A RETROSPECTIVE STUDY ON VETERINARY CLINICAL TOXICOLOGY WITH SPECIAL EMPHASIS ON VARIOUS SPOT TESTS IN EVALUATION OF TOXICITY

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ABSTRACT

This article reviews clinical toxicology with brief note on classification of poisons/residues based on toxic effect, origin, (phytotoxins and zootoxins) or use (pesticides, fungicides, herbicides etc.) and its therapeutic management in veterinary practise. Residues of chemicals can contaminate food-producing animals and contaminants in feedstuffs can carry over into animal products used as human foodstuffs. Animals are monitors of environmental safety and food safety. Special emphasis is given on to laboratory diagnosis using spot test to detect heavy metals, traces of toxicants from biological samples collected under veterolegal cases. A goal of this review is to encourage veterinary toxicologists and to update field practitioners in the forensic toxicology.

KEY WORDS: veterinary clinical toxicology, poisons, residues, spot tests and veterolegal case.

INTRODUCTION

Veterinary clinical toxicology is a very complex, yet fascinating subject as it deals with a wide variety of poisons of chemical, mineral, plant, fungal, and animal origins. It is a specialization in veterinary medicine focused on physical agents as the cause of disease. Animal feed safety, food safety and environmental safety are important aspects of veterinary toxicology. Residues of chemicals can contaminate food-producing animals and contaminants in feedstuffs can carry over into animal products used as human foodstuffs. Animals are monitors of environmental safety and food safety. Toxicology involves the knowledge of poisons, including their chemical properties, identification, and biologic effects, and the
treatment of disease conditions caused by poisons. Toxicology shares many principles with pharmacology, including the dynamics of absorption, distribution, storage, metabolism, and elimination; mechanisms of action; principles of treatment; and dose-response relationships. Determining the epidemiology of animal disease syndrome can identify large-scale contamination of animal feedstuffs with toxic substances. These incidences are of public health importance. Feed safety ensures that animal feedstuffs do not cause disease and residues of harmful chemicals in animals. Feed safety and human food safety are linked and exemplify the convergence on the “one medicine – one health” concept. Veterinary toxicology is a multi-faceted hybrid that draws on and contributes to the veterinary medical profession, the scientific field of toxicology and, broadly, to medical science.\[^1\]

A *poison* or *toxicant* is usually considered any solid, liquid, or gas that when introduced into or applied to the body can interfere with homeostasis of the organism or life processes of its cells by its own inherent qualities, without acting mechanically and irrespective of temperature.

The term *toxin* is used to describe poisons that originate from biological sources and are generally classified as *biotoxins*. Biotoxins are further classified according to origin as *zootoxins* (animal origin), *bacterial toxins* (which include *endotoxins* and *exotoxins*), *phytotoxins* (plant origin), and *mycotoxins* (fungal origin). Poisons may be categorized as organic, inorganic, metallic, or biological. A further distinction is made by some between synthetic and natural agents. The terms *toxic*, *toxicity*, and *toxicosis* are often misunderstood or misused. The word toxic is used to describe the effects of a toxicant (e.g., the “toxic” effects of organophosphate insecticides may be described as cholinesterase inhibition; vomiting, salivation, dyspnea, and diarrhoea). However, toxicity is used to describe the quantitative amount or dosage of a poison that will produce a defined effect. For example, the acute lethal dosage to cats of ethylene glycol would be described as 2 to 5 ml/kg body weight. The toxic effects of ethylene glycol are acidosis and oxalate nephrosis. Finally the state of being poisoned by a toxicant, such as ethylene glycol, is toxicosis.\[^2\]

**Classification based on toxic effects of poisons.\[^3\]**

The classification of poisons is based on the organ or system that is the ‘target’ site for the effect of the toxic chemical. Such classification that is based on the toxic effects of poisons on the body may be unsatisfactory because the same substance can have different effects on different organs of the body. It can also vary in its action between one species and another.
Therefore a relatively simple classification can be made on the basis of the structural features of the toxic chemicals that are responsible for their toxic properties and their affinity for 'target' sites in the animal body. Various diverse chemical compounds can be thus divided into two groups, namely, inorganic and organic compounds. Inorganic compounds include metals, metalloids, their salts and acids and alkalis. Organic compounds on the other hand include all carbon compounds other than carbonates, and the metallic carbides and cyanides.

