Gaseous Poisons

General Characteristics:

1- They are gases in their nature

2- Toxicity occurs due to inhalation of the gas

3-Some gases may causes irritation to the eyes and skin

4-Toxic manifestation appear very rapidly

5- Some gases have a latent period such as arsine

6- Most of them are colourless and odourless

- National Institute for Occupational Safety and Health (NIOSH) : Determined the upper limits of most gases present in the environment

Classification:

1- Simple asphyxiants
2- Chemical asphyxiants
3- Irritant asphyxiants

I- Simple asphyxiants

- Co_2 ,Methane , Nitrogen, Helium, Nitrous oxide
- Displace O_2 from inspired air
- Death occurs when O_2 is less than 10 % in air

- Carbon dioxide Co_2

- Characters & Sources

- Colourless odourless heavy gas
- Collects in bottoms of mines, wells, sewers
- Conc. in atmosphere is 0.04 %
- Generated in respiration, combustion, fermentation and decay

■ **Clinical Presentation:**

- Headache, ringing ears, muscular relaxation and collapse
- Stimulation then depression of respiration and pulse
- Death due to tissue anoxia
- Pure asphyxia should take several minutes and be associated with cyanosis and congestion
- Pure Co_2 causes instantaneous death
- minimum fatal concentration of CO2 25–30 %, the high concentrations causing sudden death being 60–80 % .

■ **Management**

1- Prevention
2- Remove patient to avoid further exposure
3- O_2 and artificial respiration
4- Respiratory and circulatory stimulant

II- Chemical Asphyxiants

Carbon monoxide (CO)

-In pure form, co is undetectable. It is colourless, odourless non-irritant lethal gas

- Called silent killer

- Specific gravity 0.97 % relative to air , so it disperse easily in air and does not stratify

- Gas is produced whenever there is incomplete combustion of carbon containing compounds occurs .

- Sources

1- External sources

A- Incomplete combustion of carbonaceous fuel e.g. Coal , wood, methane , butane, motor vehicle exhaust

B- Tobacco smoking:

- Cigarette smoke contains 3-6 % CO
- Pipes & Cigars contain 15 % CO
- Smoking one Cigarette /day produce 6 % CoHb

C- Methylene chloride: common industrial solvent metabolized in the liver to Co.

2- Internal sources

- Normal CoHb level ranges from 1-2 %
- Produced as a byproduct of protoporphyrin braekdown
- This low level is insignificant to health person

■ Manner of Toxicity:

I- Accidental

A- Occupational

1. Firemen without respiratory protection
2. Coal miners
3. Exhaust from motor cars
3. Brick kilns
4. Paint removers

B- Household

1. Leakage from sources of domestic supply e.g. faulty gas appliances and heating systems.
2. Use of coal stoves in closed spaces for heating

II- Suicidal

Running of car engine in a closed space(victim either sits in the car in a closed garage with the engine running or leads a pipe from the exhaust through the window of the car)

III- Homicidal : Rare

Mechanism of Toxicity:

