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SPECIAL ISSUE

ON

POISONING BY AGRO-CHEMICALS

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POISONING BY AGRO-CHEMICALS

by

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The increasing use of agro-chemicals in this country has led to an increase in the frequency of poisoning caused by these chemicals. Only crude data are available to indicate the extent of the problem and according to the hospital admissions during the year 1975 to 1977, about 15000 patients are admitted to hospital per year for the treatment of poisoning by agro-chemicals of whom approximately 1000 patients per year die.

This special issue of the "Prescriber" has been prepared to assist medical practitioners and others who may be called upon to deal with cases of poisoning.

Poisoning may occur either accidentally, by intent or as a result of accidental exposure. Whatever the circumstances, prompt treatment is essential. This requires quick identification of the agro-chemical involved, and the chemical class which the agro-chemical belongs.

To facilitate rapid identification and institution of appropriate treatment the agro- chemicals are listed according to their trade names in alphabetical order, the chemical class and the official name being indicated in each case.

Agro-chemicals are classified as follows:—

1. **Pesticides**

Organochlorines (chlorinated hydrocarbons)
Organophosphates
Carbamates
Pyrethrum and synthetic pyrethroids
Miscellaneous

2. **Rodenticides**

Coumarins
Thallium
Zinc phosphate

3. **Snail bait**

Metaldehyde

4. **Herbicides (Weedicides)**

Acetamides
Acetanilides
Arsenic compounds
Bipyridyls
Carbamates
Phenols & Nitrated Cresols
Propionic acid derivatives
Triazines

5. **Fungicides**

Copper compounds
Dithiocarbamates
Organo-mercury compounds
Chlorobenzene compounds
Benzimidazole and miscellaneous compounds

TRADE NAMES AND CHEMICAL CLASS

1. Pesticides

Trade Name	Official Name	Class
Abar	Leptophos	Organophosphate
Accothion	Fenitrothion	Organophosphate
Actellic	Pirimiphos	Organophosphate
Agridol	Ethyl Parathion	Organophosphate
Agrothion	Fenitrothion	Organophosphate
Aldrex	Aldrin	Organochlorine
Aldrimul	Aldrin	Organochlorine
Aldrin	Aldrin	Organochlorine
Aldri Powder	Aldrin/Methyl Parathion	Organochlorine/ Organophosphate
Ambush 25 EC	Permethrin	Synthetic Pyrethroid
Anthio	Formothion	Organophosphate
Arkotine	Heptachlor/BHC	Organochlorine
Asuntol	Coumaphos	Organophosphate
Azodrin	Monocrotophos	Organophosphate
Bandrin	Endrin	Organochlorine
Bast	Dimethoate	Organophosphate
Bassa	BPMC	Carbamate
Basudin	Diazinon	Organophosphate
Baycarb	BPMC	Carbamate
Baygon bait	Propoxur	Carbamate
Baygon sprav	Propoxur & Dichlorvos	Carbamate/ Organophosphate
Baythion	Phoxim	Organophosphate
Bayrusi	Quinalphos	Organophosphate
Baytex	Fenthion	Organophosphate
BHC	Gamma benzene hexachloride	Organochlorine
Bidrin	Dicrotophos	Organophosphate
Birlane	Chlorfenvinphos	Organophosphate
Bolstan	Mercaptophos	Organophosphate
BPMC	BPMC	Carbamate
Brassicol	Pentachlorobenzene	Organochlorine
BUX	Metalkamate	Carbamate
Bufencarb	Metalkamate	Carbamate
Carbicron	Dicrotophos	Organophosphate
Carbophenothion	Carbophenothion	Organophosphate
Carbaryl	Carbaryl	Carbamate

Trade Name	Official Name	Class
Chlordane	Chlordane	Organochlorine
Chlordox	Chlordane	Organochlorine
Chlorobenzilate	Chlorobenzilate	Organochlorine
Cidial	Phenthoate	Organophosphate
Ciodrin	Crotoxyphos	Organophosphate
Coopex	Permethrin	Pyrethroid
Curaterr	Carbofuran	Carbamate
Daphone	Dimethoate	Organophosphate
DDT	Dicophane	Organochlorine
Deebug	Chlordane	Organochlorine
Deenol	Dicophane	Organochlorine
Dedevap	Dichlorvos	Organophosphate
Demeton	Demeton-C-Methyl	Organophosphate
Diazinon	Diazinon	Organophosphate
Dicarbam	Carbaryl	Carbamate
Dicial	Phenthoate	Organophosphate
Dicofol	Dicofol	Organochlorine
Didimac R 5	Dicophane	Organochlorine
Didimol	Dicophane	Organochlorine
Dieldrin	Dieldrin	Organochlorine
Dieldrex	Heptachlor/ Aldrin	Organochlorine
Dimecron	Phosphamidon	Organophosphate
Dimethoate	Dimethoate	Organophosphate
Dipterex	Trichlorphon	Organophosphate
Di-syston	Disulfoton	Organophosphate
Dursban	Chlorpyriphos	Organophosphate
Ecopro-17	Abate	Organophosphate
Ekalux	Quinalphos	Organophosphate
Ekatox	Ethylparathion	Organophosphate
Ektafos	Dicrotophos	Organophosphate
Elsan-50	Phenthoate	Organophosphate
Endosulfan	Endosulfan	Organochlorine
Endrex	Endrin/Methyl Parathion	Organochlorine/ Organophosphate
Endrin	Endrin	Organochlorine
Fenthion	Fenthion	Organophosphate
Fenitrothion	Fenitrothion	Organophosphate
Folidol	Parathion	Organophosphate
Folidol-E	Ethyl parathion	Organophosphate
Folimat	Omethoate	Organophosphate