**Gastrointestinal Syndrome**

This is seen in poisoning from oak, acute copper, E. coli, Salmonella, carbamate fungicides, ANTU, acute crotalaria, paraquat herbicides, thallium, bracken fern, warfarin, etc. The important clinical signs include: bloody diarrhea, hematuria, enteritis, sometimes GI irritation. Death may occur within one to a few days. In case of wild indigo, staphylococcus, peptides and amides, and borates deaths seldom occur. Severe gastrointestinal syndrome is caused by pokeweed, sneezweed, inorganic arsenic herbicides. Death may occur within 1-2 days with bloody diarrhea, hemolytic crisis (pokeweed), and excess salivation (sneezweed).

**Neuromuscular (paresis) effects**

Some poisons causing these effects act rapidly, some act slowly. Oleander, buttercup, buckeye, lead (in poultry) act rapidly causing death in 1-2 days.

Others include: death comas, poison hemlock, organophosphates. All the above cause in coordination plus GI syndrome whereas jimsonweed, 2, 4-D, organic tin, ticks cause only in coordination. Other examples of toxicants causing CNS effects include botulism, horse tail, bracken fern (in horses), pigweed, etc.

**Bone-Teeth-Hoof-Hair Deformities**

*Ergot-sloughing* of tips of tail, ears, teats, etc. *Fluoride* bone and teeth lesions Selenium (chronic), poison vetch, copper deficiency, chronic thallium, lead nitrate, chlorinated naphthalene, are all associated with hair problem.

**Kidney lesions**: Depending on the types of renal damage caused by toxicants kidney lesions are categorized as follows

1. **Hematuria**

   This may be accompanied by fever as in the case of bracken fern or there may be no fever as seen in poisoning from oak, cassia, inorganic: mercury or cadmium.
II. Hemoglobinuria
This is caused by crucifers (mustard) with photosensitization. Similar condition is seen in chronic copper toxicity.

III. Oxalates
These are formed in poisoning from amaranthus (which also causes perirenal edema), halogeton, black greasewood, rhubarb, and ethylene glycol.

IV. Perirenal Edema
Amaranthus and oak (with as cites); nightshade (no as cites).

V. Degenerative Changes in Kidneys
These can be caused by chronic organic mercury compounds, chronic thallium, sulfonamides and so forth.

Liver lesions
These are caused by several toxicants such as selenium (acute) aflatoxins, crotalaria, tannic acid, carbon tetrachloride, phenothiazines, gossypol, tarweed, etc.

Photosensitization
Phenolic fungicides (necrosis of contact tissue); vehicles for insecticides; blue-green algae (death within 1-2 days); crucifers (with hemoglobinuria), snow-on-the-mountain (without hemoglobinuria); phenothiazines, alfalfa; St. Johnswort, horse brush.

Hemorrhagic Syndrome
Bracken fern (with fever); those that do not have fevers include: sweet clover, warfarin, pindone, radiation, mycotoxins, crotalaria.

Fever: Castor bean, oleander, have rapidly acting toxic materials and death may occur within 1-2 days. On the other hand, milkweed, bracken fern, buckeye, locust, etc. are known to be slow acting.

Abortions and/or Anomalies
Fusarium fungal metabolities, poison vetch, lupine, broom weed, ergot, 2, 4, 5-T, nitrates. The following cause birth defects: chronic selenium, veratrum, oak, locoweed, jimsonweed, hem-lock, lead, and mercury.
Furthermore, an analytical toxicologist endeavours to separate poisons into characteristic groups and several classifications can be made according to the analytical procedures involved. A typical subdivision is

1. Volatile poisons
2. Metallic poisons
3. Toxic anions
4. Non-volatile organic poisons isolated by solvent extractions
5. Miscellaneous poisons

Finally, poisons may often be classified conveniently by their origin (plant poisons) or use (pesticides).

**Common toxicological problem encountered in domestic and farm animals**

**Dogs and Cats:** Pesticides, garbage, ethylene glycol, heavy metals, biotoxins (toads, snakes, and ticks), phyto-toxins, mycotoxins, drug reactions.

