ETHYLENE GLYCOL TOXICOSIS IN DOGS AND CATS

Jill A. Richardson, DVM, and Sharon M. Gwaltney-Brant, DVM, PhD
ASPCA Animal Poison Control Center

Profile

DEFINITION

Poisoning with agents containing ethylene glycol (EG) (antifreeze; aircraft de-icer; solvents; hydraulic brake fluids; windshield washer fluids; agents in condensers, heat exchangers, and home solar units).[1,2] EG is also used in portable basketball goal post bases and toilets of recreational vehicles/homes in colder climes.

Systems. Gastrointestinal tract, CNS, cardiopulmonary, and renal. Vomiting usually within the first few hours; CNS depression, ataxia, weakness, tachypnea, polyuria/polydipsia occur within 1 to 6 hours. By 18 to 36 hours, acute renal failure develops.[3,5]

RELATIVE SPECIES SENSITIVITY

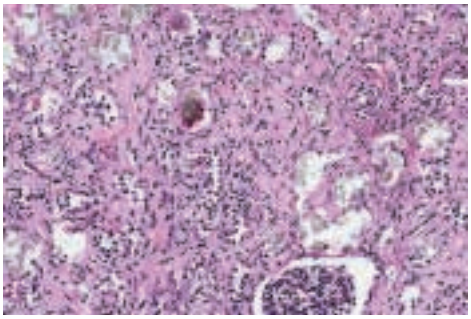
Most Sensitive: cat, rabbit, human
Moderately Sensitive: dog, cattle, pig, rodent
Least Sensitive: fish, poultry

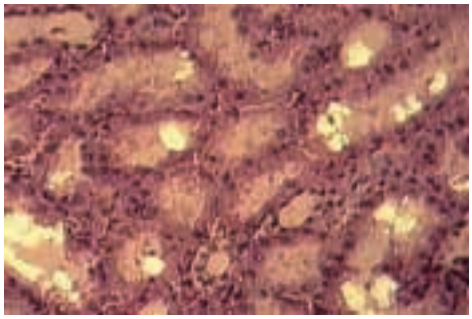
Toxic Doses. Toxic dose has not been established. Lethal dose of undiluted antifreeze (95–97%) by volume is 4.4 to 6.6 ml/kg (dogs) and 1.4 ml/kg (cats).[2,5] Most acute toxicity data based on doses causing early death (acidosis/intoxication) do not account for animals surviving initial stages that later succumb to kidney failure. Thus, any suspected exposure should be considered potential toxicosis, with steps to determine extent. **When in doubt, treat as toxic.**

SIGNS

History.

- Exposure/possible exposure
- Roaming or unsupervised animal
- Evidence of exposure (radiator leaks, chewed container)
- Vomiting, lethargy, ataxia





Calcium oxalate crystals, shown above, can be produced as ethylene glycol is metabolized.

- Fluorescence under Wood's lamp of urine, stomach contents, paws/muzzle

Physical Examination. Clinical signs categorized into 3 stages: neurologic (initial inebriation from alcohol), cardiopulmonary (severe acidosis/electrolyte disturbances), renal (direct injury from toxic metabolites created by breakdown of EG and from tubular injury from calcium oxalate crystals).[1–4,6] However, may be asymptomatic; clinical signs often change during course; and stages can overlap, be skipped, or go unobserved. Death can occur at any stage.

Stage 1—Neurologic. Typically begins ≤ 30 minutes, lasting to 12 hours.[1–4,6] Vomiting common, most likely because EG irritates gastric mucosa.[2,3] Initial ataxia, disorientation, stupor, polyuria/polydipsia.[3,4,6] These are early indicators of possible poisoning, important because early intervention is more likely to save an animal. Coma and death possible, or animal may appear to partially/fully recover. By 6 to 12 hours, neurologic status may worsen due to severe metabolic acidosis.[3,4,6] Isosthenuria in dogs by 3 hours from osmotic diuresis/serum hyperosmolality-induced polydipsia.[2,3]

Stage 2—Cardiopulmonary. Generally from 12 to 24 hours. Tachypnea, tachycardia, CNS depression, seizures, pulmonary edema may occur.[1–4,6] High anion gap and severe metabolic acidosis typical.[3,4,6] Most deaths in humans reported at this stage.[1]

Stage 3—Oliguric renal failure. As early as 12 hours, especially in cats, but generally within 24 to 72 hours.[2,3,5,6] Azotemia, depression, anorexia, vomiting, abdominal pain, and oliguria progressing to anuria.[1,3] Low urine specific gravity and glucosuria with calcium oxalate crystals possible.[3,4] Clinical pathologic abnormalities include increased osmolar gap/anion gap, hyperglycemia, hyperkalemia, decreased blood pH, hypocalcemia.[1,3] BUN and creatinine generally do not increase until ≥ 12 hours after exposure; thus, testing has little benefit.