Trade Name	Official Name	Class
Folithion	Fenitrothion	Organophosphate
Formothion	Formothion	Organophosphate
Fosferno 50	Ethyl parathion 50%	Organophosphate
Furadan	Carbofuran	Carbamate
Gammalin 20	Gamma benzene hexachloride (BHC) 20%	Organochlorine
Gammexane D 120	Gamma benzene hexachloride (BHC) 10%	Organochlorine Organophosphate
Gardona	Trichlorfon	Organophosphate
Guthion	Azinphos-Methyl	Organochlorine
Heptachlor	Heptachlor	Organochlorine
Heptamul	Heptachlor	Organochlorine
Hexidole	Gamma benzene hexachloride (BHC)	Organophosphate
Hinosan EC 50%	Edinphenphos	Organochlorine
Hopcin EC	BPMC	Carbamate
Hortex	Gamma benzene hexachloride (BHC)	Organochlorine
Intox 8	Chlordane	Organochlorine
Jacutin	Gamma hexachlor cyclohexane	
Kelthane	Dicofol	Miscellaneous
Kilemol	Chlordane/ dichlorvos + malathion	Organochlorine/ Organophosphate
Lannate	Methomyl	Carbamate
Lebaycid	Fenthion	Organophosphate
Leeseal	Chlordane	Organochlorine
Lepton	Leptophos	Organophosphate
Linoamul	Gamma benzene hexachloride	Organochlorine
Lindale	Gamma benzene hexachloride	Organochlorine
Lindane	Gamma benzene hexachloride	Organochlorine
Lorsban	Chloropyriphos	Organophosphate

Trade Name	Official Name	Class
Mortein	Pyrethrum	Pyrethrum
Mafu	Dichlorvos & Fenthion	Organophosphate
Makuna	Chlordane	Organochlorine
Malacide	Malathion	Organophosphate
Maladrex	Malathion	Organophosphate
Malathion	Malathion	Organophosphate
Malatox	Malathion	Organophosphate
Matacil	MPMC	Carbamate
Mecbal	MPMC	Carbamate
Mercaptophos	Fenthion	Organophosphate
Metacide EC	Methyl parathion	Organophosphate
Metasystox	Dimeton-S-Methyl	Organophosphate
Metalkamate	Metalkamate	Carbamate
Methamidophos	Methamidophos	Organophosphate
Methomyl	Methomyl	Carbamate
MLT-50	Malathion	Organophosphate
Monitor	Methamidophos	Organophosphate
Monocrotophos	Monocrotophos	Organophosphate
Morex	Dimethoate	Organophosphate
Morocide	Binapacryl	Miscellaneous
Morestan	Quinomethionate	Miscellaneous
MPMC	MPMC	Carbamate
Niran	Ethyl Parathion	Organophosphate
Nogos	Dichlorvos	Organophosphate
Nuvacron	Monocrotophos	Organophosphate
Nuvan	Dichlorvos	Organophosphate
Octachlor	Chlordane	Organochlorine
Octadex	Chlordane	Organochlorine
Oftanol	Isophenphos	Organophosphate
OKO	Dichlorvos	Organophosphate
Orthom	Acepate	Organophosphate
Papthion	Phenthoate	Organophosphate
Parathion-M	Methyl parathion	Organophosphate
Parathion	Ethyl parathion	Organophosphate
Paracide	Paradichlor-benzene	Organochlorine
PDCB	Paradichlor-benzene	Organochlorine
Perfekthion	Dimethoate	Organophosphate
Phenthoate	Phenthoate	Organophosphate

Trade Name	Official Name	Class
Phosdrin	Mevinphos	Organophosphate
Phosvel	Leptophos	Organophosphate
Phoxim	Phoxim	Organophosphate
Pirimphos methyl	Pyrimiphos methyl	Organophosphate
		Organophosphate
Quinalphos	Quinalphos	Organophosphate
Rogor	Dimethoate	Organophosphate
Rogrow	Dimethoate	Organophosphate
Run bug	Malathion	Organophosphate
Sayfos	Menazon	Organophosphate
Sevidol	Carbaryl	Carbamate
Sevin	Carbaryl	Carbamate
Shelltox	Pyrethrum 25%	Pyrethrum/ piperonyl butoxide
Shelltox Red	Pyrethrum/ chlorfenvinphos	Pyrethrum/ organophosphate
Shelltox with vapona	Pyrethrum/ dichlorvos	Pyrethrum/ organophosphate
Silfix	Chlordane + mercury cpd	Organochlorine
Sumicidin	Fenvalerate	Pyrethroid
Sumithion	Fenitrothion	Organophosphate
Supersumithion	Fenitrothion	Organophosphate
Supona	Chlorfenvinphos	Organophosphate
Tamaron	Methamidophos	Organophosphate
Tedion	Tetradifon	Miscellaneous
Tedion V-18	Tetradifon	Miscellaneous
Telodrin	Isobenzan	Organochlorine
Tetradifon	Tetradifon	Miscellaneous
Texastan	Camphechlor	Organochlorine
Thiodan	Endosulfan	Miscellaneous
Tokuthion	Prothiophos	Organophosphate
Toxaphene	Camphechlor	Organochlorine
Trichlorphon	Trichlorphon	Organophosphate
Trithion	Carbopenothion	Organophosphate
Unden	Propoxur	Carbamate
Unimal	Malathion	Organophosphate
Vapona	Dichlorvos	Organophosphate
Volaton 500EC	Phoxim	Organophosphate

