Human Medication Intoxications

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You have asked…

How do I treat common human medication intoxications in veterinary patients?

The expert says…

Because veterinary patients commonly present for human medication ingestion, veterinarians should have treatment plans for patients that have ingested these medications, including over-the-counter and prescription medications (eg, NSAIDs), new classes of antidepressant medications, amphetamines (used in the treatment of attention deficit-hyperactivity disorder [ADHD]), marijuana, and vitamin D.

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General Approach

As with any emergent patient, it is imperative to stabilize airway, breathing, and circulation first. Carefully questioning the owner often reveals the ingested toxin. Asymptomatic patients that present <4 hours after ingestion may benefit from emesis induction with apomorphine (dogs) or xylazine (cats). If the patient is stable, administration of activated charcoal may be warranted (for treatment agents see **Table 1**, next page). In addition, contraindications for emesis (**Contraindications for Inducing Emesis**, next page) should be considered before induction. When needed, gastric lavage should be performed in an anesthetized, intubated patient to provide comfort and limit risk for aspiration.

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Table 1 Agents Used for Toxic Exposures

Agent	*Dose/Schedule*	*Notes/Adverse Events*
20% IV lipid emulsion	1.5 mL/kg bolus, then 0.25 mg/kg/min for 30–60 min	Discontinue if lipemia develops or if treatment is ineffective
Acepromazine	0.05–0.2 mg/kg IV or IM	Titrate dose; hypotension
Activated charcoal	1–2 g/kg PO q8h	Enterohepatic recirculation or extended release warrants additional doses
Apomorphine	0.03 mg/kg IV (dogs)	Sedation
Chlorpromazine	0.5 mg/kg IV	Sedation
Cyproheptadine	1.1 mg/kg (dogs) q4–6h 2–4 mg total dose (cats) q4–6h	Oral or rectal dosing
Diazepam	0.25–0.5 mg/kg IV q1h; repeat up to 3 doses	Continuous infusion option
Maropitant	1 mg/kg SC	
Methocarbamol	50–100 mg/kg slow IV	Sedation
Midazolam	0.2–0.4 mg/kg IV or IM	Sedation
Misoprostol	2–5 µg/kg PO q8h	Wear gloves to handle (GI/uterine contractions, abortifacient possibility)
Ondansetron	0.6–1 mg/kg SC	
Pamidronate	1.3–2 mg/kg IV diluted over 2 hours	May repeat in 4–7 days
Pantoprazole	0.5–1 mg/kg slow IV q24h	Give diluted
Phenobarbital	4 mg/kg IV, 4 doses as needed	Loading dose; titrate based on level of sedation
Propofol	4–8 mg/kg induction IV	0.1–0.6 mg/kg/min infusion for continuous sedation; requires intubation
Propranolol	0.02–0.06 mg/kg slow IV 0.1–0.2 mg/kg PO q8h	Titrate dose to effect
Xylazine	0.4–1.1 mg/kg IM (cats)	Yohimbine reversal

Contraindications for Inducing Emesis

Patient Considerations	Toxin Considerations
Respiratory distress and/or disease	Corrosives
Seizures	Acid substance
Neurologic impairment	Alkali substance
Bradycardia	Sharp objects
Weakness	Potential danger to staff
Inability to protect airway	Significant vomiting already occurred
GI and/or abdominal disease	Underlying disease predisposing to aspiration (eg, laryngeal paralysis, megaesophagus)

General supportive care measures depend on clinical signs and may include antiemetic therapy, IV fluid therapy, blood pressure monitoring, oxygen therapy, and/or symptomatic supportive care.

NSAIDs

NSAIDs, which interrupt prostaglandin production by inhibiting cyclooxygenase, can result in decreased blood flow to renal and GI systems. Liver and platelet function may also be affected with large-quantity or chronic ingestion. Toxicosis severity and treatment duration may be impacted by dehydration; time until decontamination and/or treatment; and preexisting hepatic, renal, or GI disease. Vomiting and diarrhea, secondary to GI irritation, are the most common signs, but they may not be evident initially.

Accidental NSAID ingestion untreated for >8 hours can cause risk for acute renal failure and GI ulceration. When large doses are ingested, decontamination and ≥48 hours of twice-maintenance IV fluids are recommended to prevent renal injury. GI protectants (eg, misoprostol, H_2-blockers, proton pump inhibitors, sucralfate) may be beneficial. Misoprostol can help maintain GI blood flow. Proton pump inhibitors and H_2-blockers help reduce gastric acid while sucralfate coats the ulcerated region; both should be administered if a GI ulcer is suspected. Simple renal injury may respond to fluids and medications; oliguria and anuria require intensive monitoring and/or dialysis. Liver injury has been reported with some NSAID ingestion and may warrant further therapy. Baseline serum biochemistry and daily monitoring for at least 48 to 72 hours are recommended.