KINETICS AND METABOLISM

EG is rapidly absorbed, although food in stomach may slow absorption.[1,3] Clinical signs may occur as early as 30 minutes; peak blood concentrations in dogs 1 to 4 hours after ingestion.[6] Plasma half-life is 3 to 5 hours; therapeutic ethanol or fomepizole may increase half-time to 12 to 72 hours.[1,3] Primarily eliminated through kidneys; about 50% excreted unchanged.[3,4]

Metabolism occurs primarily in liver, forming toxic acidic metabolites (see EG Metabolism).[1,2,5,6] Oxalic acid is directly cytotoxic to the renal tubular epithelium and induces

continues

ETHYLENE GLYCOL TOXICOSIS IN DOGS AND CATS

hypocalcemia by binding to serum calcium to form calcium oxalate crystals.[1–3,6] Crystals precipitate in renal tubules, causing mechanical injury and possibly obstructive uropathy.[2]

Dx Diagnosis

Differentials. See algorithm on page 18 for ataxia, an early, sentinel sign of EG toxicity. Diagnostic differentials for EG toxicity can be found in standard reference texts.

Causes of Increased Anion Gap.
- Decreasing unmeasured cations (hypocalcemia, hypomagnesemia, hypokalemia)[7]
- Increasing unmeasured anions (ketoacids, lactate, phosphates, sulfates, salicylates, methanol, albumin)[7]
- Most common cause (dogs and cats) is metabolic acidosis due to lactic acidosis, renal failure, or ketoacidosis[7]

Ruleouts for acute renal failure.
- Acute decompensation of chronic renal failure
- Cholecalciferol/vitamin D_3 derivatives, hypervitaminosis D, hypercalcemia
- Glomerulonephritis
- Grapes or raisins
- Heavy metal intoxication (lead, mercury, cadmium, arsenic, zinc)
- Heatstroke
- Hemoglobinuria, myoglobinuria
- Hypoadrenocorticism
- Hypovolemia
- Leptospirosis
- Nephrotoxic antibiotics
- NSAID overdose
- Oxalic acid ingestion
- Plant ingestion (*Lilium* species, *Hemerocallis* species [in cats only], rhubarb leaves, and *Oxalis* species)
- Septic shock
- Crotalid snake bite
- Urinary obstruction

Ruleouts for metabolic acidosis.
- Acute renal failure
- Aspirin or NSAID overdose
- Diabetes mellitus
- Ethanol, methanol, other short-chain alcohols
- Metaldehyde toxicosis

BLOOD AND URINE ANALYSIS

Early diagnosis/treatment essential. Diagnosis based on history, clinical signs, laboratory testing. Peak EG levels reached ≤ 1 to 4 hours, but tests can be done from 0.5 to 12 hours.[3] One kit is available for veterinary use (EGT Kit—PRN Pharmacal). Labeled for dogs with levels > 50 mg/dl, this kit can be invaluable in determining if treatment is warranted.[3,4] False-positive results can occur (presence of propylene glycol in some activated charcoal solutions and injection solutions such as pentobarbital and diazepam, other glycols, or formaldehyde in the circulation).[3,4,6] Blood should be taken before administering any such solutions/agents. Products causing false positives are metaldehyde, glycerin/glycerol, or diethylene glycol. Other alcohols (e.g., ethanol, methanol, or isopropanol) do *not* interfere.[3]

Cats are more sensitive to EG than dogs, and kit may not diagnose toxicosis. Positive results are significant, but negative results do not rule out toxicosis. Some human laboratories run quantitative EG analysis. Any detectable EG warrants treatment.