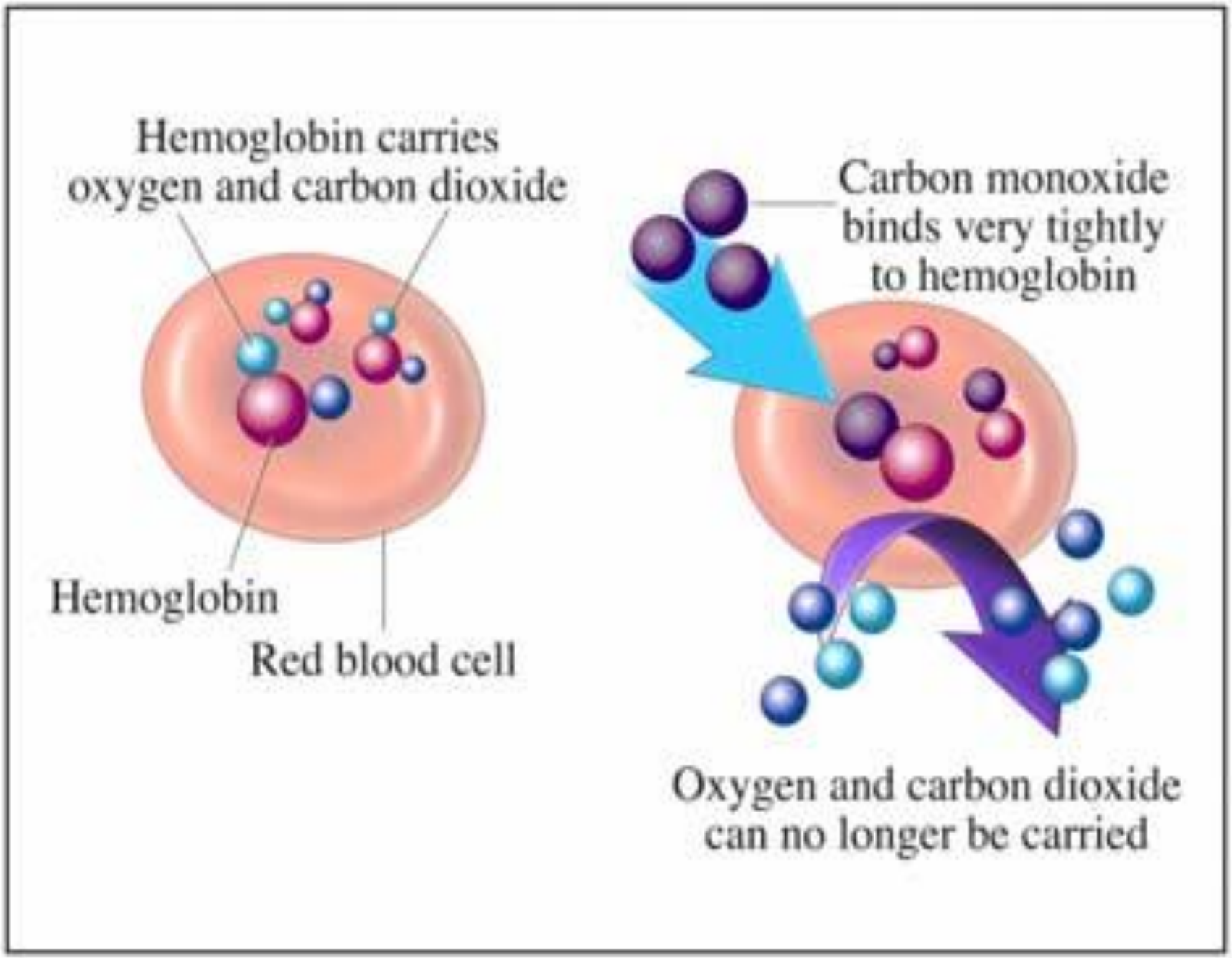
- Its poisonous qualities are due to its great affinity for haemoglobin, as it has a combining power some 250 times greater than O_2. This means that even small concentrations of CO can displace O_2 from the red blood cells and progressively diminish the ability of the blood to transport O_2 to the tissues. Strong concentrations of CO, such as a leak from a laboratory cylinder, can kill within a few moments.

- Co has significant affinity for iron or copper containing proteins and compete with o_2 for the active sites in metalloproteins including myoglobin, cytochrome C oxidae, cytochrome P-450 oxidase.

- High affinity for myoglobin (which facilitate oxygen transfer to mitochondia) so interferes with cellular respiration

- This produce hypoxic stress which first affect tissues of high metabolic rates such as myocardium and brain.

- The stability of CoHb makes it a cumulative poison, and the blood will continue to absorb the gas from the lungs if it is present in the inspired air, so that remarkably small concentrations of inspired CO may eventually prove fatal.


Hemoglobin carries
oxygen and carbon dioxide
Hemoglobin
Red blood cell
Carbon monoxide
binds very tightly
to hemoglobin
Oxygen and carbon dioxide
can no longer be carried

Factors affecting the severity of poisoning:

1- Concentration of CO
2- Period of exposure
3- Volume of pulmonary ventilation
4- Hb Concentartion
5- Infants, pregnant women, persons with high metabolic rates, patients with cardiovascular disease.

