VETERINARY TOXICOLOGY

Or POISONS USED FOR KILLING ANIMALS
IMPORTANT POINTS TO BE REMEMBERED

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- **Vetypedia** (Facebook - https://www.facebook.com/102160448212309?referrer=whatsapp)
- **Vetypedia** (Instagram) - https://instagram.com/vetypedia?igshid=u4c61r7i10xo
- **Vety exam Aspirants** (Telegram group for MCQs Discussion- https://t.me/joinchat/Gmb76hZrKdbwsTIMqadZsw)

TOXICOLOGY

- Toxicology is the study of poisons and their effects on living organisms
  - 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.

Branches of Toxicology

Poison

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- A POISON is any substance which, when ingested, inhaled, absorbed, or when applied to, injected into, or developed with in the body, in relatively small amounts, may cause damage to body structure or disturbance of function through its chemical action.
- Poison & poisonous animals have been of interest to humans since the dawn of creation.
- **M.J.B. Orfila** Spanish physician –
  - **referred as founder to toxicology – Father of toxicology**
- **GERHARD SCHRADER – Father of Nerve Agents**

Gerhard Schrader

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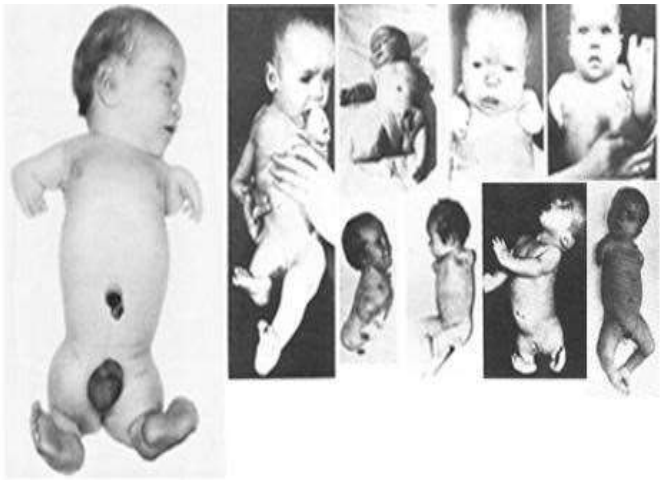
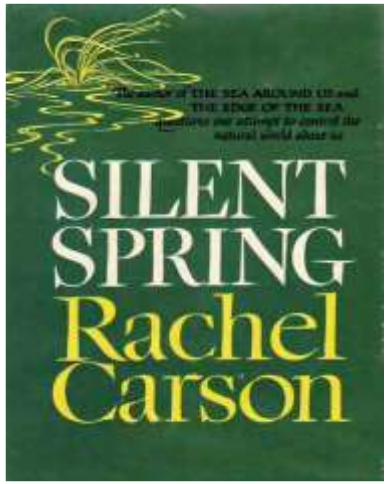
Major disasters related to Toxicology

Thalidomide - phocomelia (malformation of the limbs)

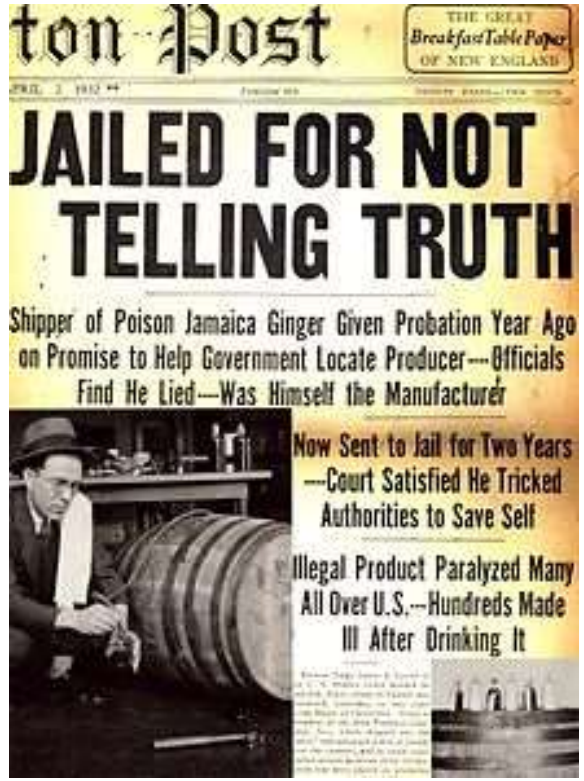


Minamata disease-Japan
Methylmercury

DDT on birds –
Silent Spring by Rachel Carson



Ginger Jake Syndrome
(Jake Leg Blues)
Organophosphate induced delayed polyneuropathy (OPIDN)
Tri-ortho-cresyl phosphate
Jamaica Ginger extract,(Jake)



Nuclear accident in Pennsylvania in 1979

Bhopal gas tragedy due to methylisocyanate in 1984
Chernobyl nuclear accident in 1986 -Ukraine

Fukushima Daiichi nuclear disaster

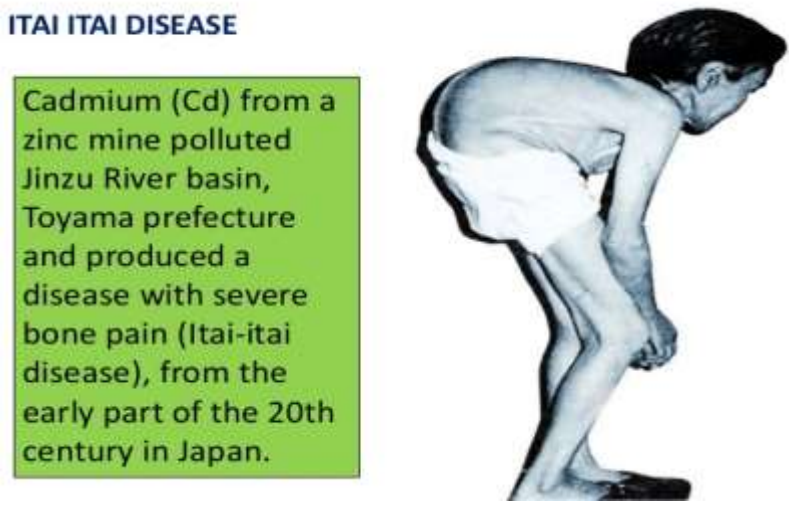
TOXICITY

It is the quality or degree of poisoning in animals and humans

In relation to frequency and duration of exposure

i) **Acute toxicity**: during 24 hour period
ii) **Sub acute toxicity**: for 30 days or less
iii) **Sub chronic toxicity**: for 1 to 3 months
iv) **Chronic toxicity**: for 3 months or more

Itai-itai disease- Japan- Cadmium (Cd)



In relation to time of development and duration of induced effects

i) **Transient/reversible/temporary toxicity**: for shorter duration of time e.g, necrosis produced by organic solvents
ii) **Persistent/permanent/irreversible toxicity:** persist throughout the life span and are of permanent nature e.g, testicular injury produced by dibromochloropropane
iii) **Immediate toxicity**: shortly after a single exposure to a toxicant e.g, cyanide toxicity
iv) **Latent or delayed toxicity**: has late onset of action e.g, peripheral neuropathy produced by some organophosphorus insecticides
v) **Cumulative toxicity**: progressive toxicity or harmful effect produced by summation of incremental injury resulting from successive exposures e.g, liver fibrosis produced by ethanol

DOSE

It is the total amount of a substance applied or administered all at one time to an individual to get the desired pharmacological or toxicological response

a) **Lethal dose**: lowest dose that causes death

b) **Median lethal dose**: lowest dose of a toxicant that is lethal to 50% of the animals

c) **Lethal concentration**: lowest concentration that causes death (milligrams of compound per kilogram of feed or water)

d) **Median lethal concentration**: concentration of a toxicant in feed or water that is lethal to 50% of animals exposed to it

e) **No observed adverse effect level (NOAEL): highest** dose level/concentration, causes no observable adverse effects

f) **Low observed adverse effect level (LOAEL): lowest** dose level/concentration, causes observable adverse effects

g) **Maximum residue limit/level**: maximum amount of a pesticide or drug residue that is legally permitted or recognized. It refers to pesticides and maximum residue level refers to drugs

h) **Acceptable daily intake**: estimated amount of a substance in food or drinking water that can be ingested daily over a lifetime by humans without appreciable health risk. It is used for food additives and term tolerable daily intake is used for containments

SOURCE OF POISONING

a) **Malicious Poisoning**: unlawful or criminal killing of animals
b) **Accidental poisoning**: accidentally or is added unintentionally in its feed, fodder or drinking water

Source of poisoning in animals includes

Natural sources:

a) Plants: Lantana, Datura etc
b) Animals: Accidental biting or stinging by poisonous reptiles, amphibians, snakes etc
c) Micro-organisms: Toxins produced by certain fungi and bacteria
d) Minerals: Metals and non metals from environment

Man made/Anthropogenic sources:

a) Industrial wastes: industrial effluents and wastes (chromium, nickel, lead) often leads to outbreak of poisoning in animals
b) Agrochemicals: pesticides, fertilizers in agricultural practices
c) Household products: like disinfectants (mainly phenol), solvents, cleaners etc
d) Therapeutic agents: improper dosage , by improper route or in improper species often give rise to toxic effects
e) Food and feed: in excess amount will cause harmful effects. Excessive use of feed additive like saccharin are known to produce adverse effects in laboratory animals
f) Environmental Pollution: Pollutants in air, water and soil can cause harmful effects in animals
g) **Improper managemental practices**: Poor ventilation, over crowding and diet deficient in essential nutrients aggravate toxic effects of certain poisons (CO_2, ammonia, CH_4 etc)

