SECTION IV

TOXICOLOGY

(A) A GUIDELINE FOR ATTENDING POISONING CASES

Whenever cases suspected for Toxicity are reported to a Veterinarian, it is necessary to follow a certain line of action to tackle the problem from all possible angles.

The diagnosis of poisoning cases is many times very difficult. There is no single test by which laboratory can identify all types of poisons. Therefore while requesting for toxicological analysis, the details of circumstantial, symptomatic and pathological evidences along with suitable material on suitable preservative can give some clue to the Toxicology Laboratory" for arranging the specific diagnostic tests.

- (I) Historical or circumstantial evidences:— On reaching the actual site of exposure one should critically observe the premises and collect history on following, points:—
 - (i) No. and kind of animals (i.e. Species, Sex, Age group, stall fed/grazing) affected and died, whether animals belonging to one owner or different owners, whether from one locality or from different localities of the village are affected.
 - (ii) Source of feed fodder and drinking water (Collect the samples for analysis if doubt arises). It should also be noted whether all affected animals have a common source of feed/fodder/water.
 - (iii) Presence of poisonous plants (names) in the vicinity or grazing area and history of consuming such plants (Collect sample).
 - (iv) History of use of any Insecticide/Pesticide/Rhodenticide etc. in the animal house, premises in the pasture or crop field.
 - History of use of Chemical Fertilizers in the nearby field, fodder plot or their chances of mixing in the food or water,
 - (vi) Findings of empties (Tins/Bags) of insecticides, Chemicals, paints, fertilizers, medicines etc. near the site of poisoning.
 - (vii) History of spraying, dusting, deworming, vaccination or treatment undertaken recently in the affected animals.
 - (viii) Presence of any industry in the vicinity of place of exposure which is likely to pollute the source of drinking water, pasture or atmosphere.
 - (II) Symptomatic Evidence:— The close observations of the ailing animals may give some clue to diagnosis. This should include:—
 - (i) Incubation period i.e. time taken for exhibition of symptoms from the time of exposure.
 - (ii) Whether symptoms developed immediately after-consumption of feed/fodder or water.
 - (iii) Severity of symptoms acute/sub-acute/chronic.
 - (iv) Body temperature.
 - (v) Smell of breath/froth.
 - (vi) Any discolouration of visible mucous Membranes (m.m.).
 - (vii) Discolouration of urine.
 - (viii) Symptoms like salivation, lacrimation, nasal discharge, contraction/dilation of pupils.
 - (ix) Presence of constipation, diarrhoea, blood, mucous in the faeces.
 - (x) Buccal lesions if any.
 - (xi) Skin condition i.e. change in the body coat, odematous swellings, necrosis slouging of skin.
 - (xii) Various symptoms indicating disturbances in digestive (e.g. colic), respiratory (e.g. Dyspnoea), Cardiovascular (Pulse, oedematous swelling) and nervous system (e.g. convulsions)-shivering, spasms).
 - (xiii) Abnormality of teeth, bones, joint etc.

The following material should be collected from the ailing animals for toxicological as well as for Pathological/Bacteriological / Parasitological investigations.

- (1) Whole blood (citrated) in sterile vial on ice at least 5 ml.
- (2) Blood serum at least 2 ml.
- (3) Blood smears on clean slides-two.
- (4) Urine available.
- (5) Faecal sample.

(III) Pathological Evidence: — Whenever carcase is available post-mortem examination should be invariably performed, after obtaining requisition letter. Even delayed post-mortem material can be useful for Toxicological Investigations. The detailed post-mortem findings should be recorded carefully and the copy of the post-mortem report should be sent with the material collected. For toxicological investigations the collection of stomach/rumen content, intestinal content, liver and kidney in sufficient quantity on common salt is a must. If hydrocynic acid poisoning is suspected a muscle piece in 1 per cent mercuric chloride solution is useful.

Along with this, the formaline preserved tissue from important organs should be sent for histopathological examination. Similarly, to rule out the bacterial/viral infection suitable material should be sent on ice in sterile container (i.e. heart blood, lymphnodes, important organ, tissues).

Preventive Measures:— To prevent further exposure to the poison, measures like removal of animals from the place of exposure, change of source of feed, fodder and water should be advised.

Treatment of ailing cases:— If the nature of poison could be known the specific antidote can be used to treat the ailing cases. But in absence of it, symptomatic treatment and supportive treatment (e.g. fluid therapy, anti-histaminic drugs, liver stimulants) can be useful to save the life of ailing animal Adequate fluid therapy will also help in elimination of the toxin from the body through urine.

An universal oral antidote can be prepared as below :-

(i) Activated	charcoal	2	Parts
(ii) Magnesium	n oxide	 2	Parts
(iii) Tannic acid	i	1	Part
(iv) Kaoline		 1	Part

Dose .-

Cattle/Horse	250 gms	2 to 3 times a day
Calves	30 gms.	- Do -
Sheep/Goat/Pig.	15 gms.	- Do -

Dog 30 gms. shaken up with 300 ml. of water and administered during 24 hours.

In cattle and sheep this should be followed with purgative i. e. Mag-Sulph. 450 gms. in Cattle and 120 gms. in Sheep.

(B) COMMON VETERINARY POISONS

NON-VOLATILE

1. Insecticides.—

- (a) Organo Phosphates.— e.g. Malathion, Rogor, Dalf, Tick-20, Folidol-M, Sumithion, Diazinal.
- (b) Organochloro Compounds.— e.g. D.D.T., Aldrin, Endrin, Gammexine.
- (c) Organichloro Sulphur.— e.g. Endosulphan.
- (d) Organochloro Phosphorous.— e.g. Nuvon, Phosphamidon.
- (e) Carbonate compounds.— e.g. Sevin, Zineb, Baygon, Deetran, Tyrolan.

2. Plant Alkaloids .-

- (a) Cyno genetic plants.— e.g. Jowar, Hiverpoda, Dhatura, Ergot, Nerium, Canabis, Opium Stry-Chnine, Cocaine, Nicotana-tobacum, Belladona.
- Sulpha Drugs.— e.g. Sulphadiazine, Sulphaguanidine, Sulphadimidine, Sulphamerasine, Sulphamethazine, Sulphamilamide etc.

4. Miscellaneous Group.—

- (a) Fertilizers.— e.g. Ammonium Sulphate, Urea.
- (b) Anthelmentics.— e.g. Phenothiazine.
- (c) Sodium Salts.— e.g. Sodium chloride.
- (d) Other drugs.— e.g. Phenol, Barbiturates, Trypanocides, Purgatives.

Volatile.— e.g. Carbon Tetrachloride, Chloroform, Carbon Monoxide, Chloral Hydras, Kerosine, Formaldehyde, Turpentine oil, Ether etc.

Inorganic.— e.g. Mercury, Lead, Arsenic, Lime, Nickel, Copper, Iron, Phosphorous, Sulphur, Amtimony, Nitrates/ Nitrites, Solenium, Fluorine, Molybdenum, Acids/Alkalies.

(C) POISONS AND TREATMENT IN GENERAL

(A) Classification of Poisons :-

- 1. Poisons causing death by Anoxia. e.g. Nitrates, Corbon monoxide.
- 2. Corrosive Poisons. e.g. Acids, Alkalies, Heavy Metals, Gases.
- 3. Poisons with selective, toxicity nervous system. e.g. Anaesthetics, Alcohol, Narcotics.

(B) Metabolism of Poisons :-

Absorption of poison is through three portal entries 1. Lungs, 2. Skin, 3. Gut.

All soluble poisons are absorbed through vascular mucous, membranc of gut.

Inhalation of toxic substances through lungs.

Pesticides and Nicotine are absorbed through intact skin.

(C) Distribution and accumilation of poison :-

- 1. Poisons absorbed through intestine pass via portal vessels to liver where liver tries to detoxify foreign compounds.
- Some poisons are selectively deposited in certain organs/and tissues e.g. Iodine in thyroid glands, lead in bones, D.D.T. in fat.