2. Rodenticides

Trade Name	Official Name	Class
Actosin	Warfarin	Coumarin
Dethmor	Warfarin	Coumarin
Drat	Chlorphacinone	Coumarin
Kil Rat	Zinc phosphide	Phosphorus
No Rat	Zinc phosphide	Phosphorus
Racumin	Coumatetralyl	Coumarin
Ratak	Difenacoum	Coumarin
	Zinc Phosphide	Phosphorus
Zelio paste	Thallium sulphate	Thallium

3. Snail Bait

Meta	Metaldehyde	Metaldehyde
Metabait	Metaldehyde	Metaldehyde
Slug It	Metaldehyde	Metaldehyde
Snail Bait	Metaldehyde	Metaldehyde
Snail Killer	Metaldehyde	Metaldehyde
Snail X	Metaldehyde	Metaldehyde

4. Herbicides (Weedicides)

Afalon	Linuron	Carbamate
Agroxone	MCPA	Acetamide
Bassafon	Dalopon sodium	Propionic acid derivative
Brushkiller	2.45T & 2.4D	Acetamide
Dalapon	Dalapon	Propionic acid derivative
Dalspray	Dalapon	Propionic acid derivative
Diuron	Diuron	Carbamate
Dowpon	Dalapon sodium	Propionic acid derivative
3.4oD.P.A.	Propanil	Propionic acid derivative
Fernoxone	2.4D	Acetamide
Finopal	2.4D & 2.4.5.T	Acetamide
Gesaguard 50	Prometryn	Triazine
Gesapax 50	Ametryn	Triazine
Gesamil	Propazine	Triazine
Gesatop	Simazine	Triazine
Gramevin	Dalapon sodium	Propionic acid derivative

Trade Name	Official Name	Class
Gramoxone	Paraquat	Bipyridyl
Hedonal M	MCPA	Acetamide
Hedonal D	2.4.D	Acetamide
Karmex	Diuron	Carbamate
Lasso	Alachlor	Acetanilide
Linurol	Linuron	Carbamate
Lorox	Linuron	Carbamate
M 50	MCPA	Acetamide
Machete	Butoclor	Acetanilide
MCPA	MCPA	Acetamide
Morice	MCPA	Acetamide
Palormone	MCPA	Acetamide
Peecep	Pentachlorphenol	Phenols & Nitrated Cresols
Phordene	MCPA	Acetamide
Planotox	MCPA	Acetamide
Propanex 45	Propanil	Propionic acid derivative
Ramrod	Propochlor	Acetanilide
Rogue - 34	Propanil	Propionic acid derivative
Roundup	2.4.D/Chloro isopropyl acetanilide	Acetamide
Sandolin A	Dinitrocresol	Phenols & Nitrated Cresols
Saturn-G	Benthiocarb	Carbamate
Sencor	Metribuzin	Thiazine
Shell D	2.4.D	Acetamide
Sodium arsenite	Sodium arsenite	Arsenic compound
Spontox	2.4.D & 2.4.5.T	Acetamide
Stam-F-34	Propanil	Propionic acid derivative
Surcopur	Propanil	Propionic acid derivative
Telvar	Monuron	Carbamate
Tordon	MCPA	Acetamide
Tribunil	Methabenzthiasuron	Carbamate
Tributon	2.4.D & 2.4.5.T	Acetamide
Ustinex PA	Diuron/Amitrol	Carbamate
Weedkiller	2.4.5.T	Acetamide

Trade Name	Official Name	Class
Weedkiller D	2.4.D	Acetamide
Weedkiller P.20	Pentachlorophenol	Phenols & Nitrated Cresols

5. Fungicides

Agrosan	Methoxyethyl mercuric chloride	Mercury compound
Antimucin	Phenylmercuric nitrate	Mercury compound
Antracol	Propineb	Dithiocarbamate
Benomyl	Benomyl	Miscellaneous
Blitox	Copper oxychloride	Copper compound
Blendox	Copper dust	Copper compound
Bordeaux Mixture	Copper sulphate & lime	Copper compound
Captan	Captan	Miscellaneous
Carbox	Copper oxychloride	Copper compound
Carboxim	Carboxim	Miscellaneous
Ceresan Wet	Methoxyethyl mercuric chloride	Mercury compound
Ceresan dry	Phenylmercuric nitrate	Mercury compound
COC	Copper oxychloride	Copper compound
Colloidox	Colloidal Copper	Copper compound
Cupracol	Colloidal Copper	Copper compound
Cuprantol	Copper spray	Copper compound
Cupramox	Copper oxide 50%	Copper compound
Cuprasan	Copper zinc	Copper compound
Cuprasana	Copper oxychloride	Copper compound
Cupravit	Copper oxychloride	Copper compound
Cupravit blue	Copper hydroxide	Copper compound
Cuprokylt	Copper oxychloride	Copper compound
Dithane A 40 & D 22	Nabam	Dithiocarbamate
Dithane M 45	Mancozeb	Dithiocarbamate
Etrofolan	Isoprocarb	Dithiocarbamate
Fermate	Ferbam	Dithiocarbamate
Fomac	Quintozene	Chlorobenzene compound

