BVSC & AH STUDY BOOK 6 OF 7

Veterinary Toxicology

Seventeen chapters on poisons and poisoning: principles, pesticides, rodenticides, plants, metals, feed and gas hazards, photosensitisation, household and pet toxins, venoms, drug toxicities, antidotes, environmental toxicology and medico-legal work. Each chapter ends with a 10-question self-test.

17 chapters | **170** practice questions | **No** dose tables

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Start here

Pick a chapter from the list on the left (or the top bar on a phone). Chapter 1 gives the principles and Chapter 15 is a quick reference for antidotes and decontamination. The other chapters group poisons by source: pesticides, rodenticides, plants, metals, feed and gases, household and pet hazards, venoms and drugs. Each chapter has six parts: Learn, Concepts, Compare, Species, Quiz and Revise. Your quiz progress is saved on this device only. In a real poisoning, contact a veterinary poison service or toxicologist at once.

CHAPTER 1 OF 17

General principles of toxicology

How poisons enter, move through and leave the animal, how dose and response are related, and how toxicity is tested. The chapter also sets out the logic of decontamination, supportive care, antidotes and history taking in a poisoned patient.

After this chapter you can

- Describe toxicokinetics in terms of absorption, distribution, biotransformation and excretion, and link them to toxicity

- Explain the dose-response relationship, threshold, LD50 and the limits of LD50 as a measure of safety
- Distinguish acute, sub-acute and chronic toxicity studies and state what each is designed to find out
- Explain the reasoning behind decontamination, supportive care and the use of antidotes
- Take a structured history in a suspected poisoning case and know when to seek urgent specialist help

What toxicology asks

Toxicology studies the harmful effects of chemicals on living animals. The old statement that 'the dose makes the poison' remains the foundation: almost any substance is harmless at a low enough exposure and harmful at a high enough one. A **toxicant** is any substance that causes harm. A **toxin** is a toxicant made by a living organism, such as a plant, fungus, bacterium or snake.

In practice the veterinarian asks four linked questions. What was the exposure? How much reached the body? What is it doing at its target? What can be done to stop it or reverse it? Every section of this chapter answers one of those questions.

Toxicokinetics

Toxicokinetics describes what the body does to the poison. The same four steps as in pharmacokinetics apply, but the amounts are often large enough to overload normal handling.

- **Absorption.** Poisons enter by the gut, skin, lungs, eye or injection. Route matters. A lipid-soluble pesticide can be absorbed through the skin of a dog bathed in it, and inhaled gases act within minutes. Food in the stomach, gut motility and the form of the product (liquid, powder, granule) change the speed.
- **Distribution.** Fat-soluble agents such as organochlorines are stored in fat and released slowly, so signs can appear long after exposure, for example when the animal loses weight. Binding to plasma protein holds a poison in the blood but can be displaced by other drugs.
- **Biotransformation.** The liver usually converts poisons to water-soluble products that can be excreted. Sometimes this makes the agent *more* toxic, which is called

bioactivation. Phosphorothioate insecticides are converted to the more active oxon form. Species differ greatly here: cats are poor at glucuronidation, so phenols and some drugs linger and cause toxicity.

- **Excretion.** Kidney, bile, lungs and milk are the main routes. Compounds that are excreted in bile and reabsorbed (enterohepatic recirculation) stay in the body longer, which is why repeated activated charcoal is sometimes helpful.

Dose-response, threshold and LD50

As exposure rises, more animals in a group respond and the response in each animal becomes more severe. Plotted against the logarithm of dose, the proportion of animals responding forms an S-shaped curve. Many toxic effects have a **threshold**, a dose below which no effect is seen, because the body's defences can cope with small amounts. Some effects, such as certain cancers caused by genotoxic agents, are treated as having no safe threshold.

The **LD50** is the dose calculated to kill half of a group of test animals under stated conditions. **LC50** is the same idea for concentration in air or water. A low LD50 means high acute toxicity. The figure is useful for ranking and labelling products, but it has limits that students should know:

- It describes death only, not illness, organ damage or long-term effects.
- It depends on species, strain, sex, route and vehicle, so a value in rats cannot be applied directly to a cat, a horse or a cow.
- It says nothing about the slope of the curve. Two agents with the same LD50 may differ sharply in how close the lethal dose is to a harmless one.

The related ideas are the **NOAEL** (no observed adverse effect level) and the **margin of safety**, which compare the dose that causes harm with the dose that is intended or likely.

Acute, sub-acute and chronic toxicity

Toxicity is classified by how long exposure lasts and how quickly signs appear.

- **Acute toxicity** follows a single exposure or several within a day. Signs are usually fast. Acute studies give the signs, target organs and the approximate lethal range, and help to choose doses for longer studies.

- **Sub-acute (repeat-dose short-term) toxicity** follows daily exposure for a few weeks. It shows target organs, effects on blood and chemistry values, and a first estimate of a no-effect level.
- **Sub-chronic and chronic toxicity** follow exposure for months to most of the animal's life. They detect cumulative harm, organ damage, effects on growth and reproduction, and cancer. Chronic data underlie decisions on residues in food, such as the acceptable daily intake used to set maximum residue limits. Indian limits are notified by the regulator and change over time, so check the current notification.

Cumulative poisons such as lead and fluoride build up until a threshold is crossed, so chronic cases have a vague history.

Decontamination, supportive care and antidotes

Treatment follows a sequence: stabilise first, then reduce further absorption, then speed elimination, then give a specific antidote if one exists.

Stabilise. Airway, breathing, circulation, seizure control, temperature and fluid balance come before anything else. Supportive care is often the most important part of treatment, because most poisons have no antidote.

Decontamination. The aim is to reduce the amount that reaches the blood.

- *Skin and coat:* wash with water and mild detergent, with the handler wearing gloves, so the animal does not keep absorbing the agent or lick it off.
- *Eye:* flush with clean water or saline.
- *Gut:* emptying the stomach by induced vomiting helps only if done soon after a recent ingestion and the animal is alert. It is wrong in an animal that is convulsing, depressed or unable to protect its airway, after corrosives, and after some petroleum products, because of the risk of aspiration or further injury. Some species, such as horses and ruminants, cannot vomit effectively. In ruminants rumen lavage or rumenotomy may be considered for a large recent load.
- *Adsorbents:* activated charcoal binds many poisons in the gut and prevents absorption. It is less useful for alcohols, metals and corrosives. It carries a risk in animals that are depressed or vomiting.

Enhancing elimination uses fluid diuresis, repeated charcoal for poisons that recirculate in bile, and dialysis in referral centres.

Antidotes. An antidote works by one of a few logical routes: blocking the receptor (atropine against muscarinic over-stimulation), reactivating the enzyme (oximes), binding the poison (chelators for metals), supplying the missing substance (vitamin K_1 for anticoagulant rodenticides), or blocking harmful metabolism (fomepizole for ethylene glycol in dogs). An antidote must match the mechanism, and it does not replace supportive care.

History, examination and urgent help

A careful history often identifies the poison before any laboratory test. Ask in a structured way.

1. What species, age, weight and how many animals are affected, and how many are healthy?
2. What was the possible source: feed, water, pasture, plants, pesticides, drugs, household products, rodent baits, snakebite?
3. When did exposure happen, and by what route? How long between exposure and first signs?
4. Any recent change in feed, dipping, spraying, pasture or new medicines? Were the dosing or dilution instructions followed?
5. What treatment has already been given by the owner, such as home remedies or induced vomiting?

Ask to see the container or label and record the active ingredient, not just the trade name. Keep the packaging, because many brands share an active ingredient.

Examine for patterns: pupils, salivation, tremor, gait, heart rate, temperature, gum colour, breath odour and urine. Collect samples *before* treatment where possible: vomit or stomach contents, feed, water, blood, urine, and tissues at post-mortem.

Seek urgent help from a poison information service, veterinary college toxicology unit or senior colleague when the agent is unknown, when several animals are affected, when signs are progressing, or when human exposure is possible. Protect yourself and the owner while handling contaminated animals.

Concept cleared

Bioactivation

Some poisons are harmless until the liver converts them to an active product. In such cases the liver is the site where toxicity begins, so liver function and species enzyme differences change the outcome.

Threshold and the log dose-response curve

Below a threshold the body's defences cope. Above it the proportion of animals affected rises in an S-shaped curve against log dose. The slope matters as much as the LD50.

Stabilise before decontaminate

A seizing, hypoxic or shocked animal needs airway, circulation and seizure control first. Decontamination that is started too early can itself cause harm, such as aspiration.

Antidote matches mechanism

Antidotes act by blocking a receptor, reactivating an enzyme, binding the poison, replacing a missing factor or blocking toxic metabolism. Without a known mechanism, treatment stays supportive.

Memory aid: Order of treatment: SDEA

Stabilise, Decontaminate, Enhance elimination, Antidote. Think of the order when a poisoned animal arrives, and add history-taking running alongside all four.

Compare and contrast

Acute, sub-acute and chronic toxicity

Feature	Acute	Sub-acute	Chronic
Exposure	Single or within 24 hours	Repeated for some weeks	Months to much of a lifetime
Main aim	Signs, target organs, lethal range	Target organs, early no-effect level	Cumulative damage, cancer, reproduction
Typical use	Labelling and dose selection	Planning longer studies	Residue and safe-intake decisions

Toxicant and toxin

Term	Meaning	Example
Toxicant	Any substance that causes harm	Organophosphate, lead, copper
Toxin	Harmful product of a living organism	Snake venom, aflatoxin, botulinum toxin
Antidote	Agent that counteracts a specific poison	Atropine, vitamin K1, fomepizole

Species notes

Cats

Poor glucuronidation makes cats sensitive to phenols, some drugs and many topical products meant for dogs. Grooming increases oral exposure after skin contact.

Dogs

Scavenging behaviour makes dogs the commonest victims of baits, household chemicals and discarded food. They vomit readily, so emesis is an option only within the right time and condition limits.

Cattle and buffalo

The rumen slows absorption of some poisons, dilutes them, and can also convert some compounds into more toxic ones. Vomiting is not possible,

Horses

Horses cannot vomit and have a delicate gut, so colic and ileus complicate many poisonings. Stomach tube and charcoal are the usual decontamination route.

so rumen lavage may be needed for a recent large intake.

Poultry
Birds are exposed through feed and water, and signs appear in the whole flock. Flock-level history and feed testing are essential.

Clinical pearls

Self-test: 10 questions

1. Which statement about LD50 is correct?

LD50 is a lethality measure for a specific species and route. It does not describe sublethal or chronic effects.

2. Bioactivation means that:

Metabolism can produce a more toxic compound, for example conversion of phosphorothioates to oxons.

3. Cats are particularly sensitive to many phenolic compounds because they:

Deficient glucuronide conjugation slows elimination of phenols and some drugs in cats.

4. Inducing vomiting is inappropriate in which situation?

A convulsing or depressed animal cannot protect its airway, so vomiting risks aspiration.

5. The first priority in a poisoned, collapsing animal is:

Life-support measures come before decontamination and specific therapy.

6. Repeated doses of activated charcoal are most logical for poisons that:

Charcoal in the gut binds poison re-secreted in bile and prevents reabsorption.

7. A 'threshold' in a dose-response relationship means:

Below threshold, body defences cope and no measurable effect occurs for many toxic endpoints.

8. Which study type is mainly used to detect cancer and cumulative damage?

Chronic studies, covering months to much of the lifespan, are designed to find cumulative and late effects.

9. Vitamin K1 is an antidote that works by:

Anticoagulant rodenticides block vitamin K recycling, and vitamin K1 restores the pathway.

10. Which action is most useful in history taking?

Active ingredient, timing, route and group pattern guide diagnosis and treatment, and the container helps confirm the agent.

Key points to revise

1. Toxicity depends on agent, dose, route, duration and the animal's own characteristics.
2. Toxicokinetics covers absorption, distribution, biotransformation and excretion.
3. Bioactivation can make a compound more toxic after metabolism.

4. The dose-response curve is S-shaped against log dose and many effects have a threshold.
5. LD50 measures lethality only and is species and route specific.
6. Acute, sub-acute and chronic studies answer different questions about toxicity.
7. Treatment order: stabilise, decontaminate, enhance elimination, give antidote if available.
8. A structured history, label and sample collection often identify the poison.

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- Ch 3. Pyrethroid, amitraz and other insecticide poisoning (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Overview of Veterinary Toxicology
- Merck Veterinary Manual: Introduction to Poisoning (pet owner version)
- ASPCA Animal Poison Control

CHAPTER 2 OF 17

Organophosphate and carbamate poisoning

Organophosphates and carbamates inhibit acetylcholinesterase and cause cholinergic overstimulation. The chapter explains the mechanism, the muscarinic, nicotinic and central signs, the logic of atropine and oxime treatment, species differences and prevention.

After this chapter you can

- Explain how organophosphates and carbamates inhibit acetylcholinesterase and why acetylcholine accumulates
- Recognise muscarinic, nicotinic and central nervous signs of poisoning

- Explain why atropine and oximes are used and what each can and cannot reverse
- Describe differences between organophosphates and carbamates, and between species
- List practical prevention measures and know when to seek urgent help

Why these insecticides matter

Organophosphates (OP) and carbamates are among the most widely used insecticides, acaricides and ectoparasiticides in farming and household pest control. They are also among the commonest causes of poisoning in Indian dogs, cattle and buffalo, because they are cheap, easy to buy and often stored carelessly or applied at the wrong dilution.

Both groups act on the same enzyme. Understanding one enzyme and one neurotransmitter explains nearly every sign, the logic of the antidotes, and why the two groups behave a little differently in the clinic.

Mechanism of toxicity

Acetylcholine (ACh) is released at parasympathetic nerve endings, at the neuromuscular junction, at autonomic ganglia and at many sites in the brain. Its action is normally stopped within a fraction of a second by **acetylcholinesterase** (AChE), which splits it into choline and acetate.

OP and carbamate compounds attach to the active site of AChE and block it. ACh then accumulates and keeps stimulating its receptors. The result is continuous overstimulation, known as a **cholinergic crisis**.

- **Organophosphates** phosphorylate the enzyme. The bond is very stable and is broken down only very slowly, so inhibition is effectively irreversible. In time the inhibited enzyme undergoes **aging**, a loss of a chemical group that makes the bond permanent. After aging the enzyme cannot be reactivated, and recovery depends on making new enzyme.
- **Carbamates** carbamylate the enzyme. This bond breaks down much sooner, so inhibition is reversible and signs are usually shorter and often milder.

Many OPs, such as the phosphorothioates, are inactive until the liver converts them to the oxon form. This explains why signs may be delayed by some hours after exposure, especially after skin contact, and why the liver's enzyme activity affects the severity.

Routes of exposure include ingestion of baits, treated seed or contaminated feed, skin absorption from dips, sprays and pour-ons, and inhalation. Overdosing or wrong dilution of a veterinary product is a frequent cause in practice.

Clinical signs

Signs fall into three groups, depending on which receptor is overstimulated.

- **Muscarinic** (parasympathetic): salivation, lacrimation, urination, defaecation, vomiting, colic and diarrhoea, bronchial secretion and bronchoconstriction, slow heart rate and constricted pupils. Death usually results from respiratory failure caused by fluid in the airways and bronchospasm.
- **Nicotinic** (neuromuscular junction and ganglia): muscle fasciculations and tremors, then weakness and paralysis. Overstimulation keeps the end-plate depolarised, so the muscle stops responding. Heart rate may rise instead of fall, because ganglia are also stimulated.
- **Central:** restlessness, ataxia, depression, seizures and coma.

Students often remember the muscarinic signs with the word *SLUDGE* or *DUMBELS*. A common confusion is to expect every case to show miosis and a slow heart rate. Pupils and heart rate can be misleading because of nicotinic effects. Look at the whole pattern: wetness, tremor and respiratory distress together point strongly to this poisoning.

Onset may be acute, within minutes to a few hours. Some OPs also cause an **intermediate syndrome** some days later, with weakness of neck and limb muscles, and certain compounds cause **delayed neuropathy**, which damages nerves and appears one to three weeks after exposure. Poultry are the classic model for delayed neuropathy.

Diagnosis

Diagnosis rests on history of exposure, the typical combination of signs and response to treatment. Laboratory support includes measuring **cholinesterase**

activity in whole blood or in brain tissue after death. A marked fall supports the diagnosis, but red cell and plasma enzyme levels vary by species, and carbamate inhibition can reverse in a stored sample, so interpret with care. Identification of the compound in stomach contents, feed or bait is the most reliable confirmation. Many OP signs mimic other conditions such as heat stress, snake or scorpion envenomation and other causes of colic.

Treatment: decontamination, atropine and oximes

Treatment aims to protect the airway, reduce further absorption, block the effects of excess ACh and, for OPs, reactivate the enzyme.

Decontamination. Wash the skin and coat thoroughly, with gloves on, for dermal exposure. For recent ingestion, activated charcoal is used and emesis is not suitable when the animal is convulsing, depressed or secreting heavily. In ruminants, rumen lavage may be considered after a large recent intake.

Atropine is a competitive antagonist at *muscarinic* receptors. It reverses secretions, bronchospasm and slow heart rate, which are the signs that kill. The end point is judged by drying of bronchial secretions and improved breathing, not by pupil size or heart rate alone. Atropine does *not* act on nicotinic receptors, so tremors and muscle weakness continue. It also does not reactivate AChE. Over-treatment causes atropine toxicity, with dry mouth, tachycardia and, in horses and ruminants, ileus. Dose by a veterinary reference or the clinician in charge.

Oximes such as pralidoxime attach to the phosphorylated enzyme and pull the OP group off, restoring the enzyme. They are the only treatment that acts on nicotinic signs. Oximes work only *before aging*, so they are most useful early. They are less useful in carbamate poisoning because carbamylated enzyme recovers spontaneously, and some authorities advise caution with certain carbamates. Oximes enter the brain poorly, so they help mainly at the muscle and peripheral sites.

Supportive care includes oxygen, airway suction, fluids, and seizure control. Drugs that worsen cholinesterase inhibition, such as some muscle relaxants and phenothiazine tranquillisers, are avoided. Check a current veterinary pharmacology reference for the precise list. Animals may need support for days with OPs, because recovery depends on regenerating the enzyme.

Species differences and prevention

Sensitivity varies with species, with the particular compound, and with the product formulation. Cats and young animals are generally more sensitive. Cats are the species most often affected by dog flea and tick products used wrongly, and some OP compounds are far more dangerous in cats than in dogs. Horses show colic and ileus early. Ruminants show salivation, diarrhoea and tremor, and sudden death can occur after dips or sprays used incorrectly. Birds, fish and bees are very sensitive to many compounds, which matters for wildlife and for the environment.

Prevention is the most effective step.

- Use only products labelled for the species, follow the label for dilution and interval, and do not apply dog products to cats.
- Do not combine OP products with other cholinesterase inhibitors, including some dewormers, in the same animal at the same time.
- Store pesticides in original labelled containers, away from feed, water and children. Never reuse empty containers.
- Keep animals off sprayed pasture and crops for the interval stated on the label, and observe the withdrawal periods for milk and meat. Indian rules on banned or restricted pesticides and maximum residue limits are updated from time to time, so check the current notification.
- Wear gloves and protective clothing when dipping, and wash hands after use. Human exposure is a real risk for the farmer and the veterinarian.

Seek urgent help from a poison information service or a senior veterinarian when there is respiratory distress, seizures or multiple animals, or when a person has also been exposed.

Concept cleared

Cholinergic overstimulation

AChE normally ends the acetylcholine signal. When it is inhibited, acetylcholine builds up at muscarinic, nicotinic and central synapses, and all signs follow from that single change.

Aging of the enzyme

After a period, the phosphorylated enzyme loses a chemical group and becomes permanently inactive. Oximes work only before this happens, which is why early treatment matters.

Atropine limits

Atropine blocks muscarinic receptors only. It controls secretions and bronchospasm but does nothing for muscle tremors or paralysis and does not restore the enzyme.

Reversible and irreversible inhibition

Carbamates form a bond that breaks within hours, so recovery is relatively quick. OPs form a stable bond, and recovery depends on reactivation or new enzyme synthesis.

Memory aid: DUMBELS

Diarrhoea, Urination, Miosis, Bronchospasm and bronchorrhoea, Emesis, Lacrimation, Salivation. These are the muscarinic signs. Remember that tremor and weakness are nicotinic and need separate thought.

Compare and contrast

Organophosphates and carbamates

Feature	Organophosphate	Carbamate
Bond with AChE	Phosphorylation, very stable, aging	Carbamylation, spontaneous recovery
Duration	Long, days	Shorter, hours
Oxime use	Useful early, before aging	Often unnecessary, caution with some agents
Delayed neuropathy	Possible with some compounds	Not typical

Atropine and pralidoxime

Feature	Atropine	Pralidoxime
Action	Blocks muscarinic receptors	Reactivates phosphorylated AChE
Reverses	Salivation, bronchospasm, bradycardia	Nicotinic signs such as weakness, plus enzyme function
Limitation	No effect on muscle tremors	Fails after aging, poor brain entry
Main end point	Dry airway secretions, better breathing	Improved muscle strength

Species notes

Dogs

Commonly poisoned by baits, spilled concentrates and wrong use of dips. Salivation, vomiting, diarrhoea, tremor and seizures are typical. Breeds with the MDR1 mutation need extra care with some other drugs used in treatment, so check the current reference.

Cats

Particularly sensitive to many OP compounds, and exposed through grooming after skin application of dog products or flea sprays. Weakness and tremor may stand out more than wetness.

Cattle and buffalo

Poisoned by dips, sprays, treated seed and contaminated fodder. Salivation, diarrhoea, tremor, laboured breathing and sudden death are seen. Milk and meat residues are a food safety issue.

Horses

Colic and ileus appear early. Atropine must be used cautiously because it slows the gut and may worsen colic.

Poultry

Flocks show weakness, diarrhoea, and paralysis. Some compounds cause delayed neuropathy, with

weakness days later, and birds are used as the test model for this.

Clinical pearls

Self-test: 10 questions

1. The primary enzyme inhibited by OP and carbamate insecticides is:

Inhibition of acetylcholinesterase leads to accumulation of acetylcholine.

2. Which sign is mediated by nicotinic receptors?

Fasciculations and weakness arise at the neuromuscular junction, which has nicotinic receptors.

3. Atropine in OP poisoning is mainly effective against:

Atropine blocks muscarinic receptors but does not affect nicotinic receptors or the enzyme.

4. Aging of phosphorylated acetylcholinesterase means:

After aging the bond becomes permanent, so oximes no longer help.

5. Compared with OPs, carbamate inhibition of AChE is:

Carbamylated enzyme hydrolyses spontaneously within hours.

6. The best end point for atropine therapy is:

Bronchial secretion and bronchospasm cause death, so airway drying is the clinical goal.

7. Which statement about oximes is correct?

Oximes restore enzyme and improve muscle function but do not replace atropine.

8. A species in which atropine must be used cautiously because of ileus is the:

Atropine reduces gut motility and can worsen colic in horses.

9. Delayed neuropathy after certain OPs is best demonstrated in:

Hens are the classical test species for delayed OP neuropathy.

10. Which prevention step is correct?

Correct product use, labelled storage and observing withdrawal periods protect animals, handlers and food.

Key points to revise

1. OPs and carbamates inhibit acetylcholinesterase and cause cholinergic crisis.
2. OP inhibition is long lasting and can age, while carbamate inhibition is reversible.
3. Signs are muscarinic, nicotinic and central.
4. Respiratory failure is the usual cause of death.
5. Atropine blocks muscarinic effects only.
6. Oximes reactivate phosphorylated enzyme and work best early.
7. Cats, young animals and horses need special care.
8. Prevention depends on correct product, label use, storage, and protection of handlers.

Go deeper on this site

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- Ch 1. General principles of toxicology (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

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- ASPCA Animal Poison Control

CHAPTER 3 OF 17

Pyrethroid, amitraz and other insecticide poisoning

Pyrethrins and pyrethroids, amitraz and the isoxazoline class are widely used ectoparasiticides with very different mechanisms. The chapter covers their toxicity, with emphasis on cat pyrethrin poisoning, amitraz in dogs and horses, and cautions with isoxazolines.

After this chapter you can

- Explain how pyrethrins and pyrethroids act on sodium channels and why cats are at special risk
- Describe the mechanism and signs of amitraz toxicity in dogs and why horses are particularly vulnerable
- Explain how isoxazolines work and why caution is advised in animals with a seizure history
- Apply decontamination, supportive care and antidote reasoning to these poisonings
- List prevention measures and know when to seek urgent help

Three groups, three mechanisms

Modern flea, tick and mite products rely on compounds that are far more toxic to insects and mites than to mammals. That selectivity is not complete. Poisoning

occurs when the wrong species is treated, when the dose is not matched to body weight, when products are used together, or when animals groom or lick treated coats.

