Advice for Health Professionals

CARBON MONOXIDE POISONING: NEEDLESS DEATHS, UNNECESSARY INJURY

Carbon monoxide (CO) poisoning remains an important public health problem with people still dying from accidental acute carbon monoxide poisoning and others are injured from sub-lethal poisonings, which can often lead to lasting neurological damage in victims. These victims are exposed to CO in their homes, but are also at risk from exposure in holiday residences, including caravans.

Every year, there are still approximately 30 accidental deaths from acute CO poisoning in England and Wales and over 200 non-fatal poisonings requiring hospital admission. However, new data suggests there are likely to be even more cases of non-fatal poisonings in people who attend A&E, are treated for carbon monoxide poisoning, but who do not require admission to hospital – this is of great concern as CO poisoning can lead to chronic health problems. The number of people exposed to CO, but who remain unaware of the cause and do not present at their GPs surgery or local hospital is unknown.

Such deaths and accidental poisonings are preventable: through greater public awareness of the signs and symptoms of CO exposure and increased vigilance amongst health professionals.

An information leaflet for patients and the public is also available (link provided at the end of this letter) giving guidance on preventing CO poisoning and what to do if they suspect they have been poisoned. On occasion in the past, colleagues have advised patients to stay at home and keep warm when the symptoms they presented with were in fact those of CO poisoning – the results can be fatal.

Background

Carbon monoxide is a colourless, odourless gas that causes the accidental deaths of approximately 30 people each year. Around 200 people each year in England and Wales are seriously injured by CO and new data suggests that a similar number of people are treated but not admitted to hospital each year from accidental poisoning by CO. Poisoning by CO is almost certainly under-diagnosed and there could well be a large number of people being exposed and suffering the ill effects of exposure. Older people, children, pregnant women and their babies and those with breathing problems or cardiovascular disease are at increased risk. Poisoning can result in lasting neurological damage.

How to diagnose carbon monoxide poisoning

Recognising CO 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 CO poisoning are shown below:

Headache	90% of cases
Nausea and vomiting	50% of cases
Vertigo	50% of cases
Alteration in consciousness	30% of cases

Subjective weakness 20% of cases

Exposure to high concentrations of carbon monoxide leads to collapse and death within minutes.

Prolonged exposure to concentrations of carbon monoxide 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 disappear when away from the house, e.g. on holiday, or at work 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 staining on or around an appliance (e.g. stoves, boilers or fires), such as on the walls;
- Smoke or excessive condensation accumulating in rooms due to faulty flues: though you cannot smell carbon monoxide, you can often smell other combustion products;
- Yellow or orange, instead of blue, flames from gas appliances or boiler pilot lights.

Clinical signs

Neurological examination is key in determining a chronic poisoning event and 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 vital.

The cherry red skin colour is not a common sign of poisoning. This is produced when COHb) concentrations exceed about 20% and is rarely seen in life.

Investigations

Detailed advice on investigations for CO poisoning can be obtained from TOXBASE (www.toxbase.org) or the National Poisons Information Service (NPIS) (telephone 0844 892 0111).

Carbon monoxide can be measured in expired air. Breath analyzers are used in smoking cessation clinics and should be used in surgeries which have such devices. There are also analyzers which 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 PO2 and calculating the % saturation of haemoglobin with oxygen will be misleading as the PO2 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 recorded due to the similar light absorbance of carboxyhaemoglobin and oxyhaemoglobin.

- Rapid measurement of expired air 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.

Rapid measurement of expired air CO and pulse CO-oximetry are useful in diagnosis. For interpretation of blood sample results and more detailed advice on CO poisoning refer to TOXBASE (www.toxbase.org) or contact the National Poisons Information Service (NPIS) (telephone 0844 892 0111).

Management

- Remove patient and co-habitants 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;
- Arrange checking of appliances and flues and measurement of CO concentration in the house before allowing anyone back; and
- Contact social services, if necessary.

Indications for hyperbaric oxygen therapy (HBOT)

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.

HBOT is also thought to be of use for extensive exposure to CO and if neurological damage is suspected, its use should be on a case-by-case basis.