Other Diagnostic Procedures.
- Measure anion gap (>25 mEq/L) or serum osmolality (> 20 mOsm/kg).[2–4,6]
- Crystalluria (not conclusive)[2]
- Calcium oxalate crystals in urine can be seen 6 hours after ingestion; may be octahedral (envelope-like), prismatic (spindles, hippurate-like), or dumbbell shaped.[1]
- Examine urine under Wood's lamp; will show fluorescein dye up to 6 hours.[3]

Diagnosing Toxicosis.
- Increased osmolar gap (>20 mOsm/kg)
- Decreased blood pH
- Increased anionic gap (>25mEq/L)
- Toxin in serum or urine
- Hypocalcemia
- Early-phase ataxia (1–3 hours)
- Calcium oxalate crystalluria
- Fluorescence of urine or vomitus on Wood's lamp

Postmortem Findings.
Renal: Proximal tubular degeneration/necrosis, calcium oxalate deposition; calcium oxalate crystals in intestinal mucosa, liver, heart, brain[3,4]
CNS: Cerebral edema, multifocal hemorrhage, inflammatory cell infiltration[3,4]

EG METABOLISM
1. EG oxidation to glycoaldehyde via alcohol dehydrogenase.
2. Glycoaldehyde oxidation to glycolic acid via mitochondrial aldehyde dehydrogenase.
3. Glycolic acid oxidation to glyoxylic acid, the most toxic metabolite. Short half-life of glycoxylic acid prevents accumlation of toxic concentrations.[1]
4. Glyoxylic acid oxidation through several pathways to produce oxalic acid, glycine, formic acid, hippurate, benzoic acid, and other compounds.
5. Oxalic acid binds to serum calcium and forms calcium oxalate crystals.

Tx Treatment

Must be timely and aggressive. **Failure to initiate in first several hours may result in irreversible renal damage/death.** Goals are to stabilize, prevent absorption, interfere with toxin metabolism, provide supportive care.

Induction of emesis helpful only < 1 hour. Feeding dry bread before emesis may increase success. Emesis contraindicated with hyperactivity, tremor, seizures: consider gastric lavage.[8] Effective emetic is 1 teaspoon 3% hydrogen peroxide per 5 lb, not to exceed 3 tablespoons.[8] Usually occurs in minutes; dose can be repeated once if initially unsuccessful. Another option is apomorphine hydrochloride, administered topically to eye or parenterally at 0.03 mg/kg IV or 0.04 mg/kg IM in dogs; 0.04 mg/kg IV or 0.08 mg IM or SC in cats.[8] CNS/respiratory depression, ataxia, excitement, protracted vomiting can occur, especially after IV administration.[8]

Activated charcoal can be given (1–3 g/kg, 1–3 hours after ingestion). Effectiveness is controversial—aliphatic alcohols may not be well adsorbed by charcoal.[1,3] Take samples for EG analysis before activated charcoal to prevent false positives resulting from detection of glycols in these compounds.

Altering Kinetics. IV fomepizole and ethanol have been successful. Compounds aim to delay/prevent breakdown of toxin to more toxic metabolites, allowing excretion of unchanged parent compound in urine.

Fomepizole inhibits alcohol dehydrogenase; considered preferred treatment for dogs[3,4,16] but may not be effective in cats.[3,5,9,10] Unlike ethanol, does not cause hyperosmolality, metabolic acidosis, CNS depression [3,9,11,16]

Animal-safe photo courtesy Animal Poison Control Center

Administer every 12 hours for 36 hours. Dosing is 20 mg/kg slow IV over 15 to 30 minutes, then 15 mg/kg slow IV at 12 and 24 hours, then 5 mg/kg at 36 hours.[3,9,10]

Ethanol competes with EG as substrate for alcohol dehydrogenase; can be used in dogs and cats.[3,4,6,10] Inexpensive and readily available; has serious drawbacks (worsening of metabolic acidosis and CNS depression), making evaluation of degree of toxicosis difficult.[11,13] Ethanol treatments are time-intensive and require constant monitoring. Ideally, administer 8.6 ml/kg 7% ethanol solution bolus, maintain at 100 mg/kg/hr up to 200 mg/kg/hr constant-rate infusion.[10] Or, make a 20% ethanol solution (dogs: 5.5 ml/kg IV every 4 hours for 5 treatments, then every 6 hours for 4 treatments; cats: 5.0 ml/kg IV every 6 hours for 5 treatments, then every 8 hours for 4 treatments.)[4,5,10]

For dogs, a negative EG test means treatment with fomepizole or ethanol may be discontinued; however, fluid therapy and supportive care should continue until animal fully recovers.