CLASSIFICATION OF TOXICANTS

a)On the basis of source

Plant toxicants	Strychnine, morphine, opium, curare
Animal toxicants	Zootoxins and venom
Minerals	Copper, lead, selenium
Synthetic toxicants	Organophosphates, pyrethroids

b)On the basis of organ/system affected

Neurotoxicants	Organophosphates, barbiturates, nicotine
Hepatotoxicants	CCl_4, ether, paracetamol, Lantana camera
Nephrotoxicants	Oxalates, lead, arsenic, cadmium, aminoglycosides
Pulmonotoxicants	ANTU (alpha- napthyl thiourea),Paraquat
Haematotoxicants	Warfarin, phenothiazines
Dermatotoxicants	Coal tar compounds, arsenic, mercury

c)On the basis of physical state

Gaseous toxicants	Hydrocyanic acid, sulphur dioxide
Liquid toxicants	Sulphuric acid, nicotine
Solid toxicants	Strychnine, opium, atropine
Dust toxicants	Asbestos dust, silicon dust

d)On the basis of analytical behaviour

Volatile toxicants	Hydrocyanic acid, alcohol, phenol
Extractive toxicants	Alkaloids, glycosides
Metals and metalloids	Copper, mercury, zinc

e)On the basis of toxic effects

Carcinogens	Thiouracil, nickel
Mutagens	UV light, nitrogen mustards
Teratogens	Triazines, thalidomide
Clastogens	UV light, caffeine

f)On the basis of chemical nature

Inorganic toxicants	
Metals	Lead, copper, mercury
Non-metals	Phosphorus, sulphur, nitrates
Acids and alkalies	HCl,KOH
Organic toxicants	
Hydrocarbons	Benzene, cyclopropane
Halogenated hydrocarbons	BHC, DDT , CCl_4
Alcohols and phenols	Ethyl alcohol, phenol
Ethers	Diethyl ether
Aldehydes and ketones	Formaldehyde
Organic acids	Formic acid, salicylic acid
Esters	Organophosphates, pyrethroids
Amines	Adrenaline, amphetamine
Amides	Sulphonamides
Glycosides	Cyanogenetic glycosides
Alkaloids	Nicotine, atropine
Proteins	Ricin, abrin

g)On the basis of use

Insecticides	Organophosphates
Fungicides	Pentachlorophenol
Herbicides	2,4-D
Rodenticides	Warfarin
Food additives	Benzoic acid, ascorbic acid

Contd...

h)Toxicity potential

Group	Lethal dose
Extremely toxic	< 1mg/Kg
Highly toxic	1-50 mg/Kg
Moderately toxic	50-500 mg/Kg
Slightly toxic	0.5-5g/Kg
Relatively non toxic	5-15g/Kg
Practically harmless	>15g/Kg

i)On the basis of mechanism of action

Cholinesterase inhibitors	Organophosphates, carbamates
Sulphydryl(SH-) inhibitors	Mercury, arsenic, thallium, lead
Protoplasmic toxicants	Heavy metals = lead, cadmium
Methaemoglobin producers	Nitrates, chlorates
Cytochrome oxidase inhibitors	Cyanide
Krebs cycle inhibitors	Fluoroacetate
Uncoupler of oxidative phosphorylation	Dinitrophenols

j)On the basis of environmental, public health consideration

Air pollutants	Carbon monoxide
Water pollutants	Pesticides
Food and feed contaminants	Mycotoxins, antibiotics
Radiation hazards	X-rays, radioactive minerals

Common toxicological problem in domestic and farm animals:

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

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

Zoo Animals: Largely malacious and quite variable situations, poisonous plants,accidental-organophosphates/warfarin baits.

Exotic Animals: Largely due to feed additives.

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

Rabbits: milkweed, toxic plants, neck paralysis and in coordination.

Turtles: paint on shell produces lump-back deformities.

Cattle:Heavy metals,pesticides, dietary/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,white snake root, larkspur, etc; pesticides, anthelmintics,lead, nitrate, sulfur, fluoride.

Horses: Poisonous plants-oleander, bracken fern, castor bean, locoweed,lupine,selenium containing,crotalaria,cyanide; pesticides, snake and insect bites, other aflatoxins, heavy metals, toxic gases.

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.

Clinical signs

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- must be used to initiate a search for poisons and should not be used alone for diagnosis. List of signs associated with certain poisons are given below
- **Central nervous system**
  - **Convulsions**: Ammonium salts, cyanide, lead, nitrite, strychnine, organochlorine and organophosphates, **Coma**: Bromide, carbon monoxide, alcohol, phenol
  - **Ataxia**: Bromide, alcohol, thallium, hypnotics
- **Digestive system:**
  - **Nausea, vomition, diarrhoea**: Arsenic, fluoride (acute poisoning), copper, selenium (acute poisoning), organophosphorus
  - **Constipation**: Lead, alcohol, thallium, oxalate **Hypersalivation**: Organophosphorus and carbamates
  - **Dry mouth**: Atropine, opioids, phenothiazines **Gastrointestinal bleeding**: Aspirin, caustics, oral anticoagulants
- **Respiratory system**
  - **Dyspnoea**: Alcohol, hypnotics, opioids, organophosphates, carbamates, **Hyperpnoea**: Aspirin, ethylene glycol, atropine (early stages)
  - **Slow respiration**: Selenium and atropine (late stages) **Pulmonary oedema**: Aspirin, ANTU, paraquat
- **Skin, hair and nails**
  - **Cyanosis**: Due to methaemoglobin formation. Example nitrite, chlorate, nitrobenzene **Alopecia**: Thallium, x-radiation, arsenic (chronic toxicity)
  - **Photosensitization**: Phenothiazine, *Lantana camara* **Staining of skin**: Iodine (black), bromide (deep blue), picric acid (yellow) and silver nitrate (blue black)
- **Eyes:**
  - **Dilation**: Belladona, atropine, cocaine, amphetamine, nicotine (late stages) **Constriction**: Opium derivatives, organophosphorus, carbamates, nicotine (initial stages)
- **Urinogenital tract**
  - **Urinary retention**: Opioids, atropine **Frequent micturition and incontinence**: Organophosphorus and carbamates
  - **Haemoglobinurea**: Chlorate and copper (chronic) **Colouring of urine**: Phenol and cresol (dark green), picric acid (yellow colour), phenothiazine (pink)
- **Body temperature**
  - **Hyperthermia**: Organochlorines, pentachlorophenol, dinitrophenol **Hypothermia**: Alcohol, opioids, sedatives
- **Musculoskeletal system**
  - **Incoordination**: Ammonium salts, carbon monoxide, cyanide, lead, nitrite **Lameness**: Fluoride (chronic), selenium (chronic), ergot

a) Different toxins produce same type of signs or toxins of same group produce different signs. BHC and aldrin (organochlorines) produce CNS excitation and depression respectively

b) One toxicant is neutralized or potentiated by another toxicant. For example, alcohol potentiates CNS depressant effect of barbiturates

c) Same toxicant producing different signs in different species. For example morphine produces CNS excitation in cats and depression in most animals

d) Some signs of poisoning like vomiting and seizures can also be produced by infectious diseases or metabolic and endocrine disorders

Important findings

Smell of GIT	Poison
Ammonical	Ammonia and Urea
Bitter Almond	Cyanide & Benzaldehyde
Fruity	Alcohols and Phosphorous
Garlic	Arsenic and Phosphorous
Rotten Egg	Hydrogen Sulphide
Shoe Polish	Nitrobenzene
Mouse	Hemlock,Metaldehyde,Acetaldehyde
Phenolic	Phenols,Phenolic compounds
RottenGarlic/HorseRadish Smell(Se)	Acetylene/Fish smell(ZnPhosphide)

Skin/MM color	Poison
Jaundiced (pale)	Phosphorous,Naphthalene,Arsine
Cyanotic (blue)	CO,Nitrite, Phenacetin
Red/Pink	Alcohol,Cyanide
Yellow	Acute Copper poisoning

Color/ GIT contents	Probable poison
Greenish Blue color	Copper
Black color	Sulphuric acid
Yellow or Orange Green color	Chromium salts
Yellow	Nitric/Picric Acid

GENERAL LINE OF TREATMENT OF POISONING

1.**Removing source** of poison ,preventing further exposure

2. **Delaying** further **absorption**

Emetics- Dogs,Cats & Pigs only

I) **Locally acting emetics**: Ipecacuanha (ipecac) syrup, hydrogen peroxide, hypertonic sodium chloride solution, copper sulphate and zinc sulphate solutions

II) **Centrally acting** like apomorphine hydrochloride and xylazine

Universal Antidote, 2 parts of activated charcoal (for adsorption), Kaolin, 1 part tannic acid/ strong tea (for precipitation), 1 part magnesium oxide/milk of magnesia (for catharsis).

3.**Hastening elimination** of absorbed poison- Laxatives, Diuretics

4. Providing **supportive therapy:**Temperature regulation, Cardiovascular / CNS / Respiratory

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Important Points:Tests&Breeds:

- **Heavy Metals-Reinsch test -Lead – Grunwald test**
- **Arsenic-Marsh test**
- **Cyanide – Copper Guaiac test, Sodium Picrate paper test**
- **Nitrite – Cooking test,diphenylamine blue/diazotization test.**
- **Libermann's test- for confirming Carbolic acid/Phenol**
- **Primates. Guineapigs, Bats, Birds - Can't synthesize Vit.C**
- **Methotrexate-Not toxic to G.pigs, Rabbits**
- **Belladona -not toxic to rabbits**
- **Red squill-not toxic to dogs**
- **Sheep-more susceptible to Cu poisoning**
- **Collies breed of Dogs-Ivermectin toxicity**
- **Greyhound Dog - More susceptible to barbiturate toxicity**
- **Bedlington Terrier –Genetic predisposition for copper accumulation**
- **Rat, very sensitive for ANTU is BROWN or NORWAY rat.**
- **Suffolk sheep –OPIDN**
- **Boxer Dog - Sensitive to acepromazine**
- **Hereford cattle -be less susceptible to Cyanide toxicosis**
- **Akita Dog, have a heritable predisposition to the hemolytic effects of onions**
- ***Congenital primary hyperoxaluria in Beefmasters.***
- **The PIVKA test assesses for proteins induced by vitamin K antagonists**
- **Tongue stress test and grain test are sensitive measures for botulism**


Bedlington Terrier


BROWN or NORWAY rat


Boxer

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Antidotes

Toxicant	Antidote/agent	Product/complex
Heparin	Protamine	Complex formation
Venoms	Antivenin	Complex formation
Digoxin or Digitalis	Digoxin immune Fab	Complex formation
Antimony	Dimercaprol, penicillamine	Chelation
Arsenic	Dimercaprol, penicillamine, BAL,Thioctic acid,DMPS,DMSA	Chelation
Atropine	Physostigmine	Receptor antagonism
Diazepam	Flumazenil	Receptor antagonism
Toxicant	Antidote or drug therapy	Product/MOA
Carbamates	Atropine	Receptor antagonism
Curare	Neostigmine	Antagonism
Cyanide	Sodium nitrite, sodium thiosulphate	Alternate pathway
Ethylene glycol,Methyl Alcohol	Ethanol or 4-Methylpyrazole (not cats)	Alternate pathway
5-fluorouracil	Thymidine	Alternate pathway
Iron	Desferrioxamine	Chelation
Lead	Dimercaprol, BAL, Calcium disodium EDTA , D Penicillamine *or* DMSA, Succimer, dogs	Chelation
Opioids (morphine)	Naloxone	Receptor antagonism
Organophosphates	Atropine (*or* glycopyrrolate) *and* (and carbamates) 2-PAM or Oximes	Receptor antagonism
Paracetamol/ Acetaminophen	N-acetyl cysteine or sodium sulfate	Alternate pathway
Warfarin/Coumarin anticoagulants	Vitamin K=phytonadione	Antagonism
Copper	D-penicillamine *or* zinc acetate, molybdenum salts	Chelation
Lead	Calcium disodium EDTA	Chelation
Molybdenum	Copper	Enhance renal excretion