(D) Elimination of Poisons :-

- 1. Insoluble poison is thrown out through faeces i.e. Lead, arsenate.
- 2. Volatile poison is excreted through lungs in expired air e.g. Cyanides.
- 3. Irritant poison is excereted through kindney.
- 4. Urine examination helps in detection of flourides.
- 5. Milk examination helps in detection of lead, arsenic and insecticide poisoning.

(E) Diagnosis of Poisoning:-

- 1. Vomiting, Diarrhoea, Abdominal pains. Zinc, Copper, Arsenic, Iron salts. Acids, Alkalies, Phenols, Turpentine.
- 2. Convulsions-Ammonia salts, Cyanides, Nitrates and Nitrites, Phenol, Strychnine.
- 3. Coma-Bromides, Carbon monoxide, Nicotine, Alcohol.
- 4. Muscular inco-ordination Ammonium salts, Boric Acid, Cyanide, Nicotine, Nitrates, Oxalates.
- 5. Dilatation of Pupil-Nicotine, Water hemlock, Hyoscine.
- 6. Contraction of Pupils-Opium derivatives.
- 7. Slow respiration-Atropin, Hypnotics
- 8. Rapid Respiration-Ammonium salts, Nicotin, Urea.
- 9. Dyspnoea-Carbon monoxide, Cyanides, Sulpuur Dioxide.
- 10. Lameness-Flourine, Ergot, Insecticides.
- (F) Common Antidotes: Any poison remaining in the stomach should be neutralized as far as possible by administration of suitable chemical antidotes.

1. Universal oral Antidote :-

Preparation -

(a) Activated Charcoal... 2 Parts.(b) Magnesium oxide... 2 Parts.(c) Tannic acid... 1 Part.(d) Kaoline... 1 Part.

Does :-

1. Cattle, Horses 250 gms. 2 to 3 times a day.

Calves
 Goat, Pigs.
 Sheep, Goat, Pigs.
 To pms.
 Do pms.

4 Dogs 30 gms. with 300 ml. water well shaked.

Mixture of this antidote should be given in three parts of a day.

- N. B.: Antidote given should be followed by saline purgatives 450 gms. mag. sulph. in cattle.
- Tannic Acid: In the form of solution in water, Tannic acid effectively precipitates aluminium, lead, silver and the alkaloids (Useless against arsenic trioxide.)
- 3. Iodine solution (15 drops of the tincture in half a glass of water) effetive for precipitation of lead, mercury, silver and certain alkaloides of quinine and strychnine.
- 4. Acid neutralization.- By giving lime water, mag. carbonate.

- 5. Alkali neutralization.— Vinegar (5 per cent acetic acid), lemonjuice, citric acid 5-10 per cent solution, Potassium permanganate solution has some value in oxidation of phosphorus. (Dilute solution, of KMnO₄ 1 in 2000).
- 6. Milk and egg white neutralize heavy metals and phenols.
- 7. Demulcents.- Mixture of eggs, sugar and milk to linseed-tea or oatmeal-gruel allays irritation caused by poison.

(C) Tteatment of Poison :-

1. Removal of Poison unabsorbed :-

- (a) On skin.-Wash with cold water and don't use hot water. Do not rub the affected part or area.
- (b) In gastrointestinal tract.-
- 1. Gastric lavage promptly either by neutralizing agent or with water.
- Emetics may be used when gastric lavage is immediately not available, Two table spoon of table salt in warm water, or one tea spoon of dry mustard powder.
- 3. Cathartics.- After gastric lavage only, doses of sodium sulphate one to two tea spoonful may be given.
- 4. Only activated charcoal is a good absorbent for organic and inorganic compounds. It may be mixed with gum acacia in equal parts with plenty of water and should be given only after gastric lavage.

2. Removal of poison absorbed :-

- (a) Through kidney.-
- (i) Diuresis with water (Not saline)
- (ii) Electrolytes,
- (iii) Parenterally. 5 per cent to 10 per cent solution of glucose (No saline water should be used)
- (b) Through lungs .- fresh air.
- (c) Intestinal Tract.- Sodium sulphate, cathartics may be used.

3. Symptomatic Treatment :-

- (a) Maintainance of clear airway by removing obstructions, Block by falling of tongue back and position may be corrected.
- (b) Control of respiratory depression by giving stimulants.
- (c) Stimulants: Caffine is an effective stimulant.
 - Dose. 0.5 gm. I/V Repeat if required every two hours.
- (d) Control of shock.-
 - (i) Protection from cold.
 - (ii) Inflation of Oxygen.
 - (iii) Replacement of losses of water and electrolytes.
- (e) Control of convulsions.- Give barbiturates by I/V route, if it is not possible then give by mouth.
- (f) Control of Infection.-

Secondary infections like bronchopheumonia, Skin Vesicless, and infection, of urinary tract may be corrected by giving proper antibiotic.

(D) HANDLING THE SPECIMEN MATERIAL FOR TOXICOLOGICAL EXAMINATION

- (1) The Toxicology Unit of Disease Investigation Section, undertakes the diagnostic work only. The Disease Investigation Section is not recognised for medicolegal cases. No material for such purpose be sent to the Disease Investigation Section. But it should be sent to the concerned Forensic Science Laboratory through Police authority.
 - (2) Material.- The material required and the preservative to be used etc. is given in the para No. 7 below.
- (3) Container.- The material be sent in wide mouth bottles of the appropriate capacity. The containers should be chemically clean.
- (4) Label.- Proper labeling is a "must" in absence of which the material is likely to be mixed with other materials and leads to a Confused stage of affairs in the laboratory. Label should be self-explainatory giving all details required. The label be typed or use of ball pen be made.
 - (5) Sealing.- (A) In case of Medicolegal cases the sealing is very important. Hence following points be noted.
 - (i) The bottles should be sealed with official seals. In absence of official seal the use of common articles like coins, keys, Buttons etc. should be avoided strictly.
 - (ii) The seal is to be done in presence of Panchas.
 - (iii) The copy of the seal used be sent along with the *Post-mortem* report in the space provided for the same in the report.
 - (B) In case of diagnostic case, material the sealing to stop the leakage is enough.

(6) Reporting.— The material sent to Disease Investigation Section (D. I. S.) for diagnosis should accompany the information about the symptoms, course of the disease, post-mortem lesions seen, treatment given and response observed along with the post-mortem examination report. The indication of a poison or a group of poisons is a must. It is of no use to send material and request to examine for "Toxicity, Poisoning".

The material sent to Forensic Science Laboratory should accompany the post-mortem report in proper prescribed form and in the covering letter the following information be sent—

- (a) Description of the animal or bird poisoned.
- (c) Indian Penal Code, Section under which the case is being investigated
- (7) Material required for Toxicological Examination.-
- (a) For sending the material for toxicological examination the thumb rule is that the organ/s by which the poison enters into the body and the organ/s by which it is excreted should be sent for examination. In case of cumulative poison the organ/s (where the poison is stored into the body) be sent for examination.

The following are the examples of the information given above, but is not exhaustive.

Sr. No.	Specimen/Material	Type of poison suspected				
1	Bile	Organic pesticides.				
2	Blood	Many types including nitrate, barbiturates,	warfrain.			
3 .	Bones	Chronic lead poisoning.				
4	Brain	Alkaloids, barbiturates.				
5	Fat.	Organic pesticides.				
6	Feed	All types.				
7	Hair	Chronic Arsenic poisoning.				
8	Intestinal Contents	Sub-acute poisoning.				
9	Kidney	Lead, thallium, ethiylene glycol				
10	Liver	Many types, including Arsenic Oxalates, alka	loids, pesticides			
11	Rumen Content	Many acute poisonings.				
12	Stomach content	Many acute poisonings.				
13	Urine	Most types, preferred for thallium or nitra	te.			

7(b) By and large the following material may be sent for Toxicological Examination to Forensic Science Laboratory or to Disese Investigation Section for medicological / dignostic investigation respectively.