This chapter deals with three groups that are unrelated in mechanism: **pyrethrins and pyrethroids** (sodium channel modifiers), **amitraz** (an alpha-2 adrenergic agonist) and the **isoxazolines** (inhibitors of ligand-gated chloride channels). Organophosphates and carbamates were covered in the previous chapter.

Pyrethrins and pyrethroids

Pyrethrins are natural extracts of chrysanthemum flowers. **Pyrethroids** such as permethrin, cypermethrin, deltamethrin and flumethrin are synthetic and more stable. They are used on dogs, cattle, buffalo, sheep and goats, in sprays, dips, ear tags, collars and spot-on products, and as household insecticides.

Mechanism. They bind to voltage-gated sodium channels in nerve membranes and keep them open for longer than normal. Nerve cells fire repeatedly, giving tremor, hyperexcitability and, with strong exposure, seizures. Type II compounds that contain a cyano group, such as cypermethrin and deltamethrin, produce a more severe syndrome with salivation and choreoathetosis, a writhing movement. Type I compounds, such as permethrin, cause mainly tremor, often called the T syndrome.

Mammals are protected because they have a higher body temperature, which speeds closing of the channel, and efficient breakdown by esterases and liver enzymes. Mammalian toxicity is therefore low in healthy animals when used as labelled.

Why cats are at risk. Cats are poor at glucuronidation, one of the main routes of pyrethroid metabolism, so permethrin lingers. Many cat poisonings come from the owner placing a concentrated dog spot-on product on a cat, or from a cat living with a recently treated dog and grooming it. Signs begin within hours and include hypersalivation, ear and skin twitching, tremor, ataxia, hyperthermia, seizures, and in severe cases death. Cats are more sensitive than dogs, and young, small or debilitated animals are at greater risk.

Dogs and other species usually show milder tremor, salivation and vomiting after an overdose or ingestion. Fish and bees are extremely sensitive, so dips must not be discharged into ponds.

Treatment of pyrethroid poisoning

There is no specific antidote, so treatment is decontamination and supportive care.

- **Skin:** bathe with lukewarm water and a mild liquid detergent, with gloves. Cold water may cause chilling and hot water increases absorption. Prevent the animal from licking the coat.
- **Tremor and seizures:** centrally acting muscle relaxants such as methocarbamol are commonly used for tremor. Benzodiazepines or other anticonvulsants may be added for seizures. Dose by a current veterinary reference.
- **Temperature:** cool the hyperthermic animal gradually and warm the hypothermic one. Provide fluids, and maintain breathing.
- Emesis is considered only for recent ingestion in an alert animal and is avoided when tremor or seizures are present.

Intravenous lipid emulsion is used for severe cases in some centres, but the evidence is limited, so treat it as an option to discuss with a specialist. Because pyrethroids are broken down fairly quickly, most animals recover with good care within a few days.

Do not confuse pyrethroid tremor with organophosphate poisoning. Pyrethroids do not inhibit cholinesterase, and atropine is not the treatment.

Amitraz

Amitraz is a formamidine acaricide used in dips and sprays for ticks and mites, for example in demodectic mange treatment in some regions and in livestock tick control. It is also used as a collar in some countries.

Mechanism. Amitraz and its metabolite act as agonists at **alpha-2 adrenergic receptors**, the same receptors targeted by xylazine and medetomidine. It also has weak monoamine oxidase inhibiting action and effects on octopamine receptors in the mite. Alpha-2 stimulation causes sedation, bradycardia, low blood pressure, reduced gut motility and raised blood glucose, because insulin release is suppressed.

Dogs. Common causes are overdose or wrong dilution of a dip, ingestion of a collar, and licking of treated coats. Signs are depression, ataxia, slow heart rate, hypotension, low body temperature, vomiting, mydriasis, hyperglycaemia and in

severe cases coma. Signs often resolve within a day or two. Small dogs are at higher risk of overdose.

Horses. Horses are very sensitive. Amitraz is not intended for use on equids. Exposure causes marked reduction of gut motility, colic, impaction and sometimes death. The label cautions should be treated as a warning against any use in horses. Ponies, donkeys and foals should be treated with the same caution.

Ruminants are relatively tolerant of properly used products, but sedation and ruminal stasis can occur after overdose. Observe label withdrawal periods for milk and meat, and check current Indian regulations.

Treatment. Decontaminate the skin by bathing and prevent grooming. Alpha-2 antagonists such as atipamezole or yohimbine reverse the sedation and bradycardia and are the antidote logic here, because they compete for the same receptor. In a bradycardic animal, anticholinergic drugs are used with caution because they can raise blood pressure and cause tachycardia, so seek veterinary advice. Monitor heart rate, blood pressure, temperature, blood glucose and gut function. Horses need colic assessment and gut support.

Isoxazolines and other products

Isoxazolines are oral or topical compounds, among them fluralaner, afoxolaner, sarolaner and lotilaner, given to dogs and cats for fleas, ticks and mites. They inhibit insect and mite ligand-gated chloride channels, particularly the GABA-gated ones, so the parasite shows uncontrolled excitation and dies. Binding to the mammalian receptor is much weaker, giving a wide safety margin in most animals.

Regulators, including the United States FDA, have issued cautions that some animals given isoxazolines have shown neurologic signs such as muscle tremor, ataxia and seizures. The cautions advise **care in animals with a history of seizures or epilepsy**. The product label and the current regulatory statement should be checked before prescribing, because wording can change. Reasonable practice is to ask about neurological history, discuss alternatives for such animals, and tell owners what to watch for. Signs when seen are usually managed with supportive care and, if needed, seizure control.

A common confusion is to assume that isoxazolines act on the same channel as pyrethroids, or that they are organophosphates. They are not. They share an effect

on the nervous system but through different targets, so the antidote logic differs.

Other products. Macrocyclic lactones such as ivermectin act on glutamate-gated chloride channels in parasites. Dogs with the MDR1 mutation are at risk of neurotoxicity because less drug is pumped out of the brain, so products for such animals need extra care. Never use fipronil, imidacloprid or similar spot-ons in rabbits or other species not named on the label without advice.

Prevention and urgent help

Prevention for all these groups: use species-specific labelled products, calculate by body weight, avoid overlapping products, separate treated dogs from cats until dry, store products out of reach, wash hands and keep children away from freshly treated animals. Contact a poison information service or specialist promptly for cats with tremor, any seizing animal, horses exposed to amitraz, or any large ingestion.

Concept cleared

Sodium channel prolongation
Pyrethrins and pyrethroids keep voltage-gated sodium channels open, so nerves fire repeatedly. The outcome is tremor, hyperexcitability and seizures, rather than cholinergic wetness.

Cat glucuronidation gap
Cats lack efficient glucuronidation, so permethrin is removed slowly. A dog spot-on applied to a cat is the classic cause of severe tremor and seizures.

Alpha-2 agonism by amitraz
Amitraz acts at the same alpha-2 receptor as xylazine, producing sedation, bradycardia, ileus and hyperglycaemia. Alpha-2 antagonists such as atipamezole reverse these effects.

Selectivity is relative

Isoxazolines bind insect and mite chloride channels more strongly than mammalian ones, but in animals with a seizure tendency the margin may be smaller, so caution is advised.

Memory aid: Match the pattern

Wet and weak means cholinesterase inhibitor. Twitchy and hot means pyrethroid. Sleepy and slow with high glucose means amitraz. Seizure in a treated epileptic means think isoxazoline.

Compare and contrast

Mechanism and clinical picture

Group	Target	Typical signs	Key treatment idea
Pyrethroids	Voltage-gated sodium channel kept open	Tremor, salivation, hyperthermia, seizures	Bathe, muscle relaxant, supportive care
Amitraz	Alpha-2 receptor agonist	Sedation, bradycardia, ileus, hyperglycaemia	Bathe, alpha-2 antagonist, monitor
Isoxazolines	Parasite GABA-gated chloride channel	Rare tremor, ataxia, seizures	Supportive care, caution with seizure history
OP and carbamates	Acetylcholinesterase	Wetness, tremor, respiratory distress	Atropine, oxime for OPs

Species susceptibility

Agent	More vulnerable species	Reason
Permethrin	Cats	Poor glucuronidation, grooming
Amitraz	Horses and ponies	Marked effect on gut motility
Isoxazolines	Animals with seizure history	Possible lowering of seizure threshold
Pyrethroids	Fish, bees	High sensitivity to low concentrations

Species notes

Cats

Highest risk for pyrethroid poisoning, especially from dog spot-ons. Tremor, salivation, hyperthermia and seizures need prompt decontamination and muscle relaxant therapy. Seek urgent help.

Dogs

Amitraz overdose and collar ingestion cause sedation and bradycardia. Small dogs are at higher risk of overdose. MDR1 breeds need care with macrocyclic lactones.

Horses

Amitraz is dangerous, with ileus, colic and impaction. Do not use products labelled for other species on horses.

Cattle, buffalo, sheep and goats

Pyrethroid sprays, pour-ons and dips are common, and mistakes in dilution cause toxicity. Observe milk and meat withdrawal periods, and check current Indian notification.

Rabbits and small mammals

Many ectoparasiticides for dogs are unsafe in these species, so use only products and doses specified for them.

Clinical pearls

Self-test: 10 questions

1. Pyrethroids toxicity arises mainly from:

Pyrethroids keep sodium channels open, leading to repetitive nerve firing and tremor.

2. Which species is at greatest risk from dog spot-on permethrin products?

Cats metabolise pyrethroids slowly because they lack effective glucuronidation.

3. A suitable drug logic for pyrethroid tremor is:

Pyrethroid tremor is central and is treated with muscle relaxants. Atropine and oximes are for cholinesterase inhibitors.

4. Amitraz acts as an agonist at:

Amitraz and its metabolite stimulate alpha-2 receptors, similar to xylazine.

5. Which sign is typical of amitraz toxicity in a dog?

Alpha-2 stimulation causes sedation, bradycardia and suppressed insulin release.

6. Why is amitraz dangerous in horses?

Alpha-2 effects on the equine gut can produce ileus, colic and impaction.

7. The antidote logic for amitraz poisoning uses:

Competitive antagonists at the alpha-2 receptor reverse the sedation and bradycardia.

8. Isoxazolines act on:

They inhibit parasite chloride channels. This differs from pyrethroids and organophosphates.

9. A caution advised by regulators for isoxazoline products concerns animals with:

Neurologic adverse events such as tremor and seizures were reported, so care is advised in animals with seizure history.

10. Which decontamination step is correct for a cat with dermal pyrethroid exposure?

Washing reduces ongoing absorption and licking. Alcohol and hot water can worsen absorption.

Key points to revise

1. Pyrethroids prolong opening of sodium channels in nerve membranes.
2. Type II pyrethroids with a cyano group cause salivation and more severe signs.
3. Cats are especially sensitive to permethrin because of poor glucuronidation.
4. Pyrethroid treatment is decontamination and supportive care with muscle relaxants.
5. Amitraz is an alpha-2 agonist with sedation, bradycardia and ileus.
6. Horses are very sensitive to amitraz, so avoid use.
7. Isoxazolines inhibit parasite GABA-gated chloride channels, with caution in seizure history.
8. Prevention relies on species-specific labelled use and correct dosing by weight.

Go deeper on this site

- Ch 4. Rodenticide poisoning (in this book)
- Ch 2. Organophosphate and carbamate poisoning (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Overview of Veterinary Toxicology
- Merck Veterinary Manual: Introduction to Poisoning (pet owner version)
- ASPCA Animal Poison Control

CHAPTER 4 OF 17

Rodenticide poisoning

Rodenticides are the commonest household poisons met in small-animal practice. This chapter links each group (anticoagulants, zinc phosphide, cholecalciferol, bromethalin, strychnine) to its mechanism, so that signs, timing, antidote and monitoring follow logically.

After this chapter you can

- Explain how anticoagulant rodenticides cause delayed bleeding and why vitamin K1 reverses it
- Describe why zinc phosphide releases phosphine gas and what this means for decontamination and staff safety
- Explain how cholecalciferol causes hypercalcaemia and kidney injury
- Describe the mechanism of bromethalin and strychnine and why no simple antidote exists for bromethalin
- Apply an approach to an unknown rodenticide exposure: history, decontamination, monitoring and when to seek urgent help

Why rodenticides are a veterinary problem

Rodenticides are made to kill mammals, and dogs, cats, pigs, poultry and wildlife are mammals or eat poisoned rodents. Most cases are accidental: a dog eats bait left in a house or godown, a cat eats a poisoned rat, or a farm animal reaches stored grain mixed with bait. Malicious baiting of dogs also occurs, especially with strychnine.

The first task is to learn **which product** was used. Many baits look alike (coloured grain, wax blocks, pellets) but the groups differ in mechanism, in time to onset and in antidote. Ask for the packet or its active ingredient. If it cannot be identified, treat as an unknown exposure and decontaminate, then monitor for all the main groups.

Merck's veterinary overview notes that use has been shifting from anticoagulants towards bromethalin and cholecalciferol in some countries, so an old assumption that every bait is an anticoagulant is unsafe.

Anticoagulant rodenticides

Clotting factors II, VII, IX and X need vitamin K to become active. During activation vitamin K is oxidised to an epoxide, and the enzyme **vitamin K epoxide reductase (VKORC1)** recycles it back. Anticoagulants such as warfarin, coumatetralyl, bromadiolone and brodifacoum block this enzyme. The liver keeps making factors, but they are inactive.

Signs are **delayed** because factors already in blood keep working until they are used up. Factor VII has the shortest half-life, so the prothrombin time (PT) rises first, usually within a day or two, while bleeding appears after several days. Animals present with lethargy, pale gums, breathlessness (bleeding into the chest or lungs), haematomas, nose bleeds, blood in faeces or sudden weakness. The animal is often seen only when bleeding has started.

First-generation compounds (warfarin) act for a short time and often need repeated feeding. **Second-generation** compounds (brodifacoum, bromadiolone, difethialone) are potent, stay long in the liver and may be lethal after a single feed. Their effect lasts weeks, so treatment is long.

Antidote and monitoring. The antidote is **vitamin K1 (phytonadione)**, which supplies active vitamin K and bypasses the blocked enzyme. A common confusion is vitamin K3 (menadione), which is not an adequate substitute. Oral K1 is absorbed better with fat in the meal. Because second-generation agents persist, treatment continues for weeks. PT is measured before treatment, during it, and again a few days after the last dose to confirm that clotting does not fall back. Severe bleeding needs plasma or whole blood, which gives factors at once, because vitamin K takes many hours to act. Avoid unnecessary needle sticks and jugular puncture in bleeding patients.

Early decontamination (induced vomiting if recent and safe, activated charcoal) is useful. Treating after a known ingestion, before signs appear, is easier than treating bleeding. **Secondary poisoning** of dogs, cats and raptors that eat poisoned rodents is possible, mainly with second-generation products.

Zinc phosphide (and related phosphides)

Zinc phosphide is a grey-black powder in baits. Its own toxicity is small until it meets **stomach acid**, which converts it into **phosphine gas**. Phosphine inhibits cellular respiration and damages the gut, liver, kidney, lungs and heart. A full stomach with high acid output produces more gas, and signs begin within hours.

Signs include vomiting (the vomit may smell of garlic or fish), abdominal pain, restlessness, rapid breathing, collapse and sometimes convulsions. There is **no specific antidote**; treatment is decontamination and supportive care. Concepts that matter: keep the stomach less acidic with antacids so that less gas forms; control seizures; support circulation and breathing; and protect staff. Vomitus and stomach contents give off phosphine, so handle them in a ventilated area, not in a closed room.

Aluminium and magnesium phosphide, sold as grain fumigant tablets, act in the same way and release gas on contact with moisture. They are far more dangerous and are a frequent cause of human and animal deaths in India. Check the current notification and local rules on their sale and storage.

Cholecalciferol (vitamin D3)

Cholecalciferol baits do not look dangerous, because vitamin D is a vitamin. In large amounts, however, it is converted to active metabolites that raise calcium absorption and release from bone. The result is **hypercalcaemia and hyperphosphataemia**. Calcium and phosphorus deposit in soft tissues (**mineralisation**) of the kidney, heart, stomach and blood vessels, and the kidneys fail.

Signs are **delayed**, often a day or more after ingestion: vomiting, loss of appetite, excessive drinking and urination, weakness, then signs of acute kidney injury. The common confusion is that the animal looks well on the first day. Monitoring of serum calcium, phosphorus, urea and creatinine is continued for several days even if signs are absent.

Treatment concepts: early decontamination; intravenous fluids to protect the kidney and increase calcium loss in urine; drugs that lower calcium or block bone release (such as calcitonin or bisphosphonates, used under specialist advice); and low-

calcium, low-vitamin-D diet. Recovery can take weeks because vitamin D is stored in fat. Calcium-containing products and vitamin D supplements must be avoided.

Bromethalin

Bromethalin is a neurotoxin. It is converted to a metabolite that **uncouples oxidative phosphorylation** in brain mitochondria. ATP falls, the sodium-potassium pump fails, fluid collects between the myelin layers and the brain and spinal cord swell. Higher doses give an **acute convulsive form** with tremors, hyperexcitability, seizures and death. Lower doses give a **delayed paralytic form** with hindlimb weakness, ataxia, depression and recumbency over days. Cats are especially sensitive.

Merck's manual states plainly that **no antidote exists** for bromethalin. Everything depends on early decontamination and repeated activated charcoal under veterinary advice, plus supportive care, seizure control and nursing. Any pet that may have eaten a bromethalin bait, including a cat, should reach a clinic quickly, before signs begin.

Strychnine and the overall approach

Strychnine blocks **glycine**, the inhibitory transmitter of the spinal cord. Without inhibition, motor neurons fire freely. The animal shows muscle stiffness, an extended posture with arched neck (opisthotonos), and violent seizures set off by touch, light or sound, while it remains **conscious** and in distress. Death comes from exhausted or paralysed breathing muscles. Signs start within a few hours of ingestion. Dogs are the usual victims, often through malicious baiting.

Treatment concepts: control the seizures first (muscle relaxants such as methocarbamol, or sedatives), keep the animal in a quiet, dark place and handle it minimally, then decontaminate once it is stable, and support breathing. Sampling of stomach contents and tissue helps a legal case. Check the current notification on the status of strychnine in India.

Approach to any rodenticide exposure: find the product and time; decontaminate early if safe; do not induce vomiting in a convulsing or already weak animal; arrange monitoring for the likely group; keep baits where pets and livestock cannot reach them; use bait stations; and collect dead rodents. **Seek urgent help** for any

pet with known bromethalin, cholecalciferol or phosphide exposure, any animal with bleeding, tremors or seizures, or when the product is unknown.

Concept cleared

Concept cleared: Delayed bleeding
Think of a shop that stops getting new stock. Shelves look full for a few days because of what is already inside. Only when old stock is used up do shelves empty. Anticoagulants stop new active clotting factors, so bleeding begins only when the old factors have decayed.

Concept cleared: Phosphine and stomach acid
Zinc phosphide is like a sealed gas cylinder that opens only in acid. The powder is harmless on the shelf, but the stomach acid opens it and releases phosphine. Less acid (antacid) means less gas.

Concept cleared: Vitamin K1 as a bypass
Vitamin K recycling is a blocked road. Vitamin K1 given by mouth is a new bypass that reaches the destination without using the blocked road, so clotting factors can be activated again.

Concept cleared: Strychnine and the brake
Glycine is the brake of the spinal cord. Strychnine cuts the brake cable, so every small stimulus sends the motor system into full throttle.

Memory aid: Rodenticide quick code
Anticoagulant = bleeding after Days (Vitamin K1). Phosphide = gas in the stomach (garlic smell). Cholecalciferol = Calcium goes up, kidneys go down. Bromethalin = Brain swells, no antidote. Strychnine = Stiff and Startled by sound, glycine blocked.

Compare and contrast

Rodenticides at a glance

Agent	Main mechanism	Onset	Antidote or key treatment
Anticoagulants	Block VKORC1, inactive factors II, VII, IX, X	Days	Vitamin K1, plasma or blood if severe
Zinc phosphide	Phosphine gas from stomach acid	Hours	No antidote; decontaminate, antacid, support
Cholecalciferol	Hypercalcaemia, soft tissue mineralisation	Delayed, 1 day or more	No antidote; fluids, calcium-lowering drugs, monitor
Bromethalin	Uncoupled oxidative phosphorylation, brain oedema	Hours to days	No antidote; early decontamination, support
Strychnine	Glycine blockade in spinal cord	Within hours	Control seizures, quiet, support

First- versus second-generation anticoagulants

Feature	First generation (e.g. warfarin)	Second generation (e.g. brodifacoum)
Potency	Lower	High
Doses needed for rodents	Often repeated feeding	Often a single feed
Duration in the body	Short	Long, weeks
Treatment with vitamin K1	Short course	Long course, repeat PT

Species notes

Dog

The commonest victim, because it eats baits. Strychnine is often malicious. Dogs may also eat a poisoned rat.

Cat

Especially sensitive to bromethalin, and may be secondarily poisoned by eating poisoned rodents.

Pig

Free-range pigs may eat baits and poisoned rats; pigs also vomit, which can protect or contaminate. Treat as for other monogastrics and seek advice.

Cattle and buffalo

Occasionally exposed through feed contaminated with bait or fumigant residue. Ruminal acid is lower, but gas release can still occur with phosphides, so seek veterinary help.

Poultry

Birds may eat baited grain. Safe baiting practice is to place baits in covered stations out of reach.

Clinical pearls

Self-test: 10 questions

1. Anticoagulant rodenticides act by inhibiting:

They block VKORC1, so vitamin K cannot be recycled and factors II, VII, IX and X remain inactive.

2. Why is bleeding delayed after anticoagulant ingestion?

Active factors already in blood keep clotting until they decay.

3. Which test usually becomes abnormal first after anticoagulant poisoning?

Factor VII has the shortest half-life, so PT rises first.

4. The correct vitamin for anticoagulant rodenticide poisoning is:

Vitamin K1 is the adequate antidote; K3 is not a reliable substitute.

5. Zinc phosphide becomes especially dangerous because it:

Gastric acid converts it into phosphine gas.

6. Which statement about handling phosphide vomitus is correct?

Phosphine can be released from the vomitus and stomach contents.

7. Cholecalciferol poisoning is characterised by:

Excess vitamin D raises calcium and phosphorus and mineralises soft tissue, including kidney.

8. For bromethalin poisoning, which statement is true?

Merck states that no antidote exists; management is decontamination and support.

9. Bromethalin acts mainly by:

ATP loss causes pump failure and brain oedema.

10. A dog is conscious with extended limbs and seizures triggered by noise. Which poison is most likely?

Strychnine blocks glycine in the spinal cord, giving stimulus-triggered rigidity while the animal remains aware.

Key points to revise

1. Rodenticide groups differ in mechanism, onset and antidote.
2. Anticoagulants block VKORC1 and cause delayed bleeding; PT rises first.
3. Vitamin K1 is the antidote; severe bleeding also needs plasma or blood.

4. Second-generation anticoagulants persist for weeks.
5. Zinc phosphide releases phosphine gas in stomach acid and has no specific antidote.
6. Cholecalciferol causes hypercalcaemia, mineralisation and kidney injury.
7. Bromethalin causes brain oedema, with no antidote; cats are very sensitive.
8. Strychnine blocks spinal glycine and causes stimulus-triggered seizures while conscious.

Go deeper on this site

- Ch 5. Herbicide and fungicide poisoning (in this book)
- Ch 3. Pyrethroid, amitraz and other insecticide poisoning (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Anticoagulant rodenticide poisoning in animals
- Merck Veterinary Manual: Phosphide poisoning in animals
- Merck Veterinary Manual: Bromethalin poisoning in animals
- Merck Veterinary Manual: Strychnine poisoning in animals

CHAPTER 5 OF 17

Herbicide and fungicide poisoning

Most herbicides and fungicides are of low toxicity to mammals, but a few, such as paraquat, copper and organomercury compounds, are dangerous. This chapter explains why, with mechanisms, signs, management concepts and prevention.

After this chapter you can

- Explain why glyphosate is of low toxicity to animals and why the surfactant matters
- Describe how paraquat injures the lung and why oxygen and delay in decontamination matter

- Describe the low toxicity of 2,4-D and its indirect hazards
- Explain copper toxicity and species differences, especially in sheep
- Describe organomercury poisoning and the prevention of seed-dressing accidents

General principles

Herbicides are designed to act on plant processes such as amino acid synthesis, hormone control or photosynthesis. Animals lack many of these targets, so most modern herbicides have low acute toxicity to mammals. Poisoning usually follows access to **concentrated product**, contaminated water or feed, spillage, or an animal licking a freshly sprayed surface. Chronic low-level exposure is hard to diagnose.

Fungicides are different. Many contain heavy metals (copper, mercury) that harm animals as well as fungi. Seed treated with a fungicide is a classical hazard when leftover seed is fed to animals.

