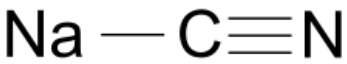
Cyanide poisoning

Hydrogen cyanide (HCN (https://pubchem.ncbi.nlm.nih.gov/compound/768)), ***potassium cyanide*** (KCN (https://pubchem.ncbi.nlm.nih.gov/compound/Potassium-cyanide)), ***sodium cyanide*** (NaCN (https://pubchem.ncbi.nlm.nih.gov/compound/8929)) is one of the **fastest acting and most violent poisons**. Lethal dose: 1 mg/kg body weight. Gas smells after the bitter tonsils (this ability is genetically determined, not everyone feels it). It is slightly lighter than air. It was used by the Nazis in gas chambers (the so-called *Zyklon B*).



Sodium cyanide (NaCN)

The **salts** are also highly lethal, the lethal dose of KCN (*potassium cyanide*) is **100-250 mg** (on the tip of a knife). Over time, the toxicity of the salts decreases, because thanks to the CO_2 in the air, they turn into **carbonates**.

Potassium cyanide (KCN)

Sources of exposure

Exposure to salts in **galvanising plants** – use for plating (gold plating, silver plating, copper plating), extraction of gold (https://pubchem.ncbi.nlm.nih.gov/compound/23985) and silver (https://pubchem.ncbi.nlm.nih.gov/compound/23954) from ores... When salts come into contact with acid, HCN is released, it is also released when burning – especially plastics with nitrogen (https://pubchem.ncbi.nlm.nih.gov/compound/NitrogenNitrogen) in the molecule (polyurethanes (https://www.britannica.com/science/polyurethane)). It is also produced from bitter **almonds** (after glucose is split off from amygdalin – the lethal dose is about 30 pieces). Another source is *organic cyanides* - eg **sodium nitroprusside** (medicine for hypertensive crisis).

Pathophysiology & Etiology

It causes a blockage of cellular respiration by binding to Fe^{3+} in **cytochrome oxidase** in the mitochondria. Inhibition of **cytochrome oxidase** causes interruption of **oxidative phosphorylation**, **lactate** rises and metabolic acidosis occurs. Venous blood becomes arterialised (has a lot of oxygen) and is lighter red.

Our organism metabolises cyanide by the hepatic *thiosulfate sulfotransferase* (rhodanase) to **SCN**, which is excreted in the urine. The gas is absorbed very quickly and can cause **sudden death**. After ingestion of salt, HCN is produced in the stomach by the action of **HCl**, the onset of the effect is slower, depends on the acidity in the stomach, when the salt was formed, and the amount of food ingested.

Clinical features

After inhalation of **HCN**: dizziness, confusion, convulsions, vomiting, tachycardia, unconsciousness, death by respiratory arrest.

After ingestion of salt, the symptoms develop within tens of minutes, the breath smells after what is usually described as "bitter almonds".

Potassium / sodium cyanide – poison

Diagnosis

Acid-Base Balance (ABB): metabolic acidosis (MAc).

Oxygen saturation: arterial O_2 saturation is sufficient, in addition there is increased venous oxygen saturation.

Differential diagnosis

It is necessary to distinguish other **intoxications** - carbon monoxide (CO) and carbon disulfide.

Therapy

1. First, the victim must be removed from the HCN-contaminated environment.
2. When he is not breathing - controlled breathing ⚠
3. If a person is fine after cyanide inhalation, we can assume that the patient will be fine without any further treatment, however, we have to take into account the latency after oral cyanide intoxication,

Conservative therapy – HCN has an affinity mainly in Fe^{3+}, so it does not bind to normal hemoglobin, but it binds well to methemoglobin, so methemoglobinemia is induced with the help of oxidizing agents (4-dimethylaminophenol, nitrates); this method has its risks, but it has a quick effect.

A new method of therapy – binding to **hydroxycobalamin** (from vitamin B12), the treatment is risk-free but expensive.

STEP 1 of therapy

- Inhalation of *amylium nitrosum* (Amyl nitrite) from a broken glass tube,
- will make 5% methemoglobinemia, this help is also provided by a amateur,
- *hydroxycobalamin* inj. **4–10 g i.v.**

STEP 2 of therapy

- *Natrium thiosulfate* inj. **4–12 g i.v.**,
- does not have severe side effects, but the effect is slower.

References

References

- National Library of Medicine: National Center for Biotechnology Information. *Cyanide toxicity* [online]. StatPearls Publishing, ©2022. The last revision 2022-02-17, [cit. 2022-11-27]. <https://www.ncbi.nlm.nih.gov/books/NBK507796/>.

Bibliography

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