Industrial Toxicology

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Industrial toxicology

Inorganics chemicals – metals:

- Lead (Pb)
- Mercury (Hg)
- Arsenic (As)
- Cd, Cr, Mn, V, P – professional poisoning are rare

Chemical asphyxiants:

- Carbon monoxide (CO)
- Hydrogen cyanide (HCN)
- Hydrogen sulpide (H2S)

Lead (Pb)

- The **inorganics forms of lead** (mainly as the sulphide PbS) have tha same action in the body. **Organics leads** compounds, primarily tetraethyl – and tetramethyl – forms, act similary to each other, but differently from inorganic salts.
- Uses: pipes, sheet metal, foil, ammnunition, pigments, anti-knock additive to petrol (organic compounds only).
- Metabolism: poorly absorbed through the gut (10%), but dependent on calcium and iron in the diet. Pulmonary absorption is more efective. Transported in a form bound to red cell membrane and mainly store in the hone. Excretion mainly urinary. The half-live is long (5-10 ears)

Lead (Pb)

Lead interferes with haem synthesis by preventing the conversion of delta aminolevulinic acid (ALA) to porphobilinogen and incorporation of iron into protoporphyrin IX to form haem.

Health effects:

inorganic form:

- Acute effects: non specific with lassitude, abdominal cramps and constipation,myalgia and anorexia, encephalopathy, acute renal failure.
- Chronic effects: peripheral motor neuropathy(espec. wrist drop) and anemia are the main late manifestations.

Organic form: differs with inorganics effcts – in associated with psychiatric manifestation (insomnia, hyperexcitability, mania).

Leads (Pb)

Diagnostic laboratory tests:

- anemia normochromic, reticulocytosis, blood-lead, elevation in erytrocyte protoporphyrin, urinary d-ALA, or urinary coproporphyrin.

Treatment:

If necessary, **calcium EDTA** or **penicilamine** can be given.

The latter can be administered orally.

Organic lead poisoning does not respond to such **chelation therapy.**

Mercury (Hg)

- Uses: sctientific instruments, amalgams, silvering, solders, pharmaceuticals, paints, explosives.

Salts **Hg** are rapidly absorbed by all routes:
inhalation, ingestion, skin contact.

Inorganic salts **Hg** are more readibily absorbed throught the gut and excreted by the kidneys than organics.

Mercury (Hg)

- *Acute exposure **Hg**:* rare in industry, is characterised by febrile illnes with pneumonitis. If severe, it can cause oliguric renal failure.

- *Chronic exposure **Hg**:* slow onset with peculiar neuropsychiatric disorder (erethism), with features of anxiety neurosis, timidity and paranoia. Accompanied by gingivitis, excessive salivation, intention tremor, dermatographia, scanning speech. Upper motor neuron lesion and visual field constriction are more commonly associated with organics mercurialism.

Mercury (Hg)

- *Biological monitoring:* **mercury** in urine or blood.
- Treatment: **BAL, penicillamine**
- Prognosis: for patients with organics poisoning (methyl or ethyl mecury) is poor, often fatal.

Arsenic (As)

- It is a **by-product** of both ferrous and non ferrous smelting.
- **Arsin** (AsH3) is a gas - the most toxic form of arsenic. Arsenic is **general protoplasmic poison**.
- Uses: alloys, insecticides, fungicides, rhodenticides, pigments, decolorizer in glass and paper-making.

- *Acute effects **As**:* severe respiratory irritation, nausea, vomiting, diarrhea, abdominal pain, hemolysis, oliguria, shock.
- *Chronic effects **As**:* gastrointestinal symptoms, encephalopathy, peripheral neuropathy - mainly sensory, hyperkeratosis and hyperpigmentation, liver damage, carcinogenic changes in skin and lungs.

Arsenic (As)

- **Arsenic** levels in urine, hair and nails may be useful in the detection: of systematic absorption of arsenic.

- Therapy: specific chelator BAL i.m., non-specific for the skin and respiratory disturbances.

- Professional poissoning of other inorganic chemicals as **Cadmium (Cd), Chromium (Cr), Manganese (Mn), Vanadium (V), Phosphorus (P)** – are rare.

Chemical asphyxiants

The mechanism by with chemical asphyxiants cause their toxic effects is **producing tissue hypoxia**.

Carbon monoxide (CO)

Uses: by-products of mining, smelting, petrochemical processes and many processes involving combustion.

Metabolism: toxic effect CO - producing tissue hypoxia.

CO reversibly combines with haemoglobin to produce **carboxyhaemoglobin (COHb).**

CO also binds to muscle **myoglobin** and to intracellulare **cytochome oxidases**.

Carbon monoxide (CO)

*Acute **CO** poisoning*: typically, individuals with

- COHb lewels below 1% are asymptomatic, and even
- COHb lewels between 10-30% produce effects that are sometimes nondesciptive –hedeache, faitness, nausea, vomiting. Increased respiratory rate. Increased heart rate.
- COHb 30-40%: as above, plus dimness of vision, decreased blood preassure, musculare incoordination, cherry red skin discoloration.

Carbon monoxide (CO)

- COHb 40-60% aa above, plus generalized weakness, mental confusion
- COHb 60% and higer: coma, intermittens convulsions, depressed heart action and respiratory rate, and possibly death.
- COHb over 90%: death within a few minut.
- *Chronic **CO** poissoning:* headache, organic brain damage if asphyxiation was prolonged.

Carbon monoxide (CO)

- *Biological monitoring:* **COHb levels.**

Treatment **CO** poisoning:

- remove from exposure and give **pure or hyperbaric oxygen**. Cerebral edema may result from central hypoxia. **Diuretics** and **glucocorticoids** may be appropriate to prevent its apperance or reduce its severity.

Hydrogen cyanide (HCN)

- **Hydrogen cyanide** and its derivates

are used in electroplating, metallurgy and extraction gold and siver metals from ores, production of syntetic fibres and plastics, and as fumigand and fertilizer.

Metabolism: inhibits the action of **cytochrome oxidase**, thus disrupting oxygenation at the tissue cell level.

Hydrogen cyanide (HCN)

Acute poisoning ***HCN***:

can ocure from inhalation and also absorption through the skin, with rapid onset of hedeache, hypopnoea, tachykardia, hypotension, convulsion and death.

Chronic poisoning ***HCN***: none

Hydrogen cyanide (HCN)

- *Biological monitoring:* blood cyanid concentration.

- Treatement: remove contaminated clothing and wash the skin. Administer **amylnitrite** inhalation, 3% **sodium nitrite** i.v., and 25% **sodium thiosulphate** solution i.v.

  Dicobalt EDTA i.v. is advocated for the uncouscious pacient, with a definitive history of a cyanide exposure, dispatche the pacient immediately to hospital.

Hydrogen sulphide (H2S)

Metabolism: it inhibits cytochrom oxidase (cf HCN) and causes increase in **sulphmethaemoglobin**.

*Acute poissoning **H2S**:* lacrimation, photophobia and mucous membrane irritation in low concentation. In high concentration pneumonitis, paralysis of the respiratory centre can cause sudden unconscionsness.

*Chronic poissoning**H2S**:* keratitis, skin vesicles.

Treatement: removal from exposure, administer **oxygen** and **amyl or sodium nitrite**. Other therapy is symptomatic.