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# POISONS AND THE PACHYDERM

## RESPONDING TO POISONING IN ASIAN ELEPHANTS – A FIELD GUIDE

Jacob V. Cheeran

Wildlife Trust of India

10 Years in Service of Nature

IFAW.org

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A Better World for Animals and People

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# PREFACE

An exponential increase in human population has led to the shrinking of natural animal habitats resulting in an inevitable conflict between the two. In India, elephant habitat is slowly but surely being replaced by human settlements and associated activities like agriculture and industries thereby disrupting traditional elephant paths and reducing their food supply. The decrease in habitat area has been forcing the animals to frequent the adjoining crop fields only to raid and destroy them. This also results in the death of several hundreds of animals and people every year. This rise in the rate of human and animal mortality has become inevitable as Asian elephants have less and less natural habitat to feed and roam. The human-elephant conflict is now the major reason for individual elephant deaths through indiscriminate poisoning, shooting and trapping.

It therefore, becomes imperative to devise and implement strategies which ensure the long-term survival of the species. This publication which is a collaborative attempt by the Wildlife Trust of India and globally acknowledged elephant experts, is meant to serve as a guide for veterinarians, biologists and forest personnel engaged in the conservation of the megaherbivore.

Although we have cited cases from different parts of the country on elephant poisonings, there is no substantial evidence of the kinds of poisons used to carry out the heinous act. This publication deals in detail with various types of poisons used and is meant to serve as a ready-reckoner for detection of toxin used to exterminate the pachyderm. This report will help serve as a guiding tool for detection of such cases in future. We hope that this collective effort would help reduce the mortality rate due to poisoning in pachyderms and help in the conservation of the species.

**Vivek Menon**
Executive Director
Wildlife Trust of India

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**Dr Jacob Cheeran**

# INTRODUCTION

## What is the need for this guide?

Due to a rapid increase in human population, the elephant's habitat has seen a significant reduction in recent years. Wild elephant populations are now mostly small and isolated with their migratory routes having been cut off by human settlements. The expansion of this interface has increased the intolerance among local people living within and at the peripheries of elephant habitats, towards the animal. As a result of this growing conflict between humans and elephants, several cases of elephant poisoning are being reported from many parts of the country.

To combat this, the Wildlife Trust of India has come forward with the novel idea of providing a field guide for foresters and enforcement officers, veterinarians, laboratory technicians and toxicologists to help identify the exact poison used and suggest methods to prevent mortality of the animal.

## The objectives of the guide are to provide:

1. Analytical data on the elephants that have been poisoned in the past five years in India.
2. A basis for the identification of the source of poisoning.
3. Guidelines to forest staff, biologists and veterinarians to help identify and differentiate cases of poisoning by looking at the clinical symptoms.
4. Guidelines for the clinical management of conditions that arise from the exposure to poison, in an elephant.
5. Guidelines to the forest staff and veterinarians in taking necessary steps on seeing a dead elephant.
6. Information on steps to be taken to prevent poisoning.

# HOW TO TACKLE ELEPHANT POISONING CASES

On finding an ailing elephant in the wild, distinguish the incident as a potential case of poisoning from other causes such as gunshot injuries, electrocution, accidental falls, infighting, vehicular accidents, disease or other stressor(s).

**Collect case history. Proceed further in the investigation by:**

1. securing the area if necessary,
2. recording clinical signs of the elephant(s),
3. recording any changes in the immediate vicinity of the animal, including weather, chemical use, natural chemical exposures, illnesses and deaths in other biota,
4. conducting preliminary interviews in the neighbouring villages.

**Dead Animal**

1. Examine carcass & continue to collect history.
2. Collect circumstantial evidence.
3. Conduct post-mortem examination.
4. Collect samples for laboratory analysis.
5. Develop differential diagnosis list if suspected poisoning.
6. Develop a shortlist of possible poison based on probability of exposure and consistency of effects.

**Live Animal**

1. Approach apparently poisoned elephant & administer supportive and detoxification treatment immediately as warranted.
2. Look for circumstantial evidence.

Confirm the cause of disease using chemical analysis and identifying possible sources of the poison.

Forest Department to comb the area in search of other elephants suffering from similar poisoning. All enforcement agencies to address the source of poisoning, whether intentional, cumulative or accidental.

Vets, Forest Department & other administrative & enforcement agencies determine & implement strategies to prevent future cases of poisoning.

# ELEPHANT POISONING IN INDIA

**Elephant deaths caused by poisoning since 1997 in different states of India**

| STATE | YEAR | CASES |
|---|---|---|
| Arunachal Pradesh | 1997-2005 | 2 |
| Assam | 1997-2005 | 29 |
| Jharkhand | 2002-2003 | 2 |
| Karnataka | 1997-2005 | 2 |
| Kerala | 1997-2003 | 2 |
| Meghalaya | 1997-2005 | 1 |
| Orissa | 1997-2003 | 3 |
| Tamil Nadu | 1997-2005 | 1 |
| Uttar Pradesh | 1997-2004 | 2 |
| Uttaranchal | 2001-2004 | 2 |
| West Bengal | 1997-2004 | 3 |
| **TOTAL** | | **49** |

Source: WTI elephant mortality database

# TOXICOLOGY AND TOXICANTS

**Toxicology** is the study of the adverse effects of chemicals on living organisms. It is the study of symptoms, mechanisms, treatments and detection of poison. **Poisons** are substances that can cause damage, illness or death to living organisms, usually by a chemical reaction or other activity on a molecular scale when sufficient quantity is absorbed by an organism. Poisonous substances that are a specific product of the metabolic activities of a living organism *eg.* plant, animal, fungus, bacteria, etc. are known as biotoxins. **Toxicosis** is a condition resulting from poisoning. Poisoning may also be due to some malicious intent. Hence, in accidental poisoning, where intentions are unknown, some authors prefer to use the term toxicosis. Any substance, be it biological or chemical in origin when administered or imbibed in excess becomes a poison. **Tolerance** is the ability of an organism to withstand the toxic effects of a substance on repeated exposure. Tolerance may be acquired or innate. Acquired tolerance is mostly due to repeated exposures. Innate tolerance is largely due to genetic selection. An example of innate tolerance is the resistance of rodents to high doses of atropine and of birds to strychnine.

Toxicology has developed as a parallel discipline with other biological streams; such as food, pharmaceutical, industrial, forensic, environmental, ecological, medical and veterinary sciences. The production of food additives, including flavours, preservatives, colouring agents, and organoleptic compounds has made it necessary for toxicology to become relevant in food science especially as these compounds are mass-produced, to provide a longer shelf life, thereby, increasing consumer appeal. **Forensic toxicology** deals with the legal aspects of poisoning. The development of plant protection chemicals in agriculture, chemicals used in post-harvest technology as well as detergents, adhesives, paints, cleansers, bleaches and solvents; chemicals used in industry and effluents from the industrial production units have contributed to the development of **environmental toxicology**. **Clinical** (medical and veterinary) **toxicology** deals with poisoning, its causes, diagnosis, treatment and clinical management.

## Effects of toxicants

The action of any toxicant is expressed as:

1) **Acute toxicity:** Refers to the effect produced in the first 24 hours after exposure.
2) **Chronic toxicity:** Refers to effects produced on prolonged exposure for over a period of three months or more.

Other expressions used are sub-acute or sub-chronic toxicity.

When two poisons act by a similar mechanism on the same organ, their combined effect can

be additive (2+2 = 4) or synergistic (2+2 > 4). There are instances where two toxicants may act antagonistically with one agent mitigating the ill effect of the other (2+2 < 4). This mechanism is made use of while treating a case of poisoning. A toxicant, once it enters the body, may accumulate if the body is unable to excrete it at a rate faster than the rate of absorption. This process is known as **bio-concentration**. Moreover, bio-magnification occurs in ecosystems, when different trophic levels pass an accumulative toxicant on to one another, potentially resulting in extremely high residues in animals at the top of long food chains. Some plants absorb toxic chemicals and are termed as accumulators.

## Metabolism of poisons

A poison may be absorbed through mucous membranes, including those of the gastro-intestinal tract, skin (dermal), lungs (pulmonary), eyes, as also through injections. The rates and extent of absorption depend mostly on the solubility of the poison. Lipid soluble substances are readily absorbed through the intact skin. Water-soluble compounds are excreted in urine via the kidney while fat-soluble compounds are excreted through bile and are also deposited as fat. It may also be noted that the target organ may not be the place with the highest concentration.

Metabolism of toxicants generally leads to detoxification through reduced concentration, but in some instances, compounds are metabolically converted to compounds more toxic than the parent molecules. Organophosphorus insecticides like malathion and parathion are examples of such poisons. Malathion becomes malaoxon and parathion becomes paraoxon, both of which are highly toxic. Many toxicants are metabolised in two phases. Phase I often involves oxidation, reduction, or hydrolysis of the compound while phase II involves conjugation or synthesis. Such products are most often eliminated by the liver via bile. Some conjugates undergo microbial deconjugation in the lower digestive tract and are reabsorbed as an active compound. Such a process is termed **enterohepatic recycling**.

## Elimination and biotransformation

Toxicants that enter the body may accumulate, be metabolised, and/or be eliminated. The time required for the disappearance of half of the compound from the body is referred to as half-life ($t_{1/2}$) and is specific for each compound and to each species of animal. Individual variation related to strain, sex, age, physiological condition, hydration and nutritional status can also influence half-lives of different xenobiotics. Elimination depends on the concentration of the compound in the blood (the usual case). A constant fraction gets eliminated per unit time (first-order kinetics). However, in some cases, elimination pathways are saturated, and thus a constant amount is eliminated per unit time (zero order kinetics). Different components of the body like blood, muscle, and fat may have variable different elimination rates. Many chemicals are rapidly eliminated from the blood while they may be

very gradually eliminated from tissues like adipose tissue, kidney or liver. It is important, therefore, to understand the rates of uptake and elimination of toxicologically important xenobiotics.

## Expressing toxicity

The quantitative expression traditionally used as a measure of relative toxicity, is the **$LD_{50}$**. It is the dose predicted to be lethal to 50% of the animals when used as a test sample in a population. Because of concerns about the suffering of animals in $LD_{50}$ studies, the use of the Minimum Lethal Dose (**MLD**) has become more commonplace and may eventually replace the $LD_{50}$, even though the latter provides greater statistical confidence. $LD_{50}$, when expressed, also carries information about the route of administration and the species of animal for which it is relevant. When $LD_{50}$ is calculated for mammals, the rat is often taken as the experimental animal. The route of administration may be oral (by mouth), dermal (skin), or by inhalation. Often, oral $LD_{50}$ is expressed unless otherwise specified. Depending on the level of toxicity, $LD_{50}$ is classified as follows:

| | |
|---|---|
| 1. Extremely toxic | < 1 mg/kg. |
| 2. Highly toxic | 1 - 50 mg/kg. |
| 3. Moderately toxic | 50 -500 mg/kg. |
| 4. Slightly toxic | 0.5 - 5 g/kg. |
| 5. Practically non toxic | 5 -15 g/kg |
| 6. Relatively harmless | > 15 g/kg |

Other expressions used, are No Effect Level (**NEL**), Maximum Non-toxic Dose (**MNTD**), Maximum Tolerated Dose (**MTD**), Approximate Lethal Dose (**ALD**), Accepted Daily Intake (**ADI**) and Permissible Level (**PL**).

The term $LC_{50}$ (the concentration predicted to kill 50% of the test animal population) can be applied to drinking water, diets, or air. With aquatic animals, the expression $LC_{50}$ generally pertains to concentrations in the water.

**Ecology** is the study of all interactions among animals and their environments. Toxicology is the study of all the chemically-mediated harmful effects of all chemicals on all life forms. **Ecotoxicology** is the study of the direct and indirect adverse effects on all life forms and on their interactions with one another and with the environment. Long food chains involving plants (producers), herbivores and a series of predatory animals (consumers) typically results in the the highest residues of metabolically recalcitrant xenobiotic compounds in animal tissues.

## Toxicity and risk

The terms risk and toxicity are often confused and wrongly used synonymously. **Toxicity** indicates the amount of a toxicant necessary to produce a detrimental effect, while **risk** refers to not only toxicity but also the likelihood of poisoning under the conditions of use, disposal, and fate in the environment. Toxicity depends upon the dose, species, route of exposure (oral, dermal, inhalation, and parentral) duration of exposure, nature of the chemical, etc. The example in the table below exemplifies the distinction between these terms.

| Compound | $LD_{50}$ (mg/kg) | Toxicity | Dose (kg/ha or ml/lt) | Risk Ratio |
|---|---|---|---|---|
| X | 300 | Low | 100.00 | 3:1 |
| Y | 20 | High | 1.00 | 20:1 |

A lower $LD_{50}$ will indicate high toxicity. Risk takes into account not only its low $LD_{50}$ but also its form, quantity of intake, probability of intake and duration of intake.

Many pesticides are not water-soluble; therefore, organic solvents are added for effective administration. This commonly increases the risk, because of the added toxicity contributed by the solvent and an enhanced rate of absorption of the pesticides.

Often synergists are added to have a better killing effect on insects. A typical example is piperonyl butoxide, which is added to pyrethrins to enhance their insecticidal property. While some insecticides are resin-impregnated, others are sustained release preparations. These may hasten or delay absorption of the active ingredient. A delayed absorption often decreases toxicity as it gives ample time for the body to handle the poison and considerable amounts may be excreted in the feces without even being absorbed. Flavouring agents are often added to bait to encourage the animal to consume them more readily.

Most insecticides used in agriculture and forestry are sprayed as emulsions. A fine emulsion acts over a large surface area, which increases risks of adverse effects. Some commercial pesticides contain toxic quantities of impurities, like halogenated dioxins, which are formed due to high temperature conditions during manufacturing processes. The 2,3,7,8-isomer of tetrachlorodibenzodioxin (TCDD) is one of the most poisonous of the 70 odd chlorinated dioxins known. This contaminant is especially of concern in the herbicide 2,4,5-T.

The nutritional status of the animal is another factor that contributes to toxicity. Malnutrition reduces the glycogen level of the liver and reduces its capacity for bio-transformation. Protein deficiency over a long period results in inefficient microsomal activity. Pesticide toxicity is augmented by these conditions. Pregnancy and lactation adds to increased stress levels in the animal. Substances like anticoagulants are more toxic to pregnant

animals. In some cases, such as with methyl mercury and polychlorinated biphenyls (PCBs), the young are readily exposed *in utero* and are highly susceptible to effects, such as impaired cognitive abilities.

The body weight of the animal often contributes to toxicity as well. Very large animals naturally require large doses of poisons to produce toxic effects. Elephants consume large quantities of food and water and hence the large body size does not spare them in this regard. The metabolic rate of large animals is relatively lower than that of smaller species. As a result, the larger animal may not be able to detoxify or eliminate the xenobiotic as efficiently as a smaller animal. Hence, it has been seen that the lethal dose for a larger animal may be lower than that required for a smaller animal on a mg/kg body weight basis.

# DIFFERENT TYPES OF POISONING

**ACCIDENTAL, INTENTIONAL AND CUMULATIVE POISONING**

## Accidental poisoning

As the name indicates, this type of poisoning is purely accidental and the victims are non-target animals, which may include elephants. Accidental poisoning arises by accidental baiting of non-target organisms or through pollutants.

Baiting: Since elephants are large animals, accidental ingestion of poison meant for other small animals is unlikely to cause fatalities. But it may be noted that crackers used against vermin, like pigs, that are often bitten by young, inexperienced elephants, have caused severe injuries and sometimes death.

## Poisoning from pollutants happens due to:

1. Lack of safety measures,
2. Poor worker-protection standards,
3. Ambiguous rules and regulations,
4. Non-compliance with rules and regulations,
5. Accidents during transportation,
6. Indiscriminate use of pollutants,
7. Cumulative effect of pollutants,
8. Insufficient or improper storage facilities,
9. Residues in fodder and
10. Foraging on crops sprayed with pesticides.

## Intentional poisoning

These cases are with malicious intent and occur largely due to conflicts that arise between man and elephants.

Ordinarily, intentional poisoning, as a result of human-elephant conflict, is common in fringe areas of sanctuaries and reserve forests. Elephants raid crops depending upon the type and palatability of crop. They often go to another range once the crop is harvested. Barriers in the form of elephant proof-trenching and live-wire fencing are usually not effective in preventing depredation. Other than destroying the crops of these farmers, marauding elephants also damage property and even injure or kill people. Elephants are usually poached for tusks and in some places for meat also. The hide is used as leather and the foot made into foot-stools or baskets.

## Methods of poisoning

Frustrated farmers attempting to rid themselves of these pests often make use of normally available poisons, which are commonly pesticides. Most of the pesticides have a special odour especially due to solvents and adjuvants. The offending odour is masked by using palatable feeds such as fruits and molasses as bait. Often pineapple, watermelon, jackfruit and other large fruits and vegetables are used to conceal granular insecticides (eg. Furadan Ò).

Taking advantage of the elephants' preference for salts, urea mixed with molasses or any other sweetening agent is used for poisoning. Elephants being simple stomached animals, suffer from ammonia toxicity following consumption of non-protein nitrogen (NPN). Ruminants classically experience ammonia toxicosis from NPN, whereas monogastrics show better tolerance. Posterior fermenters also experience ammonia toxicosis but generally with higher doses than in ruminants.

Elephants frequently partake of illicit brew that is used to produce locally distilled spirit. Perhaps because of its sweet and sour taste, elephants seem to like it. The olfactory senses of elephants are well developed and thus the animal is attracted to the brew by its strong, inviting smell. Also, habituation of elephants to the feeling of euphoria - a state of well-being – may add to the vulnerability of elephants for malicious baiting using this. Persons engaged in this "business" often try to poison these marauding elephants by adding poisonous substances to the brew. Arrow poisoning using metallic arrow tips has recently started surfacing in certain sanctuaries. Arrow poisons are generally muscle relaxants. Since tribals essentially develop arrow poisons to hunt animals for consumption, the poisons they use are not absorbed when ingested.

Experience indicates that, if the death of an elephant is reported from an area where the problem of human-elephant conflict exists, the possibility of malicious poisoning cannot be ruled out.

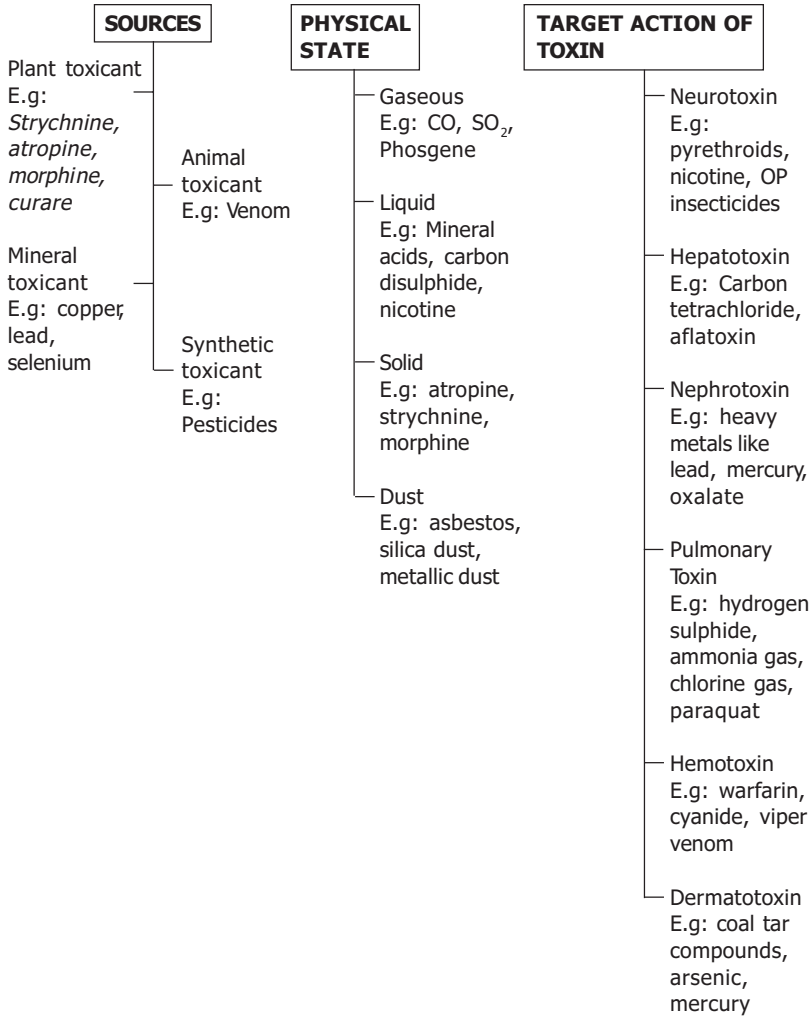
## Cumulative poisoning

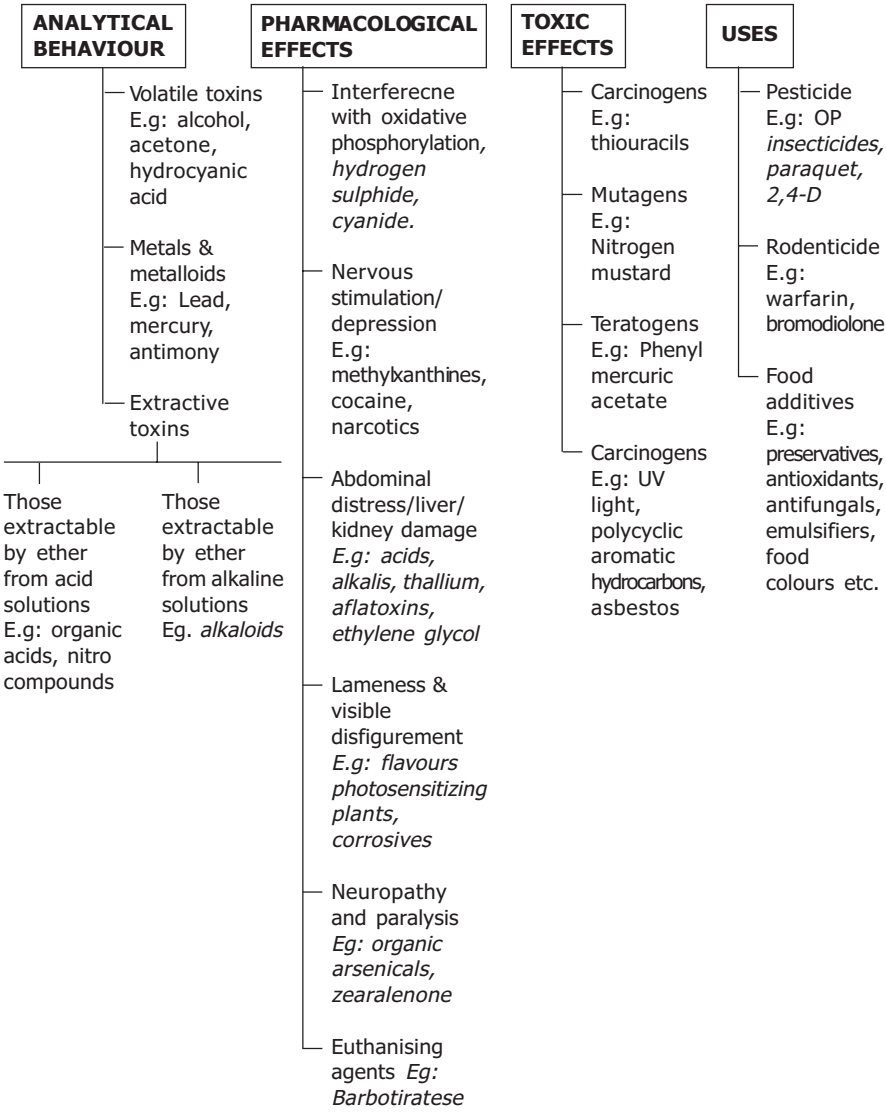
A poison or any other xenobiotic (foreign) compound is said to be cumulative when the rate of absorption of the poison exceeds the ability of the body to metabolise or excrete it. Continued exposure and inclusion either by ingestion, inhalation or through the skin will result in an incremental increase in the levels of the poison in the body. This is also referred to as **bio-accumulation.**

Examples of cumulative poisons are those of lead, mercury, arsenic and insecticides containing organochlorine. Of these, methyl mercury and many of the organochlorine containing insecticides are prone to **bio-magnification**.

As elephants are slotted low on the food chain (herbivores), cumulative poisoning may occur.

# CLASSIFICATION BASED ON CHARACTERISTICS OF THE TOXICANT






Inorganic poisons include mainly metals, metalloids, halogens, nitrates & other inorganic salts

## Cumulative injury

A toxicant can cause cumulative injury. Whenever concentrations reach toxic levels, the effects are often irreversible. A neurotoxicant similar to caffeine does not cause cumulative injury.

# PROCEDURAL APPROACH

## Suspecting poisoning

In the author's experience, many cases of poisonings in elephants are accidental but instances of deliberate or criminal poisonings cannot be ruled out. If there is a suspicion of criminal poisoning, specimens should be collected in duplicates and placed in sealed containers in the presence of a witness. A complete set of specimens should be available to both plaintiff and defending parties for independent analysis. The veterinarian should make detailed observation of the clinical, pathological and epidemiological findings and record them in detail. Photographs of the affected animals and the surroundings should be taken for future reference. In all legal cases, an accurately documented chain of custody of specimens and a thorough record of the investigation and related analyses and other diagnostic methods and results must be maintained.

The success of the toxicological examination depends on submission of the following:

Appropriate specimens.

A thorough historical account.

A thorough record of clinical signs where possible.

Attempted treatment and any response to treatment.

Necropsy findings and histopathological observations.

Analysis of appropriate specimens for the suspected poison or metabolic products derived from the poison.

Ruling out other non-infectious as well as infectious causes.

If a known poison is suspected, a specific analysis should be requested in those cases. Exceptions would include a toxicant which is no longer detectable or for which either no method of analysis is available or can be developed (e.g. lethally toxic plant for which the toxic compounds are unknown).

## Sources of poisoning

Natural sources of poison include:

(A) Plants: There are hundreds of toxic plants, but intoxication due to foraging is very rare as animals feed selectively except when there is scarcity of fodder or accidental consumption. Cyanide poisoning from plants is quite common.

(B) Animals: Envenomation by poisonous reptiles or insect stings can lead to poisoning.

(C) Minerals: Certain geographical areas that are rich in minerals like arsenic, fluorine, mercury, selenium, molybdenum and can be sources of poisoning.

Man-made sources are often results of industrialisation and other reasons arising out of modern living methods:

(A) Industrial toxicants: These include solid wastes, gases, vapours, and suspended particles e.g. chromium, nickel, lead, chlorine, sulphur dioxide, cyanide, and caustic compounds such as sodium or potassium hydroxide that can produce toxic conditions.

(B) Agrochemicals: These are sprayed in extensive areas like plantations and pose fatal risks to animals. Rubber plantations when sprayed with copper fungicides may contaminate cover crops, which are often legumes and good forage material.

(C) Domestic materials: Lead paints, phenolic disinfectants, solvents, and refrigerants (radiator coolants contain glycols and drained coolant which when ingested can cause toxicity) are common sources of poisoning. A recent problem of environmental pollution by plastics can cause mechanical obstruction of the GI tract and can even lead to the death of the animal.

## Diagnosis and treatment

### Diagnosis

While diagnosing a case of possible poisoning, an effort should be made to compare the known toxic potential of the offending element or compound with the:

1. History and temporal progression of events
2. Clinical signs
3. Local evidence of exposure and environmental evidence of toxic manifestations
4. Postmortem and histopathological findings
5. Clinical pathological effects.
6. Analytical evidence of exposure or toxic residues
7. Experimental evidence of target organ toxicity and potency
8. Response to treatment.

## History

History is the most important clue, that helps to pinpoint the cause of poisoning. In case of captive elephants, it is important to question the owners and mahouts thoroughly, as they are likely to forget or omit certain subtle but important points in the history. In case of a wild elephant the following may be noted:

Whether the animal was feeding in a new area or was raiding on a cultivated crop or in an area that has experienced human habitation in the recent past.

Any chance of sprays or baits for a non-target animal must be included. In cases of malicious or intentional poisoning, there will be attempts to conceal the evidence.

What is the period of possible exposure to toxicants in the region (both natural and manmade)?

What is the level of assurance of exposure to toxicants in the region?

The period of observation before and after the onset of abnormal symptoms in the animal

When did all the animals last appear to be normal?

The size of the herd from which the individual originated.

Whether an individual, more than one or the entire herd was affected.

If there were more than one affected animals, how many of them showed clinical signs? How many were ailing or dead? Were some animals still lingering? When was the first sick animal or carcass noticed?

Did the problem persist in the herd or in that area?

Were animals of different taxa affected?

If the animal is dead, when was it last observed alive and healthy?

Was the death observed and if so, when did it occur and what preceded death?

Ascertain possible lapse between time of death and sighting of carcass.

Whether the animal had shown any signs of struggling/purging/frothy discharge from the mouth.

## Clinical signs

If there is any chance to see the animal before its death, it is important to note any clinical manifestations, such as hyper-reactivity, excitement, depression, discordant movements, ataxia, seizures, vomiting, blindness, pressing the head on stationary objects, arched back, urination, defecation, abnormal faecal matter, or abnormal colour of urine.

## Local evidence

Identification and inspection of the nearest waterhole is very important to detect the source and quality of the drinking water. It is important to check whether there is any chance of associating foraging or drinking to the signs of poisoning and subsequent deaths. Other symptoms like ataxia, excitation, salivation, and seizures are a bad prognosis. Elephants rarely vomit like carnivores but they regurgitate more often.

### In addition to these, the following need to be checked

Specific environmental conditions such as evidence of pollution

Evidence of exposure to toxic plants

Evidence of exposure to dump sites, industrial wastes

Evidence of exposure to pesticides

Presence of snakes

Chances of consuming bait

The smell of the left over fodder or the contents of the mouth

If there are flies feeding on the vomitus or ingesta, they may be trapped by covering the contents with a basket and wait for a while to see if they die. This may be done with the stomach contents also. This method will give a clue in case of poisoning with insecticides.

## Postmortem findings

Evidence is collected by performing necropsies and by collecting appropriate samples. Gross lesions are sometimes absent in cases of lethal toxicoses. The presence or absence of lesions can be important in diagnoses, and should be recorded. Record as many details as possible.

*External signs*: The yellowish colouration of the skin in phosphorus and acute copper poisoning is not easy to discern in elephants. A cherry coloured upper palate is an indication of cyanide or carbon monoxide poisoning. Red colour can also indicate poisoning due to nitrates or nitrites. Corrosive lesions on the skin, mouth and lips could be a result of poisoning with strong acids or alkalis. Electrocution gives a burnt appearance to the skin especially on the trunk, with which the animal could have touched the live wire.

*Internal lesions:* Lesions vary according to the toxicant. Appearance of lesions may be lacking in lethally poisoned animals. Lesions are more commonly seen simultaneously with changes consistent with gastroenteritis, fatty liver, necrosis of liver, lesions on renal cortex, and pulmonary edema. Contents in the stomach and esophagus like suspected plant materials, foreign objects and remnants of poisons may give some indication as to the cause of poisoning. Lesions of the liver may indicate that the toxicant could be a poison that localizes or is bioactivated in the liver. Lesions in the kidneys often signal the presence of agents that are excreted by and concentrated in the kidneys. Appropriate organs and tissues should be collected in the prescribed manner for further analysis.

## Analytical evidence

Chemical analysis of the toxicants will be a major pointer; however it should not be the sole criteria for pinpointing the cause of death.

Some of the poisons may hydrolyse, decompose or volatilize from the body after a period of time. At times the toxicants may be seen only in the tissues. Quite often even analytical procedure may not give a clear indication as to the cause of death.

## Experimental evidence

Feeding of suspected poisonous food or water to lab animals like rats or mice can be helpful. Often an extract is also used, when water is suspected to be the source of the poisoning. The clinical manifestations exhibited by the experimental animals can be closely monitored, but reproduction of the same in field conditions is often not possible. Sometimes the toxicant is known to affect certain species only and collecting experimental evidence becomes difficult *e.g.* rodents are resistant to *Datura* poisoning.

## Response to treatment

Response to specific antidotes can give a good indication of what the poison could have been. Animals poisoned by an organophosphorous or carbamate insecticide show favourable response to atropine. However, opportunities to treat poisoned wild elephants are rare.

## Treatment

The basic principle is to prevent further absorption of the poison, i.e., change the source of food and water. Washing the skin and other such measures are not possible with wild elephants. Captive elephants may be washed down with detergent and water in many cases. Supportive therapy like control of the seizure, maintenance of respiration and the treatment of shock are also not possible in a wild herd. Spasms can be controlled by darting xylazine, and atropine also can be administered by darting in OP poisoning.

## Sample collection from dead animals for verification of poison

Because of autolysis and toxicant hydrolysis, evaporation and other losses, it is generally best to examine any dead animal before examining the animal's surroundings. In cases where the animal or its internal organs may be exposed, it is possible for such investigations to be hazardous to the investigator. Contamination of clothing and sampling equipment could also happen. It is necessary therefore, to thoroughly clean the equipment and change clothes before moving beyond the animal to survey the environmental sources of exposure.

Samples should be collected from the animal as well as from sources available in the immediate vicinity/neighbourhood

In cases where the animal is still alive, the following materials may be collected for analysis;

substances in the oral cavity

regurgitated mass

blood

urine

faeces

water

feed

In the case of dead animals, besides the abovementioned biospecimens, the viscera of the animal must also be collected and preserved in 10% buffered neutral formalin for histopathological examination. The visceral parts which may be collected include:

part of the stomach with contents

part of the intestine with its contents

liver

kidney, lung, heart and brain tissues

Brain (special equipment will be needed for removal from an elephant). If the cranium cannot be opened, specimens may need to be removed via the foramen magnum.

