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Volatile poisons

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1-Ethyl alcohol

Physical characters:

Volatile, clear, colorless, 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 lung or the 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 beverage.

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 to CO_2 and water.
- 10% is excreted unchanged mainly by the lungs and kidneys.
- Little amounts are excreted in sweet, saliva, bile and other body secretions.

Mechanism of toxicity

Alcohol causes increase in extra-cellular water leading to……..

- Alcohol depress the CNS in a descending manner from the cortex to the medulla. Leading to disturbances in the motor and thinking processes, and behavioral changes.
- Alcohol is not a CNS stimulant, it is a CNS depressant from the start.

Acute toxicity

Four stages

I- Stage of euphoria:

Blood alcohol level 100 mg/dl (0.1%)

Charechteristics:

1- Sense of well-being, confidence and cheerfulness.

2- An aggressive person seeks a quarrel, while a shy person becomes confiding.

3- Aggressiveness and automatism, crimes may be committed.

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

In motor care accidents 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 the level of alcohol.

II- Stage of inebriation:

Blood alcohol level reaches about 250 mg/dl (0.25%)

1- Smell of alcohol.
2- Flushed face.
3- Rapid week pulse.
4- Subnormal temperature.
5- Staggering gait.
6- Slurred speech.
7- Blurring of vision.
8- Vomiting.
9- Hiccough.

III- Paralytic stage:

Blood-alcohol level is 400 mg/ dl (0.40%).
Characterized by:
1- Cyanosed face.
2- Dilated pupils
3- Stertorous breathing.

IV- Stage of coma:

Blood-alcohol level reaches the LD_{50} about 500ml/dl (0.50%).
It is a highly dangerous stage.
1- Marked respiratory depression and
2- peripheral vascular collapse.
3- Medulla is affected with a critical outcome.
4- May end by death due to respiratory and vasomotor centers depression.

Management:

According to the stage.

1- Hospitalization.

2- Gastric lavage.

3-Management of the respiratory depression.

4-Management of the circulatory depression.

5-Management of increased intracranial tension.

6- Antidotes.

7- Hemodialysis.

2- Methyl alcohol (Methanol)

Uses: Solvent, Paint thinner, heat…….

The commonest rout of toxicity is by ingestion.

Toxicokinetics:

Absorption and distribution are 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….why 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 bicarbonate level.
2-Increased anion gap two 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 resemble those of ethanol but to a lesser extent

Following a latent period 3- 36 h. there is a abrupt onset of:

1-GIT symptoms, nausea, vomiting, intense colicky pain and diarrhea.

2-CNS. Symptoms, dizziness, headache and delirium.

3-Cold clammy extremities.

4-Optic hyperemia, eye pains, blurred vision and blindness.

5-Respiratory and circulatory depression.

Coma, convulsions and death.

Treatment

1-Gastric lavage.
2-Correction of acidosis.
3-Ethanol is the specific antidote given orally or intravenously.
4-Hemodialysis or peritoneal dialysis.
5-Leukovorin calcium.
6-Methyl pyrazole (4-MP).

3- Hydrocyanic acid (Prussic acid)

- **Physical characters**: Rapidly killer, readily volatilized to hydrogen cyanide, has a characteristic bitter almond odor.

- **Cyanide gas** is produced by the action of dil. Mineral acids (eg. HCl) with cyanide salts.

Toxic states

*Accidental: The most common.

*Suicidal: Salts are most commonly used than the acid.

*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:

1- Tissue anoxia.

2- Inhibition of functions of all tissues.

Clinical manifestations

In hydrogen cyanide vapors, onset is within seconds the patient fall immediately unconscious with a cyanide cry. Death occurs within minutes.

With cyanide salts: Onset 30-60minutes with:

1- smell of bitter almond

2- Open glistening fixed eyes with dilated uncreative pupil.

3- Nausea and vomiting.

4- Hyperpnea followed by dyspnea.

5- No cyanosis instead of anoxia.

6- Convulsions followed by death.

Treatment: It must be urgent

1- Historically methylene blue was used.
2- "Cyanide Kit" contains three components:
a) Amyl nitrite
b) Sodium nitrite (3% solution) 10ml solution injected very slowly.
c) Sodium thiosulphate (25% solution) iv injection.

*Drawbacks of cyanid kits:

- The induced methemoglobinemia may add to the already present hypoxia.
- The use of nitrites carry the risk of lowering blood Pressure.

More safe antidotes

*Hydroxy cobalamine (vit. B12 precursor).
*Di cobalt EDTA as a chelating agent.

Role of oxygen therapy

1) it may displace cyanide from cytochrome oxidase.
2) It is recommended to be given with nitrite.

4- Kerosene

- One of the volatile hydrocarbons
- **Physical characters**: Volatile, colorless, irritant liquid with a characteristic odor.
- **Uses:** household 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 lung.

2) CNS depression in large doses.