Antidotes – contd…

Toxicant	Antidote/agent	Product/complex
Iron	Desferrioxamine, Sodium bicarbonate	Iron desferrioxamine complex, Ferrous carbonate
Silver nitrate	Sodium chloride	Silver chloride
Strychnine, nicotine	Potassium permanganate	Oxidized product
Fluoride	Calcium lactate, Calcium gluconate	Calcium fluoride
Methanol	Ethanol	Competitive inhibition
Fluoroacetate	Acetate	Competitive inhibition
Bromide	Chloride	Enhance renal excretion

Toxicant	Antidote/agent
Amitraz	Tolazine *or* yohimbine
Cadmium	Calcium disodium EDTA *or* D-Penicillamine
Cholecalciferol	Pamidronate disodium
Crimidine	Pyridoxine (vitamin B6)
Mercury (inorganic)	Calcium disodium EDTA *or* D-Penicillamine,*or BAL,* DMSA
Mercury (organic)	Dimercaptosuccinic acid? (experimental)
Pyriminil	Nicotinamide
Sodium fluoroacetate *or* sodium fluoroacetamide	Monoacetin (acetin, glyceryl monoacetate), acetic acid, *and* ethanol
Thallium	Diphenylthiocarbazone (dithizone)
Xylazine	Yohimbine
Zinc	Calcium disodium EDTA *or* D-Penicillamine

Toxicant	Antidote/agent
Nitrate, Nitrite, Chlorate	Methylene Blue
Opiates	Nalorphine
Alkaloids	Tannic acids
Barbiturates	Bemegride
Carbon Monoxide	O2, HBO

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Arsenic Poisoning – *"king of poisons" and "poison of kings"*

- The most common arsenic compound in general use is arsenic trioxide. **Inorganic**:more toxic > Organic
- ***Arsenites*** *(As3+ or trivalent) are 5-10 times more toxic than Arsenates (As5+ or pentavalent) due to higher solubility.*
- Most toxic gaseous form - storage **batteries** is **ARSINE** (AsH_3).
- Arsenic - ground water is endemic in **WEST BENGAL**.
- Arsenic poisoning in sheep is **DIPPING**. *[Eg. Sodium arsenite; Lead arsenate]*

- *high affinity for* ***sulphydryl*** *groups(****Lipoic*** *acid), So it accumulates in* ***hair*** *and* ***nails*** *(–SH rich keratin)*
- *Potent capillary toxin*
- *Acute –* ***Rice watery Diarrhoea***
- *Chronic –* ***Brick red MM's***
  - *Pigs – Demyelination,* ***Sitting Dog Posture***
  - *Blindness -* **arsinilic acid**
  - *Horses –* ***Tying up***

Rx:

- ✓ Gastric lavage, Emesis, Purgatives, Demulcents, $Fe(OH)_3$, Hypo?
- ✓ Specific antidote for arsenic poisoning is – **BAL-3mg**/kg or **DIMERCAPROL**.
  - Superior water soluble derivatives of BAL are MESO-DIMERCAPRO-SUCCINIC ACID (**MDSA=Unithiol**) and DI-MERCAPTO-SUCCINIC ACID (**DMSA=Succimer**)
- ✓ Thioctic acid -50mg/kg
- ✓ D-Penicillamine

- **PM Lesions** Intense **rose-red** inflammation of GIT.
  - Soft and **yellow** liver.
  - Edematous and **congested** lungs.
  - Haemorrhages in the heart, peritoneum, kidneys and liver.
  - Inflammation of proventriculus and gizzard in birds.
  - **Horny** layer of the **gizzard** may be **sloughing** off.

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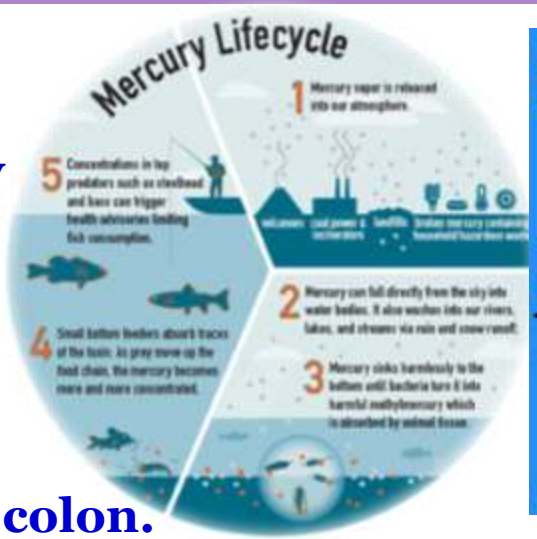
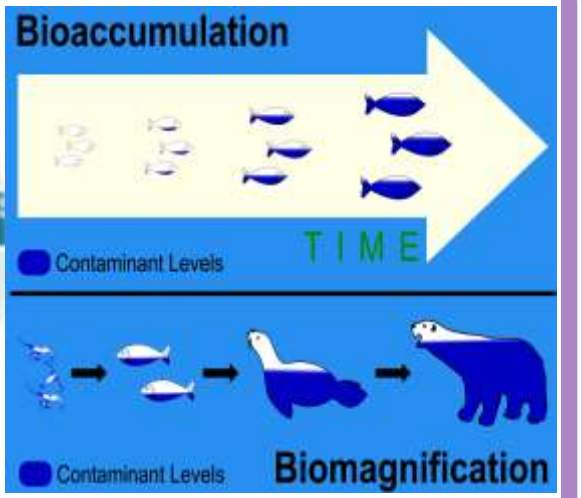
MERCURY POISONING



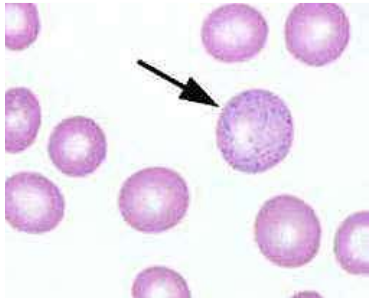


- Minamata Disease - city in Japan in 1956 - **methyl mercury**
- Ruminants- Cattle (Calves and Cows)- Highly sensitive
- Organic(highly soluble) > inorganic(Insoluble,monovalent)
- Elemental Hg by inhalation highly toxic
- Selenium and Vit E decreases toxicity of Hg
- Inorganic mercury - in erythrocytes and plasma.
- Soluble mercury salts (Hg +2, Mercuric) - **Stored in mm of colon.**
- Insoluble salts (Hg +, Mercurous) accumulate in the renal cortex and localize in the lysosomes.
- Mercury easily crosses the BBB. **Alkyl organic mercury -** accumulates in brain- **Most toxic form of Hg**
- Organic mercury poisoning –NEUROLOGICAL *(Ataxia,incoordination,convulsions,abnormal behaviour etc)*
- Inorganic mercury poisoning - GASTROENTERITIS.
- Elemental mercury poisoning - PULMONARY SYMPTOMS
- Humans-Mercurial Ptyalism
- Diagnosis - level of mercury in the cortex, brain and liver. (> 10-15 ppm)

- **Rx:** Administration of **egg white, activated charcoal, sodium thiosulphate** (to bind mercury), saline cathartic and oral d-penicillamine.
- **D-penicillamine** only if the gut is free of significant ingested mercury and only if the renal function is proper.
- **Dimercaprol: (BAL):** not with organic mercurials (contra indicated as it cause release of Hg from other tissues)
- Mesodimercaptosuccinic acid (DMSA) (Succimer) - less toxic than dimercaprol.

LEAD POISONING or PLUMBISM

- Lead acetate (sugar of lead) - white lotion. Petrol - tetraethyl lead
- Lead – Plumbers, Grass near highways, Batteries, Paints
- Soluble Salts (Lead Acetate) > Insoluble Salts(lead Oxide)
- indicator for lead in the environment is DOG, Pig - Resistant
- **Bones – Sink for Lead**
- Central neurotransmitters affected by lead - GABA and DOPAMINE.
- HAEMOPOETIC SYSTEM is extremely sensitive
- Lead decreases 'haeme' synthesis –inhibits- δ-AMINO LEVULINIC ACID SYNTHETASE (ALA-D synthase).
- **Basophilic Stipplings** (BS) – Dogs Cattle-Acute-Blindness,Tetany,Spasms (Neurological)
- Tubular cells of kidney is INTRANUCLEAR INCLUSION BODIES. (Eosinophilic)
- Dog-GIT,Horses-Resp'y–Roaring-RECURRENT LARYNGEAL nerve paralysis.
- X-ray reveals Opaque lead lines in metaphyses of bone
- Diagnosis-estimation of ALA-D SYNTHASE enzyme in blood
- Rx:: Antidote - Di sodium Calcium Edentate
- Thiamine, BAL, DMPS, DMSA, d-Penicillamine, Vit.D, CBG, Sedatives, Oral MgSo4, Barbiturates & Diazepam (Seizures), Dexa & Mannitol(Cerebral Edema), Broad Antibiotics



COPPER POISONING

- Sheep are affected most , Poultry are more resistant
- Hepatotoxic substances- Precipitate Cu toxicity
- High levels of Zinc and Ascorbic acid- decreases Cu absorption and its toxicity
- Deficiency of Manganese, Iron, Vit-B- Cu accumulate in body
- Cu Toxicity-**Molybdenum deficiency** in sheep - Cu to Mo ratio (ideal-6:1 if it is 10:1 or more-toxicity)
- Progressive hepatocyte damage - elevated SGOT, lactic dehydrogenases, plasma arginase and plasma bilirubin.
- Liver damage- large Cu into circulation-Lysis of erythrocytes-Haemolytic crisis. (absent in non-ruminants).
- Kidneys fail because the renal tubules get clogged with haemoglobin and necrosis of tubules and glomeruli
- Faeces of the affected animals contain mucous and are of deep green colour
- **Port wine coloured** urine, Kidney-bluish black- **Gun-metal Kidney-** due to the haemolytic crisis
- **Diagnosis-Copper** in body fluids, tissues 8000-10000 ppm in faeces;
- Acute poisoning- >15 ppm wet wt. in Kidney, Chronic poisoning - 5-20 µg/ml in blood and >150 ppm in liver-
- Blue-green ingesta Elevated levels for liver enzymes like SGOT etc.
- **Rx**:: Gastrointestinal sedatives to reduce irritation.
- Administration of d-Penicillamine and Calcium versenate are useful.