Supportive Care. Preventing further kidney damage/maintaining fluid, electrolyte, acid–base balance are crucial. Fluid therapy is foundation of acute renal failure treatment, comprising fluid diuresis with isotonic alkalinizing crystalloid solution (e.g., lactated Ringer's) twice maintenance rate.[10,12] Goals are to correct fluid and electrolyte imbalances, improve renal blood flow, initiate diuresis. Monitor patient to avoid volume overload and possible pulmonary edema. Oliguria necessitates attempts to increase urine output (furosemide, 1 mg/kg/hr for 4 hours then 3 to 8 µg/kg/min to maintain urine flow).[10] Dopamine may increase urine output (dogs); administer at 2 to 3 µg/kg/min.[11] Consider peritoneal dialysis in oliguric/anuric animals. Sodium bicarbonate can be used for acidosis (i.e., blood pH < 7.10 to 7.15 or total CO_2 < 10-12 mEq/L).[3,5,10,11] Continue treatment until animal is clinically normal, with at least 24 hours' normal renal function, acid-base parameters.

Precautions.
Overhydration: Electrolyte imbalances, volume overload, possibly pulmonary edema
Overuse of sodium bicarbonate: Ionized calcium deficits, CSF acidosis, cerebral edema
Ethanol: Worsening of metabolic acidosis, CNS depression

ALTERNATIVE THERAPY

High doses of fomepizole may be safe and effective in cats if therapy is initiated < 3 hours after ingestion; > 4 hours, mortality rate is 100%.[17] Initiating alcohol therapy at home in cases of time delay in obtaining veterinary help has been reported.[10] In these cases, 2.25 ml/kg PO 40% alcohol (e.g., vodka, rum) can be administered orally.[10]

Hemodialysis considered superior to peritoneal dialysis in eliminating EG/metabolites in humans but limited availability in veterinary medicine.[1] Pyridoxine and thiamine—cofactors for EG metabolism—recommended in human literature adjunctively.[2,4]

continues

of Periodontology from the University of Pennsylvania. He was instrumental in bringing dentistry to the forefront of veterinary medicine. Dr. Emily is director of exotic animal dentistry at the Denver Zoological Gardens and is author and co-author of 3 veterinary textbooks and multiple dental publications. The "Peter Emily Awards" are presented annually to outstanding contributors to veterinary dentistry by the American Veterinary Dental College.

Kirk Gelatt, VMD
Distinguished Professor
University of Florida College of Veterinary Medicine
Dr. Gelatt's more than 3 decades of academia has included teaching more than 2,500 veterinary students and the training of 36 residents and postdoctoral fellows in comparative ophthalmology. He has presented more than 290 professional talks both nationally and internationally and has published a number of articles, abstracts, chapters, and books. Dr. Gelatt's research interests have concentrated on canine glaucomas, inherited cataracts in the dog, clinical pharmacology of drugs that change intraocular pressure, and ophthalmic surgery.

Gregory F. Grauer, DVM, MS, Diplomate ACVIM (Internal Medicine)
Professor/Head, Department of Clinical Sciences
Kansas State University College of Veterinary Medicine
Dr. Grauer received his DVM from Iowa State University and then completed his postgraduate training at Colorado State University (CSU). He was a faculty member at University of Wisconsin and CSU prior to his current position at K-State. Dr. Grauer's clinical and research interests involve the small animal urinary system. He has published and lectured widely in the US and abroad on this topic.

Deena G. Gregory, DVM, MS
Pharmacy Coordinator
Oklahoma State University Veterinary Teaching Hospital
Dr. Gregory earned her DVM and MS in veterinary pathology from Oklahoma State. She completed a residency in veterinary toxicology and was in small animal practice for 8 years following graduation. Dr. Gregory has served in her current position for 6 years and before that as research assistant for the biochemistry department.

