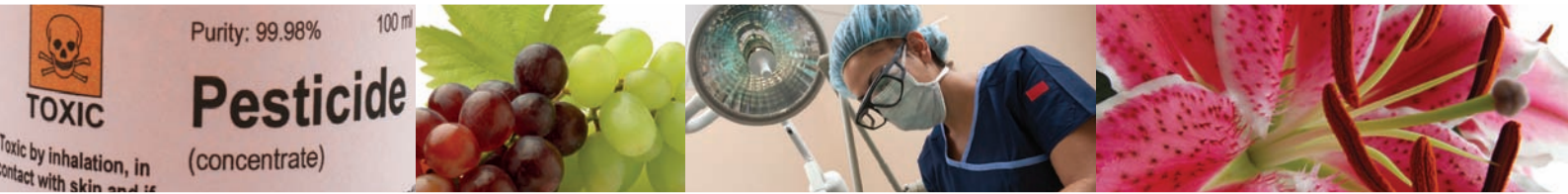


# Toxic Times



**Welcome to our newly named 'Toxic Times', the seasonal Newsletter from the Veterinary Poisons Information Service.**



Many thanks to all who took the time to send in suggestions for a name. The winning name was supplied by Doug Alexander of AlphaPet Veterinary Clinics. Well done Doug, we have added 5 case credits to your account as a thank you. We have also awarded a runner-up prize of 2 case credits to Dave Fisher from Brentknoll Veterinary Centre in Worcester, for his suggestion 'Toxicity Times' – a very close match to the winning title.

The festive season can be difficult for pets. Their usual home environment changes, becoming cluttered with strange trees, flowers, bright decorations, interesting packages, odd noises (crackers / fireworks), crisps, nuts, and attractive food aromas.

In addition there may be unfamiliar visitors. Consequently, pets may not be as closely observed as usual. Strong cardiac or other medicines taken by elderly relatives may pose a risk to animals if left around the home. Dustbins and food waste can be of interest to the scavenger too. Many foods are toxic to animals – chocolate, onions, macadamia and other nuts, blue cheese, fruit cakes and puddings, mince pies, and so on. This newsletter contains some information on several seasonal poisonings, but accidents will inevitably occur. However, remember that the VPIS office is staffed 24 hours a day, even over Christmas and New Year, to provide you with case-specific poisons advice whenever you need it. Merry Christmas!

**By Alex Campbell**





# Christmas trees

Christmas trees including those of the *abies* (fir), *pinus* (pine) and *picea* (spruce) species all belong to the Pinaceae family. They are considered to be of low toxicity, but ingestion may cause a mild gastrointestinal upset (vomiting and diarrhoea). However ingestion of plant material could cause a mechanical obstruction if a large amount is ingested or physical injury from sharp needles. Contact dermatitis has been reported in humans but this is more likely to occur in those who have chronic exposure (e.g. florists, wood workers) and therefore unlikely to occur in companion animals. Christmas tree preservative is similar to cut flower food and is added to the water to prolong the life of the tree. Ingestion of the preservative is unlikely to be a problem if ingested.

By Karen Sturgeon

# Batteries

Ingestion of batteries by pets is common at this time of year as they are found in many things under the tree (particularly children's toys). The severity of poisoning is generally dependent on the type of battery. The batteries most frequently eaten are button batteries or small cylindrical batteries (e.g. AA or AAA). The brand, type and size of the battery should be determined if possible. The battery code is also important and should be determined as VPIS may be able to identify a button battery by its code. Most batteries contain either strong liquid acids / alkalis and many have significant metal content. Ingestion may result in local burns and / or corrosive injury of the throat, oesophagus and stomach. In rare cases, systemic metal poisoning may occur (with mercury button batteries presenting the most significant risk). Electrical discharge is a primary cause of injury; the high electrolyte composition of tissue fluid allows batteries to generate a direct current, causing cell death and electrical burns. Corrosive damage to the battery casing may also occur in the gut; leakage of the contents may again cause severe caustic injury. However, most batteries pass through the gastrointestinal tract uneventfully. Complications usually arise when the battery becomes lodged; most commonly, and with the most serious complications, in the oesophagus. Treatment is largely supportive and may involve repeat abdominal x-rays and, in some cases, surgical removal of the battery. We would recommend contacting the VPIS for specific guidelines determined by the type of battery ingested.