Sr. N	o. Biological Material	Quantity Required	Preservative Used
1.	(a) Rumen Contents and the abomasal contents (in case of ruminants)	1 Kg.	For Sr. Nos. 1 to 4 use saturated solution of common salt.
	(b) Corp with gizzard and proventriculus.	Whole 10 in Nos.	Add sufficient salt, so that some salt remains undissolved.
2.	Small Intestine with its content.	250 gms. to 1 kg.	As above.
3.	Liver.	1 Kg.	As above.
4.	Half of each kidney	Half of each kidney. In case of birds whole kidneys at least ten in number.	As above.
5.	Urine	All available.	For each 200 to 500 ml. Of urine use 1 gm. of Sodi. Benzoate or 5 ml. of-Hydrochloric acid.
6.	Blood	100 ml. or whatever is available.	For every 10 ml. Of blood use mixture of Pot. Oxalate (30 mgs.) Sodi. Fluoride (10 mgs.)
7.	Suspected Taxi Cmaterial (Fodder, Seeds, Water, Chemicals etc.)	Sufficient Quality	

(E) IMPORTANT POISONS IN LIVESTOCK, THEIR SOURCES, ROUTE OF ENTRY, SYMPTOMS, LESIONS AND TREATMENT

Sr. No.	Poison	Source	Route / Species	Symptoms	P. M. findings	Treatment
1	2	3	4	5	6	7
1	Abrin	Seed of plant Abris precatorius (Gunj).	Cattle, Sheep Goats Horse, Pig, Either oral or prick by needle.	Local oedematous Swelling, Violent Purging, Spasms, inco- ordination, Stiff Joint anorexia Salivation, Nasal discharge.	Needle in Skin extensive oedema of S/C tissue. Haemorrhage in regional lymphnodes and on endocardium. Congestion of Vagus nerve.	 Antiabrin hyperimmune serum s/c and orally Pepsin hydrochloride acid orally. Saline purgative symptomatic and Suportive treatment.
2	Acids	Accidents in treatment, salts in plant, Insect bite, effluent from industries, Spray on potato etc.	Oral/Skin eyes Rare among Livestock.	Vomition, colic, convulsions, diarrhoea, carrosion of mucous membrance of buccal cavity, shallow breathing.	Dark blood, cyanosis of body, Inflammatory patches in G. I. Tract Subendocardial haemo- rrhage, burn wounds on the body in contact.	 (1) Do not use emetics/catheretics. (2) Dilute and Neutralise in situ with ample water / milk. (3) Burn should be flooded with ample water and then wash with dilute alkali like soap water or lime water. (4) Eyes wash with plenty of cold water, drop of castor oil, antibiotic eye oinment. (5) Give demulscents like milk, egg white and vegetable oil.
3	Alkali	Contaminated food, Soil, Water, Effluent from industries.	Birds Livestcok/oral Skin Eyes.	Shallow respiration muscular tremors, Tetany, dyspnea.	Nothing specific Burns and necrosis of the body in contact.	 (1) 1, 4 and 5 as in acid. (2) Neutralise by washing with weak acid like 2 per cent Acetic acid or venegar.
4	Aflatoxin	Fungus Aspergillus Flavus through feed.	Duckling Poultry, Broilers, Pigs Lethal does in ducklings 0.5 mg. Per B. W.	Blindness, Circling, grinding of teeth. In calves eversion of rectum, falling down Pigs- Jaundice and extreme apathy.	Fibrosed liver and neerotic foci. Kidney surrounded by wet fat, Ascites Haemorrhages in intestine and peritoneal Cavity, Hepati- ctumors in ducks.	No specific treatment Chick mash should be replaced. Avoid long stored food for feeding.
5	Antimony Compound e.g. Tartar emetics antimosan etc.	Accidents in treatment and over dosing.	Dogs Ruminants Cat Horse oral/skin and Parentral.	Vomition, colic, Diarrhoea, ataxia, stiffnes, convulsions, General Weakness.	Acute Gastroentritis Violent Purging Inflammation of G. I. Tract, Necrosis of liver and Kidney oedema of C. N. S. Increase in extra cellular fluid.	 Dimercaprol/ (BAL) 2 to 3 mg/1b B.W. 1/m is available as 10 per cent solution in oil for I.M. use. It should be given every 4 hrs. for first two days, four times on third day and twice daily for next 10 days untill recovery is complete. Tannic acid 5 to 10 gms. for adult cattle. Calcium hydroxide (lime water) Dose-mix 1 part in 4 parts of Drinking water. Magnesium oxide or milk of magnesia Dose 30 gms for adult cattle. Use oily purgatives with demulscents.

1	2	3	4	5	6	7
6	ANTU(Rhodenticide) Alphanapthylthic urea.	Lethal dose mg/kg. B. W. Cat-75 Dog-10 Pigs-25 Horse-30 Ruminants-50 Contaminated Feed ingestion of Rat, died of ANTU.	All animals and poultry Dogs are more susceptible oral.	Sudden on-set Salivation, Vomition Dyspnea, Coughing In co-ordination increased heart rate and sound muted due to accumulated fluid, foam from nostrils and mouth. Watery diarrhoea coma and death.	Cyanosis oedema of lungs Inflammation of G. I. Tract. Hydropericardium Liver enlarged and dark congestion and degenerative changes in Kidney.	 Use emetics or Gastric lavage. There is no specific antidote. Sedation with barbiturate to reduce dyspnea. Pressing of chest to remove fluid. 10 per cent Silicone may be administred to break some of the foam in the bronchi. Note: No treatment is likely to help on animal that is already comatose.
7	Arsenic compounds e.g. Calcium, Copper, Zinc, lead, magnessium arse- nated stovarsal atoxy.	Arsenical dip Dust, plants and surface Soil, water Accident in treatment	Farm animals e.g. Cattle Sheep, Pigs oral/Skin.	Acute: Vomition Colic, Salivation trembling, in co-ordination, paralysis of hind limbs, anorexia, thirst, dehydration, Cold extremities, Grinding of teeth, oedema of eyelids, Ulceration of buccal mucus membrane. Chronic.— Indigestion, thirst wasting, dry stairing, coat brick red colour of visible mucuous membrane poor growth, drop in milk sloughing of skin, abortion, sterility, oedema of eyelids.	Drying and cracking of skin, congestion of lungs, rupture of blood vessels and exudation of serum in tissues haemorrhages on pericardium, intense rose red inflammation of G. I. Tract, In Cattle, intestinal contents are blood tinged with mucous Haemorrhages on liver and Kidney. Pigs:—Congestion of mucus membrane of larynx and Trachea. Poultry:—Reddening of crop, duodenum, liver pale and friable.	In acute cases treatment is of little value. (1) Residual arsenic in the gut should be removed by administration of an only demulscent. Avoid drastic purgatives as dehydration is severe. Sodium thiosulphate (hypo) I/V and orally. (2) Dose: Cattle 8 to 10 gm. In the form of 10 per cent solution I. V. and 20 to 30 gms. Of water sheep and Goat ¼ dose of Cattle. Treatment should be continued till recovery occures (i.e. 3 to 4 days). (3) Dimercaprol (BAL) is useful in organic arsenic poisoning but not useful in inorganic arsenic poisoning for dose see antimony. (4) Freshly prepared ferric hydroxide solution ferric chloride 3 parts calcined magnesia I part water 17 parts. Dose - 500 gm. Repeat after 24 hrs. But efficiency is doubtful. It only postpones death. (5) In carnivora-Vomiting, gastric lavage with warm water and warm soap water, enema with warm soap water is indicated. (6) In herbivora a large dose of saline purgative is desirable followed by demulscents. (7) Supportive treatment like perentral fluid therapy to check dehydration and astringent preparations given orally to reduce the loss of body fluid.