Common sources of error: assuming that a product that is safe on the label is safe when swallowed as concentrate; forgetting that *formulation* (solvent, surfactant) can be more toxic than the active ingredient; and giving the wrong decontamination. Always get the product name, active ingredient, formulation and time of exposure.

Glyphosate

Glyphosate is a broad-spectrum systemic herbicide. It blocks the plant enzyme **EPSP synthase** in the shikimate pathway, which makes aromatic amino acids. Mammals have no shikimate pathway, so the main target is absent. Merck notes that its immediate toxicity is very low at most environmental concentrations.

A frequent confusion: glyphosate is a phosphorus-containing compound (a phosphonate) but is **not** a cholinesterase inhibitor, so atropine and pralidoxime are not indicated just because the label says "organophosphorus". Concentrated formulations include **surfactants** that irritate the eye, skin and gut and may cause more of the clinical effect than glyphosate itself. Exposure in dogs and cats can cause eye and skin irritation, drooling, vomiting and unsteadiness, which usually resolve when exposure stops. Management is to wash skin and eyes, give fluids

and symptomatic care. Questions about longer-term effects are still being studied, and regulatory status is debated, so check the current notification.

In practice, a poisoning history should include what was sprayed, when, the mixing strength if known, and whether the animals drank from puddles, troughs or ponds near the field. Keep the container and label for the veterinarian and the poison information service. Rinse skin and coat with plenty of water, and prevent the animal from licking itself until it has been washed.

Paraquat

Paraquat is a bipyridyl herbicide and is among the most dangerous. Its toxicity rests on **redox cycling**: inside cells it accepts an electron, then passes it to oxygen, making superoxide and other reactive oxygen species, and the cycle repeats. This consumes NADPH and causes lipid peroxidation and cell death. It is actively taken up into lung cells through a polyamine uptake system, so the lung accumulates it. Damage is greatest in the lung, kidney and liver.

Signs after swallowing: mouth and throat ulceration, vomiting, diarrhoea, then kidney and liver injury. Over days a **progressive pulmonary injury** develops with breathlessness and, in survivors, fibrosis. Merck states that there is no specific treatment and that oxygen therapy and fluids are contraindicated, because extra oxygen increases radical formation. Management is early and aggressive decontamination with adsorbents (activated charcoal, bentonite or Fuller's earth) and cathartics, and supportive care. Because the lung injury is delayed, an animal may look better for a day or two before it deteriorates, and the prognosis for a significant dose is poor.

Paraquat has caused many human and animal poisonings in India. Check the current notification on its sale, restriction or ban, and always handle it as a high-risk product. Seek urgent veterinary and poison-centre help in any suspected ingestion.

2,4-D and phenoxy herbicides

2,4-D is a synthetic plant growth regulator, mimicking the plant hormone auxin, so that weeds grow uncontrollably and die. The group is described as essentially non-toxic to animals at normal exposures, with the exception of one older member (silvex). In very large exposures animals may show vomiting, drooling, weakness

and muscle stiffness or twitching, and some dogs show kidney or liver effects. Treatment is supportive: remove the animal from the source, give fluids, use adsorbents, and monitor.

The more important hazards are indirect. Plants sprayed with growth regulators may become more palatable, so livestock graze poisonous weeds they would normally avoid, and stressed plants may accumulate nitrate, a point that links to the nitrate chapter. Always keep animals off sprayed pasture for the period given on the label.

Copper fungicides

Copper sulfate with lime (Bordeaux mixture) and copper oxychloride are used on grapes, vegetables and other crops. **Species differ greatly**. Sheep are very sensitive because they absorb and store copper in the liver and excrete little. Cattle, goats and pigs tolerate more. Chronic exposure, for example from grazing sprayed orchard or vineyard ground or from feed with too much copper, builds up in the liver for weeks without signs. Then a stressor releases copper suddenly and causes a **haemolytic crisis**: jaundice, dark red-brown urine (haemoglobinuria), weakness, and kidney damage.

Acute swallowing of concentrate is a gastrointestinal irritant, with colic, salivation, green diarrhoea and shock. Management concepts: stop the source, give symptomatic and fluid support, and use a veterinarian's advice on chelators and other copper-lowering therapy. Prevention is the key: keep sheep away from sprayed ground and from feed formulated for other species.

Organomercury fungicides and prevention

Organomercury compounds were widely used as seed dressings. Mercury binds sulfhydryl groups on many enzymes and proteins. Alkyl mercury compounds (such as methyl mercury) cross the blood-brain barrier and cause **neurological signs**: blindness, incoordination, tremor and paralysis, often appearing weeks after exposure. Inorganic and phenyl mercury compounds more often cause gut and **kidney** injury. Human outbreaks have followed the eating of dressed seed grain, and animals fed with it were also affected.

Treatment is difficult once nerve damage is present. Remove the source, give supportive care, and consult a toxicologist about chelating agents such as

dimercaprol or succimer. Most countries have banned or restricted organomercurials, but old seed stocks may still exist. Check the current notification for India.

Prevention in practice: never feed treated seed or the washings of seed to animals; store pesticides in labelled, locked containers away from feed; never reuse pesticide containers for feed or water; observe the withholding period before grazing sprayed fields; wash sprayer equipment away from water sources; and keep poison-centre or veterinary toxicology contacts ready. **Seek urgent help** for any suspected paraquat or mercury exposure, and for sheep with jaundice and dark urine after grazing sprayed ground.

Concept cleared

Concept cleared: Why glyphosate is low toxicity
Glyphosate blocks a factory line that exists in plants but not in animals. Animals buy these amino acids ready-made from food, so the blocked line does not hurt them.

Concept cleared: Redox cycling
Paraquat is like a ball bounced between two walls. Each bounce makes a spark of free radicals, and the ball is not used up, so one molecule can cause damage again and again.

Concept cleared: Oxygen and paraquat
Oxygen is the fuel for paraquat's sparks. Giving extra oxygen to a paraquat-poisoned animal supplies more fuel for radical injury, which is why it is avoided.

Concept cleared: Copper storage in sheep
Sheep keep copper in the liver like a bank deposit that grows quietly. Stress can cause a sudden withdrawal of the whole amount into blood, and red cells burst.

Memory aid: Herbicide and fungicide quick code

Glyphosate = Gentle (no cholinesterase). Paraquat = Pulmonary, Poor outlook, avoid Pure oxygen. 2,4-D = Dull toxicity, but Dangerous weeds eaten. Copper = Chronic store in Sheep. Mercury = Mind and kidney, from Mouldy-treated seed.

Compare and contrast

Herbicide and fungicide poisoning at a glance

Agent	Mechanism	Main signs	Key point
Glyphosate	Blocks plant EPSP synthase; surfactant irritant	Irritation, drooling, vomiting	Low toxicity; not a cholinesterase inhibitor
Paraquat	Redox cycling, free radicals, lung uptake	Mouth ulcers, delayed lung injury	No specific treatment; avoid oxygen; poor prognosis
2,4-D	Plant auxin mimic	Usually mild; vomiting, weakness	Indirect hazards from palatable or nitrate-rich weeds
Copper fungicides	Hepatic copper accumulation, haemolysis	Jaundice, haemoglobinuria; GI signs if acute	Sheep very sensitive
Organomercury	Binds sulfhydryl groups	Neurological or renal signs	Treated seed grain is the main hazard

Glyphosate versus true organophosphate insecticides

Feature	Glyphosate	Organophosphate insecticide
Target	Plant EPSP synthase	Acetylcholinesterase
Cholinergic signs	No	Yes (salivation, miosis, tremor)
Atropine useful	No	Yes
Typical toxicity	Low	Moderate to high

Species notes

Dog

Often exposed by licking sprayed lawns or concentrate. Glyphosate and 2,4-D usually cause mild signs, while paraquat can be fatal.

Cat

Grooming of sprayed fur causes oral exposure. Keep cats indoors during and after spraying.

Sheep

Highly sensitive to copper, including copper fungicide residues; may develop a haemolytic crisis.

Cattle and buffalo

Graze sprayed pasture or eat treated seed. Observe withholding periods and avoid weeds growing after spray, which may hold nitrate.

Pig

May be given waste grain; treated seed is a hazard, including mercury-treated seed.

Clinical pearls

Self-test: 10 questions

1. Glyphosate acts on which plant enzyme?

It blocks EPSP synthase in the shikimate pathway, which animals lack.

2. Glyphosate poisoning should not be treated with atropine routinely because:

Glyphosate does not inhibit cholinesterase, so cholinergic signs are not expected.

3. Much of the irritation from glyphosate products is due to:

Formulation additives can be more irritant than the active ingredient.

4. The main mechanism of paraquat toxicity is:

Paraquat repeatedly accepts and passes on electrons, generating radicals.

5. Paraquat damage is most notable in the:

It is concentrated by lung cells and causes progressive pulmonary injury.

6. Which is advised in paraquat ingestion?

Oxygen worsens radical injury; early adsorption limits absorption.

7. 2,4-D belongs to the group called:

It is a phenoxy acid, a plant auxin mimic of low animal toxicity.

8. Which species is most sensitive to copper?

Sheep accumulate copper in the liver and excrete little.

9. A haemolytic crisis in sheep after chronic copper exposure produces:

Sudden copper release from the liver ruptures red cells.

10. The usual source of organomercury poisoning in animals is:

Seed dressings contained mercury and leftover treated grain can be fed by mistake.

Key points to revise

1. Most herbicides are low toxicity unless concentrate is swallowed.
2. Glyphosate blocks plant EPSP synthase and is not an anticholinesterase.
3. Paraquat causes free-radical injury and delayed lung damage with a poor prognosis.
4. Early adsorbent and cathartic use matters in paraquat poisoning.
5. 2,4-D is of low toxicity but changes plant palatability and may raise nitrate.
6. Sheep are very sensitive to copper; chronic build-up then haemolytic crisis.
7. Organomercury causes neurological and renal damage; treated seed is the hazard.
8. Check current Indian notifications for banned or restricted pesticides.

Go deeper on this site

- Ch 6. Plant poisoning in Indian livestock (in this book)
- Ch 4. Rodenticide poisoning (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Overview of herbicide poisoning in animals
- Merck Veterinary Manual: Organic herbicides toxic to animals
- Merck Veterinary Manual: Toxicology

CHAPTER 6 OF 17

Plant poisoning in Indian livestock

Most livestock plant poisoning follows hunger, drought, flood or a change in feed, not wickedness of the plant. This chapter covers cyanogenic plants, nitrate and oxalate, oleander and related cardiotoxic plants, common toxic weeds, and the seasonal patterns behind outbreaks.

> **After this chapter you can**
>
> - Explain how cyanogenic glycosides release cyanide and how cyanide blocks cell respiration

- Describe nitrate to nitrite conversion and methaemoglobinaemia in ruminants
- Explain how soluble oxalates cause hypocalcaemia and kidney damage
- Describe how cardiac glycoside plants such as oleander affect the heart
- Link outbreaks with season, hunger, drought, rain and fodder practices

Why animals eat poisonous plants

Livestock usually avoid poisonous plants because of taste, but poisoning happens when choice is removed. Typical settings are overgrazed or drought pasture, hungry animals moved to a new field, animals transported and let out hungry, early monsoon flush when only new shoots are green, floods that wash seeds or roots into fields, and garden or roadside cuttings thrown to animals. Animals unfamiliar with a local plant are at greater risk than those raised there.

Toxicity depends on **dose, part of the plant, stage of growth, weather, species and rumen function**. A plant may be safe at one stage and deadly at another. Diagnosis rests on history, plants seen in the field, rumen content examination for plant fragments, and laboratory tests where available. Because specific antidotes are few, prevention and early removal from the source are the foundation. Always contact a veterinarian early.

Cyanogenic plants

Plants such as young or stressed sorghum and Sudan grass, cassava, some Prunus species and certain wild plants hold **cyanogenic glycosides** (for example dhurrin and linamarin). These are harmless while intact. When the plant is wilted, frosted, trampled, chewed or fermented in the rumen, plant enzymes (beta-glucosidases) and rumen microbes split the glycoside and release **hydrogen cyanide**.

Cyanide binds to **cytochrome oxidase** in mitochondria, so cells cannot use oxygen even though blood carries plenty. This is **histotoxic hypoxia**. The blood stays bright red because oxygen is not extracted. Signs begin minutes after eating: distress, rapid laboured breathing, salivation, tremors, staggering, convulsions and sudden death. Ruminants are more susceptible than monogastrics because the rumen provides enzymes and a suitable pH for release.

Risk is highest with **young regrowth, drought-stressed plants and first rain after drought**, with hungry animals, and with fresh cut fodder. Cutting and wilting, then

ensiling or full drying lowers cyanide, though caution is still needed. The antidote concept is two-step: sodium nitrite makes methaemoglobin, which attracts cyanide away from cytochrome oxidase, and sodium thiosulfate supplies sulfur for the enzyme rhodanese to convert cyanide to thiocyanate. Merck also mentions hydroxocobalamin. Treatment must be given fast by a veterinarian, so move animals off the pasture and call for help early.

Nitrate and nitrite

Fast-growing or stressed plants can accumulate **nitrate**, which is not very toxic by itself. Common sources: sorghum, maize, oats, millets, and weeds such as Amaranthus (pigweed) and Chenopodium, especially after drought followed by rain, heavy nitrogen fertiliser, cloudy weather or herbicide stress. Contaminated well water can also contribute.

In the rumen, microbes convert nitrate to **nitrite**. Nitrite oxidises haemoglobin iron to give **methaemoglobin**, which cannot carry oxygen. Blood is a typical **chocolate brown** and mucous membranes look muddy. Signs are weakness, rapid breathing, tremors, staggering, and sudden death. Pregnant animals may abort later. The common confusion is that cyanide gives bright red blood and nitrite gives brown blood, and that nitrite is used therapeutically as an antidote to cyanide, yet nitrite itself is the toxin here.

Merck gives methylene blue as the treatment, to reduce methaemoglobin back to haemoglobin, together with removal of the source. Hungry animals eating suddenly are at highest risk, and feeding grain or gradual introduction can lower risk. Hay does not lose nitrate quickly; silage reduces it.

Oxalate plants

Soluble oxalates occur in Rumex (dock), Oxalis, Amaranthus, Portulaca and some tropical grasses. Oxalate binds calcium, so blood calcium falls (**hypocalcaemia**), producing weakness, tremors and recumbency. Calcium oxalate crystals can also form in kidney tubules and cause **renal failure**.

Rumen microbes of adapted animals break down oxalate, so sudden intake by unadapted, hungry animals is most risky. Treatment is calcium by a veterinarian and supportive fluids. Some tropical grasses with high oxalate cause long-term calcium

imbalance, mainly in horses; this is a different, slower problem from acute oxalate poisoning.

Oleander and other cardiotoxic plants

Oleander (*Nerium oleander*, kaner) and yellow oleander (*Thevetia peruviana*) are common garden and roadside shrubs in India. They contain **cardiac glycosides** that inhibit the sodium-potassium ATPase. Intracellular sodium and then calcium rise, the heart becomes irritable, and potassium rises in blood. Poisoning looks like digoxin overdose: colic, salivation, diarrhoea, weakness, irregular, slow or rapid pulse, heart block and sudden death. A small amount of fresh or dry leaf can be fatal, and drying does not remove toxicity.

Other plants with cardiac glycoside-like action include Calotropis (akand, which also has irritant latex) and other members of the same plant family. Treatment concepts: remove animals from the source, give activated charcoal early, monitor the ECG, correct potassium problems, and use antiarrhythmic drugs under veterinary advice. Antibody fragments for digoxin have been used in selected cases. Prevention is important: never throw oleander prunings into animal enclosures, never use oleander wood for skewers or fodder, and fence off shrubs near sheds.

Toxic weeds and seasonal risks

Several weeds in India are important. **Lantana camara** contains lantadenes that damage the liver and stop bile flow, causing jaundice and **photosensitisation** (sunburn of unpigmented skin) in cattle and buffalo. **Castor** (*Ricinus communis*) contains ricin, a protein synthesis inhibitor that harms gut and organs. **Ipomoea carnea** (besharam) has been reported to cause nervous signs in goats and sheep. **Cassia occidentalis** seeds in feed have been reported to cause muscle degeneration. Plants with pyrrolizidine alkaloids, such as Crotalaria, cause slow, cumulative liver damage. **Parthenium** mainly causes skin allergy and may taint milk and meat, so treat reports with care.

Seasonal pattern, in outline: **early monsoon** flush and first rain after drought (cyanide, nitrate); **summer and drought** (hunger, shortage of fodder, wilting plants); **floods and post-flood** (new weed growth, mouldy fodder); **winter** frost-damaged sorghum; and **festival and garden waste** seasons (oleander, castor). Prevention:

provide adequate clean feed before turnout, avoid grazing hungry animals on risky crops, cut and dry or ensile sorghum, test suspect fodder for nitrate, identify and remove weeds, fence hazardous shrubs, and teach farmers to call early. Treat sudden death in the field as a possible plant poisoning until shown otherwise.

When sudden deaths occur in a herd, collect rumen content, fodder samples and plant specimens in separate clean containers and send them with a full history. Move the survivors to a safe paddock with clean fodder and water. Do not slaughter sick animals for meat or milk use until a veterinarian has advised on withdrawal and safety.

Concept cleared

Concept cleared: Cyanide release
A cyanogenic plant is a locked box. Chewing, wilting or the rumen unlocks the box and cyanide spills out. The animal is not low on oxygen in blood; its cells simply cannot use it.

Concept cleared: Bright red versus chocolate brown
Cyanide stops cells using oxygen, so blood stays red. Nitrite changes haemoglobin so it cannot carry oxygen, so blood turns brown. Same hypoxia, different place in the chain.

Concept cleared: Oxalate and calcium
Oxalate is a thief of calcium. It binds calcium in the gut and blood, leaving muscles short, and the crystals can then clog the kidney.

Concept cleared: Hunger decides
Most plant poisonings are about opportunity. A hungry animal eats what a fed animal ignores, so the cheapest prevention is good feeding.

Memory aid: Plant poisoning quick code

Cyanide = Cherry-red blood, Cells cannot breathe. Nitrate = Nitrite makes Brown blood (methylene Blue). Oxalate = Oxalate Overtakes calcium (hypocalcaemia, kidney). Oleander = Odd rhythm, like digoxin. Lantana = Light-sensitive skin (photosensitisation).

Compare and contrast

Plant poisonings at a glance

Toxin	Example plants	Mechanism	Key sign or test	Treatment concept
Cyanogenic glycosides	Young sorghum, cassava	Cyanide blocks cytochrome oxidase	Sudden death, bright red blood	Sodium nitrite and thiosulfate, or hydroxocobalamin
Nitrate	Sorghum, maize, Amaranthus	Nitrite makes methaemoglobin	Chocolate brown blood	Methylene blue
Oxalate	Rumex, Oxalis	Binds calcium, kidney crystals	Hypocalcaemia, renal failure	Calcium, fluids
Cardiac glycosides	Oleander, Thevetia	Na-K ATPase inhibition	Arrhythmia, colic, sudden death	Charcoal, ECG monitoring, antiarrhythmics

Cyanide versus nitrate poisoning

Feature	Cyanide	Nitrate/nitrite
Site of block	Cytochrome oxidase	Haemoglobin (methaemoglobin)
Blood colour	Bright red	Chocolate brown
Onset	Minutes	Hours
Antidote	Nitrite plus thiosulfate	Methylene blue

Species notes

Cattle and buffalo

Goat

Most often affected by cyanide, nitrate and Lantana because they graze coarse pasture and cut fodder. Rumen conversion makes them more sensitive to cyanide and nitrate.

Browse many shrubs and are exposed to oleander, Ipomoea and other shrubs when grazing is short.

Sheep
Graze close to the ground and are exposed to weeds; oxalate plants and oleander clippings are risks.

Horse
Monogastric, so less affected by cyanide and nitrate from plants than ruminants, but sensitive to oleander and to some oxalate grasses over time.

Dog
Chew garden shrubs and may be exposed to oleander and castor seeds; call a veterinarian early.

Clinical pearls

Self-test: 10 questions

1. Cyanide poisoning blocks:

Cyanide binds cytochrome oxidase and stops cells using oxygen.

2. The colour of blood in acute cyanide poisoning is typically:

Oxygen is not extracted by tissues, so venous blood stays bright red.

3. Which situation raises the risk of cyanide poisoning from sorghum?

Young and stressed plants hold more cyanogenic glycoside.

4. Why are ruminants more vulnerable to plant cyanide?

Rumen enzymes and pH release HCN.

5. In nitrate poisoning of cattle the toxic step is conversion of nitrate to:

Nitrite oxidises haemoglobin to methaemoglobin.

6. Chocolate brown blood suggests:

Methaemoglobin cannot carry oxygen and gives brown blood.

7. The treatment given for nitrite methaemoglobinaemia is:

Methylene blue helps reduce methaemoglobin to haemoglobin.

8. Soluble oxalate plants cause poisoning mainly through:

Oxalate binds calcium, causing hypocalcaemia and kidney crystals.

9. Oleander poisoning is similar to overdose of:

Both inhibit Na-K ATPase and cause arrhythmia.

10. Photosensitisation with jaundice in cattle after grazing is most characteristic of:

Lantadenes damage the liver and bile flow, so phylloerythrin accumulates and sensitises skin.

Key points to revise

1. Plants are most dangerous when animals are hungry or the plant is stressed.
2. Cyanogenic glycosides release HCN, which blocks cytochrome oxidase.
3. Nitrate is reduced to nitrite in the rumen and forms methaemoglobin.
4. Methylene blue treats nitrite methaemoglobinaemia.
5. Oxalate binds calcium and may injure kidneys.
6. Oleander glycosides inhibit Na-K ATPase and cause arrhythmia.
7. Lantana causes liver injury and photosensitisation in cattle.
8. Prevention: feed before turnout, remove weeds, fence shrubs, call early.

Go deeper on this site

- Ch 7. Heavy metals and mineral poisoning (in this book)
- Ch 5. Herbicide and fungicide poisoning (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Cyanide poisoning in animals
- Merck Veterinary Manual: Nitrate and nitrite poisoning in animals
- Cornell University: Plants poisonous to livestock

CHAPTER 7 OF 17

Heavy metals and mineral poisoning

How lead, arsenic, copper, selenium, fluoride, molybdenum and zinc injure animals, why species differ so much, and how chelators work. The emphasis is on mechanism, recognising the pattern, removing the source and preventing exposure.

After this chapter you can

- Explain how lead and arsenic damage cells through sulphydryl binding and enzyme inhibition

- Describe why sheep are especially prone to copper poisoning and how molybdenum and sulphur interact with copper
- Distinguish acute from chronic selenium and fluoride toxicity and name the typical lesions
- Recognise zinc toxicity from foreign bodies in dogs and its haemolytic effect
- Explain the principle of chelation, the main chelating agents and their limitations

Why metals and minerals poison

Metals are not broken down by the body. They bind to proteins, replace essential minerals or sit in tissues for a long time. That is why many metal poisonings are **chronic or cumulative**, and why a single clinical case often points to a problem in the whole herd, feed or water supply.

Two broad groups exist. Classical toxic metals such as lead and arsenic have no useful role in the body. Essential trace minerals such as copper, selenium, zinc and molybdenum are needed in small amounts, but the gap between enough and too much can be narrow. Deficiency and toxicity of the same mineral can occur in the same district.

Always think of three questions: what is the source, which species is exposed, and is the exposure acute (one large intake) or chronic (small repeated intake)?

In practice, a good history is the most valuable test. Ask about recent changes in feed, new mineral mixtures, repainting or renovation, burning of waste, new water sources and access to scrap. Laboratory confirmation (blood, liver, kidney, feed and water analysis) should be arranged through a veterinary diagnostic laboratory, and samples should be collected before treatment where possible. Report herd-level problems to the local animal husbandry authority, because the source may harm other animals and people.

Lead

Lead is among the commonest toxic metals in animals. Sources include old paint, batteries, lead shot, used engine oil, putty, linoleum and pasture contaminated by smelters or burning of lead-containing waste. Young, curious cattle and dogs are most often affected.

Mechanism. Lead binds sulphydryl groups and inhibits enzymes of haem synthesis (such as ALA dehydratase and ferrochelatase). It also behaves like calcium, interferes with calcium-dependent processes, and damages the small blood vessels of the brain, causing oedema and neuronal injury.

Signs. In cattle the picture is mainly nervous: blindness, head pressing, bellowing, muscle twitching, teeth grinding and convulsions. In dogs gastrointestinal signs (vomiting, abdominal pain) often appear together with nervous signs. Nucleated red cells and basophilic stippling without marked anaemia support the suspicion, but diagnosis rests on blood lead measurement and the history.

Management concepts. Stop further exposure first. Chelators are used to bind absorbed lead, and supportive care controls seizures and brain swelling. If lead objects remain in the gut, removal is needed, because chelation alone can leave a continuing source.