Toxic and fatal dose

- Toxic manifestations appear at conc. of 0.01 % CO in the atmosphere (20 % COHb)
- 0.1 % CO in the air produces 50 % COHb
- 1% CO in the inspired air produce immediate death

❑ Clinical Picture

COHb %	Signs & Symptoms
0 -10	No symptoms
10 - 20	Slight headache and exertional dyspnea
20 - 30	headache and throbbing in the temples, dizziness, easy fatigue
30 - 40	Severe headache , weakness, dizziness, confusion, dimness of vision, nausea, vomiting and collapse
40 - 50	Same as above with syncope and increased pulse and respiratory rates
50 - 60	Syncope, convulsions , chyne –stokes respiration
60 - 70	Coma, convulsions, depressed heart and respiratory rates, pulmonary oedema and death
70 - 80	Respiratory failure and death within few hours
80 - 90	Death in less than an hour
> 90	Death within few minutes

Sequelae of CO poisoning:

1- Complete recovery in 2-4 days
2- Little residual effects
3- If coma persists for more than 24 hours , there will be serious neurological manifestations in the form of: Twitching, chorioform movements, seizures, delayed neuropath, deterioration of personality, memory impairment, visual and hearing impairment, blood diathesis, kidney damage, skin lesions.

Chronic poisoning

1- Changes in higher cerebral functions
2- Increased incidence of coronary vascular disease
3- low grade hypoxia
4- changes in the CNS
5- Degenerative changes in kidneys , liver and heart

■ Acute effects of CO:

❖The earliest symptoms are non-specific(headache, dizziness and nausea) confused with other illness as influenza. Children show non-specific symptoms including nausea, headache, vomiting and colic making diagnosis difficult to establish. Continued exposure to CO can lead to cardiac symptoms. The CNS is most sensitive to CO .Acutely, there are headache, dizziness and ataxia; with longer exposure, syncope, seizers or coma can result. Retinal haemorrhages have been seen in patients with exposure more than 12 hours.

❖Longer exposure with decreased levels of consciousness result from metabolic acidosis from lactate production accompanying the tissue hypoxia. Although mynecrosis and even compartment syndrome, patients rarely develop renal failure. Cherry red colour of the skin is seen only after excessive exposure. Another uncommon phenomenon is the development of cutaneous bullae following sever exposure.

■ Long term problems

- Delayed effects of acute CO poisoning are varied including dementia, amnesia, psychosis, parkinsonism, paralysis chorea, cortical blindness, peripheral neuropathy and incontinence. These effects can be preceded by lucid interval ranged from days to weeks.Children may show behavioural and learning difficulties after sever poisoning.Older patients(>30yrs)appear to be much susceptible to delayed sequelae.

❖Laboratory tests used to diagnose CO poisoning

1- Cooximeter which estimate the percentage of the total Hb saturated with CO by a spectrophotometric manner

2- Breath sampling method can also be used

3- Arterial blood gas analysis can be used in severe poisoning to confirm the presence of metabolic acidosis

4- Spectroscopic test (Ordinary and revision spectroscope)

Treatmet

I- Prophylactic measures

II- Curative measures

1- Remove the victim from the CO source
2- if CoHb is less than 15% fresh air and rest are sufficient
3- If CoHb is more than 15% O_2 should be given
4- Pure O_2 decrease the half-life of CO from 4-5 hours to 40 minutes.HBO decrease the half-life to 23 minutes

❖Indications of HBO:

1- CoHb level is 40% or more
2- Occurrence of syncope, seizers, visual disturbance or coma
3- Persistent neurological findings
4- Cardiac arrhythmias or myocardial ischemia
5- Pregnancy with CoHb 15% or more
6- Metabolic acidosis
7- Neuropsychiatric abnormalities

❖Mechanism of action of HBO

- O_2 under tension will be dissolved in plasma oxygenating tissues directly
- HBO accelerates CoHb disassociation and shifts the OxyHb disassociation curve back to the right