- **D-penicillamine**- sheep- 50 mg/Kg –orally; alone or in combination with BAL; Dogs: 10-15mg/kg BID
- Ammonium tetrathiomolybdate (1.7-3.4 mg/day three treatments on alternate days by IV or SC).
- Daily administration of ammonium molybdate and sodium thiosulphate are useful. Oral Zinc supplements-decrease intestinal absorption of Cu (5-10 Mg/ Kg). Supportive therapy:
- Anti oxidant therapy with vit E To treat shock, colic pains. Alkalization of urine Supplementation of sheep rations with 2-16 ppm Mo

MOLYBDENUM POISONING

- Chronic molybdenum poisoning is known as **Tearts Or Teart Disease (Peat Scours)**
- Cattle are the most susceptible (Cattle > sheep. Young animals > old). Non-ruminants are less susceptible
- Demyelination –**Enzootic Ataxia** (Austr), **Sway back**(UK) in lambs,
- Decreased activity of tyrosinase-diminished melanin depigmentation- **Spectacle eye**, **achromotrichia**,
- Progressive atrophy of myocardium- Sudden death (**Falling /Pacing disease** in cattle)
- **Rx::CuSo4(**1-2 g/adult cow/day; 0.25 g/45 kg body wt in sheep/day.
- **Copper glycinate-** SC- 60mg for calves and 120 mg for cattle.

FLOURIDE POISONING / FLUOROSIS

- Dogs- Acute type of poisoning. Herbivores- Chronic type
- >2-4 ppm dietary intake- may be toxic. The MPL 40-50 ppm-for cattle,200 ppm-chicken.
- Bone is natural sink for fluoride like lead (96-99% absorbed F-hydroxyapatite of bone)
- F- interferes with cations like Ca++, Mn++ and Mg++
- malformed enamel and dentin- brown or black mottling & discolouration of teeth (**Dental fluorosis)**
- Bones:Skeletal fluorosis-**Osteoporosis**,**exostosis**,chalky white,aplastic anemia,perosis,Sclerosis
- Periodic and intermittent; moving type lameness
- **Hypocalcemia**, **Hyperkalemia.** PINEAL GLAND- accumulates highest fluoride
- Cattle-3000 ppm or more(400-1200 ppm normal). Sheep-5000ppm(250 ppm normal).Urine 15-20 ppm(2-6 ppm normal)

Rx:: Non specific– ACUTE: calcium gluconate, Al salts, milk, Quinidine- to prevent hyperkalaemia, Dialysis
CHRONIC: Al2(So4)3,Ca2Co3,Defluorinated Po4, Feed correction

Phosphorus Toxicosis

- Deficiency - **Osteomalacia And Rickets**
- **Yellow/White** phosphorus-Toxic whereas **Red** Phosphorus-Non-toxic.
- Feeding of excess of **wheat bran** -very rich phosphorus and low in calcium causes **Bran Disease** in horses.
- **Meat** high in phosphorus and low in calcium causes **Hyperphosphataemia** in dogs.
- Phosphorus -eliminated by lungs-exhaled air a smell of **garlic-like** and a **glow** in dark.
- Similarly, the vomitus of GI tract contents may be **luminous** and have the same odour.
- Acute **protoplasmic poison**, direct **cardiotoxic** effect –cardiovascular collapse.
- Hepatic necrosis. On dermal exposure, white phosphorus results in painful **chemical burn injuries**
- Acute toxicity-abdominal pain, colic, profuse vomiting (occasionally haematemesis), severe diarrhoea (often haemorrhagic), and a **garlic-like odour** from the breath.
- **Jaundice** -profound depression, shock, coma and death. Massive hepatic failure is followed by convulsions and death. progressive **oliguria** and **anuria**..
- **Chronic toxicity : "Phossy jaw"**
- **Rx:: Non Specific**-careful gastric lavage with potassium permanganate or 0.2% copper sulphate, Non absorbable oil (e.g. mineral oil) -. Activated charcoal and sorbitol or saline cathartic may be useful.
- A diet high in cysteine and carbohydrates but low in fat -to combat liver damage.
- High doses of vitamin B and ascorbic acid.
- Hypoprothrombinaemia and haemorrhage- vitamin K1 : Dehydration, loss of electrolytes - fluid therapy
- Prognosis -guarded to grave

NITRITE/NITRATE POISONING

- Nitrates **are** non-toxic .
- **Ruminants**(Cattle)>monogastric animals. Pigs(Young>old) are susceptible to preformed nitrites
- In ruminants, **nitrate** is converted into **nitrite** and then into **ammonia**(**Reductase** enzyme).
- In RBCs, nitrite converts haemoglobin into **methaemoglobin** (Met-Hb), itself into nitrate.
- Draught, Frost, Hail damage, adverse weather-more plant accumulation
- One mole nitrite + two moles of haemoglobin (loss of an electron) and ferrous form (Fe2+) of iron in Hb is converted to ferric form (Fe3+) → resulting in methaemoglobin formation.
- this met-Hb –ferric form is converted back to ferrous form by diaphorase-I and Diaphorase-II.
- But during toxicity, formation of met-Hb exceeds the turnover of the enzymes.
- When **20**% of Hb is converted to Met Hb, **toxic symptoms** are noticed.
- When **80**% of Hb is converted, **anoxia** and clinical signs are noticed and **death** occurs.
- Acute-cyanosis (**5-15%MetHb**),Anoxia(**20-50% met-Hb**);violent struggling (**50-60%** met-Hb)
- **Chronic:**.Anorexia, dyspnoea, loss of condition, poor wt. gain, abortion, still birth, goitre/thyroid dysfunction (sheep & pigs):Nitrate: large quantities-caustic effect-diarrhoea, salivation, abd. pain.
- **PM Lesions** Acute- haemorrhagic gastroenteritis, **Dark brown or coffee/chocolate coloured blood** which clots improperly (due to Met-Hb), **Brown** staining of tissues, Congestion of the intra-abdominal organs, Peticheal haemorrhages on the serous surface, Dilatation of the blood vessels, Generalised cyanosis and Blood stained pericardial fluid are common
- **Methylene blue** intravenously at the rate of 4-8 mg/kg in cattle and sheep (can be given up to 20 mg/kg) . Dogs; 5-10 mg/Kg Slow IV Methylene blue is an oxidising agent which is reduced to leucomethylene blue by the action of **NADPH2 - reductase**. This leucomethylene blue converts methaemoglobin to haemoglobin. Ascorbic acid is found to be useul.

Crop plants	Weeds
Alfalfa (Medicago sativa) Barley (Hordeum vulgare) Blackberry (Rubus fruticosus) Fescue (Festuca spp.) Linseed (Linum usitatissimum) Maize/Corn (Zea mays)* Oats (Avena sativa) Rape (Brassica napus)* Rye (Secale cereale) Sorghum (Sorghum spp.)* Soybeans (Glycine max) Sudan Grass (Sorghum sudanense)* Sugar beat (Beta vulgaris)* Sweet Clover (Melilotus officinalis) Turnip tops (Brassica rapa) Wheat (Tnticum spp.)	Canada Thistle (Cirsium arvense) Cheeseweed (Malva parviflora) Dock (Rumex crispus) Fat hen/Lambsquarters (Chenopodium album) Fireweed (Kochia)* Jimson weed (Datura) Johnson Grass (Sorghum helepense)* Marshmallow (Althaea officinalis) Mintweed (Salvia reflexa) Nightshade (Solanum spp.) Red root or Pigweed (Amaranthus retroflexus)* Russian thistle/Tumbleweed (Salsola) Smartweed (Polygonum hydropiperoides) Variegated thistle (Silybum marianum)* White ragweed (Franseria discoloui)* Wild sunflower (Helianthus)

* = Plants of high risk

Nitrate (ppm)		Recommendations for use in livestock
Forage	Water	
0- 6,500	0-400	Generally considered safe
6,500 - 20,000	400 - 1,300	Caution: Possible toxicity
>20,000	>1,300	Danger: Potential for toxicity high

Toxicant	Mechanism of action	Blood colour	Treatment
Nitrate/Nitrite	Methaemoglobin formation	Brown	Methylene blue; ascorbic acid
Sodium chlorate	Methaemoglobin formation	Brown	Methylene blue; ascorbic acid
paracetamol	Methaemoglobin (formation	Brown	N-Acetylcysteine
Naphthalene	Methaemoglobin formation	Brown	Methylene blue; ascorbic acid
Silo gases (Nitrogen dioxide, nitric oxide)	Lung irritation; slight methaemoglobin formation	Slight brown	Methylene blue
Cyanide	Cytochrome oxidases inhibition	Cherry red	Sodium nitrite and sodium thiosulphate
Carbon monoxide	Carboxyhaemoglobin formation	Bright red	Oxygen + 5% Carbon dioxide
Methylene chloride	Carboxyhaemoglobin formation	Bright red	Oxygen + 5% Carbon dioxide
Carbon dioxide	Oxygen displacement	Dark	Oxygen

HYDROGEN CYANIDE/CYANIDE POISONING

- **Hydrocyanic** acid or **prussic** acid- **most toxic**.
- **Ferrocyanide** and **thiocyanates**-practically harmless.
- Young and immature plants, plants growing rapidly after drought, wilted and frost bitten plants are more toxic.
- Hydrogen cyanide is released from plant during maceration as in chewing from cyanogenic glycosides by **β-GLYCOSIDASE** present in mesenchymal cells by Hydrolysis. Toxicity if >200ppm
- In liver, cyanide ions to thiocyanate, through **Rhodanese enzyme**
- Toxicity bcz of binding with **Cytochrome Oxidase**,inhibits, the ferric (Fe3+) heme moeity form of mitochondrial cytochrome c oxidase – **Histotoxic anoxia**
- Dyspnoea, Tachycardia, **Bitter Almond breath**, **Bright red MM(pink to Cyanotic), Bright Cherry Red Venous blood**, Severe asphyxia convulsions, Resp'y Paralysis.Chronic form of cyanide toxicity due to cassava root is called as KONZO.
- Chronic poisoning- Goiter, Myelomalacia (cystitis-ataxia syndrome),urinary incontinence, incoordination of hind limbs, Arthrogryposis
- **Positive Picrate paper test, Dark skeletal muscles**
- Rx:: high-affinity **decoy chemical receptor approach-Sodium nitrite,Sodium thiosulfate,Hydroxocobalamin (vitamin B12),** Cobaltous chloride- oral-5% solution- 10mg/ kg ,**Sulfanegen** (as the sodium or triethanolamine salt- prodrug that generates 3-mercaptopyruvic acid (3-MP)) -mass poisoning incidents.
- 4-dimethyl-aminophenol (DMAP; IM at 5 mg/kg) or hydroxylamine hydrochlorine (IM at 50 mg/kg) (**Alternative inducers of methemoglobinemia)**

