Carbon monoxide poisoning

There are approximately 400 admissions to hospital with Carbon Monoxide (CO) poisoning in England each year and around 40-50 deaths. CO exposure can lead to anoxic brain injury and this section explains what CO poisoning is, its symptoms, treatment and how to prevent it.

What is Carbon Monoxide (CO)?

CO is a colourless and odourless gas, making its presence difficult to detect. It is formed when domestic fuels such as gas, coal, wood and charcoal are burned and by petrol engines. When fuel burns in an enclosed room, the oxygen in the room is gradually used up and replaced with carbon dioxide. If carbon dioxide builds up in the air, the fuel is prevented from burning fully and starts releasing carbon monoxide instead.

How is Carbon Monoxide (CO) poisoning caused?

CO is so dangerous because it binds very tightly to haemoglobin in the red blood cells and so reduces the amount of oxygen which can be carried in the bloodstream. Haemoglobin is the molecule in the blood that oxygen binds to in order to be carried around the body. The binding of CO to haemoglobin is actually more than 200 times stronger than for oxygen, so the CO effectively takes up all the space on the haemoglobin. In addition, CO interferes with the delivery of oxygen from haemoglobin into the body tissues.

These effects severely reduce the oxygen carrying capacity of the blood and limit the availability of oxygen to the body, with the brain and heart being particularly vulnerable. This can lead to anoxic brain injury. Pregnant women and the foetus are particularly susceptible to the toxic effects of CO.

Sources of CO Poisoning

- Central heating boilers
- Gas-powered generators
- Charcoal grills, propane stoves, and charcoal briquettes for both cooking and heating indoors
- Motor vehicles
- Fire
- Boats
- Power washers and other gas powered tools

An elevated COHb level of 2% for non-smokers and >9% COHb level for smokers strongly supports a diagnosis of CO poisoning.

At-risk Populations include:

- Babies and infants
- The elderly
- People with chronic heart disease, anemia or respiratory illness.

Confirmation of Diagnosis

The key to confirming the diagnosis is measuring the patient's carboxyhemoglobin (COHb) level.

> Carbon Monoxide levels can be tested either in whole blood or exhaled air.
>
> It is important to know how much time has elapsed since the patient has left the toxic environment, because that will impact the COHb level. If the patient has been breathing normal room air for several hours, COHb testing may be less useful.

The most common technology available in hospital laboratories for analyzing the blood is the multiple wavelength spectrophotometer, also known as a CO-oximeter. Venous or arterial blood may be used for testing.

A fingertip pulse CO-oximeter can be used to measure heart rate and oxygen saturation, and COHb levels. The conventional two-wavelength pulse oximeter is not accurate when COHb is present

COHb levels do not correlate well with severity of illness, outcomes or response to therapy so it is important to assess clinical symptoms and history of exposure when determining type and intensity of treatment.

Other testing, such as a fingerstick blood sugar, alcohol and toxicology screen, head CT scan or lumbar puncture may be needed to exclude other causes of altered mental status when the diagnosis of carbon monoxide poisoning is inconclusive.

Note: carbon monoxide can be produced endogenously as a byproduct of haeme metabolism. Patients with sickle cell disease can have an elevated COHb level as a result of haemolytic anemia or haemolysis

What is hypoxic brain injury / anoxic brain injury?

The brain needs a continuous supply of oxygen to survive and it uses 20% of the body's oxygen intake. Oxygen is needed for the brain to make use of glucose, its major energy source. If the oxygen supply is interrupted, the functioning of the brain is disturbed immediately and irreversible damage can quickly follow. Consciousness will be lost within 15 seconds and damage to the brain begins to occur after about four minutes without oxygen.

A complete interruption of the supply of oxygen to the brain is referred to as cerebral anoxia. If there is still a partial supply of oxygen, but at a level which is inadequate to maintain normal brain function, this is known as cerebral hypoxia. In practice, these two terms tend to be used interchangeably.

Causes of anoxic brain injury

There are many potential causes of cerebral anoxia, including:

Carbon monoxide inhalation.

Cardiac or respiratory arrest.

Irregular heart rhythm or poor function of the heart muscle after a heart attack, resulting in inefficient supply of blood to the brain.

Very low blood pressure (shock), resulting from blood loss (haemorrhage) or disturbed heart function.

Suffocation.

Choking.

Strangulation.

Very severe asthma attack.

Complication of general anaesthesia (where there has been inadequate oxygen supply or cardiac arrest).

Near drowning.

Exposure to high altitudes.

Smoke inhalation.

Poisoning.

Drug overdose.

Electric shock.

Symptoms and treatment of carbon monoxide poisoning

Symptoms

In acute carbon monoxide (CO) poisoning (rapid onset, with short-term exposure), the symptoms will depend on the degree of exposure:

Mild: Headache, nausea and vomiting are the features of mild CO exposure, often along with a general feeling of malaise. These non-specific symptoms may be misdiagnosed as more common illnesses, such as flu, gastroenteritis or food poisoning. This may lead to CO poisoning being overlooked initially, unless there is a clear history of exposure.

Moderate: As the degree of CO poisoning becomes more marked, there may be a generalised feeling of weakness, with dizziness, unsteadiness and problems with concentration and thinking. More obvious changes in behaviour, confusion and drowsiness develop and there may be shortness of breath and chest pains.

Severe: In severe CO exposure, serious deterioration can occur quite quickly, with seizures, coma and death. MRI scans may show changes in the basal ganglia and the white matter.

Long-term effects

Like other types of anoxic brain injury, acute CO poisoning may lead to quite severe long-term neurological problems, with disturbances in memory, language, cognition, mood and behaviour. The damage to the basal ganglia, which is a particular feature of CO poisoning, may lead to a movement disorder resembling Parkinson's disease.

An unusual feature of acute CO poisoning is the delayed deterioration in neurological condition which may be seen in some cases, occurring anything from a few days to as long as five to six weeks after the initial exposure. The reason for this is not entirely clear, but changes in the white matter seem to be involved. It has been suggested that these may result from demyelination, in which there is loss of the fatty, insulating myelin sheath of the nerve axons, therefore impairing their ability to conduct electrical nerve impulses.

Chronic Carbon Monoxide (CO) exposure

Chronic (persistent and long-term) exposure to lower levels of CO, as can occur with faulty domestic boilers, may go unrecognised. The symptoms include milder versions of those seen in acute CO poisoning, with headache, nausea, dizziness, light-headedness, fatigue and sleepiness, difficulty concentrating and memory problems, as well as changes in mood.

People may be aware that something is wrong, but be unable to identify exactly what is the matter, or may attribute the problems to overwork, stress or depression. If symptoms disappear while away at work, reappearing on returning home, or if other people in the same premises develop similar symptoms, it may become more obvious that there is an environmental cause.

Although most people seem to recover following chronic low level CO exposure when the source is removed, it can lead to anoxic brain injury. There have been some documented cases of subtle Magnetic Resonance Imaging (MRI) abnormalities and long-term neuropsychological effects.

Treatment of Carbon Monoxide (CO) poisoning

Treatment of acute exposure to CO involves immediate removal from the source of the poisoning and administration of 100% oxygen, together with general supportive medical care.

Hyperbaric oxygen therapy is sometimes advocated for severe cases of CO poisoning and involves giving pure oxygen at increased pressures in a hyperbaric chamber. It has been suggested that this may improve the long-term neurological outcome, although it remains controversial. Hyperbaric oxygen therapy is a specialised technique, which is only available in a few centres. It may also be associated with complications of its own and it is not used routinely.