Trade Name	Official Name	Class
Hexachloro-benzene	Hexachlorobenzene	Chlorobenzene compound
Kauritil	Copper oxychloride	Copper compound
Lonacol	Zineb	Dithiocarbamate
Manzate	Maneb	Dithiocarbamate
Manzate - D	Maneb	Dithiocarbamate
Manzate - 200	Mancozeb	Dithiocarbamate
Mildrex	Zineb/Copper Oxychloride	Dithiocarbamate
Miltox 50	Copper oxychloride	Dithiocarbamate
Mipcin	Isoprocarb	Copper compound
M Z 4	Ethylene bisdithio-carbamate/ZN/Mn	Dithiocarbamate
Oxyear	Copper oxychloride	Dithiocarbamate
Panogen	Methylmercury dicyanamide	Copper compound Mercury compound
Paris Green	Copper aceto-arsenite	Copper compound
Parzate	Nabam	Dithiocarbamate
Perenox	Copper oxide 50%	Copper compound
Perezin	Copper Zinc	Copper compound
Pericloud	Copper oxychloride dust	Copper compound
Polyram M	Maneb	Dithiocarbamate
Propineb	Propineb	Dithiocarbamate
Tiezene	Zineb	Dithiocarbamate
Tillex	Methoxyethyl mercuric chloride	Mercury compound
Unisol	Methoxyethyl mercuric chloride	Mercury compound
Vitigran	Copper oxychloride	Copper compound
Zebenide	Colloidal Copper	Copper compound
Zineb	Zineb	Dithiocarbamate
Zerlate	Ziram	Dithiocarbamate

CLINICAL MANIFESTATIONS & MANAGEMENT OF POISONING

(1) ORGANOCHLORINES

Poisoning may follow ingestion, accidentally or with suicidal intent, inhalation of the spray or dust or absorption through the skin particularly in oily solution.

Clinical Manifestations

Initial clinical manifestations of poisoning are the result of stimulation of the central nervous system.
They consist of:—

Headache, nausea, vomiting, dizziness, paraesthesiae, muscle fibrillation, tremors and convulsions.

If poisoning is severe this is followed by depression of the CNS and lead to coma and sometimes death from respiratory failure.

Treatment

If poisoning is due to absorption through the skin, the skin should be washed promptly with soap and running water.

Poisoning resulting from ingestion should be treated with a gastric lavage and administration of a saline purgative. Oils and oil laxatives should be avoided as they potentiate absorption of the toxicant.

Treatment for the effects of CNS stimulation is primarily symptomatic.

Phenobarbitone 100mg is preferable prophylactically in the absence of CNS signs and symptoms but if tremors and convulsions are present a faster acting drug such as **amylobarbitone** 100-200mg should be used and repeated if necessary. The dose must be adjusted to sedate without inducing sleep.

Calcium gluconate (10ml of a 10% solution) given slowly iv.v may be helpful in addition to sedation to control convulsions.[1]

Patient who experience a convulsion should be observed carefully for at least one week.

Sudden physical stimuli should be avoided as they may precipitate convulsions.

In the presence of respiratory depression, oxygen therapy and artificial respiration may be needed.

Avoid the use of adrenaline.

(2) ORGANOPHOSPHATES

Clinical Manifestations

Organophosphates are anticholinesterases. They cause irreversible inhibition of cholinesterases. In poisoning caused by organophosphates, acetylcholine accumulates greatly in excess of physiological levels at cholinergic nerve endings such as the motor and postganglionic parasympathetic nerves and in the brain. Stimulation of cholinergic receptors by excess acetylcholine gives rise to the clinical manifestations.

Action at the postganglionic parasympathetic nerve endings leads to nausea, excessive salivation, vomiting, abdominal colic, diarrhoea, tenesmus and involuntary defaecation; rhinorrhoea, dyspnoea, pulmonary rales and rhonchi and frothing; miosis, lacrimation and blurred vision.

Its effects at the neuromuscular junction results initially in muscular twitching usually of the tongue and face and in severe poisoning, generalized twitchings, muscular weakness followed by paralysis.

Accumulation of acetylcholine in the brain results in headache, giddiness, confusion and convulsions. This may be later followed by coma and paralysis of the respiratory centre.

Management

(1) General Measures

If the compounds have been swallowed the stomach should be washed out with a solution of sodium bicarbonate.

If absorption has occurred through the skin, all clothing that might be contaminated must be removed at once and the areas exposed to the chemical should be thoroughly washed with soap and water.

In the presence of coma cyanosis and respiratory embarrassment, care must be taken to keep the airway clean. It may be necessary to pass an endotracheal tube or perform a tracheostomy. Excessive secretions in the mouth and throat must be removed by suction. Careful watch must be kept on the respiration. If respiratory depression occurs, pulmonary ventilation must be maintained by artificial means. Oxygen inhalation may be required.