**Poultry:** Pesticides (very sensitive to insecticides), feed and water additives, fungi, bacterial toxins, gases and fumigants, heavy metals.

**Zoo Animals:** Largely malicious and quite variable situations, drug reactions, poisonous plants, accidental-organophosphates and warfarin baits.

**Exotic Animals:** Largely due to feed additives.

**Mink:** Botulism, chronic lead, phenolic wood preservatives, stilbesterol, etc.

**Rabbits:** Milkweed, toxic plants, neck paralysis and in coordination common.

**Turtles:** Paint on shell produces lump-back deformities.

**Cattle:** Heavy metals, pesticides, dietary and environmental contaminants (e.g., urea, nitrate, cyanide, mycotoxins), poisonous plants; snake and insect bites, drug adverse reactions.

**Sheep and Goats:** Poisonous plants - photo sensitizers, cyanogenetic, selenium, oxalate, lupine, sneezeweed, laurels, white snake root, larkspur, etc; Pesticides, anthelmintics, others - lead, nitrate, sulfur, fluoride.

Swine: Salt, coal-tar (pitch) and petroleum products, nitrates, wood preservatives, heavy metals, organic arsenicals, fungal toxins, poisonous plants, gossypol, insecticides, botulism, edema disease (endotoxins), rodenticides.

Therapeutic management of intoxication
Clinical management of intoxication can be organized into five areas
(1) ABCs of the critically ill patient: airway, breathing, circulation
(2) Gaining control of seizures or tremors
(3) Assessment of metabolic and medical derangements and institution of a plan for their management
(4) Gastrointestinal decontamination
(5) Supportive care

A brief note on antidotes.[4]
Animal disease emergency is a frequently presenting complaint in general and emergency clinics of Veterinary practice. Acute poisonings represent a diagnostic and therapeutic challenge for veterinary professionals, whether single or multiple animals are involved. The key to success in treating animals with toxicosis is early diagnosis and therapy. Prognosis varies considerably, depending on the toxin involved, the amount of exposure and the length of time that has elapsed between the exposure and presentation to the hospital.

The general approach to the poisoned animal includes stabilization of vital signs and institution of supportive care measures, obtaining a detailed clinical history and samples of the suspected toxic substance or source (e.g., feed samples, plants) if possible, decontamination to prevent further absorption of the toxicant, elimination of absorbed toxicants and administering a specific antidote if one exists and is available.

Selective antidotes and non-selective supportive drugs are used in the treatment of poisoning. Antidote (Greek word, anti = against, dotes = what is given) is an agent that selectively neutralizes or antagonizes adverse effect of a poison. Multifarious ways of antidotes action includes enzyme inhibition, receptor antagonism or chelation and are classified on the basis of their mechanism into chemical, functional and pharmacological antidotes.
Table - I: brief list of selective antidotes and their therapeutic indications