By Lennie Hawkins



# Cannabis

Recent analysis of VPIS data shows that incidents of cannabis poisoning are relatively common around Christmas – some may argue that it is, after all, the season to be jolly! Most instances involved dogs eating their owner's, or a visitor's, personal "stash". Other reports have included sniffer dogs that ingested this drug while in training or even on the job.

Cannabis is the collective term for the psychoactive substances obtained from the dried leaves and flowers of the plant *Cannabis sativa*. The plant itself contains around 1-6% THC (delta-9-tetrahydrocannabinol) – the main toxic component. The mechanism of action of THC is not clear, but may be linked to changing levels of biogenic amines in the CNS.

Onset of effects is usually rapid (1-3 hours) and may persist for one to three days. Effects may be delayed if the cannabis is enclosed in cling film or a bag. Animals may display behavioural changes such as agitation, barking and may also appear to hallucinate. Some pets appear to develop a heightened sense of smell and may be more interested in sights and sounds in general. Other effects include: weakness, ataxia, drowsiness, dilated pupils, photophobia, tachycardia and hyperaesthesia. Shaking, twitching and convulsions have been reported in some instances.

By Nick Sutton



# Chocolate (theobromine) toxicity



It's that time of year again when the VPIS prepares for the onslaught of chocolate enquiries. Our chocolate calls increase dramatically over the Christmas period, with nearly 350 theobromine enquiries taken last December alone. Ensure you have an adequate supply of activated charcoal in stock, as this can be particularly useful in the management of theobromine toxicity.

## VPIS rough treatment guide

You may want to consider treatment in a dog that has ingested more than the quantities in the adjacent table. These treatment doses are based on the maximum theobromine content of the various types of chocolate.

Type of chocolate	Rough treatment dose
White chocolate	2.2g/kg
Milk chocolate	9g/kg
Dark/plain chocolate	1.25g/kg

By Nick Sutton

## Carbon monoxide

Now the weather has turned wintery and everyone is at home with their heating on full, we need to be on the lookout for carbon monoxide poisoning. Carbon monoxide (CO) is a colourless, odourless, tasteless, non-irritating, flammable gas, formed when there is incomplete combustion of organic fuels. Many incidents of CO poisoning are associated with the use of badly installed, poorly maintained or malfunctioning domestic combustion appliances using gas, oil or solid fuel, or the use of such appliances in inadequately ventilated areas. Animals may be exposed to carbon monoxide through sleeping close to a faulty heating appliance.

### Carbon monoxide (CO) causes its toxic effects in two main ways:

1) by replacing oxygen on the haemoglobin molecule forming carboxyhaemoglobin. This reduces the amount of oxygen, the haemoglobin molecule can carry, and results in impairment of oxyhaemoglobin formation and causes cellular hypoxia.

2) When CO binds to the haem site of a haemoglobin molecule it undergoes a conformational change, with a resulting increase in the affinity of the remaining haem groups for oxygen. This prevents the release of bound oxygen to peripheral tissues.

CO saturates myoglobin at a concentration 3 times greater than it saturates skeletal muscle. The resultant hypotension and myocardial depression cause ischaemia and potentiates the hypoxia induced by impaired oxygen delivery. Animals may also exhibit vomiting, drowsiness, tachycardia, tachypnoea and ataxia. Deafness and blindness can occur and in cats anisocoria and protrusion of the third eyelid has been reported. In severe exposures lactic acidosis, hypotension, convulsions, coma and death may occur. The mainstay of treatment is administration of 100% oxygen for a prolonged period.