A specimen of the gut content is necessary so that ingested material can be analysed. A sample of tissue can be used (usually liver), to prove that absorption has occurred. Liver and kidney are often sites of concentration and an analysis of the tissue from any of these organs can support the findings.

Brain tissues are used for such assays to detect the presence of cholinesterase and residues of organochlorine insecticides.

Retinal tissue may be assayed for cholinesterase as well (submit intact eye).

If an animal has been exhumed and the viscera missing, soil under the digestive tract and liver could still be preserved and examined to identify the presence of toxic elements or biologically recalcitrant toxicants (such as organochlorines).

Uterus and foetus would be useful in suspected cases of abortion.

Burnt bones and ashes of cremated bodies should be preserved for analysis. The skeleton or the remnant bones are important material for analysis in the case of exhumed bodies.

Apart from the collection of tissues for toxicology, fixed specimens of tissues of all major organs (in 10% neutral buffered formalin) should be preserved and submitted for comprehensive histopathologic examination.

## Samples and sampling

**Dry sample:** Feed, fodder and other materials suspected to be contaminated by the poison should be sampled only after conducting a summary of inquiry into the circumstances that caused the poisoning. In case of animals kept in captivity, samples of the remains found in the stall could provide some direct clues. Bottles, packets and some unusual materials also have to be examined and sampled if required. When fodder is sampled it is important that the materials on the ground found in a powdered form should also be included. Samples should be collected at random. When samples of feeds are taken, it should be ensured that representative samples of particles of all sizes are included. Careful examination of the feed is necessary so as to analyse all the unusual materials found in it. Colour, smell and appearance are also indicators of accurate sampling. Fodder should be opened and sorted through to acquire plants for identification. When possible, intact plants should be saved, as flowers, fruits, seeds, stems, roots are essential for taxonomic identification.

**Wet samples:** When wet samples are collected, ensure that a sample of country brewed liquor is also included. If the poison is water soluble, the aqueous portion will be the critical component. In case of insoluble materials, the sedimented portion should be collected. Sand particles of uniform size settled at the bottom can sometimes provide a preliminary indication of the presence of materials such as carbofuran (Furadan) and phorate (Thimet).

## Quantity

A suitable quantity of materials should be submitted for analysis; 1 kg of liver and proportionate amount of other viscera is suggested to cover all the contingencies. Careful sampling and packing of specimen is necessary to avoid loss of important material due to degeneration.

## Preservation for toxicologic analyses

Tissues and fluids for toxicological analysis should be as fresh as possible. Freezing and shipping on dry ice are the preferred methods of preservation. All specimens for analysis must be kept separately to avoid cross contamination between specimens. Where freezing is not feasible, packing with ice is preferred.

Adequate refrigeration is critically important when submitting body fluids and materials for nitrate analysis, as these salts are rapidly metabolised by microorganisms leaving insignificant levels for analysis. Refrigeration prevents microbial growth and helps ensure that the salts are preserved.

No preservative should be added to the samples except in the case of suspected nitrate poisoning and only when freezing or adequate refrigeration facilities are not available. Chemical preservation of materials is done in 50% ethanol (1 ml/g/tissue). The tissues should be placed in a container and submerged in sufficient alcohol.

Alternatively, tissues may also be preserved in saturated sodium chloride. Preservation using solid common salt could also be done. The tissues should be placed in a container, with sufficient quantity of common salt covering them completely. A sample of the saturated solution of common salt used for preservation should also be collected in separate glass bottles for analysis to exclude the presence of any poison in it. **Note:** Immersion of the tissues in either ethanol or salt solution will reduce the accuracy of quantitative assessment of tissue residues and will increase the possibility of hydrolysis of some unstable compounds.

## Blood is preserved using

1. Sodium fluoride 20 mg/ml of blood
2. Sodium citrate solution containing 10 g of sodium citrate and 200 mg of mercuric chloride dissolved in 100 ml of distilled water. One drop of this solution is sufficient for each ml of blood.

The appropriate volume of fluid prepared as described should be kept in a clean, dry glass bottle. The fluid on drying sticks to the bottle. These bottles are then cooled at room temperature and the samples preserved *in situ*.

## Containers and packing

The specimen should be packed in glass or plastic to prevent contamination by lead present in soldered joints of the cans. When possible, specially cleaned jars with Teflon-coated lids should be used for sample collection. If not possible then the specimens for metals analysis should be placed in plastic bags prior to freezing, refrigeration, or chemical fixation. Tissues to be frozen for organic analysis should be carefully wrapped in aluminium foil, placed in a plastic bag and then frozen or refrigerated.

Metal tops on jars should also be separated from the tissue by a layer of plastic or other impervious material. Specimens should be packed individually. The most appropriate containers for visceral tissue collection are wide-mouthed glass bottles of about two litres capacity equipped with airtight stoppers. These bottles should be numbered and labelled properly. The labels should include the name of the species or the serial number, tissue type, date and time of collection before shipping to the laboratory. A separate laboratory specimen form mentioning the details of the case, nature of the contents preserved, place and date of

preservation, preservatives used and duly signed by the veterinarian should accompany the container. An accompanying detailed post-mortem report with the tentative diagnosis would be of immense help.

## Analytical errors and contaminations

There is always a possibility of contamination and analytical errors during the procedures. The following measures are recommended to minimise them.

**Blood samples:** Plated needles or soldered syringes that can add relatively large amounts of Cu, Cd, Zn, Fe, or other elements should not be used. In case of live animals if the sample is to be analyzed for potassium, any contraction or tourniquet to raise the veins could raise the serum potassium levels up to 17%.

**Tissue samples:** These should be collected using clean stainless steel knives. For microdeterminations a glass microtome blade or Teflon-coated blade should be used. Tissues or organs meant for metal analysis should not be placed in aluminium, stainless steel, plated steel or galvanized trays.

**Dry samples:** When using hot-air ovens, perforated metal shelves should be checked for any corrosion. Airborne contaminants, particularly those containing calcium, copper, iron, aluminium and zinc are often present in laboratory atmosphere.

**Grinding:** This should be carried out using a pestle and mortar or in a blender with liquid nitrogen.

**Dry ashing:** This may cause metal loss through volatilisation, especially if a local hot-spot develops due to self-combustion. Carbon residues can alter results. Dry ashing can yield insoluble metal silicates that could result in low recoveries during subsequent processing.

**Wet ashing:** By using nitric, perchloric and sulphuric acids, losses can be minimised as this is done at lower temperatures. However As, Sb, Hg, Sn and Cr may volatilise unless appropriate precautions are taken. Appreciable blank levels may arise through the use of large amounts of acid.

**Solution storage:** Polythene bottles are considered superior to the glass ones for storing reagents.

**Filtration:** This could lead to contamination, so centrifugation is preferred.

**Glassware:** Try to use separate glassware for each type of analysis.

**Spoilage:** Samples should not be left overnight without refrigerating or deep freezing. Repeated freezing and thawing is known to denature proteins or proteinaceous toxins which may be the analytes of interest. Protein degradates from tissues could also interfere with analysis.

## Supportive evidence collection

**Crime scene search**

If a wildlife crime has been committed, an important starting point for investigations is searching for evidence at the scene of the crime. For this, one should follow a certain regime for evidence collection and subsequent documentation of the same. The following should be done:

1. Record the exact time of arrival of the investigating team on the scene.
2. Check for survivors and render appropriate first aid.
3. Check to see if authorities have been notified.
4. Document weather and description of the area
5. Document unusual behaviour of animals (the victim and others) in the vicinity.
6. Pay attention to the wind direction and velocity and its particulate concentration.
7. Pay attention to the existence of unusual odours such as sweet, almond, rotten egg, sulphur, etc.
8. Isolate the scene with a rope or tape.
9. Isolate witnesses and remove other onlookers from the immediate area.
10. Record names, addresses, telephone numbers etc. of the people present.
11. Interview witnesses.
12. Keep written notes and/or audio recordings.
13. Take as many photos and video recordings as possible.
14. Sketch area and positioning of the victim.
15. Search for evidence.
16. Take footprints of animals and humans in the area.
17. Collect evidence.
18. Search for wounds or marks of assault on the animal.
19. If death has occurred in a water body, do not wade through the water collecting carcasses without proper protection. The water may be the source of the toxicant which could be absorbed through intact or broken skin or the mucous membranes. Ideally, a mask and gloves should be worn while searching for evidence.

## How to search for evidence

Trained personnel should be deployed to collect evidence. One of the methods mentioned below may be used as a suitable one.

1. **Straight line:** Start searching point to point in a straight line. Several parallel lines can be made and the team of members be asked to walk on them.
2. **Quadrants:** Divide the area into quadrants. Search one quandrant at a time. It is useful if a number of teams are available for collection of evidence in a large area.
3. **Spiral manner:** Cover the area in a spiral manner (starting from inside out or vice versa). This is useful if there are limited people on the scene and the area is flat and wide open and one is looking for large objects. It is best to start with the areas which are least likely to be involved and then go on to examine the areas most likely to be involved. In this way, all the areas where the animals, their food, or their water may have been exposed can be checked out.
4. Send in the team members from point to point and then back again.
5. **Sector (Wheel/pie):** Send the team members in a radial manner from the centre to the circumference of the circular area. It is useful when the scene is large and one has to collect different kinds of evidence spread all over the place.

## Collection of evidence from crime scene

Collection of evidence from the scene of crime has to be done in a systematic, scientific and precise manner in order for the evidence to have some significance in the final investigation. If the evidence collection is done correctly, it stands a greater chance of being admitted in the event of prosecution. The following evidence is commonly available at the poaching site.

## Blood

1. **Liquid blood**

(i) Collect blood from animal's body in a test tube using a dropper. Seal it tightly with a stopper.

(ii) If a pool of blood has formed samples should be picked up on a gauze pad or other clean, sterile pieces of cloth and refrigerated or frozen immediately. When freezing or refrigeration of the sample is not possible, the same should be air-dried in a clean dish at room temperature and transported to the laboratory quickly.

(iii) Delays beyond 48 hours could greatly reduce the value of the sample for toxicological tests or render it useless.

2. **Dry blood**

(i) If blood is found on the soil, samples should be collected along with the soil in a test tube and sealed. Additional soil without blood should also be collected.

(ii) If blood smears are found on small solid objects, the whole stained object should be collected and sent to the laboratory.

(iii) If dried blood is found on large solid objects, cover the stained area with a clean paper and seal the edges down with tape to prevent loss or contamination. If the sample is too big to be delivered to the laboratory, scrape the stained portion on to a clean piece of paper which could be folded and sealed in an envelope.

## Footprints/pugmarks

1. Photograph prints before taking impression.
2. Cover the prints with glass. Sketch from above.
3. The prints should be viewed from 90º to avoid parallax error.
4. Fill print with plaster of Paris. Allow it to dry and collect the cast.

## Hair

1. Use forceps to collect hair for evidence.
2. Do not bend hair while collecting.
3. Collect whole hair samples.
4. Place samples in a plastic bag (not paper envelope), seal and label it.

## Firearms

1. Record the serial number, make, model and caliber of the weapon. Marking firearms is important since duplicate serial numbers are sometimes found on different guns of the same make.
2. Use gloves to pick up the firearms and note whether they have been fired or not.
3. Do not clean the bore, chamber or cylinder prior to submitting the firearm to the forensic laboratory.
4. Do not insert any object into the barrel of the gun.
5. Note position of lock, hammer and catch (if present on the firearm) and do not tamper with it.

## Fingerprint

1. Photograph fingerprints before lifting them.
2. If fingerprint tape is available use it to collect print.

## Bullet

1. Take out bullet from animal's body wi thout scratching it. Cutting around the bullet and submitting it with some tissue still intact around it could reduce the risk of scratches. Place it in a plastic bag, seal and label it.
2. Cartridge cases found outside should also be collected in separate plastic bags, sealed and labelled.
3. In case a bullet is embedded in a hard object (eg. tree), cut around the bullet and place the whole block in a plastic bag. Seal and label it.

## Things that can go wrong

1. Failure to recognise the evidence.
2. Presence of too many by-standers and improper cordoning of the scene of the crime.
3. Too may officers adding to contamination of evidence.
4. Using hit-or-miss type search methods.
5. Lack of organization and communication among the search team members.
6. Failure to search outside the immediate crime scene.
7. Failure to search area for witnesses.
8. Failure to take proper notes, photographs or video recordings.
9. Contaminating the evidence by improper handling.
10. Placing wet/stained items in plastic bag before sufficient drying.
11. Packing more than one item in the same packet.
12. Improper collection of fingerprints.
13. Jumping to conclusions and making the scene fit theories.
14. Failure to restrict information.
15. Delay in sending the samples to the nearest laboratory.
16. Failure to interview all witnesses in a timely and thorough manner.

## Conducting a criminal investigation

Once the evidence is collected, the next step is to conduct an investigation. For an investigation to begin, a crime need not have been committed and this could also be the beginning of an effort to mislead the effort. The investigative process can be divided into five phases.

1. **Intelligence gathering**

   Try to identify the suspects.

   Through various, preferably redundant sources, try to determine the activity regime of the suspects.

   Document and validate until all the relevant information has been confirmed.

   Analyse the submitted intelligence reports (including plausibility, sequential factors, confirmations, inconsistencies, and apparent knowledge gaps.

2. **Decision to conduct the investigation**

   The final decision to conduct the investigation must be taken after the background reports have been analysed. At this stage, one could close the case if the background information is contradictory or inadequate.

3. **Planning the investigation**

   An investigation can only be carried out if there is sufficient manpower.

   Adequate and trained supervisory staff.

   Coordinator of the investigation who would also serve as the "team leader"

   How many investigators/officers are available to provide for surveillance, evidence analysis, and technical support?

   The equipment available is adequate and well maintained

   There are adequate arrangements for storage of evidence

4. **Implementing the plan**

   A plan can only be implemented if

   A certain degree of flexibility exists while analysing the data.

   A continuous upgrading of information is done.

   Simultaneous identification of defendants, suspects and charges is done.

   Evidence is subjected to systematic and precise evaluation and analysis.

   Search and arrest warrants are obtained and executed.

   Interrogations are conducted and recorded in the presence of two independent witnesses.

   A draft of and the final written reports are made available.

   Evidence can be produced in court within 24 hours.

   Synopsis of the investigation is released to the media only after being duly considered and warranted.

   A lawyer is briefed after preparation and submission of evidence.

5. **Evaluate your results**
   Evaluate to see if the investigation would be a deterrent to future illegal activities and whether the case results could add to existing results for better management of crime.

## Approaching poisoned elephants for treatment

The treatment of wild animals especially elephants, has always been a real challenge. But with the advent of safe and modern immobilizing agents it has become easier.

Sick or injured wild elephants or those with maggot infested wounds may at times be found in water bodies such as ponds or reservoirs, attempting to avoid being disturbed by flies. Even when under the stress of adverse conditions such as painful wounds, arthritis and poisoning, elephants are normally found standing and not recumbent. Elephants are very reluctant to lie down when they are sick or feeling weak. They can sleep while standing. In one instance, a sick elephant stood for 18 months. Since getting up is not a task easily or quickly accomplished, elephants only lie down as a last resort and the animal invariably succumbs to the situation. The absence of a pleural cavity and the animal's massive size can cause postural congestion of the lungs during recumbency. Consequently, pneumonia-like condition may set in and at times lead to death.

The use of tranquilliser guns and kunkies (trained elephants used by the Forest Department for training other elephants or capturing rogue elephants) comes in handy when dealing with elephants suffering from poisoning in the wild. The use of kunkies is always advisable as it proves to be a safer way of coming close to the injured elephant. They can also physically help the immobilised animal to get up during recovery. However, it may be noted that kunkies may refuse to approach the animal being treated if it is in musth. Even otherwise, many captive elephants are reluctant to approach or even refuse to go close to a wild elephant.

Of course, elephants are animals with strong herd instincts and herd mates may not leave the immobilized animal. Hence, they may have to be driven away using appropriate methods like firecrackers, guns and kunkies. The herd mates must be kept at bay and the team should be aware that they may return at any time during the immobilization and treatment procedures. Furthermore, a strong maternal instinct also poses a problem when the animal in need of treatment is a cow elephant with a calf. Most often, both will have to be immobilized to handle either of them.

Darting an animal should be avoided after it has had a full drink of water. Darting should be avoided in the afternoons or near water holes. If etorphine is used for immobilisation, the nature of the terrain should be taken into consideration to prevent the animal from stumbling and falling.

## Treatment in lateral recumbency

The drug of choice in this situation is Immobilon L.A. (etorphine with acepromazine). The recommended dose is 2-mg/mt body weight, i.e., approximately 1 ml (2.25 mg/ml) is needed for a little over a ton of body weight. If the dart is properly lodged in the right place, usually on the rump or shoulder, the drug will act within a few minutes and the animal assumes recumbency. If any delay or insufficient sedation is noticed, it is possible that the injection might have gone subcutaneous or there is formation of haematoma at the site of the injection. The latter is not uncommon when using a detonating syringe for injection. If the animal falls in a sternal position it should be pulled over to a lateral position. If the animal remains in sternal recumbency for about 15 to 20 minutes, it is likely to die from asphyxia.

Once the animal is down, it should be left for a while to ensure that the animal is under deep sedation. Prodding the animal in the rear with a long stick or checking its response by gently tossing a few stones at the animal will help to ascertain the degree of sedation.

Once sedated to a safe level, the animal should be physically bound. The hind limbs should be tied to a tree or strong peg. The two hind limbs could also be tied together using a chain if available or with a rope using a figure of "8" knot. Thick ropes made from either locally available fibres or polypropylene could be used. An iron ring of 8 or 10 cm diameter should be attached to the tip of the rope for easy and quick noose formation, especially when the elephant is in standing position. The forelimbs should be similarly immobilised with a rope and tied to a tree or a peg. The trunk may also be noosed. However, it is important to ensure that the opening of the trunk remains free for the animal is able to breathe properly.

The safest place to stand near an elephant reclining in this position is at the dorsal side of the neck. The entire dorsal side of the animal's body is safe. All nooses should be tied in such a way that they can be cut or loosened easily at short notice.

Intravenous injections are given on the ear veins. Identify the vessels on the ear, i.e., vein or artery by feeling for the pulse. If the artery is punctured instead of the vein, the blood would gush into the I.V. bottle. The animal should be constantly watched for signs of recovery. These would include slight movements of the trunk, eyelids and earflaps. If more sedation is required, depending upon signs of recovery, approximately 1/3rd of the original dose can be administered.

In etorphine immobilization, the body temperature should always be checked. If it rises significantly, water should be poured on the body. A sprayer could be effectively used for this. Kunkies could also be used to pour water on the body of an elephant immobilized by etorphine (Immobilon LA). Kunkies should be made to stand parallel to the immobilised animal on either side (if the animal is in standing posture, depending on the area to be handled) and spray water.

## Treatment while in standing posture

If the animal is found to be in a standing position, it should be treated in that position. The best drug for immobilizing an elephant in the standing position is xylazine, administered at the rate of 100 - 120 mg/mt body weight. If the animal is excited and suffering from a painful condition, the required dose may be two or three times more than the normal dose. Any disturbance during induction would prolong the induction time. The peak effect is obtained after 45 minutes if there is no disturbance. If Acepromazine and Ketamine are added as adjuvants, the animal may develop "sunburn" (photosensitivity) when exposed to sunlight. An additional dose of xylazine can be given if sedation is not satisfactory even after 50 to 60 minutes of administration.

Under these circumstances, noosing is done first on hind limbs and then the forelimbs.. Noosing the forelimb prevents the animal from turning back.

The safest place to stand is near the belly or hindquarters. If threatened, one should move closer to the posterior end of the body.

In xylazine immobilization, the animal may snore. Though this does not in any way indicate the depth of anaesthesia, it could also be due to the relaxation of the vocal chords.

Once the operation is over, all the ropes should be removed. All the assistants should move to a safe distance or climb trees before administering the reviving agent (Revivon - Diprenorphine) I/V. Alternately, the dose of Diprenorphine can be split and given I/V and I/M in equal volumes. In case of xylazine, the reversal drug of choice is Yohimbine, or Yohimbine + 4 aminopyridine, atipamazole (Antisedan). Atipamazole @ 0.02-0.06/Kg body weight, Yohimbine @ of 0.12 mg/Kg. Diprenorphine is given two times the dose of etorphine. Immobilon is available as a preparation containing 2.25 mg/ml of etorphine and 10 mg/ml Acepromazine. The antidote Revivon contains 3 mg/ml of diprenorphine which reverses only the effects of Etorphine and not acepromazine.

## Case studies

1. **Sonitpur District, Assam**

The recent surge of incidents along the Assam and Arunachal Pradesh border amply indicates the worsening scenario faced by conservationists today. Between July and December 2001, at least 17 elephants were found dead in the Sonitpur district of Assam, with the highest number being in and around Nameri National Park. Five more were found the following year in the same region. Due to regular crop depredation; destruction of property and in some cases, loss of life, the situation has taken a turn for the worse with the tolerance level of local villagers' plummeting. Consequently, in this part of Assam, many elephants were "eliminated as pests".

Sonitpur district is situated between 26°30'N and 27°02'N latitudes and 92°17'E and 93°47'E longitudes and occupies 4,921.45 km$^2$ of land, accounting for 6.27% of the total geographical area of Assam (Assam Remote Sensing Application Centre, 1990). The district is bound by Arunachal Pradesh in the north, Darrang district in the west, Lakhimpur district in the east and the river Brahmaputra and Nowgong district in the south. Land use patterns in 1990 showed 56.54% of the district being utilized for agricultural purposes, with tea gardens that are found mainly along the northern belt of the district constituting 18.74% of the total agricultural land. 20.53% of the district was forestland at that time (Assam Remote Sensing Application Centre, 1990).

## Evidence of poisoning

1. Of the 14 unnatural deaths, 5 were found in paddy fields around the town of Tezpur and the rest were found in Nameri National Park.
2. The paddy cultivation within 500 m radius of the carcasses found outside Tezpur was completely damaged. The damage pattern indicated that the elephants repeatedly walked through the paddy field, damaging crops. There were no signs of bullet injury on the bodies and a total of three dead elephants were found within a 1.5 km$^2$ area on the same day.
3. Of the 9 carcasses found in Nameri National Park, 8 were found in or in close proximity of streams. It is common for poisoned animals to go in search of water in an attempt to relieve their discomfort.
4. A pathologist from the Department of Pathology at the College of Veterinary Science, Khanapara Campus, Guwahati visited the sites of where the carcasses were found. However, in each case, the carcass was considerably decomposed, and there was no likelihood of lesions being meaningful for investigation purposes (Prof. Chakraborty, pers.comm.[1]). The only option remaining was to take the liver, stomach and intestinal contents for toxicological tests, which revealed death to be caused by poisoning by Dimecron®, an organophosphorous based pesticide. Eight more samples were sent for toxicological tests by the Forest Department and all eight cases were poisoning.
5. A majority of victims were calves, sub-adults and adult females, which indicates that the target unit for poisoning were herds, as these tend to do maximum damage to crop fields during the depredation period. Also, a majority of the victims were calves or sub-adults as is expected in cases of malicious poisoning because these younger animals often have not developed enough sense to be wary of feed, water or liquor contaminated with poisons.
6. Investigations in the affected villages corroborated the hypothesis of poisoning to be the cause of death.

# DESCRIPTIONS OF INDIVIDUAL POISONS

# ARSENIC

## Introduction

Arsenic (As) is a common environmental poison found naturally in soil and water. It occurs as a cumulative poison within the body, remaining for long as deposits in bone, skin and hair. It is often found in combination with other metals, particularly iron, as arsenic pyrite (FeAs) and iron-arsenic sulphide. There are two elemental arsenics, trivalent and pentavalent arsenic salts (sodium, potassium and calcium salts of arsenates and arsenites respectively which are used as baits for slugs). Inorganic arsenicals were previously used in treatment of eczema and other skin conditions till recently, but owing to their toxicity, they were replaced by organic arsenicals. Currently, even these are rarely used as therapeutic agents and are not a very common source of poisoning in animals. Weak and dehydrated animals are more susceptible to arsenic poisoning. Interestingly, the metal may confer some amount of tolerance to its toxicity. Drinking water containing more than 0.25% of arsenic is potentially toxic especially to large animals.

## Possible sources

1. Drinking water may be contaminated with arsenic from:

   a) Effluents from heavy metal smelters like that of Cu, Pb & Zn near water holes.

   b) Electroplating industry and some processes used for the production of computer chips also release arsenic.

   c) Defoliants and herbicides (sodium or potassium arsenite/arsenate), rodenticides (arsenic trioxide) and wood preservatives (arsenic pentoxide).

   d) Organic arsenicals used as growth promoters in poultry and pig industry (arsenalic acid and sodium arsenilate).

2. Elephants like ash and ash of wood products treated with preservative may be a source of poisoning.

3. Some areas in India have a high concentration of arsenic in the soil. Some recent investigations show that tube wells in Bangladesh and West Bengal are highly contaminated with arsenic. Arsenic contaminated soils or burn-piles can be licked by animals that crave salt or minerals.

4. Malicious poisoning using arsenic trioxide in water holes or salt-licks can also lead to poisoning.

## Verification of toxicosis

### Clinical signs

1. A single, sufficient dose is enough to cause a slow and painful death over several hours or days.
2. In acute cases, major effects are seen in the gastrointestinal tract and the cardiovascular system. Clinical signs appear rapidly within a few hours and may last between a few hours to several weeks depending on the amount ingested. Among other symptoms of arsenic poisoning are:

a. Destruction of microvascular integrity, resulting in the exudation of the plasma. Blood loss and hypovolemic shock are observed.

b. Severe gastro-enteritis, vomiting, and rice-gruel like diarrhoea sometimes tinged with blood.

c. Severe colic, dehydration, weakness, depression, weak pulse, and cardiovascular collapse.

d. Clonic convulsions and subsequent coma may precede death.

3. In a per-acute form, the animal may be found dead.
4. In a sub-acute form, signs of colic, anorexia, depression, staggering gait, weakness, blood-tinged diarrhoea, or faeces with mucus, polyuria, anuria, partial paralysis of hind limbs, trembling, and haematuria are observed.
5. Chronic forms of toxicosis are rare but manifestations may occur as wasting, poor body condition, brick-red mucus membranes, normal temperature and irregular weak pulse. Reproductive disorders like sterility and abortion may be reported. These cases may be noticed in areas polluted by industrial effluents.

### Post mortem findings

1. Inflamed and reddened (extremely hyperaemic) gastrointestinal mucosae followed by oedema, ruptured blood vessels and necrosis of the epithelial and sub-epithelial tissue.
2. Necrosis may progress resulting in the perforation of the gastric or intestinal walls.
3. Gastrointestinal fluids are foul-smelling and blood-tinged with shreds of epithelial tissue.
4. Liver, kidney and other visceral organs show diffused inflammation.
5. Degeneration of fats and necrosis of liver, tubular changes in kidney is also noticed.
6. In cases of cutaneous exposure, skin may show necrosis with a dry and leathery appearance.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) Drop one or two pieces of bright copper strip after acidifying the test solution with dilute HCl, boil for 5-10 mins | Steel-grey or black deposition | Arsenic present |
| b) 1 ml of the suspected solution is taken in a test tube. Add a little Zn powder followed by dilute $H_2SO_4$. Boil a paper moistened with $AgNO_3$ to this. | $AgNO_3$ paper turns yellow and finally black | Silver arsenide, silver nitrate complex decomposes to release silver |

## Diagnosis

1. On chemical examination, suspected tissues (liver/kidney/stomach contents), are found to contain more than 1 ppm of arsenic. In cases of toxicosis, concentrations over 3 ppm. are noticed.
2. The concentration of arsenic in the stomach contents within 24-48 hours of ingestion, is diagnostically significant.
3. The concentration of the arsenic in the urine may be high for several days after ingestion.

## Differential diagnosis

1. Lead poisoning (plumbism) may show similar nervous and behavioural symptoms but digestive tract manifestations are likely to be much less severe.
2. Consumption of irritants such as certain plants, chlorate, and zinc phosphide, high doses of other heavy metals, as well as cholinesterase-inhibiting (organophosphorus and carbamate) insecticides in acute form may cause similar symptoms as the ingestion of blister beetles (*Epicauta* spp.) in horses.

## Lethal dose

Oral dose of sodium arsenite (trivalent) in most species is between

1-25 mg/kg – cats being the most sensitive.

Pentavalent arsenates are five times less toxic than other arsenates.

Average lethal dose for large animals like horses and cows is 10-45 g and 15-45 g respectively and for elephants it is likely to be at least at these levels.

## Treatment & management

1. The classical treatment with Dimercaprol/British Anti-Lewisite (BAL) was developed against Lewisite, which was used as a war gas in the olden days.
2. Water soluble analogs of BAL can be given orally.
3. Water-soluble preparations of BAL like MDSA (Methane Disulfonic Acid) or DMSA (Dimethyl Sulfonic Acid) are available; however these do not work where very high doses of arsenic have been ingested.

   a. Recommended dose for large animals is 3 mg/kg.

   b. Course: Treatment is to be repeated every 4hrs for the first 2 days, then every 6hrs on the 3rd day and thereafter twice a day for the next 10 days or till recovery.
4. Treatment with thioctic acid.

   a. Recommended dose for large animals can be using thioctic acid alone at 50 mg/kg I/M TD (thrice a day) as 20% solution or with BAL (3 mg/kg I/M).

   b. Course: The dose should be repeated every 4hrs for first two days, QD (four times a day) for the next three days and BD (twice a day) for the next 10 days or till recovery.
5. In large animals, BAL alone may not be effective. In that case other effective treatments can be considered.
6. Sodium thiosulphate may be administered orally at the rate of 20-30 mg in 300 ml water for large herbivores.
7. D-pencillamine has also been reported to be effective in man.
8. Activated charcoal with a cathartic followed by administration of a gastro-intestinal protectant such as kaolin pectin and supportive fluid therapy is recommended.
9. In cases of acute poisoning, the prognosis is grave.
10. In mild cases, morbidity is high but recovery may take 2-4 weeks.

Any of the treatments listed above can be resorted to by the veterinarian on observing the progress of clinical condition and outcome of the treatment and, depending on the availability of drugs, severity of poisoning, ease of administration etc. as well.

# LEAD

## Introduction

Lead (Pb)and its compounds can cause poisoning in many large animals. Young and pregnant animals are more susceptible. Poor health and previous exposure could make animals vulnerable to toxicosis. The presence of calcium and iron in the food reduces the absorption of lead through the digestive tract. Lead inhibits the release of several essential enzymes causing toxic or degenerative effects on the nervous system. Lead is ubiquitous in nature and small amounts are expected everywhere such as in soil, water, in plants and even in animal tissues. Normal forage samples for herbivores may contain 3-7 ppm lead or even less. Lambs feeding on forage containing lead at 45-65 ppm have not been observed to suffer any serious side effects. However silage with 150 ppm has killed cattle and herbage with 216-914 ppm has killed calves. The following are the concentrations of lead leading to poisoning and deaths of 175 cattle and calves:

Ingesta – 3427 (0-1,46,200) in ppm wet weight.

Liver – 43 (0-1,300) in ppm wet weight.

Kidney – 137 (2-2355) in ppm wet weight.

The presence of high lead concentrations in liver, kidney and ingesta should lead to presumptive diagnosis. In many poisoning cases, concentrations of lead in blood and tissues are enough to establish a diagnosis.

## Possible sources of lead contamination are:

1. Paint, grease, lead weights used in fishing lines or fishing nets, drapery weights, storage batteries, solder, buckshot or lead shots, in pollutants emanating from smelters or automobiles settling on the forage, rejected motor oil spills on pastures, oil filters, water waste from lead plumbing/glazed crockery units and lead parasiticide sprays. Elephants shot with lead pellets may suffer from chronic plumbism if the pellets do not get encapsulated.

## Verification of toxicosis

### Clinical signs

1. Major signs are associated with disturbances to the gastrointestinal and nervous systems.
2. The onset may take a few hours, days or weeks depending on the quantity ingested.
3. In chronic cases, clinical symptoms may take weeks or months to develop. Effects could include ataxia, blindness, salivation/spastic twitching of eyelids and convulsions.
4. In sub-acute cases, clinical symptoms include anorexia, constipation, colic, dullness, occasional diarrhoea, blindness, head-pressing, hyperaesthesia and incoordination.

**Post mortem findings**

1. Very few gross lesions are observed.
2. In acute cases, oil or flakes of paint or battery may be seen in the GI tract.
3. Evidence of gastroenteritis is present.
4. Tubular necrosis and degeneration of kidneys is observed along with the presence of intranuclear acid fat inclusion bodies.
5. In chronic cases, presence of excess porphyrins can be detected in plasma fluorescence under ultra violet rays.
6. Pronounced rigormortis is seen if the animals have high body temperature and convulsions prior to death.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) Add a little HCl to the test solution prepared by ashing (Refer Appendix II on Pg. 116) | White precipitate | Lead chloride |
| b) Add a little potassium chromate solution to the test solution prepared by ashing (Refer Appendix II on Pg. 116) | Yellow precipitate | Lead chromate |

## Diagnosis

Concentration of lead in the blood at 0.35 ppm, in the liver at 10 ppm or the kidney cortex at 10 ppm is indicative of lead poisoning in most species. Certain haematologic abnormalities like anaemia, anisocytosis, poikilocytosis, polychromasia, basophilic strippling, metarubricytosis and hypochromia are indicative but not confirmatory of lead poisoning.

## Differential diagnosis

1. In the case of poisoning by organochlorine insecticides, there is no blindness.
2. Encephalitic diseases must be ruled out from history, observation and other clinical tests.
3. Urea poisoning shows prominent colic but diarrhoea is not very prominent. There are convulsions as if induced by strychnine followed by rigidity instead of depression. There is no blindness, head-pressing, compulsive hypermotility or similar nervous symptoms.
4. Organophosphorous or carbamate insecticide poisoning shows pronounced parasympathetic signs, no behavioural changes and quick response to atropine.

