

ANS - TOXICITIES

SOURCES OF MUSCARINIC AGONISTS

- Veterinary Medicines
- Pilocarpine, bethanecol
- Plants
- Fungal toxin in red, white & alsike clovers (toxin-slaframine)
 Mushrooms (Amanita muscaria)



ANS - TOXICITIES

SOURCES OF CHOLINESTERASE INHIBITORS

- Veterinary Medicines
- Physostigmine, neostigmine (NMB reversal)
- Plants
- Blue-green algae [anatoxin-a(s)]
- Cholinesterase Inhibitors • Organophosphates
- carbamates

ANS - TOXICITIES

SOURCES OF NICOTINIC AGONISTS

Toxicants with Nicotinic Effects

- Plants • Tobacco (Nicotiana)

- Poison Hemlock (Conium maculatum)
 Sophora, Laburnum
 Chemicals
- Levamisole (anthelmintic)
 - (annemanc)

(COD)



ORGANOPHOSPHORUS Toxicity

TOXICITY

- HIGHLY VARIABLE 100'S OF OP Compounds

- Rats

 \cdot LD 50 ranges from <1 mg/kg to > 4 grams/kg

- Birds and Fish very sensitive

ORGANOPHOSPHORUS Toxicity

MECHANISM OF ACTION:

 Inhibition of acetylcholinesterase at cholinergic receptors (IRREVERSIBLE when aged)
 Inhibition of acetylcholinesterase (RBCs)

ORGANOPHOSPHORUS Toxicity

CLINICAL SIGNS (MUSCARINIC):

- SLUDDE

- Salivation
 Lacrimation
- Lacrimation
 Urination
- Defaecation
- Dyspnoea
- Emesis

ORGANOPHOSPHORUS Toxicity

CLINICAL SIGNS (MUSCARINIC):
sweating
brady or tachy cardia depending on adrenaline release
pinpoint pupils (usually)
nasal discharge

ORGANOPHOSPHORUS Toxicity

• CLINICAL SIGNS (NICOTINIC)

- Tremors
- Weakness
- Paralysis

ORGANOPHOSPHORUS Toxicity

- · CLINICAL SIGNS (CNS):
- Nervousness
- Apprehension
- Ataxia
- Convulsions
- Coma
- Small animal: ± seizure, hyperactive,
- hyperreflexive
- Large animal: rarely seizure, ±
- hyperactive

ORGANOPHOSPHORUS Toxicity

- Muscarinic Signs: SLUDDE
- Nicotinic Signs: Muscle Tremors
- CNS: Anxiety, hyperactivity, clonictonic seizures

ORGANOPHOSPHORUS Toxicity

Intermediate Syndrome

- CATS and DOGS
- anorexia, diarrhoea, weakness,
- muscle tremors,
- abnormal posture and behaviour,
- clonic-tonic seizures

ORGANOPHOSPHORUS Induced Delayed Neuropathy OPIDN

Mechanism of Action

• Caused by inhibition of neuropathy target esterase (NTE)

- $\boldsymbol{\cdot}$ Loss of myelin and axons in the spinal cord
- Known Substances Causing OPIDN: •• leptofos, fenitrothion, trichlorfon,
- reptotos, tenitrotnion, tricn
 trichloronat and others

ORGANOPHOSPHORUS Toxicity

• DIAGNOSIS

- History

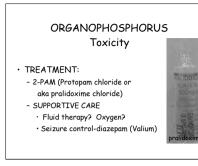
- Garlic odour?
- decreased acetylcholinesterase activity
- Test dose of 0.02 mg/kg atropine

ORGANOPHOSPHORUS Toxicity

- TREATMENT:
 DECONTAMINATE
- (dermal vs oral exposure) - Atropine sulphate • Part given Intravenously, • the rest Subcutaneously

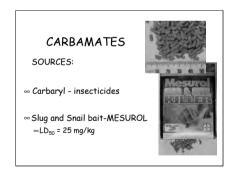
- 0.25-1 mg/kg







 TREATMENT:
 2-PAM (Protopam chloride or aka pralidoxime chloride)
 SUPPORTIVE CARE



CARBAMATES

· MECHANISM OF ACTION

SAME AS OPs except reversible binding to acetylcholinesterase

CARBAMATES

• CLINICAL SIGNS: - Similar to Organophosphorus compounds

CARBAMATES

 TREATMENT
 Similar to Organophosphorus compounds EXCEPT DO NOT USE 2-PAM
 2-PAM is not necessary and may be harmful

ANS Toxicities

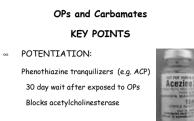
ORGANOPHOSPHATES DDX

- Amitraz
- Pyrethrins
- Cationic Surfactants e.g. benzalkonium chloride
- Garbage intoxication (endotoxins)

OPs and Carbamates

KEY POINTS

- ∞ SOURCES-numerous
- ∞ Do NOT accumulate (in fat) *
- ∞ RAPIDLY EXCRETED
- ∞ Comp Inhib ACETYLCHOLINESTERASE
- ∞ "AGING" OF OP-ENZYME complex







STAVE

POTENTIATION:

Levamisole:

-nicotinic stimulation

Aminoglycosides:

-blocks acetylcholinesterase

OPs and Carbamates

KEY POINTS

- ∞ Muscarinic: SLUDDE + other clinical signs
- $^{\infty}$ Nicotinic and CNS effects

 ∞ ATROPINE AND 2 - PAM (oxime)

(2-PAM is not necessary with carbamate poisoning)

OPIDN

- $\, \approx \,$ Cattle drenched with trichloronat
- ∞ Trichloronat pasture insecticide
- $\,\,\sim\,\,$ OPIDN Onset 2-3 weeks after drenching