

## Chapter 23

# CARBON MONOXIDE TOXICITY

Carbon monoxide (CO) is a gas produced by the incomplete combustion of carbon containing compounds. It is a component of the smoke from **engine exhausts**, slow combustion **stoves** and **cigarette** smoke. It can also be produced in divers' air compressors (see Chapter 24).

CO breathed in anything more than trace amounts can be lethal. It binds avidly to the oxygen (O<sub>2</sub>) binding sites of haemoglobin (Hb) in the blood, preventing the haemoglobin from carrying O<sub>2</sub>. CO bound to haemoglobin forms carboxyhaemoglobin (HbCO). If a sufficient number of the O<sub>2</sub> binding sites are occupied by CO, death from hypoxia ensues (see Chapter 20).

CO also binds with components of the energy-producing biochemical pathways in the cells, interfering with fundamental cellular function.



Fig. 23.1

## CLINICAL FEATURES

Symptoms are those of progressive hypoxia due to the reduction in the oxygen transport by the blood. They vary with the **carboxyhaemoglobin** content of the blood as shown in the following table:

Concentration of CO in Breathing Gas	% Carboxy-haemoglobin	Effects on a Diver
400 parts per million (ppm)	7.2%	Nil or slight
800 ppm	14.4%	headaches dizziness, nausea breathlessness with exertion
1600 ppm	29.0%	confusion, vomiting, collapse.
3200 ppm	58.0%	paralysis, or loss of consciousness
4000 ppm	72.0%	coma
4500 ppm	87.0%	death

**Table 23.1**

The effects of CO are cumulative and are related to the concentration breathed and the duration of exposure. A concentration of 400 ppm may produce symptoms in an hour while 1200 ppm will need only 20 minutes. As the carboxyhaemoglobin (HbCO) level falls, following removal of the CO contamination, the clinical state may lag due to CO bound in the tissue, or from enzyme or protein damage. The classical "cherry pink" colour is only seen in the acute and early cases, before respiratory failure develops. Despite the above table, HbCO levels may not correlate directly with mortality or morbidity.

The effects of CO poisoning are greatly increased by increased pressure at **depth**, if the oxygen pressure is kept consistent. A 400 ppm contamination which would not produce clinical effects at atmospheric pressure will be equivalent to  $4 \times 400$  ppm (or 1600 ppm) at 30 metres depth (4 ATA), a concentration sufficient to cause serious toxicity. Because the oxygen partial pressure reduces with ascent, the symptoms of mild CO poisoning may only become serious during or after ascent.

Serious **brain damage** is a frequent complication of significant CO toxicity due to prolonged hypoxia of the brain. (See Case History 24.1)

Because the contaminated compressed air may be given to other divers who employ the same supplier, it is possible that they may also be affected. This may have implications not only on the differential diagnosis of the victim, but for prevention of other casualties.

## TREATMENT

The diver should be rapidly **isolated** from the contaminated gas and have **100% O<sub>2</sub>** administered by mask. The administer **basic life support** (see Chapter 42) including CPR should be applied where appropriate.

**Hyperbaric O<sub>2</sub> (HBO)** is the treatment of choice. The high partial pressure of O<sub>2</sub>, which occurs in a hyperbaric chamber, will dissolve enough O<sub>2</sub> in the blood plasma to meet the bodies needs without participation of the haemoglobin system. Oxygen is breathed at a partial pressure of 2 ATA or more to sustain life while the CO slowly detaches from the haemoglobin and is breathed out through the lungs, allowing the haemoglobin to resume its normal O<sub>2</sub> transport role.

If hyperbaric O<sub>2</sub> is to be of value it should be instituted as soon as possible, preferably within 6 hours of poisoning. Delay in treatment may result in irreversible and progressive brain damage.

## PREVENTION

The major danger to any diver is from carbon monoxide contamination of the compressed air supply. Sources of contamination include:

### **Direct contamination by CO from gasoline engine exhausts.**

This may come either from the compressor motor itself, or from other nearby motors or gas exhaust outlets. The classic case occurs where the compressor air inlet hose is located downwind from the compressor motor exhaust.

### **Contamination produced by the breakdown of unsuitable lubricants.**

The incorrect use of hydrocarbon-based lubricants used to lubricate an air compressor is a common cause, however it may also result from overheating of the compressor. Both carbon and nitrogen oxides can be formed.

### **The intake of polluted atmospheric air to fill air cylinders.**

It is important for suppliers of compressed air to regularly check the quality of the air being compressed, to ensure that this and other pollutants are not included in divers' air supplies. Adequate filtration systems are necessary on all compressors, and these should always be properly maintained.