Herbicides

A. Type of Herbicide

1. Inorganic herbicides: arsenicals & Chlorates (used infrequently)
2. Organic synthetic herbicides (commonly used)

B. Applications

# Post-emergent herbicides
# Pre-emergent herbicides

1. Agriculture: row crops, small grain crops, forages
2. Commercial use: residential lawns, industrial sites, road crew

C. Exposure

1. Environmental residues
   a. Excessive application rates or use outside of label restrictions
   b. Urban runoff
2. Inadvertent consumption of concentrated or mixed herbicide
3. Grazing freshly sprayed pasture or eating treated hay
D. Diagnosis

1. Cannot be diagnosed based on circumstantial evidence alone; evidence of substantial and repeated exposure is necessary.
2. Efforts to rule out other diagnoses
3. Chemical analysis

Phenoxy Herbicides (2,4-D, MCPA, silvex)

A. Chemical structure: are chlorinated phenoxy derivatives of fatty acids

B. Exposure

1. Dogs with access to freshly sprayed lawns
2. Drink concentrates or tank mixes
3. Potentiate the toxic effects of some plants
   a. increase the nitrate content of certain plants
   b. increase the palatability of certain toxic plants

C. Toxicokinetics

1. Absorption: readily from the GI. Dermal absorption is slower and less complete.
2. Distribution: rapidly distributed to the liver, kidney and brain
3. Metabolism: metabolized by hydrolysis
4. Excretion: is excreted unchanged in the urine
   a. Canine excrete phenoxy herbicides relatively slowly
   b. Excretion is markedly enhanced by using alkaline diuresis
   c. half life: about 18 hrs.
D. Mechanism of toxic

1. Depress ribonuclease synthesis, uncouple oxidative phosphorylation & increase the number of hepatic peroxisomes

2. In dogs may directly affect muscle membranes

E. Toxicity

1. Acute toxicity
   a. 200 mg/kg in cattle & 100 mg/kg in pigs
   b. Oral LD₅₀: 500 mg/kg in rats & 100 mg/kg in dogs
   c. Multiple lethal dosage in dogs is 25 mg/kg for 6 days

2. Chronic toxicity: no evidence

F. Diagnosis

1. Clinical sign

<table>
<thead>
<tr>
<th>Species</th>
<th>Gastrointestinal</th>
<th>Signs</th>
<th>Neuromuscular</th>
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</thead>
<tbody>
<tr>
<td>Cattle</td>
<td>Anemia, rumen atony, diarrhea, ulceration of oral mucosa, biot, rumen stasis</td>
<td>Depression, muscle weakness</td>
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<tr>
<td>Swine</td>
<td>Vomiting, diarrhea, salivation</td>
<td>Tremors, ataxia, weakness</td>
<td></td>
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<tr>
<td>Dogs</td>
<td>Vomiting, diarrhea, bloody feces</td>
<td>Mydriasis, ataxia, posterior weakness, periodic tonic spasms only at high dosages, altered electromyogram with increased insertion activity</td>
<td></td>
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</tbody>
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2. Lab.
   a. Alkaline phosphatase, Lactate dehydrogenase, and creatinine phosphokinase are increased.
   b. Chemical analysis of forages, urine, or renal tissue
G. Treatment

1. Detoxification
   a. Dermal exposure
   b. Ingestion: Activated charcoal

2. Supportive therapy
   a. Symptoms of diarrhea & rumen atony
   b. IV fluid administration (promote diuresis)
   c. High quality diet & treatment with rumen inoculants

Dipyridyl Herbicides (Paraquat, Diquat)

A. Characteristics

1. Nonvolatile, water soluble and stable
2. Degradable in sunlight
3. Rapidly and completely inactivated in soil by bacterial activity
4. Strongly bound to clays in soil

B. Sources

1. Concentrated forms (5-20% solution) for agricultural use & dilute forms for home lawns & gardens
2. Contact (desiccant) herbicide
C. Exposure
1. Consume concentrates or tank mixes
2. Malicious poisoning of dogs

D. Toxicokinetics
1. Absorption is limited
   a. Less than 20% is absorbed when ingested
   b. Less than 10% is absorbed through the skin
2. Distribution: concentrates in the lung (10 times higher than other tissue)
3. Metabolism: is minimal
4. Excretion: in the urine as unmetabolized

E. Mechanism
are reduced by NADPH. Electron transfer occurs from paraquat to oxygen
1. Singlet oxygen reacts with lipids
2. Lipid free radicals are generated
3. The lungs are most affected

F. Toxicity
1. Acute toxicity
   a. Oral LD50 ranges from 25-75 mg/kg
   b. Dermal LD50 is approximately twice the oral LD50
2. Chronic toxicity: dogs consuming 170 ppm dietary paraquat for 2 months developed intoxication
3. Factor affecting toxicity: Clay soil limit paraquat’s toxicity
G. Diagnosis

1. Clinical sign
   a. Acute toxicosis
      1. Early signs: vomiting, depression, ataxia, dyspnea & seizure
      2. Respiratory sign (delayed for 2-7 days): tachypnea, dyspnea, moist rales & cyanosis

2. Laboratory
   a. Radiographic changes in the lungs
   b. Analysis of urine, herbicide solutions or suspect baits

H. Treatment

a. Detoxification
   1. Emetics
   2. Bentonite or Fuller’s earth is the preferred adsorbent (activated charcoal)
   3. Saline cathartics

b. Supportive
   1. Assisted ventilation (oxygen is contraindicated)
   2. Renal function should be monitored

c. Specific antidotes are not available.
   1. Biochemical antagonists
      - Superoxide dismutase
      - Acetylcysteine
      - Ascorbic acid
      - Niacin & riboflavin
Fungicides

A. Application
1. Protect tubers, fruits & vegetable
2. At the time of planting
3. Applied directly to ornamentals, trees, field crops, & cereals

B. Potential toxicosis
Flagrant misuse, accidents and carelessness

C. Toxicity: little information is available for farm animals and pets

D. Diagnosis
1. Clinical sign: not specific (anorexia, depression, weakness, and diarrhea)
2. Laboratory: Chemical analysis of contaminated feeds and forages
E. Treatment: Detoxification and supportive therapy

Pentachlorophenol

A. Application: Fungicide, herbicide, bactericide & wood preservative

B. Exposure
   1. Some treated wood is available
   2. accidental

C. Source
   is synthesized by the successive chlorination of phenol.

D. Kinetics
   1. Absorption is rapid
   2. Metabolism is conjugated with glucuronic acid
   3. Excretion in the urine

E. Mechanism
   1. Uncouples oxidative phosphorylation (fever is a reflection of energy lost as heat instead of stored as high energy phosphate bond)
   2. Direct irritant
   3. Neurotoxic (cellular energy deficit & hyperglycemia)
F. Toxicity
1. LD₅₀ 100-200 mg/kg
2. Subacute toxicity: occurs in cattle at dosage 15-20 mg/kg
3. Chronic toxicity ranges form 40-70 mg/kg

G. Diagnosis
1. Clinical sign
   a. Acute toxicity: restlessness, fever, elevated BP, hyperperistalsis, weakness, seizure and collapse.
   b. Chronic: weight loss, progressive anemia, low thyroxine levels and decreased milk production.
2. Lab.: analysis blood & urine

H. Treatment
1. Detoxification
2. Supportive
   a. Hyperthermia
   b. Balanced electrolyte solutions without glucose