5. Other toxicants that need to be excluded are metaldehyde, fluoroacetate, arsenic and mercury.
6. Possibility of tetanus also needs to be excluded.

**Lethal dose**

1. For cattle, the acute oral lethal dose of lead salts as lead acetate is approximately 600-800 mg/kg.
2. The chronic oral lethal dose of lead acetate is 6-7 mg/kg/day for cattle and 2.4 mg/kg/day for horses given for 6-8 weeks. However, only a small portion (<10 %) of this is absorbed due to the formation of insoluble complexes in the digestive tract, that are excreted in the faeces. The lethal dose for elephants is not available but figures for horses are to go by as they are also monogastric hindgut fermeters.

## Treatment & management

1. If the quantity ingested is large and signs of acute involvement of nervous system are perceived, treatment may not be successful.
2. In livestock, calcium disodium edetate (Ca EDTA) is given as I/V or S/C at the rate of 100 mg/kg/day divided into two doses daily for three days, to be repeated two days later.
3. Thiamine (2-4 mg/kg/day) and zinc supplements may alleviate some of the clinical symptoms and reduce deposition in tissue.
4. D-pencillamine has been tried in dogs (100 mg/kg/day) for two weeks.
5. A sulphate cathartic may help to remove the lead from the gastrointestinal tract.
6. Barbiturates and tranquilizers have been used for symptomatic treatments. However, whether phenothiazine tranquilizers will precipitate seizures is not known.
7. Corticosteroids and an osmotic diuretic may reduce cerebral oedema.

# MERCURY

## Introduction

Mercury (Mg) exists in both organic and inorganic forms in nature. Toxicity varies depending on which of these forms was ingested. Non-ruminants are more resistant than ruminants to mercury toxicosis. The presence of selenium in the diet decreases its toxicity, best documented in marine mammals. Elemental mercury is converted to alkyl forms like methyl by microbes in rivers, lakes and seas.

## Possible sources

1. Grains or seeds treated with mercurial fungicides, effluents from industries where mercury is used (e.g. electrical equipment including switches, thermostats, amd fluorescent light tubes, thermometers, barometers, mildew-proof paints, ointments, mirrors, small storage batteries, chloroalkaline – bleach, plastics, hydrochloric acid, fumes of fossil fuels like coal, volcanic action), and dental amalgams.

## Verification of toxicosis

### Clinical signs

1. Blindness, excitation, abnormal behaviour, chomping, incoordination, and convulsions.
2. The onset of clinical manifestations in case of organic mercurial ingestion is slow in most instances of poisoning.
   a. Redness of the skin, conjunctivitis, lacrimation and stomatitis are noticed.
   b. CNS effects are also noticed in organic mercurial poisoning.
3. Organic mercurials are absorbed generally and accumulation takes place in the brain and to some extent in the kidney and muscles.
4. Ingested inorganic mercury is poorly absorbed through the gastrointestinal tract, but due to its corrosive nature, high enough doses could produce colic, diarrhoea and renal damage.
5. In severe cases, inorganic mercury poisoning produces polydypsia and anuria.

### Post mortem findings

1. Lesions in the gastrointestinal tract leading to gastric ulcers, necrotic enteritis and colitis.
2. Congestion in the lungs and gastrointestinal mucosa is a possibility.
3. Renal lesions in swollen kidneys, which are pale with renal tubular necrosis.
4. With organo-mercurials and elemental mercury, degenerative lesions in the brain are noticed.

5. Organo-mercurials are not corrosive, hence, enteritis and ulcers are not common, in case they are ingested.
6. Histopathologic changes include fibrinoid degeneration of cerebral arterioles, neuronal necrosis, cortical vacuolation, axon swelling and gliosis in the CNS. The cerebrum is the most severely affected.
7. Organic mercurial ingestion leads to tissue degeneration in the heart and liver as well as tubular necrosis in the kidneys.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) Add a few drops of KOH solution to the test solution prepared by ashing (Refer Appendix II on Pg. 116) | Yellow orange precipitate | Mercuric hydroxide |
| b) Add a little stannous chloride solution to the test solution prepared by ashing (Refer Appendix II on Pg. 116) | White precipitate changing to grey and black | Mercuric chloride changing to mercury |
| c) Add a little potassium iodide solution to the test solution prepared by ashing (Refer Appendix II on Pg. 116) | Red precipitate | Mercuric chloride changes to mercuric iodide |

## Diagnosis

It is often difficult to pinpoint mercury poisoning on the basis of clinical signs and lesions. Laboratory analysis is essential to differentiate between the normal concentrations of mercury in tissues and feed (<1 ppm) and concentrations leading to acute poisoning. In cases of poisoning the concentration of mercury in kidneys, liver and brain could be elevated (>10 ppm).

## Differential diagnosis

Tremors and ataxia are the prevalent symptoms.

**Lethal dose**

1. The lethal dose for elephants is not available. The toxic dose of mercuric chloride for for horse and cattle is 8 gm when taken in orally.
2. With mercurous chloride, the toxic dose is almost double that of mercuric chloride.

## Treatment & management

1. A chelating agent like Dimercaprol can be given at the rate of 3 mg/kg I/M every 4 hrs for the first 2 days, every 6 hrs on the 3rd day and every 12 hrs for the next 10 days or until recovery.
2. Daily oral administration of D-pencillamine at 15-50 mg/kg body weight.
3. I/V administration at 8 hr intervals of sodium thiosulphate (20%) solution at 10 ml for 50 kg body weight could improve the action of Dimercaprol.
4. Egg white and activated charcoal are also given, orally if possible.
5. Supplemental selenium and vitamin E may also help.
6. A saline cathartic or sorbitol administration could promote faster clearance of the toxicant from the intestinal tract.

# SELENIUM

## Introduction

Selenium (Se) is an essential element that leads to toxicosis when consumed in excess amounts. Trace amounts of it in the diet are required to prevent deficiency diseases like the white muscle disease in cattle. Consumption of either organic or inorganic Se can cause poisoning. Organic salts are more soluble and can cause Se toxicosis faster. Plants are a major source of Se toxicosis. Many plants accumulate Se and transform it into organic compounds, which when foraged by animals could lead to poisoning.

Plants are classified according to their ability to accumulate Se:

1. Obligate accumulators or indicator plants – these grow well in Se rich soil, contain as much as 15,000 ppm of Se and enrich the top soil (e.g. *Astragalus* – poison vetch, *Stanleya* – princess' plume, *Oonopsis* – golden weed, *Xylorrhiza* – woody aster).
2. Facultative accumulators – these do not require Se for growth but accumulate it to a level of 25-100 ppm (e.g. *Castilleja* - paintbrush, *Comandra* – bustard toad flex, *Grayia* - hopsage, *Grindelia* - gumweeds, *Gutierrezia* – snake weed, *Machaeranthera* – tangy aster, *Penstemon* – beard tongue, *Syderanthus* – iron weed).
3. Non-accumulator plants – these can accumulate Se only to a level of 1-25 ppm if grown on Se rich soil, are passive accumulators and are mostly crops like corn, wheat, barley and gram.

When accumulator plants are consumed, the disease known as blind-staggers results. Chronic Se poisoning usually develops when animals consume seliniferous plants containing 5-40 ppm of Se. Seliniferous plants usually grow on alkaline soils that receive little rainfall.

## Possible sources

1. Plants are a major source of selenium toxicosis and so fodder containing more than 5 ppm of Se is considered harmful.
2. Industrial effluents from industries manufacturing rectifiers, xerographs, photoelectric cells, ceramics and certain electronic instruments, copper and steel industries. However, all these are organic sources that can be treated easily in effluent treatment plants.

## Verification of toxicosis

### Clinical signs

1. A garlicky odour in the animal's breath is an indicator of Se toxicosis

2. Hair loss is also a good indicator of chronic Se poisoning.
3. Lameness, stiff joints, dullness, cracking of the nails.
4. Poor appetite.

**Post mortem findings**

1. Acute selenium toxicosis is indicated by pulmonary congestion and oedema as well as degenerative changes in the liver and kidneys.
2. In chronic selenium toxicosis, lesions leading to necrosis and cirrhosis of the liver, enlargement of the spleen with localised haemorrhagic areas, congestion of the renal medulla, epicardial petechiae, hyperaemia and ulceration of the stomach and small intestine and erosion of the articular surface (particularly of tibia), could be seen.
3. Ascites is present in most cases.
4. In chronic selenosis, lesions with transverse lines of abnormal growth on the feet, cardiomyopathy, chronic fibrosis and cirrhosis are observed.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Add a little perchloric acid and warm gently. Cool and add a few drops of KI solution | Brown color | Iodine liberation |

## Diagnosis

1. Clinical signs, post-mortem findings and laboratory analyses of feed and forage, blood and tissues like kidney, liver can confirm the diagnosis.
2. In acute cases, blood selenium level may be 25 ppm and in chronic cases around 1-4 ppm.
3. Kidney and liver may contain 4-25 ppm Se, both under acute and chronic conditions
4. Levels of Se in the blood are usually 1-2 ppm in alkali disease and 1.5-5 ppm in blind staggers.

## Differential diagnosis

Acute toxicosis may be confused with pneumonia, anthrax, infectious hepatitis, enterotoxaemia and pastuerellosis. Chronic toxicosis may be confused with ergotism, molybdenosis, fluorosis and laminitis. Rotten horse-radish/garlic smell in fresh carcass is suggestive of an acute toxicosis. However, absence of such an odour does not necessarily

rule out Se toxicity. A test for selenium in tissues revealing sufficiently high concentrations is confirmatory.

## Lethal dose

1. Maximum tolerance level for selenium is considered to be 2-5 ppm.
2. A single oral dose in the range of 1-5 mg/kg can cause acute poisoning and is lethal for most animals.

## Treatment & management

1. No specific treatment for Se poisoning is available.
2. Feeds high in protein, linseed oil meal and sulphur have all been tried experimentally as antidotes. Even arsenic salts have been tried but practical utility is limited.
3. Use of chelating agents like BAL is contraindicated.
4. Vitamin E has a synergistic action in treatment.

# COPPER

## Introduction

Copper (Cu) is widely distributed in the environment. Being commercially important, its toxicity, in either acute or chronic form, is easily noticed. Low levels of molybdenum or sulphate influence copper poisoning.

## Possible sources

1. Effluents from the vicinity of mines and smelters.
2. Contaminated forage sprayed with copper fungicides.
3. Contamination in water holes (like ponds) that are treated with $CuSO_4$ for killing snails.
4. Exposure to toxic amounts of copper algicides.
5. Phytogenous factors can lead to secondary copper poisoning.
6. Consumption of plants like subterranean clover (*Trifolium subterraneum*) could lead to mineral imbalance and cause excessive copper retention.
7. Certain plants with very low levels of Mb, such as *Heliotropium europium* or *Senecio* spp. when ingested for several months may cause hepatogenous chronic copper poisoning. These plants contain hepatotoxic pyrrolizidine alkaloids, which lead to retention of excessive copper in the body.
8. Feeding/foraging on crops sprayed with copper fungicides (e.g. Bordeaux mixture).

## Verification of toxicosis

### Clinical signs

1. Acute poisoning causes gastroenteritis with abdominal pain, diarrhoea, anorexia, dehydration, haemolysis and hemoglobinuria occurring even after 3 days if the animal survives.
2. At times, chronic cases may develop into acute ones showing a sudden onset of depression, weakness, recumbency, constipation, anorexia, excessive thirst and jaundice.

### Post mortem findings

1. Severe gastritis with erosion and ulceration of the mucosa.
2. Jaundice is observed especially in chronic poisoning.
3. Pale yellow liver, enlarged pulpy spleen, bluish-black kidneys often referred to as "gun metal kidneys" especially if haemolytic crisis has occurred, renal tubular necrosis and excessive fragmented erythrocytes in the spleen are seen.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) Add a little $NH_4OH$ to the test solution prepared by ashing (Refer Appendix II on Pg. 116), observe & then add excess of the reagent | Blue precipitate with excess $NH_4OH$, intense blue colouration | Blue precipitate due to copper hydroxide & blue colour due to copper ammonium |
| b) Potassium ferro cyanide is added to neutral or fairly acid solution | A reddish brown precipitate | Copper ferro-cyanide is formed |

## Diagnosis

1. Since copper is a normal constituent of blood and tissues, diagnosis depends upon finding markedly elevated concentration in the same.
2. Diagnosis depends on existing clinical signs. Bluish-green ingesta and faeces (with acute toxicoses), elevated faecal copper levels (8000-10,000 ppm) all indicate copper poisoning.
3. In chronic cases, Cu levels upto 5-20 micrograms/ml are found as opposed to a normal level of approximately 1 microgram/ml.
4. Blue-green ingesta, deep-green coloured faeces and increased faecal copper levels (8000-10,000 ppm) and elevated levels in the kidney (>15 ppm wet weight) are indicative of acute copper toxicosis.

## Differential diagnosis

Data/information is inadequate.

### Lethal dose

1. 200-800 mg/kg body weight causes acute toxicity in cattle.
2. Daily intake of 3.3 mg/kg will produce chronic toxicity.

Signs of copper toxicosis may be similar to those observe in non-toxicologic and other toxicologic haemolytic diseases or hepatitis. A test for copper levels in tissues becomes essential.

## Treatment & management

1. Often not very successful.
2. Oral administration of pencillamine at a dose of 10-15 mg/kg twice daily.
3. Calcium versenate has also been tried.
4. Dietary supplementation with zinc acetate could prevent the absorption of Cu.

# MOLYBDENUM

## Introduction

Molybdenum (Mo) is an essential metabolite, micronutrient and an integral part of certain enzymes. It is present in all biological fluids and tissues including bones. Non-ruminants are more resistant to Mo toxicosis as compared to ruminants. Mo toxicosis often results from an imbalanced Cu: Mo ratio in the fodder and diet. Mo has a strong inverse reaction with copper and sulphur.

## Possible sources

1. Mining wastes.
2. Fertilisers containing Mo.
3. Smoke from steel and bauxite plants and oil refineries. Brick manufacturing plants that use clay that is high in molybedenum, steel mines and some industries that produce aluminium.
4. Plants can absorb and accumulate water soluble molybdates from contaminated soil.

## Verification of toxicosis

### Clinical signs

1. Lack of thriftiness, anaemia, emaciation and alopecia with loss of hair pigmentation.
2. Diarrhoea.

### Post mortem findings

1. Findings not specific
2. Osteoporosis and exostosis are seen but are not common findings.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Data/information is inadequate. | | |

## Diagnosis

A provisional diagnosis can be made when low copper levels in blood and tissues are found along with the apparent clinical signs of copper deficiency. Diarrhoea that stops within a few days of oral dosing with copper is helpful in confirming the diagnosis. Abnormal concentrations of molybdenum and copper in the blood or liver tissues through high dietary intake of molybdenum are indicative of Mo poisoning. Achromotrichia (i.e. depigmentation of hair) occurs, especially around the eyes and the hair coat becomes dull and rough.

## Differential diagnosis

Often confused with many other enteritides, often due to internal parasitism, especially since the two can occur simultaneously.

## Lethal dose

1. In the diet, a Cu : Mo ratio of 6:1 is ideal, 2:1 to 3:1 is border line and <2:1 is toxic.
2. Dietary Mo of 10 ppm can cause toxicity regardless of copper intake and as little as 1 ppm may be hazardous if copper content is <5 ppm (dry weight basis).

## Treatment & management

1. Injection of copper glycinate subcutaneously provides good treatment.

# CADMIUM

## Introduction

Cadmium (Cd) is a light, soft white metal soluble in acidic media. Environmental pollution with Cd is on the rise due to industrial activities. In nature, cadmium is often found in association with zinc and lead. It produces toxic effects due to its propensity to bind with proteins. Normally plants contain cadmium in relatively low concentrations from 0.01 ppm to 1.0 ppm, but those growing in highly contaminated areas may contain a higher concentration (>400 ppm).

## Possible sources

1. Units manufacturing plastics (plastic stabilizers), solder alloys, batteries (nickel cadmium), photo cell and rubber tyres.
2. Contamination of soil and forage by smelter plants of zinc, copper and lead.
3. Leachates from metal plating, alloys, small cadmium-nickel batteries and antiseborrhoeic shampoos.
4. From plants fertilised with cadmium-contaminaed sewage sludge or fertilizers.
5. From herbicides.

## Verification of toxicosis

### Clinical signs

1. Chronic toxicosis is more common than acute.
2. Gastrointestinal disorders, colic, diarrhoea, spasms, hypotension and collapse
3. Cough, pulmonary emphysema, anaemia and proteinuria are noticed
4. Severe kidney failure.

### Post mortem findings

1. Damages the mucosae of the GI tract and affects hepatic function.
2. Hepatic damage, nephrosis, osteoporosis, degeneration of gonads, CNS and blood vessels.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) Add dilute $H_2SO_4$<br>b) Add $NH_4OH$ drop by drop in excess | White precipitate<br>White ppt soluble in excess of $NH_4OH$ | Cadmium sulphate<br>Cadmium hydroxide |

## Diagnosis

Case history, chemical analysis and clinical symptoms help in establishing a diagnosis.

Increased urinary threonine and serine are indications of toxicosis.

Chemical analysis of cadmium in feed and body organs should be done to establish the cause of the toxicosis.

## Differential diagnosis

Data/information inadequate.

## Lethal dose

1. In the diet, a Cu : Mo ratio of 6:1 is ideal, 2:1 to 3:1 is border line and <2:1 is toxic.
2. Dietary Mo of 10 ppm can cause toxicity regardless of copper intake and as little as 1 ppm may be hazardous if copper content is <5 ppm (dry weight basis).

## Treatment & management

1. Administration of Selenium and vitamin D to reduce osteoporosis.
2. Disodium calcium EDTA (ethylene diamine tetraacetic acid) can also be tried.
3. Use of BAL is contraindicated.

# IRON

## Introduction

The body utilises iron (Fe). Certain enzyme systems in the body handle iron metabolism. Accidental ingestion of pharmaceutical preparations can lead to toxicity. Young animals are more susceptible to iron toxicity. Excess iron absorbed over a period of time accumulates as hemosiderin or ferritin. The normal iron concentration is usually around 100-300 micrograms per decilitre and 100-300 ppm.

## Possible sources

1. Forages high in iron.
2. As leachates from food and water.

## Verification of toxicosis

### Clinical signs

1. Chronic poisoning causes hemosiderosis.
2. Other toxic effects include chronic hepatic failure, weight loss, icterus, and depression.

### Post mortem findings

1. Hepatic lesions are seen.
2. Liver becomes friable and could become swollen or may shrivel.
3. Liver looks pale, tan or mottled reddish brown in colour.
4. Haemorrhages are seen in the gastrointestinal tract and liver.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) Add a little ammonium thiocyanate solution to the test solution prepared by ashing (Refer Appendix II on Page 116) | Blood red colour | Ferric thiocyanate |
| b) Add a little potassium ferrocyanide solution to the test solution prepared by ashing | Blue colour | Ferric ferrocyanide (Prussian blue) |

## Diagnosis:

1. History, clinical signs and necropsy lesions help confirm diagnosis.

2. Elevated total serum iron levels.
3. A high concentration of iron in the blood and tissues (50-100% above the normal level) indicate toxicosis.

## Differential diagnosis

Data/information inadequate.

### Lethal dose

1. Since rate of elimination is slow, an oral dose >150 mg/kg will lead to toxicosis.

## Treatment & management

1. Generally, iron toxicosis is grave.
2. Deferioxamine (Desferal) may trap the circulating iron by chelation. (To be given as I/V drip at the rate of 0.175 mg/kg/minute.). Hypotension is a side effect of this agent and the dosage regimen for different species of animals are yet to be determined.
3. Repeated phlebotomy and oral administration of large doses of ascorbic acid are recommended for the quick elimination of iron from the body.
4. Supportive treatment with vitamin E and selenium can be undertaken.

# ZINC

## Introduction

Zinc (Zn) is an essential element for animals and birds. It is a micronutrient essential for skin nourishment as well as a component of several enzymes. Poisoning resulting from consumption of a zinc-containing compound is common in domestic animals. Soluble salts of Zinc (like chloride, sulphate, acetate) are more toxic than insoluble salts (like carbonate and oxide). Young animals are more susceptible to Zinc poisoning.

## Possible sources

1. Inhalation of industrial fumes containing zinc salts.
2. Chronic poisoning is seen in animals living in close proximity to Zinc ore processing or smelting works.

## Verification of toxicosis

### Clinical signs

1. In cases of acute toxicity, soluble salts cause painful local irritation and corrosion of GI tract resulting in vomiting and diarrhoea, colic, blood in faeces, abdominal pain and hypotension.
2. Liver damage leading to icterus of the visible mucus membrane.
3. Chronic toxicity leads to stunted growth, weakness, lameness and moderate anaemia.

### Post mortem findings

1. Gastroenteritis, renal tubular necrosis, haematuria, proteinuria and uraemia.
2. In chronic cases of poisoning, arthritic and osteoporotic lesions are seen.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) Add a few drops of NaOH. Observe & then add excess $NH_4OH$ to the test solution prepared by ashing (Refer Appendix II on Page 116) | White precipitate which dissolves in excess $NH_4OH$ | White precipitate is zinc hydroxide. It dissolves in excess $NH_4OH$ forming sodium zincate |
| b) Add a few drops of potassium ferri-cyanide solution to the test solution prepared by ashing (Refer Appendix II on Pg. 116) | White precipitate | Zinc ferro-cyanide |

## Diagnosis

1. Clinical symptoms as well as analysis of feed, blood (serum or plasma) and hair can confirm the diagnosis.

## Differential diagnosis

Data/information inadequate.

## Lethal dose

1. $LD_{50}$ for zinc chloride is 100 mg/kg body weight.
2. Dietary concentration of 1 g/kg of feed may cause toxicity – fortunately, feeds at such concentrations are often unpalatable for animals.
3. Dietary zinc exceeding 2000 ppm can cause chronic toxicity.

## Treatment & management

1. Calcium disodium EDTA at 110 mg/kg body weight per day in three or four divided doses mixed with 5% dextrose I/V and then S/C or D-penicillamine at 110 mg/kg for 1-2 weeks orally.
2. Sodium carbonate 1% solution and egg albumin are used for conversion to insoluble salts, thereby minimising absorption.

# THALLIUM

## Introduction

Toxicosis usually occurs more commonly with thallous (Tl) salts (thallous sulphate and thallous acetate) and less with thallic salts. Young animals are more ssusceptible to thallium poisoning than adults. Chronic poisoning with thallium is not common but may occur due to accumulation in the body. Thallium sulphate was used formerly as a rodenticide.

## Possible sources

1. Salts of thallium will cause toxicity.
2. In the industry, thallium is used as a dye, optical lenses and jewellery.

## Verification of toxicosis

### Clinical signs

1. Colic, gastroenteritis, diarrhoea, dyspnoea, blindness, fever, conjunctivitis, gingivitis with extreme hyperaemia, and tremors or seizures.
2. In chronic cases, dermatitis characterized by alopecia, erythema and hyperkeratosis, cracking of the skin.
3. Hyperemia of the conjunctiva.
4. Nervous signs are sometimes observed.
5. Death results due to malnutrition, dehydration and secondary infection.
6. There could be softening of bones leading to rickets.

### Post mortem findings

1. In cases of acute poisoning, ulceration, haemorrhagic gastroenteritis and inflammation of respiratory mucosa are noticed.
2. Many tissues may show local necrosis.
3. Degenerative changes are noticed in the heart, kidney and liver tissues.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Data/information inadequate. | | |

## Diagnosis

Case history and clinical signs.

## Differential diagnosis

1. Symptoms of poisoning may be confused with those of other debilitating diseases with secondary complications, as also with signs of bacterial, fungal and parasitic skin infections.

## Lethal dose

1. In rats, acute oral LD50 of thallous sulphate is 16-25 mg/kg body weight.
2. In other species this varies from 10-25 mg/kg body weight.

## Treatment & management

1. The specific recommended chelating agent, though not very effective, is diphenyl thiocarbazone (dithizone) at 70 mg/kg administered orally TID (thrice a day) for 6 days.
2. In chronic cases, smaller doses of the above may be tried orally.
3. Dithizone should not be administered intravenously (I/V).
4. Administration of potassium chloride solution (I/V) at a concentration of 9.3 mg/ml infused at the rate of 1.96 ml/min is recommended.
5. All potassium salts are to be given I/V very slowly.
6. Prussian blue 100 mg/kg orally BID will enhance faecal excretion of the toxicant.

# FLUORIDE

## Introduction

The terms fluorine (F) and fluoride are used interchangeably. Fluorides are widely distributed in the environment and originate naturally from rocks, soils or as a result of industrial activities. Fluorine at a concentration of 1-2 ppm is sufficient for metabolic activities. Since it has a high affinity for calcium, it replaces the hydroxy apatite moiety in the bones. Hence, during the growth phase small amounts of fluorine will make the enamel less soluble, thereby protecting it. At the same time excessive fluorine can also make the enamel brittle. Faulty mineralization is seen in case of toxicosis. Endemic fluorosis exists in many parts of India and Africa. Toxicity depends upon the solubility of inorganic fluorine in water. In terms of toxicity, sodium fluoride and sodium fluorosilicate (silicofluoride) are the most toxic while calcium fluoride (fluorspar) is the least.

## Possible sources

1. Many forages especially legumes and plants that grow on soils rich in fluoride (seeds or grains do not accumulate excessive amounts of the element).
2. Effluents from phosphate fertilizer factories.
3. Natural sources like fluorspar ($CaF_2$), cryolite ($Na_3AlF_6$), apatite [$CaF_2.3Ca_3(PO_4)_2$] and rock phosphate ($CaHPO_4$).
4. From apatites that are used as phosphate fertilizers and as a source of Ca in mineral mixtures.
5. Fluorine fumes emanating from fertilizers like superphosphate which settle on forages.
6. Fluorine emitted during smelting in the case of aluminium smelters that use a mixture of fluorine containing bauxite and cryolite.
7. Some steel ores may contain fluorine which are released during certain enamelling processes and could contaminate forages. Depending on the wind direction, the pollutant may spread over a distance of 5-10 km.
8. Drinking water may contain large quantities of fluorine as a result of which endemic fluorosis becomes common (seen in parts of Andhra Pradesh like Ongole).
9. Brick manufacturing units using phosphate limestone may contaminate nearby fields.
10. Dust and gases from volcanic eruptions are also sources of contamination.
11. Water in areas with historic or ongoing volcanic activity or in hotsprings.
12. Ingestion of sodium fluorosilicate/fluoroacetate (used as a rodenticide) can cause toxicosis.

## Verification of toxicosis

**Clinical signs**

1. Acute fluoride poisoning is rare.
2. In chronic poisoning, periodic and intermittent lameness, mottled teeth, exostosis and sclerosis are seen.
3. Growing bones in young animals like ribs, mandible and long bones are often affected.
4. Decreased food and water intake is a common feature.
5. Spontaneous fractures can occur.
6. Excessive wearing of teeth.
7. Exostosis that can be palpated along long bones and also across the mandible.
8. In cattle, the teeth (typically discoloured and with premature wear) and long bones (exostoses with the joints spared) are affected.

**Post mortem findings**

1. Presence of fractures.
2. Exostosis along long bones and also across the mandible.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) Ash from the suspected material is taken in a test tube & warmed in conc. $H_2SO_4$ | Vapours having the property of etching (greasy surface look). A wet glass rod introduced into the vapours produces white ppt | Hydrogen fluoride vapours and formation of silicic acid (an acid containing silicon) |
| b) Use fluoride sensing electrode to estimate the level of fluoride. Feed & mineral mixture are digested with conc. HCl to prepare the test solution | Compare using the standard NAF solution | Can be detected in small conc. levels 0.01 ppm |

## Diagnosis

1. Circumstantial evidence such as the location of a nearby factory, which can be the cause of contamination of food and water sources.
2. Elevated fluorine levels in various tissues such as plasma, bone, dental tissues and urine, is an indication of toxicosis.

3. Fluoritic cattle may show up to 3000 ppm or more fluoride in the bone (normal values being between 400 and 1200 ppm).
4. Urine may also contain 15-20 ppm of fluoride as compared to 2-6 ppm found in normal animals.
5. Clinical signs may suggest osteoporosis and deficiency of Ca, P and vitamin D.
6. Lameness may even be due to accidents.

## Differential diagnosis

Mineral (Ca, P) deficiencies, vitamin D deficiency, and tetracycline toxicosis (in domestic animals and human beings).

## Lethal dose

1. Tolerance limit in large animals is 40-50 ppm.

## Treatment & management

1. Since chronic fluorosis is often a result of environmental pollution, the remedy consists of reducing further emissions (e.g. improved pollution control equipment in a fertilizer or aluminium manufacturing plant). Animals may need to be removed from the area and samples of forage and soils tested repeatedly until risks have been reduced.
2. Supplementation of diet with Calcium, Phosphorous and vitamin D are indicated.
3. Aluminium oxide, sulphate and chloride, calcium carbonate, calcium aluminate, magnesium metasilicate or boron have been found to decrease the absorption or to increase the excretion of fluoride.

# PHOSPHORUS

## Introduction

Phosphorus (P) is an essential element included in body tissues as phosphate. As an element, it appears yellow, white, red and black. It is hepatotoxic when absorbed. Only yellow phosphorus is used as a rodenticide and is easily available in the market. It is a protoplasmic poison, strong irritant, corrosive and has a necrotising effect on the stomach mucosa. Yellow phosphorus also acts directly on the heart. The first and most pervasive systemic effect of phosphorus is peripheral vascular dilatation. The response to phosphorus varies little amongst species.

## Possible sources

1. Chemical industry, units involved in the manufacture of gunpowder, fertilizers and incendiary ammunitions.
2. Rat poison, containing elemental phosphorous.

## Verification of toxicosis

### Clinical signs

1. Phosphorus leads to structural damage of vital organs and severely disrupts their metabolic function. Resultant hypoglycaemia, azotemia, and inhibition of glycogen formation in the liver and other disorders confirm toxicosis.
2. An acute initial phase with vomiting and diarrhoea leading to gastrointestinal, abdominal and circulatory signs occurs within hours of ingestion.
3. If the quantity ingested is significant, shock, cyanosis, incoordination and hepatic failure with potential hepato-encephalopathy may ensue followed by coma and death.
4. Colic, garlic odour from the mouth and stomach contents, liver damage and icterus could also be noticed.
5. In chronic cases, the main finding is "phossy jaw", that is initial toothache followed by loosening of the teeth.
6. There is often an offensive discharge from the sockets; the bone is exposed and often shows signs of necrosis and formation of a sequestrum; there is gross disfigurement and debility finally leading to death.
7. Death is usually due to liver necrosis and heart failure.

### Post mortem findings

1. Fatty tissue degeneration, swollen liver, gastrointestinal irritation, necrosis and haemorrhage confirm diagnosis.

2. If death is immediate, then few signs can be noticed other than irritation of the oesophagus and stomach. Perforation may also have occurred.
3. If the animal dies several days after poisoning, fatty tissue degeneration in the liver, heart and kidney are striking evidences. This is also observed, though to a lesser extent in all other organs including the brain.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Fresh sample of the material suspected to be contaminated with zinc phosphide is treated with aluminium phosphide, then treated with conc. HCl & immediately introduced to cadmium mercuric iodide paper moistened with acetic anhydride | Orange colour | Cadmium phosphide |

## Diagnosis

1. Vomit showing luminiscence (phosphorescence) at night is indicative of the presence of phosphorus.
2. Case history, clinical signs and chemical tests confirm the diagnosis.

## Differential diagnosis

Should be differentiated from zinc phosphide, metaldehyde, aflatoxins, iron, and infectious hepatitis viruses.

## Lethal dose

1. White phosphorous in finely divided form can cause toxicity in horses and oxen (0.5-2 g), pigs (0.05-0.3 g), dogs (0.05-0.1 g) and fowl (0.02 g).

## Treatment & management

1. There is no antidote for phosphorus poisoning. However, an early diagnosis followed with induced vomiting or gastric lavage can save the animal.
2. Treatment with 0.1% potassium permanganate or 2% hydrogen peroxide (to oxidise the toxicant to harmless phosphates) and mineral oil (to prevent absorption) could help.

3. Non-absorbable oil like liquid paraffin would solubilise the phosphorus and prevent its absorption.
4. Activated charcoal, an adsorbent for many poisons could be of help. Osmotic purgatives such as sodium sulphate, magnesium sulphate, sorbitol or mannitol can be administered orally or rectally. Sorbitol is often mixed with activated charcoal in the management of poison.
5. 1% $CuSO_4$ is used as an emetic in other animals but is not effective in elephants. Administration of $CuSO_4$ solution can lead to the formation of a non absorbable copper phosphide complex.
6. Prognosis is not good because of the resultant cardiovascular dysfunction and severe damage to the kidney and liver.

# NITRATE & NITRITE

## Introduction

Many species of animals are susceptible to nitrite ($NO_2$) and nitrate ($NO_3$) poisoning. Ruminants are more susceptible as the ruminal flora changes nitrate to ammonia – this can occur in the caecum of elephants, though to a lesser extent. Nitrite is approximately 10 times more toxic than nitrate. Nitrates containing water may support a significant population of coliform bacteria, which can also affect the health of the animal adversely. Toxicity is primarily due to the reaction of the nitrite ion with iron in haemoglobin, causing its oxidation to form methemoglobin which results in anoxia.