❖Complications of HBO

1- Oxygen induced seizers
2- Barotrauma in the form of discomfort, tympanic membrane rupture, tension pneumothorax

❖Symptomatic treatment:

1- Warmth
2- Diuretics and glucocorticoids
3- Dizepam for seizers
4- complete bed rest for 10 days
5- Observation for delayed complications

❖Post-mortem picture

1- Froth around mouth and nostrils
2- Cherry-red hypostasis and in the internal organs
3- Brain shows oedema , petechial hemorrhage, degenerative changes in the basal ganglia
4- Myocardial degeneration and lung oedema

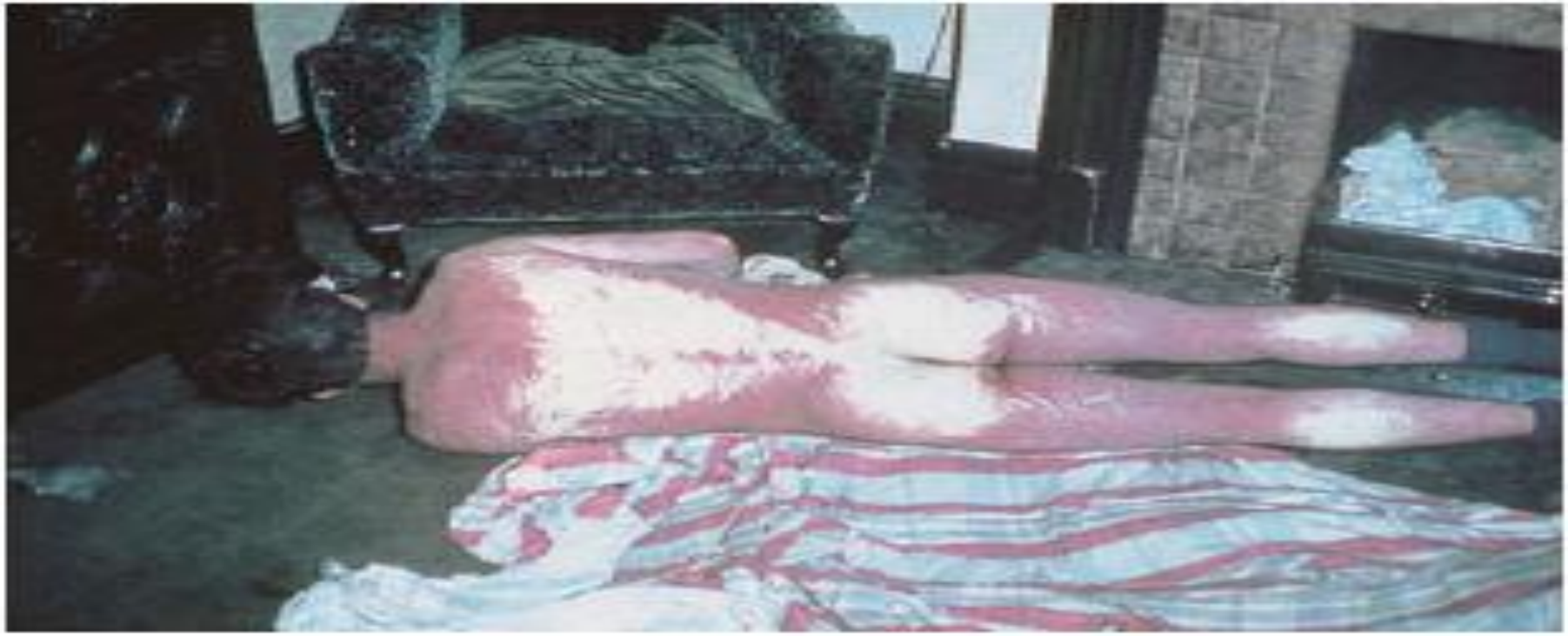


Figure 28.1 Cherry-pink hypostasis of carbon monoxide poisoning.