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1	2	3	4	5	6	7
8	Attropic Alkaloids.	Plants-Dhatura Belladona, Stromonium Accidents in Treatment.	All Classes of Livestock Route oral and parentral.	Rapid irregular week pulse and respiration. Dilation of pupil frequent, urination. Muscular spasms Impaired vision Dry mouth.	Nothing Specific Inflamation of various organs Congestion of brain.	 Emetics and Gastric lavage in simple stomach animals. Activated charcoal to delay absorption. Symsptomatic treatment. Fluid therapy intravenously to maintain urine output.
9	Boric Acid, Cumulative poison.	Accidental ointment excessive use of calborogluconate food Preservative.	All animals M. L. Dose: — Dog - 2.5 gm. to 3 gm. Per kg. B. W.	Gastro intestinal irritation Nausea, vomiting diarrhoea increasing shock and death.	Cerebral oedema inflammation of alimentary track, degenerative changes in the liver and Kidney: Boric acid get concentrated in liver, kidney and fat.	Symptomatic treatment may be given large ½ doses of isotonic saline and of plasma as an antidote.
10	Cadmium	Pasture in the Vacinity of industries using Zinc. Environmental Pollution due to mines. Smelting plants, metallurgical galvanotechnical and dye work. Some phosphate fertilizers cadoxide anthranilate used as antihelmities for treatment of ascariasis in Pig. Cadmium plated vessels.	Cattle, Cows, Pigs Camel, Man Route oral and inhalation.	Acute- Reduced feed intake, severe G.I. Disorder, vomiting abdominal plain diarrhoea, spasm, collapse Irritation to respiration tract, coughing, laboured-respiration and vomiting. Chronic- Stunted growth, Chronic coughing anaemia proteinuria Pigs and Calves show symptoms of parakeratosis.	Cardiac hypertrophy gastro enteritis, morphological changes occur mainly in liver lungs and kidneys Also seen in testes. Pulmonary oedema and emphysema followed by bronchopneumonia. In chronic poisoning morphological changes may not be evident.	 (1) Dimercarprol (BAL) @ 2 to 3 mg/ lb B. W., i/m to be given every four hours for first two days. Four times on third day and twice daily for next 10 days until recovery is complete. (2) General and Symptomatic treatment of poisoning. (3) In acute poisoning gastric lavage.
11	Carbamate Insecticide.	Consumption of forage inadvertently sprayed by carbamate insecticides consumption of contaminated drinking water Accidental ingestion by any means.	Cattle Sheep Goat Swine	Similar as in organo-phosphorus poisoning.	Same as in organo-phosphorus poisoning.	Same as in organo-phosphorus poisoning.
12	Castor bean (Ricin Poison-Toxalbumin)	Feeding of plants in drought, feeding of uncooked castor cake.	Horses are more susceptible than Cattle Sheep and Pigs Poultry is resistant Route oral, MLD for cow is 2 gms/ Kg. B. W.	Nausea dullness powerful heart beats. Teatanic spasms in horse profuse sweating, Bloody purgation in cattle. Birds- Dullness Dropping of wings Paleness of comb and wattles.	Gastroenteritis mesenteric lymph Modes enlarged, Liver, kidney and spleen enlarged. In poultry oesophages, Crop and gizzard are inflamed.	 Emetice/Gastric lavage and catherosis. Specific antiserum is the best antidote. Symptomatic treatment. Fluid therapy. Orally sodium bicarbonate may be given to make urine alkaline to prevent. Precipitation of hemoglobin products in the kidney.

1	2	3	4	5	6	7
13	Chloral hydras	Accidents in treatment.	Horse, Dog other Livestock Route oral or parentral.	Relaxation of the voluntary muscles Staggering gait Dilatation of pupil, lowering of body temperature.	Nothing Specific.	 Artificial respiration. Analeptic C.N.S. Stimulant drugs e.g. Nikthamide / Leptazol should be used. Glucose saline i/v.
14	Chlorinated Hydrocarbons e.g. D.D.T. B.H.C., Aldrin, Endrin, Endoslul- phate.	Through intact skin, spraying on Pastures, fat soluble.	Cattle, Sheep Poultry Route of exerction is in milk.	Hypersensetive, Spasms of cervical muscles, forequarters and finally hind quarters. Salivation grinding of teeth, convulsions and respiratory failure. Violent shaking of body is prominent in organophosphorus poisoning.	Cloudy swelling of most of viscera - Small haemorrhages occur at random through out the body. - Endocardium is whitish in colour. - Lungs congested dark in colour. - Brain and spinal cord oedematus. - degeneration of liver.	 Treatment with anti dote as in Insecticide poisoning. Depreserts to control convulsions, Barbitraruates intraperitoneally. Saline Purgatives No oily purgatives If through skin, wash with plenty of cold water. Calcium borogluconate i/v. chloral hydrate in large animals. Supportive treatment.
15	Chlorates and chlorine.	Chlorinated lime (bleaching powder) used in error of lime wash Contamination of whey with hypochlorites used for disinfecting whey containers.	Route oral Skin Inhala- tion and Gas. Cattle buffalo, Sheep, Goat, Pig, Dog.	In aucts cases sudden death, confusing anthrax, Colic, purging, haematura, hypoxia, dyspnoea, general weakness. — increased respiratory efforts — cyanosis.	Body shows general cyanosis Blood is of dark chocolate colour pronounced brownish discoloura- tion of various organs and tissues, Gastroenteritis may be seen.	 Gastric lavage. Saline purgative. Methylene blue. 10mg/ kg. I/v. as 2 to 4 per cent sol. Per 100 lb body (45 kg. body wt.) to be repeated. In Dog blood transfusion. Glucose, solution intravenously and Egg albumen orally.
16	Chlorates e.g. Sodium chlorate (Weed killer)	Pastures and Dip.	Cattle more susceptible due to saline teste to Pasture Route-oral, skin.	Cyanosis and dyspnoea, Staggering, Purging and abdominal pains, haematuria and sudden death in acute cases confusing anthrax.	General Cyanosis Dark chocolate colour blood. Tarry blood from nostrils, Anus and Valva.	 Gastric lavage and saline purgatives methylene blue i/v 2 to 4 per cent solution @ 10mg/kg body weight. In dog glucose solution i/v, blood transfusion, egg albumen orally. Inhalation of oxygen.

17 Chromium

18

Drinking water contaminated with effluent from Chromium plating works Access to Zinc chromate used in hydroelectric engineering for cold Galvanising Accidental ingestion of potassium dichromate in horses

3

Cattle Calves Horses through ingestion.

Cattle - profuse scouring leading in chronic cases to dehydration Horses loss of appetite transient sickness, rapid and irregular heart beat, raised temperature respirations at first slow and then rapid and shallow, evanotic mucus membrane, abdominal pain and intense thirst.

Acute - Cattle - Severe Congestion, inflammation through out the intestine, sloughing of gastric mucus membrance.. In chronic cases rumen and abomasums showed severe ulceration.

6

Horses - couniunctival buccal and gastric mucus membrane showed signs of erosion and intestinal mucosa sloughed off, small and large gut contained blood stained fluid.

No Specific Treatment however symptomatic treatment of poisoning may be carried out.

Copper Compounds e.g. Copper Sulphate / Chloride/ Oxichloride / Oxide and Carbonates.

- Fungicide Pesticide Foot bath Contamination of feed and drinking water with copper salts, excessive use of copper sulphate is snail eradication. Chronic poisoning when soil is rich in copper. Ingestion of plants leading to abnormal copper metabolism e.g. Helitropum Echinum spp. Excess of Copper content in mineral mixture licks.

Route oral/wound and Cumulative poison Species Sheep Cattle Dog Pigs, dose MLD Cattle 200 mg/kg Sheep 20 mg/kg.

Acute - Nausea, Vomiting, Salivation Purgation, abdominal pain, convulsions and paralysis, faeces are deep green in colour

Chronic-

Constipation followed by diarrhoea, softening and of hair, anaemia, arthiritis, thickening of skin, Jaundice, bone deformity, infertility in both sexes.

Acute - Gastroenteritis, erosion and ulceration of mucus membrane of abomasum. congestion of liver, spleen and kidney, Contents of Stomach and gut are bluish in colour.

Chronic - Icterus, liver enlarged, fraglile and vellowish in colour Gall bladder B. with thick greenish brown bile, bending of ribs, overgroth with long bones, myocarditis, cystitis.

