CHAPTER 11

Household products

Chapter outline

In this chapter some examples of poisoning with household products and miscellaneous chemicals and treatment of poisoning will be considered:

- Introduction
- Carbon monoxide
- Antifreeze: ethylene glycol
- Cyanide
- Alcohol
- Glue sniffing and solvent abuse
- Treatment of poisoning and antidotes

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 8) 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. Many deaths in children under 10-years of age and adults 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 11.1). 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.

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 a 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).

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 11.1 *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 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 11.2). Carbon monoxide binds *more avidly* than oxygen, however, and the resulting carboxyhaemoglobin 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 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:

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

where **M is 220**, at pH 7.4 in man. [COHb] and [HbO_2] are the concentration of carboxyhaemoglobin and haemoglobin respectively. [Pco] and [P_{O2}] 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

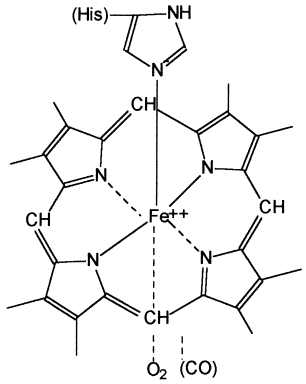

FIGURE 11.2 *The haem moiety of the haemoglobin molecule showing the binding of the oxygen molecule to the iron atom. As shown in the diagram, carbon monoxide (CO) binds at the same site as the oxygen molecule, but it is bound much more tightly. (His is the side chain of the amino acid Histidine.)*

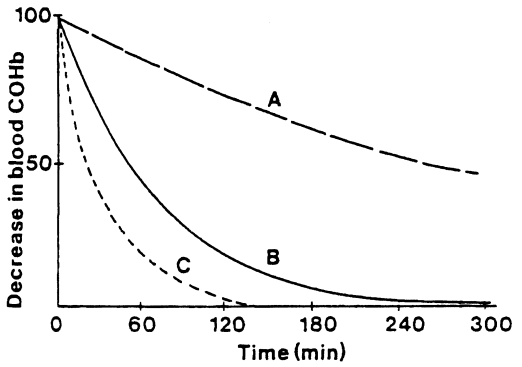

FIGURE 11.3 *The dissociation of carboxyhaemoglobin in the bloodstream of a patient poisoned with carbon monoxide. The graphs show the effects of breathing air (A), oxygen (B) or oxygen at increased pressure (2.5 atmospheres) (C) on the rate of dissociation. Data from Meredith, T. J. and Vale, J. A. (1981), Antidotes, pp. 33–45, Figure 5.9, in* Poisoning – Diagnosis and Treatment, *J. A. Vale and T. J. Meredith (Eds), London: Update Books.*

the immediate environment. As the 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 11.3). 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 3.2). 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**:

$$CH_3OH \rightarrow HCHO \rightarrow HCOOH$$

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.

Cyanide

Cyanides of various kinds are found both naturally in the environment and as a result of human activity. Cyanide is often associated with homicidal or suicidal poisoning and there are some well known cases, but there are many ways in which humans can be exposed. For example, fatalities in fires may result from cyanide poisoning because certain substances, such as plastics, generate **HCN** gas when burning. Cyanides are extensively used in the metal industry and in mining for the extraction of gold, for example. Therefore there is potential for occupational poisoning and contamination of the environment with cyanides. Indeed, there are several documented cases of pollution of rivers from mining operations. In February 2000 a serious case of river pollution occurred in Romania where cyanide from a mining operation leaked into the river Tiza destroying most of the fish and then as it moved down the river it also destroyed many fish in the rivers and tributaries in Hungary. Inorganic cyanides are often found in laboratories and were once used around the home for the removal of pests such as wasps nests.

