Chapter 10

Household Products

Introduction

This group of potential poisons comprises many substances, some of which fit into one or more of the other categories already discussed. For example, the herbicide paraquat (see Chapter 7) is widely used by domestic gardeners as well as by horticulturalists, and consequently it is often found in the home. Drugs too are often found around the household, however, these have been discussed already and will not be further mentioned.

Household products feature in poisoning cases usually after accidental ingestion by children and occasionally in suicide cases. The majority of enquiries relating to childhood poisoning, especially in children under 5-years-old, are in connection with non-medicinal, mainly household products or toxic substances to which people may be exposed in the home. However, the number of deaths due to substances used in the home is small, six in the UK in 1978 and 21 in the USA in 1976. The majority of deaths in children under 10-years of age are due to carbon monoxide and consequently this will be discussed in detail.

Some of the potentially toxic substances found in the home are corrosive and some are generally only ingested intentionally. Bleach is perhaps the substance most commonly involved in poisoning cases. Other substances include strong detergents such as dishwasher powder, drain cleaners which are generally caustic (i.e. corrosive), and kettle descalers which are corrosive (Figure 10.1). When bleach is ingested orally it causes burning to the throat, mouth and oesophagus. The tissue damage results in oedema in the pharynx and larynx. In the stomach the presence of endogenous hydrochloric acid generates hypochlorous acid which is an irritant, and chlorine gas which may be inhaled causing toxic effects in, and damage to, the lungs. However, serious injury from ingestion of bleach rarely occurs as it requires relatively large quantities and this is usually intentional rather than accidental.

Hydrocarbon solvents such as turpentine substitute and white spirit are often used for cleaning paint brushes. They may be dangerous by aspiration which can lead to a chemical pneumonitis. Having a low viscosity and being volatile, the solvent spreads through the lungs easily and therefore can affect a large area.

AYLESBURY PLUS, WEDNESDAY, SEPTEMBER 16 1987

Agonising death of haunted woman

A HAUNTED Wendover woman died an agonising death after drinking kettle descaler.

In the last year Mrs Heidi Mason, 44, of Orchard Close, Wendover, had tried to kill herself with pill overdoses, a razor blade and a plastic bag after becoming a victim of serious depression.

She was found, bleeding from the mouth, half-conscious but dying, in the grounds of St John's psychiatric hospital, Stone, where she was a voluntary patient, on June 4.

Her stomach was almost entirely eaten away by the acid and her mouth and throat badly blistered.

But despite her history of suicide attempts, Bucks coroner Rodney Corner refused to record a verdict of suicide at Mrs Mason's inquest in Aylesbury on Friday.

He recorded an open verdict, saying he could not be certain she had intended to kill herself this time.

Dr Julian Candy, psychologist at St John's, told the inquest that Mrs Mason believed that people around her knew certain things about her past.

'Mrs Mason suffered from self-blame, guilt and depression, and discussed suicide with me on several occasions,' said Dr Candy.

'She had an intense feeling of hopelessness. A number of deaths in the family including her mother's and stepfather's caused her great distress.'

Pathologist Dr Andrew Tudway said that Mrs Mason's stomach was almost entirely corroded and her mouth and throat ulcerated by the formic acid in the kettle descaler.

Figure 10.1. A headline reminds us of the potential toxicity of household substances. In this case kettle descaler containing corrosive formic acid was taken intentionally.
Taken from the newspaper, *Aylesbury Plus,* 16 September 1987, with permission.

Carbon Monoxide

This highly toxic gas is still a major cause of poisoning deaths in the UK despite the fact that a major source, coal gas, has been replaced by natural gas. Several hundred deaths occur annually and carbon monoxide poisoning is still the major cause of death from poisoning in children. The gas is found in car exhausts and results from the inefficient burning of hydrocarbon fuels in engines as well as in stoves and boilers especially where there is poor ventilation. There have, in fact, been a number of poisonings recently, some with fatal outcomes which have been highlighted in the press and on television in the UK. In one recent case, a birds nest had blocked the chimney of a holiday cottage and so when the fire was lit, the lack of ventilation caused the fire to produce carbon monoxide. All the members of the family subsequently died in the house from carbon monoxide poisoning (see Emsley, Bibliography).

Carbon monoxide is a very simple poison and its mode of action has been understood for many years. Poisoning with it is also relatively simple to treat. In 1895 Haldane conducted experiments with carbon monoxide using himself as a subject. He carefully documented the effects as the concentration of carbon monoxide in his blood stream rose towards lethal levels. Through his studies and the earlier work of Claude Bernard in 1865 we now know much about the mechanism of action of carbon monoxide as a poison.

