INDUSTRIAL TOXICOLOGY

A. INORGANIC CHEMICALS - METALS

Lead (Pb)

The inorganic forms of lead (mainly as the sulphide PbS) have the same action in the body. Organic lead compounds, primarily tetraethyl- and tetramethyl- forms (TEL, TML), act similarly to each other, but differently from inorganic salts.
***Uses*: Pipes, sheet metal, foil, ammunition, pigments, anti-knock additive to petrol (organic compounds only).**

***Metabolism*: Poorly absorbed through the gut (10%) but dependent on calcium and iron content in the diet. Pulmonary absorption is more effective. Transported in a form bound to red cell membrane and mainly stored in the hone. Excretion mainly urinary. The half-life is long (5-10 years).**

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 (especially wrist drop) and anemia are the main late manifestations.

Organic form - differs from inorganic effects –

is associated with psychiatric manifestation (insomnia, hyperexcitability, mania).

Diagnostic laboratory tests:

anemia normochromic, and reticulocytosis. Blood-lead, elevation in erythrocyte protoporphyrin, urinary d-ALA, or urinary coproporphyrin.

Treatment:

If necessary, calcium EDTA or penicillamine can be given. The latter can be administered orally. Organic lead poisoning does not respond to such chelation therapy.

Mercury (Hg)

***Uses*: scientific instruments, amalgams, silvering, solders, pharmaceuticals, paints, explosives.**

Salts are rapidly absorbed by all routes - inhalation, ingestion, skin contact . Inorganic salts are more readily absorbed through the gut and excreted by the kidneys than organic.

Acute exposure: rare in industry, is characterized by febrile illness with pneumonitis. ff severe, it can cause oliguric renal failure.

Chronic exposure: slow onset with pecu1iar neuropsychiatric disorder (erethism) with features of anxiety neurosis, timidity and paranoia. Accompanied by gingivitis, excessive salivation, intention tremor, dermatographia, scanning speech. Upper motor neuron lesions and visual field constriction are more commonly associated with organic mercurialism.

***Biological monitoring*: mercury in urine or blood.**

Treatment: BAL, penicillamine

Prognosis for patients with organic poisoning (methyl or ethyl mercury) is poor, often fatal.

Arsenic (As)

It is a by-product of both ferrous and non-ferrous smelting.

Arsine **(AsH3) is a gas - the most toxic form of arsenic.**
Arsenic is general protoplasmic poison.
Uses: alloys, insecticides, fungicides, rodenticides, pigments, decolorizer in glass and paper-making.

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

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

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

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

B. CHEMICAL ASPHYXIANTS

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

Carbon monoxide (CO)

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

Metabolism: the mechanism by which CO causes its toxic effect is producing tissue hypoxia.

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

CO also binds to muscle myoglobin and to intracellular cytochrome oxidases.

Acute CO poisoning: typically, individuals with COHb levels below 1% are asymptomatic, and even COHb levels between 10 to 30% produce effects that are sometimes nondescriptive-headache, faitness, nausea and vomiting. Increased respiratory rate. Increased heart rate.

COHb 30-40% : as above, plus dimness of vision, decreased blood preassure, muscular incoordination, cherry-red skin discoloration.

COHb 40-60% : as above, plus generalized weakness, mental confusion.

COHb 60% and higher: coma, intermittent convulsions, depressed heart action and respiratory rate, and possibly death.

COHb over 90% : death within a few minutes.

***Chronic CO poisoning*: headache, organic brain damage if asphyxiation was prolonged.**

<u>Biological monitoring</u>: COHb levels.

<u>Treatment</u>: remove from exposure and give pure or hyperbaric oxygen. Cerebral edema may result from central hypoxia. Diuretics and glucocorticoids maybe appropriate to prevent its appearance or reduce its severity.

<u>Hydrogen cyanide (HCN)</u>

Hydrogen cyanide and its derivates are used in electroplating, metallurgy, and extraction of gold and silver metals from ores, production of synthetic fibers and plastics, and as a fumigand and fertilizer.

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

Acute poisoning: can occur from inhalation and also absorption through the skin, with rapid onset of headache, hypopnoea, tachykardia, hypotension, convulsion and death. The rapidity of the onset of symptoms necessitatates the treatment statim.

Chronic poisoning: none.

<u>Biological monitoring</u>: blood cyanide concentration.

<u>Treatment:</u> 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 unconscious pacient with a definitive history of cyanide exposure, dispatch the pacient immediately to hospital.

<u>Hydrogen sulphide (H2S)</u>

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

Acute poisoning: lacrimation, photophobia and mucous membrane irritation in low concentration. In high concentration pneumonitis, paralysis of the respiratory centre can cause sudden unconsciousness. Chronic poisoning : keratitis, skin vesicles. Treatment: removal from exposure, administer oxygen and amyl or sodium nitrite. Other therapy is symptomatic.