check acid-base status blood gas
  - Check ECG in all patients, check cardiac enzymes in younger patients with chest pain or symptoms suggestive of ischemia
  - Consider CNS imaging in patients with altered mental status to rule out other etiologies
  - Standard pulse oximetry cannot screen for CO exposure

Treatment

- **Secure Airway, Breathing, and Circulation**
- **Intubates as clinically indicated**
- **Apply high-flow oxygen** to all CO poisoned patients regardless of pulse oximetry or arterial pO2
- **Direct fire department to assess for environmental exposure and remove victims**

Hyperbaric oxygen (HBO)

- **CO level>25 percent**
  - (>20 percent if pregnant)
- **Loss of consciousness**
- **Severe metabolic acidosis (PH< 7.1)**
- **Concern for end-organ ischemia (chest pain, ECG changes, altered mental status)**
- **Caution to patients with unpalliated ductal dependent cardiac lesion**

Algorithm for using normobaric and hyperbaric oxygen following carbon monoxide exposure

- **Potential CO exposure victims**
- **Evaluate for hypoxemia or hypoxemic respiratory failure**
- **Check co-oximetry for arterial or venous blood**
- **Consider CNS imaging**
- **High-flow oxygen**
- **Intubate if clinically indicated**
- **Hyperbaric oxygen if necessary**
- **100 percent FiO2 therapy with CO-oximetry and pulse oximetry**