Arsenic

Arsenic exists in organic and inorganic forms. **Inorganic trivalent arsenicals** are the more toxic. Sources include old herbicides, wood preservatives, sheep and cattle dips and contaminated water. Arsenic in groundwater is a recognised public health problem in parts of India and neighbouring countries, so check the current local advisories.

Mechanism. Trivalent arsenic binds sulphydryl groups of enzymes, especially those needing lipoic acid, and so blocks cellular energy production. It also damages capillaries, especially in the gut.

Signs. Acute poisoning causes severe abdominal pain, salivation, vomiting in species that can vomit, watery or bloody diarrhoea, weakness, collapse and death, often within a day. Chronic exposure is subtler: poor condition, skin and gut changes.

Antidote reasoning. Dithiol chelators such as dimercaprol and succimer offer competing sulphydryl groups so that arsenic leaves the enzymes. They work best early, before damage is established.

Copper and molybdenum

Sheep are very sensitive to copper because they absorb it efficiently and store it in the liver with poor biliary excretion. Poisoning is often *chronic*: copper builds up silently over weeks, then a stressor triggers a sudden release causing a **haemolytic crisis** with jaundice, dark haemoglobin-stained urine and death. Typical causes are feeds made for cattle or pigs given to sheep, or contaminated pasture.

Molybdenum and sulphur bind copper in the rumen as insoluble thiomolybdates, lowering copper absorption. Excess molybdenum therefore causes *secondary copper deficiency* in cattle (scouring, poor growth, loss of coat colour), while a low molybdenum intake makes sheep copper poisoning more likely. This is a common confusion: molybdenum toxicity is really a copper deficiency state.

Dogs. Certain breeds, such as the Bedlington terrier, inherit a defect in copper handling and develop chronic liver disease.

Prevention depends on correct feed formulation. Treatment aims to bind or reduce liver copper, but advanced haemolysis carries a poor outlook.

Selenium, fluoride and zinc

Selenium. It is essential (part of glutathione peroxidase) yet toxic in excess, with a narrow safety margin. Selenium replaces sulphur in amino acids and promotes oxidative damage. Chronic poisoning in grazing animals, linked to seleniferous soils and accumulator plants, causes hair loss, hoof deformity and lameness. Acute poisoning is usually due to dosing or supplement errors and causes respiratory distress, sudden death or cardiac failure. Some regions of India are reported to have high-selenium soils; check local data.

Fluoride. Sources are high-fluoride groundwater, rock phosphate supplements and industrial emissions. Fluoride enters bone and teeth, forming fluorapatite. Chronic **fluorosis** gives mottled, worn teeth (only if exposure occurred while teeth were forming), bony outgrowths, stiffness and lameness. Fluorosis is reported from several Indian states. There is no specific antidote, so prevention by water testing and low-fluoride supplements is the main tool.

Zinc. Dogs swallow galvanised nuts, bolts, some coins and zinc-containing ointments. Gastric acid releases zinc, which causes **intravascular haemolysis**, and may injure pancreas, liver and kidney. The foreign body should be removed; supportive care follows.

Chelation: principles and cautions

A **chelator** grips a metal ion with several binding sites, forming a stable ring-like complex that is water soluble and excreted, mainly in urine or bile. The choice depends on the metal.

- **Calcium disodium EDTA**: lead and zinc. It is given by injection, and it can injure kidneys. Do not confuse it with disodium EDTA, which can cause dangerous hypocalcaemia.
- **Dimercaprol (BAL)**: arsenic. It is an oily, painful injection.
- **Succimer (DMSA)**: lead, arsenic; oral and generally better tolerated.
- **D-penicillamine**: copper and lead, mainly oral.
- **Tetrathiomolybdate**: reduces copper uptake.

Cautions. A chelator is not a substitute for removing the source. Chelators may also remove essential minerals, and some can increase absorption of metal still in the gut. Dosing must follow a current formulary and veterinary toxicology advice, not a textbook. In any suspected metal poisoning, contact a poison information service or toxicologist early.

Concept cleared

Cumulative toxicity
Metals persist in tissues. Small daily intakes add up until clinical disease appears, often after a stressor.

Sulphydryl binding
Lead and arsenic inactivate enzymes by binding their sulphydryl groups. Chelators with their own sulphydryl groups compete for the metal.

Mineral antagonism
Molybdenum and sulphur lock copper in the gut. Excess of one mineral can produce deficiency of another.

Source control first

Antidotes and chelators cannot work well while the animal is still being exposed. Find and remove the source.

Memory aid: Remember the metal and its target

Lead hits head and heme, arsenic attacks the gut, copper kills sheep, fluoride fixes in bone, zinc breaks blood.

Compare and contrast

Metals at a glance

Agent	Main mechanism	Typical presentation	Antidote concept
Lead	Blocks haem synthesis, vascular and neuronal injury	Blindness, convulsions (cattle); GI plus nervous (dogs)	Calcium EDTA, succimer, source removal
Arsenic (inorganic)	Sulphydryl binding, energy block, capillary damage	Acute haemorrhagic gastroenteritis	Dimercaprol, succimer
Copper	Liver storage then haemolytic release	Jaundice, haemoglobinuria (sheep)	Prevention; tetrathiomolybdate, penicillamine
Fluoride	Replaces hydroxyl in bone and teeth	Dental mottling, lameness	None specific; prevention
Zinc	Haemolysis, organ injury	Anaemia, dark urine (dogs)	Remove object, supportive

Acute versus chronic mineral poisoning

Feature	Acute	Chronic
Exposure	One large intake or error	Repeated small intake
Onset	Hours to days	Weeks to months
Examples	Arsenic, selenium dosing errors, zinc object	Copper in sheep, fluorosis, selenium on pasture
Main aim	Decontaminate and chelate early	Find the source, correct diet and water

Species notes

Sheep

Highly sensitive to copper; chronic accumulation then haemolytic crisis. Avoid feeds meant for other species.

Cattle

Commonest victims of lead (nervous signs); molybdenum excess causes secondary copper deficiency.

Dogs

Lead and zinc from household objects; inherited copper storage disease in some breeds.

Horses

Lead from pasture near contaminated sites can cause laryngeal and nervous signs; fluoride affects teeth and bone.

Pigs and poultry

Sensitive to selenium excess and to arsanilic-type feed additives if overdosed or mixed wrongly.

Clinical pearls

Self-test: 10 questions

1. Which enzyme pathway is a main target of lead?

Lead inhibits ALA dehydratase and ferrochelatase, so haem synthesis is impaired.

2. Typical presentation of lead poisoning in a calf is:

Cattle show predominantly nervous signs.

3. Why do dithiol chelators help in arsenic poisoning?

Competing sulphydryl groups pull arsenic away from enzymes.

4. Which species is most sensitive to chronic copper accumulation?

Sheep absorb and store copper efficiently and excrete it poorly.

5. Excess dietary molybdenum in cattle mainly causes:

Thiomolybdates bind copper in the rumen.

6. Mottled and worn teeth with bony outgrowths suggest:

Chronic fluoride affects developing enamel and bone.

7. A dog that swallowed galvanised nuts develops anaemia and dark urine. The likely mechanism is:

Zinc released in the stomach damages red cells.

8. A caution about calcium disodium EDTA is:

It can damage kidneys; disodium EDTA risks hypocalcaemia.

9. What should come first when managing any metal poisoning?

Antidotes are poorly effective if exposure continues.

10. Selenium excess in grazing animals commonly produces:

Chronic selenosis causes hair and hoof changes and lameness.

Key points to revise

1. Metals bind enzymes and persist, so many poisonings are chronic or cumulative.
2. Lead blocks haem synthesis and injures brain vessels; cattle show blindness and convulsions.
3. Inorganic trivalent arsenic binds sulphydryl groups and damages gut capillaries.
4. Sheep store copper and may suffer a sudden haemolytic crisis after stress.
5. Molybdenum and sulphur reduce copper availability; excess molybdenum causes secondary copper deficiency.
6. Selenium has a narrow margin between need and toxicity.
7. Fluorosis damages developing teeth and bone and has no specific antidote.
8. Chelators are metal specific, can be toxic, and never replace source removal.

Go deeper on this site

- Ch 8. Feed-related toxicities (in this book)
- Ch 6. Plant poisoning in Indian livestock (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Lead poisoning in animals
- Merck Veterinary Manual: Copper poisoning in animals
- Merck Veterinary Manual: Fluoride poisoning in animals

CHAPTER 8 OF 17

Feed-related toxicities

Poisonings that arise from what animals eat or drink: urea and other non-protein nitrogen, salt and water deprivation, nitrate, ionophores and the major mycotoxins. The chapter links each toxin to its mechanism, its species pattern and the practical prevention step.

After this chapter you can

- Explain why ruminal pH and adaptation determine urea toxicity and how acidifying the rumen helps
- Describe salt poisoning as water deprivation sodium toxicosis and the danger of rapid rehydration
- Explain nitrate to nitrite to methaemoglobin and its antidote reasoning
- Describe ionophore toxicity and the species that are most at risk
- Distinguish aflatoxin, ergot and the main Fusarium toxins by source, mechanism and signs

Feed as a source of poison

Many livestock poisonings are not caused by a wild plant or a spilt chemical but by a **feed that is normally safe**. Urea, salt, nitrate and ionophores are useful in the right amount and in the right species. Poisoning follows mixing errors, sudden changes, poor adaptation or feeding a product to the wrong species.

Three themes recur. First, **the rumen** changes both toxicity and safety. Second, **mixing and storage** errors cause herd outbreaks. Third, mycotoxins arise when feed is stored damp or warm, so prevention starts long before the animal eats.

Because many animals share the same ration, a feed problem usually appears as several animals sick or dead within a short period. This pattern is itself a diagnostic clue.

When investigating a suspected feed problem, stop the suspect feed at once, keep a sealed sample of the batch for analysis, and record which animals ate it and when. Check feed mill records for mixing errors and for any recent change of ingredient or supplier. Samples of feed, rumen contents, liver and serum are useful,

but they must be collected and stored correctly. In suspected cases involving milk or meat, ask the authorities about withholding and residue rules and check the current notification.

Urea and non-protein nitrogen

Ruminants can use urea as a cheap nitrogen source because rumen microbes split it with **urease** into ammonia and rebuild it as microbial protein. Poisoning occurs when ammonia is released faster than microbes can use it.

Why ruminal pH matters. Ammonia exists as ammonium ion (poorly absorbed) in an acid rumen, but as free ammonia (readily absorbed) when pH rises. Excess ammonia overwhelms the liver, and the blood ammonia then rises sharply.

Risk factors: unadapted animals, fasted animals eating a large amount quickly, poor mixing, a low-energy diet, and access to fertiliser bags. Non-ruminants do not have the rumen urease and are much less susceptible.

Signs: restlessness, salivation, tremors, bloat, laboured breathing, incoordination, bellowing and rapid death.

Reasoning for treatment: acidifying the rumen with dilute acetic acid and giving cold water slows urease and traps ammonia as ammonium. This is an emergency, because treatment is only effective early. Prevention is gradual adaptation, even mixing and never feeding urea to calves with an undeveloped rumen.

Salt poisoning and water deprivation

Sodium chloride is not very toxic by itself when water is freely available. The true problem is **water deprivation sodium ion toxicosis**. Pigs, poultry and cattle with limited water that eat salty feed, whey, brine or salted waste develop hypernatraemia.

Mechanism. High sodium draws water out of brain cells. The brain protects itself by building up small organic solutes. If water is then given freely and quickly, water follows these solutes into the brain, causing **cerebral oedema**. This is why many animals worsen or die after sudden access to water.

Signs. In pigs: thirst, wandering, blindness, circling, head pressing, then convulsions, often in a dog-sitting posture. Brain lesions with eosinophilic cuffing

are classic in pigs. Ruminants show nervous signs and diarrhoea.

Management and prevention. Withdraw the salt source and restore water slowly and in small amounts, under veterinary supervision. Prevention is constant clean water and care with salty by-products.

Nitrate and nitrite

Nitrate comes from heavily fertilised or drought-stressed plants such as sorghum, oats and some weeds, and from contaminated well water. Nitrate itself is of low toxicity. In the rumen microbes reduce it to **nitrite**, which oxidises the iron of haemoglobin from the ferrous to the ferric state, forming **methaemoglobin** that cannot carry oxygen.

Ruminants are more susceptible than monogastrics because of this ruminal conversion, and a hungry animal eating suddenly is at highest risk.

Signs. Rapid breathing, weakness, muddy or chocolate-brown mucous membranes and blood, tremors, collapse and death. Pregnant cows may abort. The animal appears short of oxygen even though the lungs are normal.

Antidote reasoning. Methylene blue acts as an electron carrier that helps convert methaemoglobin back to haemoglobin. It is used by a veterinarian, because overuse has its own risks. Ensiling reduces nitrate, and testing feed and water before feeding is the best prevention.

Do not confuse nitrate poisoning with cyanide: cyanide blocks tissue use of oxygen and gives bright red blood.

Ionophores

Ionophore antibiotics such as monensin, lasalocid, salinomycin and narasin improve feed efficiency in ruminants and control coccidia in poultry. They carry ions across cell membranes and destroy ion gradients.

Mechanism. In toxic amounts they cause calcium overload and energy failure in muscle cells, with especially severe injury to the heart and skeletal muscle.

Species. Horses are extremely sensitive and may be poisoned by cattle or poultry feed containing monensin, often from a mixing error. Dogs, cats and turkeys are

also sensitive. Cattle tolerate a limited range. Some drugs, notably tiamulin, slow ionophore metabolism and can precipitate toxicity in pigs and poultry.

Signs. Inappetence, colic, sweating, muscle weakness, ataxia, recumbency, fast irregular heart rate, myoglobinuria and sudden death. Survivors may have permanent heart damage.

Management. There is no specific antidote. Remove the feed and give supportive care, and activated charcoal early in recent ingestion. Prevention is strict labelling, dedicated mixing equipment and never feeding ionophore feed to horses.

Mycotoxins: aflatoxin, ergot and Fusarium

Mycotoxins are fungal metabolites produced in the field or in storage. Heat, humidity and damaged grain favour them, and cooking or pelleting does not destroy most of them. Several can occur together.

- **Aflatoxin** (*Aspergillus flavus*, *A. parasiticus*): found in groundnut cake, maize and cottonseed. The liver activates aflatoxin B1 to a reactive epoxide that binds DNA and proteins. Effects: liver damage, poor growth, immunosuppression, bleeding and cancer risk. Ducklings, turkeys and young pigs are very sensitive. Dairy animals excrete aflatoxin M1 in milk, which is a public health issue. Check the current Indian limits for feed and food.
- **Ergot** (*Claviceps* species on grain and grasses): alkaloids act on adrenergic, serotonin and dopamine receptors, causing vasoconstriction and gangrene of tail, ears and feet, and in some cases nervous signs and poor milk.
- **Fusarium toxins:** T-2 toxin and other trichothecenes block protein synthesis (oral and gut lesions, bleeding); deoxynivalenol causes feed refusal and vomiting in pigs; **zearalenone** is oestrogenic (swollen vulva, prolapse, infertility); fumonisins cause leukoencephalomalacia in horses and pulmonary oedema in pigs.

Prevention rests on dry storage, testing, discarding mouldy feed and, where appropriate, binders; no binder works against all toxins.

Concept cleared

Rumen as a reactor

Microbes convert urea to ammonia and nitrate to nitrite. Rumen pH and adaptation decide whether the product is useful or dangerous.

Water deprivation toxicosis

Salt is dangerous when water is short. Rapid rehydration after hypernatraemia can cause brain oedema.

Wrong species, wrong feed

Ionophore feed safe for cattle or poultry can kill horses. Always check the label and the species.

Storage creates toxins

Mycotoxins develop in damp or warm feed and survive processing, so prevention is at harvest and storage.

Memory aid: Feed troubles: U-S-N-I-M

Urea, Salt, Nitrate, Ionophore, Mould: check the feed for each of these when a group of animals falls sick together.

Compare and contrast

Feed toxicities compared

Toxin	Key mechanism	Main species at risk	Characteristic sign
Urea	Rumen ammonia overload	Ruminants	Tremor, bloat, bellowing
Salt	Hypernatraemia, brain oedema on rehydration	Pigs, poultry, cattle	Wandering, convulsions
Nitrate	Methaemoglobin	Cattle, sheep	Brown blood, dyspnoea
Ionophores	Calcium overload in muscle	Horses, dogs, turkeys	Heart and muscle damage

Mycotoxins compared

Mycotoxin	Source fungus	Target	Typical outcome
Aflatoxin	Aspergillus	Liver	Hepatic injury, immunosuppression, milk residue
Ergot alkaloids	Claviceps	Blood vessels, receptors	Gangrene of extremities
Zearalenone	Fusarium	Oestrogen receptors	Vulvovaginitis, infertility
Fumonisin	Fusarium	Brain (horse), lung (pig)	Leukoencephalomalacia, pulmonary oedema

Species notes

Cattle and buffalo
Susceptible to urea and nitrate because of rumen conversion; also exposed to aflatoxin through cake and silage, with M1 in milk.

Sheep and goats
Similar to cattle for urea and nitrate; small body size makes errors in mixing serious.

Horses
Extremely sensitive to ionophores; fumonisin causes

Pigs
Classic salt poisoning with water deprivation; sensitive to

leukoencephalomalacia.

deoxynivalenol and zearalenone.

Poultry

Aflatoxin, ionophore mixing errors and salt excess cause losses; ducklings and turkeys are most sensitive to aflatoxin.

Clinical pearls

Self-test: 10 questions

1. Why is urea toxicity mainly a ruminant problem?

Rumen microbial urease splits urea into ammonia, which is absorbed if released too fast.

2. Which rumen condition increases ammonia absorption?

At high pH more ammonia is in the free, absorbable form.

3. The rationale for giving dilute acetic acid orally in urea poisoning is to:

Acid slows urease and converts ammonia to ammonium.

4. Why can rapid watering be harmful in salt poisoning?

Brain cells have built up solutes; sudden water entry causes swelling.

5. Which species classically show salt poisoning with dog-sitting posture and eosinophilic cuffing?

This is the classic porcine picture.

6. Methaemoglobin forms when nitrite:

Ferric haemoglobin cannot carry oxygen.

7. Which species is exceptionally sensitive to monensin?

Horses are highly sensitive and suffer heart and muscle injury.

8. Aflatoxin M1 is important because it:

M1 is a milk metabolite and a public health concern.

9. Gangrene of the tail and ear tips from contaminated grain suggests:

Ergot alkaloids cause vasoconstriction and dry gangrene.

10. Swollen vulva and prolapse in young gilts suggest:

Zearalenone is oestrogenic.

Key points to revise

1. Urea toxicity depends on ammonia release, rumen pH and lack of adaptation.
2. Acidifying the rumen and cooling it slows urea breakdown and traps ammonia.
3. Salt poisoning is mainly water deprivation sodium toxicosis; rapid water access worsens brain oedema.
4. Nitrite oxidises haemoglobin to methaemoglobin; methylene blue is the antidote concept.
5. Ionophores cause heart and skeletal muscle damage and are especially dangerous to horses.

6. Aflatoxin B1 is hepatotoxic and carcinogenic, and aflatoxin M1 appears in milk.
7. Ergot causes vasoconstriction and gangrene; zearalenone is oestrogenic.
8. Mycotoxin control is dry storage, testing and discarding bad feed.

Go deeper on this site

- Ch 9. Toxic gases and algal toxins (in this book)
- Ch 7. Heavy metals and mineral poisoning (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Nitrate and nitrite poisoning in animals
- WHO: Mycotoxins fact sheet
- Merck Veterinary Manual: Copper poisoning in animals (mineral feed toxicity background)

CHAPTER 9 OF 17

Toxic gases and algal toxins

How carbon monoxide, ammonia, hydrogen sulphide and nitrogen dioxide poison animals in sheds, pits and silos, and how cyanobacterial toxins make water deadly. The chapter stresses mechanism, rescuer safety, ventilation and prevention.

After this chapter you can

- Explain how carbon monoxide produces tissue hypoxia despite normal oxygen in the lungs
- Describe the irritant action of ammonia and its role in poultry and pig housing
- Explain hydrogen sulphide toxicity from manure pits and why smell is an unreliable warning
- Describe silo filler's disease from nitrogen dioxide

- Classify cyanobacterial toxins and describe the clinical pattern, prevention and first response

Gas and water hazards in the animal environment

Animals live and breathe in the same air for hours. Poor ventilation, fermentation and combustion can turn a shed, manure pit or silo into a hazard. Surface water can also turn toxic when cyanobacteria bloom. These poisonings are usually **sudden, affect several animals and sometimes harm people**.

Gases fall into three action types: **asphyxiants** that stop oxygen delivery or use (carbon monoxide, hydrogen sulphide), **irritants** that burn airway lining (ammonia, nitrogen dioxide), and simple oxygen displacement (carbon dioxide, methane).

The most important practical lesson is safety: many human deaths happen when a person enters a pit or silo to rescue an animal. **Never enter a confined space without proper breathing apparatus and trained help.**

Prevention is largely about design and habit: adequate ventilation sized for the number of animals, maintained heaters, covered and ventilated pits, gas detectors where available and a written emergency plan. Staff should be trained to raise the alarm instead of entering confined spaces alone. After any suspected gas event, ventilate fully before re-entry, examine all animals, and seek urgent veterinary and medical advice.

Carbon monoxide

Carbon monoxide (CO) is colourless and odourless. It comes from incomplete combustion: faulty or unvented heaters, charcoal or gas brooders in poultry sheds, engines running in closed spaces, generators and fires.

Mechanism. CO binds to haemoglobin with far greater affinity than oxygen, forming **carboxyhaemoglobin**. This reduces oxygen carrying capacity and also shifts the oxygen dissociation curve so the remaining oxygen is released poorly. CO also binds to cytochrome oxidase and myoglobin. The lungs are normal and the arterial oxygen tension is normal, yet tissues are starved. This is a common confusion: a normal pulse oximeter reading can be falsely reassuring, because it cannot tell carboxyhaemoglobin from oxyhaemoglobin.

Signs. Weakness, headache-like depression, rapid breathing, incoordination, collapse, convulsions and death. Mucous membranes may look bright cherry red, but this is not always seen. Chicks in brooders may die in numbers. Survivors can have delayed brain injury and abortion.

Response. Move animals into fresh air and give high-concentration oxygen, which displaces CO. Prevention: maintenance of heaters, ventilation and CO detectors.

Ammonia

Ammonia is formed by bacterial breakdown of urine and faeces, especially in wet poultry litter and poorly ventilated pig and calf houses. It has a sharp smell that people notice quickly.

Mechanism. Ammonia dissolves in the moist lining of the eyes and airways, forming an alkaline solution that injures cells and paralyses cilia. This allows bacteria and viruses to colonise the respiratory tract. Chronic low-level exposure is therefore a *predisposing cause* of respiratory disease, not just a direct poison.

Signs. Watery eyes, conjunctivitis and corneal damage (including keratoconjunctivitis in chickens), nasal discharge, coughing, reduced growth and increased susceptibility to infections such as colibacillosis. Very high levels cause acute respiratory distress.

Prevention. Good litter management, dry floors, adequate ventilation and sensible stocking density. A simple rule: if a person finds the air irritating at animal height, it is too much for the animals.

Do not confuse airborne ammonia in sheds with ammonia produced in the rumen after urea feeding.

Hydrogen sulphide

Hydrogen sulphide (H_2S) is released when manure or slurry decomposes without oxygen. The risk is highest when a pit is agitated, emptied or stirred, because trapped gas is suddenly released. The gas is heavier than air and collects in low places.

Mechanism. H_2S inhibits cytochrome c oxidase, the final enzyme of the respiratory chain, so cells cannot use oxygen. This is similar to cyanide in effect. At lower levels

it irritates the eyes and airways.

Warning smell. It smells of rotten eggs at low levels, but higher levels *paralyse the sense of smell*, so absence of smell does not mean safety.

Signs. Sudden collapse and death in pigs and other animals in the building (knock-down), with breathing failure. Lower exposure: eye irritation, coughing, nausea and pulmonary oedema. Workers who enter pits to rescue animals are often victims.

Prevention. Ventilate before and while agitating slurry, remove animals and people from the building during pit work, and never enter pits unprotected. Treatment is removal to fresh air and supportive care with oxygen.

Nitrogen dioxide and silo gases

When green forage with high nitrate is ensiled, fermentation produces **nitrogen oxides**, mainly nitrogen dioxide (NO2). The gas appears within hours and may persist for days to a few weeks after filling. It is yellow to brown, heavier than air and may collect at the silo base and near the feed outlet, entering the animal house or a person's lungs.

Mechanism. NO2 is poorly soluble, so it reaches deep into the lungs, where it forms nitric and nitrous acids that damage the air-blood barrier. The result is pulmonary oedema, and some days or weeks later, scarring bronchiolitis may develop. This delayed phase is a typical feature of **silo filler's disease** in people; animals may show coughing, rapid breathing and sudden death if exposed in poorly ventilated spaces.