Sharon Gwaltney-Brant, DVM, PhD, Diplomate ABVT, ABT
Senior Toxicologist/Manager of Veterinary Toxicology Training
Animal Poison Control Center
Dr. Gwaltney-Brant is an on-line consultant for the Veterinary Information Network and has authored or co-authored several peer-review articles and book chapters on toxicology and pathology. She also enjoys participating in draft work and water work with her Newfoundlands, and is a member of the Research Advisory Committee of the Newfoundland Club of America.

John L. Harvey, DVM, PhD, Diplomate ACVP
Professor/Chair, Department of Physiological Sciences
University of Florida College of Veterinary Medicine
Dr. Harvey received his DVM from Kansas State University and his PhD from the University of California-Davis. In addition to his faculty position, he is also chief of the clinical pathology service at the teaching hospital. Dr. Harvey is past president of the American Society for Veterinary Clinical Pathology and currently serves on the editorial board of *Comparative Haematology International*. He speaks widely and contributes to numerous publications.

Heidi L. Hoefer, DVM
West Hills Animal Hospital
Huntington, NY
Dr. Hoefer is a graduate of Ross University School of Veterinary Medicine. She completed an internship in small animal medicine and surgery at The Animal Medical Center in New York and continued there, first, as a resident in avian and exotic medicine then in a staff appointment for 9 years. She has written widely and lectured nationally and internationally. Her clinical practice is limited to birds, reptiles, and small mammals.

Gail A. Kunkle, DVM, Diplomate ACVD
Professor/Director, Blanche Saunders Dermatology Laboratory
University of Florida College of Veterinary Medicine
In addition to serving as an officer and member of many professional associations, Dr. Kunkle is known for her education of students and residents, as well as continuing education for practitioners in her specialty of dermatology. Dr. Kunkle's research interests include feline and canine allergy, infectious diseases, and ectoparasites. She is the recipient of a number of awards, including the Woman Veterinarian of the Year and the ACVD Award for Excellence.

Michael S. Leib, DVM, MS, Diplomate ACVIM
Professor/Staff Internist/Specialist, Gastroenterology and Endoscopy
Virginia Maryland Regional College of Veterinary Medicine
Dr. Leib received his DVM from the University of Georgia and his MS at Colorado State University. He has been at Virginia Maryland Regional College since 1983, where he was chief of small animal medicine from 1985-1990. Dr. Leib has received several teaching awards; been investigator on more than 25 funded research projects; authored numerous publications, including a textbook; and served in various editorial and continuing education capacities.

Heidi B. Lobprise, DVM, Diplomate AVDC
Dallas Dental Service Animal Clinic
Dallas, TX
Dr. Lobprise graduated from Texas A&M University. She then completed a residency in veterinary dentistry. Dr. Lobprise has published many articles and co-authored two books, including *The Veterinarian's Companion to Common Dental Procedures.* She has lectured internationally and is past president of the Texas Academy of Veterinary Practitioners and American Veterinary Dental Society.

Denny Meyer, DVM, Diplomate ACVIM & ACVP
Service Chief of Small Animal Medicine/Clinical Pathology
University of Florida College of Veterinary Medicine
Dr. Meyer has contributed extensively to clinical, research, and veterianry teaching programs at the University of California-Davis, University of Florida-Gainesville, and Colorado State University. He has spent 12 years in the development of novel drugs for human indications and is recognized nationally and internationally for his expertise in clinical pathology, diagnostic cytology, and hepatic histopathology. He has co-authored 3 texts and numerous articles and book chapters regarding those areas.

Paula F. Moon-Massat, DVM, Diplomate ACVA
Independent Contractor
New England Veterinary Anesthesia Services
Dr. Moon-Massat's current responsibilities at New England Veterinary Anesthesia Services include technical and medical writing as well as providing clinical anesthesia for clients such as the University of California-Davis. Additionally, she contributes to continuing education seminars and wet laboratories, as well as editing and reviewing for NAVC and the *Journal of Veterinary Anesthesia and Analgesia.* Her current research interests include improving the anesthetic management of critically ill patients.

