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# Chapter 7: Poison treatment

In 1 collection

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<sup>1</sup>VulPro

1 Works for me This protocol is published without a DOI.

# VulPro

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#### **ABSTRACT**

This protocol outlines the steps to take in the event of a likely poisoning incident of multiple vultures.

#### ATTACHMENTS

Vulture\_Rehabilitation\_Man ual\_Version\_2.0\_.pdf

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Vulture Rehabilitation Manual

#### **KEYWORDS**

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**Vulture Rehabilitation Manual** 

## ATTACHMENTS

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#### Agrochemical poisoning

#### Possible drug culprits:

• DDT is not likely to be a culprit, as large quantities would have to be ingested to have a lethal or sublethal clinical effect

## Organophosphates:

- Fenthion
- Methamidophos
- Diazinon
- Chlorfenvinphos
- Fenamiphos
- Cadusafos

#### Carbamates:

- Aldicarb
- Carbofuran (granular and liquid)
- Methomyl

#### Clinical signs:

In general, if a bird has survived a poisoning incident and is able to make it to a rehabilitator, these are the clinical signs most likely to be seen:

- Incoordination, stumbling, falling over and an inability to balance (ataxia)
- High- or goose-stepping (over-exaggerated lifting of legs when walking)
- Seizures (severe)

#### Organophosphate (OP) poisoning

More specific characteristics of each drug reaction include:

- Fenthion relatively slow onset of about a half hour, pupil constriction (miosis), vomiting, tremors, paralysis.
  Prognosis is very poor. Birds may fly for half an hour before toxic effects ground them. Recovery can take as long as 8 months.
- 2. Methamidophos rapid onset of symptoms within 5 minutes, prognosis very poor. Birds are typically not found further than 50m from the source.
- 3. Diazinon very rapid onset of symptoms within 3 to 5 minutes, prognosis fairly good with no long term effects. Birds will be found within 100m of the source.
- 4. Chlorfenvinphos rapid onset of symptoms within 5 minutes, prognosis poor. No long term effects.
- 5. Fenamiphos rapid onset of symptoms, prognosis very poor with virtually no survivors. Birds found within 100m of source.
- 6. Cadusafos slow onset of symptoms, up to half an hour after exposure. Prognosis very poor and survival unlikely. Birds can move very far from the source.

It is important to note, recovered birds will occasionally re-present symptoms long after the initial treatment of the poisoning event. When the bird initially ingests the poison, the body distributes a proportion of the poison into fat reserves. Should the bird lose weight post-recovery, these fat stores are utilised and the poison is released back into the bloodstream. Typically, the symptoms will be mild as the volume of poison is much smaller.

#### Carbamate poisoning

- 1. Aldicarb immediate onset of symptoms with pupil constriction, paralysis, some vomiting, tremors and hypothermia. Prognosis extremely poor. Birds found within a few metres of source.
- 2. Carbofuran (granular) onset of symptoms from 5 to 30 minutes after exposure. Usually sub-lethal concentrations are ingested so prognosis is less guarded. Symptoms are like those seen from exposure to Aldicarb. Birds may get back to their nests where they and their chicks die.
- 3. Carbofuran (liquid) immediate onset of symptoms with very poor prognosis. Birds found at the source.
- 4. Methomyl immediate onset of symptoms, prognosis extremely poor with no survival, birds found at source.

## **NSAID** poisoning

## Possible drug culprits:

All NSAIDs except Meloxicam (Meloxicam®, Metacam®, Mobic®) are considered toxic to vultures. The degree of toxicity varies between different NSAIDs, but every individual vulture reacts differently. Some birds may react badly

to one drug while others appear unharmed.

