Carbon monoxide intoxication

Carbon monoxide is a non-irritating, odorless gas. It is lighter than air, but mixes freely with air. Its presence in the meeting is thus influenced more by the air flow. It is formed by imperfect oxidation of carbonaceous substances. Poisoning is very often overlooked.

See the Oxides of Carbon page for more information .

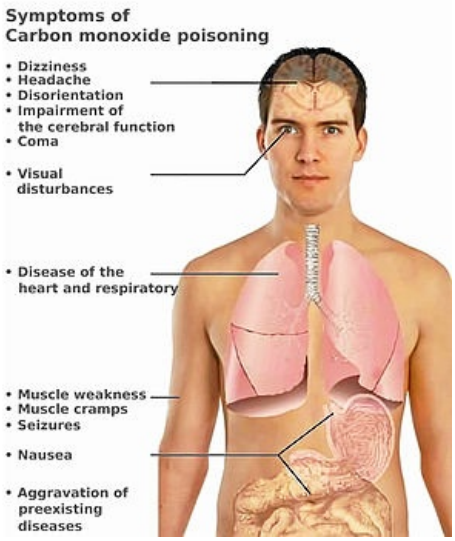
Professional exposure

- Risk workplaces - boiler rooms, lignite mines where coal smolders (up to 50% CO in mine gases, 38% in water gas , 11% in exhaust gases),
- unprofessionally - from natural gas (bathrooms - karma).

Etiopatogeneze

The bond to Hb is about 210 times higher than that of oxygen . It forms carboxyHb and thus occupies oxygen binding sites. The result is tissue anoxia. As COHb increases, the oxygen dissociation curve shifts to the left. COHb is bright red, which is most pronounced in the venous blood. CO 2 production decreases , leading to hypocapnia. CO also binds to cytochrome P450 and myoglobin , there is a decrease (myocardial contractility). It also appears to have a specific cytotoxic effect.

In addition to the concentration in the air, it also depends on physical activity - firefighters inhale the most (higher minute volume). The first damaged organs are the brain and myocardium due to the highest demands on oxygen (there is a physiological reduction in blood flow in other organs).



Příznaky otravy oxidem uhelnatým

The COHb bond is reversible - it makes sense to bring the affected person to fresh air. When in the fresh air, COHb halves in 4 hours (in oxygen therapy per hour).

In non-smokers, the common presence of COHb is about 1%, in smokers 5% or more.

Clinical picture

Acute

They are not specific, it is necessary to determine the level of COHb to verify the diagnosis. In addition to the laboratory, it is possible to use a pulse cooxymeter - an instrument that is able to distinguish the wavelengths of COHb and HbO2. Conventional oximeters do not differentiate these hemoglobin and report a false acceptable saturation value in a severely hypoxic patient.

- **10 % COHb** – mild concentration disorder;
- **20 % COHb** – mild headache, dizziness ;
- **30 % COHb** – headache , nausea , vomiting , exertional dyspnoea ;
- **40–50 % COHb** – confusion, persistent headache, even coma and convulsions ;
- **nad 60 % COHb** – deep coma, death.

Chronic

- Pseudoneurastenic syndrome , extrapyramidal symptoms, or organic psychosyndrome ;
- neurological symptoms - parkinsonism with mutism , agnosia , visual disorders;
- personality changes, increased irritability, verbal aggression;
- we often find necrosis in the *globus pallidus* , as well as in other BG , hippocampus and white matter.

Hyperbarická komora

Investigation methods

Blood collection for COHb determination must be performed as soon as possible. Saturation measurement (pulse oximetry or sampling) - false normal results (bright red COHb color). In the acute stage - we find MAC , elevated blood glucose . Changes in CNS - CT , MRI . PET can detect ischemic foci in an acute condition (but it is difficult to estimate the prognosis).

Therapy

Remove to fresh air immediately. The causal treatment is oxygen therapy , in severe poisonings - hyperbaric oxygen therapy .

Links

External links

- Carbon monoxide poisoning - interactive algorithm + test (https://www.mzcr.cz/intoxikaci-oxidem-uhelnatym-je-dobre-nepodcenovat/)
- Stránky MZ ČR (https://www.mzcr.cz/intoxikaci-oxidem-uhelnatym-je-dobre-nepodcenovat/)

Source

- BENEŠ, Jiří. *Study materials* [online]. [feeling. 24.02.2010]. < http://jirben.wz.cz >.

References

- PELCLOVA, Daniela. *Occupational diseases and intoxication.* 2nd edition. Prague: Karolinum, 2006. 207 pp. ISBN 80-246-1183-X .