## Possible sources

1. Crops grown on soil treated heavily with nitrogenous fertilizers.
2. Herbicides like 2, 4-D and 2, 4, 5-HT which increase the nitrate content in plants.
3. Water holes with water from nitrate rich soils or fertilizer factory effluents are another source for toxicosis – this is seen in certain deep wells where seepage has occurred from nitrate rich soils and could contain nitrate levels as high as 17,000 ppm.
4. Decaying organic matter, industrial effluents and limestone may all increase the soil and water nitrate concentration.
5. Nitrate concentration in plants are enhanced by low Molybdenum levels.
6. Rapidly growing plants during the hot and humid weather may cause high nitrate concentrations.
7. Immature plants during drought could acquire a high nitrate concentration. Drought-stricken plants could have extremely high nitrate concentrations. Immediately after a prolonged drought, the overly stressed plants may take up high concentrations of nitrate from soil yet lack the ability to use it in synthesis of biomolecules. When this occurs, plant nitrate residues can be particularly high.
8. Certain plants like cereal grains (oats, millets and rye), sunflower, sorghum and corn (maize) accumulate nitrate, primarily in the stems and leaves.
9. Plants like red root, white rag weed, Russian thistle, variegated thistle, smart weed, pig weed, lamb's quarter, jimson weed, fire weed, dock and Johnson's grass are known to be nitrate accumulating plants.
10. Nitrates used in pickling and in brine used in the preservation of meat.
11. Waste water from slaughter houses and meat processing units may contaminate nearby water bodies causing toxicosis.
12. Potassium nitrate is used in gunpowder and ammonium nitrate for dynamite production, and thus effluents from ammunition factories are also sources for nitrate toxicosis.

## Verification of toxicosis

### Clinical signs

1. In acute cases, signs appear quickly - within 0.5-4 hrs of ingestion.
2. Common symptoms include a weak pulse, below normal body temperature, muscular tremors, weakness and ataxia.
3. In some cases, the animal may die suddenly without showing clinical signs Expected effects include terminal anoxia and convulsions during the last hour may be noticed.
4. The course of the disease may last for 12-24 hrs.

### Post mortem findings

1. In the peracute form of nitrate toxicosis, hardly any clinical signs are seen.
2. Dark chocolate-coloured blood that clots poorly. The chocolate colour of blood is due to methemoglobin formation. However, methemogobin is converted back to haemoglobin after some time and hence the chocolate colour is not seen if the post-mortem is done very late.
3. Brown-stained tissues, congestion and inflammation of parenchymatous organs, petechial and large haemorrhages on the serous surfaces are observed.
4. Dilatation of blood vessels is noticed.
5. Blood-stained pericardial fluid and generalized cyanosis is observed.

## Chemical test (for Nitrite)

| Procedure | Observation | Reaction |
|---|---|---|
| Take fresh aqueous extract of the materials suspected to have been contaminated with nitrite and acidify with dil $H_2SO_4$ and add starch | Blue colour | Nitrite oxidizes KI to $I_2$ which turns starch blue |

## Diagnosis

1. Blood containing methhemoglobin has a chocolate brown colour and at times even appears dark red. This is indicative of nitrate toxicosis. Methhemoglobin analysis giving results as a percent concentration has certain diagnostic value.
2. An approved and reliable field test for nitrate in suspected forages is the use of diphenyl amine blue (1% DPAB in concentrated $H_2SO_4$) and nitrate test strip. The nitrate strips will have a colour chart which can be used to determine its presence.
3. Nitrate test strips can be used to test water supplies, serum, plasma, ocular fluid and plasma to confirm the diagnosis.

## Differential diagnosis

1. Signs of nitrate toxicosis may need to be distinguished from those of poisoning due to cyanide, urea, , poisonous gases (CO, $H_2S$), aminophenols, aniline dyes and chlorates
2. Also, it is also necessary to diffentiate symptoms of infectious diseases that affect oxygen carrying capacity (e.g. haemolytic bacterial infections and parasites) causing unexpected death.

## Lethal dose

1. Minimum lethal dose in cattle is 0.65-0.75 g/kg for sodium nitrate and 1 g/kg for potassium nitrate.

## Treatment & management

1. Administration of methylene blue solution 1% either in water for injection or with normal saline.
2. Suggested dose is 4-10 mg/kg or more depending upon the severity of exposure
3. Higher doses of upto 22 mg/kg are well tolerated by ruminants.
4. 1% methylene blue in distilled water or isotonic saline, given at the rate of 4-22mg/ Kg body weight or more depending upon the severity of exposure is recommended. Treatment should be repeated every 6-8 hours.

# UREA

## Introduction

Mono-gastric animals are less susceptible to oral urea poisoning than ruminants. Unlike ruminants, neither alkaline hydrolysis nor urease activity occurs in the upper digestive tracts of mono-gastric animals. In elephants, the caecum contains urease and also has an alkaline medium. Elephants have been found to consume urea applied as a fertilizer on crops like pepper cultivated close to the forests, probably because of its salty taste. Urea is known to have been utilised maliciously for the poisoning of elephants.

## Possible sources

1. Urea and other inorganic and organic sources of nitrogen used in the ruminant's rations as a source of non-protein nitrogen.
2. Fertilisers.

## Verification of toxicosis

### Clinical signs

1. Clinical signs occur within 20-90 mins of ingestion in ruminants.
2. Non-ruminant herbivores such as horses and elephants take a longer time to exhibit symptoms.
3. Muscle tremors especially of the face and the ears, exophthalmia, colic, frothy salivation, frequent urination and head-pressing are common symptoms.
4. As the condition progresses, there is cyanosis, dyspnea, anuria and hyperthermia
5. In non-ruminant herbivores, death may occur within 3-12 hrs.
6. In animals that survive (those that do not succumb within 12-24 hrs), the following changes are observed: increased Packed Cell Volume (PCV). Increased serum ammonia, glucose, lactate, potassium, phosphorus, Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT) and Blood Urea Nitrogen) BUN levels are observed.

### Post mortem findings

1. No specific lesions are seen.
2. Carcass will bloat and decompose rapidly, especially so in humid climates.
3. Pulmonary congestion, oedema and petechial haemorrhages could be seen in the gastro intestinal tract.

4. Mild bronchitis and catarrhal gastroenteritis could be observed.
5. If carcass is fresh when opened, the contents of the caecum will have an ammoniacal odour. A recently dead animal may exhibit a caecal pH e" 7.5 thus suggesting ammonia poisoning.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) Aqueous extract is boiled with NaOH. A glass rod dipped in conc. HCl is introduced into the test tube | A smell of ammonia and intense white fumes emanate | Ammonium chloride |
| b) For quantitative estimation, a weighed quantity of the sample is mixed with NaOH and distilled. The ammonia liberated is absorbed into a known excess N/10 HCl | Urea is estimated by the back titration of excess of N/10 HCl | $CO(NH_2) + 2NaOH$ à $2NH_3 + Na_2CO_3$ |

## Diagnosis

1. Animals dead for more than a few hours in high temperatures or more than 12 hrs in moderate climates undergo significant autolysis and hence may be of no diagnostic value.
2. Apart from clinical signs, laboratory analysis for ammoniacal nitrogen in both the anti- and post-mortem specimens are essential.
3. Specimens from the suspected sources must be frozen immediately and thawed only at the time of analysis.
4. The GI contents should be preserved with a few drops of saturated mercuric chloride solution for each 100 ml of the specimen.
5. Concentrations greater than or equal to 2 mg/100 ml of ammoniacal nitrogen in blood or serum indicates excess NPN exposure, thereby confirming diagnosis.

## Differential diagnosis

It is necessary to differentiate the symptoms from those of nitrate, nitrite, cyanide, lead, and neurotoxic pesticide poisonings and signs of acute infectious diseases, encephalopathy and enterotoxaemia.

## Lethal dose

1. In mono-gastric herbivores such as horses, urea ingestion at a rate of 4 g/kg becomes lethal.
2. Ammonia salts are lethal in these species at a dose of 1.5 g/kg.

## Treatment & management

1. Oral administration of vinegar or dilute acetic acid is done to lower the pH in order to slow urease and protonate $NH_3$ formation so as to release less readily absorbed $NH_4^+$.
2. I/V fluids like calcium gluconate and magnesium solutions are used to relieve the tetanic seizures.
3. Since most cases of urea poisoning recognised in elephants to date have been with malicious intent, a local enquiry could reveal the problem easily. It is, however, difficult to prevent toxicosis in areas where elephants raid crops frequently and human-animal confrontations are inevitable.

# SALT POISONING

## Introduction

Sodium (Na) performs a number of essential functions in the body and sodium chloride (NaCl) is an essential and natural ingredient of all feeds. Salt licks are often provided at regular intervals in many reserve forests. Herbivores, especially elephants, have a craving for salts. Salt toxicosis is very unlikely as long as the salt regulating functions of the body are intact and fresh drinking water is available *ad libitum*. However, all species are susceptible to salt toxicosis. A salt hungry animal may consume large quantities of salt from a salt lick in a single feeding and thus get overdosed. Salt poisoning is more common in summer.

## Possible sources

1. Salt toxicity is directly related to water scarcity and availability of salts.
2. Salt poisoning occurs when salt licks are provided in large numbers to attract animals.
3. Drinking water containing more than 1.5% salt may prove toxic – it is generally recommended that drinking water should contain less than 0.5% of total salts for it to be safe for any species.

## Verification of toxicosis

### Clinical signs

1. Salivation, serous nasal discharge, urinary incontinence or polyuria followed by anuria.
2. Diarrhoea may be followed by constipation.
3. Colic at normal body temperature.
4. Nervous signs include hyperaesthesia, partial or complete blindness or deafness, moving in circles, muscle tremors, shivering or twitching.
5. Clonic or tonic seizures could also be observed.

### Post mortem findings

1. Congestion of the gastric mucosa or inflammation with pinpoint ulceration.
2. Faeces become dark coloured and could be either fluid or dry.
3. Oedema of skeletal muscles and hydropericardium.
4. Fluid accumulation in the body cavities and pericardium.
5. Prominent cerebral oedema could be seen.
6. Testes could become oedematous and enlarged.

## Chemical Test

Since salt poisoning is quality dependent, qualitative test is not important.

## Diagnosis

1. Mainly from clinical signs and case history indicating water toxicity.
2. A concentration of sodium serum in the cerebrospinal fluid (CSF) of more than 160 mEq/l, is compatible with salt poisoning
3. If brain (cerebrum) concentration of sodium is > 18,000 ppm (wet weight), then this is suggestive of salt toxicosis.
4. Laboratory analysis of suspected fodder must be conducted to confirm the diagnosis.

## Differential diagnosis

1. Symptoms may be similar to those due to toxicity by encephalopathic poisons such as lead, chlorinated hydrocarbon insecticides and urea.

## Lethal dose

1. In large animals the oral lethal dose of salt is 2.2 g/kg body weight - this is variable depending on the availability of water – i.e. toxicity is very unlikely if water is available *ad libitum*.

## Treatment & management

1. The basic principle as in the case of other toxicosis is the removal of the source of poison at the earliest.
2. Providing fresh water will help. Water must be provided in small quantities at regular intervals. Intake of large quantities of water would cause cerebral oedema. Excess water may exacerbate the nervous symptoms due to the brain oedema and could even cause death of the animal.
3. In severe cases, the animal may die in spite of the treatment administered.

# PESTICIDES

Pesticides are often used as plant protection chemicals for improving the production of the crops. The major group of agricultural pesticides include insecticides, acaricides, herbicides, fungicides, rodenticides and fumigants.

There are many ways in which animals are maimed when exposed to pesticides. Just like any other poison, pesticides can enter the body on ingestion by inhaling or through the dermal layer. In elephants, oral poisoning is very common. This can be accidental or intentional. Accidental poisoning often results when the elephants forage on crops sprayed with insecticides. Certain plantation crops like rubber are sprayed with fungicides like copper oxychloride prior to the monsoon season. Large areas are sprayed with such chemicals for protection against phytopthera. These chemicals contaminate not only the upper canopy of the trees but also legumes grown as ground cover crops, which provide good forage. Environmental pollution is yet another cause for poisoning of wild animals. It may also be noted that pesticides, at times are also used for the treatment of various parasitic infections in domestic animals.

The **Minimum Residual Level** for pesticides in food is explained by the following formula:

$$\textbf{MRL mg/kg food} = \frac{\text{ADI x Body weight in kg}}{\text{Quantity of food in grams}}$$

$$\textbf{Accepted Daily Intake (ADI)} = \frac{\text{NEL x 70 kg}}{100}$$

(NEL = No Effect Level in mg/kg)

# INSECTICIDES

A large number of insecticides were synthesized extensively after the advent of DDT during the Second World War. The general classification of insecticides is as follows:

1. Organochlorine (chlorinated hydrocarbons) insecticides e.g. DDT, BHC (HCH- gamma isomer of hexachlorocyclohexane), chlordane, dieldrin, endrin, heptachlor, methoxychlor.
2. Organophosphorus insecticides (OP compounds) e.g. malathion, dichlorvos (DDVP), disyston, diazinon.
3. Carbamate insecticides e.g. carbaryl, carbofuran, propoxur.
4. Pyrethroids e.g. fenvalerate, alethrin, deltamethrin.
5. Formamidine acaricides and insecticides e.g. amitraz.
6. Natural products e.g. nicotine, rotenone.

## Toxicity based classification of pesticides

| | **Group I** | **Group II** | **Group III** | **Group IV** |
|---|---|---|---|---|
| Label | *Red* | *Yellow* | *Blue* | *Green* |
| **Warning** | Poison | Poison | Danger | Caution |
| **Toxicity** | Extremely toxic | Highly Toxic | Moderately Toxic | Slightly Toxic |
| **Oral $LD_{50}$** | 1-50 g | 51-500 g | 501-5000 g | > 5000 g |
| **Compounds** | Aldicarb | DDT | Copper oxychloride | Zineb |
| | Carbofuran | Lindane, DDVP | Kitazine | Maneb |
| | Phosphomidons | Herogan | Baygon | Plant insecticides |
| | Phorate | Sumithion | Warfarin | Plant insecticides |
| | Zinc phosphide. Mainly used as a rodenticide. | Gremaxone | Warfarin. Commonly used as an anticoagulant rodenticide. | Zineb and maneb |
| | Parathion | Labacid | | |
| | Endosulfan | Ekalex, 2,4-D - herbicide | | |

## Coloured labels indicating the toxic level of the specimens


Group I label


Group II label


Group III label


Group IV label

# ORGANOCHLORINE INSECTICIDES

## Introduction

These organochlorine (OC) compounds are contact insecticides and ectoparasiticides. Among them, DDT was the first to be used widely. Since most of them are not easily degradable in the environment, their use has now been discontinued in many countries. DDT has been almost completely banned worldwide except in certain countries for malaria eradication. The use of another previously popular compound, Benzene Hexa Chloride (BHC) whose correct name is Hexachlorocyclohexane (HCH), has now been restricted to its gamma isomer known as Lindane which has potent insecticidal properties. Other commonly used OC compounds include aldrin, chlordane, dieldrin, heptachlor, methoxychlor and toxaphane. Depending on the chemical structure, this group of insecticides can be further subdivided into the following categories:

1. Diphenyl aliphatics: This group includes the first synthetic insecticide & DDT (Dichloro Diphenyl Trichloroethane). Other compounds are methoxyclor & dicofol. Methoxychlor is atypical in its comparatively efficient elimination from the mammalian systems.
2. Cyclodiene compounds (polycyclic chlorinated compounds): Examples are aldrin, dieldrin, endrin, chlordane, heptachlor, toxaphane, and endosulphan. Of these, endosulphan is extensively used even today due to its broad spectrum of activity and safety to beneficial insects and pollinators.
3. Aryl hydrocarbons: eg. BHC.

OC compounds are highly stable and their half-life varies from one to several years, e.g. DDT has a half-life of 3-10 years and toxophane has a half-life of 10 years. These compounds have a high lipid solubility and hence cumulative in nature. This leads to their bio-magnification in the food chain. They are readily absorbed by the skin and the mucous membranes. Absorption through the gastrointestinal tract is poor except only if they are in an oily solution. Organochlorines are metabolized by microsomal enzymes in the liver. Unabsorbed ingested compounds are excreted in the faeces, unchanged. The route of elimination from the body is mainly through the bile, urine and milk. They act on the body as stimulants of Central Nervous System (CNS) and there are no specific antagonists. In some animals, hepatotoxic effects are observed.

## Possible sources

1. OC compounds are available in the market and can be used for intentional poisoning.
2. Leakage from old dump sites occasionally lead to residual accumulation.

## Verification of toxicosis

### Clinical signs

1. In acute toxicity, signs are common for all species. The onset of symptoms is sudden, often taking only a few minutes. In chronic cases it could take upto several days.
    a. Affected animals show hypersensitivity to stimuli and become timid and apparently apprehensive.
    b. Muscle twitching, starting from the eyelids and neck, anterior portions of the body followed by the hindquarters is observed.
    c. Restlessness, staggering gait and circular movements are common.
    d. The animal may strike out at objects as if blind or may even step over imaginative objects.
    e. Unusual postures and head pressing are seen in some animals.
    f. This is followed by intermittent/clonic seizures finally leading to convulsions that could be brought about by external stimuli.
    g. Subsequently, the convulsions become persistent leading to ophisthotonous, paddling, and clamping of the jaws.
    h. Convulsions involving muscles exertion results in rising body temperature and sometimes respiratory failure. The animal may appear normal between the seizures.
    i. Frothy salivation, mydriasis, diarrhoea, urinary incontinence and bradycardia/ tachycardia are also noted.
2. In chronic toxicity, clinical symptoms often appear due to the release of the compound stored in the body fat.
    a. This process is hastened by starvation, excess physical activity and sudden fall in body temperature.
    b. These stresses cause adrenaline release, which initiates lipolysis.
    c. The stored compound thus finds its way into the blood stream and when concentrations are sufficiently high in the nervous system, they cause clinical signs of intoxication or even death.
    d. Vocalisation, inability to distinguish surroundings, loss of interest in food and water are seen.
    e. Nervous symptoms are similar to those seen in encephalitis or meningitis.

### Post mortem findings

1. Hardly any lesions are seen but cyanosis occurs if death is sudden.
2. Protracted illness may result in chronic cases and lesions like congestion of the lungs, liver and kidney could happen.

3. If the animal has died when the body temperature was very high, a blanched appearance of all organs is observed.
4. Heart stops at systole and haemorrhages are seen on the epicardium.
5. Lesions on the lungs, if observed are suggestive of pneumonia.
6. Pulmonary oedema may be noticed if the animal is affected for more than a few hours. Blood tinged froth is noticed in the respiratory system.
7. Nervous symptoms are due to receptor activities in sodium and potassium channels (for DDT and analogs) and at GABA-mediated chloride channels (for cyclodienes).

## Chemical test

| | Procedure | Observation | Reaction |
|---|---|---|---|
| **Test for DDT/BHC** | a) The residue is dissolved in n-hexane & ethanol. Small quantity of NaOH pellet is added. Evaporated in a water bath. Cool. 4 drops of $CCl_4$ added. Shake. Add a mixture of $H_2SO_4/HNO_3$. Shake | Green colour | DDT/BHC present |
| | b) Heat a small quantity of the extract with 0.5% solution of hydroquinone in sulphuric acid | Wine red colour | Specific for DDT |
| **Test for dieldrin, endrin & aldrin** | To a little of the extract prepared in xylene add 2 ml of conc. $H_2SO_4$ | Intense red purple colour | Purple (endrin) Red (dieldrin) Slow generation of colour (aldrin) |
| **TLC test for OC compounds** | a) (For very small quantities). Prepare a TLC plate. Develop the residue plated on it using hexane or benzene 90/10. Spray diphenylamine reagent (0.5 g in 100 ml acetone or ethyl alcohol). Keep in sunlight | Gray spot | OC compound present |

## Diagnosis

1. Chemical analysis of the appropriate samples leads to confirmed diagnosis. Suspected food material, stomach contents, fat, kidney, liver and brain should be subjected to chemical analysis.
2. Since OC compounds are present extensively in the environment, a positive laboratory report need not necessarily indicate poisoning by these compounds unless they are present in very high concentrations.
3. Concentrations expressed in ppm, are suggestive of toxicosis when supported by case history, clinical signs and postmortem findings. Most veterinary toxicologists rely only on concentrations in the brain and gut for confirmed diagnoses. GI contents could suggest recent intake of high oral doses.
4. Residues in the brain rather than in the body fat are of diagnostic significance.
5. The liver and kidney of the dead animal may contain ppm concentrations if the exposure has been recent and in excess.
6. A piece of liver and kidney can be put in a jar with some flies to see if they die to verify toxicosis. Although non-specific, this must be confirmed by laboratory analysis.

## Differential diagnosis

1. Salt poisoning – history and absence of elevated body temperature.
2. Strychnine poisoning – convulsions are tonic and not clonic and there is no behavioural aberrations or locomotor irregularity.
3. Cholinesterase-inhibiting insecticides like OPs and carbamates could cause behavioural changes. No hyperthermia may be observed but there would be a quick response to atropine.
4. Lead poisoning – fewer abnormal postures in most animals, less frequent hypothermia and less frequent convulsions.
5. Urea – less abnormal posture and hallucination induced behaviour. Convulsions are tonic and severe in nature. Ammoniacal smell of the stomach contents.

## Lethal dose

1. Acutely toxic oral dose of some chlorinated hydrocarbon insecticides for rats:

| Common Name | Rat acute oral $LD_{50}$ (mg/kg) |
|---|---|
| Endrin | 3-43 |
| Aldrin | 38-60 |
| Dieldrin | 40-64 |
| Toxaphane | 40-94 |
| Heptachlor | 40-162 |
| Endosulphan | 18-110 |
| Lindane | 76-177 |
| DDT | 87-400 |
| Mirex | 235-312 |
| Chlordane | 283-590 |
| TDE | 400 |
| Methoxychlor | 5000-6000 |
| Perthane | 6600-9000 |

## Treatment & management

1. OC compounds have no specific antidotes.
2. Dermal contamination should be treated by washing with detergents.
3. A cold water spray may also help to bring down the body temperature.
4. Activated charcoal is recommended orally.
5. Convulsions can be controlled by using muscle relaxants or anesthetics like barbiturates and chloral hydrate.
6. A calm environment should be maintained to reduce the external stimuli which can lead to convulsions and delirium.
7. Oral administration of phenobarbitone is also recommended as a line of treatment. (Phenobarbitone 12-20 g I/V can be given)
8. Calcium borogluconate (CBG) I/V with glucose saline could be administered to avoid liver damage and neutralize the preconvulsive hyperkalemia.
9. CBG in a dose of 2-3 litres and activated charcoal at a dose of 1-2 g per kg body weight are recommended for administration, however, this is difficult in large animals.

10. Xylazine at a dose rate of 100-150 mg/ton is worth trying and can also be given I/M.
11. If CNS depression has already set in, further administration of CNS depressants is contraindicated.
12. Prognosis is grave as the recovery may take a long time and it is not practically possible to continue the treatment over such long periods. This depends on the dose of the poison and the time frame available for treatment. There seems to be some redistribution of toxin from the brain over a day or two in some poisoned small animal patients.

# ORGANOPHOSPHORUS INSECTICIDES

## Introduction

Organophosphates have largely replaced OC compounds both in veterinary practice as well as in agriculture. Hence, occurrences of cases of poisoning using these compounds have been noted. Many of these compounds are used as insecticides, acaricides, soil nematicides, fungicides and in a variety of similar agrochemicals. Compared to organochlorine insecticides, they are much less persistent in the environment and hence biomagnification is less. They are very unstable in hot, humid climates. They exhibit varying degrees of toxicity and environmental degradation. Based on their chemical structure they are classified into several groups. Their classification based on their mode of action is as follows:

1. Direct acetylcholine esterase inhibitors: Eg. dichlorvos (DDVP), DFP, TEPP.
2. Indirect acetylcholine esterase inhibitors: These agents require the activation of oxone in the body for their toxic action, eg malathion, parathion and chlopyrifos.

Commonly used OP compounds are chlorfenvinphos, chlorpyriphos, coumaphos, demeton, diazinon, dichlorvos, dimethoate, ecothiophate, fenthion, malathion, methyl parathion, parathion (now banned), phorate, phosmet, profenphos, ronnel, (Fenchlorphos), ruelena, tetrachlorvinphos, tetraethyl pyrophosphate (TEPP), Trichlorfon.

OP compounds are absorbed by all body surfaces as also through the GI tract, lungs and eyes. They are rapidly distributed throughout the body. They do not accumulate in specific tissues or in fat as in the case of OC compounds. Among OP compounds, chlorpyrifos is retained longer in the body and exhibits more toxic effects as well.

## Possible sources

Contaminated feed and water, consuming crops/forage that is dusted/sprayed with organophosphates and drinking water out of empty pesticide containers. OP compounds are available in the market both for agriculture and veterinary use.

## Verification of toxicosis

### Clinical signs

1. Symptoms of poisoning suggest excessive cholinergic stimulation.
2. Exposure to vapour or aerosol can cause adverse respiratory symptoms like nasal discharge, tightness of the chest, wheezing due to broncho-constriction and excessive bronchial secretion.

3. Exposure of the eyes causes miosis (constricted pupil) and increased intra ocular pressure. In systemically exposed animals, miosis or mydriasis could be seen.
4. Symptoms are classified into muscarinic, nicotinic and central:

   a. Muscarinic effects are the first to appear and include hypersalivation, miosis, frequent urination, colic, diarrhoea, bradycardia related to vagal stimulation, and dyspnea due to both bronchoconstriction and increased bronchial secretions as a result of reduced stimulation of the muscles of respiration from the medulla.

   b. Nicotinic effects include fasiculations of the muscles and weakness.

   c. Central effects are indicated by nervousness, ataxia, apprehension and seizure. Excitement is the reason for tachycardia in some animals.
5. Signs appear within minutes to hours of ingestion of the compound and are influenced by the specific compound involved – its dose, and the route of entry.
6. In acute poisoning, the primary clinical signs are respiratory distress, collapse and death resulting from paralysis of the respiratory muscles.

**Post mortem findings**

1. In acute poisoning, no specific lesions are noticed.
2. Pulmonary oedema, congestion, cyanosis, haemorrhages and oedema of the heart, bowel as well as other organs may be noticed.
3. Cerebral oedema and necrotic patches on the skeletal muscles could occur
4. The intestinal tract may be dilated and fluid filled.
5. Animals that survive for more than a day may lose some weight and become dehydrated due to continuous purging.

## Chemical test

| | Procedure | Observation | Reaction |
|---|---|---|---|
| **Parathions** | a) The test for OP compounds having P-nitro phenol ring in it. The ether extract is used. A portion of the residue is extracted in ethyl alcohol and taken in a china dish. Add a piece of KOH. Warm. This is known as the PNP (paranitrophenol) test. | Intense yellow colour | Formation of P-nitrophenol |
| | b) The residue is hydrolysed using alcoholic potash and reduces to P-aminophenol using Zn/HCl mixture by boiling. Cool and add $NaNO_3$ in excess to diazotise the amino group. Add beta napthol in NaOH. | Orange colour | Diazotisation & coupling produce phenol azo beta napthol |
| **All OP compounds (Thin Layer Chromato-graphy Test)** | a) For every quantity of OP compound, prepare TLC plate. Develop the OP using hexane: acetone 90:10. Spray palladium chloride solution (0.5 gm/100 ml water). Add a drop of conc. HCl | Brown yellow colour | Applicable to all OP compounds containing sulphur |
| **Detection of elements for OP, OC & carbonate compounds (to be done after preliminary tests)** | The results are fused with metallic sodium & extracted with water, boiled and filtered. Using the sodium fusion extract, the following tests are conducted | | |
| | a) Add freshly prepared ferrous ammonium sulphate followed by drops of HCl to dissolve the ppt. Add drops of neutral ferric chloride | Green blue colour | Prussian blue. Presence of nitrogen |
| | b) A small quantity of the extract is boiled with conc. $HNO_3$. Add freshly prepared ammonium molybdate, boil & cool | Yellow colour | Presence of phophorus |
| | c) To a small quantity of the extract dilute solution of sodium nitroprusside is added | Violet pink colour | Presence of sulphur |
| | d) Boil with conc. $HNO_3$. Add dilute $AgNO_3$ | White ppt followed by yellow ppt | White ppt indicates chlorine & yellow – bromine |

## Diagnosis

1. In the field, diagnosis of acute poisoning relies mainly on the case history and symptoms, as well as recognition of the offending insecticide and inhibition of chlolinesterases in the patient.
2. Determination of the choline esterase activity in the blood and the brain can be done, however, results need not always be corroborative with the clinical signs. The important point is the rate at which the enzymatic activity is reduced, but this is of limited value under field conditions.
3. Inhibition of cholinesterase, is usually largely irreversible, even upon prolonged incubation.
4. Frozen samples of stomach contents could be analysed for the pesticides.
5. OP compounds are generally stable in acids.
6. An inhibition of 50-70% of activity may indicate clinical manifestations.
7. A depression of acetylcholine esterase activity to less than 25% of the normal value is considered to be compatible with a case of acetylcholine esterase inhibitor poisoning.
8. In field conditions, the response to atropine treatment is a good indicator of OP poisoning.

## Differential diagnosis

1. Depression of the choline esterase activity is noticed both in organophosphorus and carbamate poisoning.
2. Examining the reversibility of cholinesterase activity is important to help distinguish toxicosis due to carbamates from organochlorines.
3. The carbamates tend to be shorter acting than most OPs.

## Lethal dose

1. A number of physical and chemical factors affect the toxicity of these compounds:

   a. Rate of decomposition (water, sunlight, alkali, microbes, heavy metals like Cu and Fe are all decomposing agents that augment the process of decomposition).

   b. Storage for long periods may sometimes result in toxic isomers.

   c. Toxicity varies between species. This is due to the differences in the enzymatic activation and degradation of the compounds. It may be noted that mammals possess a group of enzymes known as phosphoryl phosphatases, which make them less vulnerable to toxicosis compared to insects in which these enzymes are absent. Some of the OP compounds act synergistically.

d. Often organic solvents are used along with emulsifying agents in certain commercial formulations. Hence these are more toxic than the pure OP compound. Some of the pesticides are incorporated with sticking agents to prevent washing out in rains. These sticking agents can cause pesticides to adhere to animals. Formulations are often dust, wettable powders, emulsifiable concentrates and solution concentrates.Their $LD_{50}$ varies from 1-5 mg/kg (Dimefon) to 4000 mg/kg (Tetrachlorvinphos).

## Treatment & management

1. Treatment is aimed at blocking the action of the toxicant, reviving the enzyme and preventing further absorption of the toxicant.
2. Atropine blocks the central and peripheral effects of cholinesterase inhibitors at the level of muscarinic receptors when given at the dose of 0.1-0.2 mg/kg. The ideal dosing schedule is to give 1/3 of the total dose I/V and the rest I/M. The I/M administration is for protracted action. Dilated pupils, alert appearance and the cessation of salivation are signs suggesting atropinisation. However, animals should be monitored primarily to observe easing of respiration (as respiratory failure is the usual cause of death). Atropine does not counter the nicotinic cholinergic effects like muscle fasiculations and muscle paralysis. Hence, massive OP toxicosis may still cause death even under the atropine umbrella. Excessive atropine administration could cause excitement and delirium.
3. Diphenhydramine (4 mg/kg orally) may help to control refractory nicotinic signs. However, it should not be used in combination with atropine.
4. The second line of treatment aims at reactivating the inhibited choline esterase activity. Reactivating drugs are 2-PAM (Pyridine – 2 aldoxime), DAM (Diacetyl monoxime), MINA (Mononitroso acetone) and obioxime chloride. The dose of 2-PAM in animals is 20-50 mg/kg as a 10% solution I/M or slow I/V, repeated when needed. It may be required to repeat the I/M treatment at 8-12 hour intervals, 2-3 times. Early administration of the oximes could help to wrest out the enzyme quickly. Once the phosphorylated enzyme becomes "aged", the response to reactivation decreases. Ideally the oximes should be given within 24-48 hours.
5. Washing the skin in the case of dermal contamination prevents further absorption. In case of ingestion, activated charcoal (3-6 g/kg as a water slurry) may be administered orally. This will help to eliminate the OP compounds through the faeces. Phenothiazine tranquilisers should not be used. Succinyl choline treatment should also not be carried out for at least 10 days after poisoning.

# CARBAMATE INSECTICIDES

## Introduction

These are anti cholinesterase insecticides, similar in action to OP compounds. Unlike OP compounds, their inhibition of the choline esterase enzyme is not permanent. Hence, enzyme reactivators like oximes are not used in treatment of toxicosis.

## Possible sources

1. Contaminated feed and water, eating of crops or forage dusted or sprayed with carbamates and drinking of water from empty pesticide containers.
2. Carbamate insecticides are extensively used in agriculture. One granular formulation namely carbofuran (Furadan®) is used for malicious poisoning.

## Verification of toxicosis

### Clinical signs

1. Symptoms are similar to that of OP compounds poisoning as the mechanism of action is similar.
2. Excessive salivation, diarrhoea, abdominal cramping, dyspnea, miosis, cyanosis, muscle fasiculations and convulsions are all signs of poisoning.
3. In extreme cases, tetany may be followed by weakness and paralysis.
4. As with OPs, death is usually from hypoxia due to respiratory failure.

### Post mortem findings

1. Similar to those of OP compounds.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) The residue is boiled with Tollen's reagent (0.1 N $AgNO_3$ & 5 N $NH_4OH$ in 1:5 proportion) | Mirror formation | Reduced to Ag |
| b) Ethyl methyl ketone extract developed on TLC and sprayed with Tollen's reagent. Heated in an oven between 80-100°C | Black spots | For all carbamates |

## Diagnosis

1. Clinical history and symptoms confirm diagnosis.
2. Chemical analysis of the stomach contents could help.
3. Inhibition of cholinesterase enzyme which is usually reversible upon prolonged incubation.
4. Positive response to atropine in a clinical setting.