<table>
<thead>
<tr>
<th>Name of the antidote</th>
<th>Therapeutic indications</th>
<th>Dosage for large ruminants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine sulfate</td>
<td>Organophosphate/ carbamate insecticide toxicosis</td>
<td>Single dose @ 0.03-0.06 mg/kg, IM, SC, IV or Multiple doses up to 0.2 mg/kg</td>
</tr>
<tr>
<td>Ammonium molybdate And Ammonium tetrathiomolybdate</td>
<td>Copper poisoning</td>
<td>Ammonium molybdate @ 200 mg per head PO once daily for 3 weeks</td>
</tr>
<tr>
<td>British Anti-Lewisite (BAL) / Dimercaprol</td>
<td>Intoxication caused by Arsenic and Mercury (Anyimony, Bismuth, Cadmium, Chromium, Cobalt, Gold And Nickel)</td>
<td>@ 3 mg/kg, IM – every 4 hours for first 2 days followed by every 6 hrs. for 3rd day and 2 times daily for next 10 days until recovery.</td>
</tr>
<tr>
<td>Ethylene Diamine Tetra-Acetic Acid (EDTA)</td>
<td>Lead poisoning</td>
<td>@ 110 mg/kg as 1-2% solution in 5 % dextrose, IM, IP or slow IV – 2 times daily for 4 to 5 days. If required, dose may be repeated for 3 to 5 days depending on the severity.</td>
</tr>
<tr>
<td>Methylene blue</td>
<td>Nitrite/nitrate and chlorate poisoning</td>
<td>@ 4-8 mg/kg slow IV as 1% solution in normal saline. repeat in 6 - 8 hrs. (~ 20 mg/kg, if poisoning severity is high)</td>
</tr>
<tr>
<td>Pralidoxime (2-PAM)</td>
<td>Organo phosphorous Poisoning</td>
<td>@ 25 – 50 mg/kg, slow IV infusion, followed by IM route at 8-12 hrs. intervals. *It should not be used after 24-36 hrs. of poisoning.</td>
</tr>
<tr>
<td>Phytonadione (vitamin K1)</td>
<td>Anticoagulant rodenticide poisoning; sweet clover (dicumarol) poisoning</td>
<td>@ 0.5- 2.5 mg/kg IM or diluted with normal saline and given slowly IV; subsequent doses IM or SQ</td>
</tr>
<tr>
<td>Sodium thiosulphate (Na₂S₂O₃)</td>
<td>Cyanide poisoning in conjugation with sodium nitrite (Sometimes used for the treatment of copper, arsenic and Mercury poisoning)</td>
<td>Cyanide poisoning: 500 mg/kg slow IV as 25% solution &amp; 30g/cow, PO (to detoxify HCN in rumen if poisoning occurs through oral cyanogenic plants). Arsenic poisoning: 8-10 g total, IV as 10-20 % solution and 20-30g total dose, per os dilute in 300 ml of distil water. Copper poisoning: 500 mg per os, in conjugation with 200 mg of ammonium or sodium molybdate daily for upto 3 weeks.</td>
</tr>
<tr>
<td>Tolazoline</td>
<td>Reversal of Xylazine/ Xylazine over dosage</td>
<td>@ 2-4 mg/kg</td>
</tr>
<tr>
<td>Tetanus antitoxin/antitetanic serum</td>
<td>Prophylaxis and or therapeutic management of tetanus</td>
<td>@ 1,50,000-3,00,000 I. U. total dose, slow IV.</td>
</tr>
<tr>
<td>Vitamin K1</td>
<td>Anticoagulant rodenticides, sweet-clover toxicosis</td>
<td>@ 0.5-2.5 mg/kg IV, IM, SC.</td>
</tr>
</tbody>
</table>
Identification of poisons

Veterinary toxicology faces problems related to the general increase in the use of chemicals and especially to their increased use in livestock husbandry and agriculture. Clinical and laboratory methods are used in determining a disease is caused by a chemical or physical agent.

Sample of unknown origin - The test will be direct, if the sample is liquid. If the sample is solid, it should be dissolved in 10 ml of water (heat, if necessary). If it is not dissolved in water, shake up a small portion with 95% of ethyl alcohol and take a clear liquid. If neither solvent dissolves the solid, it should be shaken with ether.
Spot tests for metals.\textsuperscript{[5]}: One drop of unknown solution or one pinch of unknown solids at 3 sites marked as 1, 2, and 3. One drop of 10% Ammonium carbonate is added to sample 1, one drop of colourless 20% ammonium sulphide to sample 2 and one drop of 15% potassium iodide to sample 3. Colour obtained is thus matched with standard reference colours of inorganic salts of various metals.

Table - IV: chromatic references obtained from inorganic salts.