While it is important to treat any animal coming in with suspected CO poisoning promptly, the owners of the animal may also require medical attention. Any suspect appliance should be switched off and serviced. Cats and dogs (and children) have higher alveolar ventilation due to an increased metabolic rate and may show signs and symptoms of carbon monoxide poisoning before adult humans.

By Karen Sturgeon

## Forthcoming events

VPIS will be exhibiting at the following event – why not visit our stand and meet the team.

VPMA Congress, 27-29th January 2011, Stand 16, Chesford Grange Hotel, Kenilworth. The VPMA Annual Congress, now in its 17th year, is established as the event in the veterinary calendar for practice managers, owners and practice staff. The 2011 Congress has been re-vamped to give you a 'better than ever' programme of top quality management CPD in a dedicated, relaxed, professional environment away from the hustle and bustle of practice. For further details visit [www.vpma.co.uk](http://www.vpma.co.uk)



# Antifreeze in cats and dogs

Winter is associated with an increase in antifreeze exposure in domestic animals. It is a liquid product added to water in radiators of vehicles to prevent freezing and improve hot weather performance. Hence antifreeze is readily available in the domestic environment as drivers top up or drain their vehicle radiators. Most antifreeze contains methanol or ethylene glycol.



The toxicity of methanol is well recognised in primates (including humans) but it is much less toxic to cats and dogs. This is because they are able to rapidly metabolise methanol to non-toxic compounds. Ingestion of methanol in cats and dogs usually causes inebriation similar to that seen with ethanol but serious poisoning is very rare.

Ethylene glycol ingestion, however, is very dangerous in domestic animals, particularly cats. It is sweet-tasting and very palatable. Even a relatively small quantity can cause serious toxicity. It is not ethylene glycol itself which is hazardous but the metabolites produced by the action of alcohol dehydrogenase (ADH) on the parent compound. ADH converts ethylene glycol to glycoaldehyde which is then metabolised to glycolic acid. This is the principle cause of acidosis. One of the metabolites of glycolic acid is oxalate, which causes renal damage, and hypocalcaemia by binding to calcium to form calcium oxalate.

Clinical effects from ethylene glycol poisoning occur in 3 stages. Stage 1 (30 minutes -12 hours) presents with ataxia, tachycardia, weakness and convulsions. There may also be polydipsia, polyuria, dehydration and tachypnoea. In

stage 2 (12-24 hours) there is tachypnoea, tachycardia and pulmonary oedema. There may be a period of transient recovery followed by anorexia, severe depression, coma and convulsions. In the final stage (24-72 hours) there is oliguria, azotaemia, vomiting, anorexia and severe depression. Laboratory findings include increased osmolality, acidosis, raised urea and creatinine, low urine specific gravity, calcium oxalate crystals in the urine, proteinuria, glucosuria, haematuria and albuminuria, hyperglycaemia, hypocalcaemia, hyperphosphataemia and hyperkalaemia.

Therapy for ethylene glycol is principally aimed at blocking the action of ADH to prevent the formation of the toxic metabolites. This is achieved by the administration of ethanol, the preferred substrate of ADH. This allows renal excretion of the unmetabolised parent compound before toxic metabolites can be formed. The longer the delay between ingestion and initiation of treatment the less favourable the prognosis. Unfortunately, in many cases ingestion is not witnessed and the animal presents in the third stage of poisoning. Once renal damage has occurred antidotal therapy is of limited benefit. Most feline cases reported to VPIS have a fatal outcome.

By Nicola Bates

## Beat the VAT man!

Remember that the VAT rate rises to 20% on the 4th January 2011. The VPIS payment system for its case credits will reflect the new and increased tax rates automatically from midnight on the 3rd January 2011. Practices running low on credit may wish to purchase some additional credits at the old rate before this change!