Drug Therapy

(a) Atropine

Atropine must always be used first as it directly counteracts the effects of excessive acetylcholine accumulation by competitive antagonism. Atropine antagonises the parasympathomimetic as well as the central effects mediated by acetylcholine. It has no effect on the action of acetylcholine at the neuromuscular junction namely atimulation manifested by muscle fasciculation, followed later by muscle weakness, flaccid paralysis and respiratory failure.

Atropine must be given promptly and in large doses. A dose of 2mg i.m. or i.v. is sufficient in mild cases. If the symptoms are not relieved or if the condition of the patient is seen to deteriorate at the end of 10—15 minutes, atropine is repeatedly administered in doses of 2mg i.v. at 10—15 minute intervals until symptoms are controlled and signs of atropine toxicity appear, namely flushing of the skin and an increase in pulse rate to 120—140 per minute. The patient should be kept fully atropinised and under observation for

at least 24 hours. This blocks the action of excess acetylcholine until such time as the cholinesterase inhibitor is detoxified and excreted. If symptoms return after stoppage of atropine, further doses should be given. Closely supervision is indicated for at least 2—3 days.

Persons poisoned with organophosphate compounds tolerate atropine to a much greater extent than do normal people and large amounts of atropine are required.

Atropine should not be given to a syanotic patient until hypoxia is relieved as it may give rise to ventricular fibrillation and cardiac failure, in an intoxicated subject with asphyxia. In severe poisoning up to 100mg has been given in a 24 hour period. The usual problem is failure to give enough than giving too much.

(b) Cholinesterase Reactivators

Pralidoxime 'Protopam" "Antidote Pam"

Pralidoxime belongs to a group of compounds known as oximes which act as reactivators of cholinesterase by breaking the relatively irreversible binding of the phosphoryl group of the organic phosphates releasing the cholinesterase. The released enzyme is then available to destroy the acetylcholine which is causing the symptoms. This reactivating effect is most marked at the neuromuscular junction. It is less marked at the autonomic effector sites and insignificant in the CNS. It is therefore mainly of value in reversing skeletal muscle paralysis particularly the respiratory muscles.

Pralidoxime is most effective if administered immediately after poisoning. Generally it is of little value if given more than 36 hours after exposure has been stopped. However when the poison has been swallowed, pralidoxime may be given even later as there is slow absorption of the poison from the lower bowel. Pralidoxime should be started at the same time as atropine.

Pralidoxime is available as the chloride in powder form in vials containing lg of the powder. The powder is dissolved in 20ml of water and injected slowly i.v. at a rate

not exceeding 10ml (500mg) per minute. The dose for children is 20-40mg/mg body weight.

After about an hour a second dose of 1g may be given if muscle weakness has not be relieved. Additional doses may be given cautiously if muscle weakness persists, to a total of 4g in 24 hours. If intravenous administration is not feasible pralidoxime should be given i.m.

Obdoxime "Toxogonin"

It is another cholinesterase reactivator available commercially in ampoules containing 250mg in 1ml. A dose of 250mg is given initially i.v. diluted in saline and if a satisfactory response is not obtained a further ampoule should be given once or twice at an interval of 2 hours.

Other Drugs

If convulsions interfere with respirations, Diazepam may be given i.v. with care in a dose of 5-15mg at the rate of 0.5ml/minute. Otherwise in general, barbiturates, tranquillizers and morphine are contraindicated.

If pulmonary oedema occurs **frusemide** 20mg should be given i.v. as well.

(3) CARBAMATES

They act as rapidly reversible inhibitors of cholinesterase by blocking the site of attachment of the enzyme to the substrate by strong attachment to both the anionic and esteratic sites. There is consequent accumulation of actylcholine, producing symptoms.

They are water soluble and gain entrance through the gastrointestinal, respiratory and skin surfaces. The symptoms occur rapidly but are reversible, recovery occurring within minutes or hours. No deaths have been reported.

Symptoms & Signs

They are those of excess acetylcholine and consist of:—

Tremors and convulsive seizures of muscles.

Increased secretion of bronchial lacrimal salivary and sweat glands.

Diarrhoea and vomiting

Bradycardia

Miosis

Treatment

(1) Stomach wash

(2) Treat with atropine as in the case of organophosphate poisoning, **Pralidoxime is not indicated in carbamate poisoning** as it is not effective in reactivating carbamylated acetylcholinesterase although highly effeffctive in the case of phosphorylated acetylcholinesterase.

(4) PYRETHRUM AND SYNTHETIC PYRETHROIDS

Pyrethrum is one of the oldest insecticides known to man. The source of the material is the flower of the pyrethrum plant Chrysanthemum cinerariaefolium. The active constituent are the esters called pyrethrins and cinerins.

Pyrethrum is an important constituent of **Shelltox** which contains in addition, **piperonyl butoxide** 0.1%. The latter has a synergistic action enhancing the insecticidal properties especially of pyrethrins and increase their stability. Pyrethrins have low toxicity. Shelltox contains 0.2% pyrethrins so that serious poisoning is highly improbable. Synergists like piperonyl butoxide possess even lower acute toxicities in laboratory animals than do pyrethrins. Therefore severe poisoning from the active ingredients is rare and is more often due to kerosene the common solvent in pyrethrins sprays. **Shelltox with Vapona** contains in addition the organophosphate dichlorvos. **Shelltox Red** contains the organophosphate chlorfenvinphos instead of dichlorvos.