<table>
<thead>
<tr>
<th>Salts of metals</th>
<th>Ammonium carbonate</th>
<th>Ammonium sulphide</th>
<th>Potassium iodide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antimony</td>
<td>White</td>
<td>Orange</td>
<td>-</td>
</tr>
<tr>
<td>Arsenic</td>
<td>-</td>
<td>Yellow forms slowly, but soluble in excess</td>
<td>-</td>
</tr>
<tr>
<td>Barium</td>
<td>White</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Bismuth</td>
<td>White</td>
<td>Brownish black</td>
<td>Dark brown but soluble in excess</td>
</tr>
<tr>
<td>Cadmium</td>
<td>White</td>
<td>Yellow</td>
<td>-</td>
</tr>
<tr>
<td>Copper</td>
<td>Light blue</td>
<td>Black</td>
<td>Brown</td>
</tr>
<tr>
<td>Lead</td>
<td>White</td>
<td>Black</td>
<td>Bright yellow</td>
</tr>
<tr>
<td>Mercuric salts</td>
<td>White</td>
<td>Black</td>
<td>Green turning red</td>
</tr>
<tr>
<td>Mercurous salt</td>
<td>White</td>
<td>Brown, brown to black</td>
<td>Green, yellow red mixture “parrot colour”</td>
</tr>
<tr>
<td>Silver</td>
<td>White</td>
<td>Black</td>
<td>White turning purplish in light</td>
</tr>
<tr>
<td>Zinc</td>
<td>White</td>
<td>White, soluble in excess</td>
<td>-</td>
</tr>
</tbody>
</table>

