22 February 2002

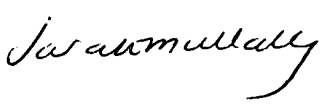
Carbon Monoxide: The Forgotten Killer

Dear Doctor

It is understandable that people should think of carbon monoxide (CO) poisoning as being a problem of the past. However, CO in the home is responsible for a considerable number of deaths each year and for many more cases of sub-lethal poisonings, which can often lead to lasting neurological damage in victims. Many more people are likely to be exposed and suffer from ill effects, but be unaware of the cause.

Yet these deaths and accidents can be prevented: both by greater awareness amongst the public and greater vigilance amongst health professionals of the signs and symptoms of exposure in their patients. By bringing this to your attention, I hope that this indiscriminate killer will not be forgotten.

We would be grateful if Nurse Directors in NHS Trusts could arrange for this letter to be circulated to community nurses, midwives and health visitors working within their Trust.



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Chief Medical Officer

Sarah Mullally
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From the Chief Medical Officer and the Chief Nursing Officer

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PL/CMO/2002/2, PL/CNO/2002/2

For Action

- District Directors of Public Health
- Medical Directors of NHS Trusts
- Chairs of Primary Care Groups and Primary Care Trusts
- General Practitioners
- Nurse Executive Directors
- Lead nurses at PCTs
- HA Nurse Advisors

For information

- Regional Directors
- Regional Directors of Public Health
- Chief Executives of HAs
- Chief Executives of NHS Trusts
- Regional Nurse Directors
- Obstetricians
- Paediatricians
- Consultants in Accident and Emergency Medicine

Summary

This letter brings together the most up-to-date information on CO poisoning and updates the CMO letter of September 1998. It sets out the main sources of CO in the home; describes the signs and symptoms which should be looked for; explains the investigations which may be necessary to establish whether a case of CO poisoning has occurred; describes how cases should be managed; and gives sources of further advice and information.

Background

Carbon monoxide is an odourless, colourless gas that causes the accidental deaths of about 50 people and seriously injures nearly 200 in the United Kingdom each year. Poisoning by carbon monoxide is almost certainly 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.

Sources of carbon monoxide

Carbon monoxide is produced by the incomplete combustion of carbon-containing fuel: gas (domestic or bottled), coal, coke, oil and wood. Gas stoves, fires and boilers, gas powered water heaters, paraffin heaters, solid fuel powered stoves, boilers and room heaters are all potential sources. Inadequate maintenance leading to poor combustion of fuel and inadequate removal of waste products as a result of blocked and partially blocked flues and chimneys are the main causes of poisoning. Such faults can occur in all types of property and the idea that carbon monoxide poisoning is limited to poorer homes and student accommodation is false. Newly occupied houses with gas powered heating systems are sometimes the site of accidents.

Carbon monoxide can seep into properties via shared flues and chimneys and people may be poisoned by carbon monoxide produced next-door. Extraordinary errors, such as the venting of gas fires into cavity walls, can lead to poisoning of people living above those using the fire. Integral garages can be a source of carbon monoxide if car engines are run without adequate ventilation.

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PL/CMO/2002/2, PL/CNO/2002/2

Date: 22 February 2002

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This letter is also available on the Internet at:
http://www.doh.gov.uk/cmo/letters.htm

How to diagnose carbon monoxide poisoning

The diagnosis of carbon monoxide poisoning is not at all easy as it may simulate many other conditions: unless poisoning is suspected the diagnosis will 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 signs 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. These include difficulties 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; and
- 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; and
- Yellow instead of blue flames from gas appliances.

Clinical signs

The cherry red skin colour produced when 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, Rhomberg'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 vital.

Investigations

Carbon monoxide can be measured in expired air. Monitors are available that convert CO concentration into COHb concentration from the standard equilibration curve. If such devices are used, they must be used quickly: there is no point in taking a measurement if the patient has spent hours away from the source of CO. Measurements taken the next day at the surgery may be misleading.

COHb can be measured in blood by any clinical chemistry laboratory. Venous blood should be taken into anti-coagulant and sent to the laboratory. COHb should be measured directly: measuring PO_2 and calculating the % saturation of haemoglobin with oxygen will be misleading as the PO_2 in CO poisoning may well be normal. Several suitable instruments are available, for example: the radiometer co-oximeter.

Pulse oximetry in cases of suspected carbon monoxide poisoning is not recommended because false high oxygen saturations are likely to be displayed due to the similar light absorbance of carboxyhaemoglobin and oxyhaemoglobin.

Rapid measurement of expired air CO is useful in diagnosis.
Blood COHb is also useful.
Expired air CO and blood COHb are poor guides to prognosis and the need for hyperbaric treatment.

Management

- Remove patient and relatives from source of CO;
- 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; and
- Arrange checking of appliances and flues and measurement of CO concentration in the house before allowing anyone back.

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; or
- The patient is pregnant.

People to consult

For CO measurements in the house:

Local CORGI(Council for Registered Gas Engineers) engineer
Local Environmental Health Department
Health and Safety Executive Helpline: **0800 300 363**

Advice on the management of poisoning:

Refer to the local National Poisons Information Service Centre

Last points

Carbon monoxide detectors are available (British Standard 7860) and could be recommended. These detectors alarm at high concentrations of CO and so protect against acute poisoning: they do not protect against chronic exposure to lower levels of CO.

An in-depth review of the health effects of domestic CO exposure is contained in **Indoor Air Quality in the Home (2): Carbon Monoxide, Assessment A5**. Institute for Environment and Health, Leicester, 1998, 203pp.

Leaflets:

1. Good Air Quality In Your Home. From the Department of the Environment, Food and Rural Affairs. Tel: 0845 955 6000.

2. Danger! Fires and Heaters Need Air and Carbon Monoxide Poisoning – This Leaflet Could Save Your Life. From the Department of Trade and Industry's Consumer Safety Unit Order Line. Tel: 0870 150 2500.

3. Gas Appliances – Get Them Checked, Keep Them Safe. From the Health and Safety Executive. Tel: 0207 717 6816 or contact HSE Books on 017 8788 1165.

Appendix

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 CO in the fetus is longer than that in the mother.

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.

Produced by Department of Health

26833 1P 45k Feb 02 (CWP)

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