## Differential diagnosis

1. It is difficult to differentiate from OP poisoning.

## Lethal dose

1. Table of common carbamate insecticides with acute oral $LD_{50}$ in rats

| Common Name | Rat acute oral $LD_{50}$ (mg/kg) |
|---|---|
| Aldicarb | 0.6-1 |
| Carbofuran | 5-11 |
| Methomyl | 17 |
| Aldoxycarb | 27 |
| Aminocarb | 30-60 |
| Dioxacarb | 60-80 |
| Methiocarb | 60-130 |
| Promecarb | 74-90 |
| Propoxur | 83-104 |
| Metalkamate | 87-170 |
| Isocarb | 128 |
| Bendiocarb | 143 |
| Primicarb | 147 |
| Carbaryl | 307-850 |
| Ethiofencarb | 411 |

## Treatment & management

1. If a sample of the suspected poisonous container can be obtained, antidotes may be found noted on the container. Symptoms of carbamate and OP insecticides poisoning are almost the same. In the former only atropine is used for treatment while in the latter both atropine and oximes are used. Hence, if the antidote mentioned on the container is only atropine, it can be inferred (reverse inference) that the poison under suspicion is a carbamate and not an OP.

# PYRETHROIDS

## Introduction

This group consists of the oldest natural insecticides in use. Originally they were sourced from the pyrethrum (*Chrysanthemum*) species, but due to their poor stability, new synthetic derivatives have been developed. Most synthetic pyrethroids are esters of carboxylic acids. Some may be similar to natural pyrethrins like allethrin, while others like flucythrinate are different compounds. Commonly used pyrethroids are allethrin, cypermethrin, decamethrin, fenvalerate and tetramethrin. They are not quickly absorbed through the skin.

## Possible sources

Ingestion of insecticides (plant protection chemicals), which are used extensively in agriculture and synthetic pyrethroids, which are used as veterinary ectoparasiticides. Direct ingestion of concentrated insecticides is also possible.

## Verification of toxicosis

### Clinical signs

1. Signs may appear within minutes of ingestion or may at times be delayed in cases of dermal absorption.
2. Since they are not cumulative, chronic toxicity is not common.
3. Early toxicosis results in hypersalivation, diarrhoea, mild tremors and hyperexcitability. At this stage, poisoning may be confused with OP or carbamate toxicosis.
4. In severe cases, hyperthermia or hypothermia, severe tremors, disorientation and seizures are observed.
5. Death may result due to respiratory failure.
6. Some pyrethroids may produce allergic reactions.

### Post mortem findings

1. Not specific and seldom corroborated with clinical symptoms.

## Diagnosis

1. Based on case history and symptoms.
2. Chemical analysis is difficult under field conditions. Samples can be submitted to the lab. But unfortunately in India there are no such laboratories where analysis can be done. No database is therefore, available.

## Differential diagnosis

Poisoning may be confused with OP, carbamate or paraquat toxicosis.

## Lethal dose

1. Mammalian toxicity is comparatively less.

## Treatment & management

1. Seizures are controlled by barbiturates (pheno/pento barbiturates 6 mg/kg body weight I/V). Phenobarb is the preferred anticonvulsant for most species. Pentobarbital must be used as an anesthetic, as stimulation at lower doses is observed (e.g. seen during recovery).
2. Methocarbamol 50-200 mg/kg body weight and diazepam 0.2-2 mg/kg could be tried.
3. If oral administration is possible, slurry of activated charcoal (2-8 g/kg) followed by saline purgatives, magnesium sulphate, sodium sulphate at the rate of 0.5 mg/kg could be tried.
4. In cases of dermal absorption, the skin may be washed smoothly with soap but without scrubbing as this will promote dermal absorption.

# FORMAMIDINE INSECTICIDES

## Introduction

In veterinary practice, amitraz is the only commonly used formamidine insecticide. It is readily absorbed orally as well as parentrally.

## Possible sources

Insecticides such as amitraz.

## Verification of toxicosis

**Clinical signs**

1. Signs are similar to those produced by choline esterase inhibitors or pyrethroids.
2. Bradycardia, ataxia, depression, disorientation, anorexia, diarrhoea and seizures are observed.
3. Chronic toxicity is unlikely.

**Post mortem findings**

1. Liver enlargement is observed.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Data/information inadequate | | |

## Diagnosis

From case history and clinical signs.

## Differential diagnosis

Data/information inadequate.

## Lethal dose

1. Acute oral $LD_{50}$ in rats is 800 mg/kg body weight.

## Treatment & management

1. No specific antidotes are available.
2. Activated charcoal and saline cathartics could help.
3. Yohimbine administration could be tried at the rate of 0.125 mg/kg I/V.
4. Tolazoline administration is recommended.
5. Atropine should not be used.

# NATURAL INSECTICIDES

## Nicotine

### Introduction

Nicotine is obtained from the tobacco plant – *Nicotiana tobaccum* and is highly toxic. Due to its strong smell, natural ingestion is very rare. It is readily absorbed through the lungs and GI tract. Nicotine is an alkaloid and is absorbed even through the intact skin.

### Possible sources

The tobacco plant – *Nicotiana tobaccum*. A decoction of tobacco leaves is used both as a natural insecticide as well as for malicious purposes in villages.

### Verification of toxicosis

**Clinical signs**

1. Severe convulsions of clonic type may be noted.
2. Confusion and respiratory distress are commonly observed.
3. In the terminal stages, the animal becomes comatose before dying.

**Post mortem findings**

1. Pale mucous membranes, dark blood, haemorrhages on heart, lungs and congestion of the brain.

### Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Data/information inadequate. | | |

### Diagnosis

From history and clinical symtoms.

### Differential diagnosis

OP and carbamate insecticides; paraquat, zinc phosphide, organochlorine insecticides, and metaldehyde toxicosis produce similar symptoms.

### Treatment & management

1. No specific antidote is available.
2. Use of activated charcoal is advised. Gastric lavage and artificial respiration are not possible in elephants.
3. Mildly affected animals may recover quickly with symptomatic treatment.
4. In acute cases, prognosis is very poor.

# HERBICIDES

## Introduction

As the word indicates, these are phytotoxic chemicals used to destroy various weeds and other harmful plants, both in agriculture as well as on industrial premises. Some are used to defoliate plants prior to harvest. Others are used to kill undesirable plants prior to or after planting the desired ones. They are classified differently depending upon their chemical nature and the group of plants upon which they act. Very few poisoning cases are reported due to these chemicals. Some inorganic hebicides, rarely used nowadays, are arsenicals and sodium chlorate. Their poisoning is naturally due to the arsenic and chlorate moieties respectively. Some commonly used group herbicides are:

1. Chlorophenoxy acid derivatives such as 2,4-dichlorophenoxy acetic acid (2,4-D, Feronoxone), 2,4,5-trichlorophenoxy acetic acid (2,4,5-T), MCPA, MCPP.
2. Dinitro compounds or dinitrophenols e.g. DNP, DNOL, DNPP
3. Bipyridyl compounds e.g. Diquat, Paraquat. The latter is extensively used
4. Triazine compounds like atrazine are extensively used in forestry and agriculture.

## Possible sources

Ingestion of foliage sprayed with insecticides/weedicides. Ingestion of spilled concentrates.

## Verification of toxicosis

### Clinical signs

1. Phenoxy acid derivatives or chlorophenoxy compounds produce GI symptoms like diarrhoea and salivation, as well as depression, weakness, especially of the posterior portion, tremors, paralysis and rarely, respiratory paralysis.
2. Poisoning by bipyridyl compounds produce GI and CNS symptoms. There may be difficulty in breathing due to pulmonary oedema with paraquat. Pulmonary effects can be progressive, lethal, or chronic and debilitating with paraquat.

### Post mortem findings

1. Phenoxy acid derivatives or chlorophenoxy compounds:
   a. Gastritis, swollen and fragile liver.
   b. Oral ulcers could be present.
   c. Bright green undigested feed may be seen as a result of gastrointestinal stasis.
   d. The kidneys are enlarged and show renal congestion with tubular degeneration.
   e. Mesentric lymph nodes and vessels could be enlarged and hyperaemic.

f. Hydropericardium with epicardial haemorrhage could also be seen.

2. Paraquat (Gramaxone):
   a. Pulmonary congestion, oedema and haemorrhage.
   b. Tongue ulcers are observed.
   c. Other findings include failure of lungs to collapse once the thoracic cavity is opened, areas of haemorrhage, fibrosis and atelectasis.
   d. Microscopic lesions include necrosis of alveolar epithelium followed by progressive alveolar and interstitial fibrosis, and alveolar emphysema.
   e. Renal proximal tubular degeneration and moderate centrilobular hepatic degeneration could also be seen. Renal lesions and failure may be noted more frequently with diquat.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Data/information inadequate. | | |

## Diagnosis

Case history, clinical signs and lesions confirm diagnosis.

## Differential diagnosis

Data/information inadequate.

## Lethal dose

1. For trizine compounds oral $LD_{50}$ ranges between 1000-5000 mg/kg depending on the compound.

## Treatment & management

1. Treatment for poisoning by chlorophenoxy acid derivatives includes prevention of absorption and symptomatic care as no specific antidotes are available. Administration of fluids and slight alkalinization of the urine may help in promoting elimination.
2. Treatment for toxicosis from bipyridyl compounds is done symptomatically as no specific antidotes are available. These compounds bind tightly to clays, thus clays are preferred as adsorbents over other more expensive products like activated carbon.

# FUNGICIDES

## Introduction

Fungicides are used extensively in agriculture to eradicate fungal diseases in plants and are used to treat seeds to avoid seed-borne plant diseases. They are classified into two groups: organic and inorganic.

Among organic fungicides, organo mercuric compounds (phenyl mercuric chloride, ethyl mercuric acetate and methyl mercury) are of toxicological importance. Common inorganic fungicides include copper sulphate itself and other copper sulphate containing compounds like Bordeaux mixture. A group of fungicides include dithiocarbamates (e.g. Zineb, Maneb), chlorophenols (e.g. Pentachlorophenol, Trichlorophenol), phthalimides (e.g. Captan, Captafol), benzimidazoles (e.g. Benomyl, Thiophanate) and organotin compounds (e.g. Triphenyl tin, Tributyl tin, Triethyl tin). Other miscellaneous fungicides are pentachloro nitrobenzene (PCNB) and dinocap. Most of these have a very high $LD_{50}$ and clinical poisoning cases are very unlikely. Organotins are rather toxic and difficult to counter. They may be encountered in wood preservatives and antifouling paints.

## Possible sources

1. Plants and seeds treated with fungicides.
2. Consumption of polluted water, herbage or seeds treated with mercury fungicides.

## Verification of toxicosis

### Clinical signs

1. Symptoms of poisoning resulting from ingestion of inorganic fungicides like bordeaux mixture are identical to those for copper poisoning.
2. Mercuric fungicide poisoning symptoms include confusion, ataxia, staggering gait, prostration, convulsions and finally death.
3. Large animals may show Central Nervous System depression.
4. Bronchopneumonia, epistaxis, haematuria, hyperpyrexia and blood in faeces are some other symptoms.
5. Eczema, pustules, ulcers and keratinisation could be seen on the skin.
6. Dehydration and weakness, finally leading to death.

### Post mortem findings

1. Congestion and necrosis of the gastrointestinal tract
2. Enlarged and congested kidney with petechiae and ecchymosis

3. Degeneration of the parenchymatous organs like the liver
4. Lesions of the brain including demyelination and axonal degeneration are observed.

## Chemical test

Test for copper in case of suspected copper fungicide poisoning and for zinc in zinc fungicide poisoning.

## Diagnosis

They are a large variety of chemicals which are copper (organic and inorganic), zinc (organic) and mercury based (organic mercurials) as well as other synthetic chemicals. Since there are a wide variety of chemicals in this group it is difficult to identify them using simple lab tests, often requiring sophisticated instrumental diagnosis for confirmation.

## Differential diagnosis

Data/information inadequate.

## Lethal dose

Data/information inadequate.

## Treatment & management

1. Includes British Anti-Lewisite (BAL) administration in addition to symptomatic treatment.

# RODENTICIDES

## Introduction

Rodenticides are used to destroy rodents, especially rats and mice. They are also poisonous to other vertebrate vermin. Due to their easy availability and extensive use, elephants could also be affected. There have been many instances where captive elephants were poisoned with zinc phosphide, a popular rat poison. Although a large number of rodenticides are available, only those with clinical significance of toxicosis are being discussed.

1. Elemental phosphorus: smeared preferably on oil-based bait. Rats are attracted and consume the bait.
2. Zinc phosphide: A gray powder with 14-18% phosphorus and 70-80% zinc. It is an irritant of the gastro-intestinal tract leading to gastroenteritis. It can also damage the lungs and could be associated with profound seizures.
3. Other rodenticides of recent origin are anticoagulants: warfarin, bromodioline, pindone.
4. Cholecalciferol (Vitamin D3) is another rodenticide, which is not used in India and also is not toxicologically important in elephants.
5. ANTU, alpha naphthyl thiourea.

## Possible sources

Accidental ingestion of baits extensively available in the market.

## Verification of toxicosis

### Clinical signs

1. In acute cases of zinc phosphide poisoning, depression, tremors, colic, ataxia, weakness, prostration, gasping, dyspnea and struggling are observed.
   a. Convulsions could cause a rise in body temperature
   b. Coma followed by death may occur in these cases
2. In poisoning cases due to the use of anticoagulants, animals may die from bleeding from even minor wounds.

### Post mortem findings

1. Elemental phosphorus:
   a. Severe gastroenteritis, fatty liver, multiple haemorrhages and black tarry blood that does not clot.
   b. The gastric contents have a garlic odour and could be phosphorescent.

c. Lesions due to severe hepatitis and renal damage could be present.

d. There could also be evidence of jaundice.

2. Zinc phosphide:

   a. The stomach contents would have a characteristic "dead fish" or acetylene odour due to emission of phosphine gas which could be a toxic hazard to persons conducting the post-mortem examinations

   b. Gastroenteritis, which is often haemorrhagic, is noticed.

   c. Other infrequent lesions could be visceral congestion and pulmonary oedema. Other non-specific and inconsistent lesions are myocardial degeneration, pale yellow fatty liver and degeneration of the liver and the kidneys.

3. Anti-coagulants:

   a. Haemorrhages of the mucous membranes, sub-cutis and internal haemorrhaging of the internal organs and other parts of the body may be observed. Most haemorrhages are ecchymosis, rather than petichae.

   b. Blood clotting does not occur and centrilobular hepatic necrosis resulting from anaemia and hypoxia is observed.

4. ANTU, alpha naphthyl thiourea:

   a. Cyanosis and dark coloured arterial blood could be observed.

   b. Thoracic cavity partially filled with colourless or pale reddish fluid is noted.

   c. Oedematous lung, froth in the trachea and bronchi, hydrothorax and hyperaemic tracheal, bronchial and gastric mucosa are noticed.

   d. In ANTU, pulmonary oedema, hemorrhage due to trauma, alfatoxicosis, bracken fern, mouldy sweetclover ingestion or moldy lespedeza from anticoagulants, liver and lung damage by zinc phosphide (includes paraquat, pyrrolizide alkaloids) are observed.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Data/information inadequate. | | |

## Diagnosis

History of use and availability of rodenticides in the vicinity, clinical signs and post-mortem lesions.

## Differential diagnosis

Data/information inadequate.

## Lethal dose

Data/information inadequate.

## Treatment & management

1. Sodium carbonate is used in the treatment of zinc phosphide poisoning to neutralize the stomach acidity.
2. Further, artificial respiration and demulcents are not possible in elephants.
3. Vitamin K is a specific antidote used in cases of poisoning due to ingestion of anticoagulants.

# STRYCHNINE

## Introduction

Strychnine is an alkaloid obtained from the seeds of the tree *Strychnos nuxvomica*. It is also found in the seeds of a climbing shrub *Strychnos ignati* – a plant found in southeastern Asia and the Philippines. The seeds also contain other alkaloids like brucin, loganin, caffeotannic acid and proteins. Mammals are highly susceptible to strychnine poisoning, more so than birds. Strychnine has a long history of being used as a rodenticide. The chemical is rapidly absorbed through the GI tract with the CNS being the site of action. Absorption from the small intestine is complete. A high concentration may be seen in the blood, liver and kidney tissues. Strychnine retains its toxicity indefinitely in the bait and carcass of the animal due to its stability as an alkaloid. As a result of the poison's rapid action, the affected animal is usually found close to the bait.

## Possible sources

1. The seeds of the tree *Strychnos nuxvomica*.
2. Decoctions with strychnine produced in villages. Small quantities are added as a stimulant for ruminal atony. Strychnine as such is used as a rodenticide all over the world. The therapeutic use of strychnine in human medicine is banned.

## Verification of toxicosis

### Clinical signs

1. Strychnine inhibits competitively and reversibly the inhibitory neurotransmitter glycine in the spinal cord and the medulla. This results in maximum response to even a minimal stimuli.
2. Stimulation of extensor muscles promoting extensor rigidity leading to tonic and clonic seizure could be noted.
3. Death occurs as a result of asphyxia combined with acidosis.
4. The onset of action is very rapid, with signs appearing within 30-60 minutes of ingestion. Poisoned animals may die in less than an hour after ingestion if dose is high.
5. Absorption on a full stomach is delayed.
6. Restlessness and muscular twitching could progress to intermittent tetanic seizures that could appear spontaneously or be initiated by the slightest stimuli like touch, clapping of hands or even a sudden bright light. These spasms which result from mild stimuli increase in severity.
7. Breathing could become laboured and irregular.

8. In advanced conditions, extensor rigidity could prevail and the animal could show an ophisthotonous condition ("Sea-horse like stance").
9. Death is mainly due to spasm of respiratory muscles, resulting anoxia and exhaustion due to paralysis of the respiratory centre.

**Post mortem findings**

1. Strychnine can be detected in the carcass even months after the death of the animal.
2. Subcutaneous or intramuscular haemorrhages could result from trauma along with cyanosis and asphyxia.
3. Rigor mortis sets in fast due to muscular contractions.
4. Depending on the dose and the time prior to death, the highest concentrations of strychnine could be found in the digestive tract, blood, liver or kidney tissues.
5. Although strychnine is a neurotoxin, it does not appear to concentrate in nervous tissues.

## Chemical test

| Procedure | Observation | Inference |
|---|---|---|
| Mix residue with conc. $H_2SO_4$ & add a pinch of $MnO_2$ carefully | Pink colour is developed at the boundary | Strychnine present |

## Diagnosis

1. Tentative diagnosis is from history of exposure and clinical signs exhibited by the animal.
2. Strychnine can be detected in the stomach contents, liver, kidney and even in the urine.

## Differential diagnosis

1. Strychnine poisoning should be differentiated from poisoning with urea or other non-protein nitrogen sources to metaldehyde, anti-choline esterase insecticides like organophosphates and carbamates, organochlorines, fluoroacetate, nicotine, zinc phosphide and 4-aminopyridine.

## Lethal dose

1. Oral $LD_{50}$ in large animals varies from 0.1-1 mg/kg body weight.

## Treatment & management

1. Prognosis is good, provided prompt and quick treatment can be instituted.
2. The main objective of the treatment would be to control the convulsions and prevent asphyxia and exhaustion.
3. The animal must be kept in a quiet environment without giving any chance for sensory stimuli.
4. Gastric lavage with $KMnO_4$ is not possible in elephants.
5. Diuretics and urinary acidifiers could be used to accelerate urinary excretion.
6. Activated charcoal is also recommended.
7. Pentobarbital anesthesia is the treatment of choice to control convulsions.
8. Chloral hydrate with pentobarbitone could also be tried.
9. Phenobarbitone is the most effective and long-acting among the barbiturates. Phenobarb is preferred because it acts longer than pentobarb and frequency of administration can be reduced as and when required. The product is also much cheaper.
10. The dose of phenobarbitone varies from 1-15 mg/kg in dogs, however in all cases of poisoning it is the effect that determines the dose, depending on the degree of poisoning. In most animals, the dose of pentobarbitone required is 4.4 mg/kg I/V.
11. Dose of chloral hydrate in large animals is 23-30 g/100 kg and its action is very quick. However, chloral hydrate is not easily available. Phenobarbitone and even other barbiturates are not commonly available.
12. Alternate drugs that can be used are glyceryl guaiacolate-5%, 100 mg/kg I/V. Glyceryl guaiacolate (guaifenesin) is a centrally acting muscle relaxant, mild analgesic and sedative and a skeletal muscle spasmolytic.
13. Other drugs that could be used are xylazine and methocarbamol.
14. Diazepam is recommended in animals, but in elephants the volume required could be a problem.
15. Midazolam is a better compound as it is more potent and is worth trying.
16. Treatment can be continued until the animal shows no further sign of rigidity or muscle contractions while recovering from anesthesia.
17. Recurrence of convulsions could continue as long as there is significant absorption from the gastrointestinal tract.
18. Acepromazine, a phenothiazine derivative has not been found to be useful.
19. Because of rapid elimination from the system, strychnine may not be detected in the urine of a recovered animal.
20. Use of drugs like ketamine and morphine are contraindicated.

# CYANOGENIC PLANTS

## Introduction

Most herbivores instinctively avoid poisonous and toxic plants. However, accidental ingestion of toxic plants has been reported in captive elephants. This happens when they are tethered for long periods of time in areas where such plants grow in plenty. It also happens when elephants raid crops that contain certain poisonous substances containing cyanogens could be released upon chewing. Cyanogenetic plants belong to one of four broad categories of plants that could poison elephants in the wild or during crop raiding. These also include bracken ferns, lathyrus and plants containing oxalates.

There are nearly a hundred species of plants that yield hydrocyanic acid upon acidic or enzymatic hydrolysis during digestion or when crushed or chewed upon. Cyanogenetic glycosides in the plants release hydrocyanic acid (HCN) when hydrolysed by beta glycosidase. HCN is also released when the cell structure is disrupted or damaged by freezing, chopping and chewing. Therefore, upon ingestion after chewing by elephants, such plants release the poison. In certain forages like sorghum, the leaves are richer in HCN than the stems. Seeds of some plants do not contain any HCN. New shoots of young, rapidly growing plants contain large amounts of cyanogenetic glycosides. Microbial action in the rumen can also release hydrocyanic acid. However, since elephants are non-ruminants, they are less affected. In monogastric animals the gastric hydrochloric acid destroys the plant enzymes that hydrolyse the cyanogenetic glycosides, but acid hydrolysis can occur to some extent. This explains why monogastric animals are less susceptible to toxicosis. Even in ruminants, cattle are less susceptible than sheep and goats. In elephants that raid crops like corn and sorghum, the rapid ingestion causes fast release of the gas from the cyanogenetic glycosides. Furthermore, those raiding animals that are already starving may be more susceptible to cyanide poisoning. Depredation coupled with drought may also increase the incidence of poisoning. Since cyanide is rapidly metabolized and eliminated from the body, there is normally little chance for toxicity. Poisoning from the cyanide radical is due to the blockage of the enzyme cytochrome oxidase. This when blocked allows the cyanide radical to combine with the ferric iron of the cytochrome oxidase. This alters the ability of the tissues to consume oxygen and produce ATP. Cyanide also reacts with methhaemoglobin leading to cyan methhaemoglobin formation. However, this is not of much clinical importance, except with regard to therapeutic induction of methhaemoglobinemia. Fresh fodder, i.e. which has cyanide as HCN on a wet basis, is considered dangerous as animal feed. On drying, cyanide volatilises and hence the toxicity of the forage is reduced. Forage containing < 100 ppm HCN net weight is a safe feed. General criteria on a dry weight basis are as follows:

1. 750 ppm – hazardous
2. 500-750 ppm – doubtful
3. < 500 ppm – safe

## Possible sources

1. Some common plants containing cyanogenetic glycosides are *Triglochin maritime* (arrow grass), *Hoecus lunatus* (velvet grass), *Sorghum* spp (Johnson grass, Sudan grass, common Sorghum), *Prunus* spp (apricot, peach, chokecherry, pincherry, wild black cherry).
2. Tapioca or cassava (*Manihot utillisima*) and rubber (*Hevea braziliensis*) contain cyanogenetic glycosides and crop-raiding elephants, especially in Kerala, have access to these crops.
3. When plants die due to frost, new shoots that grow from the base may be dangerous as they are rich in glycosides and elephants like to graze on them
4. Nitrate fertilizers as well as herbicides like 2,4-D on plants can increase the cyanide concentration in the plants.
5. Malicious cyanide poisoning is possible and is being done in animals like dogs. But it is generally not encountered in elephants.

## Verification of toxicosis

### Clinical signs

1. Symptoms occur between 15-20 minutes to a few hours after consumption of the toxic plant.
2. Initially excitement followed by rapid respiration, dysponea, tachycardia, excessive lacrimation, salivation, frequent urination and defecation are observed.
3. Muscle fasciculation is common, leading finally to generalized spasms and death.
4. Mucus membranes, gum and sclera turn bright red and then become cyanotic.
5. Animals stagger and struggle before dying.
6. Several asphyxial convulsions are observed before death.
7. Though respiration stops first, the heart continues to beat for several minutes in the apparently 'dead' condition.
8. The whole episode may not last for more than an hour. Animals that survive for more than two hours, usually recover.

### Post mortem findings

1. Congestion and haemorrhaging in the gastrointestinal tract, trachea, lungs and heart could be noted.
2. Blood does not clot and is observed to be bright red in colour since oxygen is not transported from the blood to the tissues.

3. A smell of bitter almonds emanates from the stomach when opened. This could be hazardous for individuals undertaking the post-mortem examination. Conducting the examination out of doors would reduce the risk.
4. Laboratory analysis of stomach contents, liver and muscle would reveal the presence of HCN.
5. If the postmortem is delayed for some reason, the muscle tissues should be sent for laboratory analysis.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| a) Make distillate alkaline with NaOH and concentrate to about 1 ml. Add a freshly prepared 5% solution of ferrous sulphate and 1-2 drops of a 3% solution of ferric chloride. Shake mixture well and allow to stand for 2 min. Warm gently & acidify with dilute HCl | A blue ppt | Ferric ferrocyanide (Prussian Blue) |
| b) Make distillate alkaline with KOH and concentrate to 1 ml. Add a little yellow ammonium sulphide & evaporate to dryness on a water bath. Extract the dry residue with a few drops of 20% HCl & add a few drops of neutral $FeCl_3$ | Blood-red colour which disappears on the addition of $HgCl_2$ | Ferric thiocyanate which reduces to colourless ferrous thiocyanate |

## Diagnosis

1. Sudden onset of symptoms, bright-red mucus membrane and presence of HCN in the stomach content confirms the diagnosis.
2. Sources of poisoning like remnants of suspected plants, heparinized whole blood, muscle and liver tissues should be sent for cyanide analysis.
3. Ante-mortem blood or blood collected within four hours after death is preferred for analysis.
   a. The specimen should be sealed in airtight containers, refrigerated or frozen
   b. If cold storage is not possible, the specimen should be immersed in 1-3% mercuric chloride solution.

## Differential diagnosis

Cyanogenic plant poisoning should be differentiated from nitrite, nitrate, urea, insecticides (OP), carbamate, (OC) poisoning and acute septicaemic diseases.

## Lethal dose

1. Minimum lethal concentration of cyanide in the blood is approximately 3.0 micrograms per ml.

## Treatment & management

1. Sodium nitrite, 10% in normal saline or in distilled water is administered I/V at the rate of 15-20 mg/kg body weight. This is followed by sodium thiosulphate 20% I/V treatment at the rate of 500 mg/kg.

   a. Sodium nitrite changes some of the ferrous haemoglobin to ferric/methaemoglobin. This methaemoglobin competes with ferric cytochrome oxidase for $CN^-$ and regenerates the cytochrome oxidase to form cyan-methaemoglobin.

2. Nitrite administration should be done very carefully to avoid lethal methhaemoglobinemia.
3. The antidote should be administered immediately. This is, however, difficult with wild elephants.
4. Sodium thiosulphate, on reacting with $CN^-$ in the blood stream or with that which is liberated from cyan methhaemoglobin forms thiocyanate, which is then excreted. Sodium thiosulphate is also recommended orally up to 100-200 g to detoxify any remaining HCN in the stomach.
5. Methylene blue at an I/V dose of 4-22 mg/kg could be given in place of sodium nitrite to produce methaemoglobin. Methylene blue is preferred when nitrate poisoning is suspected, since clinical signs of nitrate and prussic acid poisoning are similar.
6. Sodium nitrite is preferred if a diagnosis of cyanide intoxication is to be confirmed.
7. Plenty of excess salt licks and minerals with extra sulphur help to protect against prussic acid poisoning.
8. A large dose of hydroxy cobalamine (Vit $B_{12A}$) or aquacobalamin (Vit $B_{12B}$) is also recommended. This helps to form a salt complex with additional cyanide in the blood.
9. Cobalt chloride and cobalt EDTA have been used to prevent cyanide poisoning.
10. Oral medication with vinegar, 5 liters in 12-20 liters of cold water may help to slow down the microbial hydrolysis.
11. Activated carbon also helps in adsorbing cyanide.
12. If the animal survives for a day or two, and if further intoxication is prevented, chances are reasonable that it would survive.

# LATHYRUS

## Introduction

Lathyrism is a disease that cripples animals and humans and is produced by the ingestion of plants belonging to the genus *Lathyrus*.

Two types of lathyrism have been identified: osteolathyrism and neurolathyrism. Osteolathyrism occurs as a result of ingesting plants like *Lathyrus odoratus* and *Lathyrus pusillius*. The toxic constituent of lathyrus plants is Beta-N-(gamma-1-glutamyl)-aminopropionitrile (BAPN). BAPN irreversibly inhibits the enzyme, lysyl oxidase. The inhibition of this enzyme prevents cross linking between polypeptide chains in collagen and elastin. This results in weakness of the bones and blood vessel walls. This syndrome is endemic in large domestic animals feeding on these plants. This has been seen in elephants, especially in captive ones.

Neurolathyrism is a neurological condition observed in horses, cattle and human beings. It is often caused by the long-term consumption of seeds of *Lathyrus sativus* and *L. hirsuta*. Outbreaks of lathyrism are commonly observed during periods of famine when large quantities of lathyrus are consumed.

## Possible sources

Plants belonging to the genus *Lathyrus* (Wild Pea, Common Vetch). These plants are primarily found growing in eastern and central India.

## Verification of toxicosis

### Clinical signs

1. Lathyrism is often chronic in nature.
2. Lameness, pain in the feet, difficulty in getting up and in the case of elephants – refusal to lie down indicates toxicity.

### Post mortem findings

1. Often complete de-myelination of the posterior one half to one third of the lateral white columns on both sides of the spinal cord could be seen.
2. Degeneration of laryngeal muscles and recurrent laryngeal nerves has also been noticed.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Data/information inadequate. | | |

## Diagnosis

Case history and clinical signs.

## Differential diagnosis

Delayed neuropathy from orgnosphosphorus compounds, myelinopathy due to heavy metal toxicoses, intramyelenic oedema and secondary axonal damage by uncoupling agencts like bromethalin could be ruled out.

## Lethal dose

Data/information inadequate.

## Treatment & management

1. Nonspecific treatment is administered.
2. Only supportive therapy can be given based on the symptoms.

# BRACKENFERN

## Introduction

Some plants, mainly ferns like *Pteridium aquilinum*, *Pteris aquilina* and *Equisetum arvense and Equisetum hyamale* are widely distributed in temperate regions and upland areas. All parts of the plant, both leaves and rhizomes, contain toxic principles and the concentration of the toxic principles varies with the time of the year. Grazing on bracken fern is usually for want of suitable forage. Animals often like young tender shoots and leaves. Poisoning may be detected after a drought when the preferred fodder is not available. Bracken fern toxicity in non-ruminants is due to the presence of thiaminase, which destroys $B_1$ (thiamine or aneurine) resulting in the deficiency of the vitamin.

## Possible sources

1. Ferns like *Pteridium aquilinum* and *Pteris aquilina*.

## Verification of toxicosis

### Clinical signs

1. It is a cumulative poison which produces signs within 1-3 months depending on the quantity consumed, the animal species and the time of the year.
2. When significant quantities of bracken fern are ingested, signs of acute poisoning due to thiamine deficiency can be seen in simple stomached animals.
3. In ruminants, there could be signs of bone marrow depression.
4. Non-ruminant herbivores are more susceptible to vitamin $B_1$ deficiency.
5. Bone marrow poisoning causing aplastic anaemia in cattle could be noted.
6. Anorexia, incoordination, crouching stands, arched neck and feet placed apart could be observed.
7. In extreme cases, convulsions, clonic spasms and ophisthotonus can be noted.
8. Pyrexia may rarely be encountered.

### Post mortem findings

1. At necropsy, the heart is dilated.
2. There could be multiple haemorrhages all over the carcass in acute poisoning.
3. Enzootic haematuria has been noted in cattle.
4. Increased incidence of urinary tract cancer could also be another potential outcome of chronic exposure to bracken fern.

5. In horses, no specific lesions are seen grossly, however, brain damage from thiamine deficiency has been documented.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Data/information inadequate. | | |

## Diagnosis

1. Identification of plants like horse tail (*Equesetum arvense*) and turnip (*Beta vulgaris*) in the grazing area could indicate the reason for thiamine deficiency.
2. Other neurological disorders and poisoning due to *Crotalaria* sp or ragwort (*Senecio jacobea*) could also be observed.
3. High blood thiaminase level would also indicate toxicity.

## Differential diagnosis

Data/information inadequate.

## Lethal dose

Data/information inadequate.