Plants	Glycoside
Bitter almond,Wild cherry	Amygdaline
Sorghum,Millet,Jowar,Sudan grass	Dhurrin
Linseed , Velvet grass and Wild clover, Other plants are Acacia, Arrow grass. Maize , Peach, etc	Linamarin





Cyanide (HCN) Content of Forages		
As-fed Basis	Cyanide Content (DM Basis)	Comments
<200 ppm	<600 ppm	No cyanide poisoning.
200 to 600	600 to 1800	Potentially toxic
>600 ppm	>1800 ppm	Potentially very toxic. Drying, ensiling or allowing it to mature should reduce the cyanide content

SELENIUM POISONING/DOG MURRAIN(Ireland)

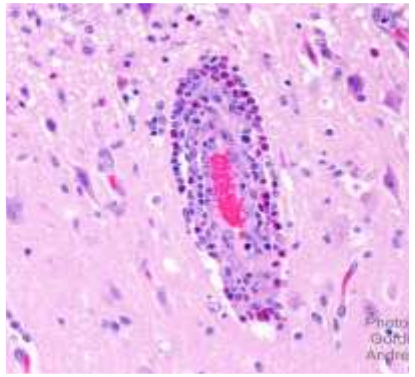
- Toxicity ranking:**Natural organoselenium>selenite(+4)=selenate(6+)>selenide(-2)> elemental Se.**
- Vit E deficiency- increases toxicity. Young>Old animals
- High protein diet reduces toxicity.
- MOA: Glutathione depletion and lipid peroxidation.
- Selenium replaces sulphur in amino acids (cysteine and methionine).
- Cytotoxic-pro-oxidant catalytic activity of selenide anions
  - produce superoxide anions-ROS- oxidative damage

Obligate accumulators/ indicator plants	Facultative accumulator	Non accumulators
Astralagus, Stanleya, Oonopsis,Xylorrhiza	Aster, Astriplex and Acacia	Corn, wheat, Barley, Grass and Hay
Requires selenium for growth	Not for growth but if present in soil, they accumulate it	For formation of seleno-cysteine and selenomethionine
100-15,000 ppm	25-100 ppm	1-25 ppm
Unpalatable with garlic odour and not readily consumed by plants	Some are palatable and are readily eaten by animals	Palatable plants



- Acute- colic,bloat,**dark watery diarrhoea**,polyuria, fever, mydriasis,
- Peculiar **rooted-to-one-spot stance** with head and ears lowered
- **Subacute (Blind staggers) → Chronic (Alkali disease)**
- Lameness-**Erosion** of the **Articulate Surface Of Long Bones**.
- Shedding is incomplete and old hoof fuses with new hoof and form abnormally **long rocker shaped hoof**.
- Diagnosis:Blood-1-4 Ppm(up To 25 Ppm In Acute); Liver&Kidney-4-25ppm;Urine-0.1-8ppm;Milk-1-3 Ppm;Hoof-5-20 Ppm;Hair-5-10ppm
- **Rx:** Removal of source, saline purgatives and high protein diet are said to be useful.
- **Acetylcysteine** a substitute for glutathione may be effective (loading dose-140mg/kg b.wt, then 70mg/kg daily in 4 divided doses)
  - In chronic selenium toxicity, addition of **copper** to the diet is useful to prevent selenosis. Addition of inorganic arsenicals enhances biliary excretion of selenium and increasing the dietary levels of sulphur containing proteins is also beneficial.
- **Contra indications**:
  - Dimercaprol (BAL): inhibits recovery from Se poisoning Vit E: has got synergisticaction with Se

SALT POISONING/ Hypernatremia



- also known as **water deprivation syndrome, or water deprivation–sodium ion intoxication**
- **Poultry**(Young chicks) and **Pigs** (most sensitive species) are more susceptible.
- **The acute oral lethal dose of salt in swine, horses, and cattle is ~2.2 g/kg; in dogs, it is ~4 g/kg.**
- **Sheep appear to be the most resistant species, with an acute oral lethal dose of 6 g/kg.**
- Anorexia, excessive **thirst**, salivation,initially diarrhoea followed by constipation
- **Stumbling**, walking **backwards** or in circles, **Peddling** of limbs, recurrent convulsive **seizures**, **dragging** of hindfeet while walking, Recumbency, coma and death.
- **Eosinophilic meningoencephalitis** is noticed in **swine- eosinopenia**, **eosinophilic cuffs around vessels in the cerebral cortex** and adjacent meninges, and cerebral edema or necrosis.
- Cattle **do not** develop eosinophilic cuffs but can have edema of the skeletal muscles as well as hydropericardium. **Chickens** can also have **hydropericardium**.
- **Rx**: Immediate removal of offending feed or water. Cold water-Acetic acid treatment can be given.
- 19-38 litres of **water** and 3.8litres of 5% **Acetic Acid** can be administered to an adult cow.
- By diluting the ruminal contents and by decreasing the ruminal pH and temperature, **hydrolysis of urea** can be slowed.
- Water intake should be limited to **0.5% of body weight** at hourly intervals until normal hydration is accomplished, usually taking several days.
- Slightly hypertonic IV fluids has been recommended to reduce the likelihood of cerebral edema. A solution - 170 mEq/L sodium - initially, -concentration decreased as clinical signs improve.
- Monitoring serum sodium concentration-levels should be lowered at a rate of 0.5–1 mEq/L/hr. If brain edema is suspected, **mannitol**, **dexamethasone**, or **DMSO** may be helpful.
- In dogs, a series of warm water enemas -intervals of several hours -reduce acutely increased sodium levels

PHYTOTOXINS - INTRODUCTION

Toxic Principles

Alkaloids

1. Tropane or atropine like - Atropa belladonna, Datura ,Hyoscyamus, Erythroxylon
2. Pyrrolizidine alkaloids - Crotolaria, Heliotropium
3. Pyridine/piperidine alkaloids - Conium, Lobelia
4. Pyrrolidine alkaloids - Nicotiana tabacum
5. Purine alkaloids - Coffea, Theobroma
6. Quinoline alkaloids - Cinchona
7. Isoquinoline alkaloids -Papver somniferum
8. Indole alkaloids - Claviceps, Strychnos(Strychnine&Brucine
9. Quinolizidine alkaloids -Laburnum
10. Steroidal glycoalkaloids - Solanum
11. Steroidal alkaloids -Veratrum
12. Diterpenoid alkaloids - Aconitum
13. Phenylamine alkaloids - Ephedra

Terpenes

- Monoterpenes - Cantharidine, Anamirta cocculus
- Sesquiterpenes - Helenium microcephalum
- Diterpenes - Aconitine from Aconitum sp
- Triterpenes - Lantadenes from Lantana sp

- **Glycosides**

1. Cyanogenetic glycosides - Sorghum vulgare ,
2. Gossypol - cotton seed
3. Steroidal glycosides - Digitalis purpurea
4. Coumarin glycosides - Ipomoea sp
5. Anthraquinone glycosides - Cassia fistula
6. Mustard oil glycosides - Brassica nigra

Proteinacious compounds

- Toxalbumins/proteins - Abrin from Abrus precatorius Ricin from Ricinus communis
- Peptides - Amatoxins from Amanita sp.
- Amines - Mimosine from Mimosa pudica

Organic acids

- Oxalic acid - Beta vulgaris

Resins and resinoids

- Cannabin from Cannabis sativa

OXALATE POISONING

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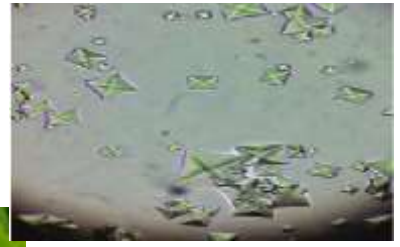
- The oxalates rich plants **are *Amaranthus, Astriplex, Beta vulgaris, Oxalis spp, Rumex spp and Halogeton glomeratus***.
- Large amount of oxalic acid are formed on damp hay/straw or silage infested with with ***Aspergillus niger***.
- **Calcium metabolism** is upset after chelation with oxalates and leads to **hypocalcemia**,affects electrical activity of the heart, muscles and nerves, **blood clotting mechanism** and causes enhanced **bleeding**,**milk** production & **bone growth** in lactating and pregnant animals.
- Kidneys- **Oxalate Nephrosis**. Oxalates get crystallize in **brain** tissues,**Rumen epithelium-haemorrhages**& in blood vessels
- Acute-Dullness, anorexia, lowered head, salivation, rapid shallow breathing, cardiac failure and oliguria.
- In sub acute-Stiff gait, frequent attempts to **urine** (**red brown in colour**) and recumbency.
- Chronic - renal damage→**uraemia**. Oxalate Calculi - **Cattle(SIGMOID FLEXURE), *Rams(URETHRAL PROCESS)***
- After death on post mortem examination, haemorrhages are found in visceral organs and lungs are filled with **dark red blood**.