Table - V: Identification of toxicants by chemical tests.\textsuperscript{[5]}

<table>
<thead>
<tr>
<th>Suspected Poison</th>
<th>Extraction</th>
<th>Test procedure</th>
<th>End point</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>Traces of atropine can be extracted by Stas-Otto process, slightly acidified by tartaric acid and rendered alkaline by small quantity of sodium carbonate</td>
<td><strong>Gerrard’s test</strong> - add 1 or 2 drops of 2% mercuric chloride solution to the test tube containing unknown sample</td>
<td>A rapid development of red colour</td>
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<td>Yellow colour turning red on warming</td>
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<td></td>
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<td></td>
<td>No change in colour</td>
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<tr>
<td>Arsenic (As)</td>
<td>Add 10 ml of aqueous saturated magnesium nitrate with magnesium oxide</td>
<td>Test no -1: A piece of mossy/pure Zinc is placed in test tube with Unknown sample.</td>
<td>Filter paper turning red indicates presence of arsenic, hydrogen sulphide or similar reducing</td>
</tr>
<tr>
<td>Substance</td>
<td>Procedure</td>
<td>Result</td>
<td></td>
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<tr>
<td>Magnesium Pyroarsenate</td>
<td>To make it alkaline and add 20 g of unknown sample to it and gently heat the mixture till the mass softens and chars. Then heat more strongly to get grey ash of magnesium pyroarsenate. Soak the ash in 25 ml of distilled water, filter and clear supernatant can be used for detection of As.</td>
<td>Filter paper turns yellow which may turn black after applying water indicates presence of As.</td>
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<tr>
<td>Barbiturates</td>
<td>5 ml of Unknown sample is dissolved in glacial acetic acid.</td>
<td>A white precipitate indicates presence of barbiturates.</td>
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<tr>
<td>Cocaine</td>
<td>Taken aqueous solution of unknown sample and add few drops of 30 percent aqueous potassium dichromate. Metzger’s test – a yellow precipitate obtained from the prepared extract, which dissolves on shaking. Now add few drops of conc. HCL.</td>
<td>Formation of orange yellow needle precipitates indicates the presence of cocaine.</td>
<td></td>
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<tr>
<td>Copper</td>
<td>Prepare aqueous solution of unknown sample. Add few drops of ammonium hydroxide to aqueous solution of unknown sample till the formation of greenish-blue precipitate soluble in excess of ammonium hydroxide.</td>
<td>Appearance of deep blue or purple solution indicates copper</td>
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<tr>
<td>Hydrocyanic acid (HCN)</td>
<td>HCN may be separated from organic matter acidified with tartaric acid by distillation with steam. It is present in first portion of the distillate. Test 1: dip strips of filter paper into a mixture containing 20 ml of aqueous mercuric chloride, 10ml of aqueous 2.5% methyl orange solution and 2 ml of glycerin. Dry it. Test 2: Scheerer’s test – take a strip of filter paper and dip in saturated solution of picric acid. Dry and soak in 10 percent sodium carbonate or sodium hydroxide solution till it gets dry under room.</td>
<td>Paper held to the mouth of unknown will turn pink in 2 min. indicates the presence of HCN. If HCN is given off, the paper turns pink or brick red.</td>
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<td><strong>Sindhu.</strong></td>
<td><strong>World Journal of Pharmacy and Pharmaceutical Sciences</strong></td>
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<td></td>
<td><strong>temperature. Take 5 ml of suspected sample in conical flask and add 10 percent of tartaric acid. Fix the filter paper in the neck of the tube and seal the neck. Heat the sample.</strong></td>
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<td><strong>Test 3: take 1 ml supernatant of centrifuged gastro-intestinal content in a test tube and add 2 ml of 10% sodium hydroxide solution, then add 10% HCL in sufficient quantity which can dissolve ferrous oxide precipitate formed.</strong></td>
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<td></td>
<td><strong>Formation of blue colour indicates presence of cyanide in the sample</strong></td>
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<tr>
<td><strong>Lead</strong></td>
<td><strong>Suspected sample must be mineralised to remove organic materials. Mineralisation may be done by either heating at high temperature (600°C) to produce ash or by boiling biological materials in 4:1 mixture of concentrated sulphuric acid and nitric acid in kjeldahl flasks (wet mineralisation).</strong></td>
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<td><strong>Test 1: Add 1 or 2 drops of dithizone reagent dissolved in chloroform to a small amount of the sample and shake thoroughly.</strong></td>
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<td><strong>Test 2: take 3 ml of clear aqueous solution of unknown and add few drops of 30% aqueous potassium dichromate solution. Then add 30% acetic acid solution until acid to litmus paper. A yellow precipitate of lead chromate indicates lead.</strong></td>
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<td><strong>Test 3: collect a small amount of scrapings from stomach wall and add four drops of concentrated nitric acid. Heat gently till dry and add a few drops of water and 2 drops of 10% potassium iodide solution. Presence of a marked yellow colour indicates lead.</strong></td>
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<tr>
<td><strong>Morphine</strong></td>
<td><strong>Extraction of morphine from biological matter is like atropine by Stas-Otto-process.</strong></td>
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<td><strong>Test 1: Husemann’s test – add 2 or 3 drops of concentrated sulphuric acid to the suspected residue and heat the mixture on a water bath for about an hour. Cool and add one or two drops of concentrated nitric acid or a crystal of potassium nitrate. A reddish violet colour which changes immediately to blood red and then to reddish yellow and finally fades away indicates morphine.</strong></td>
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<tr>
<td></td>
<td><strong>Test 2: Add a few drops of 5% neutral Appearance of blue colour which may fade,</strong></td>
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<tr>
<td>Component</td>
<td>Procedure</td>
<td>Result</td>
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<tr>