Symptoms

Symptoms included incoordination, tremors, muscular paralysis and rarely respiratory paralysis. Piperonyl butoxide may in large doses cause vomiting and diarrhoea. Symptoms of kerosene poisoning may be present.

(5) MISCELLANEOUS PESTICIDES

The only one which is relatively toxic is **endosulfan.** There is however no accumulation in the body due to its rapid decomposition.

Treatment

Vomiting should be induced if swallowed. The effects of endosulfan are similar to those of chlorinated hydrocarbons and similar methods of treatment should be followed.

RODENTICIDES

1. Zinc Phosphide

It is a crystalline grey powder with a pungent phosphorus like odour. It releases phosphine on contact with water. Rodents seem to like the rotten fish odour and readily accept this type of bait.

Symptoms

Inhalation of zinc phosphide dust is followed in several hours by vomiting, diarrhoea, cyanosis, rapid pulse, fever and irritability. Ingestion causes nausea and vomiting.

Treatment

Treatment is mainly symptomatic for poisoning from inhalation. If it is ingested, gastric lavage is required and potassium permanganate 1:5000 solution should be used to oxidise any remaining phosphide present.

2. Thallium

Thallium sulphate 1% is an effective rodenticide. The mean lethal dose for an adult is about 1g. Single doses of 4mg/kg body weight have caused toxic symptoms in children.

Symptoms

Ingestion is followed after a latent period of 12—24 hours by a severe gastroenteritis characterised by paroxysmal abdominal pain, nausea, vomiting and diarrhoea which may be bloody. Delirium, convulsions and coma may appear rapidly but more often the acute reaction subsides followed by the gradual development of mild gastrointestinal disturbances, polyneuritis, convulsions, tremors, encephalopathy, skin eruptions and hepatorenal injury. Signs of pulmonary oedema and pneumonia may precede death from respiratory failure. A prominent delayed effect is alopecia. Diagnosis can be confirmed by finding thallium in the urine.

Treatment

Immediate measures include administration of an emetic or gastric lavage. Activated charcoal in a dose of 0.5g/kg twice daily for 5 days absorbs thallium and prevents absorption but this is not substantial.

Potassium chloride orally in a dose of 3-5g for 5—10 days is also given, as potassium salts promote urinary thallium excretion but only to a limited extent.

Recently **Prussian blue** (Potassium Ferric hexacyanoferate II) has been advocated for oral use the aim being to replace the potassium ion with thallium in the lattice of the prussian blue molecule rendering the thallium less absorbable. There is experimental evidence that thallium is much more strongly bound to prussian blue than to activated charcoal. Prussian blue is not toxic as it is not absorbed. It is given in a dose of 10g twice daily for 10—14 days.

Some recommend administration by duodenal tube because of pyloric spasm and gastric dilation in such patients.

Peritoneal dialysis is useful as substantial amounts appear in the peritoneal dialysate. **Haemodialysis** is of little value.

Dimercaprol (BAL) is used but there is no clear evidence of its value.

Calcium disodium acetate has been found disappointing in a few cases where it was used.

Other measures which have been recommended include i.v. injection of 0.3-1.0g. of sodium iodide in an attempt to precipitate insoluble thallium iodide in the tissues followed by 12-20ml of 3% sodium thiosulphate i.v. later, to mobilize thallium in small quantities for excretion. Proof of efficacy of this measure has not been clearly established.

Benzhexol is valuable in controlling distressing tremors and severe ataxia that accompanies thallium intoxication.

3. Coumarin Derivatives

These are fairly safe where humans are concerned. A single large dose is rarely if ever fatal. A lethal dose is considered to be over 100mg of the pure compound which is equal to about one pound of rat bait.

Symptoms

The main manifestation of poisoning is bleeding. The toxicity of a single large dose is slight and oniy when it is taken over a period of several days or on reepated ingestion that the bleeding tendency appears.

There may be epistaxis, massive purpura, petechiae at the knees and elbows, weakness, pallor, haematuria and rectal bleeding.

Treatment

Perform gastric lavage if large amounts (about ¼lb.) has been swallowed and if no more than 2 or 3 hours have elapsed. A cathartic such as an ounce of sodium or magnesium sulphate in 250ml of water should be given. If bleeding occurs or if the prothrombin time is prolonged more than twice normal give vitamin K. If the haemoglobin level is low and bleeding severe, fresh whole blood should be given.

SNAIL BAIT

Metaldehyde is the only substance in this group.

The effects of metaldehyde are due to it being changed to acetaldehyde.

Clinical Manifestations

Ingestion causes nausea, abdominal pain, severe vomiting, the face becomes flushed, there is rise of temperature, muscular rigidity, twitching and choreiform movements. In severe poisoning, convulsions, coma and death from respiratory failure may occur.

Treatment

Treatment consists of immediate gastric lavage and symptomatic therapy.

HERBICIDES (WEEDICIDES)

Carbamates

They are of very low toxicity and symptoms and treatment have been discussed under carbamates in the section on insecticides.