Note - In all animals treatment is of no use once hameolytic crisis starts

- (1) Calcium E.D.T.A. (available as 5 ml ampoule of 20 per cent solutions) Dose by I/v-15-25 mg/kg (i.e. 0.08-0.125ml - of 20 per cent Solution) per kg./B./W in 500 ml of 5 per cent Dextrose solution given very slowly twice daily. By I/M 12.5 mg/kg every four to six hours. Dilute each does with an equal volume of 1 per cent Procaine.
- (2) Penicilamine (Cuprimine) oral dose for small animals 100 mg./kg B. W. Daily (Maximum) 1 gm daily) divided into 4 doses for no longer than one week.
- (3) B.A.L. (Dimercaprol) has a doubtful value.
- (4) In simple stomach animals gastric lavage with 1 per cent solution of postassium ferrocyanate is advised to form insoluble Cupric ferrocynide.
- (5) For severe cases associated with anorexia and haematuria intramuscular injection of 52 to 100 mg of cortisone every 6 to 8 hours or 10 mg of prednesolone three times a day.

Compounds.

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20

Copper

Ergot (Selerotium of fungus the Claviceps Purpurea, Ergotoxin is the main toxic alkaloid).

Grazing Pasture contaminated with ergot fungus Cereal grass and crops like bajara-Cakes and feed (Repeated Ingestions).

Oral Route Spp. Cattle Man Pig and other animals.

Acute — (Nervous Symptoms) hyper-excitability, in coordination, tremors blindness fatal convulsions hind limbs first affected Coma and death.

5

Chronic - (Gangrenous Symptoms) Diarrhoea, Lameness, Coldness insensibility and sloughing of extremities for e.g. hooves, tail, ear, abortion mummification of foetus.

Injury to Capillary endothelium, blocking of vessels dry gangrene of feet, ears and tail and slough off without pain. Ulceration of mucus membrane of alimentary tract.

6

(7) Activated charcoal powder orally.

(6) Feeding of skimmed milk, white of an egg as demulscent.

(8) In chronic copper poisoning in Sheep 50 to 500 mg. of Ammonium molybdate and 0.3 to 1 gm. of Sodium sulphate orally daily for three weeks.

Preventions-

- (1) If Coper Poisoning is of soil origion the soil should be enriched with molybdenum about 300 grm/hectare.
- (2) Medicated salt bricks containing molybdenum should be used (e.g. Nacl 100 kg + 70 kg Gypsum + 0.5 kg Sodium Molybdate).
- (3) In Cases of exposure to risk following dose is reco recommended per sheep 0.3 to 10 grm. Sodium Sulphate and 50-100 grm of ammonium molybdate orally per day.
- (4) A Solution of Sodium Sulphate and ammonium molybdate may be sprayed on hay and feed for 2 to 3 weeks.

There is no treatment for ergot poisoning other than rest and removal of the source of the fungus.

21	Fluorine (Sodium, Barium and Calcium	- Smoke and dust from Industrial area affec-	Route — oral, and inhalation.	Acute- Vomiting anorexia, Colic, increased thirst, lacrimation,	Concentration in bones and teeth, Teeth easily break, colour of bone	(1) Gastric lavage with dilute lime water every four hours.	
	Salts.	ting plants soil, water - Feeding of Rock phophate in	Species—Calves Dairy Cattle, sheep, Goat, Hourse, Pig and	Pupils dilated, Mottling of teeth, Difficulty in chewing, lameness arthritis, green coloured faeces	changes to chalky, white and roughened, Exostosis of bones, haemorrhagic gastritis and	(2) Feeding of aluminium sulphate or chloride 0.53 per cent of diet.	
		Phosphorus deficiency. Drinking water from wells or artesian bores Dust and gases from	Poultry.	with mucus, anaemia, coma and death. Chronic- loss of appetite, constipation, followed by diarrhoea,	enteritis, Errosion and ulceration of Mucous membrane of Abomasum, Congestion of spleen liver kidney, hemorrhagic patches	(3) Special cakes, containing calcium, phosphorus, Vit. D to be fed to diminish fluorine absorption in the intestine.	
		Volcanic erruption.		emaciation, softening, and loss of hairs, thickness of skin, Jaundice, deformity of bones, over growth	on the heart bearing of ribs.	(4) The Calcium salts be given i/v to replace the precipitated calcium.	
				of long bones, infertility in both sexes.		(5) Parentral administration of glucose solution is recommonded because of interference by fluorine with glucose metabolism.	
						Prevention - Avoid Contaminated feed and water by mopping out the exposed territories.	
2 Fuel oils Kerosine (1)	(1) Contamination of	Oral / Intact Skin /	(1) Vomiting Shivering Consti-	Haemorrhages in lungs, Fatty	(1) Oxygen therapy.		
	Diesel.		feed and water in inhalation of Vapours storage. Cattle, pig and other	pation loss of appetite, Rapid and Shallow, respiration,	degeneration of liver. Broncho- pneumonia oedema and emphy-	(2) Gastric Lavage.	
		storage. (2) Licking of discarded	animal.	Muscular weakness.	sema of lungs in cattle Congestion	(3) Glucose I/v.	
		oil by grazing cattle.			of udder.	of udder.	(4) Use of olive oil to dilute the kerosine and diminish the rate of absorption.
						(5) Antibiotic injection.	
	Gossypol (Cotton	Cotton Seed Cotton	Oral-young Calves and	Loss of appetite, Slowing of	Gastroenteritis, Congestion of	(1) No specific antidote.	
	Plants).	Seed Cake and meal.	Preganent animals are	growth, Restlessness. Dyspnoea,	spleen and lymphnode, Oedema	(2) Symptomatic treatment.	
			more susceptible. Cattle, Sheep, Pigs, Dog Poultry	Ascitis, Convulsions, Foam from nostrils in chronic cases diarrhoea,	of lungs. Hydropericardium ascitis, Generalised oedema	Preventive measures-	
			and rabbits.	Vomiting and Weakness.	Necrotic Changes in liver and	(1) Cooking of cake before feeding.	
				Kidney,	Kidney,	(2) Add 1% calcium hydroxide or 0.1% ferrus sulphate to cake for detoxification of gossypol.	
	Iron	Excessive medication	Route - Oral, Parenteral.	Drowsyness, Vomition, pale skin,	Anaemic changes, blood stained	Milk of magnesia	
		i.e. ferrous sulphate if	Species - Piglets.	dark faeces, death due to acute	contents of stomach and intestine	Milk of lime	
		given in massive doses.	Dogs Children.	haemorrhage with diarrhoea, Symptoms of shock.	ulceration of stomach necrotic lesions in the liver. Severe	Shock - glucose and noradrenaline.	
					myodegeneration of skeletal		

. 3

(1) Accidental ingestion of paints based on lead compound.

3

- (2) Pesticide, containing lead.
- (3) Carelessly discarded lead batteries.
- (4) Air, Water and pasture, contamination due to industrial pollution.
- (5) Smoke from vehicle near petrol pump.
- (6) Contamination of feed, fodder, and water with greese, petrol, motor oil.
- (7) Use of lead pipe for drinking water.

Cattle Sheep Horse Dog and Cat, Pig, Poultry man. Goats are rather resistent.

Acute - Affected animals walk, in circle, push head against fences. appears to be blind, grinding of teeth, excessive salivation.

Muscular twitching, Violent blinking of eyelids, convulsions, weakness and prostrations.

Gastro enteritis

Elevated body temperature, weak pulse and respiration.

Chronic - Anorexia, depression, progressive, wasting, muscular weakness and General prostration are characteristic Blue line on gum, oedema of optic disc. Brisket and leg.

Constipation in some Animals.

Pustular erruptions on the skin

Grinding of teeth, Occasional attacks of colic. Heart and respiratory action becomes weak.

Abortion and sterility.

Sheep - Anorexia anaemia, faeces dark with offensive odour. aboration

Pigs- Slight diarrhoea, teeth grinding, anorexia, blindness, convulsions, incordination.

Dog - Vomiting along with haemoglobinurea. Paralysis of massetor muscle. Anaemia.