<td>Ferric chloride to the unknown powder.</td>
<td>indicates morphine. Red colour changing to black indicates apomorphine.</td>
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<td>Joint reaction - Mercury (Hg and Hg)</td>
<td>Dissolve the unknown in a mixture of 1 ml of concentrated sulphuric acid and 3 ml of concentrated hydrochloric acid. Add a few drops of 20% aqueous stannous chloride.</td>
<td>Formation of gray to black colour indicates the presence of mercury.</td>
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<tr>
<td>Nicotine</td>
<td>From the suspected sample, nicotine is extracted by alkaline steam distillation or alkaline Stas-Otto extraction.</td>
<td>A rose colour indicates nicotine. If formaldehyde solution is used in excess, a green colour is formed.</td>
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<tr>
<td>Joint reaction - Nitrate (NO₃⁻) and Nitrite (NO₂⁻)</td>
<td>Add 5 ml of diphenylamine reagent (1% in concentrated sulphuric acid) to 5 ml of sample.</td>
<td>The solution will take on a blue colour, if nitrite or nitrate is present.</td>
<td></td>
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<tr>
<td>Nitrite (NO₂⁻)</td>
<td>Test 1: take 2 drops of unknown on a slide and add 2 drops of sulphanilamide solution (1% in 1.5 N HCl) and add 2 drops of N-1-naphthylethylene diamine dihydrochloride (NEDD) (0.02% in absolute alcohol) solution.</td>
<td>Development of pink colour indicates nitrite.</td>
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<td></td>
<td>Take a sample of blood without anticoagulant in a test tube from a normal animal and a nitrite intoxicated animal and let them clot. Then put them in a boiling water bath for &gt; 45 min.</td>
<td>Nitrite containing blood will be salmon pink, do not pull away from tube and has either level or concave surface.</td>
<td></td>
</tr>
<tr>
<td>Nitrate (NO₃⁻)</td>
<td>Take 1 ml of suspected material and 1-4 ml of salicylic acid (5% in concentrated sulphuric acid).</td>
<td>Development of yellow colour indicates nitrate.</td>
<td></td>
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<tr>
<td>Oxalates</td>
<td>In a test tube take 1 ml clear stomach content or urine and add 0.2 ml concentrated ammonium hydroxide solution. Heat it on flames to evaporate the solution.</td>
<td>Formation of an orange red colour soluble in ethanol, indicates the presence of oxalates in the sample.</td>
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<td></td>
<td>To the residue add 40 mg of thiobarbituric acid crystals and gently reheat on burner.</td>
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<tr>
<td>Phenols/ Carbolic acid</td>
<td>Liebermann’s test – dissolve a crystal or drop of unknown in concentrated sulphuric acid and cool. Add a small crystals of sodium or potassium nitrite.</td>
<td>A deep blue or green colouration will be produced. When poured on a beaker of water, it is turned red which is again turned green or blue on adding alkali solution indicates presence of phenols.</td>
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<tr>
<td>Strychnine</td>
<td>Strychnine content in suspected sample can be separated by alkaline-chloroform extraction</td>
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<td></td>
<td>Test 1: Mandelin’s test – add 2 drops of ammonium vanadate (1% in concentrated sulphuric acid) solution to the unknown solution. Blue colour changes to brilliant violet.</td>
<td>To the coloured solution add 30% ammonium hydroxide solution, the colour changes to brilliant reddish-violet indicates presence of strychnine.</td>
<td></td>
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<tr>
<td></td>
<td>Test 2: take 1 ml of unknown solution and add 0.5 ml dilute tincture of iodine</td>
<td>Decolourization and precipitation of tincture of iodine indicates strychnine.</td>
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<td>Test 3: in a clean and dry petridish, take 1-2 ml of clear stomach content, urine or serum. Add equal quantity of concentrated sulphuric acid, mix well and add a few crystals of potassium dichromate and mix well. Examine the plate for change of colour for 5 – 10 min.</td>
<td>A change in colour from yellow orange to greenish blue indicates strychnine.</td>
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<td>Zinc</td>
<td>Add 5 ml of potassium ferrocyanide to 10 ml of unknown sample. Wait till white precipitate of zinc ferrocyanide is formed.</td>
<td>To the precipitate, add few drops of bromine water</td>
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<td>Lay 3 strips of filter papers on the cotton pas, one strip soaked in 5% silver nitrate solution, one in 5% mercury dibromide and one in 5% lead acetate solution. All the strips must have been completely dried over a moderate flame.</td>
<td>Formation of greenish yellow or yellow colour which on boiling forms a green or bluish green precipitate confirms the presence of Zn.</td>
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<td>Zinc phosphide</td>
<td>Take 10 ml of unknown sample in conical flask and add 10% of tartaric acid. Place a cotton wool pad soaked in 5% lead acetate solution on the top of the acidified solution.</td>
<td>A positive result is indicated if the paper soaked in a silver nitrate turns black, mercury dibromide paper turns yellow and lead acetate paper remains unchanged.</td>
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<td>If hydrogen sulphide penetrates the cotton wool the lead acetate paper turns black.</td>
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<td>To test the small quantities of zinc phosphide, place the test tube in a dark chamber and assess the result for the reaction after 24 hrs.</td>
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CONCLUSION
Veterinary toxicology, as a sub-specialty in veterinary medicine, had an applied origin – the diagnosis and treatment of toxicosis in domestic animals and companion animals. That important role continues today. However, the field has broadened to include concern for contaminants in human food products originating from animals and for contributing to the conduct and interpretation of safety/risk evaluations for pharmaceuticals, food additives, consumer products and specific chemicals. Veterinary toxicologists recommend treatments and antidotes for animals that have been poisoned, and treat poisoned animals. They also understand both normal and disease processes extending from the molecular level to the integrated mammalian organism and, indeed, populations, have an array of opportunities for making significant contributions to society.

REFERENCES