Naturally occurring cyanides, such as **amygdalin**, which is a **cyanogenic glycoside**, are found in the kernels and pips of fruits such as apricots, peaches and apples. The most significant natural source of cyanide, however, is **Cassava** which is an important food crop eaten in Africa. The Cassava plant has to be prepared with care in order to avoid poisoning with cyanide. This is usually done by washing and soaking the plant in water. This allows the enzymes in the plant to degrade the cyanogenic glycoside, **Linamarin**, and then the water soluble cyanide ion is washed out. If this is not done and the plant is eaten without this careful preparation, then the cyanogenic glycosides can be degraded by the enzymes in the human gut and the cyanide released in sufficient quantities to poison the unfortunate person who eats the plant.

Hydrogen cyanide is rapidly fatal when inhaled as a vapour but a dose of about 300 mg sodium or potassium cyanide when taken by mouth might take some time to kill the victim depending on factors such as the presence of food in the gut.

The mechanism by which cyanide is toxic involves inhibition of the respiratory chain in the mitochondria. The cyanide binds to **cytochrome aa_3** and blocks the movement of electrons down the respiratory chain (Figure 11.4). This means that cellular metabolism is compromised and ATP production is drastically reduced, although not stopped completeley. Organs such as the brain and heart are affected particularly as they require ATP and have limited capacity to cope with a deficit. The heart has only enough ATP to last three

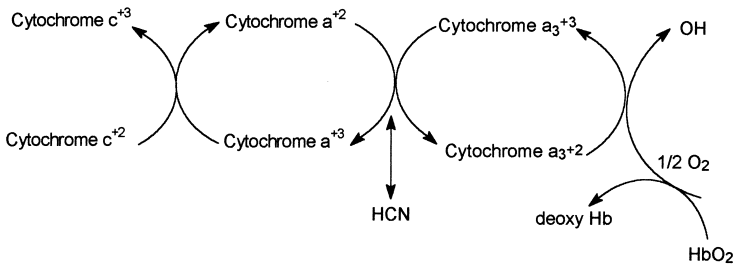

FIGURE 11.4 *The site of action of cyanide in the mitochondrial electron transport chain. Hb: haemoglobin; HbO_2: oxyhaemoglobin.*

minutes and so cyanide may cause death as a result of heart or respiratory failure. There are several antidotes to cyanide poisoning. One method of treatment is to boost detoxication by giving the patient **thiosulphate** which is involved in an endogenous reaction catalysed by the enzyme **rhodanese**. This converts cyanide into thiocyanate which is less toxic and is excreted into the urine. Increasing the availability of thiosulphate increases the capacity of this reaction to detoxify cyanide. Other methods of treatment involve the use of substances to remove the cyanide such as chelating agents like **dicobalt edetate** (see antidotes, this chapter).

Chronic, non-fatal exposure to cyanide such as that which occurs as a result of eating Cassava may cause other pathological effects such as paralysis due to damage to the spinal cord. This is a significant toxic effect in some parts of Africa where Cassava is a staple diet. An additional factor in the poisoning is the poor diet in parts of Africa where Cassava is eaten which means that local people may be deficient in sulphur amino acids and hence have low levels of thiosulphate. Consequently, they are less able to detoxify the cyanide. A secondary effect is **goitre** (enlarged thyroid) due to the thiocyanate detoxication product of cyanide causing interference in the uptake of iodine essential for the function of the thyroid.

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 h^{-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 1500 mg l^{-1} to coma and death at levels of more than 5000 mg l^{-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.

Antidotes and the treatment of poisoning

This section is relevant to a number of the examples and substances covered in various chapters and consequently is placed in this penultimate chapter of the book.

Poisoning of humans may result from the accidental or intentional overdosage with drugs or other chemicals. This usually, but not always, follows oral ingestion and is normally an acute poisoning episode. Such poisoning requires treatment and falls within the remit of clinical toxicology. However, repeated or chronic exposure may lead to acute poisoning, and exposure via inhalation or skin absorption can also feature in acute poisoning cases.

For some chemicals there are specific antidotes which can be administered, and which reduce the toxicity of the substance swallowed. There are, however, relatively few of these. In most cases poisoning is treated by means other than the use of antidotes.