Acetamides

Those substances are considered to be plant hormones. Human toxicity is low and deaths occur only rarely.

Acetanilides

Human toxicity is low and there have been hardly any cases of poisoning reported.

Clinical manifestations

Mild symptoms of ingesting these herbicides are those of irritation of the throat and gastrointestinal tract. Severe poisoning is characterised by coma, convulsions, muscle twitching, weakness and urinary incontinence.

Local effects include irritation of mucous membranes and eyes.

Recovery is usually complete in 24 hours. Prolonged exposure of the skin to 2.4D has been reported to cause a peripheral neuropathy.

Treatment

Treatment consists in removing any poison left in the stomach by gastric lavage and symptomatic treatment of toxic manifestations.

Triazines

They produce only mild symptoms and no deaths have been reported. No specific treatment.

Bipyridyls

Paraquat "Gramoxone"

Paraquat is the most important member of a group of herbicides called the bipyridyls. It has the property of killing all green tissue with which it comes into contact while it is rendered inactive by contact with the soil so that the land can be immediately resown.

Paraquat is available as a reddish brown liquid as a 20% W/V solution in the undiluted state.

Poisoning and deaths have only resulted from the ingestion of the concentrated liquid. The proportion of paraquat absorbed from the gut is unknown. An oral dose of about 15ml. or ½fl. oz. of the liquid concentrate is likely to be fatal. There is very little absorption from the diluted spray material through the intact skin. Once absorbed from the gut it is preferentially accumulated by lung tissue.

Symptoms

The immediate effects are due to local irritation and consist of vomiting, abdominal discomfort and diarrhoea which may be bloody. There is usually in addition, soreness of the mouth and throat and dysphagia. With very large dose the central nervous system may be involved giving rise to tremors and convulsions.

Manifestations of kidney and liver damage such as oliguria, anuria, elevated blood area and jaundice due to

centrilobular necrosis of the liver may appear within 2 or 3 days of ingestion. The severity of the damage depends on the amount absorbed.

Pulmonary symptoms may develop gradually a few days later. Those include dyspnoea with pulmonary oedema or haemorrhage progressing to marked pulmonary fibrosis and death from respiratory insufficiency. Abnormal lung function tests, may be the only abnormality in the stage before respiratory symptoms occur. Death is due to anoxia as a result of gross impairment of gaseous exchange processes. Electron microscopy reveals damage to the epithelial lining of the lung and there is biopsy evidence of a fibroblastic reaction obliterating alveolar spaces.

Local effects

Severe irritation and inflammation of the cornea and conjunctiva may follow accidental splashing of liquid paraquat concentrate in the eyes. These may be extensive denudation of the superficial layers of the corneal and conjunctival epithelium. Paraquat may produce inflammation and in severe cases blistering of the skin. Inhalation of the spray mist of a dust of paraquat causes epistaxis and soreness of the throat.

Treatment

1. Vomiting should be induced as soon as possible.

2. A gastric lavage should be given. This should be done with care because of possible oesophageal damage.

3. A large volume of a 30% suspension of Fullers Earth (Surrey finest powder grade) available from Laporte Industries Ltd., should be given together with a purgative every 24 hours for several days. Fullers Earth is available in 300G packets and should be added to one litre of water and shaken to form a suspension.

4. Of the treatments designed to help elimination of paraquat, forced diuresis is the treatment of choice

because paraquat is excreted in the urine unchanged, most of it in the first 24 hours.

5. Oxygen is definitely contraindicated or should be delayed as long as possible since it has been shown experimentally to enhance the toxicity of paraquat in the early phase of poisoning.

Immunosuppressive drugs such as corticosteroids have been used to prevent the lung reaction but with inconsistent results.[1]

Arsenic Compounds

Sodium Arsenite is widely used as a herbicide. Symptoms of poisoning depends on the amount ingested or inhaled. Ingestion of large amounts usually cause a severe gastroenteritis with a burning pain in the oesophagus, vomiting, copious, watery and blood diarrhoea. Circulatory collapse may follow, with generalized weakness. Cold and clammy skin, fall of blood pressure, convulsions and coma are the terminal signs and death may occur from circulatory failure.

Inhalation of arsenic compounds leads to dyspnoea, cyanosis, cough with frothy sputum and pulmonary oedema. Contact with the eyes causes conjunctivitis, lacrimation, pain and oedema of the eyes. Delayed effects include dermatitis, peripheral neuropathy, encephalitis, oliguria, anuria and jaundice.

Treatment

Attempts should be made to remove ingested arsenic by gastric lavage followed by a saline cathartic.

The specific antidote **dimercaprol** should be given. It is available as an injection in ampoules containing 100mg/ml in peanut oil in 3ml containers, and should be given by intramuscular injection. The dose is 2.5mg/kg body weight for mild poisoning and 3mg/kg for severe poisoning. In mild poisoning four such doses are given on the first two days, 2 doses on the third day and once daily thereafter for 10 days. In severe poisoning the recommended dose is given six times daily for the first 2 days, four doses on the third day and twice daily thereafter for 10 days.

Other measures include correction of dehydration to prevent circulatory collapse, oxygen therapy, and artificial respiration for respiratory failure.