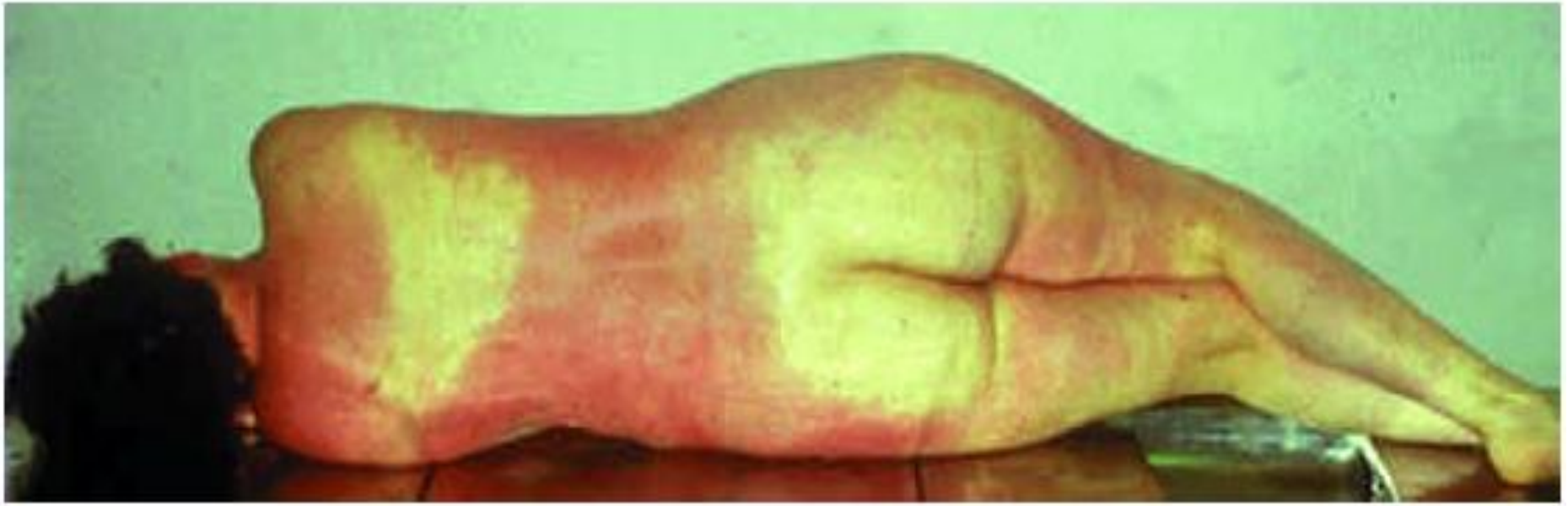


FIGURE 9.59 Carbon monoxide poisoning causes red livor mortis. Red livor mortis also occurs in cyanide poisoning and from the cold.

III- Hydrogen sulphide

Physical characters

- Colourless, characteristic rotten egg odour, heavier than air, accumulates in underground locations and sewers and wells.

Sources

1- Cases of putrefaction
2- Some industries as petroleum and rubber
3- Liquid manure tanks in agriculture and rural latrines

Mechanism of action

- Irritation of skin, eyes, respiratory and GIT
- Inhibition of cytochrome oxidase resulting in tissue hypoxia
- Direct depressant effect on CNS including respiratory centre
- Paralysis of the olfactory nerve

Toxic dose

- 0-0.05% causes irritant effects
- 0.1% causes immediate death
- 2000 ppm is fatal after a single breath

❖Clinical picture

- Conjunctivitis with pain, lacrimation and photophobia
- Rhinitis, tracheitis, bronchitis, pneumonia, pulmonary oedema and greenish cyanosis
- Higher concentration lead to resp. Collapse, convulsions, paralysis of CNS and death

❖Treatment

1- Prevention of further exposure
2- Wash the eyes with copious amount of water
3- Washing the skin
4- Induction of methehemoglobinaemai with nitrites
5- Symptomatic treatment

❖Post-mortem picture

Externally: greenish cyanosis and hypostasis
Internally: Hemorrhagic pulmonary oedema, greenish discolouration of brain and viscera

IV- Irritant Asphyxiants

Examples:

sulpher oxide, nitric oxide, nitrogen dioxide, phosphides, phosphene, chlorine, bromine, fluorine and phosgen

