

Carbon Monoxide

Properties

Carbon monoxide is a colourless, odourless, tasteless and non-irritant gas that is slightly less dense than air and is sparingly soluble in water. It is the product of the incomplete combustion of carbonaceous fuels and may itself be used as a fuel, burning in air to yield carbon dioxide (the product of complete combustion). The lower flammable limit in air is 12.5% by volume.

Toxic effects - physiological basis

The acute toxicity of carbon monoxide has long been recognized and is well documented.

Carbon monoxide is absorbed via the lungs in to the bloodstream, where it replaces oxygen by attaching chemically to hemoglobin forming carboxyhemoglobin. This reduces the oxygen carrying capacity of the blood. In addition the dissociation of oxyhemoglobin is also affected so that the supply of oxygen to tissues is further reduced.

The symptoms of exposure depend on the degree of saturation of the hemoglobin with carbon monoxide. Below a saturation level of 20% slight behavioral changes, e.g. impaired vigilance and sense of time, slight reductions in work capacity and mild headache, have been noted. At saturation levels of 20 to 50% severe headache, nausea, weakness, impaired judgement and fainting on exertion are common symptoms. At saturation levels of 50% or greater loss-of consciousness, severe depression and ultimately death may result. Further details of symptoms at various saturation levels are given in Appendix I

Pre existing severe respiratory or cardiovascular disease may render the body more susceptible to the effects of oxygen deprivation associated with even low concentrations of carbon monoxide. There is some evidence to suggest that other populations, such as those living at high altitudes, young children, older adults and pregnant women, may also have a heightened susceptibility to carbon monoxide exposure.

The amount of carboxyhemoglobin in the blood depends on the following factors:

- a) concentration of carbon monoxide in inhaled air,
- b) duration of exposure;
- c) degree of activity of exposed individual (respiration rate),
- d) individual susceptibility (see table 1)

In uncontaminated air carbon monoxide is eliminated solely through the respiratory system, at first rapidly but later more slowly. Average elimination rates vary between 30% and 50% per hour, depending on the amount of uncontaminated air breathed.

Raised blood carboxyhaemoglobin levels may be found in people with no exposure to carbon monoxide. Smoking also results in raised levels. The average carboxyhaemoglobin level in the blood of cigarette smokers is about 5 to 7% but may range up to about 15% in heavy smokers. Non smokers with no exposure may show carboxyhaemoglobin levels in the range 0.1% to 2.5%.

The effects of long term exposure to low levels of carbon monoxide have not been examined in any detail. There are suggestions however, that long term exposure to elevated levels may be associated with the development of hardening of the arteries (atherosclerotic cardiovascular disease).

Summary of toxic effects

Carbon monoxide has no smell and it is important to remember that it has a cumulative effect. Individuals vary considerably in their reaction to concentrations of toxic gases but the descriptions in Table I are typical.

Table I *Carbon monoxide in air*

Parts per million	Effects
50	Recommended Exposure Limit (8 hours time weighted average concentration)
200	Headache after about 7 hours if resting or after 2 hours exertion
400	Headache with discomfort with possibility of collapse after 2 hours at rest or 45 minutes exertion
1200	Palpitation after 30 minutes at rest or 10 minutes exertion
2000	Unconscious after 30 minutes at rest or 10 minutes exertion

Note: Low concentrations of individual gases (when measured in air) are recorded as parts per million i.e. 1 molecule of gas per million molecules of air

First Aid

A doctor should examine all persons who are affected by inhalation of carbon monoxide contaminated air as soon as possible. Until the affected person is receiving care from a qualified medical practitioner the following treatment should be administered:

Carry the patient to a warm uncontaminated atmosphere and loosen clothing at the neck and waist

If unconscious: begin artificial respiration at once, preferably by the mouth to mouth method if breathing has ceased continue artificial respiration until breathing recommences or until told to stop by a doctor

In other cases' keep the patient at rest, avoiding unnecessary exertion. After recovery, the patient should be taken home, and not allowed to walk there.

Sources of Carbon Monoxide Emissions

Sources of Carbon Monoxide

Carbon monoxide occurs naturally in the environment. Small quantities are produced by normal body chemistry, resulting in background levels of carboxyhaemoglobin of 0.1 to 1.0%

The most significant sources of man-made pollution and general exposure include cigarette smoking, motor vehicle exhaust fumes and other combustion processes. Serious acute exposures have resulted from running portable gas or paraffin fired heaters in poorly ventilated confined spaces.

The combustion of solid or liquid fuels produces some carbon monoxide as a function of burner design and efficiency. Flue gases, either in the stack, at the stack outlet or as blow-back through a boiler may be a serious risk

Open flame heaters

All heaters and cookers fuelled by natural gas, liquefied petroleum gas (LPG) or by paraffin, require an adequate supply of fresh air to ensure complete combustion and minimal formation of carbon monoxide. The operation of flueless equipment where the products of combustion are recirculated into the general atmosphere of a room also requires an adequate supply of make-up fresh air to ensure an adequate supply of oxygen for efficient combustion and to dilute the products of combustion to a safe level.

Problems have arisen with the use of flueless heaters in ill-ventilated rooms. Certain types of water heater have potential for flame impingement on cold surfaces (e.g. water tubes) which can give rise to excessive carbon monoxide formation.

Carbon monoxide concentrations of several hundred parts per million with reduced levels of oxygen and high levels of carbon dioxide can soon be generated in sealed rooms if flueless heaters are used.

Inefficient burner design or poor maintenance may lead to excessive carbon monoxide formation under 'normal' operation of gas-fired appliances. In such cases leaks from flues or re-entry of combustion products into buildings may result in toxic concentrations.

Internal combustion engines

Internal combustion engines powered by petrol, LPG or diesel oil may produce significant quantities of carbon monoxide depending on engine design, carburation, operating temperature, engine load etc. Other toxic fumes and gases such as oxides of nitrogen carbon dioxide lead compounds and hydrocarbon particulates may also be formed. Carbon monoxide emissions may range from 1 to 10% (10.000 to 100.000 p.p.m.) in the exhaust from a petrol driven engine at idle. Lower levels are emitted under normal load. Diesel engines usually produce significantly lower levels of carbon monoxide but may generate higher levels of oxides of nitrogen as compared with petrol and LPG engines.

In busy areas the level of background carbon monoxide may be high at certain times during the day. Under certain conditions the gases produced by these various processes maybe drawn into the dwelling giving rise to a misleading complaint of fume emission.

APPENDIX I

EFFECTS OF DIFFERENT BLOOD CARBOXHAEMOGLOBIN LEVELS

Blood % saturation with carboxyhaemoglobin	Symptoms
0-10	None
10-20	Tightness across forehead
20-30	Headaches
30-40	Severe headaches, weakness, dizziness, nausea vomiting
40-50	Collapse, increased pulse rate and respiratory rate
50-60	Coma, intermittent convulsions
60-70	Depressed heart action, death possible
70-80	Weak pulse, slowed respiration, death likely
80-90	Death in minutes

"Extracted from Canada Safety Council Data Sheet on carbon monoxide

Solid Fuel Association, 95 High Street, Clay Cross, Chesterfield S45 9DZ
 Tel: 01773 835400 Website: solidfuel.co.uk Email: sfa@solidfuel.co.uk