Rx: Shift to oxalate free pastures

- ✓ **Calcium borogluconate** as 25% solution (300-500ml)
- ✓ Prior to feeding of oxalate rich plants provide **dicalcium phosphate** (25%) to animals as preventive feed.
- ✓ **Lime water** orally

Prognosis-Poor



POISONOUS PLANTS

- **Datura :** Genera of the *Solanaceae* (**Nightshade**) -*Datura stramonium* (**jimson weed/thornapple**), *Atropa belladonna* (**deadly night shade**), *Hyoscyamus niger* (**black henbane**), Solan*um nigrum* (**black nightshade**), *Physalis virginiana* (**ground cherry**).
- Contain **alkaloids or glyco-alkaloids**, -pharmacological and toxicological actions.
- Source of **malicious and accidental** poisoning.
- *Datura* exists in more than dozen varieties (e.g. *D. stramonium/metal/tatula/fastuosa/meteloides/quercifolia/alba/ niger).*
- **Toxic principles: tropane alkaloids** like **Hyoscine**(scopolamine), **Atropine** (dl-hyoscyamine),**Hyoscyamine**, **Daturine** (mixture of **atropine** and **hyoscyamine**) and **Solanine** (glycoalkaloid).
- Nitrites, fatty acid glycerides, etc. are also present.
- Seeds-drug of abuse in humans-hallucinogenic effects: **Pigs -very sensitive** to datura seeds-2.2-5.7 mg/kg/day =toxic signs.
- Classic **Anticholinergic poisoning**, inhibit all **muscarinic** receptors in the body(neurotransmitter acetylcholine)
- High doses of atropine may also block **nicotinic** receptors at the autonomic ganglia and neuromuscular junctions.
- **Dryness of mouth and throat**, dysphagia and difficulty in swallowing. followed by staggering gait, incoordination, dry-hot skin - rise in body temperature, photophobia, dilated pupils, cycloplegia, urine retention, tachypnoea and tachycardia. Bloat and rumen atony in cattle and colic in horses. Behavioural changes (in man).
- Very high doses - coma, skeletal muscle paralysis and death from respiratory failure.
- **Diagnosis:** Placing a **drop of urine from poisoned animal in the eye of a cat -mydriasis**.
- **Treatment:** No specific antidote
- ***Physostigmine:*** Parasympathomimetic drug- to stimulate cholinergic secretions and activities.
- Cattle: 0.02 mg/kg, slow IV over 5 minutes. Dose may be repeated, if required.
- Dogs: 0.05 mg/kg, slow IV over 5 minutes.
- Horses: 0.1 -0.6 mg/kg, IM or slow IV over 5 minutes.
- **Diazepam-** If marked excitement is present. Cold water and ice-packs to reduce fever, artificial respiration.
- ***Contraindications:*** Phenothiazine tranquilizers should not be used for sedation and controlling CNS excitation- they possess anticholinergic action.

LECTIN CONTAINING PLANTS

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- *Ricinus communis* (castor bean- **Ricin**), *Abrus precatorius* (rosary pea, jequirity bean- **Abrin**)
- Lectins -concentrated in seeds -when seeds are crushed and fed -toxicity.
- Castor beans and rosary peas -used for **malicious poisoning and criminal activities**.
- Castor beans contain a toxic alkaloid **ricinine**, an irritant and purgative **ricinoleic acid**, and a powerful **haemagglutinin RCA *(Ricinus communis* agglutinin**).

- Humans/horses> goats,/cattle/dogs. Horse-**tumultuous heart**
- Castor beans at 0.01% body weight cause toxicosis in horses and at 0.02% -cattle.
- In humans poisoning –as little as 1mg ricin or 1-3 beans. Abrin is much more toxic than ricin.
- One seed of rosary peas, approximately 0.5 cm in diameter, can kill a small child or medium sized dog. (**Abrin(**Rathi)**-Sui/Needle poisoning)**

- **Clinical signs :** incoordination, dyspnoea, hyperpyrexia, hemolytic anaemia, convulsions, opisthotonos, paralysis and coma followed by death. Renal failure oliguria and sometimes uraemia may be seen in acute cases.

- Death in 1-10 days-hypovolaemic shock/paralysis of respiratory centre.
- Non-fatal cases =secondary infections.
- In horses, -trembling, sweating, cold extremities, weak pulse and shivering.
- In dairy cattle, lactation stops abruptly. Hallucinations –in human beings.

- Depression, roughened feathers, droopy **wings**, **greyish wattles** and combs and decreased egg production -in poultry.
- **Rx:N**o specific Rx. Treatment is directed at removing the seeds from the digestive tract and providing the supportive therapy. Emesis and gastric lavage -followed by administration of activated charcoal. Demulcents,Appropriate fluid and electrolyte therapy.

- Anti-ricin serum - may be used in castor bean poisoning, if readily available.

Poisonous plants- Contd…

- **Nuxvomica(*Strychnos nuxvomica)*-alkaloids (Strychnine and brucine),glycoside(loganin),(caffeotannic acid.)**
  - **Saw horse stance, Bait shyness.**

- **Oleander and Related Cardiotoxic Poisoning**
  - *Nerium odorum (white* or sweet scented oleander) and *Thevetia neriifolia* (yellow oleander) **cardio- active glycosides - nerioside, oleandroside, oleandrin, nerin, folinerin and digitoxigenin**
  - *Nerium odorum* contains -**nerin** ;*Cerbera thevetia or Thevetia neriifolia* –**thevitin and cerberin**. *Nerium Indicum* contains **odoroside**, a steroidal glycoside - inhibit the **Na +/K+-ATPase system (potassium pump)** –
  - Agonal haemorrhages-on heart and serous and mucous membranes
  - *Digitalis purpurea* (foxglove plant), *Convallaria majalis* (lily of the valley), *Apocynum cannabinum* (dogbane), *Asclepias* spp. (milkweed), *Taxes Sp*. (yew) and *Urginea* spp.(squill).

- Ipomea *turpethum* (Indian Jalap or white turpeth), I. *orizabensis/violacea/carnea,/purga* (Jalap)= morning glory
  - Cathartic resin=**Scammonin** (jalapin), in *I. turpethum* (Indian jalap) is **turpethin** and in *I.hederaceae* is **pharbitisin**.
  - *I.violacea* ***seeds*-lysergic acid**, I.carnea **leaves-Saponins**
  - **Bizzarre** behaviour,barking & disorientation in dogs.

- **Brackern fern poisoning** – Pteridium aquilinum(B.Fern)/Equisetum arvense(Horse tail)-**Thiaminase- Thiamine deficiency-Aplastic anemia(Ptaquiloside)-Enzootic Heamturia,Quercitin(Procarcinogenic)**
  - **Sheep-Bright blindness(Progressive retinal atrophy=PRA),Horse-Brackern staggers,Cattle-Hyphema**
  - Haemangioma,haemangiosarcomas, Papillomas, Fibromas, Adenomasand Transitional Cell Carcinomas may occur
  - **Rx:Thiamine,DL-Batyl alcohol(bonemarrow stimulant)**
  - **Blood/Platelet transfusion**

LANTANA and other plants-PHOTOSENSITIZATION

Plants causing liver damage	Plants causing biliary occlusion	Plants causing both liver damage and biliary occlusion
Contain Pyrrolizidine alkaloids, Senecio spp,Crotolaria spp, Heliotropium spp.The active pyrroles formed from pyrrolizidine alkylate with DNA and impair cell division. Hence, there is hepatomegaly and karyomegaly and eventually cell dies (necrosis) **Pigs** are most susceptible fld by poultry, cattle, horses, goats, sheep	Saponins from some plants have been suggested to cause inflammation and obstruction of the biliary tract Agave poisoning cause photosensitization by occlusion	Lantana camara, an ornamental plant causes photosensitization due to presence of two polycyclic triterpenes Lantadene A and Lantadene B, which cause damage to liver and bile ducts. Among various varieties of Lantana camara, the red flowered variety is considerably toxic. Animals eat this plant in draught conditions. **Sheep** is most susceptible

Primary photosensitization	Secondary photosensitization	Inherited or congenital photosensitization
directly ingested or ingested material is converted to active photodynamic metabolite which reach skin and interact with UV light to produce PS. Examples are Hypericum perforatum (Saint John's wart), Fagopyrum esculentum (buckweed). They produce hypericin and fagopyrin as photodynamic agents respectively. Other plants are Ammi majus (bishops weed), clovers and alfalpha. Drugs and chemicals like phenothiazines, sulphonamides, tetracyclines and acridine dyes	most frequent type of photosensitization in livestock and is caused by phylloerythrin, a bacterial break down product of chlorophyll. Normally, it is removed by the liver and is excreted in bile but if the liver is severely damaged or if the bile duct is occluded, the phylloerythrin accumulates in the peripheral blood to cause photosensitization. Pyrrolizidine alkaloids containing plants, green algae (Microcystis), horse brush and some mould toxins (Sporidesmin) produce secondary photosensitization	It may be due to congenital porphyria (abnormal synthesis of porphyrins). It occurs due to inhibition of heme synthesis due to blockade of certain enzymes uroporphyrinogen decarboxylase, coproporphyrinogen oxidase and ferrochelatase which results in accumulation of porphyrinogens proved to be photosensitive.

POISONOUS ANIMALS

Venom and poison (toxin)?

- **Venom is produced by a specialized gland,either by biting or stinging.**
- **Poison (toxin) is in the tissues ,enters through food. Eg:Puffer tetrado toxin**

Snake Poisoning

- Snake venom-aminoacids, polypeptides, glycopeptides and biogenic amines,with enzymatic&non-enzymatic portion.
- **two types::neuro and cardio or haemotoxicity.**
- Venoms=necrotising, anticogaulant, coagulant, neurotoxic, cardiotoxic and haemolytic fractions.
- Cobra and krait venom is **neurotoxic** while viper and rattle venom is **haemotoxic**.
- Venomous snakes belong to several families of snakes such as Elapidae, Crotalidae, Viperidae, Hydrophiidae and Colubridae
- But mainly classified into 2 classes: 1) the elapines, which include the cobra, mamba, and coral snakes; and 2) the two families of viperines, (a) the true vipers (e.g., Russell's viper) and (b) the pit vipers (e.g., rattlesnakes).
- There are 4 common poisonous snakes in India. They are **1. Indian cobra (Nag) 2. Indian krait *(Bangam)* 3. Russel's viper *(Daboia)* and *4*. Saw-scaled viper *(Phoorsa).***
- **Pre-synaptic Toxin(B-bungarotoxin),Post-synaptic Toxins(A/K-bungarotoxins).**
- ***Viperines*** bites-severe **local tissue** damage,pain,dark blood oozes
- ***Elapines*** bites-pain/swelling are less but systemic **neurological** signs predominate
- Rx**: Polyvalent antivenom**
  - Total dose- 100 ml – 5-10 ml at the site of bite and remaining IV. If required adrenaline 1:1000 (0.5 to 1 ml) is administered to counter hypersensitivity & Broad-spectrum antibiotics to stop infections. Resp'y Assistance,Diuresis,Fluid therapy,TT.
  - **No Antihistamines,** No Alcohol to clean wound-vasodilation– spread of venom?