Carbon monoxide reacts with the haemoglobin in red blood cells. It does this by binding to the iron atom of the haem molecule in the same way as oxygen (Figure 10.2). Carbon monoxide binds more avidly than oxygen, however, and the resulting haemoglobin cannot carry out its normal function of transporting oxygen. Therefore, there is competition for binding to haemoglobin between oxygen and carbon monoxide and the concentration of the latter is a crucial factor. As carbon monoxide binds much more avidly to the

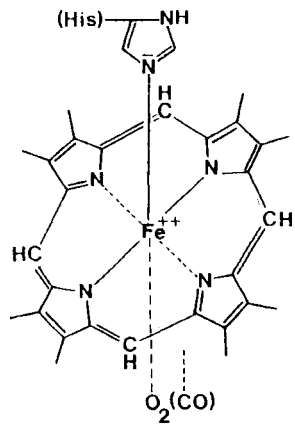

Figure 10.2. The haem moiety of the haemoglobin molecule showing the binding of the oxygen molecule to the iron atom. As shown in the diagram, carbon monoxide (CO) binds at the same site as the oxygen molecule, but it is bound much more tightly. (His is the side chain of the amino acid Histidine.)

iron atom the concentration of the toxic gas necessary to saturate the haemoglobin is much less than that of oxygen in air. This was determined by Haldane and is shown by his equation:

$$\frac{[COHb]}{[HbO_2]} = \frac{M[P_{CO}]}{[P_{O2}]}$$

where M is 220, at pH 7.4 in man. [COHb] and [HbO_2] are the concentration of carboxyhaemoglobin and haemoglobin respectively. [Pco] and [Po_2] are the partial pressures of carbon monoxide and oxygen respectively.

Consequently, for 50 per cent saturation of haemoglobin with carbon monoxide, where 50 per cent of the haemoglobin in the blood is carboxyhaemoglobin, the concentration of carbon monoxide need only be 1/220 of that of oxygen in the air or about 0.1 per cent. A level of 50 per cent carboxyhaemoglobin would certainly be lethal for a human after a relatively short time. As carbon monoxide is also odourless and tasteless, it is an extremely dangerous poison. The result of carbon monoxide poisoning is that the tissues are starved of oxygen and suffer ischaemic damage. Energy production is reduced, only anaerobic respiration being possible and, hence, there is an accumulation of lactic acid causing acidosis.

The symptoms of carbon monoxide poisoning depend on the concentration to which the victim is exposed. There is often headache, mental confusion, agitation, nausea and vomiting. The skin becomes characteristically pink due to the carboxyhaemoglobin in the blood. The victim hyperventilates and will eventually lose consciousness and suffer respiratory failure. There may be brain and cardiac damage resulting from the hypoxia, and also cardiac arrythmias and other malfunctions of the heart can occur.

Treatment is relatively simple, especially for mild cases and involves removing the victim from the source of carbon monoxide, or causing fresh uncontaminated air to be introduced into the immediate environment. As the

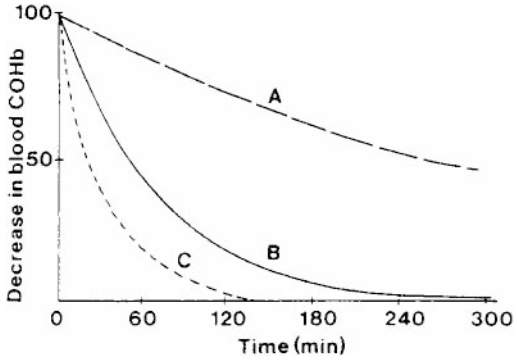

Figure 10.3. The dissociation of carboxyhaemoglobin in the bloodstream of a patient poisoned with carbon monoxide. The graphs show the effects of breathing air (A), oxygen (B) or oxygen at increased pressure (2.5 atmospheres) (C) on the rate of dissociation.
Data from Meredith, T.J. and Vale, J.A. (1981), Antidotes, pp. 33–45, Figure 5.9, in *PoisoningDiagnosis and Treatment,* J.A.Vale and T.J.Meredith (Eds), London: Update Books.

concentration of carbon monoxide in the ambient air and hence the inspired air falls, the carboxyhaemoglobin dissociates and the carbon monoxide is expired. This rate of loss of carbon monoxide from the blood (half-life around 250 minutes) can be increased by making the patient breathe oxygen rather than air (half-life reduced to 50 minutes). For severe poisoning cases the use of oxygen at elevated pressures (2.5 atmospheres) will reduce the half-life of elimination to around 22 minutes (Figure 10.3). The reductions in half-life with oxygen at elevated concentrations and pressures can be predicted from the Haldane equation.

Antifreeze: Ethylene Glycol

Antifreeze liquid contains one or sometimes two toxic compounds which may feature in poisoning, either accidental or suicidal. The major constituent of antifreeze is ethylene glycol but this may sometimes be combined with methanol.

Ethylene glycol is a dihydric alcohol and a sweet tasting liquid, which has effects on the state of mind similar to those of ethanol. It may sometimes be consumed instead of normal alcohol by alcoholics. It is very toxic, however, and fatal poisoning may occur after as little as a cupful of antifreeze.

It is not intrinsically toxic but requires metabolism. There are various intermediate metabolic products terminating in oxalic acid (Figure 2.13). The intermediate acidic metabolites cause acidosis directly and also by increasing the level of NADH which is then utilized in the production of lactic acid. As well as being acidic, oxalic acid damages the brain by crystallizing there. Calcium oxalate crystals may also form in the kidney tubules and cause damage.

