BASICS

Rodenticide toxicity can be caused by any of several types of rodent poisons that fall into two general categories, anticoagulants and non-anticoagulants. **ANTICOAGULANT RODENTICIDES** work by interfering with the activation of Vitamin K, a critical component in the production of blood clotting factors in the liver. **NON-ANTICOAGULANT RODENTICIDES** vary in their mechanism of action and include bromethalin, strychnine, cholecalciferol, and zinc phosphide.

Rodenticides are **TOXIC** to many species of birds and mammals including pets, farm animals, and wildlife species. The time between **EXPOSURE AND DEVELOPMENT** of clinical signs is dependent upon the specific chemical and amount consumed.

Ingestion of a significant amount of **ANTICOAGULANT** rodenticides results in interference with blood coagulation and spontaneous bleeding. Specific **CLINICAL SIGNS** can include widespread bruising, bleeding into body cavities, and blood in the urine or feces; if the bleeding is sudden and significant, then cardiovascular shock and death can result. Bleeding can occur **INTERNALLY OR EXTERNALLY** and can affect any part of the body.

**NON-ANTICOAGULANT** rodenticide toxicity symptoms are more variable and are dependent on the chemical and dose. The **CLINICAL SIGNS** include rapid onset of seizures, muscle tremors, limb weakness, ataxia, neurologic signs, respiratory paralysis, anorexia, nausea, vomiting, diarrhea, and lethargy.

There are currently **NO BLOOD TESTS FOR BIRDS** for anticoagulant rodenticide exposure.

Diagnosis of non-anticoagulant rodenticide toxicity is based on detection of the chemical in the **DIGESTIVE SYSTEM OR TISSUES** of the animal.

Vitamin K is used to treat anticoagulant rodenticide intoxication and help restore normal coagulation. The treatment for non-anticoagulant rodenticide poisoning is typically only supportive care.
twitching, stiffened neck, dilated pupils, seizures, and hypersensitivity. Animals can have severe muscle rigidity and seizures which increase in frequency. Eventually exhaustion or respiratory paralysis can lead to death.

**CHOLECALCIFEROL** (Vitamin D3) increases serum phosphate and calcium and may lead to kidney failure, cardiac abnormalities, and hypertension, as well as nonspecific clinical signs. Calcification of soft tissues because of the high calcium and phosphorus levels is possible.

**ZINC PHOSPHIDE** releases phosphine gas into the bloodstream following contact of the bait with stomach acid. Nonspecific clinical signs can include anorexia, nausea, vomiting with or without blood, diarrhea, and lethargy.

**DIAGNOSIS** Anticoagulant rodenticide toxicity is suspected when an animal has signs of spontaneous hemorrhage and rodenticide in the blood or liver. Because of the delay between ingestion and clinical signs, the bait or poisoned rodent is usually not present in the digestive tract by the time the animal is sick.

In **DOMESTIC ANIMALS**, a special blood test called a coagulation panel will show prolonged prothrombin time (PT), activated partial thromboplastin time (APTT), or thrombin time along with normal fibrinogen, fibrin degradation products, and platelet counts.

Laboratory detection of anticoagulant in the blood, liver, or kidney **DOES NOT NECESSARILY CONFIRM** rodenticide poisoning as cause of death in wild animals because many live wild animals have exposure to these compounds.

Bromethalin poisoning can be diagnosed based on history, clinical signs, and the presence of the chemical in liver, kidney, brain, or fat. Zinc phosphide poisoning is difficult to diagnose because phosphine gas doesn’t remain long in the stomach.

**TREATMENT** Because the second generation anticoagulant rodenticides can be very long acting, treatment may be required for weeks to months.

When ingestion is recent, stomach flushing or induction of vomiting followed by activated charcoal administration can help prevent more absorption. Cholecalciferol treatment may include medications to increase calcium excretion in the urine and help prevent absorption from the digestive tract.