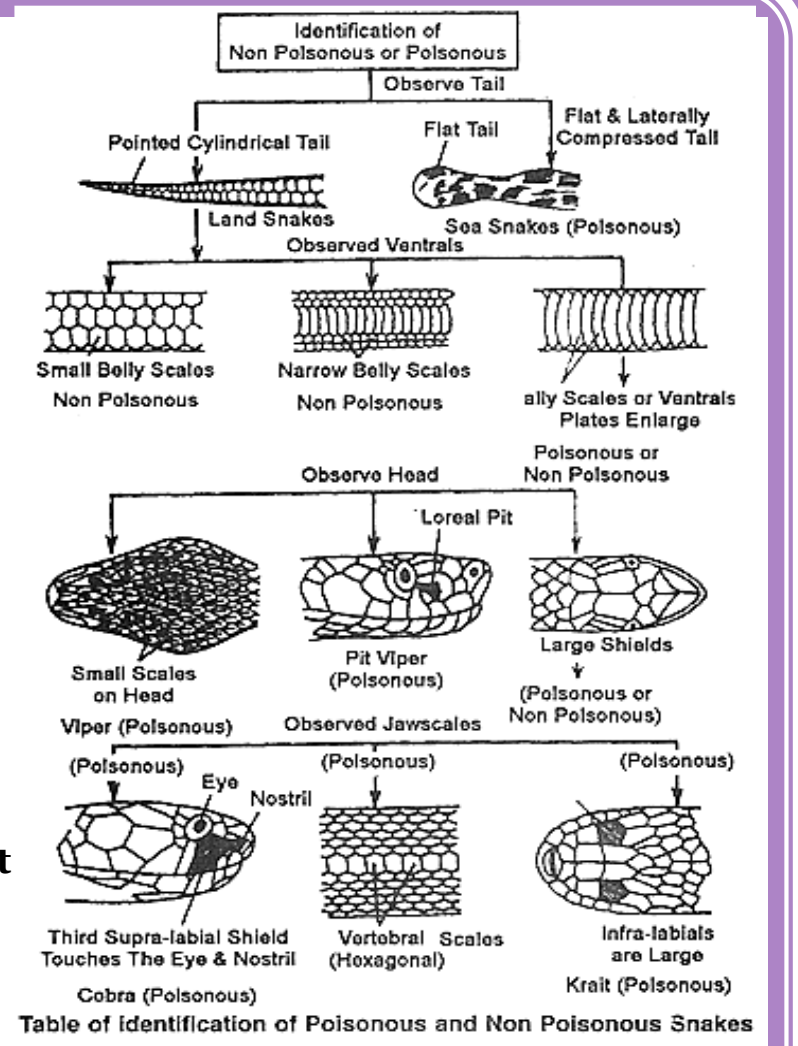

Table of Identification of Poisonous and Non Poisonous Snakes

Other POISONOUS ANIMALS-Contdd

- Order HYMENOPTERA*(Apidae [Honey bees]; Vespidae[Wasps] and Formicidae [Ants].)*
- **TETRADOTOXIN** (TTX)-*(10X >snake venom; 100X>black widow spider; 10000X>cyanide)*
  - From TETRAODON(**Puffer**) /**FUGU** fish (in **Liver & Ovary**),by bacteria ***Pseudoalteromonas Tetradonis***
  - -potent neurotoxin=blocks Na+ channel in CNS & PNS.
- Ciguatera (CIGUATOXIN (CTX) from the consumption of REEF fish. *(Reef fish such as barracuda, eel etc).*
  - **Most heat resistant toxin =By Dinoflagellate GAMBIERDISCUS TOXICUS**
- **Tuna & Mackerel fish-** Scrombroid poisoning,by HISTAMINE,*(Others puterscien and cadaverine)*

Scorpions Venom– neurotoxins- main-cardio, nephro, haemolysins, agglutinins, coagulins,, histamine, phospholipases, phosphodiesterases , hyaluronidase,serotonin etc.

- Two general classes, Alpha(prolongs action potential)&Beta-receptor toxins.
- The most dangerous species of scorpion is *LEIURUS QUINQUESTRIATUS*. *(The venom contains potent neurotoxins)*

Spider Poisoning : Toxic principle is proteins=protease, hyaluronidase, sphingomyelinase D and esterase.

- They have direct lytic effect on RBCs.
- Most venomous spiders in the world- **Brown recluse spider, Hobo spider and Black widow spider.**
- **Some spider venoms can kill a mouse at a dose as low as 0.006 mg.**
- The most potent neurotoxin=venom-**black widow spider(*Lactrodectus mactans*)** is **α-LACROTOXIN**.

Honeybees is **barbed** sting,**Wasps-Plain** stinger.

- The antigen in honey bee venom=allergies or anaphylaxis is **MELLITIN**.
- Cytotoxin in Ant venom is **FORMIC ACID**. The piperidine alkaloid-fire ant venom is **SOLENOPSIN**
- **Ascending Flaccid Tick Paralysis-dogs**=*Rhipicephalus*,*Dermacenter*=blocks Na+ channels.
- Ixodid ticks=blocks AcTH receptors.
- **Toads=Bufotoxin=Parotid glands/Skin=*Bufo marinus*=Bufodienolides(Cardiac Glycosides)-Rx:Propranolol(Dogs)**

ORGANOPHOSPHORUS INSECTICIDES (ORGANOPHOSPHATES)

- **organic esters of Phosphoric or Phosphonic acid**
- **Insecticides,Acaricides, Rodentcides, Nematicides And Herbicides**
- **Most dangerous organophosphorus compounds are nerve agents (Sarin, Soman, tabun)**

➢ **Dimethoate is more toxic in cattle than sheep.**
➢ **Cats are sensitive to acute toxicity of OP insectides.**
➢ **Suffolk sheep (England) pre-disposition to OPIDN**
➢ **Age: Young animals are more prone to directly acting organophosphorus insecticides and adults are susceptible to indirectly acting organophosphates.**
➢ **Delayed neuropathy occurs more in adults than younones.**
➢ **Among animals adult hens are highly susceptible to delayed neuropathy.**
➢ **increases the concentration of AcTh** & activation of muscarinic and nicotinic receptors in central and peripheral nerves and in neuro-muscular junctions
➢ **Blocks AChe enzymes=esteratic** site and stable enzyme-OP complex (phosphorylated enzyme) is formed due to irreversible binding
➢ **OPIDN-t**richlorphon, Diisoprophyfluorophosphate (DFP), monocrotophos, fenitrothion, haloxon, chlorpyriphos etc.
➢ Humans, Chickens And Cats Whereas Others Are Non Susceptible Rats, Mice, Rabbits And Guinea Pigs
➢ Salivation,Lacrimation,Urination,Defecation=SLUD

On the basis of action "Directly acting"	On the basis of action "Indirectly acting"
phosphates and pyrophosphates (P=O oxon bond),act directly on acetylcholinesterase without metabolic conversions	phosphorothioates (P=S thionate bond) and their active metabolites LESS TOXIC
e.g., dichlorvos, DFP,Tabun, tetraethylpyrophosphate (TEPP), trichlorphon,Serin,Soman,Diazinon	e.g., Melathion, parathion, fenthion,Fenirothion etc

On the basis of chemical structure	
Phosphates & pyrophosphates	TEPP, dichlorvos
Phosphorothioates	Parathion
Phosphonates	Trichlorfon
Phosphoramidates	Phospholan
Phosphorothiolates	Echothiophate
Phosphorohalides	Diisoprophylfluorophosphate/DFP
Phosphorocyanides	Tabun

✓ Rx:**Atropine sulphate**
✓ **Cholinesterase reactivators/Oximes(**not after 24-36 hours).
  ✓ **Pyridine-2-aldoxime (2-PAM)** Aging
  ✓ **Diacetylmonoxime (DAM), Toxagonine**
✓ Diazepam
✓ Supportive : Gastric lavage, emesis, activated charcoal etc

CARBAMATES

- cyclic or aliphatic derivatives of carbamic acid
- inhibit acetylcholinesterase enzymes but these insecticides occupy both **anionic site and esteratic sites of AChE(Carbamoylation)**
- as **reversible** anticholinesterases agents.
- **Rx:** Atropine sulphate
- **2-PAM should not be used in carbamate poisoning**
- **(**Carbamates bind both at esteratic and anionic site of AchE**)**

Oximes	**Aldicarb, methomyl, thiocarb**
Naphthyl carbamates	Carbaryl (sevin)
Phenyl carbamates	Propoxur
Heterocyclic methyl carbamates	Pyrolan and isolan
Heterocyclic dimethyl carbamates	Carbofuran and furadan

Organophosphates poisoning	Organochlorine poisoning
Profuse and watery salivation	Frothy sticky salivation
Delayed neuropathy	Not observed
Body temperature sub normal	Hyperthermia
Pupil constricted (miosis)	Normal/mydriasis
Behavioural changes not common	Highly behavioural changes
Aimless running in straight direction	Circling movement
Normal posture	Abnormal posture
Quick onset of acute signs	Comparatively delayed
Atropine	Sedatives

Carbamate poisoning	Organophosphorus poisoning
Reversible inhibition of cholinesterase	Irreversible inhibition of cholinesterase
Both anionic and esteratic sites of cholinesterase are occupied	Only esteratic site is occupied
No inhibition of carboxylesterase (liver enzyme)	Inhibitor of carboxylesterase
Reactivation of inhibited ChE is easier	Reactivation of inhibited ChE is difficult
Cholinesterase enzyme reactivators (PAM, DAM) should not be used	Can be used before 24-36 hours
Less severity poisoning and development of acute signs	Acute signs are prominent and severe
Duration of poisoning less	Comparatively more duration

ORGANOCHLORINES INSECTICIDES

- chlorinated hydro-carbon compounds having C, H, Cl and sometimes oxygen atoms with a number of C-Cl bonds
- **Young animals>adults, Females>males,**
- **Cats>Others & Fish>mammals>birds**
  - **(Glucuronic acid conjugation is absent)**
- **Cyclodienes**>aliphatic alkyl compounds
  - LD_{50} of DDT in dogs =500-750mg/Kg
  - LD_{50} of Aldrien and dieldrin in dogs= 65mg/Kg
- **Neurotoxicity** by closure of K-channel and opening of Na-channel in neurons
- Binds with **GABA** receptors and impede Cl-conductance in cell(excitation-cyclodienes)
- Uncoupling of oxidative phosphorylation & also endocrine disruptors
- C/S:Twitching,Excitement,Circling,Behavioural,HeadPressing, TonicClonic convulsions,Salivation,Mydriasis,Increased Secretions
- **Rx::Sedatives**: Diazepam, phenobarbitone, xylazine
  - Supportive treatment: Gastric lavage, emesis, purgatives, activated charcoal etc

Diphenyl aliphatic compounds	**DDT, Methoxychlor etc**
Aryl hydrocarbons	BHC (lindane), Mirex etc
Cyclodienes	Aldrin, dieldrin, endosulphan

Differentials

Salt poisoning (NaCl)	**Similar signs are produced .Absence of high body temperature**
Strychnine Poisoning(No behavioural aberrations)	There are convulsions but they are tonic
Fluoroacetate Poisoning (Excitation with external stimuli absent)	Convulsion although tonic-clonic become weak with time.
Organophosphates and carbamates (There are no behavioural changes)	.Body temperature not elevated in fact there is hypothermiaTreatment with atropine remove toxic signs
Lead poisoning (No rise of temperature)	No abnormal posturing. Convulsions are less common.
Urea poisoning (Colic is an important sign.)	No abnormal posturing or jumping over unseen objects although there is behavioural change like bovine bonkers.Convulsions are tonic i

FUNGICIDES

I. Inorganic fungicides ::Sulphur Metals e.g. copper sulphate, bordeaux mixture (copper sulphate + calcium hydroxide), cadmium chloride and mercuric chloride.