Antony S. Moore, MVSc, Diplomate ACVIM (Oncology)
Professor/Head, Section of Oncology and Harrington Oncology Program
Tufts University School of Veterinary Medicine
Dr. Moore has been affiliated with Tufts University since 1988. Dr. Moore received his veterinary degree from the University of Sydney and completed his residency in veterinary oncology at the University of California-Davis. He was recipient of the MSD AgVet Award for Creativity in Teaching in 1996 and the Tufts University Outstanding Faculty Award in 1997. He is co-author of two books, *Managing the Veterinary Cancer Patient* and *Feline Oncology.*

Jacqueline Neilson, DVM, Diplomate ACVB
Owner, Animal Behavior Clinic
Portland, Oregon
Dr. Neilson graduated from the University of Florida and after 2 years in private practice was selected as first Friskies PetCare Companion Animal Behavior Resident at UC Davis, where she completed her residency program and board certification. Dr. Neilson then returned to Portland and opened a behavior referral practice. She also acts as an industry consultant, a visiting instructor at the University of Florida, and lectures across the country about animal behavior.

Karen L. Overall, MA, VMD, PhD, Diplomate ACVB & ABS Certified
Research Associate
Psychiatry Department
University of Pennsylvania School of Medicine
Dr. Overall has been a regular columnist for both *Canine* and *Feline Practice* journals and currently writes a bimonthly column for *DVM Newsmagazine.* Dr. Overall's research interests focus on development of genetic and behavioral animal models for human psychiatric illness,

Our review board is growing. Look for additional introductions in the future.

particularly those involving anxiety, panic, and aggression. She previously was at the university's School of Veterinary Medicine, where she ran the Behavior Clinic for 12 years. Dr. Overall is the author of two books on small animal behavior.

Dale Paccamonti, DVM, MS, Diplomate ACT
Professor/Chief, Section of Theriogenology and Preventative Medicine
Louisiana State University School of Veterinary Medicine
Dr. Paccamonti's primary research interests are in equine theriogenology, but he is also actively involved in research in canine reproduction. He teaches third- and fourth-year veterinary students at LSU's Veterinary Teaching Hospital, where he is responsible for clinical theriogenology cases in all species. Dr. Paccamonti is author or co-author of numerous articles, book chapters, and abstracts and is currently president of the American College of Theriogenologists.

Karen Rosenthal, DVM, MA, Diplomate ABVP
Director of Special Species Medicine
University of Pennsylvania
Following her internship at The Animal Medical Center in New York, Dr. Rosenthal stayed on, completing a residency in avian and exotic animal medicine and surgery. Dr. Rosenthal continued as a staff member for 5 years. She then became the national director of avian and exotic animal services for Antech Diagnostics and, concurrently, was chief veterinarian for the Long Island Reptile Museum. She started the Special Species Service at the University of Pennsylvania in 1999. Her special interests include endocrine diseases of ferrets and imaging techniques.

Joseph Taboada, DVM, Diplomate ACVIM
Professor/Associate Dean
Louisiana State University School of Veterinary Medicine
Before coming to LSU, Dr. Taboada was a member of the faculty and director of the Small Animal ICU at the University of Florida. During his years at LSU he has won 11 teaching awards; has spoken at over 100 meetings; and has published over 30 chapters and 50 articles. Dr. Taboada's special interests are feline medicine, hepatology, fungal infectious diseases, and endoscopy. He is past president of the specialty of Internal Medicine for ACVIM and current president-elect of ACVIM.

Donald E. Thrall, DVM, PhD
Professor
North Carolina State University College of Veterinary Medicine
Before coming to NCSU, Dr. Thrall held faculty positions at University of Georgia and University of Pennsylvania. His primary imaging interests are diagnostic radiographic and computed tomographic imaging, particularly of tumors, and experimental and clinical radiation oncology. His current research interest is hyperthermia as an adjunct to cancer therapy. He has authored or co-authored over 200 publications and is editor of *Veterinary Radiology & Ultrasound,* as well as the *Textbook of Veterinary Diagnostic Radiology.*

ETHYLENE GLYCOL TOXICOSIS IN DOGS AND CATS

RELATIVE COST OF TREATMENT*

Uncomplicated (mild or no clinical signs, no evidence of renal injury): $$$-$$$$
Includes physical examination, decontamination, EG test; baseline clinical laboratory values, ethanol or fomepizole therapy (comparable cost, $$-$$$, with added nursing/acid-base monitoring and correction and hospitalization for ethanol treatment), additional serum chemistry values through treatment, and final EG test.