Horse - Colic, lead line on gums, roaring due to paralysis of laryngeal muscle, partial paralysis, with knuckling over pastern joint.

Gastroenteritis with haemorrhages.

Liver becomes pale.

Kidneys are congested and contain areas of haemorrhages.

Petechial to ecchymotic haemorrhages in heart, excessive fluid accumilation around brain and spinal cord.

After opening the carcass a typical smell and greenish red colouration of muscle is seen.

- Acute lead poisoning is always almost fatal.
- (1) Attempts be made to remove lead by giving saline purgatives emetics and by gastric, lavage.
- (2) Oral dosing with small amounts of
- (3) Milk, egg white or Tannic acid are
- (4) Calcium versenate (Calcium
- 12.5 per cent solution i/v. by continuous therapy may be repeated after, 7 days interval. This therapy is not free from danger. Toxic effects are damage to kidney, bone marrow, cardiac muscle and disturbed electrolyte balance if high doses are used.

2	3	4	5	6	7
0 Nitrate and Nitrites.	Artificial manures Containing Sodium Potassium Ammonium Nitrate well water plants, grown on soil contai-ning these salts. Crops like oat, Sugar, beet, Turniptops, Accidental mixing with feed and water.	Oral Cattle, Sheep, Goat, Pigs are most susceptible NB - Nitrates are converted into nitrites and are toxic. Due to Preservation of hay for long time.	 Sudden on set beginning with dyspnoea, violent respiratory, efforts, Salivation. Abdominal pain and diarrhoea. Muscular weakness and incoordination, convulsions cyanosis of visible mucus membrane, Abortion, drop in milk, death due to anoxia. 	Tarry blood, thickened pleura, Ascities, Necrotic foci on liver. - Petechial haemorrhages on serous surfaces, - In peracute case no characteristic lesion is seen.	 Methylene blue 4 per cent solution I/V is the best treatment. (9 mg/kg B. W.) Repeat if required. Large doses of mineral oil to slow the absorption of nitrates. Supportive treatments.
1 Snake Venom	Bite of Poisonous snake e.g. Cobra, krait, viper, etc. Four types of toxins. (1) Necrotizing (2) Coagulant (3) Neurotoxin (ex. Krait, Viper) (4) Haemolytic (e.g. Viper) Secondary Bacterial infection may be present.	All domestic Animals. Route S/c through fang marks more on legs and lips.	General Symptoms- Restlessness, excitement, depression Pupils dialated, not responding to stimulus, muscular twitching, in co-ordination, Convulsions, collapse and death. Local - Swelling at the site which develops rapidly. Acute cases - Symptoms appear within 15 min. of bite, Excitement anxiety coagulation of blood is completely lost, death due to pulmonary thrombosis of excessive haemorrhage. Subacute - More of local Swelling symptoms appear within one hour of bite excitement, convulsions, coagulability of blood not affected, Death due to paralysis of respiratory centre. Bite by other type snakes.— Local swelling deve lops rapidly and causes sever pain, excitement, anxiety. If neurotoxin is more dialation of pupil, Salivation, hyper esthesia, tetany, depression and terminal paralysis.	 Presence of fang marks in the centre of swollen area. Venom of both crotaline and Veperine snakes produce varying degree of swelling. Elapid venom causes only slight swelling and myonecrosis. 	 (A) Local Treatment (1) Clipping of hairs around the site of bite. (2) Tourniquet above the bitten part to restrict venous circulation to be released at 20 min. interval. (3) Immediate excision of bitten part (1 sq. inch) in size and 0.5 Cndeep. (4) Massage the part towards the incision (5) Suction of incised part may be beneficial. (6) Infilteration of antivenom around the site. (7) Systemic Treatment (8) Systemic Treatment (9) Polyvalent antivenom in large dose be given i/v as soon as possible unless specific antivenom is available. Dose: large animal 1 unit per 70 kg B.W. or more. Small animals - 5 units per 9 to 18 kg b. w. (2) Release of tourniquet. (3) Broad spectrum antibiotic to control local infection at the site. (4) Antitetanic serum 1500 to 3000 In U. S/c. (5) Cortisone acetate @ 50 mg I/m

water.

poultry.

salts.

pasture.

ment.

(4) Leakage

(4) Sea-fish meal for

(1) Accidental Injection

(2) Poisoning in Cattle

may be caused by

urea used as a

fertilizer and spread

unevenly on the

urea in Livestock

feed as non-protein

mitrogen supple-

(3) Uneven mixing of

Ammonia Gas.

Ammonium

Sodium chloride or salt poisoning Urea and Ammonia

2

- (1) Excessive intake of Poultry Chicks and salt. Briolers, Suckling Pigs, Dog and Cattle. Route (2) Salted meat in dogs and pigs. oral. (3) Scarcity of drinking
- Tremors and Twiching of skeletol muscles, pulse rapid and weak, Intense thirst.
 - In poultry wet faeces and leg Paralysis.
 - In swine convulsions, clamping of Jaws, salivation and diarrhoea.
 - Increase in respiratory rate. forceful breathing marked jugular pulse, painful coughing, Pulmonary oedema is characteristic.
 - Sever colic groaning, Shevering, staggering.
 - Tremor, fibrillar muscular twitching.
 - Salivation, photophobia, conjunctivitis and keratitis.
 - Bloating, tonic and Clonic spasms.
 - Death after violent struggling and bellowing.

- Gastro enteristis
- Generalised tissue oedema
- ureters full fo chalky deposits
- Carcass appears to be water logged
- Pigs thirst, Salivation, Cerabral Oedema.
- Large haemorrhagic patches on mucous membrane of stomach and intestine.
- Oedema and ulcers on intestinal m. m.
- Liver enlarged pale and friable.
- Blood fluidy and lighter in colour than normal.
- There were numerous petechae in the skin and throughout the carcase.
- Pulmonary oedema bronchi filled with purulent casrt containing debris of mucousmembrane.
- Nephritis.

Sheep, Goat inhalation.

Route-Oral Cattle,

birds.

Cattle - Violent Vomiting diarrhoea with blood in faeces, Abdominal pain, anorexia, dyspnea, icteric colouring, of visible m/m Rapid decline in milk production in cows, arched back and tucked up abdomen causing death within

Cattle - Generalise Pulmonary emphysema pale, flabby myocardium, blood spotting in the cortex and medulla of kidneys, marked degenerative changes in liver, Acute Gastroenteritis. Fibrinus exudates in forth stomach.

- (6) Supportive fluid therapy the sedative to control pain and excitement. Antihistanminic drug are contraindicated in the treatment of snake poisoning in dog.
- (1) Toxic feed/water should be removed.
- (2) Provide large quantity of safe drinking water.
- (3) Calcium gluconate.
- (4) In poultry immediate change of feed.
- (5) Symptomatic and supportive treatment.
- (1) Remove the source of Ammonia.
- (2) Provide ventilation and fresh air.
- (3) Administer weak acid like 2% Acetic Acid citric acid or 2% Venegar. Dose - 3 to 5 Lit. for adult cattle.
- (4) Use demulscents like milk and white of egg.

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- (5) Glutanic acid 50-200 gm in Cattle to be given orally in lukewarm water.
- (6) Give symptomatic treatment and antibiotics to prevent secondary bacterial, infections Convulsions can be controlled by careful administration of phenobarbitone sodium.
- (7) The only really and effective treatment is prompt and efficient emptying of Rumen either via a large bore tube or by Rumenotomy.
- (1) Removal of source of Zinc.
- (2) there is no known treatment for acute zinc phosphide poisoning.
- (3) Sod. Carbonate 1 per cent solution is administered as antidote to produce insoluble Zinc Carbonate.
- (4) Tannic acid and egg albumin may have similar effect.

- Zinc Poisoning
- (1) Chronic Poisoning may occur in the proximity of zinc ore. Rosting plants or other zinc processing work, either by contamination of pasture or drinking water.

Young animals are more susceptible Cattle, Sheep, Goat, Horse, Dog, Swine Route

Oral Inhalation of fumes from factory.

20 hrs.