Clinical manifestations

1- GIT ……. Nausea, vomiting, colic and blood tinged stools.
2- Sensation of burn in the throat with
3- kerosene smell in breath.
4- Pulmonary irritation progresses to pulm. Edema and chemical pneumonia.
5- CNS depression weakness, drowsiness, slow shallow respiration, coma and convulsions.
6- Death early due to resp. depression or delayed due to pneumonia.

Diagnosis:

*Smell and clinical picture.
*X- ray on the abdomen.
*X-ray on the chest.

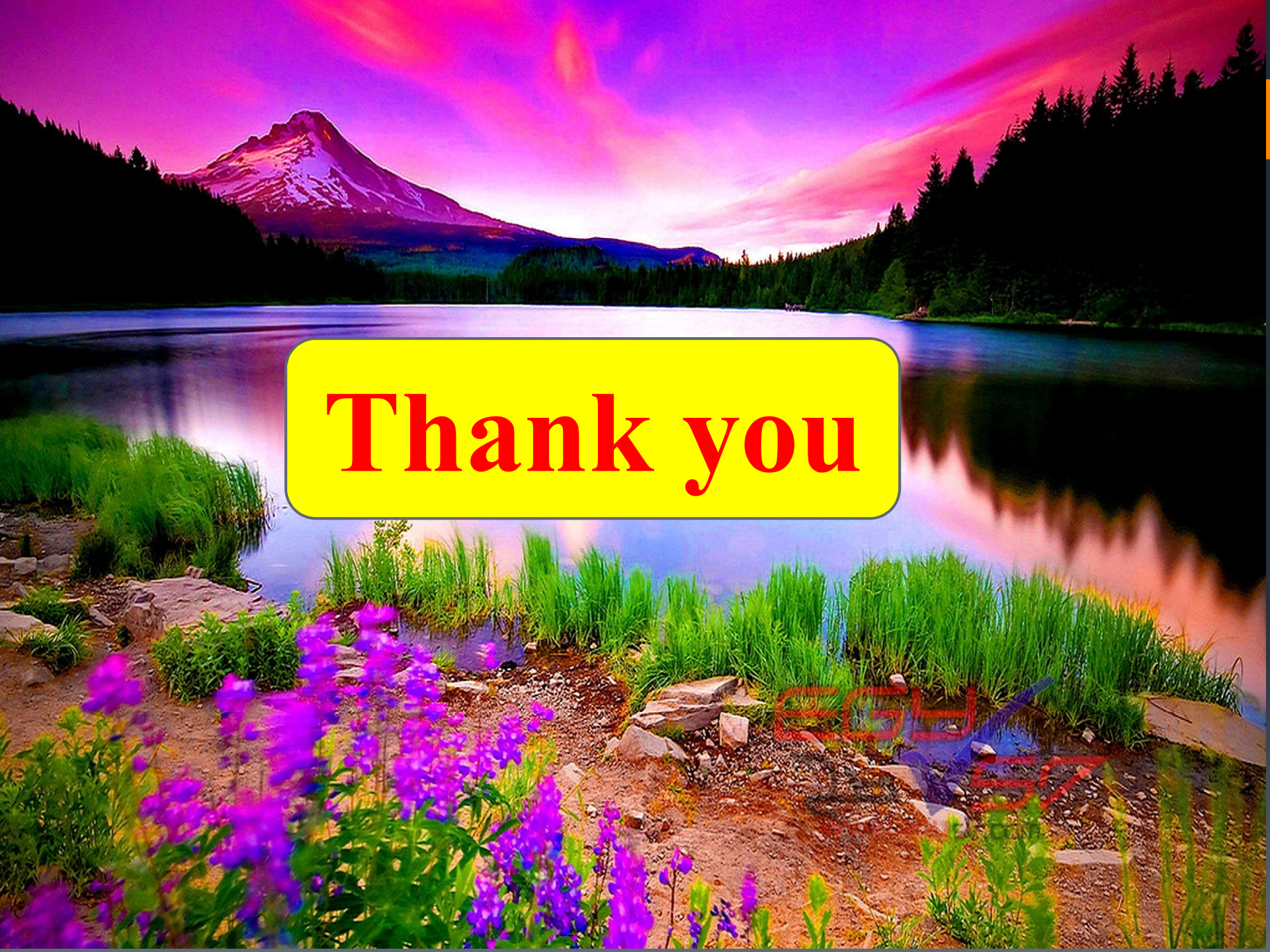
Treatment:

*Emesis is contraindicated.
*Gastric lavage after using a cuffed endotracheal tube.
*Oxygen inhalation and respiratory stimulant.
*Antibiotics.
*Cortisone to reduce pulmonary edema.

Chronic kerosene poisoning

*Due to inhalation of small doses in industry.

*Characterized by dizziness, anosmia, weakness, weight loss, anemia, pain in limbs and parathesia.


Thank you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