COCAINE TOXICOSIS
Don't Get "Snowed"

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Cocaine that has been left out is more likely to be sniffed or ingested by dogs because of their indiscriminate eating habits.

Any amount of cocaine can be toxic to an animal, and animals from many walks of life are at risk for exposure to this drug.[1] Animals whose owners keep the drug in their home are at greatest risk, but there have also been cases in which police dogs were exposed or the drug was brought into a home without the pet owner's knowledge. Certain health food teas are also an exposure risk.[2] Therefore, veterinary technicians should be prepared to recognize and treat cocaine toxicosis in all animals.

Cocaine has been used for centuries as a stimulant and is currently well known for its illicit use (it is a schedule II drug). Cocaine is a natural alkaloid from the *Erythroxylon coca* plant. It is processed from the leaves of the plant into cocaine hydrochloride. Street cocaine is diluted (i.e., "cut"; often with mannitol, lactose, sucrose, cornstarch, inositol) many times before it reaches the user. Other common adulterants include lidocaine, procaine, tetracaine, caffeine, amphetamine, and quinine. The amount of adulterant in cocaine directly affects the toxic potential of the drug by either diluting it or by increasing the effects. Cocaine hydrochloride is commonly reprocessed to form the cocaine alkaloid (i.e., freebase).[3-5] Cocaine has many street names that technicians should know in case owners use the terms.

In addition to its pharmaceutical and illicit uses, cocaine is also found in health food stores in such products as medicinal teas made from decocanized tea leaves. The concentration of cocaine in these leaves can be identical to that in untreated cultivated coca leaves.[2]

Most cases of cocaine exposure in animals occur through ingestion of illicit cocaine. Dogs seem to be more likely than cats to ingest cocaine, probably a result of dogs' indiscriminate eating habits. Any amount of cocaine in a companion animal may result in toxicosis.[1] As little as 20 mg of the drug has caused death in humans.[6]

MECHANISM OF ACTION

Cocaine is rapidly absorbed when inhaled or ingested. Plasma levels can be detected at 15 and 30 minutes, respectively. Cocaine's powerful central nervous system (CNS) stimulant effect is probably due to alteration of nerve transmission within the CNS. By increasing the amount of the neurotransmitter dopamine within nerve synapses, cocaine causes increased excitation of postsynaptic neurons. Cocaine is broken down within both the liver and the plasma and is excreted primarily in the urine.[2]

CLINICAL SIGNS

An animal may develop signs of cocaine toxicosis in as little as 30 minutes after exposure. Animals often present initially with CNS excitement

Common Names for Cocaine[2,a]

Baseball[b]	Her
Bernice	Leaf
Bernies	Nose candy
Blow	Rich man's drug
C	Rock
Champagne	Snow
Coke	Speedball[c]
Crack	Stardust
Dama blanca	White girl
Flake	White lady
Girl	
Gold dust	

[a]These names are important to know for communicating with clients.
[b]Also used for freebase.
[c]Also used for heroin.

Toxicology Brief is contributed by veterinary technicians at the American Society for the Prevention of Cruelty to Animals–Animal Poison Control Center, 1717 S. Philo Rd., Suite 36, Urbana, IL 61802; hotline: 888-4ANI-HELP (888-426-4435) or 900-443-0000 (a $45 consultation fee is charged to the caller's telephone bill); email: sharont@napcc.aspca.org (for nonemergency information only); Web site: **www.apcc.aspca.org**.

Medical Use of Cocaine

Cocaine has been beneficial in human and veterinary medicine as a topical and local anesthetic as well as a mydriatic, although it is no longer commonly prescribed. It is a controlled substance used mainly by otolaryngologists, plastic surgeons, and emergency room physicians for its local anesthetic and vasoconstrictor effects in nasal surgery and emergency nasotracheal intubation.[3–5] It is available in a 1% to 4% solution as an eye anesthetic or in a 10% to 20% solution as a nasopharyngeal anesthetic.[6]

(i.e., hyperactivity, erratic behavior, seizures). Other signs may include vomiting, hypersalivation, hyperthermia, tachyarrhythmias, cardiac arrest, respiratory arrest, and death. After the initial signs have subsided, pulmonary edema or pneumonia may develop.[1,2]

DETECTION

If veterinary staff or pet owners suspect cocaine toxicity, it may be beneficial to obtain an over-the-counter test kit. These tests are readily available in drug stores, inexpensive, and easy to conduct. They are designed to detect cocaine metabolites in the urine and may be able to do so for up to 3 days after exposure. Positive test results can be confirmed at a human hospital or diagnostic laboratory. Cocaine can be detected in plasma, stomach contents, and urine; therefore, any of these matrices may be submitted to an outside laboratory. Individual laboratories should be consulted for their preferences or restrictions.[2]

TREATMENT AND PROGNOSIS

Stabilizing the patient is of primary importance. Neurologic and cardiac signs should be addressed first. If these signs are not present, the staff should proceed with decontamination efforts. Decontamination should be implemented if or when the animal is in stable condition. Vomiting should be induced only if the animal is asymptomatic and the exposure was recent. Gastric lavage (after sedation) can be performed after a symptomatic animal has been stabilized. Activated charcoal does bind to cocaine, so its use in these exposures is quite valuable.[1]

Tremors and seizures can be treated with diazepam. Refractory seizures may require barbiturates. If the animal has extreme hyperexcitation or seizures, chlorpromazine or acepromazine may be preferred. Naloxone has proven to be beneficial for hyperactivity in humans.[7] Tachyarrhythmias may be controlled with a β-blocker such as propranolol.[1]

Supportive care, including IV fluids and thermoregulation, is necessary. It is also important to monitor the animal's acid–base status and correct it as necessary.[1]

With early decontamination and treatment, the prognosis for exposed animals is good. A poor prognosis is given when a significant dose is involved and signs are prolonged.

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