1 2	3	4	5	6	7
	 (2) Consumption of feed mixture Containing, excess amount of Zinc salt particularly in pigs and fowl. (3) Metalic Zinc from 		In Chronic cases inhibits animal growth and induces fatigue. Pigs - Anorexia, unthriftiness, weakness convulsions, enlargement of joints, (Particularly shoulder).	Pigs - Non specific degenrative arthiritis. Oesteophoric, lesions in bone gastroenteritis pale flabby myocardium, blood spotting in cortex and medulla of kidney, degenerative lesions in the liver.	(5) use of emetic and laxative to remove the source.(6) Symptomatic treatment.
*	Zinc coated containers or pipes.				
	(4) Ingestion of Rhodenticide baits containing Zinc Phosphide.				
	(5) Contamination of Pasture by spray of fungicide containing Zinc salts.				
	(6) Contamination of water, milk, milk products, kept for long time in galvanized iron vessels.				
35 Oxalates	Oxalate containing plants (Oxalate content highest at leafy stage.)	Oral – Ruminants, Horse, Dog.	Dullness, loss of appetite Ruminal Stasis, Constipation, Lowering of head, Excessive salivation with frothing, Nasal discharge, Progressive incoordination, deep and irregular breathing, followed by subcutenious oedema at perineal, brisket, dewlap and abdominal region, oligourea and anuria, coma and death.	Lungs may be Dark red / purplish and filled with blood, petecheal or larger haemorrhages may be seen. Various tissues may show cyanosis, pereneal oedema and ascitis, accumulation of calcium oxalate crystals in kidneys and urinary bladder	Calcium carbonate (Lime Water) orally@one lit / animal twice in a day. Ruminotonics, Purgatives in early stages. Fluid in early stages, Inj. Calcium borogluconate 25% s/c Cattle: 300 - 500 ml. S & G: 50 - 100 ml.

No. DIS/TOXI - - 85,
Office of the Joint Director of Animal Husbandry,
Disease, Investigation Section,
M. S., Pune - 411 007.
Date:

CIRCULAR No. 1

PREPARATION OF PICRATE PAPERS FOR TESTING HCN

Take 0.5 gms. of picric acid (powder) and 5.0 gms. of Sodium Carbonate in a beaker. Add 100 ml. of distilled water, to prepare pieric acid solution. Cut the filter-paper into small rectangular strips. Dip the strips into this readymade solution. Remove the filter paper strips outside the solution, after they become completely soaked up with the solution. Allow the filter-paper strips (which will be now yellow in colour) to dry at room temperature. These strips are now picrate papers ready for the use of testing HCN poison.

SPOT TEST FOR HCN .-

Materials :-

- 1. Samples suspected for HCN-poisoning.
- 2. Chloroform.
- 3. Spirit lamp.
- 4. Test tubes.
- 5. Picrate Papers.
- 6. Beakers and Rods.

Procedure :-

- 1. Sample received is to be cut into small pieces and taken in the beaker.
- 2. Take about 10 gms. of sample into test tube.
- 3. Add few drops of chloroform into the test tube.
- 4. Heat the test tube on spirit lamp.
- 5. Hold the picrate papers for 3-4 minutes at the mouth of test tube against fumes.

Observations and: - 1. No change in colouration of picrate paper, indicates that sample does not contain HCN.

Inference :-

If the picrate paper turns brownish to red; then it is suggestive of presence of HCN in the Sample tested.

Note: Test is to be repeated for confirmation on large samples.

CIRCULAR No. 2

SPOT TEST OF NITRATE OR NITRITE POISONING IN SERUM, BLOOD AND URINE

(I) Nowadays there is an increasing tendency to use chemical fertilizers in cultivation for increasing the yield. Continuous feeding of such fodder to livestock, may, some times, lead to excessive nitrate/ nitrite, intake resulting in poisoning.

Therefore to detect nitrate/nitrite in serum, urine / blood, fodder the following quick field test may be useful.

Procedure:— Take 0.5 to 1.0 ml. of Diphenylamine Blue Reagent ((DPB) in the testing plate or glass slide and place one or two drops of suspected material into the reagent. If Blue Colour diffuses out of this material after 1 to 2 minutes, then the test is, positive for nitrate.

NB. :- DO NOT STIR

PREPARATION OF DIPHENYLAMINE BLUE REAGENT (DPB)

Take 1 gram of diphenylamine and dissolve the same in 100 ml. of concentrated sulphuric acid to give an almost water clear solution with a faint tint of blue colour.

If the diluted reagent is required for detection of lower concentration, 80 per cent Sulphuric acid is to be added in equal quantity of the above solution (DPB).

Reference: — Taken from "Observations on the Diagnosis of Nitrate poisoning In Sheep" by D. M, Helwing and B. P. Setehell; The Australian Veterinary Journal, January. 1960.

SPOT TEST FOR NITRATES IN PLANTS

Reference :- From "Disease of Livestock" by Hungerford.

Preparation of Reagent (DPB)

Take 0.5 gram of Diphenylamine in 20 ml. of water. Add concentrated Sulphuric acid to make 100 ml. Cool and Store in brown bottle.

For half strength add 80 per cent Sulphuric acid in equal quantity in the above solution.

Procedure :- Place one drop of the reagent on the freshly cut surface, of the plant which is to be tested.

Observation: If green colour turns to blue colour, then the test is positive for nitrates.

Inference: — Green to blue with half strength indicate 2% nitrate (which is dangerous to cattle).

(Sd.) X X X,

Joint Director of Animal Husbandry, Disease Investigation Section, M. S., Pune - 411 007.

DISEASE INVESTIGATION SECTION AUNDH, PUNE - 411 007

TOXICOLOGY UNIT

CIRCULAR No. 6

Sub. :— Control Measures to avoid Hydrocyanic Acid poisoning (HCN).

In circular No. 5 pathogenisis, toxic doses, symptoms, treatment etc. were described. The feeding of plants or fodder containing cyanogenic glucosides is on increase especially on Government Farms. It is, therefore, desirable, to note carefully the following circumstances about the possibility of Hydrocyanic Acid poisoning and take suitable steps in feeding of the livestock to avoid plant poisoning.

- 1. (A) Circumstances in relation to plant / fodder:— Mature cyanogenetic plants usually contain smaller amounts of the glucosides than younger plants. Generally, less hydrocyanic acid is produced from the dried plant than from fresh ones. The concentration of the Glucosides is more in leaves than in stems and the amount varies with and within strains. Nitrate fertilization and irrigation increase the glucoside content of such plants as Sorghums (Viz. Jawar, Maize and allied species). Spraying with 2, 4 D has similar effect on beets. The application of nitrogenous fertilizers is known to increase the amount of glucoside twenty fold, particularly when applied to poor soils. Second growth plants stunted by lack of water, frost, injury, or other unfavourable conditions are particularly dangerous wilted plants, including those resulting from trampling and from storm damage or frost, are considered to be of the greatest danger because hydrocyanic acid is immediately available to animals after consuming such plants,. Most of the potential hydrocyanic acid is reduced when plants are dried slowly, certain feeds, such as sorghums grown and harvested during the period of poor rainfall, are particularly dry at the time of harvest, dry completely in a short time, and thus retain enough glucosides to remain dangerous. Caution must be used in feeding any known cyanogentic plant with unsatisfactory growth, regardless of the cause.
- (B) Circumstances in feeding:— The feeding of concentrates tend to prevent the liberation of HCN from Sorghum which may be consumed within about 24 hours thereafter. Large amount of dextrose (Molasses) tend to reduce, harmful effect. Water deficient sorghum can be utilized, with safety if converted into silage adding sufficient water to insure fermentation. Hungry and greedy cattle should not have free access to sorghum, its poisoning depends on the rate of liberation of HCN, If cattle are fed with some dry fodder before they have access to sorghum the speed of intake will be reduced. Thus chances of H. C. N. poisonings will be lessened. If fodder is suspected to contain excessive amount of glucoside, prevention of poisoning may be made by adding 5% sulphur to salt licks.