GENERAL METHODS

1 One of the most common general methods for the treatment of poisoning is the use of substances to remove the toxicant from the gastrointestinal tract. This can be done in one of two ways:

   a Use of **emetics**. These substances cause the poisoned patient to vomit and so eject the poison from the gastrointestinal tract. **Syrup of Ipecachuana** is one such emetic which can be used in children.

   b The use of **absorbants**. Absorbants such as **Fullers Earth** or activated charcoal remove the substance from the gastrointestinal tract by absorption and strong binding. This treatment is effective for many different types of poisons.

2 Another commonly used general method of treatment is to increase elimination from the body. This can be done by changing the acidity or alkalinity of the urine or by simply increasing the urine flow. The urine flow is increased by making the patient imbibe large amounts of water or by injecting fluid intravenously. So-called **forced diuresis** is not without hazard, however. Increasing the elimination by changing the acidity or alkalinity of the urine is effective for drugs and chemicals or their metabolites which are charged, i.e. acids or bases. The basis of this is that acids will be more soluble in urine which has an alkaline pH and vice versa, bases will be more soluble in acidic urine. Changing the urine to a more alkaline pH can be achieved by giving the poisoned patient **sodium bicarbonate** either orally or by intravenous injection. This is used effectively with **aspirin** poisoning (see Chapter 5) or **barbiturate poisoning**. Conversely, the urine may be acidified by giving the patient **ammonium chloride**. This may be used for the treatment of **amphetamine** poisoning. As well as increasing elimination into the urine this technique also changes the pH of the plasma which may increase distribution of the drug out of the tissues into the blood. This occurs in the case of both phenobarbital and salicylic acid, which being acidic, become more ionized in the plasma as the pH rises. The ionized forms of these drugs are less able to penetrate the tissues, especially the brain, thereby reducing the toxicity.

3 Drugs and toxicants can also be directly removed from the blood by the techniques of **haemodialysis** and **haemoperfusion**. Both involve the passage of the blood of the poisoned patient through an apparatus which either allows diffusion of the toxicant through a semi-permeable membrane into another fluid (haemodialysis) or removes the toxicant from the blood by adsorption onto charcoal or a resin (haemoperfusion).

ANTIDOTES

Antidotes act in various ways but there are few in relation to the number of toxicants available. Antidotes can act in one of a number of ways.

1 **Chelating agents**. These act by reacting with the compound to form a water soluble complex which can be eliminated. The toxicant is thereby removed from the body. Examples of chelating agents used for the treatment of poisoning are **dicobalt edetate** used for the treatment of cyanide poisoning (see this chapter), penicillamine used for treating lead poisoning (see Chapter 9) and **dimercaprol** used against the poison

gas Lewisite and also for heavy metal poisoning.

2 Antidotes which specifically **increase** the **detoxication** of a reactive metabolite. A good example of this is **N-acetylcysteine**, used for the treatment of paracetamol overdoses (see Chapter 5). N-acetylcysteine increases the detoxication of the reactive metabolite of paracetamol (p-benzoquinoneimine) by increasing the production of glutathione. Therefore the reactive metabolite is diverted away from the toxic interaction with liver protein and the toxicity is reduced. Stimulation of the detoxication of cyanide by increasing its metabolic conversion to thiocyanate using **thiosulphate** administration is an alternative antidote for cyanide poisoning (see this chapter).

3 **Inhibition of metabolism**. An alternative antidotal strategy for situations where a chemical is toxic as a result of metabolism is to block the metabolic transformation. For example, ethylene glycol, often used in antifreeze, is toxic as a result of metabolism first by **alcohol dehydrogenase** and then by other enzymes with the final toxic product being **oxalic acid**. Competitive inhibition of alcohol dehydrogenase with **ethanol** blocks this metabolism and allows the ethylene glycol to be eliminated unchanged. Poisoning with **methanol**, which also involves metabolism by alcohol dehydrogenase, can be treated similarly.