The following drugs are proven to be toxic to vultures, but all others (except Meloxicam) cannot be ruled out:

- Diclofenac Sodium (Diclofenac®, Voltaren®)
- Ketoprofen (Ketofen®)
- Phenylbutazone (Tomanol®, Phenylarthite®, Equipa-lazone®, and Fenylbutazone®)
- Flunixin (Finadyne®, Cronyxin®, Pyroflam®, Hexasol®)
- Vedaprofen (Qaudrisol®)
- Carprofen (Rimdayl Aquous®)

#### Clinical signs:

- Dehydration ranging from slight to severe
- Generalised signs of weakness
- Drooping head
- Periods of 'zoning out' but may regain normal stance and consciousness when interacting with people.
- Depressed appearance
- Wings held slightly out from the body.

#### Lead poisoning

Lead poisoning, subsequent to the ingestion of spent ammunition fragments by scavenging birds is a long recognised complication in wildlife free living populations and in admitted casualty birds. Incidence varies depending on global and local location, species of scavenger and time of year, varying from: 31% above normal levels in white backed vultures in Botswana, 35% in Griffon vultures in Spain and 12% in Cape vultures in South Africa.

Levels below  $10\mu g/dl$  are considered normal, levels above  $40\mu g/dl$  may lead to clinical signs associated with toxicity, but even levels above  $10\mu g/dl$  have been postulated to cause some deleterious effects. Naidoo et al. (2012) investigated a captive Cape Vulture breeding colony, exposed to high lead concentrations within their enclosures at the South African National Zoological Gardens in Pretoria. In this case-study, they described signs of decreased egg hatchability, embryonic death and abnormal chick development concurrent with whole blood lead concentrations ranging between 50 and 100  $\mu g/dl$  in the adult birds.

Research into swans showed that birds with lead levels of less than 25  $\mu$ g/dl, birds did not suffer any greater incidence of power cable collisions, birds with lead levels ranging 25-41  $\mu$ g/dl suffered a significantly higher collision incidence, whilst birds with levels over 41 $\mu$ g/dl, had a lower collision incidence. It was postulated that birds with the highest levels were too weak to fly, hence their lower collision incidence.

In view of this data, the authors encourage the routine collection and testing of blood lead levels from all admitted vultures, with chelation therapy being administered to all birds with levels in excess of 10µg/dl.

#### Possible Sources:

Vultures can ingest lead from several sources. The most common source is lead bullets used in hunting of game or euthanasia of farm stock. As bullets typically splinter on impact with any bones, even if the body part around the area of impact is removed, fragments of lead will be found a significant distance from the point of entry. Lead shotgun pellets will also scatter throughout a carcass.

Additional sources of lead include fishing tackle weights, refuse areas, old agricultural gates etc. In addition to direct ingestion of fragments, lead can leach into water sources.

#### Symptoms:

- Seizures and neurological issues
- Incoordination, in flight or when walking
- Paralysis (in severe cases), often sitting on the ground, with their inter-tarsal joints on the floor
- Vultures are often emaciated and malnourished, as they are unable to adequately forage and feed
- Sudden onset of acute blindness

*Diagnosis:* this should be based on the presence of lead particles in the gastrointestinal tract on x-ray, or an elevated blood lead level on testing.

Blood lead levels in vulture blood is normal if:- <10ug/dl (= <0.48 umol/l) Blood levels between 10-20 ug/dl (0.48 – 0.96umol/l) indicates exposure which is unlikely to be of clinical significance.

Any vulture with a lead level above 20 ug/dl (= 0,96umol/l), should receive chelation (EDTA) therapy.

Blood lead levels are often elevated in vultures in the absence of particulate lead in the gut, which is consequent to the consumption of ballistic fragments (i.e. fragments of hunters' bullets).

Even in the absence of any nervous signs consistent with lead poisoning, there is now very good evidence of elevated lead levels in many vultures presented with trauma, power line injuries and other illnesses, which are considered to be associated with long term low levels of lead toxicosis.

ABSTRACT

This protocol outlines the steps to take in the event of a likely poisoning incident of multiple vultures.

