Carbon monoxide
Sources of carbon monoxide exposure
Mechanism of action

Tissue hypoxia

Binds to myoglobin and cytochrome oxidase
Shift oxyhaemoglobin dissociation curve to left
Accelerate cellular death by apoptosis
Factors affecting COHB saturation

- Fetus (lower arterial oxygen level)
- Infants and other with high metabolic rate
- Anemic (low oxygen reserve)
- Arteriosclerotic heart disease
- Respiratory insufficiency
- Debilitating conditions
- Old age
Clinical features

Level of COHB can be dissimilar in individuals simultaneously exposed to the same source
<table>
<thead>
<tr>
<th>Percentage</th>
<th>Clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>10%</td>
<td>no appreciable symptoms</td>
</tr>
<tr>
<td>10-20%</td>
<td>Shortness of breath, mild headache, flushed skin</td>
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<tr>
<td>20-30%</td>
<td>Throbbing headache, muscular weakness and incoordination</td>
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<tr>
<td>30-40%</td>
<td>Nausea, vomiting collapse, impaired judgment, dim vision</td>
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<tr>
<td>40-50%</td>
<td>Loss of memory, palpitation and dyspnea</td>
</tr>
<tr>
<td>50-70%</td>
<td>Intermittent convulsion, respiratory paralysis and death</td>
</tr>
<tr>
<td>70%</td>
<td>Rapidly fatal due to respiratory arrest</td>
</tr>
</tbody>
</table>
Delayed effects

- Serious neurological symptoms
  - Delayed neuropathy
  - Demyelination
  - Twitching
  - Visual hearing impairment
  - Seizure
  - Parkinson's dyskinesia

- Skin lesions
  - Erythema, blisters
Chronic low levels cause
- tiredness
- lethargy
- shortness of breath
- irritability
- visual impairment
- Kidney damage

Heart disease and atherosclerosis
Diagnostic test

- COHB level in blood
- ECG
- Chest Radiograph
- CT Scan
- MRI
DIFFERENTIAL DIAGNOSIS

Flu-like viral syndromes

Depression

Chronic fatigue syndrome

Chest pain and migraine or other headaches

Acute respiratory distress syndrome

Altitude sickness

Meningitis
treatment
   ➢ Removal from source

   ➢ 100% oxygen

   ➢ Normobaric oxygen
   ➢ Hyperbaric oxygen

Oxygen delivered at 3x atm pressure
Half life of CO reduced to 23 mins
Indication for hyperbaric oxygen

- Loss of consciousness
- COHB above 40%
- Myocardial infarction
- Metabolic acidosis
- End organ dysfunction
Postmortem findings

- Cherry pink discoloration
- Blistering of dependent areas of body
- Blood and muscles will be pink
- Pulmonary edema
- Focal necrosis in myocardium
Postmortem changes in brain
Carbon dioxide
Properties

- Heavy
- Odorless
- Colorless
- Slightly acidic taste
- In normal air present to extent of 0.04%
Carbon dioxide

• Produced from complete combustion of carbon containing compounds.
• Formed during fermentation reactions and decomposition of organic matter.
• Other sources of exposure are refrigerating plants, manholes, wells, cellars, and rebreathing in close spaces.
Mode of action

• It produces o2 lack and tissue anoxia.
• Pure co2 may cause vagal inhibitory response with spasm of glottis and instant death
Symptoms

Vary with conc. Of gas

20-30%

40% conc.

AT 50% conc.
FATAL DOSE
Treatment
Autopsy findings

• Marked cyanosis
• Deep congestion
• Dilatation of pupils
• Petechial hemorrhages
• Sometimes froth
• Dark fluid blood
• Left side of heart is empty.
Medico legal importance
Hydrogen sulphide
• Byproduct of organic decomposition
• Petroleum industry
• Tanning
• Present in coal pits, gas wells
• Serious toxicity occurs in poorly ventilated spaces
• Rapidly absorbs from lungs

• Detoxification occurs rapidly
  • with the red blood cells and liver mitochondria.

• Irritant to mucous membrane
Pathophysiology

• Intracellular toxin
• Inhibits cytochrome oxidase and disrupt electron transport
• Anaerobic metabolism causes lactate accumulation and metabolic acidosis
• Low doses
  • Mucous membrane and respiratory irritant
• High doses
  • Direct respiratory depression
Clinical presentation

• **Acute exposure**
  • Irritant to eyes and respiratory tract

• **Short exposure of high concentration**
  • Knockdown effect
  • Characterize by sudden, brief loss of consciousness followed by complete recovery
• Central nervous system effects
  • Headache, lethargy, agitation, weakness, seizure and coma
  • Rapid loss of consciousness and respiratory paralysis occur at 1000 ppm

• Cardiac effects
  • Dysrhythmias, bradycardia, myocardial depression conduction defects

• Gastrointestinal effects
• Respiratory effects
  • Bronchitis and pneumonitis, cough, hoarsness and pulmonary edema
• Ocular effects
  • Conjunctivitis, eye pain, lacrimation, photophobia, keratoconjunctivitis (gas eye)
• Olfactory effects
  • Olfactory fatigue
Delayed and chronic complication

- Interstitial pulmonary fibrosis
- Chronic olfactory deficit
- Neuropsychiatric disorders
- Weight loss
Postmortem findings
Diagnostic test

• Whole blood sulphide level

• Thiosulphate is a metabolite of sulfide can be detected in the blood and urine

OTHER TEST

• Chest radiograph
• Arterial blood gases
• CT scans
Treatment

• Removal from source
• Self contained breathing apparatus
• Adequate ventilation and circulation
• **Antidote**
  • Induction of methaemoglobinemia and treatment with HBO
  • Amyl nitrite inhaled for 30 sec
  • Sodium nitrite 300 mg (10ml of 3% soln) I/V over 4 mins.
  • Thiosulfate is not required b/c body spontaneously detoxify sulfmethemoglobin.
Postmortem findings

• Visceral congestion
• Scattered petechial
• Hemorrhagic pulmonary edema
• Greenish discoloration of gray matter, viscera and bronchial secretions
WAR GASES
War gases

- Lacrimators or tear gases
- Lung irritants or choking gases
- Vesicant or blister gases
- Nasal irritants or vomiting gases
- Nerve gases
- Miscellaneous
Lacrimators

• Tear gas
• Chloracetophenone (C.A.P)
  • Finely divided powder
• Bromobenzyl cyanide (B.B.C)
  • Heavy, oily dark brown liquid
Lung irritants

• Chlorine
• Phosgene
Vesicants

• Blister gas
• Discharge from artillery shells
• Mustard gas
• Lewisite

• After absorption it causes hemolysis of red cells and produce signs of arsenic poisoning
Nerve gases