## Treatment & management

1. Thiamine injections at the rate of 5 mg/kg initially I/V every three hours followed by I/M treatment for several days.
2. Oral supplementation of thiamine for one to two weeks have also been recommended.
3. Antibiotics could also be given to prevent secondary bacterial complications.
4. Habitat improvement is suggested. Deep ploughing and use of herbicides like glyphosate could help but are not practical in the wild.

# OXALATE CONTAINING PLANTS

## Introduction

Although oxalate poisoning in animals by the chemical oxalate is not common, many plants that are palatable contain oxalates. Of the many different species that contain oxalates, halogeton (*Halogeton glomeratus*) and oxalis (*Oxalis pescaprae*) are the most important. They contain oxalic acid as oxalate in the soluble form and could produce acute poisoning. In the case of *Oxalis*, it is often chronic or sub-acute and only occasionally manifests in an acute form. Some fungi also contain oxalate and when consumed, may contribute to oxalate poisoning. Oxalates in plants may be salts of calcium, sodium, ammonium, potassium and magnesium. Of these, sodium and potassium salts are more common. These could also react with calcium in the GI tract to form insoluble salts. Ruminants are less susceptible to oxalate poisoning, however, once absorbed it combines with blood calcium and could cause hypocalcaemia. It ccould damage the kidney tubules due to the deposition of insoluble calcium oxalate crystals. The central nervous system could also be affected when these crystals are deposited in the brain tissues. Oxalates can also cause the breakdown of red-blood cells in the blood.

## Possible sources

1. Major oxalate containing plants belong to the genus *Rumex* or other genera belonging to the families Chenopodiaceae and Oxalidaceae.
2. There are about 70 different species of plants that contain oxalate, some of them being Halogeton (*H. glomeratus*), oxalis (*O. pescaprae*), sour sob, rheum or rhubarb (*Rheum. rhaponticum*), *Sarcobatus* or grease wood (*S. vermiculatus*) and beta or sugar beet (*Beta vulgaris*).
3. Sometimes certain grasses may accumulate oxalate from the soil in concentrations that could be harmful.

## Verification of toxicosis

### Clinical signs

1. Dullness, lowered appetite, salivation, weakness, dysponea, dilated pupils, muscle twitching, convulsion, tetany and finally death from shock. These symptoms are due to hypocalcaemia produced by the oxalate.
2. In sub-acute poisoning, stiff gait and frequent urination are noted.
3. The urine will often be reddish-brown in colour.

**Post mortem findings**

1. Swollen kidneys with oxalate crystal deposits on cut surfaces.
2. Oxalate crystals in the renal tubules that can be seen with the naked eyes.
3. Tissues may show cyanosis and lungs may be filled with dark, purplish blood.
4. Microscopic examination would show renal tubules filled with calcium oxalate crystals.
5. Ruptured tubules could also be noticed.

## Chemical test

| Procedure | Observation | Reaction |
|---|---|---|
| Data/information inadequate. | | |

## Diagnosis

1. Feeding history, clinical signs of hypocalcemia.
2. Urine may contain albumin and at times blood.
3. Reduced PCV and high BUN values with hyperkalemia could be noticed.
4. Suspected plants could be analysed for the presence of oxalate.

## Differential diagnosis

Data/information inadequate

## Lethal dose

Data/information inadequate.

## Treatment & management

1. No specific treatment.
2. Supportive therapy like providing plenty of fresh water to flush the system of oxalates.
3. Calcium borogluconate could be administered through I/V.
4. In advanced cases, the prognosis is poor.

# APPENDIX I

## GUIDELINES FOR SUBMITTING SPECIMENS FOR TOXICOLOGICAL EXAMINATIONS

| | Suspected Xenobiotic for analysis | Specimen required | Amount required | Remarks |
|---|---|---|---|---|
| 1 | Ammonia/urea | Whole blood or serum | 5 ml | Frozen or may add 1-2 drops of saturated mercuric chloride |
| | | Urine | 5 ml | |
| 2 | Arsenic | Liver, kidney | 100 g | |
| | | Whole blood | 15 ml | |
| | | Urine | 50 ml | |
| | | Ingesta | 100 g | |
| | | Feed | 1-2 kg | |
| 3 | Chlorinated hydrocarbons | Cerebrum, ingesta, fat | 100g | Use only glass containers. Avoid aluminium for wrapping the specimens |
| | | Liver, Kidney | 100 g | |
| 4 | Copper | Kidney, liver | 100 g | |
| | | Serum | 2-5 ml | |
| | | Whole blood | 10 ml | |
| | | Feed | 1-2 kg | |
| | | Faeces | 100 g | |
| 5 | Cyanide | Forage | 1-2 kg | Rush the samples to the l lab, frozen in airtight containers |
| | | Whole blood | 10 ml | |
| | | Liver | 100 g | |
| 6 | Fluoride | Bone | 20 g | Ideal sample will show l esion in organal bone |
| | | | | |
| | | Water | 100 ml | |
| | | Forage | 100 g | |
| | | Urine | 50 ml | |

| | | | | |
|---|---|---|---|---|
| 7 | Herbicides | Treated weeds | 1-2 kg | |
| | | Urine | 50 ml | |
| | | Ingesta | 500 g | |
| | | Liver & kidney | 100 g | |
| 8 | Lead, Mercury | Kidney | 100 g | |
| | | Whole blood | 10 ml | |
| | | Liver | 100 g | |
| | | Urine | 15 ml | |
| 9 | Mycotoxins | Brain, forage, liver, kidney | 100 g | Airtight container, plastic bag. For dry feeds, use cloth bags |
| 10 | Nitrate | Forage, stomach & gut contents | 1-2 kg | |
| | | Water | 100 ml | |
| | | Body fluids | 10-20 ml | |
| 11 | Organo-phosphates Organo-carbonates | Feed | 100 g | |
| | | Ingesta | 100 g | |
| | | Liver | 100 g | |
| | | Urine | 50 ml | |
| 12 | Oxalates | Fresh forage | 100 g | |
| | | Kidney | 100 g | Fixed in Formalin |
| 13 | Sodium (NaCl) | Brain | 100 g | |
| | | Serum | 5 ml | |
| | | CSF | 1 ml | |
| | | Feed | 1-2 kg | |
| 14 | Zinc phosphide | Liver, kidney, gastric contents | 100 g | |

# APPENDIX II

## ISOLATION OF TOXIC MATERIALS

Isolation and purification of the toxic material is a necessary pre-requisite for ensuring accuracy of the chemical analysis and identification of the active principles. The presence of interfering substances can either give false results or mask the expected colour of the end product, thus causing confusion. Important tests used for the isolation are:

1. Ashing
2. Direct solvent extraction (organic poisons and minerals)
3. Steam distillation (volatile poisons)
4. Stas-Otto extraction (non-volatile organic poisons).

### 1. Ashing

The representative sample of the suspected materials is mixed with concentrated $HNO_3$ and incinerated in a crucible. The ash is cooled and extracted with distilled water. For rumen contents, boiling with charcoal could cause decolourisation of the extract. Tests for the cat ions Cu, Zn, Fe, Pb, Hg, Sb, As, Al, and Ba could be conducted as explained later. The suspected material is mixed with NaOH and incinerated and the ash is collected for testing fluorine.

### 2. Direct solvent extraction

**Aqueous extract**

The representative sample of the suspected material is extracted with distilled water. Water-soluble compounds will dissolve in the aqueous portion. Urea, phosphates, nitrates and chloral may be extracted in this manner. This extract could be used for identification.

**Acid ether extract**

The representative sample of the suspected material is mixed with a little concentrated HCl and extracted with solvent ether and kept overnight. Ether portion is filtered through anhydrous sodium sulphate and the solvent removed by keeping the extract in a water bath. The residue is used for detecting organophophorus insecticides, salicylates, barbiturates, phenols, sulpha drugs and glycosides. Chromatographic analysis such as TLC may be required in case the quantity of the residue is less. Identification of the procedure is described later.

**Benzene extract**

The representative sample of the suspected material is soaked in benzene or hexane and kept overnight if possible. Benzene/hexane portion is filtered through anhydrous sodium sulphate and evaporated on a water bath. The residue is used for the identification of OC insecticides.

**Ethyl methyl ketone (EMK) extract**

The representative sample of the suspected material is soaked in EMK and kept overnight. EMK portion is filtered through anhydrous sodium sulphate and evaporated to dryness on a water bath. The residue is used for identification of carbamate insecticides.

**Alkaline chloroform extract**

The representative sample of the suspected material is made alkaline with a few ml of ammonium hydroxide and soaked in chloroform. It is mixed carefully to control emulsification. The extract is kept overnight and then the chloroform portion is decanted carefully. A separating funnel may be required for effective separation. The extract is evaporated on a water-bath and residue is used to test for the presence of alkaloids and basic drugs such as antihistaminics, phenothiazine and tranquilisers.

**Ethyl acetate extract**

A representative sample of the suspected material is acidified with dilute $H_2SO_4$ and soaked in 1:1 mixture of ethyl acetate and methyl alcohol kept overnight. The non-aqueous portion is separated and dried by filtering through the anhydrous sodium sulphate. The extract is evaporated to dryness and tested for the presence of steroids.

## 3. Steam Distillation

**Basic volatile materials**

The representative sample of the suspected material is made alkaline with NaOH and steam distilled. If the suspected material is contaminated with methyl alcohol or ethyl alcohol, it will appear in the first 10-15 ml distillate portion and can be identified chemically. If the toxic material suspected is nicotine, the distillate is collected in N/10 HCl and then chemically tested.

**Acid distillate of volatile materials**

Acid distillation is done in the presence of tartaric acid for distilling out HCN. The distillate can be tested for identifying HCN.

## 4. **Stass-Otto Extraction**

When the toxic principle is expected only in very small quantities in the suspected material, Stass-Otto extraction procedure is adopted. About 50 g of the representative sample of the suspected material is digested in a china dish over a water bath using acetic acid until the residue becomes fat free. The residue is digested with absolute alcohol and filtered. The alcohol is evaporated on a water bath and the residue is extracted with distilled water. The filtered extract is divided into two portions. A portion is acidified, extracted with ether and ether evaporated off.

# APPENDIX III

## ISOLATION TECHNIQUES & TOXIC PRINCIPLES

| | **Techniques** | **Toxic Principles Isolated** | |
|---|---|---|---|
| | | Type | Name |
| 1 | Ashing | a) Heavy Metals | Cu, Zn, Fe, Pb, Hg |
| | | b) Metalloids | Sb, As |
| | | c) Other cations | Al, Ba |
| | | d) Anions | F (Hydrofluoric acid) |
| 2 | Direct Solvent Extraction | a) Aqueous | Urea, water soluble minerals |
| | | b) Ether (Acidic) | Organophosphorus insecticides, acidic drugs such as salicylate barbiturates, phenol, sulpha drugs, glycosides |
| | | c) Benzene | Organochlorine |
| | | d) Ethyl methyl ketone | Carbamate insecticides |
| | | e) Chloroform (alkaline) | Alkaloids and basic drugs such as antihistaminics, phenothiazine tranquilizers |
| | | f) Ethyl acetate | Steroids |
| 3 | Steam Distillation | a) Alkaline media | Basic volatile materials Methyl, alcohol, ethyl alcohol |
| | | b) Acid media | Acid volatile materials HCN, formic acid, chloral hydrate, OP & OC insecticides, herbicides |
| 4 | Stass-Otto Extraction | a) Acid ether extract | Acidic & neutral drugs such as salicylates, barbiturates, carbonates, glycosides, sulpha drugs |
| | | b) Alkaline chloroform | Alkaloids, antihistaminics, phenothiazine tranquilizers and anti-depressants |
| | | c) Neutral ethyl acetate | Steroids |

# APPENDIX IV

## QUALITATIVE TESTS FOR IDENTIFICATION OF TOXIC MATERIALS

| | Suspected Material | Procedure | Observation | Reaction |
|---|---|---|---|---|
| **I** | **Heavy Metals** | ***Take a portion of the extract (ashing) & conduct the following tests*** | | |
| 1 | **Copper** | a) Add a little $NH_4OH$, observe & then add excess of the reagent | Blue ppt & with excess $NH_4OH$, intense blue colouration | Blue ppt due to copper hydroxide & blue colour due to copper ammonium complex |
| | | b) Potassium ferro cyanide is added to neutral or fairly acidic solution | A reddish brown ppt | Copper ferro-cyanide is formed |
| 2 | **Zinc** | a) Add a few drops of NaOH. Observe & then add excess NaOH | White ppt which dissolves in excess NaOH | White ppt is zinc hydroxide. It dissolves in excess NaOH forming sodium zincate |
| | | b) Add a few drops of potassium ferro-cyanide solution | White precipitate | Zinc ferro-cyanide |
| 3 | **Mercury (Mercuric)** | a) Add a few drops of KOH solution | Yellow orange ppt | Mercuric oxide |
| | | b) Add stannous chloride solution drop by drop | White ppt changing to grey and finally black | Mercurous chloride changing to mercury |
| | | c) Add a little potassium iodide solution | Red ppt | Mercuric iodide is formed |
| | **Mercury (Mercurous)** | a) Add a few drops of KOH or $NH_4OH$ solution and warm | Black ppt | Mercury |

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| | | b) Add stannous chloride solution drop by drop | White ppt. Changing to grey and finally black by excess addition of stannous chloride | Mercury |
| | | c) Add a little potassium iodide solution | Yellowish green precipitate becomes grey or greyish black | Mercurous iodide turns to mercury |
| 4 | **Iron** | a) Add a little ammonium/ potassium thiocyanate solution | Blood red colour | Ferric thiocyanate |
| | | b) Add a little potassium ferrocyanide solution | Blue colour | Ferric ferrocyanide (Prussian blue) |
| 5 | **Lead** | a) Add a little dilute HCl | White ppt | Lead chloride |
| | | b) Add a little potassium chromate solution | Yellow ppt | Lead chromate |
| **II** | **Metalloids** | | | |
| 1 | **Antimony** | a) Reinsch's Test: Drop in one or two pieces of bright copper strip after acidifying the test solution with dilute HCl. Boil for 5-10 minutes | Bluish black deposit of antimony oxide on copper | Antimony present |
| | | b) Take the test solution on a spot plate, add one drop of conc. HCl, followed by a crystal of sodium nitrate. Stir well. Add 2 drops of Rhodamine-B reagent | Violet colour changing to blue | Rhodamine antimony complex |

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| 2 | **Arsenic** | a) Reinsch's Test: Drop one or two pieces of bright copper strip after acidifying the test solution with little dilute HCl, boil for 5-10 minutes | Steel grey or black deposition of arsenic oxide on copper strip | Arsenic present |
| | | b) Gutzeit Test: 1 ml of the suspected solution is taken in a test tube. Add a little Zn powder followed by dilute $H_2SO_4$. Boil. A paper moistened with $AgNO_3$ is kept at the mouth of the test tube | $AgNO_3$ paper turns yellow and finally black | Silver arsenide, silver nitrate complex decomposes to silver |
| **III** | **Other Cations** | | | |
| 1 | **Aluminum** | a) Add $NH_4Cl$ and $NH_4OH$ in excess<br>b) Add KOH, white ppt dissolves in excess KOH. The ppt reappears on adding $NH_4Cl$ | White gelatinous ppt<br>Aluminum hydroxide forms potassium aluminate | Aluminum hydroxide<br>Potassium aluminate decomposes to form aluminum hydroxide in the presence of $NH_4Cl$ |
| 2 | **Barium** | a) Add dilute $H_2SO_4$ | White ppt insoluble in conc HCl | Barium sulphate |
| | | b) Add neutral potassium chromate solution | Yellow ppt | Barium chromate is insoluble in acetic acid. |
| **IV** | **Anions** | | | |
| 1 | **Fluorine** | a) Ash is taken in a test tube & warmed in conc $H_2SO_4$ | HF vapours having the property of etching (greasy surface). A wet | Fluorine present |

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| | | | glass rod introduced into the vapours get white ppt of silicon tetrafluoride | |
| | | b) Add a few drops of calcium chloride solution | white precipitate of calcium fluoride | Fluorine present |
| 2 | **Phosphide** | Treat the sample with conc. HCl and immediately introduce cadmium mercuric iodide paper wet in acetic anhydride | Orange color of cadmium phosphide (phosgene reacts with cadmium) | Cadmium phosphide |
| 3 | **Nitrite** | Take fresh aqueous extract of the materials suspected to be contaminated with nitrite and acidify with dil $H_2SO_4$ and add starch | Blue colour | Nitrite oxidizes KI to $I_2$ which turns starch blue |
| **V** | **Water soluble organic poisons** | | | |
| 1 | **Urea** | a) Aqueous extract is boiled with NaOH. A glass rod dipped in conc HCl is introduced into the test tube | A smell of ammonia and intense white fumes of ammonium chloride | Urea present |
| 2 | **Chloral hydrate** | a) A little of the aqueous extract or steam distillate is mixed with resorcinol. Make the mixture alkaline with 2N-NaOH. Boil for a while gently | Pink colour gradually turns red in cold due to quinone formation. After one hr it exhibits a green fluorescence in UV light | Chloral hydrate present |

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| | | b) Nessler's reagent is added to little of the distillate | Yellow ppt turns reddish brown & finally black. Sodium tetra-iodo mercurate changes to mercury | Chloral hydrate present |
| 3 | **Hydrocyanic acid** | a) Prussian blue test: The distillate is made alkaline with NaOH and concentrated to about 1 ml. Add a freshly prepared 5% solution of ferrous sulphate, 1-2 drops of a 3% solution of ferric chloride. The mixture is shaken well and allowed to stand for 2 min. Warmed gently & acidified with dilute HCl | Development of Prussian blue ppt of ferric ferrocyanide | HCN present |
| | | b) Thiocyanate test: The distillate is made alkaline with KOH and concentrated to 1 ml. Add a little of yellow ammonium sulphide & evaporated to dryness on a water bath. Extract the dry residue with a few drops of 20% HCl & add a few drops of neutral $FeCl_3$ | Blood red colour which disappears on the addition of $HgCl_2$. Ferric thiocyanate which reduces to colourless ferrous thiocyanate | HCN present |

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| 4 | **Formic Acid** | a) Neutralise the sample with calcium hydroxide and evaporate to dryness. Extract the residue with water. Add mercuric chloride drop by drop to a portion of the extract | Formation of white ppt of mercurous chloride | |
| | | b) Another portion of the aqueous extract is mixed with $AgNO_3$ | White ppt, which turns black on boiling. Silver mirror formation | Deposit of silver |
| 5 | **Methyl alcohol** | a) 2-4 ml of the sample is taken in a test tube. Add drop wise 3% $KMnO_4$ acidified with phosphoric acid until the solution remains pink. The excess permanganate is decolo-urised by adding 10% oxalic acid in drops. Then add a pinch of chromot-ropic acid. Add 1.5 ml of conc. $H_2SO_4$ carefully down the sides of the test tube so that it will underlay the distillate | A purple ring at the interface | Methanol oxidized to formaldehyde |
| | | b) Heat 3 ml sample with 5 ml potassium dichro-mate solution and 1 ml conc $H_2SO_4$ | Green colour | Methanol oxidized to formaldehyde |
| 6 | **Ethyl alcohol** | a) Add a few drops of 10% NaOH followed by 2 ml of Iodine in KI, gently heat on a waterbath. | Yellow ppt | Iodoform is formed |

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| | | b) Take 2-3 ml of the distillate in a test tube. Add 0.5 g anhydrous sodium acetate followed by 1 ml conc $H_2SO_4$. Boil & add dilute sodium carbonate | Fruity odour of ethyl acetate | Ethyl acetate |
| **VI** | **Acidic & neutral drugs** | | | |
| 1 | **Salicylic acid** | a) Dissolve the residue containing salicylic acid in a little cold water. Add a drop of neutral $FeCl_3$ | Violet color that persists in acetic acid. | Characteristic test of the phenolic group |
| | | b) To a little of the salicylic acid add three drops of con. $H_2SO_4$ and three drops of methyl alcohol. Heat on a waterbath. Pour into dil. $Na_2CO_3$ in a beaker. | Smell of oil of winter green | Methyl salicylate is formed |
| | | c) Dissolve the residue containing salicylic acid in a little water. Boil & add a few drops of potassium nitrite, 2 drops of 10 % copper sulphate & boil for 2 mins. | Red colour | Characteristic test of sodium salicylate |
| 2 | **Acetyl salicylic acid (Aspirin)** | a) Dissolve the residues in cold water & add a few drops of neutral ferric chloride | No violet colour | Absence of free phenolic group |

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| | | b) Dissolve the residue in cold water & add 5 drops of 10 % $KNO_3$, 2 drops of glacial acetic acid & 1 drop of 10% $CuSO_4$. Boil for 2 mins | Red colour | Presence of acetyl salicylic acid |
| 3 | **Barbiturates** | a) Millon's test: Take aqueous extract & add a few drops of Millon's reagent | White gelatinous ppt | Diethyl barbituric acid is formed |
| | | b) Take ethanol extract of the residue. Add 2 ml of 1% solution of cobalt nitrate in ethanol. Put 2 pellets of NaOH. | Blue colour | Organometallic complex with cobalt |
| | | c) If there is excess barbiturates, approx. 10 mg, dissolve the residue in 10 % formaldehyde. Add 4 ml of conc $H_2SO_4$ & allow to stand for 2 mins | i) weak yellow<br>ii) greenish yellow<br>iii) wine red<br>iv) reddish brown<br>v) bright yellow<br>vi) colourless | i) Barbital, pentobarbital<br>ii) Aminobarbital<br>iii) Pheno barbital<br>iv) Cyclo barbital<br>v) Diethyl barbituric acid<br>vi) Apobarbital |
| **VII** | **Organophos-phorous insecticides** | | | |
| 1 | **Parathions** | a) The test for OP compounds having P-nitro phenol ring in it - the ether extract is used. A portion of the residue is extracted in ethyl alcohol & kept in a | Intense yellow colour | Formation of P-nitrophenol |

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| | | china dish. Add a piece of KOH. Warm the contents. This is known as PNP (paranitrophenol) test. | | |
| | | b) The residue is hydrolysed using alcoholic potash & reduces to P-aminophenol using Zn/HCl mixture by boiling. Cool & add $NaNO_3$ in excess to diazotise the amino group. Add beta napthol in NaOH. | Orange colour | Diazotisation & coupling produce phenol azo beta napthol |
| 2 | **All OP compounds (TLC Test)** | a) Sample dissolved in hexane is developed on a TLC plate using hexane/ acetone 90/10. Spray palladium chloride (0.5 g/100 ml water containing a drop of conc. HCl) | Brown to yellow colour | Applicable to all OP compounds containing sulphur |
| 3 | **Detection of elements for OP, OC & carbamate compounds (to be done after preliminary tests)** | The residue is fused with metallic sodium & extracted with water. Boil and filter using the sodium fusion extract. The following tests are then conducted | | |

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| | | a) Add freshly prepared ferrous ammonium sulphate followed by drops of HCl to dissolve the ppt. Add drops of neutral ferric chloride | Green blue colour | Prussian blue. Presence of nitrogen |
| | | b) Little of the extract is boiled with conc. $HNO_3$. Add freshly prepared ammonium molybdate, boil & cool | Yellow colour | Presence of phophorus |
| | | c) To a little of the extract, dilute solution of sodium nitroprusside is added | Violet pink colour | Presence of sulphur |
| | | d) Boil with conc. $HNO_3$. Add dilute $AgNO_3$ | White ppt followed by yellow ppt | White ppt indicates chlorine & yellow – bromine |
| **VIII** | **Organochlorine Insecticides** | | | |
| 1 | **Test for DDT/ BHC** | a) The residue is dissolved in n-hexane & ethanol. Several NaOH pellets are added. Evaporate in a water bath. Cool. Add 4 drops of $CCl_4$. Shake. Add a mixture of $H_2SO_4/HNO_3$. Shake | Green colour | DDT/BHC present |
| | | b) Heat a small quantity of the extract with 0.5% solution of hydroquinone in sulphuric acid | Wine red colour | Specific for DDT |

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| 2 | **Test for Dieldrin, Endrin & Aldrin** | To a little of the extract prepared in xylene add 2 ml of conc. $H_2SO_4$ | Intense red purple colour | Purple (endrin) Red (dieldrin) Slow generation of colour (aldrin) |
| 3 | **TLC test for OC compounds** | a) (For very small quantities) Sample dissolved in hexane is developed on TLC using hexane or benzene 90/10. Spray diphenylamine reagent (0.5 g in 100 ml acetone or ethyl alcohol). Keep in sunlight | Gray spot | OC compound present |
| **IX** | **Carbamate insecticides** | a) The residue is boiled with Tollen's reagent (0.1 N $AgNO_3$ & 5 N $NH_4OH$ in 1:5 proportion). | Mirror formation | Reduced to Ag |
| | | b) Ethyl methyl ketone extract developed on TLC & sprayed with Tollen's reagent. Heated in oven between 80-100°C | Black spots | For all carbamates $AgNO_3$ is reduced to Ag |
| **X** | **Alkaloids** | | | |
| 1 | **Strychnine** | The residue is mixed with conc. $H_2SO_4$ & a pinch of $MnO_2$ is added carefully | Pink colour is developed at the boundary of the liquid | Strychnine present |
| 2 | **General test for alkaloids** | a) To a little alcoholic solution, add Dragendorff reagent | Orange yellow ppt | Violet – atropineOrange – strychnineRed violet – nicotine |

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| | | b) Develop the extract on a TLC plate in methyl alcohol, ammonium hydroxide & spray Dragendroff reagent | Orange yellow spots | |
| **XI** | **Glycosides** | | | |
| 1 | **Cerebrin (Odollam)** | Mix residues with conc. $H_2SO_4$, keep for 2 hrs | Violet colour | |
| 2 | **Yellow oleander (nerin)** | Treat with boiling conc. HCl | Blue-bluish green colour | |
| 3 | **Thevetin** | Treated with conc. $H_2SO_4$ | Crimson colour | |
| 4 | **Amygdalin (Tapioca, rubber)** | Treated with conc. $H_2SO_4$, Boil over water bath | Yellowish brown colour slowly changes to bright pink | |
| 5 | **General test for all glycosides** | The residue is dissolved in 1 ml of acetic acid containing 5% ferric sulphate. This solution is allowed to float on the surface of a mixture of conc. $H_2SO_4$ (100 part) & 5% ferric sulphate (1 part) | Immediate blue colour (green) in acetic acid layer & slow crimson colour in $H_2SO_4$ layer | Thevatin & cerebrin form slow green in acetic acid, & immediate crimson in $H_2SO_4$ is neriin. |
| **XII** | **Tannins** | a) Treat with methanolic 5% phosphomolybdic acid & boil | Blue colour | |
| | | b) Treat with Folin-ciocalteau reagent | Blue colour | |

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| **XIII** | **Resins** | | | |
| 1 | ***Calotropis gigantica*** | a) Treat with conc. HCl. Keep for a while | Immediate blue colour. Colour disappears on heating or keeping | |
| | | b) Treat with conc. $H_2SO_4$ | Green colour changing to brown or violet | |
| **XIV** | **Toxalbumin (Sui poisoning)** | | | |
| 1 | **Abrin** | Two drops of the extracted saline solution are added to 2 ml of defibrillated blood (undiluted) in a small test tube | The red cells agglutinate into a mass like that of sealing wax | Agglutination reaction |

# APPENDIX V

## LIST OF SPECIAL REAGENTS

1. **Beam's Reagent:** Dissolve 5 g of KOH in 20 ml of water. The solution is made up to 100 ml by adding water.
2. **Beta napthol-alkaline:** Dissolve 4 g beta napthol in 100 ml of 2-N sodium hydroxide.
3. **Borate buffer solution** (pH 9.5): To 4 g of boric acid, add 70 ml of water and 27 ml of NaOH solution. The solution is made up to 100 ml with water.
4. **Bromine solution:** A saturated solution of bromine in water is known as bromine solution or bromine water.
5. **Bromocresol Green:** Dissolve 0.5 g of bromocresol green in 20 ml of ethanol and dilute the solution to 100 ml with ethanol.
6. **Chromotropic acid:** 50 mg of chromotropic acid is mixed with 0.5 ml of 75% concentrated sulphuric acid. The paste is now dissolved in 10 ml of concentrated sulphuric acid and the same acid is used to adjust the final volume.
7. **Congo Red:** 0.4 g of Congo red is dissolved in 20 ml of 50% ethanol and the final volume of the solution is made up to 100 ml with 50% ethanol.
8. **p- Dimethyl amino benzaldehyde (p-DMAB):** 1 g of p-DMAB is dissolved in 100 ml of ethanol. The solution is acidified with 10 ml of dilute HCl.
9. **Draggendorff reagent:**

   a. 2 g of Bismuth subnitrate is dissolved in 25 ml of glacial acetic acid and to it 100 ml of water is added

   b. 40 g of potassium iodide is dissolved in 100 ml water.

   c. 10 ml of (a) is mixed with 10 ml of (b) and 25 ml of glacial acetic acid is added. The solution is diluted with 100 ml of water.
10. **Flurescein spray reagent:** 10 ml of the saturated solution of Flurescein in acetic acid is diluted with 15 ml of acetic acid and to it 25 ml of strong hydrogen peroxide is added
11. **F.P.N. reagent:** To 5 ml of 5% W/V solution of ferric chloride in water, add 45 ml of 20% W/W perchloric acid and 50 ml of 50% W/W nitric acid.
12. **Iodopalatinate spray reagent:** 0.25 g of palatinic chloride is dissolved in 100 ml of 5% solution of potassium iodide in water.
13. **Liebermann's reagent:** 10 g of potassium nitrate is dissolved in 20 ml of concentrated sulphuric acid and the contents are diluted to 100 ml by the same acid.

14. **Marquis reagent:**

    a. For chromatography: to 1 ml of formaldehyde solution (i.e. Formalin) add 10 ml of concentrated sulphuric acid. The reagent should not be sprayed but poured on paper or TLC plate.

    b. For colour test: 2 drops of formalin are mixed with 1 ml of concentrated sulphuric acid

15. **Mayer's Reagent:**

    a. 1.355 g of mercuric chloride is dissolved in 50 ml of water by warming.

    b. 5 g of potassium iodide is dissolved in 20 ml of water.

    Solution (a) and (b) are mixed together and made up to 100 ml with water.

16. **Millon's Reagent:** 23.5 g of mercury is dissolved in 34 ml of cold fuming nitric acid. The solution is diluted with cold fuming nitric acid and then diluted with water up to 100 ml.

17. **N-I-Napthyl ethylene-diamine-dihydrochloride solution:** 1% in ethanol.

18. **Nessler's reagent:** 3.5 g of potassium iodide and 1.25 g of mercuric chloride are dissolved in 80 ml of water. A cold saturated solution of mercuric chloride in water is added to the above solution with constant stirring till red precipitate is obtained. 12 g of sodium hydroxide is now added to it. A little more saturated solution of mercuric chloride is mixed and the contents are made up to 100 ml with water. The solution is allowed to stand and is then filtered.

19. **Palladium chloride solution:** 0.5 g of palladium chloride is dissolved in 100 ml of water and the solution is acidified to pH 3 with 2-3 drops of concentrated hydrochloric acid.

20. **Phosphate buffer:** (pH 4.5) 1.361 g of palladium dihydrogen phosphate is dissolved in 75 ml of water and the pH is adjusted to 4.5 by adding 0.1 N potassium hydroxide drop wise and the contents are diluted to 100 ml with water.

21. **Picric acid solution:** Saturated solution of trinitrophenol (picric acid) in water.

22. **Fehling solution:**

    a. 34.64 g of copper sulphate is mixed with 0.75 ml of sulphuric acid and the contents are diluted to 500 ml with water.

    b. 176 g of sodium potassium tartarate and 77g of sodium hydroxide are mixed and dissolved in small quatity of water.

    c. Equal volumes of (a) and (b) are mixed immediately before use.

23. **Schiff's reagent:** 0.2 g of basic fuchsin is dissolved in 120 ml of hot water and then cooled. 20 ml of 10% W/V solution of sodium hydrogen sulphite and 2 ml of hydrochloric acid is added to the above solution. The final volume of the solution is adjusted to 200 ml with water. The solution should be stored in cold and protected from light.

24. **Silver ammonium nitrate solution:** 2.5 g of silver nitrate is dissolved in 80 ml water. Dilute ammonia solution is added drop wise. A precipitate is formed. Excess of dilute ammonia is added to the above solution till the precipitate dissolves. The final volume of the solution is adjusted to 100 ml with water.
25. **Silver nitrate spray:** 1.0 g of silver nitrate is dissolved in 90 ml of redistilled ethanol and 5 ml concentrated ammonium solution (sp.gr. 0.88) is added to it. The volume is made up to 100 ml with ethanol.
26. **Sodium nitroprusside solution:** 1 g of sodium nitroprusside is dissolved in 100 ml of water.
27. **Starch iodide paper:** Few drops of 0.5% starch solution are poured on a piece of Whatman No. 1 filter paper. The paper is then dipped in 0.4% solution of potassium iodide in water.
28. **Vanillin-sulphuric acid reagent:** 1.0 g vanillin is dissolved in 100 ml of concentrated sulphuric acid.
29. **Zwikker's reagent:** 40 ml of 10% copper sulphate solution in water is mixed with 10 ml of pyridine and the contents are diluted to 100 ml with water.
30. **Diphenylamine solution:** 1 g of diphenylamine is dissolved in 100 ml of redistilled ethanol.
31. **Frohde's reagent:** 0.5 g of ammonium molybdate is dissolved in 50 ml water and the solution further diluted to 100 ml with water.
32. **Iodine solution:** Iodine is dissolved in water in the presence of potassium iodide so as to produce 0.1 N solution of iodine.
33. **Mercurous sulphate reagent:** 5 g of mercurous oxide is dissolved cautiously in 20 ml of concentrated sulphuric acid. The solution is then made up to 250 ml with slow addition of water.
34. **Diphenyl carbazone solution:** 1.0 g of diphenyl carbazone is dissolved in 100ml of redistilled chloroform
35. **Rhodamine B reagent:** 0.05 g of Rhodamine B in 100 ml of 15% KCl prepared in 2N HCl

# APPENDIX VI

## TOXICITY ASSESSMENT

### Acute toxicity testing / $LD_{50}$

The simplest and most common test is a response study, which maintains death as the criterion for toxicity. It is designed to determine the $LD_{50}$ or the lethal dose for 50% of the animals. Great variations are recorded when $LD_{50}$ studies are performed with the same chemical being exposed to different species of animals Rat is considered as the standard species of animal for toxicity. These values cannot be extrapolated to provide an estimate of the amount of the material that would kill 50% of the animals or humans exposed to it.