4 **Antidotes acting on a receptor**. **Morphine** poisoning, which causes respiratory depression, can be treated by blocking the receptor where it acts with the drug **naloxone**. Another example is the treatment of the cholinergic crisis, which occurs as a result of **organophosphate** poisoning, with **atropine**, which antagonizes the effect of the excess acetylcholine on receptors (see Chapter 8).

5 **Reversal of receptor blockade** or inhibition. Again with poisoning with organophosphates such as **parathion**, the inhibition of the acetylcholinesterase by the metabolite paraoxon can be treated with **pralidoxime** which removes the organophosphate from the enzyme, thereby regenerating it. Another example is the treatment of **carbon monoxide** poisoning with **oxygen**. Because the binding of the carbon monoxide to the haemoglobin is reversible, with a sufficiently high concentration of oxygen this can be displaced. The presence of carbon dioxide also helps to facilitate the dissociation of the carboxyhaemoglobin.

6 Use of **antibodies** or **antibody fragments**. For toxins such as snake venoms, **antivenoms** may be available which specifically bind the protein(s) in the venom. Occasionally antibodies may also be used for the treatment of poisoning with drugs such as **digoxin**.

Summary and learning objectives

There are many chemicals in use in and around the modern home which are potentially poisonous, including potent pesticides, solvents, and strong detergents and acids. A not infrequent culprit of poisoning, often leading to fatalities in the home, is ***carbon monoxide*** resulting from poor ventilation of stoves, fires and boilers and also found in car exhaust. This gas binds strongly to haemoglobin, thereby competing with and depriving the tissues of oxygen. Symptoms include severe headache, confusion,

acidosis, unconsciousness and death from respiratory/cardiac failure. ***Ethylene glycol*** used in antifreeze may sometimes be ingested, leading to acidosis, kidney and brain damage due to metabolism to toxic aldehydes and acids. A chemical which may be found in the home and also in plants (e.g. Cassava) as well as in factories and laboratories is ***cyanide*** (usually found as a salt). Cyanide binds to cytochrome aa_3 in the mitochondrion, thereby stopping the flow of electrons and depleting ATP. Death is usually rapid as a result of cardiac or respiratory failure.

Alcohol is a ubiquitous but potentially toxic drug which accounts for many cases of liver damage (cirrhosis).

Solvents and other volatile substances may be inhaled by 'glue-sniffers', leading to unconsciousness or sometimes heart failure.

Treatment of poisoning involves use of emetics, absorbants, diuresis and pH manipulation, haemodialysis or haemoperfusion. For some toxic chemicals antidotes exist such as chelating agents, enzyme inhibitors, receptor blockers, antibodies, and agents that increase detoxication or facilitate displacement of toxicants from binding sites.

Questions

Q1. Indicate which of the following are true and which false.
Ethanol is used as an antidote for the treatment of ethylene glycol poisoning because it:

a facilitates the excretion of ethylene glycol
b blocks the metabolism of ethylene glycol
c increases the detoxication of ethylene glycol
d chelates ethylene glycol
e none of the above.

Q2. Carbon monoxide is poisonous because:

a it binds to cytochrome aa_3 in the mitochondria
b it binds to haemoglobin
c it causes respiratory alkalosis
d it causes a failure of respiration
e it forms methaemoglobin.

Q3. Indicate which of the following are true with reference to ethylene glycol:

a it is an alcohol
b it causes metabolic acidosis
c it is metabolized to lactic acid
d it crystallizes in the brain
e it is metabolized by aldehyde dehydrogenase.

Q4. Indicate which of the following are true:

a solvents can cause death by sensitization of the myocardium
b alcohol is toxic because it stimulates the central nervous system
c alcohol is metabolized to formic acid
d the major target organ for methanol toxicity is the liver
e large doses of alcohol cause blindness
f high doses of alcohol lower blood sugar.

Q5. Which of the following has been used as an antidote:

a Lewisite
b dicobalt edetate
c thiocyanate
d N-acetylcysteine
e pralidoxime.

SHORT ANSWER QUESTION

Q6. Write notes on the various types of general treatment for poisoning.

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