Silos also fill with carbon dioxide, which displaces oxygen and silently asphyxiates.

Prevention. Keep people and animals away from fresh silage, ventilate adjacent areas, and follow local silo safety guidance. Seek urgent medical or veterinary help after any exposure, even if the first symptoms seem mild.

Blue-green algae and water-borne toxins

Cyanobacteria (blue-green algae) multiply in warm, still, nutrient-rich water, especially in summer. Wind can push a bloom to a shoreline where animals drink or dogs swim and lick their coats. The water may look green, turn like paint, or form scum, but sometimes shows no obvious change.

Main toxin groups:

- **Hepatotoxins** such as microcystins block protein phosphatases, collapsing the liver cell skeleton and causing liver bleeding. Signs appear within hours to a day or two: vomiting, bloody diarrhoea, weakness, pale or jaundiced membranes, shock.
- **Neurotoxins** such as anatoxin-a (nicotinic receptor agonist) and saxitoxins (sodium channel blockers) cause tremors, paralysis, breathing failure and death within minutes to hours. Anatoxin-a(s) is an anticholinesterase, producing heavy salivation.
- **Cylindrospermopsin** injures liver and kidney more slowly.

Response. There is no specific antidote for most. Remove animals from the water at once, provide clean water, decontaminate (rinse coat; activated charcoal or emesis only on veterinary advice) and give supportive care. Boiling does not remove the toxins. Livestock owners should seek urgent help if animals sicken after water contact.

Prevention includes fencing off affected water, providing alternative water and reducing nutrient run-off into ponds.

Concept cleared

Asphyxiant versus irritant
Carbon monoxide and hydrogen sulphide block oxygen delivery or use; ammonia and nitrogen dioxide burn the airway.

Normal lungs, starved tissues
In CO poisoning the lungs work normally but haemoglobin is occupied, so tissues are hypoxic.

Smell is not a safety test
Hydrogen sulphide paralyses smell at high levels, and carbon monoxide has no smell at all.

Rescuer safety

Never enter a pit, silo or enclosed shed without breathing apparatus and trained backup.

Memory aid: Gas safety: CAHN

Carbon monoxide, Ammonia, Hydrogen sulphide, Nitrogen dioxide. Ask for each: where from, how it acts, and how to ventilate and escape.

Compare and contrast

Toxic gases compared

Gas	Source	Mechanism	Main outcome
Carbon monoxide	Faulty heaters, brooders, engines	Carboxyhaemoglobin, tissue hypoxia	Collapse, cherry-red membranes sometimes
Ammonia	Wet litter, manure	Alkaline irritant to eyes and airway	Conjunctivitis, respiratory disease
Hydrogen sulphide	Manure pits, slurry agitation	Cytochrome c oxidase inhibition	Sudden knock-down deaths
Nitrogen dioxide	Fresh silage	Acid injury deep in lung	Pulmonary oedema, later bronchiolitis

Cyanobacterial toxins

Toxin type	Example	Target	Speed and signs
Hepatotoxin	Microcystin	Liver cells	Hours to days; vomiting, bloody diarrhoea, jaundice
Neurotoxin	Anatoxin-a, saxitoxin	Neuromuscular junction, sodium channels	Minutes to hours; tremor, paralysis
Anticholinesterase	Anatoxin-a(s)	Cholinesterase	Salivation, tremor
Cytotoxin	Cylindrospermopsin	Liver, kidney	Slower; organ failure

Species notes

Poultry

Chicks in brooders are vulnerable to carbon monoxide; ammonia from wet litter damages eyes and airways.

Pigs

Slurry pit hydrogen sulphide causes sudden deaths during agitation; ammonia predisposes to respiratory disease.

Cattle and buffalo

Exposed to silo gases and drinking from pond blooms; livestock may die within hours of ingesting toxic algae.

Dogs

Swim in and lick algal scum, with very rapid neurotoxic or hepatotoxic illness; vomiting after water contact is an emergency.

Horses

Poorly ventilated stables with wet bedding build ammonia and contribute to airway disease.

Clinical pearls

Self-test: 10 questions

1. Carbon monoxide causes hypoxia mainly by:

CO occupies haemoglobin and reduces oxygen delivery.

2. Why can a pulse oximeter mislead in CO poisoning?

The device cannot distinguish the two forms and may read normal.

3. The first priority in suspected CO poisoning is:

Oxygen displaces CO from haemoglobin.

4. Chronic ammonia exposure in poultry houses mainly:

It paralyses cilia and injures mucosa.

5. Hydrogen sulphide inhibits:

This blocks cellular respiration.

6. Why is smell an unreliable warning for hydrogen sulphide?

Olfactory fatigue at high concentrations removes the warning.

7. The main hazard during slurry pit agitation is:

Agitation releases trapped gas, causing knock-down deaths.

8. Silo filler's disease is caused by:

NO_2 from fermenting silage damages the deep lung.

9. Microcystins mainly cause:

They inhibit protein phosphatases in hepatocytes.

10. A dog develops tremor and paralysis within an hour of swimming in a green pond. The likely toxin group is:

Anatoxins and saxitoxins act fast on nerves and muscle.

Key points to revise

1. Carbon monoxide binds haemoglobin and causes tissue hypoxia with normal lungs.
2. Ammonia is an irritant that damages eyes and airways and predisposes to infection.
3. Hydrogen sulphide inhibits cytochrome c oxidase and abolishes the sense of smell at high levels.
4. Nitrogen dioxide from fresh silage injures deep lung and can cause delayed bronchiolitis.
5. Silos and pits are confined spaces; never enter without breathing apparatus.
6. Microcystins are liver toxins; anatoxins and saxitoxins are fast neurotoxins.
7. Most cyanobacterial poisoning has no specific antidote, so removal from the water and supportive care are key.
8. Prevention is ventilation, maintenance, water management and supervised access.

Go deeper on this site

- Ch 10. Photosensitisation (in this book)
- Ch 8. Feed-related toxicities (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- CDC: Carbon monoxide poisoning
- CDC NIOSH: Hydrogen sulfide immediately dangerous to life or health documentation
- CDC: Harmful algal blooms

CHAPTER 10 OF 17

Photosensitisation

Photosensitisation is a sunlight-triggered skin disease caused by photodynamic compounds in the skin. This chapter separates primary, hepatogenous and drug-induced forms, links them to common plants and drugs, and explains the management logic of shade, source removal and liver support.

After this chapter you can

- Define photosensitisation and explain the photodynamic mechanism of skin injury
- Distinguish primary, aberrant-pigment, hepatogenous and drug-induced forms
- Explain why liver damage leads to phylloerythrin-based photosensitisation in herbivores
- Name plants and drugs commonly associated with each form
- Outline the management principles and the points that need urgent veterinary help

What photosensitisation is

Photosensitisation is an abnormal reaction of the skin to light. It is not simple sunburn. A **photodynamic agent** must first reach the skin, either from inside the body (through the blood) or by direct contact. When light falls on that agent it absorbs energy and passes it to oxygen, producing reactive oxygen species. These damage cell membranes in the epidermis and the dermal vessels, so the skin becomes inflamed and may die.

Three ingredients are therefore needed: a photodynamic compound, light of the right wavelength, and exposed skin. Take away any one and the disease does not appear. This is the basis of most management advice.

Lesions are seen on **unpigmented or thinly haired skin** that is exposed to sun, such as the white face, muzzle, ears, eyelids, teats, coronets and back. Pigmented skin and well-haired skin are protected because the pigment or coat absorbs or blocks the light. A black cow with a white face and white feet will show lesions only on the white areas.

A common confusion is to treat photosensitisation as an allergy. It is a toxic, dose-related light reaction and does not need prior sensitisation by the immune system, so a first exposure can cause disease. Another is to expect it only in hot weather: bright light on a cool day is enough, and cloud does not block all the relevant light.

Four ways the photodynamic agent arises

Classification depends on where the photodynamic compound comes from.

1. **Primary (type I) photosensitisation.** The animal eats or is exposed to a preformed photodynamic compound. The liver is normal. The compound is absorbed and reaches the skin directly.
2. **Aberrant endogenous pigment synthesis (type II).** An inherited defect in haem synthesis (porphyria) lets porphyrins build up. This is a genetic disease, reported in cattle, pigs and cats, and it is not a poisoning.
3. **Hepatogenous (type III) photosensitisation.** Liver damage or bile obstruction stops the animal clearing **phylloerythrin**, a normal breakdown product of chlorophyll. It builds up in blood and reaches the skin. This is the commonest form in grazing livestock.
4. **Other or unknown causes.** Some classifications keep a group for cases where the mechanism is not understood.

Drug-induced photosensitisation sits mainly with the primary type: the drug or its metabolite is itself the photodynamic agent. The common confusion is to call every photosensitisation 'liver disease'. Only the hepatogenous form is, and a normal liver does not exclude photosensitisation.

Primary and drug-induced forms

In primary photosensitisation the animal's liver can be completely normal, so there is usually **no jaundice** and liver enzymes are not markedly raised. The skin disease appears within hours to a few days of eating the plant and exposure to sun.

Plants usually named include **St John's wort** (*Hypericum perforatum*, hypericin), **buckwheat** (*Fagopyrum*, fagopyrin) and members of the carrot family such as *Ammi* species (bishop's weed), which contain furocoumarins that can act on contact as well as after ingestion. Check regional flora lists for which species are present in your area.

Drugs reported to sensitise skin to light include phenothiazine, tetracyclines, sulfonamides and some other antimicrobials and diuretics. The practical point is that a drug may be safe in a shaded, housed animal and cause lesions in the same animal at pasture in strong sun. Always ask about sun exposure when starting such

a drug and when reviewing an unexplained skin reaction in a treated animal. Reaction is individual and not every treated animal is affected.

Hepatogenous photosensitisation

Ruminants and horses have gut microbes that break down chlorophyll in green forage to **phylloerythrin**. This is absorbed and normally taken up by the liver and excreted in bile. If the liver is damaged or the bile ducts are blocked, phylloerythrin stays in the circulation and reaches the skin. A healthy animal on lush pasture therefore does not react, while a liver-injured animal on the same pasture does.

Plants and toxins that injure the liver or bile ducts include **Lantana camara** (a widespread weed in India, with lantadenes that cause cholestasis), **Tribulus terrestris** (puncture vine) in sheep, certain tropical grasses such as *Brachiaria* and *Panicum* with saponin-containing fungal-linked disease, bog asphodel (*Narthecium*), and blue-green algae (cyanobacteria) in water. The fungal toxin **sporidesmin** from *Pithomyces chartarum* on pasture causes facial eczema in sheep and cattle in some countries. Verify which are relevant locally.

Because the cause is the liver, these animals are often **jaundiced**, may be dull and lose weight, and show raised liver enzymes and bile acids. The prognosis depends on the liver injury as much as on the skin.

Clinical signs and diagnosis

Signs follow the same pattern in every form. Early: restlessness, rubbing, shaking of the head and ears, reddening and swelling of exposed skin. Later: oedema of the face and ears (sometimes called 'big head' in sheep), serous exudate, crusts, cracking, and in severe cases necrosis and sloughing of skin. The eyes may show photophobia, tearing and corneal cloudiness. Animals seek shade and avoid sun.

Secondary problems matter: fly strike and bacterial infection of damaged skin, weight loss because grazing in daylight stops, and in hepatogenous cases the signs of liver failure, including hepatic encephalopathy.

Diagnosis combines the history (plant or drug access, sun exposure), distribution of lesions on unpigmented skin, and tests of liver function when hepatogenous disease is suspected. Differentials include sunburn, contact dermatitis, bacterial or

fungal skin disease, and in cattle certain viral diseases causing teat and muzzle lesions.

Management concepts and when to seek help

Management follows the three ingredients.

- **Remove the light.** Move the animal into a shed or dense shade at once and graze at night or on cloudy days until recovery.
- **Remove the source.** Stop the plant, mouldy or contaminated feed, or the causative drug. Review the pasture and water supply.
- **Treat the skin.** Clean, protect against flies and secondary infection, and give analgesia. Anti-inflammatory treatment is chosen by the clinician.
- **Support the liver** in hepatogenous cases, with good-quality feed and fluids as needed. There is no antidote for most causes.

Call a veterinarian urgently for any animal with swollen face or eyelids, corneal damage, jaundice, neurological signs, or when several animals in a group are affected, because this suggests a pasture or feed problem that will spread. Prevention means weed control (for example removing Lantana from grazing land), avoiding grazing of risky plants during growth stages and sunny periods, and caution with photosensitising drugs in light-skinned animals kept outdoors.

Concept cleared

Concept cleared: Photodynamic reaction

Think of a solar panel connected to a firecracker. The agent in the skin catches the light like the panel and hands the energy to oxygen, which goes off as reactive oxygen species and burns the cells around it. No light, no spark, so shade is a treatment.

Concept cleared: Why the liver matters in type III

The liver is the dustbin for phylloerythrin. If the bin is blocked or broken, the waste piles up in the blood and spills into the skin. A normal grass diet then becomes a source of light-sensitive waste.

Concept cleared: Why only white skin

Pigment and hair act like a sunscreen and a hat. Where they are absent, light reaches the agent in the tissue, so lesions follow the white patches of the animal.

Concept cleared: Primary versus hepatogenous

In primary disease the poison itself is the sensitiser and the liver is healthy. In hepatogenous disease the poison harms the liver and the liver failure creates the sensitiser. Ask: is the liver sick? Jaundice and raised liver enzymes point to type III.

Memory aid: The three ingredients

Think A-L-S: Agent, Light, Skin that is unpigmented. Remove any one and the disease stops. For the four forms remember P-A-H-D: Primary, Aberrant pigment, Hepatogenous, Drug.

Compare and contrast

Forms of photosensitisation

Form	Source of photodynamic agent	Liver	Examples
Primary (type I)	Eaten or applied preformed compound	Normal	St John's wort, buckwheat, Ammi species, some drugs
Aberrant pigment (type II)	Inherited porphyrin defect	Normal function	Congenital porphyria in cattle, pigs, cats
Hepatogenous (type III)	Phylloerythrin retained	Damaged or obstructed	Lantana, Tribulus, cyanobacteria, sporidesmin
Drug-induced	Drug or metabolite in skin	Usually normal	Phenothiazine, tetracyclines, sulfonamides

Sunburn versus photosensitisation

Feature	Sunburn	Photosensitisation
Cause	UV light alone	Light plus a photodynamic agent
Group effect	Any unprotected skin	Only animals exposed to the agent
Systemic signs	Rare	Possible, such as jaundice in type III
Control	Shade and sunscreen	Shade plus removing the agent

Species notes

Cattle

Hepatogenous disease from Lantana is a classic problem in cattle where the weed is common. White-skinned areas of Holstein-type and crossbred animals are most affected; congenital porphyria is also described.

Sheep

Often affected by hepatogenous forms, with facial swelling ('big head') and ear oedema, as with Tribulus and facial eczema; wool covers much of the body, so face and ears show the lesions.

Horse

Can develop photosensitisation on white-marked limbs and muzzle; liver disease from toxic plants or feed contamination should be considered.

Pig

White-skinned breeds are prone to sunburn-like lesions; buckwheat and porphyria are described causes.

Cat and dog

Rare compared with livestock. Light-coated cats on drugs or with porphyria can develop ear and nose lesions; keep indoors in strong sun.

Clinical pearls

Self-test: 10 questions

1. Which three factors are required for photosensitisation to occur?

A photodynamic agent, light and exposed lightly pigmented skin are all required.

2. Phylloerythrin is formed from which dietary substance?

Gut microbes break chlorophyll down to phylloerythrin.

3. A cow with jaundice, raised liver enzymes and skin lesions on white areas most likely has which form?

Liver damage with jaundice points to hepatogenous photosensitisation.

4. Which plant is a common cause of hepatogenous photosensitisation and is widespread in India?

Lantana contains lantadenes that cause cholestasis. The others cause primary forms.

5. St John's wort causes photosensitisation through which compound?

Hypericin is the photodynamic pigment in Hypericum perforatum.

6. Congenital porphyria is best described as

It is a genetic defect that allows porphyrins to accumulate.

7. Which is the most important first step in managing a photosensitised animal?

Removing light interrupts the photodynamic reaction.

8. Why do lesions appear on white-skinned areas?

Pigment and haircoat shield against the light that activates the agent.

9. Which drug group has been reported to cause drug-induced photosensitisation?

Tetracyclines, sulfonamides and phenothiazine are reported photosensitisers.

10. Several animals on the same pasture develop facial swelling and jaundice. What is the best action?

Group outbreaks suggest a feed or pasture source that needs urgent investigation.

Key points to revise

1. Photosensitisation needs a photodynamic agent, light and exposed unpigmented skin.
2. Light activates the agent, which produces reactive oxygen and injures skin.
3. Primary form: preformed agent eaten or contacted, liver normal.
4. Hepatogenous form: liver damage prevents phylloerythrin excretion.
5. Congenital porphyria is an inherited form, not a poisoning.
6. Drugs such as phenothiazine, tetracyclines and sulfonamides may sensitise skin.
7. Management: shade, remove the source, skin care, liver support.
8. Urgent help is needed for jaundice, eye damage, neurological signs or multiple animals affected.

Go deeper on this site

- Ch 11. Household and pet toxins (in this book)
- Ch 9. Toxic gases and algal toxins (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Photosensitization in Animals
- Merck Veterinary Manual: Overview of Veterinary Toxicology
- ASPCA: Toxic and Non-Toxic Plants

CHAPTER 11 OF 17

Household and pet toxins

Many of the commonest poisonings in dogs and cats come from ordinary household items: foods, plants, medicines, antifreeze and cleaners. This chapter explains the mechanism, signs, species differences and decontamination logic for chocolate, grapes, xylitol, onion and garlic, lilies, antifreeze, human painkillers and cleaning products.

After this chapter you can

- Explain the mechanism of toxicity of chocolate, xylitol, onion and garlic, lilies and ethylene glycol
- Recognise the typical clinical signs and their time course for each toxin
- Identify the species that is most at risk for each toxin
- Explain when decontamination is appropriate and when it is harmful
- Describe prevention advice and when to seek urgent veterinary help

Why household toxins matter

Pets explore with their mouths, and homes contain foods, plants, medicines and chemicals meant for people. Pet owners also tend to share human food or leave medicines within reach. In India, additional exposure comes from foods such as raisins in sweets, onion and garlic in cooked household food, and from floor and toilet cleaners and 'phenyl' used on floors.

The approach to any suspected exposure is the same. Identify the product and the animal, estimate how much and how long ago, and ask what signs are present. Then decide whether to decontaminate, which antidote or supportive care is needed, and how long to monitor. The most important lesson is that **timing and the toxin both decide the plan**. A step that helps within an hour (such as induced

vomiting) can be harmful if the animal is already sedated, convulsing or has swallowed a corrosive.

This chapter gives concepts only. Treatment decisions and quantities must come from a veterinarian or a poison information service, which can calculate risk from the product and body weight.

Chocolate, grapes and xylitol

Chocolate contains the methylxanthines **theobromine** and caffeine. They block adenosine receptors, inhibit phosphodiesterase and increase catecholamine effects, so the heart and nervous system are overstimulated. Dogs eliminate theobromine slowly, and it is recirculated through bile, so signs last long. Dark chocolate, cooking chocolate and cocoa powder are more dangerous than milk chocolate because they contain more methylxanthine per gram. Signs: vomiting, restlessness, thirst, rapid heart rate, arrhythmia, tremors, and in severe cases seizures and high temperature. Management uses decontamination when the ingestion is recent, repeated activated charcoal because of the recirculation, and supportive care with heart monitoring.

Grapes and raisins can cause acute kidney injury in dogs. The toxic agent has not been clearly identified, and individual dogs vary unpredictably, so any known ingestion should be taken seriously. Early signs are vomiting and lethargy within hours, followed by reduced urine output and kidney failure over one to three days. Early decontamination and fluid therapy with kidney monitoring are the basis of treatment.

Xylitol is a sweetener in sugar-free gum, sweets and some other foods. In dogs it triggers a large release of insulin, causing sudden **hypoglycaemia** with weakness, ataxia, collapse and seizures. Larger exposures can also cause liver injury. Blood glucose, liver values and electrolytes must be monitored. Do not induce vomiting in a dog that is already weak or dull. Check how xylitol is handled in a poison service protocol, as activated charcoal binds it poorly.

Onion, garlic and lilies

Onion and garlic (the genus *Allium*) contain sulphur compounds that cause **oxidative damage to red cells**. Haemoglobin is denatured into Heinz bodies, the

cells are removed early, and haemolytic anaemia and methaemoglobin can result. The effect is dose-related and cumulative, and signs may appear only after a delay of one to several days: pale or yellow gums, dark urine, weakness, rapid breathing. Cooked, dried and powdered forms are still toxic. **Cats** are very sensitive, and some dog breeds, notably Japanese breeds, are reported to be more sensitive. There is no specific antidote. Treat with decontamination if recent, then supportive care, oxygen and transfusion if needed.

True lilies and daylilies (*Lilium* and *Hemerocallis*) are a major cause of acute kidney failure in **cats**. Leaves, flowers, pollen and even vase water are dangerous, and a small amount can be enough. The toxin is not fully identified, and it injures the renal tubules. Dogs and horses are generally considered not at risk from lilies. Early signs: vomiting, drooling, lethargy and loss of appetite within hours. If untreated, kidney failure follows over a day or two, with changes in urination. Immediate veterinary treatment, with decontamination and intravenous fluids started early, greatly improves the outcome.

Antifreeze and ethylene glycol

Ethylene glycol is found in antifreeze and coolants. It has a sweet taste and attracts animals. The parent compound is only mildly toxic. The harm comes from **metabolism**. Alcohol dehydrogenase converts it to glycolaldehyde, then to glycolic, glyoxylic and finally oxalic acid. Glycolic acid causes a severe metabolic acidosis, and oxalate binds calcium to form **calcium oxalate crystals** that damage the renal tubules.

Signs appear in stages. First, within hours, the animal appears drunk, with ataxia, vomiting and increased thirst. Later, there are cardiopulmonary signs and acidosis. Finally, over one to three days, acute kidney failure with little or no urine develops. Cats progress faster and have a lower tolerance than dogs.

The antidote logic is important for exams. Antidotes such as **fomepizole** (and ethanol where that is not available) compete for alcohol dehydrogenase so that ethylene glycol is not converted to its toxic products. They work only if given early, before most of the compound has been metabolised, and the cat dose differs from the dog dose, so use a protocol. Once kidney failure is established, antidotes are of little use and the outlook is poor. Therefore any suspected ingestion is an **emergency**, and owners should come in at once rather than wait for signs.

Prevention: store antifreeze securely, clean up spills, and consider less toxic products.

Human painkillers and cleaning products

Human **painkillers** are a common source of poisoning. Ibuprofen, naproxen and aspirin inhibit cyclo-oxygenase and cause gastric ulceration and kidney injury. **Paracetamol** is particularly dangerous to cats, which are poor at glucuronidation, and leads to methaemoglobin, haemolysis and facial swelling. Medicines made for people are not scaled for pets, so never advise owners to give human tablets. Chapter 12 covers these in more detail.

Cleaning products divide into groups with different problems. Strong acids and alkalis (some toilet cleaners, drain cleaners) are corrosive and burn the mouth, oesophagus and stomach. Bleach irritates. Cationic detergents and disinfectants can burn tissues. Phenol-based products such as 'phenyl' can be absorbed through skin and by licking, and cats are again vulnerable because of poor glucuronidation. Signs: drooling, pawing at the mouth, vomiting, pain and respiratory distress from fumes.

The golden rule for corrosives is **do not induce vomiting**, because the substance burns twice on the way back up. Rinse the mouth and skin, remove from the area, keep the container and call for veterinary help.

General approach, prevention and urgent help

Decontamination reasoning in brief:

- **Vomiting** may help only when the ingestion is recent, the animal is alert, and the substance is not corrosive or a hydrocarbon. It should be done by or on the advice of a veterinarian.
- **Activated charcoal** binds many toxins in the gut but does not bind everything, and it can be dangerous if the animal is sedated or may inhale it.
- **Antidotes** exist for only a few of these toxins, such as fomepizole for ethylene glycol. For the others, supportive care (fluids, glucose, oxygen, heart monitoring) keeps the animal alive while the body recovers.
- **Monitoring time** depends on the toxin: some, like lily, grapes, ethylene glycol and onion, cause problems days after the exposure, so a well-looking animal is

not safe.

Prevention: keep foods, medicines and chemicals in closed cupboards, keep plants out of reach of cats, use child-proof containers, and educate owners not to share human food. Teach owners to keep the packaging, estimate the amount and time, and phone a veterinarian or a poison information service at once. Do not wait for signs, because signs often start after the best window for treatment has passed.