II. Organic fungicides

1. Organomercury compounds e.g. phenylmercuric chloride, phenylmercuric acetate, ethylmercury acetate and methylmercury.

2. Chlorophenols, e.g. pentachlorophenol and trichlorophenol.

3. Dithiocarbamates e.g. ferbam, maneb, thiram and zineb.

4. Organotin compounds e.g. triphenyltin, tributyltin and triethyltin.

5. Phathalimides e.g. captan, folpet and captafol.

6.Benzimidazoles, e.g. benomyl, thiophanate methyl and fuberidazole.
7.Azoles e.g. cyproconazole, etridazole and penconazole.

8. Halogenated substituted monocyclic amines e.g. chlorothalonil, tecnazine and dichloran.

9. Morpholines e.g. dodemorph, fenpropiomorph and tridemorph.

10.Miscellaneous fungicides, e.g. cycloheximide,pentachloronitrobenzene (PCNB), pyrazophos,terrazole and dinocap.

HERBICIDES

- **I. Inorganic herbicides** Arsenicals e.g. sodium arsenite. Chlorates e.g. sodium chlorate.
- **II. Organic herbicides ::**
- Chlorophenoxy herbicides e.g.2, 4-D, 2,4,5-T, mecoprop, MCPA and MCPB.
- Dinitro herbicides e.g.dinitroorthocresol (DNOC) dinoseb.
- Bipyridyl herbicides e.g. paraquat and diquat.
- Triazine herbicides e.g. atrazine, simazine and propazine.
- Substituted urea herbicides e.g. monuron, diuron and isouron.
- Carbamates and thiocarbamates
- e.g. barban, propham, cycloate and butylate. Uracils
- e.g. bromacil, isocil and terbacil. Chlorobenzoic acid
- e.g. chloramben and dicamba. Chloracetanilides
- e.g.alachlor, butachlor and metolachlor Organophosphates
- e.g. glyphosate and glufosinate.

Others...

- CHLORATE TOXICITY-haemoglobin into methaemoglobin-dark tarry blood from natural orifices which clots readily-Methylene blue is antidote
- UREA AND ITS TOXICITY:: In ruminants 0.3-0.5g/kg may be toxic unaccustomed. Oral LD 50 in sheep and cattle is 1-1.5g/kg
  - **Peracute-bovine** bonkers.. Rx:;Acetic acid (5%) ruminal infusion @ 2-8 L in cattle and @ 2 L in sheep lowers rumen pH,Iced water ruminal infusion decrease urease activity,Rumen tympany is corrected with trochar and canula,Convulsions are controlled by pentobarbitone
- Thallium decreases the activity of SH-enzymes (succinic dehydrogenase and MAO).
- Zinc phosphide is generally available as grey-black powder with garlic like odour-Phosphine gas inhibits cytochrome C oxidase which causes tissue **hypoxia-Mad dog running**= Stomach contents may have a characteristic **acetylene or dead fish odour**
- Alpha naphthyl thiouria (ANTU): massive pulmonary oedema and leakage of fluid into air passages=**Drowning in its own fluids,**
  - Rx::Competitive ANTU antagonists like ethylenethiouria or 1-ethyl-1-phenyl thiouria
- Red squill-bulbs of the Mediterranean sea onion Urginea maritime, a plant with large bulbs. It contains a mixture of glycosides of which scilliroside
- Fluoroacetate: As such it is non toxic, however, it becomes toxic after conversion into fluorocitrate. Fluorocitrate competitively blocks enzyme aconitase of the Krebs cycle RxGlycerol monoacetate, ethanol and acetic acid/acetate can be used .

Others..

- Bromethalin-uncoupler of oxidative phosphorylation in the mitochondria of CNS. Un-coupling results in less production of ATP which in turn leads to less energy for Na-K-ATPase activity
- Pyriminil-destruction of pancreatic beta cells and development of insulin deficient diabetes mellitus
- Reserpine-alkaloid obtained from Rauwalfia serpentine plant.
- Norbromide-selective rodenticide against rats (receptors are present only in rats) but not effective against mice
- Alpha-chloralose-It is a fast acting narcotic agent used occasionally as rodenticide and avicide

RADIATION TOXICOLOGY

<table>
<tr><th colspan="3">Non genetic Effcts</th><th>Genetic Effects</th></tr>
<tr><th colspan="2">Whole body irridation</th><td rowspan="2">Partial irridation</td><td rowspan="2"></td></tr>
<tr><th>Early effects</th><td>Late effects</td></tr>
<tr>
<td rowspan="2">Prodromal syndrome- It appears within 48 hours of irradiation and is mediated through autonomic nervous system and signs are anorexia, nausea, vomition at lower doses 4-5 gy and diarrhoea, fever and listless at higher doses
Neurological syndrome-It is observed after high dose of radiation > 100 gy producing death from cerebrovascular damage in most mammalian species within hours
Gastrointestinal syndrome- This is characterized by prodromal phase followed by prolonged diarrhoea, loss of apetite, loss of fluid, dehydration, weight loss. Dose of 5-10 gy can cause death from gastrointestinal injury with in few days
Haemopoitic syndrome Proliferating haemopoietic stem cells are highly radiosensitive and are sterilized by radiations thereby reducing the blood supplies of red and white cells. 2.5-5 gy may cause death in several weeks</td>
<td>Clinical signs appear after month or years. These include incidence of neuplasms, leukemia, local tissue changes like lens opacity, shortening of life</td>
<td rowspan="2">A part of body or organ gets exposure
Skin-Fall out of skin especially by beta radiations
Reproductive system-Fertility in males and females decrease
GIT-Inflammation, ulceration and necrosis of GIT
Respiratory system-Ulceration, atropy, fibrosis, pneumonitis, edema occur in respiratory tract
Nervous system-Demyleniation and necrosis of neurons accompanied by signs like numbness, weakness and paralysis
Endocrine glands-Gamma radiations and internally deposited radio iodine can cause hypothyroidism and growth retardation
Urinary system-Kidneys are highly sensitive followed by bladder and ureter. Inflammation, ulceration, fibrosis are common signs in bladder
Connective tissue-Muscles, bones and cartilages are relatively resistant to direct cytocidal action of radiation. Damage to bone marrow may lead to anaemia, leucopaenia, lymphopaenia and tumor formation</td>
<td rowspan="2">Damage to germ cells may lead to mutation. Point mutation, addition or deletion, translocation are some common changes in genetic material by radiations which are carried to future generations. Mutation appear either as recessive or in dominant form

Rem (radiation equivalent man) is the amount of any radiation that produces a biological effect equivalent to that resulting from 0.01 gray of gamma radiations. The unit Rem takes into account the damage caused to humans tissue by the radiations</td>
</tr>
<tr>
<td>Units of radiations
Radiation dose-in terms of quantity of absorbed energy per unit mass. The SI unit is the gray (gy) which is defined as 1 joule of energy per kilogram. The older unit of Rad (radiation absorbed dose) which is still in use is equal to 0.01 joules of energy per kilogram of tissue. Thus, one gray is equal to 100 Rad..
a) Cells in early G-phase,Zygotes,Highly proliferative like haemopoetic system more sensitive
b) Young and old persons are more radiosensitive</td>
</tr>
</table>

LABORATORY SAMPLES

Sample	Amount	Condition	Examples of Toxicoses
Antemortem			
Whole blood	1-3 mL	EDTA or heparin anticoagulant	Lead, arsenic, mercury, selenium, pesticides, anticoagulants
Urine	10-50 mL	Plastic screw- capped vial	Drugs, some metals, paraquat, alkaloids
Serum	5 mL	Remove from clot; element tubes	Trace metals (no rubber contact if testing for zinc), some drugs, ethylene glycol, electrolytes, botulinum, iohexol
Cerebrospinal fluid	1 mL	Clot tube	Sodium
Gastrointestinal contents	100 g	Obtain represen tative sample	Pesticides; plant, metal-, and feed related poisons
Body fluids	10-20 mL	Clot tubes	Anticoagulants
Hair	1-5 g	Rarely useful	Call laboratory; chronic selenosis
Postmortem			
Urine, serum, body fluids	1-50 mL	Same preparation and tests as for antemortem samples; get serum from heart clot	Drugs, arsenic
Liver	100 g	Plastic (foil for organics)	Pesticides, metals, botulinum
Kidney	100 g	Plastic (foil for organics)	Metals, compound1080 (sodium monofluoroacetate), calcium, ethylene, glycol, cholecalciferol
Brain	50%	Cut sagittally; put half in plastic for analysis (fix other half for pathologic examination)	Organochlorines, sodium, bromethalin
Fat	100 g	Foil in plastic	Organochlorines
Lung	100 g	Plastic	Paraquat
Pancreas	100 g	Plastic	Metals (zinc)
Gastrointestinal contents	100 g	Obtain representative sample	Pesticides/baits; plant-, metal-, or feed-related toxicants
Bone	100 g	One long bone	Fluoride
Miscellaneous		Injection sites, spleen	Some drugs (barbiturates in spleen)
Environmental			
Baits/sources	200 mL or g	Clean mason jar(liquid); plastic vial (write chemical name if available)	Unidentified chemicals, organics
Feed	1 kg	Plastic, box; must be representative	Mycotoxins, feed additives, plants, pesticides
Plants	Entire plant	Fresh or pressed, send all parts	Identification, chemical assay
Water	1 L	Clean mason jar; foil under lid for organics, plastic lid for metals	Metals, nitrates, pesticides, algae, salt, organics

*Submit samples frozen except for blood or if very dry. Amounts given are optimum amounts; smaller samples may be

Further Reading & Refs:

- ✓ Sandhu H. S. & Brar R. S: Text Book of Veterinary Toxicology.
- ✓ Garg S. K: Veterinary Toxicology.
- ✓ Wikipedia & Google images.
- **Illustrated Objective Toxicology for Veterinary Exams, Dr.M.Alpha Raj**

https://t.me/joinchat/Gmb76hZrKdbwsTIMqadZsw

THANK YOU!!!!!

Any Suggestions & Corrections Are Welcome

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- **Vety exam Aspirants** (Telegram group for MCQs Discussion- https://t.me/joinchat/Gmb76hZrKdbwsTIMqadZsw)

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