If lineseed meal is to be fed, it should be fed in small quantities without soaking and gruel containing linseed meal should be thoroughly boiled to drive off free HCN.

No. VET/84/159/85/AH-9
Office of the Director of Animal Husbandry,
Maharashtra State, Pune - 411 001.
18th January 1985.

CIRCULAR

Subject:—Poisoning due to consumption of Jute seeds.....

Until recently jute was not a common crop in Maharashtra. However, because the jute seed fetches a lucrative price of Rs. 8 per kilo, the cultivaters are attracted to sow jute for seeds, and the area under jute cultivation is gradually increasing every year.

Recently two incidences of toxicity due to jute seeds have been reported, one from Tuljapur in Osmanabad District and the other from Dhanora, Taluka Jalgaon (Jamod) of Buldhana District. At Tuljapur, 15 bullocks engaged in threshing operation of jute, exclusively consumed the jute seeds, out of which 6 bullocks subsequently died. At Dhanora, a herd of about 375 heads of cattle grazed exclusively on the dried pods of jute plants, out of which 132 animals subsequently died.

It has therefore become necessary to prevent the animal population from consuming the jute plants, pods and seeds. All the animal owners in the State must therefore, be alerted in this respect so that they will not allow their animals to have access to jute plants, pods or seeds. The findings of the above two incidences are outlined below for the guidance of the field Veterinarians, to enable them to take prompt action in such cases.

Jute Plant :-

The jute plant belongs to the Genus Corchorus. Although there are many species of corchorus, only two species are known to yield jute fibre namely (i) Corchorus capsularis and (ii) Corchorus Olitorius. Out of these two species the seeds of Corchorus Olitorius are sweet and may be relished by animals, whereas the seeds of Corchorus capsularis being bitter in taste, may not be ordinarily eaten by animals. The sun-hemp which is cultivated for green manuring is altogether different from this variety of jute.

Active Principles :-

A cardio-active glycone isolated from seeds of Corchorus species is isomeric with (i) Corchor-genin, (ii) Corchortoxin and (iii) Strophanthidine. Their action is similar to Digitalin.

Toxic Dose :-

Cattle when consume the jute seeds at the rate of 5.0 gm. per kilogram body weight or more may show toxic effects. For buffaloes the dose may be little higher, whereas for smaller ruminants, it may be little less. For pigs, the toxic dose is 0.5 gm. per kilogram body weight and above.

Clinical Symptoms :-

The following symptoms were observed in the affected animals at the two places mentioned above :-

- (i) Suspension of rumination.
- (ii) Impaction with tympany.
- (iii) Dyspnoea.
- (iv) Diarrhoea followed by dysentry.
- (v) Colour of the faeces-blackish.
- (vi) Groaning suggestive of mild colic.
- (vii) Passing of undigested seeds in the faeces.
- (viii) Subnormal temperature before death.

Course :-

The first deaths were reported 36 hours after the consumption of seeds and the last deaths were reported on the 5th day after eating the jute seeds. So the course may be 1 to 5 days.

Post-mortem Lesions :-

The following gross changes were reported in the dead animals which were autopsized :-

- (i) Haemprrhagic enteritis,
- (ii) Seeds in the ingesta of four stomachs and intestines,
- (iii) Sero-sanguinous effusion in the pericardial sae,
- (iv) Petechial haemorrhages on the trachea, endocardiam, liver and ecchymoses in the abomasum,
- (v) Lungs congested and emphysematous,
- (vi) Kidney-Congestion in the medula,
- (vii) Liver-Enlarged with rounded borders,
- (viii) Colour of blood dark coffee coloured,
- (ix) Spleen-no enlargement.

Antidote :-

1. Attempts are in progress to ascertain the specific ancidote against jute poisoning. However, in the absence of knowledge about the specific antidote "POWDERED CHARCOAL" is found to be the most effective universal antidote. The dosage of the powdered charcoal is as stated below:—

Cattle ... 200 to 225 gms.

Buffaloes ... 250 to 300 gms.

Horses ... 200 to 225 gms.

Calves ... 25 to 30 gms.

Pigs ... 20 to 25 gms.

Sheep ... 25 to 30 gms.

Goats ... 25 to 30 gms.

Dogs ... 20 gms.

Two to three times a day after thoroughly mixing with sufficient water.

(If there are no symptoms of Gastro-enteritis, the administration of powdered charcoal may, be followed by a suitable purgative).

- 2. Methylene Blue: Methylene blue in doses of 5.0 miligram per Kilogram body weight, disolved in sufficient quantity of distilled water and injected intravenously can also be tried as a general antidote.
- 3. Sodium Hyposulphate: Sodium hyposulphate or sodium thiosulphate can also be tried as a general antidote in combination with sodium nitrate in the following doses:—

Cattle and Buffaloes:— Sodium Nitrate - 3 gms.
Sodium hyposulphate - 15 gms.
Distilled water - 200 ml.

(To be administered as Intravenous injection)

Sheep, Goat and pigs — The dose should be 1/5th of large animal dose i. e. 40 ml of the above injectable "sodium hyposulphate, which is known as hypo available in Photography shops can also be given" orally in suitable doses.

Symptomatic Treatment :-

Along with the administration of Universal/General antidotes as mentioned above, symptomatic treatment should also be undertaken like :—

- (i) Injections of relatively large quantities of glucose saline i/v to check dehydration.
- (ii) Soothing drugs in cases of Gastroentcritis.
- (iii) Stimulants in case of nervous depression (The symptom of nervous depression was not noticed in cases of jute poisoning).
 - (iv) Tranquilizers or Sedatives in case of convulsions (Convulsions were also not observed in jute poisoning).

Supportive Therapy :-

- (i) Supportive therapy should consist of 20% Dextrose solution i/v for energy in convulsing cases.
- (ii) Belamyl, Livogen or Liv 52 as liver tonics.
- (iii) Avil or Antihistaminic drugs wherever necessary.

IMPORTANT NOTE :-

REMOVE THE CONTACT OF THE ANIMALS FROM SUSPECTED POISON IMMEDIATELY

Material to be collected and sent for Laboratory Examination

By and large the following Material may be sent to the Laboratory for confirmation of the toxicity:-

Sr. No. 1	Material to be collected 2	Quantity required 3	Mode of collection <i>i.e.</i> (Preservative)	
1	Stomach Contents (a) Rumen and abomasal contents in case of ruminents	About one kilogram	Saturated solution of common salt. Add sufficient salt so that some salt remains un- disolved or on ice with a messenger	
	(b) Crop with gizzard and proventri- culus of Poultry.	Complete organs.	- do -	
2	A loop of small intestine with its contents.	About one kilogram.	- do -	
3	Liver	1 kilogram or whole liver from poultry	Saturated solution of common salt. Add sufficient salt so that some salt remains undissolved or on ice with a messenger.	
4	Kidney	Half of each kidney. In case of birds whole kidneys from 10 birds should be sent.	- do -	
5	Urine	Whole urine available in the bladder.	For each 200 to 500 ml. of urine use one gram of Sodium benzoate or 5 ml. chloroform or 5 ml. of dil. HCL.	
6	Blood	100 ml. or as much is available.	For every 10 ml. of blood use 30 mgs. of pot. oxalate and 10 mgs. of Sodium fluoride.	
7	Suspected toxic material. (Fodder, seeds, water, chemical etc.)	Sufficient Quantity.		

For clinical toxicological examination about 225 ml. of urine overlayed with a layer of Toluene and 100 ml. of citrated blood may be sent.

Conclusion :-

All the Veterinarians are requested to alert the cultivators on the possibility of poisoning due to consumption of jute seeds, so that the cultivators will not allow their animals to eat the jute seeds. In the event of any incidence of toxicity due to jute seeds, the guidelines suggested above should be immediately followed to save the affected animals. Wide publicity may be given to alert the farmers, through the media of work campaigns, farmers Shibirs, Public meetings, Radio news, T. V. broadcast etc. Information needed on any other point can be sought from Disease Investigation Section, Pune - 411 007.

(Sd.)

for Director of Animal Husbandry, Maharashtra State, Pune - 411 001.