Concept cleared

Concept cleared: Lethal synthesis

Ethylene glycol is like a harmless-looking parcel that becomes a bomb when opened by the body's own enzyme. Fomepizole blocks the enzyme and the parcel passes out unopened.

Concept cleared: Delayed toxicity

Onion, grapes, lilies and antifreeze look harmless for hours or days. Damage builds quietly in red cells or kidneys. This is why the history, not the current appearance, decides the response.

Concept cleared: Why cats are different

Cats have limited glucuronidation, so drugs and phenols that rely on it, such as paracetamol and phenolic cleaners, linger and cause harm. Add the lily kidney effect and ethylene glycol sensitivity, and cats need extra caution.

Concept cleared: When not to make a pet vomit

If the swallowed substance is corrosive, if the pet is weak, sedated or convulsing, or if inhalation into the lungs is likely, vomiting adds injury. Ask a veterinarian first.

Memory aid: Home hazard list

Think C-G-X-O-L-A-P-C: Chocolate, Grapes, Xylitol, Onion, Lilies, Antifreeze, Painkillers, Cleaners. Keep them away from the pet and the pet away from them.

Compare and contrast

Key household toxins

Toxin	Main target	Key mechanism	Main at-risk species
Chocolate	Heart and brain	Methylxanthines, adenosine block	Dogs
Grapes and raisins	Kidney	Unknown agent, tubular injury	Dogs
Xylitol	Pancreas and liver	Insulin release, hypoglycaemia	Dogs
Onion and garlic	Red cells	Oxidative damage, Heinz bodies	Cats, dogs
Lilies	Kidney	Tubular injury	Cats
Ethylene glycol	Kidney	Metabolites, acidosis, oxalate crystals	Dogs and cats

Decontamination decisions

Situation	Vomiting	Notes
Recent, alert, non-corrosive	May be considered by a veterinarian	Does not work well after a long interval
Corrosive acid, alkali or cleaner	Avoid	Rinse and get veterinary care
Weak, sedated or seizing	Avoid	Risk of inhalation
Xylitol or ethylene glycol	Specific advice needed	Antidote or glucose care may be the priority

Species notes

Dog

Most chocolate, grape and xylitol cases are in dogs, which eat indiscriminately. Dogs are also affected by onion, garlic and antifreeze.

Cat

Very sensitive to lilies, onion and garlic, paracetamol, phenolic cleaners and antifreeze. Cats groom, so skin contamination becomes oral.

Horse

Lilies are not considered a concern in horses, but chocolate by-products, onions and cleaning chemicals in the stable can still cause harm.

Cattle and small ruminants

Antifreeze and household chemicals dumped near yards can be consumed. Onion in large amounts, such as waste, can cause haemolysis in ruminants.

Birds

Avocado, chocolate and fumes from some non-stick or cleaning products are of concern; seek advice for each product.

Clinical pearls

Self-test: 10 questions

1. The main toxic compounds in chocolate are

Theobromine and caffeine are methylxanthines.

2. Xylitol causes which early problem in dogs?

It stimulates insulin release in dogs.

3. Onion toxicity mainly damages which cells?

Oxidative damage leads to Heinz bodies and haemolysis.

4. Which species is most at risk of kidney failure from lilies?

Lily kidney injury is a feline problem.

5. Why is ethylene glycol dangerous?

Metabolites cause acidosis and calcium oxalate crystals in kidney tubules.

6. Fomepizole acts by

It blocks formation of toxic metabolites.

7. Which step is inappropriate after swallowing a strong toilet cleaner?

Corrosives re-injure the oesophagus and mouth on the way up.

8. Why is repeated activated charcoal useful in chocolate poisoning?

Recirculation prolongs exposure, and charcoal traps it in the gut.

9. A dog ate raisins and looks normal. What is correct?

Kidney injury follows after a delay, and early treatment works best.

10. Why are cats particularly sensitive to phenolic cleaners and paracetamol?

Cats clear these compounds slowly.

Key points to revise

1. Household poisoning is decided by the toxin, amount, time and species.
2. Chocolate: methylxanthines, long-lasting signs in dogs.
3. Grapes and raisins: kidney injury of unpredictable risk in dogs.
4. Xylitol: insulin release and hypoglycaemia in dogs.
5. Onion and garlic: oxidative haemolysis, delayed signs.
6. Lilies: kidney failure in cats from small exposures.
7. Ethylene glycol: toxic metabolites; fomepizole works only early.
8. Corrosives: no vomiting; rinse and seek help.

Go deeper on this site

- Ch 12. Recreational drugs and human medicines in pets (in this book)
- Ch 10. Photosensitisation (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Chocolate Toxicosis in Animals
- Merck Veterinary Manual: Ethylene Glycol Toxicosis in Animals
- ASPCA: People Foods to Avoid Feeding Your Pets

CHAPTER 12 OF 17

Recreational drugs and human medicines in pets

Pets are often poisoned by human drugs left within reach or given by well-meaning owners. This chapter covers cannabis, opioids, antidepressants, ADHD stimulants, sleep aids, paracetamol and NSAIDs, with the mechanism of each, the signs to recognise, species differences and a sensible response.

After this chapter you can

- Explain the mechanism of toxicity of cannabis, opioids, antidepressants, stimulants, sleep aids, paracetamol and NSAIDs

- Recognise the characteristic clinical picture of each drug class
- Explain why cats and dogs respond differently to paracetamol and NSAIDs
- Describe the antidote and decontamination reasoning for each class
- Give owners a clear first-response plan and know when urgent help is needed

Why human drugs poison pets

A tablet that is safe for a 60 kg person may be a serious dose for a 5 kg dog or cat. Pets also metabolise drugs differently from humans, so even a normal human dose can behave like an overdose. Common routes of exposure are tablets left on a table, a handbag or a bedside, dogs chewing blister packs and bottles, owners giving human painkillers to relieve pain, and cannabis or edible products dropped or left open.

The first step is always the **history**: which drug, which formulation (immediate or extended-release, or combination products), how many tablets, how long ago, and the weight and species of the animal. Keep the package. Many human medicines contain more than one drug, and some preparations also contain xylitol or chocolate.

Do not try home remedies or induce vomiting without veterinary advice. Some of these drugs cause sedation or seizures within minutes, and vomiting then risks inhalation. This chapter gives concepts only. For quantities, use a veterinarian or poison information service, and check any local rules on controlled substances.

Cannabis and opioids

Cannabis (marijuana, hashish and bhang preparations, and edible products) contains **THC**, which acts on cannabinoid CB1 receptors in the brain. Dogs are the commonly affected species and are very sensitive. Signs appear within hours: wobbling and ataxia, depression or sedation, dribbling urine, slow heart rate, low body temperature, dilated pupils, vomiting, and sometimes agitation or tremors. Severe cases may progress to seizures or coma. Most animals recover with supportive care in a quiet, warm place, but they need monitoring because of the risk of aspiration and low temperature. Edibles are a hazard because of added

chocolate, xylitol or other ingredients. Legal status of cannabis in India is specific and conditional; check the current notification before advising owners.

Owners may be reluctant to admit exposure. Reassure them that treatment depends on honesty, and that clinicians treat the pet, not the owner.

Opioids (morphine, codeine, tramadol, fentanyl patches, buprenorphine, heroin) act on mu receptors. In dogs the typical picture is sedation, slow breathing, slow heart rate, low temperature and small pupils. Cats may be excited with dilated pupils. Tramadol also affects serotonin and noradrenaline, so seizures and serotonin-type signs can occur. The antidote is **naloxone**, which reverses respiratory depression, but its action is shorter than that of many opioids, so the animal may become sedated again and needs prolonged monitoring. Skin patches should be recovered and not left on the animal.

Antidepressants, ADHD drugs and sleep aids

Antidepressants. Selective serotonin reuptake inhibitors and related drugs increase serotonin. Large exposures can cause **serotonin syndrome**: agitation, tremors, increased heart rate, high temperature, dilated pupils, vomiting and diarrhoea, and sometimes seizures. Some animals instead become sedated. Tricyclic antidepressants affect the heart rhythm and the nervous system. Bupropion is known for causing seizures. Treatment is supportive: control of tremors and seizures, cooling, fluids and monitoring of heart rhythm. Drugs that block serotonin, such as cyproheptadine, may be used by a clinician. Avoid vomiting if signs are developing.

ADHD stimulants (amphetamines, methylphenidate and related drugs) are potent central nervous system and heart stimulants. Pets show hyperactivity, panting, dilated pupils, fast heart rate, high blood pressure, tremors, high temperature and seizures. Extended-release capsules prolong the period of risk. Intensive care with sedation, cooling, fluids and heart monitoring is the mainstay, and the signs can persist for many hours.

Sleep aids. Benzodiazepines and 'Z-drugs' such as zolpidem act on the GABA-A receptor. They cause sedation and ataxia in many animals, but in some dogs and cats they cause paradoxical agitation, so you cannot predict the response. Respiratory depression is more likely when taken with alcohol or opioids. Flumazenil

can reverse benzodiazepines in some cases; a clinician decides. Melatonin products are generally considered low risk, but combinations and other ingredients may not be.

Paracetamol

Paracetamol (acetaminophen) is metabolised in the liver by conjugation. A small part is converted into a reactive metabolite which is normally neutralised by glutathione. When too much is taken, glutathione runs out and the metabolite damages cells.

Cats are the most sensitive species because they have poor glucuronidation, so more drug is pushed down the toxic pathway, and their red cells are prone to oxidation. Even a single tablet can be dangerous. The result is **methaemoglobin** (chocolate-brown blood, blue-grey gums, difficulty breathing), Heinz body haemolysis, and swelling of the face and paws. **Dogs** are more tolerant, but higher exposures cause liver injury, and sometimes methaemoglobinaemia and dry eye (keratoconjunctivitis sicca).

The antidote is **N-acetylcysteine**, which replenishes glutathione. It works best when started early. Decontamination, oxygen and supportive care are added. A key exam point: never give paracetamol to a cat, and do not advise it for dogs without veterinary direction. Many combination cold remedies contain paracetamol.

NSAIDs and other human painkillers

Non-steroidal anti-inflammatory drugs such as ibuprofen, naproxen, diclofenac and aspirin inhibit cyclo-oxygenase. Prostaglandins protect the stomach lining and maintain kidney blood flow, so inhibition causes **gastric irritation and ulceration, bleeding and acute kidney injury**. High exposures can also cause neurological signs. Signs include vomiting (sometimes with blood), black stools, abdominal pain, lethargy, poor appetite and later increased thirst or reduced urine output. Signs may be delayed for a day or more.

Species differences are important. Cats clear many NSAIDs slowly, so smaller amounts cause problems. Dogs differ between drugs and individuals. Several drugs are licensed for animals, but human products are not dosed or formulated for them, and an animal NSAID should only be used as prescribed. Never give another NSAID

or a corticosteroid on top of a suspected overdose, because the risk of ulcer and kidney damage adds up.

Management includes decontamination if recent, repeated activated charcoal in some cases because of bile recirculation, stomach protection, fluid therapy to support the kidneys, and monitoring of kidney values for several days. A well-looking animal after an NSAID overdose is not out of danger.

Recognition and response

Think of drug poisoning when a pet suddenly shows odd behaviour: wobbling, unusual sleepiness, agitation, tremors, vomiting, fast or slow heart rate, abnormal pupils or collapse, especially in a household with drugs or a young animal exploring. Match the picture to the class:

- Wobbly, urinating and dilated pupils: think cannabis.
- Slow breathing and small pupils in a dog: think opioid.
- Agitated, hot, tremors, fast heart: think stimulant or antidepressant.
- Blue-grey gums and swollen face in a cat: think paracetamol.
- Vomiting blood and later kidney signs: think NSAID.

Response: remove access to any remaining drug, keep the packaging, note the time, and contact a veterinarian or a poison information centre immediately. Do not give milk, salt water or other home emetics. Human urine drug screening kits are unreliable in animals and should not be used alone to rule drugs in or out. Treat the animal according to its signs even if the history is uncertain.

Seek urgent help for any seizure, collapse, breathing difficulty, severe agitation, or ingestion of extended-release, combination or fentanyl products. Prevention: store medicines high and closed, give pets only medicines prescribed for them, and tell owners that a safe medicine for them may not be safe for their pet.

Concept cleared

Concept cleared: Same drug, different animal

Drug handling is like a factory line. Humans have many workers for paracetamol. Cats have too few glucuronidation workers, so the extra drug goes to the toxic side line. Species, not just weight, sets safety.

Concept cleared: Antidotes versus supportive care

Only some drugs have a true antidote: naloxone for opioids and N-acetylcysteine for paracetamol. For cannabis, stimulants and antidepressants, treatment means keeping the body safe (airway, temperature, seizures, heart) until the drug wears off.

Concept cleared: Short antidote, long drug

Naloxone is like a short-acting shield against a long-acting arrow. When it wears off, the opioid is still there and the animal may become sedated again, so watch after reversal.

Concept cleared: Extended-release products

A slow-release capsule is a time bomb with a long fuse. Signs may start late and last much longer, so monitoring continues beyond the first hours.

Memory aid: Cannabis signs

Cannabis in dogs, think S-A-D-P: Sedation, Ataxia, Dribbling urine, Pupils dilated, with a slow heart rate and low temperature. For cats remember: no paracetamol, no human NSAIDs.

Compare and contrast

Drug class recognition

Drug class	Mechanism	Typical signs	Specific antidote
Cannabis (THC)	CB1 receptor activation	Ataxia, dribbling urine, slow heart, sedation	None; supportive care
Opioids	Mu receptor activation	Sedation, slow breathing, small pupils (dog)	Naloxone
SSRIs and antidepressants	Raised serotonin or heart effects	Agitation, tremor, high temperature, seizures	None; serotonin blocker may be used
ADHD stimulants	Catecholamine release	Hyperactivity, fast heart, high temperature	None; supportive care
Paracetamol	Reactive metabolite, methaemoglobin	Blue-grey gums, face swelling (cat), liver injury (dog)	N-acetylcysteine
NSAIDs	COX inhibition	Vomiting, ulcers, kidney injury	None; gastroprotection and fluids

Cat versus dog

Drug	Cat	Dog
Paracetamol	Highly sensitive: methaemoglobin, haemolysis	Less sensitive: liver injury at higher exposure
NSAIDs	Slow clearance, small exposures can harm	Ulcers and kidney injury, varies by drug
Opioids	May be excited	Usually sedated
Cannabis	Affected but fewer reports	Very sensitive, common

Species notes

Dog

Most exposures are in dogs, which chew packaging. Cannabis, antidepressants, stimulants and

Cat

Highly sensitive to paracetamol and many NSAIDs. Cats may be excited rather than sedated by opioids. Small

NSAIDs are frequent. Extended-release capsules and blister packs can make quantities hard to judge.

exposures are enough to cause harm.

Horse

Medicines given by owners or stored in the barn are a risk. Equine NSAIDs have narrow margins in foals and dehydrated animals, and human drugs should not be used.

Cattle and small ruminants

Rarely affected by recreational drugs, but cannabis plants and discarded medicines can be eaten. NSAIDs and other drugs must be used only in the labelled way.

Birds and small mammals

Small body size makes any exposure serious. Ferrets, rabbits and birds need immediate advice after any ingestion.

Clinical pearls

Self-test: 10 questions

1. THC produces its effects mainly by acting on which receptor?

THC is a CB1 agonist in the brain.

2. Which antidote reverses opioid respiratory depression?

Naloxone is an opioid antagonist.

3. A cat that swallowed paracetamol has blue-grey gums and a swollen face. What has formed?

Methaemoglobin cannot carry oxygen and causes cyanosis.

4. Why are cats sensitive to paracetamol?

Limited conjugation sends more drug through the toxic pathway.

5. Serotonin syndrome includes which combination?

Excess serotonin causes neuromuscular and autonomic hyperactivity.

6. NSAID toxicity mainly affects which organs?

COX inhibition reduces protective prostaglandins in the gut and kidney.

7. Which is a reason to avoid inducing vomiting at home?

Rapid CNS signs make aspiration likely.

8. Why must dogs be watched after naloxone?

Re-sedation can occur.

9. Extended-release stimulant capsules are especially dangerous because

Slow release extends the exposure window.

10. What is the best first response to suspected drug ingestion in a pet?

Early advice allows timely decontamination and the right antidote.

Key points to revise

1. Human drugs poison pets through size and species metabolic differences.
2. THC acts on CB1 receptors; signs are ataxia, sedation and dribbling urine in dogs.
3. Opioids depress breathing; naloxone reverses but is short acting.
4. Antidepressants and stimulants cause tremors, high temperature and seizures.
5. Sleep aids can sedate or cause paradoxical agitation.
6. Paracetamol causes methaemoglobinaemia in cats; N-acetylcysteine is the antidote.
7. NSAIDs cause ulcers and kidney injury, with delayed signs.
8. Do not induce vomiting at home without advice; call a veterinarian or poison centre.

Go deeper on this site

- Ch 13. Venoms, bites and stings (in this book)
- Ch 11. Household and pet toxins (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Overview of Veterinary Toxicology
- ASPCA: People Foods to Avoid Feeding Your Pets
- ASPCA Animal Poison Control

CHAPTER 13 OF 17

Venoms, bites and stings

How venoms from snakes, scorpions, toads, bees and spiders injure animals, which signs point to which toxin family, and the reasoning behind first aid and antivenom use in Indian practice. The focus is on mechanism and decision-making, not on doses.

After this chapter you can

- Distinguish venom, poison and toxin, and name the main Indian venomous snakes

- Contrast neurotoxic, haemotoxic and local tissue-damaging snake venoms and the signs each produces
- Explain the principle of polyvalent antivenom and why it is time-critical and not always sufficient
- Describe the toxic mechanisms and clinical picture of scorpion, toad, bee and spider exposures
- List correct and incorrect first-aid steps and say when urgent veterinary help is needed

Venom, poison and toxin

A **toxin** is any harmful substance made by a living organism. A **venom** is a toxin mixture delivered actively through a bite or sting, by a fang, stinger or spine. A **poison** harms when swallowed, inhaled or absorbed. A toad is therefore poisonous (its skin secretion harms an animal that mouths it), while a cobra is venomous.

Venoms are not single chemicals. They are cocktails of enzymes, peptides and small molecules, and each component targets a different system. This is why one bite can cause nerve paralysis, bleeding and tissue death together, and why the clinical picture depends on the species of the animal that bit.

Indian snakes and their venom types

In India the medically important snakes are traditionally called the **big four**: the spectacled cobra, the common krait, Russell's viper and the saw-scaled viper. Other snakes can be dangerous locally, but the standard antivenom is built around these four.

- **Cobra** (elapid): venom contains post-synaptic neurotoxins that block the acetylcholine receptor at the neuromuscular junction, plus cytotoxins that cause painful local swelling and necrosis.
- **Krait** (elapid): venom acts mainly at the nerve terminal (pre-synaptic) and can cause progressive paralysis with little local swelling, so the bite may be missed. Bites often occur at night.
- **Russell's viper and saw-scaled viper**: venom is haemotoxic. It activates or consumes clotting factors, producing a consumption coagulopathy with

bleeding, along with swelling at the site. Russell's viper envenoming can also injure the kidney.

A common confusion is to think that paralysis always means a neurotoxic snake and bleeding always means a viper. Overlap exists, so treat the whole animal, not only one sign.

Snake envenomation in animals: signs and reasoning

Dogs, cats, cattle, buffalo, horses and small ruminants are bitten, usually on the face, muzzle or limbs while grazing or investigating. Many bites are unwitnessed, so the history may only be sudden swelling, weakness or collapse.

Signs follow the venom type. Neurotoxic bites cause drooling, ptosis, difficulty swallowing, weakness and respiratory failure. Haemotoxic bites cause swelling, bruising, bleeding from the bite and mucosae, and prolonged clotting. A bedside whole-blood clotting test, in which blood left in a clean glass tube fails to clot within a set time, is used in human medicine to show venom-induced coagulopathy and is also a practical clue in animals. Check current clinical guidance for how to use it.

Bite size, species and body size matter. A small animal receives a larger venom load for its weight and may deteriorate faster. A dry bite, in which no venom is injected, also happens, so absence of signs early on does not prove safety. Watch the animal, do not assume.

First aid and antivenom concepts in India

The aims of first aid are to slow venom spread, avoid harm and reach a veterinarian quickly. Sensible steps are to keep the animal calm and as still as possible, limit the movement of the bitten part, and transport without delay.

Harmful practices include cutting or incising the bite, sucking the wound, tight tourniquets, ice, applying herbal or chemical pastes and delaying treatment to consult a healer. They add infection, tissue loss or ischaemia and waste time. Specific bandaging methods such as pressure immobilisation are recommended for some neurotoxic bites in some settings and are debated for others, so follow current guidance and do not improvise.

Antivenom is the only specific treatment. Indian polyvalent antivenom is made by immunising horses and purifying antibody fragments or whole immunoglobulin that

bind venom from the big four. It neutralises venom that is still circulating. It cannot reverse damage already done and is less reliable against venoms of other snakes. Because it is a foreign protein, anaphylactoid reactions can occur, so adrenaline and supportive drugs should be ready. Antivenom supply and its use in animals vary and should be confirmed locally, since shortages and quality problems are widely reported by WHO.

In neurotoxic cases, anticholinesterase drugs are sometimes tried as a supportive measure and ventilatory support may be life-saving. Wound care, fluids, analgesia and monitoring of clotting and renal function complete the plan.

Scorpion, toad, bee and spider exposures

Scorpions. The Indian red scorpion is the best-known dangerous species. Its venom acts on sodium and potassium channels and triggers a massive release of catecholamines, the so-called autonomic storm. Signs include intense pain, salivation, vomiting, sweating, restlessness, rapid or irregular heartbeat, high or low blood pressure and, in severe cases, pulmonary oedema. Treatment is mainly supportive and symptom-directed; specific antivenom exists but availability varies.

Toads. Dogs that mouth or lick toads absorb bufadienolides from skin glands. These compounds inhibit sodium-potassium ATPase in a digoxin-like way. Signs begin quickly: profuse salivation, pawing at the mouth, vomiting, tremor, arrhythmia and seizures. Immediate rinsing of the mouth, with the head held down so that water runs out and is not swallowed, is the key first step, followed by urgent veterinary care.

Bees and wasps. A single sting causes local pain and swelling. Allergic animals may develop anaphylaxis with collapse, vomiting, facial oedema and respiratory distress. Mass stinging, from many stings, behaves like a true venom overdose, with haemolysis, muscle damage and kidney injury. A honeybee sting stays in the skin and should be flicked or scraped off quickly.

Spiders. Clinically significant spider bites are uncommon in Indian veterinary practice. Classic examples elsewhere are widow spiders, whose latrotoxin causes painful muscle rigidity, and recluse spiders, whose venom causes tissue necrosis. Any suspected case should be managed on its clinical signs.

Prevention and when to seek urgent help

Prevention reduces most cases. Keep grass and rubbish short around animal sheds, store feed away from rodents that attract snakes, light walkways at night, and keep dogs on a lead in snake habitats during the monsoon. Control nesting sites of bees near animal housing.

Treat any of the following as an emergency: a bite or sting by a known or suspected snake, rapid swelling of the face, drooling with difficulty breathing, collapse, bleeding that does not stop, or repeated stings. Do not wait to see how the animal looks in an hour. Phone the veterinarian, begin gentle first aid and transport. If a dead snake is brought, handle it carefully, since reflex bites can occur, and use it only to help identification and never to delay care.

Concept cleared

Venom is a cocktail
Each venom contains many components. The mix, not a single chemical, explains why paralysis, bleeding and necrosis can occur together.

Neurotoxic versus haemotoxic
Cobra and krait venoms mainly act at the neuromuscular junction. Russell's and saw-scaled viper venoms mainly disturb clotting and tissue. Overlap is common.

Antivenom neutralises, it does not repair
Antivenom binds free venom. Early use helps most because it cannot undo damage already done.

Poisonous versus venomous
Toads are poisonous because their secretion harms when mouthed. Snakes, scorpions and bees are venomous because they inject toxin.

Memory aid: Bite response: CALM

C: Calm and keep still. A: Avoid cutting, sucking and tourniquets. L: Limit movement of the limb. M: Move to the veterinarian at once.