Clinical picture

- In low concentration: skin and upper respiratory tract irritation
- In high concentration: severe resp. Distress, laryngeal oedema and pulmonary oedema , nausea, vomiting, fatigue, and coma
- Acute oedema of glottis may cause severe sudden death

Treatment

1. Prevention of further exposure
2. Inhalation of oxygen
3. Antibiotics
4. Washing of eyes and skin thoroughly with water

Volatile Poisons

I- Ethyl alcohol

❖Physical characters

Volatile, clear, coloureless , liquid having burning sensation when swallowed

❖Mode of toxicity: almost accidental

❖M.L. Importance: - Criminal acts
- Traffic accidents

❖Toxicokinetics:

▪ Administration

The most common route is ingestion, but absorption can occur by the lungs or skin

▪ Absorption:

After ingestion begins in the stomach, but it is greater in the small intestine

▪ Factors affecting absorption

State of the stomach

Factors enhance or delay stomach emptying

Amount, alcohol conc. and composition of the beverages

Distribution: parallel to water content and blood supply of the tissues

❖Metabolism and excretion

- 90% of alcohol is oxidized mainly in the liver(by the aid of dehydrogenase enzyme) to acetaldehyde then CO_2 and water.10% is excreted unchanged mainly by the lungs and kidneys. Little amounts are excreted in sweat, saliva, bile and other body secretions

❖Mechanism of toxicity:

- Alcohol causes increase in extra-cellular water leading to:
- Alcohol depress CNS in a descending manner from the cortex to the medulla
- Leading to disturbances in the motor and thinking processes , and behavioural changes
- Alcohol is not a CNS stimulant; it is a depressant from the start.

❖Acute toxicity: Four stages

I- Stage of Euphoria : Blood alcohol level : 100 mg/dl (0.1 %)

Characteristics:

1- Sense of well being, confidence and cheerfulness

2- Aggressiveness and automatism, crimes may be committed

3- Prolongation of the reaction time leading to traffic accidents.

- In car accident, the driver must be examined for alcohol ingestion by the following tests:

1- The breath smell of alcohol

2-Finger nose test

3- Get a match out of a box

4- Walking along a strait line

5- The drunkometer to estimate alcohol level

II- Stage of inabriation: Blood alcohol level : 250mg/dl (0.25%)

1- Alcohol smell

2- Flushed face

3- Rapid weal pulse

4- Subnormal temperature

5- Staggering gait

6- Slurred speech

7- Blurring of vision

8- Vomiting

9- Hiccough

III- Paralytic stage: Blood alcohol level : 400 mg/dl (0.40 %)

-Cyanosed face, dilated pupils, stertorous breathing

IV- Stage of Coma: Blood alcohol level : 500mg/dl (0.50 %)

-Marked respiratory depression and peripheral vascular collapse

- Medulla is affected with a critical outcome.

- May end with death due to respiratory and vasomotor centres depression

❖Management: according to the stage

1- Hospitalization
2- Gastric lavage
3- Management of respiratory depression
4- Management of circulatory depression
5- Management of increased intracranial tension
6- antidote; methyle phenidate(Ritalin) and prednisolone
6- Haemodialysis

II- Methyl alcohol (Methanol)

-**Uses**: Solvent, paint thinner, antifreeze, source of heat

- The most common route of toxicity is by ingestion

- Toxicokinetics:

- Absorption and excretion : as ethanol

- Metabolism: occurs as 1/7-1/5 the rate of metabolism in ethanol leading to accumulation of methanol and its metabolites in the circulation leading to delayed toxic effects.

-Its metabolism leads to formation of formaldehyde and formic acid; Formaldehyde is 33 times as toxic as methanol.

-Distribution

-According to the water content of the tissues... it is highly toxic to the eye

-Mechanism of toxicity

1-sever metabolic acidosis due to accumulation of formic acid and lactic acid with decrease of bicabronate level.

2-increased anion gap tow or more times above normal value.

3-CNS depression and mild inebriation

4-Eye damage due to accumulation of formaldehyde enhanced by acidosis

❖Clinical manifestations:

-Symptoms resembles those of ethanol but to a lesser extent following a latent period 3-36h.