The first step in the metabolism of ethylene glycol involves the enzyme alcohol dehydrogenase and this provides the key to the treatment of poisoning. The preferred substrate for this enzyme is ethanol and so when ethanol is present *in vivo* it is preferentially metabolized. The metabolism of ethylene glycol is therefore blocked. The treatment for ethylene glycol poisoning is, therefore, administration of ethanol (whisky or some similar spirit in an emergency) by mouth or pure ethanol can be infused intravenously until all of the ethylene glycol has been excreted from the body. Haemoperfusion or haemodialysis may also be used to remove ethylene glycol from the body.

The Austrian wine scandal of 1986 involved ethylene glycol being used to sweeten the wine. The amounts used however would not have been acutely toxic although the chronic toxic effects of ethylene glycol, deposition of calcium oxalate kidney stones, might have been a cause for concern. The fact that wine also contains ethanol raises the interesting possibility that the toxicity would be reduced by the continued presence of the antidote!

Methanol, which may sometimes be present in antifreeze, is also found in methylated spirits (industrial spirit). It is also very toxic due to its metabolism to formaldehyde and formic acid:

$$CH3OH \rightarrow HCHO \rightarrow HCOOH$$

The former may cause blindness if the dose of methanol is not rapidly fatal. Again, as methanol is metabolized by alcohol dehydrogenase, treatment of poisoning involves administration of ethanol and correction of metabolic acidosis, in the same way as for ethylene glycol poisoning. It may be that the presence of large amounts of ethanol in methylated spirits confers some protection on those unfortunates who drink it either accidentally or intentionally as a substitute for alcoholic drinks.

Alcohol

Ethanol is perhaps our most ubiquitous drug and features in far more cases of poisoning or adverse effects than more notorious drugs such as heroin or cocaine. Many members of the general public probably do not consider alcohol to be a drug but it has both pharmacological and toxic effects. The effects of ethanol vary with the dose and there is some evidence that it may even have beneficial effects at low doses (see Saul, Bibliography). Ethanol is rapidly absorbed from the gut and distributes into body water. About 90 per cent is metabolized to acetaldehyde, acetic acid and then carbon dioxide and water at a rate of 10–20 ml hr^{-1}. After acute doses the major effect of ethanol is depression of the central nervous system. The pharmacological effects may be desirable if the dose is low but after higher doses the effects become exaggerated, progressing through increasing visual impairment, muscular incoordination and slowed reaction times and after large, toxic doses unconsciousness and death. A lethal dose in an adult is between 300 and 500 ml (equivalent to about a litre of whisky) if taken in less than an hour. Large doses may cause a reversible change in the liver known as fatty liver where triglycerides accumulate in the hepatic cells. The effects of ethanol can be related to the plasma level and range from mild intoxication at levels of between 500 and 1500mg 1^{-1} to coma and death at levels of more than 5000 mg 1^{-1}. At this level respiratory depression, hypotension, hypothermia and hypoglycaemia will occur. The hypoglycaemia is largely due to the inhibition of gluconeogenesis by ethanol.

After chronic exposure to ethanol the liver is the main target organ although the brain may also suffer. Cirrhosis of the liver occurs after chronic abuse of alcohol and in this toxic effect the architecture of the liver is altered by the replacement of normal tissue by collagen so that it functions less efficiently. The biochemical basis of the hepatic effects of ethanol are complex and involve alterations in the level of cofactors such as NADH and disturbances in intermediary metabolism. Thus, a shift in the redox potential with an increased NADH/NAD ratio leads to impaired mitochondrial oxidation of fatty acids such that more triglyceride is synthesized. Ethanol, however, causes various other metabolic effects which contribute to the toxicity.

Ethanol is now classified as a carcinogen from epidemiological evidence in man which associates cancer of the liver and parts of the gastrointestinal tract to its use.

Glue Sniffing and Solvent Abuse

Glue sniffing and solvent abuse became common among teenagers during the 1970s and 1980s. Many different types of solvent were used with correspondingly different effects. Toluene is a solvent commonly found in glues and mainly causes narcosis. Some of the halogenated solvents such as those used as aerosol propellants are more hazardous, as they may cause sensitization of the myocardium to catecholamines leading to ventricular arrhythmias. This effect can lead to sudden death from heart attack, especially under certain conditions such as fright, and over 120 deaths occurred in the UK in 1991. Solvents are found in many different household products including glues, paints, paint strippers, aerosols, varnishes, cleaning fluids and fire extinguishers and so the scope for abuse is enormous. The acute toxic effects of solvents are mainly narcosis or anaesthesia and the more serious sensitization of the heart. The chronic effects are in many cases unknown but may include changes in personality and general morbidity. There are, however, known cases of chronic cardiac toxicity due to trichloroethane exposure.

Questions

1. Describe the mechanism of toxicity of carbon monoxide and explain how it is treated. Why is it a potentially very dangerous compound?
2. Compare and contrast the toxicology of ethylene glycol and methanol.
3. 'Ethanol is a toxic drug widely available to the general public.' Discuss this statement.

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