Sources of carbon monoxide

Carbon monoxide is produced not just by malfunctioning or poorly flued gas appliances but by the incomplete combustion of all carbon-containing fuels: gas (domestic or bottled), coal, coke, oil, biofuel and wood. Stoves, fires and boilers, water heaters, paraffin heaters and room heaters are all potential sources. Caravans, boats and mobile homes are also at risk

as they often use portable appliances which use these fuels, and exhaust gases from vehicle engines and generators of electricity can also contain high levels of CO. During incomplete combustion, carbon, hydrogen and available oxygen combine to form carbon dioxide, water, heat and CO. Any disruption of the burning process or shortage of oxygen can increase CO production and its accumulation to dangerous levels.

Inadequate installation or maintenance of fossil fuel and wood-burning appliances leading to poor combustion of fuel, inadequate removal of waste products because of blocked and partially blocked flues and chimneys, and insufficient ventilation are the main causes of CO poisoning. Such faults can occur in all types of property, and the idea that carbon monoxide poisoning is restricted to poorer homes and student accommodation is false. Owner-occupied houses with newly installed oil-powered cooking ranges can also be the site of accidents.

Carbon Monoxide can also seep into properties via shared flues and chimneys and people may be poisoned by carbon monoxide produced by an appliance in a neighbouring property. Dangerous 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 also be a source of carbon monoxide if car engines are run without adequate ventilation.

People to consult

For CO measurements in the house:

- *For gas:* Gas Safe Registered engineer – call **0800 408 5500** (Gas Safe is the Health and Safety Executive appointed provider of the gas installer registration scheme) or visit https://www.gassaferegister.co.uk/

- Health and Safety Executive Gas Safety Advice Line – call **0800 300 363**. http://www.hse.gov.uk/gas/domestic/index.htm

- *For oil:* Local OFTEC (Oil Firing Technical Association) engineer – call **01473 626298** or visit https://www.oftec.org.uk/

- *For solid fuel*: Local HETAS (Heating Equipment Testing and Approval Scheme) engineer – call **01684 278170** or visit https://www.hetas.co.uk/

Advice on the management of poisoning

Follow advice on TOXBASE (www.toxbase.org) or refer to the National Poisons Information Service (NPIS) on **0844 892 0111** for more detailed advice on the management of CO poisoning and interpretation of blood sample results.

Last points

Audible CO alarms are available (European Standard EN 50291, showing a British Standards Kitemark or LPCB – Loss Prevention Certification Board logo) and should be recommended. Further advice on alarms is available through the Gas Safe Register website at www.gassaferegister.co.uk. These alarms are available in homeware or DIY stores, and the alarm manufacturer's instructions for installation and maintenance should be followed.

You can also buy CO detection patches and ‘black-spot’ indicators, but these will not wake you and warn you if dangerous levels of CO develop. It is important to remember that fitting an audible CO alarm is not an alternative to having appliances, flues and chimneys serviced and tested.

Leaflets and further information

1. *Carbon monoxide: You can’t smell it, you can’t see it and it can kill!* Leaflet for the general public, available at https://gov.wales/carbon-monoxide-poisoning-leaflet

2. NHS Choices information on CO poisoning**:** www.nhs.uk/carbonmonoxide

3. Public Health England information on CO: https://www.gov.uk/government/publications/carbon-monoxide-poisoning

4. *Gas Appliances – Get them checked. Keep them safe*. Leaflet produced by the Health and Safety Executive (HSE), available at: http://www.hse.gov.uk/pubns/indg238.pdf

5. HSE has also prepared a range of information on CO poisoning: http://www.hse.gov.uk/gas/domestic/co.htm

6. Public Health Wales also has information about CO on their website: http://www.wales.nhs.uk/sitesplus/888/page/50368

Appendix A

Mechanisms of action of carbon monoxide

Carbon monoxide (CO) gas enters the blood system via the lung. Inhaled CO combines with haemoglobin to form carboxyhaemoglobin (COHb). Once this reaction occurs, the capacity of haemoglobin to carry oxygen is much reduced. Carbon monoxide binds to haemoglobin with about 240 times the affinity of oxygen and 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 microvasculature, 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 CO 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 fetal 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.