### $ED_{50}$

This is defined as the dose required to produce some therapeutic response in 50% of the test animals. It is also known by other terms such as $LD_{50}$ - Lethal Dose 50%, MLD - Medium Lethal Dose, $ED_{50}$ - Effective Dose 50%, and MED - Medium Effective Dose.

# APPENDIX VII

## POISONOUS PLANTS LIST

### Group 1: Plants containing alkaloid poisons

| Sl. No | Name of the Plant | Toxic principle | Toxic action |
|---|---|---|---|
| 1. | *Strychnos nux vomica* | Strychnine and brucine | Direct spinal cord stimulant |
| 2. | *Nicotiana tabacum* | Nicotine | Ganglionic blocking and action on nicotine receptors |
| 3. | *Atropa belladonna* (Deadly night shade) | Atropine, Hyoscine, Hyoscyamine | Parasympatholytic action, acetylcholine receptor blocking and nervous signs |
| 4. | *Datura stramonium* (Thorn apple) | Atropine, Hyoscyamine, Hyoscine | Parasympatholytic action, acetylcholine receptor blocking and nervous signs |
| 5. | *Hyoscyamus niger* (Henbane) | Hyoscyamine and hyoscine | Parasympatholytic action. Acetylcholine receptor blocking and nervous signs. |
| 6. | *Rauvolfia serpentina* | Reserpine | Tranquilizing effect, blocking nor-epinephrine release, fall in BP and body temperature |
| 7. | *Vinca rosea* (Periwinkle) | Vincrystine and vinblastin | Antineoplastic, high dose causes neurotoxicity |
| 8. | *Aristolochia spp.* | Aristolochine | Poisoning similar to aloe poisoning (alion), acute gastro enteritis, collapse and death |
| 9. | *Heliotropium europium* | Pyrrolizedine alkaloids | Acute hepatotoxicity and haemoglobinuria |
| 10. | *Ipomoea spp.* | Lysergic acid alkaloids | Hallucinogenic action. Nervous system poisoning, ataxia |
| 11. | *Papaver somniferum* (White Poppy) Opium | Morphine, Codeine and Thebaine | CNS depressant, drug addiction |
| 12. | *Coffea arabica* (Coffee plant) | Caffeine, Theophylline and Theobromine | CNS stimulant |
| 13. | *Anamirta paniculata* (Fish berry) | Picrotoxin | Powerful CNS stimulant |
| 14. | *Colchicum* spp | Colchicine | Powerful purgative, prevents mitosis by inhibiting spindle formation |

| | | | |
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| 15. | *Veratrum spp* | Veratrine | Teratogenicity (Cyclops) |
| 16. | *Aconitum spp.* | Aconite | Cardiotoxicity |
| 17. | *Solanum tuberosum* (Potato) | Glyco akaloid (Solanin) | Acute gastro enteritis and haemolysis of red cells and cardiac arrest. |
| 18. | *Cannabis indica* (Ganja, Marijuana) | Delta-9-tetrahydrocannabinol) | Psychotropic agent, CNS depressant, hallucinations |
| 19. | *Mimosa* spp | Mimosin | Severe allergic reaction - bloating, cardiac depression and death |

## Group 2: Plants containing glycoside poisons

***A: Cardiac Glycosides***

| Sl. No. | Name of the Plant | *Toxic principles* | Toxic action |
|---|---|---|---|
| 1. | *Digitalis* spp (Fox glove and *Stropanthus kombe*) | Cardiac glycoside, Digitoxin, Gitoxin Strophanthin | Cardiac action and vagal toxicity |
| 2. | *Nerium oleander* (Yellow Oleander)<br><br>*Nerium indicum* (Indian Oleander) | Cardiac Glycosides<br><br>Oleandroside & Neriocide | Severe vomiting associated with respiratory problems<br><br>Arterio-venous heart block |
| 3. | *Thevetia spp.* | Cardiac glucoside, Thevetin | Produce digitalis like action on heart |
| 4. | *Rhamnus cathartica* (Buck thorn) | Glycoside - Emodin | Acute diarrhea and colic |
| 5. | *Cerebra odollam* | Cardiac glycoside cerebrin | Cardiotoxicity |

***B: Cyanogenetic Glycosides***

| Sl. No. | *Name of the Plant* | Toxic principles | Toxic action |
|---|---|---|---|
| 1. | Prunus spp | Cyanogenetic glycosides Amygdalin, Prulaurasin | |
| 2. | Tapioca (leaf and immature tuber and tuber peel) | Amygdalin and HCN | |
| 3. | Rubber leaf | Hydrocyanic acid | Cyanide poisoning |
| 4. | *Sorghum vulgaris* (millets) | HCN | |

| | | | |
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| 5. | Sudan Grass (*Sorghum vulgare* var. *sudanense*), Arrow grass (*Triglochin* sp) | HCN | HCN poisoning |
| 6. | *Zea mays* (Maize) | HCN | |
| 7. | *Trifolium repens* (White Clover) | Lotaustralin | |
| 8. | *Linum catharticum* (Purging flax and Linseed cake)<br><br>Fruit trees like plum (*Prunus domestica*), choke, cherry (*Prunus laurocerasus*), peach (*Prunus persica*), apricot (*Prunus armeniaca*), apple (*Pyrus mallus*) and almond (*Amygdalus communis*); the kernels and leaves of these plants contain cyanogenetic glycoside. | Linnamerin- HCN released under the action of the enzyme linamerase<br>Amygdalin | Cyanide poisoning and goitrogenic in lambs<br><br>Cyanide poisoning, tissue anoxia cyanhaemoglobin formation and hindrance of $O_2$ transport by inhibiting cytochrome oxidase enzyme |
| 9. | Jowar (*Sorghum vulgare*) | HCN | Cyanide poisoning |

## Group 3: Oxalate containing toxic plants

| Sl. No. | Name of the plant | Toxic Principles | Toxic action |
|---|---|---|---|
| 1. | *Beta vulgaris, Oxalis spp Rumex spp. Trianthema spp, Sarcobatus spp,* Colocasia leaf, Spinach, Rhubarb | Oxalates and Oxalic acid | Acute crystalluria and nephrotoxicty |

## Group 4: Nitrate containing plants

| Sl. No. | Name of the plant | Toxic Principles | Toxic Action |
|---|---|---|---|
| 1. | *Amaranthus spp* (Pig Weed), *Bassia spp., Beta spp.,* Oat hay poisoning (Sugar beet sprayed with 2.4-D) | Nitrate | Gastroenteritis and methhaemoglobinaemia |

## Group 5: Phytotoxins – Toxalbumin

| Sl. No. | Name of the plant | Toxic Principles | Toxic Action |
|---|---|---|---|
| 1. | *Abrus precatorius* Coral bead vine seed | Abrin (It is a toxalbumin) | Sui (Needle like projections) poisoning in animals |
| 2. | *Ricinus communis* (Castor bean) | Ricin - Toxalbumin | Acute gastro enteritis, cardiotoxicity |

## Group 6: Fixed oils

| Sl. No | Name of the Plant | Toxic Principle | Toxic Action |
|---|---|---|---|
| 1. | *Ricinus communis* | Irritant oil | Acute purgation, abortifacient |
| 2. | *Croton tigilium* | Irritant oil | Drastic purgative |

## Group 6: Miscellaneous

| Sl. No | Name of the Plant | Toxic Principle | Toxic Action |
|---|---|---|---|
| 1. | Cotton seed cake (Gossypol) | Toxic principle in the pigment gland. Polyphenolic binaphthalene derivative-gossy purp-urin and gossy verodurin inactivated by cooking | Young ruminants and monogastric animals are more susceptible. Affects $O_2$ transport and haemolysis of RBCs |
| 2. | Yellow sweet clover, White sweet clover (*Melilotus officinalis, M. alba* used as hay on spoilage by mould) | Coumarin converted to dicoumarol | Interferes with prothrombin production, prolonged clotting time and internal hemorrhage, Vit. K synthesis is affected |

| | | | |
|---|---|---|---|
| 3. | Bracken fern poisoning | Contains enzyme – thiaminase | Thiamine deficiency hemorrhage. High blood pyruvic acid level. |
| 4. | *Mimosa pudica* and *Leucena leucocephala* | Amino acid mimosin | Hemorrhagic gastritis and depilatory agent |
| 5. | *Lathyrus sativa* (Indian pea) | Beta amino propionate | Lathyrism, degeneration of vagus and recurrent laryngeal muscles and laryngeal muscles |
| 6. | *Calotropis gigantea* | Latex resin irritant | Irritant resin with proteolytic action, digitalis like action in heart, caustic purgative and emetic |
| 7. | *Semicarpus anacardium* (Marking Nut) | Anacardic acid, sremicarpol (Phenolic components | Highly irritant, vesicant |

## Group 7: Mycotoxins

***A: Larger Fungi***

| Sl. No | Name of the Plant | Toxic Principle | Toxic Action |
|---|---|---|---|
| 1. | Poisonous mushrooms *Amanita spp.; A. phalloides* - death cap; *A. verna*- Foolish mushroom; *A. virosa* Destroying angel; *A. muscaria* | *A. Phalloides* contains phallodin, alpha and beta amantin. Muscarin alkaloid also contains hallucinogenic substance - bufotenin | Phallodin - Hepatotoxic and nephrotoxic. Amanitin cause hypoglycaemia Neurotoxicity- Severe vomiting and gastro enteritis – |

***B: Micro Fungi***

| Sl. No | Name of the Plant | Toxic Principle | Toxic Action |
|---|---|---|---|
| **1.** | **Hepatotoxic Plants** | | |
| | a) *Sporodesmin bakeri* | Sporodesmin | Hepatotoxicity. Hepatobiliary blocking causes secondary photosensitization |
| | b) *Aspergillus flavus* | Aflatoxin (B1. B2, G1 & G2) | Interaction between aflatoxin and vitamin D in poultry with respect to bone calcification. In cattle, cirrhosis, liver tumor and jaundice |

| | | | |
|---|---|---|---|
| | c) *Pencillium rubrum* | Rubra toxin - A and Rubra toxin B | Haemmorhage and necrosis in vital organs like liver |
| **2.** | **Nephrotoxic plants** | | |
| | a) *Aspergillus ochraceus* | Ochratoxin | Myotoxic |
| | b)*Pencillium virindication* | Citrinin | Nephropathy (Mould nephrosis) enlarged kidney in poultry |
| | c) *P. Palitams* | | |
| **3.** | **Neurotoxins** | | |
| | a) *Pencillium cyclopium* | Peniterm - A | Hypochromic microcytic anaemia |
| | b) *P. puberalum* | | Neurological and renal effects |
| | c) *P. patulum* | Patulin | Neurological and renal effects |
| | d) *Aspergillus fumigatus* (Mouldy silage) | Fumitremorgen | Neurological effect |
| | e) *P. citrovirude* in rice | Citrovirin | Neurological effect in man |
| **4.** | **Oestrogenic mycotoxins** | | |
| | a) *Fusarium gramanicum* | Phenolic macrolide Zearalenone (F2 toxins) | Direct effect on female reproductive system inducing estrus and atrophy of the ovary. Production of still born babies |
| **5.** | **Ergot alkaloid** | | |
| | *Claviceps purpurea* | Ergotoxine and ergotamine | Cardiovascular effect, narrowing of capillaries and necrosis of extremities, gangrene, development of thrombosis. Hallucination and ergotism in man |
| **6.** | **Photosensitizing plants** | | |
| | a) *Hypericium* spp | Photodynamic substances – Hypericin | |
| | b) *Fagopyrum* (Buck wheat) | Fagopyrin | |
| | c) *Panicum* spp | | |
| | d) *Tribulus terrestris* (Devil's thorn) | Photodynamic substance | Geldikkop in sheep, hepatic damage and enzootic icterus |
| | e) *Lantana camara* | Lantadine A, Lantadine B | |
| **7.** | **Goitrogens** | | |
| | a) Rape seed and other brassicae (Kale, cabbage, turnip) | Antithyroid substances, 1,5-vinyl-2 thiooxazolidine | Goitrogenic effect. Affects thyroid slowly |

| | | | |
|---|---|---|---|
| | b) *Sinapis nigra* | 3-buteny 7l isothiocy | Acute gastro enteritis |
| | (Black mustard seed) | anate glycoside singrin | |
| | | enzyme myrosin decompose singrin to yield alkyl isothiocyanate | |
| | | (volatile oil of mustard) and to give isopropyl isothiocyanate | |

# APPENDIX VIII

## FLUID THERAPY

The water content of the animal body is 55-60%. Of this 60%, 40% is intra cellular fluid (ICF) and 20% is extra cellular fluid (ECF). Of the 20% of extra cellular fluid, 15% is interstitial fluid and 5% is plasma. The osmotic pressure of ICF and ECF are the same, but the electrolyte concentration differs. The pH of ECF is 7.4 and ICF is 7.0.

### Normal water balance

The water balance is maintained by continuous water intake and excretion. Intake of water is through liquid diet, solid food and by oxidation of food (100 g protein = 40 g water, 100 g carbohydrate = 55 g of water and 100 g fat = 107 g water). Excretion of water occurs via the kidney, skin, lung, faeces, tears, saliva, milk and semen. (around 50-60% of water excretion is through the kidneys). The water intake and output will be the same in healthy animals. Fatty animals require less water compared to thin and muscular ones. Abnormal gain of water is through the parentral administration of fluids. Abnormal water loss occurs in diarrhea, vomition, fistula, ascites, peritonitis and bleeding.

***Electrolyte composition***

| | ***ECF (mEq/Lit)*** | ***ICF (mEq/Lit)*** |
|---|---|---|
| Cations | | |
| Na | 143 | 10 |
| K | 5 | 150 |
| Ca | 5 | - |
| Mg | 2 | 40 |
| **TOTAL** | **155** | **200** |
| | | |
| **Anions** | | |
| Cl | 106 | - |
| $HCO_3$ | 20 | 10 |
| $HPO_4$ | 2 | 140 |
| $SO_4$ | 2 | 10 |
| Organic acids | 10 | - |
| Protein | 15 | 40 |
| **TOTAL** | **155** | **200** |

## Dehydration

The skin is a vast reservoir of water (skin comprises 16 % of the body weight and holds 70% of the water). We can assess the degree of dehydration by observing the loss of turgor (tone), elasticity and dryness of the skin.

The calculation of the water requirement depends on the turnover of water and abnormal loss. Turnover in mature animals is 65 ml/day and in immature and lactating animals is 130 ml/day (a 20 kg dog require 20 x 65 = 1300 ml/day, 600 kg horse require 600 x 65 = 39000 ml/day). Since the turnover is more in young individuals the dehydration will be more serious in them e.g., calf diarrhea, puppy diarrhea.

## Points to be kept in mind

1. Dehydration affects young animals faster than adults.
2. Very active animals require more water.
3. Animals under anesthesia will require more water.
4. Drugs like corticosteroids and diuretics increase water requirements.
5. Old animals with chronic diseases (impaired renal function) will require more water than normal adults.
6. Individuals will require more water in hot, humid weather.

### The following are clinical signs indicating varying degrees of dehydration:

1. **4% dehydration:** No evidence of clinical dehydration, will have a history of water loss, slightly leathery skin
2. **6 % dehydration:** Leathery skin, when skin is lifted up, it will peak but will return to normal very slowly, dry, mucus membranes
3. **8 % dehydration:** Lack of skin pliability and elasticity, skin will peak and stay, mucus membranes and tongue will be dry, eyeball will be soft and shrunken
4. **12% dehydration:** Circulatory insufficiency and there will be all signs of circulatory collapse
5. **15 % dehydration:** Acute shock

Calculate the requirement and give at a rate of 15-30 ml/kg/hr in large animals. The animal can tolerate up to 90 ml/kg/hr initially but then decrease the rate. Try to establish the renal function. If no urine is passed within 2-4 hours of injection, the rate must be reduced to 20 ml/kg/hr.

The easiest and least dangerous route is oral. No strict tonicity, no strict volume and asepsis. Per-rectal route can be tried in young ones. K, Na, Cl solutions will be best absorbed. Most commonly used routes of administration are I/V, S/C and I/P.

## Treatment for dehydration

Acute imbalances will respond to acute therapy. Chronic imbalances will respond only with treatment and diet. First, it must be determined whether the abnormality is due to volume, osmology, or acid-base imbalance. Volume losses need electrolytes and water. Osmological imbalances require electrolytes and acid base imbalances do not respond immediately.

**Lactated potassium saline solution (Darrow's solution)**

| | |
|---|---|
| KCl | - 240 to 280 mg |
| NaCl | - 381-420 mg |
| Na lactate | - 590 mg |
| Dist. water | - 100 ml |

**Ringer solution**

| | |
|---|---|
| NaCl | - 6.0 g |
| KCl | - 0.3 g |
| CaCl | - 0.22g |
| Dist. water | - one liter |

**Acidifying solution**

| | |
|---|---|
| NaCl | - 8.9 g |
| $NH_4Cl$ | - 19 g |
| Dist. water | - one litre |

**Alkalinizing solutions**

| | |
|---|---|
| NaCl | - 6 g |
| KCl | - 0.3 g |
| CaCl | - 0.22 g |

Na lactate 60% solution 5 ml
Dist. water to one litre.

**Sodium bicarbonate**

13 g sodium bicarbonate per litre.

**High Sodium solution**

| | |
|---|---|
| Lactate ringer | - 1 lit |
| Sod. bicarb | - 5 g |

**High pot sol.**

| | |
|---|---|
| Normal saline | - 1 Litre |
| KCI | - 2.5 g |

Potassium supplementation is administered in malnutrition, loss of G.I secretion, G.I. intubation and fistula. A high potassium solution may cause cardiac arrest. For this reason, the injection is given very slowly. The maximum concentration is 80 mEq/liter. In potassium toxicity give calcium gluconate I/V. Contraindications are glucose, insulin, cation exchange resins, renal dysfunction, Cushing's syndrome, haemolytic reaction.

Calcium - Calcium gluconate is preferred over calcium chloride since it is less of an irritant. It is used to treat hypocalcemia, calcium tetany, milk fever and alkalosis, and to stimulate the heart when adrenaline is failing. Warm the solution to body temperature and administer 2 ml/min I/V. Contraindicated in ventricular fibrillation.

# APPENDIX IX

## PESTICIDES REGISTERED FOR USE IN THE COUNTRY UNDER SECTION 9(3) OF THE INSECTICIDES ACT, 1968, (as on 30/09/1998) INCLUSIVE OF PLANT GROWTH REGULATORS

In India, no pesticide can be imported, manufactured, distributed or used within the country without registration under the Insecticides Act, 1968. So far 141 pesticides (inclusive of plant growth regulators) stand registered on a regular basis for their import, manufacture and use in the country. Out of these 141, 13 pesticides are for restricted use in the country. The list of 140 pesticides registered in the country, 20 banned pesticides and 18 pesticides which have been refused registration are all given below.

NAME OF PESTICIDE

| **A** | **Registered pesticides for regular use** |
|---|---|
| 1 | Acephate |
| 2 | Alachlor |
| 3 | Aldicarb |
| 4 | Allethrin |
| 5 | Alpha-naphthylacetic acid |
| 6 | Aluminium phosphide |
| 7 | Anilofos |
| 8 | Atrazine |
| 9 | Alpha-cypermethrin |
| 10 | Aureofungin |
| 11 | *Bacillus thuringiensis* (B.t.) |
| 12 | Barium carbonate |
| 13 | Fipronil |
| 14 | Benthiocarb (thiobencarb) |
| 15 | Benomyl |
| 16 | Bitertanol |

| | |
|---|---|
| 17 | Bromadiolone |
| 18 | Butachlor |
| 19 | Captafol |
| 20 | Captan |
| 21 | Cartap hydrochloride |
| 22 | Carbaryl |
| 23 | Cabendazim |
| 24 | Carbofuran |
| 25 | Carboxin |
| 26 | Chlorobenzilate |
| 27 | Chlorfenvinphos |
| 28 | Chloromequate chloride (ccc) |
| 29 | Chlorothalonil |
| 30 | Chloropyriphos |
| 31 | Copper oxychloride |
| 32 | Copper sulphate |
| 33 | Coumachlor |
| 34 | Coumatetralyl |
| 35 | Cuprous oxide |
| 36 | Cyfluthrin |
| 37 | Cypermethrin |
| 38 | Dalapon |
| 39 | Decamethrin (Deltaamethrin) |
| 40 | Dichloro diphenyl trichloroethane (DDT) |
| 41 | Dichloropropene and dichloropropane mixture (DD mixture) |
| 42 | Dichlorvos (DDVP) |
| 43 | Dicofol |
| 44 | Doflubenzuron |

| | |
|---|---|
| 45 | Dimethoate |
| 46 | Dinocap |
| 47 | Dithianon |
| 48 | Diuron |
| 49 | Dodine |
| 50 | Diazinon |
| 51 | Dieldrin |
| 52 | d-trans allethrin |
| 53 | 2, 4-D Sodium, amine & ester (salts) |
| 54 | Ethyline dichloride & carbon tetrachloride |
| 55 | EDCT mixture (3:1 ethyline dibromide & carbon tetra chloride mixture) |
| 56 | Edifenophos |
| 57 | Ethofenprox (Etofenprox) |
| 58 | Ethephon |
| 59 | Ethylene dibromide (EDB) |
| 60 | Ethion |
| 61 | Fenitrothion |
| 62 | Fenarimol |
| 63 | Fenobucarb (BPMC) |
| 64 | Fenthion |
| 65 | Fenvalerate |
| 66 | Ferbarm |
| 67 | Fluchloralin |
| 68 | Fluvalinate |
| 69 | Formothion |
| 70 | Fosetyl-Al |
| 71 | Gibberellic acid |
| 72 | Glyphosate |

| | |
|---|---|
| 73 | Hexaconazole |
| 74 | Iprodione |
| 75 | Isoproturon |
| 76 | Kitazin (Indole-3-butyric acid) |
| 77 | Lambdacy halothrin |
| 78 | Lime sulphur |
| 79 | Lindane (Gamma BHC) |
| 80 | Malathion |
| 81 | Maleic hydrazide (MH) |
| 82 | Mancozeb |
| 83 | Metaldehyde |
| 84 | Methabenzthiazuron |
| 85 | Methoxy ethyl mercury chloride (MEMC) |
| 86 | Methyl bromide |
| 87 | Methyl chlorophenoxy acetic acid (MCPA) |
| 88 | Metalaxyl |
| 89 | Metoxuron |
| 90 | Methomyl |
| 91 | Metolachlor |
| 92 | Metribuzin |
| 93 | Monocrotophos |
| 94 | Myclobutanil |
| 95 | Methyl parathion |
| 96 | Neem products |
| 97 | Nickel chloride |
| 98 | Oxadiazon |
| 99 | Oxycarboxin |
| 100 | Oxydemeton-methyl |

| | |
|---|---|
| 101 | Oxyfluorfen |
| 102 | Para-dichlorobenzene |
| 103 | Paraquat dichloride |
| 104 | Penconazole |
| 105 | Pendimethaalin |
| 106 | Permethrin |
| 107 | Phenthoate |
| 108 | Phorate |
| 109 | Phosalone |
| 110 | Phosphamidon |
| 111 | Prallethrin |
| 112 | Pretilachlor |
| 113 | Pirimiphos-methyl |
| 114 | Profenofos |
| 115 | Propanil |
| 116 | Propetamphos |
| 117 | Propiconazole |
| 118 | Propoxur |
| 119 | Pyrethrins (Pyrethrum) |
| 120 | Quinalphos |
| 121 | Sodium cyanide |
| 122 | Streptomycin + Tetracycline |
| 123 | Sulphur |
| 124 | Temephos |
| 125 | Triadimefon |
| 126 | Triallate |
| 127 | Triazophos |
| 128 | Trichlorofon |

| | |
|---|---|
| 129 | Tricyclazole |
| 130 | Tridemorph |
| 131 | Trifluralin |
| 132 | Thiram |
| 133 | Thiometon |
| 134 | Thiophanate-methyl |
| 135 | Trichloro acetic acid (TCA) |
| 136 | Validamycin |
| 137 | Warfarin |
| 138 | Zinc phosphide |
| 139 | Zineb |
| 140 | Ziram |
| 141 | Kasugamycin |
| | |
| **B** | **Banned Pesticides** |
| 1 | Aldrin |
| 2 | Calcium cyanide |
| 3 | Chlordane |
| 4 | Copper acetoarsenite |
| 5 | Dibromochloropropane (DBCP) |
| 6 | Endrin |
| 7 | Ethyl Parathion |
| 8 | Ethyl Mercury Chloride |
| 9 | Heptachlor |
| 10 | Menazon |
| 11 | Nicotine sulphate |
| 12 | Nitrofen |
| 13 | Paraquate dimethyl sulphate |

| | |
|---|---|
| 14 | Pentachloro nitrobenzene (PCNB) |
| 15 | Pentachlorophenol (PCP) |
| 16 | Phenyl Mercury Acetate (PMA) |
| 17 | Sodium Methane Arsonate (MSMA) |
| 18 | Tetradifon |
| 19 | Toxaphene |
| 20 | BHC |
| | |
| **C** | **Pesticides Refused Registration** |
| 1 | Ammonium sulphamate |
| 2 | Azinphos ethyl |
| 3 | Azinphos methyl |
| 4 | Binapacryl |
| 5 | Calcium arsenate |
| 6 | Carbophenothion |
| 7 | Chinomethionate (Morestan) |
| 8 | Dicrotophos |
| 9 | EPN |
| 10 | Fenthion acetate |
| 11 | Fenthion hydroxide* |
| 12 | Lead arsenate |
| 13 | Leptophos (phosvel) |
| 14 | Mephosfolan |
| 15 | Mevinphos (phosdrin) |
| 16 | 2,4,5-T* |
| 17 | Thiodemeton/disulfoton |
| 18 | Vamidothion |

*These pesticides are manufactured in India for export only

## Persistence of Chlorinated Hydrocarbon Insecticides in Soil

| Insecticide | Average (lb/acre) | Time for 95% disappearance |
|---|---|---|
| Aldrin | 1-3 | 1-6 (3) |
| DDT | 1-2.5 | 4-30 (10) |
| Dieldrin | 1-3 | 5-25 (8) |
| Lindane | 1-2.5 | 3-10 (6.5) |
| Heptachlor | 1-3 | 3-5 (3.5) |
| Chlordane | | 15 yrs |
| Dicofol | | 1 yr |
| Endosulfan | | 100 days |
| Methoxy chlor | | Less persistent |

Source: Training course manual on Pesticide residue analysis

Analysis of Pesticides residue content in Fish, Fishery Products and processed water (Central Institute of Fisheries Technology).

## PESTICIDE

### a) Insecticides

| Sl No. | Generic Name | Trade Name | Uses/Indication | Dose |
|---|---|---|---|---|
| 1 | Endosulphan | Thiodan, Hildan, Starsulfan, Hexasulfan, Corosulfan, Thiokill (all 35% EC), Endocell, | Broad spectrum action safe for beneficial insects and pollinators | 0.05% spray |
| 2 | Carbaryl | Parysulfan, Hexasulphan, Thiotex (all 4% DP) Sevin (5%,10% DP and | For controlling vegetable pests. Broad spectrum action. Used | |

| | | | | |
|---|---|---|---|---|
| | | 50% WP) Hexavin (5%, 10% DP), Killex carbaryl (50% and 80% WP), Carvint (10% and 85% WP) | against a wide range of pests. Not effective against mites. | 2 kg ai/ha 2.5 kg/ha of 50% WP |
| 3 | Carbaryl+ Lindane | Sevidol 8% | Broad spectrum action used for pest control in rice | 1.5 kg/ ai/ha |
| 4 | Carbofuran | Furadan,Hexafuran (3 G) | For pest control in rice | 0.5-0. 75 kg ai/ha |
| 5 | Aldicarb | Temik (10% G) | For nematode control in rice, pepper and banana | 0.02% solution Dip 1 g. ai/ha |
| 6 | Methyl parathion | Metacid, Parataf(2% DP and 50% EC) Paramet M and Kloflox-(50% EC) Ekatox (2% DP) | Knock down action harmful to natural enemies – DP used for control of cardamom thrips | 0.05% spray |
| 7 | Fenitrothion | Sumithion (5% DP &50% EC) Foliothion,Accothion (50% EC) | Broad spectrum, action contact & stomach poision | 0.05% 1L/ ha of 50 EC |
| 8 | Mercaptothion | Malathion (25% WP & 50% EC) Malamar, Malatox,Starmal (50 EC) Cythion 5% DP & 50% EC. | Controlling vegetable storage pests- safer insecticide | .1% spray |
| 9 | DDVP | Nuvan,Divap,Marvex super, Vapona 76% E/af (100% EC) | Less residual action contact and fumigant action safer insecticide | 0.05% |
| 10 | Quinalphos | Ekalux, Kinalux, Quinal-phos, (25% EC, 5% G & 1.5% DP) | Broad spectrum used in cardamom insecticide & acaricide | 1L/ha 0.25 to 0.05% spray 1.5 kg/ha (G) 25 kg DP/ha |

| | | | | |
|---|---|---|---|---|
| 11 | Phosalone | Zolone 35% EC | Broad spectrum insecticide & acaricide | 1 L/ha, 0.07% spray |
| 12 | Fenthion | Lebaycid (50% EC) | Rice pests | 1 L/ha 0.05% spray |
| 13 | Dimethoate | Rogor, Tara 909, Coroth-oiate,Nugor, Hillthoate, Killex, Metasystox (25 & 30% EC) | Systemic insecticide & nematicide | 1 L/ha (30EC) 0.03 to 30% EC) 0.05% spray |
| 14 | Methyl dematon | Metasystox, (25% EC) | Systemic effective against sucking pests | 1 L/ha 0.05% spray |
| 15 | Formothion | Anthio (25% EC) | Systemic insecticide & acaricide | 1 L/ha 0.05% spray |
| 16 | Monocrotophos | Nuvacron, Monophos, Monocil, Corphos, J K mono, Kadette, Phoskill | Systemic insecticide-long residual action | 600 ml/ha 0.05% spray |
| 17 | Phosphamidon | Dimecron 86 EC Umecron 85 EC JK Midon 86% | Systemic insecticide & acaricide | 250 ml/ha (85 EC) 0.05% spray |
| 18 | Phorate | Thimet, Phorate, Umet, JKPhorate (10% G) | Systemic insecticide & nemacticide in rice & banana | 1.5 kg/ha |
| 19 | Trichlorfon | Dipterex (50% EC) | Effective for chewing insects, feeble contact action | 800 ml/ha 0.1% spray |
| 20 | Thiometon | Ekatin (25% EC) | Systemic insecticide | 0.1% spray |
| 21 | Chlorpyriphos | Dursban (20% EC) | Used in rice, also as root dip and termite control | 0.02% spray |

| | | | | |
|---|---|---|---|---|
| 22 | Phenthoat | Phendal, Elsan ( 50% EC, 2% DP) | Broad spectrum | 750 ml/ha of 50 EC 0.05 kg D/ha |
| 23 | Triazophos | Hostathion (40% EC) | Used in rice | 625 ml/ha |

## b) Fungicides

| | | | | |
|---|---|---|---|---|
| 1 | Copper oxychloride | Blitox 50 W, Blue copper, Esso copper Cupramer 50 W, Starcop 50 W, Fytolan 50 W Killex copper 50 W | Foliar spray and drenching | 0.3-0.4% spray |
| 2 | Sulphur | Cossan, Thiovit 80 WP, Micro sul, Sultaf 80 W, Sulfex 80 WP | Foliar spray against powdery mildew and mites | 0.1-0.5% spray |
| 3 | Ziram | Cuman, JK Ziram | Foliar spray, Residual activity, protective fungicide | 0.2-0.4% spray |
| 4 | Zineb | Indofil Z-78, Zineb 75 W, Hexathane 75 W, Sandoz Zineb | Foliar spray | 0.2%-0. 4% spray |
| 5 | Thiram | Thiride 75 WP, Hexathir 75 WP, JK Thiram 75 W | Foliar spray, soil and seed treatment | 0.2-0.3% spray |
| 6 | Mancozeb | Indofil M-45, Maneb 75 WP, Hithane M-45, Shield 75 WP, Uthane M-45, Zebthane 45 WP | Foliar spray | 0.2-0.4% spray |
| 7 | Ediphenphos | Hinosan 50 EC, H-Phos 50 EC | Foliar spray for the control of blast and sheath blight of rice | 0.1% spray of 50 EC |
| 8 | PCNB | Brassicol 75 WP | For soil application and seed treatment | 0.1% spray of 75 WP |
| 9 | Dinocap | Karathane 25 WP and 48 EC | Foliar spray for powdery mildew control of cucurbits and rose | 0.05% spray of 45 EC 300 g/ha of 25 WP |

| | | | | |
|---|---|---|---|---|
| 10 | Captan | Captan 75 WP, Hexacap 75 WP | Seed treatment | 1.5 g/kg seed |
| 11 | Captafol | Foltaf 80 WP, Difoltan 80 WP | Foliar fungicide for the control of sheath blight of rice and Sigatoka disease of banana. | 0.1-0.3% spray |
| 12 | Carbendazim | Bavistin 50 WP, B engard 50 WP, Zoom 50 WP, Sten 50 Saivistin 50 WP, JK stein 50 WP Akozim 50 W | Systemic-foliar spray for control of powdery mildew in ornamentals and sheath rot of rice | 0.1% spray 500 g/ha of 50 WP |
| 13 | Benomyl | Benlate 50 WP | Systemic-foliar spray against blast of rice | 0.1-0.2% spray |
| 14 | Carboxin | Vitavax 75 WP & 80 WP | Foliar spray for seed treatment in rice | 500 g/ha of 80 WP |
| 15 | Tridemorph | Calixin EC | For control of stem bleeding of coconut and pink disease of rubber | 0.1-0.2% spray |
| 16 | Kitazin | Kitazin 48 EC | Foliar spray against blast of rice | 0.1% spray of 45 EC |
| 17 | Pyroquilon | Fongorene 50 WP | Seed treatment | 2 g/kg seed |
| 18 | Tricyclazole | Beam 75 WP | Seed treatment | 2 g/kg seed |
| 19 | Triadimefon | Bayleton 5 WP, 25 WP & 100 EC | Systemic fungicide effective against powdery mildew and rust fungi | 0.1% spray |
| 20 | Hexaconazole (Triazoles) | Contaf 5 EC | Systemic, effective against powdery mildews, blight and rusts | 200-400 ml/ha |
| 21 | Metalaxyl | Ridomil | Systemic against oomycetous fungi | 3 ml/L |
| 22 | Aureofungin | Aureofungin sol | Antifungal antibiotic | 50 g/ha |
| 23 | Validamycin | Val idamycin A 3% | Control of sheath blight | 2 ml/L |

| | | | | |
|---|---|---|---|---|
| 24 | Antibacterial Materials | Agrimycin-100 Streptocyclin, Plantomycin, Paushamycin | For control of bacterial diseases | 15 g/300L/ ha 750 g/ 500 L/ha |