- 1 If you attend the site of a likely poisoning incident, i.e. several birds dead or ill around a carcass, consider and respect the site as a 'crime scene'. It is imperative to avoid any damage to evidence as this is vital information in bringing any future legal action against a suspect.
- 2 Contact the local law enforcement authorities in the area of the poisoning event.
- If any vultures remain alive, treat these as a priority. The observed clinical signs will vary with respect to the type of poisoning. The three main categories of poisonings are: agrochemicals, non-steroidal anti-inflammatory drugs (NSAIDs) and lead.

## Treatment of Organophosphate Poisoning

4 Rapid availability and administration of the correct antidote is imperative. If not treated correctly within 24-48 hours, therapy is often ineffective.



It is important to note, recovered birds will occasionally re-present symptoms long after the initial treatment of the poisoning event. When the bird initially ingests the poison, the body distributes a proportion of the poison into fat reserves. Should the bird lose weight post-recovery, these fat stores are utilised and the poison is released back into the bloodstream. Typically, the symptoms will be mild as the volume of poison is much smaller.

- Antidote: treat with 2 PAM (Pralidoxime Chloride), at 50 mg/kg once intravenously slowly over a © 00:05:00 © 00:10:00 period, repeated after © 06:00:00 if necessary, then every © 24:00:00 as required.
- 6 Provide supportive care: sling as necessary, control seizures, fluid and nutritional support.
- 7 If the poisoning is OP and treatment is not started within 24 hours, therapy may make clinical signs worse. In this event, stop treatment.

#### Treatment of Carbamate Poisoning

- 8 Antidote: treat with Atropine 2 mg/kg by intramuscular, or better still, intravenous injection. If the bird responds and later deteriorates, treatments can be repeated as often as necessary. If there is no improvement after © 00:05:00, but saliva is still present in the mouth, repeat the treatment at 4mg/kg.
- 9 For as long as there is no improvement and yet also no deterioration and still saliva in the mouth, keep repeating the dose, doubling each time, © 00:05:00 between each dose.
- 10 Maintain treatment until post-treatment deteriorations cease.
- 11 Provide supportive care: sling as necessary, control seizures, fluid and nutritional support.

## Treatment of Unidentified OP/Carbamate Poisoning

- Antidote: Atropine at 2 mg/kg plus 2 PAM at 25 mg/kg, repeated as necessary (i.e. if clinical signs improve then deteriorate again). Typically treatment will be required every **© 02:00:00 © 04:00:00**.
- 13 If the poisoning is organophosphate and treatment has not started within 24 hours, therapy may make clinical signs worse. In this event, stop PAM treatment, but maintain Atropine. In such cases, then dose atropine as above under 'Carbamate' heading.

## Treatment of NSAID Poisoning

- There is no specific or reliably effective treatment. Give the bird IV fluid therapy via a drip at twice maintenance rates (i.e. give 100 ml/kg/day). This is critical to reduce the severe dehydration.
- 15 Reduce uric acid levels by administering Allopurinol at 30 mg/kg by mouth twice daily until recovery.
  - Nothing will reverse the kidney damage caused by the toxic effect.
- 16 If the bird is showing significant clinical signs, then death is typically inevitable and euthanasia is indicated.

## Treatment of Lead Poisoning

Administer EDTA – 35 mg/kg twice daily by intramuscular or intravenous injection for 5 days, after any particulate lead has been removed from the gastro-intestinal tract.

- 18 If lead is shown to be in the gut, then stabilise initially for 24 hours with intensive fluid and chelation therapy. A veterinary surgeon may then remove the lead from the proventriculus, under anaesthetic, with an entubated patent, the bird is tied to a tilted table, with the body above the head at 45 degrees, the lead particles are removed by gastric lavage.
- 19 Do not continue chelation therapy beyond five days after the particles are removed, as it can cause renal damage.
- 20 If nervous signs persist, then perform a repeat test of blood lead level 7 days after the cessation of EDTA therapy. If it remains elevated, then a repeat of 5 days of therapy is justified.
- Important: in all lead poisoning cases, administer fluid therapy (preferably via IV drip) to reduce the likelihood of kidney failure and to concurrently treat any seizure activity.