Complicated (severe clinical signs, acidosis +/- renal injury): $$$$-$$$$$
Includes physical examination, EG test, correction of emergent signs (e.g. seizures, acidosis), IV fluid administration, baseline clinical laboratory values, ethanol or fomepizole therapy (comparable cost, $$-$$$ with added nursing/acid-base monitoring and correction with ethanol treatment), additional serum chemistry values through treatment, hospitalization, treatment of acute renal failure ($$-$$$$$), and final EG test.

* $= <$100, $$ = $100-250, $$$ = $250-500, $$$$ = $500-1000, $$$$$ = >$1000 (Actual costs will have regional variations.)

Follow-Up

Patient Monitoring.

- BUN
- Creatinine
- Calcium
- Acid–base balance
- Urine specific gravity
- Urine production

Prognosis and Course. Depends on degree of exposure, time before treatment, aggressiveness of treatment. Good if therapy starts < 8 hours and no azotemia.[4] Feline mortality rates may be as high as 97%; canine rates, 59% to 70%.[3,4] Oliguria/anuria indicates grave prognosis. Animals may fully recover or have residual renal insufficiency requiring lifetime maintenance.

Tx ... at a glance

TIME OF EXPOSURE

- < 1 hour: Induce emesis
- 1–3 hours: 1-3 g/kg activated charcoal

THEN

- Dogs: fomepizole—20 mg/kg slow IV over 15-30 mins every 12 hrs for 36 hrs; then 15 mg/kg slow IV at 12 and 24 hrs; then 5 mg/kg at 36 hrs. Tx of choice

OR

- Cats or dogs: ethanol—8.6 ml/kg 7% ethanol solution bolus, maintain at 100 mg/kg/hr up to 200 mg/kg/hr constant-rate infusion

OR

- 20% ethanol solution (homemade)
  - Dogs: 5.5 ml/kg IV every 4 hrs, 5X; then every 6 hrs, 4X
  - Cats: 5.0 ml/kg IV every 6 hrs, 5X; then every 8 hrs, 4X

PLUS

- Fluid therapy (diuresis with isotonic alkalinizing crystalloid, e.g., lactated Ringer's), twice maintenance rate