-There is abrupt onset of:

1- GIT symptoms;Nausea, vomiting,intense colic pain and diarrohea

2- CNS symptoms:Dizzness, headache and delirium

3- Cold clammy exterimities

4- Optic hyperemia , pain in the eyes, blurred vision and blindness

5- Respiratory and circulatory depression

6- Coma, convulsion and death

❖Treatment

1- Gastric lavage

2- Correction of acidosis

3- Ethanol is the specific antidote given orally or I.V.; higher affinity to alcohol dehydrogenase decrerasing the metabolism of methanol

4- Heamodialysis or peritoneal dialysis

5-Leukovorin calcium enhance metabolism of formaldehyde to carbon dioxide

6- Methyle pyrazole ; potent inhibitor of alcohol dehydrogenase in animals

III- Hydrocyanic Acid (Prussic Acid)

Physical characters:

- Rapid killer, readily metabolized to hydrogen cyanide, has a characteristic bitter almond odour
- Cyanide gas is produced by the action of diluted mineral acids e.g HCl with cyanide salts

Toxic states

- A- Accidental : the most common
- B- Suicidal: Salts are most commonly used than the acid
- C- Homicidal : rare

Absorption:

- The acid is readily absorbed from the skin and mucous membranes
- Inhalation of HCN..... Reaction within seconds and death within minutes
- Ingestion of salts..... Delayed action to few hours.

Mechanism of toxicity:

Cyanide combines with ferric ions of the enzymes leading to enzymatic paralysis resulting in :

- Tissue anoxia
- Inhibition of the functions of all the tissues

Clinical manifestations:

A- In hydrogen cyanide vapours ; onset within seconds the patient fall immediately unconscious with a cyanide cry. Death occur within seconds

B- With cyanide salts: Onset 30-60 minutes with

1- smell of bitter almond
2- open glistening fixed eyes with dilated unreactive pupils
3- Nausea and vomiting
4- Tachypnea followed by dyspnea
5- No cyanosis instead of anoxia
6- Convulsions followed by death

❖Treatment:

- Cyanide kit; contains three components
- Amyl nitrite
- Sodium nitrite (3% solution) 10 ml solution injected very slowly
- Sodium thiosulphate (25% solution) I.V. Injection

❑ Drawbacks of cyanide kit:

The induced metHb may add to the alraedy present hypoxia

The use of nitrite carries the risk of lowering blood pressure

❑ More safe antidotes:

1- Hydroxy cobalamine (vit B12 precursor)

2-Di cobalt EDTA (Ethylenediaminetetraacetic acid) as a chelating agent

❑ Role of oxygen therapy:

-It may displace cyanide from cytochrome oxidase

- It is recommended to be given with nitrite

IV- Kerosene

Physical characters:

Volatile colourless, irritant liquid with a characteristic odour

Uses: householf fuel and in some industries

Mode of poisoning: commonly accidental among children less than 5 years. Inhalation by workers in rubber industry

Fatal dose about 20 ml

Action:

1- mainly irritation of the lungs due to inhalation, aspiration or excretion by the lungs
2-CNS depression in large doses

Clinical manifestations:

- GIT..... Nausea, vomiting, colic and blood tinged stools
- Sensation of burn in the throat with kerosene smell in breath
- Pulmonary irritation progresses to pulmonary oedema and chemical pneumonia.
- CNS depression, weakness, drowsiness, slow shallow respiration, convulsions and coma
- Death early due to respiratory depression or delayed due to pneumonia

Diagnosis:

1. Smell and clinical picture
2. X-ray on the abdomen
3. X-ray on the chest

Treatment

1. Emesis is contraindicated
2. Gastric lavage after using a cuffed endotracheal tube
3. Oxygen inhalation and respiratory stimulants
4. Antibiotics
5. Cortisone to reduce pulmonary oedema

Chronic kerosene poisoning

- Due to inhalation of small doses in industry
- Characterized by dizziness , insomnia, weakness, weight loss, anaemia, pain in limbs and parathesia