Antidepressant Medications

Most antidepressant medications work via reuptake inhibition or altered transport of serotonin, norepinephrine, or dopamine (**Table 2**). Many are rapidly absorbed and come in extended-release or long-acting formulas. Clinical signs of toxicosis can vary, depending on the medication; selective serotonin reuptake inhibitors (SSRIs) can result in serotonin syndrome, affecting the cardiovascular (eg, hyper- or hypotension, tachycardia), GI (eg, vomiting, diarrhea), and neurologic (eg, sedation, agitation, ataxia, tremors, seizures) systems.[1]

Standard decontamination is recommended but should be avoided in symptomatic patients. Supportive and symptomatic care are the mainstay of therapy. Fluid therapy does not enhance elimination but may help correct dehydration and acidosis; sodium bicarbonate may be given to patients with severe acidosis. Passive and/or active cooling may be necessary for hyperthermic patients. Propranolol or esmolol may be used for supraventricular tachyarrhythmias, norepinephrine or epinephrine for hypotension, and benzodiazepines for sedation or treatment of tremors or seizures.[1-3] Methocarbamol has been

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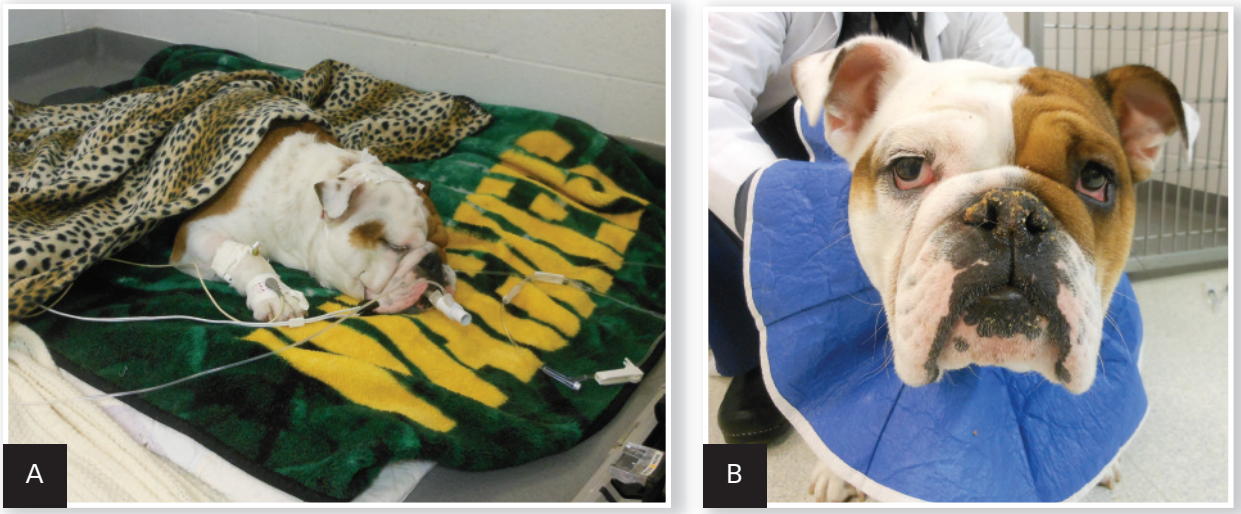
Table 2 **Common Antidepressant Medications**

Generic Name	*Common Brand Names*	*Mechanism*
Amitriptyline	Elavil	SNRI (TCA)
Atomoxetine	Strattera	NRI
Bupropion	Wellbutrin	NDRI
Clomipramine	Anafranil	SNRI (TCA)
Duloxetine	Cymbalta	SNRI
Fluoxetine	Prozac	SSRI
Mirtazapine	Remeron	NaSSA
Paroxetine	Paxil	SSRI
Selegiline	Anipryl, L-deprenyl	MAOI
Sertraline	Zoloft	SSRI
Venlafaxine	Effexor	SNRI
Viloxazine	Vivalan	NRI

Supportive and symptomatic care are the mainstay of therapy.