Phenols and Nitrated Cresols

Pentachlorophenol (PCP) and dinitro-O-cresol (DONC) are the best known members of the group. They are used as pesticides as well as herbicides.

Clinical manifestations

Symptoms vary in severity depending on the rate of absorption. In mild poisoning there is appetite loss, nausea and vomiting, dizziness, headache restlessness, dyspnoea, feeling of constriction in the chest and excessive sweating. In severe cases there is high fever as they increase the rate of body metabolism. With DNOC there is yellow pigmentation of the selera.

Treatment

If the compound has been swallowed, vomiting should be induced. If absorption has occurred through the skin, the contaminated clothing should be removed and the skin thoroughly washed.

There is no specific antidote and treatment is symptomatic. If the temperature is elivated the patient should be sponged with ice cold water. Copious drinks should be given to replace fluids and oxygen should be administered. Where restlessness and agitation are present the patient should be sedated with diazepam.

Alcohol and atropine are contraindicated.

Propionic Acid Derivatives

There has been a report of acute haemolysis following ingestion of approx. 50ml of **propanil. The patient was** drowsy, dyspnoeic and cyanosed and eventually renal failure supervened. Treatment is symptomatic.

FUNGICIDES

These fungicides are considered to be essentially harmless to man and animals. They or their vehicle may cause bronchitis, conjunctivitis dermatitis or pharyngitis.

Therapy if any is supportive and symptomatic as for carbamate insecticides.

Copper preparations

Soluble and unsoluble copper salts are used extensively in agriculture or fungicides. These include copper oxychloride, copper oxide, colloidal copper, copper dust and copper sulphate.

Ingestion of copper salts causes vomiting and a haemorrhagic gastrititis with colicky abdominal pain and a diarrhoea with bloody stools. This may be followed by a severe headache, cold sweat and weak pulse. The patient may become somnolent comatose develop convulsions and death may result.

Those who survive may develop liver and kidney damage and haemolytic anaemia. Splashing of copper dust or salts on the eye can cause severe conjunctivitis, oedema of the lids and even ulceration of the cornea.

Prolonged inhalation of these compounds can produce severe congestion of the nasal mucosa and ulceration.

Treatment

A 1% solution of potassium ferrocyanide should be given quickly to precipitate the copper ferrocyanide. If this is not available, milk should be used and the stomach should then be cleansed by a gastric lavage.

Dithiocarbamates

These fungicides are considered to be relatively harmless to man and animals. They or their vehicle may cause bronchitis, conjunctivits, dermatitis or pharyngitis. Therapy is symptomatic.

Organomercuric Compounds

They are mainly used as seed dressings and acute poisoning due to consumption of treated seed grain has been reported. There have been a few instances in which people were poisoned by eating meat from domestic animals that had been fed treated grain. Poisoning has also been reported in Japan along the Minamata river by eating fish and shell fish. Mercury containing waste from a plastic factory had been diverted into the river about two years previously.

Chronic poisoning is not uncommon and is associated with repeated exposure in the manufacture. treating seeds. or eating of treated seeds.

Organomercuric compounds can be classified into 3 types and symptoms vary with the type.

(1) Alkylmercury compounds

e.g. methylmercury dicyanamide "Panogen"

Mild poisoning manifests as headache, paraesthesia of the tongue, lips, fingers and toes. These symptoms usually disappear gradually. In severe poisoning there is fine tremor, inco-ordination which if severe may result in inability to stand or carry out other voluntary movements. Muscle atrophy, flexure - contractures or generalized myoclonic movements may occur. Occasionally stuper and coma may occur. Recovery may occur slowly. Deaths from infections or pneumonia may occur in protracted cases.

(2) Alkoxy alkylmercury compounds

e.g. methoxyethyl mercuric chloride. "Ceresan Wet. Tillex., Agrosan, Unisol".

Symptoms of poisoning are loss of apetite, flatulence. diarrhoea, loss of weight, tiredness, and headache. Albuminuria often occurs and is accompanied by generalised oedema. CNS effects include numbness of the fingers and toes. weakness and ataxia.

(3) Arylmercury compounds

e.g. Phenylmercuric nitrate. "Antimucin" "Ceresan dry"

Symptoms are related to effects on the blood and include weakness due to anaemia and infections secondary to the leucopenia.

Treatment of Organomercuric Poisoning

Prompt removal from further exposure is important. If the material has been swallowed. the stomach should be washed out and either eggwhite or a 5% solution of sodium bicarbonate given. Treatment is essentially symptomatic.

Chlorobenzene Compounds

e.g. Hexachlorobenzene, Quintozene "Foame"

Prolonged consumption of grain treated with hexachlorobenzene has resulted in disease. There is blistering and epidermolysis of the skin particularly of the hands and face. Blisters may become infected or may heal leaving pigmented scars. Infections of the deeper tissues particularly of the hands may lead to suuurative arthritis and osteomyelitis. There may be increased pigmentation of the skin. Hepatomegaly, subnormal temperature, weight loss and muscle atrophy were common.

Recovery occurs if consumption of the treated grain is stopped. Mortality is about 10%.

Benzimidazole and Miscellaneous Compounds

e.g. Benomyl, Captan, Carboxim

They have a much lower acute toxicity than the other fungicides. They are eliminated relatively rapidly from the body and delayed effects are unknown.

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