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 of 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

Percentage	Clinical features
10%	no appreciable symptoms
10-20%	Shortness of breath, mild headache, flushed skin
20-30%	Throbbing headache ,muscular weakness and incoordination
30-40%	Nausea, vomiting collapse, impaired judgment ,dim vision
40-50%	Loss of memory ,palpitation and dyspnea
50-70%	Intermittent convulsion ,respiratory paralysis and death
70%	Rapidly fatal due to respiratory arrest

Delayed effects

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

  - **Skin lesions**
  - Erythema, blisters

Chronic

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 23mins

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,keratoconjectivitis (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