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CARBON MONOXIDE POISONING

A guide for GPs and other medical professionals

This fact sheet covers:

- Sources of carbon monoxide
- How to diagnose carbon monoxide poisoning
- Clinical signs
- Management of carbon monoxide poisoning
- Mechanisms of action of carbon monoxide
- Bibliography

Carbon Monoxide (also known as CO) is a colourless, odourless poisonous gas and is a common yet preventable cause of death from poisoning worldwide. On average between 1 and 2 people die each year in Ireland from unintentional CO poisoning in the home in incidents related to domestic heating or other fossil fuel installations*. Because of the non-specific nature of its symptoms, poisoning by carbon monoxide is most probably under-diagnosed and there could well be a large number of people being exposed and suffering the ill effects of exposure. Children, pregnant women and their babies and those with cardiovascular disease are at increased risk. Poisoning can result in lasting neurological damage.



* In total, approximately 40 people die each year in Ireland from CO poisoning. Approximately half of the deaths from unintentional CO poisonings result from the inhalation of smoke from fires. Other significant causes are vehicle exhausts and deaths in industrial / commercial settings.

Sources of carbon monoxide

- Carbon monoxide is produced by the incomplete combustion of carbon-containing fuel including gas (natural, piped or bottled), coal, coke, oil and wood.
- Potential sources include the following: Gas stoves, fires and boilers, gas powered water heaters, paraffin heaters, solid fuel powered stoves, boilers and room heaters.
- Gas fuel leaks per se are not a source of carbon monoxide unless there is a combustion process of some sort involved.
- Inadequate maintenance leading to poor combustion and partially blocked or damaged flues and chimneys are the main causes of indoor carbon monoxide accumulation.
- Carbon monoxide can also seep into properties via shared flues and chimneys.
- Integral garages can be a source of carbon monoxide if car engines are run without adequate ventilation.
- All forms of carbon fuel combustion may lead to production of carbon monoxide gas.

How to diagnose carbon monoxide poisoning

The diagnosis of carbon monoxide poisoning can be difficult as it may simulate many other conditions: Unless poisoning is suspected the diagnosis may be missed. The onset of symptoms is often insidious and may not be recognised by either the patient or the doctor.

The commonest symptoms and an indication of their approximate frequency in carbon monoxide poisoning are shown below:

- Headache - 90%
- Nausea and vomiting - 50%
- Vertigo - 50%
- Alteration in consciousness - 30%
- Subjective weakness - 20%



Whilst exposure to high concentrations of carbon monoxide leads to collapse, chronic exposure to lower concentrations may lead to the symptoms and signs of influenza or food poisoning (apparently classic cases of food poisoning of a whole family may be produced by carbon monoxide poisoning). Prolonged exposure to concentrations that produce only minor symptoms may, in some cases, be associated with lasting neurological effects including difficulty in concentrating and emotional lability. Complaints about such problems should alert the doctor to the possibility of carbon monoxide poisoning.

Clues to the diagnosis

The following are suggestive of domestic carbon monoxide poisoning:

- More than one person in the house affected.
- Symptoms better when away from the house e.g. on holiday, but recur on returning home.
- Symptoms related to cooking: stove in use.
- Symptoms worse in winter: heating in use.

The following signs may be recognised in the home:

- Black sooty marks on the radiants of gas fires.
- Black sooty marks on the wall around stoves, boilers and fires.
- Smoke accumulating in rooms due to faulty flues: Though you cannot smell carbon monoxide you can smell other combustion products.
- Yellow instead of blue flames from gas appliances.

Clinical signs

The cherry red skin colour produced when carboxyhaemoglobin (COHb) concentrations exceed about 20% is rarely seen in life. Neurological signs must be looked for: A neurological examination, including tests of fine movement and balance (finger-nose movement, Romberg's test, normal gait and heel-toe walking), a mini-mental state examination and testing of short term memory and the ability to subtract 7, serially, from 100, are useful.

Carbon monoxide is produced continuously in the body as a by-product of haem breakdown. This leads to a normal baseline COHb concentration of about 0.5%. In pregnancy and especially in haemolytic anaemias this can rise towards 5%. Cigarette smoking leads to COHb concentrations of up to about 13% in heavy smokers.

Management of carbon monoxide poisoning in primary care setting

Monitors are available that measure carbon monoxide concentration in expired air and convert this value into COHb concentration in blood. If such devices are used, they must be used soon after likely exposure: there is no point in taking a measurement if the patient has spent hours away from the source of carbon monoxide. Measurements taken the next day at the surgery may be misleading. Alternatively, if a monitor is not available, and carbon monoxide poisoning is suspected, the patient may be referred to the local Accident and Emergency Department for further assessment.

Management

- Remove patient and others from source of carbon monoxide.
- Give 100% oxygen.
- A tightly fitting mask with an inflated face-seal is necessary for the administration of 100% oxygen.
- Consider referring for hyperbaric oxygen treatment. (see below).
- If a natural gas appliance is suspected, advise to arrange a FREE safety check by calling 1850 79 79 79.

Indications for hyperbaric oxygen

There is debate about the added value provided by hyperbaric oxygen. A COHb concentration of >20% should be an indication to consider hyperbaric oxygen and the decision should be taken on the basis of the indicators listed below:

- Loss of consciousness at any stage.
- Neurological signs other than headache.
- Myocardial ischaemia/arrhythmia diagnosed by ECG.
- The patient is pregnant.

Mechanisms of action of carbon monoxide

Carbon monoxide binds to haemoglobin with about 240 times the affinity of oxygen and also causes a left shift in the oxyhaemoglobin dissociation curve. These effects combine to reduce oxygen delivery to the tissues. In addition, carbon monoxide is transported dissolved in plasma and binds to intracellular myoglobin and mitochondrial cytochrome enzymes. Binding to cytochrome A3 is thought to play an important part in the toxicity of this gas.

Recent studies have shown that carbon monoxide may function as a local transmitter substance in the body playing a role in controlling permeability of the micro-vasculature and may increase adhesion of inflammatory cells and platelets to

the capillary endothelium.

Carbon monoxide poisoning leads to leakage of fluid across cerebral capillaries and thus to cerebral oedema. In those who have been exposed to enough carbon monoxide to produce unconsciousness, delayed neurological damage due to leuko-encephalopathy may occur. Damage tends to be focused on those parts of the brain lying at the boundaries of the fields supplied by two cerebral arterial systems, e.g. the basal ganglia. Neurological damage seems to be the result of free radical generation and lipid peroxidation. It is possible that the binding of carbon monoxide to cytochrome A3 reduces the capacity of cells to deal with free radicals.

Carbon monoxide bound to

haemoglobin has a half-life of about 320 minutes under normal circumstances. This can be reduced by exposing the patient to 100% oxygen: This reduces the half-life to 80 minutes; or to 100% oxygen at 2 atmospheres pressure (hyperbaric oxygen) which reduces the half-life to 23 minutes. The half-life of carbon monoxide bound to mitochondrial cytochromes may well be much longer than that of carboxyhaemoglobin and hyperbaric oxygen has been suggested as being important in attacking this binding site. Carbon monoxide binds to fetal haemoglobin and shifts the already left-shifted oxyhaemoglobin dissociation curve further to the left. The half-life of carbon monoxide in the fetus is longer than that in the mother.