Compare and contrast

Major Indian snake venoms

Snake	Main venom action	Typical picture	Special concern
Spectacled cobra	Post-synaptic neurotoxin plus cytotoxin	Local swelling, necrosis, paralysis	Respiratory failure
Common krait	Pre-synaptic neurotoxin	Little local change, progressive weakness	Bite may go unnoticed
Russell's viper	Procoagulant and haemotoxic	Swelling, bleeding, clotting failure	Kidney injury
Saw-scaled viper	Procoagulant and haemotoxic	Swelling, bleeding, clotting failure	Small snake, easily missed

Other venomous or poisonous exposures

Source	Toxic mechanism	Key signs	Immediate step
Indian red scorpion	Channel toxins, catecholamine surge	Pain, salivation, arrhythmia, oedema	Urgent supportive care
Toad	Digoxin-like bufadienolides	Salivation, tremor, arrhythmia	Rinse mouth outward
Honeybee	Melittin, allergens	Swelling, anaphylaxis, mass-sting haemolysis	Remove sting, seek help
Widow spider	Latrotoxin	Pain, rigidity	Supportive care

Species notes

Dogs

Most commonly bitten on the head and limbs. Also the usual victims of

Cats

Small body size means a larger venom load per kilogram and faster

toad poisoning because they mouth toads.

deterioration, so early signs deserve urgent attention.

Cattle and buffalo
Usually bitten on the muzzle or lower limbs while grazing. Swelling of the muzzle can obstruct breathing.

Horses
Muzzle and limb bites are common. Antivenom is a foreign horse-derived protein, so sensitisation and reactions should be considered.

Small ruminants
Light body weight makes venom effects rapid, and bites in grazing flocks may be unwitnessed.

Clinical pearls

Self-test: 10 questions

1. Which statement correctly defines a venom?

Venom is delivered actively through a bite, sting or spine. Substances that harm when swallowed or absorbed are poisons.

2. Which snake is part of the Indian big four?

The big four are the spectacled cobra, common krait, Russell's viper and saw-scaled viper.

3. Cobra post-synaptic neurotoxins mainly act by

Post-synaptic neurotoxins occupy the acetylcholine receptor on the muscle end plate, producing flaccid paralysis.

4. Which feature is typical of krait envenomation?

Krait venom acts at the nerve terminal and often leaves little visible local change, so the bite can be missed.

5. Russell's viper envenomation commonly causes

Its venom activates and consumes clotting factors, so bleeding and prolonged clotting are typical, and kidney injury can follow.

6. Which first-aid step is inappropriate after a snake bite?

Cutting and sucking cause tissue damage and infection and do not remove meaningful venom.

7. Polyvalent antivenom in India is best described as

It is a purified antibody preparation raised in horses that binds free venom, but it cannot repair damage that has already occurred.

8. Toad poisoning in dogs resembles which drug effect?

Bufadienolides inhibit sodium-potassium ATPase, giving cardiac glycoside-like arrhythmias along with salivation and tremor.

9. The immediate first step after a dog mouths a toad is to

Rinsing removes secretion from the mucosa. Forced salt is dangerous, and delay allows absorption.

10. Mass bee stinging may cause

A very large venom load acts like an overdose of melittin and other components, causing systemic injury.

Key points to revise

1. Venom is injected, poison is ingested or absorbed, toxin is the general term.
2. The big four Indian snakes are cobra, krait, Russell's viper and saw-scaled viper.
3. Cobra and krait venoms are mainly neurotoxic.
4. Vipers cause coagulopathy, swelling and, in Russell's viper, kidney injury.
5. Keep the animal calm, still and move it quickly to a veterinarian.
6. Avoid cutting, sucking, tourniquets, ice and herbal pastes.
7. Polyvalent antivenom neutralises circulating venom but cannot reverse established damage.
8. Scorpion venom causes an autonomic storm and toad toxin acts like digoxin.

Go deeper on this site

- Ch 14. Drug toxicities and adverse drug events (in this book)
- Ch 12. Recreational drugs and human medicines in pets (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index
- Veterinary Drug Concepts Clearly Explained

Sources

- WHO fact sheet: Snakebite envenoming
- WHO SEARO guidelines for the management of snakebites
- Merck Veterinary Manual: Overview of toxicology

CHAPTER 14 OF 17

Drug toxicities and adverse drug events

Why therapeutic drugs become toxic in veterinary patients, with the characteristic overdose and adverse patterns of NSAIDs, ivermectin, anaesthetics, antibiotics and hormones. The chapter stresses species differences, recognition and the logic of response.

After this chapter you can

- Distinguish dose-related, idiosyncratic and species-specific adverse drug events
- Explain the mechanisms of NSAID toxicity in dogs, cats and vultures
- Describe ivermectin neurotoxicity and the role of MDR1 (ABCB1) in dogs
- Recognise typical adverse effects of anaesthetics, antibiotics and hormones in different species
- Outline a general response to a suspected drug toxicity and the need for reporting

Why drugs become toxins

Almost any drug can poison an animal if the exposure is too high, too prolonged or given to the wrong species or patient. Adverse drug events fall into a few groups. **Dose-related** events are an extension of the normal action (hypoglycaemia from too much insulin). **Idiosyncratic** events are unpredictable and not clearly dose-related. **Species-specific** events arise because enzymes, transporters or physiology differ between animals.

Common causes in practice are calculation or conversion errors, wrong species, wrong route, repeated dosing in an animal with kidney or liver disease, interacting drugs and owner dosing with human medicines. This is why the earlier chapters on pharmacokinetics matter: toxicity is usually the clinical result of a drug accumulating, not being cleared or acting where it should not.

Young, old, pregnant, dehydrated, underweight and sick animals are at higher risk, because drug distribution, metabolism and excretion differ in them. Always match the drug, dose and interval to the individual animal, and recheck the label for species before every use.

NSAIDs

NSAIDs inhibit cyclooxygenase. COX-1 products protect the gastric mucosa and maintain renal blood flow, so excessive or non-selective inhibition causes **gastric ulceration** and, in dehydrated or hypotensive animals, **acute kidney injury**. Signs

include vomiting, black tarry stools, inappetence, abdominal pain, polyuria or oliguria and lethargy. Combining an NSAID with a corticosteroid greatly raises ulcer risk, and switching between NSAIDs without a gap is a classic error.

Cats clear many NSAIDs and paracetamol slowly because they have limited glucuronidation. Paracetamol in cats causes methaemoglobinaemia (chocolate-brown blood, cyanosis, facial oedema) and red-cell damage. Dogs are also affected at higher exposures, with liver injury more prominent. Human NSAID tablets should never be given to pets without veterinary advice.

Vultures are a landmark case. Gyps vultures feeding on carcasses of cattle treated with diclofenac developed renal failure and visceral gout, and populations in South Asia collapsed. Regulatory action against veterinary diclofenac followed in India and neighbouring countries; check the current notification for what is permitted today.

Ivermectin and related macrocyclic lactones

Ivermectin binds glutamate-gated chloride channels in parasites, causing paralysis. In mammals the same drug is normally kept out of the brain by the efflux pump **P-glycoprotein**, encoded by the **MDR1 (ABCB1)** gene. Dogs with the mutant gene, such as Collies, Shelties, Australian Shepherds and related herding breeds, lack functional pump and the drug crosses the blood-brain barrier. It then acts on GABA-gated chloride channels, producing neurotoxicity.

Signs are mydriasis, salivation, tremor, ataxia (the so-called blind staggers), disorientation, blindness, bradycardia, coma and respiratory depression. Overdose in non-mutant dogs can produce the same picture, especially when large-animal formulations are used on small animals. Treatment is supportive and prolonged, as there is no true antidote. Some reports use intravenous lipid emulsion; ask a toxicologist or check current guidance.

Calves, cats and some other species are also susceptible to overdose, and certain reptiles such as tortoises and turtles are sensitive and should not be treated casually. Always check species and breed before using macrocyclic lactones, and check MDR1 status when in doubt.

Anaesthetics, sedatives and local anaesthetics

Most anaesthetic toxicity is dose-related depression of the brain, heart and breathing. Overdose of propofol, thiopentone, inhalants or opioids produces apnoea, hypotension and prolonged recovery. Several species differences are important.

- **Ruminants** are very sensitive to xylazine, showing profound sedation, salivation, bloat and bradycardia. Alpha-2 effects can be reversed with specific antagonists such as atipamezole or yohimbine.
- **Sighthounds** recover slowly from thiobarbiturates because they have little body fat to redistribute drug and metabolise it slowly.
- **Cats** can develop Heinz-body anaemia after repeated propofol, and repeated ketamine can cause prolonged recovery or seizures if overdosed.
- **Local anaesthetics** are safe at the site, but accidental intravenous injection or overdose causes seizures and cardiac toxicity; bupivacaine is especially cardiotoxic. Cats are sensitive to lidocaine at higher exposures.

Opioid overdose is reversed with naloxone, and benzodiazepine overdose with flumazenil, but both reversals wear off sooner than the drug being reversed, so the animal must be watched.

Antibiotics and antimicrobials

Antibiotic toxicity illustrates how a drug that is selectively toxic to microbes can still harm host tissue.

- **Aminoglycosides** (gentamicin, amikacin) are nephrotoxic, ototoxic and can cause neuromuscular blockade. Risk rises with dehydration, long courses and combination with loop diuretics.
- **Fluoroquinolones** can damage cartilage in growing dogs and cause retinal degeneration in cats, particularly with overdose of enrofloxacin.
- **Tetracyclines** bind calcium and can stain developing teeth or bone, and intravenous rapid injection can cause hypotension and collapse.
- **Gut flora disruption.** Some oral antibiotics, such as lincosamides and penicillins, cause fatal enterocolitis in rabbits, guinea pigs, hamsters and horses because they overgrow harmful bacteria.
- **Procaine penicillin** can cause excitement, tremor and collapse in horses if accidentally injected intravascularly.

- **Chloramphenicol** is a risk to humans handling it and is not allowed in food-producing animals in many countries; check the current notification in India.

Residues in milk and meat are an additional kind of toxicity, which is a public-health issue and discussed under withdrawal periods.

Hormones and recognition and response

Hormones are potent and have narrow margins. Excess insulin produces hypoglycaemia with weakness, tremor and seizures. Excess glucocorticoid causes polyuria, polydipsia, immunosuppression, delayed wound healing, ulcers and, in pregnancy, abortion or premature birth in ruminants. Prostaglandin F2 alpha and oxytocin given to the wrong animal can cause abortion or uterine rupture; prostaglandins are also hazardous to handlers, including pregnant women and asthmatics. Oestrogens in dogs can suppress bone marrow, and long-acting progestins risk pyometra and mammary changes. Anabolic steroids and beta-agonists such as clenbuterol are subject to restriction in food animals; check the current notification.

Recognition and response. Suspect a drug event when signs start soon after a new drug, a dose change or an owner-given medicine. First stop the suspected drug. Then support the airway, breathing, circulation and fluids, treat seizures and arrhythmia, give an antidote if one exists, and use decontamination if the exposure was oral and recent. Record the drug, batch, dose given and time. Report serious or unexpected events to the manufacturer and to the competent authority through the channel currently in use; check the latest reporting arrangements in India. Phone a poison information service or toxicologist early.

Concept cleared

Three types of adverse event
Dose-related, idiosyncratic and species-specific events differ in predictability. Species-specific events are the most testable in veterinary exams.

Transporters decide the brain dose

P-glycoprotein keeps drugs like ivermectin out of the brain. A mutation in MDR1 removes this barrier and makes normal doses toxic.

Limited glucuronidation in cats

Cats clear many NSAIDs and paracetamol slowly. The drug persists and metabolites that damage red cells accumulate.

Selective toxicity is relative

An antibiotic harmless to the host in one species can disrupt gut flora or damage kidney, cartilage or retina in another.

Memory aid: Response to suspected drug toxicity: STOP-ABC

S: Stop the drug. T: Take a history and record batch and time. O: Obtain urgent advice. P: Protect the patient and others. A: Airway. B: Breathing. C: Circulation, then antidote and decontamination if suitable.

Compare and contrast

Drug groups and characteristic toxicity

Drug group	Main mechanism of harm	Typical signs	At-risk animals
NSAIDs	COX-1 loss, reduced renal perfusion	Vomiting, melaena, kidney injury	Dehydrated animals, cats, vultures
Ivermectin	GABA channel effect in brain when pump absent	Tremor, ataxia, blindness, coma	MDR1 mutant dogs, young animals
Aminoglycosides	Tubular and inner ear toxicity	Polyuria, azotaemia, deafness	Dehydrated or renal patients
Alpha-2 agonists	Excess adrenergic stimulation	Deep sedation, bradycardia, bloat	Ruminants

Reversal agents for drug overdoses

Drug taken	Reversal agent	Key point
Opioids	Naloxone	Reversal is shorter than opioid effect
Benzodiazepines	Flumazenil	May unmask seizures
Xylazine, medetomidine	Atipamezole or yohimbine	Atipamezole is more selective for alpha-2
Ivermectin	None specific	Supportive care

Species notes

Dogs
Herding breeds with the MDR1 mutation are sensitive to ivermectin and some other drugs. Sighthounds recover slowly from thiobarbiturates.

Cats
Poor glucuronidation makes paracetamol and many NSAIDs dangerous. Enrofloxacin overdose can cause blindness.

Cattle and sheep
Very sensitive to xylazine. Hormonal overdose or wrong timing can cause abortion. Residues are a public-health concern.

Horses
Antibiotic enterocolitis and procaine penicillin reactions are well-known adverse events. NSAID overdose causes right dorsal colitis and ulcers.

Rabbits and rodents
Oral antibiotics that disturb gut flora can be fatal, so choose agents carefully.

Clinical pearls

Self-test: 10 questions

1. A drug effect that is unpredictable and not clearly related to dose is called

Idiosyncratic reactions occur in a few individuals regardless of dose, unlike dose-related events.

2. Why do NSAIDs cause gastric ulcers?

Prostaglandins maintain mucus, bicarbonate and mucosal blood flow, and COX inhibition reduces them.

3. Paracetamol toxicity in cats is characterised by

Limited glucuronidation allows toxic metabolites to oxidise haemoglobin, giving chocolate-coloured blood and cyanosis.

4. Vulture population decline in South Asia was linked to

Vultures developed renal failure and visceral gout after feeding on carcasses of cattle recently treated with diclofenac.

5. MDR1 mutation in Collies increases sensitivity to ivermectin because

Without the efflux pump the drug penetrates the blood-brain barrier and acts on GABA channels.

6. Which animals are especially sensitive to xylazine?

Ruminants show profound sedation, bradycardia, salivation and bloat at amounts that are mild in other species.

7. Aminoglycoside toxicity risk rises with

Dehydration reduces renal perfusion and increases drug accumulation in tubular cells.

8. Oral antibiotics such as lincosamides are dangerous in

Hindgut fermenters can suffer fatal clostridial overgrowth when normal flora are disrupted.

9. What is the first step when a drug toxicity is suspected?

Stopping the cause and stabilising vital functions come before any other measure.

10. Naloxone reversal of an opioid overdose should be followed by

Naloxone has a shorter duration than many opioids, so sedation and respiratory depression can return.

Key points to revise

1. Adverse events are dose-related, idiosyncratic or species-specific.
2. NSAIDs cause gastric ulcer and renal injury by reducing protective prostaglandins.
3. Diclofenac residues caused severe vulture declines in South Asia.
4. Ivermectin neurotoxicity occurs when P-glycoprotein is absent or overwhelmed.
5. Ruminants are very sensitive to xylazine.
6. Aminoglycosides injure kidney, ear and neuromuscular transmission.
7. Some antibiotics cause fatal enterocolitis in rabbits, guinea pigs and horses.
8. Stop the drug, support the patient, give an antidote if available and report serious events.

Go deeper on this site

- Ch 15. Antidotes and emergency decontamination quick reference (in this book)
- Ch 13. Venoms, bites and stings (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Concepts
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Overview of pharmacology
- Merck Veterinary Manual: Overview of toxicology
- Merck Veterinary Manual: Introduction to poisoning (pet owners)

CHAPTER 15 OF 17

Antidotes and emergency decontamination quick reference

A concept-level reference matching common poisons to antidotes, with the mechanism and cautions for each, and the reasoning behind emesis, activated charcoal and lavage. It stresses when decontamination should not be attempted and when to call for urgent help.

After this chapter you can

- Match common poisons to their antidotes and explain each mechanism
- State the main cautions and limits of antidotes
- Explain when emesis is appropriate and when it must not be induced
- Describe how activated charcoal, cathartics and lavage work and where they fail
- Apply a stepwise approach to a poisoned animal, including ruminant and skin or eye exposure

The stepwise approach

The first priority in any poisoning is the patient, not the poison. Secure the airway, breathing and circulation, control seizures and temperature, and only then think about removing or neutralising the poison. The usual order is **stabilise, prevent further exposure, decontaminate, give an antidote if one exists, enhance elimination and give supportive care**.

Most poisons have no antidote. Good supportive care is therefore the commonest treatment. Obtain a history of the agent, the amount, the route and the time of exposure, keep the container or sample, and contact a poison information service or toxicologist early. This chapter is a concept summary; doses and current protocols must come from a current formulary or specialist advice.

How antidotes work

Antidotes work in a handful of ways. Knowing the mechanism helps you remember both the use and the limit.

- **Competitive receptor blockade or reversal:** atropine blocks muscarinic receptors, naloxone blocks opioid receptors, flumazenil blocks benzodiazepine binding and atipamezole blocks alpha-2 receptors.
- **Enzyme reactivation:** pralidoxime reactivates acetylcholinesterase inhibited by organophosphates.
- **Replacing a depleted substance:** acetylcysteine restores glutathione after paracetamol, and vitamin K1 restores clotting factor synthesis after anticoagulant rodenticides.
- **Blocking toxic metabolism:** fomepizole inhibits alcohol dehydrogenase so ethylene glycol is not converted to toxic acids.
- **Chelation:** calcium EDTA, dimercaprol and succimer bind metals such as lead and arsenic so they can be excreted.
- **Binding or neutralising the toxin:** antivenom and digoxin-specific antibody fragments bind their targets, and Prussian blue binds thallium in the gut.
- **Other mechanisms:** methylene blue helps convert methaemoglobin back to haemoglobin, and cyanide antidotes act by forming less toxic complexes.

Antidote quick reference and cautions

The following pairings are commonly taught. Each antidote has limits.

1. **Organophosphate and carbamate:** atropine controls muscarinic signs. Pralidoxime works best early, before the enzyme becomes permanently inhibited (ageing), and is generally not needed for carbamates. Atropine is titrated to drying of airway secretions, not to heart rate alone.
2. **Paracetamol:** acetylcysteine. Cats also need treatment for methaemoglobinaemia.
3. **Methaemoglobin-forming agents:** methylene blue, with caution in cats because it can itself cause Heinz-body anaemia.
4. **Ethylene glycol:** fomepizole in dogs; the timing window is short, and cats need earlier treatment. Ethanol is an older alternative.

5. **Anticoagulant rodenticides:** vitamin K1 for weeks, as some compounds persist; check clotting after stopping.
6. **Opioids and benzodiazepines:** naloxone and flumazenil, which have shorter actions than the poison.
7. **Lead and arsenic:** chelators, with attention to the kidney and to preventing further oral intake.
8. **Cyanide:** nitrite and thiosulfate or hydroxocobalamin are used; hedge by checking current protocols because nitrites form methaemoglobin.
9. **Snakebite:** species-specific antivenom, as covered in the previous chapter.
10. **Isoniazid:** pyridoxine. **Heparin:** protamine.

A frequent confusion is to treat the antidote as a cure for any exposure. It is not: an antidote helps only when it is matched to the agent, given in time and accompanied by supportive care.

Emesis: when and when not

Inducing vomiting removes stomach contents shortly after ingestion. It helps most when the poison was swallowed recently, usually within about one to two hours, is not corrosive or a hydrocarbon, and the animal is alert. In dogs, apomorphine is commonly used; in cats, alpha-2 agonists such as xylazine are commonly used, and cats respond poorly to many dog methods. Hydrogen peroxide and salt are widely advised by laypeople but carry risk, and salt can cause dangerous sodium poisoning, so veterinary guidance should be sought first.

Do not induce vomiting when:

- The animal is already vomiting, seizing, collapsed, depressed or has a poor gag reflex, because of aspiration risk.
- The poison is a **corrosive** (strong acid, alkali), because it burns the oesophagus a second time on the way back.
- The poison is a **hydrocarbon** such as petrol or kerosene, because aspiration causes severe pneumonia.
- The material is sharp, or the animal has a condition likely to worsen with vomiting.
- The species cannot vomit safely: **ruminants, horses, rabbits and rodents** do not vomit effectively.

- The animal has taken a drug that will itself cause rapid sedation or seizures.

Activated charcoal, cathartics and lavage

Activated charcoal has a very large surface area and adsorbs many toxins in the gut, preventing absorption. Repeat doses can also interrupt enterohepatic circulation of drugs excreted in bile and can pull some drugs back from the blood into the gut. It is poor at adsorbing alcohols, ethylene glycol, strong acids and alkalis, and many metals, and it is dangerous if aspirated. Charcoal with sorbitol may cause diarrhoea and fluid shifts; in salt poisoning it can contribute to hypernatraemia, so it should be used under advice.

Cathartics hasten gut transit but have limited proven benefit and can cause dehydration, so they are not routine.

Gastric lavage is used for an anaesthetised animal with a cuffed endotracheal tube in place, to remove stomach contents when emesis is not suitable. It is not suitable for corrosives or sharp objects.

In **ruminants**, activated charcoal and adsorbents are given by stomach tube. Ruminal lavage or rumenotomy to remove toxic plant matter and transfer of rumen fluid from a healthy animal may be used. Because the rumen holds a large volume that is released slowly, exposure may continue for hours after ingestion.

For **skin exposure**, wear gloves and wash with water and mild detergent while preventing hypothermia. For the **eye**, irrigate with copious clean water or saline. Avoid neutralising a corrosive with another chemical, since the reaction generates heat.

Enhancing elimination and when to seek urgent help

Some poisons can be removed faster once absorbed. Fluid diuresis speeds renal excretion of water-soluble agents, and urinary pH manipulation helps for some weak acids. Dialysis can remove agents such as ethylene glycol or some drugs when kidneys fail. Lipid emulsion has been used for lipophilic drugs; consult current guidance.

Seek urgent help at once for: seizures, collapse, difficulty breathing, blue or dark-brown mucosae, persistent vomiting or bloody stools, known ingestion of rodenticide, antifreeze, pesticides, human drugs, or any corrosive, and any

ingestion by a very young or small animal. Delay hurts because time-sensitive antidotes such as fomepizole and pralidoxime lose value as hours pass.

Prevention is the best antidote: store chemicals and drugs out of reach in labelled containers, avoid giving human medicines to animals, read labels on pesticides, and keep animals away from treated fields until the safe interval has passed.

Concept cleared

Match the antidote to the mechanism
Antidotes block, reactivate, replace, chelate or bind. If you know the poison's mechanism, the antidote usually follows.

Most poisons have no antidote
Supportive care is the main treatment in most poisonings. An antidote adds to it and does not replace it.

Timing limits decontamination
Emesis and charcoal help mostly in the first hours. Late attempts add risk without benefit.

Safety of the airway comes first
Many decontamination methods fail because of aspiration. Protect the airway before using them.

Memory aid: No vomiting: CHAOS
C: Corrosives. H: Hydrocarbons. A: Already unconscious or seizing. O: Obstructed or sharp object risk. S: Species that cannot vomit, such as ruminants, horses and rabbits.

Compare and contrast

Poison and antidote pairs

Poison	Antidote	Mechanism	Caution
Organophosphate	Atropine, pralidoxime	Block muscarinic effects, reactivate enzyme	Pralidoxime works best early
Paracetamol	Acetylcysteine	Restores glutathione	Cats also need methaemoglobin care
Ethylene glycol	Fomepizole	Blocks alcohol dehydrogenase	Narrow time window
Anticoagulant rodenticide	Vitamin K1	Restores clotting factors	Long courses may be needed
Lead	Calcium EDTA, succimer	Chelation	Renal monitoring

Decontamination methods

Method	Best used	Main limit or risk
Emesis	Recent ingestion, alert dog or cat	Aspiration, corrosives, hydrocarbons, ruminants
Activated charcoal	Many drugs and plant toxins	Does not bind alcohols, glycols, many metals
Gastric lavage	Anaesthetised animal with cuffed tube	Corrosives, sharp objects
Skin or eye wash	Dermal or ocular exposure	Cold stress, never use chemical neutralisers

Species notes

Dogs

Apomorphine is the usual emetic. Ethylene glycol and chocolate type exposures are classic reasons for early help.

Cats

Emesis uses different drugs from dogs. They are sensitive to methylene blue and paracetamol and respond late to ethylene glycol.

Cattle, buffalo, sheep and goats

Horses

Cannot be made to vomit. Use charcoal by stomach tube, ruminal lavage or rumenotomy as indicated.

Cannot vomit. Gastric reflux and nasogastric tubing with charcoal are possible, with care for aspiration.

Rabbits and rodents
Cannot vomit. Rely on charcoal and supportive care, and watch for hypothermia.

Clinical pearls

Self-test: 10 questions

1. What is the first priority in a poisoned animal?

Life-threatening problems are treated before the poison is addressed.

2. Pralidoxime acts by

It removes the organophosphate from the enzyme, and works best before ageing makes the bond permanent.