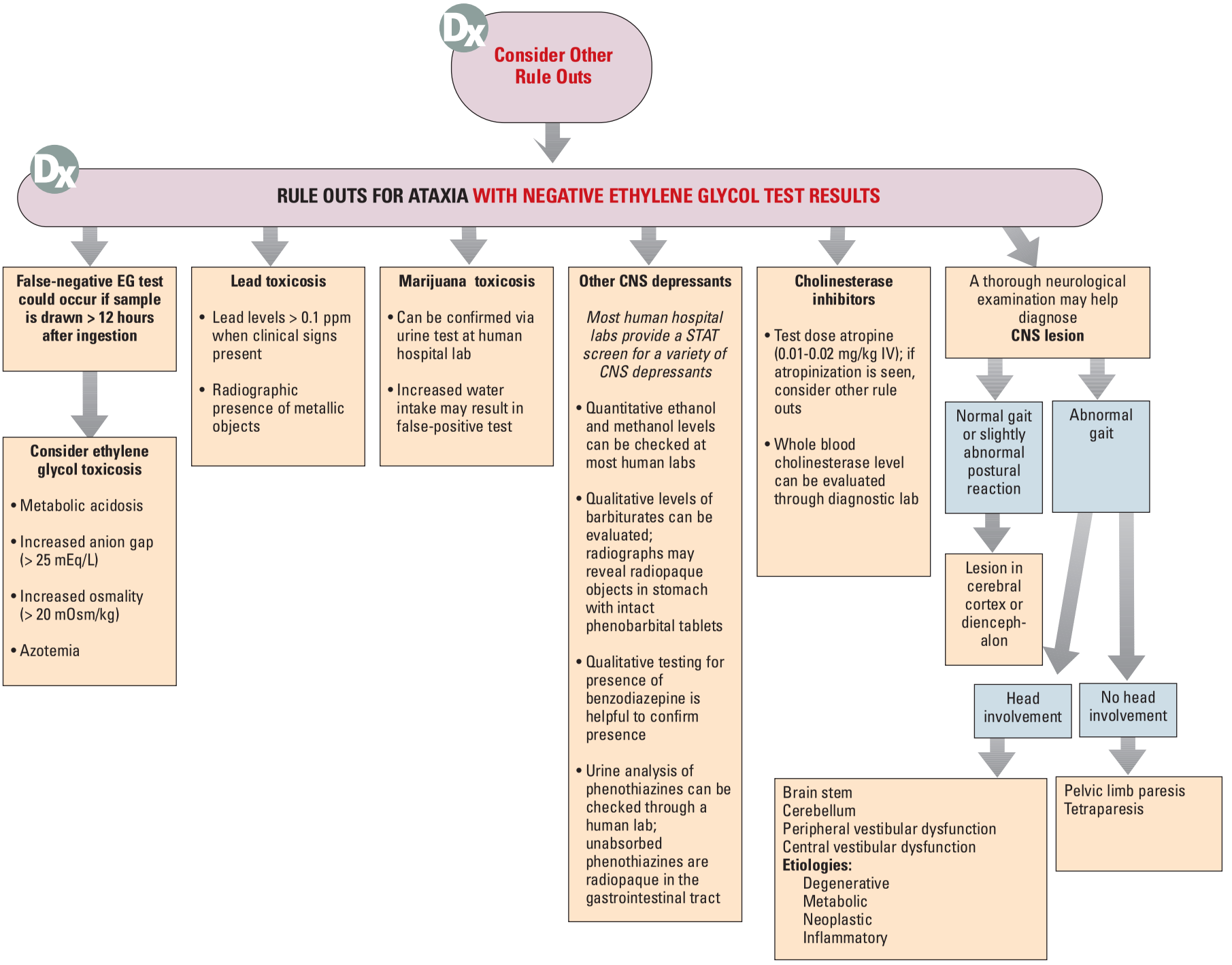
Advice for Owners. The veterinary community can help spread the word about antifreeze safety. Motorists can help prevent accidental EG ingestion. Among considerations are using antifreeze that contains propylene glycol, which is less toxic than EG.

FUTURE CONSIDERATIONS

Researchers at Colorado State University are investigating use of fomepizole in EG-exposed cats.

See Aids & Resources, back page, for references, further reading, and contacts.

EVALUATING ATAXIA IN SUSPECTED ETHYLENE GLYCOL TOXICITY


Dx
Consider Other Rule Outs
Dx
RULE OUTS FOR ATAXIA WITH NEGATIVE ETHYLENE GLYCOL TEST RESULTS
False-negative EG test could occur if sample is drawn > 12 hours after ingestion
Consider ethylene glycol toxicosis
• Metabolic acidosis
• Increased anion gap (> 25 mEq/L)
• Increased osmality (> 20 mOsm/kg)
• Azotemia
Lead toxicosis
• Lead levels > 0.1 ppm when clinical signs present
• Radiographic presence of metallic objects
Marijuana toxicosis
• Can be confirmed via urine test at human hospital lab
• Increased water intake may result in false-positive test
Other CNS depressants
Most human hospital labs provide a STAT screen for a variety of CNS depressants
• Quantitative ethanol and methanol levels can be checked at most human labs
• Qualitative levels of barbiturates can be evaluated; radiographs may reveal radiopaque objects in stomach with intact phenobarbital tablets
• Qualitative testing for presence of benzodiazepine is helpful to confirm presence
• Urine analysis of phenothiazines can be checked through a human lab; unabsorbed phenothiazines are radiopaque in the gastrointestinal tract
Cholinesterase inhibitors
• Test dose atropine (0.01-0.02 mg/kg IV); if atropinization is seen, consider other rule outs
• Whole blood cholinesterase level can be evaluated through diagnostic lab
A thorough neurological examination may help diagnose CNS lesion
Normal gait or slightly abnormal postural reaction
Abnormal gait
Lesion in cerebral cortex or dienceph-alon
Head involvement
No head involvement
Brain stem
Cerebellum
Peripheral vestibular dysfunction
Central vestibular dysfunction
Etiologies:
Degenerative
Metabolic
Neoplastic
Inflammatory
Pelvic limb paresis
Tetraparesis