MAOI = monoamine oxidase inhibitor, NaSSA = noradrenergic and specific serotonergic antidepressant, NDRI = norepinephrine-dopamine reuptake inhibitor, NRI = norepinephrine reuptake inhibitor, SNRI = serotonin-norepinephrine reuptake inhibitor, SSRI = selective serotonin reuptake inhibitor, TCA = tricyclic antidepressant



1 **One-year-old intact male bulldog presented after ingestion of the owner's Adderall (amphetamine–dextroamphetamine). The dog presented hyperthermic and tachycardic and developed seizures. It required sedation, antiepileptic medications, and β-blockers to control signs (A). Endotracheal intubation and intermittent oxygen supplementation were required because of heavy sedation. Thirty-six hours later (B), the dog was normothermic, had a normal heart rate, and no longer had seizures. Several days after presentation, it was reportedly doing well.**

recommended as an alternative therapy for tremors. Phenothiazine use remains controversial. Seizures not responsive to benzodiazepines may be treated with barbiturates. Although it is not specifically studied in toxicologic overdoses, status epilepticus not responsive to benzodiazepines and barbiturates may respond to propofol or inhalant (isoflurane) anesthesia.[3-8] Specific therapy includes serotonin antagonism with cyproheptadine.

ADHD Medications

ADHD amphetamines (**Table 3**) are stimulants that inhibit norepinephrine and dopamine reuptake in the brain. Clinical signs are neurologic (eg, agitation, shaking/trembling, circling, seizures, disorientation, coma, death), cardiovascular (eg, tachycardia, hypertension, potential reflex bradycardia), and GI (eg, vomiting, abdominal pain). Standard decontamination should be used judiciously because of possible inability to protect the airway.

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Additional treatment (**Figure 1**) is symptomatic and supportive; phenothiazines (eg, acepromazine) are the first-line agents for excessive stimulation and may also be used to treat hypertension and hyperthermia. Keeping the patient in a quiet, dark room to minimize stimulation may help. Use of benzodiazepines in patients with stimulatory signs is controversial; however, they may be used for seizures. Seizures not responsive to benzodiazepines may respond to barbiturates or propofol.[3-9] Excessive muscle fasciculations may also be treated with methocarbamol.

Table 3 **Common ADHD Medications**

Generic Name	*Common Brand Names*
Amphetamine–dextroamphetamine	Adderall
Dexmethylphenidate	Focalin
Dextroamphetamine	Dexedrine, Dextrostat
Lisdexamfetamine	Vyvanse
Methylphenidate	Ritalin, Concerta, Daytrana, Metadate

IV fluids and cooling may be necessary for patients with elevated body temperatures. Because some ADHD medications are lipophilic, IV lipid solutions may be considered; however, there are little data available on their use with these medications.

Marijuana

Marijuana (*Cannabis sativa*), legally prescribed for humans in select states, contains Δ^9-tetrahydrocannabinol (THC), which alters neurotransmitter activity. Patients typically present with altered mental status, ataxia, and dilated pupils; potential agitation, seizures, and/or coma may result in death. Additional signs can include vomiting, diarrhea, arrhythmias, tachypnea, and incontinence. In dogs, over-the-counter urinary tests are unreliable for diagnosis.

Decontamination should be avoided when altered mental status is present, and emesis is often unrewarding. Supportive care is the mainstay of treatment. Because renal elimination of THC is minimal, the benefit of diuresis is questionable; fluids may be used to treat dehydration. Benzodiazepines can be used safely for agitation, seizures, and tremors; monitoring temperature, respirations, and heart rate is essential. Most patients recover uneventfully within 24 to 96 hours; however, ingestion of large quantities may require aggressive supportive care. IV lipid infusions have been advocated anecdotally and used successfully by the author.

Vitamin D

Vitamin D_3 is available as a sole supplement, in multivitamins (which may also contain toxic levels of iron, xylitol, and vitamin A), or in rodenticides. Initial clinical signs (8–12 hours after ingestion) are vague and include lethargy, vomiting, diarrhea, and inappetence resulting in hypercalcemia and renal failure (occurring 36–48 hours after ingestion), which cause polyuria/polydipsia, weakness, hematemesis, and arrhythmias. Decontamination should be initiated immediately, as vitamin D_3 has a prolonged action and treatment can be challenging and costly if hypercalcemia develops. When decontamination is not possible or is incomplete, IV lipid emulsion may also help prevent hypercalcemia, as vitamin D is fat soluble. Ionized calcium levels should be monitored q24h for 5 to 7 days. When ionized hypercalcemia is present, therapies (eg, saline diuresis, furosemide, steroids, salmon calcitonin) may need to be combined. Pamidronate inhibits osteoclast function and has prolonged activity, making it an attractive alternative if other therapy is unavailable or ineffective. ■ cb

See **Aids & Resources**, back page, for references & suggested reading.