3. Acetylcysteine is the antidote for

It restores glutathione, which neutralises the toxic metabolite of paracetamol.

4. Fomepizole prevents toxicity of ethylene glycol by

Blocking the enzyme stops conversion of ethylene glycol to toxic metabolites that injure the kidney.

5. Vitamin K1 is used in poisoning by

These poisons block vitamin K recycling, and giving vitamin K1 restores clotting factor synthesis.

6. Vomiting should NOT be induced after ingestion of

Hydrocarbons are easily aspirated and cause severe pneumonia.

7. Which species cannot vomit effectively?

Ruminants, horses, rabbits and rodents do not vomit effectively, so other decontamination methods are used.

8. Activated charcoal is poor at binding

Small polar molecules such as alcohols and glycols are not adsorbed well.

9. Gastric lavage should be done only when

Without airway protection lavage risks aspiration.

10. For a skin exposure to a chemical you should

Washing removes the agent. Neutralising reactions can produce heat and further injury.

Key points to revise

1. Treat the patient before the poison.
2. Most poisons have no antidote.
3. Atropine and pralidoxime are used for organophosphates.
4. Acetylcysteine is used for paracetamol and fomepizole for ethylene glycol.
5. Vitamin K1 is used for anticoagulant rodenticides.
6. Emesis is useful only soon after ingestion in alert dogs and cats.

7. Do not induce vomiting after corrosives, hydrocarbons, or in seizing animals and species that cannot vomit.
8. Skin and eye exposures need washing, not chemical neutralisation.

Go deeper on this site

- Ch 16. Environmental, poultry and wildlife toxicology (in this book)
- Ch 14. Drug toxicities and adverse drug events (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Concepts
- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Overview of toxicology
- Merck Veterinary Manual: Introduction to poisoning (pet owners)
- WHO fact sheet: Snakebite envenoming

CHAPTER 16 OF 17

Environmental, poultry and wildlife toxicology

How poisons move through the environment, feed chain and carcasses to harm animals that were never treated directly. The chapter uses diclofenac and Gyps vultures as the central case, then covers pesticide residues, water and soil contaminants and the common poultry toxicoses.

After this chapter you can

- Explain how diclofenac in livestock carcasses caused vulture declines in South Asia
- Describe bioaccumulation and why persistent pesticide residues matter in food animals
- List common water and soil contaminants and the reasoning behind their toxic effects
- Identify the main poultry-specific toxicoses and their usual sources on farms

- Apply prevention and urgent-help principles to environmental and flock poisonings

Why environmental toxicology is different

In clinical toxicology one animal is exposed and one animal is treated. In environmental toxicology the poison reaches animals indirectly: through water, soil, plants, feed, or the body of another animal. The affected species may never have been given the substance by a veterinarian.

Three ideas link this chapter. First, **exposure route** matters as much as toxicity. Second, a substance that is safe in the species it was meant for can be dangerous to a different species that eats the carcass. Third, many problems appear as a flock or herd event, or as a slow population decline, so the diagnosis depends on pattern recognition and on laboratory support, not on a single sick patient.

Diclofenac and vultures

Diclofenac is a non-steroidal anti-inflammatory drug (NSAID) used in cattle and buffalo for pain and fever. In South Asia, Gyps vultures (white-rumped, long-billed and slender-billed) feed on carcasses of dead livestock. When a treated animal died shortly after dosing, its tissues still held drug residue, and the vultures that fed on it were exposed.

The mechanism is **acute kidney failure**. Affected birds show lethargy, drooping of the neck, and death within days. At post-mortem the typical finding is **visceral gout**: white chalky urate deposits on the liver, heart, air sacs and joints, because failing kidneys cannot excrete uric acid. Vultures are very sensitive to this drug, whereas many other species tolerate it far better. The reason for the sensitivity is not fully settled, so students should state it as a species difference and not claim a single proven mechanism.

Populations of these vultures fell by more than ninety per cent in a short period. Because vultures breed slowly and a small fraction of carcasses carrying residue is enough to kill many birds, the decline was steep. The loss has wider meaning: carcasses were not cleared, scavenging dogs increased, and public health risks such as rabies exposure were raised.

India restricted veterinary diclofenac in 2006. Meloxicam has been promoted as a vulture-safe alternative, and other NSAIDs have raised concern in later work. Please check the current notification from the regulator for the present status of each drug. The lesson for a veterinarian is practical: choose carefully, do not dispense human-formulation vials for animals, and dispose of carcasses of recently treated animals responsibly.

Pesticide residues and bioaccumulation

Pesticides reach animals through sprayed fodder, contaminated water, dips and sprays, and treated seed. Two terms are important. **Bioaccumulation** means a substance builds up in the tissues of one organism over time. **Biomagnification** means concentration rises at each step of the food chain, so predators and fish-eating or carcass-eating birds carry the highest levels.

Persistent organochlorines, such as DDT and related compounds, are fat-soluble and resist breakdown. They collect in body fat and appear in milk and eggs. They act mainly on the nervous system and can also affect reproduction and eggshell quality in birds. Organophosphates and carbamates break down faster, but they act on cholinesterase and cause acute cholinergic signs, which are covered in the earlier pesticide chapters.

For food animals, the concern is **residues** in milk, meat and eggs. Maximum residue limits (MRLs) and withdrawal periods exist to protect consumers. Indian limits are set by the food safety authority and change over time, so check the current notification before quoting any value. Good practice includes using products only as labelled, keeping treated animals from freshly sprayed pasture for the stated interval, and recording treatments.

A common confusion: a low acute toxicity does not mean low residue risk. A fat-soluble, persistent chemical can be safe in a single exposure yet still accumulate.

Water and soil contaminants

Water and soil carry both natural and industrial toxicants. The usual reasoning is to ask what the substance does, where it accumulates, and which animals are most exposed.

- **Fluoride.** Occurs naturally in groundwater in some regions. Long exposure damages teeth and bones (fluorosis), causing mottled teeth, stiffness and lameness. Young animals are affected during tooth and bone development.
- **Arsenic.** Natural or industrial contamination of groundwater. It blocks cellular energy pathways and damages the gut lining, liver and kidney.
- **Lead and cadmium.** From batteries, paints, mining and industrial waste. Lead affects the nervous system and blood formation; cadmium accumulates in kidney and liver.
- **Nitrates and nitrites.** From fertiliser, manure and some plants. Nitrite converts haemoglobin to methaemoglobin, so tissues are starved of oxygen although the animal breathes normally. Ruminants convert nitrate to nitrite in the rumen and are especially at risk.
- **Blue-green algal blooms.** In stagnant, warm ponds. Some species release toxins affecting the liver or nervous system, and sudden death after drinking is typical.
- **Salinity.** Very salty water or sudden access after deprivation can cause nervous signs.

Prevention rests on testing water sources, fencing stagnant ponds, safe disposal of batteries and oil, and providing an alternative clean water supply. If several animals on one source become ill together, think of water early.

Poultry-specific toxicoses

Poultry are kept in large numbers with shared feed and water, so a single error can affect the whole flock. Birds also have fast metabolism and small body size, and they lack some of the safety margins of mammals.

- **Mycotoxins.** Aflatoxin from mouldy grain damages the liver and depresses growth, immunity and egg production. Ochratoxin and trichothecenes such as T-2 toxin cause kidney damage and mouth lesions. Poor storage in humid weather is the main cause.
- **Ionophore coccidiostats.** Monensin, salinomycin and related drugs are safe at the intended level but toxic when mixing errors occur or when given with certain other drugs, such as tiamulin. They damage muscle, so birds show weakness, leg paralysis and reluctance to move. Feed mixing and label checking are the prevention.

- **Salt toxicity.** Excess salt in feed, or water deprivation, causes thirst, wet droppings and nervous signs.
- **Carbon monoxide and gases.** From poorly ventilated brooders. Chicks may be found dead or dull, and the problem is a ventilation fault.
- **Other feed errors.** Wrong premix levels, contaminated feed from equipment shared with other species, and pesticide-treated seed.

The investigation approach is to look at the flock pattern, the timing relative to a new feed batch, and then to submit feed, water and fresh carcasses to a laboratory. Withdraw the suspect feed first and provide clean water; that single step often limits further loss.

Diagnosis, prevention and seeking urgent help

Environmental and flock cases follow a common sequence. Stop the exposure, protect the survivors, collect samples, and treat supportively. Many of these toxicants have no specific antidote, so removing the source is the most valuable action.

Wildlife cases need extra care. Dead wild animals may carry zoonotic disease or poison residues, so handle with gloves, report to the forest or wildlife authority, and do not dispose of carcasses in a place where scavengers will feed. Mass deaths of wild birds should be reported without delay, since the cause may be a poison, an infectious disease or both.

Seek urgent help from a poison laboratory, a toxicology colleague, or the public health and animal husbandry authorities when: several animals die suddenly, a human may have been exposed (for example by eating affected eggs or meat), a protected species is involved, or the source is an industrial discharge. Keep notes and samples, since these may later be needed in a legal investigation, discussed in the next chapter.

Prevention is a matter of habits: use drugs as labelled, store feed dry, test water, avoid human-formulation drugs in livestock, and train farm workers to report unusual deaths early.

Concept cleared

Secondary poisoning

An animal is harmed by eating the tissues of another animal that carries the poison. Vultures and diclofenac are the standard example.

Bioaccumulation and biomagnification

Persistent, fat-soluble substances build up in an organism and become more concentrated at each step of the food chain.

Species differences in sensitivity

A drug safe in cattle can be lethal in vultures. Safety data from one species cannot be assumed for another.

Flock-level thinking

In poultry, think of shared feed and water first. A single mixing or storage error can explain a whole house of sick birds.

Memory aid: Environmental case checklist: SWEEP

Source, Water, Exposure route, Exposed species, Prevention. Run through these five in order when a group of animals falls ill.

Compare and contrast

Persistent and non-persistent pesticides

Feature	Organochlorines	Organophosphates and carbamates
Persistence	Long, resist breakdown	Short, break down faster
Storage	Fat, milk, eggs	Little accumulation
Main concern	Residues and chronic effects	Acute cholinergic signs
Food-chain effect	Biomagnify	Rarely biomagnify

Common poultry toxicoses

Toxicosis	Usual source	Typical clue
Aflatoxicosis	Mouldy grain	Liver damage, poor growth, immunosuppression
Ionophore toxicity	Mixing error or drug interaction	Leg weakness, paralysis
Salt toxicity	Excess salt or water deprivation	Thirst, wet droppings, nervous signs
Carbon monoxide	Faulty brooder ventilation	Dull or dead chicks

Species notes

Vultures
Highly sensitive to diclofenac, with kidney failure and visceral gout after feeding on treated carcasses.

Cattle and buffalo
The treated species; they tolerate the drug but become the source of exposure for scavengers if they die soon after treatment.

Ruminants
Rumen bacteria convert nitrate to the more harmful nitrite, so nitrate-rich fodder and water are higher risk.

Poultry
Sensitive to mycotoxins and ionophores; shared feed means whole-flock outbreaks.

Dogs and wild scavengers
Exposed through carcasses and contaminated water; may act as sentinels of poisoning in the area.

Clinical pearls

Self-test: 10 questions

1. The typical post-mortem finding in vultures poisoned by diclofenac is:

Kidney failure leaves urates unexcreted, which deposit on visceral surfaces as visceral gout.

2. Vultures were exposed to diclofenac mainly by:

This is secondary poisoning through carcass tissue.

3. Biomagnification means:

Persistent substances become more concentrated at higher trophic levels.

4. Which pesticide group is most associated with storage in fat and appearance in milk and eggs?

Organochlorines are persistent and fat-soluble.

5. Nitrite poisoning produces signs mainly because of:

Nitrite oxidises haemoglobin so it cannot carry oxygen.

6. Which of these animals is especially vulnerable to nitrate poisoning from fodder?

Rumen microbes reduce nitrate to nitrite.

7. A flock shows leg weakness after a new feed batch containing coccidiostat. The best suspicion is:

Ionophore overdose or interaction damages muscle.

8. The first step in a suspected feed-borne flock poisoning is to:

Removing the source limits further exposure.

9. Chronic fluoride exposure mainly affects:

Fluorosis causes dental mottling and skeletal changes.

10. Which statement about Indian MRLs is best?

Limits are revised, so rely on the current official notification.

Key points to revise

1. Diclofenac residues in carcasses caused kidney failure and visceral gout in Gyps vultures.
2. India restricted veterinary diclofenac in 2006; check the current notification for NSAID status.
3. Vulture sensitivity is a species difference whose exact mechanism is not fully settled.
4. Bioaccumulation builds up in one organism; biomagnification rises along the food chain.
5. Organochlorines are persistent and stored in fat, milk and eggs.
6. Nitrite causes methaemoglobinaemia and is a particular risk in ruminants.
7. Poultry toxicoses include mycotoxins, ionophores, salt and gas exposure.
8. Removing the source is the first and often the most effective step.

Go deeper on this site

- Ch 17. Medico-legal toxicology in India (in this book)
- Ch 15. Antidotes and emergency decontamination quick reference (in this book)
- Veterinary drugs list: classes, uses and cautions
- Veterinary Drug Index

- Veterinary Drug Concepts Clearly Explained

Sources

- Merck Veterinary Manual: Toxicology (contents)
- World Organisation for Animal Health (WOAH)
- US FDA Center for Veterinary Medicine

CHAPTER 17 OF 17

Medico-legal toxicology in India

How a veterinarian handles a suspected poisoning that may reach a court: collecting and preserving samples, the outline of laboratory methods, post-mortem findings, chain of custody, reporting and giving evidence. The focus is on reasoning and procedure, not on dosing.

After this chapter you can

- Describe which samples to collect in a suspected poisoning and how to preserve them
- Outline what TLC, GC-MS and ELISA do and where each is useful
- Explain chain of custody and why a break in it weakens evidence
- Recognise post-mortem findings that suggest poisoning and their limits
- Prepare a clear report and understand the basics of court procedure

Why medico-legal work needs a different mindset

A clinical poisoning is treated for the sake of the patient. A **medico-legal** poisoning may also involve a crime, an insurance claim, a dispute between owner and supplier, or a case of cruelty. Examples include malicious poisoning of dogs or cattle, poisoning of racing animals, suspected feed adulteration, and insurance deaths.

The veterinarian is first a clinician and second a witness. Treatment must never be delayed to protect evidence, but every step should be recorded so that the evidence stays reliable. A careful note made at the time is worth more than a long memory later.

Laws and procedure codes in India have been revised recently. Names of sections and acts may differ from older textbooks, so check the current statute and the instructions of the state department before relying on any section number.

Sample collection and preservation

The aim is to obtain the samples most likely to hold the poison and to keep them unchanged until analysis. The reasoning is: collect what the poison reaches first, what stores it, and what removes it.

- **Stomach or rumen contents and vomitus.** The most useful sample when the poison was swallowed, since it may hold the actual material.
- **Liver.** The main site of metabolism and storage; useful for many drugs and metals.
- **Kidney.** Useful for metals and for substances excreted in urine.
- **Blood and urine.** From live animals or fresh carcasses.
- **Brain.** Needed for certain conditions, such as cholinesterase measurement in suspected organophosphate poisoning.
- **Feed, water, bait and suspected container.** Often the most important evidence, since it ties the poison to the source.

Use clean glass or good plastic containers, one for each organ, and never mix samples. Do not use formalin for samples meant for chemical analysis, because it interferes with testing. Cool or freeze samples as advised by the laboratory. Some laboratories in India traditionally ask for visceral samples in a saturated salt solution or in rectified spirit; follow the instructions of the receiving laboratory and state the preservative on the label. Always send a sample of the preservative alone as a control.

Seal each container, label it with case number, animal, organ, date and time, and sign the label. Collect a separate set for histopathology in formalin, as this is a different question from chemistry.

Laboratory methods in outline

The veterinarian does not need to run these tests but should know what each can and cannot answer.

- **Thin-layer chromatography (TLC).** A mixture is spotted on a plate and separated as a solvent moves through it. Spots are compared with known standards. It is simple, cheap and useful for screening, but it is less specific and less sensitive.
- **Gas chromatography-mass spectrometry (GC-MS).** Gas chromatography separates volatile compounds, and mass spectrometry identifies each by its fragment pattern. It is a confirmatory method widely accepted in legal work for many pesticides and drugs. Other forms, such as liquid chromatography with mass spectrometry, cover less volatile compounds.
- **ELISA.** An antibody-based test used for rapid screening of specific toxins or drugs. It is quick, but it can cross-react with related compounds, so a positive result usually needs confirmation by chromatography.

A common error is to treat a screening result as proof. Courts look for a method that identifies the compound with confidence. A second point is that a negative result does not rule out poisoning, since the substance may have degraded, been metabolised, or not been tested for. The veterinarian should give the laboratory a clear history and suspected agents so that the right panel is chosen.

Chain of custody

Chain of custody is the written record showing who held a sample, from collection to the court, and that it was not tampered with. Each transfer is recorded with name, date, time and signature.

The practical steps are: seal samples in front of a witness where possible, write a forwarding note listing the items, hand them directly to the police or the laboratory messenger, and keep a receipt. Keep copies of every document. If a sample sits in an unlocked cupboard or travels without a record, the defence can argue that it was changed, and the evidence may be set aside.

Think of it like a relay baton: any moment where no one can say who held it breaks the chain.

Post-mortem findings in suspected poisoning

A post-mortem should be done early, in a systematic way, with photographs and measured notes. Findings that raise suspicion include sudden death in a previously healthy animal, several deaths together, unusual smell from the stomach contents,

discoloured or ulcerated gut lining, congestion of organs, and visible bait or plant material.

Some patterns point toward groups of poisons. For example, a chemical smell or a bright blue-green colour in stomach contents may suggest a particular pesticide. Pale tissues with bleeding suggest anticoagulant rodenticides. Corrosive injury suggests acids or alkalis. Kidney changes suggest nephrotoxic agents.

The limits are important. Many poisons leave **no specific lesion**. Putrefaction can hide or mimic findings. An infectious disease such as anthrax or clostridial disease can look similar and must be considered. For this reason the post-mortem is a guide to which samples to send, not a final answer. State only what you observed, and avoid declaring a cause of death before laboratory results arrive.

Reporting and court procedure

A good report is plain, factual and dated. It should contain the identity of the animal and owner, history, clinical signs, treatment given, post-mortem findings, samples collected with their preservation, and the chain of custody. Opinions should be separated from observations, and any uncertainty stated honestly.

When the case is a police matter, the report goes to the investigating officer. The laboratory report then arrives separately. The final opinion on cause of death is formed by combining history, lesions and laboratory results.

If called to court, the veterinarian appears as an expert witness. Bring the original notes, and be ready to explain how samples were collected and sealed. Answer only what is asked, in simple language, and do not stretch beyond the evidence. Admit what is unknown. Attendance on the summons date matters, so tell the court in advance if there is a genuine difficulty.

Relevant laws can include provisions on cruelty to animals and on harming animals, and the rules of evidence for experts. Because these Acts have recently been revised, confirm the present provisions with the legal authority before quoting them. Seek advice from a senior colleague or the state veterinary council if unsure how to proceed.

Concept cleared

Evidence, not just diagnosis

In a medico-legal case every step must be documented so that another person could verify it later.

Screening versus confirmation

TLC and ELISA suggest; GC-MS and related methods confirm. A court needs confirmation.

Chain of custody

A signed record of every hand that held the sample, proving it was not changed.

Absence of lesions is not absence of poison

Many poisons leave no specific post-mortem change, so samples must be collected anyway.

Memory aid: Medico-legal steps: SCRIPT

Secure the scene, Collect samples, Record everything, Identify and label, Pass on with chain of custody, Testify honestly.

Compare and contrast

Laboratory methods

Method	Principle	Role
TLC	Separation on a plate, compared with standards	Simple screening
ELISA	Antibody binding to target	Rapid screening, may cross-react
GC-MS	Separation then identification by fragments	Confirmation

Samples and what they show

Sample	Value	Note
Stomach contents	Actual material swallowed	Collect early
Liver	Metabolism and storage	Drugs, metals
Kidney	Excretion	Metals, nephrotoxins
Brain	Specific tests	Cholinesterase in suspected organophosphate case
Feed or bait	Source of poison	Often decisive

Species notes

Dogs

Most common victims of malicious bait poisoning; vomitus and stomach contents are valuable samples.

Cattle and buffalo

Rumen contents are bulky and mixed, so collect from several areas and also sample feed and fodder.

Horses

Poisoning of competition animals involves blood and urine sampling, and strict sealing procedures.

Poultry

Submit several fresh carcasses and feed, since the question is usually about the flock.

Wildlife

Often needs the forest authority; handle carcasses carefully and report promptly.

Clinical pearls

Self-test: 10 questions

1. Which preservative should be avoided for samples meant for chemical analysis?

Formalin interferes with chemical testing; it is kept for histopathology samples.

2. Why is a control sample of the preservative sent?

It rules out contamination from the preservative.

3. Which method is generally regarded as confirmatory?

Mass spectrometry identifies compounds by fragment pattern.

4. ELISA results should be treated as:

Antibody tests may cross-react.

5. Chain of custody refers to:

It shows the sample was handled and not changed.

6. Which sample is often the most valuable in a suspected oral poisoning?

It may contain the actual poison.

7. A post-mortem with no specific lesions:

Many poisons leave no specific lesion.

8. The brain is collected in suspected organophosphate cases for:

Reduced cholinesterase supports exposure to these compounds.

9. In a report, the veterinarian should:

Clear separation protects the credibility of the evidence.

10. Before quoting a section of law in court, one should:

Indian criminal and evidence laws have been revised recently.

Key points to revise

1. Medico-legal cases require documentation at every step.
2. Collect stomach contents, liver, kidney, blood, urine, brain where relevant, and feed or bait.
3. Use separate labelled and sealed containers and follow the laboratory's preservative advice.
4. TLC and ELISA are screening tools; GC-MS confirms.
5. Chain of custody records each person who held the sample.
6. Many poisons leave no specific lesions, and infectious diseases can mimic poisoning.
7. Reports must separate observation from opinion.
8. Check current statutes and notifications before quoting legal provisions.

Go deeper on this site

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How to use Book 6: a short discussion

Toxicology is the study of how substances harm living animals and what can be done about it. In veterinary practice poisoning is a daily emergency: a dog that has eaten chocolate or a human painkiller, a herd that has grazed a toxic weed after the first rains, a flock exposed to a contaminated feed, a cat that has walked through a freshly treated floor. This book covers seventeen of the most important areas, arranged so that principles come first and detail follows.

Principles before poisons

The first chapter explains how a toxic substance is absorbed, distributed, changed and removed, why dose and timing matter, and how the general approach to a poisoned animal is organised: stabilise, find out what was taken, prevent further absorption, give an antidote when one exists, and support the animal while the body clears the poison. Reading this chapter first makes every later chapter easier, because most poisons follow the same logic with a different target.

Grouped by source

The middle chapters group poisons by where animals meet them: pesticides and rodenticides, herbicides and fungicides, plants, heavy metals, feed contaminants, toxic gases and algae, photosensitising agents, household products, human medicines and venoms. This follows the way a case arrives in clinic, with a history of exposure, so you can move from the history to the likely poison and then to the signs and the response.

Species and Indian conditions

Species differences matter greatly. Cats handle many chemicals poorly, ruminants are vulnerable to plants and feed errors that other species tolerate, and horses react badly to certain feed additives. The book also reflects Indian conditions, including seasonal plant poisoning, pesticide use on farms, snakebite and the fate of vultures

after exposure to a veterinary painkiller. Where Indian rules or availability are mentioned, the text asks you to check the current notification instead of trusting a fixed list.

Antidotes and the law

Chapter 15 gathers antidotes and decontamination methods into a quick reference that explains the mechanism of each and the cautions that apply, without doses. The final chapters deal with environmental, poultry and wildlife toxicology and with medico-legal work: how to collect and preserve samples, how laboratories analyse them, how a chain of custody is kept and what is expected in a report or in court.

Taking a poisoning history

The most valuable tool in toxicology is a careful history. Ask what the animal could have reached, when, in what form and how much, and whether other animals were exposed. Ask about recent feed changes, pesticide use, new plants in the pasture, human medicines in the house and any recent treatment given at home. Keep the packaging or a sample of the suspected material, and take samples early, because some poisons disappear from the body quickly. The chapters repeatedly show how the history points toward the poison, and how the clinical signs then confirm or challenge your first idea.

Using this book safely

This is a study book. It does not contain toxic-dose tables or treatment doses, and it must not be used to manage a real poisoning by itself. In an emergency, contact an animal poison service, a toxicologist or your clinic's senior veterinarian straight away. Use the self-test at the end of each chapter, repeat it after a week, and read the Species notes and Revise points before an examination.

The later chapters link to the drugs and anaesthesia material in Books 2 to 5, because many poisonings are treated with drugs whose mechanisms you have already studied.