## c) Herbicides

| | | | | |
|---|---|---|---|---|
| 1 | 2-4-D sodium salts | Fernoxone 80 WSP | Controls broad leaf weeds | 1.25 kg/ha |
| 2 | 2,4-D Ethyl | Agrodone concentrate 48 | -do- | 2.9 L/ha |
| 3 | Thiobencarb | Saturn 50 EC | Pre-emergence weed control in rice | 4 L/ha |
| 4 | Pendimethalin | Stomp 50 EC | -do- | 3 L/ha |
| 5 | Butachlor | Butachlor 50 EC Delehlor 50 EC, Machete 50 EC | -do- | 2 L/ha |
| 6 | Oxyfluorfen | Goal 23.5 EC | Pre-emergent weed control in rice and vegetables. | 85 ml/ha |
| 7 | Pretilachlor | Refit 50 EC | Pre-emergent weed control in rice | 2 L/ha |
| 8 | Anilofos | Aniloguard 30 EC, Arozin 30 EC | Early post emergent weed control | 1.3 L/ha |
| 9 | Diuron | Klass 80 WP, Hexuron 80 WP | Pre-emergence weed control in plantations and non crop area | 1.875 kg/ha |
| 10 | Atrazine | Atrazine 50 WP | Pre-emergence weed control in sugar cane and maize. | 2 kg/ha |
| 11 | Paraquat dichloride | Gramaxone 24 WSC | Non selective contact, post-emergent herbicide | 2 L/ha |
| 12 | Glyphosate | Round up 41 WSC, Weedoff 41 WSC, Glycel 41 WSC | Non selective post-emergent systemic herbicide | 2 L/ha |
| 13 | Dalapon | Hexapon 85 WSP, Dalapon 80 WSP | Control of grasses in tea, coffee and rubber | 3-7 kg/ha |

EC=Emulsifiable Concentrate, SC=Solution Concentrate, ai=Active Ingredient, WP=Wetable Powder (Sprayable Powder), DP=Dusting Powder, WSC=Water Soluble Concentrate, ha=Hectare, G=Granules

## RESIDUAL TOXICITY OF INSECTICIDES

| Sl.No. | Name of chemical | Waiting period |
|---|---|---|
| 1 | DDT | 2-3 weeks |
| 2 | BHC | 15-20 days |
| 3 | Lindane | 5-7 days |
| 4 | Aldrin | 2 years |
| 5 | DDVP | 24 hrs |
| 6 | Endrin | 2 weeks |
| 7 | Phosphamidon | 8-10 days |
| 8 | Monocrotophos | 3-5 weeks |
| 9 | Trichlorophon | 10 days |
| 10 | Parathion | 5-12 days |
| 11 | Methyl Parathion | 7-10 days |
| 12 | Fenthion | 10-14 days |
| 13 | Quinalphos | 1 week |
| 14 | Phorate | 3 weeks – 3 months |
| 15 | Formothion | 7 days |
| 16 | Phosalone | 2-3 weeks |
| 17 | Carbaryl | 21-30 days |
| 18 | Carbofuran | 20-30 days |
| 19 | Malathion | 3-5 days |

## RESIDUAL TOXICITY OF INSECTICIDES IN VEGETABLES

**(Waiting period)**

| Vegetable | Carbaryl | Fenitrothion | Quinalphos | Malathion | Fenthion |
|---|---|---|---|---|---|
| Ladys finger | 5 days | 3 days | 3 days | 3 days | 1 day |
| Bittergourd | 11 days | 4 days | 5 days | 4 days | 1 day |
| Brinjal | 5 days | 4 days | 3 days | 3 days | 1 day |

## Photosensitisation (light sensitisation)

It is a local or generalised reaction to bright sunlight causing inflammation and itching which in some cases is very severe.

**Primary photosensitisation:** This is caused by ingestion of photodynamic substances (i.e. substances following ingestion accumulate in the skin where it is activated by sunlight and cause tissue damage) Some wild plants like St John's wort ( *Hypericum perforatum*) and fagopyrin found in buckwheat ( *Fagopyrum esculentum*), certain drugs used in drug immobilisation (Ketamine and Phenothiazines e.g. Acepromazine, Propionyl promazine) can cause photosensitisation if exposed to excessive sunlight.

**Secondary photosensitisation:** This is also known as hepatogenous photosensitisation and occurs mainly due to liver damage resulting in poor or no excretion of phylloerythrin (a product of chlorophyll degeneration in the body). Phylloerythrin accumulates in the tissues including skin where it produces photodynamic activity. Ragwort ( *Senecio jacobea*) causes chronic liver disease in large animals. The sensitised animal suffers from dermatitis only when bright sunlight penetrates the skin sufficiently deep. The lesions caused by this type of photosensitisation are seen mostly on the back of elephants. It is seen in a triangular shape starting from the neck to the thoracic region, the base being the neck.

Treatment primarily involves taking the animal away from bright sunlight, administration of anti-histaminic, corticosteroids and local astringents and local application of lotions containing coppersulphate, zinc sulphate or tannic acid. These help to harden and protect the skin. The nature of photosensitivity should be investigated to determine the causal factors to prevent recurrence.

## NOTE: SNAKEBITES

Even though snakes are an inherent part of the elephant's natural habitat, rarely are elephant deaths recorded as a result of snakebites. Generally, small animals are more affected by snakebites than larger ones. Amongst human beings, children become victims of snakebites more often. This is true in the case of elephants as well. Calves are more susceptible as they have not yet learnt to be cautious against poisonous snakes. Their innate nature to be curious and inquisitive, and their use of trunks as a tool for this inquisitiveness, often leads to bites on the trunk.

Common poisonous snakes seen in India are vipers, cobras, kraits and king cobras. Of these, the venom of vipers (Russell's viper and saw scaled viper) is haemotoxic and that of cobras and kraits are neurotoxic. The bite of a viper produces a severe tissue reaction causing swelling, pain and sometimes bleeding. Hence in a viper bite, the bite site can be

located easily due to the severe local inflammatory reaction. Blood loses its coagulative property and the common test in a suspected case of a viper bite is the test for clotting time. However, it is not possible to conduct this test in a wild elephant. Since the venom of cobras and kraits are neurotoxic, muscle weakness will be evident and ptosis will be the first observable sign. Muscle weakness arises out of the curare like action of the poison. Passive venous congestion of all organs is a notable change.

In all cases of snakebites, the specific antidotes are anti-venoms. These are difficult to obtain and the quantity required for elephants is massive. The anti-venom is usually a combination for neurotoxin and haemotoxin and hence known as poly-anti-venom. Needless to say, anti-venom is specific for each snake and hence must be used only in specific cases. The supportive therapy, especially in viper bites, is to administer a heavy dose of corticosteroids, which help to prevent anaphylactic and allergic shock. Regarding the administration of antihistaminics, there are differences of opinion. Diphenhydramine hydrochloride (Benadryl) is used in several human cases. Calcium is often included in the supportive therapy as well. Theoretically, a neurotoxin with curariform action must be countered with neostigmine.

## WASP & HORNET STINGS

There have been instances where elephants when chased, disturbed wasp nests and were stung. Some human beings develop severe anaphylactic shock necessitating hospitalisation but elephants do not seem to develop any serious shock from wasp stings. There is no evidence for scorpion stings leading to death or serious illness in elephants. In one elephant of Kaziranga National Park severe serous exhudation was noted due to stings of bees. (Chakraborty, *pers. Comm.*)

## IRRITANT PLANTS

Cases of accidental consumption of irritant plants have been reported in captive elephants. These are mainly ornamental or cultivated plants like colocasia. Most of these irritations are caused by the presence of oxalate crystals, which are treated by local emollients applied to the mouth; however, this is not practical in wild conditions. The affected animal may be off feed due to an inflamed buccal cavity. The animal is also noticed to dribble saliva. It may drink water as a means to ease the irritation and pain.

## NOTE: WATER

Water presents a major source of toxicity compared to forage. There are many chemical compounds that are present in water, both of natural origin as well as contamination from different sources such as agro chemicals and industrial activities. The quality of water is estimated considering the following parameters:

1. **Total Dissolved Solids (TDS):** Constituents dissolved in water, which give its TDS value, affect the quality of water. TDS of up to 1000 mg per litre is not of much hazard to livestock and poultry. A level of 1000-5000 mg per litre may produce transient diarrhoea or reduced water intake in animals. TDS ranging from 5000-7000 mg per litre may produce toxic effects in large animals and TDS over 7000 mg per litre is unfit for animal consumption.
2. **Elemental Constituents:** Of the constituents dissolved in water, calcium, magnesium, sodium bicarbonate, chlorine, silicate and sulphate make major constituents ranging from 10-1000 mg per litre; iron, boron, strontium, potassium, fluoride, nitrate and phosphate are secondary constituents and range from 0.1-10.0 mg per litre. Arsenic, cadmium, copper lead, molybdenum and zinc are minor constituents (<0.1 mg per litre). Large increases in concentration in any or more of these constituents affects the quality of water.
3. **Hardness:** Salts containing calcium and magnesium affect the hardness of water. Its content > 120-180 mg per litre affects this quality.
4. **pH:** Elevated pH of water (>9.0) due to high ammonia levels affects its quality, such that it becomes highly toxic to fish. Heavily polluted water, fresh domestic sewage or treated effluents may contain 1-10 mg per litre of ammonia in water. The recommended safe level is <0.5 mg per litre.
5. **Sediments:** Undissolved materials remain as a suspension in water. They may absorb or transport a variety of organic and inorganic toxicants, which are not soluble in water (e.g. plant protection chemicals, heavy metals).
6. **Salinity:** High salinity reduces water intake by animals. Decreased feed intake often accompanies reduced water intake.
7. **Bacterial load:** There is a common laboratory test for bacterial count, which gives a measure of water quality in relation to animal excreta. Generally, coliform bacteria (*E. coli* levels greater than or equal to 5000 per 100 ml is considered to be unfit for consumption). The safe upper limit of toxic substances in drinking water is given below:

| **Substance** | **Safe upper limit** (mg per litre, PPM) | **Substance** | **Safe upper limit** (mg per litre, PPM) |
|---|---|---|---|
| Arsenic | 0.2 | Mercury | 0.01 |
| Cadmium | 0.05 | Nickel | 1.0 |
| Chromium | 1.0 | Nitrate | 100.0 |
| Cobalt | 1.0 | Nitrite | 10.0 |
| Copper | 0.5 | Vanadium | 0.1 |
| Fluoride | 2.0 | Zinc | 25.0 |
| Lead | 0.1 | | |

# GLOSSARY

**Abortion**-Termination of pregnancy before the stage of full term of gestation.

**Acidosis**-Excessive acidity in body fluids, due to accumulation of acids or excessive l oss of bicarbonate

**Adipsia**-Stopping drinking water

**Alkaloid**-Natural nitrogen-containing bases found in plants **Alkyl**-Any of a series of univalent groups of the general formula CnH2n+1 derived from aliphatic hydrocarbons

**Alopecia**-Loss of hair

**Anaemia**-a deficiency of red blood cells

**Anaesthesia**-Partial or complete loss of sensation with or without loss of consciousness

**Anisocytosis**- Condition which is characterized by a considerable variation in the size of cells, especially red blood cells.

**Anorexia**-A prolonged eating disorder due to loss of appetite

**Anoxia**-Deficiency of oxygen

**Anticoagulant**-Medicine that prevents or retards the clotting of blood

**Antidote**- A substance which can counteract a form of poisoning.

**Anuria**-Absence of urine formation

**Aplastic anaemia** – Anaemia resulting out of decreased formation of erythrocytes and haemoglobin

**Aqueous**- Similar to or containing or dissolved in water

**Arthritis**- Inflammation of a joint or joints.

**Ascites**-Accumulation of serous fluid in the peritoneal cavity

**Asepsis**-The state of being free of pathogenic organisms

**Asphyxia**- A condition of severe lack of oxygen supplied to the body.

**Ataxia**- Failure or irregularity of muscular co-ordination, especially that manifested when voluntary muscular movements are attempted

**Atelectasis**-Collapsed or airless condition of the lung.

**Atropine**- A poisonous crystalline alkaloid extracted from the nightshade family, used as an antispasmodic and to dilate the pupil.

**Axon**- Long nerve fiber that conducts away from the cell body of the neuron

**Azotemia**-Accumulation in the blood of nitrogen-bearing waste products (urea) that are usually excreted in the urine

**Biotransformation**-The series of chemical alterations of a compound (e.g., a drug) which occur within the body, as by enzymatic activity.

**Blanch**-Turn pale

**Bloat**- Condition in which stomach becomes overstretched by excessive gas content

**Bradycardia**-Abnormally slow heartbeat

**Brittle**-Having little elasticity; hence easily cracked or fractured or snapped.

**Bronchopneumonia**-Pneumonia characterized by acute inflammation of the walls of the bronchioles

**Carcass**- The dead body of an animal especially one slaughtered and dressed for food

**Cardiomyopathy**-A disorder (usually of unknown origin) of the heart muscle

**Cardiovascular**- The organ system which circulates blood around the body of most animals.

**Catarrhal**-Inflammation of mucous membrane particularly of nose and throat with increased production of mucous.

**Cathartic**-A purging medicine; stimulates evacuation of the bowels

**Cation**-A positively charged ion

**Caustic**-Concentrated solutions of strong bases, such as the hydroxide of alkali metals and alkaline earth metals.

**Centrifuge**- An apparatus that uses centrifugal force to separate particles from a suspension.

**Cerebrum**- Anterior portion of the brain consisting of two hemispheres

**Cessation**- To stop

**Chelation therapy-** A process involving the use of chelating agents to remove heavy metals from the body

**Chomp**- To make a snapping noise with the jaws in chewing

**Cholinergic**- Releasing or activated by acetylcholine or a related compound

**Cholinesterase**- An enzyme that hydrolyses acetylcholine (into choline and acetic acid)

**Cirrhosis**- Chronic hepatitis caharacterised by fibrosis, degeneration and hyperplasia of hepatic cells

**Clonic**- Alternate contraction and relaxation of muscles

**Colic**- A disease with severe pain and flatulent distension of the abdomen without diarrhoea

**Colitis**- Inflammation of the colon.

**Collagen**- A fibrous scleroprotein in bone.

**Coma**- An abnormal deep stupor/sleep from which patient cannot be aroused by external stimuli

**Congestion-** Excessive accumulation of blood or other fluid in a body part.

**Conjugation**-The state of being joined together

**Conjunctivitis**- Inflammation of conjunctiva (membrane that covers sclera)

**Conservation**- An occurrence of improvement by virtue of preventing loss or injury or other change

**Convulsion**- Temporary alterations in brain functions due to abnormal electrical activity of a group of brain cells that present with apparent clinical symptoms and findings.

**Cerebral or brain cortex**- An area of brain rich in neurons and the site of most sophisticated neural processing

**Corticosteroid**- A class of steroid hormones that are produced in the adrenal cortex.

**Cumulative**- Increasing by successive addition

**Cutaneous**- Relating to or existing on or affecting the skin

**Cyanosis**- Slightly bluish, greyish, or dark purple discoloration of the skin due to the presence of abnormal amount of reduced haemoglobin in the blood

**Dart**- A type of missile thrown or shot.

**Delirium**-A usually brief state of excitement and mental confusion often accompanied by hallucinations

**Demyelination**- Loss of the myelin covering of some nerve fibers resulting in their impaired function

**Dermatitis**- Inflammation of the skin.

**Detonate**-Process producing pressure much higher than normal combustion.

**Dilation**- The act of expanding something

**Diuretic**-Any drug that tends to increase the flow of urine from the body.

**Dyspnoea**- Air hunger resulting in laboured or difficult breathing

**Ecchymoses**- The escape of blood from ruptured blood vessels into the surrounding tissue to form a purple or black-and-blue spot on the skin

**Eczema**- Inflammatory conditions of the skin; particularly with vesiculation in the acute stages

**Elastin**-A fibrous scleroprotein found in elastic tissues such as the walls of arteries.

**Electrocution**- Killing by electric shock

**Electrolyte**- A solution that conducts electricity

**Electroplate**- The use of a solution of a metal salt, and an electrical direct current to coat an electrically conducting item with a layer of the metal making up the metal salt.

**Emphysema**-Pathological distension of the tissues by gas or air in the interstices

**Emulsion**-A colloid in which both phases are liquid

**Encephalitis**- Inflammation of the brain

**Encephalopathy**- Any disorder or disease of the brain

**Endemic**- Native to or confined to a certain region.

**Enforcement**- The act of enforcing; insuring observance of or obedience to

**Enterotoxemia**- A disease of cattle and sheep that is attributed to toxins absorbed from the intestines

**Epicardium**-The outer layer of the heart

**Epidemiology**- The branch of medical science dealing with the transmission and control of disease

**Epistaxis**- Bleeding from the nose

**Epithelium**- Membranous tissue covering internal organs and other internal surfaces of the body

**Erythema**- Abnormal redness of the skin resulting from dilation of blood vessels.

**Erythrocytes**- Mature blood cell that contains hemoglobin to carry oxygen to the bodily tissues

**Ester**- Formed by reaction between an acid and an alcohol with elimination of water

**Exhume**- Dig up dead bodies for reburial or medical investigation.

**Exophthalmia**-The protrusion of the eyeball so that the eyelids will not cover it

**Exostosis**- A benign outgrowth from a bone (usually covered with cartilage)

**Extensor-** A skeletal muscle whose contraction extends or stretches a body part

**Facultative**- Able to exist under more than one set of conditions

**Fasciculation**- Muscular twitching of contiguous groups of muscle fibers

**Fibrillation**- Muscular twitching involving individual muscle fibers acting without coordination

**Fibrin**- Protein responsible for the clotting of blood

**Fibrosis**- Development of excess fibrous connective tissue in an organ

**Fistula**- An abnormal passage leading from a suppurating cavity to the body surface

**Fluorescence**- Light emitted during absorption of radiation of some other (invisible) wavelength

**Fungicide**- Any agent that destroys or prevents the growth of fungi

**Galvanize**- Put a coat on.

**Gastroenteritis**- Inflammation of the stomach and intestines

**Gingivitis**- Inflammation of the gums.

**Glaze**- Give a shiny surface to clay pots, cups etc.

**Haemolysis-** Lysis of erythrocytes with the release of hemoglobin

**Haemorrhage**- Flow of blood from a ruptured blood vessels

**Hematoma**- A tumour like collection of blood as a result of hemorrhage as bruises or may develop on organs.

**Hematuria**- The presence of blood in the urine

**Hemoglobinuria**-Presence of hemoglobin in the urine

**Hemolysis**- Lysis of erythrocytes with the release of hemoglobin

**Hemosiderin**- Granular brown substance composed of ferric oxide; left from the breakdown of hemoglobin

**Hemosiderosis**-Abnormal deposit of hemosiderin

**Heparinise**-Adding heparin (injectable anticoagulant)

**Histopathology**-A branch of pathology that is concerned with the diagnosis of disease based on the microscopic examination of cells and tissues

**Hydrolysis**- A chemical reaction in which water reacts with a compound to produce other compounds; involves the splitting of a bond and the addition of the hydrogen cation and the hydroxide anion from the water

**Hydropericardium-** Accumulation of water in the pericardial sac without inflammation

**Hydrothorax**- Accumulation of fluid in the pleural cavity (the space between the lungs and the walls of the chest)

**Hyperaemia**- Increased blood in an organ or other body part

**Hyperaesthesia**- A state of exalted or morbidly increased sensibility of the body, or of a part of it.

**Hyperkalemia**- Higher than normal levels of potassium in the circulating blood

**Hyperkeratosis**- A skin condition marked by an overgrowth of layers of horny skin.

**Hyperpnea**- Energetic (deep and rapid) respiration that occurs normally after exercise or abnormally with fever or various disorders

**Hyperthermia**- A condition resulting from excessive exposure to heat without systemic disturbances

**Hypertonic**- In a state of abnormally high tension

**Hypocalcaemia**- Abnormally low level of calcium in the blood.

**Hypochromia**- An anemic condition in which the percentage of hemoglobin in red blood cells is abnormally low.

**Hypoglycaemia**- Abnormally low blood sugar usually resulting from excessive insulin or a poor diet

**Hypotension**- The condition of having blood pressure that is too low

**Hypothesis**- A proposed explanation for a phenomenon

**Hypothermia**- Subnormal body temperature

**Hypovolemic**- Of or relating to a decrease in the volume of circulating blood

**Hypoxia**- Decreased concentration of oxygen in the inspired air

**Icterus**- Yellowing of the skin, mucous membranes, excretions and the whites of the eyes caused by an accumulation of bile pigment (bilirubin) in the blood.

**Immobilise**- Cause to be unable to move.

**Inappetance**- Lack of craving or desire or wish; uninterested

**Incendiary**- A bomb that is designed to start fires

**Incinerate-** Become reduced to ashes.

**Incontinence**- Involuntary urination or defecation

**Inflammation**- A response of body tissues to injury or irritation; characterized by pain and swelling and redness and heat

**Ingesta**- Solid and liquid nourishment taken into the body through the mouth

**Inorganic**- Lacking the properties characteristic of living organisms

**Insufficiency**- The condition of being inadequate for its purpose

**Interface**- The overlap where two phenomena affect each other or have links with each other.

**Intoxication**- The physiological state produced by a poison or other toxic substance

**Kunki/Kumky**- Monitor elephants specially trained to handle wild elephants that are used in wild elephant capturing, driving deprading elephants etc.

**Lavage**- Washing out a hollow organ (especially the stomach) by flushing with water

**Legume**- An erect or climbing bean or pea plant of the family Leguminosae.

**Lesion**- Any visible abnormal structural change in a bodily part

**Lipolysis**- The breakdown of fat stored in fat cells

**Lymph node**- Glands of lymphatic system acting as filters.

**Maggot**- The larva of the housefly and blowfly commonly found in decaying organic matter.

**Medulla**- Lower or hindmost part of the brain; continuous with spinal cord

**Meningitis**- The inflammation of membranes covering the brain and the spinal cord.

**Mesentric**- Of or related to mesentry.

**Metabolism**- The organic processes (in a cell or organism) that are necessary for life.

**Methemoglobin**- A form of oxygen carrying protein hemoglobin in which iron in the haem group is in ferric state and not in ferrous state of normal haemoglobin which is unable to carry oxygen.

**Microsomal**- Relating to microsomes

**Microtome**- Scientific instrument that cuts thin slices of something for microscopic examination.

**Monogastric**- Having a single stomach

**Morbidity**- The percentage/ratio of deaths in an area to the population in a particular disease outbreak.

**Mortar**- A bowl-shaped vessel in which substances can be ground and mixed with a pestle.

**Mottle**- Mark with spots or blotches of different color or shades of color as if stained.

**Musth**- A condition in bull elephants, characterized by a thick, dark brown secretion from the temporal glands and showing unusually aggressive behaviour, and caused by a rise in reproductive hormones.

**Myasis**- Reflex contraction of the sphincter muscle of the iris in response to a bright light (or certain drugs) causing the pupil to become smaller

**Mydriasis**- Reflex pupillary dilation as a muscle pulls the iris outward; occurs in reduced response to a decrease in light or certain drugs

**Myelin**- A white fatty substance that forms a medullary sheath around the axis cylinder of some nerve fibers.

**Myocardium**-Muscular tissue of the heart.

**Myopathy**- Any pathology of the muscles that is not attributable to nerve dysfunction

**Necropsy**- An examination and dissection of a dead body to determine cause of death or the changes produced by disease

**Necrosis**- The localized death of cells or tissues (as from infection or the interruption of blood supply) in a living individual.

**Nephrosis**- A renal (kidney-related) syndrome characterized by edema and large amounts of protein in the urine and usually increased blood cholesterol

**Neuron**- Primary cells of nervous system

**Neurotoxin**- A toxin that acts specifically on nerve cells.

**Neurotransmitter**- Transmits nerve impulses across a synapse

**Nystagmus**- Jerking movement of eyeball either from left to right (horizontal nystagmus) or from above to below (vertical) or in circular direction (rotary nystagmus).

**Obligate**- Restricted to a particular condition of life

**Oedema**- Swelling from excessive accumulation of serous fluid in tissue

**Opisthotonous**- Severe spasm in which the back arches and the head bends back and heels flex toward the back

**Organic**- Of or relating to or derived from living organisms

**Organoleptic**- Effect or impression produced by any substance on the organs of touch, taste, or smell, and also on the organism as a whole.

**Osmosis**- Diffusion of molecules through a semipermeable membrane from a place of higher concentration to a place of lower concentration until the concentration on both sides is equal

**Osteoporosis**- Abnormal loss of bony tissue resulting in fragile porous bones attributable to a lack of calcium

**Oxidation**- The process of oxidizing; the addition of oxygen to a compound with a loss of electrons

**Pallor**- Paleness

**Parasympathetic-** A division of sympathetic and the parasympathetic system influences organs toward restoration and the saving of energy.

**Parenchyma**- The tissue characteristic of an organ, as distinguished from associated connective or supporting tissues.

**Parenteral**- Administered by means other than through the alimentary tract (as by intramuscular or intravenous injection)

**Pericardium**- Double-layered serous membrane that surrounds the heart

**Pervasive**- Spread throughout

**Pestle**- A club-shaped hand tool for grinding and mixing substances.

**Petechiae**- Small crimson, purple, or livid spots, like flea-bites, due to extravasation of blood

**Pharmacology-** The science or study of drugs: their preparation, properties, uses and effects.

**Phenolic**- A thermosetting resin

**Phlebotomy**- Surgical incision into a vein or a procedure that involves removing blood from the body for treatment.

**Phosphorescent**- A property of emitting light for a period of time after the source of excitation is taken away

**Pipette**- Measuring instrument consisting of a graduated glass tube used to measure or transfer precise volumes of a liquid by drawing the liquid up into the tube.

**Plaintiff**- A person who brings an action in a court of law

**Plasma**- Colorless watery fluid of blood and lymph containing no cells and in which erythrocytes, leukocytes and platelets are suspended

**Pleura**- The thin serous membrane around the lungs and inner walls of the chest.

**Poikilocytosis**- An increase in the number of abnormally shaped red blood cells.

**Polychromasia**- The reaction of red cells to stain as indicated by the appearance of bluish or grayish colored erythrocytes.

**Polydipsia**- Excessive thirst

**Polypeptide**- A peptide containing 10 to more than 100 amino acids.

**Polypropylene**- Thermoplastic polymer unusually resistant to many chemical solvents, bases and acids.

**Polyuria**- Excessive discharge of urine

**Porphyrins**- Any of various pigments distributed widely in living tissues

**Predispose**- Make susceptible

**Prognosis-** A medical opinion of the likely effect or a result of an illness.

**Prostration**- A condition marked by dizziness and nausea, weakness and lying flat on the ground caused by depletion of body fluids and electrolytes

**Proteinuria**- The presence of excessive protein (chiefly albumin but also globulin) in the urine

**Protoplasm**- a viscous translucent material that makes up the substance of all living cells.

**Purgative**- A preparation used for the purpose of encouraging defecation.

**Pustule**- A small inflamed elevation of skin containing pus

**Pyrethrins**- A group of closely related compounds used as insecticides, traditionally of plant origin now synthetic as well.

**Pyrexia**- A rise in the temperature of the body.

**Recumbency**- The act of leaning, resting, or reclining.

**Reduction**- Any process in which electrons are added to an atom or ion (as by removing oxygen or adding hydrogen)

**Resin**- Any of a class of solid or semisolid viscous substances obtained either as exudations from certain plants or prepared by polymerization of simple molecules

**Rhizome**- A horizontal plant stem with shoots above and roots below serving as a reproductive structure.

**Rigormortis**- A recognizable sign of death that is caused by a chemical change in the muscles, causing the limbs of the corpse to become stiff.

**Rumen-** The first compartment of the stomach of a ruminant; here food is collected and returned to the mouth as cud for chewing

**Ruminant**- Any of various cud-chewing hoofed mammals having a stomach divided into four (occasionally three) compartments

**Sclerosis**- Any pathological hardening or thickening of tissue.

**Seborrhoeic**- A disorder causing an excessive discharge of sebum from the sebaceous glands.

**Seizure**- A sudden fit or attack of illness

**Serous**- Of or producing or containing serum

**Serum**- Watery fluid of the blood that resembles plasma but contains fibrinogen.

**Shock**- Inability of the body to supply enough oxygen to meet tissue requirements.

**Smelting**- Extract (metals) by heating

**Spasm**- Violent and involuntary muscular contraction

**Stall-fed**- Kept and fed in stall in order to fatten for the market.

**Stasis**- An abnormal state in which the normal flow of a liquid (such as blood) is slowed or stopped

**Sterility**- Inability of the female to become pregnant or the male to impregnate a female

**Stomatitis**- Inflammation of the mouth.

**Strippling**- Dab paint up and down to give contrast or texture

**Subcutaneous**- Relating to or located below the epidermis.

**Synergistic**- Used especially of drugs or muscles that work together so the total effect is greater than the sum of the two (or more)

**Systole**- The contraction of the chambers of the heart (especially the ventricles) to drive blood into the aorta and pulmonary artery

**Tachycardia**- Abnormally rapid heartbeat

**Taxa/ taxon-** Animal or plant group having natural relations

**Tetany**- Clinical neurological syndrome characterized by muscular twitching and cramps and (when severe) seizures

**Therapeutic**- The act of remediation of a health problem after the diagnosis.

**Tonic**- Pertaining to or characterised by tension or contraction especially muscular contraction.

**Toxicologist-** One who studies the nature and effects of poisons and their treatment.

**Toxicology-** The branch of pharmacology that deals with the nature and effects and treatments of poisons

**Tranquiliser**- A sedative drug that depresses the central nervous system which causes calmness, relaxation, reduction of anxiety, sleepiness, slow breathing, staggered gait and slow uncertain reflexes.

**Tremor**- An involuntary vibration.

**Tubular**- Having hollow tubes

**Twitching**- Sudden muscle spasm

**Ubiquitous**- Being present everywhere at once

**Ulcer**- A circumscribed inflammatory and often suppurating lesion on the skin or an internal mucous surface resulting in necrosis of tissue

**Uremia**- Accumulation in the blood of nitrogen-bearing waste products (urea) that are usually excreted in the urine.

**Vacuolation**- The state of having become filled with vacuoles

**Vermin**- Any of various small animals or insects that are pests; e.g. cockroaches or rats

**Viscera**- Internal organs of a body.

**Wasting**- Emaciating; causing loss of strength or size

**Waterhole**- A natural hole or hollow containing water

**Xenobiotic**- Related to a substance foreign to the body or ecological system.

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# POISONS AND THE PACHYDERM

## RESPONDING TO POISONING IN ASIAN ELEPHANTS – A FIELD GUIDE

Elephants have always played a significant role in Indian society. They have been used as beasts of burden, as living tanks or battering rams by medieval Mughal armies and as one of the most versatile timber moving machines by loggers. In modern times, they are a major tourist attraction, and are used akin to the best four-wheel drive vehicles in inaccessible terrain. They have also been, for times immemorial, revered as Ganesha, the Hindu god of wisdom, prosperity and good fortune.

Today, with rising human populations, which are rapidly encroaching on what was once a habitat for these animals; human-elephant conflicts are not just leading to a large crop and property loss, but also killing a large number of people across India. It is, therefore, no surprise that the Gods are slowly turning into pests, fit to be eliminated. Moreover, the greed for illegal ivory is also claiming a large number of tuskers.

With a variety of synthetic poisons, pesticides and herbal toxins available, elephants have been at the receiving end in the last few years. Veterinarians, para-vets, wildlife managers and conservationists across India are facing situations where they need to react rapidly to cases where elephants have been poisoned. This reference volume, which lists out every known poison affecting Asian elephants, its symptoms, effects and antidotes, will thus prove handy to all those dealing with poisoned elephants, primarily to save them, and if not, to at least know, what killed them.

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10 Years in Service of Nature

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